Obstructive sleep apnoea in the general population: highly prevalent but minimal symptoms

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ABSTRACT The aim was to assess the prevalence of obstructive sleep apnoea (OSA) as defined by an apnoea–hypopnea index (AHI) ⩾15 in the middle-aged general population, and the interrelationship between OSA, sleep-related symptoms, sleepiness and vigilance.

A general population sample of 40–65-year-old Icelanders was invited to participate in a study protocol that included a type 3 sleep study, questionnaire and a psychomotor vigilance test (PVT).

Among the 415 subjects included in the study, 56.9% had no OSA (AHI <5), 24.1% had mild OSA (AHI 5–14.9), 12.5% had moderate OSA (AHI 15–29.9), 2.9% had severe OSA (AHI ⩾30) and 3.6% were already diagnosed and receiving OSA treatment. However, no significant relationship was found between AHI and subjective sleepiness or clinical symptoms. A relationship with objective vigilance assessed by PVT was only found for those with AHI ⩾30. Subjects already on OSA treatment and those accepting OSA treatment after participating in the study were more symptomatic and sleepier than others with similar OSA severity, as assessed by the AHI.

In a middle-aged general population, approximately one in five subjects had moderate-to-severe OSA, but the majority of them were neither symptomatic nor sleepy and did not have impaired vigilance.

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Overall, 15% of the general population had untreated OSA without symptoms, sleepiness or decreased vigilance http://ow.ly/StiqS

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Introduction

Obstructive sleep apnoea (OSA) was considered a very rare disorder 30 years ago [1], but recent epidemiological studies show an astonishingly high prevalence. Moderate to severe OSA has been found in up to 50% of men and 25% of women in the middle-aged population as defined by an apnoea–hypopnea index (AHI) ⩾15 [2, 3].

Over the past 30 years, obesity levels have risen dramatically, partly explaining the increase in OSA prevalence [1]. However, increased obesity may not be the only explanatory variable as the measurement techniques and scoring criteria have also changed markedly over the same period [4–6]. These changes make the relevance of the AHI ⩾15 cut-off levels in today's studies unclear, as these cut-off levels were set using measurement techniques and scoring criteria other than those currently used [4].

The AHI as the sole criterion for having OSA is problematic, as AHI rises continuously with age and is highest in the elderly [7], while the association between OSA and hypertension, cardiovascular diseases [8, 9] and relative mortality rate is much higher in younger subjects with OSA [10]. In addition, previous studies have shown that AHI is poorly associated with many of the classical symptoms and the physiology of OSA as measured by, for example, quality of life, sleepiness and performance [11, 12].

Recent studies have shown that many of those diagnosed with OSA do not suffer from subjective sleepiness [13], previously considered a cardinal symptom of OSA [4]. Sleepiness is not a requirement for diagnosis of OSA, as shown by the diagnostic criteria of the International Classification of Sleep Disorders, third edition (ICSD-3) [14]. Also, while studies have shown an improvement in objective vigilance with positive airway pressure (PAP) treatment [15, 16], a relationship between OSA severity and vigilance in the middle-aged general population has not been found [17].

The aim of this study was to assess the prevalence of OSA as measured by AHI ⩾15, in a well-defined middle-aged general population sample, and to assess the relationship of OSA with sleep-related symptoms, subjective sleepiness and objective vigilance.

Material and methods

Subjects

The subjects in the present study were Icelandic general population subjects in the European Community Respiratory Health Survey (ECRHS) [18]. The study participants were first studied in 1990 [18], again in 2000 [19] and for the third time in 2012 (online supplementary material). Among the 522 subjects contacted for participation in ECHRS III, 40 refused to participate, eight had moved, two had died, six were untraceable and a further two did not find the time to participate before the study was closed in May 2013. Nine subjects responded over the phone whereas a total of 455 came to the outpatient clinic for participation, or 87% of those invited.

Consent for the study was granted by the national bioethics committee (VSN-11-121) and by the data protection authority of Iceland, and written consent was obtained from all the research subjects.

