Research article

**Beverage specific alcohol intake in a population-based study: Evidence for a positive association between pulmonary function and wine intake**

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**Abstract**

**Background:** Lung function is a strong predictor of cardiovascular and all-cause mortality. Previous studies suggest that alcohol exposure may be linked to impaired pulmonary function through oxidant-antioxidant mechanisms. Alcohol may be an important source of oxidants; however, wine contains several antioxidants. In this study we analyzed the relation of beverage specific alcohol intake with forced expiratory volume in one second (FEV1) and forced vital capacity (FVC) in a random sample of 1555 residents of Western New York, USA.

**Methods:** We expressed pulmonary function as percent of predicted normal FEV1 (FEV1%) and FVC (FVC%) after adjustment for height, age, gender and race. To obtain information on alcohol intake we used a questionnaire that reliably queries total alcohol and beverage specific recent (past 30 days) and lifetime alcohol consumption. Results: Using multiple linear regression analysis after adjustment for covariates (pack-years of smoking, weight, smoking status, education, nutritional factors and for FEV1%, in addition, eosinophil count), we observed no significant correlation between total alcohol intake and lung function. However, we found positive associations of recent and lifetime wine intake with FEV1% and FVC%. When we analyzed white and red wine intake separately, the association of lung function with red wine was weaker than for white wine.

**Conclusion:** While total alcohol intake was not related to lung function, wine intake showed a positive association with lung function. Although we cannot exclude residual confounding by healthier lifestyle in wine drinkers, differential effects of alcoholic beverages on lung health may exist.

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Background

Impaired pulmonary function is a strong predictor of cardiovascular and all-cause mortality in the general population independent of smoking [1–4]. The factors that influence pulmonary function are not completely understood and oxidant exposure could play a negative role [5–7]. Support for this hypothesis comes from studies that report a positive association between antioxidant vitamin status and lung function [8–11]. Several studies have indicated that alcohol exposure may contribute to oxidative burden and, therefore, it may be linked to impaired pulmonary function through oxidant-antioxidant mechanisms [12–15]. However, while alcohol itself can act as an oxidant, wine has antioxidant properties [12,13,16,17].

An early report by Cohen et al. [18] did not show an effect of total alcohol intake on lung function, but a number of cross-sectional studies subsequently have shown high alcohol intake to be negatively associated with lung function and positively related to the occurrence of respiratory symptoms [19–24]. However, several recent studies failed to show a negative association of alcohol intake with lung function or respiratory symptoms [25–27]. Thus, the evidence is contradictory and oxidants or antioxidants associated with different alcoholic beverages may be responsible for these conflicting results [12–14,28]. Only one study has investigated the association between beverage-specific alcohol intake and pulmonary function. In that study, Cohen et al. [18] did not find beverage-specific effects on pulmonary function. However, there is evidence that alcoholic beverages could differ in their effects on lung health due to differences in their constituents from a study linking beverage-specific alcohol intake to lung cancer risk [29]. In addition, no study has investigated lifetime alcohol intake and white and red wine intake separately in relation to pulmonary function. Therefore, the goal of this study was to describe the relation of beverage-specific alcohol intake with pulmonary function (FEV1 and FVC) in a general population sample. In order to investigate whether effects of recent alcohol intake differ from long term effects we considered both recent as well as lifetime beverage specific alcohol intake in this analysis.

Materials and Methods

We report here on data from a general-population sample drawn from Erie and Niagara Counties in New York State, USA, between September 1995 and December 1998. A detailed description of the study design, participant recruitment and methodology has been reported previously [11]. The ethics review board of the University at Buffalo, New York, USA, approved the study.

Study population

In brief, we utilized New York State Department of Motor Vehicles and Health Care Finance Association lists to randomly select participants aged 35–79 years. Of the 4946 initially contacted eligible subjects 2537 (1,322 female and 1,215 male) participated (51.3 percent). The following exclusion criteria applied to the current analysis: race other than Caucasian or African-American (n = 33), missing information on diet (n = 111), missing information on height, weight, smoking status, laboratory values or education (n = 177), missing pulmonary function tests (n = 250), unacceptable or not reproducible pulmonary function tests (n = 108), a history of COPD, asthma or pulmonary fibrosis (n = 249) or missing information on alcohol intake (n = 54). The remaining 1555 participants (814 women and 741 men) are included in this report. The excluded participants for whom information was available were comparable to the included participants in regards to gender and race. We did not find a statistically significant difference between included and excluded participants for age, height and weight, but as a result of the higher prevalence of asthma and COPD excluded participants had lower values of FEV1 and FVC.

