



# Alcohol consumption and sleep quality: a community-based study

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## Abstract

**Objective:** To assess the association between total alcohol intake, specific alcoholic beverages and sleep quality in a community-based cohort.

**Design:** A cross-sectional study.

**Setting:** The Kailuan community, China.

**Participants:** Included were 11 905 participants who were free of a history of CVD, cancer, Parkinson's disease, dementia and head injury in or prior to 2012. Alcohol consumption (amount and frequency intake) and alcoholic beverage type were collected in 2006 (baseline) and 2012. Participants were grouped into non-, light- (women: 0–0.4 serving/d; men: 0–0.9 serving/d), moderate- (women: 0.5–1.0 serving/d; men: 1.0–2.0 servings/d) and heavy- (women: >1.0 servings/d; men: >2.0 servings/d) drinkers. Overall sleep quality was measured in 2012 and included four sleep parameters (insomnia, daytime sleepiness, sleep duration, snoring/obstructive sleep apnoea).

**Results:** We observed a dose–response association between higher alcohol consumption in 2006 and worse sleep quality in 2012 ( $P_{\text{trend}} < 0.001$ ), after adjusting for age, sex, socio-economic status, smoking status, physical activity, obesity, plasma lipid profiles, diabetes and hypertension. A similar association was observed when alcohol consumption in 2012 was used as exposure. Alcohol was associated with higher odds of having short sleep duration (adjusted OR for heavy- *v.* non-drinkers = 1.31; 95 % CI: 1.09, 1.57) and snoring (adjusted OR for heavy- *v.* non-drinkers: 1.38; 95 % CI: 1.22, 1.57). Consumption of hard liquor, but not beer or wine, was significantly associated with poor sleep quality.

**Conclusions:** Higher alcohol consumption was associated with poorer sleep quality and higher odds of having snoring and short sleep duration.

**Keywords**  
Alcohol  
Sleep disorders  
Community  
Insomnia  
Daytime sleepiness  
Sleep duration  
Snoring

Alcohol has been suggested to have a hypnotic effect on the body because it can suppress the function of the central nervous system<sup>(1–3)</sup>. In contrast, alcohol also could interrupt the sleep cycle and increase the risk of sleep disorders by interrupting the respiratory system during sleep and competing with neuroimmune and neurotransmitter systems<sup>(1,2,4–6)</sup>. Alcohol tends to reduce sleep onset latency and decrease the rapid eye movement sleep percentage of the total sleep period and results in a higher frequency of waking during sleep<sup>(1,2)</sup>. Previous human studies, including a clinical trial and several observational studies, found that alcohol intake may increase the odds of sleep disorders<sup>(1,7,8)</sup>. Although these results demonstrate a possible connection between alcohol consumption and sleep disorders, these studies were limited by a small

sample size or lack of detailed information on sleep disorder type and alcohol consumption (i.e., amount and type of alcohol consumed)<sup>(1,7,8)</sup>. Further, most previous studies of the association between alcohol intake and sleep quality focused on excessive drinking, inducing participants with alcoholism, alcohol dependence and alcohol use disorders (AUD)<sup>(6,9–11)</sup>. The impact of alcohol intake on sleep quality in community populations without excessive drinking remains unclear. Additionally, previous studies have only investigated the effect of alcohol consumption on one or two sleep parameters (i.e., insomnia, daytime sleepiness or snoring/obstructive sleep apnoea (OSA))<sup>(4,5,12)</sup>. The impact of habitual alcohol consumption on overall sleep pattern remains unknown.

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Therefore, we conducted a cross-sectional study using a community-based cohort to examine the association of alcohol intake, specifically the amount and type of alcoholic beverages consumed, with sleep quality. Overall sleep quality was evaluated by combining four sleep parameters including insomnia, daytime sleepiness, sleep duration and snoring. We also examined the association between alcohol intake and each individual sleep parameter. We hypothesised that higher alcohol intake would be associated with poorer sleep quality, as suggested by low overall sleep quality score and higher odds of having individual sleep disorders.

