Effects of Inspiratory Muscle Training on Obstructive Sleep Apnea: A Systematic Review and Meta-analysis

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Abstract

Rationale: Inspiratory muscle training (IMT) is a relatively new intervention for obstructive sleep apnea (OSA) with the aim of improving respiratory muscle strength and mitigating OSA-related symptoms. However, its effects on key clinical endpoints remains unclear.

Objectives: To evaluate the effects of IMT on OSA severity, sleep quality, respiratory function, and cardiovascular outcomes.

Methods: We systematically searched PubMed, EMBASE, CENTRAL, and Web of Science for randomized control trials (RCTs) assessing IMT in adults with OSA. Studies assessed IMT versus control/placebo with outcomes, including the apnea-hypopnea index (AHI), Pittsburgh Sleep Quality Index (PSQI), Epworth Sleepiness Scale (ESS), maximal inspiratory pressure (MIP), lowest oxygen saturation ($LSaO_2$), blood pressure (BP), and body mass index (BMI).

Results: Ten RCTs (166 IMT vs. 157 control participants) found significant improvements in OSA with IMT across multiple domains. The intergroup difference in mean BMI reduction (mean difference [MD], -1.48; 95% credible interval [CI], -2.39 to -0.57), mean PSQI gain (-3.15; -3.69 to -2.62), mean $LSaO_2$ gain (2.86; 1.01 to 4.71), mean ESS reduction (-3.18; -4.50 to -1.87), mean MIP gain (25.54; 11.09 to 40.00), the percentage of predicted values for forced vital capacity (FVC) (FVC%) predicted gain (17.20; 9.53 to 24.87), and mean systolic blood pressure (SBP) reduction (-6.63; -13.26 to -0.00) indicated the benefit of IMT over the control therapy. However, there was no significant improvement in AHI (1.00; -2.57 to 4.56). Heterogeneity primarily stemmed from differences in intervention protocols and baseline disease severity among the patients.

Conclusions: IMT resulted in clinically meaningful symptomatic benefits across OSA phenotypes by enhancing respiratory strength, sleep quality, and cardiovascular health. However, the lack of

significant improvement in AHI suggests that IMT may not impact primary pathophysiological markers of OSA. Thus, IMT may be an adjunctive intervention, rather than a primary therapy, for reducing event frequency in OSA.

Introduction

Obstructive sleep apnea (OSA) is a respiratory sleep disorder characterized by loud snoring, disturbed sleep architecture, and excessive daytime sleepiness. Untreated OSA may lead to serious systemic complications including neurocognitive impairment, type 2 diabetes mellitus, drug-resistant hypertension (HT), and cardiovascular diseases (1-4). According to the latest data, nearly one billion adults aged 30-69 years are affected by OSA worldwide (5).

Continuous positive airway pressure (CPAP) is considered the gold standard treatment for OSA (6). Nevertheless, long-term CPAP adherence is poor with approximately 30-60% of patients discontinuing therapy within the first year due to interface discomfort, claustrophobia, and lifestyle restrictions (7,8). Therefore, in recent years, practical and well-tolerated therapies, such as inspiratory muscle training (IMT), have been increasingly explored (9).

IMT involves training specific muscles, including the diaphragm and external intercostals, to improve inspiratory muscle strength and endurance (10). In the context of OSA, inspiratory muscle dysfunction, particularly diaphragmatic dysfunction, may contribute to disease burden (11). Specifically, ineffective respiratory mechanics and increased breathing work in collapsed airways in patients with OSA lead to muscle fatigue, heightened fluctuations in intrathoracic pressure, and heightened sympathetic nervous system activity. IMT may alleviate these symptoms. Although the benefits of IMT in other conditions, such as facilitating weaning from mechanical ventilation (12) and improving the prognosis of chronic obstructive pulmonary disease (COPD) (13), have been well established, its application in OSA remains underexplored. IMT can reduce the apnea-

hypopnea index (AHI) by 25-40%, but existing trials exhibited marked heterogeneity in protocol design, outcome measures, and population characteristics (14-23). Furthermore, comprehensive studies evaluating the efficacy of IMT across all OSA characteristics, including the respiratory event frequency, sleep quality, inspiratory muscle strength, and cardiovascular parameters, are lacking.

Therefore, this meta-analysis aimed to address the following question: in adults with OSA, what is the efficacy of standalone IMT compared to sham training or no intervention on AHI and key secondary outcomes, including sleep quality, daytime sleepiness, respiratory muscle strength, pulmonary function, nocturnal oxygenation, and cardiovascular parameters? In this context, 'standalone' refers to the use of IMT as an independent therapeutic intervention with no other treatments involved. This approach allowed for assessment of the specific contribution of IMT in improving the severity of OSA and its associated manifestations.

Methods

This study followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) reporting guidelines (24). This study was prospectively registered in the International Prospective Registry of Systematic Reviews (PROSPERO) database (www.crd.york.ac.uk/prospero/) on October 10, 2022 (CRD42022366263). This systematic review and meta-analysis did not use individualized patient data, therefore institutional review board approval was not required.

Literature Search Strategy

Two independent reviewers (D.W. and C.Z.) systematically searched four electronic databases - PubMed, EMBASE, CENTRAL, and Web of Science- from study inception to November 12,

2025. Comprehensive details of the search strategies, including all keywords and syntaxes used for each database, are reported in the Supplementary Methods (eMethods).

