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.

Self-reported alcohol consumption and all-cause mortality during 20 years of follow-up in a Swedish population

Michel Erik Said Watz

michel.watz.cherigui@gu.se

University of Gothenburg

Kristin Ottarsdottir

University of Gothenburg

Margareta Inger Hellgren

University of Gothenburg

Yufang Zheng

University of Gothenburg

Ying Li

University of Gothenburg

Bledar Daka

University of Gothenburg

Ulf Lindblad

University of Gothenburg

Research Article

Keywords: Alcohol Drinking, Cohort Studies, Mortality, Risk Factors, Survival Rate, Public Health

Posted Date: November 11th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-10922947/v1

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

Additional Declarations: No competing interests reported.

Self-reported alcohol consumption and all-cause mortality during 20 years of follow-up in a Swedish population

Authors published names: Watz MES[1], Ottarsdottir K[1], Hellgren MI[1], Zheng Y[1], Li Y[2], Daka B[1], Lindblad U[1]

Authors full names (+highest academic degree): Michel Erik Said WATZ CHERIGUI [1] (MD), Kristin OTTARSDOTTIR[1] (MD, PhD), Margareta Inger HELLGREN[1] (MD, PhD), Yufang Zheng[3] (PhD), Ying LI[2] (PhD), Bledar DAKA[1] (MD, PhD), Ulf LINDBLAD[1] (MD, PhD)

1 Family Medicine, School of Public Health and Community Medicine, Institute for Medicine, Sahlgrenska Academy, University of Gothenburg, Gothenburg

2 Biostatistics, School of Public Health and Community Medicine, Institute for Medicine, Sahlgrenska Academy, University of Gothenburg, Gothenburg

Number of tables: 3

Number of figures: 1

Number of additional files: 3

Corresponding author: Michel Watz

Contact info:

Address: Department of Public Health and Community Medicine, University of Gothenburg, Guldhedsgatan 5 A, Box 453, 405 30 Göteborg, Sweden

Email address: michel.watz.cherigui@gu.se

Telephone: +46 31-786 00 00

Article summary

Abstract

Background

While excessive alcohol consumption increases mortality, the threshold remains uncertain. This study aimed to investigate the association between self-reported alcohol consumption and all-cause mortality in a representative Swedish population.

Methods

In 2002–2005, a random sample of 2,816 participants aged 30–75 years (mean 48 years) from two Swedish communities was recruited (participation rate 76%) focusing on cardiometabolic risk factors, lifestyle, and social determinants of health. Self-reported exposure to alcohol was categorised based on mean weekly ethanol intake: abstainers, occasional drinkers (<20 g/week), low (20 to <60 g/week), moderate (60 to <120 g/week), high (120 to <240 g/week), and highest consumption (≥240 g/week). Mortality follow-up data were available through 31 December 2022.

Hazard ratios (HR) for all-cause mortality were estimated using multivariable Cox regression with occasional drinkers as the reference group. Analyses were adjusted for demographic, lifestyle, socioeconomic and cardiometabolic risk factors.

Results

During a mean follow-up of 19.2 years, 301 deaths occurred. In the final model, the highest consumers had a HR of 2.0 for mortality (95% CI 1.07-3.75). There was no evidence that low or moderate consumption influenced mortality risk (HR=0.84; 95% CI 0.59-1.19 and HR= 1.17; 95% CI 0.76-1.81, respectively). Abstainers had an increased mortality after adjustments for age and sex (HR=1.38; 95% CI 1.02-1.86), but it was attenuated and non-significant after further adjustments.

55 **Conclusion**

56 These findings are consistent with current recommendations limiting alcohol consumption to
57 no more than 10 standard drinks per week and provide no evidence for a protective effect of
58 moderate drinking on mortality.

59

60 **Keywords**

61 Alcohol Drinking, Cohort Studies, Mortality, Risk Factors, Survival Rate, Public Health

62

63

Manuscript

Introduction

Excessive alcohol consumption is well-established as a risk factor for cardiovascular disease and death, though the precise level at which alcohol becomes harmful remains uncertain. Approximately 26% of alcohol-related deaths are due to cardiovascular disease[1]. Nevertheless, there is a substantial body of research from population-based studies that has reported reduced mortality in light to moderate drinking compared to no drinking[2, 3]. These conflicting findings have sometimes been attributed to residual confounding present in abstainers[4], who include people with poorer health. The use of occasional drinkers, sometimes defined as consuming less than 20 g of alcohol per week, as reference group has been adopted in some studies to reduce the "sick quitter" effect and other health-related biases present in the abstainer category. It is unlikely that direct physiological effects of <20 g of ethanol per week would materially affect mortality.