**Method**

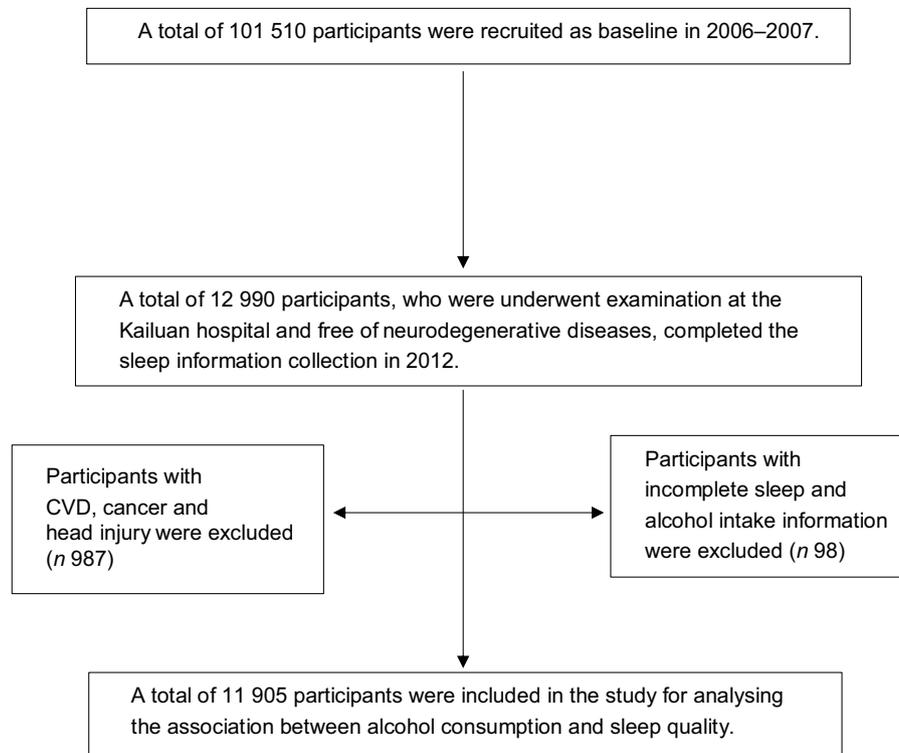
**Participants**

The analysis was based on a subset of a Chinese cohort, the Kailuan Study, being conducted in the Kailuan community in Tangshan city, China<sup>(13)</sup>. In the Kailuan Study, 101 510 participants (81 110 men and 20 400 women) from the ages of 18–97 were recruited in 2006–2007 at eleven hospitals (study sites)<sup>(13–15)</sup>. These hospitals provide primary care for all participants of the Kailuan Study. All participants completed standardised questionnaires, laboratory assessments and underwent physical examination. Every 2 years, the questionnaires, laboratory assessments and clinical examination were repeated.

In 2012, information on sleep habits was collected in 12 990 participants (10 725 men and 2265 women) who had completed the survey in the Kailuan Hospital (the largest study site of the Kailuan Study) and were free of neurodegenerative diseases, as detailed previously<sup>(13,14)</sup>. We excluded participants with CVD, cancer and head injury (*n* 987) and participants who had incomplete sleep and alcohol intake information (*n* 98), leaving 11 905 participants (9776 men and 2129 women) in the current analysis. Fig. 1 showed the flow chart of the study.

