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Original Article

# Association between alcohol intake and subjective cognitive complaints in southwest Nigeria: a cross-sectional observational study ☆

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ABSTRACT

*Background:* Alcohol, a widely abused drug, is a general CNS depressant that is involved in an impaired neurological functioning in a dose-dependent manner and purportedly, in the development of adverse cognitive functions in humans.
*Objective:* To assess crosss-sectionally whether alcohol consumption is associated with the risk of subjective cognitive complaints (SCCs).
*Material and Methods:* A cross-sectional study of 1299 participants with diverse age groups, ethnicity and socioeconomic levels recruited from six public hospitals in three different states in the southwest Nigeria between March 2016 and April 2016 was done. Prevalence of subjective cognitive complaints by the level of alcohol intake was measured using standardized questionnaire. Factor analyses (explorative and confirmatory) were used to validate the cognitive complaint questionnaire while conditional multiple logistic regression analysis was used to investigate the association between alcohol intake and SCCs.
*Results:* After adjustment for age, marital status, level of education, ethnicity, smoking status and physical activity (basic adjustment), participants in the highest compared with the lowest quintile of alcohol intake had a significantly increased odds of SCCs (odds ratio [OR], 1.95; 95% confidence interval [CI], 1.39–2.74; *P* for trend <0.001). Additional adjustment for body mass index, depression, hypercholesterolemia, cardiovascular diseases, insomnia, stress and family histories of diabetes and hypertension (multivariable adjustment), did not substantially affect this relationship (OR, 2.02; 95% CI, 1.40–2.93; *P* for trend <0.001). When stratified by gender, results were similar and stronger for men in the basic (OR, 2.28; 95% CI, 1.34–3.88, *P* for trend <0.001) and multivariable (OR, 2.47; 95% CI, 1.37–4.47; *P* for trend <0.001) adjusted models but completely attenuated in the multivariable adjusted model for women (OR, 1.60; 95% CI, 0.94–2.69; *P* for trend = 0.08).
*Conclusion:* High intake of alcohol is associated with higher risk of SCCs in men. This relationship is independent of cardiovascular risk factors.


## 1. Introduction

Alcohol is not only a general CNS depressant affecting brain functions in a dose-dependent manner but also constitutes an agent for substance induced psychotic disorder.[1] Several studies have contributed toward understanding the dose and task-related ethanol's effect on the brain and their findings are valuable in disclosing the types of functions and the neural circuits underlying impairments due to ethanol's action.[2] Such studies revealed that the effects of alcohol, one of which may involve adverse cognitive functioning (i.e., mental activities that involve storing, retrieving and using information), are mediated through a number of target sites in the brain, principally $GABA_A$ and NMDA receptors.[3,4] However, proper cognitive functioning is essential for adequate and healthy performance in life.

SCCs are cognitively based errors that occur during the performance of a task that a person is normally successful in executing. Such complaints involve frequent forgetfulness, and difficulties in concentrating, making decision and thinking clearly, hence, depicting the experience of having problems with cognitive functioning. For instance, some individuals may experience such cognitive problems of having had to throw away something that they meant to keep and retaining those things they meant to throw out. Interestingly, these SCCs are common among not only the elderly people[5,6] but also the non-elderly adults.[7,8]

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The prior observations that mid-to-moderate drinking of alcohol can adversely affect various aspects of cognitive functioning[9] give reasons to expect that SCCs may be associated with alcohol intake. The focus of this present study therefore was to investigate the relationship between alcohol intake and subjective perceptions of cognitive symptoms. Thus, two independent self-report measures of (1) dietary intake that allows quantification of the amount of alcohol intake and (2) cognitive complaints that permit a quick and easily administered snapshot of cognitive concerns were used. It was predicted that a high- compared to a low level of alcohol intake would be associated with high SCCs.

## 2. Material and methods

### 2.1. Study population

In total 1299 people participated in the survey. The research participants consisted of 807 women and 492 men that were between 18 and 87 years of age. Participants were recruited from three different States of Lagos, Ogun and Oyo in the southwest of Nigeria and were selected across six public hospitals between March 2016 and April 2016. Letters were sent to the necessary units within these hospitals. An information sheet was sent out with the questionnaire. This included a description about the aims of the project. All participants were informed about the study, had the opportunity to ask questions and signed an informed consent before enrolling in the study. The following inclusion criteria were applied: (1) 18 years of age and older, (2) good health status. The exclusion criteria included (1) serious illness, severe hearing or visual impairment precluding a reliable assessment of cognitive complaints, (2) persistent impairment of consciousness, (3) a history of severe head trauma or neurosurgery, (4) previous prolong mental retardation. The study had approval by the Tai Solarin University of Education Institutional Review Board.

