SCIENTIFIC INVESTIGATIONS

Association of Alcohol Consumption and Sleep Disordered Breathing In Men And Women

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Study Objectives: Experimental evidence indicates that alcohol use near bedtime may exacerbate sleep disordered breathing (SDB). However, scarce research has examined the relation between moderate habitual alcohol use and objectively assessed SDB, and it is unclear whether patients with SDB, or those at risk for SDB, should be counseled to avoid alcohol regardless of proximity to bedtime. In this population-based epidemiology study, our objective is to measure the association of SDB with usual alcohol consumption habits.

Methods: Men (N = 775) and women (N = 645)—initially randomly selected from a working population—participating in the Wisconsin Sleep Cohort Study were evaluated for alcohol consumption and SDB. The apnea-hypopnea index (AHI, events/hour) was determined by in-laboratory polysomnography. AHI>5 defined "mild or worse" SDB and AHI>15 defined "moderate or worse" SDB. Alcohol consumption (drinks/day) was assessed by questionnaire. Potential confounding or interacting variables such as smoking, body mass index, and medication use, were measured by clinical assessment and questionnaire.

Results: Relative to men who consumed less alcohol, for each increment of one drink per day, men who consumed more alcohol had 25% greater odds of mild or worse SDB (OR = 1.25, 95% CI = 1.07-1.46, p = 0.006). Among women, minimal to moderate alcohol consumption was not significantly associated with increased risk of SDB.

Discussion: In men, increased usual alcohol consumption was associated with increased risk of mild or worse SDB. Persons with SDB might benefit from generally reduced alcohol consumption and not just avoidance near bedtime.

Keywords: Sleep-disordered breathing, sleep apnea, alcohol use

Citations: Peppard P; Austin D; Brown R. Association of Alcohol Consumption and Sleep Disordered Breathing In Men And Women. *J Clin Sleep Med 2007*;3(3):265-270

Sleep disordered breathing (SDB)—a risk factor for hypertension,[1,2] vascular disease,[3,4] and impaired functioning[5,6]—afflicts millions of adults in the United States.[7] Although alcohol use is widespread in the U.S., few studies have examined the association of alcohol use habits and SDB. Those few studies have taken 1 of 2 approaches: experiments examining the acute effect of alcohol doses on SDB, or epidemiologic analyses of correlations between self-reported alcohol use and SDB. Most,[8-15] but not all,[16, 17] studies in which defined quantities of alcohol were administered to healthy subjects or patients with SDB just prior to sleep have shown effects on respiration, including increasingly frequent and longer-duration hypopnea and apnea events. A few epidemiologic investigations of predictors of self-reported snoring[18-21] or sleep apnea[22] found alcohol use was associated with increased SDB severity, but others failed to demonstrate associations.[22-24] Tanigawa and colleagues,[25] reporting data from an epidemiologic study of 1425 Japanese men specifically designed to assess alcohol use and SDB, observed an increased risk of SDB in alcohol-consuming men compared to nondrinkers. The association was graded—with heavier drinkers at greater risk—and was most pronounced in leaner persons.

Given the findings of experimental studies, it is prudent to counsel patients with SDB, or at risk for SDB (e.g., overweight or snoring patients), to avoid alcohol use in the hours prior to bedtime. However, it remains unclear whether patients who habitually consume light to moderate levels of alcohol should reduce consumption regardless of time of day. While experimental data are best suited for examining short-term effects of alcohol on sleep respiration, the association of long-term alcohol use habits along the spectrum of nondrinkers to moderate drinkers can be practically examined only in observational studies. The objective of this report from the Wisconsin Sleep Cohort Study is to examine associations between typical alcohol use and SDB in adult men and women. We focus on the clinical question posed above—the association of habitual alcohol use along the spectrum of nondrinkers to moderate drinkers—by limiting analyses to 1420 participants who: 1) reported steady drinking habits over the 5 years prior to SDB evaluation; 2) consumed fewer than 6 drinks/day on average; and, 3) consumed no more than 2 drinks during the day of the sleep study to minimize possible "acute" effects of alcohol on the measurement of SDB.

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METHODS

Participants

The Wisconsin Sleep Cohort Study is an ongoing epidemiologic investigation of the natural history of SDB. Informed consent documents and study protocols—details of which have been reported previously[1,26]—were approved by the University of Wisconsin-Madison Health Sciences Institutional Review Board.

