**CAMBRIDGE UNIVERSITY PRESS**

# Association of Alcohol Consumption with Cognition and Functionality in Older Adults Aged 75+ Years: The Pietà Study

Mariana Alves de Almeida[1] [ID], Maira Tonidandel Barbosa[1,2], Elisa de Paula França Resende[1,2,3], Viviane Amaral Carvalho[1], Ana Paula Borges Santos[3], João Carlos Barbosa Machado[2], Vivian Proença Lara[4], Karina Braga Gomes[4], Thais Helena Machado[1,5] and Paulo Caramelli[1]

[1]Behavioral and Cognitive Neurology Research Group, Departamento de Clínica Médica, Faculdade de Medicina, Universidade Federal de Minas Gerais, Belo Horizonte, MG, Brazil, [2]Faculdade de Ciências Médicas de Minas Gerais, Belo Horizonte, MG, Brazil, [3]Hospital das Clínicas da Universidade Federal de Minas Gerais/EBSERH, Belo Horizonte, MG, Brazil, [4]Faculdade de Farmácia, Universidade Federal de Minas Gerais, Belo Horizonte, MG, Brazil and [5]Departamento de Fonoaudiologia, Faculdade de Medicina, Universidade Federal de Minas Gerais, Belo Horizonte, MG, Brazil

### Résumé

La rélation entre l'alcool et la cognition est encore controversée. II s'agit d'une étude transversale populationnelle menée au Caeté (MG), Brésil où 602 personnes âgées de 75+ ans, 63.6% femmes et avec une éducation moyenne de 2.68 ans, ont été soumises à des évaluations cliniques détaillées et classées face au nombre de boissons alcooliques consommées par semaine. Les prévalences de consommation d'alcool antérieure/actuelle étaient de 34.6% et 12.3%, respectivement. Aucune association n'a émergé entre les diagnostics cognitifs et la consommation d'alcool. L'absence de consommation actuelle d'alcool était associée à la démence (OR = 2.34; IC à 95%: 1.39–3.90) et à une pire fonctionnalité (p = 0.001). La consommation antérieure de cachaça (liqueur de canne à sucre) a augmenté la chance du diagnostic de démence de 2.52 (IC à 95%: 1.25–5.04). Cette association entre la consommation de cachaça et démence n'a pas été registré auparavant.

### Abstract

The relationship between alcohol consumption and cognition is still controversial. This is a cross-sectional population-based study conducted in Caeté (MG), Brazil, where 602 individuals aged 75 + years, 63.6% female, and with a mean education of 2.68 years, were submitted to thorough clinical assessments and categorized according to the number of alcoholic beverages consumed weekly. The prevalence rates of previous and current alcohol consumption were 34.6% and 12.3%, respectively. No association emerged between cognitive diagnoses and current/previous alcohol consumption categories. Considering current alcohol intake as a dichotomous variable, the absence of alcohol consumption was associated with dementia (OR = 2.34; 95%CI: 1.39–3.90) and worse functionality (p = 0.001). Previous consumption of cachaça (sugar cane liquor) increased the risk of dementia by 2.52 (95%CI: 1.25–5.04). The association between the consumption of cachaça and dementia diagnosis has not been described before.

## Introduction

Dementia is a prevalent clinical disorder among older adults, with approximately 50 million cases worldwide, projected to reach 82 million in 2030 and 152 million in 2050 (World Health Organization & Alzheimer's Disease International, 2020). Because of increases in life expectancy and greater risk factor exposure, the most substantial increase in dementia prevalence is projected for individuals in low- and middle-income countries. In the absence of disease-modifying treatments for dementia, risk factor reduction is a fundamental strategy for preventing dementia. In the pursuit of this objective, the 2020 report from the Lancet Commission for Dementia Prevention, Intervention and Care estimated that 40% of global dementia cases could be prevented or delayed if 12 key modifiable risk factors for dementia were eliminated. One of the identified modifiable risk factors for dementia is excessive or harmful alcohol use during mid-life (Livingston et al., 2020; Suemoto et al., 2023).

Establishing the relationship between alcohol and cognitive performance is challenging due to inconsistencies in existing data and inherent limitations of observational studies (Rao, 2018;

Sabia et al., 2018; Schwarzinger, Pollock, Hasan, Dufouil, & Rehm, 2018). These studies vary greatly in terms of types of alcoholic beverage, consumption categories and methodologies. In addition to this heterogeneity, self-report questionnaires lack reliability, and alcohol consumption patterns vary considerably throughout life, which makes it difficult to remember and report accurate alcohol consumption in research settings (Sabia et al., 2018).

Previous research suggests that the association between alcohol consumption and cognition is non-linear. Several authors propose a J- or U-shaped relationship, in which a lower risk of dementia or cognitive decline would be associated with light to moderate alcohol consumption compared to abstinence and heavy consumption (Cooper et al., 2009; Mukamal et al., 2003; Orgogozo et al., 1997; Peters, Peters, Warner, Beckett, & Bulpitt, 2008; Ruitenberg et al., 2002; Sabia et al., 2018; Salvador et al., 2022; Xu et al., 2017). A Brazilian community-based and cross-sectional study with 1145 older adults obtained findings that also suggest a non-linear relationship between alcohol and cognition (Lopes, Furtado, Ferrioli, Litvoc, & Bottino, 2010). Although wine shows stronger evidence in some studies (Orgogozo et al., 1997; Peters et al., 2008) most do not differentiate between types of alcoholic beverages.

Conversely, prolonged and excessive alcohol consumption (usually defined as greater than 14 doses per week) can lead to structural damage and permanent brain dysfunction (Sachdeva, Chandra, Choudhary, Dayal, & Anand, 2016). In fact, excessive and prolonged alcohol consumption seems to be associated with alcohol-related dementia, Korsakoff syndrome (Ridley, Draper, & Withall, 2013), and an increased risk of any dementia (Boff, Sekyia, & Bottino, 2015). A study carried out in France with hospitalized patients showed that the main risk factor for dementia of any aetiology, especially early-onset (<65 years), was alcohol use disorders (Schwarzinger et al., 2018).

Regarding alcohol abstinence, some studies indicate a higher dementia risk when compared to light or moderate consumption (Cooper et al., 2009; Mukamal et al., 2003; Ruitenberg et al., 2002; Sabia et al., 2018). One of the studies proposes that part of this risk of dementia in abstainers is attributable to a higher risk of cardiometabolic disease in this group (Sabia et al., 2018). Furthermore, the association between alcohol consumption and dementia may be influenced by the presence of the Ɛ4 allele of the *Apolipoprotein E* (*APOE*) gene. Some studies have shown that carriers of this allele are at greater risk of dementia with increased alcohol consumption (Anttila et al., 2004), but others have found no interaction (Koch et al., 2019; Ruitenberg et al., 2002).

Despite recurrent observations of an association between light-to-moderate alcohol intake and superior cognitive performance when compared to non-drinkers, studies are not robust enough to define a causal relationship (Mukamal et al., 2003; Ruitenberg et al., 2002; Sabia et al., 2018). More recent research has questioned these observations, with new evidence suggesting an association between brain damage and moderate alcohol intake (Daviet et al., 2022). A 2017 study of 550 older men showed that moderate drinking was associated with a threefold increased risk of hippocampal atrophy, compared with abstainers. The study also revealed that adverse brain outcomes, including hippocampal atrophy, were present even among those with moderate alcohol intake (Topiwala et al., 2017).

