Research Square

Preprints are preliminary reports that have not undergone peer review.
They should not be considered conclusive, used to inform clinical practice,
or referenced by the media as validated information.

# Alcohol and cognitive ageing: the gendered role of social connectedness

**Eva Teyssier**
Institute for Research on Socio-Economic Inequality (IRSEI), University of Luxembourg
**Dina Maskileyson**
Institute for Research on Socio-Economic Inequality (IRSEI), University of Luxembourg
**Anja Leist**
anja.leist@gero.uni-heidelberg.de

Heidelberg University

**Research Article**

**Keywords:** Alcohol, cognitive ageing, social networks, gender, SHARE, sustainable ageing

**Posted Date:** November 20th, 2026

**DOI:** https://doi.org/10.21203/rs.3.rs-11680128/v1

**License:** This work is licensed under a Creative Commons Attribution 4.0 International License. Read Full License

**Additional Declarations:** No competing interests reported.

## Abstract

Preserving cognitive function in later life is a precondition for autonomy and a central concern of sustainable ageing. The sometimes-observed cognitive “advantage” of low-to-moderate alcohol consumption remains unresolved. Because alcohol use in Europe is embedded in social practices, this study investigates whether social connectedness confounds or modifies its relationship with cognitive health. Using longitudinal data from the Survey of Health, Ageing and Retirement in Europe (SHARE), we followed older adults in 19 countries across an average of four waves over eight years. We applied gender-stratified linear mixed models to assess cognitive trajectories, recognising the gendered nature of drinking behaviours and social networks. Men were predominantly frequent drinkers, whereas women were more likely to abstain, and female abstention correlated with lower social integration. In fully adjusted models, introducing social connectedness attenuated the main effect of alcohol on cognitive scores by 10–17% in both genders, consistent with partial confounding by social factors. The longitudinal buffering effect of social integration, however, diverged by gender. For women, social connectedness buffered age-related cognitive decline, and this “protective” effect was magnified among moderate and frequent drinkers. In men, alcohol and social connectedness operated independently. Part of the cognitive benefit of social integration is therefore “masked” as an effect of drinking, and the mechanisms linking alcohol, social networks and cognition are gendered. In both cases, the modifiable resource these findings point to is social connection rather than alcohol use. Sustaining it supports independent living and reduces future demand on formal and informal care.


## Introduction

Population ageing raises the question of how societies can sustain the well-being and autonomy of older people without overburdening the generations that follow. Preserving cognitive function is central to that question; unimpaired cognitive function is a requirement for living independently and performing daily activities. Dementia is one of the leading causes of death and disability among older people (World Health Organization, 2026), but neurodegenerative illnesses are not an inevitable part of ageing. With no known cure, public health efforts focus on identifying modifiable risk factors to reduce the burden of dementia on the individual and society, framing prevention as a continuous, lifelong process (Listabarth et al., 2022; Livingston et al., 2024; World Health Organization, 2026).

Excessive alcohol consumption is one such modifiable risk factor, and global consensus indicates there is no safe amount of alcohol intake (Livingston et al., 2024; World Health Organization, 2024) (George et al., 2026; Livingston et al., 2024; World Health Organization, 2024). Nonetheless, observational studies frequently find that light-to-moderate alcohol intake is associated with a lower risk of dementia than no alcohol at all (Mewton et al., 2023; Wiegmann et al., 2020). These results remain heterogenous, lack confirmation by genetic analysis such as Mendelian randomisation, and are recognised to be subject to systematic selection bias or residual confounding (Anstey & Peters, 2018; Listabarth et al., 2022; Topiwala et al., 2025). Recent work by Listabarth et al. (2022) using data from the Survey of Health, Ageing and Retirement in Europe (SHARE) confirmed this cognitive advantage for moderate drinkers and found better baseline outcomes for women and highly educated individuals. However, the mechanisms behind this apparent “protection” remain unclear. We expand on their work in two ways: first, we apply a gender-stratified model.

This stratification is critical because biological sex, hormone exposure, and societal norms intersect to influence dementia risk (Livingston et al., 2024), yet gender and ethnic differences remain understudied (Erol & Karpyak,

2015; Listabarth et al., 2022; World Health Organization, 2026). Women typically show a higher age-adjusted incidence of dementia, and genders often operate with distinct drinking patterns and social network structures (Erol & Karpyak, 2015; Listabarth et al., 2022; World Health Organization, 2026). Analysing men and women together risks masking these distinct pathways.

Second, we introduce social connectedness as a potential lifestyle confounder, to clarify this paradoxical "protective" effect of alcohol use. In Western Europe, moderate drinking is correlated with a wide range of favourable social and lifestyle factors (Allen et al., 2018; S. E. Collins, 2016; Wiegmann et al., 2020). For older adults, drinking is linked with social engagement and active community participation (Giné-Garriga et al., 2021; Kelly et al., 2018; Kersey et al., 2022). Conversely, social isolation and loneliness are established risk factors for cognitive decline (Giné-Garriga et al., 2021; Livingston et al., 2024). Regular contact is hypothesised to preserve cognitive function through neural stimulation, stress reduction, and mitigated neuroinflammation (Arnhold et al., 2026; M. A. Collins et al., 2009). While this could be due to reverse causation, it also presents a plausible pathway: the apparent benefits of moderate drinking may simply be confounded by the true neuroprotective effect of the social engagement that accompanies social drinking (Kawakami et al., 2023; Kelly et al., 2018; Listabarth et al., 2022). If so, prevention efforts would do better to support social participation than to focus on alcohol alone.

We frame this study within the concept of sustainable ageing, understood as promoting the well-being, autonomy and social inclusion of older adults today without compromising the social, economic and institutional resources available to future generations (Arpino et al., 2026). Dementia, as a major cause of dependency in later life, is central to this agenda (World Health Organization, 2026). Cognitive health also has an inclusion dimension because protective resources, including social networks, are not equally distributed across social groups and may differ systematically by gender (Arnhold et al., 2026; Erol & Karpyak, 2015; Kersey et al., 2022; Livingston et al., 2024). Alcohol consumption is relevant to sustainable ageing for a different reason. Any apparent cognitive benefit associated with drinking must be considered alongside the substantial health and societal harms attributable to alcohol. If associations between moderate drinking and better cognitive outcomes are instead explained by the greater social connectedness of some drinkers, the appropriate target for intervention is the social conditions that support sustained relationships in later life.

This study contributes to the sustainable ageing agenda in two ways. *First*, it distinguishes social connectedness, a potentially modifiable and sustainable social resource, from alcohol consumption itself. *Second*, it examines whether social connectedness helps explain the association between alcohol consumption and cognitive health, and whether these relationships differ by gender, thereby identifying both the mechanism through which a potentially actionable social resource may operate and the groups for whom it may matter most.

We investigate these questions using longitudinal data from SHARE. Specifically, we address the question: *"To what extent is the observed association between moderate alcohol consumption and higher cognitive function explained by social connectedness, and does social connectedness modify the rate of cognitive decline?"*

We answer this question in two steps: first, we analyse the confounding effect of social connectedness, expecting the baseline cognitive "advantage" of alcohol to shrink once social integration is added (H1); second, we evaluate its buffering or moderating effect on cognitive trajectories over time, hypothesising that social connectedness flattens the curve of cognitive decline (H2). In addition, we apply a gender lens to these pathways, recognising that both drinking patterns and the structure of social networks are shaped by gendered

cohorts experiences and norms, and thus positing that both mechanisms operate distinctly for men and women (H3) (Arnhold et al., 2026; Erol & Karpyak, 2015). While women often maintain diverse, friend-based bridging networks that expose them to novel cognitive stimulation, men rely much more heavily on spouse-centred bonding ties for emotional and cognitive support (Arnhold et al., 2026).

## Data and methods

Study population

Data comes from the Survey of Health, Ageing and Retirement in Europe (SHARE), an ongoing longitudinal survey of a representative sample of the population aged 50 and older that started in 2004, with biennial assessment. The survey details have been published elsewhere (Börsch-Supan et al., 2013). This paper uses data from SHARE waves 1, 2, 4, 5, 6, 7, 8, and 9.

Our sample is composed of respondents who participated in at least two waves of SHARE, one of which in waves 1 to 5 as the alcohol frequency question was only asked then. Participants had to be over 50 at baseline, with no diagnosis of Alzheimer's disease. 19 of the 28 available countries are represented in our sample, as some countries only started participating in SHARE after wave 5 (see Fig. 1). Of the 159 267 respondents in our 8 waves, we were left with 75 770 eligible participants for analysis (see supplementary materials Figure S1)

**Figure 1** *Countries included in our analytical sample with number of participants per country*

Assessments and variables

The variable of interest, *cognitive function*, was assessed as a composite score of three different cognitive tests: verbal fluency (number of animals named in 60 seconds), immediate recall (immediate recall of as many words as possible from a list of 10 words) and delayed recall (recall of the same words after a standardised period of time). Following previous operationalisation (Bertogg & Leist, 2021; Leist et al., 2021; Listabarth et al., 2022), the summary score was created by averaging the standardized (at baseline) z-scores of those tests.