Questionnaires and anthropometric measurements

Subjects answered questions on smoking, general health and chronic diseases and listed their medication use in a manner similar to that of the previous ECHRS studies [18, 19]. Additionally, a questionnaire on sleep habits, symptoms and disorders was administered to all subjects, developed by the Sleep Apnea Genetics International Consortium (SAGIC) [20]. The SAGIC questionnaire uses the same format as the Basic Nordic Sleep Questionnaire [21] when asking about symptoms of insomnia, snoring, nocturnal sweating, nocturnal gastro-oesophageal reflux (nGOR), *etc.* with the addition of a new "don't know" response alternative on a scale of 1–6: 1) never or very seldom; 2) less than once a week; 3) once to twice a week; 4) three to five times a week; 5) every day or almost every day of the week; and 6) don't know. The Epworth Sleepiness Scale (ESS) [22] was administered, as well as questions on restless leg syndrome (RLS) [23] (defined in [24]). Habitual snoring was defined as reported snoring more than three times a week in the Berlin questionnaire [25]. All questionnaires were translated from English into Icelandic and back-translated to assure accuracy. Height and weight were measured in the same manner for all participants.

Assessment of vigilance

Vigilance performance was assessed using the psychomotor vigilance test (PVT) [26] (PVT-192; CWE, Inc., Ardmore, PA, USA). The test was performed over a 10-min period after a single 1-min acclimation practice, between 09:00 h and 16:30 h on the day following the sleep study. The following PVT outcomes were analysed in a similar manner as in previous publications [17, 27]: 1) mean response speed defined as the reciprocal of mean response time (RT) in milliseconds (ms^{-1}); 2) number of transformed lapses (RT ⩾500 ms) subjected to the Tukey transformation ($\sqrt{x}+\sqrt{(x+1)}$); 3) number of response errors (false

starts and wrong button presses) subjected to the Tukey transformation; 4) mean of the reciprocal of the fastest 10% RTs; 5) mean of the reciprocal of the slowest 10% RTs; 6) slope of linear regression line across the 10 min of the task; and 7) sum of Tukey-transformed lapses and response errors. Five subjects had >20 errors, indicating poor compliance with the study instructions, and were therefore removed from the PVT analysis.

Whole-night studies

All subjects who participated in the ECRHS III were invited for a type 3 sleep study [28] using a T3 device (Nox Medical, Reykjavik, Iceland). Nasal airflow was recorded through a cannula. Chest and abdominal movements were measured using respiratory inductive plethysmography belts. Pulse and oxygen desaturation were measured by a finger probe oximeter based on a four-beat exponential average (Nonin Medical Inc., Plymouth, MN, USA). Body position, activity and audio were measured using sensors situated on the chest.

The sleep studies were scored by two trained sleep technologists. Studies had to have ⩾4 h of scorable oxygen saturation and more than two out of three respiratory traces: cannula flow, thorax and respiratory inductive plethysmography belts. Subjects were invited to undertake a repeat study if the study was not of acceptable quality for analysis. Total analysis time was assessed based on questionnaires and the sleep technologist's review of the study. Sleep studies were scored in accordance with the American Academy of Sleep Medicine (AASM) 2007 manual [6], using the accepted hypopnea classification requiring a ⩾30% drop in respiratory flow for ⩾10 s with ⩾4% oxygen desaturation.

Statistical methods

For bivariate analysis, the Chi-squared test and t-test (one-way ANOVA if more than two groups) were used for nominal and continuous variables, respectively, whereas logistic and linear regression were used for the respective multivariable analyses. Means are presented as ±SD, unless otherwise indicated. Additional sensitivity analyses were performed, using the AHI and oxygen desaturation index linearly (square-root transformed) and AHI >15 or AHI >30 cut-offs in events per hour instead of the four OSA severity categories. All of these analyses led to the same conclusions as the primary analysis. STATA (12.0; StataCorp, College Station, TX, USA) was used for all statistical analyses.

Results

The study cohort

Among the 455 participants, 28 declined to participate in a whole-night study, 11 recordings were technically unacceptable and one subject declined the weight measurement (fig. 1). No differences were found between the excluded and included subjects (online supplementary table S1).

