


# Exercise and obstructive sleep apnoea: a 24-week follow-up study

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## ABSTRACT

**Objective** Report on long-term follow-up results in the apnoea hypopnea index (AHI) and self-reported daytime sleepiness in participants with moderate to severe obstructive sleep apnoea at 12 weeks after completion of a high-intensity exercise training or control intervention.

**Methods** Twenty-six participants with obstructive sleep apnoea (body mass index (BMI) 37 (36–39) kg/m, age 52 (49–55) years, apnoea-hypopnoea index 40.5 (31.3–50.2) events/hour), randomised to either 12 weeks of supervised high-intensity interval training (HIIT) (4×4 min of treadmill running or walking at 90%–95% of maximal heart rate) or no intervention (control), underwent a sleep evaluation follow-up 24 weeks after intervention initiation. Respiratory measures during sleep were registered at baseline, 12 weeks (postintervention) and 24 weeks (long-term follow-up).

**Results** At the 24-week follow-up, there were no statistically significant differences between the groups in the AHI (HIIT 30.7 (17.2–44.1) and control 38.7 (22.8–54.5) events/hour), Epworth score (HIIT 7.0 (4.7–9.3) and control 5.5 (3.9–7.0)), mean oxygen saturation (HIIT 93.2 (92.5–93.9) and control 92.0 (91.1–92.8)) or oxygen desaturation events (HIIT 32.9 (20.4–45.4) and control 44.3 (27.3–61.3) n/hour). BMI remained unchanged from the baseline in both groups. In the HIIT group, only two participants reported having continued with HIIT at 24 weeks.

**Conclusion** The effect of 12 weeks of supervised high-intensity exercise training on AHI and self-reported daytime sleepiness was lost at the 24-week follow-up.

## INTRODUCTION

Obstructive sleep apnoea (OSA),<sup>1</sup> sleep deprivation and daytime sleepiness are associated with physical inactivity,<sup>2–4</sup> and both OSA and physical inactivity are associated with obesity, hypertension, type 2 diabetes and cardiovascular disease.<sup>5</sup> Modest improvements in the apnoea hypopnea index (AHI), self-reported sleepiness, maximal oxygen uptake ( $VO_{2max}$ ) and quality of life have been reported after relatively short exercise interventions.<sup>6–12</sup> In general, adherence to exercise training after a structured rehabilitation programme is moderate to good, depending on the population studied and the programme investigated.<sup>13–16</sup> Promising results have been reported in studies of long-term follow-ups in patient activity and home-based rehabilitation

## WHAT IS ALREADY KNOWN ON THIS TOPIC

- ⇒ Obstructive sleep apnoea is associated with physical inactivity.
- ⇒ Exercise interventions improve obstructive sleep apnoea.
- ⇒ Knowledge of the effects of long-term adherence to high-intensity exercise training on obstructive sleep apnoea is limited.

