PREVENTING ALCOHOL-RELATED HARM

RESEARCH SUMMARY

Date Compiled: December 2025

Key takeaways from included research:

- Researchers found that using strong causal language (e.g., "alcohol causes cancer") in warning labels did not increase perceived risk or change drinking intentions but reduced credibility and increased negative reactions. In contrast, softer modal language (e.g., "alcohol may cause cancer") lowered perceived risk but made messages seem more credible and less reactive, highlighting tradeoffs in how alcohol–cancer warnings are phrased.
- A Japanese study of nearly 59,000 adults found that stopping alcohol use was linked to small but measurable reductions in blood pressure, even among light-to-moderate drinkers. Starting to drink led to dose-dependent increases in blood pressure for both men and women, regardless of beverage type. These findings suggest that even modest alcohol reduction or cessation can help lower blood pressure and support heart health.
- An Australian study found that adolescents who began drinking earlier in life showed higher levels of heavy drinking, alcohol-related harms, and alcohol use disorder by age 20, compared to those who began drinking later. The study supports delaying alcohol initiation until adulthood to reduce long-term risks and harm.
- A new study of over 559,000 adults examined whether alcohol use is linked to dementia. Traditional observational results suggested light drinkers seemed to have lower dementia risk than non-drinkers or heavy drinkers, but this was likely due to reverse causation—people in early stages of dementia often reduce drinking. Genetic analyses showed a clear, steady increase in dementia risk with higher alcohol consumption, with no evidence that light drinking is protective. A higher risk was also seen among people with alcohol use disorder. Overall, the study concludes that any level of alcohol use may increase dementia risk, and reducing alcohol problems could help prevent dementia by up to 16%.

WARNING LABEL MESSAGES ABOUT THE CANCER RISK ASSOCIATED WITH ALCOHOL: EFFECTS OF CAUSAL LANGUAGE
November 2025

Objectives: To investigate the effects of causal language in warning messages about alcohol and cancer risk.

Methods: In an online factorial experiment conducted in 2023, 799 US adults were randomly assigned to view one of eight messages employing different combinations of causal verbs (e.g., "causes") and modal verbs (e.g., "may") to describe the alcohol-cancer relationship. Analyses compared the effects of causal language on perceptions of causal certainty, alcohol-related cancer risk, message credibility, message reactance, and alcohol consumption intentions.

Results: Causal language did not affect perceived causal certainty, perceived cancer risk, or alcohol consumption intentions, but decreased perceived message credibility and increased reactance. Modal language decreased perceived causal certainty and cancer risk, increased perceived credibility of messages containing "causes," and decreased reactance to all messages.

Conclusions: Causal language in alcohol warning messages has complex, countervailing effects: higher-certainty causal verbs do not increase risk perceptions but increase negative reactance, whereas higher-uncertainty modal verbs decrease risk perceptions but also reactance. These results suggest causal language may pose potential tradeoffs for risk communication efforts, raising the need for caution and more research to understand its effects.

Source: Payne, J. B., Rohde, J. A., Garrido, C. O., Jesch, E., Klein, W. M., Berrigan, D., ... & Han, P. K. (2025). Warning label messages about the cancer risk associated with alcohol: effects of causal language. *Addictive Behaviors*, 108453. https://doi.org/10.1016/j.addbeh.2025.108453

BLOOD PRESSURE AFTER CHANGES IN LIGHT-TO-MODERATE ALCOHOL CONSUMPTION IN WOMEN AND MEN: LONGITUDINAL JAPANESE ANNUAL CHECKUP ANALYSIS
October 2025

Background: Alcohol consumption is a known contributor to elevated blood pressure (BP). However, the association between changes in light-to-moderate alcohol consumption (eg, ≤1 drink for women and ≤2 drinks for men daily), especially cessation, and BP remains unclear.

Objective: In this study, the authors sought to evaluate the associations between alcohol cessation/initiation and subsequent BP changes, focusing on sex-specific estimates, light-to-moderate intake, and type of alcoholic beverage.

Methods: We analyzed data from adults undergoing annual health checkups at a Japanese preventive medicine center from October 2012 to March 2024. Systolic blood pressure (SBP) and diastolic blood pressure (DBP) changes between consecutive visits were evaluated. Alcohol intake was self-reported as standard drinks (1 drink = 10 g ethanol). Associations between alcohol cessation/initiation and BP changes were assessed with the use of generalized estimating equations, adjusted for demographics, clinical history, and lifestyle behaviors.

