

## Original Contribution

### Increased Prevalence of Sleep-Disordered Breathing in Adults

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Sleep-disordered breathing is a common disorder with a range of harmful sequelae. Obesity is a strong causal factor for sleep-disordered breathing, and because of the ongoing obesity epidemic, previous estimates of sleep-disordered breathing prevalence require updating. We estimated the prevalence of sleep-disordered breathing in the United States for the periods of 1988–1994 and 2007–2010 using data from the Wisconsin Sleep Cohort Study, an ongoing community-based study that was established in 1988 with participants randomly selected from an employed population of Wisconsin adults. A total of 1,520 participants who were 30–70 years of age had baseline polysomnography studies to assess the presence of sleep-disordered breathing. Participants were invited for repeat studies at 4-year intervals. The prevalence of sleep-disordered breathing was modeled as a function of age, sex, and body mass index, and estimates were extrapolated to US body mass index distributions estimated using data from the National Health and Nutrition Examination Survey. The current prevalence estimates of moderate to severe sleep-disordered breathing (apnea-hypopnea index, measured as events/hour,  $\geq 15$ ) are 10% (95% confidence interval [CI]: 7, 12) among 30–49-year-old men; 17% (95% CI: 15, 21) among 50–70-year-old men; 3% (95% CI: 2, 4) among 30–49-year-old women; and 9% (95% CI: 7, 11) among 50–70 year-old women. These estimated prevalence rates represent substantial increases over the last 2 decades (relative increases of between 14% and 55% depending on the subgroup).

adult; middle age; obesity; sleep

Abbreviations: AHI, apnea-hypopnea index; BMI, body mass index; NHANES, National Health and Nutrition Examination Survey; SDB, sleep-disordered breathing.

The apnea and hypopnea events of sleep-disordered breathing (SDB) have substantial harmful health consequences. Immediate effects include intermittent hypoxia, fragmented sleep, and exaggerated fluctuations in heart rhythm, blood pressure, and intrathoracic pressure (1). In turn, these acute physiologic disruptions evolve into long-term sequelae, such as hypertension and cardiovascular morbidities (1–3), decrements in cognitive function (4, 5), mood and quality of life (6, 7), and premature death (8, 9).

In 1993, data collected over a 4-year period from the Wisconsin Sleep Cohort Study uncovered a high prevalence of SDB assessed using polysomnography in a working population-based sample of adults 30–60 years of age (10). The findings were corroborated by other US population-based studies (11), but these prevalence rates were estimated

more than a decade ago (12, 13). The most important modifiable causes of SDB in adult populations are overweight and obesity. Weight gain and loss have been consistently associated with increasing and decreasing SDB severity, respectively, in observational and intervention studies (14–16). Over the last few decades, the prevalence rates of overweight and obesity experienced epidemic trajectories in the United States (17–20), which is likely to have resulted in increased occurrence of obesity-related outcomes, including SDB.

In the United States, a systematic program for monitoring SDB prevalence over time does not exist. High-quality objective assessments of SDB are time-consuming, expensive, and burdensome to subjects, typically requiring overnight continuous monitoring of multiple physiologic processes,

including sleep/wake state and respiratory functioning. As a consequence, despite the high prevalence of and broad range of negative health outcomes from SDB, there is presently no national monitoring system for tracking SDB prevalence in a fashion analogous to the manner in which iterations of the US National Health and Nutrition Examination Survey (NHANES) are used to track the prevalence rates of health exposures and outcomes such as overweight and obesity, hypertension, and blood lead levels. However, if a robust SDB prevalence estimation model is developed that has as input parameters factors that are routinely tracked and accurately estimated, that model could allow for serial estimations of SDB prevalence.

In the present study, we developed models of SDB prevalence as a function of sex, age group, and weight status categories, the 3 most important factors in SDB prevalence in US populations (11). We applied the models to adult (30–70 years of age) SDB prevalence in the early 1990s and in a recent time period (2007–2010), framing a time interval that corresponded with a rapid expansion of the US obesity epidemic. This was performed by combining information from the Wisconsin Sleep Cohort Study, which since 1988 has performed more than 4,500 overnight in-laboratory SDB evaluations on 1,520 study participants, and the NHANES in an approach that increased our new estimates' generalizability and replaced previous, now-outdated prevalence estimates (10, 16). We did this in 3 steps: 1) using data from participants in the ongoing Wisconsin Sleep Cohort Study, we modeled sex, age, and body mass index (BMI) strata-specific prevalence estimates of SDB; 2) we used NHANES (21) data to estimate US population distributions of corresponding sex, age, and BMI strata for 2 periods, the early 1990s (representing the initiation of the Wisconsin Sleep Cohort) and the late 2000s; and 3) we applied the Wisconsin Sleep Cohort SDB prevalence estimates to the 2 periods. This process yielded estimates of US adult SDB prevalence in the early 1990s and late 2000s.