Interview and Examination

The examination included an in-person interview about lifestyle habits, a self-administered questionnaire, anthropometric measurements and spirometry. Between 6:30 and 9:30 am we obtained spirometric measurements from participants, standardized according to 1994 American Thoracic Society guidelines [30]. To derive forced expiratory volume in one second (FEV1) and forced vital capacity (FVC) prediction equations we used multiple linear regression with values obtained from lifelong nonsmokers who did not report a history of chronic lung disease for men (n = 277) and women (n = 418) separately. We chose the model that explained the greatest fraction of variability (R2) in lung function.

We obtained the following equations for men,

\[
\text{predicted FEV1} = -0.550 - 0.0359 \times \text{age (years)} + 3.603 \times \text{height (m)} - 0.478 \times \text{race}
\]

\[
\text{predicted FVC} = -2.229 - 0.0382 \times \text{age (years)} + 5.210 \times \text{height (m)} - 0.739 \times \text{race}
\]

and for women,

\[
\text{predicted FEV1} = -0.184 - 0.0298 \times \text{age (years)} + 2.829 \times \text{height (m)} - 0.309 \times \text{race}
\]

\[
\text{predicted FVC} = -1.413 - 0.0318 \times \text{age (years)} + 4.104 \times \text{height (m)} - 0.514 \times \text{race}
\]
with race as a dummy variable (Caucasian = 0, African-American = 1). We then calculated FEV₁ and FVC in percent of the predicted value (FEV₁% and FVC%) adjusted for age, gender, height and race for all participants [11].

**Alcohol intake**

We obtained information about alcohol intake with a computer assisted in-person interview [31]. Interviewers underwent extensive training and standardization. Alcohol consumption was assessed for the past 30 days and during participants' lifetimes [32,33]. We defined participants who reported consumption of less than 12 drinks during their lifetime as lifetime abstainers (never drinkers) and those who reported 12 or more drinks during their lifetime but never consumed alcoholic beverages in the past 30 days as former drinkers (non-current drinkers).

We asked participants who drank during the past 30 days which of four major types of alcoholic beverages beer, wine, wine cooler, and hard liquor they had drunk at least once. Participants defined their usual drink size in ounces for each beverage they drank using bottles of various sizes and sample glasses with drink sizes marked on the side to improve the accuracy of their estimates. Participants were then asked how often they drank during the last 30 days, and those who drank less often than once a week were asked how many drinks they usually had on a day when they drank alcohol. Respondents who drank once a week or more often were asked about their pattern of drinking – how many Fridays in the past 30 days did they drink, how many drinks did they usually have on a Friday when they drank, and what beverage(s) did they drink? These questions were repeated for Saturdays, Sundays, and weekdays. We also asked quantity-frequency questions for times participants drank more than usual. This data was used to estimate the number of drinks consumed of each beverage over the past 30 days, which was then multiplied by drink size to get beverage-specific ounces of alcohol consumed. We then calculated beverage-specific grams of alcohol using the following conversion factors: 0.045, 0.121, 0.04, and 0.409 grams of alcohol per ounce, for beer, wine, wine cooler and hard liquor, respectively.

We used the Cognitive Lifetime Drinking History (CLDH) to obtain information on lifetime alcohol intake [31–33]. Prior to the interview, participants completed a lifetime events calendar on which they recorded the date and their age when significant events in their lives occurred. This calendar was used during the interview to help them remember what they were doing during different periods of their lives and whether drinking alcohol was involved. Participants reported how old they were when they started drinking alcohol regularly – at least once a month for six months and when their drinking patterns changed. This information was used to define intervals during their lives when drinking patterns were fairly homogeneous. For each interval, participants were asked questions on drinking pattern comparable to those asked for the past 30 days and asked to estimate what percentage of the drinks consumed during that interval came from the specific beverages consumed during that interval. This information was used to estimate beverage-specific grams of alcohol consumed in the interval, and these estimates were summed across intervals to arrive at lifetime totals. Total alcohol intake was expressed in kg during participants’ lifetimes.