Results: Among 359,717 visits from 58,943 participants, the cessation cohort (53,156 visits, 25,621 participants, median age 50.5 years, 52.1% women) revealed that cessation was associated with dose-dependent reductions in BP. Among women, cessation of 0.5 to 1.0 drinks per day did not correspond to a statistically significant change in SBP (−0.44 mm Hg; 95% CI: −0.93 to 0.06 mm Hg) and corresponded to a change of −0.41 mm Hg (95% CI: −0.77 to −0.05 mm Hg) in DBP, and cessation of 1.0 to 2.0 drinks per day corresponded to changes of −0.78 mm Hg (95% CI: −1.53 to −0.04 mm Hg) in SBP and −1.14 mm Hg (95% CI: −1.68 to −0.61 mm Hg) in DBP. Among men, cessation of 0.5 to 1.0 drinks per day did not correspond to a statistically significant change in SBP (−0.27 mm Hg; 95% CI: −0.81 to 0.27 mm Hg) or DBP (−0.39 mm Hg; 95% CI: −0.77 to 0.01 mm Hg), and cessation of 1.0 to 2.0 drinks per day corresponded to changes of −1.03 mm Hg (95% CI: −1.70 to −0.35 mm Hg) in SBP and −1.62 mm Hg (95% CI: −2.11 to −1.12 mm Hg) in DBP. The initiation cohort (128,552 visits, 31,532 participants, median age 50.0 years, 70.4% women) demonstrated dose-dependent increases in BP after alcohol initiation, and the overall direction of the effect was consistent across sexes. Beverage-specific analyses revealed similar BP effects regardless of alcohol type in both cohorts.

Conclusions: Even at low levels, drinking was associated with higher BP, and cessation was associated with lower BP in both sexes. These findings suggest that alcohol cessation is a broadly applicable strategy even among light-to-moderate drinkers for BP management. In this large dataset, BP changes in women associated with consumption at levels as low at 0.5 to 1 drinks per day are detectable.

Source: Suzuki, T., Fukui, S., Yoneoka, D., Aoki, J., & Fujiwara, T. (2025). Blood Pressure After Changes in Light-to-Moderate Alcohol Consumption in Women and Men: Longitudinal Japanese Annual Checkup Analysis. *JACC*. https://doi.org/10.1016/j.jacc.2025.09.018

DOES ALCOHOL USE AND RELATED HARM DIFFER BASED ON THE AGE OF INITIATION TO ALCOHOL? RESULTS FROM A PROSPECTIVE COHORT STUDY

October 2025

Background and Aims: There is evidence to suggest that earlier initiation to alcohol increases the rate and severity of alcohol consumption. However, this often overlooks the fact that earlier initiation also means a longer drinking history. This paper estimated differences in patterns of alcohol use and harm across adolescence and early adulthood, allowing for the fact that the patterns over time may differ depending on the age at which initiation occurred.

Design: Prospective cohort study.

Setting: Australia.

Participants: The Australian Parental Supply of Alcohol Longitudinal Study (APSALS), a cohort of n = 1906 adolescents recruited in adolescence (mean age 12.9) from 107 Australian schools and followed up until adulthood (11 annual waves total, 2010–2021).

Measurements: We defined age of initiation as the age at which alcohol consumption was first reported in the study. Our outcomes were amount of alcohol consumed, monthly heavy episodic drinking, alcohol-related harm and self-reported symptoms of alcohol use disorder in the years following initiation.