A recently updated meta-analysis showed no clear association with mortality in consumers of between 9 and 175 g of pure ethanol per week, compared to those who consumed less[5]. The 2023 Guidelines from the National Board of Health and Welfare (NBHW) in Sweden were partly based on this meta-analysis, which has generated some debate[6-9]. According to the new guidelines, the lower threshold for so-called "risk use" was harmonised for men and women at 120 g per week, lowering the previous limit for men from 168 g[10]. Individuals classified as having risk use are recommended to be offered medical intervention where appropriate.

However, uncertainty remains regarding the mortality threshold associated with different levels of alcohol consumption in contemporary population-based cohorts, particularly in relation to recently defined national risk-use thresholds. Therefore, in this study we

investigated the association between levels of alcohol consumption and all-cause mortality using a population-based cohort and explored the potential dose-response relationship. We evaluated the newly defined NBHW risk use thresholds in relation to mortality. We also hypothesised that the excess mortality risk observed among abstainers would be explained by comorbidity and other risk factors.

Methods - Design

This community-based study used data from the Vara–Skövde Cohort. Between 2002 and 2005, a random sample of 2,816 participants aged 30–75 (mean age 48 years) from two rural communities in Sweden were recruited from the population census register and examined, with a focus on cardiometabolic risk factors, lifestyle, and psychological symptoms (participation rate 76%). Details on the baseline survey of this cohort have previously been described[11]. The study size was arrived at from similar cohort studies conducted by the research group[11]. The mortality was retrieved from the Swedish cause of death register including all the deaths in Sweden since 1952. A representative sample of 1954 individuals were invited to a follow-up examination (participation rate 68%) undergoing a second examination. The studies were approved by the medical faculty's research ethics committee at the University of Gothenburg. All participants gave written informed consent.

Methods - data collection

Data on alcohol consumption during the previous 30 days were collected using a validated questionnaire[12]. This consisted of quantity-frequency questions and additional questions regarding the number of days of atypical high consumption (see Additional file 1). Data on mortality were collected from the National Mortality Register until 31 December 2022. Data on lifestyle was collected using standard questionnaires, including leisure time physical activity (LTPA)[11] and current smoking. LTPA was dichotomized into sedentary or physically active. Level of education was self-reported and was categorised as lower (primary

and secondary education, ≤9 years) or higher. A single-person household was defined as an individual living alone. Waist circumference was measured between the lowest rib margin and iliac crest by a specially trained nurse. A supine resting blood pressure was measured in accordance with guidelines from the European Society of Hypertension[13]. In the case of elevated blood pressure, three additional measurements were made and a diagnosis of hypertension was given according to JNC7 guidelines[14]. A standard oral glucose tolerance test (OGTT) was performed, and previously undiagnosed cases of diabetes were identified in accordance with 1999 WHO guidelines[15]. Self-reported data on hypertension, diabetes mellitus, as well as previous cardiovascular disease, here defined as congestive heart failure, atrial fibrillation, angina pectoris, coronary artery bypass surgery and stroke, were systematically collected using a structured interview by a study nurse[11].

Primary outcome measures

The main outcome was all-cause mortality. The patients were censored either due to death or the date of the retrieval of the death register, i.e. 2022-12-31.

Methods – exposure of alcohol consumption

The categories were based on commonly consumed amounts and existing guidelines (Table 1).

Table 1. Alcohol consumption and crude mortality

Self-reported amount of consumed alcohol per week	
Abstainers	no drinking in the past 12 months
Occasional drinkers	<20 g/week
Low consumption	20 up to <60 g/week
Moderate consumption	60 up to <120 g/week
High consumption	120 up to <240 g/week
Highest consumption	≥ 240 g/week

Definition of the consumption categories. g=grams

To assess stability of alcohol consumption over time in the cohort, a McNemar-Bowker test was performed in crosstabulation of the consumption category of each participant in baseline and at the follow-up examination, respectively. The average change in grams of ethanol per week and standard deviation was also calculated.