A total of 415 were therefore included in the study cohort: n=400 with usable sleep studies and 15 patients previously diagnosed and already on treatment for OSA (table 1). The mean±SD age of the cohort was 54.7±6.8 years (range 42–66 years), BMI was 28.2±5.0 kg·m^{-2}, 16.6% were current smokers and 30.6% had doctor-diagnosed hypertension. In addition, 2.9% of the cohort had doctor-diagnosed type 2 diabetes, 2.7% had ever had a stroke and 2.7% had been diagnosed with angina, heart attack or coronary heart disease.

Males had a higher severity of OSA on average and were more likely to report habitual snoring than females. However, females had a higher prevalence of sleep-related symptoms such as nocturnal sweating, dry mouth upon awakening and morning headaches, difficulties initiating sleep and RLS symptoms than males (table 1).

Prevalence of OSA

The sleep study for the 400 subjects with previously untreated OSA showed that 236 subjects (59.0%) had no OSA, 100 (25.0%) had mild OSA (AHI 5–14.9), 52 (13.0%) had moderate OSA (AHI 15–29.9) and 12 (3.0%) had severe OSA (AHI ⩾30) (table 2). Subjects with OSA were more likely to be male, older and obese than other subjects (fig. 2). Sex, age and BMI were all significant variables in explaining the AHI in a linear regression analysis, explaining a total of 23.5% of the variance in AHI levels. However, the OSA severity groups did not differ with regard to smoking history or hypertension. In addition, subjects with moderate or severe OSA were not more likely to have cardiometabolic disease than subjects with no or mild OSA.

OSA and sleep-related symptoms

The four OSA severity groups were assessed with regard to sleep-related symptoms. A significant difference between groups was only found for habitual snoring ($p<0.0001$). Other sleep-related symptoms did not differ between the OSA severity groups (table 2). A logistic regression for individual sleep-related symptoms, adjusting for age, sex and BMI confirmed these findings.

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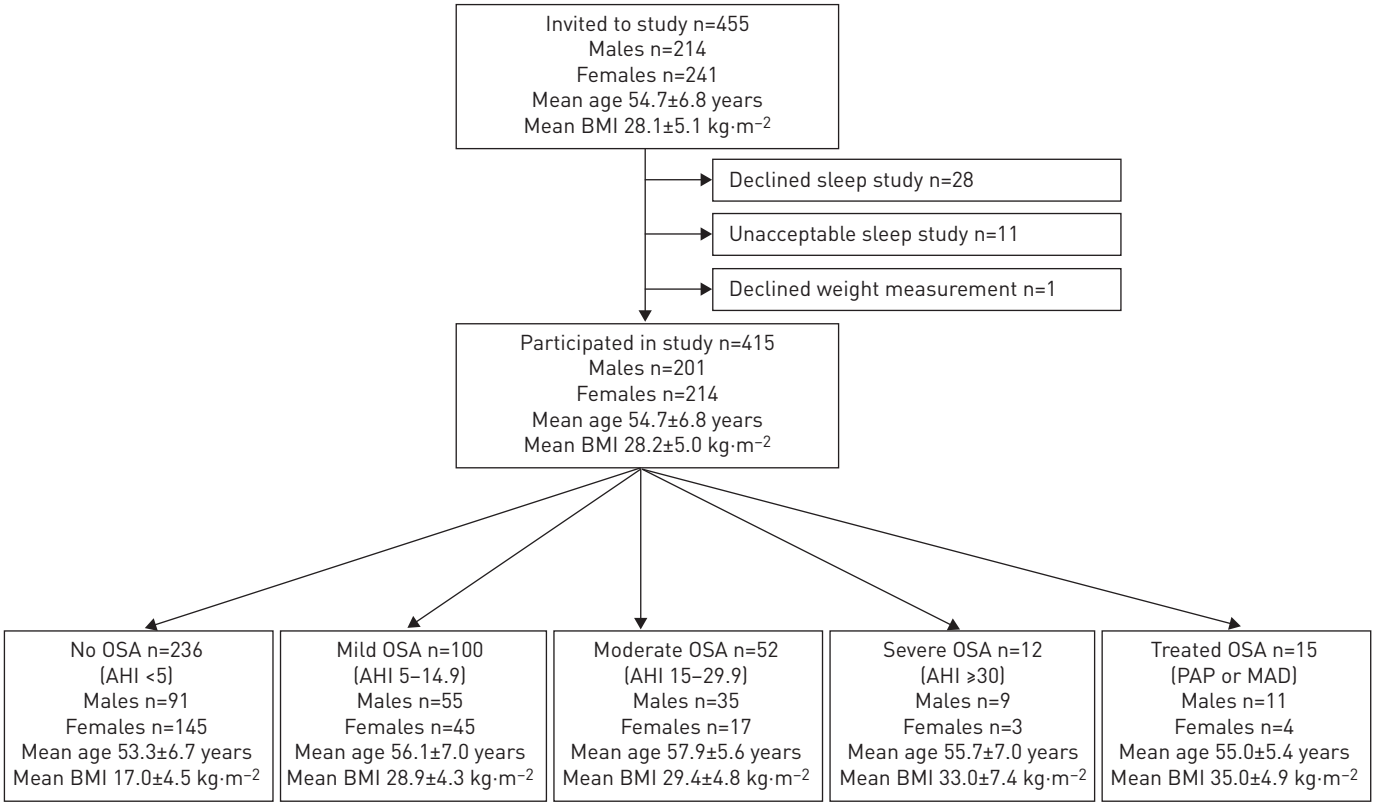