Findings: Those who initiated at age 12 had a lower risk of consumption [risk ratio (RR) 0.01; 95% confidence interval (CI) = 0.01–0.02] in the year following initiation (age 13), compared with those who initiated at age 18. Similarly, those who initiated at age 15 had a lower risk of alcohol use disorder in the year after initiation (RR 0.66; 95% CI = 0.52–0.83), compared with those who initiated at age 18. However, at age 20, those who initiated at age 12 showed higher consumption (RR 1.57; 95% CI = 1.18–2.09), monthly heavy episodic drinking (RR 1.24; 95% CI = 1.02–1.51) and alcohol-related harms [incidence-rate ratio (IRR) 1.73; 95% CI = 1.21–2.46] than those who initiated at age 18. Similar results were seen for symptoms consistent with DSM-IV alcohol dependence (RR 1.20; 95% CI = 1.05–1.38), DSM-IV alcohol abuse (RR 1.54; 95% CI = 1.04–2.29) and DSM-5 alcohol use disorder (RR 1.36; 95% CI = 1.12–1.65). However, there was evidence of ageing out, with risk of heavy episodic drinking and alcohol-related harm peaking around age 20 and then declining, regardless of when initiation occurred.

Conclusions: Later initiation to alcohol appears to be associated with more rapid escalation in drinking and related harm, but lower 'peak' harm than earlier initiation. The findings support the current guidelines recommending adolescents avoid alcohol until adulthood, and reinforce the need for public health intervention targeting both children and parents.

Source: Clare, P. J., Yuen, W. S., Henderson, A., Kypri, K., Bruno, R., Slade, T., ... & Peacock, A. (2025). Does alcohol use and related harm differ based on the age of initiation to alcohol? Results from a prospective cohort study. *Addiction*. https://doi.org/10.1111/add.70183

ALCOHOL USE AND RISK OF DEMENTIA IN DIVERSE POPULATIONS: EVIDENCE FROM COHORT, CASE–CONTROL AND MENDELIAN RANDOMISATION APPROACHES

October 2025

Objectives: To investigate the relationship between alcohol consumption and dementia.

Design: Prospective cohort and case–control analyses combined with linear and non-linear Mendelian randomisation.

Setting: Two large-scale population-based cohorts: the US Million Veteran Programme and the UK Biobank. Genetic analyses used summary statistics from genome-wide association studies (GWAS).

Participants: 559 559 adults aged 56–72 years at baseline were included in observational analyses (mean follow-up: 4 years in the US cohort; 12 years in the UK cohort). Genetic analyses used summary data from multiple large GWAS consortia (2.4 million participants).

Main outcome measures: Incident all-cause dementia, determined through health record linkage, and genetic proxies.

Results: During follow-up, 14 540 participants developed dementia and 48 034 died. Observational phenotype-only analyses revealed U-shaped associations between alcohol and dementia risk: higher risk was observed among non-drinkers, heavy drinkers (>40 drinks per week; HR 1.41, 95% CI 1.15 to 1.74), and those with alcohol use disorder (AUD) (HR 1.51, 95% CI 1.42 to 1.60) compared with light drinkers. In contrast, Mendelian randomisation genetic analysis identified a monotonic increase in dementia risk with greater alcohol consumption. A 1 SD increase in log-transformed drinks per week was associated with a 15% dementia increase (inverse-variance weighted (IVW) OR 1.15, 95% CI 1.03 to 1.27). A twofold increase in AUD prevalence was associated with a 16% increase in dementia risk (IVW OR 1.16, 95% CI 1.03 to 1.30). Alcohol intake increased dementia, but individuals who developed dementia also experienced a decline in alcohol intake over time, suggesting reverse causation—where early cognitive decline leads to reduced alcohol consumption—underlies the supposed protective alcohol effects in observational studies.

Conclusions: These findings provide evidence for a relationship between all types of alcohol use and increased dementia risk. While correlational observational data suggested a protective effect of light drinking, this could be in part attributable to reduced drinking seen in early dementia; genetic analyses did not support any protective effect, suggesting that any level of alcohol consumption may contribute to dementia risk. Public health strategies that reduce the prevalence of alcohol use disorder could potentially lower the incidence of dementia by up to 16%.

Source: Topiwala, A., Levey, D. F., Zhou, H., Deak, J. D., Adhikari, K., Ebmeier, K. P., ... & Gelernter, J. (2025). Alcohol use and risk of dementia in diverse populations: evidence from cohort, case–control and Mendelian randomisation approaches. *BMJ Evidence-Based Medicine*. https://www.doi.org/10.1136/bmjebm-2025-113913

In the News: LaMotte, Sandee. (2025, September 25). There is no safe amount of alcohol when it comes to dementia, study finds. *CNN Health*. https://www.cnn.com/2025/09/23/health/alcohol-dementia-risk-study-wellness