Methods - data analysis

Descriptive statistics were presented as means with standard deviations for continuous variables and frequencies with percentages for categorical variables. Kaplan–Meier curves were visually inspected across categories. There was no crossing of the survival curves nor any indication of changing separation over time. Therefore, the proportional hazards assumption was accepted prior to Cox regression. Individuals with missing data at baseline on any included variables were excluded from all analyses (See Additional file 2 – flow chart of the study population).

Cox proportional hazards regression was used to estimate hazard ratios (HRs) for all-cause mortality during the follow-up time across predefined categories of alcohol consumption, with occasional drinkers as the reference group. Follow-up time was defined as the time in calendar years from inclusion in the study to date of death or end of follow-up time (2022-12-31). Baseline covariates were used to adjust for possible confounding across different models. Analyses were performed with four consecutive models to assess the impact of confounders, with covariates added sequentially. Model 1 was adjusted for age and sex. Model 2 added waist circumference, smoking, and LTPA. Model 3 added single household status and education level. Model 4 additionally included previous cardiovascular disease, diabetes mellitus, and hypertension. As a sensitivity analysis, the same analyses were repeated limited to 10-year follow-up. An interaction between sex and alcohol consumption was included in the model to assess the possibility of a sex modification effect. All tests were two-sided, and

statistical significance was defined as $p<0.05$. All statistical analyses were performed using IBM SPSS Statistics version 29 (IBM Corp., Armonk, NY, USA). The study was reported in accordance with the STROBE guidelines (see Additional file 3), and the checklist was used in the revision of the manuscript[16].

Results

After exclusion of 221 individuals with missing data on one or more of the included variables, 2,595 remained. Characteristics at baseline are displayed in Table 2. [insert Table 2 here]

Women reported less alcohol consumption, and only 14.2% and 7.1% in the high (120 to <240 g/week) and highest (≥240 g/week) consumption categories were women, corresponding to 1.6% and 0.2% of all women in the cohort, respectively. Crude mortality was highest among abstainers (20.2%) and among participants with the highest alcohol consumption (28.6%), and consistently the prevalence of established risk factors for mortality were higher in these categories.

At 10 years follow-up, in a sub-sample of 1214 individuals for which data was available, no significant change was seen when comparing individual allocation to each alcohol consumption category at baseline vs follow-up examination (McNemar-Bowker 0.59). The mean change in alcohol consumption for the entire cohort was -5 grams per week (SD 62 grams).

During a mean follow-up of 19.2 years, 301 deaths occurred. No statistically significant interaction between sex and alcohol consumption was observed in relation to mortality ($p>0.20$). An illustration of cumulative survival over 20 years follow-up by alcohol consumption category is shown in Figure 1.