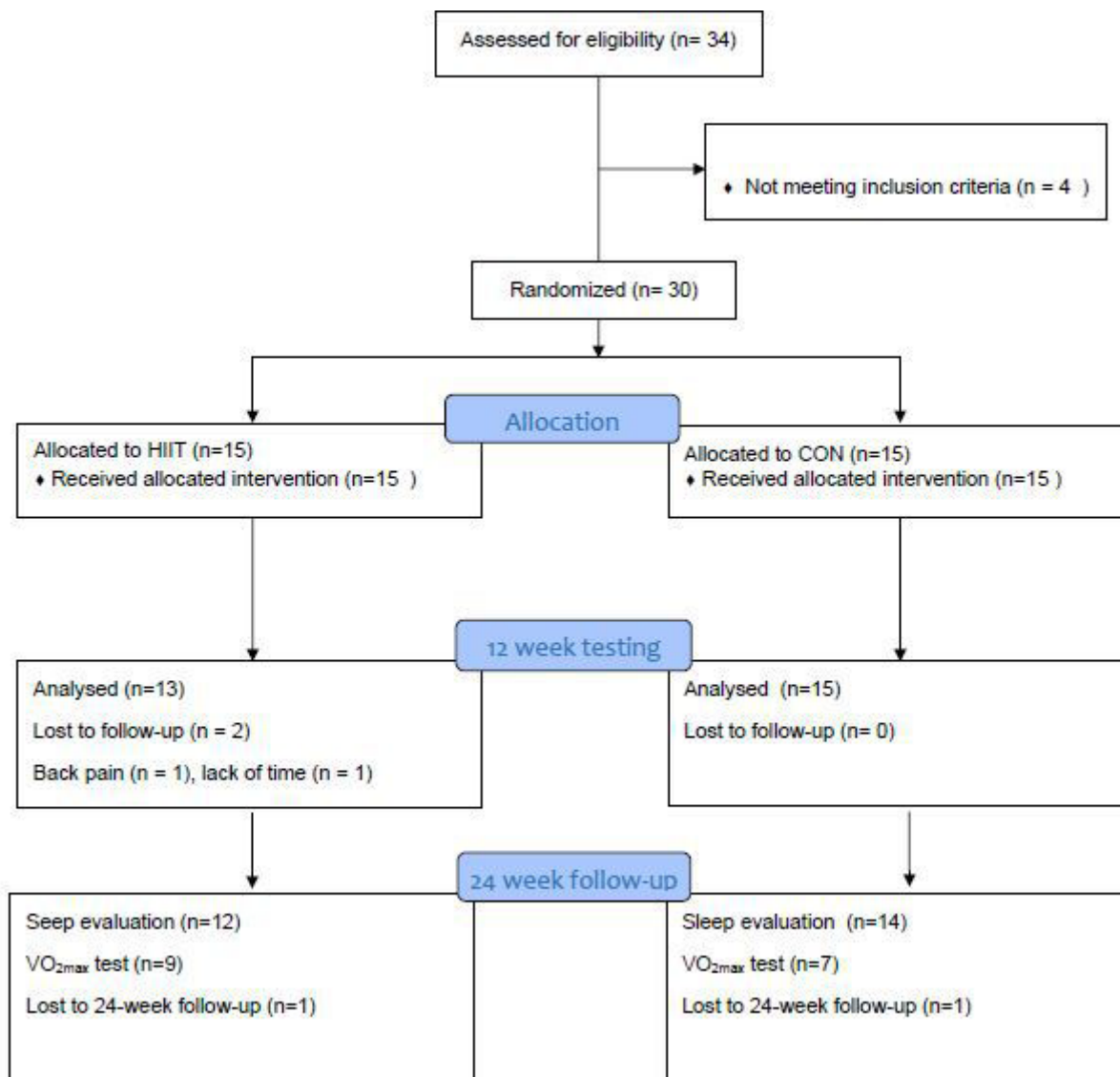
## WHAT THIS STUDY ADDS

- ⇒ Few participants with obstructive sleep apnoea continued with high-intensity exercise training beyond the supervised intervention.
- ⇒ Adherence to long-term high-intensity exercise training is challenging for participants with obstructive sleep apnoea.
- ⇒ The small positive effect of 12 weeks of supervised exercise on the apnoea hypopnea index (AHI) and self-reported daytime sleepiness was most likely lost due to detraining in participants with moderate obstructive sleep apnoea.

## HOW THIS MIGHT AFFECT RESEARCH, PRACTICE AND POLICY

- ⇒ Future studies should examine the exercise intensity, mode and volume that is most likely to achieve long-term adherence to regular exercise in participants with obstructive sleep apnoea.
- ⇒ The effects of long-term high-intensity or moderate exercise training on obstructive sleep apnoea should be investigated further.
- ⇒ The underlying mechanisms connecting improved AHI and daytime sleepiness with regular exercise training should be further investigated.

studies.<sup>13–15</sup> In a study of the combination of weight reduction and increased physical activity, improvement in OSA was sustained 4 years after the active 1-year intervention.<sup>17</sup> To our knowledge, little data exist on the effects of long-term adherence to high-intensity exercise training on AHI or daytime sleepiness in patients with OSA. In this study, we report long-term follow-up data on AHI and daytime sleepiness in participants with moderate to severe OSA: here, 'long-term' is 24 weeks after the initiation of the study and 12 weeks after the participants completed either a supervised high-intensity interval training (HIIT) intervention or a control intervention.<sup>7</sup>



**Figure 1** Study flowchart. CON, control group; HIIT, high intensity interval training group,  $VO_{2max}$ , maximal oxygen uptake.