**Measurement of alcohol consumption**

Information on alcohol intake (both amount and type) was collected via questionnaires during the Kailuan Study, in 2006 (baseline) and 2012<sup>(13,15)</sup>. To reduce the possibility of reverse causality (the presence of sleep disorders may change alcohol intake) we used the alcohol intake from the 2006 questionnaire as the primary exposure. However, we also examined the 2012 alcohol intake as a secondary exposure to understand the potential short-term impact of alcohol intake on sleep. In the questionnaire, participants reported their alcohol intake over the past 12 months, including consumption (yes or not), beverage type (beer, wine, hard liquor), amount of alcohol consumed and frequency of intake. Alcohol consumption was calculated (in g/d) via the frequency of intake (times/d) multiplied by the usual amount of alcoholic beverage consumed and the corresponding average ethanol content of that



**Fig. 1** Flow chart of the study



beverage (5.0 g for 100 g beer, 12.0 g for 100 g wine, 40.0 g for 100 g hard liquor)<sup>(13,15)</sup>. A standard drink (classified as 'one serving' in the study) contained around 14.0 g of ethanol<sup>(15)</sup>.

The US Department of Agriculture and US Department of Health and Human Services define one standard drink as a beverage containing 0.6 fl oz or 14.0 g ethanol on the 'Dietary Guidelines for American 2015–2020'<sup>(16)</sup>. Thus, based on the definition of one standard drink, we classified participants into the following categories of alcohol consumption: non-drinkers, light drinkers (women: 0–0.4 serving/d; men: 0–0.9 serving/d), moderate drinkers (women: 0.5–1.0 serving/d; men: 1.0–2.0 servings/d) and heavy drinkers (women: >1.0 servings/d; men: >2.0 servings/d)<sup>(16)</sup>. For the alcoholic beverage types, we defined three categories: beer, wine and hard liquor. Within each category, we divided participants into two groups, a 'yes' group containing people who drank the indicated type of alcoholic beverage and a 'no' group containing people who did not drink any alcohol or did not drink the indicated type of alcoholic beverage.

The validity of the self-reported alcohol consumption data has been confirmed in previous studies. One study from our lab demonstrated a dose–response relationship between alcohol consumption and HDL-cholesterol concentrations in a cross-sectional analysis conducted in 71 379 Kailuan Study participants<sup>(15)</sup>.

### **Measurement of sleep quality**

In the Kailuan Study, the majority of the sleep data was collected in 2012 via questionnaires administered verbally by trained interviewers<sup>(13,15,17)</sup>. OSA information was collected in 2014. The primary outcome of the current study was overall sleep quality, based on four sleep parameters including insomnia, daytime sleepiness, sleep duration and snoring, as detailed previously<sup>(17)</sup>.

### **Insomnia**

The insomnia status of participants was assessed via a Chinese version of the Athens Insomnia Scale (AIS)<sup>(12,18)</sup>. The AIS is a self-report questionnaire designed to assess the insomnia status of a person in the past month via eight questions about sleep situations<sup>(18)</sup>. The score of each question within the AIS ranges from 0 to 3 (0 = no event, 1 = mild, 2 = moderate, 3 = severe), and the maximum total score of the AIS is 24 (8 × 3 = 24)<sup>(12)</sup>. A participant with a total score scale ≥6 was classified as having insomnia<sup>(12)</sup>.

### **Daytime sleepiness**

Daytime sleepiness data were collected via a Chinese version of the Epworth Sleepiness Scale (ESS)<sup>(19,20)</sup>. To support the validation of the Chinese version of ESS, a previous study demonstrated an acceptable test–retest reliability ( $\rho = 0.74$ ,  $P = 0.01$ ) of the Chinese version of ESS among the Chinese population<sup>(19)</sup>. The ESS includes eight items,

each scored from 0 to 3, to evaluate a person's feeling of falling asleep while engaging in daily activities, in specific circumstances<sup>(20)</sup>. A person with a higher score for each item represents a higher tendency of falling asleep. The total score of the ESS ranges from 0 to 24<sup>(20)</sup>. A person with a score ≥10 was classified as having excessive daytime sleepiness<sup>(20)</sup>.

### **Sleep duration**

Information regarding the sleep duration of participants was collected via self-report surveys<sup>(14)</sup>. Participants reported their total sleep hours during a usual night. In the current study, sleep duration was classified into four groups (<6, 6–7, 7–8, ≥8 h/d).

