

# Obstructive sleep apnea is a common disorder in the population— a review on the epidemiology of sleep apnea

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**Abstract:** The prevalence of obstructive sleep apnea (OSA) defined at an apnea-hypopnea index (AHI)  $\geq 5$  was a mean of 22% (range, 9-37%) in men and 17% (range, 4-50%) in women in eleven published epidemiological studies published between 1993 and 2013. OSA with excessive daytime sleepiness occurred in 6% (range, 3-18%) of men and in 4% (range, 1-17%) of women. The prevalence increased with time and OSA was reported in 37% of men and in 50% of women in studies from 2008 and 2013 respectively. OSA is more prevalent in men than in women and increases with age and obesity. Smoking and alcohol consumption are also suggested as risk factors, but the results are conflicting. Excessive daytime sleepiness is suggested as the most important symptom of OSA, but only a fraction of subjects with AHI  $>5$  report daytime sleepiness and one study did not find any relationship between daytime sleepiness and sleep apnea in women. Stroke and hypertension and coronary artery disease are associated with sleep apnea. Cross-sectional studies indicate an association between OSA and diabetes mellitus. Patients younger than 70 years run an increased risk of early death if they suffer from OSA. It is concluded that OSA is highly prevalent in the population. It is related to age and obesity. Only a part of subjects with OSA in the population have symptoms of daytime sleepiness. The prevalence of OSA has increased in epidemiological studies over time. Differences and the increase in prevalence of sleep apnea are probably due to different diagnostic equipment, definitions, study design and characteristics of included subjects including effects of the obesity epidemic. Cardiovascular disease, especially stroke is related to OSA, and subjects under the age of 70 run an increased risk of early death if they suffer from OSA.

**Keywords:** Epidemiology; population-based; sleep apnea; prevalence

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## Introduction

Obstructive sleep apnea (OSA) is characterized by recurrent episodes of partial and complete airway obstructions during sleep with repetitive apneas and hypopneas as a result (1). The disease severity is measured using the apnea-hypopnea index (AHI), i.e., the mean number of apneas and hypopneas per hour of sleep. OSA is defined when the AHI is  $\geq 5$  and OSA syndrome when AHI  $\geq 5$  is accompanied with daytime sleepiness (1).

The American Association of Sleep Medicine defined daytime sleepiness as mild, moderate and severe in relation to impact on social life during the daytime (1). The Epworth Sleepiness Scale (ESS) is, however, the most often used measure to define daytime sleepiness (2,3). Diagnostic equipment and definitions of oxygen desaturations, apnea, hypopnea, OSA and daytime sleepiness has, changed over time, which in turn affects estimates of the prevalence of sleep apnea.

In the first prevalence surveys when sleep apnea was considered a rare disorder, sleep recordings were only performed in sub-samples with a high risk of OSA in a first-stage screening procedure, and the estimated prevalence in the whole population was based on the assumption that there was no OSA at all among the remaining participants. The estimated prevalence of OSA syndrome in these studies ranged from 0.7% to 3.3% (4-8).

Patients in sleep clinic cohorts have all been referred for the diagnostic sleep test because of symptoms suggestive of the diagnosis and they are most frequently heavy snorers suffering from daytime sleepiness. Epidemiological studies on sleep apnea will identify all subjects with OSA defined as AHI  $\geq 5$ . However, only part of them will have symptoms such as snoring and daytime sleepiness reflecting subjects eligible for sleep apnea investigation on clinical grounds. This article reviews the epidemiology of OSA on prevalence and associated factors including possible risk factors and consequences.

## Prevalence of OSA

We identified eleven population-based epidemiological studies from US, China, Spain, India, Korea, Japan and Sweden published between 1993 and 2013. All eleven studies were done in two stages. In stage one, they send postal questionnaires to a random sample of the population. In stage two, they investigated a random sample of responders from stage one, with oversampling of subjects who reported snoring and daytime sleepiness and then weighted their results to the population. The prevalence of OSA defined at an AHI  $\geq 5$  were a mean of 22% (range, 9-37%) in men and 17% (range, 4-50%) in women (Table 1, Figure 1) (3,8-17). OSA syndrome defined as apnea-hypopnea index  $\geq 5$  and excessive daytime sleepiness occurred in 6% (range, 3-18%) of men and in 4% (range, 1-17%) of women (Figure 1). The prevalence in different studies has increased with time and OSA in the last studies was reported in 37% of men and in 50% of women (3,16). The differences over time could be due to different equipment and definitions for the apnea-hypopnea scoring. There are also differences in study design and populations. The results may also be affected by an increased amount of obese subject due to the obesity epidemic.

