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Effects of continuous positive airway pressure on energy intake in obstructive sleep apnea: A pilot sham-controlled study

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Abstract

Obesity is among the leading risk factors for obstructive sleep apnea (OSA). A reciprocal relationship between obesity and OSA has been proposed, which may be due to excessive food intake. We conducted a pilot study to test the effects of continuous positive airway pressure (CPAP) on energy intake (EI) in OSA patients using a sham-controlled crossover design. In-laboratory total daily EI was assessed after 2 mo of active and sham CPAP. Four men were enrolled (age $\pm$ SEM: 51.8 ± 2.1 y; body mass index: 31.5 ± 1.5 kg/m²). All received active treatment first. Meals (breakfast, lunch, dinner, snack) were served in excess portions at fixed times and additional palatable snacks were freely available throughout the day. Total EI was lower after active (3744 ± 511 kcal/d) vs. sham (4030 ± 456 kcal/d) CPAP but this difference was not significant ($p = 0.51$) due to variability in the free snack intake. When only fixed eating occasions were considered, daily EI was significantly lower in the active (3105 ± 513 kcal/d) vs. sham (3559 ± 420 kcal/d) condition ($p = 0.006$). This small pilot and feasibility study is the first to utilize a sham-controlled design to investigate the effects of CPAP treatment on objective measures of EI. Findings suggest that CPAP may cause a reduction in fixed meal intake. In demonstrating feasibility of study methodology, our study also suggests a larger randomized sham-controlled trial be conducted to fully characterize the effects of CPAP treatment on EI and energy balance overall.

Keywords

Sleep apnea; Food intake; Obesity; CPAP

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Conflict of interest

None of the authors declare any conflicts of interest related to this study.

ClinicalTrials registration

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1. Introduction

Obesity is among the leading risk factors for obstructive sleep apnea (OSA), and OSA may promote weight gain as a result of excessive energy intake (EI) [1]. A hormonal profile that would predispose to high EI appears to exist in OSA patients, who have increased leptin [2–10], suggestive of leptin resistance, and increased ghrelin [4,5,10,11], relative to controls. Increased liking for high-fat foods [12] and preference for calorie-rich foods high in fat and carbohydrate [13] were observed in association with OSA severity. Batool-Anwar et al. investigated the effects of continuous positive airway pressure (CPAP) on food intake in OSA patients using a Food Frequency Questionnaire (FFQ), and found that CPAP treatment reduced consumption of trans-fat in women, but did not affect overall EI [14]. However, some authors suggest that subjective tools like the FFQ may in fact lead to inaccurate conclusions, including a systematic underestimation of actual EI, and their use should be abandoned [15]. It is therefore critical to conduct systematic investigations of how CPAP affects EI using objective measures under real-life and laboratory conditions. To our knowledge, the current pilot study is the first to utilize ad libitum food intake monitoring as an objective laboratory-based behavioral measure of food intake in OSA patients in response to treatment with CPAP using a placebo-controlled crossover design.

2. Materials and methods

Participants were overweight ($n = 1$) or obese ($n = 3$) adult males, who had been diagnosed with OSA (apnea-hypopnea index [AHI] ≥ 5), and underwent a CPAP titration, but not yet initiated CPAP. Exclusion criteria were AHI > 50 , type 2 diabetes, untreated severe hypertension, being a habitual driver, or having recent near-miss or auto accidents. Participants agreed to refrain from driving throughout the study. Eating disorders were not ruled out with formal screening tools. However, in a taking of medical history, none of the participants indicated a positive response when asked if they have problems with eating or their appetite. Baseline characteristics are presented in Table 1. Procedures were approved by the Institutional Review Boards of St. Luke's-Roosevelt Hospital and Columbia University, and were conducted in accordance with the Declaration of Helsinki. All participants provided written informed consent before enrollment.

This was a placebo-controlled crossover study modeled after prior designs [16]. After enrollment, participants began using CPAP at their titrated setting (active) at home each night for 2 mo, after which, participants became inpatients for a 2-d period. On the first inpatient day, participants were fed a controlled weight maintenance diet [17]. Macronutrient composition was 30% energy from fat (12.5% saturated fat), 55% energy from carbohydrates, and 15% energy from protein. Meal times were fixed at 09:00, 13:00, 16:00, and 19:00, for breakfast, lunch, snack, and dinner, with 30% of daily energy requirements coming from each meal and 10% from the snack. Participants used CPAP at their prescribed setting during the scheduled in-lab sleep episode (23:00–7:00).