FIGURE 1 Flowchart of the study participants. BMI: body mass index; OSA: obstructive sleep apnoea; AHI: apnoea–hypopnea index; PAP: positive airway pressure; MAD: mandibular advancement device.

Relationship between sleep disordered breathing and sleepiness

Subjective sleepiness

Among the 400 subjects, 371 fully answered the ESS questionnaire. Lower age and higher BMI were significantly related to the total ESS score but no effects of sex were found. No differences were found in subjective sleepiness between OSA severity groups as assessed by the total ESS score or ESS ⩾10 (table 3 and fig. 3a). Regression analyses adjusting for age, sex and BMI confirmed these findings.

Objective vigilance

Males had on average faster reaction speeds in the PVT than females (reaction speed 4.2±0.5 ms^{-1} *versus* 4.1±0.4 ms^{-1}, p=0.007) but also made more errors than females (3.0±1.8 ms^{-1} *versus* 2.6±1.6 ms^{-1}, p=0.01). The reaction speed decreased significantly with higher age and higher BMI.

A significant difference in objective vigilance, as assessed by reaction speed as well as number of lapses, was found between OSA severity groups (table 3 and fig. 3b). However, the differences found were driven solely by the AHI ⩾30 group (n=12). A linear regression analysis adjusted for sex, age and BMI confirmed these findings as no overall differences were found between the OSA severity groups (table 3). However, the AHI ⩾30 group was significantly or borderline different from the "no OSA group" as a reference group in the adjusted analysis with regards to mean reaction speed (p=0.02), fastest 10% reaction speed (p=0.07), slowest 10% reaction speed (p=0.06), number of lapses (p=0.06) and total errors (p=0.046).

To assess whether a lower AHI cut-off would be more appropriate than the classical AHI ⩾30 for severe OSA, the moderate OSA group was divided in two by the median value (AHI 19.2). However, no significant differences were found with regards to any PVT performance parameters between those with moderate "low" and moderate "high" OSA severity as defined by the AHI.

No significant relationship was found between objective vigilance as assessed by any of the above PVT variables and subjective sleepiness as assessed by the total ESS score.

Clinical follow-up of subjects diagnosed with untreated OSA

After the study finished, all 64 subjects who had an AHI ⩾15 (44 males and 20 females) were invited for an interview with a sleep specialist to discuss potential treatment options. This was part of a clinical

TABLE 1 Characteristics of the population included in the sleep study analysis as well as those who refused to have a sleep study or had an unusable study