### 2.2. Assessments

#### 2.2.1. Alcohol intake

This was measured using items from a standard food frequency questionnaire with ratings involving a 10 point scale from – Never to – Everyday. The questionnaire is similar to a fairly recent validated food frequency questionnaire (FFQ).[10] The FFQ was analysed using feta software.[10]

#### 2.2.2. SCCs

This study measures SCCs by using Cognitive Failures Questionnaire (CFQ) scores.[11] The CFQ is a 25-item self-report measure of failures in attention, perception, memory, and action. Participants were asked to indicate on a 5-point scale how often they have experienced each failure in the past months, from 0 (*never*) to 4 (*very often*). Examples of CFQ items include #2, "Do you forget why you went from one part of the house to the other?" and #19, "Do you daydream when you ought to be listening to something?" The total score of the measure ranged from 0 to 100 and the median score was put at 50.0.

### 2.3. Medical history, anthropometric data and lifestyle factors

Anthropometric data, lifestyle factors and medical history were obtained as part of the standard FFQ. Body mass index was calculated as weight in kilogrammes divided by the square of height in metres. Physical activity was expressed as metabolic equivalent task (MET)-hours based on self-reported types and durations of activities per week. One MET-hour is equivalent to energy expenditure while sitting quietly for 1 h. Smoking status was obtained and depicted as current, former and never. Medical history of chronic diseases involving diabetes, hypertension, depression, cardiovascular diseases, insomnia, hypercholesterolemia and stress were defined through questions asking whether doctor informed them with having the condition in the past year, and were dichotomized to either yes or no.

### 2.4. Demographics

Data from items referring to age, gender, education, ethnicity and marital status included in the demographic section of the standard FFQ were obtained for this analysis. Gender was self-reported as either male or female. Age was treated as continuous measure. Education level was treated based on four levels — No formal education, primary education, secondary education, college education and university education. Ethnicity consisted of four subgroups — Yoruba, Hausa, Ibo, and others. There were five categories of marital status — married, single, widowed, divorced and separated.

### 2.5. Statistical analyses

The CFQ as a measure of SCCs was assessed for internal consistency using Cronbach's alpha reliability coefficient. We validated the construct on Nigerian sample using exploratory factor analysis (EFA) and confirmatory factor analysis (CFA).

Continuous variables are presented as means and standard deviations or medians and interquartile ranges and were compared among quintiles of alcohol separately for men and women using One-way ANOVA or the Kruskal-Wallis H test. Proportions were compared using the $X^2$ test.

Alcohol intake of participants were categorised into quintiles. Conditional logistic regression was used to investigate the association between alcohol intake and SCCs. To test for linear trends across categories, we used log-transformed alcohol levels. In the basic model, we adjusted for demographic and lifestyle factors involving age (continuous), marital status (single, married, divorced or separated), level of education (non-formal, primary, secondary, college or university), ethnicity (Yoruba, Hausa, Ibo or others), smoking status (never, past or current) and physical activity (quintiles). In our multivariable model, we further adjusted for body mass index (continuous) and history of diabetes (yes/no), hypertension (yes/no), depression (yes/no), cardiovascular diseases (yes/no), insomnia (yes/no), hypercholesterolemia (yes/no) and stress (yes/no).

We next examined the impact of a doubling in alcohol intake on subjective cognitive complaints by estimating the odds ratios (ORs) associated with an increase of (continuous) log-transformed alcohol intake by units on a log scale, which corresponds to a doubling in alcohol intake on the original scale. These analyses were carried out for crude, basic and multivariable models as indicated above.

We repeated all the analyses above separately for men and women using the crude, basic and multivariable models and estimated their individuals ORs. We assessed the goodness of fit of the models using the method described by Hosmer and Lemeshow.[12] All *P*-values are two-tailed, and $P < 0.05$ was considered statistically significant. All analyses were performed using SAS software, version 9.2.1 (SAS Institute Inc., Cary, NC, USA).

