Exercise and Sleep-Disordered Breathing: an Association Independent of Body Habitus

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Study Objectives: The degree to which physical exercise habits are related to sleep-disordered breathing is not known. We sought to investigate the association between a single-item exercise question and laboratory-assessed sleep-disordered breathing.

Design: A population-based cross-sectional epidemiologic study of adults measured the association between exercise and sleep-disordered breathing. Hours of weekly planned exercise were assessed by questionnaire. Sleep-disordered breathing was assessed by 18-channel in-laboratory polysomnography and characterized by the apnea-hypopnea index.

Setting: Polysomnography was conducted at the University of Wisconsin General Clinical Research Center sleep laboratory.

Patients and Participants: Participants included 1104 men and women, aged 30 to 60 years, enrolled in the Wisconsin Sleep Cohort Study.

Measurements and Results: Associations were modeled using linear and logistic regression, adjusting for body mass index, skinfold measurements, age, sex, and other covariates. Adjusted mean (95% confidence interval) apnea-hypopnea index was 5.3 (4.4, 6.2) events per hour for participants who exercised 0 hours per week; 3.9 (2.8, 5.0) events per hour for those with 1 to 2 hours of exercise; 3.2 (2.2, 4.2) events per hour for those with 3 to 6 hours of exercise; and 2.8 (1.0, 4.6) events per hour for those with > 7 hours of exercise (P trend < .001). Similarly, the odds of having moderate or worse sleep-disordered breathing (apnea-hypopnea index > 15 events per hour) significantly decreased with increasing level of exercise.

Conclusion: Independent of measures of body habitus, lack of exercise was associated with increased severity of sleep-disordered breathing.

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Data Collection

Sleep Cohort participants complete a baseline overnight protocol that includes nocturnal polysomnography and other tests. Every 4 years thereafter, baseline participants are invited for follow-up studies. Only baseline studies were used for this report.

Overnight protocols are conducted at the University of Wisconsin General Clinical Research Center. Participants arrive for overnight studies in the early evening. Sleep technicians obtain written informed consent. Information on medical history and current medication use, smoking, alcohol use, education, age, and other sociodemographic factors are obtained by interview and questionnaire. Blood pressure and body habitus measurements—including height and weight; waist, neck, and hip girths; and biceps, triceps, subscapular, and suprailiac skinfold thicknesses—are performed. Body mass index (BMI) is calculated from height and weight (kg/m²).

Following body habitus assessment, technicians affix polysomnography leads to participants and perform calibrations. An 18-channel polysomnography recording system (Polygraph model 78, Grass Instruments, Quincy, Mass) is used to assess sleep-state, respiratory, and cardiac parameters. Sleep state is determined by electroencephalography, electrocochleography, and chin electromyography. These signals are used to score sleep stage for each 30-second epoch of the polysomnographic record, using conventional criteria.49 Arterial oxyhemoglobin saturation, oral and nasal airflow, nasal air pressure, and thoracic cage and abdominal respiratory motion are used to assess SDB events. Oxyhemoglobin saturation is continuously recorded using pulse oximetry (Ohmeda 3740, Englewood, Colo). Stalk-mounted thermocouples (ProTec, Hendersonville, Tenn) detect oral and nasal airflow. A pressure transducer (Validyne Engineering Corp., Northridge, Calif) continuously measures air pressure at the nares via nasal prongs. Respiratory induc- tion plethysmography (Respiritrace, Ambulatory Monitoring, Airdsley, NY) continuously records thoracic cage and abdominal excursions. Sleep-state and respiratory-event scoring are performed by trained sleep technicians and reviewed by an expert polysomnographer. Each 30-second epoch of the polysomnographic records is visually inspected and scored for abnormal breathing events. Cessation of airflow lasting 10 or more seconds is defined as an apnea event. A discernable reduction in the sum of rib cage plus abdomen respiratory inductance plethysmography amplitude associated with a 4% or greater reduction in oxyhemoglobin saturation is defined as a hypopnea event. The average number of apnea events plus hypopnea events per hour of objectively measured sleep defines the apnea-hypopnea index (AHI), the summary parameter of SDB.

Exercise information was obtained by a single question on the initial mailed surveys that were filled out by all Sleep Cohort invitees. The question was: “About how many hours per week— if any— do you spend at regular planned exercise (such as jogging, sports, exercise class, workouts at home or a gym)?” Also collected on the mailed surveys were several questions concerning daytime sleepiness and typical hours of sleep on weekdays and weekends and naps. Two particular questions—one assessing the frequency of feeling unrested during the daytime regardless of hours of sleep (“unrefreshing sleep”) and a second assessing the frequency of feelings of excessive daytime sleepiness—were used to assess the possibility that SDB, by contributing to daytime sleepiness, might be a factor in reduced habitual exercise.

