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Original Article

Associations of Individual Beverage Types and Substitution with Dementia Risk: A UK Biobank Cohort Study

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ABSTRACT

Objectives: The role of beverage consumption in dementia prevention, particularly regarding substitution effects and interactions with modifiable risk factors, remains unclear. This study aimed to evaluate the associations of major beverage types and their substitution effects with the risk of all-cause dementia.
Design: A prospective cohort study.
Setting and participants: We included 118,963 dementia-free participants (2006–2010 baseline) with complete dietary questionnaires from the UK Biobank.
Measurements: Self-reported intake of sugar-sweetened beverages, artificially sweetened beverages, natural juices, coffee, and tea was assessed through 24-h dietary recall. The primary outcome was incident all-cause dementia, ascertained through linked primary care, hospital admission, and mortality registration data. Associations between beverage intake and dementia risk were evaluated using Cox proportional hazards models, yielding hazard ratios (HRs) and 95% confidence intervals (CIs). Substitution modeling assessed the effects of replacing one beverage with another. Interaction analyses explored variations by modifiable risk factors, including obesity, hypertension, depression, or dyslipidemia.
Results: Over 13.45 years, 992 all-cause dementia cases were recorded. Higher sugar-sweetened beverage intake (>1 glass/day) was associated with an increased risk of all-cause dementia (HR, 1.61; 95% CI, 1.28–2.02; $P < 0.001$). Coffee and tea consumption were associated with a lower risk of all-cause dementia. Substituting sugar-sweetened beverages or artificially sweetened beverages with coffee or tea significantly reduced the risk of all-cause dementia. These protective associations were strongest among individuals with obesity, hypertension, depression, or dyslipidemia.
Conclusion: Replacing sugar-sweetened beverages or artificially sweetened beverages with coffee or tea was associated with a reduced risk of dementia, particularly among individuals with modifiable risk factors. These findings support beverage substitution as a simple, targeted strategy for mitigating the risk of dementia.

1. Introduction

Dementia is a growing public health concern, characterized by a progressive decline in cognitive function that interferes with independent daily living [1,2]. The global prevalence of dementia is projected to increase substantially from 57 million cases in 2019 to 152 million by

Abbreviations: ACD, all-cause dementia; AD, Alzheimer's disease; APOE, apolipoprotein E; ASBs, artificially sweetened beverages; BMI, body mass index; CI, confidence interval; CVD, cardiovascular disease; HDL, high-density lipoprotein; HR, hazard ratio; ICD, International Classification of Diseases; LDL, low-density lipoprotein; NJ, natural juices; SSBs, sugar-sweetened beverages; TDI, Townsend deprivation index; VaD, vascular dementia.

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2050 [3]. Among all-cause dementia (ACD) cases, Alzheimer's disease (AD) is the most common subtype, accounting for approximately 60–70% of cases, followed by vascular dementia (VaD), which accounts for approximately 25% [4]. Emerging evidence from nutritional epidemiology highlights the potential of dietary interventions in mitigating dementia risk [5]. Among dietary factors, beverage consumption is a modifiable component that can influence metabolic processes, nutrient intake, and fluid balance [6,7]. Optimal beverage consumption has also been implicated in other significant aspects of healthy aging, such as a reduced risk of frailty [8]. However, limited research has focused on the association between beverage consumption and the development of dementia in large, population-based cohorts. The 2024 update of the *Lancet Commission on Dementia Prevention* emphasized the importance of lifestyle modification for dementia prevention, estimating that up to 45% of cases could be prevented by addressing factors such as hypertension, obesity, depression, and diabetes [9]. However, data on how specific beverage choices may contribute to dementia prevention remain scarce.

Using data from the UK Biobank, a large, prospective population-based cohort, this study aimed to examine the association between habitual beverage consumption, including sugar-sweetened beverages (SSBs), artificially sweetened beverages (ASBs), natural juices (NJs), coffee, and tea, and the risk of incident ACD. The study also assessed whether substituting one beverage type for another affects this risk and whether these associations differ by modifiable risk factors such as obesity, hypertension, dyslipidemia, and depression.