Study Selection Criteria

Eligible studies were randomized controlled trials (RCTs) that met the following PICO-based criteria: Participants (P) were adult patients (≥18 years) with suspected or confirmed OSA, defined by an AHI of ≥5 events/h as identified by polysomnography (PSG) and in accordance with current American Academy of Sleep Medicine (AASM) recommendations; Intervention (I) consisted of standalone IMT (any protocol intensity/duration); Comparator (C) was sham IMT or no intervention; Outcomes (O) included the primary outcome of AHI, and secondary outcomes comprising body mass index (BMI), neck circumference (NC), Pittsburgh Sleep Quality Index (PSQI), sleep efficiency (SE), lowest oxygen saturation ($LSaO_2$), Epworth Sleepiness Scale (ESS), maximal inspiratory pressure (MIP), maximum expiratory pressure (MEP), forced vital capacity (FVC), the ratio of forced expiratory volume in 1 s to forced vital capacity (FEV_1/FVC), forced expiratory volume in one second (FEV_1)%, percentage of predicted FVC (FVC%), systolic blood pressure (SBP), diastolic blood pressure (DBP), heart rate (HR), and plasma norepinephrine (PNE). We excluded studies that combined IMT with other therapies, focused on any sleep-breathing disorder other than OSA, were review articles, letters to the editor, or trials involving non-IMT devices or interventions.

Data Extraction

Two authors (D.W. and C.Z.) evaluated the titles and abstracts of the literature search results and independently selected studies based on the above inclusion criteria. For each study, the year of publication, authors, study design, sample size, participant age, sex distribution, and baseline

characteristics were recorded. Discrepancies were resolved through consensus discussions with a third investigator.

Quality Assessment and Risk of Bias

The risk of bias was independently assessed by two investigators (Wu, D. and Zuo, C.) using the Cochrane Risk of Bias tool (considering selection, performance, detection, reporting, attrition, and other biases). Each domain was rated as having a "high," "moderate," or "unclear" risk of bias. The overall risk of bias in each study was labeled as the highest risk in the first five domains.

Data Synthesis and Statistical Analysis

Meta-analysis was performed using the Cochrane Review Manager (RevMan), version 5.4.1 (The Cochrane Collaboration). The data of participants receiving IMT and controls were pooled and analyzed based on the mean difference (MD) with a 95% confidence interval (CI). For studies that reported data as medians or interquartile ranges, the corresponding means and standard deviations were derived using the conversion method described by Hozo et al. Heterogeneity was assessed using the I^2 statistic (with $I^2 > 50\%$ indicating substantial heterogeneity) and Cochran's Q test ($p < 0.100$ indicating statistically significant heterogeneity). A fixed-effects model was applied when heterogeneity was not significant ($I^2 > 50\%$ and $p < 0.100$), whereas a random-effects model was used for significant heterogenic data ($I^2 > 50\%$ or $p < 0.100$). For substantial heterogeneity, the potential causes were explored using prespecified subgroup analyses. Additionally, correlation analyses of changes in MIP and clinical outcomes were conducted using Spearman's rank correlation in the GraphPad Prism software.

Results

Literature Search

The screening procedure is illustrated in Figure 1. In total, 391 references were retrieved through electronic database search. After removing duplicate studies, 264 references were retained. Thirty articles were excluded after screening the title and abstract. After reviewing the full texts, 19 articles were retrieved and assessed for eligibility. Among these, five non-RCTs, one study with high heterogeneity, one with non-normally distributed data, and two with erroneous interventions were excluded. Ultimately, 10 studies met the inclusion and exclusion criteria and were included in the meta-analysis (14-23).

Quality Assessment and Risk of Bias

The overall bias judgments are summarized in Supplementary Figures S1 and S2, which provide detailed risk-of-bias assessments for each study across all Cochrane domains. Regarding the risk of selection bias, all included studies in random sequence generation were at low risk (14-23). Six of the studies on allocation concealment were at low risk (14,16-18,21,22), whereas the risk of the remaining studies was unclear, as they only mentioned randomization without clearly describing their method of allocation concealment (15,19,20,23). Among these, seven were at low risk of performance bias (14,16,17,20-23). Regarding detection bias, five studies had low risks and adopted blinding of outcome assessment (14,16,19,21,22). In addition, four studies were at low risk of attrition bias (15,17,19,21) while six studies were at high risk and adopted incomplete outcome data (14,16,18,21-23). Moreover, most studies had a low risk of reporting bias (14,16-22), and only two studies had unclear risks of selective reporting (15,23). Finally, eight studies were at a low risk (14,16-22), one was unclear (23), and another was at a high risk and adopted another bias (15).

Study Characteristics

The characteristics of the enrolled studies are summarized in Table 1, and their baseline characteristics are presented in Table 2.

Four studies were conducted in Brazil (14,16,17,21), two in the United States (22,23), one in France (18), one in Turkey (15), one in Taiwan (19), and one in Egypt (20). All the studies were published after 2016. All the studies were RCTs (14-23) and had a group that used IMT as the intervention. Two studies used loads of 30% of MIP (15,19), one study used loads of 40% of MIP (14), one study used loads of 50% to 60% of MIP (16), one study used loads of 50% to 75% of MIP (21), two studies used loads of 70% of MIP (17,18), and three studies used loads of up to 75% of MIP (20,22,23). Regarding the intervention duration, one study performed IMT for 1 h (17), three studies performed IMT for 6 weeks (18,22,23), one study performed IMT for 8 weeks (21), and five studies performed IMT for 12 weeks (14-16,19,20).

Evaluation of Efficacy Outcomes

The findings of the meta-analyses based on the 10 included studies are presented as forest plots (Figure S3). Quantitative synthesis was performed for the following outcomes: the primary outcome AHI and secondary outcomes BMI, NC, PSQI, SE, $LSaO_2$, ESS, MIP, MEP, FVC, FEV1/FVC, FEV1%, FVC%, SBP, DBP, HR, and PNE. These outcomes included the mean difference and 95% CI for each study as well as the pooled estimate, as shown in Supplementary Figure S3.