Alcohol consumption is prevalent globally, but its patterns are strongly influenced by socioeconomic, cultural, and ethnic factors across diverse regions. Few published studies have addressed this topic in Latin America and other low- and middle-income countries. Furthermore, there is a dearth of research evaluating populations with lower education levels, particularly among older and oldest-old individuals.

The objective of this study was to investigate the associations between alcohol consumption and cognitive function and functional capacity, in a community-based population sample of older adults aged 75+ years in Caeté, Minas Gerais, Brazil. It was hypothesized that excessive alcohol consumption would be associated with poorer cognitive performance and functionality in older adults. Effect modification of *APOE* genotype was further explored.

## Methods

### Participants

The study data were extracted from the Pietà Study, a population-based investigation conducted in Caeté, Minas Gerais, Brazil, between October 2007 and July 2008. Based on the 2007 census, the municipality had a population of 39,039 individuals, among whom 1,251 (3.2% of the total population) were aged 75+ years (Caramelli et al., 2011).

Participants were recruited through an active search for individuals aged 75 years or older, residing in both urban and rural areas. This search was conducted by community healthcare agents and involved the dissemination of information through local media channels (radio and newspapers). Additionally, the research team visited the city's two long-term care institutions. In total, 639 individuals (constituting 51.1% of the target population) consented to participate in the study and signed the informed consent form.

From the initial group of 639 participants in the Pietà study, 18 participants with indeterminate cognitive diagnosis were excluded from the current analysis. Additionally, 19 participants lacking alcohol consumption information were excluded. Consequently, the final sample for statistical analysis comprised 602 participants, with an average age of 81.1 years, spanning from 75 to 99 years. Within this group, 383 participants (63.6%) were female, while 219 (36.4%) were male. The average duration of education, as measured in lifetime years, was found to be 2.68 ± 2.73 years. Notably, over 80% of the individuals were of middle or middle-low socioeconomic status, according to the Brazilian Association for Market Research Institutes scale (Associação Brasileira de Empresas de Pesquisa, 2003).

### Procedure and measures

Participants provided data across three phases: (1) completion of sociodemographic and self-report psychosocial questionnaires and interview, (2) clinical assessment, and (3) blood sample collection. Briefly, Phase 1 was conducted by trained interviewers who visited participants in their living residence (independent home or long-term care dwelling). Structured questionnaires indexed participant sex and socioeconomic status (Associação Brasileira de Empresas de Pesquisa, 2003), global functional performance using the Brazilian Older Americans Resource and Services Multidimensional Functional Assessment Questionnaire (BOMFAQ; Pfeiffer, 1978) and alcohol consumption. The comprehensive clinical evaluation in Phase 2 was administered by board-certified geriatricians, neurologists, and a psychiatrist, each experienced in assessing older adults with neurological and psychiatric expertise. Participants underwent a battery of clinical and neuropsychological tests to index their history of neurological and clinical diagnoses, global functioning, cognitive functioning, and psychiatric functioning

(see Supplemental Material). Following clinical evaluation, participants underwent neuropsychological assessments, including the Mattis Dementia Assessment Scale (Porto, Fichman, Caramelli, Bahia, & Nitrini, 2003), Rey Auditory-Verbal Learning Test (Malloy-Diniz, Parreira Lasmar, de Sena Rabelo Gazinelli, Fuentes, & Salgado, 2007), Consortium to Establish a Registry for Alzheimer's Disease (Bertolucci et al., 2001), phonemic verbal fluency test, and Frontal Assessment Battery (Beato, Nitrini, Formigoni, & Caramelli, 2007). Finally, Phase 3 entailed the collection of blood for biomarker analyses, including *APOE* genotyping in 295 individuals (Lara et al., 2016). For further details on the study protocol and measurements, see Caramelli et al. (2011).

### Alcohol consumption
*Current* and *previous* (i.e., more than 6 months before the interview) alcohol intake was assessed in Phase 1 by trained interviewers. This information was gathered directly from the study participant or, in cases of cognitive impairment, from a close informant (e.g., family relative). Information on dose, frequency, duration, and types of beverages consumed was collected. The quantity of each type of beverage ingested per day, week, or month was queried, with a minimum dose of 0.1 per week as the inclusion threshold for current users.

In this study, the alcoholic beverage dose was defined as 14 grams, equivalent to one can of beer (350 ml) or one dose (45 ml) of *cachaça*, whisky, spirits, or a glass of wine (150 ml). Cachaça, a distilled alcoholic beverage derived from sugar cane fermentation, possesses an alcohol content ranging from 38% to 48%. Beer typically contains around 5% alcohol by volume, wine approximately 12%, whisky at least 40%, and other spirits around 40% or less. Beer is produced through the fermentation of cereals, mainly malted barley, while wine results from the fermentation of grape juice. Whisky is a distilled alcoholic beverage derived from cereals. Male and female participants were categorized based on the number of alcohol doses consumed per week throughout their lives, based on the categorization used in previous studies: no consumption (abstainers), 0.1–7 doses per week (light consumption), 7.1–14 doses per week (moderate consumption), and more than 14 doses per week (heavy drinking) (Koch et al., 2019).

The diagnoses of alcohol abuse and dependence were determined by the semi-structured Mini International Neuropsychiatric Interview (MINI; Lecrubier et al., 1997). Alcohol abuse and dependence were defined when >1 and ≥3 positive responses were obtained, respectively (American Psychiatric Association, 1994).

### Cognitive diagnosis
Following a consensus meeting among the research team, the participants received categorization into one of three cognitive diagnoses: dementia (American Psychiatric Association, 1987), cognitive impairment no-dementia (CIND; Graham et al., 1997), and no cognitive impairment (NCI). Notably, individuals with an undetermined cognitive profile (18 participants) were excluded from the analyses, as mentioned above.

### Functional assessment
In the current study, functional capacity was determined by three separate measures. Global functional performance was measured using the BOMFAQ (assessed in Phase 1; Pfeiffer, 1978). The Pfeffer Functional Activities Questionnaire (FAQ assessed in Phase 2; Pfeffer, Kurosaki, Harrah, Chance, & Filos, 1982) was administered to informants of participants with cognitive impairment. Finally, participants were classified according to the Functional

Assessment Staging in Alzheimer's Disease (FAST assessed in Phase 2; Reisberg, 1988).

### Statistics
The data were analyzed using the Statistical Package for Social Science (version 23.0). Pearson's chi-square test was employed to assess associations between categorical variables, and in cases where it was more suitable, Fisher's exact test was used. The chi-square test was selected for variables with more than three ordinal categories, as well as when over 20% of the cells had an expected count below five or when the expected value was less than the unit. For comparing non-categorical variables in two independent groups without a normal distribution, the nonparametric Mann–Whitney test was applied. The significance level chosen for statistical significance was 5%.

Multivariate analysis was conducted using the ordinal logistic regression method. In the final adjustment of the multivariate model, all variables displaying a p-value <0.20 in the association analysis with the outcome (cognitive diagnosis) were initially included. Subsequently, these variables were iteratively eliminated one by one, starting with those exhibiting the highest p-values, until only variables with a p-value <0.05 remained in the model. This study was approved by the Research Ethics Committee of the Universidade Federal de Minas Gerais (reference 126/07) in Belo Horizonte, Brazil, and all participants or their legal representatives signed the informed consent forms.

