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Effect of continuous positive airway pressure on glucose metabolism in adults with type 2 diabetes: a systematic review and meta-analysis of randomized controlled trials

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Abstract Obstructive sleep apnea (OSA) has many serious consequences, and one of these may be the exacerbation of type 2 diabetes mellitus (T2DM). Reports on the effect of continuous positive airway pressure (CPAP) on glucose metabolism in people with T2DM and OSA are conflicting. Therefore, the purpose of this review was to examine the effect of CPAP treatment on glucose metabolism by synthesizing findings from randomized controlled trials. The PRISMA review protocol was developed and registered in PROSPERO. A systematic search of PubMed, CINAHL, Embase, Web of Science, PsycInfo, and Cochrane was conducted from inception to March 2017. The Cochrane risk of bias tool was used to assess the study quality. Review Manager (v5.2) was used for the meta-analyses, and the standardized mean difference was calculated. Six studies consisting of 496 participants were included in this review. The meta-analyses indicated that CPAP treatment did not have significant impact on glucose metabolism measured by A1C (mean difference = 0.05, 95% CI − 0.14 to 0.24, $P = 0.61$), fasting insulin level (mean difference = − 2.34, 95% CI − 8.19 to 3.51, $P = 0.43$), and fasting glucose (mean difference = − 0.05, 95% CI − 0.52 to 0.42, $P = 0.84$). As expected, CPAP treatment can improve daytime sleepiness (mean difference = − 2.68, 95% CI − 3.91 to − 1.54, $P < 0.001$). Findings of this meta-analysis do not substantiate a positive effect of CPAP on glucose metabolism in people with T2DM and coexisting OSA. Future large-scale clinical trials with a longer treatment duration and better CPAP compliance are warranted.

Keywords CPAP · Diabetes · Glycemic control · Meta-analysis · OSA · RCT

Introduction

Worldwide, 415 million people had diabetes in 2015, which is projected to be 642 million by 2040 [1]. Over 90% of all cases are type 2 diabetes mellitus (T2DM). Every 6 s, one person dies from diabetes, resulting in 5.0 million deaths in 2015 [1]. Around 12% of global health expenditure was spent on diabetes; the estimated direct costs for individuals with diabetes was estimated to be 2.3 times higher compared to their non-diabetic counterparts [2]. Multiple physiological and behavioral factors are considered responsible for the drastic increase in diabetes prevalence, among which obstructive sleep apnea (OSA) is shown to be an independent risk factor [3–5]. OSA is characterized by repetitive upper airway obstruction that results in a cessation or significant reduction in airflow during sleep, which causes intermittent hypoxia and sleep fragmentation [6]. With the rise of obesity and aging, the prevalence of OSA is expected to increase [7]. In the USA alone, 12% of the adult population is estimated to have OSA [8], with estimates rising to as high as 71% in people with T2DM [9]. OSA brings many serious health consequences, and one of these may be the exacerbation of T2DM.

OSA can be treated with various methods including oral appliances, surgery, and continuous positive airway pressure (CPAP). The most common treatment choice is CPAP, which

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helps to keep the airway open by pressurizing the air in the upper airway [10]. CPAP treatment is effective in reducing daytime sleepiness [11], but its effect on glucose metabolism (e.g., A1C, insulin sensitivity, and fasting glucose) is unknown. Theoretically, intermittent hypoxia and sleep fragmentation caused by OSA could influence the metabolism and contribute to the development of T2DM by activating the sympathetic nervous system, systemic inflammation, hypothalamic-pituitary-adrenal axis, and appetite-regulating hormone alterations [12, 13]. Empirical evidence on the effect of CPAP treatment on glucose metabolism is growing. Four reviews have included studies conducted in people with OSA [14–17], with inconsistent findings. One review concluded that CPAP could improve insulin sensitivity in non-diabetic and pre-diabetic patients [17]. In contrast, another report did not find a significant improvement in insulin sensitivity after CPAP treatment [16]. In people with T2DM and coexisting OSA, two previous meta-analyses found that CPAP treatment was effective in improving insulin sensitivity, with no effect on A1C [18, 19]. However, both reviews included the same studies, most of which were non-randomized.

