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) 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.

Citation: Peppard PE; Young T. Exercise and sleep-disordered breathing: an association independent of body habitus. *SLEEP* 2004;27(3):480-4.

INTRODUCTION

SLEEP-DISORDERED BREATHING (SDB) IS ASSOCIATED WITH BEHAVIORAL[1-8] AND CARDIOVASCULAR[9-13] MORBIDITY EVEN IN ITS MILD MANIFESTATIONS. These associations are of particular concern because untreated SDB is highly prevalent among adults in the United States and other countries.[14-22] Continuous positive airway pressure can effectively prevent SDB events. However, this therapy presents a substantial and continuing patient burden, and thus its use in mild or asymptomatic SDB has been questioned. Modification of causal risk factors of SDB, then, is likely to be an important way to address the high prevalence of SDB. As we noted in a recent review,[23] there are several putative, potentially modifiable, risk factors for SDB, including obesity, alcohol and tobacco use, nasal congestion, and estrogen depletion in menopause. Presently, though, the only population-based intervention strategy rigorously and consistently supported by research evidence is weight loss.

Lack of physical exercise is likely to be causally associated with SDB, since obesity is a strong risk factor for SDB,[24,25] and inactivity is, in turn, linked to obesity.[26] However, despite the existence of a substantial literature documenting the relation between excess weight and increased prevalence of SDB in population studies,[18,21,22,24,25,27-32] as well as between weight loss and reduced severity of SDB in clinical studies of SDB patients,[33-46] the relation between exercise and SDB remains largely unexamined, since the population studies did not report exercise behavior and the clinic studies focused primarily on weight loss due to caloric restriction via dietary or surgical intervention.

Disclosure Statement

Grant support: R01HL62252, RR03186, and RO1AG14124 from the National Institutes of Health

Submitted for publication August 2003

Accepted for publication November 2003

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To our knowledge, only 2 exercise intervention studies examined the effect of exercise regimens on SDB severity.[47,48] These studies were small (a combined total of 20 subjects), lacked control groups, and were limited to predominantly middle-aged men (90%) with SDB. Both studies involved a 6-month supervised exercise protocol that included weekly aerobic exercise. The studies found reductions in measures of SDB following the exercise interventions without[47] or with[48] concomitant change in mean body weight. Thus, exercise may protect against or reduce SDB severity. However, mechanisms are unclear. Authors of both studies speculated that exercise-related training of the ventilatory muscles, including those of the upper airway, may be, in part, responsible for some of the observed improvement in SDB.

Clearly there is a need for continued study of the role of exercise in SDB. This report presents cross-sectional findings of the association between exercise and SDB from the Wisconsin Sleep Cohort Study, a large population-based (nonclinical) epidemiologic study of the natural history of SDB. This on-going study of middle-aged men and women represents the full spectrum of SDB—from none to severe—assessed by attended in-laboratory overnight polysomnography. With a sample of 1104 study participants, we address 2 specific questions: (1) How strong is the association of weekly hours of planned exercise and SDB? and (2) Is there an association between exercise and SDB that is independent of body habitus?

METHODS

Sample

Study protocols and informed consent documents were approved by the institutional review board of the University of Wisconsin Medical School. From 1989 to 1992, mailed questionnaires were sent to all men and women aged 30 to 60 years employed in a diverse set of job classifications at 5 State of Wisconsin agencies. The questionnaires obtained information on sleep problems and habits, weekly hours of exercise, and demographic and other data. Of the 4284 questionnaires sent, 3513 properly completed questionnaires were returned (an 82% response rate). A stratified random sample of eligible questionnaire respondents is continually recruited to participate in the ongoing Wisconsin Sleep Cohort Study. To date, 2912 persons have been invited to participate. Criteria

precluding Cohort participation include pregnancy, unstable or decompensated cardiopulmonary disease, airway cancers, and recent upper respiratory surgery. The average response rate for the continuing recruitment into the Sleep Cohort is approximately 51%. For this report, participants were also excluded if they had sleep studies with unusable physiologic parameters or less than 4 hours sleep time; medical treatment for SDB; physician-diagnosed stroke or cardiovascular disease; or missing data for key variables, including exercise or measures of body habitus. Following these additional exclusions, 1104 Sleep Cohort participants were available for analysis.

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/m^2).

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, electrooculography, 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 inductance plethysmography (Respitrace, Ambulatory Monitoring, Ardsley, 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 used to define 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 used to define a hypopnea event. The average number of apnea events plus hypopnea events per hour of objectively measured sleep define 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 workdays 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 $\log_e(\text{AHI}+1)$ 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*

Characteristic	Exercise (hours per week) 0	1 to 2	3 to 6	7 or more	All
N (percentage of entire sample), %	410 (37)	271 (25)	331(30)	92 (8)	1104
Men, %	57	43	52	58	54
Age, y	47 (8)	47 (8)	48 (8)	46 (8)	47 (8)
AHI, events/h	6 (12)	3 (8)	3 (6)	2 (4)	4 (9)
0 ≤ AHI < 5, %	75	84	84	90	82
5 ≤ AHI < 15, %	15	10	12	7	12
AHI ≥ 15, %	10	5	4	3	6
Typical sleep time (h/day)	7.6 (1.0)	7.6 (0.9)	7.4 (0.9)	7.3 (0.9)	7.5 (0.9)
Excessive daytime sleepiness, %	19	20	14	14	17
BMI, kg/m^2	30 (7)	28 (6)	29 (6)	28 (5)	29 (6)
Median BMI, kg/m^2	29	27	28	26	28
Skinfolds total†, mm	96 (40)	87 (36)	89 (42)	80 (42)	90 (40)
Neck girth, cm	38 (4)	37 (4)	37 (4)	37 (4)	38 (4)
Hypertensive‡, %	29	25	21	27	25
Cigarette smoker, %	18	10	11	11	13
Alcohol, drinks/wk	3 (5)	3 (5)	3 (6)	4 (6)	3 (5)
Education beyond high school, %	72	83	77	73	76

*Statistics are weighted to reflect the stratified sampling design. Data are presented as mean (SD) unless otherwise indicated.
†Sum of triceps, biceps, subscapular and suprailiac skinfold measurements.
‡Blood pressure ≥140/90 mmHg or current use of antihypertensive medications.
AHI refers to apnea-hypopnea index; BMI, body mass index.