Data Analysis

Descriptive and regression analyses were performed with SAS software, release 8.02 (SAS Institute, Inc., Cary, NC). Since the exercise data were collected by mailed survey and other data used for this report were collected at subsequent overnight sleep studies, there was a time lag between assessment of exercise and all other data. For the majority of participants, the exercise data were collected 2 or fewer years prior to the overnight sleep studies. Supplemental analyses examining the importance of length of time (eg, by excluding participants with large time lags) between exercise and SDB assessment failed to show any significant effect on our findings.

Regression models were used to measure the association between exercise and SDB while controlling for potential confounding variables and examining possible interactions. Multiple linear regression models were used to assess the association of hours of weekly exercise (the primary predictor variable, modeled categorically and continuously) and the AHI (a continuous outcome variable). Models of untransformed AHI and log(AHI+1) were examined. Age, sex, smoking habits (never/ever/current-use status and cigarette packs per week) and alcohol use (usual weekly consumption and amount consumed 24 hours prior to sleep study), education level, menopausal status, unrefreshing sleep, excessive daytime sleepiness, and use of antihypertensive medications were investigated as interacting and confounding factors. Multiple measures of body habitus including BMI; weight; height; skinfold measurements; neck, hip, and waist girths; and waist to hip girth ratio were examined as potential mediating, interacting, or confounding variables. Covariates that substantially altered the regression coefficient for the exercise variables were retained in final models. Interactions between the covariates and exercise were tested for statistical significance. The statistical significance (2-tailed P value < .05 for main effects, < .01 for interactions) of regression coefficients was assessed by t tests.

Final linear regression models presented here used untransformed AHI as the outcome variable and categories of weekly exercise (none, 1 to 2 hours, 3 to 6 hours, and 7 or more hours) as the predictor variable. Untransformed AHI was used so that least-squares mean AHI levels could be examined by category of weekly exercise. However, since the AHI is a skewed variable in the sample, we examined each participant’s influence on final model parameter estimates. No participant was found to be unduly influential. In addition, because the AHI distribution is highly skewed, the sampling distribution of mean AHI may not be approximately normally distributed. To investigate this, Monte Carlo simulations of the sampling distribution of the mean of the AHI were conducted and indicated that 95% confidence intervals calculated under asymptotic normality assumptions from samples of 300 or more would be expected to symmetrically cover the mean of the AHI (or a β-coefficient in a linear regression model where AHI is the outcome variable) approximately 95% of the time.

SDB was also modeled as a binary outcome with logistic regression models. Two sets of models were examined: (1) exercise as a predictor of the odds of having an AHI < 5 versus an AHI ≥ 5 events per hour (ie, “mild or worse” SDB) and (2) exercise as a predictor of the odds of having an AHI < 15 versus an AHI ≥ 15 events per hr (ie, “moderate” or worse SDB). Confounding, interaction, and the role of body habitus were assessed as described above for linear regression analyses. Both linear and logistic regression models were weighted to reflect the stratified random sampling scheme used to select Sleep Cohort invitees from the sampling frame of mailed sleep survey respondents.

RESULTS

Table 1 provides summary statistics for all of the study participants and also broken down by categories of self-reported hours of weekly exercise. Most of the sample participated in 2 or fewer hours of exercise weekly. Men tended to be most represented at the extremes of the exercise distribution (ie, in the 0 or 7+ hours per week categories). Among persons exercising 1 to 2 hours, women were more prevalent than men.
Table 1—Summary of Key Variables by Categories of Exercise and for All Participants*

