https://doi.org/10.5664/jcsm.8322

JCSM | Journal of Clinical Sleep Medicine

SCIENTIFIC INVESTIGATIONS

Effects on obstructive sleep apnea severity following a tailored behavioral sleep medicine intervention aimed at increased physical activity and sound eating: an 18-month follow-up of a randomized controlled trial

Sören Spörndly-Nees, PhD[1]; Pernilla Åsenlöf, PhD[1]; Eva Lindberg, MD, PhD[2]; Margareta Emtner, PhD[1,2]; Helena Igelström, PhD[1,2]

[1]Department of Neuroscience, Physiotherapy, Uppsala University, Uppsala, Sweden; [2]Department of Medical Sciences, Respiratory, Allergy, and Sleep Research, Uppsala University, Uppsala, Sweden

Study Objectives: Positive effects have been reported following a behavioral sleep medicine (BSM) intervention targeting physical activity and eating behavior in addition to continuous positive airway pressure (CPAP) treatment in patients with obstructive sleep apnea (OSA). Long-term follow-up remains to be explored. The aim was to examine the long-term effects of a tailored BSM intervention addressing physical activity and eating behavior in addition to CPAP treatment in patients with moderate to severe OSA combined with overweight and physical inactivity. Further, the aim was to identify variables at baseline, associated with treatment success regarding OSA severity.

Methods: Sixty participants (body mass index: 34.5 ± 5.0 kg/m^2; apnea-hypopnea index [AHI]: 43.7 ± 21.2 events/h) completed the randomized controlled trial with a follow-up at 18 months. The participants were randomized to either a control group treated with CPAP or an experimental group treated with CPAP and a BSM intervention targeting physical activity and eating behavior changes. OSA was categorized as mild (AHI: 5 to <14.9 events/h), moderate (AHI: 15 to <29.9 events/h), or severe (AHI ≥30 events/h).

Results: Being in the experimental group was associated with a larger improvement (B = −9.353, *P* = .029) in AHI at the 18-month follow-up compared with being in the control group when adjusting for baseline AHI and body mass index. Improvement in OSA category occurred more frequently in the experimental group participants (n = 11; 36.7%) compared with the control group (n = 2; 6.7%). Deterioration in OSA category was found in 1 (3.3%) participant in the experimental group and 3 (10%) in the control group.

Conclusions: The importance of a BSM intervention as an adjunct treatment in patients with OSA is emphasized due to its long-term benefits.

Clinical Trial Registration: Registry: ClinicalTrials.gov; Name: Lifestyle changes in obstructive sleep apnea; Identifier: NCT01102920.

Keywords: physical activity, eating habits, obstructive sleep apnea, behavioural sleep medicine

Citation: Spörndly-Nees S, Åsenlöf P, Lindberg E, Emtner M, Igelström H. Effects on obstructive sleep apnea severity following a tailored behavioral sleep medicine intervention aimed at increased physical activity and sound eating: an 18-month follow-up of a randomized controlled trial. *J Clin Sleep Med*. 2020;16(5): 705–713.

BRIEF SUMMARY

Current Knowledge/Study Rationale: We have reported improvements in obstructive sleep apnea (OSA) severity after a behavioral sleep medicine intervention that aimed to increase physical activity and healthy eating in patients with OSA and overweight. Knowledge on the potential additional effects of behavior change in combination with continuous positive airway pressure treatment on a long-term basis is of clinical importance.

Study Impact: The intervention effects in reducing the apnea-hypopnea index and OSA severity in the long term were evident in a cluster of individuals, characterized by lower baseline apnea-hypopnea index and percentage body fat, higher physical activity level, and better glucose control. These results may help the clinician in selecting treatment alternatives in addition to the continuous positive airway pressure treatment, wherein the patient with more severe OSA may need more support than that offered in the current behavioral sleep medicine intervention.

INTRODUCTION

Obstructive sleep apnea (OSA) is a highly prevalent condition[1] associated with quality-of-life impairment as well as cardiovascular diseases and mortality.[2,3] Continuous positive airway pressure (CPAP) is considered as the most effective treatment for the condition and is currently the standard treatment.[4,5] Not all patients tolerate this treatment and adherence is often low,[6] limiting the overall effectiveness of CPAP.[7] Recent reviews, presenting studies on physical exercise as treatment of OSA, report an independent effect of physical exercise on OSA severity and daytime sleepiness.[8–10] Diet restrictions in patients with obesity and OSA have been found to induce weight loss and significantly reduce OSA symptoms.[10,11] Given the association between OSA and overweight and obesity,[12,13] it may be important to combine CPAP treatment with behavioral interventions targeting unhealthy eating behavior and sedentary lifestyle. Physical activity and dietary changes may, as such, be a treatment alternative or an adjunct treatment to CPAP treatment. Knowledge on potential additional effects of behavior

Downloaded from jcsm.aasm.org by 65.21.193.86 on February 24, 2022. For personal use only. No other uses without permission.
Copyright 2022 American Academy of Sleep Medicine. All rights reserved.

change, in combination with CPAP treatment on a long-term basis, is of clinical importance. We have reported improvements in OSA severity on 6-month follow-up after a behavioral sleep medicine (BSM) intervention that aimed to increase physical activity and healthy eating in patients with OSA and overweight.[14] However, the long-term effects of the intervention on OSA severity remain to be reported.

The overall aim was to examine whether a tailored BSM intervention addressing physical activity and eating habits has any additional effects on CPAP treatment at 18-month follow-up in patients with moderate to severe OSA and overweight or obesity. Furthermore, the aim was to identify variables at baseline associated with treatment success regarding OSA severity.