## 3. Results

### 3.1. Validation of the CFQ: A measure of SCCs

The data from CFQ had a Gaussian distribution and a reliability with high internal consistency (Cronbach's $\alpha$ was 0.93). A scree plot graphing the eigen values revealed a sharp break in the curve

after the first factor leading to the retention of one factor. This factor had an eigen value of 13.87 and accounted for 14.03% of the variance. Consequently, evaluation of a model consisting of this single factor for goodness of fit revealed that the Bentler-Bonnet Normed Fit Index (NFI) and the Comparative Fit Index (CFI) had values of 0.92 and 0.93 respectively.

### 3.2. Alcohol intake and SCCs

Characteristics of participants according to quintiles of alcohol intake are presented in Table 1a and b. Participants with higher alcohol intake had significantly high SCCs in both gender. Marital status was significantly associated with alcohol intake in both gender. However, age, depression, diabetes, hypertension, hypercholesterolemia, cardiovascular disease, insomnia, stress and physical activity were not associated with alcohol intake in both gender. Men with higher alcohol intake had significantly high body mass index and were more likely to smoke. Among men, education and ethnicity were significantly associated with alcohol intake. In women, no significant difference was observed for body mass index, smoking status, education and ethnicity among the quintiles of alcohol intake.

Table 2 shows the estimated ORs of SCCs across quintiles of alcohol intake among participants. Participants in the highest compared with lowest quintile of alcohol consumption had a significantly increased odds of SCCs (OR, 2.20; 95% CI, 1.60–3.02, *P* for trend on a log scale <0.001). This relationship was only slightly affected after basic adjustment (OR, 1.95; 95% CI, 1.39–2.74; *P* for log trend <0.001), moreover, further adjustment for depression, diabetes mellitus, hypertension, insomnia and stress did not substantially affect this relationship (OR, 2.02; 95% CI, 1.40–2.93; *P* for log trend <0.001). Results were similar and stronger for men in the crude, basic and multivariable analyses but completely attenuated in the multivariable adjusted model for women (OR, 1.60; 95% CI, 0.94–2.69; *P* log for trend <0.001).

We next examined the impact of a doubling in alcohol intake on SCCs in this population by calculating the crude, basic and multivariable adjusted ORs associated with an increase of (continuous) log-transformed alcohol intake by 2 units on a log-scale, which represents a doubling in alcohol intake on the original scale; again, as can be seen in Table 3, similar results were found in the combined analysis and when stratified by gender, the effect was more pronounced in men but completely attenuated in women in the crude, basic and multivariable adjusted models.

## 4. Discussion

The present study validated the CFQ as a measure of SCCs and tested the relation between alcohol intake and SCCs in a cross-sectional sample of Nigerian adults. The validation analyses indicated that the single factor structure resulting from the EFA of the CFQ was confirmed by the CFA. This provides evidence that suggests that SCCs construct as measured by CFQ appears to be culturally invariant thus reinforcing the one-factor model. More importantly, this study found that high alcohol intake was associated with higher odds of SCCs. This relationship remained even after adjustment for life style factors and cardiovascular risk factors suggesting that either of these factors may not fully explain the relationship between SCCs and alcohol intake. Of note, this relationship was gender-dependent as when stratified by gender, results were similar and stronger for men but completely attenuated for women in the final adjustment for the joint life style and cardiovascular risk factors. This indicates that SCCs was specifically associated alcohol intake in men and that such relationship in men may not be explained by cardiovascular risk factors or factors associated with life style.

### 4.1. The CFQ reveals a single factor model

On the CFQ validation, Broadbent et al.[11]claimed that the CFQ should be used only to assess the single construct of cognitive complaint. He supported this claim by stating the high internal consistency of the scale. We validated this scale in this Nigerian sample and confirmed the unidimensionality of the construct using EFA and CFA. For our study, the goodness of fit was indicated by the NFI and the CFI. The value of the NFI indicates the proportion in the improvement of the overall fit of the researcher's model relative to the null model.[13] The CFI can be interpreted in a similar way to the NFI, except that it is not influenced by sample size.[13] Practically, fit values approaching 1.0 indicates a better match between the proposed model and the theoretical model. The NFI was 0.92, and the CFI was 0.93. These values fell above the recommended 0.90,[13] suggesting acceptance of the single-factor model by the criterion.