### **Snoring and obstructive sleep apnoea**

In 2012, self-reported snoring, including self-reported snoring and breathing stops (i.e., apneas), was collected via a questionnaire<sup>(14,17)</sup>. A participant with self-reported breathing stops must have had >10 s of breathing stops before breathing re-covered and had more than an estimated fifty breathing stops per night<sup>(14,17)</sup>. Participants reported the frequency of snoring based on three levels (never/rare, occasional or frequent).

In 2014, the OSA status and anthropometric assessments were measured via a STOPBANG questionnaire by a trained interviewer, as detailed previously<sup>(13,14,21)</sup>. The STOPBANG questionnaire evaluates a participant's risk of OSA and contains eight binary variables including snoring, daytime tiredness, observed apnoeas, blood pressure, BMI (BMI, >35 kg/m<sup>2</sup>), age (>50 years), neck circumference (>40 cm) and sex (men)<sup>(13,14)</sup>. A person with three or more positive scores of the eight binary variables is considered to have an intermediate or high risk of OSA<sup>(13,21)</sup>. The validation of the STOPBANG questionnaire in a Chinese population is supported by a previous study demonstrating high sensitivity (91–94%)<sup>(22)</sup>. In our secondary analysis, we examined the relation between alcohol and OSA status.

### **Overall sleep quality**

As detailed elsewhere, overall sleep quality was calculated based on the combination of the four sleep parameters assessed in 2012, each scored 0–2, with a total score of 8 (0 = best sleep quality, 8 = worst sleep quality)<sup>(17)</sup>. A person without insomnia (AIS score <6) or with insomnia (AIS score ≥6) was scored either 0 or 2. A person without daytime sleepiness (ESS score <10) or with excessive daytime sleepiness (ESS score ≥10) was scored either 0 or 2. Participants with a sleep duration of <6.0 h/d or ≥8.0 h/d was scored 2, while a sleep duration of 6.0–6.9 h/d was scored 1 and a sleep duration of 7.0–7.9 h/d was scored 0. Self-reported snoring was classified into three score groups (none = 0, occasional = 1, frequent = 2).

**Table 1** Demographic and clinical characteristics of participants according to alcohol consumption status in 2006\*,†

	None		Light		Moderate		Heavy	
	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Age (year)	47.8	0.14	42.7	0.22	48.9	0.54	47.4	0.28
Women (%)	30.0		4.80		0.25		0.40	
Education (%)								
Primary	7.7		5.6		13.2		10.8	
Middle	83.6		78.0		80.9		85.7	
College	8.7		16.4		5.9		3.5	
Occupation (%)								
Blue-collar	91.8		88.3		95.3		96.0	
White-collar	8.2		11.7		4.7		4.0	
Income (%)								
<500 RMB/month	30.6		36.3		31.1		41.7	
500–1000 RMB/month	51.0		36.4		46.8		37.1	
>1000 RMB/month	18.5		27.3		22.1		21.3	
Smoking status (%)								
Never	69.9		28.5		19.4		12.3	
Past smoker	5.9		8.6		7.6		6.5	
Current smoker	24.2		63.0		73.0		81.2	
Hypertension (%)								
No	18.1		15.5		9.2		9.0	
Prehypertension	28.8		35.5		28.3		25.4	
Hypertension	53.1		49.1		62.5		65.5	
Diabetes (%)								
No	64.9		68.0		61.3		59.3	
Prediabetes	22.5		22.9		25.4		27.0	
Diabetes	12.6		9.1		13.3		13.7	
Physical activity (%)								
Never	7.2		10.5		6.9		14.0	
Every time more than 20 min								
<4 times/week	79.5		75.6		76.0		71.1	
≥4 times/week	13.3		13.9		17.2		14.9	
BMI (kg/m <sup>2</sup> )	24.8	0.05	24.9	0.08	24.5	0.18	24.7	0.09
HDL-cholesterol (mmol/l)	1.58	0.01	1.60	0.01	1.66	0.02	1.74	0.01
LDL-cholesterol (mmol/l)	2.11	0.01	2.16	0.01	2.15	0.04	2.20	0.02
TAG (mmol/l)	1.44	0.02	1.47	0.03	1.41	0.07	1.69	0.03
Urate (μmol/l)	277	1.07	305	1.72	306	4.06	307	2.15
Sleep quality score	1.63	0.02	1.80	0.03	1.69	0.06	1.85	0.03