METHODS

The study was a prospective randomized controlled trial with follow-up at 18 months. The participants were randomized to either a control group treated with CPAP or an intervention group treated with CPAP and a BSM intervention targeting physical activity and eating behavior changes (see flow chart in **Figure 1**). Data were collected from March 2010 to March 2012. The study was approved by the local ethics committee in Uppsala, Sweden (dnr2009/004), and is registered at ClinicalTrials.gov (clinical trial number NCT01102920).

Procedure

Patients were considered eligible if overweight (body mass index [BMI] ≥25 kg/m^2) and diagnosed with OSA with an apnea-hypopnea index (AHI) ≥15 events/h and prescribed first-time CPAP treatment. Eligible patients were consecutively addressed by the examining physician at the sleep clinic at Uppsala University Hospital and were provided written information about the study and asked about their interest in study participation. Patients who volunteered were interviewed by phone regarding their leisure-time physical activity. Those reporting a physical activity level less than the recommended 150 minutes per week of moderate physical activity[15] were scheduled for an appointment with a study nurse for written consent and baseline measurements.

Data collection

All measurements were done by a study nurse blinded to the study condition. Sociodemographic data including age, sex, civil status, and education were collected by questionnaires at baseline. Information on comorbidities included self-reported type 2 diabetes mellitus, heart failure, hypertension, and lung diseases (chronic obstructive pulmonary disease, asthma, lung fibrosis, and cancer). Assessments of study variables were made at baseline and at 18-month follow-up.

OSA severity

AHI was assessed by ambulatory recording using the type 3 Embletta device (Embletta Systems) at baseline and follow-up. Signals recorded and included for analysis were nasal pressure through a nasal cannula attached to a pressure transductor oronasal thermistor, pulse oximetry, piezoelectric belts for respiratory effort, and body position. At follow-up, the participants were instructed not to use CPAP for 3 consecutive nights prior to the recording. The recordings were blinded to the physician who scored and analyzed the data (E.L.). Apneas were defined as cessation of airflow in nasal pressure for ≥10 seconds with continued abdominal and thoracic movements. Hypopneas were defined as ≥50% reduction in baseline airflow for ≥10 seconds combined with an oxygen desaturation of ≥3%.[16] AHI was calculated as the number of events per hour of sleep. Registrations were considered valid when exceeding 240 min of recording time. OSA severity was categorized into the following AHI groups: no OSA (AHI: 0–4.9 events/h), mild OSA (AHI: 5–14.9 events/h), moderate OSA (AHI: 15–29.9 events/h), and severe OSA (AHI ≥30 events/h). Treatment success was defined as the transition of an OSA severity category to a lower OSA category.

Physical activity

Physical activity and sedentary time were assessed by a SenseWear Armband Pro 3 (BodyMedia, Inc.) worn by the participants for 7 consecutive days at baseline and follow-up. This device objectively registers the participants' level of physical activity and, with the use of metabolic equivalents (METs), allows for classification of time spent in sedentary time and moderate or vigorous physical activity corresponding to <1.5 METs, 3 METs, or >6 METs, respectively. SenseWear Professional software v8.1 (BodyMedia, Inc.) was used for calculating sedentary time, number of steps, and moderate-to-vigorous physical activity (MVPA). Data were considered valid when comprising 10 hours of data per day from ≥90% of the waking time for ≥4 days.[17]

Eating habits

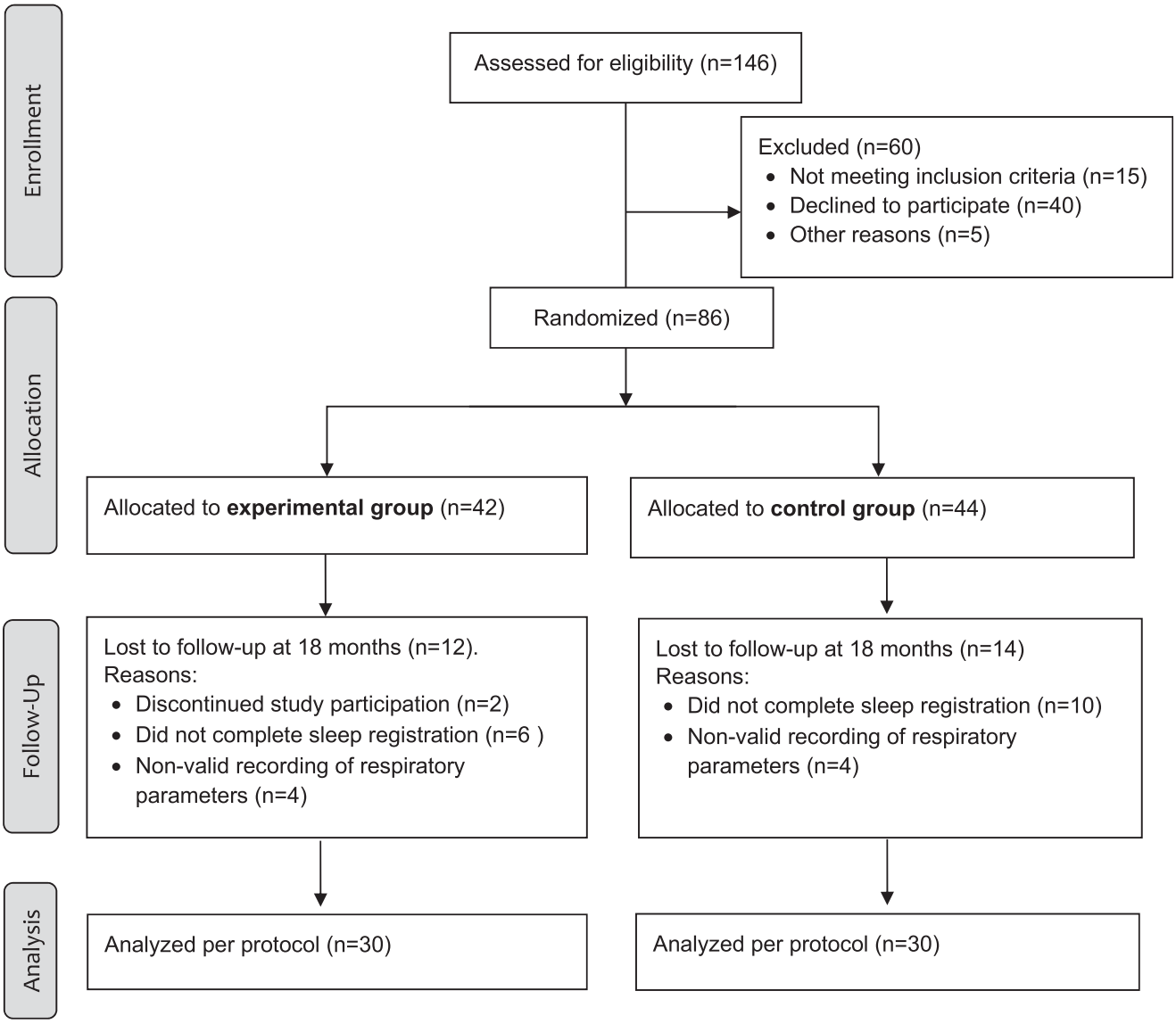
The Swedish National Board of Health and Welfare has developed guidelines regarding disease prevention and questionnaires on lifestyle factors to screen for individuals at risk for disease due to unhealthy habits, including eating habits.[18] The individual is asked to reflect on his/her eating habits during a normal week regarding consumption of the following: (1) vegetables and/or root crops (fresh, frozen, or prepared); (2) fruit and berries (fresh, frozen, or prepared); (3) fish or shellfish as a main course; and (4) buns/cake with coffee, chocolate/candy, chips, or soda/cordial. Each item included 4 categorical answers from 0 (= once a week [items 1 and 2], a couple of times per month [item 3]) to 3 (= twice a day or more often [items 1 and 2], 3 times per week [item 3]). In item 4 the score was reversed; thus, ranging from 0 (= every day) to 3 (= a couple times per month or less). A total score, ranging from 0 to 12, was calculated. A value of <4 points indicates unhealthy eating, whereas individuals with a score of ≥9 points mainly adhere to the Swedish Nutrition Recommendations.