## MATERIALS AND METHODS

### Participants

Informed consent documents and study protocols for the Wisconsin Sleep Cohort Study, which have been described previously (22), were approved by the University of Wisconsin-Madison Health Sciences Institutional Review Board. All employees of 5 state agencies in south-central Wisconsin who were 30–60 years were mailed a survey containing questions about sleep habits, health, and demographic characteristics in 1988. Of the 6,947 state employees who received the survey, 5,091 (73%) completed and returned it. From these respondents, a sampling frame was constructed from which a stratified random sample of 2,884 persons (nonpregnant and without recent airway cancer, airway surgery, or decompensated cardiopulmonary disease) was selected for invitation to participate in overnight in-laboratory polysomnography (sleep) studies to be repeated at 4-year intervals as part of the Wisconsin Sleep Cohort. Data for this report were collected between 1988 and June 2011 from 1,520 eligible participants with at least 1 adequate

sleep study (53% of those invited for baseline studies). The primary reported reason for nonparticipation was the burden of sleeping overnight in a sleep laboratory.

Sleep studies were performed at the Clinical Research Unit of the University of Wisconsin Hospital and Clinics. The majority of participants had undergone 2 or more (up to 6) sleep studies, all of which were used for this analysis (416 participants underwent exactly 1 study, 172 underwent 2 studies, 298 underwent 3 studies, 325 underwent 4 studies, 245 underwent 5 studies, and 64 underwent 6 studies). Not all subjects had the maximum possible number of studies (6) because: 1) not all subjects had yet had the opportunity to be invited for multiple repeat studies (protocols are ongoing and repeat studies accrue at a rate of approximately 200 per year); 2) 83 subjects had studies that produced sleep data of insufficient quality (e.g., in-laboratory sleep time <4 hours); 3) 112 subjects have died since the study began; and 4) 28 subjects who participated in a baseline study refused future studies or were not locatable. The participation rate for ongoing follow-up studies, that is, the proportion of those invited for follow-up studies that agree to participate, is approximately 80%. For the present analysis, studies from 31 subjects older than 70 years of age were excluded because there were too few observations from subjects in that age range.

### Measurements

Sleep studies included polysomnography and other assessments, such as ascertainment of BMI, which was calculated as measured weight (in kilograms) divided by height (in meters) squared (23). Data on medical history, medication use, alcohol use, smoking habits, and self-reported sleep habits, problems, and daytime sleepiness were obtained using questionnaires. For this study, daytime sleepiness was assessed using the Epworth Sleepiness Scale (24, 25), a widely used, validated questionnaire that was added to the Wisconsin Sleep Cohort Study in 1993 and that asks subjects to rate their likelihood of falling asleep in a variety of common situations. Possible scores range from 0 (least sleepy) to 24 (sleepiest), with daytime sleepiness defined as a score of >10, a commonly used clinical definition of excessive sleepiness.

A polysomnography system (Grass Instruments, Quincy, Massachusetts) was used to assess sleep state and respiratory and cardiac parameters. Sleep state was determined by electroencephalography, electrooculography, and electromyography. Arterial oxyhemoglobin saturation, oral and nasal airflow, nasal air pressure, and thoracic cage and abdominal respiratory motion were used to detect SDB events. Oxyhemoglobin saturation was recorded using a pulse oximetry device (Ohmeda 3740, Englewood, Colorado). Thermocouples (ProTec, Hendersonville, Tennessee) was used to detect airflow. A pressure transducer (Validyne Engineering Corp., Northridge, California) measured air pressure at the nares. Respiratory inductance plethysmography (Respirtrace, Ambulatory Monitoring, Ardsley, New York) was used to record thoracic and abdominal excursions. Sleep state and respiratory event scoring were performed by trained sleep technicians. Each 30-second epoch of the polysomnographic records was scored for sleep stage using conventional

criteria (26) and for breathing events. Cessation of airflow lasting 10 seconds or longer defined an apnea event. A discernible reduction in the sum of thoracic plus abdomen respiratory inductance plethysmography amplitude associated with a 4% or more reduction in oxyhemoglobin saturation defined a hypopnea event. The average number of apnea plus hypopnea events per hour of sleep were used to determine the apnea-hypopnea index (AHI), our summary parameter of SDB. From 1988 to 2000, sleep studies were scored using a paper-based recording system; from 2000 on, studies were scored by sleep technicians using a computer-screen system. All statistical modeling of SDB prevalence adjusted for the pre- and post-2000 instrument changes; this effectively removed instrumentation-related influences on SDB assessments after 2000.