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>0</th>
<th>1 to 2</th>
<th>3 to 6</th>
<th>7 or more</th>
<th>All</th>
</tr>
</thead>
<tbody>
<tr>
<td>N (percentage of entire sample), %</td>
<td>410 (37)</td>
<td>271 (25)</td>
<td>331 (30)</td>
<td>92 (8)</td>
<td>1104</td>
</tr>
<tr>
<td>Men, %</td>
<td>57</td>
<td>43</td>
<td>52</td>
<td>58</td>
<td>54</td>
</tr>
<tr>
<td>Age, y</td>
<td>47 (8)</td>
<td>47 (8)</td>
<td>48 (8)</td>
<td>46 (8)</td>
<td>47 (8)</td>
</tr>
<tr>
<td>AHI, events/h</td>
<td>6 (12)</td>
<td>3 (8)</td>
<td>3 (6)</td>
<td>2 (4)</td>
<td>4 (9)</td>
</tr>
<tr>
<td>≤ AHI ≤ 5, %</td>
<td>75</td>
<td>84</td>
<td>84</td>
<td>90</td>
<td>82</td>
</tr>
<tr>
<td>5 ≤ AHI ≤ 15, %</td>
<td>15</td>
<td>10</td>
<td>12</td>
<td>7</td>
<td>12</td>
</tr>
<tr>
<td>AHI ≥ 15, %</td>
<td>10</td>
<td>5</td>
<td>4</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>Typical sleep time (h/day)</td>
<td>7.6 (1.0)</td>
<td>7.6 (0.9)</td>
<td>7.4 (0.9)</td>
<td>7.3 (0.9)</td>
<td>7.5 (0.9)</td>
</tr>
</tbody>
</table>

*Statistics are weighted to reflect the stratified sampling design. Data are presented as mean (SD) unless otherwise indicated.

Table 2—Mean AHI by Levels of Weekly Exercise*  

<table>
<thead>
<tr>
<th>Exercise, h/wk</th>
<th>Adjusted† AHI</th>
<th>Habitus-adjusted‡ AHI</th>
<th>P value for trend</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>5.5 (4.6, 6.4)</td>
<td>5.3 (4.4, 6.2)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>1-2</td>
<td>3.4 (2.3, 4.5)</td>
<td>3.9 (2.8, 5.0)</td>
<td>&lt;.005</td>
</tr>
<tr>
<td>3-6</td>
<td>2.6 (1.8, 3.8)</td>
<td>3.2 (2.2, 4.2)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>≥ 7</td>
<td>2.0 (0.1, 3.9)</td>
<td>2.8 (1.0, 4.6)</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

*Data are presented as mean (confidence intervals). AHI refers to apnoea-hypopnoea index.

DISCUSSION

We observed an association between increased exercise and reduced degree of SDB. This moderate association was independent of age, sex, and other covariates and even persisted when accounting for measures of body habitus. Since overweight and obesity are strongly associated with SDB, and since exercise is linked to healthier body habitus profiles, we expected that, after controlling for measures of body habitus, some or all of the association between exercise and SDB might disappear. Associations did slightly diminish with control for body habitus, but, nonetheless, a moderate association remained. As a frame of reference for the magnitude of the associations, consider, for example, the habitus-adjusted odds ratio of 0.39 for moderate or worse SDB for participants reporting 3 to 6 hours per week of exercise relative to those reporting no exercise (final column of Table 3). From the same model, the decrement in BMI units needed to obtain a similar odds ratio is ~6.5 kg/m² (data not shown).

The slight attenuation of the exercise-SDB association following adjustment for measures of body habitus implies that body habitus might be acting as an intermediary variable between exercise and SDB, as a confounding variable, or both. If indicative of a causal relation, the persistence of the exercise-SDB association may indicate that exercise protects against SDB via additional mechanisms beyond favorable changes in body habitus profile. If so, exercise in addition to caloric restriction, as compared to weight reduction via caloric restriction alone, may be a preferred method of lifestyle modification in persons with obesity-related SDB.

Consistent with our findings, in their studies of exercise interventions, Giebelhaus and colleagues and Norman and colleagues saw mean...
ingual reductions in SDB severity following 6-month supervised exercise training regimens in small groups of persons with moderate SDB. In the Giebelhaus et al study, 47 11 subjects participated in a 6-month protocol of 2 hours of weekly aerobic exercise plus 2 hours of weekly weight lifting. While there was no mean change in BMI, participants did experience a reduction in AHI from a mean of 33 events per hour to 24 events per hour (−27%). The authors speculated that engagement of the pharyngeal and glossal muscles during exercise may have had a training effect on those muscles and, thus, helped maintain patency during periods of nocturnal susceptibility to upper airway obstruction. Additionally, while there was no net weight change in the 11 participants, there may have been changes in weight distribution and body composition that could be expected from an extended regimen of aerobic and weight-training exercise that might, in turn, lead to reductions in susceptibility to SDB events.

