



Lifestyle Modification in Patients with Obstructive Sleep Apnea

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Obstructive sleep apnea (OSA) is a disorder in which the upper respiratory tract is closed repeatedly during sleep. Treatment of OSA includes continuous positive airway pressure (CPAP), intraoral devices, and upper respiratory tract surgery, among which CPAP is known as the most effective treatment method. However, even in the case of CPAP, adherence rates during long-term follow-up are lowered considerably due to inconvenience of wearing, and low adherence rates limit its therapeutic effect. Therefore, studies have been carried out to confirm the effect of lifestyle modification which corrects modifiable risk factors of OSA, such as obesity. The aim of this study was to examine the effects of weight loss and aerobic exercise to improve the severity of OSA, and to examine the effect of drinking and smoking on OSA. In consideration of the difficulty of modifying one's lifestyle through traditional individual counseling, we propose the use of smart devices in the management of patients' lifestyles.

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Key Words Lifestyle, Obstructive sleep apnea, Weight loss, Exercise.

INTRODUCTION

Obstructive sleep apnea (OSA) is a disorder in which the upper airway obstruction is repeated during sleep. If left untreated, it causes hypoxia, daytime sleepiness, loss of concentration that can result in traffic and other accidents in the short term, and complications such as high blood pressure [1], cardiovascular disease [2], dementia [3], and depression [4] in the long run.

Obesity, age, male gender, alcohol consumption, and upper airway collapsibility are well known risk factors for OSA, and cigarette smoking has been reported to increase the risk of OSA [5]. Obesity is one of the most important risk factors for OSA, and the incidence and severity of OSA has been positively correlated with body mass index (BMI) [6]. The prevalence of OSA in obese people was shown to be twice that of normal weight people, and a 10% increase in body weight increased the apnea-hypopnea index (AHI) by 32% [7].

The most common treatment method for OSA is continuous positive airway pressure (CPAP), intraoral devices, and upper airway surgery. Among them, CPAP maintains an open airway during sleep and is known as the most effective treatment method for improving AHI and daytime sleepiness [8]. The American Academy of Sleep Medicine recommends a CPAP or oral device for the treatment of mild-to-moderate OSA patients. For patients with severe OSA, CPAP is recommended as the first-line treatment and an intraoral device is recommended as a secondary treatment method [9]. However, even among CPAP users, adherence rates during long-term follow-up were low due to inconvenience of wearing, and these low adherence rates limited the therapeutic effectiveness of CPAP [10].

Evidence suggests that weight loss or exercise may reduce the severity of OSA, and drinking and smoking have been found to be associated with an increased risk of OSA. Therefore, behavioral therapy through lifestyle modification as a means to treat OSA was investigated in

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this study. We will discuss lifestyle modification as a treatment method of OSA by examining the results of research on each modifiable item, including weight loss, exercise, alcohol consumption and smoking.

WEIGHT LOSS AS A TREATMENT FOR OBSTRUCTIVE SLEEP APNEA

Many studies (Table 1) on weight-loss attempts in patients with OSA used a very low-calorie diet of only 600–800 kcal a day for the first 8 to 12 weeks, followed by diet consultation [7,11-17]. In a study of weight loss in patients with mild OSA for one year, a 10% reduction in average body weight (from 101.2 to 90.5 kg) reduced AHI by approximately 40% (from 10 to 6 events/h). Since the subjects had mild OSA patients, approximately 60% of the patients were cured with weight loss alone [15]. Despite the fact that there was no further consultation or intervention for one year thereafter, at 2-year follow-up the average BMI of patients decreased further from -3.5 kg/m^2 (at 1-year follow-up) to -2.5 kg/m^2 (at 2-year follow-up), and the average decrease of AHI was maintained at -4.0 events/h (at 1-year follow-up) and -4.6 events/h (at 2-year follow-up) [13]. In a study of weight loss during one year in moderate-to-severe OSA patients, AHI decreased by about 16.9% (from 42.5 to 32.0 events/h) with a 2.2 kg/m^2 reduction in BMI (from 29.8 to 27.6 kg/m^2) [11]. In addition, a meta-analysis of studies examining the effects of weight loss on OSA showed that weight loss reduced the severity of OSA [18].