### Statistical analyses

**Modeling SDB Prevalence in the Wisconsin Sleep Cohort Study.** SAS software, version 9.2 (SAS Institute, Inc., Cary, North Carolina) and SUDAAN software, version 10.0.1 (Research Triangle Institute, Research Triangle Park, North Carolina) were used for analyses. To efficiently use all available data, up to 6 sleep studies per subject were used for prevalence estimation. To produce robust standard errors, generalized estimating equation (27) logistic models (SUDAAN logistic procedure) were used to regress the absence or presence of 4 distinct definitions of SDB on age, sex, and BMI. Because SDB prevalence is highly dependent on age, sex, and BMI, and to better accommodate application of SDB prevalence estimates from the Wisconsin Sleep Cohort to adult populations with varying age, sex, and BMI distributions, we chose the largest number of strata for which we could measure age  $\times$  sex  $\times$  BMI strata-specific prevalence estimates with acceptable precision. BMI was categorized into 4 strata (<25, 25–29, 30–39, and  $\geq$ 40) and age was categorized into 2 strata (30–49 and 50–70 years). For the regression modeling, BMI strata were assigned the strata mean sample values (23.0, 27.6, 33.9, and 45.4, respectively). Age, sex, BMI, and BMI squared, as well as pairwise and higher-order interactions among these factors, were tested for statistical significance (2-sided  $P < 0.05$ ); significant terms were retained in the final models. Supplementary models included alcohol use, cigarette smoking, and race/ethnicity as covariates. However, inclusion of those factors did not substantially affect estimates of SDB prevalence rates and were not included in final models.

Four separate regression models were fitted as described above, 1 for each of the 4 SDB definitions, all of which are commonly used in clinical settings or in epidemiologic studies. The 4 SDB outcome definitions are: 1) mild to severe SDB ( $AHI \geq 5$ ); 2) moderate to severe SDB ( $AHI \geq 15$ ); 3) mild to severe SDB with daytime sleepiness ( $AHI \geq 5$  and Epworth Sleepiness Scale score  $>10$ ); and 4) moderate to severe SDB with daytime sleepiness ( $AHI \geq 15$  and Epworth Sleepiness Scale score  $>10$ ). Subjects who were diagnosed with and being treated for SDB at the time of the sleep studies were classified as having an  $AHI >15$ . Note that each of the 4 separately modeled SDB definitions is binary, partitioning all subjects in the sample into 1 of 2

groups. Individual subjects could meet none, some, or all of the 4 definitions.

**Extrapolation of Wisconsin Sleep Cohort Study estimates to the US population.** We estimated the prevalence of people in the United States in each BMI stratum (<25, 25–29, 30–39, and  $\geq$ 40) from the entire NHANES III (28) data set (1988–1994) and from combined data from NHANES 2007–2008 (29) and 2009–2010 (30). We used sampling weights provided by NHANES and included only subjects who were 30–70 years of age and not pregnant. We then estimated US prevalence of SDB in age- and sex-specific strata by applying Wisconsin Sleep Cohort Study–estimated age, sex, and BMI strata–specific SDB prevalence rates to NHANES–estimated age and sex strata–specific BMI distributions. This produced US estimates of SDB prevalence rates in each age and sex stratum.

**Confidence interval estimation.** A bootstrap procedure was used to generate 95% confidence intervals for the prevalence estimates: 999 samples of subject identifiers (for Wisconsin Sleep Cohort subjects) or NHANES clusters (using NHANES strata and population sampling units reweighted using the Rao-Wu rescaling bootstrap method (31)) were selected using the SAS Surveyselect procedure, version 9.2. For each of the 999 samples, stratum-specific prevalence estimates were calculated as described above. The 2.5th and 97.5th percentiles for each estimate were used to construct confidence intervals. These confidence intervals reflected the variability in both the strata-specific estimates of SDB prevalence from the Wisconsin Sleep Cohort and in the estimates of the proportions of the US population represented by each age, sex, and BMI stratum for both examined time periods. The confidence intervals also accounted for the varying number of observations (sleep studies) per subject by bootstrap sampling of subjects rather than individual sleep studies.