2. Methods

2.1. Study population and data collection

This study utilized data from the UK Biobank, a large, population-based prospective cohort study that recruited over 500,000 participants aged 40–69 years between 2006 and 2010 in England, Wales, and Scotland [10,11]. Detailed study protocols are available on the UK Biobank website (https://www.ukbiobank.ac.uk/). The recruitment process involved postal invitations sent to over 9 million individuals registered with the UK's National Health Service to attend one of the 22 centers [12]. Dietary assessments were conducted up to five times between April 2009 and June 2012 using the Oxford WebQ, a web-based, self-administered 24-h recall questionnaire. The Oxford WebQ presents participants with 21 broad food groups, further expanding to include over 200 commonly consumed foods and beverages. Ethical approval for the study was obtained from the North West Multi-Centre Research Ethics Committee, and participants provided written informed consent [13].

Among 502,369 UK Biobank participants assessed at baseline, the following were excluded: (1) 228 with dementia at baseline, (2) 291,249 without 24-h dietary recall data, and (3) 91,929 with missing baseline data. After exclusions, 210,892 participants who completed at least one dietary questionnaire and had no dementia at baseline were eligible for inclusion. Of these, 118,963 participants were included in the final analysis. Fig. 1 presents a flowchart illustrating participant selection.

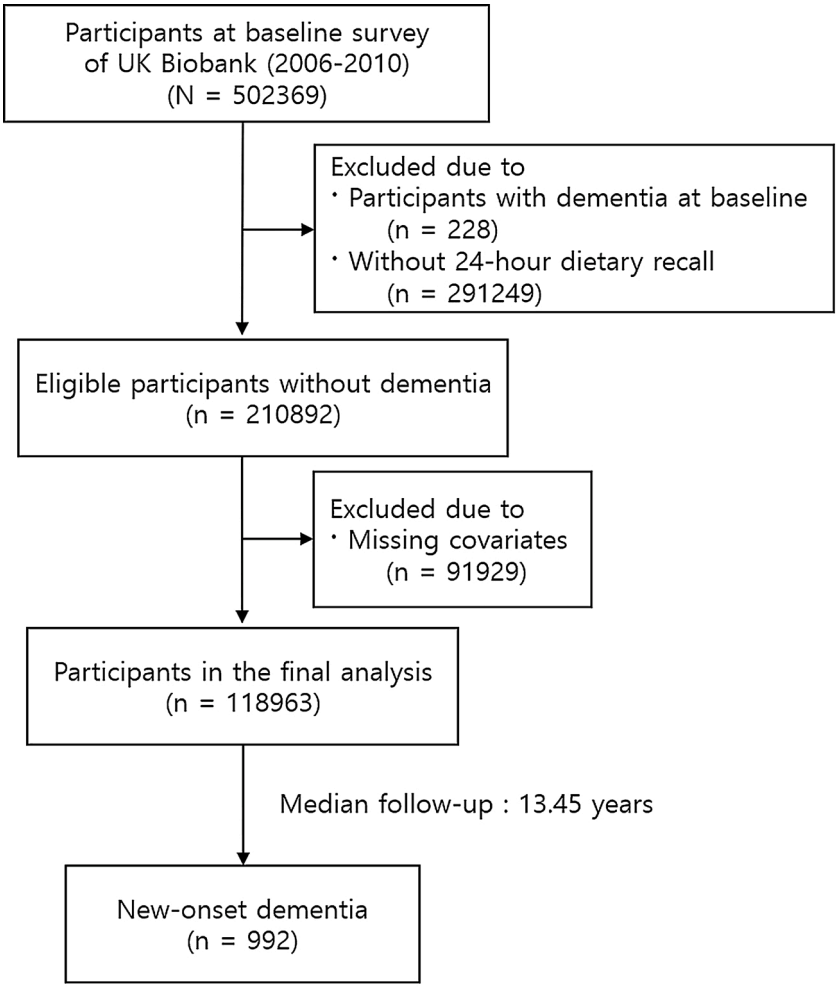

Fig. 1. Flowchart illustrating participant selection for the study population. The diagnosis of dementia was made using health-related outcomes defined by the UK Biobank preprocessing algorithm.