Since the publication of previous reviews, more research has been published, particularly clinical trials. There is a need to review the emerging best evidence and provide an up-to-date examination of the effectiveness of CPAP treatment on glucose metabolism. Therefore, the objective of this systematic review and meta-analysis was to examine the effect of CPAP treatment on glucose metabolism including A1C, insulin sensitivity, fasting glucose, and mean glucose level. We synthesized findings from existing randomized controlled trials (RCTs) conducted in adults with T2DM and coexisting OSA.

Methods

We developed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) protocol [20] and registered it in the international prospective register of systematic reviews (PROSPERO) (registration number: 42017059085). We followed the PRISMA guideline in developing this review [21].

Search strategy

A systematic search was conducted in PubMed, CINAHL, Embase, Web of Science, PsycInfo for dissertation/thesis, and Cochrane from inception to March 2017. ClinicalTrial.gov was searched for potential completed trials. A review of the reference lists from relevant studies was conducted to identify additional studies. There was no language restriction. Combinations of the following search terms were used: (1) sleep apnea, sleep apnoea, sleep disordered breathing, OSA, or SDB; (2) diabetes; and (3) CPAP or continuous positive airway pressure. The inclusion criteria were (1) RCT; (2) studies conducted in adults (aged 18 years or over) with T2DM and OSA; and (3) studies investigated CPAP treatment. The exclusion criteria were (1) glucose metabolism not measured; (2) review, abstract, editorial, and reply; or (3) secondary analysis.

The PRISMA flow chart [21] was used to guide the selection of studies. Initial screening was conducted by one reviewer (BZ) based on the title/abstract. Full-text of potential studies was independently reviewed by two reviewers (BZ and CM) to determine the final inclusion based on the above inclusion and exclusion criteria. Disagreements were resolved by a third reviewer.

Data extraction

A standard matrix was developed by the team to extract information from each study. The extracted data included study characteristics (e.g., sample size, intervention, and outcomes) and participant characteristics (e.g., age, gender, and diabetes duration). Data were independently extracted by two reviewers (BZ and CM). Discrepancies were resolved by a third reviewer. If the data we needed could not be extracted directly from the text, we computed them using other available data. If the data were not reported in an original article, we attempted to contact the authors.

Outcome measures

The primary outcome was the change in the A1C level before and after treatment. A1C is an indicator of the overall glucose for the past 2 to 3 months and has been widely used as the "gold standard" for glycemic control [22]. Secondary outcomes included fasting glucose, mean glucose level, and insulin sensitivity measured by fasting insulin level or homeostasis model assessment (HOMA) index, such as HOMA-IR (insulin resistance). The HOMA index is a method assessing insulin sensitivity from fasting glucose and insulin [23]; it has been widely used as a robust, standard tool in diabetes research [24]. Body mass index (BMI) and daytime sleepiness measured by Epworth Sleepiness Scale (ESS) were also included as secondary outcomes. When the outcomes at different time points were reported, data at the later point were used.

Quality appraisal

The Cochrane risk of bias tool [25] was used to independently assess study quality by two reviewers (BZ and CS). Each study was evaluated from six aspects: random sequence generation, allocation concealment, blinding of the participant, blinding of outcome measures, incomplete data, and selective reporting. The discrepancy was resolved by a third reviewer.

Data analysis

Review Manager (v5.2 for Windows, Cochrane Collaboration, Oxford, U.K.) was used for statistical analyses. Statistical significance was set at $P < 0.05$. For outcomes reported in two or more studies, pooled mean differences with the 95% CI were calculated for each outcome, using the inverse variance method. Forest plots were used to present the results of individual studies and the pooled effect size. The funnel plot for the primary outcome (i.e., A1C) was used to examine publication bias, and asymmetry of the plot suggests publication bias. Heterogeneity among studies was examined by Cochrane Q test and I^2 value ($I^2 > 50\%$ considered significant) [26]. A fixed-effects model was used if no heterogeneity was detected, and a random-effects model was used otherwise [27]. In the case of heterogeneity, we performed sensitivity analyses to test the robustness of the pooled estimates, using leave-one-out approach.