When the mechanism of weight loss to improve severity of OSA was examined, weight loss was found to increase the area of the upper airway by reducing velopharyngeal fat and alleviate collapsing of the upper airway by reducing mechanical burden. In addition, accumulation of abdominal visceral fat due to obesity makes ventilation difficult by increasing abdominal pressure. Weight loss can improve the severity of OSA by reducing abdominal pressure and improving thoracic exercise and ventilation. Some studies have reported that weight loss contributes to the normalization of neuromechanical control of the upper airway during sleep by reducing fat-induced inflammation-related cytokines, and resolving hormone control disorders caused by obesity [19,20].

AEROBIC EXERCISE AS A TREATMENT FOR OBSTRUCTIVE SLEEP APNEA

When studies on the effects of weight-loss in patients with OSA were examined (Table 2), moderate-intensity aerobic exercise was performed 3–4 times a week for about 30 to 45 minutes for 3 months in many cases [21-25]. In a study of three-month aerobic exercise in patients with mild-to-moderate OSA,

there was no reduction in body weight, but AHI decreased by about 28% (from 15.19 to 11.01 events/h) [23]. In a study of aerobic exercise for 3 months in patients with chronic heart failure, there was no weight loss but AHI decreased by 34% (from 25.2 to 16.7 events/h), and the sleep efficiency increased by 7% (from 74.4 to 79.6%) [22]. And in a study of 4-week exercise in patients with moderate-to-severe OSA and coronary artery disease, AHI decreased by 34% (from 31.1 to 20.5 events/h), overnight change in leg fluid volume decreased by 20% (from -579.0 to -465.8 mL), and overnight change in upper airway cross-sectional area increased from -0.2 cm^2 to $+0.09 \text{ cm}^2$ [21].

Although exercise is one method of reducing body weight among life style-related treatment methods, meta-analysis of studies on the effects of aerobic exercise on OSA showed that the effects of exercise were not limited to weight loss [26,27]. In the meta-studies, there was no significant weight loss due to exercise, but AHI in patients with OSA decreased, and sleep efficiency and daytime sleepiness were improved. This means that exercise itself has a therapeutic role in OSA treatment. In dog studies, stimulation of the gastrocnemius muscle and sciatic nerve increased the activity of the genioglossus muscle [28]. In human studies, results have shown that exercise was associated with increased respiratory muscle strength [26,29]. Exercise has also been reported to reduce the fluid retention in the legs, thereby preventing rostral fluid shift to the upper body at night, where it can cause collapse of the upper airway during sleep [21,30]. The effect of exercise on OSA appears to be due to the interaction of these multiple mechanisms, rather than the effect of only one of these mechanisms.

EFFECTS OF ALCOHOL INTAKE ON OBSTRUCTIVE SLEEP APNEA

Table 3 shows that the risk of OSA was about 1.36 to 1.56 times higher for persons who consumed alcohol than for those who did not [31-34]. In a study of 20 healthy men who consumed an excess of 1 g of alcohol per body weight, alcohol intake increased the frequency of apnea from 20 to 110 times over the entire night's sleep, while desaturation events increased from 118 to 226 times [35]. In a study in which 0.5 g of alcohol was consumed per body weight in patients with mild-to-moderate OSA, there was no increase in the length of the apnea or aggravation of hypoxia, but the AHI increased by 37% (from 7.1 to 9.7 events/h) [36]. In a meta-analysis of studies dealing with the association between alcohol and OSA, the risk of having OSA increased by about 25% from alcohol consumption [37].

The mechanism by which alcohol induces OSA is through lowering genioglossus and hypoglossal motor nerve activities [38,39], decreased sleep arousal [35], increased nasal mucosal swelling, and increased nasal resistance [39]. In addition, alcohol is known to cause segmented sleep regardless of sleep apnea