2.2. Exposure assessment

Beverage intake was classified into five main categories: SSBs, including fizzy drinks and squash; ASBs, including low-calorie soft drinks; NJs, including pure orange juice, grapefruit juice, and other pure fruit or vegetable juices; coffee; and tea, including black tea and green tea. To categorize beverage consumption levels, intake of each beverage type was classified into three groups: 0 servings per day, >0 and ≤1 serving per day, and >1 serving per day, as previously described [14]. Daily beverage intake was assessed using the question "How many glasses, cans, or cartons containing 250 mL of beverages did you drink yesterday?"

2.3. Outcome measures

The primary outcome was ACD. The two major subtypes, AD and VaD, were assessed as secondary outcomes. The diagnosis of dementia was based on primary care, hospital admission, and mortality registration data during the follow-up period [15,16]. The International Classification of Diseases 9th and 10th Revision coding systems were used to record dementia diagnoses in the UK Biobank [17] (Supplementary Table S1).

2.4. Covariates

Sociodemographic data (age, sex, ethnicity, Townsend deprivation index [TDI], household income, education level), lifestyle factors (smoking status, alcohol intake, physical activity), physical examination measurements (body mass index [BMI]), laboratory measurements (glucose, total cholesterol, low-density lipoprotein [LDL] cholesterol, high-density lipoprotein [HDL] cholesterol, triglyceride), comorbidities (hypertension, diabetes, cardiovascular disease [CVD]), apolipoprotein E (APOE) ε4 carriers, and total energy intake were accounted for to control confounding bias. The TDI was computed using national census data and assigned based on postal codes, considering factors such as unemployment, non-car ownership, non-home ownership, and household overcrowding, with a higher TDI indicating a greater degree of deprivation. Lifestyle factors were evaluated using a touchscreen questionnaire and included alcohol consumption, smoking status, and physical activity. Physical activity levels were assessed using metabolic equivalent (MET) scores calculated according to the validated guidelines of the International Physical Activity Questionnaire (IPAQ). BMI was calculated by dividing weight by height squared (kg/m^2). Obesity was defined as BMI ≥30 kg/m^2, whereas central obesity was classified based on waist circumference thresholds (≥88 cm for women and ≥102 cm for men). Comorbidities were defined as self-reported physician-diagnosed cases or medical records from the International Classification of Diseases 10th Revision (ICD-10) codes as follows: hypertension (I10–13 and I15), depression (F32–33), diabetes (E10–14), dyslipidemia (E78), CVD (I20–25, I60–64), and chronic kidney disease (CKD) (I12–13 and N18). APOE genotype was coded as APOE ε4 non-carriers (low genetic risk) and APOE ε4 carriers (high genetic risk).

2.5. Statistical analysis

Baseline characteristics are presented as numbers with percentages for categorical variables and as means with standard deviations for continuous variables. Cox proportional hazards models were used to estimate the association between beverage intake and dementia. Hazard ratios (HRs) and corresponding 95% confidence intervals (CIs) for dementia were calculated. Four multivariable-adjusted models were employed. Model 1 was adjusted for age and sex. Model 2 was adjusted for sociodemographic characteristics (age, sex, ethnicity, TDI, household income, education level) and lifestyle factors (smoking status, alcohol intake, physical activity). Model 3 was further adjusted for variables in Model 2, and additional adjustments for BMI, glucose, total cholesterol, LDL-cholesterol, HDL-cholesterol, triglyceride, and comorbidities. Model 4 was adjusted for the variables in Model 3 and APOE ε4 carrier status. Kaplan–Meier survival curves were used to assess the cumulative incidence of dementia according to beverage intake. To minimize potential bias arising from baseline age differences between participants who developed dementia and those who did not, we conducted a sensitivity analysis using age- and sex-exact matched data. Substitution analysis assumes that a reduction in one food or nutrient is accompanied by an increase in another, with total energy intake held approximately constant [18,19]. Within this framework, we estimated the difference in dementia risk associated with a one-serving/day higher intake of one beverage type and a corresponding one-serving/day lower intake of another beverage type, in the same model. To examine additive interactions between modifiable risk factors and beverage intake, p-values for interaction and 95% CIs were calculated, using the stratum without risk factors as the reference category. A two-sided P-value < 0.05 was considered statistically significant. All analyses were conducted using R version 4.3.0 (R Foundation for Statistical Computing, Vienna, Austria)

