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Effects of CPAP on energy expenditure in obese obstructive sleep apnoea patients: A pilot study☆

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Summary

We conducted a placebo-controlled crossover pilot study investigating the effects of 2 mo of active and sham continuous positive airway pressure (CPAP) on energy expenditure (EE) via whole-room indirect calorimetry in three obese obstructive sleep apnoea (OSA) patients. Total 24-h (active: 2970 ± 254 kcal/d, sham: 2705 ± 217 kcal/d; $p = 0.015$) and mean sleeping (active: 1.60 ± 0.20 kcal/min; sham: 1.47 ± 0.17 kcal/min; $p = 0.038$) EE were significantly increased after active vs. sham CPAP. Findings suggest that CPAP may correct a hypoxia-related adaptive decrease in thermogenesis.

Keywords

Sleep apnoea; Energy expenditure; Obesity; CPAP; Indirect calorimetry

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Conflict of interest

None of the authors have any conflict of interest related to this manuscript.

Author contributions

AS and M-PS-O designed the study; AS, TF, RR, and M-PS-O performed experiments; AS, TF, RR, and M-PS-O analysed data and interpreted results; AS and M-PS-O drafted the manuscript; AS, TF, RR, and M-PS-O edited and revised manuscript; AS, TF, RR, and M-PS-O approved final version of manuscript

Introduction

The regulation of energy expenditure (EE) in obstructive sleep apnoea (OSA) is complex. Frequent nocturnal arousals, increased respiratory effort, and enhanced sympathetic outflow may increase EE in OSA [1]. However, hypoxemia reduces metabolic rate [2]. Findings on EE in OSA are mixed: some report increases [1,3,4] or no difference [5,6] in sleeping and 24-h EE in patients vs. controls. Few have assessed the effects of continuous positive airway pressure (CPAP) on EE using indirect calorimetry [1,6]. These found that ~2—3 mo of treatment either reduced [1] or had no effect [6,7] on sleeping and resting EE, respectively, vs. base-line. This is the first placebo-controlled crossover study to investigate the effects of CPAP on EE in obese OSA patients using whole-room indirect calorimetry (WRIC).

Methods

Participants were recently diagnosed with OSA but CPAP naïve. Following enrolment, participants began using CPAP at their titrated setting at home. After 2 mo, participants entered the WRIC chamber at ~08:00 for measurements of 24-h EE. WRIC details have been previously described [8]. In the chamber, participants were fed a weight-maintenance diet (30% energy from fat, 55% from carbohydrates, 15% from protein), with energy intake estimated from the Harris—Benedict equation with an activity factor of 1.5 [8]. At 15:15 and 20:15, participants performed 15 min of cycling on a stationary bicycle (65 W, 50 rpm). In-lab sleep was recorded via wrist actigraphy (Actigraph LLC, Pensacola, FL). The first intervention phase was followed by a 1-mo washout (no CPAP). Participants then crossed over into the sham phase, consisting of 2-mo sham treatment and a second 24-h lab visit.

Sham-CPAP devices delivered a sub-therapeutic pressure <1 cm H_2O, measured via gauge manometer, despite normal settings [9]. A flow-restricting connector (BiPAP test adaptor 0.25-inch, Respironics, Murrysville, PA) was attached to the machine outlet. Four extra 4-mm holes were drilled into the mask for expelling excess CO_2. Noise and settings of sham and active devices were identical.

Procedures were approved by the Institutional Review Board of St. Luke’s-Roosevelt Hospital/Columbia University. All participants provided written informed consent.

Results

Participants were 3 obese males, with mild-to-moderate OSA and daytime sleepiness (Table 1).

Twenty-four-hour EE was significantly higher during active vs. sham CPAP (Table 2). Mean EE throughout the wake episode was significantly higher in active vs. sham. Mean EE during the sleep episode was significantly higher in active vs. sham. Mean EE during the afternoon, but not evening, exercise session was significantly higher in active vs. sham. Wake episode EE remained significantly higher in active vs. sham when the exercise sessions were removed.

No difference in sleep latency was observed (Table 2). Significant increases in total sleep time and sleep efficiency were observed in active vs. sham. Wake after sleep onset was significantly lower in active vs. sham.

Discussion

Our finding of a ~9% increase in sleeping EE after active vs. sham CPAP contrasts with the only other study to measure daily EE via WRIC after CPAP, which observed a ~16% decrease in sleeping EE [1]. A sham condition was not used in that study; pre- and post-treatment were compared [1]. Our reported increase in sleeping EE may be related to the adaptive decrease in thermogenesis found in OSA patients [5,10]. Specifically, a negative correlation was reported between time spent in a hypoxic state, indicative of high OSA severity, and daily and sleeping EE [10]. A higher severity of OSA-related hypoxia is also associated with lower than expected daily and sleeping EE [5]. Our results suggest that CPAP may correct this adaptive decrease in thermogenesis.