## RESULTS

Key descriptive statistics for 1,520 study participants (96% non-Hispanic white) who were assessed for SDB at a total of 4,563 sleep studies performed between 1988 and 2011 are presented in Table 1. Women made up 45% of the sample. Most sleep studies (62%) were undertaken by older (50–70 years of age) participants, which reflects the fact that many follow-up sleep studies were performed after a majority of the baseline cohort (ages 30–60 years in 1988) aged into the 50–70-year-old range. A large minority (47%) of sleep studies were provided by obese (BMI  $\geq 30$ ) participants. One third of the sleep studies identified mild to severe SDB ( $AHI \geq 5$ ).

Modeled estimates of the prevalence of mild to severe SDB ( $AHI \geq 5$ ) and of moderate to severe SDB ( $AHI \geq 15$ ) by sex, age, and BMI strata are presented in Tables 2 and 3, respectively. Analogous prevalence estimates for SDB co-occurring with daytime sleepiness (Epworth Sleepiness Scale score  $>10$ ) are presented in Table 4 (mild to severe SDB with sleepiness) and Table 5 (moderate to severe SDB with sleepiness). The prevalence of SDB was substantially higher in men, older subjects, and subjects with higher BMIs.

**Table 1.** Descriptive Characteristics of Participants, Wisconsin Sleep Cohort Study, Wisconsin, 1988–2011

Characteristic	% <sup>a</sup>
Sex	
Male	55.1
Female	44.9
Age, years	
30–49	37.7
50–70	62.3
Body mass index <sup>b</sup>	
<25	19.5
25–29.9	33.5
30–39.9	36.2
≥40	10.8
Apnea-hypopnea index <sup>c</sup>	
<5	67.3
5–14	21.4
≥15	11.3
≥5 and ESS score <sup>d</sup> >10	14.5

Abbreviation: ESS, Epworth Sleepiness Scale.

<sup>a</sup> For sex, the value is the percentage of participants ( $n=1,520$ ); for age, body mass index, and apnea-hypopnea index, the value is the percentage of polysomnography studies ( $n=4,563$ ; individual participants contributed multiple observations, potentially ranging across age, body mass index, and sleep-disordered breathing categories).

<sup>b</sup> Weight (kg)/height (m)<sup>2</sup>.

<sup>c</sup> Events/hour.

<sup>d</sup> The ESS was added to Wisconsin Sleep Cohort Study protocols in 1993 and was available from 3,623 polysomnography studies by 1,291 participants.

Two interactions on the logistic scale involving age (sex  $\times$  age and BMI  $\times$  age) were statistically significant ( $P < 0.05$ ) in prevalence models and are reflected in the prevalence estimates shown in Tables 2–5. For all definitions of SDB, there was a stronger relationship between age and SDB in women than in men. For example, as shown in Table 2, among overweight men (BMI 25–29.9), the prevalence of mild to severe SDB was approximately 2-fold higher in older men than in younger men (37% vs. 18%, respectively); among overweight women, the prevalence was approximately 5-fold higher in older women than in younger women (20% vs. 4%, respectively). Also, for all definitions of SDB (Tables 2–5), BMI was more strongly related to SDB prevalence in the younger age stratum.

Table 6 presents age- and sex-specific SDB prevalence estimates that were determined by extrapolating Wisconsin Sleep Cohort Study age-, sex-, and BMI-specific SDB prevalence estimates to US age, sex, and BMI distributions estimated from 1988–1994 NHANES data and recent (2007–2010) NHANES estimates. All age and sex strata were estimated to have increased prevalence rates of SDB, with younger age categories of men and women experiencing the largest relative prevalence increases.

**Table 2.** Model-based<sup>a</sup> Prevalence Estimates of Mild to Severe Sleep-Disordered Breathing, Wisconsin Sleep Cohort Study, Wisconsin, 1988–2011

Body Mass Index <sup>b</sup> by Age, years	Estimated Prevalence of AHI <sup>c</sup> ≥5	
	% <sup>d</sup>	95% CI
<i>Men</i>		
30–49		
<25	7.0	5.0, 9.3
25–29.9	18.3	15.2, 21.6
30–39.9	44.6	48.7, 50.2
≥40	79.5	71.1, 86.2
50–70		
<25	18.9	14.8, 23.8
25–29.9	36.6	32.8, 40.3
30–39.9	61.4	57.0, 65.5
≥40	82.8	77.1, 87.7
<i>Women</i>		
30–49		
<25	1.44	0.82, 2.23
25–29.9	4.2	2.7, 5.8
30–39.9	13.5	9.8, 17.7
≥40	43.0	33.0, 54.2
50–70		
<25	9.3	6.8, 12.3
25–29.9	20.2	16.4, 24.4
30–39.9	41.1	35.6, 46.7
≥40	67.9	60.6, 75.1

Abbreviations: AHI, apnea-hypopnea index; CI, confidence interval; SE, standard error.