*Alcohol frequency* was assessed using a variable that captured frequency of intake over the past 6 months (waves 1 and 2) or the past 3 months (waves 4 and 5). The original seven harmonised categories ("almost every day", "5 or 6 days a week", "3 or 4 days a week", "once or twice a week", "once or twice a month", "less than once a month", and "not at all in the last 6 months") were tracked for each participant. Because this data was only available up to wave 5, we defined each participant's exposure by their maximum reported frequency across these waves, thus making *alcohol frequency* as a time-invariant variable. The variable was collapsed into 4 categories for analysis: "Abstainer", "Occasional (< 1 week)", "Moderate (1–4 days/week)", and "Frequent (5 + days/week)". Although this design categorises individuals who subsequently reduce their intake according to their highest historical consumption, this approach is preferred to underestimating early exposure and helps mitigate the confounding effects of the "sick quitter" bias.

*Social connectedness* is assessed using a composite measure scale created and validated in previous work (Litwin et al., 2013; Litwin & Stoeckel, 2015a, 2015b) based on the subjective mapping process of the participant's personal social networks. The social connectedness scale is based on 5 variables:

- Network size of the respondent

- Proximity of the network: the number of cited social network members living within 25km
- Contact frequency
- Support: the number of cited people with very close emotional ties
- Diversity: the number of different types of relationships present within the network

Each of the five variables is assigned a score from 0 to 4 and summed into a raw score (0–20). This total is then converted into a final social scale score ranging from 0 to 4 based on specific cut-offs (0 = 0, 1 = 1–5, 2 = 6–10, 3 = 11–15, and 4 = 16–20), with 4 corresponding to high connectedness (Litwin et al., 2013; Litwin & Stoeckel, 2015b). Within our analytical sample, the mean social connectedness score was 2.27 (SD = 0.89) (see Table 1). Because of the scarcity of reporting of this variable (only available in wave 6, 8 and 9), social connectedness is treated as a time-invariant trait in our model. To fix this baseline exposure across the longitudinal cognitive observation period, we utilised the maximum recorded value for each individual, capturing their highest reported capacity for social integration.

Selected based on established modifiable risk factors for dementia (Livingston et al., 2024), covariates could be either time-invariant or time-varying across waves. Time-invariant covariates included educational attainment (categorised via ISCED-97) and “ever smoked daily”. Time-varying covariates included age (centred at 65), living with partner, log-transformed income (adjusted for purchasing power parity), physical activity (coded by frequency of vigorous activities), and depression (measured by the 12-item EURO-D composite scale) (Börsch-Supan et al., 2013).

# Statistical analysis

To trace cognitive decline trajectories, we fitted linear mixed-effects models (LMMs) with random intercepts for unique ID, household ID, and country. Age entered linearly, centred at 65 years, without random slope. Practice effects were controlled via a dummy variable distinguishing the first test from subsequent tests (Vivot et al., 2016; Weuve et al., 2015).

To address longitudinal dropout bias, we applied stabilised inverse probability weights, predicting attrition using age, gender, education, drinking group, time in the study, and entry wave. Weights were stabilized using the ratio of marginal to conditional probabilities of remaining in the study. Missing social scale data (n = 18,219) were handled via multiple imputation, incorporating all analytical variables including baseline cognitive z-scores.

We tracked the alcohol coefficient using a 4-stage hierarchical structure: Model 1 (M1) estimated the alcohol-by-age interaction alongside covariates; Model 2 (M2) added social connectedness; Model 3 (M3) added chronic conditions; and Model 4 (M4) introduced a 3-way interaction (alcohol*age*social connectedness) to test modifying effects. To explore cross-national variation, an interaction with regional drinking culture was evaluated as a supplementary analysis. More details are provided in the Supplementary materials. Finally, robustness checks confirmed model stability by: excluding frequent drinkers (as heavy drinking is an established risk factor; Livingston et al., 2024) and testing binary drinking status; modelling social connectedness categorically (as done by Arnhold et al., 2026); testing individual random slopes for age and alternative practice effect adjustments; and verifying consistency across complete case, naïve unweighted, and restricted baseline models. Analyses were conducted in R (v. 4.6.2) utilising the lme4 (v. 2.0.1) package for modelling, mice (v. 3.19.0) for imputations, and tidyverse (v. 2.0.0) for data management.

# Results

Sample characteristics

Table 1 details the characteristics of the 75,770 individuals in the analytical sample (55.29% women, n = 41,896). At entry, the mean age was 64 years (SD = 10), with participants contributing an average of 4.41 assessment waves over a mean follow-up of 8.71 years (SD = 4.53). Alcohol consumption and social integration patterns diverged by gender: abstainers formed the largest group among women (32.03%; Table 1 & Fig. 2), whereas the share of men rose with drinking frequency and was largest among frequent drinkers (38.82%; Table 1 & Fig. 2). Furthermore, higher social connectedness corresponded to a lower likelihood of abstaining (Fig. 2). This inverse relationship was present in both sexes but more pronounced in women due to their higher overall abstention rate. Confirmed by the significant 3-way interactions between alcohol frequency, age, and gender ($p \leq 0.05$), analyses were stratified by gender. Finally, after LMMs inherently excluded missing covariates, the fully adjusted models utilised a final sample of 74,990 individuals (41,483 women and 33,507 men, see Figure S2 in supplementary materials for details).

Table 1
Participants characteristics by gender

| Characteristics | Overall N = 75,770[1] | Male N = 33,874[1] | Female N = 41,896[1] | p-value[2] |
|---|---|---|---|---|
| Education level | | | | **< 0.001** |
| Low | 31,570 (42.10%) | 12,620 (37.66%) | 18,950 (45.68%) | |
| Medium | 24,080 (32.11%) | 11,326 (33.80%) | 12,754 (30.74%) | |
| High | 19,345 (25.80%) | 9,561 (28.53%) | 9,784 (23.58%) | |
| Missing | 775 | 367 | 408 | |
| Age at entry | 64 (10) | 64 (9) | 64 (10) | 0.404 |
| Age at last assessment | 73 (10) | 73 (9) | 73 (10) | **< 0.001** |
| Alcohol consumption frequency | | | | **< 0.001** |
| Abstainer | 18,416 (24.31%) | 4,997 (14.75%) | 13,419 (32.03%) | |
| Occasional (< 1/wk) | 16,516 (21.80%) | 5,248 (15.49%) | 11,268 (26.90%) | |
| Moderate (1–4 days/wk) | 20,556 (27.13%) | 10,479 (30.94%) | 10,077 (24.05%) | |
| Frequent (5 + days/wk) | 20,282 (26.77%) | 13,150 (38.82%) | 7,132 (17.02%) | |
| Maximum social scale | 2.27 (0.89) | 2.15 (0.89) | 2.37 (0.88) | **< 0.001** |
| Missing | 18,219 | 8,590 | 9,629 | |
| Global cognition at entry | 0.00 (0.83) | -0.04 (0.80) | 0.03 (0.86) | **< 0.001** |
| Global cognition at last assessment | -0.14 (0.96) | -0.19 (0.91) | -0.11 (0.99) | **< 0.001** |
| Household size at entry | 2.23 (1.04) | 2.36 (1.04) | 2.13 (1.02) | **< 0.001** |
| Household size at last assessment | 1.93 (0.87) | 2.07 (0.86) | 1.81 (0.86) | **< 0.001** |
| Living with spouse/partner at entry | | | | **< 0.001** |
| Yes | 57,283 (75.60%) | 28,656 (84.60%) | 28,627 (68.33%) | |
| No | 18,487 (24.40%) | 5,218 (15.40%) | 13,269 (31.67%) | |
| Living with spouse/partner at last assessment | | | | **< 0.001** |

[2]Pearson's Chi-squared test; Wilcoxon rank sum test; Fisher's exact test

| Characteristics | Overall N = 75,770[1] | Male N = 33,874[1] | Female N = 41,896[1] | p-value[2] |
|---|---|---|---|---|
| Yes | 49,437 (65.25%) | 26,522 (78.30%) | 22,915 (54.69%) | |
| No | 26,333 (34.75%) | 7,352 (21.70%) | 18,981 (45.31%) | |
| Number of AD diagnosis since baseline | 1,980 (3.68%) | 848 (3.54%) | 1,132 (3.79%) | 0.122 |
| Missing | 21,909 | 9,892 | 12,017 | |
| Income per household | 35,129 (48,962) | 38,010 (48,757) | 32,799 (49,004) | **< 0.001** |
| Missing | 1,730 | 763 | 967 | |
| Euro Depression scale | 2.40 (2.21) | 1.89 (1.94) | 2.80 (2.33) | **< 0.001** |
| Missing | 31 | 17 | 14 | |
| Ever smoked daily | | | | **< 0.001** |
| No | 40,542 (53.59%) | 13,128 (38.81%) | 27,414 (65.55%) | |
| Yes | 35,108 (46.41%) | 20,702 (61.19%) | 14,406 (34.45%) | |
| Missing | 120 | 44 | 76 | |
| Number of chronic illnesses | 1.15 (1.22) | 1.16 (1.21) | 1.15 (1.23) | **0.038** |
| Missing | 43 | 12 | 31 | |

[1]n (%); Mean (SD)

[2]Pearson's Chi-squared test; Wilcoxon rank sum test; Fisher's exact test

Baseline associations: alcohol, social connectedness, and cognition

To evaluate if social engagement drives alcohol's apparent cognitive benefits, we constructed gender-stratified hierarchical LMMs on the imputed dataset. In the baseline adjusted models, reporting any drinking frequency was associated with significantly higher baseline cognitive score than abstinence (Table 2). Specifically, moderate drinking (1–4 days/week) was associated with a higher baseline cognitive score (in standard deviation units) for both women (β = 0.1667, 95% CI: 0.1506–0.1829, p ≤ 0.001) and men (β = 0.1217, 95% CI: 0.1011–0.1423, p ≤ 0.001). Results were similar for occasional drinking and frequent drinking (Table 2, Table S1 for full table in supplementary materials).