	All subjects	Males	Females	p-value
Subjects n	415	201	214	
Demographics				
Age years	54.7±6.8	54.8±6.9	54.6±6.8	0.74
Males	48.4			
BMI kg·m^{-2}	28.2±5.0	28.6±4.4	27.8±5.4	0.10
Smoking				
Current	16.6	15.4	17.8	0.34
Former	44.6	48.3	41.1	
Never	38.8	36.3	44.1	
Ever had hypertension	30.6	28.4	32.7	0.34
Sleep study				
Apnoea–hypopnea index$^{\#}$	7.3±9.3	9.5±10.4	5.2±7.7	**<0.0001**
Oxygen desaturation index$^{\#}$	6.6±8.2	8.6±9.0	4.7±6.9	**<0.0001**
Hypoxia time$^{\#,\P}$ %	4.7±12.9	5.3±12.4	4.2±13.4	**0.02**
Minimum $Sa_{O_2}$$^{\#}$ %	85.2±5.4	84.7±4.9	85.8±5.5	**0.02**
Symptoms				
Snoring ⩾3 per week	42.3	53.7	31.1	**<0.0001**
Nocturnal sweating ⩾3 per week	10.2	5.9	14.2	**0.007**
Dry mouth when awakening ⩾3 per week	18.9	14.4	22.8	**0.046**
Morning headache ⩾1 per week	14.5	10.3	18.2	**0.03**
Difficulties initiating sleep ⩾3 per week	12.6	7.9	17.1	**0.006**
Difficulties maintaining sleep ⩾3 per week	37.6	37.8	37.4	0.94
Early morning awakenings ⩾3 per week	13.5	13.2	13.7	0.87
Nocturnal GOR symptoms ⩾1 per week	6.1	6.5	5.9	0.81
RLS symptoms	15.9	11.1	20.3	**0.01**

Data are presented as mean±SD or %, unless otherwise stated. Bold type represents statistical significance. BMI: body mass index; Sa_{O_2}: arterial oxygen saturation; GOR: gastro-oesophageal reflux; RLS: restless legs syndrome. $^{\#}$: n=15 subjects currently on treatment for obstructive sleep apnoea were excluded from comparison. The data were square-root transformed or cubic transformed for statistical analysis as appropriate to normalise distribution; ¶: Sa_{O_2} <90%.

follow-up and not part of the original study protocol. Altogether, 31 (48.4%) accepted the invitation. Of these, 27 were advised to seek treatment for OSA in the interview, 26 started PAP treatment, and one started mandibular advancement device (MAD) treatment. Of the 26 subjects starting PAP treatment, 18 were still on PAP treatment 2 years later.

Subjects accepting PAP treatment were on average more obese, sleepier and reported more nocturnal sweating than the other subjects with an AHI ⩾15 (online supplementary table S2). They were also borderline and more likely to report difficulties maintaining sleep and nGOR. However, no differences were found with regard to sex, age, OSA severity, smoking, hypertension or objective vigilance.

Subjects with treated OSA

The 15 subjects receiving treatment for OSA when the study started were 11 males and four females with a mean age of 55.0±5.4 years. and mean BMI of 35.0±4.9 kg·m^{-2} (online supplementary table S3). 13 were using PAP treatment and two were using MAD. There had been a mean 6.8±4.7 years since their OSA diagnosis. When diagnosed, OSA severity was high but diverse. The mean ESS score at baseline was 10.2±4.8, significantly higher than for all other subjects in the cohort (p=0.008) as well as those with previously untreated moderate-to-severe OSA (p=0.03).

Discussion

In a middle-aged general population in Iceland, 15.4% had an AHI ⩾15 and a further 3.6% were already on OSA treatment, indicating an OSA prevalence of 19% in this population. However, the majority of the subjects with previously untreated OSA did not report sleep-related symptoms, were not sleepy and did not have decreased objective vigilance.

The prevalence of OSA in the general population in this study was similar to other recent studies [1]. These more recent studies have reported a much higher prevalence than earlier studies [1]. We believe that the

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TABLE 2 Characteristics of the study population (n=400) with no previous diagnosis of obstructive sleep apnoea (OSA) divided by OSA severity