Anthropometrics

Data were collected on body weight in kilogram (kg) and height, waist circumference, neck circumference, and sagittal abdominal diameter in centimeters (cm). Participants wore light clothing without shoes during the measurements. BMI was

Downloaded from jcsm.aasm.org by 65.21.193.86 on February 24, 2022. For personal use only. No other uses without permission.
Copyright 2022 American Academy of Sleep Medicine. All rights reserved.

Figure 1—Flow chart of study participation from recruitment to follow-up at 18 months.



calculated as weight divided by height squared. The amount of body fat in percentage was measured using the Tanita BC-418MA Body Composition Analyzer (Tanita Corporation of America) and rounded to 1 decimal point.

Neck circumference was measured in an upright position with a measuring tape around the neck just below the larynx. Both waist circumference (standing position) and sagittal abdominal diameter (supine) were measured midway between the lower rib and the anterior superior iliac crest at the end of a normal exhalation.

CPAP adherence

At follow-up, CPAP adherence was assessed by questioning how many hours per night and nights per week the participants used their CPAP device. Compliance was defined as mean CPAP usage ≥4 hours/night, ≥5 nights/week, and noncompliance was defined as CPAP usage <4 hours/night and/or <5 nights/week.

Daytime sleepiness

Daytime sleepiness was assessed using the Swedish version of the Epworth Sleepiness Scale.[19,20] The perceived risk of dozing off was assessed in 8 different situations on a 4-point scale, with 0 meaning "Would never doze off" and 3 indicating a "High chance of dozing off" in the specific situation. The total sum of scores ranged from 0 to 24 points, and scores >10 indicated excessive daytime sleepiness.[21]

Readiness for change

In 2 separate questions related to the targeted behaviors, the participants scored their readiness to change their behavior regarding engagement in physical activity and eating habits on a 5-point graded scale: (1) precontemplation stage (not being ready for change), (2) contemplation (considering change), (3) preparation (preparing for change), (4) action (taking action for change), and (5) maintenance stage (maintaining change).[22]

Downloaded from jcsm.aasm.org by 65.21.193.86 on February 24, 2022. For personal use only. No other uses without permission.
Copyright 2022 American Academy of Sleep Medicine. All rights reserved.

Self-efficacy

Participants' self-efficacy for physical activity was assessed with the Exercise Self-Efficacy Scale.[23] The confidence in engaging in physical activity in 18 specific situations was scored using 18 numerical rating scales (NRSs), ranging from 0 (not being confident) to 10 (being highly confident). The total sum score ranged from 0 to 180 points, where higher scores indicated higher self-efficacy.

Further, self-efficacy was assessed on 2 separate NRSs, answering how confident the participants were in their ability to enhance their physical activity level and improve their eating habits.

Fear of movement/activity avoidance

Selected parts of the Tampa Scale of Kinesiophobia (TSK)[24] were used to assess fear of movement/activity avoidance. The TSK was originally developed to capture perceptions regarding fear of movement and (re)injury when in pain. Seven of the original 17 questions were chosen and included the following:

1. I am afraid that I might injure myself if I exercise.
2. My body is telling me I have something dangerously wrong.
3. Pain always means I have injured my body.
4. I am afraid that I might injure myself accidentally.
5. It is really not safe for a person with a condition like mine to be physically active.
6. I cannot do all the things normal people do because it is too easy for me to get injured.
7. No one should have to exercise when he/she is in pain.