## MATERIALS AND METHODS

### Study design

Twenty-eight participants with OSA completed a 12-week intervention. Participants were randomised to either a HIIT group or a control group that did not receive supervised intervention (CON).<sup>7</sup> Participants were investigated at the baseline, at 12 weeks and at 24 weeks. In the current paper, we report sleep data from the long-term postintervention follow-up, at the time point of 24 weeks after study initiation and 12 weeks after completion of the intervention. Twenty-six participants completed the 24-week follow-up sleep evaluation (HIIT  $n=12$ , CON  $n=14$ ), and 16 participants underwent a  $VO_{2max}$  test (figure 1). The inclusion criteria were a body mass index (BMI)  $\geq 30$  kg/m<sup>2</sup> and an AHI  $\geq 15$ , and the exclusion criteria were any disability precluding exercise intervention and known ischaemic cardiovascular disease. The primary study outcome was AHI at 12 weeks, measured using respiratory polygraphy. Secondary outcomes

included the Epworth sleepiness questionnaire (Norwegian version)<sup>18</sup> and the long-term postintervention follow-up.

### Exercise training intervention

The full exercise intervention is described in Karlsen *et al.*<sup>7</sup> Briefly, HIIT involved 4×4 min of treadmill walking or running at 90%–95% of maximal heart rate ( $HR_{max}$ ), two times a week, for 12 weeks.<sup>19</sup> The target exercise intensity was achieved using heart rate monitors. Treadmill speed or grade was increased throughout the training period to maintain the target exercise intensity. The control group was encouraged to continue their normal lifestyle during the intervention period. After the intervention, the participants in the HIIT group were asked to continue the exercise training programme at home, while participants in the control group were given the opportunity to perform one HIIT session in the laboratory in order to

sustain their motivation to continue the study and meet for the 24-week follow-up.

At the 24-week follow-up, participants in the HIIT group were asked whether they had continued HIIT beyond the supervised intervention. In addition, the participants were asked to fill out the International Physical Activity Questionnaire (IPAQ) short form for the last 7 days.<sup>20 21</sup> The data were analysed and categorised according to the questionnaire's user manual ([www.ipaq.ki.se/scoring.htm](http://www.ipaq.ki.se/scoring.htm)). The levels of physical activity were classified based on weekly metabolic equivalent per minutes (MET-minutes) (the sum of the metabolic energy requirements of specific activities or tasks performed in 1 week. One metabolic equivalent, one MET=3.5 mL of oxygen consumption·kg<sup>-1</sup>·min<sup>-1</sup>), with the categories of low (<600), moderate (600–3000) or high (≥3000) physical activity.<sup>22</sup>

### Demographics

Body weight and body composition were measured using a bioimpedance scale (Omron BF500, Omron Healthcare, Japan). Systolic and diastolic blood pressures were measured using a polygraph (SunTech Medical, Tango+) in line with standard procedure. Fasting venous plasma and serum samples were collected for standard haematology, and biomarkers were analysed at the Department of Clinical Chemistry at St. Olav's University Hospital.

### Sleep evaluation

The participants underwent one full night of data collection using respiratory polygraphy with ambulatory equipment (Embletta, Embla, Ontario, Canada) at home, without the use of CPAP, at baseline, 12 weeks and 24 weeks. The morning after, participants reported their sleeping patterns and answered the Epworth sleepiness questionnaire. A specialist in clinical neurophysiology analysed the registrations blinded for information of intervention using Somnologica studio V.5.1 (Micromed S.p.A. Mogliano Veneto (TV), Italy) and standardised guideline criteria.<sup>23–26</sup> Further details of the sleep evaluation methodology are found in Karlsen *et al.*<sup>7</sup>

### Cardiopulmonary exercise testing

VO<sub>2max</sub> was measured during treadmill running/walking at baseline, 12 weeks and 24 weeks using indirect breath-by-breath calorimetry (MMX-II, CORTEX Biophysik GmbH, Germany). Specifically, the participants warmed up for 15 min at ~70% of HR<sub>max</sub> before being fitted with the test equipment, and the workload gradually increased until the participants reached exhaustion. VO<sub>2max</sub> was defined as the mean of the three highest consecutive 10 s measurements where the VO<sub>2</sub> levelled off despite an increase in intensity, with a respiratory exchange ratio value above 1.10.