\*Values are mean (SE) adjusted for age and sex, or percentages.

†Participants were categorised into non-drinkers, light drinkers (women: 0–0.4 serving/d; men: 0–0.9 serving/d), moderate drinkers (women: 0.5–1.0 serving/d; men: 1.0–2.0 servings/d) and heavy drinkers (women: >1.0 servings/d; men: >2.0 servings/d).

### Measurement of potential covariates

Potential covariates were selected because they either had association with alcohol consumption or had association with sleep quality<sup>(6,15,23)</sup>. Information on age, sex, occupation (two groups: blue-collar/white-collar), education level (three groups: primary, middle, and college or higher), income level (three groups: <500, 500–1000 and >1000 RMB/month), smoking status and physical activity was collected via a questionnaire in 2006 and repeated every 2 years<sup>(24)</sup>. The smoking status of participants was categorised into three groups: never, past smoker and current smoker. Physical activity, with a definition of lasting at least 20 min, was divided into three groups, including never, <4 times/week and ≥4 times/week<sup>(24)</sup>.

The weight and height of each participant were measured by trained study staff (nurses and physicians). BMI (in kg/m<sup>2</sup>) was calculated by dividing body weight (in kg) by the square of height (in m<sup>2</sup>). BMI was then divided into categories of normal weight (<24 kg/m<sup>2</sup>), overweight

(24–27.9 kg/m<sup>2</sup>) and obese (≥28 kg/m<sup>2</sup>)<sup>(25,26)</sup>. Brachial blood pressure was measured twice with participants in the seated position using a mercury sphygmomanometer. The average of two measurements of systolic and diastolic blood pressure was calculated. Participants were assigned to the hypertension group if their systolic or diastolic blood pressure was elevated (> 140 or 90 mmHg, respectively) or they self-reported a history of hypertension<sup>(27)</sup>. Prehypertension was defined as systolic blood pressure between 120 and 139 mmHg or diastolic blood pressure between 80 and 90 mmHg<sup>(27)</sup>.

Fasting blood samples were collected from participants at the Kailuan Hospital to measure the concentration of glucose, HDL-cholesterol, LDL-cholesterol, TAG and urate using an autoanalyzer (Hitachi 747; Hitachi). Diabetes was defined as a self-reported history of a diagnosis of diabetes, a fasting blood glucose ≥7.0 mmol/l or the use of an oral hypoglycaemic agent<sup>(28)</sup>. Prediabetes was defined as a fasting blood glucose concentration ranging from a 5.6 to 6.9 mmol/l<sup>(28)</sup>.

**Statistical analysis**

The statistical analyses were conducted using SAS software, version 9.4 (SAS Institute Inc.). The significance level of the two-sided hypothesis tests was 0.05.

We used a linear regression model to calculate the mean differences and 95 % CI for overall sleep quality score across different alcohol intake categories, adjusting for age, sex, education level, occupation, income level, smoking status, hypertension, diabetes, physical activity, BMI and plasma concentrations of TAG, HDL-cholesterol, LDL-cholesterol and urate. Three models were conducted. Model 1 was adjusted for age and sex. Model 2 was additionally adjusted for education level, occupation, income level, smoking status, hypertension, diabetes, physical activity and BMI. Model 3 was further adjusted for some potential intermediators in the alcohol-sleep pathway, including plasma concentrations of TAG, HDL-cholesterol, LDL-cholesterol and urate although we were aware of the possibility of over-adjustment.