Table 1. Studies that analyzed the impacts of weight loss on OSA

Author	Study design	Study participants	Prescribed regimen	Effects of intervention
Ng et al. (2015) [11]	Randomized, controlled, parallel study of 12 months intervention	Age: 30–80 years BMI: > 25 kg/m ² AHI: > 15 events/h 78 men, 26 women	Intervention group Diet counseling once a week for the initial 4 months followed by once a month for the remaining 8 months Goal to lower the intake amount of the participants by 10–20% from that in the ordinary days Control group Advices on the basic lifestyle modification at baseline and Month 6	BMI (kg/m ²) Intervention group: baseline 29.8 ± 3.6; 12-month 27.6 ± 3.9 Control group: baseline 30.5 ± 4.2; 12-months 29.9 ± 4.2 (p < 0.001)* AHI (events/h) Intervention group: baseline 42.5 ± 19.7; 12-month 32.0 ± 20.7 Control group: baseline 42.5 ± 20.0; 12-month 39.6 ± 19.5 (p = 0.024)* ESS Intervention group: baseline 11.9 ± 5.3; 12-month 8.4 ± 4.1 Control group: baseline 10.2 ± 4.7; 12-months 9.2 ± 4.9 (p = 0.004)*
Kiuna et al. (2013) [12]	Randomized, controlled trial with follow-up at 1, 2, and 4 years	Age: 45–75 years BMI: ≥ 25 kg/m ² (or ≥ 27 kg/m ² if taking insulin) Type 2 diabetes AHI: ≥ 5 events/h 108 men, 156 women	Intervention group Goal to lower the body weight by 10% for the initial one year Diet using portion-controlled diets (liquid meal and snack bar) only for initial 4 months After that, lower the intake amount of portion-controlled diets for 8 months Moderate-intensity activity such as fast walking for 175 minutes per week Personal counseling once a month with additional contacts by telephone, mail, or e-mail from Year 2 to 4 Control group Group training on the diet, physical activities related to the management of diabetes three times a year	Changes from baseline at the 4-year follow-up Weight (kg) Intervention group: ↓5.2 ± 0.7, control group: ↓0.8 ± 0.7 (p < 0.001)* AHI (events/h) Intervention group: ↓4.0 ± 1.6, control group ↓3.7 ± 1.6 (p = 0.001)*
Tuomilehto et al. (2010) [13]	Randomized, controlled study of 1 year and 2-year follow-up	Age: 18–65 years BMI: 28–40 kg/m ² AHI: 5–15 events/h 53 men, 18 women	Intervention group Very low-calorie diet with 600–800 kcal/day for initial 12 weeks Maintain the fat amount in the diet not more than 30% for the next 9 months and counseling to increase the level of physical activities 14 times of counseling with the nutritionist for the first year No counseling for the second year Control group Basic lifestyle counseling at Month 0, 3, and 12 Very low-calorie diet with 800 kcal/day for initial 8 weeks After that, group meeting once a week for support	Odds ratio for having mild OSA at the 2-year follow-up 0.35 (95% CI: 0.12 to 0.97; p = 0.045)* Changes from baseline at the 2-year follow-up AHI (events/h) Intervention group: ↓4.6 ± 4.9, control group: ↓0.5 ± 9.3 (p = 0.049)* Cured patients, n (%) Intervention group: 57 (20), control group: 31 (11) (p = 0.045)* BMI (kg/m ²) Intervention group: ↓2.4 ± 2.1, control group: ↓1.0 ± 2.6 (p = 0.028)*
Nerfeldt et al. (2010) [14]	Prospective single group intervention study with follow-up at 2 years	Age: 30–69 years BMI: ≥ 30 kg/m ² AHI: ≥ 10 events/h 24 men, 9 women	Basic lifestyle counseling at Month 0, 3, and 12 Very low-calorie diet with 800 kcal/day for initial 8 weeks After that, group meeting once a week for support	AHI (events/h) Baseline 43 ± 24; 2-year 28 ± 19 (p = n.s.)† ODI (events/h) Baseline 42 ± 23; 2-year 23 ± 15 (p = 0.010)† Arousal index (events/h) Baseline 24 ± 15; 2-year 11 ± 11 (p = 0.019)† BMI (kg/m ²) Baseline 40 ± 5; 2-year 35 ± 3 (p = 0.003)† ESS Baseline 9 ± 4; 2-year 5 ± 3 (p = 0.026)†

Table 1. Studies that analyzed the impacts of weight loss on OSA (continued)