### Results

#### Participant characteristics
Of the 602 individuals evaluated, 394 (65.4%) reported no lifetime consumption of alcoholic beverages, while 208 (34.6%) indicated prior consumption. Within the group of individuals with a history of previous alcohol consumption, 74 (12.3% of the total sample) continued drinking alcohol up to the evaluation and 134 (22.3% of the total sample) who had ceased the habit were classified as former drinkers. All individuals currently consuming alcohol had a history of previous alcohol consumption. With respect to dose, the average current alcohol intake was 5.82 units per week and the average past consumption was 6.40 units per week. The mean duration of alcohol consumption was 25.13 years, considering all patients with a history of alcohol use. Current alcohol abuse and dependence were diagnosed in 11 (1.8% of the total sample) and 8 individuals (1.3% of the total sample), respectively.

Among the 208 participants with previous alcohol consumption, 186 could be categorized based on the past weekly alcohol consumption, while this information was unavailable for 22 participants. In total, 102 participants (16.9% of the total sample) were categorized as having engaged in previous light consumption (0.1–7 doses per week), 32 (5.3% of the total sample) as moderate consumption (7.1–14 doses per week), and 52 (8.6% of the total sample) as heavy consumption (>14 doses per week). The remaining 394 participants (65.4%), who denied alcohol consumption throughout their lives, were classified as abstainers. Among the 74 participants (12.3% of the total sample) with current alcohol consumption, 59 (9.8% of the total sample) were categorized as light consumption, 10 (1.7% of the total sample) as moderate consumption, and 3 (0.5% of the total sample) as heavy consumption. This information was unavailable for two participants.

**Table 1.** Association between alcohol consumption and cognitive diagnoses

| Variables | Groups (cognitive diagnoses) | | | |
|---|---|---|---|---|
| | NCI | CIND | Dementia | *p*-Value |
| **Previous consumption of alcoholic beverages**[a] | | | | 0.109 |
| No (394) | 174(60.8%) | 113(70.6%) | 107(68.6%) | |
| Yes (208) | 112(39.2%) | 47(29.4%) | 49(31.4%) | |
| Total (602) | 286 | 160 | 156 | |
| **Categories – previous consumption of alcoholic beverages**[a] | | | | 0.523 |
| Abstainers (394) | 174(63.7%) | 113(72%) | 107(71.3%) | |
| Light (102) | 57(20.9%) | 23(14.6%) | 22(14.7%) | |
| Moderate (32) | 18(6.6%) | 8(5.1%) | 6(4%) | |
| Heavy (52) | 24(8.8%) | 13(8.3%) | 15(10%) | |
| Total (580) | 273 | 157 | 150 | |
| **Current consumption of alcoholic beverage**[a] | | | | 0.002[b] |
| No (528) | 238(83.2%) | 142(88.8%) | 148(94.9%)[+] | |
| Yes (74) | 48(16.8%)[+] | 18(11.3%) | 8(5.1%) | |
| Total (602) | 285 | 160 | 156 | |
| **Categories – current consumption of alcoholic beverage**[c] | | | | 0.009[b] |
| Abstainers + former drinkers (528) | 238(83.5%) | 142(89.3%) | 148(94.9%)[+] | |
| Light (59) | 39(13.7%)[+] | 13(8.2%) | 7(4.5%) | |
| Moderate (10) | 7(2.5%) | 2(1.3%) | 1(0.6%) | |
| Heavy (3) | 1(0.4%) | 2(1.3%) | 0(0%) | |
| Total (600) | 285 | 159 | 156 | |
| **Categories – current consumption of alcoholic beverage**[c] | | | | 0.013[b] |
| Abstainers (394) | 174(61.1%) | 113(71.1%) | 107(68.6%) | |
| Former drinkers (134) | 64(22.5%) | 29(18.2%) | 41(26.3%) | |
| Light (59) | 39(13.7%)[+] | 13(8.2%) | 7(4.5%) | |
| Moderate (10) | 7(2.5%) | 2(1.3%) | 1(0.6%) | |
| Heavy (3) | 1(0.4%) | 2(1.3%) | 0(0%) | |
| Total (600) | 285 | 159 | 156 | |
| **Type of alcoholic beverage** | | | | |
| Beer[a] | | | | 0.307 |
| No (104) | 52(50%) | 24(55.8%) | 28(63.6%) | |
| Yes (87) | 52(50%) | 19(44.2%) | 16(36.4%) | |
| Total (191) | 104 | 43 | 44 | |
| Cachaça[a] | | | | 0.020[b] |
| No (52) | 37(35.9%)[+] | 7(16.3%) | 8(19%) | |
| Yes (136) | 66(64.1%) | 36(83.7%) | 34(81%) | |
| Total (188) | 103 | 43 | 42 | |
| Whisky[c] | | | | 1.000 |
| No (176) | 95(93.1%) | 41(95.3%) | 40(93%) | |
| Yes (12) | 7(6.9%) | 2(4.7%) | 3(7%) | |
| Total (188) | 102 | 43 | 43 | |
| Wine[a] | | | | 0.140 |
| No (132) | 66(64.7%) | 34(81%) | 32(72.7%) | |
| Yes (56) | 36(35.3%) | 8(19%) | 12(27.3%) | |

*(Continued)*

**Table 1.** *Continued*

| Variables | Groups (cognitive diagnoses) | | | |
| | NCI | CIND | Dementia | *p*-Value |
|---|---|---|---|---|
| Total (188) | 102 | 42 | 44 | |
| Other types[c] | | | | 0.885 |
| No (181) | 98(96.1%) | 40(95.2%) | 43(97.7%) | |
| Yes (7) | 4(3.9%) | 2(4.8%) | 1(2.3%) | |
| Total (188) | 102 | 42 | 44 | |

Abbreviations: NCI, no cognitive impairment; CIND, cognitive impairment no dementia.
[a]Chi-square test.
[b]p < 0.05; +significant association; frequency (%).
[c]Fisher's exact test.

**Table 2.** Final model of ordinal logistic regression for the cognitive diagnoses

| Variables | Estimate | p-Value | OR | 95%CI |
|---|---|---|---|---|
| Age | 0.129 | <0.001 | 1.14 | 1.06–1.21 |
| *Cachaça* | 0.925 | 0.009 | 2.52 | 1.25–5.04 |
| Schooling | −0.062 | 0.043 | 0.94 | 0.89–0.99 |
| Absence of current alcohol consumption | 0.844 | 0.001 | 2.34 | 1.39–3.90 |
| Sex (female) | 0.387 | 0.020 | 1.47 | 1.06–2.04 |

Abbreviations: OR: *odds ratio*; CI: confidence interval.