Ad libitum EI was measured throughout the waking period on laboratory day 2. Meal content was the same as on day 1, but food items were served in excess (100% more than baseline). Fixed meals (breakfast, lunch, snack, dinner) were served at the same times as day

1, and additional palatable snack choices (“free snacks”: Oreos, Welch's fruit snacks, M&Ms., Cheez-Its, Hershey's kisses, chocolate chip cookies, dried cranberries, almonds, trail mix) were freely available. This approach has been utilized to assess the effects of sleep restriction on ad libitum food intake [18–20]. Participants were instructed to eat until they felt comfortably full. All foods were dispensed by research personnel and weighed, pre and post consumption after each eating episode. EI was analyzed using University of Minnesota Nutrition Data System for Research. Participants were discharged from on the morning of day 3.

Sleep in the laboratory was recorded with the GT3x ActiGraph monitoring device (Actigraph LLC, Pensacola, FL), a tri-axial accelerometer, worn on the non-dominant wrist. Sampling rate for the device was 60 Hz, and data were reintegrated into 60-s epochs for scoring and analysis purposes. Data were analyzed with Actilife5 software (Actigraph LLC, Pensacola, FL) to determine total sleep time (TST), sleep efficiency (SE), and wakefulness after sleep onset (WASO) using Cole-Kripke criteria. Participants also completed the functional outcomes of sleep questionnaire (FOSQ), a 30-item questionnaire to assess impact of sleep disorders on various daytime domains, and had measures of body mass index (BMI) taken at baseline and after the active and sham CPAP phases.

After a 1-mo washout, participants crossed over into the sham phase. Participants were provided with the sham CPAP devices, and instructed to use the CPAP at the titrated level for 2 mo. This was followed by the second laboratory period (repeat of first phase). After the second phase, participants were debriefed and instructed to return to active CPAP.

Sham CPAP devices were constructed as done by others [16]. A flow-restricting connector (BiPAP test adaptor 0.25-inch, Respironics, Murrysville, PA) was attached to the machine outlet and the front of the tubing. Four extra 4-mm holes were drilled into the mask near the portholes to allow participants to expel excess CO₂. Pressure was set at the individual's normal titrated (active) setting. Modified CPAP devices delivered a pressure < 1 cm H₂O, measured via gauge manometer (TMS-13700, Tiara Medical Systems, Lakewood, OH), confirming sham devices delivered sub-therapeutic pressures. Sham devices were well-tolerated with no reported adverse events. Sham devices were identical to active devices in terms of noise and settings. Components of the CPAP system manipulated to produce the sham CPAP were replaced with functional parts so that active treatment could resume after the study.

Two-tailed paired-samples *t*-tests were used to compare variables between active and sham conditions. Data are expressed as mean ± SEM.

3. Results

Individual and mean BMI and FOSQ scores at baseline and after active and sham CPAP phases are shown in Table 2. No differences were seen between conditions.

Mean total ad libitum EI including fixed meals and free snacks was 3744 ± 511 kcal in active and 4030 ± 228 kcal in sham ($t_3 = -0.74$, $p = 0.51$, Cohen's $d = 0.36$). Three of the four participants increased their total daily EI during sham vs. active, whereas one

participant showed a decrease (Fig. 1). A large degree of variability in contributing to daily EI was introduced by the free snacks (Fig. 2). EI from free snacks ranged from 24 to 1644 kcal. The participant who decreased his EI after sham vs. active CPAP consumed 1644 kcal from free snacks during active CPAP. This represents 31.3% of his total daily EI intake, which was higher than his percent daily energy consumed during breakfast (22.0%), lunch (17.0%), dinner (22.5%) and snack (7.2%). We therefore conducted an analysis of total daily ad libitum EI from fixed meals after removing EI from free snacks. When free snacks were removed from the daily totals, total EI from fixed meals (breakfast, lunch, snack, and dinner) was 3105 ± 257 kcal in active and 3559 ± 210 kcal in sham CPAP ($t_3 = -6.99$, $p = 0.006$, Cohen's $d = 0.97$). In this analysis, total daily EI increased for all participants during sham vs. active CPAP (range of between-group differences: 325–636 kcal) (Fig. 3). Similar results were observed when only breakfast, lunch, and dinner were considered, i.e. when fixed and free snacks were removed (active: 2875 ± 286 kcal/d vs. sham: 3371 ± 249 kcal/d, $t_3 = -9.95$, $p = 0.002$; Cohen's $d = 0.92$; range of between-group differences: 420–642 kcal).