### 4.2. Alcohol intake is associated with SCCs in men

Though link between duration and lifetime quantity of drinking and the development of cognitive problems remains unclear, previous studies have suggested that cognitive performance worsens in direct proportion to the severity and duration of alcohol use disorders.[14–17] In addition, it was also suggested that some cognitive deficits are possible even in people who are not heavy drinkers.[18] Interestingly, our results in men are in line with these studies as we observed a more pronounced SCCs when alcohol intake was doubled in the regression analysis. While a further research is needed to determine how a person's pattern of drinking is related to SCCs, one possible hypothesis to explain the gender-mediated relationship between alcohol intake and SCCs in our study is that the gender differences observed might be associated with cardiovascular diseases or risk factors, for which similar gender variations were observed. For instance, among the quintiles of alcohol in this study, we observed some cardiovascular risk factors involving stress symptoms, smoking, and BMI that were significant in men but not in women. Interestingly, these factors may induce cerebral microvascular damages or neurodegeneration in men than in women, explaining why men reported more cognitive complaints in this study. In line with this and as noted earlier, we have evidence in our data that the different cardiovascular risk can at least in part explain the gender differences.

As an extension to the above hypothesis, one might speculate that the higher odds of SCCs in relation to alcohol in men may reflect a more cognitively demanding life for men than for women. This is more so, in Nigeria where labour force participation rate for male ages 15 and above was about 78% as against 37% for female of the age range based on national estimates according to the World Bank collection of development indicators,[19] thus reinforcing the observed association between alcohol intake and SCCs in men.

One strength of the present study was the setting: the SCC items and alcohol intake were part of a general health survey collecting a large number of other relevant health data, but without specific focus on cognitive complaint or alcohol consumption. In addition, the alcohol intake was derived from the general food frequency questionnaire (FFQ). Moreover, the sample covered a wide age range and geographical area and the number of participants was relatively high. The participation in this study presupposed relatively high cognitive function being that the participants must read the introduction letter, sign the consent form and fill in several questionnaire.