**Table 3.** Association between alcohol consumption and scores on functional assessment instruments

| Alcohol consumption | Previous consumption | | | Current consumption | | |
| | Yes | No | p-Value | Yes | No | p-Value |
|---|---|---|---|---|---|---|
| BOMFAQ[1] | 16(14–22) | 18(14–25.25) | 0.040* | 14(14–17) | 18(14–25) | <0.001* |
| | n = 202 | n = 382 | | n = 73 | n = 511 | |
| FAQ[1] | **1**(0–7) | 1(0–6) | 0.896 | 0(0–2) | 1(0–7) | 0.001* |
| | n = 194 | n = 381 | | n = 73 | n = 502 | |
| FAST[1] | 2(1–3) | 3(1–4) | 0.088 | 2(1–3) | 3(1–4) | <0.001* |
| | n = 198 | n = 367 | | n = 70 | n = 495 | |

Notes: [1]Mann–Whitney test; [1]Higher scores reflect lower functioning; *p < 0.05; Median (1° quartile–3° quartile).
Abbreviations: BOMFAQ, Brazilian Older Americans Resource and Services Multidimensional Functional Assessment Questionnaire; FAQ, Functional Activity Questionnaire; FAST, Functional Assessment Staging in Alzheimer's Disease.

### Association between alcohol consumption and cognitive diagnosis

As shown in Table 1, no significant associations emerged between prior alcohol consumption (208 patients) and cognitive diagnoses, both when alcohol consumption was considered as a dichotomous variable and when categorized by weekly alcohol dose (see Table 1).

Irrespective of past alcohol use (former drinkers or abstainers), no current alcohol consumption was associated with dementia. Furthermore, current alcohol consumption was significantly associated with NCI. When alcohol was categorized by weekly alcohol dose, light alcohol consumption showed a significant association with NCI (see Table 1).

In terms of the type of alcoholic beverages consumed in the past, 136 (72.3%) reported *cachaça* consumption, 87 (45.5%) consumed beer, 56 (29.8%) consumed wine, 12 (6.4%) consumed whisky, and 7 (3.7%) consumed other types of alcoholic beverage. Only the

absence of *cachaça* consumption was associated with the absence of cognitive impairment (see Table 1).

For the multivariate analysis, ordinal logistic regression was employed, initially incorporating variables with a p-value below 0.20 from the univariate analysis (past consumption of wine and *cachaça*; current and previous alcohol consumption as dichotomous variables; categories of current alcohol consumption) involving the main outcome (cognitive diagnoses) and in the demographic data (age, education, marital status, and sex).

After the multivariate analysis, *cachaça* consumption, age, schooling, absence of current alcohol consumption, and female sex remained statistically significant in the model. Specifically, *cachaça* consumption increased the chance of the individual being in the dementia group compared to the CIND and NCI groups by 2.52 (95%CI: 1.25–5.04) times. Each additional year of age raised the chance of being in the dementia group by 1.14 times (95%CI: 1.06–1.21). An increment of 1 year of schooling was associated with

**Table 4.** Distribution of the occurrence of psychiatric and neurological disorders according to previous/current alcohol consumption and the association between these variables

| Psychiatric/neurological diagnosis | Previous alcohol consumption | | |
| --- | --- | --- | --- |
| | Present (n = 208) | Absent (n = 394) | *p*-Value |
| Stroke (54)[a] | 21(10.1%) | 33(8.4%) | 0.482 |
| Hydrocephalus (2)[b] | 0(0%) | 2(0.5%) | 0.547 |
| TBI (10)[b] | 6(2.9%) | 4(1%) | 0.102 |
| Epilepsy (16)[a] | 7(3.4%) | 9(2.3%) | 0.442 |
| Current major depression (67)[a] | 18(8.7%) | 49(12.5%) | 0.160 |
| Recurrent major depression (25)[a] | 8(3.9%) | 17(4.3%) | 0.784 |
| Current melancholic major depression (20)[a] | 6(2.9%) | 14(3.6%) | 0.663 |
| Current dysthymia (43)[a] | 11(5.3%) | 32(8.2%) | 0.199 |
| Parkinsonism (66)[a] | 21(10.1%) | 45(11.5%) | 0.620 |
| Current psychosis (10)[b] | 2(1%) | 8(2%) | 0.506 |
| Lifelong psychosis (10)[b] | 2(1%) | 8(2.1%) | 0.506 |
| Current psychosis mood disorder (5)[b] | 2(1%) | 3(0.8%) | 1.000 |
| Lifelong psychosis mood disorders (5)[b] | 2(1%) | 3(0.8%) | 1.000 |
| Current GAD (20)[a] | 7(3.4%) | 13(3.3%) | 0.971 |
| Psychiatric/neurological diagnosis | Current alcohol consumption | | |
| | Present (n = 74) | Absent (n = 528) | *p*-Value |
| Stroke (54)[a] | 1(1.4%) | 53(10%)+ | 0.014[c] |
| Hydrocephalus (2)[b] | 0(0%) | 2(0.4%) | 0.769 |
| TBI (10)[b] | 0(0%) | 10(1.9%) | 0.602 |
| Epilepsy (16)[b] | 0(0%) | 16(3%) | 0.241 |
| Current major depression (67)[a] | 2(2.7%) | 65(12.4%)+ | 0.013[c] |
| Recurrent major depression (25)[b] | 2(2.7%) | 23(4.4%) | 0.757 |
| Current melancholic major depression (20)[b] | 1(1.4%) | 19(3.6%) | 0.494 |
| Current dysthymia (43)[a] | 3(4.1%) | 40(7.6%) | 0.266 |
| Parkinsonism (66)[a] | 3(4.1%) | 63(12%)+ | 0.041[c] |
| Current psychosis (10)[b] | 0(0%) | 10(1.9%) | 0.621 |
| Lifelong psychosis (10)[b] | 0(0%) | 10(1.9%) | 0.621 |
| Current psychosis mood disorder (5)[b] | 0(1%) | 5(1%) | 1.000 |
| Lifelong psychosis mood disorders (5)[b] | 2(1%) | 3(0.8%) | 1.000 |
| Current GAD (20)[b] | 4(3.1%) | 16(5.4%) | 0.295 |

Abbreviations: TBI, traumatic brain injury; current GAD, current generalized anxiety disorder.
[a]Pearson's chi-square test.
[b]Fisher's exact test.
[c]p < 0.05; +significant association; frequency (%).

a 0.94-fold (95%CI: 0.89–0.99) chance of being in the dementia group compared to the NCI and CIND groups. The absence of current alcohol consumption is linked with a 2.34 (95%CI: 1.39–3.90) chance of being in the dementia group compared to the NCI and CIND groups. The female sex was associated with a 1.47-fold (95%CI 1.06–2.04) chance of being in the dementia group compared to the NCI and CIND groups (see Table 2).

### Association between alcohol consumption and functionality

Participants who continued their current alcohol consumption exhibited better functionality compared to a combined group of abstainers and former drinkers, as indicated by scores on the FAST, BOMFAQ, and FAQ scales (see Table 3). Previous consumption of alcoholic beverages was significantly associated with enhanced functionality using the BOMFAQ questionnaire. However, no significant associations were observed between previous alcohol consumption and functionality based on the other questionnaires.