We have previously demonstrated construct validity of the questionnaires. In a study involving 147 subjects [32], we compared the average daily volume of alcohol consumed during the 12–24 months prior to the interview as estimated from the alcohol questionnaire with those estimated from two different food frequency questionnaires (FFQ), the Health Habits and History Questionnaire and the Harvard Semiquantitative Food Frequency Questionnaire. FFQs have been shown to produce valid estimates of alcohol intake, with correlations of about 0.5–0.9. In this study, the CLDH compared favorably to the FFQs with correlations between 0.7 and 0.8, and therefore we can infer reasonable validity. Because the FFQs query intake over a one year period, we did not estimate 30 day intake in that study. Furthermore, the repeatability of the CLDH over an average two week period was excellent (r = 0.84) indicating that this questionnaire performs well in measurement of short term alcohol intake.

**Statistical methods and analysis**

We examined the distributions of the continuous variables for all relevant variables to determine if they were normally distributed and calculated mean values and standard deviation (SD). We also performed descriptive analyses separating drinkers by beverage types consumed and compared drinkers of specific beverages but there were only few participants who consumed one type of alcoholic beverage exclusively. For the analyses related to alcohol intake we used recent intake (last 30 days) and lifetime alcohol intake with ounces converted into metric units. To investigate the association between the alcohol intake variables we calculated Pearson’s correlation coefficients. For both total and white and red wine separately we observed weak correlations (r < 0.2) with beer and liquor intake for recent and lifetime total intake. The correlation between red and white wine intake were high for both recent (r = 0.8) and lifetime (r = 0.5) intake. However, the correlations between recent and lifetime alcohol intake were only weak to moderate ranging from r = 0.13 for liquor, r = 0.17 for total alcohol, r = 0.29 for wine to r = 0.37 for beer intake. These correlation coefficients did not differ significantly between men and women.
Table 1: Characteristics of participants

| Variable (unit)         | Value (SD) |
|-------------------------|------------|
| Age (years)             | 59.9 (10.7) |
| BMI (kg/m²)‡           | 28.2 (5.2)  |
| FVC% (liter)            | 3.82 (1.01) |
| FEV₁ (liter)            | 2.90 (0.80) |
| FVC% (liters)           | 98.4 (15.2) |
| FEV₁, † (liters)        | 96.5 (16.4) |
| Women (%)               | 52.3%      |
| African-American (%)    | 6.9%       |
| Smoking                 |            |
| Never-Smoker (%)        | 42.8%      |
| Ex-Smoker (%)           | 43.5%      |
| Current-Smoker (%)      | 13.7%      |
| Pack-years of smoking   | 14.8 (23.0)|
| Alcohol Intake          |            |
| Never Drinkers          | 3.0%       |
| Current Drinkers        | 68.1%      |
| Non-Current Drinkers    | 28.8%      |
| Past 30 days alcohol intake |         |
| Total Alcohol (g/day)§  | 8.2 (17.8) |
| Beer (g/day)§           | 3.0 (8.8)  |
| White Wine (g/day)§     | 1.3 (5.0)  |
| Red Wine (g/day)§       | 1.0 (3.4)  |
| Fortified Wine (g/day)§ | 0.03 (0.5) |
| Liquor (g/day)§         | 2.8 (13.6) |
| Lifetime alcohol intake |            |
| Total Alcohol (kg)      | 210.6 (600.6)|
| Beer (kg)               | 98.7 (215.3)|
| White Wine (kg)         | 16.6 (127.1)|
| Red Wine (kg)           | 10.4 (37.6)|
| Fortified Wine (kg)     | 2.7 (35.5) |
| Liquor (kg)             | 81.6 (495.0)|

†SD, standard deviation, for categorical variables prevalence is given
‡BMI, body mass index; FEV₁, forced vital capacity in one second in percent of predicted; FVC%, forced vital capacity in percent of predicted
§ in the 30 days prior to the interview

The principal analysis we used was multiple linear regression analysis including all 1555 subjects. The total and beverage specific alcohol intake variables were not normally distributed and, therefore, we performed a logarithmic transformation. Transformation was based on Ig10 (alcohol intake +1), where alcohol intake resembles cur- riculous transformation. Because eosinophil count, education, smoking status and cumulative tobacco smoke exposure in pack-years of smoking predict FEV₁ % with the largest variance explained and therefore, we included these variables in the baseline model [11]. Because eosinophil count was not significantly correlated with FVC% only total pack years of smoking, smoking sta-

tus, education and weight were included in the baseline model predicting FVC%. In addition, we previously reported that several serum and dietary antioxidant vitamins were correlated with pulmonary function [11,34]. Therefore, we included serum antioxidant vitamins in the regression models for FEV₁ % (vitamin E, vitamin C, lutein/zeaxanthin, β-cryptoxanthin and retinol) and FVC% (vitamin E, vitamin C, β-cryptoxanthin and lutein/zeaxanthin).