## Associated factors with OSA

### Gender

OSA is more common in men than women. The male-

to-female ratio is estimated to about 2:1 in the general population (Table 1, Figure 1) and the prevalence of snoring shows similar gender differences (18-20). The male predominance is higher in clinical populations (21,22). Possible explanations for the male predominance include hormonal effects on upper airway muscles and collapsibility, gender differences in body fat distribution and differences in pharyngeal anatomy and function. Hormonal influences could play an important role in the pathogenesis of OSA, as the prevalence seems to be higher in post- versus pre-menopausal women (17,23). The pathophysiological roles of hormones are, however, unclear and the gender differences in prevalence remained also in the elderly (24).

A recent study, by Franklin *et al.*, reported that sleep apnea occurs in as much as 50% of females aged 20-70 years old in the population (3). There was no relationship between OSA and daytime sleepiness in this study. Instead hypertension, obesity and age were associated with sleep apnea in females. It is, thus, possible that sleep apnea has not been observed as a public health problem in females, as they have other signs of sleep apnea than males.

### Age

Snoring frequency increases with age up to 50 to 60 years old and then decrease in both men and women (6,18,25,26). The prevalence of OSA also increases with age independent of other risk factors including obesity (3,8,27,28). On the contrary to snoring, the prevalence of OSA still increases also after the age of 60 years (3,8,10). Bixler *et al.* reported an increase in OSA after 65 years but the frequency of OSA syndrome declined (8). The above findings indicate that self-reported snoring and doctor-diagnosed OSA syndrome display similar age distributions with a decline at older ages in contrast to the age distribution of OSA with an AHI over five that increase with age also in the elderly.

Several studies have reported little or no association between sleep-disordered breathing and morbidity and mortality at older ages, and it has been suggested that sleep apnea in seniors represents a specific entity compared with middle-aged adults (29).

### Obesity

Obesity is a major risk factor for snoring and sleep apnea and a majority of patients with OSA are overweight (3,30-32). Caloric restriction or bariatric surgery reduces the severity of sleep apnea (28,33-36). One randomized controlled study

**Table 1** Population-based studies on the prevalence of obstructive sleep apnea (OSA) and OSA syndrome

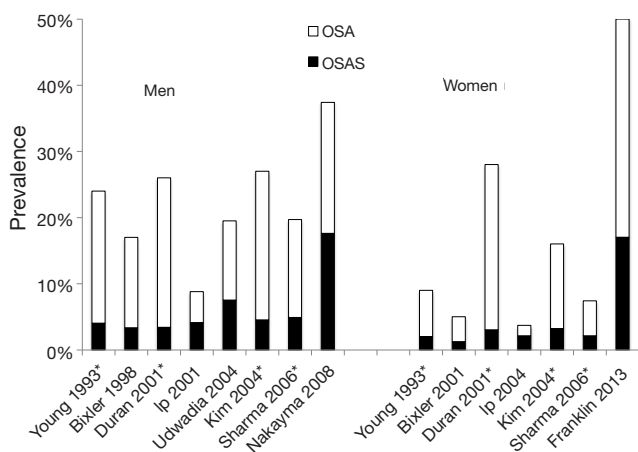
Ref./study population	Sample size gender and criteria	Estimated prevalence of AHI $\geq 5$ (%)	Estimated prevalence of AHI $\geq 15$ (%)	Estimated prevalence of OSA syndrome (%)	Methodology	Hypopnea definition
Young <i>et al.</i> , 1993 (9)						
Random sample of 3,513 state employees in Wisconsin, USA; age: 30-60 years	M: 352 W: 250 Habitual snorers (n=355) and a random sample of non-habitual snorers	M: 24 W: 9	M: 9 W: 4	M: 4.0 W: 2.0	Attended PSG	Discernible reduction in airflow and $\geq 4\%$ oxygen desaturation
Bixler <i>et al.</i> , 1998 (8)						
Random sample of 4,364 men in Pennsylvania, USA; age: 20-100 years	M: 741 Age-stratified cohorts. Oversampling of high-risk individuals	M: 17	M: 7	M: 3.3	Attended PSG	Discernible reduction in airflow and $\geq 4\%$ oxygen desaturation
Durán <i>et al.</i> , 2001 (10)						
2,148 subjects from the general population in Vitoria-Gasteiz, Spain; age: 30-70 years	M: 325 W: 235 Tentative diagnosis of OSAH (n=390) and a random sample	M: 26 W: 28	M: 14 W: 7	M: 3.4 W: 3.0	Attended PSG	50% airflow reduction and either $\geq 4\%$ oxygen desaturation or an EEG arousal
Ip <i>et al.</i> , 2004 (11,12)						
Male (n=1,542) and female (n=1,532) office workers in Hong Kong, China; age: 30-60 years	M: 153 W: 106	M: 8.8 F: 3.7	M: 5.3 W: 1.2	M: 4.1 W: 2.1	Attended PSG	Discernible reduction in airflow and $\geq 4\%$ oxygen desaturation
Udwadia <i>et al.</i> , 2004 (13)						
658 healthy men coming to hospital for routine health check in Bombay, India; age: 35-65 years	M: 250 All snorers (n=171) and 25% of non-snorers	M: 19.5	M: 8.4	M: 7.5	Home PSG	Discernible 50% reduction in airflow and $\geq 4\%$ oxygen desaturation
Kim <i>et al.</i> , 2004 (14)						
Population-based sample of 5,020 residents in Seoul, Korea; age: 40-69 years	M: 309 W: 148 Oversampling of habitual snorers	M: 27 W: 16	M: 10.1 W: 4.7	M: 4.5 W: 3.2	Home or in laboratory PSG	Discernible reduction in airflow and $\geq 4\%$ oxygen desaturation