<sup>a</sup> Estimated logistic regression model coefficients: intercept = -10.3 (SE, 1.3);  $\beta_{\text{Sex}} = -1.6$  (SE, 0.2; female = 1, male = 0);  $\beta_{\text{Age}} = 2.1$  (SE, 0.5; 30–49 years of age = 0, 50–70 years of age = 1);  $\beta_{\text{BMI}} = 0.41$  (SE, 0.07; <25 = 23.0, 25–29.9 = 27.6, 30–39.9 = 33.9, ≥40 = 45.4);  $\beta_{\text{BMI}^2} = -0.003$  (SE, 0.001);  $\beta_{\text{Age} \times \text{BMI}} = -0.041$  (SE, 0.015); and  $\beta_{\text{Age} \times \text{Sex}} = 0.82$  (SE, 0.22).

<sup>b</sup> Weight (kg)/height (m)<sup>2</sup>.

<sup>c</sup> Events/hour.

<sup>d</sup> Among 1,520 participants who contributed a total of 4,563 sleep studies.

Summing over age  $\times$  sex  $\times$  BMI strata, we estimated the overall prevalence of mild to severe SDB (AHI  $\geq 5$ ) to be 26% (95% confidence interval: 24, 28) among person 30–70 years of age for the recent time period (2007–2010). Similarly, the estimated overall prevalence of moderate to worse SDB (AHI  $\geq 15$ ) was 10% (95% confidence interval: 8, 11).

## DISCUSSION

The ongoing obesity epidemic in the United States is likely to result in “offspring epidemics” of obesity-related conditions. Overweight and obesity are strong causal factors for SDB, and in tandem with an escalation in the prevalence of obesity in the United States, the prevalence of SDB

**Table 3.** Model-based<sup>a</sup> Prevalence Estimates of Moderate to Severe Sleep-Disordered Breathing, Wisconsin Sleep Cohort Study, Wisconsin, 1988–2011

Body Mass Index <sup>b</sup> by Age, years	Estimated Prevalence of AHI <sup>c</sup> ≥15	
	% <sup>d</sup>	95% CI
<i>Men</i>		
30–49		
<25	0.93	0.49, 1.59
25–29.9	3.8	2.5, 5.3
30–39.9	16.6	12.5, 21.7
≥40	55.0	41.8, 69.2
50–70		
<25	3.6	2.0, 5.4
25–29.9	10.6	8.2, 13.0
30–39.9	29.0	24.6, 33.9
≥40	56.0	46.3, 65.3
<i>Women</i>		
30–49		
<25	0.18	0.07, 0.34
25–29.9	0.73	0.34, 1.23
30–39.9	3.6	2.0, 5.7
≥40	18.6	11.7, 27.0
50–70		
<25	1.44	0.74, 2.52
25–29.9	4.5	3.0, 6.3
30–39.9	13.9	10.4, 17.4
≥40	33.5	25.7, 40.8

Abbreviations: AHI, apnea-hypopnea index; CI, confidence interval; SE, standard error.

<sup>a</sup> Estimated logistic regression model coefficients: intercept = -15.2 (SE, 1.8);  $\beta_{\text{Sex}} = -1.7$  (SE, 0.3; female = 1, male = 0);  $\beta_{\text{Age}} = 2.7$  (SE, 0.7; 0–49 years of age = 0, 50–70 years of age = 1);  $\beta_{\text{BMI}} = 0.58$  (SE, 0.10; <25 = 23.0, 25–29.9 = 27.6, 30–39.9 = 33.9, ≥40 = 45.4);  $\beta_{\text{BMI}^2} = -0.005$  (SE, 0.001);  $\beta_{\text{Age} \times \text{BMI}} = -0.059$  (SE, 0.021); and  $\beta_{\text{Age} \times \text{Sex}} = 0.75$  (SE, 0.33).

<sup>b</sup> Weight (kg)/height (m)<sup>2</sup>.

<sup>c</sup> Events/hour.