	No OSA	Mild OSA	Moderate OSA	Severe OSA	p-value
Subjects n	236	100	52	12	
Demographics					
Age years	53.3±6.7	56.1±7.0	57.9±5.6	55.7±7.0	**<0.0001**
Males	38.6	55.0	67.3	75.0	**<0.0001**
BMI kg·m^{-2}	27.0±4.5	28.9±4.3	29.4±4.8	33.0±7.4	**<0.0001**
Smoking					
Current	17.0	17.0	17.3	25.0	0.49
Former	40.3	50.0	50.0	50.0	
Never	42.8	33.0	32.7	25.0	
Ever had hypertension	28.0	31.0	30.8	25.0	0.92
Sleep study					
Apnoea–hypopnea index[#]	1.8±1.4	9.2±2.7	20.0±3.6	43.4±9.8	**<0.0001**
Oxygen desaturation index[#]	1.9±1.6	8.1±3.6	17.4±4.6	37.8±8.0	**<0.0001**
Hypoxia time[¶] %	3.2±13.0	3.8±5.5	8.9±15.8	23.1±23.6	**<0.0001**
Minimum Sa_{O_2}[#] %	87.5±4.1	82.6±5.1	81.4±4.4	76.1±6.6	**<0.0001**
Symptoms					
Snoring ⩾3 per week	30.6	49.5	74.5	77.8	**<0.0001**
Nocturnal sweating ⩾3 per week	11.5	6.3	8.7	20.0	0.37
Dry mouth when awakening ⩾3 per week	17.1	19.5	28.6	11.1	0.33
Morning headache ⩾1 per week	15.5	12.2	17.4	0.0	0.47
Difficulties initiating sleep ⩾3 per week	13.2	10.5	16.3	20.0	0.70
Difficulties maintaining sleep ⩾3 per week	38.2	36.2	37.5	40.0	0.99
Early morning awakenings ⩾3 per week	12.0	13.7	18.4	20.0	0.61
Nocturnal GOR symptoms ⩾1 per week	5.8	6.3	6.5	20.0	0.35
RLS symptoms	13.6	17.5	25.0	10.0	0.23

Data are presented as mean±SD or percentage, unless otherwise stated. Bold type represents statistical significance. BMI: body mass index; Sa_{O_2}: arterial oxygen saturation; GOR: gastro-oesophageal reflux; RLS: restless legs syndrome. [#]: the data were square-root transformed or cubic transformed for statistical analysis as appropriate to normalise distribution; [¶]: Sa_{O_2} <90%.

"inflation" in reported OSA prevalence is at least partly explained by the changes in measurement techniques and scoring criteria for OSA over the past 30 years and is not solely explained by the increase in obesity levels. For example, in the first sleep studies a thermistor, which is not sensitive to subtle changes in airflow, was used to estimate hypopneas [29]. Also, anecdotally, a higher signal averaging time was used for the pulse oximeter in the earlier OSA studies resulting in a lower number of oxygen desaturations measured [30]. However, it is unclear to what extent this affected the number of desaturations measured as very few articles include this information. In addition, the scoring criteria have changed significantly over the years, especially for hypopneas, with some sleep laboratories using definitions requiring changes in airflow only [4], others requiring ⩾3% oxygen desaturations and/or arousals [5] and the most stringent ones requiring ⩾4% oxygen desaturations [6]. This difference in criteria has a large impact on the calculated AHI, even causing a doubling of the AHI between scoring criteria [2, 31].

The high prevalence of OSA found in the general population in this study was related to male sex, obesity and reported snoring, while women were more likely to report sleep-related symptoms. However, no relationship was found between OSA severity and diagnosed cardiometabolic diseases. Similarly, several previous studies have shown that many OSA patients do not suffer from subjective sleepiness [13, 32]. Only those few (3.6%) in the general population already on PAP treatment in our study were excessively sleepy as measured by the ESS while untreated.

We found no relationship between OSA and objective vigilance as assessed by the PVT, except in the 12 subjects with severe OSA. Therefore, subjects diagnosed with moderate OSA in the general population did not have impaired objective vigilance compared to those with no or mild OSA. Previous studies have found significantly worse performance in OSA patients compared to controls [33] and an improvement in vigilance with PAP treatment [15, 16]. However, in a community-based sample no relationship was found between OSA severity and PVT performance except in those aged ⩾65 years [17], an age range not assessed in our study cohort. Also, unlike in our study, they did not specifically test an AHI ⩾30 cut-off. Furthermore, we found no relationship between subjective sleepiness and objective vigilance performance, a dissociation known previously from sleep-deprivation studies in healthy subjects [34].