Scoring was done through a 4-point Likert scale with the anchors 1 (= I do not agree at all) and 4 (= I totally agree), and the total sum score ranged from 7 to 28 points. Higher scores indicated more fear of movement/activity avoidance.

Depressive symptoms

The Montgomery Åsberg Depression Rating Scale[25] was used to assess depressive symptoms. The individual was asked to reflect on the 3 previous days and answer 9 items, including common symptoms of depression ranging from 0 (no symptoms) to 6 (severe symptoms). The total sum score ranged from 0 to 54 points. A score of 0–6 indicated no depression, 7–19 indicated mild depression, 20–34 indicated moderate depression, and >34 indicated severe depression.[26]

Study conditions

Participants were randomly allocated to either a control group or experimental group. The randomization was administered by a senior researcher (P.Å.) after the baseline assessment. Participants were randomized in blocks of 10 individuals, using a random integer generator (www.random.org).

Control group

All participants were informed about the association between overweight and OSA and were recommended to lose weight and increase their physical activity level. A CPAP nurse taught the participants how to use the CPAP device; then, after 5–14 nights of using auto-CPAP, the participants met with the CPAP nurse and decided the final CPAP pressure.

Experimental group

The experimental group received an introduction and adjustment to CPAP similar to the control group. In addition, a BSM intervention aiming to enhance physical activity and sound eating habits was provided. The participant received 8–10 individual visits to a dietitian and physiotherapist during the first 6 months.

In short, the behavioral intervention included 7 components: (1) preparation for action and motivational analysis; (2) behavior goal setting; (3) action planning; (4) self-monitoring; (5) review of behavioral goals, action plans, and functional behavioral analysis; (6) identification of barrier and problem solving; and (7) maintenance, relapse prevention and coping planning.

Between the 6th and 18th month, 4 additional booster sessions took place, including contact per telephone twice and 2 face-to-face meetings. The aim of the booster sessions was to enhance adherence to the targeted behavioral changes. At each booster session, the individual's maintenance plan from the intervention was reviewed. After problem solving, new behavioral goals and strategies for relapse prevention and coping planning were formed, when needed.

The components were similar for all participants in the experimental group, but the content was adjusted and developed to fit the individual. More details on the components of the BSM intervention have been described previously.[27]

Adherence to BSM intervention

During the first 6 months of the study, the participants attended 8.8 (SD: 1.3) sessions. One participant dropped out of treatment after 4 sessions and did not attend any booster sessions. Of the remaining participants, all 4 booster sessions were completed by 26 participants (86.7%). When finishing the intervention, all participants had completed the first 6 components, and 29 (96.7%) completed all 7 components.

Data analysis

Data are presented as means (SDs), medians (interquartile ranges), or as numbers and proportions (%).

The Mann-Whitney *U* test and chi-square/Fisher's exact test were used for comparative statistics for data not normally distributed and nonparametric and categorical data.

For AHI as a continuous dependent variable, a multiple linear regression was performed including study condition, baseline AHI, and baseline BMI to allow for analysis of the influence of these variables. The significance level was set at $P < .05$. According to the power calculation, 144 participants were required in order to detect a between-group difference of 20%. However, due to time constrictions, inclusion was ended when 86 participants had entered the study.

RESULTS

For the 18-month follow-up, 60 participants were eligible for analysis, 30 in the control and intervention groups, respectively (**Table 1**, **Figure 1**). Those not completing follow-up measurements did not differ from those included in the final analysis regarding age, BMI, AHI, and daily average MVPA at baseline. The trajectories regarding AHI at baseline and the 6-month and

Downloaded from jcsm.aasm.org by 65.21.193.86 on February 24, 2022. For personal use only. No other uses without permission.
Copyright 2022 American Academy of Sleep Medicine. All rights reserved.

Table 1—Participants' characteristics at baseline.

	All (n = 60)	Experimental Group (n = 30)	Control Group (n = 30)
Age, mean (±SD), years	54.4 (±11.4)	56.5 (±11.9)	52.2 (±10.6)
Sex, n (%)			
Male	49 (81.7)	26 (86.7)	23 (76.7)
Female	11 (18.3)	4 (13.3)	7 (23.3)
Civil status, n (%)			
Married/cohabitant	44 (73.3)	21 (70.0)	23 (76.7)
Living apart	3 (5.0)	1 (3.3)	2 (6.7)
Single/living alone	13 (21.7)	8 (13.3)	5 (16.7)
Education, n (%)			
Elementary school	14 (23.3)	6 (20.0)	8 (26.7)
High school	31 (51.7)	17 (56.7)	14 (46.7)
College	15 (25.0)	7 (23.3)	8 (26.7)
Occupation, n (%)			
Working	44 (73.3)	19 (63.3)	25 (83,3)
Retired	12 (20.0)	8 (26.7)	4 (13,3)
On sick leave	1 (1.7)	0 (0.0)	1 (3.3)
On sickness benefits	3 (5.0)	3 (10.0)	0 (0.0)
Smoking, n (%)			
Never smoked	32 (53.3)	13 (43.3)	19 (63.3)
Quit smoking	22 (36.7)	13 (43.3)	9 (30.0)
Smoking	6 (10.0)	4 (13.3)	2 (6.7)
Comorbidities, n (%)			
Type 2 diabetes	10 (16.7)	7 (23.3)	3 (10.0)
Heart failure	2 (3.3)	1 (3.3)	1 (3.3)
High blood pressure, hypertension	34 (56.7)	18 (60.0)	16 (53.3)
Lung disease (eg, COPD, asthma, lung fibrosis, cancer)	7 (11.7)	2 (6.7)	5 (16.7)
AHI, mean (±SD), events/h	43.7 (±21.2)	42.2 (±19.5)	45.1 (±23.0)
OSA severity, n (%)			
Moderate (AHI 15–29.9 events/h)	20 (33.3)	9 (30.0)	11 (36.7)
Severe (AHI >30 events/h)	40 (66.7)	21 (70.0)	19 (63.3)
Epworth Sleepiness Scale, median (IQR)	13 (6.8)	13.5 (4.8)	13 (8.3)
BMI, mean (±SD), kg/m^2	34.5 (±5.0)	35.9 (±5.3)	33.1 (±4.4)
Percentage body fat, mean (±SD)	33.6 (±6.0)	34.3 (±5.5)	33 (±6.4)
Daily average minutes of MVPA, n	51	28	23
Mean (±SD)	75.3 (±50.9)	71.5 (±44.7)	80 (±58.2)