In the Norman et al study, 48 participants also performed aerobic exercise (but not necessarily weight training) for approximately 2 hours per week. The 9 participants in that study did experience modest net changes in weight (a 5% reduction in BMI from a mean of 31.2 kg/m² preintervention to 29.6 kg/m² postintervention) along with a reduction in waist to hip ratio. Thus, weight loss and changes in weight distribution and composition might have been one mechanism by which exercise (putatively) reduced SDB severity from a mean AHI of 22 events per hour preintervention to 12 events per hour postintervention (a 45% reduction).

The accuracy of our findings and interpretations may be limited by measurement error, uncontrolled confounding, selection bias, or statistical-model misspecification. The outcome of interest, SDB, was characterized by the AHI. We assessed the AHI by attended in-laboratory overnight polysomnography, the clinical standard. However, the AHI is but one of many possible characterizations of SDB and not necessarily the one most sensitively or specifically related to physical activity and exercise.

Of special concern, our measure of exercise is quite limited. We characterized exercise along one dimension—self-reported weekly time spent in planned exercise. We do not have information on the type or intensity of exercise, or on the frequency and duration of typical sessions of planned exercise. Although examples of exercise were provided (eg, “jogging” or “exercise class”), there is likely to be significant variation in what participants consider “planned exercise.” Measurement error in exercise likely produced an underestimate, perhaps substantial, of the association that otherwise would have been measured if pertinent aspects of physical activity and exercise had been assessed with a high degree of accuracy.

We controlled for several possible confounding variables, including age, sex, and educational attainment. However, it is possible that not all confounding factors were fully accounted for. For example, factors related to more or less healthy lifestyles (eg, dietary habits) or imprecision in characterizing tobacco habits might have lead to some misestimation of reported associations. However, we saw little evidence that confounding was an important consideration in assessing the exercise-SDB association—there were only slight changes in the coefficients of the exercise categories when we added potential confounding variables to the regression models.

An important additional consideration is that it is plausible that SDB due to SDB can lead to excessive daytime sleepiness and, perhaps, a general feeling of fatigue that may disincline persons with SDB to be physically active. If so, then a simplistic model of exercise predicting SDB may be insufficient to capture the full complexity of the relationship. As may be noted in Table 1, participants who exercised more hours per week had lower prevalences of typical feelings of excessive daytime sleepiness—despite reporting, on average, slightly fewer minutes of usual daily sleep—than participants who exercised less. However, the results from the regression analyses controlled for subjectively assessed excessive daytime sleepiness and self-reported typical occurrence of an unrefreshing night’s sleep. The presented models did not differ substantially from models that did not control for those factors, suggesting that there is an exercise-SDB association independent of feelings of sleepiness. This observation does not, however, dismiss the possibility of a coexisting reverse association between SDB, sleepiness, and lack of exercise.

Selection bias would be present in our study if the association between exercise and SDB in our defined sampling frame (sleep-survey respondents) differed from the association we measured in our sample of 1104 Sleep Cohort participants. While much of the difference between the sampling frame and the sample is due to members of the sampling frame not being randomly selected for invitation into the Cohort, approximately half of those selected for invitation refused to participate. Fortunately, because we have self-report data on snoring—a sensitive but nonspecific indicator of SDB—in both the entire invited sample and in the final study sample, we were able to compare the association between exercise and snoring in both samples. In the entire invited sample, after adjusting for age and sex, persons reporting 2 or fewer hours of exercise per week had a 34% greater odds of being a regular snorer (ie, odds ratio = 1.34) than persons reporting 3 or more hours of exercise per week. In the final participating sample for this report, the exercise-snoring odds ratio was 1.35. Thus, there was no evidence that the association between exercise and a crude indicator of SDB—self-reported snoring—was different between the invited and participating samples, indicating nonrandom selection bias was unlikely to have led to inaccurate results.

There are additional limitations of our study that are indicative of the current paucity of data examining the possibly complex relation between exercise and SDB. For example, it is not clear which aspects of SDB and exercise might be most related. Is there a minimal level of exercise intensity necessary to receive SDB-related benefits? What types of exercise (eg, aerobic vs. weight training) are most beneficial? What are the mechanisms by which exercise might protect against SDB? Our data are unable to address these questions, which are important topics for future research.

There appears to be an independent dose-response association between hours of weekly exercise and reduced severity and likelihood of SDB. Some, but apparently not all, of this association may be due to the beneficial effects of exercise on body weight and weight distribution and composition. Thus, physical activity and exercise programs may be an important component—in addition to the demonstrated effectiveness of dietary weight management—of clinical and public health efforts aimed at moderating the prevalence and severity of SDB in persons with SDB and in protecting persons currently without, but susceptible to, the future development of SDB.

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