Table 1 (continued)

Table 1 (continued)

Ref./study population	Sample size gender and criteria	Estimated prevalence of AHI $\geq 5$ (%)	Estimated prevalence of AHI $\geq 15$ (%)	Estimated prevalence of OSA syndrome (%)	Methodology	Hypopnea definition
Sharma <i>et al.</i> , 2006 (15)						
2,400 citizens in Delhi, India; exclusion criterion: several diseases; age: 30-60 years	M: 88 W: 63 Habitual snorers (n=77) and non- snorers	M: 19.7 W: 7.4	n/a	M: 4.9 W: 2.1	Attended in laboratory PSG	Discernible 50% reduction in airflow and $\geq 4\%$ oxygen desaturation
Nakayama-Ashida <i>et al.</i> , 2008 (16)						
466 male employees of a wholesale company in Osaka, Japan; age: 23-59 years	M: 322	M: 37.4	M: 15.7	M: 17.6	Home type 3 portable monitors and actigraphy	$\geq 50\%$ reduction in nasal pressure or respiratory effort and $\geq 3\%$ oxygen desaturation
Franklin <i>et al.</i> , 2013 (3)						
Population-based sample of 10,000 women in Uppsala, Sweden; age: 20-70 years	W: 400 Oversampling of habitual snorers	W: 50	W: 20	W: 17	Home PSG	$\geq 50\%$ reduction in oro-nasal thermistor and nasal pressure, and $\geq 3\%$ oxygen desaturation

OSA, obstructive sleep apnea; OSAH, obstructive sleep apnea hypopnea; PSG, polysomnography; M, men; W, women.



**Figure 1** Prevalence of OSA = AHI  $> 5$  and OSAS AHI  $> 5$  + daytime sleepiness men and women in different populations. OSA, obstructive sleep apnea; \*, men and women in the same publication.

reported a decrease in AHI using very low calorie diet (37). Another recent study reported that despite an effect of diet on AHI compared with continuous positive airway pressure (CPAP), patients were still better off with the combination of diet and CPAP than with CPAP alone (38). Men are more likely than women to increase their AHI at a given weight gain regardless of starting weight, waist circumference, age, or ethnicity (39).

Obesity is believed to predispose to OSA because of mass loading in the upper airway (40). Controversy remains whether specific measures of body habitus, such as neck or waist circumference, are better predictors of sleep-disordered breathing as compared with body mass index (BMI) alone. Neck circumference was in a population-based sample more important as a risk factor for snoring with increasing BMI in obese than in lean women (26).

Young *et al.* estimated that 58% of moderate to severe cases of OSA is due to a BMI of  $\geq 25$  kg/m<sup>2</sup> (34). This

highlights the need for effective strategies to implement long-term weight-loss programs to prevent OSA and the ongoing epidemics of obesity. Not only subjects with obesity and fat necks suffer from sleep apnea, but also lean subject and about one-third of OSA syndrome patients are non-obese (41). Franklin *et al.* reported that 39% of normal weighted women had OSA but only 0.1% of them had severe sleep apnea (3).