Results

Searching results

The initial literature search yielded 2930 relevant records. A total of 23 underwent the full-text review, and 17 were excluded based on reasons listed in Fig. 1. Six RCTs [28–33] met the inclusion and exclusion criteria and thus were included in this review. No eligible studies were identified through other sources. The searching process is shown in Fig. 1.

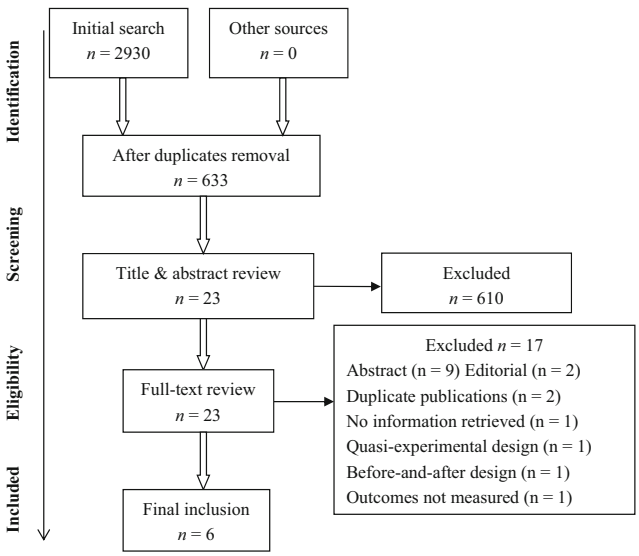

Fig. 1 PRISMA flow chart for study selection

Study characteristics

The six studies had a total of 496 participants, with the individual sample size between 19 and 298. Participants had a mean age between 55.0 and 62.4 years. The studies included a higher proportion of men, ranging from 53.8 to 100%. The intervention group received CPAP treatment, and the control group received usual care or sham-CPAP. The intervention duration ranged from 7 days to 6 months. Study characteristics are summarized in Table 1.

The risk of bias of each study is presented in Table 2. The studies typically had a low risk of selection bias and reporting bias. Three studies [30, 32, 33] used placebo or sham CPAP, and thus had a low risk of performance bias. The risk of detection bias was low in most of the studies, except in two [30, 32] where it was not clear. The risk of attrition bias was mostly low, with intention-to-treat analysis used.

Effect of CPAP treatment on glucose metabolism

Effect of CPAP treatment on A1C

Four studies [28, 29, 31, 32] were included in the meta-analysis of changes in A1C after CPAP treatment (Fig. 2a). The fixed-effects model was used, because no significant heterogeneity was detected ($\chi^2 = 1.33$, $P = 0.72$). The pooled estimates of mean difference suggested no significant difference in A1C level between the CPAP and control group (mean difference = 0.05, 95% CI − 0.14 to 0.24, $P = 0.61$).

Effect of CPAP treatment on insulin sensitivity

Two studies [29, 32] were included in the meta-analysis of changes in fasting insulin level after CPAP treatment (Fig. 2b). Significant heterogeneity was detected ($\chi^2 = 4.39$, $P = 0.04$). Therefore, the random-effects model was used. The pooled estimates of the mean difference suggested no significant difference in fasting insulin level between the CPAP and control group (mean difference = − 2.34, 95% CI − 8.19 to 3.51, $P = 0.43$). HOMA index was also used in those two studies. However, the use of different matrixes precluded us from getting a pooled result. HOMA-IR did not improve after 3-month CPAP treatment ($P = 0.092$) but had a significant improvement after 6-month intervention (intergroup adjusted difference = − 2.58, 95% CI − 4.75 to − 0.41, $P = 0.023$) [29]. In contrast, HOMA-%S did not change significantly after 3-month treatment ($P = 0.2$) [32].