3. Results

3.1. Baseline characteristics of the study population

Table 1 presents the baseline characteristics according to ACD incidence. Among the 118,963 participants, 992 (0.83%) developed ACD over a median follow-up period of 13.45 years. Participants who developed ACD were generally older, more likely to be male, and from lower-income households. They also exhibited a higher prevalence of comorbidities, along with higher glucose levels and lower total cholesterol, LDL cholesterol, and HDL cholesterol levels. The proportion of APOE ε4 carriers was significantly higher among those who developed ACD. Differences in beverage consumption intake were observed, with the group that developed dementia showing a higher proportion of individuals consuming >1 glass/day of NJs and lower consumption of coffee and tea.

3.2. Association between beverage consumption and dementia risk

Table 2 presents the results of the Cox proportional hazards regression analysis examining the association between beverage intake and ACD. SSB consumption was significantly associated with an increased risk of dementia. Compared with non-consumers, individuals who consumed >1 glass/day had a 61% higher risk of dementia in the fully adjusted model (HR, 1.61; 95% CI, 1.28–2.02; $P < 0.001$), whereas those consuming ≤1 glass/day showed no significant association. For ASBs, higher consumption was significantly associated with an increased risk of dementia in Model 1 (HR, 1.43; 95% CI, 1.08–1.91; $P = 0.014$). However, the significant association was attenuated in the fully adjusted model (HR, 1.27; 95% CI, 0.95–1.69; $P = 0.116$). NJ consumption did not show a clear association with the risk of dementia. Although moderate intake (>0–1 glass/day) was associated with a lower risk in Model 1 (HR, 0.81; 95% CI, 0.71–0.92; $P = 0.001$), this association weakened in the fully adjusted model (HR, 0.89; 95% CI, 0.78–1.02; $P = 0.095$). Coffee and tea consumption were both associated with a lower risk of dementia. Compared with non-consumers, individuals consuming >1 glass/day of coffee had a 24% lower risk of dementia (HR, 0.76; 95% CI, 0.63–0.92; $P = 0.004$), whereas those consuming >0–1 glass/day had a 37% lower risk (HR, 0.63; 95% CI, 0.52–0.76; $P < 0.001$). Similarly, tea consumption was inversely associated with dementia risk, with >1 glass/day associated with a 26% lower risk (HR, 0.74; 95% CI, 0.63–0.87; $P < 0.001$) and >0–1 glass/day with a 34% lower risk (HR, 0.66; 95% CI, 0.52–0.83; $P < 0.001$). The cumulative incidence of ACD according to beverage intake is provided in Supplementary Fig. S1. Associations between beverage intake and dementia subtypes, including AD and VaD, are presented in Supplementary Tables S2 and S3. Higher

Table 1
Baseline characteristics of the study population according to the all-cause dementia incidence.