### Smoking

Several cross-sectional epidemiological surveys observed significant associations between cigarette smoking and snoring or sleep apnea (18,19,42-46). Possible underlying mechanisms include airway inflammation and sleep instability from overnight nicotine withdrawal (47). Never-smokers who have been exposed to passive smoking on a daily basis display an increase in the odds of being a habitual snorer of 1.6 (95% CI, 1.2-2.1) after adjusting for age and BMI according to the Respiratory Health in Northern Europe Study (44). In a Swedish longitudinal study, smoking predicted the development of snoring in men younger than 60 years old but not in older ones (25).

Wetter *et al.* found a dose-response relationship between smoking and the severity of sleep apnea. Heavy smokers ran the greatest risk, while former smoking was unrelated to snoring and sleep-disordered breathing after adjustment for confounders (46). Smoking is, however, not an established risk factor for OSA. In the analysis from the Sleep Heart Health Study, smokers actually displayed less sleep apnea than non-smokers and there are still no available data on the impact of smoking on the incidence and remission of sleep apnea (48).

### Alcohol

Alcohol intake reduces motor output to the upper airways with hypotonia of the oropharyngeal muscles as a result (49). In studies performed in the laboratory, alcohol increases both the number of apneas and the duration of apnea (50,51). The results did, however, diverge, when the relationship between chronic alcohol use and snoring or sleep apnea was analyzed in epidemiological studies and an association was found by some but not by others (13,25,30,52-54). Svensson *et al.* reported that alcohol dependence was only related to snoring in lean women with a BMI of <20 kg/m<sup>2</sup> (26). It is thus possible that the alcohol-induced reduction in motor output to the upper airways is more important in lean women

without compromised upper airways from fat deposits and overweight.

### Excessive daytime sleepiness

Excessive daytime sleepiness is regarded as the most common and most important symptom of OSA. Numerous randomized controlled trials have demonstrated a significant improvement in daytime sleepiness when such patients are effectively treated with CPAP as compared to sham CPAP or oral placebo (55-60).

Daytime sleepiness is related to OSA and snoring in the general population studies (9,61-63). In the Wisconsin Sleep Cohort Study, about 23% of the women with an AHI of  $\geq 5$  reported excessive daytime sleepiness compared with only 10% of non-snoring women (9). The corresponding prevalence in men was 16% and 3% respectively. Similar findings were reported from the Sleep Heart Health Study using the ESS with a significant, progressive increase in sleepiness with increasing AHI in both older and younger subjects and independent of gender, age and BMI (61).

The evidence of apnea induced daytime sleepiness is, however, weak as only a fraction of patients with OSA in the population report daytime sleepiness. Attempts to find the suggested association between the arousals and sleepiness have also failed (64,65). Daytime sleepiness can be due to a number of factors and OSA patients may have suffered from other disorders of sleepiness than sleep apneas. Svensson *et al.* reported that snoring, but not sleep apnea (AHI >15) was related to excessive daytime sleepiness (63). The association between OSA and sleepiness is also less evident in patients with chronic disease such as in patients with congestive heart failure who report less daytime hypersomnolence regardless of whether they have OSA or not (66). Sleepiness is also frequently reported in the absence of OSA in elderly people and in patients with end-stage renal disease (67,68).

### Hypertension

Sleep apnea and hypertension are both prevalent in the community and many individuals suffer from both. Several large population-based, cross-sectional studies reported an independent association between the two conditions (3,10,69-72). Self-reported snoring is also a predictor of developing hypertension in both males and females (43,73,74). Peppard *et al.* analyzed the odds ratios for the presence of hypertension at a 4-year follow-up among 709 middle-aged participants in the Wisconsin cohort, all of

who had been investigated with polysomnography at baseline. Compared with subjects with no OSA, the adjusted odds ratio for prevalent hypertension at follow-up was 2.03 (95% CI, 1.29-3.17) for mild OSA (AHI, 5-14.9) and 2.89 (95% CI, 1.46-5.64) for moderate to severe OSA (AHI  $\geq$ 15) (75). The same group also provided data from a sub-group who were followed-up after a mean of 7 years using 24-hour blood pressure studies. Regardless of confounders including baseline blood pressure and progress of sleep apnea, there was a significant dose-response relationship between the severity of sleep apnea at baseline and the risk of developing systolic non-dipping blood pressure during sleep (76).