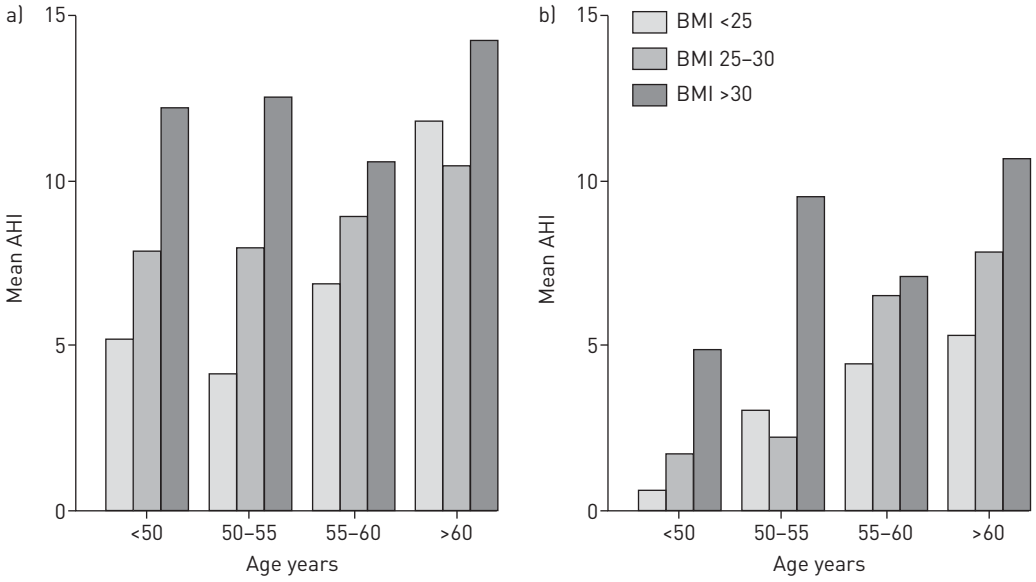

FIGURE 2 Mean apnoea–hypopnea index (AHI) by sex, age group and body mass index (BMI) category. a) males; b) females.

A simple AHI cut-off of 15 may therefore be inadequate to define who has symptomatic OSA and is in need of treatment because of daytime sleepiness or impaired objective vigilance when cardiovascular risk factors are not present. Still there are individuals in the AHI 15–30 group who, when offered PAP treatment, adjust to the treatment, especially if they are proportionally more symptomatic. In our opinion, new criteria for OSA disease severity are needed, as well as further research into new disease markers that are more related to symptoms and cardiometabolic effects than is the AHI.

The European Union has recently set new driving regulations requiring all drivers with suspected OSA as defined by an AHI ⩾15 to be referred for medical advice before issuing or renewing a driving licence, due to the relationship with excessive daytime sleepiness [35]. Also, OSA patients need to show adequate control and compliance with treatment at regular intervals to establish "continued good vigilance" and to maintain their driving licence [35]. Our study shows that these regulations are not appropriate or

TABLE 3 Subjective (Epworth Sleepiness Scale (ESS)) and objective (psychomotor vigilance test (PVT)) sleepiness for subjects within obstructive sleep apnoea (OSA) severity categories

	No OSA	Mild OSA	Moderate OSA	Severe OSA	p-value ANOVA	Adjusted p-value[#]
Subjects n	236	100	52	12		
Subjective sleepiness						
ESS score	7.0±4.0	7.0±3.8	6.7±4.1	8.3±5.4	0.74	0.90
Excessive sleepiness (ESS ⩾10)	24.8	23.9	20.8	44.4	0.51	0.95
Objective sleepiness (PVT)						
Mean reaction speed 1/RT	4.2±0.4	4.1±0.5	4.2±0.3	3.9±0.6	**0.008**	0.14
Fastest 10% reaction speed ms	5.3±0.5	5.1±0.5	5.2±0.5	5.0±0.6	**0.03**	0.22
Slowest 10% reaction speed ms	2.8±0.4	2.7±0.5	2.7±0.4	2.5±0.6	**0.03**	0.24
Minor lapses (RT >500 ms)[¶]	2.0±1.3	2.3±1.7	2.2±1.2	3.0±2.8	**0.04**	0.24
Total errors (false starts and wrong buttons)[¶]	2.8±1.7	2.7±1.7	3.1±1.7	2.1±1.3	0.20	0.39
Lapses and errors[¶]	4.8±2.2	5.0±2.4	5.3±2.4	5.1±3.5	0.61	0.90
PVT slope (time on task decrement)	−0.02±0.04	−0.02±0.05	−0.01±0.04	−0.02±0.06	0.90	0.93

Data are presented as mean±SD or %, unless otherwise stated. Bold type represents statistical significance. A total of n=371 fully answered the ESS and n=395 had valid responses in the PVT. RT: response time
[#]: linear regression analysis between OSA severity groups (logistic regression for ESS categories) adjusted for age, sex and body mass index; [¶]: transformed using $\sqrt{x}+\sqrt{(x+1)}$.