Figure 1. Cumulative adjusted survival in Cox regression full model

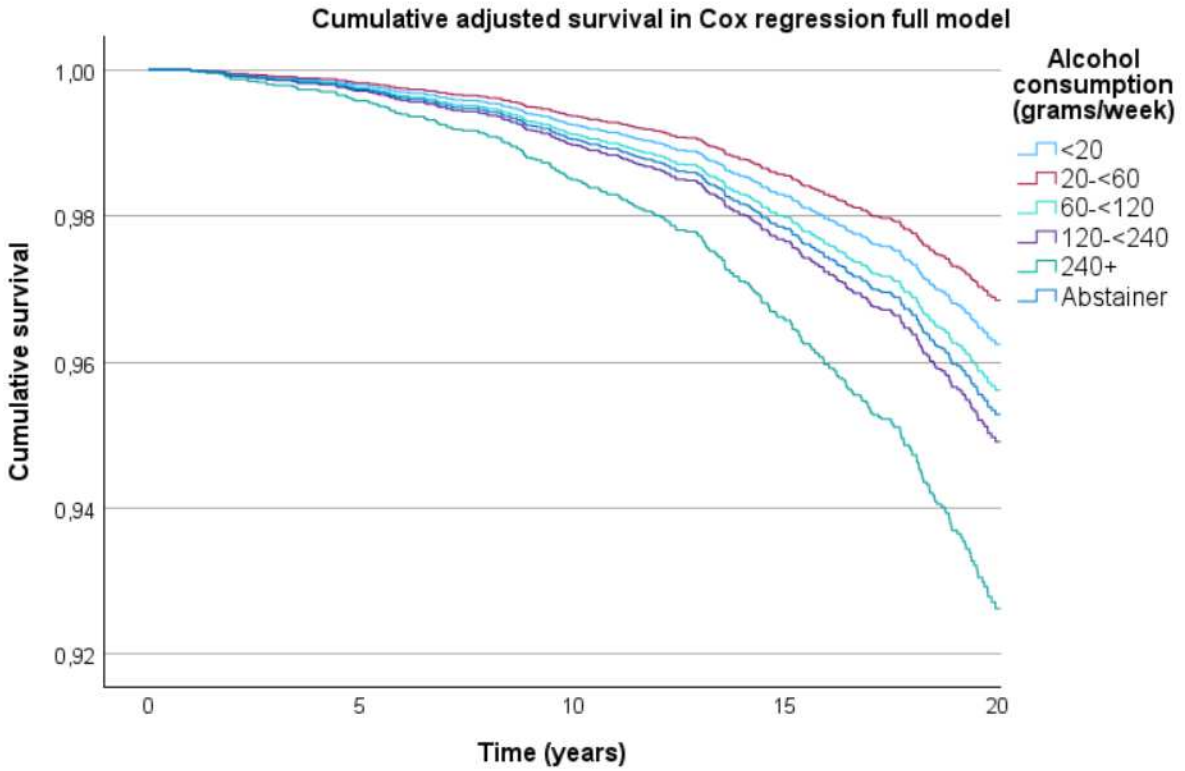


Figure 1 legend. Adjusted survival curves over 20 years follow up by categories of self-reported alcohol consumption at baseline. Curves are based on fully adjusted Cox proportional hazards model with covariates fixed at their mean values. Covariates included sex, age, waist circumference, smoking, leisure time physical activity, single household status, educational level, hypertension, diabetes mellitus, and cardiovascular disease.

During the first 10 years follow-up there was a statistically nonsignificant increase in mortality among those with the highest consumption (HR=2.5; 95% CI 0.93 to 6.80) after full adjustments compared to occasional consumers. With longer follow-up participants in the highest consumption category showed increasing separation from the other categories over time (Figure 1). Hazard ratios for mortality across the different consumption categories are shown in table 3.

Table 3. Hazard Ratio (HR) for mortality across categories of self-reported consumption in four sequentially adjusted models.

	Model 1	Model 2	Model 3	Model 4
	HR (95% CI)	HR (95% CI)	HR (95% CI)	HR (95% CI)

Abstainer	1.38 (1.02-1.86)	1.35 (1.00-1.83)	1.31 (0.97-1.78)	1.26 (0.93-1.72)
<20	1 [Reference]	1 [Reference]	1 [Reference]	1 [Reference]
20 to <60	0.79 (0.56-1.12)	0.78 (0.55-1.10)	0.83 (0.58-1.18)	0.84 (0.59-1.19)
60 to <120	0.98 (0.64-1.50)	1.05 (0.69-1.61)	1.13 (0.73-1.72)	1.17 (0.76-1.81)
120 to <240	1.22 (0.74-2.02)	1.17 (0.71-1.94)	1.25 (0.75-2.06)	1.37 (0.82-2.27)
≥240	2.00 (1.08-3.72)	1.88 (1.01-3.50)	2.00 (1.07-3.72)	2.00 (1.07-3.75)

Abbreviations: HR, hazard ratio; CI, confidence interval; NA, not applicable. Occasional drinkers (<20 g/week) were used as a reference.

Model 1 adjusted for age and sex.

Model 2 added smoking, waist circumference, and physical activity.

Model 3 added single household status and educational level.

Model 4 added diabetes mellitus, hypertension, and cardiovascular disease.

After adjustments for age and sex, the highest consumption category had a significant increased mortality risk compared with occasional drinkers (HR=2.00; 95% CI 1.08 to 3.72). This association remained after full multivariable adjustment (HR=2.00; 95% CI 1.07 to 3.75).