Beginning in 1988, female and male employees of 5 State of Wisconsin agencies, between the ages of 30-60 years, were surveyed by mail regarding sleep habits and problems (N = 5091). From these data, a sampling frame was constructed and randomly selected survey respondents (N=2884) were invited to participate in the Wisconsin Sleep Cohort. Initial exclusion criteria included pregnancy, unstable or decompensated cardiopulmonary disease, airway cancers, and recent upper airway surgery. Sleep Cohort participants had overnight sleep studies at the University of Wisconsin General Clinical Research Center. Repeat studies were carried out at 4-year intervals. For this analysis, 1 to 4 sleep studies were available for each participant.

For this report, Sleep Cohort study participants were excluded from analyses if they had inadequate sleep studies (<3 hours of sleep, or < 4 hours of sleep with <20 minutes REM sleep) in the laboratory. As of May 2005, there were 1519 eligible participants with at least 1 adequate sleep study (53% of those invited for baseline studies). The majority of persons who refused participation reported that the burden of sleeping overnight away from home in a sleep laboratory was the reason for refusal. Of the 1519 participants, 57 persons were excluded from this analysis because they reported major changes in drinking habits in the 5 years prior to their sleep studies. An additional 3 participants were excluded due to reporting heavy drinking—an average of ≥42 drinks per week. Finally, 6 participants who consumed > 2 alcohol-containing drinks after noon on the day of the sleep study and 33 participants who used continuous positive airway pressure (CPAP) devices at the time of their sleep studies (thus SDB could not be meaningfully assessed) were excluded from analyses. The final sample for this report was 1420 participants (775 men and 645 women) with a total of 3165 sleep studies.

Measurements

Study participants completed overnight studies that included nocturnal polysomnography and other clinical tests. Participants arrived for sleep studies in the early evening. Information on alcohol use, medical history, current medication use, smoking, age, and other sociodemographic factors was obtained by interview and questionnaire. Alcohol use habits were assessed by questionnaire items regarding: 1) usual weekly number of cans/bottles of beer, glasses of wine, mixed drinks or shots of liquor; 2) number and time of drinks on the day of sleep study; and, 3) changes in typical drinking habits over the previous 5 years.

Medication use, obtained by questionnaire, was coded into 23 pharmacologic categories for which there were sufficient numbers of participants using to analyze. These categories included anti-anxiety, hypnotics-benzodiazepines, stimulants, classes of antidepressants, classes of antihypertensives, and muscle relaxants.

An 18-channel polysomnography recording system (Polygraph model 78, Grass Instruments, Quincy, MA) was used to assess sleep stage, respiratory, and cardiac parameters. Sleep stage was determined by electroencephalography, electrooculography, and chin electromyography. These signals were used to score sleep stage for each 30-second epoch of the polysomnographic record, using conventional criteria.[27] Arterial oxyhemoglobin saturation, oral and nasal airflow, nasal air pressure, and thoracic cage and abdominal respiratory motion were used to assess SDB events. Oxyhemoglobin saturation was continuously recorded using pulse oximetry (Ohmeda 3740, Englewood, CO). Thermocouples (ProTec, Hendersonville, TN) detected oral and nasal airflow. A pressure transducer (Validyne Engineering Corp., Northridge, CA) continuously measured air pressure at the nares. Respiratory inductance plethysmography (Respitrace, Ambulatory Monitoring, Ardsley, NY) continuously recorded thoracic cage and abdominal excursions. Sleep stage and respiratory event scoring were performed by trained sleep technicians and reviewed by an expert polysomnographer. Each 30-second epoch of the polysomnographic records was visually inspected and scored for abnormal breathing events. Cessation of airflow lasting ≥10 seconds defined an apnea event. A discernable reduction in the sum of rib cage plus abdomen respiratory inductance plethysmography amplitude associated with ≥4% reduction in oxyhemoglobin saturation defined a hypopnea event. The average number of apnea events plus hypopnea events per hour of objectively measured sleep defined the apnea-hypopnea index (AHI), our summary parameter of SDB.

Body habitus measurements—including height and weight; waist, neck and hip girths; and biceps, triceps, subscapular and suprailiac skinfold thicknesses—were performed using standard procedures.[28] Body mass index (BMI) was calculated from height and weight (kg/m^2).