Effect of CPAP treatment on fasting glucose

Four studies [28, 29, 31, 32] were included in the meta-analysis of changes in fasting glucose after CPAP treatment

Table 1 Characteristics of included studies ($n = 6$)

Author (year), country	Age (years)	Gender (male)	BMI (kg/cm^2)	Diabetes duration (years) and A1C (%)	OSA duration (years) and AHI (event/h)	Groups (sample size)	Intervention duration and compliance	Outcomes
Morariu (2017), USA	55.6 ± 10.6	14 (61-%)	35.5 ± 6.2	Participants with A1C < 9 were included Mean A1C = 6.9 ± 1.2	Newly diagnosed OSA (AHI ≥ 10) were included Mean AHI = 38.9 ± 26.1	A: CPAP ($n = 12$) B: sham CPAP ($n = 11$)	4 weeks Mean usage: 4.1 h/night	Mean daytime glucose (6 am–6 pm)
Lam (2016), Hong Kong	55.0 ± 9.0	26 (81-%)	29.9 ± 5.3	Participants with A1C ≥ 7.0 were included Mean A1C = 8.1 ± 1.1 Mean duration = 8.8 ± 5.5	Newly diagnosed (AHI ≥ 15) were included Mean AHI = 45.3 ± 23.2	A: CPAP ($n = 32$) B: no active intervention ($n = 32$)	3 months Mean usage: 2.5 h/night	A1C Fasting glucose BMI ESS
Martinez-Ceron (2016), Spain	61.0 ± 9.0	30 (60-%)	32.5 ± 4.5	Participants with A1C > 6.5 were included Mean A1C = 7.62 ± 1.05 Mean duration = 5 (3–15)	Newly diagnosed OSA (AHI ≥ 5) were included Mean AHI = 32.1 ± 20.9	A: usual care + CPAP ($n = 26$) B: usual care ($n = 24$)	6 months Mean usage: 5.2 h/night	A1C Fasting glucose Insulin sensitivity: HOMA-IR and fasting plasma insulin
Mokhlesi (2016), USA	56.2 ± 3.2	7 (53.-8%)	36.8 ± 2.5	Newly diagnosed patients or on oral medication for > 3 m were included Mean A1C = 7.3 ± 0.4 Mean duration = 2.5 ± 1.2	Mean AHI = 39.7 ± 8.0	A: CPAP ($n = 13$) B: sham CPAP ($n = 6$)	7 days in-lab Mean usage: 7.92 h/night	24-h mean glucose
Shaw (2016), Australia and USA	62.4 ± 9.1	99 (65.-6%)	33.4 ± 5.9	Participants with 6.5 < A1C ≤ 8.5 were included Mean A1C = 7.3 Mean duration = 8.4 ± 7.3	Newly diagnosed OSA (5 < AHI < 70) Mean AHI = 28.0 ± 14.1	A: usual care + CPAP ($n = 151$) B: usual care ($n = 147$)	6 months Mean usage: 4.3 h/night at 3 months and 4.9 h/night at 6 months	A1C Fasting glucose ESS BMI
West (2007), UK	57.8 ± 10.4	44 (100-%)	36.6 ± 4.9	Mean A1C = 8.5 ± 1.8 Mean duration = 7.3	Newly diagnosed OSA (> 10 dips/h in oxygen saturation) were included	A: CPAP ($n = 20$) B: placebo CPAP ($n = 20$)	3 months Mean usage: 3.6 h/night	A1C Fasting glucose Insulin sensitivity: HOMA-%S and fasting plasma insulin ESS BMI

BMI body mass index, *OSA* obstructive sleep apnea, *AHI* apnea-hypopnea index, *CPAP* continuous positive airway pressure, *ESS* Epworth Sleepiness Scale, *HOMA-IR* homeostasis model assessment-insulin resistance

(Fig. 2c). The fixed-effects model was used, because no significant heterogeneity was detected ($\chi^2 = 0.84$, $P = 0.84$). The pooled estimates of the mean difference suggested no significant difference in fasting glucose level between the CPAP and control group (mean difference = − 0.05, 95% CI − 0.52 to 0.42, $P = 0.84$).