<sup>d</sup> Among 1,520 participants who contributed a total of 4,563 sleep studies.

among adults has increased substantially. We estimate that currently, among adults 30–70 years of age, approximately 13% of men and 6% of women have moderate to severe SDB (AHI ≥15). We also estimate that 14% of men and 5% of women have an AHI ≥5 plus symptoms of daytime sleepiness. Either of these SDB definitions fit the Medicare criteria for a positive indication of obstructive sleep apnea (32).

These prevalence estimates represent double-digit relative percentage increases, ranging from approximately 14% up to 55% depending on age group, sex, and SDB severity level, as evidenced in Table 6 by comparing data from 1988–1994 with that from 2007–2010. The estimated increases translate to millions of additional afflicted persons

**Table 4.** Model-based<sup>a</sup> Prevalence Estimates of Mild to Severe Sleep-Disordered Breathing With Concomitant Daytime Sleepiness Wisconsin Sleep Cohort Study, Wisconsin, 1993–2011

Body Mass Index <sup>b</sup> by Age, years	Estimated Prevalence of AHI <sup>c</sup> ≥5 and ESS Score >10	
	% <sup>d</sup>	95% CI
<i>Men</i>		
30–49		
<25	2.7	1.5, 4.2
25–29.9	6.8	4.7, 9.1
30–39.9	18.9	14.3, 24.0
≥40	52.9	39.3, 66.6
50–70		
<25	7.7	4.9, 10.9
25–29.9	13.8	11.0, 16.1
30–39.9	24.9	20.7, 29.4
≥40	43.0	33.8, 53.9
<i>Women</i>		
30–49		
<25	0.49	0.19, 0.91
25–29.9	1.26	0.58, 2.20
30–39.9	3.9	2.1, 6.2
≥40	16.4	10.3, 23.8
50–70		
<25	2.8	1.6, 4.4
25–29.9	5.3	3.8, 7.1
30–39.9	10.5	7.8, 13.2
≥40	20.9	15.4, 27.8

Abbreviations: AHI, apnea-hypopnea index; CI, confidence interval; ESS, Epworth Sleepiness Scale; SE, standard error.

<sup>a</sup> Estimated logistic regression model coefficients: intercept = -9.9 (SE, 1.6);  $\beta_{\text{Sex}} = -1.7$  (SE, 0.3; female = 1, male = 0);  $\beta_{\text{Age}} = 2.6$  (SE, 0.7; 0–49 years of age = 0, 50–70 years of age = 1);  $\beta_{\text{BMI}} = 0.33$  (SE, 0.09; <25 = 23.0, 25–29.9 = 27.6, 30–39.9 = 33.9, ≥40 = 45.4);  $\beta_{\text{BMI}^2} = -0.002$  (SE, 0.001);  $\beta_{\text{Age} \times \text{BMI}} = -0.07$  (SE, 0.02); and  $\beta_{\text{Age} \times \text{Sex}} = 0.70$  (SE, 0.33).

<sup>b</sup> Weight (kg)/height (m)<sup>2</sup>.

<sup>c</sup> Events/hour.

<sup>d</sup> Among 1,291 participants who contributed a total of 3,623 sleep studies.

in the United States, and, consequently, incidences of sequelae of SDB, such as cardiovascular morbidities and stroke (1, 33, 34), cognitive decline (4), depression (7), and premature death (8, 9), are likely accelerated relative to levels that would otherwise be expected.

Multiple mechanisms likely explain the associations of excess body mass and SDB, including increased upper airway collapsibility and impaired neuromuscular control of upper airway patency due to local fat deposition (35). Excess fat deposited outside of the upper airway may also contribute to breathing events via several mechanisms, including reduced lung volume, greater whole-body metabolic demand, and increased effort of breathing (24, 36).

**Table 5.** Model-based<sup>a</sup> Prevalence Estimates of Moderate to Severe Sleep-Disordered Breathing With Concomitant Daytime Sleepiness, Wisconsin Sleep Cohort Study, Wisconsin, 1988–2011

Body Mass Index <sup>b</sup> by Age, years	Estimated Prevalence of AHI <sup>c</sup> ≥15 and ESS Score >10	
	% <sup>d</sup>	95% CI
Men		
30–49		
<25	0.39	0.10, 0.87
25–29.9	1.6	0.74, 2.66
30–39.9	7.7	4.7, 11.3
≥40	35.9	20.2, 56.6
50–70		
<25	1.2	0.44, 2.5
25–29.9	3.7	2.3, 5.1
30–39.9	11.5	8.4, 14.8
≥40	28.7	19.7, 38.1
Women		
30–49		
<25	0.045	0.008, 0.140
25–29.9	0.19	0.048, 0.44
30–39.9	0.95	0.36, 1.9
≥40	6.0	2.8, 10.6
50–70		
<25	0.48	0.15, 1.09
25–29.9	1.46	0.77, 2.33
30–39.9	4.7	3.1, 6.6
≥40	13.3	9.1, 18.6

Abbreviations: AHI, apnea-hypopnea index; CI, confidence interval; ESS, Epworth Sleepiness Scale; SE, standard error.

