CHEST

Alcohol Consumption and Small Airways Obstruction

To the Editor:

Small airways obstruction (SAO) is characterized by inflammation, hypersecretion of mucus, and airway remodeling, and it is an important contributor to airflow limitation in both asthma[1] and COPD.[2] SAO can be detected early, before irreversible airflow limitation in the larger airways becomes evident. To mitigate the risk of SAO, it is essential to gain a deeper understanding of its causes.

Alcohol consumption is a major risk factor for several noncommunicable diseases, but its association with asthma and COPD remains uncertain. Experimental evidence, however, suggests that it may contribute to chronic inflammation of the airways.[3] In this study, we used data from the UK Biobank to assess the association of isolated SAO with alcohol consumption.

Methods

The UK Biobank is a cohort of approximately 500,000 adults (≥ 40 years), recruited across England, Wales, and Scotland, between 2006 and 2010.[4] Lung function was assessed prior to bronchodilator using a Vitalograph Pneumotrac 6800 spirometer. To obtain 2 reproducible maneuvers, a maximum of 3 attempts were permitted. In this study, we considered only data of 256,546 participants who had at least 2 spirograms with no cough, back extrapolated volume (< 5% FVC, or > 5% but < 150 mL), reproducible FEV_1, and FVC and a forced expiratory time ≥ 6 seconds on the best curve (curve with highest FEV_1 and FVC).[5] We defined isolated SAO as an FEV_3/FEV_6 ratio less than the lower limit of normal (LLN), with no evidence of airflow obstruction ($FEV_1/FEV_6 \geq$ LLN).[5] The LLN was calculated based on reference equations for European American individuals from the third US National Health and Nutrition Examination Survey.[6] This study was approved by the UK National Research Ethics Service (11/NW/0382, 21/NW/0157). Each participant provided written informed consent.

Participants provided information on their alcohol consumption patterns, including the frequency of intake and types of beverages consumed. Based on this information, we converted the reported consumption of each type of beverage into standard alcohol units using the following conversion factors: beer (2 units), fortified red wine (1 unit), red wine (1.5 units), spirits (1 unit), champagne (1.5 units), and other alcoholic beverages (1.5 units).[7] We calculated total alcohol consumption by summing the number of units across all beverage types[7] and converting the total into grams per week, using the standard conversion of 1 unit equivalent to 8 g pure alcohol. After excluding participants with missing alcohol consumption data, missing covariates, or airflow obstruction, the sample size for analysis was n = 168,793.

We used logistic regression models to assess the association between alcohol consumption and isolated SAO. To investigate whether the relationship was nonlinear, we fitted restricted cubic splines with knots at the 33rd, 50th, 66th, and 90th percentiles of weekly alcohol consumption distribution. The models were adjusted for age, sex, ethnicity, assessment center, smoking status, pack-years, healthy diet score, Townsend deprivation index, employment status, education, and weight. We further conducted a sensitivity analysis excluding individuals who currently smoke. We used R, version 4.4.3, and SAS 9.4 software to conduct data analysis, and considered 2-sided *P* values < .05 to be significant.

Results

Among 168,793 study participants, the proportion of high alcohol consumption (> 400 g/week in male participants; > 280 g/week in female participants) was greater among male than female participants (5.8% vs 3.2%). A total of 12,175 participants (7.2%) had isolated SAO.

Higher alcohol consumption was associated with 11% greater odds of isolated SAO (adjusted OR = 1.11; 95% CI, 1.02-1.21) compared with low consumption

TABLE 1] Association Between Alcohol Consumption and iSAO

	OR (95%CI)[a]		
	Low-Risk Drinking	Moderate-Risk Drinking	High-Risk Drinking
With/without iSAO, No.	7,511/96,767	3,995/53,217	669/6,634
Model 1	1.00 (Ref.)	1.05 (1.01-1.10)	**1.38 (1.27-1.50)**
Model 2	1.00 (Ref.)	0.98 (0.94-1.02)	**1.10 (1.01-1.19)**
Model 3	1.00 (Ref.)	0.98 (0.94-1.02)	**1.11 (1.02-1.21)**
Model 4	1.00 (Ref.)	0.98 (0.94-1.03)	**1.11 (1.02-1.21)**

The logistic regression models were used to assess the association between alcohol consumption and iSAO. Model 1: age, sex, ethnicity, assessment center. Model 2: model 1 + smoking status, pack-years. Model 3: model 2 + healthy diet score, and weight. Model 4: model 3 + Townsend deprivation index, employment status, and education. Boldface identifies $P < .05$. iSAO = isolated small airways obstruction.
[a]Participants were classified into low-risk (≤ 14 units/week), moderate-risk (15-34 units/week for female individuals; 15-49 units/week for male individuals), and high-risk drinking (≥ 35 units/week for female individuals; ≥ 50 units/week for male individuals), according to UK guidelines.

(≤ 112 g/week) (Table 1). The dose-response relationship between alcohol consumption and isolated SAO risk is shown in Figure 1. Overall, we observed a significant nonlinear relationship between alcohol consumption and isolated SAO, with the odds of isolated SAO increasing notably at 473 g/week (P for nonlinear $< .05$; Fig 1A). Sex did not modify this association. After excluding individuals who smoke from the analysis, the dose-response relationship between alcohol consumption and isolated SAO became clearer and linear (Fig 1D-F).