Effect of CPAP treatment on mean glucose level

Two studies [30, 33] examined the effect of CPAP treatment on mean glucose level. Nevertheless, the use of mean glucose over different time spans (i.e., 24 h and 6 am–6 pm) precluded us from getting a pooled estimation. Mokhlesi et al. [30] found

Table 2 Risk of bias assessment ($n = 6$)

Author (year)	Random sequence generation (selection bias)	Allocation concealment (selection bias)	Blinding of participant (performance bias)	Blinding of outcome measures (detection bias)	Incomplete data (attrition bias)	Selective reporting (reporting bias)
Morariu (2017)	Low	Low	Low	Low	Not clear	Low
Lam (2016)	Low	Low	High	Low	Low	Low
Martinez-Ceron (2016)	Low	Not clear	High	Low	Low	Low
Mokhlesi (2016)	Not clear	Not clear	Low	Not clear	Low	Low
Shaw (2016)	Low	Low	High	Low	Low	Low
West (2007)	Low	Low	Low	Not clear	Low	Low

a positive effect of CPAP treatment on 24-h mean glucose. There was a 13.7 mg/dl decrease in the mean 24-h glucose in the CPAP treatment group as compared with the 2.9 mg/dl decrease in the control group ($P = 0.01$). In contrast, Morariu et al. [33] did not find a significant change in mean glucose from 6 am to 6 pm ($P = 0.7$).

Effect of CPAP treatment on other parameters

As predicted, there was a significant decrease in ESS score in the CPAP group compared to the control group (Fig. 3a): mean difference = − 2.68, 95% CI − 3.91 to − 1.45, $P < 0.001$. The effect of CPAP treatment on BMI was not significant: mean difference = 0.11, 95% CI − 0.49 to 0.71, $P = 0.72$ (Fig. 3b).

Sensitivity analysis and publication bias

Sensitivity analyses suggested the results were robust. A funnel plot was performed for the primary outcome (i.e., A1C). The plot indicated no publication bias (Fig. 4).