The impact of snoring and OSA on hypertension is less pronounced in overweight and obese subjects when compared with normal-weights in population-based samples (43,69,71). Analyzed by age group, there is an independent relationship of snoring or OSA on hypertension among young and middle-aged participants, but not in the elderly (71-73,77,78). An AHI of  $\geq$ 15 was independently associated with hypertension in subjects aged <60 years, with an adjusted odds ratio of 2.38 (95% CI, 1.30-4.38), among 6,120 participants in the Sleep Heart Health Study, while no such relationship was found between sleep apnea and hypertension among subjects above that age (72).

Although observational studies indicate a causal relationship between OSA and hypertension the effectiveness of reducing blood pressure by treating OSA is less clear and intervention studies using CPAP have produced mixed results (79).

### Coronary artery disease

OSA frequently coexists, but is usually being undiagnosed in patients with cardiovascular disease and several cross-sectional studies support a strong association between OSA and prevalent coronary artery disease, defined as myocardial infarction and/or angina pectoris (80-82). However, sleep apnea was assessed after coronary artery disease was established in the cited studies and thereby limits the conclusion on an etiologic relationship. Cross-sectional epidemiologic studies on self-reported coronary artery disease and snoring or objectively measured OSA have reported a positive association, although of considerably smaller magnitude than that observed in case-control studies (18,83). Among 6,424 participants who underwent in-home polysomnography in the Sleep Heart Health Study, Shahar *et al.* reported that subjects with the highest quartile of AHI >11 had an adjusted odds ratio of 1.27 of

self-reported coronary artery disease after adjusting for confounders including hypertension (84). The relative high age with a mean of 64 years old of participants at study start could be an explanation to the rather modest association.

Patients with OSA had a higher incidence of coronary artery disease (16.2%) compared with snorers without OSA (5.4%) in a prospective study over 7 years (85). Efficient treatment with CPAP significantly reduced the risk of adverse cardiovascular outcomes both when it comes to primary and secondary prevention (85-87).

Population-based prospective studies on sleep apnea and incidence of coronary artery disease are still lacking.

### Stroke

Clinical cohorts suggest an important link between sleep apnea and stroke. Spriggs *et al.* followed patients with recent stroke until death or 6 months and found that previous stroke and regular snoring were the only two risk factors that adversely affected mortality (88). Yaggi *et al.* followed 1,022 patients being investigated on clinical grounds and concluded that OSA syndrome significantly increases the risk of stroke or death from any cause, and the increase is independent of other risk factors, including hypertension (89). Valham *et al.* found a dose-response relationship between AHI at baseline among patients with coronary artery disease and the incidence of stroke during a 10-year follow-up after adjusting for potential confounders (90). Moreover, in stroke survivors, the occurrence of OSA, but not central sleep apnea, was a significant predictor of early death (91).

Population based studies also support the evidence of stroke due to OSA. Munoz *et al.* reported that severe sleep apnea (AHI  $\geq$ 30) at baseline was associated with a significantly increased risk of developing an ischemic stroke (adjusted hazard ratio 2.52, 95% CI, 1.04-6.01) from a 6-year longitudinal study of a population-based cohort, initially event-free subjects aged 70-100 years (92). Arzt *et al.* investigated a younger population-based cohort of 1,189 subjects, mean age 47 years with polysomnography. During the following 4 years, 14 subjects suffered a first-ever stroke and this was related to sleep apnea defined as an AHI of  $\geq$ 20 at baseline, although the association did not reach statistical significance after adjusting for age, gender and BMI (adjusted OR 3.08, 95% CI, 0.74-12.81) (93).

### Diabetes mellitus

Sleep-disordered breathing and diabetes mellitus share several

**Table 2** Population-based studies designed to investigate the relationship between sleep-disordered breathing and mortality

Population/ref.	n	Adjusted confounders	Marker of SDB	Adj hazard ratio; all-cause mortality	Comments
Sample of men in Uppsala, Sweden; follow-up period: 10 yrs (114)	3,100	Age, BMI, hypertension heart disease, diabetes	No snoring or EDS Snoring, no EDS EDS, no snoring Snoring and EDS	1 1.1 (0.8-1.5) 1.1 (0.6-1.9) 1.8 (1.2-2.5)	In stratified analyses: significant only in men <60 yrs [adj HR 2.7, (range, 1.6-4.5)]
Wisconsin sleep cohort; follow-up period: 18 yrs (23)	1,522	Age, gender, BMI	AHI 0-<5 AHI 5-<15 AHI 15-<30 AHI ≥30	1 1.6 (0.9-2.8) 1.4 (0.6-3.3) 3.0 (1.4-6.3)	No sign interaction with age, gender or EDS
Sleep Heart Health Study Average; follow-up 8.2 yrs (115)	6,441 Mean age 62.9±11.0	Age, race, BMI, gender, smoking, diabetes, blood pressure, cardiovascular disease	AHI 0-<5 AHI 5-<15 AHI 15-<30 AHI ≥30	1 0.93 (0.80-1.08) 1.17 (0.97-1.42) 1.46 (1.14-1.86)	In stratified analyses the adj hazard ratio was only significant in men <70 [adj HR 2.09 (range, 1.31-3.33)]