Author	Study design	Study participants	Prescribed regimen	Effects of intervention
Tuomilehto et al. (2009) [15]	Randomized, controlled study of 1 year intervention	Age: 18–65 years BMI: 28–40 kg/m ² AHI: 5–15 events/h 53 men, 19 women	Intervention group Very low-calorie diet with 600–800 kcal/day for initial 12 weeks Maintain the fat amount in the diet not more than 30% for the next 9 months and counseling to increase the level of physical activities 14 times of counseling with the nutritionist for the first year Control group Basic lifestyle counseling at Month 0, 3, and 12	Changes from baseline at the 1-year follow up AHI (events/h) Intervention group: ↓4.0 ± 5.6, control group: ↓0.3 ± 8.0 (p = 0.017)* Cured patients, n (%) Intervention group: 22 (63), control group: 13 (35) (p = 0.019)* BMI (kg/m ²) Intervention group: ↓3.5 ± 2.1, control group: ↓0.8 ± 2.0 (p < 0.001)* Waist circumference (cm) Intervention group: ↓11.6 ± 6.6, control group: ↓3.0 ± 6.0 (p < 0.001)* Plasma insulin (mU/L) Intervention group: ↓5.9 ± 7.0, control group: ↓1.2 ± 3.4 (p = 0.004)* Serum triglycerides (mmol/L) Intervention group: ↓0.48 ± 1.13, control group: ↓0.06 ± 0.65 (p = 0.027)* Changes from baseline at the 1-year follow-up Weight (kg) Intervention group: ↓10.8 ± 0.7, control group: ↓0.6 ± 0.7 (p < 0.001)* AHI (events/h) Intervention group: ↓5.4 ± 1.5, control group: ↓4.2 ± 1.4 (p < 0.001)* Hemoglobin A1c, (%) Intervention group: ↓0.7 ± 0.1, control group: ↓0.2 ± 0.1 (p < 0.001)*
Foster et al. (2009) [16]	Randomized, controlled study of 1 year intervention	Age: 45–75 years BMI: ≥ 25 kg/m ² (or ≥ 27 kg/m ² if taking insulin) Type 2 diabetes AHI: ≥ 5 events/h 108 men, 156 women	Intervention group Goal to lower the body weight by 10% for one year Diet using portion-controlled diets (liquid meal and snack bar) only for initial 4 months After that, lower the intake amount of portion-controlled diets for 8 months Moderate-intensity activity such as fast walking for 175 minutes per week Control group Group training on the diet, physical activities related to the management of diabetes three times a year	Changes from baseline at the 1-year follow-up Weight (kg) Intervention group: ↓18.7 ± 4.1, control group: ↓1.1 ± 1.9 (p < 0.001)* AHI (events/h) Intervention group: ↓25 ± 17, control group: ↓2 ± 11 (p < 0.001)* ESS Intervention group: ↓3 ± 5, control group: ↓1 ± 3 (p < 0.001)* % change in weight (vs. no weight change) Estimated % change in AHI (95% CI) ↓40 (-58 to -35) ↓26 (-34 to -18) ↓14 (-18 to -9) ↑15 (+10 to +21) ↑32 (+20 to +45) ↑70 (+42 to +104) adjusted for sex, change in cigarette packs/wk, baseline BMI (kg/m ²), baseline age (all p < 0.001)†
Johansson et al. (2009) [17]	A randomized controlled trial of 9 weeks intervention	Age: 30–65 years BMI: 30–40 kg/m ² AHI: ≥ 15 events/h 63 men	Intervention group Very low-calorie diet with 2.3 MJ/day for initial 7 weeks, intake 6.3 MJ/day at week 9 upon introduction of normal food gradually for the next 2 weeks Control group Keep their diet Data collection at baseline and 4-year follow-up	Changes from baseline at the 1-year follow-up Weight (kg) Intervention group: ↓18.7 ± 4.1, control group: ↓1.1 ± 1.9 (p < 0.001)* AHI (events/h) Intervention group: ↓25 ± 17, control group: ↓2 ± 11 (p < 0.001)* ESS Intervention group: ↓3 ± 5, control group: ↓1 ± 3 (p < 0.001)* % change in weight (vs. no weight change) Estimated % change in AHI (95% CI) ↓40 (-58 to -35) ↓26 (-34 to -18) ↓14 (-18 to -9) ↑15 (+10 to +21) ↑32 (+20 to +45) ↑70 (+42 to +104) adjusted for sex, change in cigarette packs/wk, baseline BMI (kg/m ²), baseline age (all p < 0.001)†
Peppard et al. (2000) [7]	Prospective cohort study with 4-year follow up	Adult general population 385 men, 305 women	Data collection at baseline and 4-year follow-up	Changes from baseline at the 1-year follow-up Weight (kg) Intervention group: ↓18.7 ± 4.1, control group: ↓1.1 ± 1.9 (p < 0.001)* AHI (events/h) Intervention group: ↓25 ± 17, control group: ↓2 ± 11 (p < 0.001)* ESS Intervention group: ↓3 ± 5, control group: ↓1 ± 3 (p < 0.001)* % change in weight (vs. no weight change) Estimated % change in AHI (95% CI) ↓40 (-58 to -35) ↓26 (-34 to -18) ↓14 (-18 to -9) ↑15 (+10 to +21) ↑32 (+20 to +45) ↑70 (+42 to +104) adjusted for sex, change in cigarette packs/wk, baseline BMI (kg/m ²), baseline age (all p < 0.001)†

*Between intervention and control group. †From the baseline.