### Association between alcohol consumption and other psychiatric and neurological diagnoses

The association between prior alcohol consumption and other psychiatric and neurological diagnoses were assessed through

**Table 5.** Frequency of psychiatric and neurological disorders according to categories of previous/current alcohol consumption and association between these variables

| Psychiatric/neurological diagnosis | Previous alcohol consumption | | | | |
| --- | --- | --- | --- | --- | --- |
| | Abstainers (n = 394) | Light (n = 102) | Moderate (n = 32) | Heavy (n = 52) | *p*-Value |
| Stroke (53)[a] | 33(8.4%) | 11(10.8%) | 3(9.4%) | 6(11.5%) | 0.721 |
| Hydrocephalus (2)[a] | 2(0.5%) | 0(0%) | 0(0%) | 0(0%) | 1.000 |
| TBI (10)[a] | 4(1%) | 2(2%) | 0(0%) | 4(7.7%)[+] | 0.026[b] |
| Epilepsy (16)[a] | 9(2.3%) | 3(2.9%) | 0(0%) | 4(7.7%) | 0.167 |
| Current major depression (66)[c] | 49(12.5%) | 10(9.8%) | 2(6.3%) | 5(9.6%) | 0.631 |
| Recurrent major depression (24)[a] | 17(4.3%) | 5(4.9%) | 1(3.1%) | 1(1.9%) | 0.920 |
| Current melancholic major depression (19)[a] | 14(3.6%) | 3(2.9%) | 1(3.1%) | 1(1.9%) | 1.000 |
| Current dysthymia (42)[a] | 32(8.2%) | 7(6.9%) | 0(0%) | 3(5.8%) | 0.411 |
| Parkinsonism (66)[c] | 45(11.5%) | 10(9.9%) | 2(6.3%) | 9(17.3%) | 0.446 |
| Current psychosis (10)[a] | 8(2%) | 2(2%) | 0(0%) | 0(0%) | 0.868 |
| Lifelong psychosis (10)[a] | 8(2.1%) | 2(2%) | 0(0%) | 0(0%) | 0.869 |
| Current psychosis mood disorder (5)[a] | 3(0.8%) | 2(2%) | 0(0%) | 0(0%) | 0.571 |
| Lifelong psychosis mood disorders (5)[a] | 3(0.8%) | 2(2%) | 0(0%) | 0(0%) | 0.571 |
| Current GAD (20)[a] | 13(3.3%) | 4(3.9%) | 0(0%) | 3(5.8%) | 0.575 |

| Psychiatric/neurological diagnosis | Current alcohol consumption | | | | | |
| --- | --- | --- | --- | --- | --- | --- |
| | Abstainers (n = 394) | Former drinkers (n = 134) | Light (n = 59) | Moderate (n = 10) | Heavy (n = 3) | *p*-Value |
| Stroke (53)[a] | 33(8.4%) | 20(14.9%)+ | 1(1.7%) | 0(0%) | 0(0%) | 0.030[b] |
| Hydrocephalus (2)[a] | 2(0.5%) | 0(0%) | 0(0%) | 0(0%) | 0(0%) | 1.000 |
| TBI (10)[a] | 4(1%) | 6(4.5%) | 0(0%) | 0(0%) | 0(0%) | 0.100 |
| Epilepsy (16)[a] | 9(2.3%) | 7(5.2%) | 0(0%) | 0(0%) | 0(0%) | 0.254 |
| Current major depression (66)[a] | 49(12.5%) | 16(12%) | 2(3.4%) | 0(0%) | 0(0%) | 0.213 |
| Recurrent major depression (24)[a] | 17(4.3%) | 6(4.5%) | 2(3.4%) | 0(0%) | 0(0%) | 1.000 |
| Current melancholic major depression (20)[a] | 14(3.6%) | 5(3.8%) | 1(1.7%) | 0(0%) | 0(0%) | 0.922 |
| Current dysthymia (43)[a] | 32(8.2%) | 8(6%) | 3(5.1%) | 0(0%) | 0(0%) | 0.812 |
| Parkinsonism (66)[a] | 45(11.5%) | 18(13.5%) | 2(3.4%) | 0(0%) | 1(33.3%) | 0.101 |
| Current psychosis (10)[a] | 8(2%) | 2(1.5%) | 0(0%) | 0(0%) | 0(0%) | 0.906 |
| Lifelong psychosis (10)[a] | 8(2.1%) | 2(1.5%) | 0(0%) | 0(0%) | 0(0%) | 0.906 |
| Current psychosis mood disorder (5)[a] | 3(0.8%) | 2(1.5%) | 0(0%) | 0(0%) | 0(0%) | 0.793 |
| Lifelong psychosis mood disorders (5)[a] | 3(0.8%) | 2(1.5%) | 0(0%) | 0(0%) | 0(0%) | 0.793 |
| Current GAD (20)[a] | 13(3.3%) | 3(2.3%) | 4(6.8%) | 0(0%) | 3(5.8%) | 0.453 |

Abbreviation: current GAD, current generalized anxiety disorder.
[a]Fisher's exact test.
[b]p < 0.05; +significant association; frequency (%).
[c]Pearson's chi-square test.

clinical history and MINI, but no findings emerged (see Table 4). In contrast, among the group with current alcohol consumption, associations were observed between the absence of alcohol consumption and stroke, parkinsonism, and depression (see Table 4).

When participants were categorized according to the amount of alcohol consumed in the past, a statistically significant association was established between traumatic brain injury and heavy drinking, with a significance level of 5% (see Table 5). Similarly, categorizing participants by their current alcohol consumption revealed an association between diagnosis of stroke and former drinkers (see Table 5).

## Association between alcohol consumption, cognitive diagnosis, and APOE genotyping

Regarding *APOE* genotyping, the distribution observed was as follows: 6 individuals with ε2ε2 genotype, 44 with ε2ε3 genotype, 11 with ε2ε4 genotype, 154 with ε3ε3 genotype, 73 with ε3ε4 genotype, and 7 with ε4ε4 genotype (see Table 6). No association

**Table 6.** Distribution of *APOE* polymorphism according to previous alcohol consumption and cognitive diagnoses

| Variables | | Groups (cognitive diagnoses) | | | |
|---|---|---|---|---|---|
| | | NCI | CIND | Dementia | *p*-Value |
| APOE | Previous alcohol consumption | | | | |
| ε2ε2[a] | No (4) | 1(50%) | 2(100%) | 1(50%) | 1.000 |
| | Yes (2) | 1(50%) | 0(0%) | 1(50%) | |
| | Total (6) | 2 | 2 | 2 | |
| ε2ε3[a] | No (27) | 16(55.2%) | 7(77.8%) | 4(66.7%) | 0.484 |
| | Yes (17) | 13(44.8%) | 2(22.2%) | 2(33.3%) | |
| | Total (44) | 29 | 9 | 6 | |
| ε2ε4[a] | No (6) | 3(50%) | 2(66.7%) | 1(50%) | 1.000 |
| | Yes (5) | 3(50%) | 1(33.3%) | 1(50%) | |
| | Total (11) | 6 | 3 | 2 | |
| ε3ε3[b] | No (97) | 52(61.2%) | 29(64.4%) | 16(66.7%) | 0.873 |
| | Yes (57) | 33(38.8%) | 16(35.6%) | 8(33.3%) | |
| | Total (154) | 85 | 45 | 24 | |
| ε3ε4[b] | No (49) | 21(66.7%) | 17(70.8%) | 11(61.1%) | 0.817 |
| | Yes (24) | 10(32.3%) | 7(29.2%) | 7(38.9%) | |
| | Total (73) | 31 | 24 | 18 | |
| ε4ε4[a] | No (6) | 1(100%) | 2(100%) | 3(75%) | 1.000 |
| | Yes (1) | 0(0%) | 0(0%) | 1(25%) | |
| | Total (7) | 1 | 2 | 4 | |

Abbreviations: NCI, no cognitive impairment; CIND, cognitive impairment no dementia; APOE, apolipoprotein E.
[a]Fisher's exact test; frequency (%).
[b]Chi-square test.

was found between the presence of the ε4 allele, cognitive diagnoses, and previous or current alcohol consumption (see Table 7).