Statistical analyses

Analyses were performed with SAS software, release 9.1 (SAS Institute, Inc., Cary, NC). Sex-specific generalized estimating equation regression models[29] were used to measure the association between alcohol use and SDB while controlling for:1) potentially confounding and interacting variables (age, body habitus, smoking habits, and medication use); 2) SDB scoring method (using paper or electronic recordings of the polysomnograms); and 3) within-subject correlation due to multiple sleep studies per participant. We made an a priori decision to stratify analyses by sex because of the marked differences in distribution of both alcohol use and SDB in men and women.

Participants' alcohol use was parameterized as average drinks per day. This was calculated as the number of participant-reported weekly drinks of beer plus wine plus hard liquor divided by 7. For descriptive statistics, drinks per day groups were categorized as: 0 (nondrinkers); more than 0 to less than 1 drink/day (i.e., 1 to 7 drinks/week); 1 to less than 2 drinks/day; and 2 to less than 6 drinks/day. Too few participants reported drinking between 2 and 6 drinks/day to further subdivide that category. In addition to drinks/day, we examined alcohol habits using alternative approaches including estimated grams of alcohol per kg body mass per day. Supplementary exploratory analyses examined subtypes of alcohol (beer vs. wine vs. hard liquor).

Two sets of models estimating odds ratios for SDB were fit using common cutpoints for defining SDB severity: 1) AHI<5 vs. AHI≥5 events/hr ("mild or worse" SDB), and 2) AHI<15 vs.

Table 1—Characteristics of the study population[a]

| | Alcohol drinks per day | | | | |
	Nondrinker	0.1 – 0.9	1.0 – 1.9	2.0 – 5.9	All
MEN					
Participants: N (%)	281 (36)	324 (42)	120 (15)	50 (6)	775
Sleep studies[a]: N (%)	516 (30)	752 (44)	321 (19)	135 (8)	1724
Age: years, mean (SD)	51 (8)	51 (8)	52 (9)	52 (9)	52 (9)
BMI: kg/m^2, mean (SD)	30 (6)	30 (5)	29 (5)	29 (4)	30 (5)
Current smoker: N (%)	64 (12)	80 (11)	50 (16)	35 (26)	229 (13)
AHI[b] > 5: N (%)	175 (34)	261 (35)	119 (37)	56 (41)	611 (35)
AHI[b] > 15: N (%)	78 (15)	105 (14)	34 (11)	18 (13)	235 (14)
WOMEN					
Participants: N (%)	332 (51)	267 (41)	34 (5)	12 (2)	645
Sleep studies[a]: N (%)	639 (44)	657 (46)	110 (8)	35 (2)	1441
Age: years, mean (SD)	51 (9)	51 (9)	51 (9)	52 (8)	51 (9)
BMI: kg/m^2, mean (SD)	33 (8)	30 (7)	29 (6)	29 (6)	31 (8)
Current smoker: N (%)	96 (15)	78 (12)	26 (24)	13 (37)	213 (15)
AHI[b] > 5: N (%)	162 (25)	122 (19)	24 (22)	8 (23)	316 (22)
AHI[b] > 15: N (%)	55 (9)	30 (5)	6 (5)	4 (11)	95 (7)

[a]Most participants had multiple (up to 4) sleep studies. Statistics are reported using sleep studies as units of analysis.
[b]Apnea-hypopnea index (events per hour of sleep), adjusted for scoring method.

AHI≥15 ("moderate or worse" SDB). We also examined untransformed and log-transformed continuous parameterizations of SDB.

RESULTS

Table 1 displays selected characteristics by categories of alcohol use and sex. Because SDB is strongly associated with excess body weight, it is noteworthy that both male and female participants who consumed more alcohol had, on average, slightly lower body mass indices (BMI) than participants who consumed less or no alcohol.

Table 2 presents associations between alcohol consumption and mild or worse SDB (AHI≥5 events/hr) separately for men and women, adjusting for progressively more covariates. Among men there is a modest, though only borderline significant (p=0.07) positive association between drinks per day and prevalence of SDB prior to adjustment for participant characteristics. After adjustment for age and body habitus measures, the association is stronger and significant. Further adjustment for cigarette smoking and 3 classes of medications (those classes for which adjustment was found to have a moderate affect on the alcohol-SDB association) yields a final estimate for men of an approximately 25% increased odds (odds ratio = 1.25, 95% C.I. = 1.07 to 1.46) of SDB for each increment of one drink per day. There were no significant associations between moderate alcohol consumption and SDB in women. In both sex-specific analyses, further adjustment for other examined variables had no substantial effect on the presented models. For both men and women, associations between alcohol use and moderate or worse SDB (AHI≥15 events/hr) were small and not statistically significant (data not shown).