**Table 1**
Characteristics of the study population.

| | Quintile 1 | Quintile 2 | Quintile 3 | Quintile 4 | *P* value |
|---|---|---|---|---|---|
| *(a)* | | | | | |
| **Men** | | | | | |
| Alcohol intake, gday$^{-1}$ | 0.0 | 0.8 | 1.6 | 7.7 | **<0.001** |
| Median [IQR] | [0.0–0.0] | [0.7–0.8] | [1.3–1.8] | [3.3–10.2] | |
| Cognitive failures[¶] (SCCs), mean ± SD | 40.3 ± 18.1 | 38.7 ± 19.3 | 40.1 ± 19.4 | 50.5 ± 16.4 | **<0.001** |
| Age, mean ± SD, y | 32.9 ± 12.4 | 30.0 ± 9.8 | 35.3 ± 12.3 | 34.9 ± 11.2 | 0.05 |
| Education, % | | | | | **0.04** |
| No formal education | 0.5 | 4.3 | 0.0 | 0.0 | |
| Primary | 2.3 | 6.4 | 7.7 | 3.7 | |
| Secondary | 16.0 | 12.8 | 8.8 | 15.6 | |
| Tertiary-College | 27.4 | 36.2 | 29.7 | 24.8 | |
| Tertiary-University | 53.9 | 40.4 | 53.9 | 56.0 | |
| Ethnicity, % | | | | | |
| Yoruba | 82.0 | 80.0 | 68.1 | 66.4 | **0.04** |
| Hausa | 4.1 | 2.0 | 7.5 | 4.6 | |
| Ibo | 12.2 | 12.0 | 18.1 | 22.7 | |
| Other | 1.8 | 6.0 | 6.4 | 6.4 | |
| Marital status, % | | | | | **0.04** |
| Married | 52.5 | 36.0 | 54.2 | 54.1 | |
| Single | 45.6 | 54.0 | 40.6 | 38.5 | |
| Divorced | 0.5 | 2.0 | 3.1 | 3.7 | |
| Separated | 0.5 | 6.0 | 0.0 | 2.8 | |
| Widow(er) | 0.9 | 2.0 | 2.1 | 0.9 | |
| Smoking, % | | | | | |
| Never | 81.3 | 75.0 | 55.4 | 60.0 | **<0.001** |
| Former | 11.6 | 14.6 | 25.0 | 15.5 | |
| Current | 7.1 | 10.4 | 19.6 | 24.6 | |
| Body mass index mean ± SD | 25.8 ± 5.0 | 25.2 ± 3.2 | 26.3 ± 5.2 | 27.9 ± 4.6 | **<0.001** |
| History, % | | | | | |
| Depression | 10.9 | 10.4 | 5.4 | 8.9 | 0.49 |
| Diabetes mellitus | 6.5 | 6.3 | 14.0 | 8.0 | 0.17 |
| Hypertension | 8.5 | 6.3 | 10.9 | 6.2 | 0.63 |
| Hypercholesterolemia | 0.5 | 4.2 | 1.1 | 0.0 | 0.06 |
| Cardiovascular disease | 1.9 | 0.0 | 4.3 | 3.5 | 0.37 |
| Insomnia | 5.2 | 2.1 | 3.2 | 5.3 | 0.70 |
| Stress | 33.6 | 50.0 | 33.0 | 26.8 | 0.04 |
| Physical activity, MET-h/wk | 3.5 | 4.0 | 3.5 | 2.5 | 0.44 |
| Median [IQR] | [1.8–12.8] | [1.8–13.1] | [1.8–14.0] | [1.8–9.9] | |
| *(b)* | | | | | |
| **Women** | | | | | |
| Alcohol intake, gday$^{-1}$ | 0.0 | 0.8 | 1.6 | 6.4 | **<0.001** |
| Median [IQR] | [0.0–0.0] | [0.7–0.8] | [1.5–2.1] | [3.8–10.7] | |
| Cognitive failures[¶] (SCCs), mean ± SD | 39.4 ± 17.9 | 41.6 ± 18.1 | 43.2 ± 18.1 | 43.3 ± 18.0 | **0.04** |
| Age, mean ± SD, y | 31.7 ± 10.7 | 29.9 ± 9.8 | 30.5 ± 9.8 | 29.9 ± 9.2 | 0.32 |
| Education, % | | | | | 0.08 |
| No formal education | 1.7 | 0.0 | 2.9 | 0.0 | |
| Primary | 4.0 | 1.0 | 5.1 | 6.3 | |
| Secondary | 15.2 | 14.1 | 11.7 | 12.6 | |
| Tertiary-College | 33.8 | 31.3 | 27.7 | 42.5 | |
| Tertiary-University | 45.3 | 53.5 | 52.6 | 38.6 | |
| Ethnicity, % | | | | | |
| Yoruba | 81.2 | 84.0 | 76.9 | 73.8 | 0.23 |
| Hausa | 2.4 | 1.0 | 5.2 | 7.1 | |
| Ibo | 13.5 | 13.0 | 13.4 | 15.9 | |
| Other | 2.9 | 2.0 | 4.5 | 3.2 | |
| Marital status, % | | | | | **0.002** |
| Married | 52.7 | 36.9 | 50.7 | 48.4 | |
| Single | 42.0 | 56.3 | 44.9 | 43.0 | |
| Divorced | 1.5 | 1.9 | 1.9 | 2.9 | |
| Separated | 0.7 | 1.9 | 0.0 | 6.3 | |
| Widow(er) | 3.2 | 2.9 | 2.9 | 0.8 | |
| Smoking, % | | | | | |
| Never | 91.2 | 88.2 | 84.9 | 84.6 | 0.16 |
| Former | 4.5 | 6.9 | 10.1 | 10.8 | |
| Current | 4.2 | 4.9 | 5.0 | 4.6 | |
| Body mass index mean ± SD | 25.8 ± 4.2 | 26.2 ± 4.5 | 25.8 ± 4.1 | 26.3 ± 4.7 | 0.79 |
| History, % | | | | | |

**Table 1** (*continued*)

| | Quintile 1 | Quintile 2 | Quintile 3 | Quintile 4 | *P* value |
|---|---|---|---|---|---|
| Depression | 8.3 | 11.2 | 9.6 | 10.1 | 0.80 |
| Diabetes mellitus | 8.5 | 6.1 | 7.4 | 3.1 | 0.21 |
| Hypertension | 6.5 | 14.3 | 6.7 | 7.7 | 0.08 |
| Hypercholesterolemia | 1.5 | 0.0 | 1.5 | 3.1 | 0.32 |
| Cardiovascular disease | 2.5 | 1.0 | 1.5 | 3.2 | 0.65 |
| Insomnia | 5.0 | 7.2 | 6.8 | 6.9 | 0.73 |
| Stress | 35.0 | 40.0 | 28.6 | 32.8 | 0.31 |
| Physical activity, MET-h/wk Median [IQR] | 3.0 [0.8–9.0] | 3.5 [1.8–17.1] | 3.0 [1.8–10.1] | 4.2 [1.8–10.5] | 0.20 |

Notes: MET-h/wk, metabolic equivalent task-hours per week. Data are expressed as No. (%), as means ± SD, standard deviation or as median [IQR, interquartile range]. Bold characters indicate significant values.
[¶] Cognitive failures depict subjective cognitive complaints (SCCs).