Introducing social connectedness attenuated these main effects (Table 2, Fig. 3). For moderate drinking, the coefficient shrank to β = 0.1464 (95% CI: 0.1303–0.1625) for women and β = 0.1039 (95% CI: 0.0834–0.1244) for men. Across all drinking categories, the alcohol coefficients decreased by 10–17% (calculated at the percentage change: [original β – new β]/ original β). While alcohol frequency maintained an independent, significant

association with baseline cognition, this attenuation is consistent with part of the unadjusted “protective” effect of alcohol being explained by social engagement. Adding chronic illnesses in a subsequent model did not significantly alter these main coefficients. The interaction between age and alcohol frequency is significant in all models for each possible alcohol frequency.

Table 2
Hierarchical Mixed Models of Global Cognition by Gender simplified

| Characteristic | General Model | | | + Social scale | | | + Chronic illness | | |
|---|---|---|---|---|---|---|---|---|---|
| | Beta | 95% CI | p-value | Beta | 95% CI | p-value | Beta | 95% CI | p-value |
| **Women** | | | | | | | | | |
| Maximum alcohol Frequency | | | | | | | | | |
| Abstainer | — | — | | — | — | | — | — | |
| Occasional (< 1/wk) | 0.1129 | 0.0976, 0.1282 | < 0.001 | 0.0989 | 0.0837, 0.1141 | < 0.001 | 0.0988 | 0.0836, 0.1140 | < 0.001 |
| Moderate (1–4 days/wk) | 0.1667 | 0.1506, 0.1829 | < 0.001 | 0.1464 | 0.1303, 0.1625 | < 0.001 | 0.1465 | 0.1304, 0.1626 | < 0.001 |
| Frequent (5 + days/wk) | 0.1352 | 0.1173, 0.1531 | < 0.001 | 0.1211 | 0.1034, 0.1389 | < 0.001 | 0.1211 | 0.1034, 0.1389 | < 0.001 |
| Age centered at 65 | -0.0297 | -0.0305, -0.0289 | < 0.001 | -0.0291 | -0.0299, -0.0283 | < 0.001 | -0.0291 | -0.0299, -0.0283 | < 0.001 |
| Maximum social connectedness score (0–4) | | | | 0.1033 | 0.0970, 0.1097 | < 0.001 | 0.1031 | 0.0967, 0.1095 | < 0.001 |
| Number of Chronic illnesses | | | | | | | 0.0008 | -0.0023, 0.0038 | 0.620 |
| **Men** | | | | | | | | | |
| Maximum alcohol Frequency | | | | | | | | | |
| Abstainer | — | — | | — | — | | — | — | |
| Occasional (< 1/wk) | 0.0700 | 0.0468, 0.0933 | < 0.001 | 0.0580 | 0.0347, 0.0812 | < 0.001 | 0.0582 | 0.0350, 0.0814 | < 0.001 |
| Moderate (1–4 days/wk) | 0.1217 | 0.1011, 0.1423 | < 0.001 | 0.1039 | 0.0834, 0.1244 | < 0.001 | 0.1038 | 0.0833, 0.1243 | < 0.001 |
| Frequent (5 + days/wk) | 0.0789 | 0.0587, 0.0990 | < 0.001 | 0.0656 | 0.0456, 0.0857 | < 0.001 | 0.0654 | 0.0454, 0.0855 | < 0.001 |
| Age centered at 65 | -0.0277 | -0.0290, -0.0264 | < 0.001 | -0.0274 | -0.0287, -0.0261 | < 0.001 | -0.0273 | -0.0286, -0.0260 | < 0.001 |
| Maximum social connectedness score (0–4) | | | | 0.0862 | 0.0784, 0.0941 | < 0.001 | 0.0863 | 0.0785, 0.0942 | < 0.001 |

Abbreviation: CI = Confidence Interval

| Characteristic | General Model | | | + Social scale | | | + Chronic illness | | |
|---|---|---|---|---|---|---|---|---|---|
| | Beta | 95% CI | p-value | Beta | 95% CI | p-value | Beta | 95% CI | p-value |
| **Women** | | | | | | | | | |
| Number of Chronic illnesses | | | | | | | -0.0039 | -0.0073, -0.0005 | 0.024 |
| Abbreviation: CI = Confidence Interval | | | | | | | | | |

Longitudinal Cognitive Trajectories: the social buffer

Table 3 presents the gender-stratified 3-way interactions (alcohol use*age*social connectedness) (see Table S2 for full table in supplementary materials). While social connectedness partially accounted for the higher baseline cognitive scores of people who drink, its impact on longitudinal decline diverged by gender. A significant social connectedness-by-age interaction for women ($p \leq 0.001$), but not men ($p = 0.50$), indicates that it buffers age-related decline for women regardless of alcohol intake. Furthermore, a significant 3-way interaction emerged for women: high social connectedness flattened the ageing trajectory significantly more for moderate and frequent drinkers than abstainers, amplifying its base buffering effect (Fig. 4). Conversely, while male drinkers retained a baseline cognitive “advantage”, social connectedness did not differentially alter the rate of decline for occasional or moderate drinkers compared to abstainers ($p = 0.30$ and $p = 0.12$), though it did for frequent drinkers ($p = 0.018$). In both genders, the main effect of social connectedness remained significant (women: $\beta = 0.0814$, $p \leq 0.001$; men: $\beta = 0.0780$, $p \leq 0.001$), confirming a higher baseline score.

Multivariate Wald tests formally evaluated this longitudinal buffering effect by comparing full models against restricted models lacking the 3-way interaction. This interaction significantly improved model fit for women (*$F(3, 57.0) = 4.83$, $p = 0.00461$*), confirming that social connectedness modifies cognitive ageing trajectories differently across female drinking groups. For men, the interaction was not significant overall (*$F(3, 77.4) = 1.58$, $p = 0.200$*). Therefore, while men may experience independent cognitive benefits from social integration and moderate drinking, social connectedness does not alter their rate of age-related cognitive decline across these groups.

Table 3
Gender-stratified fully adjusted models for global cognition

| Characteristic | Females (main) | | | Males (main) | | |
|---|---|---|---|---|---|---|
| | **Beta** | **95% CI** | **p-value** | **Beta** | **95% CI** | **p-value** |
| Maximum drinking frequency | | | | | | |
| Abstainer | — | — | | — | — | |
| Occasional (< 1/wk) | 0.0790 | 0.0365, 0.1215 | **< 0.001** | 0.0671 | 0.0002, 0.1339 | **0.049** |
| Moderate (1–4 days/wk) | 0.1082 | 0.0627, 0.1537 | **< 0.001** | 0.0956 | 0.0440, 0.1471 | **< 0.001** |
| Frequent (5 + days/wk) | 0.1030 | 0.0461, 0.1599 | **< 0.001** | 0.0645 | 0.0127, 0.1164 | **0.015** |
| Age centered as 65 | -0.0342 | -0.0365, -0.0319 | **< 0.001** | -0.0283 | -0.0315, -0.0251 | **< 0.001** |
| Maximum social scale | 0.0814 | 0.0690, 0.0939 | **< 0.001** | 0.0780 | 0.0579, 0.0980 | **< 0.001** |
| Living with/without partner | | | | | | |
| Living with spouse/partner | — | — | | — | — | |
| Living without spouse/partner | -0.0168 | -0.0262, -0.0075 | **< 0.001** | -0.0466 | -0.0591, -0.0341 | **< 0.001** |
| Maximum drinking frequency * age (centered at 65) | | | | | | |
| Occasional (< 1/wk) * age (centered at 65) | -0.0004 | -0.0044, 0.0035 | 0.8 | -0.0026 | -0.0078, 0.0025 | 0.3 |
| Moderate (1–4 days/wk) * age (centered at 65) | -0.0026 | -0.0065, 0.0012 | 0.2 | -0.0015 | -0.0056, 0.0027 | 0.5 |
| Frequent (5 + days/wk) * age (centered at 65) | -0.0045 | -0.0082, -0.0008 | **0.019** | -0.0021 | -0.0059, 0.0018 | 0.3 |
| Maximum drinking frequency * Maximum social scale | | | | | | |
| Occasional (< 1/wk) * Maximum social scale | 0.0107 | -0.0070, 0.0283 | 0.2 | -0.0031 | -0.0351, 0.0288 | 0.8 |
| Moderate (1–4 days/wk) * Maximum social scale | 0.0191 | 0.0008, 0.0374 | **0.041** | 0.0052 | -0.0186, 0.0290 | 0.7 |
| Frequent (5 + days/wk) * Maximum social scale | 0.0115 | -0.0112, 0.0343 | 0.3 | 0.0018 | -0.0228, 0.0264 | 0.9 |
| age (centered at 65) * Maximum social scale | 0.0024 | 0.0014, 0.0034 | **< 0.001** | 0.0005 | -0.0010, 0.0021 | 0.5 |
| Abbreviation: CI = Confidence Interval | | | | | | |