To define statistical significance we used the conventional level of p < 0.05. However, we determined that interaction terms would be important if the level of significance was p < 0.1. We investigated interaction by including interaction terms of beverage specific alcohol intake, smoking status (never, former, current), gender and other covariates and stratification, but we did not observe statistically significant interactions. We also examined the regression models for multicollinearity. Significant collinearity was present only for the analyses that included both white and red wine intake. For the analyses we utilized the Statistical Package for Social Sciences and SPLUS.

Results

Demographic characteristics, spirometry and alcohol intake

Table 1 shows the characteristics of the study participants included in this analysis. Mean age was 59.6 years and, based on mean body mass index, participants tended to be overweight. The study population included slightly more women than men, approximately 43.6 percent were former smokers and 13.6 percent were current smokers. There were few never drinkers of alcohol and the majority of participants consumed several alcoholic beverages presently. For both recent daily alcohol intake and lifetime alcohol consumption we observed the highest intake for beer, followed by liquor and wine. On average, participants had consumed larger quantities of white wine than red wine. Mean alcohol intake from wine coolers was low and therefore we did not further investigate this beverage.

Demographic characteristics and spirometry by recent beverage specific alcohol intake

Table 2 shows the characteristics of participants by recent beverage specific alcohol intake. Beer only drinkers were younger than other participants, but those in the liquor only group had a higher BMI than those in the other groups. The beer only group included predominantly men while the groups of wine only, liquor only and recent abstainers included more women than men. The education level was highest in the groups with only wine intake and with mixed drinking pattern. Cumulative tobacco smoke exposure was highest in the beer drinker group and lowest among wine drinkers. Recent average alcohol intake was highest among those with a mixed drinking pattern and
Nutritional status indicators showed that wine drinkers had the highest levels of serum antioxidant vitamins. Both mean FEV1% and FVC% (corrected for age, weight and gender) were highest among participants who consumed various alcoholic beverages or wine only.

**Regression analysis of recent alcohol intake on FEV1% and FVC%**

Table 3 summarizes the results of multiple linear regression analysis for recent total alcohol and beverage specific alcohol intake after inclusion of all covariates in the base-line model. Multiple linear regression of FEV1% revealed independent associations with alcohol intake from wine for both FEV1% and FVC% when variables were entered separately. The association attained statistical significance only for white wine when we entered red and white wine intake variables simultaneously. Even after inclusion of other beverage specific alcohol variables, alcohol intake from wine was at least borderline significantly related to FEV1% and FVC%. Alcohol intake from beer and liquor showed little or no association with lung function.

**Regression analysis of lifetime alcohol intake on FEV1% and FVC%**

Next we explored the association between lifetime beverage specific alcohol intake and lung function. Table 4 shows the results of multiple linear regression analysis for lifetime total and beverage specific alcohol intake after adjustment for other covariates. Cumulative alcohol intake from both red wine and white wine was significantly and positively related to both FEV1% and FVC% when these variables were considered separately. Similar to the results for recent alcohol intake the association was stronger for white wine compared with red wine. Total alcohol intake and alcohol intake from beer and liquor were not correlated with pulmonary function. Alcohol intake from wine was significantly associated with lung function even after inclusion of alcohol intake from beer and liquor. The association of alcohol intake from white wine persisted even after inclusion of other alcohol variables in the regression model, but red wine was not significantly related to lung function (data not shown). There was no statistically significant association between lifetime wine intake and lung function after we included recent alcohol intake from wine in the regression models.