## Discussion

This study aimed to investigate the associations between current and previous alcohol consumption and cognitive impairment in an older adult population living in the community of a middle-income country. The absence of current alcohol consumption was associated with a diagnosis of dementia.

The absence of current alcohol consumption, encompassing both abstainers and former drinkers, was not only associated with dementia but also correlated with poorer functionality, stroke diagnosis, current major depression, and parkinsonism. These findings may be compatible with the hypothesis of a neuroprotective effect of alcohol suggested by some studies (Cooper et al., 2009; Mukamal et al., 2003; Orgogozo et al., 1997; Peters et al., 2008; Ruitenberg et al., 2002; Sabia et al., 2018; Salvador et al., 2022; Xu et al., 2017) or reflect the tendency of less healthy individuals to abstain from alcohol. Indeed, the higher prevalence of stroke among former drinkers specifically supports the notion that participants who are unwell or cognitively impaired may choose to cease alcohol consumption as a compensatory measure.

Surprisingly, no significant association was observed between the previous habit of alcohol consumption (dichotomous variable) or its categories (abstainers, light, moderate, and heavy consumption) and cognitive diagnosis. This null association could be attributed to a survival effect. Since the study focused on individuals aged

75+, it is plausible that those exposed to prolonged and excessive alcohol consumption in the past might have passed away before reaching this age. Others might have experienced cognitive recovery after discontinuing alcohol use. Additionally, recall bias in self-reported alcohol consumption and historical alcohol dose could have contributed to these findings.

While previous research has indicated that light to moderate alcohol consumption can reduce dementia risk and excessive consumption or abstention might heighten it (Rehm, Hasan, Black, Shield, & Schwarzinger, 2019), the current results contrastingly align with a large study in individuals with a median age of 78 years, which similarly found no association between prior alcohol consumption and dementia (Koch et al., 2019).

In the univariate analysis, current light alcohol consumption demonstrated a significant association with NCI. However, this association lost significance after accounting for covariates such as age, sex, and education. This raises the emerging concern that the apparent protective effect of light to moderate alcohol use might be confounded by factors such as socioeconomic status and cognitive reserve. A U.K. study involving individuals aged 60–74 years, excluding those with an AUDIT score equal to or greater than 8 (i.e., alcohol consumption that can cause problems), found no cognitive benefits from moderate alcohol consumption after adjusting for premorbid intelligence and health status (Cooper et al., 2009).

In the present study, no interaction emerged between previous or current alcohol consumption, the presence of the *APOE* ε4 allele and cognitive diagnoses. This interaction, although observed in other studies (Anttila et al., 2004), is still conflicting.

**Table 7.** Association between previous and current alcohol consumption, cognitive profile, and APOE ε4 allele

| | Variables | Groups (cognitive diagnoses) | | | |
| --- | --- | --- | --- | --- | --- |
| | | NCI | CIND | Dementia | *p*-Value |
| APOE | Previous alcohol consumption (295) | (154) | (85) | (56) | |
| No ε4 allele[a] (204) | No (128) | 69(59.5%) | 38(67.9%) | 21(65.6%) | 0.531 |
| | Yes (76) | 47(40.5%) | 18(32.1%) | 11(34.4%) | |
| With ε4 allele[a] (91) | No (61) | 25(65.8%) | 21(72.4%) | 15(62.5%) | 0.730 |
| | Yes (30) | 13(34.2%) | 8(27.6%) | 9(37.5%) | |
| *APOE* | Categories – previous consumption (284) | (146) | (84) | (54) | |
| No ε4 allele[b] (196) | Abstainers (128) | 69(62.2%) | 38(69.1%) | 21(70%) | 0.874 |
| | Light (37) | 22(19.8%) | 10(18.2%) | 5(16.7%) | |
| | Moderate (14) | 8(7.2%) | 3(5.5%) | 3(10%) | |
| | Heavy (17) | 12(10.8%) | 4(7.3%) | 1(3.3%) | |
| With ε4 allele[b] (88) | Abstainers (61) | 25(71.4%) | 21(72.4%) | 15(62.5%) | 0.885 |
| | Light (17) | 7(20%) | 5(17.2%) | 5(20.8%) | |
| | Moderate (4) | 1(2.9%) | 2(6.9%) | 1(4.2%) | |
| | Heavy (6) | 2(5.7%) | 1(3.4%) | 3(12.5%) | |
| *APOE* | Current alcohol consumption (295) | (154) | (85) | (56) | |
| No ε4 allele[a] (204) | No (174) | 99(85.3%) | 46(82.1%) | 29(90.6%) | 0.557 |
| | Yes (30) | 17(14.7%) | 10(17.9%) | 3(9.4%) | |
| With ε4 allele[b] (91) | No (79) | 30(78.9%) | 26(89.7%) | 23(95.8%) | 0.197 |
| | Yes (12) | 8(21.1%) | 3(10.3%) | 1(4.2%) | |
| *APOE* | Categories – current alcohol consumption (294) | (153) | (85) | (56) | |
| No ε4 allele[b] (203) | Abstainers (174) | 99(81.6%) | 46(82.1%) | 22(90.6%) | 0.659 |
| | Light (22) | 12(10.4%) | 8(14.3%) | 2(6.3%) | |
| | Moderate (6) | 4(3.5%) | 1(1.8%) | 1(3.1%) | |
| | Heavy (1) | 0(0%) | 1(1.8%) | 0(0%) | |
| With ε4 allele[b] (91) | Abstainers (79) | 30(78.9%) | 26(89.7%) | 23(95.8%) | 0.113 |
| | Light (11) | 8(21.1%) | 2(6.9%) | 1(4.2%) | |
| | Moderate (1) | 0(0%) | 1(3.4%) | 0(0%) | |
| | Heavy (0) | 0(0%) | 0(0%) | 0(0%) | |

Abbreviations: NCI, no cognitive impairment; CIND, cognitive impairment no dementia; *APOE*, apolipoprotein E.
[a]Chi-square test.
[b]Fisher's exact test; frequency (%).

Cachaça consumption increased the chance of the individual being in the dementia group in comparison to CIND and NCI groups by 2.52 times. Cachaça is a typical drink in the Brazilian territory, especially in smaller towns in the interior of the country, which correspond to more than 80% of the municipalities in Brazil. No data were found regarding the association between this type of alcoholic beverage and cognition in the scientific literature. It is possible that the association between cachaça and dementia stems from its higher alcohol content (around 40%). Given its affordability and widespread accessibility, it is disproportionately consumed by alcohol abusers in Brazil. Furthermore, other high-alcohol-content drinks possibly lacked representation in this population studied, which could explain the absence of associations with dementia.