Data are presented as mean (±SD), median (IQR), or n (%). AHI = apnea-hypopnea index; BMI = body mass index; COPD, chronic obstructive pulmonary disease; IQR = interquartile range; MVPA = moderate-to-vigorous physical activity; OSA = obstructive sleep apnea.

18-month follow-up for the intervention and the control groups are presented in **Figure 2**.

Intervention effect

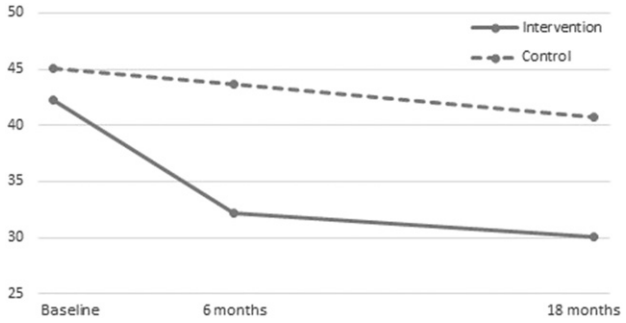
Participants in the intervention group were significantly more likely to improve AHI during the 18-month study period. According to the multiple linear regression analysis, study group and baseline values of AHI and BMI together explained 47.6% of the variance in AHI at the 18-month follow-up ($F = 16.99$, $df = 3$, $P < .0001$). Being in the intervention group implied a larger improvement in AHI at follow-up compared with being in the control group, when adjusted for baseline AHI and BMI ($P = .029$). As expected, a higher AHI at baseline was associated with higher AHI after 18 months ($P < .0001$), whereas baseline BMI was not associated with the outcome at 18 months (**Table 2**).

Changes in OSA severity from baseline to 18-month follow-up

Improvement in OSA severity occurred more frequently in the participants in the intervention group compared with those in the

Downloaded from jcsm.aasm.org by 65.21.193.86 on February 24, 2022. For personal use only. No other uses without permission.
Copyright 2022 American Academy of Sleep Medicine. All rights reserved.

Figure 2—AHI at baseline and the 6-month and 18-month follow-ups for the intervention and the control group.



AHI = apnea-hypopnea index.

Table 2—Multiple linear regression analysis of study group, AHI, and BMI at baseline on AHI at the 18-month follow-up.

Independent Variable	B	SE	*P* Value	95% CI for B	
				Lower	Upper
Intervention group	−9.353	4.173	.029	−17.712	−0.994
AHI at baseline	0.594	0.097	<.0001	0.423	0.812
BMI baseline	0.184	0.0426	.668	−0.669	1.037

R^2 for the model = 0.476. AHI = apnea-hypopnea index; B = unstandardized coefficient; BMI = body mass index.

control group, whereas deterioration was less apparent among participants in the intervention group compared with the control group (**Table 3**).

Characteristics of participants who improved in OSA severity

Those participants in the intervention group who improved their OSA severity (n = 11) were characterized by lower baseline values of AHI, body fat percentage, and glycated hemoglobin (HbA1c); they also spent more time in MVPA (**Table 4**).

DISCUSSION

Principal findings

The present study is unique as it documents the long-term effects on OSA of a BSM intervention aimed at enhancing physical activity and eating behaviors in addition to CPAP treatment. In addition to the initial treatment sessions during the first 6 months, booster sessions were provided to stimulate and maintain physical activity and eating behavior changes. At the 18-month follow-up, participants with moderate-to-severe OSA treated with CPAP and the BSM intervention reduced their AHI compared with patients treated with CPAP alone. Further, patients in the intervention group had improved OSA severity more often and worse OSA severity less often compared with the control group. The results are in line with those of a recent review and meta-analysis of 10 randomized controlled trials evaluating the effect of exercise training and/or diet on OSA severity,

Table 3—Changes in OSA category by study condition (baseline to 18 months).

Study Condition	No Change (n = 43)	Improvement (n = 13)	Deterioration (n = 4)	*P* Value
Control group	25 (83.3)	2 (6.7)	3 (10)	.008
Intervention group	18 (60)	11 (36.7)	1 (3.3)	

Data are presented as n (%). OSA = obstructive sleep apnea.

suggesting a weighted reduction in AHI of 22.3% of combined exercise and diet.[10] The meta-analysis supports the findings of the present study despite differences between the studies regarding severity of OSA, BMI, and intervention components. The duration of the interventions and follow-up period included in the review ranged from 4 to 52 weeks, and the present study adds information to the long-term perspective. Only in 1[28] of the 2 studies on the effects of combining diet and exercise was CPAP provided to the study participants, as was done in the present study. Further, the size of the treatment effect in AHI in the study by Ng et al[28] was almost similar to the present study.