Four studies involving 134 participants reported the effects of IMT on BMI (14,18,19,23). The heterogeneity test showed low heterogeneity (I^2 = 0%, p = 0.900), therefore a fixed-effects model was used. The synthesized data indicated that the IMT groups had significantly lower BMIs than the control groups (MD, -1.48; 95% credible interval [CI], -2.39 to -0.57; Figure S3, BMI). Six studies involving 177 participants reported the effects of IMT on AHI (14,15,18,19,21,23).

The heterogeneity test showed low heterogeneity (I^2 = 0%, p = 0.69), therefore, a fixed-effects model was used. The results showed no differences in AHI changes between the IMT and placebo groups (MD, 1.00; 95% CI, -2.57 to 4.56; Figure S3, AHI). Seven studies, with a total of 193 participants, reported the effects of IMT on PSQI outcomes (14-16,18,19,21,23). The heterogeneity test showed low heterogeneity (I^2 = 38%, p = 0.14), therefore, a fixed-effects model was used. The synthesized data indicated that the PSQI was significantly increased in the IMT group compared with that in the placebo group (MD, -3.15; 95% CI, -3.69 to -2.62; Figure S3, PSQI). Three studies involving 91 participants reported the effects of IMT on the $LSaO_2$ (18,19,23). The heterogeneity test showed low heterogeneity (I^2 = 0%, p = 0.910), therefore a fixed-effects model was used. The synthesized data indicated that the $LSaO_2$ was significantly increased in the IMT group compared with that in the control group (MD, 2.86; 95% CI, 1.01 to 4.71; Figure S3, $LSaO_2$). Six studies, totaling 169 participants, reported the effects of IMT on ESS outcomes (14-16,18,19,21). The heterogeneity test showed low heterogeneity (I^2 = 27%, p = 0.230), therefore a fixed-effects model was used. The synthesized data indicated that the ESS was significantly reduced in the IMT group compared with that in the placebo group (MD, -3.18; 95% CI, -4.50 to -1.87; Figure S3, ESS). Eight studies, totaling 251 participants, reported the effects of IMT on MIP (14-16,18,20-23). The heterogeneity test showed substantial heterogeneity (I^2 = 92%, p < 0.00001), therefore a random effects model was used. The synthesized data indicated that the MIP was significantly enhanced in the IMT group compared with that in the control group (MD, 25.54; 95% CI, 11.09 to 40.00; Figure S3, MIP). Five studies involving 160 participants reported the effects of IMT on SBP (16,17,20,22,23). The heterogeneity test showed substantial heterogeneity (I^2 = 73%, p = 0.005), therefore the outcomes were combined using a random effects model. The synthesized data indicated that SBP was significantly reduced in the IMT group

compared with that in the control group (MD, -6.63; 95% CI, -13.26 0.00; Figure S3, SBP). In contrast, no changes in NC, SE, MEP, FVC, FEV1/FVC, FEV1%, DBP, or HR were observed between the IMT and control groups (Figure S3).

Subgroup Meta-analyses

Subgroup analyses were performed to compare outcomes based on the AHI at baseline (mild and moderate, defined as AHI < 30 points, or severe, defined as AHI ≥ 30 points). We found that the changes in SBP (MD, -12.47; 95% CI, -15.31 to -9.62; $p < 0.00001$; $I^2 = 0\%$, $p = 0.670$) and DBP (MD, -5.08; 95% CI, -7.01 to -3.14; $p < 0.00001$; $I^2 = 0\%$, $p = 0.590$) were associated with the baseline AHI, with mild to moderate patients experiencing significant decreases after IMT (Figure 2, Figure 3).

Spearman's Correlation between the MIP and BMI, AHI, PSQI, ESS, SBP, and DBP

The correlations between the baseline and change values, including those of MIP, AHI, PSQI, ESS, SBP, and DBP, are shown in Figure 4. The results indicate that baseline characteristics may impact the magnitude of improvement following IMT. In studies in which the participants had a higher baseline MIP, we observed greater improvements in BMI, AHI, PSQI, and ESS. Conversely, studies with participants who had a higher baseline AHI (more severe OSA) tended to show less improvement in MIP.

Discussion

This systematic review and meta-analysis provides the first comprehensive evaluation of the efficacy of IMT as a standalone intervention for OSA. The most significant finding was that the IMT did not significantly reduce the AHI. This indicates that the IMT applied in these studies did not directly improve the anatomical vulnerability or collapsibility of the upper airway. Despite this limitation, our analysis indicates that IMT provides significant benefits across multiple secondary

outcomes, including sleep quality, daytime sleepiness, inspiratory muscle strength, and cardiovascular parameters. Dissociation existed between symptom improvement and unchanged AHI, suggesting that the therapeutic effect of IMT may be achieved through non-anatomical pathways rather than by directly preventing collapse. Based on these parameters, we speculate that several interconnected physiological mechanisms may be involved.

Disease-Related Risk Factors

Obesity is a predominant risk factor for OSA. Weight gain exacerbates OSA severity (25-27), whereas BMI reduction can normalize AHI levels (28,29). Our meta-analysis demonstrated a significantly lower BMI in the IMT group compared with those in the controls, aligning with mechanistic hypotheses positing that IMT enhances diaphragmatic efficiency and respiratory muscle endurance, potentially increasing daily energy expenditure and promoting weight loss.