Sleep quality was generally better during the active vs. sham condition when measured in the laboratory, although with the small sample size these differences were not statistically significant. Specifically, in active vs. sham, higher SE (90.1 ± 2.0 vs. $87.2 \pm 1.4\%$, $t_3 = 1.35$, $p = 0.27$) and TST (431.9 ± 9.7 vs. 417.9 ± 7.0 min, $t_3 = 1.40$, $p = 0.26$), and lower WASO (39.9 ± 8.6 vs. 52.8 ± 7.0 min, $t_3 = -1.35$, $p = 0.27$), were observed. Significant correlations were seen between sleep measures and EI when considering only breakfast, lunch, and dinner (SE: $r = -0.73$, $p = 0.04$; TST: $r = -0.75$, $p = 0.04$; WASO: $r = 0.72$, $p = 0.05$). These correlations of sleep measures with EI became trends when considering breakfast, lunch, dinner and fixed snack (SE: $r = -0.61$, $p = 0.10$; TST: $r = -0.63$, $p = 0.10$; WASO: $r = 0.62$, $p = 0.10$), and were no longer significant when considering fixed meals and free snacks (SE: $r = -0.08$, $p = 0.86$; TST: $r = -0.12$, $p = 0.73$; WASO: $r = 0.15$, $p = 0.73$).

4. Discussion

To our knowledge, this is the first study to objectively monitor EI after CPAP. We observed that active CPAP decreased EI vs. sham in 3 of the 4 participants when considering fixed meals and free snacks. When only fixed meals were considered (breakfast, lunch, snack, dinner), EI was significantly reduced in the active vs. sham condition in all participants, and this reduction in EI reached statistical significance. This statistically significant difference was found to have a large effect size, and considering that an energy deficit of 500 kcal/d is desirable to elicit weight loss [21], the observed difference of ~450 kcal represents a clinically meaningful change that should encourage further study in a larger trial.

Interestingly, as observed here, the effects of CPAP on EI from fixed meals seem to be more pronounced than that of free snacks. This is reminiscent of findings from Hogenkamp et al. that, in a fasted state, portion size preferences for both meals and snack foods were increased after sleep deprivation, whereas in the satiated state (i.e. following a 650 kcal breakfast), portion size preferences were increased for snacks but not meals following sleep deprivation [22]. This appears to represent the existence of an increase in homeostatic drive (represented by fixed meals), as well as a secondary hedonic or non-homeostatic mechanism of increased food intake (represented by snacks), after sleep disruption. This may further suggest that

CPAP affects EI by targeting the homeostatic as opposed to hedonic regulation of EI. Indeed, CPAP may alter EI by normalizing levels of hunger/satiety hormones, as was reported after CPAP use for ghrelin [4,5,11,23] and leptin [2,5,6,24–29]. Based on reports of increased activation in brain regions involved in motivation and reward in response to food stimuli after sleep restriction [30], future investigations should extend beyond homeostatic/hormonal mechanisms to explore if and how CPAP treatment affects the hedonic and/or cognitive controls of food intake, using functional magnetic resonance imaging.

As this is to our knowledge the first time actual measures of food intake were documented in response to CPAP, it remains unclear if a 2 mo treatment is necessary to induce effects on EI. In several studies which documented changes in hunger/satiety hormones such as leptin and ghrelin, effects have been seen in as little as 1–3 d of CPAP [25,29,31]. However, in other reports, leptin was unchanged at 2 d but reduced after 3 mo [32] and ghrelin was unchanged at 3–4 d but reduced at 1 mo [11] post treatment. It is also unclear how long sham CPAP would be needed to reverse these effects, although withdrawal of CPAP was associated with a recurrence of high AHI within a few days and an increase in sleepiness by 2 wk. [33]. Related to this, although we observed no effect on BMI after 2 mo CPAP use, the question of whether CPAP affects body weight and composition is still controversial. A recent meta-analysis of 25 controlled trials reported that CPAP use is associated with a statistically significant although modest ($+0.13 \text{ kg/m}^2$) increase in BMI [34]. However, a decrease in visceral fat was observed after 6 mo of CPAP in individuals who either did or did not also show decreased BMI after treatment [25]. Future studies should therefore look at other components of body composition in addition to BMI, and should take into account outcomes of interest when determining duration of treatment.