The main results of the present study are of a similar magnitude as that previously reported from the 6-month follow-up[14] (ie, the reduction in AHI achieved after 6 months was maintained after 18 months). The project's overall hypothesis is that targeting both eating and physical activity behaviors in a structured way and with a longer perspective will enable maintenance of relevant behavior changes and, subsequently, improvements in AHI and OSA severity. However, since this study did not include any analysis of the contribution of the different components of the intervention, it is uncertain to what extent this contributed to the positive long-term maintenance of the treatment effects seen after the first 6 months. The baseline values differed regarding BMI in the participants eligible for the final analysis. Therefore, baseline BMI was included in the multiple linear regression to adjust for the potential confounding effect. The aim of the present study was to examine the long-term effects of a tailored BSM intervention program on AHI and OSA severity and to identify baseline predictors for treatment effect. We can only speculate about the underlying mechanisms for the overall OSA improvement that was found. OSA has different phenotypes with different mechanisms, including upper airway obstruction due to obesity or craniofacial morphology, increased loop gain, change in arousal threshold, and fluid shift.[29,30] However, unfortunately, no data were collected allowing analysis of the potential impact from fluid shift or the importance of other phenotypes on treatment effect.

Characteristics for success

Our results suggest that the actual improvements in OSA severity categorization are concentrated in a smaller cluster of 36.7% of the individuals receiving the BSM intervention. Knowledge about the characteristics of this group of patients may be of clinical importance since it has the potential to assist

Downloaded from jcsm.aasm.org by 65.21.193.86 on February 24, 2022. For personal use only. No other uses without permission.
Copyright 2022 American Academy of Sleep Medicine. All rights reserved.

Table 4—Descriptive data for participants in the intervention group with or without improvement at the 18-month follow-up.

	Improved (n = 11)	No Change/Deteriorated (n = 19)	*P* Value
Age, years	55.4 (±9.6)	57.2 (±13.3)	.698
Sex, n (%)			.530
Male	10 (90.9)	16 (84.2)	
Female	1 (9.1)	3 (15.8)	
AHI at baseline, events/h	34.6 (±7.9)	46.6 (±22.9)	.048
BMI, kg/m^2	33.8 (±4.6)	37.2 (±5.4)	.093
Body fat, %	30.6 (±2.5)	36.5 (±5.7)	.001
Waist circumference, cm	115.3 (±11.0)	124.4 (±14.2)	.080
Neck circumference, cm	43.5 (±3.1)	44.7 (±4.5)	.445
Daily average minutes of MVPA, n	11	17	
Mean (±SD)	100 (±49.4)	53 (±30.4)	.004
Daytime sleepiness	13.6 (±3.3)	12.0 (±4.5)	.290
Depressive symptoms*	9 (5.0)	9 (5.0)	.840
HbA1c*	10 (38 [6.8])	19 (42 [10.0])	.038
Fear of movement*	11.7 (3.1)	12.9 (4.2)	.430
Exercise self-efficacy	90.5 (±22.7)	106.1 (±39.2)	.240
Readiness for change in physical activity, n (%)			
Precontemplation and contemplation stages	2 (18.2)	1 (5.3)	.405
Preparation stage	8 (72.7)	16 (84.2)	
Action and maintenance stages	1 (9.1)	2 (10.5)	
Self-efficacy–enhancing physical activity,* n (%)	8 (3.0)	8 (3.0)	.452
Readiness for change in eating behavior, n (%)			
Precontemplation and contemplation stages	2 (18.2)	5 (26.3)	.350
Preparation stage	3 (27.3)	8 (42.1)	
Action and maintenance stages	6 (54.5)	6 (31.6)	
Self-efficacy for improving eating behavior*	8 (2.0)	8 (2.0)	.860
CPAP adherence at follow-up, n (%)			.667
Yes	5 (62.5)	10 (62.5)	
No	3 (37.5)	6 (37.5)	
Adherence to the Swedish nutrition recommendations, n (%)			.530
No	10 (90.9)	16 (84.2)	
Yes	1 (9.1)	3 (15.8)	

Data are presented as n (%) or as mean (±SD) unless otherwise stated. All values except for CPAP adherence represent baseline assessments. AHI = apnea-hypopnea index; BMI = body mass index; CPAP = continuous positive airway pressure; HbA1c, glycated hemoglobin; IQR = interquartile range; MVPA = moderate-to-vigorous physical activity. *Median (IQR).

clinicians in allocating patients to the right kind of treatment (ie, treatment with expected positive outcomes). For example, Mendelson et al[9] highlight the need to include analysis of patients with OSA who are the most likely to succeed with the exercise training. The importance of baseline values was also highlighted in the study by Foster et al[31] including obese patients with OSA and type 2 diabetes, reporting that those with higher AHI at baseline were more likely to decrease their AHI. Overall, the participants in the experimental group who improved in OSA severity seem to be in better health. As seen in **Table 4**, baseline data indicate that they had a lower AHI, lower body fat percentage, lower HbA1c, and a higher physical activity level, contributing to the overall perception that those experiencing effects of the intervention were in better health already at baseline than those not improving in OSA severity. Even though the analyses are based on a low number of participants, this offers some hypotheses that the BSM intervention did not reach the group of participants with the greatest need for changes in health-related behaviors. This is problematic as targeting both physical activity and eating behavior may, in addition to the effect of BSM on AHI, have positive effects on comorbidities such as hyperglycemia and cardiovascular disease, known to be positively influenced by regular physical activity[32] also in obese individuals.[33] Additional studies will shed light on

Downloaded from jcsm.aasm.org by 65.21.193.86 on February 24, 2022. For personal use only. No other uses without permission.
Copyright 2022 American Academy of Sleep Medicine. All rights reserved.

mediating the effects of possible behavior changes in the present participants. Based on the current results, however, future behavior-change trials among patients with OSA could benefit from testing a stratified approach with a minimal dismantled version of the BSM intervention for patients with better baseline values and a more intensified BSM intervention (eg, including supervised exercise) for patients with worse baseline values.