Notably, NC, a surrogate marker of upper airway adipose deposition (30,31), showed no significant improvement after IMT. This null effect may reflect the localized nature of IMT, which primarily targets the inspiratory muscles (e.g., the diaphragm and external intercostals) rather than cervical adiposity. These findings underscore the selective impact of IMT on systemic versus regional adiposity in patients with OSA.

Sleep-Related Data

The primary focus of this study was to evaluate the effect of IMT on the severity of OSA. First, the AHI was assessed in six RCTs, yielding conflicting results. Three trials demonstrated improvements in AHI in the IMT group (14,18,21), whereas others reported no significant changes (15,19,23). Our meta-analysis found no significant reduction in the AHI after IMT. To holistically evaluate the efficacy of IMT in the treatment of OSA, we analyzed its effects on additional sleep-related parameters.

Our pooled analysis revealed a significant post-IMT PSQI reduction, indicating that IMT effectively enhanced sleep quality. Furthermore, as assessed using the ESS, excessive daytime sleepiness significantly improved after IMT. Moreover, we observed a significant increase in $LSaO_2$ during sleep. In addition, Vranish and Bailey (23) reported an improvement in the AI in the IMT group. Finally, our meta-analysis revealed a 4.22% increase in SE after IMT; however, the change was not significant. We attributed this outcome to the relatively small sample size used to assess SE.

Regarding the possible non-anatomical pathways underlying the effects of IMT, a key mechanism is elevation of the respiratory effort-related arousal threshold. By strengthening the inspiratory muscles, IMT reduces the perceived effort required to breathe against an obstructed airway. This allows patients to tolerate longer periods of obstruction without cortical arousal, leading to more consolidated sleep (improved PSQI), reduced sleep fragmentation, and consequently less daytime sleepiness (improved ESS) (32). Meanwhile, improved ventilatory control may attenuate sympathetic overactivity, which also explains the reduction in ESS (-3.18 points) despite no changes in AHI. The significant increase in $LSaO_2$ supports this model, indicating that events, while still occurring, may be less severe. Simultaneously, an increase in $LSaO_2$ also reduces the arousal phenomena, enabling the IMT to further consolidate the sleep architecture. Additionally, improved respiratory muscle efficiency may stabilize ventilatory control (reduce loop gain) and prevent the overshooting of ventilatory responses that perpetuate event cycling. Finally, according to the rule of thumb summarized by Fattal et al. (28), every 7-pound drop in weight is thought to reduce the AHI by 7 %. Our study showed that 12-week IMT resulted in a maximum weight loss of 2.6 kg (less than 7 pounds), which might be the reason for the failure to significantly decrease AHI. Additionally, AHI decreases in relation to a reduced

amount of fluid shifting rostrally from the legs to the neck (33). NC serves as an indicator of fluid accumulation in this context. No significant reduction in NC was observed following IMT treatment, which may explain the lack of reduction in AHI.

Collectively, these improvements in sleep quality, oxygenation, and daytime function underscore the role of IMT as a viable therapeutic adjunct for OSA, particularly in patients who are intolerant to first-line therapies, such as CPAP.

Respiratory-Related Data

Patients with OSA exhibit reduced inspiratory muscle strength, endurance, and increased fatigability (34). Elevated upper airway resistance during sleep likely amplifies inspiratory effort and mechanical load, thereby impairing inspiratory muscle function (35,36). Our meta-analysis demonstrated a significant increase in MIP, confirming that IMT effectively enhanced the inspiratory muscle capacity. IMT provides benefits such as increased inspiratory muscle strength and endurance (37,38). In addition, the degree of improvement in MIP is associated with the training load and intervention time (39). After correlation analysis, we found that MIP correlated positively with inspiratory muscle strength baseline levels and negatively with baseline AHI but not with training load or intervention time. However, because the duration of treatment in the original studies was at most 12 weeks, further clinical trials are required to confirm long-term efficacy.

IMT can improve lung function in patients with heart failure (40). Therefore, we analyzed changes in lung function. We found three primary studies (16,19,20), in which only the high-intensity group showed positive reactions. In our study, a significant increase was found in FVC% predicted values after IMT. The upper and lower respiratory tracts are connected mechanically. When IMT increases the lung volume, the cross-sectional area of the airway increases, and

ventilation is smooth. This is the respiratory function benefit of IMT and explains the possible mechanism by which IMT improves sleep in patients with OSA.

Cardiovascular Function

Prolonged sleep curtailment raises 24-hour and sleep-time BP in patients with OSA (41). These symptoms lead to cardiovascular diseases (42) such as HT, especially drug-resistant HT. Treatments that stabilize or even lower blood pressure should be used to treat OSA. Current research shows that CPAP leads to a mild reduction in aldosterone secretion and improves nighttime BP (43). Therefore, we conducted an analysis to confirm the antihypertensive effect of IMT in patients with OSA, which was mainly reflected by SBP (16,17,20,22,23). Our analysis demonstrated a significant reduction in SBP, particularly in patients with mild to moderate OSA. This finding indicates that IMT has tangible cardiovascular benefits and direct clinical relevance for risk management. While long-term outcomes regarding major adverse cardiovascular events fell beyond the scope of our included studies and constitute a critical area for future research, the observed BP-lowering effect provides a compelling physiological rationale for the potential role of IMT in mitigating OSA-associated cardiovascular risk.