Abstainers had a higher mortality risk than occasional drinkers after adjustment for age and sex (HR=1.38; 95% CI 1.02 to 1.86). After further adjustments for lifestyle factors and comorbidity, the association was attenuated and no longer statistically significant (HR=1.35; 95% CI 1.00 to 1.83). No differences in mortality risk were observed between occasional drinkers and participants with low or moderate alcohol consumption (low consumption HR=0.84; 95% CI 0.59 to 1.19; moderate consumption HR=1.17; 95% CI 0.76 to 1.81 in the final model).

Discussion

In this representative Swedish population-based cohort study, self-reported alcohol consumption of ≥ 240 g of ethanol per week was associated with a twofold increase in all-cause mortality compared with occasional drinkers, after adjustment for major risk factors for death. In fully adjusted models, no differences in mortality risk were observed among abstainers or among participants with low, moderate, or high alcohol consumption compared with occasional drinkers. The higher mortality observed among abstainers in model 1 may reflect a higher burden of major risk factors and comorbidity, underscoring concerns about residual confounding when abstainers are used as the reference group in observational studies.

The meta-analysis of Zhao et al used a similar design, with occasional drinkers as the primary reference group[5] although complementary analyses using abstainers as a reference group were also included. Alcohol consumption was examined across other levels of consumption. Low consumption (1.3 to <25 g per day) was not associated with increased mortality compared with 25–45 g per day, except in the partially adjusted model (HR=1.09; 95% CI 1.00 to 1.19). Mortality was higher in consumers of 45 to <65 per day and ≥65 g per day (HR=1.24; 95% CI 1.07 to 1.44 and HR=1.41; 95% CI 1.23 to 1.61, respectively). Zhao et al also highlighted the frequent lack of information on lifetime abstainers in many of the conducted studies, a limitation shared by the current study, supporting the use of occasional drinkers as the reference group.

Similarly, an article in The Lancet from 2018 based on individual participant data from 83 prospective studies, restricted to current drinkers, reported a progressive reduction in life expectancy from 40 years of age among individuals consuming more than 100 g of alcohol per week compared with those consuming 100 g of alcohol per week or less[17]. That study included fewer covariates and individuals with previous cardiovascular disease were excluded. Compared to our study, it included older individuals with a higher alcohol

consumption and a greater proportion of smokers. In contrast to our study, alcohol consumption was analysed in broader intervals of approximately 100 g.

Several meta-analyses or systematic reviews conducted at government agencies, for example in Canada[18] and the United States of America (USA)[19] have reached differing conclusions. In Canada, low risk consumption is defined as less than 26 g of ethanol per week, while US reviewers have concluded that moderate consumption (up to 98 or 196 grams of ethanol per week for women and men respectively) compared to lifetime non-drinking is associated with a lower risk of mortality. These divergent conclusions underline the fact that despite a vast number of studies, there is no consensus on whether alcohol itself can still be claimed to have a protective effect on mortality. Differences in policy recommendations may also reflect variations in interpretation of the evidence and broader public health priorities. In our, albeit smaller, study, we showed a clear increase of mortality among individuals consuming ≥240 g of alcohol per week. There were non-significant trends in moderate consumers towards a lower mortality, but this should be interpreted with caution.

The Global Burden of Disease Study conducted a large meta-analysis on alcohol and disease burden, and arrived at the conclusion that no level of alcohol consumption has the lowest risk of disability-adjusted life years (DALYs)[20]. This study included multiple sources for consumption including sales data, which lowers the risk of bias. The use of DALYs provides vital information on the consequences of alcohol consumption, especially at lower doses where morbidity is increased but not mortality.

A UK cohort study including adults aged 60 or older, with a median follow-up of 12.4 years found an increase in all-cause mortality among individuals consuming 140 to 280 g of alcohol per week for men and 70-140 g per week for women, compared with occasional drinkers (HR=1.10; CI 1.03 to 1.18) [21]. That study also accounted for socio-economic status, frailty, beverage type and drinking only with meals. Additionally, cancer mortality was increased

even at lower consumption levels (140 and 70 g per week for men and women respectively). Compared with the present study, the UK cohort included older individuals and focused more on other aspects of drinking pattern, which may be associated with other lifestyle choices with effects on morbidity and mortality. These differences also highlight the potential importance of socioeconomic factors on modifying the effects of alcohol. The current study design allows for exploration of the non-linear association between alcohol and mortality, often referred to as J-shaped. However, our study suggests that the elevated mortality risk observed in abstainers is due to accumulation of risk factors and comorbidity, which is supported by the absence of a difference in mortality risk between abstainers and occasional drinkers, when adjusting for confounders.