<sup>a</sup> Estimated logistic regression model coefficients: intercept = -15.9 (SE, 3.1);  $\beta_{\text{Sex}} = -2.2$  (SE, 0.5; female = 1, male = 0);  $\beta_{\text{Age}} = 2.7$  (SE, 1.1; 0–49 years of age = 0, 50–70 years of age = 1);  $\beta_{\text{BMI}} = 0.57$  (SE, 0.17; <25 = 23.0, 25–29.9 = 27.6, 30–39.9 = 33.9, ≥40 = 45.4);  $\beta_{\text{BMI}^2} = -0.005$  (SE, 0.002);  $\beta_{\text{Age} \times \text{BMI}} = -0.07$  (SE, 0.03);  $\beta_{\text{Age} \times \text{Sex}} = 1.2$  (SE, 0.5).

<sup>b</sup> Weight (kg)/height (m)<sup>2</sup>.

<sup>c</sup> Events/hour.

<sup>d</sup> Among 1,520 participants who contributed a total of 4,563 sleep studies.

Studies have demonstrated that weight loss has the potential to reduce and sometimes eliminate SDB in overweight patients (11). However, intentional weight loss with long-term weight maintenance is typically not sustained (37), and overweight persons with SDB in whom sufficient weight loss cannot be attained may require direct treatments of SDB (e.g., positive airway pressure therapy) to mitigate SDB-related outcomes. Unfortunately, like weight loss, high rates of long-term adherence to positive pressure therapy have been difficult to achieve (38).

The present analysis necessarily updates and substantially extends earlier Wisconsin Sleep Cohort Study estimates of

the prevalence of SDB (10, 16). Important advances in prevalence estimation include the fact that thousands more observations were used to model prevalence; prevalence estimates presented in Table 6 were estimated using US distributions of BMI strata and are more generalizable than are those calculated directly from the Wisconsin Sleep Cohort Study population; and the age distribution for which estimates were calculated was broader in the current analysis (ages 30–60 years in the 1993 report vs. ages 30–70 years in the present report). Modeled prevalence estimates for the 1988–1994 period are highly consistent with the directly calculated (not modeled) previous estimates, providing a validation of our present models. For example, the 1993 report found that 9% of men and 4% of women 30–60 years of age had an AHI ≥15. Here, we estimate that in the 1988–1994 period, 6% of men 30–49 years of age and 14% of men 50–70 years of age had an AHI ≥15; the analogous 1988–1994 estimates for women were 2% (30–49 years) and 7% (50–70 years). However, the prevalence estimates for SDB co-occurring with sleepiness presented here are not comparable with those estimated in 1993 because of an alternative definition of “daytime sleepiness” used in the present study. To be classified as having “daytime hypersomnolence” in the 1993 study, subjects had to report “frequent” or “habitual” feelings of excessive sleepiness in addition to waking up unrefreshed regardless of sleep quantity, as well as having uncontrollable daytime sleepiness that caused daytime impairment. In the present study, we have updated the occurrence of daytime “sleepiness” to be defined using the Epworth Sleepiness Scale (24), an instrument in wide use in both clinical and research settings.

Our findings benefited from important methodological strengths, including the use of a large nonclinical sample and laboratory-based polysomnographic SDB assessment. Nevertheless, there are study limitations that, in aggregate, may result in underestimation of SDB prevalence. The baseline population was recruited out of a working sample, and it is possible that persons with very severe SDB might have been less likely to be present in the sampling frame. However, we included data from follow-up sleep studies regardless of subjects’ subsequent employment status, and thus our estimates include information from postemployment observations. Also, although we did not find that race/ethnicity affected our SDB prevalence estimates, our sample lacked racial/ethnic diversity (96% non-Hispanic white). Some studies have included large samples of multiple racial/ethnic groups and have found that prevalence rates of SDB vary among white, black, Asian, or Hispanic subsamples (39–41). However, prevalence variations may have reflected systematic differences in BMI distributions observed among the race/ethnic groups (40, 41); if so, our method of extrapolating BMI category-specific prevalence rates from the Wisconsin Sleep Cohort Study to the more representative NHANES-estimated US BMI distributions may reduce the impact of this limitation. Additionally, BMI is an imperfect proxy for underlying parameters (e.g., fat deposition in the upper airway) that may be more germane to the development of SDB; thus, the relation of weight and SDB may be underestimated. However, the use of BMI was necessary because US population estimates of BMI distributions are available,