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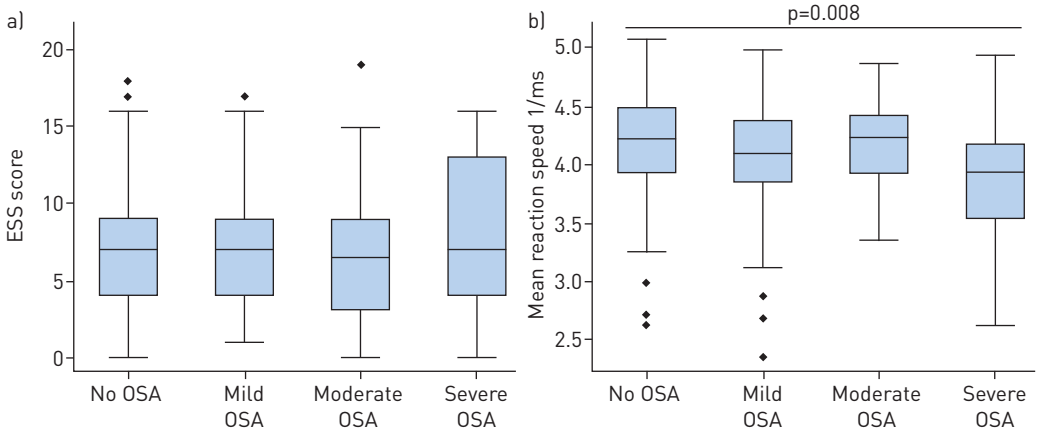

FIGURE 3 a) Subjective and b) objective sleepiness by severity of obstructive sleep apnoea (OSA), showing a) Epworth Sleepiness Scale (ESS) score (p=0.74) and b) mean reaction speed in the psychomotor vigilance test (p=0.008), driven solely by the severe OSA group.

sufficient to identify OSA subjects with excessive daytime sleepiness and/or decreased vigilance and will include many in the general population with OSA who are non-sleepy and do not have vigilance issues.

The strengths of this study include the high participation rate in a general population sample, the detailed questionnaire and measurements performed. Our study used the AASM 2007 recommended criterion for hypopnea scoring, requiring a 4% oxygen desaturation. The agreement between scorers has been shown to be higher for this criterion than other hypopnea criteria [31], which also results in more events being scored [2, 31].

The limitations of this study include the cross-sectional analysis which does not allow us to make any inferences about the long-term effects of having untreated OSA in the general population with regard to cardiometabolic effects, sleepiness or performance [36]. Also, in this article we only assessed the relationship between OSA and diagnosed cardiometabolic diseases. Differences with regards to cardiometabolic risk factors may be found when, for example, blood samples or blood pressure are assessed [37]. However, this was beyond the scope of this article. Another possible limitation is the type 3 sleep study used, instead of polysomnography for the diagnosis of OSA. However, this is clinical practice in many countries [28] and is accepted in the ICSD-3 [14]. Type 3 studies have also been shown to be comparable to polysomnography for OSA diagnosis, at least in subjects without major comorbidities [38–40]. Finally, a systematic follow-up of those using PAP treatment after participation in this study has not been performed.

Conclusions

In a large general population sample of Icelanders aged 40–65 years, almost one subject out of five had OSA as defined by the AHI ⩾15 cut-off. However, as a group, those with an AHI between 15 and 30 did not report sleep-related symptoms, excessive daytime sleepiness or impaired objective vigilance to a larger extent than the non-apnoeic group. More reliable methods are urgently needed to differentiate between those who are healthy and subjects with sleep disordered breathing who should be offered treatment.

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