**Other models examined**

We did not observe a statistically significant interaction between smoking status and alcohol intake. Because of the possibility that alcohol intake was particularly harmful in those with heavy alcohol consumption (a subgroup of the population that consumed predominantly large quantities of beer and liquor) and because an association between light drinking and lung function could be influenced by heavy drinking, we investigated whether the correlation between lifetime beverage specific alcohol intake and lung function were similar in the group with lower alcohol intake. For this analysis we included participants below the median of total alcohol intake. In this subgroup...
of the study population mean lifetime intake was 20.4 kg for total alcohol, 4.4 kg for alcohol from white wine, 2.6 kg for red wine, 6.0 kg for beer and 6.9 kg for liquor. Using multiple linear regression analysis with adjustment for the known covariates alcohol intake from white wine remained the strongest correlate of FEV\textsubscript{1}%, and FVC% (data

Table 3: Multiple linear regression coefficients for regression of recent (prior month) total alcohol and beverage specific alcohol intake on FEV\textsubscript{1}%, and FVC%

| Variable                     | FEV\textsubscript{1}%† | FVC%‡   |
|------------------------------|-------------------------|---------|
|                              | \( \beta \)              | 95% CI  | \( \beta \) | 95% CI |
| Recent alcohol intake#       |                         |         |         |
| Total alcohol                | 0.689                   | -0.737–2.116 | 0.802 | -0.554–2.157 |
| Total Wine (red and white wine) | 2.027*                 | 0.011–4.044 | 2.240* | 0.307–4.173 |
| White Wine                   | 3.058*                  | 0.603–5.514 | 2.900* | 0.543–5.257 |
| Red Wine                     | 1.211                   | -1.500–3.922 | 1.440 | -1.160–4.040 |
| Beer                         | 0.394                   | -1.396–2.184 | 0.564 | -1.144–2.267 |
| Liquor                       | 0.561                   | -1.411–2.532 | 0.129 | -1.753–2.010 |

† adjusted for smoking status (never, former, current), total pack-years of smoking, weight, education, eosinophil count and serum vitamin E, vitamin C, lutein/zeaxanthin, \( \beta \)-cryptoxanthin and retinol levels. ‡ adjusted for smoking status, total pack-years of smoking, weight, education and serum vitamin E, vitamin C, \( \beta \)-cryptoxanthin and lutein/zeaxanthin levels. * alcohol variables, expressed in gram/day, were log transformed to closer approximate a normal distribution. Transformation is based on \( \lg_{10} \) (alcohol intake + 1), where alcohol intake resembles beverage specific or total intake over past 30 days. *\( p < 0.05 \), **\( p < 0.01 \)

Table 4: Multiple linear regression coefficients for regression of lifetime total alcohol and beverage specific alcohol intake on FEV\textsubscript{1}%, and FVC%,

| Variable                     | FEV\textsubscript{1}%† | FVC%‡   |
|------------------------------|-------------------------|---------|
|                              | \( \beta \)              | 95% CI  | \( \beta \) | 95% CI |
| Lifetime alcohol intake#     |                         |         |         |
| Total alcohol                | 0.001                   | -0.999–1.017 | 0.271 | -0.687–1.228 |
| Total Wine (red and white wine) | 1.607**                 | 0.593–2.621 | 1.267* | 0.292–2.241 |
| White Wine                   | 1.825**                 | 0.660–2.990 | 1.396* | 0.277–2.516 |
| Red Wine                     | 1.277*                  | 0.008–2.546 | 1.262* | 0.044–2.480 |
| Beer                         | -0.387                  | -1.193–0.419 | -0.056 | -0.823–0.711 |
| Liquor                       | -0.042                  | -0.825–0.909 | -0.135 | -0.966–0.697 |
| Lifetime alcohol intake#     |                         |         |         |
| Total Wine (red and white wine) | 1.696**                 | 0.662–2.730 | 1.349** | 0.355–2.343 |
| Beer                         | -0.503                  | -1.007–0.791 | -0.097 | -0.884–0.691 |
| Liquor                       | -0.108                  | -1.007–0.791 | -0.324 | -1.187–0.540 |

† adjusted for smoking status (never, former, current), total pack-years of smoking, weight, education, eosinophil count and serum vitamin E, vitamin C, lutein/zeaxanthin, \( \beta \)-cryptoxanthin and retinol levels. ‡ adjusted for smoking status, total pack-years of smoking, weight, education and serum vitamin E, vitamin C, \( \beta \)-cryptoxanthin and lutein/zeaxanthin levels. * alcohol variables, expressed in kg, were log transformed to closer approximate a normal distribution. Transformation is based on \( \lg_{10} \) (alcohol intake + 1), where alcohol intake resembles beverage specific or total intake over the lifetime. *\( p < 0.05 \), **\( p < 0.01 \)
not shown). The regression coefficients (95% confidence intervals, CI) on FEV1% for wine intake were 2.024 (0.075 – 3.973) in those below the median and 1.967 (0.628 – 3.306) for those above the median.