Strengths and limitations

A major strength of our study is the high participation rate and the comprehensive assessment of key variables known to influence mortality risk, enabling robust adjustment for important confounders often unaccounted for in observational studies. The cohort is relatively modest in size, and statistical power varies between consumption categories. However, the low- and moderate-consumption categories comprise over half of the cohort, reducing, although not eliminating, the risk of type II error and supporting the robustness of the null findings in these categories. This pattern strengthens the interpretation that excess mortality is confined to the highest consumption category, but due to lack of a clear dose-response relationship, it is not certain that this constitutes a threshold effect.

The high consumption category was defined partly according to recent Swedish guidelines for *risk drinking,* which coincide with other national guidelines[10]. In this study, no statistically significant increase in mortality was observed at this level of consumption after 19 years of follow-up. As this is an observational study, it does not allow for the establishment of a cause-and-effect relationship. Nevertheless, the strong and consistent association observed between

very high alcohol consumption and mortality is consistent with a causal relationship and is well aligned with existing evidence.

The use of self-reported alcohol consumption is known to be affected by under-reporting due to recall bias, social desirability, and concerns about potential legal consequences[22, 23]. The questionnaire used in this study has previously been validated in an ecological study and was estimated to cover up to 77% of total alcohol sales[12], but some degree of under-reporting is likely. Differences in under-reporting consumption between categories may also have influenced the results[24]. Patterns of binge-drinking, which are known to impact adverse outcomes, were not examined in the present study.

Exposure to alcohol consumption was assessed at a single time point, which may have reduced the precision of long-term exposure estimation. However, alcohol consumption is generally more stable over time in older adults compared to younger individuals, who often show decreasing consumption over time[25]. Additionally, we examined the change in consumption in a representative sub-sample that was invited to a secondary examination at approximately 10 years, and consumption did not change significantly.

Conclusion and implications

High-volume alcohol consumption (≥240 g/week) was associated with increased all-cause mortality. For consumption levels corresponding to the current Swedish threshold for risk use (120–240 g/week), mortality risk estimates were elevated but imprecise and did not reach statistical significance. The findings therefore support efforts to limit high alcohol consumption while providing no evidence for a protective effect of moderate drinking. Abstainers may be a suboptimal reference group in observational studies of alcohol consumption and health due to residual confounding related to comorbidity and adverse risk profiles, which may partly explain the frequently reported J-shaped association between

alcohol consumption and health outcomes. Further research is needed to clarify the impact of alcohol consumption on specific morbidity outcomes associated with different patterns of alcohol use.

Generalisability

The findings are likely to be generalisable to middle-aged adults in similar high-income countries, predominantly Caucasian populations, but caution is warranted when extrapolating to populations with different drinking patterns, ethnic compositions, or healthcare systems.

Author Contributions

MW and UL: Conceptualisation, methodology, formal analysis, writing – original draft.

KO, MH, YZ, YL and BD: Data interpretation, writing – review and editing.

All authors: Final approval of the version to be published and agreement to be accountable for all aspects of the work in ensuring questions relating to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

Additional files

Additional file 1 (.docx) titled “Alcohol Quantity Frequency questions” is the English version of the self-assessment form used in this study for quantifying alcohol consumption.
Additional file 2 (.png) titled “Flow chart of the study population” illustrates inclusion and exclusion of participants from the study.
Additional file 3 (.docx) titled “the STROBE reporting checklist” is the reporting checklist used in the revision of this manuscript.

Statements and declarations

Ethics Approval

The study was approved by the *Medical Faculty's Research Ethics Committee* at the University of Gothenburg diary number 036-12 and 199-01.

Consent to participate

All participants provided written informed consent to participate in the study.

Consent for publication

Not applicable. No individual data is published in the study.

Declaration of conflicting interests

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Clinical trial number: not applicable

Funding statement

The authors disclosed receipt of the following financial support for the research, authorship, and/or publication of this article: This work was supported by the Skaraborg Institute, Skövde, Sweden [grant number 21/1030]; The Strategic Healthcare Committee, Region Västra Götaland [grant number VGFOUREG-993687]; by the ALF agreement / Swedish Government funding for clinical research [grant number ALFGBG-971147], [grant number ALFGBG-1006749]. Funders had no role in study design, data collection, data analysis, manuscript preparation, or decision to publish.