Characteristics[1]	Overall (n = 118963)	Non incident all-cause dementia (n = 117971)	Incident all-cause dementia (n = 992)	*P*-value
Age (years)	55.75 ± 7.98	55.69 ± 7.97	63.61 ± 5.01	<0.001
Sex, n (%)				<0.001
Male	57722 (48.5%)	57106 (48.4%)	616 (62.1%)	
Female	61241 (51.5%)	60865 (51.6%)	376 (37.9%)	
Ethnicity, n (%)				0.031
Non-white	4623 (3.9%)	4598 (3.9%)	25 (2.5%)	
White	114340 (96.1%)	113373 (96.1%)	967 (97.5%)	
Education, n (%)[2]				<0.001
Level 1	8317 (7.0%)	8162 (6.9%)	155 (15.6%)	
Level 2	28711 (24.1%)	28473 (24.1%)	238 (24.0%)	
Level 3	27991 (23.5%)	27748 (23.5%)	243 (24.5%)	
Level 4	53944 (45.3%)	53588 (45.4%)	356 (35.9%)	
Household income (£/year)				<0.001
<18000	17609 (14.8%)	17312 (14.7%)	297 (29.9%)	
18,000-30,999	28160 (23.7%)	27827 (23.6%)	333 (33.6%)	
31,000−51,999	33987 (28.6%)	33768 (28.6%)	219 (22.1%)	
52,000−100,000	30053 (25.3%)	29939 (25.4%)	114 (11.5%)	
>100,000	9154 (7.7%)	9125 (7.7%)	29 (2.9%)	
TDI, mean ± SD	−1.60 ± 2.85	−1.60 ± 2.85	−1.49 ± 2.94	0.243
Alcohol intake, n (%)				<0.001
Never	66411 (55.8%)	65957 (55.9%)	454 (45.8%)	
Previous	42990 (36.1%)	42535 (36.1%)	455 (45.9%)	
Current	9562 (8.0%)	9479 (8.0%)	83 (8.4%)	
Smoking status, n (%)				<0.001
Never	3434 (2.9%)	3390 (2.9%)	44 (4.4%)	
Previous	3504 (2.9%)	3454 (2.9%)	50 (5.0%)	
Current	112025 (94.2%)	111127 (94.2%)	898 (90.5%)	
Physical activity, n (%)				0.921
High	21837 (18.4%)	21659 (18.4%)	178 (17.9%)	
Moderate	50568 (42.5%)	50141 (42.5%)	427 (43.0%)	
Low	46558 (39.1%)	46171 (39.1%)	387 (39.0%)	
BMI, mean ± SD	26.89 ± 4.55	26.88 ± 4.54	27.28 ± 4.70	0.009
Central obesity[3]	34730 (29.2%)	34369 (29.1%)	361 (36.4%)	<0.001
SBP, mmHg	138.48 ± 19.21	138.42 ± 19.20	145.60 ± 19.79	<0.001
DBP, mmHg	81.83 ± 10.58	81.83 ± 10.58	81.83 ± 10.44	0.986
Glucose, mmol/L	5.09 ± 1.10	5.09 ± 1.10	5.29 ± 1.40	<0.001
Total cholesterol, mmol/L	5.69 ± 1.12	5.69 ± 1.11	5.52 ± 1.24	<0.001
Triglyceride, mmol/L	1.69 ± 0.99	1.69 ± 0.99	1.73 ± 1.02	0.171
LDL-cholesterol, mmol/L	3.55 ± 0.85	3.55 ± 0.85	3.43 ± 0.92	<0.001
HDL-cholesterol, mmol/L	1.47 ± 0.38	1.47 ± 0.38	1.43 ± 0.39	<0.001
Comorbidities, n (%)				
Hypertension	28307 (23.8%)	27907 (23.7%)	400 (40.3%)	<0.001
Diabetes	4743 (4.0%)	4,650 (3.9%)	93 (9.4%)	<0.001
Dyslipidemia	15936 (13.4%)	15,658 (13.3%)	278 (28.0%)	<0.001
Cardiovascular disease	8533 (7.2%)	8,332 (7.1%)	201 (20.3%)	<0.001
Chronic kidney disease	246 (0.2%)	236 (0.2%)	10 (1.0%)	<0.001
APOE ε4 carriers	33467 (28.1%)	32964 (27.9%)	503 (50.7%)	<0.001
Sugar-sweetened beverages, n (%)				0.053
0 glass/day	80132 (67.4%)	79464 (67.4%)	668 (67.3%)	
>0-1 glass/day	30864 (25.9%)	30624 (26.0%)	240 (24.2%)	
>1 glass/day	7967 (6.7%)	7883 (6.7%)	84 (8.5%)	
Artificially sweetened beverages, n (%)				0.700
0 glass/day	94255 (79.2%)	93459 (79.2%)	796 (80.2%)	
>0-1 glass/day	18165 (15.3%)	18019 (15.3%)	146 (14.7%)	
>1 glass/day	6543 (5.5%)	6493 (5.5%)	50 (5.0%)	
Natural juices, n (%)				0.035
0 glass/day	56800 (47.7%)	56308 (47.7%)	492 (49.6%)	
>0-1 glass/day	53367 (44.9%)	52956 (44.9%)	411 (41.4%)	
>1 glass/day	8796 (7.4%)	8707 (7.4%)	89 (9.0%)	
Coffee, n (%)				<0.001
0 glass/day	80684 (67.8%)	79942 (67.8%)	742 (74.8%)	
>0-1 glass/day	21042 (17.7%)	20917 (17.7%)	125 (12.6%)	
>1 glass/day	17237 (14.5%)	17112 (14.5%)	125 (12.6%)	
Tea, n (%)				<0.001
0 glass/day	78087 (65.6%)	77369 (65.6%)	718 (72.4%)	
>0-1 glass/day	13759 (11.6%)	13678 (11.6%)	81 (8.2%)	
>1 glass/day	27117 (22.8%)	26924 (22.8%)	193 (19.5%)	