When we performed regression analysis separately by type of recent alcohol intake, there was a trend that among those drinking various beverages recently wine intake was positively associated with lung function. The regression coefficient on FEV1% for wine intake was 1.743 (95% CI, -1.197 – 4.682). For comparison, it was 2.362 (95% CI, -2.490 – 7.214) in those who drank only wine. 25% of those drinking various beverages did not drink wine.

**Discussion**

In this cross-sectional population-based study we observed that both recent and lifetime alcohol intake from wine showed a positive association with pulmonary function but total alcohol intake was not significantly associated with lung function. The association between lung function and white wine was slightly stronger than that for red wine but these two variables were highly correlated and residual confounding by healthy lifestyle factors might explain the difference.

We did not detect a significant association between lung function and total alcohol intake. Some, but not all, previous studies have shown a negative association of alcohol intake with pulmonary function [18–25]. Taken all studies together, total alcohol intake might not be strongly associated with pulmonary function, but the underlying reason for the conflicting evidence may, at least in part, be related to the failure of studying beverage specific alcohol in most of these studies. Our study supports this latter hypothesis. The results suggest that among all considered alcoholic beverages, alcohol intake from wine, both recent and lifetime intake, has the strongest relation to FEV1 and FVC%. Alcohol from beer and liquor is not significantly related to pulmonary function. Evidence suggests that alcohol may increase oxidative burden [12–15]. However, while alcohol could potentially act as an oxidant, there is a large body of evidence that wine has antioxidant properties [12,13,16,17]. Our data indicate that these antioxidant properties might exist and could have positive influence on lung function. To our knowledge only one other study has investigated the effects of beverage specific alcohol intake on pulmonary function. Cohen et al. failed to find a protective effect of wine intake on pulmonary function [18]. These authors classified participants into categories of light, moderate and heavy drinkers based on the amount (drinks per occasion) and frequency (rarely, occasionally, weekly and daily) of consumption of alcoholic beverages. Thus, due to the use of a categorical variable Cohen et al. had less power to detect an association [18]. In addition, the authors analyzed pulmonary function as the ratio of FEV1/FVC only and, consequently, focused on an indicator of airway obstruction. Therefore, the results are not directly comparable to those of our study.

FEV1 and FVC are highly correlated in populations without significant degree of airway obstruction. Thus, results of the analysis between antioxidants and FEV1 or FVC will be similar unless in a population free of reported lung disease very strong effects on airway narrowing or airflow limitation are present. This study shows that both FEV1 and FVC are associated with wine intake and this leaves us with little evidence for a strong effect of alcohol intake on airway narrowing.

We found that both recent and lifetime alcohol intake from wine was positively associated with lung function. This finding is important because the correlation between the recent and lifetime alcohol intake was not strong. It could also indicate that the effects are not related to recent intake of antioxidants from wine only but that long-term intake may exert positive effects on lung function. In general, we did not find independent additional effects of lifetime alcohol consumption after we included current alcohol intake. Thus, the results indicate no independent effect of lifetime alcohol intake beyond current intake.

Our analysis also suggests that wine intake may be associated with other healthy lifestyle characteristics (table 2). Participants who drank only wine tended to be lighter, more educated, smoke less and appeared to have a healthier diet. However, our findings persisted even after adjusting for known covariates related to pulmonary function, i.e. smoking, weight, micronutrients and socioeconomic status. Nevertheless, although we adjusted for several lifestyle variables in our regression analysis, the association of wine intake with pulmonary function may be due to residual confounding by healthy lifestyle as reported previously [35–38].

If our findings are real, the observation that the association with wine intake was slightly stronger for white wine than for red wine is surprising, because it has been suggested that red wine has greater health benefits than white wine and this may be due to greater antioxidant capacity [39]. Other research has indicated that both white and red wine have beneficial health effects and similar antioxidant properties [40]. The total antioxidant capacity of wine and the relative antioxidant properties of white and red wine vary depending on the assays used and no consensus has been reached. There is strong evidence that the antioxidant properties of wine are, at least in part, due to activity of different flavonoids and other compounds [41]. In addition, white wine contains several phenols with antioxidant activity [42,43].
Alternatively, it is conceivable that the stronger association of white wine with pulmonary function compared with red wine may also be due to confounding by other, healthy lifestyle factors. Approximately 74% of the participants reported both red and white wine intake during their lifetime. Compared with those who drank red wine but not white wine, total pack years of smoke exposure was higher in participants who reported white and red wine intake together (15.7 versus 10.9 pack years, p < 0.05) and, thus, residual confounding by smoking and/or other lifestyle factors could again explain our findings. Because of the limited sample size in the subgroups of wine drinkers we were unable to provide strong data in groups who only consumed either red or white wine.