A logistic regression model was conducted to calculate the adjusted OR and 95 % CI for the odds of having each individual sleep disorder (insomnia, daytime sleepiness, sleep duration and snoring), based on alcohol intake levels, with adjustment for the aforementioned covariates.

**Results**

Participants (*n* 11 905) with higher alcohol intake were more likely to be men, current smokers, have a low education level, a low-income level (<500 and 500–1000 RMB/month), blue-collar occupation, hypertension and higher concentrations of HDL-cholesterol, LDL-cholesterol, TAG and urate (Table 1).

Higher alcohol consumption in 2006 was associated with worse overall sleep quality ( $P_{\text{trend}} < 0.001$ , Table 2), after adjusting for sex, age, education level, occupation, income level, smoking status, hypertension, diabetes, physical activity and BMI (Table 2). Similar results were found after including lipid profiles and urate concentrations (Table 2). Similar patterns were observed when we used the 2012 alcohol intake as the exposure (Table 2). We observed significant associations between higher alcohol consumption and higher odds of having short sleep duration, and snoring (Table 3). Consistently, alcohol was also significantly associated with increased odds of having OSA (Table 3).

Individual alcoholic beverages were further studied. Participants who consumed hard liquor, but not wine and beer, were more likely to have worse overall sleep quality when compared with people who did not consume liquor (adjusted mean differences = 0.18; 95 % CI: 0.13, 0.23 with  $P < 0.001$ ) (Table 4).

**Discussion**

In this community-based study with 11 905 participants, we observed a significant linear association between

**Table 2** The adjusted mean differences and 95 % CI of sleep quality score, according to alcohol consumption status\* †

	None	Light		Moderate		Heavy		$P_{\text{trend}}$
		Mean	95 % CI	Mean	95 % CI	Mean	95 % CI	
Alcohol consumption in 2006								
No	6564	3168		408		1765		
Model 1 ‡	0 (Ref.)	0.15	0.09, 0.22	0.10	-0.05, 0.24	0.24	0.17, 0.32	<0.001
Model 2 §	0 (Ref.)	0.15	0.09, 0.22	0.10	-0.05, 0.24	0.24	0.17, 0.32	<0.001
Model 3 †	0 (Ref.)	0.15	0.09, 0.22	0.10	-0.05, 0.24	0.24	0.17, 0.32	<0.001
Alcohol consumption in 2012								
Model 2 §	0 (Ref.)	0.27	0.21, 0.33	0.39	0.29, 0.49	0.54	0.31, 0.76	<0.001
Model 3 †	0 (Ref.)	0.27	0.21, 0.33	0.39	0.29, 0.49	0.54	0.31, 0.76	<0.001

Ref., Reference.

\*Participants were categorised into non-drinkers, light drinkers (women: 0–0.4 servings/d; men: 0–0.9 servings/d), moderate drinkers (women: 0.5–1.0 servings/d; men: 1.0–2.0 servings/d) and heavy drinkers (women: > 1.0 serving/d; men: > 2.0 servings/d).

†Adjusted for age, sex, education level (primary, middle or college and higher), occupation (blue-collar/white-collar), income level (<500, 500–1000 or > 1000 RMB/month), smoking status (never, past or current smoker), hypertension (no, prehypertension or hypertension), diabetes (no, prediabetes or diabetes), physical activity (never, <4 times/week or ≥4 times/week) BMI (<24, 24–28 or ≥28 kg/m<sup>2</sup>) and plasma concentrations of TAG (quartiles), LDL-cholesterol (quartiles) and urate (quartiles).

‡Adjusted for age, sex.