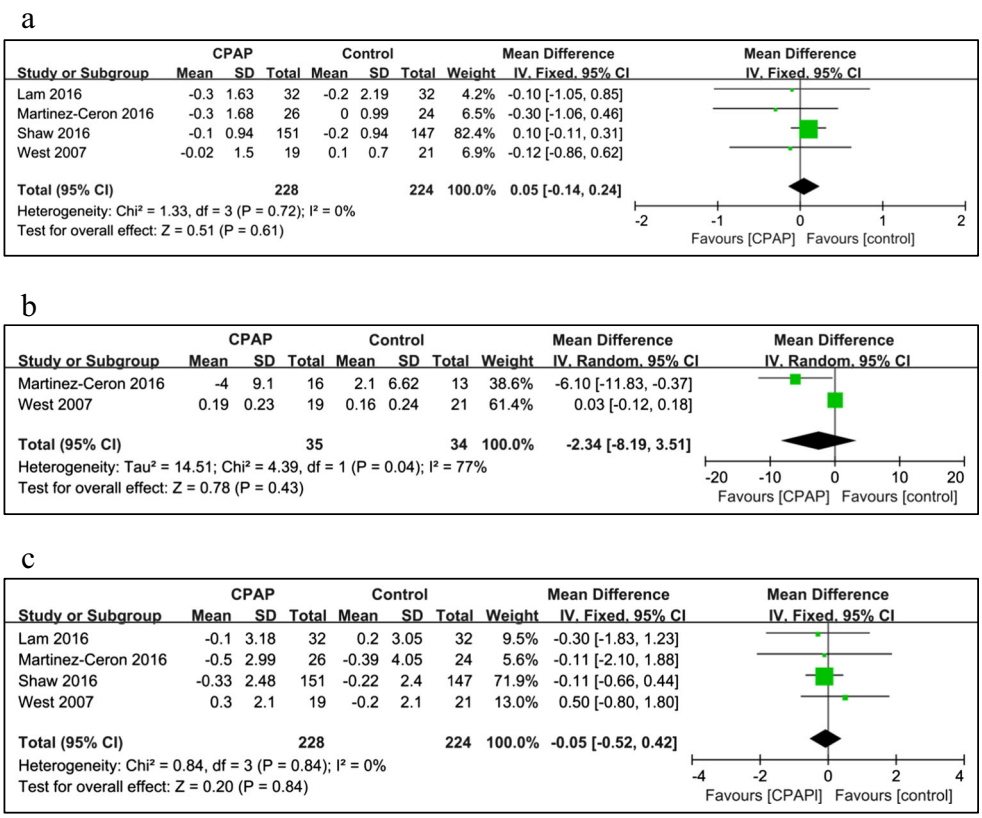

Fig. 2 Forest plots for mean differences in glucose metabolism. **a** A1C. **b** Fasting insulin. **c** Fasting glucose

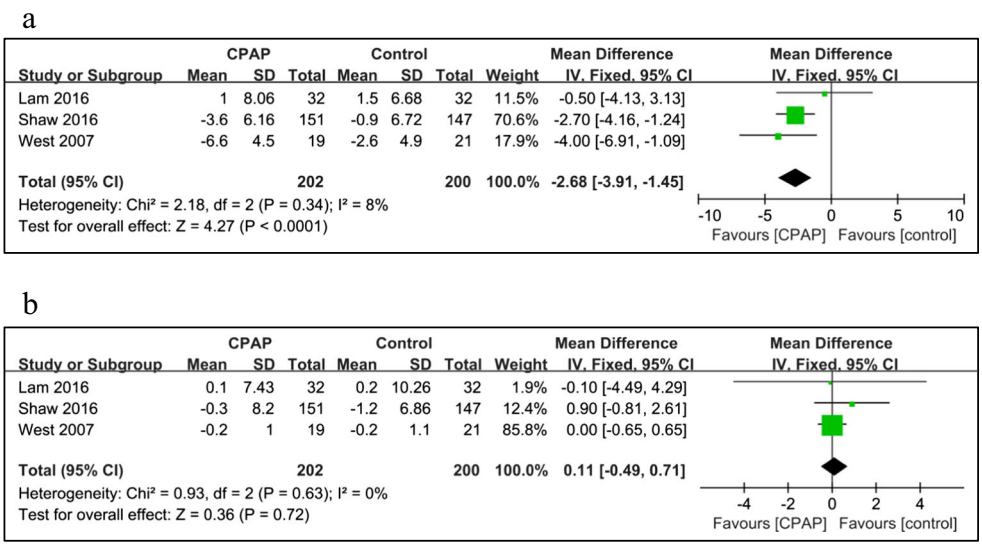

Fig. 3 Forest plots for mean differences in other parameters. **a** ESS. **b** BMI. ESS Epworth Sleepiness Scale, BMI body mass index

Discussion

The aim of this meta-analysis was to examine the effect of CPAP treatment on glucose metabolism in T2DM adults with OSA. We synthesized finding from six RCTs. Consistent with previous reviews, we found that CPAP treatment significantly reduced daytime sleepiness [11, 34]. Nevertheless, we did not find a significant effect of CPAP treatment on glucose metabolism.