**Table 6.** Sleep-Disordered Breathing Prevalence Estimates for 4 Definitions of Sleep-Disordered Breathing in US Adults Estimated Using US Body Mass Index Population Distributions, Wisconsin Sleep Cohort Study, Wisconsin

Sleep-Disordered Breathing Definition by Age, years	1988–1994, Estimated Prevalence by Sex and Age Strata		2007–2010, Estimated Prevalence by Sex and Age Strata	
	%	95% CI	%	95% CI
<i>Men</i>				
AHI <sup>a</sup> ≥5				
30–49	20.0	17.2, 23.1	26.6	22.8, 30.5
50–70	38.5	34.9, 42.4	43.2	39.4, 47.4
30–70	26.4	23.9, 28.9	33.9	30.8, 37.0
AHI ≥15				
30–49	6.2	4.4, 8.1	9.5	7.0, 12.1
50–70	13.9	11.5, 16.8	17.4	14.5, 20.6
30–70	8.8	7.3, 10.5	13.0	10.8, 15.2
AHI ≥5, ESS score >10				
30–49	8.5	6.3, 10.8	11.7	9.0, 14.7
50–70	15.3	12.6, 17.8	17.6	14.7, 20.3
30–70	10.8	9.0, 12.6	14.3	12.0, 16.4
AHI ≥15, ESS score >10				
30–49	3.1	1.8, 4.4	4.8	3.1, 6.9
50–70	5.4	4.0, 6.8	7.0	5.3, 8.9
30–70	3.8	2.9, 4.9	5.8	4.5, 7.2
<i>Women</i>				
AHI ≥5				
30–49	6.6	4.9, 8.6	8.7	6.5, 11.3
50–70	24.4	20.8, 28.2	27.8	24.0, 31.6
30–70	13.2	11.4, 15.3	17.4	15.2, 20.0
AHI ≥15				
30–49	1.9	1.2, 3.0	2.7	1.7, 4.0
50–70	7.4	5.5, 9.5	9.1	6.8, 11.4
30–70	3.9	3.1, 5.0	5.6	4.4, 7.0
AHI ≥5, ESS score >10				
30–49	2.1	1.2, 3.3	2.9	1.7, 4.3
50–70	6.6	5.1, 8.6	7.5	5.9, 9.7
30–70	3.8	2.9, 4.9	5.0	3.9, 6.3
AHI ≥15, ESS score >10				
30–49	0.55	0.24, 0.99	0.79	0.35, 1.34
50–70	2.6	1.8, 3.6	3.2	2.3, 4.4
30–70	1.3	0.9, 1.8	1.9	1.4, 2.6

Abbreviations: AHI, apnea-hypopnea index; CI, confidence interval; ESS, Epworth Sleepiness Scale.

<sup>a</sup> Events/hour.

whereas estimates for more pathophysiologically proximal parameters are not. Finally, it is possible that subjects that provided more data points (i.e., participated in a larger number of follow-up studies) had systematically lower or higher AHIs. To assess this possibility, we compared baseline mean AHIs for the 1,104 subjects who provided multiple observations with those from the 416 subjects who provided only a single (baseline) observation. Conditional

on the stratification factors (age, sex, and BMI), the baseline AHIs were not significantly different ( $P > 0.2$ ) in subjects who only provided a single observation compared with those who provided multiple observations; similarly, mean AHI for the  $n$ th sleep study was not significantly different when comparing subjects who only had  $n$  sleep studies with subjects who provided more than  $n$  sleep studies ( $n = 2–5$ ; all  $P > 0.4$ ). Thus, we found no evidence that SDB severity

was associated with the number of observations that subjects provided.

In the present study, we provided up-to-date estimates of US SDB prevalence by age, sex, and BMI strata, and our findings suggest that prevalence rates in middle-aged adults have risen substantially in recent decades. In addition to current SDB prevalence, our results and approach can be used to estimate future SDB prevalence if there are further alterations in population distributions of overweight and obesity. This information is essential for the clinical and public health sectors because of the growing high population burden of SDB, the treatability of SDB, and the myriad negative health consequences of untreated SDB.

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