In an exploratory attempt to examine independent (mutually adjusted) associations of beer, wine, and hard liquor with SDB, we found beer, but not wine or hard liquor, to be positively and significantly associated with mild or worse SDB (AHI>5) in men. The logistic regression coefficient for drinks of beer per day was 0.31 (95% C.I. = 0.12 to 0.50). Multiplying this coefficient by 2 and exponentiating yields an odds ratio of 1.86, indicating, for example, an 86% increase in the odds of prevalent SDB for per-

sons who consume 2 beers/day relative to those who consume no beer. Neither beer, wine, or hard liquor was significantly associated with any SDB in women, or in men with moderate or worse SDB (AHI≥15). Beer accounted for approximately 61% of drinking in men and 32% in women, and participants who consumed beer tended to consume more (mean[SD] = 4.5[4.8] drinks/week) than consumers of wine (2.6[2.6]) or hard liquor (3.0[3.3]). Thus, there was more power and a greater range of consumption with which to detect associations with beer compared to other forms of alcohol.

We found no evidence of interactions; that is, there were no statistically significant variations in associations between alcohol and SDB by age, body habitus, medication use, or cigarette smoking. We found no evidence that models incorporating alternative alcohol-use parameterizations—i.e., transformations (log-transformed continuous or categorical), higher-order terms (e.g. squared), or modeling grams of alcohol consumption per unit body mass—better described associations between alcohol and SDB than those models presented in Table 2.

As a check for selection bias, we examined whether there was variation in cross-sectional associations of alcohol and SDB according to study visit (1, 2, 3, and 4). There was no significant interaction. For example, the odds ratio for a 1 drink/day increment predicting mild or worse SDB in men when only baseline studies (1st study visit) were used was 1.21 (95% C.I.: 0.99, 1.49), which is similar to the corresponding odds ratio of 1.25 estimated using all available data (Table 2). We also examined the effect of including CPAP users in the analyses—defining them as having SDB. Regression models that included CPAP users yielded essentially the same results as those presented in Table 2—i.e., nonsignificant associations between alcohol use and SDB in women and a significant association between alcohol use and mild or worse SDB in men: for each increment of 1 drink/day, there was a 1.24-fold increased odds (95% C.I.: 1.06, 1.44) of SDB, compared to the odds ratio for men of 1.25 presented in Table 2. Finally, we examined the effect of adjusting for the number of drinks consumed on the day of the sleep study (0, 1, or 2 drinks consumed at least 4 hours prior to initiation of the sleep study). Again, there

Table 2—Sex-specific unadjusted and adjusted odds ratios for mild or worse SDB (AHI>5 events/hr) related to drinks per day of alcohol consumption.

	Unadjusted[a] odds ratio (95% C.I.)	Age & habitus adjusted[b] odds ratio (95% C.I.)	Fully-adjusted[c] odds ratio (95% C.I.)
MEN			
0 drinks	1.00 (reference)	1.00 (reference)	1.00 (reference)
1 drink	1.13 (0.99, 1.30)	1.22 (1.04, 1.42)	1.25 (1.07, 1.46)
2 drinks	1.29 (0.98, 1.69)	1.48 (1.09, 2.02)	1.55 (1.14, 2.12)
3 drinks	1.46 (0.97, 2.19)	1.81 (1.14, 2.87)	1.94 (1.21, 3.09)
p-value[d]	0.07	0.01	0.006
WOMEN			
0 drinks	1.00 (reference)	1.00 (reference)	1.00 (reference)
1 drink	0.80 (0.57, 1.13)	1.00 (0.71, 1.40)	0.94 (0.67, 1.33)
2 drinks	0.64 (0.32, 1.27)	1.00 (0.51, 1.96)	0.89 (0.45, 1.76)
3 drinks	0.51 (0.18, 1.43)	1.00 (0.36, 2.75)	0.83 (0.30, 2.33)
p-value[d]	0.20	0.997	0.73

[a] Adjusted only for SDB scoring method and multiple sleep studies per participant

[b] Additionally adjusted for: age, body mass index, neck and waist circumferences

[c] Additionally adjusted for: current cigarette smoking; antidepressant, antihypertensive, and cholesterol-lowering medication use

[d] P-value for linear relation between drinks per day and log(odds ratio) for SDB (AHI>5)

were no substantial differences between models including an adjustment term and those presented in Table 2. For example, for each increment of 1 drink/day, there was an adjusted 1.26-fold increase in odds (95% C.I.: 1.08, 1.48) of mild or worse SDB in men, compared to the odds ratio of 1.25 presented in Table 2.