The study sample displayed a lower prevalence of previous alcohol consumption (34.6%) in comparison to the global prevalence among adults (43%) (World Health Organization, 2020). The prevalence of current alcohol consumption within this study was 12.3% (74 individuals), which suggests a lower consumption with advanced age. However, comprehensive epidemiological data regarding the oldest population segment are limited. The SABE Study in São Paulo, Brazil showed a weighted alcohol consumption prevalence of 31.9% among those aged 60+ years, and 25.3% for the 75–79 years' age group (Pinho, 2012). A recent study encompassing 3,021 community-dwelling individuals in the United States identified a 58% prevalence among those aged 72+ years (Koch et al., 2019). This suggests that the prevalence of alcohol consumption is highly variable in different countries and regions.

The current study boasts several strengths. This study shed light on the relationship between alcohol consumption and cognition in a relatively large sample of older adults from a middle-income

country. This is particularly relevant with important public health implications considering the rising life expectancy of persons in low- and middle-income countries and widespread alcohol consumption. Furthermore, the study's participants underwent a complete clinical and cognitive evaluation by an experienced team of researchers, supplemented by ancillary tests for cognitive diagnosis. Finally, the study sampled the 75+ age group, a rapidly growing segment in low- and middle-income countries, yet one with limited population-based data.

In addition to study strengths, certain limitations warrant consideration. The use of self-reporting measurements is susceptible to recall bias, and participants might intentionally or unintentionally under-report the amount of alcohol typically ingested. The effects of chronic alcohol use depend on the amount, frequency, and type of drink, as well as the presence of comorbidities, which was not accounted for due to the study's observational nature. Furthermore, multiple tests and comparisons without correction may have increased risk of Type I error. Finally, the cross-sectional design precludes establishing a causal relationship between alcohol consumption and health outcomes.

In summary, this study underscores that the absence of current alcohol consumption in older adults aged 75+ years is linked to dementia diagnosis. However, no association emerged between past alcohol consumption and cognitive diagnoses, or between different categories of alcohol consumption – current or previous – and cognitive diagnoses. Notably, previous consumption of cachaça was significantly associated with dementia. Interestingly, those who currently consumed alcoholic beverages exhibited superior functionality.

**Supplementary material.** The supplementary material for this article can be found at http://doi.org/10.1017/S0714980824000126.

**Competing interest.** The authors declare no competing interests exist.

## References

Almeida, O. P., & Almeida, S. A. (1999). Confiabilidade da versão brasileira da escala de depressão em geriatria (GDS) versão reduzida. *Arquivos de Neuropsiquiatria*, **57**(2B), 421–426. https://doi.org/10.1590/S0004-282X1999000300013.

American Psychiatric Association. (1987). *Diagnosis and statistical manual of mental disorders: DSM-III* (3rd ed.). Washington, DC: American Psychiatric Association.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders: DSM-IV* (4th ed.). Washington, DC: American Psychiatric Association.

Anttila, T., Helkala, E. L., Viitanen, M., Kareholt, I., Fratiglioni, L., Winblad, B., et al. (2004). Alcohol drinking in middle age and subsequent risk of mild cognitive impairment and dementia in old age: A prospective population based study. *BMJ*, **329**(7465), 539. https://doi.org/10.1136/bmj.38181.418958.BE.

Associação Brasileira de Empresas de Pesquisa (ABEP). (2003). Critério de classificação Econômica Brasil. Retrieved 12 July 2007 from https://www.abep.org.

Beato, R. G., Nitrini, R., Formigoni, A. P., & Caramelli, P. (2007). Brazilian version of the Frontal Assessment Battery (FAB): Preliminary data on administration to healthy elderly. *Dementia & Neuropsychogia*, **1**(1), 59–65. https://doi.org/10.1590/S1980-57642008DN10100010.

Bertolucci, P. H., Okamoto, I. H., Brucki, S. M., Siviero, M. O., Toniolo Neto, J., & Ramos, L. R. (2001). Applicability of the CERAD neuropsychological battery to Brazilian elderly. *Arquivos de Neuropsiquiatria*, **59**(3-A), 532–536. https://doi.org/10.1590/S0004-282X2001000400009.

Boff, M. S., Sekyia, F. S., & Bottino, C. M. C. (2015). Prevalence of dementia among Brazilian population: Systematic review. *Revista de Medicina (São Paulo)*, **94**(3), 154–161. https://doi.org/10.11606/issn.1679-9836.v.94i3p154-161.

Brucki, S. M. D., Nitrini, R., Caramelli, P., Bertolucci, P. H. F., & Okamoto, I. H. (2003). Sugestões para o uso do Mini Exame do Estado Mental no Brasil. *Arquivos de Neuropsiquiatria*, **61**, 777–781. https://doi.org/10.1590/S0004-282X2003000500014.

Caramelli, P., Barbosa, M. T., Sakurai, E., Santos, E. L., Beato, R. G., Machado, J. C. B., et al. (2011). The Pietà study: Epidemiological investigation on successful brain aging in Caeté (MG), Brazil. Methods and baseline cohort characteristics. *Arquivos de Neuropsiquiatria*, **69**(4), 579–584. https://doi.org/10.1590/S0004-282X2011000500002

Cooper, C., Bebbington, P., Meltzer, H., Jenkins, R., Brugha, T., Lindesay, J. E., et al. (2009). Alcohol in moderation, premorbid intelligence and cognition in older adults: Results from the psychiatric morbidity survey. *Journal of Neurology, Neurosurgery & Psychiatry*, **80**(11), 1236–1239. https://doi.org/10.1136/jnnp.2008.163964.

Daviet, R., Aydogan, G., Jagannathan, K., Spilka, N., Koellinger, P. D., Kranzler, H. R. et al. (2022). Associations between alcohol consumption and gray white matter volumes in the UK Biobank. *Nature Communications*, **13**(1), 1175. https://doi.org/10.1038/s41467-022-28755-5.

Fahn, S., & Elton, R. L. (1987). UPDRS development committee: Unified Parkinson's disease rating scale. In S. Fahn, C. D. Marsden, D. B. Calne, & M. Goldstein (Eds.), *Recent developments in Parkinson's disease* (pp. 153–163). Florham Park: MacMillan.

Graham, J. E., Rockwood, K., Beattie, B. L., Eastwood, R., Gauthier, S., Tuokko, H., et al. (1997). Prevalence and severity of cognitive impairment with and without dementia in an elderly population. *Lancet*, **349**(9068), 1793–1796. https://doi.org/10.1016/S0140-6736(97)01007-6

Koch, M., Fitzpatrick, A. L., Rapp, S. R., Nahin, R. L., Williamson, J. D., Lopez, O. L., et al. (2019). Alcohol consumption and risk of dementia and cognitive decline among older adults with or without mild cognitive impairment. *JAMA*, **2**(9), e1910319. https://doi.org/10.1001/jamanetworkopen.2019.10319.

Lara, V. P., Caramelli, P., Teixeira, A. L., Barbosa, M. T., Carmona, K. C., Guimarães, H. C., et al. (2016). Cortisol, HDL-c, VLDL-c, and APOE polymorphisms as laboratorial parameters associated to cognitive impairment no dementia (CIND) and dementia. *Journal of Clinical Laboratory Analysis*, **30**, 374–380. https://doi.org/10.1002/jcla.21865.