| Characteristic | Females (main) | | | Males (main) | | |
|---|---|---|---|---|---|---|
| | Beta | 95% CI | p-value | Beta | 95% CI | p-value |
| Maximum drinking frequency * age (centered at 65) * Maximum social scale | | | | | | |
| Occasional (< 1/wk) * age (centered at 65) * Maximum social scale | 0.0010 | -0.0007, 0.0028 | 0.2 | 0.0014 | -0.0011, 0.0039 | 0.3 |
| Moderate (1–4 days/wk) * age (centered at 65) * Maximum social scale | 0.0024 | 0.0008, 0.0040 | **0.004** | 0.0015 | -0.0004, 0.0034 | 0.12 |
| Frequent (5 + days/wk) * age (centered at 65) * Maximum social scale | 0.0029 | 0.0013, 0.0044 | **< 0.001** | 0.0022 | 0.0004, 0.0040 | **0.018** |
| Abbreviation: CI = Confidence Interval | | | | | | |

Multiple sensitivity analyses confirmed the overall stability of the primary findings. The gendered social buffering mechanism remained robust when adjusting for macro-level regional drinking cultures (Table S3) and when incorporating random slopes for individuals to account for varying ageing trajectories (Table S4). Furthermore, the female-specific significance of both the three-way interaction and the underlying age-by-social connectedness interaction was not materially altered by using unweighted models (Table S5), treating the social scale categorically (Table S7), excluding zero-score social scale participants (Table S8), applying alternative practice effect adjustments (Table S9), running complete-case analyses on non-imputed data (Table S10), or restricting covariates (Table S11). The only notable divergence occurred when collapsing alcohol consumption into a broad 'drinker versus abstainer' category (Table S6), where the three-way interaction also became significant for men (p = 0.030). This discrepancy highlights that a simple binary classification may be an overly blunt metric that can obscures frequency-dependent nuances.

# Discussion

This study used a hierarchical mixed-modelling approach on a large, cross-national panel of older European adults to investigate whether social connectedness confounds or modifies the association between moderate alcohol consumption and cognitive function. This study supports our first hypothesis (H1): the higher baseline cognition of moderate drinkers is partially confounded by the active social integration that often accompanies it in Western Europe. As expected under H3, the mechanisms are gendered. For older women, social connectedness slows age-related cognitive decline (H2). This buffering effect is strongest among women who drink. Conversely, while older men gain a cognitive advantage from social connectedness, it does not moderate their rate of decline over time, and their drinking patterns do not alter this trajectory.

Gendered Drinking trajectories: alignment with cohort norms

The descriptive statistics of our SHARE sample showed gendered drinking patterns. Men exhibited a significantly higher consumption frequency (38.82% classified as frequent drinkers vs. 17.02% of women), whereas women were twice as likely to abstain (32.03% vs. 14.75%). Consistence with existing literature, this disparity is

structured by traditional gender roles and societal expectations: domestic responsibilities and stronger social sanctions against public drinking historically constrain women's consumptions (Cipriani et al., 2021; Erol & Karpyak, 2015). Biologically, women also experience accelerated physiological vulnerabilities (the "telescoping" effect) at lower doses, which naturally restrains consumption frequency while increasing their susceptibility to long-term harm (Erol & Karpyak, 2015).

Social connectedness as a confounding variable

Observational studies frequently report a cognitive advantage for moderate drinking compared to abstainers (M. A. Collins et al., 2009; Wiegmann et al., 2020). Among older adults, however, moderate drinking is usually a social activity that takes place within leisure and community routines (Cipriani et al., 2021). Our findings support this "lifestyle confounding" theory (Wiegmann et al., 2020). Aligning with literature identifying social isolation as a dementia risk factor (Livingston et al., 2024), social connectedness provided a "baseline cognitive advantage" in our sample. Adjusting for it attenuated the estimated "protective" effect of alcohol by 10 to 17% across both genders, suggesting that a portion of the observed "J-shape" relationship is indeed driven by residual social confounding. Despite this attenuation, cognitive estimates for all drinking frequencies remained statistically significant relative to the abstainer reference.

Social connectedness and moderate drinking among older women

Although the role of social connectedness as a confounding variable was apparent in both genders, its longitudinal buffering effect differed by gender. For women, social connectedness independently buffered age-related cognitive decline ($\beta = 0.0024$, $p \leq 0.001$), an effect absent among men ($\beta = 0.0005$, $p = 0.5$). Furthermore, our 3-way interaction showed that this buffer was significantly stronger for moderate and frequent female drinkers compared to abstainers, flattening their trajectory of cognitive ageing.

This amplification aligns with how women use alcohol as a relational tool and a medium for social bonding. Women are more likely to drink at home with close friends to cope with negative emotions, frequently framing moderate consumption (e.g., sharing a glass of wine) as a deliberate act of "self-care" and social bonding (Erol & Karpyak, 2015; Kersey et al., 2022). By acting as a social facilitator, moderate alcohol consumption helps women consolidate expansive, friend-centric networks that provide novel intellectual engagement and emotional support (Arnhold et al., 2026; Cipriani et al., 2021; Erol & Karpyak, 2015). Such ties are thought to protect cognition through the mechanisms outlined in the introduction (Chen et al., 2025; Giné-Garriga et al., 2021; Willroth et al., 2024). Because chronic stress and depression are also known modifiable risk factors that accelerate cognitive decline, moderate drinking combined with high social connectedness may describe a social setting that protects cognitive function in ageing women (Cipriani et al., 2021; M. A. Collins et al., 2009).

If this interpretation holds, the benefit derives from social contact rather than from alcohol, and such networks can be maintained without drinking. Alternatively, this three-way interaction is also consistent with reverse causation: older women who remain cognitively healthy may be better able to keep both their social networks and their moderate drinking routines into later life, a limitation we address further below.

Men's trajectories

In contrast to women, neither the social connectedness-by-age interaction ($\beta = 0.0005$, $p = 0.5$) nor the three-way interaction ($p = 0.200$) were significant for older men. Men's rate of cognitive ageing was not moderated by broad

social engagement, whatever their drinking pattern.

This difference may reflect the settings in which men drink. The *convoy model of social relations* suggests men rely disproportionately on spouse-centred ties for primary emotional and cognitive support, rather than diverse bridging networks (Arnhold et al., 2026; Kahn & Antonucci, 1980). Because a spouse provides a localised baseline of stability, a man's general social connectedness score (which weights contact frequency across a wider external network) does not systematically interact with his cognitive trajectory (Arnhold et al., 2026; Schwarzinger et al., 2018). Consequently, his broader social ties often fail to provide compensatory neuroprotection if that spousal bond is lost. While our model adjusted for "living with partner", we did not formally stratify by partnership status.

Male drinking in these cohorts has historically been more public and habitual than women's, for example at a local pub (Erol & Karpyak, 2015). Even if frequent co-presence yields a high social connectedness score (Kelly et al., 2018), the interaction in these settings may be routine and offer little of the novelty thought to promote neuroplasticity, so that the social environment of male drinking does little to protect cognition.

Finally, the null results align with SHARE evidence suggesting male vulnerability to isolation is primarily threshold dependent. A complete lack of social connection accelerates male cognitive decline, but once that basic threshold of contact is met, further engagement provides no linear buffering effects (Arnhold et al., 2026). Because absolute zero social scores were negligible in our sample, our findings fit with the broader understanding that incremental social contact does not actively moderate male cognitive aging. In the context of sustainable aging, this suggests that measuring routine social presence may be insufficient for identifying cognitive risk in older men, as their baseline vulnerabilities operate independently of these incremental social scales.