Methodological considerations

The study design including a randomized allocation to the intervention or control group along with the long intervention and follow-up are clear strengths of the study. This allows for analysis of the long-term effects of the BSM intervention, including booster sessions for increased maintenance. The BSM intervention was based on goals formulated in accordance with the prerequisites and goals of the individual participant. It is possible that behavior changes reached by some individuals were too small for a larger health effect to be seen. This may be part of the explanation as to why the intervention effects were found in those with better health already at baseline; it is possible that more progress toward meeting the recommendation of physical activity in combination with structured calorie restriction[11] is warranted for the patient with severe OSA. However, even though smaller/lighter goals might postpone intervention effects in the short term, such individually tailored behavior changes are more feasible for the individual and thus more robust for relapses in the long term.[34] In the present study, the treatment effect was maintained at the 18-month follow-up.

Even though the intended sample size was not reached, we did find an effect of the intervention. However, because of the low number of participants, the included analyses regarding variables associated with treatment success should only be seen as explorative and hypothesis generating due to the risk of both type I and type II error. In addition, it should be acknowledged that categorizing OSA severity may be insensitive to changes in AHI. Individuals close to cutoff limits may be more likely to change category. The categorization, however, is often used in addition to the AHI value in the clinical setting.[35]

The final analysis of AHI, the main outcome of the study, was conducted in 60 participants, corresponding to 69.7% of those initially randomized. This may influence the generalizability of the results to a broader population,[36] as the participants are a selected group, both by accepting participation in the study and remaining in the study, and by the effect of the intervention on individuals with better baseline health.

Conclusions and clinical implications

The addition of a BSM intervention targeting physical activity and sound eating habits proved to be effective in reducing AHI and improving OSA severity on a long-term basis compared with CPAP treatment alone. Among those receiving the intervention, the effects were evident in a cluster of individuals characterized by lower baseline AHI and percentage body fat, higher physical activity level, and better glucose control. These results may help the clinician in selecting treatment alternatives in addition to the CPAP treatment, wherein the patient with more severe OSA may need more support than that offered in the BSM to be successful in reaching an effect on OSA.

ABBREVIATIONS

AHI, apnea-hypopnea index
BMI, body mass index
BSM, behavioral sleep medicine
CPAP, continuous positive airway pressure
HbA1c, glycated hemoglobin
MET, metabolic equivalent
MVPA, moderate-to-vigorous physical activity
NRS, numerical rating scale
OSA, obstructive sleep apnea
TSK, Tampa Scale of Kinesiophobia

REFERENCES

1. Franklin KA, Lindberg E. Obstructive sleep apnea is a common disorder in the population—a review on the epidemiology of sleep apnea. *J Thorac Dis*. 2015;7(8):1311–1322.
2. Marshall NS, Wong KK, Cullen SR, Knuiman MW, Grunstein RR. Sleep apnea and 20-year follow-up for all-cause mortality, stroke, and cancer incidence and mortality in the Busselton Health Study cohort. *J Clin Sleep Med*. 2014;10(4):355–362.
3. Yaggi HK, Concato J, Kernan WN, Lichtman JH, Brass LM, Mohsenin V. Obstructive sleep apnea as a risk factor for stroke and death. *N Engl J Med*. 2005;353(19):2034–2041.
4. Giles TL, Lasserson TJ, Smith BH, White J, Wright J, Cates CJ. Continuous positive airways pressure for obstructive sleep apnoea in adults. *Cochrane Database Syst Rev*. 2006;3:CD001106.
5. Rosenberg R, Doghramji P. Optimal treatment of obstructive sleep apnea and excessive sleepiness. *Adv Ther*. 2009;26(3):295–312.
6. Weaver TE, Grunstein RR. Adherence to continuous positive airway pressure therapy: the challenge to effective treatment. *Proc Am Thorac Soc*. 2008;5(2):173–178.
7. Sawyer AM, Gooneratne NS, Marcus CL, Ofer D, Richards KC, Weaver TE. A systematic review of CPAP adherence across age groups: clinical and empiric insights for developing CPAP adherence interventions. *Sleep Med Rev*. 2011;15(6):343–356.
8. de Andrade FMD, Pedrosa RP. The role of physical exercise in obstructive sleep apnea. *J Bras Pneumol*. 2016;42(6):457–464.
9. Mendelson M, Bailly S, Marillier M, et al. Obstructive sleep apnea syndrome, objectively measured physical activity and exercise training interventions: a systematic review and meta-analysis. *Front Neurol*. 2018;9:73.
10. Edwards BA, Bristow C, O'Driscoll DM, et al. Assessing the impact of diet, exercise and the combination of the two as a treatment for OSA: a systematic review and meta-analysis. *Respirology*. 2019;24(8):740–751.
11. Johansson K, Hemmingsson E, Harlid R, et al. Longer term effects of very low energy diet on obstructive sleep apnoea in cohort derived from randomised controlled trial: prospective observational follow-up study. *BMJ*. 2011;342(1):d3017.
12. Tuomilehto H, Seppa J, Uusitupa M. Obesity and obstructive sleep apnea—clinical significance of weight loss. *Sleep Med Rev*. 2013;17(5):321–329.
13. Lindberg E, Gislason T. Epidemiology of sleep-related obstructive breathing. *Sleep Med Rev*. 2000;4(5):411–433.
14. Igelstrom H, Asenlof P, Emtner M, Lindberg E. Improvement in obstructive sleep apnea after a tailored behavioural sleep medicine intervention targeting healthy eating and physical activity: a randomised controlled trial. *Sleep Breath*. 2018;22(3):653–661.

Downloaded from jcsm.aasm.org by 65.21.193.86 on February 24, 2022. For personal use only. No other uses without permission.
Copyright 2022 American Academy of Sleep Medicine. All rights reserved.