OSA: obstructive sleep apnea, BMI: body mass index, AHI: apnea-hypopnea index, ODI: oxygen desaturation index, ESS: Epworth Sleepiness Scale, CI: confidence interval.

Table 2. Studies that analyzed the impacts of exercise on OSA

Author	Study design	Study participants	Prescribed regimen	AHI (events/h)	Effects of intervention
Mendelson et al. (2016) [21]	A randomized controlled trial with 4 weeks intervention	Age: 18–85 years AHI: ≥ 15 Coronary artery disease 30 men, 4 women	Intervention group Moderate-intensity of walking for 30 minutes per day 5 times a week Control group Not increase the physical activities during the control period	Intervention group–baseline: 31.1 ± 12.9 ; 4-week: 20.5 ± 9.4 Control group–baseline: 28.1 ± 13.5 ; 4-week: 27.0 ± 15.1 ($p = 0.047$)* Overnight change in leg fluid volume (mL) Intervention group–baseline: -579.0 ± 221.8 ; 4-week: -465.8 ± 162.8 Control group–baseline: -452.5 ± 163.5 ; 4-week: -433.6 ± 141.2 ($p = 0.02$)*	
Servantes et al. (2012) [22]	A randomized, prospective controlled study of 3 months intervention	Age: 30–70 years Chronic heart failure New York Heart Association Class II–III 21 men, 24 women	Aerobic training After three supervised aerobic exercise sessions, home-based exercise for three months 3 times/week in 1–8 weeks, 4 times/week in 9–12 weeks 30 min/session in 1–4 weeks, 45 min/session in 9–12 weeks Strength training After three supervised strength exercise session, home-based exercise for three months 3 times/week in 1–8 weeks, 4 times/week in 9–12 weeks	AHI (events/h) Group 1–baseline: 25.2 ± 24.7 ; 3-month: 16.7 ± 18.6 ($p < 0.001$) [†] Group 2–baseline: 26.4 ± 17.6 ; 3-month: 16.4 ± 11.1 ($p < 0.001$) [†] Control group–baseline: 22.8 ± 17.4 ; 3-month: 25.9 ± 18.8 ($p = 0.001$)* Sleep efficiency (%) Group 1–baseline: 74.4 ± 12.9 ; 3-month: 79.6 ± 11.9 ($p < 0.05$) [†] Group 2–baseline: 77.6 ± 11.7 ; 3-month: 83.2 ± 10.1 ($p < 0.01$) [†] Control group–baseline: 75.9 ± 13.8 ; 3-month: 71.4 ± 15.9 ($p = 0.007$)*	
			Intervention group Group 1: aerobic training Group 2: aerobic + strength training Control group Remained untrained		

Table 2. Studies that analyzed the impacts of exercise on OSA (continued)