### Statistics

We used IBM SPSS V.28 for the statistical analysis (IBM SPSS statistics, USA). Data are presented as means and 95% CI. We used a linear mixed-effect regression model

**Table 1** Demographics of the participants at baseline

Variable	HIIT (n=12)	CON (n=14)
Gender (women/men)	3/9	3/11
Age (years)	53.5 (49.3–57.7)	50.6 (45.1–56.2)
BMI (kg·m <sup>2</sup> )	37.2 (33.8–40.5)	37.5 (34.7–40.4)
Sleep apnoea duration (months)	50 (18–83)	86 (48–124)
CPAP users (n)	10	12
Systolic blood pressure (mm Hg)	134 (122–145)	137 (129–145)
Diastolic blood pressure (mm Hg)	81 (72–90)	83 (77–88)
Hb (g·dL <sup>-1</sup> )	14.7 (14.1–15.2)	14.9 (14.0–15.8)
HCT (%)	43.5 (41.7–45.4)	43.3 (40.9–45.7)
Total cholesterol (mmol·L <sup>-1</sup> )	5.25 (4.55–5.94)	5.24 (4.51–5.96)
LDL cholesterol (mmol·L <sup>-1</sup> )	3.30 (2.67–3.93)	3.29 (2.63–3.95)
HDL cholesterol (mmol·L <sup>-1</sup> )	1.23 (1.06–1.40)	1.07 (0.87–1.28)
Triglycerides (mmol·L <sup>-1</sup> )	1.58 (1.15–2.02)	1.95 (1.29–2.61)
CRP (Mg·L <sup>-1</sup> )	6.16 (4.82–7.52)	8.50 (4.25–12.75)
TSH (mU·L <sup>-1</sup> )	1.83 (1.38–2.28)	2.27 (1.79–2.76)
T4 (pmol·L <sup>-1</sup> )	15.32 (14.30–16.33)	15.27 (14.12–16.42)
HBA1c (%)	5.75 (5.52–5.98)	5.93 (5.57–6.29)
Insulin (pmol·L <sup>-1</sup> )	113.5 (68.0–159.0)	141.9 (29.9–153.9)
Leptin (pmol·L <sup>-1</sup> )	1440 (778–2102)	1656 (809–2504)

Data are reported in mean and 95% confidence interval (95% CI). BMI, body mass index; CON, control group; CPAP, continuous positive airway pressure; CRP, C reactive protein; Hb, haemoglobin; HBA1c, glycolyzed haemoglobin A1c; HCT, haematocrit; HDL, high-density lipoprotein; HIIT, high-intensity interval training; LDL, low-density lipoprotein; T4, thyroxin; TSH, thyroid-stimulating hormone.

with random intercepts to investigate changes between the groups over time from 12 weeks to 24 weeks. Data normality was investigated using QQ-plots and tests of between-group effects, and the homogeneity of variance was tested using Levene's test. Trends within groups were investigated using analysis of variance (ANOVA) for repeated measurements. A two-sided *p* value ≤0.05 was used to establish the threshold of statistical significance.

## RESULTS

### Demographics

The demographic variables were unchanged from baseline to the 24-week follow-up (table 1). BMI in the sixteen VO<sub>2max</sub>-tested participants at 24 weeks was 39.4 (33.3–45.7) and 39.0 (35.1–43.0) in the HIIT and the control group, respectively, and BMI remained unchanged throughout all study timepoints.

### Sleep data

There was no significant between-group difference in AHI, self-reported daytime sleepiness (Epworth score), mean oxygen saturation or oxygen desaturation events at

**Table 2** Sleep evaluation

Variable	HIIT (n=12)			CON (n=14)		
	Baseline	12 weeks	24 weeks	Baseline	12 weeks	24 weeks
AHI (events · h <sup>-1</sup> )	32.3 (18.1–46.6)	25.6 (12.7–38.5)	30.7 (17.2–44.1)	47.4 (33.7–61.2)	45.5 (29.9–61.1)	38.7 (22.8–54.5)
Epworth score	9.9 (7.5–12.3)	7.4 (5.0–9.9)	7.0 (4.7–9.3)	5.3 (3.1–7.5)	5.8 (3.4–8.2)	5.5 (3.9–7.0)
O <sub>2</sub> -saturation (%)	92.5 (91.5–93.5)	92.4 (91.4–93.4)	93.2 (92.5–93.9)	91.9 (91.2–92.7)	92.2 (91.5–93.0)	92.0 (91.1–92.8)
O <sub>2</sub> -desaturation events (n·h <sup>-1</sup> )	35.1 (23.5–46.6)	30.8 (16.0–45.5)	32.9 (20.4–45.4)	52.5 (36.3–68.8)	47.6 (32.7–62.5)	44.3 (27.3–61.3)

Data are reported in mean and 95% CI. n=number of participants. AHI, apnoea-hypopnoea index; O<sub>2</sub>, oxygen.