Implications for sustainable ageing

Our findings contribute to the social and care dimensions of sustainable ageing. Because the cognitive advantage of moderate drinking partly reflects social integration, policies aimed at cognitive health should target the social integration rather than drinking itself. Supporting opportunities for social participation provides older adults with diverse relational networks without creating additional health or public-safety harms. Because cognitive impairment is a major driver of long-term care needs, strengthening these social resources can reduce the burden on both formal and informal care and the younger generations financing them.

The gender differences suggest gender-specific priorities. For older women, policies should foster friend-based networks and social opportunities independent of alcohol to preserve cognitive benefits without the associated harms. Conversely, because incremental social contact does not slow cognitive decline in men (Arnhold et al., 2026), intervention should instead target critical risk threshold (such as complete isolation or spousal bereavement) where male vulnerability is more acute.

Two qualifications apply to these policies implications. First, our frequency-based measure cannot distinguish light daily drinking from heavier consumption, nor does it separate lifetime abstainers from former drinkers who stopped due to illness (detailed further in Limitations). Second, while supplementary analysis of historical drinking cultures confirm the cognitive implications of alcohol are context-dependent, the gendered social buffering effect remained robust across all regions (Room & Mäkelä, 2000) This suggests the protective role of

social connectedness persists even in cultures where drinking is less inextricably linked to socialisation, though future comparative research should examine these micro-cultural differences more granularly.

Limitations

The strengths of this study are a large, multi-country panel with nearly 9 years of follow-up, a validated social network scale, and adjustment for attrition through weights and multiple imputation. However, several limitations must be acknowledged, some common to SHARE data and similar observational studies (Arnhold et al., 2026; Listabarth et al., 2022).

First, self-reported alcohol use is susceptible to social desirability bias, potentially underestimating consumption. Because stigma may differ by gender, gender-specific reporting biases might exist. We tried to mitigate this potential underreporting by using the maximum reported alcohol frequency across all available waves.

Second, our frequency-based categories capture how often an individual drinks, not the volume consumed. Consequently, the “frequent” category likely comprises “light but frequent” drinkers (e.g., a daily glass of wine) rather than those engaging in heavy, at-risk consumption. Conversely, episodic binge drinkers, a behaviour with known cognitive risks (Cipriani et al., 2021), cannot be distinguished from true light drinkers who consume one drink per month. This risks misclassifying problematic behaviours into lower frequency tiers and could explain the persistent “advantage” of frequent drinkers over abstainers, even though heavy drinking is a recognised risk factor for cognitive decline.

Third, SHARE does not record whether participants drank alone or in company, so the social character of drinking is inferred rather than observed. More generally, the design is observational and cannot establish the direction of the associations. Reverse causation is a serious possibility: individuals experiencing subclinical cognitive decline or physical impairment may concurrently isolate themselves and stop drinking. Because SHARE life history data could not distinguish true lifetime abstainers from “sick quitters”, the persistent cognitive deficit the abstainer group could reflect residual sick-quitter bias, even after adjusting for chronic illnesses.

Finally, our cognitive composite score covers memory and verbal fluency but lacks measures of executive function or processing speed, potentially masking domain-specific decline. The longitudinal design requiring participation in at least two waves introduces a healthy survivor bias that attrition weights can only partially mitigate. Moreover, although we adjusted for a wide set of sociodemographic and health factors, residual unmeasured confounding remains a limitation. Contributors to cognitive reserve, such as lifelong occupational complexity and cognitively stimulating leisure activities, were not fully captured. Because individuals with historically high intellectual engagement may simultaneously maintain better baseline cognitive trajectories, wider social networks, and specific social drinking routines, the absence of these life-course variables means some of the observed buffering effect could reflect unmeasured earlier-life advantages rather than current behaviours. Finally, because social connectedness was only measured in wave 6, 8, and 9, this score is retrospectively applied to cognitive trajectories that may have begun in wave 1. Consequently, the network score is often observed after a significant portion of the cognitive trajectory has already unfolded, and this single-timepoint measure fails to capture dynamic network change over the follow-up period.

## Conclusion

Sustainable ageing depends on preserving cognitive function, a prerequisite for independent living and autonomy. This study demonstrates that a portion of the observed cognitive “benefit” associated with alcohol is attenuated with accounting for social connectedness. Crucially, these mechanisms are gendered. While social connectedness acts as a longitudinal buffer for women’s cognitive trajectories, an effect magnified among those who drink, men’s rate of cognitive decline is not modified by these broad social connections, regardless of their drinking frequency.

Preserving cognitive function in later life therefore depends on sustaining social networks, and the networks that matter differ for women and men. Future research should prioritise investigating the bidirectional causality of these relationships and identifying how distinct qualitative ties mitigate risk across different demographic groups. For the sustainable ageing, social connection is the protective resource worth sustaining. It supports independence in later life without the harms associated with alcohol and, where it is sustained, helps preserve autonomy and thus quality of life up to older ages.

## Declarations

# Ethics approval:

This study uses secondary data from the Survey of Health, Ageing and Retirement in Europe (SHARE). The SHARE data collection procedures are subject to continuous ethical review and have been formally approved by the Ethics Committee of the University of Mannheim and the Ethics Council of the Max Planck Society. All SHARE participants gave informed consent. The secondary use of the data in this study was approved by the Ethics Review Panel of the University of Luxembourg.

# Funding:

The author(s) declared that financial support was received for this work and/or its publication. The research for this review was supported by the University of Luxembourg’s Institute for Advanced Studies AUDACITY grant IAS_PRECIDEM_2024 and the Faculty of Humanities, Education, and Social Sciences of the University of Luxembourg.

Competing interests:

The authors have no competing interests to declare that are relevant to the content of this article.

# Author Contribution

E.T. conceptualised the study, performed the data curation and formal statistical analysis, and drafted the original manuscript. A.L. and D.M. provided supervision, methodological guidance, and critical revisions of the manuscript. All authors read and approved the final manuscript.

# Data Availability

This paper uses data from SHARE Waves 1, 2, 4, 5, 6, 7, 8 and 9 (DOIs: 10.6103/SHARE.w1.900, 10.6103/SHARE.w2.900, 10.6103/SHARE.w4.900, 10.6103/SHARE.w5.900, 10.6103/SHARE.w6.900, 10.6103/SHARE.w6.DBS.200, 10.6103/SHARE.w7.900, 10.6103/SHARE.w8.900, 10.6103/SHARE.w8ca.900, 10.6103/SHARE.w9.900, 10.6103/SHARE.w9ca900, 10.6103/SHARE.HCAP1.100) see Börsch-Supan et al. (2013) for methodological details. The SHARE data collection has been funded by the European Commission, DG RTD through FP5 (QLK6-CT-2001-00360), FP6 (SHARE-I3: RII-CT-2006-062193, COMPARE: CIT5-CT-2005-028857, SHARELIFE: CIT4-CT-2006-028812), FP7 (SHARE-PREP: GA N°211909, SHARE-LEAP: GA N°227822, SHARE M4: GA N°261982, DASISH: GA N°283646) and Horizon 2020 (SHARE-DEV3: GA N°676536, SHARE-COHESION: GA N°870628, SERISS: GA N°654221, SSHOC: GA N°823782, SHARE-COVID19: GA N°101015924) and by DG Employment, Social Affairs & Inclusion through VS 2015/0195, VS 2016/0135, VS 2018/0285, VS 2019/0332, VS 2020/0313, SHARE-EUCOV: GA N°101052589 and EUCOVII: GA N°101102412. Additional funding from the German Federal Ministry of Research, Technology and Space (01UW1301, 01UW1801, 01UW2202), the Max Planck Society for the Advancement of Science, the U.S. National Institute on Aging (U01_AG09740-13S2, P01_AG005842, P01_AG08291, P30_AG12815, R21_AG025169, Y1-AG-4553-01, IAG_BSR06-11, OGHA_04-064, BSR12-04, R01_AG052527-02, R01_AG056329-02, R01_AG063944, HHSN271201300071C, RAG052527A) and from various national funding sources is gratefully acknowledged (see share-eric.eu).