Author	Study design	Study participants	Prescribed regimen	Effects of intervention
Sengul et al. (2011) [23]	A randomized, prospective controlled study of 3 months intervention	Age: 40–65 years 5 ≤ AHI < 30 25 men	Intervention group Breathing exercise for 15 to 30 minutes and aerobic exercise for 45 to 60 minutes three times a week Control group Remained untrained	AHI (events/h) Intervention group—baseline: 15.19 ± 5.43; 3-month: 11.01 ± 5.28 (p = 0.02)† Control group—baseline: 17.92 ± 6.45; 3-month: 17.36 ± 11.18 (p = 0.11)*
Kline et al. (2011) [24]	A randomized, prospective controlled study of 3 months intervention	Age: 18–55 years AHI: ≥ 15 events/h BMI: ≥ 25 kg/m ² 24 men, 19 women	Intervention group Aerobic exercise 4 times a week for initial 4 weeks under supervision. Gradually increase the level of intensity during the period. Control group After that for 8 weeks, perform moderate-intensity of aerobic exercise 4 times a week (150 minutes per week) under supervision, and perform resistance exercise after aerobic exercise under supervision twice a week.	AHI (events/h) Intervention group—baseline: 32.2 ± 5.6; 3-month 24.6 ± 4.4 Stretching control—baseline: 24.4 ± 5.6, 3-month 28.9 ± 6.4 (p < 0.01)* ODI (events/h) Intervention group—baseline: 24.5 ± 4.2; 3-month 21.5 ± 3.7 Stretching control—baseline: 16.8 ± 4.2; 3-month 23.2 ± 5.8 (p = 0.03)*
Norman et al. (2000) [25]	Prospective single group study of 6 months intervention	Age: 32–60 years AHI: > 5 events/h BMI: < 40 kg/m ² 8 men, 1 woman	Intervention group (control group) Whole body flexibility training under supervision twice a week. Supervised 30–45 minutes aerobic exercise 3 times a week for initial 4 months Then once a week aerobic exercise under supervision with instructions to exercise at least twice more each week for the next two months	Stage N3 sleep (%) Intervention group—baseline: 12.8 ± 1.2; 3-month 13.2 ± 1.1 Stretching control—baseline: 12.4 ± 1.7; 3-month 9.2 ± 1.5 (p = 0.03)* AHI (events/h) Baseline 21.7 ± 9.0; 6-month 11.8 ± 6.8 (p = 0.002)† Sleep efficiency (%) Baseline 75.2 ± 9.0; 6-month 85.9 ± 10.5 (p = 0.003)† Weight (kg) Baseline 110.9 ± 12.0; 6-month 104.7 ± 12.3 (p = 0.001)† Resting systolic blood pressure (mm Hg) Baseline 139.2 ± 9.6; 6-month 130.4 ± 12.2 (p = 0.016)†

*Between intervention and control group. †From the baseline.

OSA: obstructive sleep apnea, BMI: body mass index, AHI: apnea-hypopnea index, ODI: oxygen desaturation index.

Table 3. Studies that analyzed the impacts of alcohol drinking on OSA

Author	Study design	Study participants	Alcohol definition	Adjustment	Adjusted OR (95% CI)
Baik et al. (2015) [31]	Case control	Age: 40–69 years General population 956 men, 807 women	Current drinker vs. no drinker	NRG1 genotype, age, sex, BMI, neck circumference	Current drinker: 1.36 (0.97–1.91) (p < 0.05)
Pan et al. (2014) [32]	Cross sectional	Age: ≥ 18 years General population 18,186 men 20,452 women	Never drinker: never alcohol use Current drinker: alcohol use within the last 12 months Past drinker: alcohol use more than 12 months ago	Age, sex, race, marital status, income, education, asthma, diabetes, hypertension	Current drinker: 1.52 (1.03–2.23) (p = 0.0329) Past drinker: 1.65 (1.09–2.49) (p = 0.017)
Kang et al. (2014) [33]	Cross sectional	Age: ≥ 19 years General population 3,559 men 4,396 women	Harmful alcohol use vs. no harmful alcohol use	Age, sex, marital status, educational level, occupation, shift work, smoking, exercise, musculoskeletal disease, diabetes mellitus, hypertension	Harmful alcohol use: 1.566 (1.302–1.884) (p < 0.001)
Peppard et al. (2007) [34]	Cohort	Age: 30–60 years General population 775 men, 645 women	Drinks per day 0 drinks: nondrinkers 1 drink: 0 < and < 1 drink/day 2 drinks: 1 ≤ and < 2 drinks/day 3 drinks: 2 ≤ and < 6 drinks/day	Age, BMI, neck, circumference, waist circumference, smoking, antidepressant, antihypertensive, cholesterol-lowering medication use	Men 0 drinks 1.0 (reference) 1 drink 1.25 (1.07–1.46) 2 drinks 1.55 (1.14–2.12) 3 drinks 1.94 (1.21–3.09) (p = 0.006) Women 0 drinks 1.0 (reference) 1 drink 0.94 (0.67–1.33) 2 drinks 0.89 (0.45–1.76) 3 drinks 0.83 (0.30–2.33) (p = 0.73)

OSA: obstructive sleep apnea, BMI: body mass index, NRG 1: neuregulin 1, OR: odds ratio, CI: confidence interval.

[40], and this increase in segmented sleeping by alcohol exacerbates OSA.