Abbreviations: TDI, Townsend deprivation index; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; LDL, low-density lipoprotein; HDL, high-density lipoprotein; APOE, apolipoprotein E.

[1] Data are expressed as mean ± standard deviation for continuous variables and number (percentage) for categorical variables.

[2] Level 1, "uneducated"; level 2, "O levels/GCSEs or equivalent" or "CSEs or equivalent"; level 3, "A levels/AS levels or equivalent" or "NVQ or HND or HNC or equivalent" or "Other professional qualifications"; level 4, "College or University degree.

[3] Central obesity was classified based on waist circumference thresholds (≥88 cm for women and ≥102 cm for men.

Table 2
Cox proportional hazard regression analysis for all-cause dementia according to beverage intake.

Beverage intake	Model 1[1]		Model 2[2]		Model 3[3]		Model 4[4]	
	HR (95% CI)	*P*-value	HR (95% CI)	*P*-value	HR (95% CI)	*P*-value	HR (95% CI)	*P*-value
Sugar-sweetened beverages								
0 glass/day	reference		reference		reference		reference	
>0-1 glass/day	0.99 (0.85–1.15)	0.882	0.99 (0.85–1.14)	0.843	0.99 (0.86–1.15)	0.916	1.00 (0.86–1.16)	0.981
>1 glass/day	1.71 (1.36–2.15)	<0.001	1.58 (1.25–1.98)	<0.001	1.59 (1.26–2.00)	<0.001	1.61 (1.28–2.02)	<0.001
Artificially sweetened beverages								
0 glass/day	reference		reference		reference		reference	
>0-1 glass/day	1.21 (1.01–1.44)	0.034	1.19 (1.00–1.42)	0.051	1.17 (0.98–1.40)	0.089	1.18 (0.98–1.41)	0.074
>1 glass/day	1.43 (1.08–1.91)	0.014	1.34 (1.01–1.79)	0.044	1.30 (0.97–1.74)	0.081	1.27 (0.95–1.69)	0.116
Natural juices								
0 glass/day	reference		reference		reference		reference	
>0-1 glass/day	0.81 (0.71–0.92)	0.001	0.87 (0.76–1.00)	0.045	0.89 (0.78–1.02)	0.082	0.89 (0.78–1.02)	0.095
>1 glass/day	1.13 (0.90–1.41)	0.298	1.22 (0.97–1.53)	0.091	1.24 (0.99–1.56)	0.064	1.23 (0.98–1.55)	0.072
Coffee								
0 glass/day	reference		reference		reference		reference	
>0-1 glass/day	0.60 (0.50–0.72)	<0.001	0.62 (0.51–0.75)	<0.001	0.62 (0.51–0.75)	<0.001	0.63 (0.52–0.76)	<0.001
>1 glass/day	0.76 (0.63–0.92)	0.006	0.76 (0.62–0.91)	0.004	0.75 (0.62–0.91)	0.004	0.76 (0.63–0.92)	0.004
Tea								
0 glass/day	reference		reference		reference		reference	
>0-1 glass/day	0.62 (0.49–0.78)	<0.001	0.65 (0.52–0.82)	<0.001	0.66 (0.52–0.83)	<0.001	0.66 (0.52–0.83)	<0.001
>1 glass/day	0.76 (0.65–0.89)	<0.001	0.74 (0.63–0.86)	<0.001	0.74 (0.63–0.87)	<0.001	0.74 (0.63–0.87)	<0.001

Abbreviations: HR, hazard ratio; CI, confidence interval; TDI, Townsend deprivation index; BMI, body mass index; LDL, low-density lipoprotein; HDL, high-density lipoprotein; CVD, cardiovascular disease; APOE, apolipoprotein E.