Observational studies have revealed that, as the time course of changes in PNE parallels BP, a heightened sympathetic drive is likely an important mediator of BP changes (44). Furthermore, Mills et al. indicated that one of the mechanisms through which CPAP reduces PNE levels is via an increase in the clearance of PNE from the circulation, and this effect is related to reductions in BP. Measurements of PNE at baseline and at the end of the study were performed in two studies (22,23). Vranish et al. reported a significant decrease in NE levels after IMT. This effect can be interpreted as IMT lowering BP by reducing PNE. However, it remains to be confirmed whether IMT reduces NE levels by altering daytime release and/or clearance rates. Furthermore, there was

no significant difference in HR between the IMT and placebo groups. This suggests that changes in BP in patients with OSA after IMT had less of an effect on HR, underscoring the cardiovascular safety profile of the intervention.

Overall, IMT could be used as an adjuvant therapy for patients with OSA who are unwilling or unable to tolerate nightly CPAP therapy. This treatment could also be offered in addition to CPAP to reduce the pressure insufflated into the airways, thereby improving patient adherence.

Limitations

This meta-analysis had several limitations. First, the number of eligible articles and sample sizes for the meta-analysis was small. This limitation restricted our ability to conduct robust subgroup or meta-regression analyses, preventing us from clearly determining the impact of intervention parameters on clinical outcomes, such as optimal IMT intensity or duration. Consequently, the high heterogeneity observed in the analyses likely stems from different training protocols and cannot be quantitatively interpreted. Second, our study was limited to studies written in English, which may have led to language bias. Finally, most of the included studies reported the short-term effects of IMT in the treatment of OSA, rather than long-term effects. Therefore, future research should consider long-term follow-ups to assess the efficacy of IMT and fully understand the role and mechanisms of IMT in the treatment of OSA.

Conclusion

This meta-analysis demonstrated that IMT effectively mitigated disease-related risk factors and improved the quality of life of patients with OSA. However, IMT does not significantly reduce the AHI. Therefore, IMT should not be considered a primary therapy to eliminate respiratory events. Instead, it is valuable as a supplemental or adjunctive intervention, particularly for patients

intolerant to CPAP therapy. These findings highlight the potential of IMT for managing OSA complicated by refractory HT, particularly in mild-to-moderate cases.

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Table 1. Characteristics of the enrolled studies

First author, Year	Country	Study Design	Patients included in Analysis	Intervention group	control group	Frequency of intervention	Duration of follow-up (weeks)
Vranish/2016	USA	RCT	24	IMT,75%MIP	IMT, 15%MIP	30 breaths/day	6
Ferreira Souza/2018	Brazil	RCT	18	IMT,50–60% MIP	IMT, 20%MIP	3 cycles of 30 breaths, twice/day, 7 days/week	12
Erturk/2020	Turkey	RCT	27	IMT,30%MIP	N/A	15 min, twice/day, 7 days/week	12
Lin/2020	Taiwan	RCT	22	IMT,30%MIP	N/A	30-45 min/day, 5 days/week	12
Moawd/2020	Egypt	RCT	55	IMT,75%MIP	IMT, ≤ 10% MIP	6 cycles of 30 breaths, three times/week	12
Nogueira/2020	Brazil	RCT	16	IMT, 50% MIP-2 weeks IMT,60% MIP-2 weeks IMT,75% MIP-4 weeks	IMT, 0%MIP	3 cycles of 30 breaths, twice/day, 7 days/week	8
Ramos-Barrera/2020	USA	RCT	25	IMT,75%MIP	IMT, 15%MIP	5 sets of 6 breaths, 5 days/week	6
Azeredo/2022	Brazil	RCT	43	IMT,40% MIP-2 weeks +10 cmH_2O-third week +20 cmH_2O-fourth week +30 cmH_2O-4 weeks	IMT, 10 cmH_2O MIP	30 breaths/day	12
Ferreira, Silvia/2022	Braszil	RCT	40	IMT,70% MIP	IMT, 0%MIP	3 sets of 30 breaths	1 hour
Labeix/2022	France	RCT	45	IMT,70% MIP	N/A	2 sets of 30 breaths, 6 days/week	6

Definition of abbreviations: RCT = randomized controlled trials; IMT = Inspiratory muscle training; MIP = maximal inspiratory pressure.

Table 2. Baseline Characteristics of Participants in the Included Studies: Demographics, AHI, ESS, and PSQI