**Table 2**
Estimated ORs of subjective cognitive complaints according to quintile of alcohol intake.

| | Quintile 1 | Quintile 2 | Quintile 3 | Quintile 4 | *P* Value[†] |
|---|---|---|---|---|---|
| **All participants** | | | | | |
| Model | | | | | |
| Crude | 1.00 | 0.86 (0.56–1.33) | 1.27 (0.91–1.79) | **2.20 (1.60–3.02)** | <0.001 |
| Basic adjusted[ǂ] | 1.00 | 0.87 (0.55–1.37) | 1.17 (0.81–1.67) | **1.95 (1.39–2.74)** | <0.001 |
| Multivariable adjusted[¥] | 1.00 | 0.76 (0.46–1.27) | 1.08 (0.73–1.61) | **2.02 (1.40–2.93)** | <0.001 |
| **Men** | | | | | |
| Model | | | | | |
| Crude | 1.00 | 0.67 (0.32–1.42) | 0.90 (0.52–1.55) | **2.29 (1.43–3.68)** | <0.001 |
| Basic adjusted[ǂ] | 1.00 | 0.60 (0.25–1.43) | 0.67 (0.36–1.26) | **2.28 (1.34–3.88)** | <0.001 |
| Multivariable adjusted[¥] | 1.00 | 0.59 (0.22–1.54) | 0.57 (0.28–1.15) | **2.47 (1.37–4.47)** | <0.001 |
| **Women** | | | | | |
| Model | | | | | |
| Crude | 1.00 | 1.02 (0.60–1.74) | 1.54 (0.99–2.39) | **1.97 (1.27–3.04)** | <0.001 |
| Basic adjusted[ǂ] | 1.00 | 1.00 (0.58–1.74) | 1.57 (1.00–2.47) | **1.64 (1.03–2.60)** | 0.05 |
| Multivariable adjusted[¥] | 1.00 | 0.83 (0.44–1.54) | 1.53 (0.92–2.53) | 1.60 (0.94–2.69) | 0.04 |

[†] *P* value for trend based on log-transformed alcohol intake.
[ǂ] Adjusted for variables involving age, marital status, level of education, ethnicity, smoking status and physical activity.
[¥] Adjusted additionally for variables involving body mass index, depression, hypercholesterolemia, cardiovascular diseases, insomnia, stress, and family histories of diabetes and hypertension.

**Table 3**
Estimated ORs of subjective cognitive complaints associated with a doubling in alcohol intake.[*]

| Model | All Participants | Men | Women |
|---|---|---|---|
| Crude | **1.95 (1.33–2.87)** | **2.45 (1.34–4.50)** | 1.64 (0.98–2.74) |
| Basic adjusted[ǂ] | **1.91 (1.26–2.90)** | **3.77 (1.85–7.71)** | 1.43 (0.82–2.50) |
| Multivariable adjusted[¥] | **2.10 (1.32–3.34)** | **4.11 (1.78–9.48)** | 1.61 (0.86–3.02) |

[*] Effect estimates and *P* values were calculated using log-transformed alcohol level as a continuous variable.
[ǂ] Adjusted for variables involving age, marital status, level of education, ethnicity, smoking status and physical activity.
[¥] Adjusted additionally for variables involving body mass index, depression, hypercholesterolemia, cardiovascular diseases, insomnia, stress, and family histories of diabetes and hypertension.

Our study has some limitations. First, this cross-sectional design cannot make inferences about the causal relationship between alcohol and SCCs in this population. This is because it is practically difficult to determine the temporal sequence between the SCC and the alcohol intake in this study. It is also possible that the observed association is confounded by a yet-to-be-determined factor; however, we adjusted our analysis for established and novel cardiovascular and cognitive function risk factors. While we excluded participants with diagnosed serious illness, severe head injury or previous mental retardation, we cannot exclude the possibility that participants had undiagnosed illness that may preclude a reliable assessment of cognitive complaints, however, any potential misclassification should be nondifferential and could therefore unlikely change the overall pattern of SCCs in the population. It should be noted that some of the variables that we adjusted for may be causal mediators instead of confounders, which would underestimate the true relationship between alcohol intake and risk of SCC.

### 4.3. Conclusion

In conclusion, our results suggest that those with increased drinking behaviour could be at an increased risk of SCCs among men, independent of traditional cardiovascular disease risk factors. The effect of alcohol on risk of SCCs merits further study.

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## Conflict of interests

The authors declare no conflict of interest.

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