Although CPAP was applied only during sleep, we observed increased waking EE, which suggests that CPAP may increase activity-induced thermogenesis and exercise thermogenesis. The increase in 24-h EE is not driven solely by increases in exercise EE, since values were still higher during active vs. sham when exercise periods were removed. No imposed standardised exercise regimen was included in the Stenlof et al. study [1], although this is common in WRIC investigations [11]. Furthermore, the diet served while in the chamber by Stenlof et al. was based on a dietary questionnaire reported by participants three days before initial laboratory entry [1]. Diet in our study was calculated based on weight maintenance energy requirements, and balanced for macronutrient content. This is important since OSA severity is associated with increased preference for calorie-rich foods high in fat and carbohydrate [12].

Our small sample size likely contributes to variance in the data, although this may be mitigated by the crossover design. Participant homogeneity may reduce some variability, but may also limit generalisability. This was not a randomised trial: all participants received the active followed by sham phase. Nevertheless, we observed that CPAP increases sleeping and daily EE. Further studies are indicated to replicate findings and assess the impact of CPAP on energy balance.

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Table 1

Participant characteristics at baseline.

Participant	Sex	Age (y)	BMI (kg/m^2)	AHI (events/h)	CPAP pressure setting (cm H_2O)	ESS
1	M	51	31.7	12.2	6	9
2	M	46	33.6	26.5	8	12
3	M	55	30.9	26.6	14	9
Mean ± SD	—	50.7 ± 4.5	32.1 ± 1.4	21.8 ± 8.3	9.3 ± 4.2	10 ± 1.7

Participants were recently diagnosed with obstructive sleep apnoea and had undergone continuous positive airway pressure (CPAP) titration, but had not yet initiated CPAP treatment. Exclusion criteria included apnoea—hypopnoea index >50 events/h, type 2 diabetes, severe hypertension (blood pressure >160/100 mmHg), and any recent near-miss or prior auto accidents. BMI: body mass index; AHI: apnoea—hypopnoea index; ESS: Epworth Sleepiness Scale.

Table 2

The effects of active and sham CPAP on EE and sleep.

	Active CPAP	Sham CPAP	*p*-value
EE			
Total 24-h EE, kcal/d†	2970 ± 254	2705 ± 217	**0.015**
Mean daily EE, kcal/min	2.06 ± 0.18	1.88 ± 0.15	**0.015**
Mean wake episode EE, kcal/min	2.32 ± 0.19	2.11 ± 0.16	**0.011**
Mean wake episode (exercise sessions removed) EE, kcal/min	2.16 ± 0.22	1.96 ± 0.18	**0.017**
Mean sleep episode EE, kcal/min	1.60 ± 0.20	1.47 ± 0.17	**0.038**
Mean exercise EE, session 1, kcal/min	6.64 ± 1.29	6.06 ± 1.29	**0.02**
Mean exercise EE, session 2, kcal/min	7.03 ± 1.42	6.60 ± 1.09	0.151
Sleep			
SOL, min	7.33 ± 2.52	10.67 ± 6.66	0.3
TST, min	441.3 ± 10.02	416.67 ± 12.10	**0.002**
SE, %	92.14 ± 2.48	86.92 ± 2.32	**0.004**
WASO, min	30.3 ± 11.37	52.0 ± 13.77	**0.034**

CPAP: continuous positive airway pressure; EE: energy expenditure; *Total 24-h EE* total 23-h EE extrapolated to 24 h; *Mean daily* mean value throughout the 23-h recording period; *Mean wake episode* mean value throughout times spanning the laboratory wake episode from 8:00 to 23:00; *Mean sleep episode* mean value throughout times spanning the laboratory sleep episode from 23:00 to 07:00. *Mean exercise* mean value throughout times spanning the exercise session in the afternoon (session 1: 15:15—15:30) and evening (session 2: 20:15—20:30). CPAP: continuous positive airway pressure; SOL: sleep onset latency; TST: total sleep time; SE: sleep efficiency; WASO: wake after sleep onset. *p*-values are by paired-samples *t*-tests, with bold indicating statistically significant differences ($p < 0.05$). Data are expressed as mean ± SD, $n = 3$.

†Total daily prescribed energy intake was 2325 ± 187 kcal.