First author, Year	Country	Group, n	Gender (M/F)	Age (years)	BMI (kg/m²)	NC	AHI (Events/h)	ESS	PSQI
Vranish/2016	USA	IMT:12	8/4	61.5±3.9	27.0±1.0	15.6±0.5	21.9±4.4	N/A	9.1±0.9
		Placebo:12	8/4	69.1± .4	28.5±1.6	14.6±0.7	29.9±8.9	N/A	9.8±0.9
Ferreira Souza/2018	Brazil	IMT:8	4/4	54.8±6.9	N/A	40.0±4.5	27.6±11.9	11.1±4.5	7.0±4.7
		Placebo:8	6/2	49.9±11.6	N/A	43.5±3.0	34.0±18.4	11.1±6.8	8.8±4.2
Erturk/2020	Turkey	IMT:15	N/A	49.66±9.08	31.00±5.42	38.66±3.30	30.00±19.33	8.93±4.41	7.46±4.15
		Control:12	N/A	47.25±7.32	32.06±3.69	42.08±2.77	38.70±23.98	9.66±5.91	7.33±3.98
Lin/2020	Taiwan	IMT:16	13/3	47.9±12.2	26.2±3.3	38.3±2.8	29.0±2.8	10.5±5.7	8.2±4.9
		Control:6	5/1	56.2±11.5	27.3±3.6	38.5±2.9	37.5±14.1	13.0±2.6	8.6±2.1
Moawd/2020	Egypt	IMT:28	20/8	55.5±9.8	29.2±3.9	N/A	32±11.7	N/A	N/A
		Placebo:27	22/5	59.5±4.8	27.9±4.8	N/A	31±10.8	N/A	N/A
Nogueira/2020	Brazil	IMT:8	3/5	58.6±5.6	32.65±3.75	38.6±3.8	31.7±15.9	12.5±4.0	7.2±3.6
		Placebo:8	1/7	60.1±2.7	30.19±9.92	35.5±3.7	31.4±20.8	14.9±5.2	7.5±3.2
Ramos-Barrera/2020	USA	IMT:15	11/4	65.9±6.0	30.7±6.2	41.6±5.2	22.9±11.0	N/A	8.6±4.0
		control:10	6/4	69.7±6.7	31.3±6.5	41.0±3.9	26.2±13.5	N/A	9.0±5.0
Azeredo/2022	Brazil	IMT:22	13/9	63±15	29.8±5.2	38.3±4.8	27.92±10.3	8±4.75	6.36±3.96
		control:21	14/7	55±15	31.2±5.2	40.2±3.6	20.72±11.13	11.72±4.77	7.36±3.96
Ferreira, Silvia/2022	Braszil	IMT:20	10/10	55.8±9.1	34.0±8.1	41.4±4.5	31.8±18.2	13.2±7.7	11.2±5.1
		control:20	12/8	55.4±11.6	33.8±6.9	41.4±4.8	30.9±16.7	14.5±7.0	11.1±4.8
Labeix/2022	France	IMT:22	21/1	61.0±8.4	27.9±4.1	38.8±2.8	24.9±7.8	7.3±5.2	6.1±3.7
		control:23	19/4	59.3±10.3	28.1±5.8	40.1±2.6	22.0±7.1	7.4±3.6	6.0±3.7

Definition of abbreviations: IMT = Inspiratory muscle training; BMI = body mass index; NC = neck circumference; AHI = apnea-hypopnea index; ESS = Epworth Sleepiness Scale; PSQI = Pittsburgh Sleep Quality Index.

Table 3. Summary of Meta-Analysis Results for Primary and Secondary Outcomes: Between-Group Differences

Date	Mean Difference	*p*
BMI	-1.48 [-2.39, -0.57]	0.001
NC	-0.82 [-3.68, 2.04]	0.570
AHI	1.00 [-2.57, 4.56]	0.580
PSQI	-3.15 [-3.69, -2.62]	<0.001
SE	4.22 [-1.06, 9.49]	0.120
$LSaO_2$	2.86 [1.01, 4.71]	0.002
ESS	-3.18 [-4.50, -1.87]	<0.001
MIP	25.54 [11.09, 40.00]	<0.001
MEP	2.19 [-15.55, 19.93]	0.810
FVC	-0.08 [-1.21, 1.05]	0.890
FEV_1/FVC	-1.00 [-4.73, 2.72]	0.600
FEV_1%	5.94 [-5.51, 17.39]	0.310
FVC%	17.20 [9.53, 24.87]	<0.001
SBP	-6.63 [-13.26, -0.00]	0.050
DBP	-2.10 [-5.79, 1.59]	0.260
HR	-0.75 [-2.27, 3.76]	0.630
PNE	-68.94 [-154.86, 16.98]	0.120

Definition of abbreviations: BMI = body mass index; NC = neck circumference; AHI = apnea-hypopnea index; PSQI = Pittsburgh Sleep Quality Index; SE = sleep efficiency; LSaO2 = lowest oxygen saturation; ESS = Epworth Sleepiness Scale; MIP = maximal inspiratory pressure; MEP = maximum expiratory pressure; FVC = forced vital capacity; FEV1/FVC = the ratio of forced expiratory volume in 1 second to forced vital capacity; FEV1 = forced expiratory volume in one second; SBP = systolic blood pressure; DBP = diastolic blood pressure; HR = heart rate; PNE= plasma norepinephrine.

Figure 1. Flow Diagram of the Literature Search

Figure 2. Forest plots of the subgroup meta-analysis for the effect of IMT on the level of SBP in patients with OSA. CI = confidence interval; IV=inverse variance.

Figure 3. Forest plots of the subgroup meta-analysis for the effect of IMT on the level of DBP in patients with OSA. AHI = apnea-hypopnea index; CI = confidence interval; IV=inverse variance.

Figure 4. Spearman's Correlation between the MIP and BMI, AHI, PSQI, ESS, SBP, and DBP. BMI = body mass index; NC = neck circumference; AHI = apnea-hypopnea index; MIP = maximal inspiratory pressure; PSQI = Pittsburgh Sleep Quality Index; ESS = Epworth Sleepiness Scale; MIP = maximal inspiratory pressure; SBP = systolic blood pressure; DBP = diastolic blood pressure