## References

1. Allen LN, Townsend N, Williams J, Mikkelsen B, Roberts N, Wickramasinghe K (2018) Socioeconomic status and alcohol use in low- and lower-middle income countries: A systematic review. Alcohol 70:23–31. https://doi.org/10.1016/j.alcohol.2017.12.002
2. Anstey KJ, Peters R (2018) Alcohol and dementia—Risk or protective factor? Nat Reviews Neurol 14(11):635–636. https://doi.org/10.1038/s41582-018-0073-0
3. Arnhold T, Weber D, Bordone V (2026) Employment Status and Later Life Cognitive Functioning: A Gendered Perspective on the Moderating Role of the Social Network. Res Aging 48(1):1–15. https://doi.org/10.1177/01640275261456210
4. Arpino B, Bordone V, Dykstra PA (2026) *Sustainable ageing: Concept, dimensions, and policy levers*(2bg39_v1). SocArXiv. https://osf.io/preprints/socarxiv/2bg39_v1/
5. Bertogg A, Leist AK (2021) Partnership and cognitive aging in Europe: Mediating factors and social stratification. Journals Gerontology: Ser B 76(6):1173–1185. https://doi.org/10.1093/geronb/gbab020
6. Börsch-Supan A, Brandt M, Hunkler C, Kneip T, Korbmacher J, Malter F, Schaan B, Stuck S, Zuber S (2013) Data Resource Profile: The survey of health, ageing and retirement in Europe (share). Int J Epidemiol 42(4):992–1001. https://doi.org/10.1093/ije/dyt088
7. Chen Y, Yin X, Wang X, Zheng X, Yang X, Zhou J, Shi M, Zhang Y (2025) Associations of alcohol drinking with incident dementia: A prospective study from the UK Biobank. Eur J Epidemiol 40(11):1345–1354. https://doi.org/10.1007/s10654-025-01304-y
8. Cipriani G, Nuti A, Carlesi C, Lucetti C, Di Fiorino M, Danti S (2021) Categorising a problem: Alcohol and dementia. Acta Neurol Belgica 121(1):1–10. https://doi.org/10.1007/s13760-020-01515-y
9. Collins MA, Neafsey EJ, Mukamal KJ, Gray MO, Parks DA, Das DK, Korthuis RJ (2009) Alcohol in moderation, cardioprotection, and neuroprotection: Epidemiological considerations and mechanistic studies. Alcoholism: Clin Experimental Res 33(2):206–219. https://doi.org/10.1111/j.1530-0277.2008.00828.x

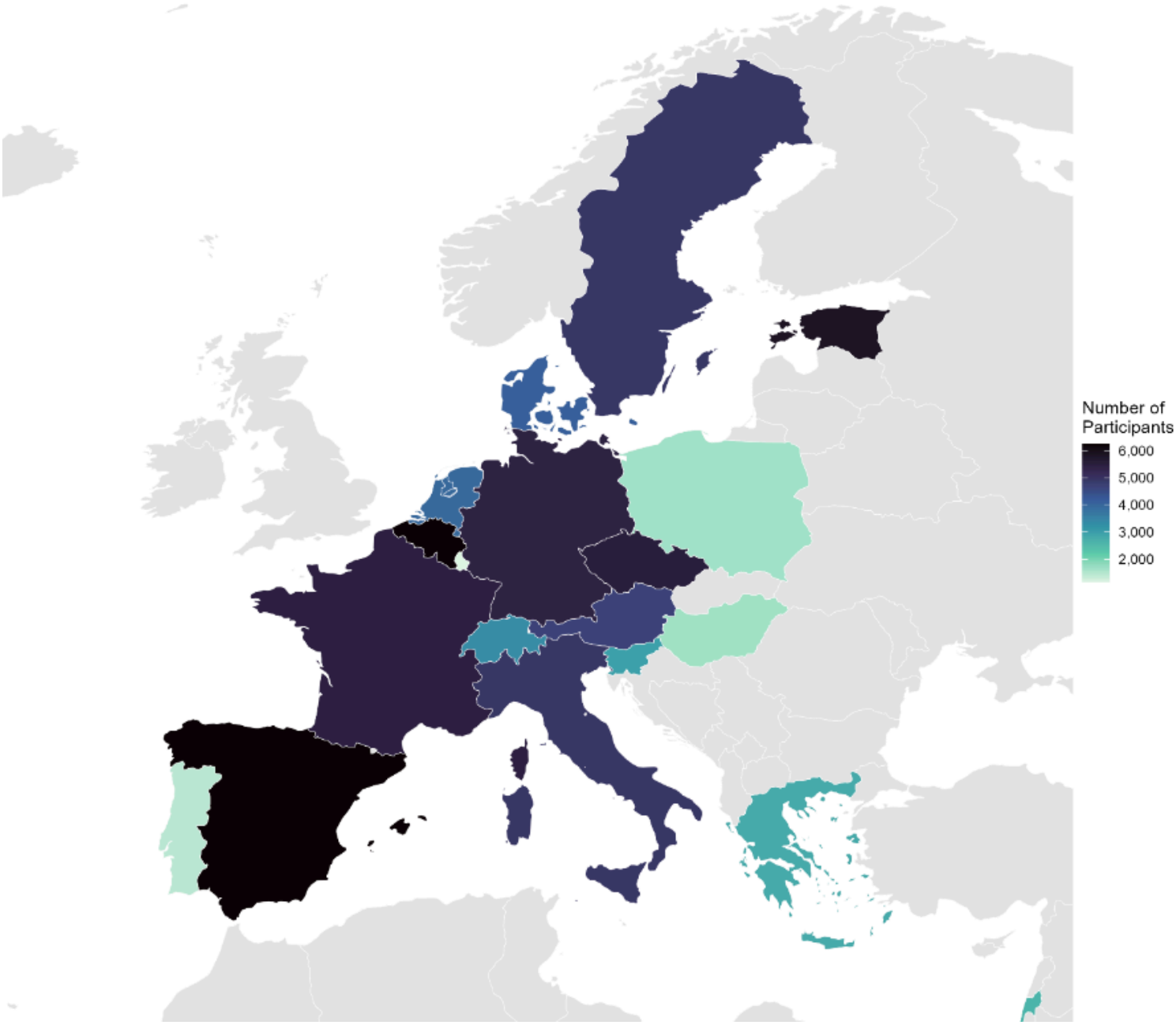
10. Collins SE (2016) *Associations Between Socioeconomic Factors and Alcohol Outcomes*. *38*(1), 83–94. https://doi.org/10.35946/arcr.v38.1.11
11. Erol A, Karpyak VM (2015) Sex and gender-related differences in alcohol use and its consequences: Contemporary knowledge and future research considerations. Drug Alcohol Depend 156:1–13. https://doi.org/10.1016/j.drugalcdep.2015.08.023
12. George S, Naimi TS, Keyes K, Martinez-Matyszczyk P, Milam AJ, Rehm J, Alonso A, Bell S, Britton A, Llamosas-Falcón L, Hobin E, Judd SE, Justice A, Kahler C, Liangpunsakul S, Jones SK, Marcus G, McGlynn K, Satre D, Shield K (2026) Alcohol Intake and Health Study: No Protective Effect at Low Levels, With Mortality Increasing to 1 in 25 at 14 Drinks Per Week. J Stud Alcohol Drug 87(4):621–638. https://doi.org/10.15288/jsad.25-00435
13. Giné-Garriga M, Jerez-Roig J, Coll-Planas L, Skelton DA, Inzitari M, Booth J, Souza DLB (2021) Is loneliness a predictor of the modern geriatric giants? Analysis from the survey of health, ageing, and retirement in Europe. Maturitas 144:93–101. https://doi.org/10.1016/j.maturitas.2020.11.010
14. Kahn RL, Antonucci TC (1980) Convoys over the life course: Attachment, roles, and social support. Life-Span Dev Behav 3:253–286
15. Kawakami S, Yamato R, Kitamura K, Watanabe Y, Kabasawa K, Takahashi A, Saito T, Kobayashi R, Oshiki R, Takachi R, Tsugane S, Yamazaki O, Watanabe K, Nakamura K (2023) Alcohol consumption, smoking, and risk of dementia in community-dwelling Japanese people aged 40–74 years: The Murakami cohort study. Maturitas 176:107788. https://doi.org/10.1016/j.maturitas.2023.107788
16. Kelly S, Olanrewaju O, Cowan A, Brayne C, Lafortune L (2018) Alcohol and older people: A systematic review of barriers, facilitators and context of drinking in older people and implications for intervention design. PLoS ONE 13(1):e0191189. https://doi.org/10.1371/journal.pone.0191189
17. Kersey K, Lyons AC, Hutton F (2022) Alcohol and drinking within the lives of midlife women: A meta-study systematic review. Int J Drug Policy 99:103453. https://doi.org/10.1016/j.drugpo.2021.103453
18. Leist AK, Bar-Haim E, Chauvel L (2021) Inequality of educational opportunity at time of schooling predicts cognitive functioning in later adulthood. SSM - Popul Health 15:100837. https://doi.org/10.1016/j.ssmph.2021.100837
19. Listabarth S, Groemer M, Waldhoer T, Vyssoki B, Pruckner N, Vyssoki S, Glahn A, König-Castillo DM, König D (2022) Cognitive decline and alcohol consumption in the aging population—A longitudinal analysis of the Survey of Health, Ageing and Retirement in Europe. Eur Psychiatry 65(1):e83. https://doi.org/10.1192/j.eurpsy.2022.2344
20. Litwin H, Stoeckel KJ (2015a) Engagement and social capital as elements of active ageing: An analysis of older Europeans. SOCIOLOGIA E POLITICHE SOCIALI 39–31. https://doi.org/10.3280/sp2014-003002
21. Litwin H, Stoeckel KJ (2015b) Social Network, Activity Participation, and Cognition: A Complex Relationship. Res Aging 38(1):76–97. https://doi.org/10.1177/0164027515581422
22. Litwin H, Stoeckel K, Roll A, Shiovitz-Ezra S (2013) Social network measurement in SHARE wave 4. In *SHARE Wave 4: Innovations and Methodology*(pp. 18–38). Munich Center for the Economics of Aging (MEA). https://share.cerge-ei.cz/pdfs/methodology/share_wave4_innovations_and_methodology.pdf
23. Livingston G, Huntley J, Liu KY, Costafreda SG, Selbæk G, Alladi S, Ames D, Banerjee S, Burns A, Brayne C, Fox NC, Ferri CP, Gitlin LN, Howard R, Kales HC, Kivimäki M, Larson EB, Nakasujja N, Rockwood K, Mukadam N