Data availability statement

The datasets generated and/or analysed during the current study are not publicly available as they include pseudo-anonymized information subject to ethical approval and applicable data protection regulations but are available from the corresponding author on reasonable request.

References

1. Rehm J, Shield KD, Roerecke M, Gmel G: **Modelling the impact of alcohol consumption on cardiovascular disease mortality for comparative risk assessments: an overview**. *BMC Public Health* 2016, **16**(1):363.
2. Ronksley PE, Brien SE, Turner BJ, Mukamal KJ, Ghali WA: **Association of alcohol consumption with selected cardiovascular disease outcomes: a systematic review and meta-analysis**. *BMJ* 2011, **342**:d671.
3. Di Castelnuovo A, Costanzo S, Bagnardi V, Donati MB, Iacoviello L, de Gaetano G: **Alcohol dosing and total mortality in men and women: an updated meta-analysis of 34 prospective studies**. *Arch Intern Med* 2006, **166**(22):2437-2445.
4. Tsai MK, Gao W, Wen CP: **The relationship between alcohol consumption and health: J-shaped or less is more?** *BMC Med* 2023, **21**(1):228.
5. Zhao J, Stockwell T, Naimi T, Churchill S, Clay J, Sherk A: **Association Between Daily Alcohol Intake and Risk of All-Cause Mortality: A Systematic Review and Meta-analyses**. *JAMA Network Open* 2023, **6**(3):e236185-e236185.
6. Helander A, Jones AW: **Gränsvärden för riskbruk av alkohol bör individanpassas**. *Lakartidningen* 2024, **121**.
7. Stenbäck MRM: **Socialstyrelsens nya alkoholråd saknar vetenskapligt stöd**. *Läkartidningen* 2024.
8. Lindén TvM, Sofia: **Förtida död är inte den enda risken med alkohol**. *Läkartidningen* 2024.
9. Stenbäck MRM: **Ingen ökad totalrisk vid låg eller måttlig alkoholkonsumtion**. *Läkartidningen* 2024.
10. Socialstyrelsen: **Interventions for risky use of alcohol - Recommendations for health care and dentistry (in Swedish)**. In.; 2023.
11. Nyholm M, Gullberg B, Haglund B, Råstam L, Lindblad U: **Higher education and more physical activity limit the development of obesity in a Swedish rural population. The Skaraborg Project**. *International Journal of Obesity* 2008, **32**(3):533-540.
12. Göransson M, Hanson BS: **How much can data on days with heavy drinking decrease the underestimation of true alcohol consumption?** *J Stud Alcohol* 1994, **55**(6):695-700.
13. Stergiou GS, Palatini P, Parati G, O'Brien E, Januszewicz A, Lurbe E, Persu A, Mancia G, Kreutz R: **2021 European Society of Hypertension practice guidelines for office and out-of-office blood pressure measurement**. *J Hypertens* 2021, **39**(7):1293-1302.
14. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL, Jr., Jones DW, Materson BJ, Oparil S, Wright JT, Jr. *et al*: **Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure**. *Hypertension* 2003, **42**(6):1206-1252.
15. Organization WH: **Definition, diagnosis and classification of diabetes mellitus and its complications : report of a WHO consultation. Part 1, Diagnosis and classification of diabetes mellitus**. In. Geneva, Switzerland; 1999.
16. von Elm E, Altman DG, Egger M, Pocock SJ, Gøtzsche PC, Vandenbroucke JP: **Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies**. *Bmj* 2007, **335**(7624):806-808.
17. Wood AM, Kaptoge S, Butterworth AS, Willeit P, Warnakula S, Bolton T, Paige E, Paul DS, Sweeting M, Burgess S *et al*: **Risk thresholds for alcohol consumption: combined analysis of individual-participant data for 599 912 current drinkers in 83 prospective studies**. *Lancet* 2018, **391**(10129):1513-1523.
18. Paradis C, Butt, P., Shield, K., Poole, N., Wells, S., Naimi, T., Sherk, A., & the Low-Risk Alcohol Drinking Guidelines Scientific Expert Panels: **Canada's Guidance on Alcohol and Health: Final Report**. In.: Ottawa, Ont.: Canadian Centre on Substance Use and Addiction; 2023.