DISCUSSION

This investigation was designed to address the important clinical question of the relation between minimal to moderate habitual alcohol consumption and SDB. Several experimental studies have demonstrated short-term effects of alcohol on SDB,[8-15] indicating that persons with or at risk for SDB (e.g., overweight patients or those who report loud snoring), should avoid alcohol near bedtime. However, little information is available to guide the clinician in counseling SDB patients about moderate habitual alcohol use. To focus the analysis on this question we excluded persons who had more than 2 drinks the afternoon prior to sleep study so that measured associations might primarily reflect habitual, not short-term, alcohol use (supplementary analyses controlling for the number of drinks consumed on the day of the sleep study yielded results nearly identical to those presented). Participants who reported habitually consuming large amounts of alcohol were also excluded.

We found that men who reported habitual alcohol consumption were more likely to have mild or worse SDB (AHI≥5 events/hr) than men who consumed no alcohol. The association was graded, with an increment of 1 drink per day in alcoholic beverage consumption associated with a 25% (95% C.I. = 7% to 46%) increase in the odds of SDB. This association was not maintained for more severe SDB in men: there were no significant associations between alcohol use and the odds of moderate or worse SDB (AHI≥15 events/hr). This may reflect less power to detect associations of alcohol and more severe SDB because there were fewer men with severe SDB in the sample. It may also reflect a "ceiling" effect whereby alcohol might produce sufficient respiratory disturbance to initiate events of SDB in susceptible persons (who, in the absence of alcohol use would be free of SDB) but not

have substantial incremental impact on persons who already have at least mild SDB. Nevertheless, mild or worse SDB is highly prevalent among in the United States, afflicting up to 1 in 4 men, and this "mild" degree of SDB is associated with cardiovascular and behavioral outcomes.[7]

Our results comparing men with an AHI≥5 vs. those with <5 events/hr were consistent with those presented by Tanigawa et al. in their epidemiologic study of Japanese men.[25] For example, they reported an odds ratio of 1.45 (95% C.I. = 1.02 to 2.04) predicting prevalent mild or worse SDB for persons drinking 0.5 to 1.0 g ethanol per kg body weight per day (or, approximately 2.5 to 5 drinks/day containing 14 g ethanol for a 70 kg man) relative to nondrinkers. Unlike Tanigawa et al., we found a weak, nonsignificant association between alcohol use and more severe SDB (AHI≥15 events/hr). This may be, in part, related to their finding that alcohol-SDB associations were stronger among leaner subjects (BMI<23.9 kg/m^2). Participants in our sample were, on average, less lean than the Japanese study population. Very few lean Wisconsin Sleep Cohort participants had moderate to severe SDB, limiting our ability to investigate the association of alcohol use and SDB in lean persons.

We found no significant associations between alcohol use and SDB in women regardless of the severity of SDB. Possible explanations for this include the limited range of alcohol consumption reported by women in our sample—fewer than 10% of women reported averaging one or more drinks/day—reducing our power to detect clinically important moderate associations. Alternatively, women may be more resistant than men to threats to nocturnal respiratory stability, as suggested by the study of Block and colleagues[30] who found men, but not women, to be susceptible to oxygen desaturation episodes during sleep after acute administration of 1 ml alcohol per kg body weight. Such protection may be due to hormonally-mediated increased ventilatory drive, anatomical differences, or other characteristics[31,32] that may provide general protection for women from events of SDB. For example, women appear to require relatively greater increases in body mass to demonstrate weight-related increments in SDB compared to men.[33]