(2024) Dementia prevention, intervention, and care: 2024 report of the Lancet standing Commission. Lancet 404(10452):572–628. https://doi.org/10.1016/S0140-6736(24)01296-0

24. Mewton L, Visontay R, Hoy N, Lipnicki DM, Sunderland M, Lipton RB, Guerchet M, Ritchie K, Najar J, Scarmeas N, Kim K, Riedel Heller S, Van Boxtel M, Jacobsen E, Brodaty H, Anstey KJ, Haan M, Scazufca M, Lobo E … Collaborators from the Cohort Studies of Memory in an International Consortium (COSMIC). (2023). The relationship between alcohol use and dementia in adults aged more than 60 years: A combined analysis of prospective, individual-participant data from 15 international studies. Addiction, *118*(3), 412–424. https://doi.org/10.1111/add.16035
25. Room R, Mäkelä K (2000) Typologies of the cultural position of drinking. J Stud Alcohol 61(3):475–483. https://doi.org/10.15288/jsa.2000.61.475
26. Schwarzinger M, Pollock BG, Hasan OSM, Dufouil C, Rehm J, Baillot S, Guibert Q, Planchet F, Luchini S (2018) Contribution of alcohol use disorders to the burden of dementia in France 2008–13: A nationwide retrospective cohort study. Lancet Public Health 3(3):e124–e132. https://doi.org/10.1016/S2468-2667(18)30022-7
27. SHARE-ERIC (2024) Survey of Health, Ageing and Retirement in Europe (SHARE) Wave 1. Release version: 9.0.0. SHARE-ERIC. Data set. 10.6103/SHARE.w1.900
28. SHARE-ERIC (2024) Survey of Health, Ageing and Retirement in Europe (SHARE) Wave 2. Release version: 9.0.0. SHARE-ERIC. Data set. 10.6103/SHARE.w2.900
29. SHARE-ERIC (2024) Survey of Health, Ageing and Retirement in Europe (SHARE) Wave 4. Release version: 9.0.0. SHARE-ERIC. Data set. 10.6103/SHARE.w4.900
30. SHARE-ERIC (2024) Survey of Health, Ageing and Retirement in Europe (SHARE) Wave 5. Release version: 9.0.0. SHARE-ERIC. Data set. 10.6103/SHARE.w5.900
31. SHARE-ERIC (2024) Survey of Health, Ageing and Retirement in Europe (SHARE) Wave 6. Release version: 9.0.0. SHARE-ERIC. Data set. 10.6103/SHARE.w6.900
32. SHARE-ERIC (2024) Survey of Health, Ageing and Retirement in Europe (SHARE) Wave 7. Release version: 9.0.0. SHARE-ERIC. Data set. 10.6103/SHARE.w7.900
33. SHARE-ERIC (2024) Survey of Health, Ageing and Retirement in Europe (SHARE) Wave 8. Release version: 9.0.0. SHARE-ERIC. Data set. 10.6103/SHARE.w8.900
34. SHARE-ERIC (2024) Survey of Health, Ageing and Retirement in Europe (SHARE) Wave 9. Release version: 9.0.0. SHARE-ERIC. Data set. 10.6103/SHARE.w9.900
35. Topiwala A, Levey D, Zhou H, Deak J, Adhikari K, Ebmeier K, Bell S, Burgess S, Nichols T, Gaziano M, Stein M, Gelernter J (2025) Alcohol use and risk of dementia in diverse populations: Evidence from cohort, case-control and Mendelian randomisation approaches. BMJ EVIDENCE-BASED Med. (WOS:001578908800001 https://doi.org/10.1136/bmjebm-2025-113913
36. Vivot A, Power MC, Glymour MM, Mayeda ER, Benitez A, Spiro A, Manly JJ, Proust-Lima C, Dufouil C, Gross AL (2016) Jump, Hop, or Skip: Modeling Practice Effects in Studies of Determinants of Cognitive Change in Older Adults. Am J Epidemiol 183(4):302–314. https://doi.org/10.1093/aje/kwv212
37. Weuve J, Proust-Lima C, Power MC, Gross AL, Hofer SM, Thiébaut R, Chêne G, Glymour MM, Dufouil C, MELODEM Initiative (2015) Guidelines for reporting methodological challenges and evaluating potential bias in dementia research. Alzheimer's Dement 11(9):1098–1109. https://doi.org/10.1016/j.jalz.2015.06.1885

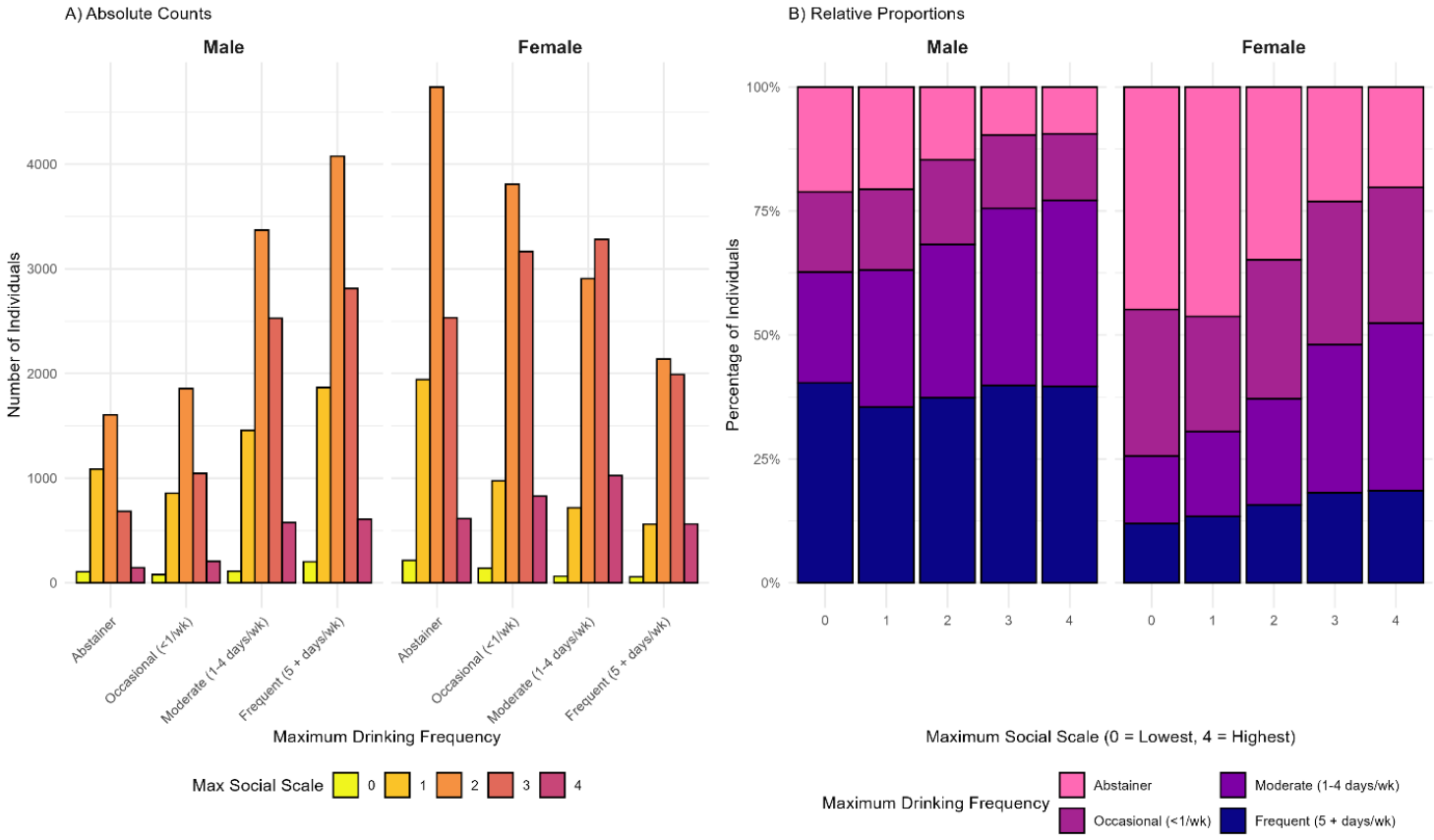
38. Wiegmann C, Mick I, Brandl EJ, Heinz A, Gutwinski S (2020) Alcohol and Dementia – What is the Link? A Systematic Review. Neuropsychiatr Dis Treat 16:87–99. https://doi.org/10.2147/NDT.S198772
39. Willroth EC, Pfund GN, Rule PD, Hill PL, John A, Kyle K, Hassenstab J, James BD (2024) A review of the literature on wellbeing and modifiable dementia risk factors. Ageing Res Rev 99:102380. https://doi.org/10.1016/j.arr.2024.102380
40. World Health Organization (2024) Alcohol—Fact sheet. World Health Organization. https://www.who.int/news-room/fact-sheets/detail/alcohol
41. World Health Organization (2026) Dementia—Fact sheet. World Health Organization. https://www.who.int/news-room/fact-sheets/detail/dementia