24 weeks (table 2). The number of CPAP users is reported in table 1. The rate of self-reported CPAP use remained constant throughout the study. In the control group, AHI was reduced by 8.7 events/h<sup>1</sup> (p=0.046) from baseline to 24 weeks.

### Cardiopulmonary exercise testing

Sixteen participants underwent VO<sub>2max</sub> testing at the 24-week follow-up (HIIT n=9, CON n=7). Reasons given by those who dropped out of the testing were lack of motivation and lack of time. There was no significant difference in VO<sub>2max</sub> between the groups at 24 weeks (table 3). The ~2 mL/kg/min increase in VO<sub>2max</sub> from baseline to 12 weeks in the HIIT group was preserved at 24 weeks in the tested participants.<sup>7</sup>

### Self-reported physical activity

At the 24-week follow-up, two participants in the HIIT group reported having continued with regular HIIT after the supervised exercise intervention. The remaining participants in the HIIT group reported regular moderate-intensity exercise (n=5) or limited/no regular exercise (n=6). From the IPAQ data on self-reported physical activity, we found no statistical differences between-groups in self-reported weekly minutes of walking, moderate physical activity, vigorous physical activity or total activity in MET-min/week at 24 weeks (table 3). As not all the participants turned in the IPAQ

data material at all study timepoints, these data serve as an indication of trends in participants' physical activity, and they must be interpreted with some caution.

### DISCUSSION

The main finding of this study was that at the 24-week follow-up—12 weeks after the training intervention was completed—there were no differences between the HIIT group and the control group in AHI or in Epworth sleepiness score. We, therefore, conclude that the 12-week exercise training effect on measures of sleep apnoea was lost after 12 weeks without supervised HIIT.<sup>7</sup> The 24-week results underscore previous findings in a long-term follow-up study demonstrating that greater lifestyle changes, or long-lasting interventions and follow-up, are needed in order to slow the progression of the burden of OSA through lifestyle changes.<sup>17</sup> Some long-term follow-up studies of supervised laboratory-based or clinic-based HIIT in cardiovascular disease patients show low to moderate adherence to exercise training,<sup>14 16 27</sup> others show that physical fitness and physical activity are maintained and that patients meet the recommended levels of moderate physical activity 1 year after a 12-week programme of either supervised HIIT or moderate continuous training.<sup>15</sup> In addition, home-based HIIT cardiac rehabilitation shows the most promise for long-term adherence after the intervention period.<sup>13</sup>

**Table 3** Maximal oxygen uptake and self-reported physical activity

Variable	HIIT (n=9)			CON (n=7)		
	Baseline	12 weeks	24 weeks	Baseline	12 weeks	24 weeks
VO <sub>2max</sub> (L/min)	3.05 (2.71–3.31)	3.28 (2.97–3.60)	3.19 (2.84–3.55)	3.38 (2.84–3.90)	3.54 (3.07–3.80)	3.33 (2.87–3.82)
VO <sub>2max</sub> (mL/kg/min)	25.6 (21.8–29.7)	27.7 (23.6–32.5)	27.2 (22.9–32.2)	26.6 (20.8–33.2)	27.1 (23.6–30.9)	25.9 (21.3–30.1)
	<b>HIIT</b>			<b>CON</b>		
	<b>n=8</b>	<b>n=9</b>	<b>n=9</b>	<b>n=11</b>	<b>n=10</b>	<b>n=10</b>
Total MET-min/ week	3641 (595–6686)	2697 (1561–3833)	2501 (288–4714)	2857 (0–5980)	1991 (773–3210)	3034 (1967–4100)
Walking (min/week)	346 (0–708)	162 (52–272)	116 (18–214)	230 (0–446)	167 (45–290)	191 (16–366)
Moderate (min/week)	158 (0–351)	262 (66–458)	129 (2–257)	148 (25–271)	132 (12–252)	189 (94–284)
Vigorous (min/week)	120 (0–264)	146 (71–223)	131 (0–266)	188 (0–436)	91 (7–175)	168 (52–284)

Data are reported in mean and 95% CI. CON, control; HIIT, high-intensity interval training; VO<sub>2max</sub>, maximal oxygen uptake.