[1] Adjusted for age and sex;

[2] Adjusted for age, sex, ethnicity, TDI, household income, education level, smoking status, alcohol intake, and physical activity;

[3] Adjusted for age, sex, ethnicity, TDI, household income, education level, smoking status, alcohol intake, physical activity, BMI, glucose, total cholesterol, LDL-cholesterol, HDL-cholesterol, triglyceride, hypertension, diabetes, and CVD;

[4] Adjusted for age, sex, ethnicity, TDI, household income, education level, smoking status, alcohol intake, physical activity, BMI, glucose, total cholesterol, LDL-cholesterol, HDL-cholesterol, triglyceride, hypertension, diabetes, CVD, and APOE ε4 carriers.

SSB consumption was significantly associated with an increased risk of AD. In contrast, higher coffee and tea consumption were associated with a lower risk of AD. ASB consumption was strongly associated with an increased risk of VaD; higher coffee intake was associated with a lower risk of VaD; and tea consumption showed no significant association with VaD risk. Additionally, the cumulative incidence of AD and VaD according to beverage intake is provided in Supplementary Figs. S2 and S3.

3.3. Sensitivity analysis

To account for potential baseline age imbalances, we performed a sensitivity analysis based on age- and sex-exact matched data (Supplementary Table S4). The findings from this rigorously matched and covariate-adjusted cohort were consistent with our main results, indicating that baseline age differences did not materially affect the observed associations between beverage intake and dementia risk.

3.4. Association between substituting 1 glass/day of a specific beverage type and dementia risk

In the substitution analyses, replacing 1 glass/day of SSB, ASB, or NJ with coffee was associated with a lower risk of incident ACD (HR [95% CI]: 0.77 [0.69–0.86], 0.85 [0.75–0.95], and 0.85 [0.74–0.96], respectively). Similarly, replacing 1 glass/day of SSB, ASB, or NJ with tea was associated with a lower risk of incident ACD (HR [95% CI]: 0.81 [0.73–0.90], 0.89 [0.80–0.99], and 0.89 [0.79–1.00], respectively) (Table 3). Replacing 1 glass/day of SSB with other beverage types was associated with a lower risk of AD, and replacing 1 glass/day of ASB with

Table 3
Substitution analysis examining the association between risk of all-case dementia and beverage intake.

All-cause dementia										
Substitution analysis[1]	Sugar-sweetened beverages		Artificially sweetened beverages		Natural juices		Tea		Coffee	
	HR (95% CI)	*P*-value	HR (95% CI)	*P*-value	HR (95% CI)	*P*-value	HR (95% CI)	*P*-value	HR (95% CI)	*P*-value
With sugar-sweetened beverages	–	–	1.11 (0.97–1.26)	0.134	1.10 (0.96–1.27)	0.174	1.23 (1.12–1.36)	<0.001	1.30 (1.16–1.45)	<0.001
With artificially sweetened beverages	0.90 (0.79–1.03)	0.134	–	–	1.00 (0.87–1.16)	0.995	1.12 (1.01–1.24)	0.032	1.18 (1.05–1.33)	0.006
With natural juices	0.91 (0.79–1.04)	0.174	1.00 (0.87–1.15)	0.995	–	–	1.12 (1.00–1.26)	0.048	1.18 (1.04–1.34)	0.011
With tea	0.81 (0.73–0.90)	<0.001	0.89 (0.80–0.99)	0.032	0.89 (0.79–1.00)	0.048	–	–	1.05 (0.96–1.15)	0.266
With coffee	0.77 (0.69–0.86)	<0.001	0.85 (0.75–0.95)	0.006	0.85 (0.74–0.96)	0.011	0.95 (0.87–1.04)	0.266	–	–

Abbreviations: HR, hazard ratio; CI, confidence interval; TDI, Townsend deprivation index; BMI, body mass index; LDL, low-density lipoprotein; HDL, high-density lipoprotein; CVD, cardiovascular disease; APOE, apolipoprotein E.