Our assessment of the association of alcohol and SDB may be limited by the standard deficiencies of observational studies—potential selection and measurement error biases and uncontrolled confounding. Considering the expense and participant burden associated with overnight laboratory-based polysomnography, this was a large study, allowing us to detect with statistical significance a modest but important association between alcohol and SDB in men. However, the Sleep Cohort experiences some participant dropout due to participant burden. Fifty-three percent of persons initially invited to the study eventually participated in at least one successful sleep study. Follow-up rates among those invited to subsequent sleep studies have been approximately 80%. Because we used multiple sleep studies from the majority of participants, we employed standard procedures for accounting for intrasubject correlation due to the use of multiple studies.[29,34] The availability of multiple studies allowed us to make a superficial check for evidence of selection bias by examining whether alcohol-SDB associations varied according to study visit among participants with multiple visits. There was no statistically significant variation (p = 0.53), suggesting that factors influencing participation in multiple visits did not lead to substantially biased associations. If similar factors influenced participation in initial studies, then it would be unlikely that an important bias due to nonparticipation affected the accuracy of the findings.

Measurement error in assessing SDB, alcohol use, or important covariates may reduce the accuracy of our findings. Random error in measuring alcohol use or SDB is likely to produce a bias toward no association,[35] which may partially explain our lack of significant findings in women. We used in-laboratory polysomnography, the diagnostic "gold standard," to classify SDB status. However, our questionnaire-based measure of alcohol use was less robust. While random error (or systematic overreporting) of alcohol consumption may have obscured associations, systematic underreporting of alcohol use in men may have exaggerated the association between alcohol and SDB. Notably, among participants in our investigation who had multiple studies, there was an intraclass correlation coefficient = 0.82 between self-reported alcohol drinks/week consumed 4 years apart. That is, most variation in self-reported drinking was due to difference among participants (i.e., 82%) rather than changes (or inconsistencies) *within* participants' self-report at multiple time points. We do not have available objective markers to validate self-reported alcohol use. However, in the Tanigawa et al. report[25] examining alcohol and SDB in Japanese men, serum γ-glutamyl transferase—an objective biomarker for regular alcohol use[36]—was well correlated with categories of self-reported alcohol use in that population.

The associations between alcohol and SDB may be confounded by variables that were associated with both alcohol use and SDB. We measured and controlled for several potential confounding factors including age, cigarette smoking, body habitus, and medications. In our sample of men, BMI was a confounding variable. In Table 1, it can be seen that the proportion of men with AHI≥5 remains steady with increasing alcohol use, then increases substantially at the highest level of alcohol use (≥2 drinks/day) *despite* a decrement in BMI with increasing alcohol use. This is the reason why adjustment for body habitus measures resulted in increased alcohol-SDB odds ratios in men.

Though this report used multiple studies per participant—and corrected for resulting intrasubject correlation using standard methods[29, 34]—we do not report here on longitudinal associations between alcohol use and SDB. There are 2 reasons for this. First, at initiation, the ongoing Wisconsin Sleep Cohort Study enrolled adults aged 30-60 years. In this sample, alcohol habits changed little over the period of study. Only 53 of 1519 participants reported major changes (e.g., ceasing use) in alcohol habits over 5-year periods prior to sleep studies. Thus, longitudinal changes in SDB status associated with substantial changes in alcohol use were not observable in a sufficiently large sample. Second, while we had 3165 sleep studies available for cross-sectional analyses, there were too few participants with ≥3 studies available for robust longitudinal analysis of what appears to be a moderate association, perhaps confined to men.

Experimental evidence is fairly consistent in demonstrating acute effects of alcohol exposure—alcohol consumed near bedtime—on initiating or exacerbating SDB, perhaps by reducing upper airway patency via reduced dilatory muscle tone, or by blunted ventilatory response to hypoxia.[10, 12, 13, 25, 37] Thus, based on the previous experimental evidence, men and women with SDB or those particularly susceptible to SDB should be advised to avoid alcohol near bedtime. By design, we attempted to exclude acute effects, since SDB patients might reasonably wonder if alcohol should be altogether avoided. Our data indicate that for very moderate levels of drinking, there appears to be little association with SDB in women. Among men, greater alcohol consumption was associated with higher risk of mild or worse SDB. There are no data available to indicate mechanisms by which habitual moderate alcohol consumption—whether via neurotoxic effects on respiratory control mechanisms, or other possible pathways—might explain "non-acute" associations. However, pending further observational and mechanistic research, our data suggest that men with SDB and those at risk for SDB can be advised to minimize alcohol consumption regardless of proximity to bedtime.

ACKNOWLEDGEMENTS

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