Sustaining motivation beyond the intervention period is a challenge with exercise training interventions. Most of the participants in the HIIT group reported not continuing HIIT after the supervised exercise period, and based on the IPAQ questionnaire, there were no significant differences between the groups in physical activity from 12 weeks to 24 weeks. Future studies should examine the exercise intensity, mode and volume that is most likely to achieve long-term adherence to regular exercise in patients with OSA.<sup>28</sup>

In the HIIT group, AHI and nightly O<sub>2</sub>-desaturation events returned to baseline values at 24 weeks. As the majority of the HIIT participants reported less regular high-intensity exercise at the 24-week follow-up, the favourable effects of 12 weeks of HIIT on sleep apnoea were most likely lost during the follow-up period, due to less structured exercise training.<sup>7</sup> However, given the small number of participants, we cannot rule out that other lifestyle changes, or random variations in the single night of respiratory polygraphy, might have influenced the results. There was also no change in BMI from baseline in the participants who underwent VO<sub>2max</sub> testing at 24 weeks; therefore, changes in body composition is unlikely to be explanatory factor behind the changes in respiratory measures during sleep.<sup>6,9</sup> The improvement in self-reported daytime sleepiness in the HIIT group from baseline to 12 weeks<sup>7</sup> persisted at 24 weeks. We speculate that this might be due to a carry-over effect from the supervised HIIT, as regular exercise is reported to have a moderate beneficial effect on sleep quality.<sup>28</sup>

With few studies investigating the relationship between physical fitness (ie, VO<sub>2max</sub>) and sleep, it is somewhat unclear how the former may mediate the relationship between regular exercise and sleep.<sup>28</sup> Possible explanatory effects of exercise on sleep discussed in the literature include improved fitness levels, changes in body composition, and changes in night-time heart rate variability and metabolism.<sup>29</sup> In addition, patients with OSA who exercise are less likely to complain about poor sleep quality than non-exercisers.<sup>30</sup> The correlation between sleepiness and OSA severity is found to be poor,<sup>30</sup> as demonstrated in a study on exercise and diet that found significant weight loss and improvement in daytime sleepiness without changes in AHI.<sup>31</sup> As discussed in extant literature,<sup>7</sup> night-time neck fluid accumulation may increase AHI; this could be reduced with physical activity, which leads to less retention of leg fluid that can shift to the neck during sleep.<sup>32,33</sup>

Despite randomised assignment to the study groups, AHI was higher in the control group than in the HIIT group at baseline, and the mean AHI was in the severe OSA category; therefore, we cannot rule out that the change over time in this group could be due to a regression towards the mean effect due to high baseline levels, or to a Hawthorn effect, with study participation stimulating increased focus on a healthy lifestyle and thereby potentially positively affecting AHI and nightly O<sub>2</sub>-desaturation events over time. This is supported by

indications of increased self-reported physical activity from baseline to 12 weeks and 24 weeks in some control group participants (n=10), which might have improved AHI in the control group at 24 weeks. However, with so few participants and only partial data on physical activity throughout the study, the results should be interpreted with caution. A previous long-term follow-up study focusing on weight reduction reported no change in AHI in the control group at 3 months and 1 year. For the 32% of the patients in the control group who had improved AHI at 3 months, this was maintained at the 1-year follow-up. Furthermore, in the total study population, changes in AHI during the 12 months of follow-up were strongly associated with weight reduction.<sup>34</sup>

### Strengths and limitations

The strength of the study is its randomised controlled design with a postintervention follow-up at 24 weeks—12 weeks after the intervention period—at which we measured both respiration during sleep and subjective daily sleepiness (Epworth scale). The study is limited by its few participants and possible low statistical power. The lack of stratification during randomisation allowed for a significantly higher baseline AHI in the CON group compared with the HIIT group. In addition, out of the 30 included participants, only 26 completed sleep quality testing at the 24-week follow-up and only 16 participants completed the VO<sub>2max</sub> testing. With partial dropout in various phases of the study, selection bias towards exercise testing cannot be ruled out, and the results should, thus, be interpreted with caution. Objectively measured physical activity throughout the study could have strengthened the study by limiting bias in self-report of physical activity. Also, the CPAP equipment used in the present study did not automatically record time in use; instead, this was self-reported by the participants and could allow for some bias.

### CONCLUSION

The effect of 12 weeks of supervised high-intensity exercise training on the AHI and on self-reported daytime sleepiness was lost at the 24-week follow-up.

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**Patient consent for publication** Consent obtained directly from patient(s)

**Ethics approval** This study involves human participants and was approved by Regional Medical Research Ethic Committee Number: 2010/1539. Participants gave informed consent to participate in the study before taking part.

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**Data availability statement** No data are available. The ethical approval does not support sharing of data.

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