Current sample size was small, although the within-subject/crossover design may mitigate some of this effect. This was not a randomized trial, so order effects cannot be excluded. The main reason for a lack of randomization is that in this small pilot study which was designed to establish feasibility of procedures and possible effects of treatment, participants were all enrolled in the active phase in a similar timeframe while the logistics and constructing the sham devices was completed. However, a baseline day was included in both phases during which participants were served a fixed weight-maintenance diet. The same food items were served in excess on the ad libitum day, and the same menu was served at both study phases. Nevertheless, a novelty effect whereby participants overate during the first phase might have been observed in one participant who consumed $\sim 30\%$ of their total daily EI from the free snacks selectively during the active phase. In a sub-analysis of total daily EI, these free snacks were removed from group analyses. The use of sham CPAP may not be without consequence, as its use may reduce sleep efficiency, increase arousal index, increase the number of hypopneas, and decrease the number apneas [35]. However, the authors of that report suggest those effects were of minimal clinical significance, and still support sham CPAP as the placebo of choice in controlled trials [35]. A further limitation of the study is that duration of nightly CPAP use during the treatment phases was not objectively monitored. Thus we do not have information on compliance during the sleep episodes outside the laboratory. A follow-up study should also include polysomnographic sleep recordings to document changes in AHI and sleep architecture.

The current pilot study was effective in demonstrating the feasibility of recruiting newly diagnosed but CPAP naïve participants and of retaining them throughout study phases. In terms of methodology, the laboratory-based ad libitum food intake protocol is considered ideal to objectively test and quantify the effects of interventions on EI [36]. Although used to investigate the effects of sleep restriction on EI [18–20, 37–39], this is the first time such a protocol has been used to document the effects of CPAP on food intake. In this small group of participants, we observed that active CPAP treatment can decrease EI consumed from meals served in the laboratory. Further studies with larger sample sizes are indicated to replicate our findings and assess the impact of CPAP treatment on energy balance. A better understanding of how CPAP affects EI, via a larger-scale follow-up randomized-controlled trial, will clarify the factors contributing to increased weight gain in OSA, and will help optimize treatments for weight loss in OSA patients.

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HIGHLIGHTS

- The presence of obstructive sleep apnea (OSA) encourages weight gain in patients.
- This was a sham-controlled pilot study to test the effects of CPAP on energy intake (EI).
- Ad libitum EI was assessed by serving meals in excess after active and sham phases.
- Daily EI from meals was significantly lower in the active vs. sham condition.
- Findings suggest that CPAP may cause a reduction in total daily food intake.

Fig. 1.

Total daily energy intake (EI) from fixed meals (breakfast, lunch, snack, dinner) and free snacks after 2 mo of active and sham CPAP. Data are shown for individuals and for group mean ($\pm$ SEM). Numbers above individuals represent difference in EI between conditions (sham – active). p-Value is by two-tailed paired sample *t*-test.

Fig. 2. Energy intake (EI) from free snacks after 2 mo of active and sham CPAP. Data are shown for individuals and for group mean ($\pm$ SEM). Numbers above individuals represent difference in EI between conditions (sham – active). p-Value is by two-tailed paired sample *t*-test.

Fig. 3.

Total daily energy intake (EI) from fixed meals only (breakfast, lunch, snack, dinner) after 2 mo of active and sham CPAP. Data are shown for individuals and for group mean ($\pm$ SEM). Numbers above individuals represent difference in EI between conditions (sham – active). p-Value is by two-tailed paired sample *t*-test.

Table 1

Individual baseline characteristics of participants. AHI: apnea-hypopnea index; CPAP: continuous positive airway pressure; ESS: Epworth sleepiness scale; M: male; SEM: standard error of the mean.

Participant	Sex	Age (y)	ESS	AHI (events/h)	CPAP pressure setting (cm H ₂ O)
1	M	51	9	12.2	6
2	M	46	12	26.5	8
3	M	55	9	26.6	14
4	M	55	7	14.0	7
Mean ± SEM	–	51.8 ± 2.1	9.3 ± 1.0	19.8 ± 3.9	8.8 ± 1.8

Table 2

Body mass index (BMI) and functional outcomes of sleep questionnaire (FOSQ) at baseline and after the active and sham CPAP treatment phases. SEM: standard error of the mean.

Participant	Baseline BMI (kg/m ²)	Post-active CPAP BMI (kg/m ²)	Post-sham CPAP BMI (kg/m ²)	Baseline FOSQ	Post-active CPAP FOSQ	Post-sham CPAP FOSQ
1	32.5	32.3	32.0	10.2	9.5	12.3
2	34.4	34.8	34.1	16.8	17.0	12.9
4	27.2	27.5	27.2	17.5	15.4	17.5
Mean ± SEM	31.5 ± 1.5	31.4 ± 1.5	31.3 ± 1.5	16.0 ± 2.0	15.3 ± 2.1	15.0 ± 1.4