Discussion

To the best of our knowledge, this is the first study to examine the association between alcohol consumption and isolated SAO, which makes it hard to compare with previous studies. However, several studies have shown that alcohol consumption may have a role in lung function impairment.[8] Taken together, these findings indicate that alcohol consumption, especially at high levels, may contribute to impaired lung function and increased risk of airflow limitation, typical of asthma and COPD. Given that alcohol consumption and

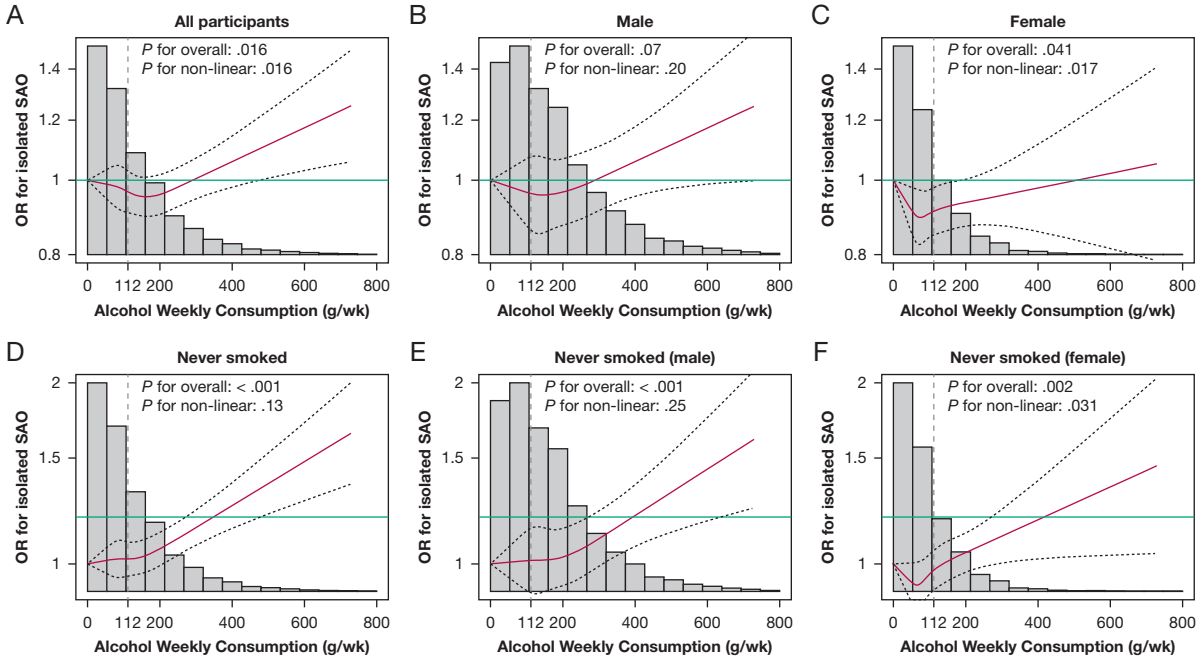

Figure 1 – *A-F, Dose-response association between alcohol consumption and isolated small airways obstruction. A, All participants; B, male; C, female; D, people who never smoked; E, male individuals who never smoked; F, female individuals who never smoked. OR (red solid lines) and 95% CIs (area between dashed lines) adjusted for age, sex, ethnicity, assessment center, smoking status, pack-years, healthy diet score, Townsend deprivation index, employment status, education, and weight. The vertical gray dashed line corresponds to the Chief Medical Officer's cutoff for low-risk drinking. SAO = small airways obstruction.*

tobacco smoking are highly correlated, we excluded individuals who smoke, and the observed association was still present, suggesting that it is independent of smoking.

The physiologic mechanisms underlying the potentially harmful effects of heavy alcohol consumption on isolated SAO are complex and are not fully understood. One possible pathogenic mechanism is that alcohol may significantly reduce levels of the antioxidant glutathione in the lungs,[9] thereby increasing susceptibility to injury (eg, higher risk of infection), which in turn might contribute to small airways impairment. Additionally, alcohol could impair innate respiratory immunity by disrupting mucociliary clearance, reducing macrophage function, and weakening the alveolar barrier.[10]

The strengths of this study include its large sample size, the use of the same model of spirometer and standardized spirometry protocol. This study also has several limitations. First, this is a cross-sectional study, which limits our ability to draw conclusions in terms of causality because of potential reverse causation. However, it is unlikely that having isolated SAO, which is underdiagnosed, would lead to increased consumption of alcohol. Measurement of alcohol consumption was based on self-reporting, which could be subject to recall bias. In addition, it is possible that we did not account for high-risk behaviors related to alcohol consumption.

In conclusion, we found that individuals with heavy alcohol consumption are more likely to show isolated SAO. Because this is a common trait in asthma and early stages of COPD, further studies are warranted to understand the biologic mechanisms underlying our findings. It is also important to raise awareness among both clinicians and the general population of the potential role of alcohol in the development of obstructive lung diseases.

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