19. National Academies of Sciences E, and Medicine; Health and Medicine Division; Food and Nutrition Board; Committee on Review of Evidence on Alcohol and Health; Stone KB, Calonge BN, editors: **Review of Evidence on Alcohol and Health**, vol. All-Cause Mortality. Washington (DC): National Academies Press (US); 2025.
20. **Alcohol use and burden for 195 countries and territories, 1990-2016: a systematic analysis for the Global Burden of Disease Study 2016**. *Lancet* 2018, **392**(10152):1015-1035.
21. Ortolá R, Sotos-Prieto M, García-Esquinas E, Galán I, Rodríguez-Artalejo F: **Alcohol Consumption Patterns and Mortality Among Older Adults With Health-Related or Socioeconomic Risk Factors**. *JAMA Netw Open* 2024, **7**(8):e2424495.
22. Hoonpongsimanont W, Ghanem G, Chen Y, Sahota PK, Carroll C, Barrios C, Lotfipour S: **Underreporting of alcohol use in trauma patients: A retrospective analysis**. *Subst Abus* 2021, **42**(2):192-196.
23. Davis CG, Thake J, Vilhena N: **Social desirability biases in self-reported alcohol consumption and harms**. *Addict Behav* 2010, **35**(4):302-311.
24. Boniface S, Kneale J, Shelton N: **Drinking pattern is more strongly associated with under-reporting of alcohol consumption than socio-demographic factors: evidence from a mixed-methods study**. *BMC Public Health* 2014, **14**:1297.
25. Shaw BA, Krause N, Liang J, McGeever K: **Age differences in long-term patterns of change in alcohol consumption among aging adults**. *J Aging Health* 2011, **23**(2):207-227.

Table 2. Characteristics of the study population by alcohol consumption category at baseline

	Abstainer N=534 (20.6%*)	<20 g/week N=714 (27.5%*)	20-<60 g/week N=790 (30.4%*)	60-<120 g/week N=367 (14.1%*)	120-<240 g/week N=148 (5.7%*)	≥240 g/week N=42 (1.6%*)	All (N=2595)
	N (%)	N (%)	N (%)	N (%)	N (%)	N (%)	N (%)
Women	343 (64.2)	466 (65.3)	371 (47.0)	88 (24.0)	21 (14.2)	3 (7.1)	1292 (49.8)
LTPA (Sedentary)	398 (74.5)	465 (65.1)	497 (62.9)	228 (62.1)	96 (64.9)	29 (69.0)	1713 (66.0)
Single household	125 (23.4)	122 (17.1)	136 (17.2)	84 (22.9)	27 (18.2)	14 (33.3)	508 (19.6)
Lower education	219 (41.0)	162 (22.7)	173 (21.9)	83 (22.6)	47 (31.8)	19 (45.2)	703 (27.1)
CVD	34 (6.4)	30 (4.2)	20 (2.5)	7 (1.9)	4 (2.7)	2 (4.8)	97 (3.7)
Diabetes	47 (8.8)	30 (4.2)	33 (4.2)	13 (3.5)	5 (3.4)	3 (7.1)	131 (5.0)
Hypertension	114 (21.3)	87 (12.2)	92 (11.6)	32 (8.7)	20 (13.5)	14 (33.3)	359 (13.8)
Smoking	99 (18.5)	93 (13.0)	149 (18.9)	59 (16.1)	27 (18.2)	10 (23.8)	437 (16.8)
	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)
Age, years	50.2 (13.3)	46.7 (11.4)	46.3 (10.3)	45.6 (9.9)	47.1 (10.5)	51.1 (12.0)	47.2 (11.4)
WC, cm	89.6 (14.1)	88.5 (13.1)	89.3 (12.0)	92.0 (11.4)	93.8 (10.4)	97.4 (11.4)	89.9 (12.7)

*Categories are based on average self-reported intake of ethanol in g/week. Abstainers reported no consumption in the previous 12 months. SD=standard deviation. WC: waist circumference. BMI: body mass index. LTPA: leisure time physical activity. CVD: cardiovascular disease. g: grams. *: row percentage*