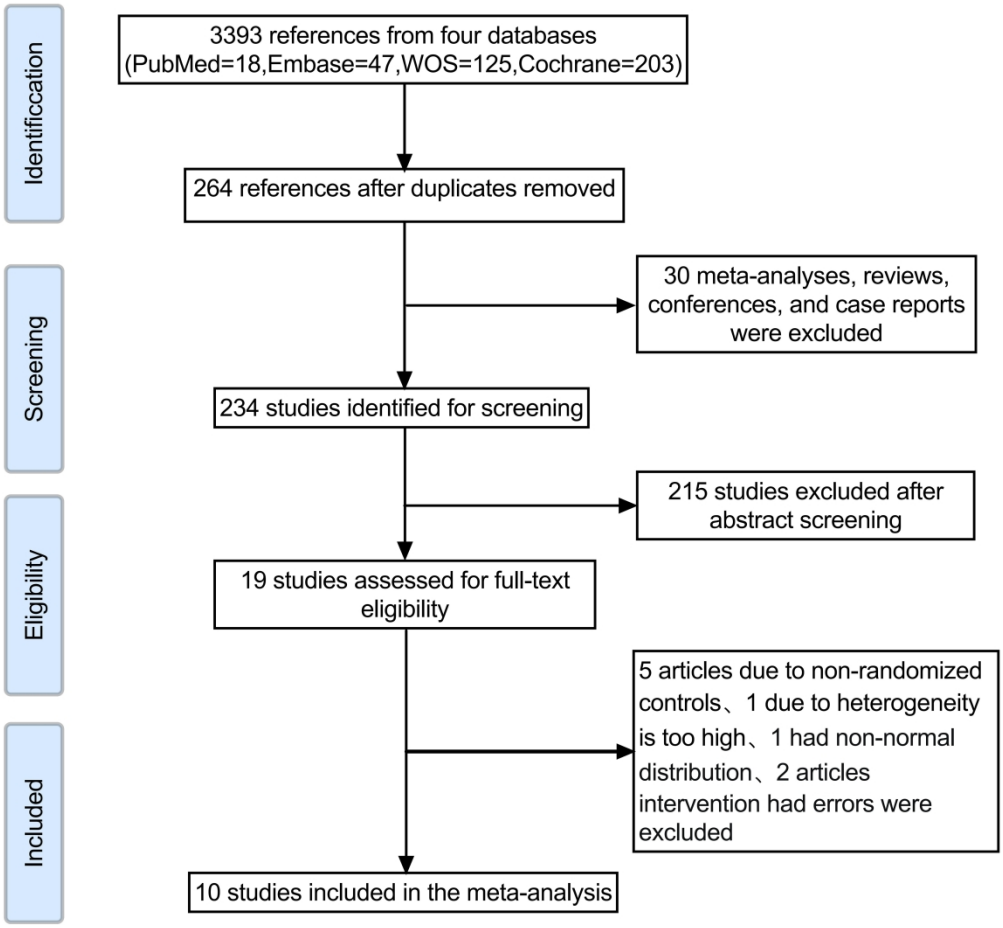

Figure 1. Flow Diagram of the Literature Search

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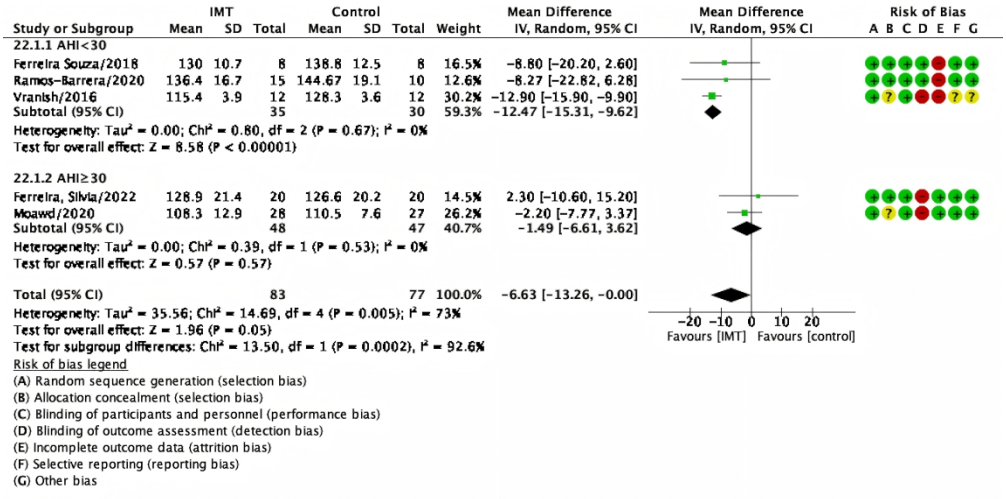

Figure 2. Forest plots of the subgroup meta-analysis for the effect of IMT on the level of SBP in patients with OSA. CI = confidence interval; IV=inverse variance.

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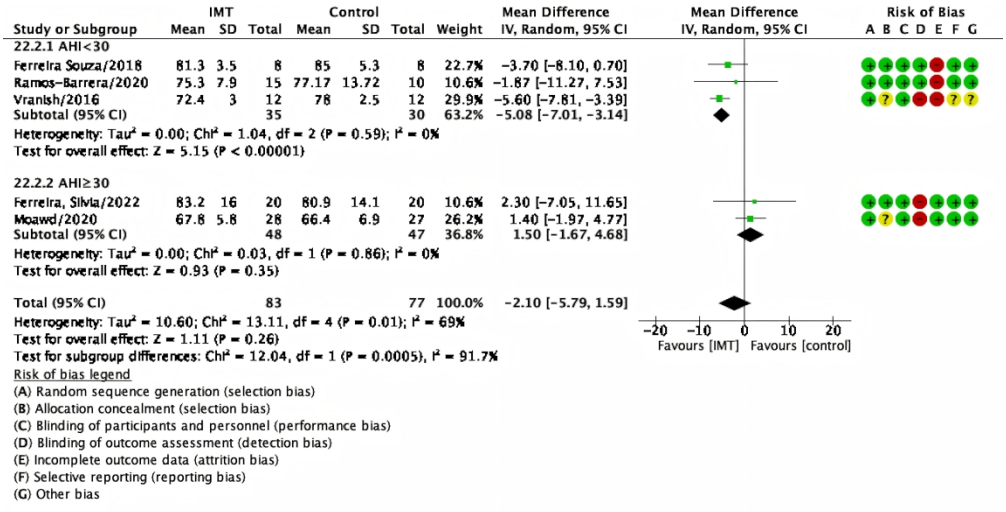

Figure 3. Forest plots of the subgroup meta-analysis for the effect of IMT on the level of DBP in patients with OSA. AHI = apnea-hypopnea index; CI = confidence interval; IV=inverse variance.