Our results did not change significantly when we restricted the analysis to participants below the median of total alcohol consumption. In this restricted sample mean alcohol intake from specific beverages differed little and alcohol intake from wine remained the strongest correlate of lung function even after adjustment for other beverage specific alcohol intake. This finding indicates that if those with the heaviest alcohol intake were excluded, the pattern of association between beverage specific alcohol intake and lung function was similar and could support a true positive effect of wine on lung health.

Because cigarette smoke is a major source of oxidants we would expect a stronger association of wine intake with pulmonary function in smokers if wine has antioxidant capacity that prevents smoking induced oxidative damage. We failed to observe a statistically significant interaction of beverage specific alcohol intake with smoking. However, we should interpret this finding with caution, because of the limited sample size of current smokers.

Our study has several limitations. First, the cross-sectional design and, following from this design, the uncertainty about the cause-effect relation represent a weakness of our study. However, we obtained a lifetime drinking alcohol history using an instrument with good test-retest reliability [33], an approach that has not previously been used. A second limitation is the limited power to perform more definite subgroup analysis by smoking status or in groups who consumed a single beverage type only. A third limitation is the moderate participation rate and, as a consequence, the restricted generalization of the results to the general public.

The strength of this study is the detailed evaluation of information on covariates, including smoking and dietary habits that have shown a relation to lung function. However, we cannot definitely exclude residual confounding of health lifestyle factors, such as diet and smoking. Wine drinkers and in particular white wine drinkers, may differ from the rest of the population in other lifestyle factors that we did not include in this analysis. Drinking patterns, such as alcohol consumption with or without food could also play a role. Since there is no information on the relation of white and red wine intake with lung function and few studies have analyzed beverage specific alcohol intake in relation to lung function, this study is important in that it suggests the importance of beverage type in assessing the relation of alcohol to pulmonary outcomes.

Conclusions
In conclusion, we found a positive association of recent and lifetime wine intake with lung function. Contrary to the expectations, (based on the findings from in vitro studies showing that red wine has higher antioxidant potentials) we found that white wine was the only wine type significantly associated with lung function. Wine drinking in general was associated with healthier characteristics and better lifestyle habits. Our findings appear independent of the potential confounding effect of numerous and well measured factors (i.e. smoking, weight, socioeconomic status, dietary antioxidant intake); however we cannot exclude the potential of residual confounding. Other unmeasured factors or errors in measurements in the factors included in the analyses could be responsible for the observed associations. In particular, we have to note that among the different types of wine, consumption of white wine was associated with healthier characteristics and better lifestyle profile than consumption of red wine. In the absence of a biological plausibility for this differential effect of red and white wine on lung function we are concerned about the potential spurious nature of the observed associations. However, flavonoids and phenols are antioxidants found in wine products that have been implicated to play a role in lung health/function and may explain the observed associations. Further studies with detailed information on potential confounders and in social and ethnic settings were the correlates of drinking habits may differ from those reported here are needed to confirm our results and help clarify whether there is a relationship between the use of specific alcoholic beverages and lung health.

Abbreviations
CI, Confidence Interval
CLDH, Cognitive lifetime drinking history
FEV₁, forced expiratory volume in one second
FEV₁%, forced expiratory volume in one second in percent of the predicted value
FFQ, Food Frequency Questionnaire
FVC, forced vital capacity

FVC%, forced vital capacity in percent of the predicted value

SD, standard deviation

SE, standard error

Competing interests
None declared.

Authors’ contributions
HJS conceived of and designed the study, participated in lung function tests, performed analyses, participated in the interpretation of results and drafted the manuscript. BJBG, SEM and DK participated in the design, statistical analyses and interpretation of the study. JLF, PM, MR, TN, MR and MT participated in its design, coordination and interpretation.

All authors read and approved the final manuscript.

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