EFFECTS OF SMOKING ON OBSTRUCTIVE SLEEP APNEA

Some studies have reported that smoking increased the risk of OSA, but other studies have reported that smoking did not increase, or even decreased the risk (Table 4) [41–44]. However, in the Wisconsin sleep cohort study, one of the largest cohort studies of OSA in the United States, smokers were twice as likely to have OSA compared to non-smokers. The amount of smoking and the prevalence of apnea were positively correlated, and those who smoked more than 2 packs per day had a 6.7-fold risk

of having mild OSA and a 40-fold risk of having moderate-to-severe OSA [44].

The mechanisms by which smoking may exacerbate OSA include changes in sleep architecture, relaxation of the upper airway muscles and decreased nerve response by nicotine, increased sleep arousal, and increased mucosal inflammation due to smoke aspiration [45]. In a study comparing the uvular mucosa of smokers and non-smokers with OSA who received uvulopalatopharyngoplasty, secretion of calcitonin gene-related peptide (inflammatory mediator) in smoker's mucosa, lamina propria edema, and mucosal thickness were increased, and this mechanism caused smoking to narrow the area of the upper airway and exacerbate OSA [46].

CURRENT STATE OF LIFESTYLE MODIFICATION AS A TREATMENT FOR OBSTRUCTIVE SLEEP APNEA

Previous research has found that weight loss and exercise decreased the severity of OSA, and that alcohol and smoking increased the risk of OSA. Based on these findings, lifestyle modifications of weight loss, aerobic exercise, alcohol abstinence

and smoking cessation are effective treatments for OSA (Fig. 1). However, lifestyle modification usually requires frequent individual counseling sessions to increase patient compliance, which is time consuming and costly. Despite the importance of lifestyle modification, in reality, patients with OSA are only counseled or encouraged to make lifestyle changes, and successful lifestyle modification cannot be achieved in this way.

Table 4. Studies that analyzed the impacts of smoking on OSA

Author	Study design	Study participants	Smoking definition	Adjustment	OR (95% CI)
Ye et al. (2014) [41]	A cross-sectional design	Age: 30-55 years General population 137 men, 61 women	Current smoker vs. no smokers	res13170573 genotypes, age, sex, BMI, LDL, hypertension	Adjusted OR Smokers 1.32 (1.03-2.78)
Koyama et al. (2012) [42]	A cross-sectional design	General population 745 men	Current smoker vs. no smokers	-	Unadjusted OR Smokers 0.74 (0.45-1.22)
Kashyap et al. (2001) [43]	A cross-sectional design	Patients who had their polysomnography data at sleep center 126 men, 88 women	Current, never, former smoker	Age, sex, BMI, and number of alcoholic drinks/week.	Adjusted OR Current vs. never smoker 2.8 (1.2-6.5) (p = 0.015) Current vs. never or former smoker 2.7 (1.2-5.7) (p = 0.011)
Wetter et al. (1994) [44]	Cohort study	General population 468 men, 343 women	Current, never, former smoker	Sex, education, age, BMI, number of cans of caffeinated soda/day, number of cups of caffeinated coffee or tea/day, number of alcoholic drinks/week	Adjusted OR Current vs. never smoker 3.05 (1.44-6.44) Former vs. never smoker 1.33 (0.77-2.30)

OSA: obstructive sleep apnea, BMI: body mass index, LDL: low-density lipoprotein, OR: odds ratio, CI: confidence interval.