Adj, adjusted; EDS, excessive daytime sleepiness.

risk factors. Insulin resistance and/or type 2 diabetes mellitus coexist with snoring or sleep apnea in general population cross-sectional studies independent of obesity and other confounders (94-101). Furthermore, an independent association between self-reported snoring and incident diabetes is reported in both males and females (102,103).

Longitudinal studies on OSA as a risk factor for future diabetes mellitus have not been conclusive. Among 1,387 participants in the Wisconsin Sleep Cohort, subjects with an AHI ≥15 did not differ significantly from those with an AHI of <5 when it came to the risk of developing diabetes mellitus over a 4-year period (OR 1.62; 95% CI, 0.7-3.6) when adjusting for age, gender, and body habitus (99). Similar findings were reported from the Busselton health study (104). On the contrary, Botros *et al.* found an independent association between sleep apnea at baseline and incident diabetes in an observational cohort study including 1,233 consecutive patients without diabetes (105). Also in a long-term follow-up of a community-based sample of men there was an independent association between oxygen desaturation index >5 at baseline and incident diabetes mellitus at follow-up after 11 years (OR 4.4; 95% CI, 1.1-18.1), after adjusting for age, BMI, and hypertension at baseline and delta BMI and years with CPAP during follow-up (106).

## Mortality

Clinic-based studies suggest that patients with OSA syndrome have a higher mortality risk (107) and that

treatment with tracheostomy or CPAP attenuates this risk (108-110). The lack of randomized, controlled interventional trials clearly limits the evidence, as non-treated patients have either been non-compliant with prescribed therapy or have for some reason not been selected for effective treatment. Clinical mortality studies might also be biased, as patients under treatment for some other serious morbidity might also be more likely to be referred for an evaluation of sleep apnea, leading to an overestimation of mortality.

The results diverge in studies investigating whether patients with OSA syndrome have a shorter survival or not. No increased mortality rate was found between apnea-hypopnea scores in two prospective studies investigating elderly populations (111,112), while a significant association was seen in another study, but in women only (113). Lavie *et al.* in a prospective study found that the apnea index was a predictor of excess mortality in the fourth and fifth decade but not in elderly men (107). This is in accordance with the results from of a population-based study from Uppsala in Sweden where men aged 30-69 years were investigated by postal questionnaire and followed over 10 years (114). Snoring men reporting excess daytime sleepiness had a significant increase in mortality, but the age-adjusted relative risk decreased with increasing age and was no longer significant after age 50 years. Snoring alone had no impact on mortality in any of the age groups (Table 2).

The impact of OSA on mortality in population-based cohorts has recently been analyzed in the Wisconsin Study (23), and in the Sleep Heart Health Study (115), and both

reported an increased mortality rate with increasing severity of sleep apnea (Table 2). Subjects with an AHI  $\geq 30$  had an adjusted hazard ratio for all-cause mortality of 3.0 (95% CI, 1.4-6.3) and 1.46 (95% CI, 1.14-1.86) respectively, compared with those with an AHI of  $< 5$ . Similar results were obtained for cardiovascular mortality in both studies and the exclusion of subjects treated for sleep apnea did not change the results. However, the adjusted hazard ratios for severe sleep apnea only remained significant in younger men  $< 70$  years in the Sleep Heart Health Study.

## Conclusions

OSA is highly prevalent in the population. It is related to age and obesity. Only a part of subjects with OSA in the population have symptoms in the form of daytime sleepiness. The prevalence of OSA and OSA syndrome has increased in epidemiological studies over time. Differences and the increase in prevalence of sleep apnea are probably due to different diagnostic equipment, definitions, study design and characteristics of included subjects. Cardiovascular disease, especially stroke is related to OSA and subjects under the age of 70 run an increased risk of early death if they suffer from OSA.

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## Footnote

*Conflicts of Interest:* The authors declare no conflict of interest.

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