15. O'Donovan G, Blazevich AJ, Boreham C, et al. The ABC of physical activity for health: a consensus statement from the British Association of Sport and Exercise Sciences. *J Sports Sci*. 2010;28(6):573–591.
16. Berry RB, Budhiraja R, Gottlieb DJ, et al. Rules for scoring respiratory events in sleep: update of the 2007 AASM Manual for the Scoring of Sleep and Associated Events. Deliberations of the Sleep Apnea Definitions Task Force of the American Academy of Sleep Medicine. *J Clin Sleep Med*. 2012;8(5):597–619.
17. Trost SG, McIver KL, Pate RR. Conducting accelerometer-based activity assessments in field-based research. *Med Sci Sports Exerc*. 2005; 37(11 Suppl):S531–S543.
18. National Board of Health and Welfare. *National guidelines for disease prevention methods. Tobacco use, alcohol risk consumption, insufficient physical activity and unhealthy eating habits*. Stockholm, Sweden: National Board of Health and Welfare; 2011.
19. Hagell P, Broman JE. Measurement properties and hierarchical item structure of the Epworth Sleepiness Scale in Parkinson's disease. *J Sleep Res*. 2007;16(1):102–109.
20. Johns MW. A new method for measuring daytime sleepiness: the Epworth sleepiness scale. *Sleep*. 1991;14(6):540–545.
21. Tsigos C, Hainer V, Basdevant A, et al. Management of obesity in adults: European clinical practice guidelines. *Obes Facts*. 2008;1(2):106–116.
22. Marcus BH, Simkin LR. The stages of exercise behavior. *J Sports Med Phys Fitness*. 1993;33(1):83–88.
23. Bandura A. Guide for constructing self-efficacy scales. In: Pajares F, Urdan T, editors. *Self-efficacy beliefs of adolescents*. Greenwich, CT: Information Age Publishing; 2006:307–337.
24. Denison E, Asenlof P, Lindberg P. Self-efficacy, fear avoidance, and pain intensity as predictors of disability in subacute and chronic musculoskeletal pain patients in primary health care. *Pain*. 2004;111(3):245–252.
25. Montgomery SA, Asberg M. A new depression scale designed to be sensitive to change. *Br J Psychiatry*. 1979;134(4):382–389.
26. Snaith RP, Harrop FM, Newby DA, Teale C. Grade scores of the Montgomery-Asberg Depression and the Clinical Anxiety Scales. *Br J Psychiatry*. 1986;148(5):599–601.
27. Igelstrom H, Emtner M, Lindberg E, Asenlof P. Tailored behavioral medicine intervention for enhanced physical activity and healthy eating in patients with obstructive sleep apnea syndrome and overweight. *Sleep Breath*. 2014;18(3):655–668.
28. Ng SSS, Chan RSM, Woo J, et al. A randomized controlled study to examine the effect of a lifestyle modification program in OSA. *Chest*. 2015;148(5):1193–1203.
29. Zinchuk AV, Gentry MJ, Concato J, Yaggi HK. Phenotypes in obstructive sleep apnea: a definition, examples and evolution of approaches. *Sleep Med Rev*. 2017;35:113–123.
30. Eckert DJ, White DP, Jordan AS, Malhotra A, Wellman A. Defining phenotypic causes of obstructive sleep apnea. Identification of novel therapeutic targets. *Am J Respir Crit Care Med*. 2013;188(8): 996–1004.
31. Foster GD, Borradaile KE, Sanders MH, et al. A randomized study on the effect of weight loss on obstructive sleep apnea among obese patients with type 2 diabetes: the Sleep AHEAD study. *Arch Intern Med*. 2009;169(17):1619–1626.
32. Lear SA, Hu W, Rangarajan S, et al. The effect of physical activity on mortality and cardiovascular disease in 130 000 people from 17 high-income, middle-income, and low-income countries: the PURE study. *Lancet*. 2017;390(10113):2643–2654.
33. Do K, Brown RE, Wharton S, Ardern CI, Kuk JL. Association between cardiorespiratory fitness and metabolic risk factors in a population with mild to severe obesity. *BMC Obes*. 2018;5(1):5.
34. Hill JO. Can a small-changes approach help address the obesity epidemic? A report of the Joint Task Force of the American Society for Nutrition, Institute of Food Technologists, and International Food Information Council. *Am J Clin Nutr*. 2009;89(2):477–484.
35. Epstein LJ, Kristo D, Strollo PJ Jr, et al. Clinical guideline for the evaluation, management and long-term care of obstructive sleep apnea in adults. *J Clin Sleep Med*. 2009;5(3):263–276.
36. Akl EA, Briel M, You JJ, et al. Potential impact on estimated treatment effects of information lost to follow-up in randomised controlled trials (LOST-IT): systematic review. *BMJ*. 2012;344:e2809.

SUBMISSION & CORRESPONDENCE INFORMATION

Submitted for publication May 6, 2019
Submitted in final revised form December 16, 2019
Accepted for publication December 16, 2019
Address correspondence to: Sören Spörndly-Nees, PhD, Department of Neuroscience, Physiotherapy, Uppsala University, Box 593, BMC, SE-75124 Uppsala, Sweden; Email: soren.sporndly-nees@neuro.uu.se

DISCLOSURE STATEMENT

All authors have seen and approved this manuscript. This study was supported by grants from the Swedish Research Council, The Swedish Heart and Lung Association, Uppsala County Council, and Swedish Sleep Research Society. The authors report no conflicts of interest.

Downloaded from jcsm.aasm.org by 65.21.193.86 on February 24, 2022. For personal use only. No other uses without permission.
Copyright 2022 American Academy of Sleep Medicine. All rights reserved.