§Adjusted for age, sex, education level (primary, middle or college and higher), occupation (blue-collar/white-collar), income level (<500, 500–1000 or > 1000 RMB/month), smoking status (never, past or current smoker), hypertension (no, prehypertension or hypertension), diabetes (no, prediabetes or diabetes), physical activity (never, <4 times/week or ≥4 times/week) and BMI (<24, 24–28 or ≥28 kg/m<sup>2</sup>).

**Table 3** The adjusted OR and 95 % CI of sleep disorders, according to alcohol consumption status in 2006\*,†

	None	Light		Moderate		Heavy		<i>P</i> <sub>trend</sub>
		OR	95 % CI	OR	95 % CI	OR	95 % CI	
No	6564	3168		408		1765		
Insomnia	1 (Ref.)	1.25	1.06, 1.47	1.10	0.78, 1.55	1.04	0.85, 1.28	0.61
Daytime sleepiness	1 (Ref.)	1.10	0.76, 1.58	0.67	0.27, 1.70	1.13	0.73, 1.76	0.71
Shorter sleep duration	1 (Ref.)	1.13	0.98, 1.31	0.99	0.72, 1.36	1.31	1.09, 1.57	0.007
Longer sleep duration	1 (Ref.)	0.89	0.78, 1.02	0.85	0.63, 1.15	0.86	0.72, 1.02	0.07
Snoring	1 (Ref.)	1.34	1.21, 1.49	0.97	0.77, 1.21	1.38	1.22, 1.57	<0.001
Obstructive sleep apnea	1 (Ref.)	1.17	1.02, 1.35	1.15	0.87, 1.53	1.47	1.25, 1.73	<0.001

Ref, Reference.

\*Participants were categorised into non-drinkers, light drinkers (women: 0–0.4 servings/d; men: 0–0.9 servings/d), moderate drinkers (women: 0.5–1.0 servings/d; men: 1.0–2.0 servings/d) and heavy drinkers (women: >1.0 serving/d; men: >2.0 servings/d).

†Adjusted for age, sex, education level (primary, middle or college and higher), occupation (blue-collar/white-collar), income level (<500, 500–1000 or >1000 RMB/month), smoking status (never, past or current smoker), hypertension (no, prehypertension or hypertension), diabetes (no, prediabetes or diabetes), physical activity (never, <4 times/week or ≥4 times/week) BMI (<24, 24–28 or ≥28 kg/m<sup>2</sup>), plasma concentrations of TAG (quartiles), LDL-cholesterol (quartiles), HDL-cholesterol (quartiles) and urate (quartiles).

**Table 4** The adjusted mean difference and 95 % CI of sleep quality by types of alcoholic beverage\*,†,‡

	No	Yes		<i>P</i>
		Mean	95 % CI	
No	7657	3953		
Hard liquor	0 (Ref.)	0.18	0.13, 0.23	<0.001
No	9782	1828		
Beer	0 (Ref.)	0.02	–0.05, 0.08	0.22
No	11 483	126		
Wine	0 (Ref.)	–0.07	–0.28, 0.14	0.87

Ref, Reference.

\*Participants were categorised into beer drinkers and non-beer drinkers (non-drinkers and other alcoholic beverage drinkers), wine drinkers and non-wine drinkers (non-drinkers and other alcoholic beverage drinkers), liquor drinkers and non-liquor drinkers (non-drinkers and other alcoholic beverage drinkers).

†Adjusted for age, sex, education level (primary, middle or college and higher), occupation (blue-collar/white-collar), income level (<500, 500–1000 or >1000 RMB/month), smoking status (never, past or current smoker), hypertension (no, prehypertension or hypertension), diabetes (no, prediabetes or diabetes), physical activity (never, <4 times/week or ≥4 times/week), BMI (<24, 24–28 or ≥28 kg/m<sup>2</sup>), plasma concentrations of TAG (quartiles), LDL-cholesterol (quartiles), HDL-cholesterol (quartiles) and urate (quartiles).