## Figures



## Figure 1

Countries included in our analytical sample with number of participants per country




## Figure 2

Social connectedness and Alcohol frequency and proportion by Gender and Social Connectedness

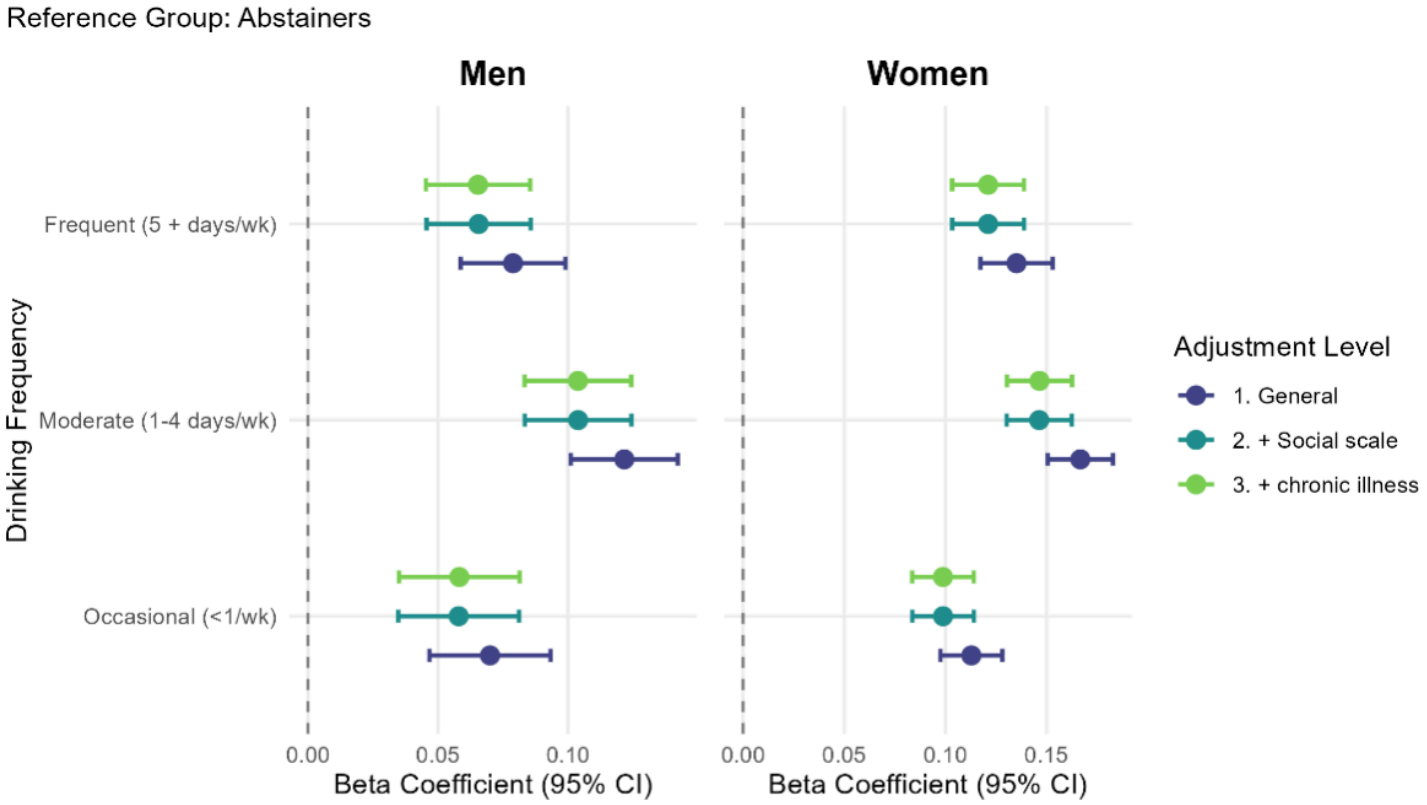



**Figure 3**

Attenuation of the main effect of alcohol on global cognition

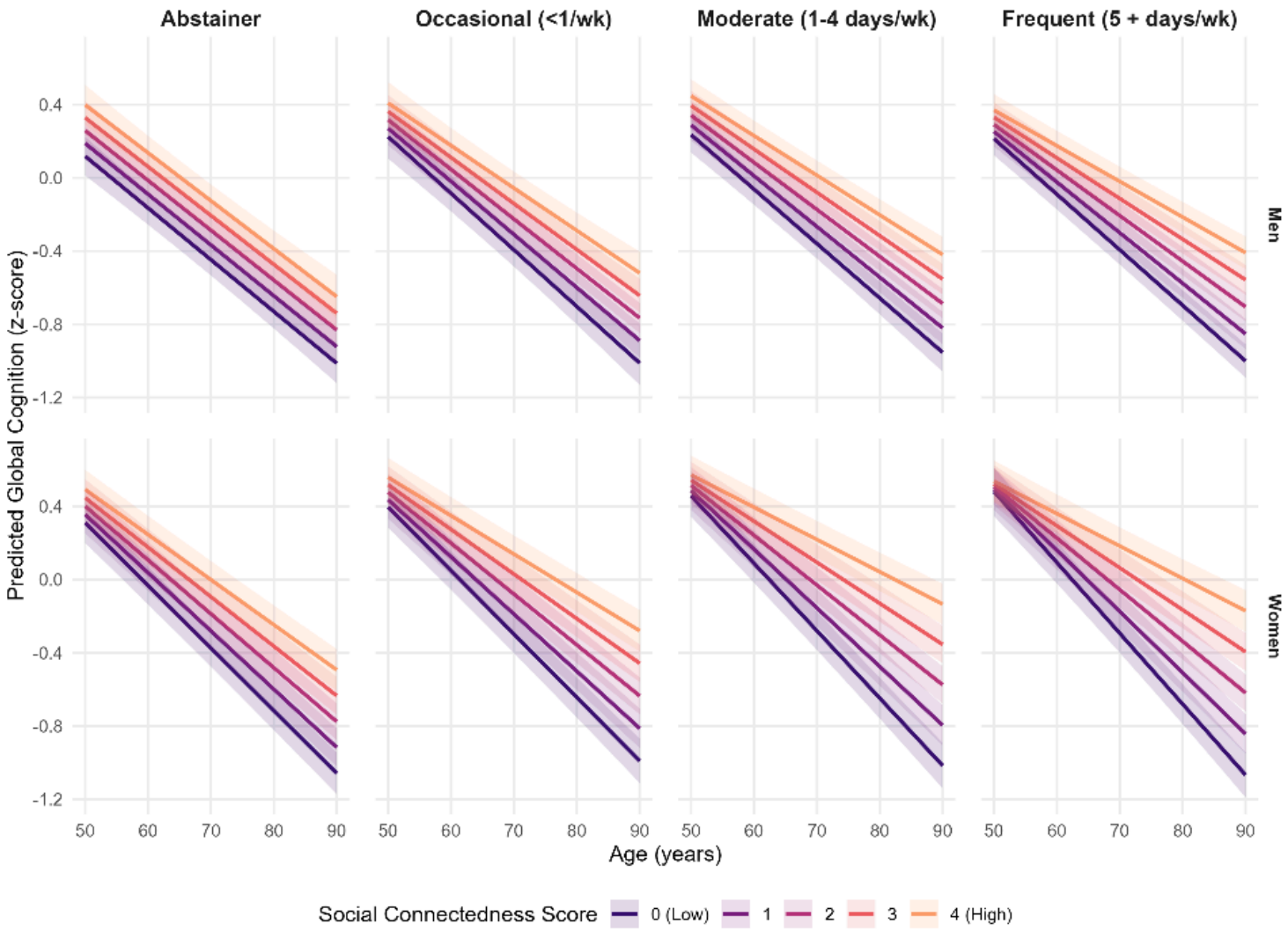



**Figure 4**

Cognitive trajectories over age - impact of social connectedness and drinking frequency

## Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- SupplementaryinformationAlcoholandcognitiveageing.docx