In this review, we found that CPAP treatment did not affect A1C level. This finding is in line with previous systematic reviews [16, 18, 19]. Specifically, Feng et al. [18] and Chen et al. [19] found no significant difference in A1C before and after the CPAP treatment in people with T2DM and OSA. Likewise, in people with OSA only, Hecht et al. [16] found no improvement of A1C after CPAP treatment (P = 0.94). Taken together, we believe that current evidence does not support the beneficial effect of CPAP treatment on A1C. Nevertheless, the findings of this meta-analysis need to be interpreted carefully. Consensus has not been reached on which measures are most indicative of OSA-related changes in glucose metabolism. The use of a crude (i.e., A1C) measure of glucose metabolism does not fully capture the problems in glucose disposal [12]. Additionally, variability in the duration and adherence to CPAP treatment might also explain the inconsistent findings. A1C is a measure of overall glucose during the past 2–3 months, which takes time to change. In this review, the duration of CPAP treatment ranged from 1 week to 6 months. It has been suggested that a longer intervention is required for the structural changes caused by β-cell damage to be corrected [13]. The commonly used 3-month intervention might not be long enough to detect the changes in A1C [28]. More importantly, adherence to the CPAP treatment could largely influence the effect of CPAP treatment. In the three studies [28, 31, 32] that did not find a significant effect of CPAP on A1C, the usage of CPAP treatment per night was between 2.5 and 4.3 h, compared to 5.2 h in the study conducted by Martinez-Ceron et al. [29] where a significant decrease in A1C was observed. Additionally, the severity of OSA likely influences the effect of CPAP treatment on A1C. Tamura et al. [35] examined the relationship between the OSA severity and A1C. In that study, OSA-induced hypoxia was independently associated with A1C regardless of the glucose tolerance, and the association was stronger in those with diabetes. Similarly, in patients with T2DM, more severe OSA was associated with a higher A1C level, independent of confounders [36]. The adjusted A1C increased by 1.93% in those with moderate OSA and 3.60% in those with severe OSA. In this review, participants' mean AHI ranged from 28.0 to 45.3 events/h. People with different OSA severity might show a different metabolic response to the CPAP, which could explain the negative finding.

We did not find an improvement in insulin sensitivity after CPAP treatment, which is inconsistent with two review findings. Feng et al. [18] synthesized findings from one RCT and two non-RCTs conducted in patients with T2DM and OSA. They found a significant improvement of insulin sensitivity, evaluated by euglycaemic hyperinsulinaemic clamp

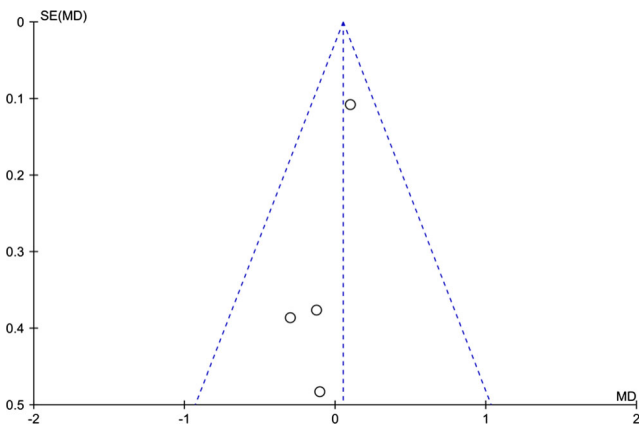

Fig. 4 Funnel plot of studies assessing A1C

(combined difference = 0.330, 95% CI 0.001 to 0.658, $P = 0.049$). However, the wide CI indicates instability of the findings. Similar to the study of Feng et al., Chen et al. [19] included the same two non-RCTs and found that CPAP treatment significantly increased insulin sensitivity (combined difference = 0.522 μmmol/kg/min, SE = 0.196, $P = 0.005$). The inconsistency between our findings and those two reviews could be explained by study design and variances in the measurement of insulin sensitivity. In our analysis, we used fasting insulin level as opposed to the clamp. Although the use of fasting insulin level is the most practical method, it might result in a high proportion of false-positive results [37]. Participant characteristic, such as OSA severity, likely affect the effect of CPAP on insulin sensitivity. Patients with more severe OSA [38] might benefit more from the treatment. Participants in the two previous reviews [18, 19] all had moderated-to-severe OSA, while participants in the present review had newly diagnosed OSA with various disease severities.

Three previous reviews examined the effect of CPAP on insulin sensitivity in non-diabetic patients with OSA. Our finding is in line with the study of Hecht et al. [16] where no significant effect of CPAP on HOMA-IR was found. In contrast, the other two reviews suggested a favorable effect [14, 15]. Disease chronicity is a well-established key factor that can affect the response to treatment. Development of T2DM is a progressive process characterized by initial insulin resistance, compensatory hyperinsulinemia, and failure of pancreatic β-cells (i.e., impaired insulin secretion) [39]. Response to CPAP treatment on insulin sensitivity could differ for patients with various diabetes severity and duration. People with poorer glycemic control might benefit more from the treatment due to a larger potential for improvement [12]. In this review, participants' mean baseline A1C ranged from 6.9 to 8.5%. Using a different A1C level as the inclusion criteria might have contributed to the inconsistency. Additionally, β-cell function progressively declines with diabetes duration [40], which likely requires various treatment regimens. In this review, diabetes duration ranged from 2.5 to 8.8 years. The inclusion of people under various treatment regimens (e.g., oral medication and insulin) could also account for the inconsistency. Moreover, even if CPAP treatment could improve insulin sensitivity in pre-diabetes or non-diabetes, it is also plausible that the same effect would not occur in established T2DM.