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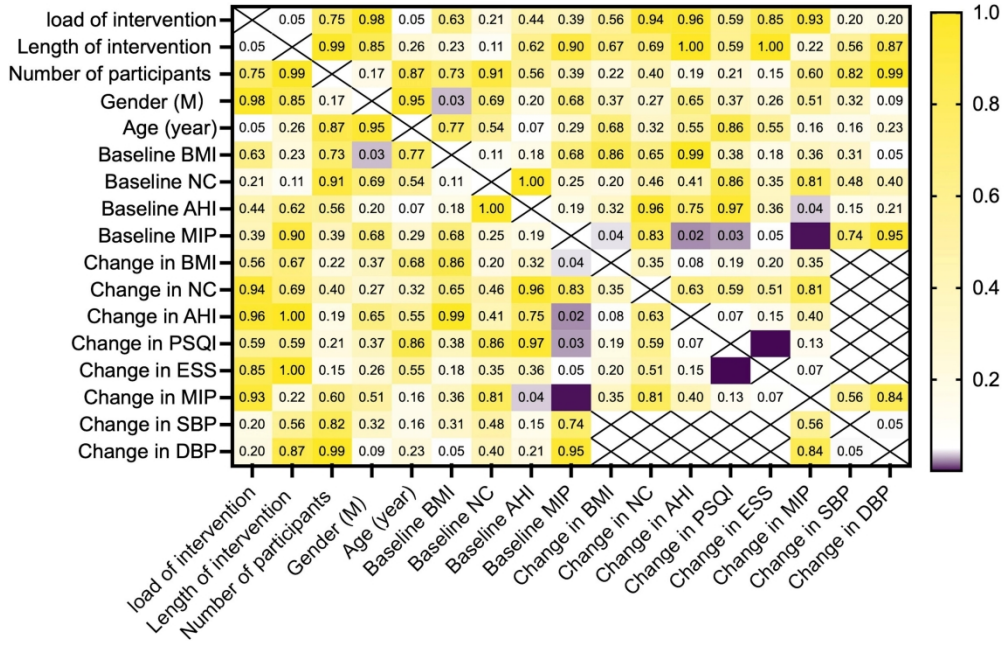

Figure 4. Spearman's Correlation between the MIP and BMI, AHI, PSQI, ESS, SBP, and DBP. BMI = body mass index; NC = neck circumference; AHI = apnea-hypopnea index; MIP = maximal inspiratory pressure; PSQI = Pittsburgh Sleep Quality Index; ESS = Epworth Sleepiness Scale; MIP = maximal inspiratory pressure; SBP = systolic blood pressure; DBP = diastolic blood pressure.

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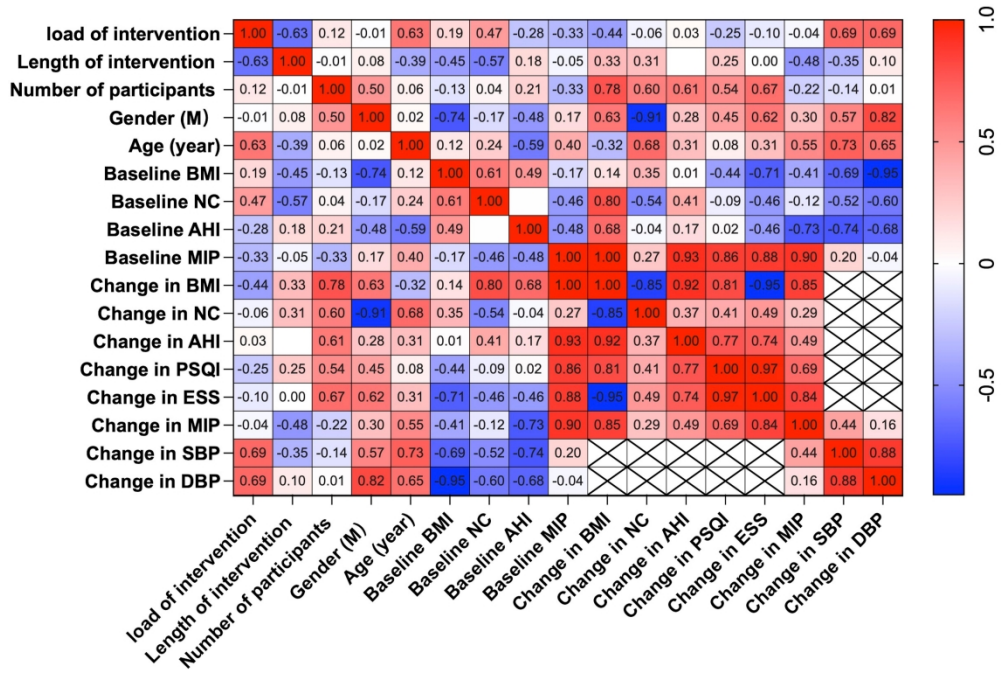

Figure 4. Spearman's Correlation between the MIP and BMI, AHI, PSQI, ESS, SBP, and DBP. BMI = body mass index; NC = neck circumference; AHI = apnea-hypopnea index; MIP = maximal inspiratory pressure; PSQI = Pittsburgh Sleep Quality Index; ESS = Epworth Sleepiness Scale; MIP = maximal inspiratory pressure; SBP = systolic blood pressure; DBP = diastolic blood pressure.

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