Lecrubier, Y., Sheehan, D., Weiller, E., Amorim, P., Bonora, I., Sheehan, K. H., et al. (1997). The mini international neuropsychiatric interview (M.I.N.I), a short diagnostic interview: Reliability and validity according to the CIDI. *European Psychiatry*, **12**(5), 232–241. https://doi.org/10.1016/S0924-9338(97)83296-8.

Livingston, G., Huntley, J., Sommerlad, A., Ames, D., Ballard, C., Banerjee, S., et al. (2020). Dementia prevention, intervention, and care: 2020 report of the Lancet Commission. *Lancet*, **396**(10248), 413–446. https://doi.org/10.1016/S0140-6736(20)30367-6.

Lopes, M. A., Furtado, E. F., Ferrioli, E., Litvoc, J., & Bottino, C. M. C. (2010). Prevalence of alcohol-related problems in an elderly population and their association with cognitive impairment and dementia. *Alcoholism: Clinical and Experimental Research*, **34**, 726–733. https://doi.org/10.1111/j.1530-0277.2009.01142.x

Malloy-Diniz, L. F., Parreira Lasmar V. A., de Sena Rabelo Gazinelli, L., Fuentes, D., & Salgado, J. V. (2007). The rey auditory-verbal learning test: Applicability for Brazilian elderly population. *Revista Brasileira de Psiquiatria*, **29**(4), 324–329. https://doi.org/10.1590/S1516-44462006005000053.

Mukamal, K. J., Kuller, L. H., Fitzpatrick, A. L., Longstreth, W. T., Mittleman, M. A., & Siscovick, D. S. (2003). Prospective study of alcohol consumption and risk of dementia in older adults. *JAMA*, **289**(11), 1405–1413. https://doi.org/10.1001/jama.289.11.1405.

Nitrini, R., Lefèvre, B. H., Mathias, S. C., Caramelli, P., Carrilho, P. E. M., Sauaia, N., et al. (1994). Testes neuropsicológicos de aplicação simples para o diagnóstico de demência. *Arquivos de Neuropsiquiatria*, **52**(4), 457–465. https://doi.org/10.1590/S0004-282X1994000400001.

Orgogozo, J. M., Dartigues, J. F., Lafont, S., Letenneur, L., Commenges, D., Salamon, R., et al. (1997). Wine consumption and dementia in the elderly:

A prospective community study in the Bordeaux area. *Revue Neurologique (Paris)*, **153**(3), 185–192.

Peters, R., Peters, J., Warner, J., Beckett, N., & Bulpitt, C. (2008). Alcohol, dementia and cognitive decline in the elderly: A systematic review. *Age and Ageing*, **37**(5), 505–512. https://doi.org/10.1093/ageing/afn095.

Pfeffer, R. I., Kurosaki, T. T., Harrah, C. H., Chance, J. M., & Filos, S. (1982). Measurement of functional activities in older adults in the community. *Journal of Gerontology*, **37**(3), 323–329. https://doi.org/10.1093/geronj/37.3.323.

Pfeiffer, E. (1978). Multidimensional functional assessment the OARS methodology: A manual (2nd ed.). Center for the Study of Aging and Human Development, Duke University. Duke University Older Americans Resources and Services Program. Retrieved November 18 2022 from https://books.google.com/books?id=ObZqAAAAMAAJ.

Pinho, R. J. (2012). Prevalência e fatores associados ao uso de álcool entre idosos do município de São Paulo/SP, Estudo SABE[dissertation]. Botucatu: Universidade Estadual Paulista "Júlio de Mesquita Filho".

Porto, C. S., Fichman, H. C., Caramelli, P., Bahia, V. S., & Nitrini, R. (2003). Brazilian version of the Mattis dementia rating scale: diagnosis of mild dementia in Alzheimer's disease. *Arquivos de Neuropsiquiatria*, **61**(2B), 339–345. https://doi.org/10.1590/S0004-282X2003000300004.

Rao, R. T. (2018). Methodological difficulties of studying alcohol consumption and dementia. *BMJ*, **362**, k3894. https://doi.org/10.1136/bmj.k3894.

Rehm, J., Hasan, O. S., Black, S. E., Shield, K. D., & Schwarzinger, M. (2019). Alcohol use and dementia: A systematic scoping review. *Alzheimer's Research & Therapy*, **11**, 1. https://doi.org/10.1186/s13195-018-0453-0.

Reisberg, B. (1988). Functional assessment staging (FAST). *Psychopharmacology Bulletin*, **24**(4), 653–659.

Ridley, N. J., Draper, B., & Withall, A. (2013). Alcohol-related dementia: An update of the evidence. *Alzheimer's Research & Therapy*, **5**(1), 3. https://doi.org/10.1186/alzrt157.

Ruitenberg, A., van Swieten, J. C., Witteman, J. C. M., Mehta, K. M., van Duijn, C. M., Hofman, A. et al. (2002). Alcohol consumption and risk of dementia: The Rotterdam Study. *Lancet*, **359**(9303), 281–286. https://doi.org/10.1016/S0140-6736(02)07493-7.

Sabia, S., Fayosse, A., Dumurgier, J., Akbaraly, T., Britton, A., Kivimäki, M., et al. (2018). Alcohol consumption and risk of dementia: 23 year follow-up of Whitehall II cohort study. *BMJ*, **362**, k2927.

Sachdeva, A., Chandra, M., Choudhary, M., Dayal, P., & Anand, K.S. (2016). Alcohol-related dementia and neurocognitive impairment: A review study. *International Journal of High Risk Behaviors and Addiction*, **5**(3), e27976. https://doi.org/10.5812/ijhrba.27976

Salvador, L., Giatti, L., Viana, M. C., Suemoto, C. K., Ducan, B. B., Molina, M. et al. (2022). Sex differences in the association between alcohol intake and cognitive decline over 4 years in a middle-aged cohort: The Brazilian longitudinal study of adult health. *European Journal of Neurology*, **29**(7), 1903–1912.

Schwarzinger, M., Pollock, B. G., Hasan, O. S. M., Dufouil, C., & Rehm, J. (2018). Contribution of alcohol use disorders to the burden of dementia in France 2008-13: A nationwide retrospective cohort study. *Lancet Public Health*, **3**, e124–e132. https://doi.org/10.1016/S2468-2667(18)30022-7.

Suemoto, C. K., Mukadam, N., Brucki, S. M. D., Caramelli, P., Nitrini, R., Laks, J., et al. (2023). Risk factors for dementia in Brazil: Differences by region and race. *Alzheimers Dement*, **19**(5), 1849–1857. https://doi.org/10.1002/alz.12820.

Topiwala, A., Allan, C. L., Valkanova, V., Zsoldos, E., Filippini, N., Sexton, C., et al. (2017). Moderate alcohol consumption as risk factor for adverse brain outcomes and cognitive decline: Longitudinal cohort study. *BMJ*, **357**, j2353. https://doi.org/10.1136/bmj.j2353.

World Health Organization (WHO) & Alzheimer's Disease International. (2020). *Dementia*: A public health priority. Retrieved 5 October 2020 from https://www.who.int/news-room/fact-sheets/detail/dementia.

Xu, W., Wang, H., Wan, Y., Chenchen, T., Li, J., Tan, L., et al. (2017). Alcohol consumption and dementia risk: A dose-response meta-analysis of prospective studies. *European Journal of Epidemiology*, **32**(1), 31–42. https://doi.org/10.1007/s10654-017-0225-3.