Consistent with previous findings, CPAP treatment did not have a significant effect on fasting glucose. Specifically, Yang et al. [14] found that 3 to 24 weeks of CPAP treatment did not improve the fasting glucose in non-diabetic patients compared to pre-CPAP. During CPAP treatment, an increase in growth hormone was observed [41, 42], and growth hormone has long been considered diabetogenic [43]. That might counter the effect of CPAP on the glucose, which helps to explain the negative findings of this review. Additionally, the sample size of included RCTs was typically determined by the primary outcome (i.e., A1C), which might not be large enough to capture the change in fasting glucose. Poor CPAP compliance could also account for the negative findings. That was further supported by the study of Mokhlesi et al. [30] where participants went through a 7-day in-lab CPAP treatment and had an average of 7.92 h CPAP usage. A significant decrease in mean 24-h glucose was observed in the CPAP group compared to the control ($P = 0.01$).

The role obesity plays in the relationship between OSA and glucose metabolism has been inconsistent. Some studies suggested the confounding role of obesity [44, 45], while others suggested that OSA was related to glucose metabolism independent of obesity [3, 46]. BMI has been, traditionally, considered an indicator of obesity. Nevertheless, controlling for BMI is not sufficient when evaluating the effect of CPAP treatment on glucose metabolism [12]. Consistent with previous findings, we did not find a significant effect of CPAP treatment on BMI in this review [14, 18].

To the best of our knowledge, this systematic review is the first that included only RCTs conducted in adults with T2DM and coexisting OSA. This meta-analysis clarifies our understanding about the causal relationship between T2DM and OSA. However, findings from this review need to be interpreted in light of the limitations. First, although we did an exhaustive literature search, the number of studies included in this review remains small, which suggests that more research in this area is needed. Second, insulin sensitivity was not measured using the hyperinsulinemic-euglycemic clamp. The clamp procedure has been considered the "gold standard" [23], but its use is limited in clinical trials due to high cost and complex operation. In this review, limiting the measure of insulin sensitivity to the clamp would result in no eligible RCTs that can be used for the meta-analysis. Third, confounding factors, including medication, eating, and physical activity, were not controlled in this review. CPAP treatment can alleviate OSA-related symptoms (e.g., daytime sleepiness and fatigue), which likely facilitate a healthier lifestyle that is beneficial for glycemic control [12]. Fourth, CPAP usage per night was not high in this review, which might have masked the beneficial effect of CPAP on glucose metabolism.

Findings from this review have important implications for both research and clinical practice. Given the small number of RCTs addressing this issue, more research is warranted. These studies ideally should be large-scale clinical trials with a longer treatment duration and better CPAP compliance. Target population could be those with more severe OSA and poorly controlled diabetes. Factors such as treatment regimen, physical activity, and eating behavior should be included as potential confounders. Although current evidence does not support the beneficial effect of CPAP treatment on glucose metabolism, CPAP is effective in reducing daytime sleepiness, which has a significant impact on daytime functioning. Therefore, timely sustained CPAP treatment of OSA should continue to be encouraged in clinical practice.

Conclusion

In view of the evidence from RCTs, CPAP is effective in alleviating daytime sleepiness. However, current findings do not substantiate a positive effect of CPAP treatment on glucose metabolism (e.g., A1C, insulin sensitivity, fasting glucose, and mean glucose level) in people with T2DM and coexisting OSA.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Informed consent For this type of study formal consent is not required.

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