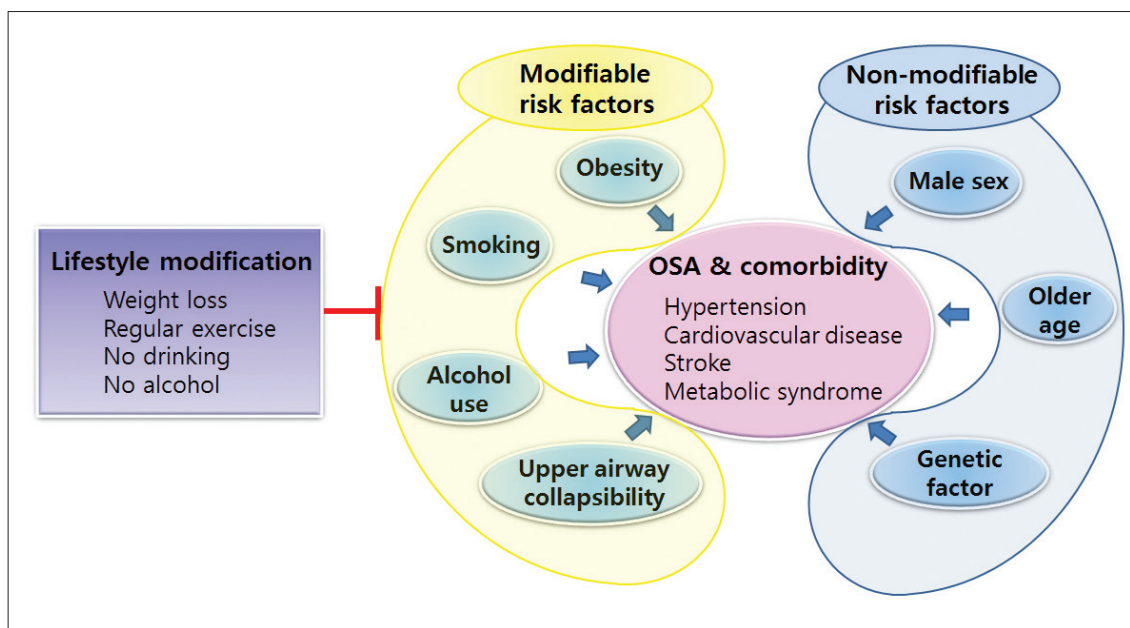


Fig. 1. Risk factors of OSA. A variety of risk factors of OSA, such as obesity, alcohol, smoking, upper airway collapsibility can be improved by lifestyle modifications. OSA: obstructive sleep apnea.

CONSTRUCTION AND USE OF LIFELOG DATA USING SMART DEVICES

Various research has been carried out to solve this problem, and developments in mobile technology have led to the creation of a user-oriented smartphone or wearable device app related to activity tracking [47,48]. Using these apps, each individual can easily record and confirm information about his or her dietary intake, physical activity, and sleep. Furthermore, by linking the hospital's electronic medical record (EMR) with the smartphone app, a system is created that can summarize and present the patient's lifestyle information, such as dietary intake, physical activity, sleep, and stress on the physician's EMR screen [49]. That data is the life log data, which is the sum of the patient's lifestyle information and the hospital's clinical data, easily and conveniently constructed using the smart device and used in the medical environment.

With this system, doctors can check items such as dietary intake and physical activity during treatment through the lifestyle summary, and encourage additional lifestyle improvements based on the patient's actual life data in the clinic. Enabling patients to schedule and coordinate activities and set diet goals for the next hospital visit can motivate the patients to evaluate his or her lifestyle data with their doctor using the hospital EMR, and is much more effective than examining it alone on their smartphone. That is, with the use of EMR-linked smartphone apps, it is now possible to boost the effectiveness of lifestyle modification [50]. In a lifestyle modification study of patients with OSA using the above system for 4 weeks, BMI was significantly lower in the experimental group that used an EMR-linked smartphone app than in the control group, and a significant improvement was shown in the lowest oxygen saturation levels and percentage of sleep time snoring at > 45 dB [50].

CONCLUSIONS

In the treatment of OSA, physicians are aware of the need for lifestyle modifications, but often fail to recommend them for patients using CPAP, intraoral devices, and surgery due to constraints of treatment time. Additionally, the medical staff may overlook or underestimate the effectiveness of lifestyle modification on OSA. However, considering the treatment effect that can be obtained through weight loss and exercise and the adverse effects of smoking and alcohol on OSA, lifestyle modification is the most basic and necessary treatment in the entire treatment course of OSA. As such, possible benefits of the lifestyle modification should be explained to the patients. In addition, efforts should be made to improve lifestyle using smart devices that are easily accessible to patients in real life, and the

results of which can be discussed together with doctors in a treatment environment. The use of smart devices represents an improvement over the traditional method based on private counseling, which is time consuming and costly both to patients and medical staff.

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Conflicts of Interest

The authors have no financial conflicts of interest.

Authors' Contribution

Conceptualization: Kim JW. Data curation: Kim JW. Formal analysis: Kim JW. Funding acquisition: Kim JW. Methodology: Kim JW. Project administration: Kim JW. Resources: Lim HJ, Kim JW. Software: Lim HJ. Supervision: Kim JW. Validation: Kim JW. Visualization: Lim HJ. Writing—original draft: Lim HJ. Writing—review & editing: Kim JW.

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