Table 2—Mean AHI by Levels of Weekly Exercise*

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

*Data are presented as mean (confidence intervals). AHI refers to apnea-hypopnea index.
†Adjusted for age, sex, use of antihypertensive medication, sleepiness (excessive daytime sleepiness—often or always; unrested feeling after sleep—often or always), alcohol and tobacco use, and highest level of educational attainment.
‡Adjusted for body mass index, skinfold thickness, age, sex, use of antihypertensive medication, sleepiness (excessive daytime sleepiness—often or always; unrested feeling after sleep—often or always), alcohol and tobacco use, and highest level of educational attainment.

Table 3—Adjusted Odds Ratios Predicting AHI ≥ 5 and AHI ≥ 15 Events per Hour by Categories of Weekly Exercise

Exercise, h/wk	Odds ratios* Predicting AHI ≥ 5		Predicting AHI ≥ 15	
	Adjusted†	Habitus-adjusted‡	Adjusted†	Habitus-adjusted‡
0	1.00	1.00	1.00	1.00
1-2 (vs 0)	0.66 (0.42, 1.02)	0.78 (0.49, 1.23)	0.51 (0.26, 1.01)	0.62 (0.29, 1.29)
3-6 (vs 0)	0.57 (0.38, 0.86)	0.67 (0.44, 1.02)	0.34 (0.17, 0.68)	0.39 (0.19, 0.80)
≥ 7 (vs 0)	0.28 (0.12, 0.63)	0.33 (0.14, 0.79)	0.25 (0.07, 0.93)	0.31 (0.08, 1.24)
P value for trend	< .001	.005	< .001	.004

*Odds ratios are presented with 95% confidence intervals in parentheses and 0 hours of exercise per week as the reference.
†Adjusted for age, sex, use of antihypertensive medication, sleepiness (excessive daytime sleepiness—often or always; unrested feeling after sleep—often or always), alcohol and tobacco use, and highest level of educational attainment.
‡Adjusted for body mass index, skinfolds, age, sex, use of antihypertensive medication, sleepiness (excessive daytime sleepiness—often or always; unrested feeling after sleep—often or always), alcohol and tobacco use, and highest level of educational attainment.

The majority of participants (82%) had no or "minimal" SDB (0 ≤ AHI < 5 events per hour).

The columns of Table 2 give the results of 2 linear regression models that attempt to measure the independent association of exercise and SDB. The first column presents estimates of least-squares mean AHI for 4 levels of hours of weekly planned exercise. Adjusting for age, sex, use of antihypertensive medication, alcohol and tobacco habits, and excessive daytime sleepiness and unrefreshing sleep, there was a significant decreasing trend in mean AHI with increasing category of weekly exercise. The next column displays results from models further adjusting for body habitus (BMI and skinfold measurements). A significant decreasing, though somewhat attenuated, trend in mean AHI with increasing exercise is seen here too.

The first 2 columns of Table 3 present models that estimate the reduced odds of having mild or worse SDB (AHI ≥ 5 events per hour) associated with increasing hours of weekly exercise relative to no weekly exercise. The first row (1 to 2 hours of exercise vs. none) indicates that persons who exercised 1 to 2 hours weekly had 0.66 times the odds (ie, a 1.00-0.66 = 34% reduced odds) of having an AHI ≥ 5 compared to those who did not exercise at all, controlling for age, sex, use of antihypertensive medication, alcohol and tobacco habits, and excessive daytime sleepiness and unrefreshing sleep. Participants who exercised 7 or more hours per week were less than one third as likely as nonexercisers to have AHI ≥ 5 events per hour. As in the analysis of mean AHI, further adjusting for BMI and skinfolds (second column, Table 3) slightly diminished, but did not eliminate, the association between exercise and SDB. The last 2 columns of Table 3 present analogous models estimating the reduced odds of having moderate or worse SDB (AHI ≥ 15 events per hour) associated with increased exercise. These results were similar to those from the models predicting mild or worse SDB. Both sets of models represented statistically significant trends in reduced odds of SDB with increasing hours of weekly exercise.

In both the logistic and linear regression analyses, models stratified by sex, age, and body habitus and models that included interaction terms between exercise and those variables indicated no strong evidence for interaction effects. In other words, the association between exercise and SDB was similar for men and women, older and younger participants, etc. Also, in both sets of regression results, the inclusion of additional measures of body habitus to the presented models (eg, waist to hip ratio, neck girth) had no further affect on the results.

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/m^2 (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[47] and Norman and colleagues[48] saw mean-

ingful 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/m^2 preintervention to 29.6 kg/m^2 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.[50]

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 is a causal factor in physical inactivity. Sleep fragmentation 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 relation. 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.

ACKNOWLEDGMENTS

We are grateful for the technical expertise of Mari Palta, PhD, Jerry Dempsey, PhD, Steven Weber, PhD, Jim Skatrud, MD, Linda Evans, Laurel Finn, Tony Jacques, Hyon Kim, Andrea Peterson, Kathryn Pluff, and Leah Steinberg and the advice and comments of F. Javier Nieto, MD, PhD.

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