‡'No' group with participants who did not drink any alcohol or did not drink the indicated type of alcoholic beverage; 'Yes' group with participants who drank the indicated type of alcoholic beverage.

alcohol intake and poor overall sleep quality 6 years later. Specifically, individuals with a higher alcohol consumption had higher odds of experiencing short sleep duration, snoring and OSA.

Our findings are consistent with the literature, although previous studies were generally conducted among populations suffering from alcoholism or alcohol abuse<sup>(11,29,30)</sup>. A recent large-scale study, including 151 567 adults with problematic alcohol consumption and post-traumatic stress disorder symptoms, found an inverse association between alcohol consumption and sleep quality<sup>(11)</sup>. Another recent study, investigating the relationship between AUD, with and without Korsakoff's syndrome and sleep quality, showed that the sleep complaints were prevalent among the participants with AUD<sup>(29)</sup>. A longitudinal study with 696 adolescents (12–19 years old) found that insomnia

and sleep problems may be a consistent problem for adolescents with AUD and may also be a risk factor for the development of AUD in adolescents<sup>(30)</sup>.

We observed a significant positive dose–response association between alcohol consumption and the odds of snoring (including OSA), and short sleep duration. The association between alcohol and snoring/OSA may be explained by the known effects of alcohol on the respiratory system, including upper airway narrowing, reducing hypoglossal muscle activity, increased nasal resistance and reduced ventilatory responses to asphyxia, which may increase the risk of snoring/OSA<sup>(5,7,8)</sup>. Alcohol may also impact sleep duration via circadian rhythm dysregulation, increased physiological arousal and competition with neuroimmune and neurotransmitter systems (i.e., Ca current in the thalamus, adenosine) caused by the metabolism of ethanol<sup>(1,2,6,31)</sup>. Alcohol may also directly disrupt the sleep cycle, which could reduce sleep duration<sup>(1,2,6,31)</sup>. Although associations between alcohol consumption and insomnia, daytime sleepiness and longer sleep duration did not reach significance, trends existed that demonstrated higher odds of these sleep disorders with increased alcohol intake, which is consistent with previous studies<sup>(6)</sup>.

We found a significant association between higher consumption of hard liquor, but not beer or wine, and poor overall sleep quality. A majority of the Kailuan Study's participants report drinking hard liquor, which is consistent with the alcohol intake culture in northern China. The significant association between hard liquor and sleep quality, not present for beer and wine, may be explained by the higher concentration of alcohol in hard liquor compared with beer and wine (5.0 g ethanol for 100 g beer, 12.0 g ethanol for 100 g wine, 40.0 g ethanol for 100 g hard liquor). The small sample sizes in the groups of beer- and wine-drinkers may also have contributed to the non-significant results.

Our study had several limitations. Sleep status was only assessed in 2012, so we cannot assess the effects of the alcohol on subsequent change in sleep quality. Therefore, our



study should be considered a cross-sectional analysis. However, using alcohol consumption at different time points (e.g., assessed 6 years prior to sleep assessment or at the same time as sleep assessment) generated similar results. Misclassification of the exposure (alcohol intake) and outcome (sleep quality) is another concern, because the alcohol consumption and sleep status data were collected via self-report questionnaires. Residual confounding is another limitation. For example, the Kailuan Study did not collect data on depression until 2016<sup>(32)</sup>, which was thus not adjusted in the current analysis. However, the prevalence of physician-diagnosed depression (0–13%) was low among the Kailuan participants<sup>(32)</sup>, although it could be underdiagnosed. Further, the Kailuan Study is located in Tangshan city, China, and included a large portion of individuals with low-to-middle education levels and low-income, which may limit the generalisability of the findings to other ethnic groups.

In conclusion, greater alcohol consumption was associated with poorer sleep quality and higher odds of having snoring and short sleep duration, in a dose–response manner. Our findings warrant replication in a prospective study conducted in populations with diverse cultural backgrounds and objective assessment of sleep status.

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