[1] Adjusted for age, sex, ethnicity, TDI, household income, education level, smoking status, alcohol intake, physical activity, BMI, glucose, total cholesterol, LDL-cholesterol, HDL-cholesterol, triglyceride, hypertension, diabetes, CVD, APOE ε4 carriers, and total energy intake.

coffee or tea was associated with a lower risk of VaD (Supplementary Tables S5 and S6).

3.5. Subgroup analyses of beverage consumption and dementia risk

Fig. 2 presents forest plots from subgroup analyses examining the associations between beverage consumption intake and the risk of ACD, stratified by key modifiable risk factors, including hypertension, obesity, depression, and dyslipidemia.

A significant interaction was observed between depression and SSB intake (*P*-interaction = 0.030). Among participants with depression, even moderate SSB intake (>0–1 glass/day) was associated with a 76% increased risk of ACD (HR, 1.76; 95% CI, 1.11–2.79). Higher SSB intake (>1 glass/day) was also associated with an elevated risk of ACD in this group (HR, 1.46; 95% CI, 0.69–3.10), although the association was not statistically significant. Among individuals without depression, high SSB consumption (>1 glass/day) was associated with a 62% increased risk of ACD (HR, 1.62; 95% CI, 1.28–2.06). A significant interaction was also found between dyslipidemia and ASBs (*P*-interaction = 0.047). Among individuals with dyslipidemia, ASB intake was associated with an increased risk of ACD, with HRs of 1.50 (95% CI, 1.12–2.03) for >0–1 glass/day and 1.80 (95% CI, 1.13–2.85) for >1 glass/day.

Coffee consumption was significantly associated with a reduced risk of ACD, particularly among individuals with hypertension and obesity. The protective effect was more pronounced in those with obesity: HRs were 0.38 (95% CI, 0.23–0.61) for >0–1 glass/day and 0.51 (95% CI, 0.34–0.77) for >1 glass/day (*P*-interaction = 0.009). In participants with hypertension, the corresponding HRs were 0.53 (95% CI, 0.42–0.67) and 0.69 (95% CI, 0.55–0.86) (*P*-interaction = 0.012).

Similarly, tea consumption showed a protective association in participants with hypertension and obesity. Among those with hypertension, tea intake was associated with a reduced risk of ACD: HRs were 0.67 (95% CI, 0.51–0.87) for >0–1 glass/day and 0.66 (95% CI, 0.54–0.79) for >1 glass/day (*P*-interaction = 0.031). In individuals with obesity, the protective association was also significant: HRs were 0.56 (95% CI, 0.33–0.93) for >0–1 glass/day and 0.48 (95% CI, 0.33–0.69) for >1 glass/day (*P*-interaction = 0.020).

Supplementary Fig. S4 displays subgroup analyses stratified by diabetes and chronic kidney disease, showing no significant interactions between these conditions and beverage consumption in relation to ACD risk. Supplementary Tables S7 and S8 present subgroup analyses for the associations between beverage intake and the risks of AD and VaD, respectively, stratified by modifiable risk factors.

4. Discussion

This large prospective cohort study revealed that habitual beverage consumption was significantly associated with the risk of developing ACD. Specifically, daily intake of more than one glass of SSBs was associated with a substantially increased dementia risk, whereas both coffee and tea consumption, even at moderate levels, were consistently linked to reduced risk. These inverse associations remained robust after adjusting for multiple variables. Substitution analyses further revealed that replacing one daily serving of SSBs, ASBs, or NJs with coffee or tea was associated with a significantly lower dementia risk. Notably, the associations between beverage intake and dementia risk were stronger in individuals with modifiable risk factors, particularly those with obesity, hypertension, dyslipidemia, or depression, suggesting possible synergistic interactions. These findings highlight the value of tailoring dietary recommendations to high-risk populations.

Prior research on beverage consumption and dementia prevention has yielded inconsistent findings [20–22]. Some cohort studies, including the Framingham Offspring [23] and Rotterdam studies [24], reported weak or inconclusive associations between ASBs and coffee

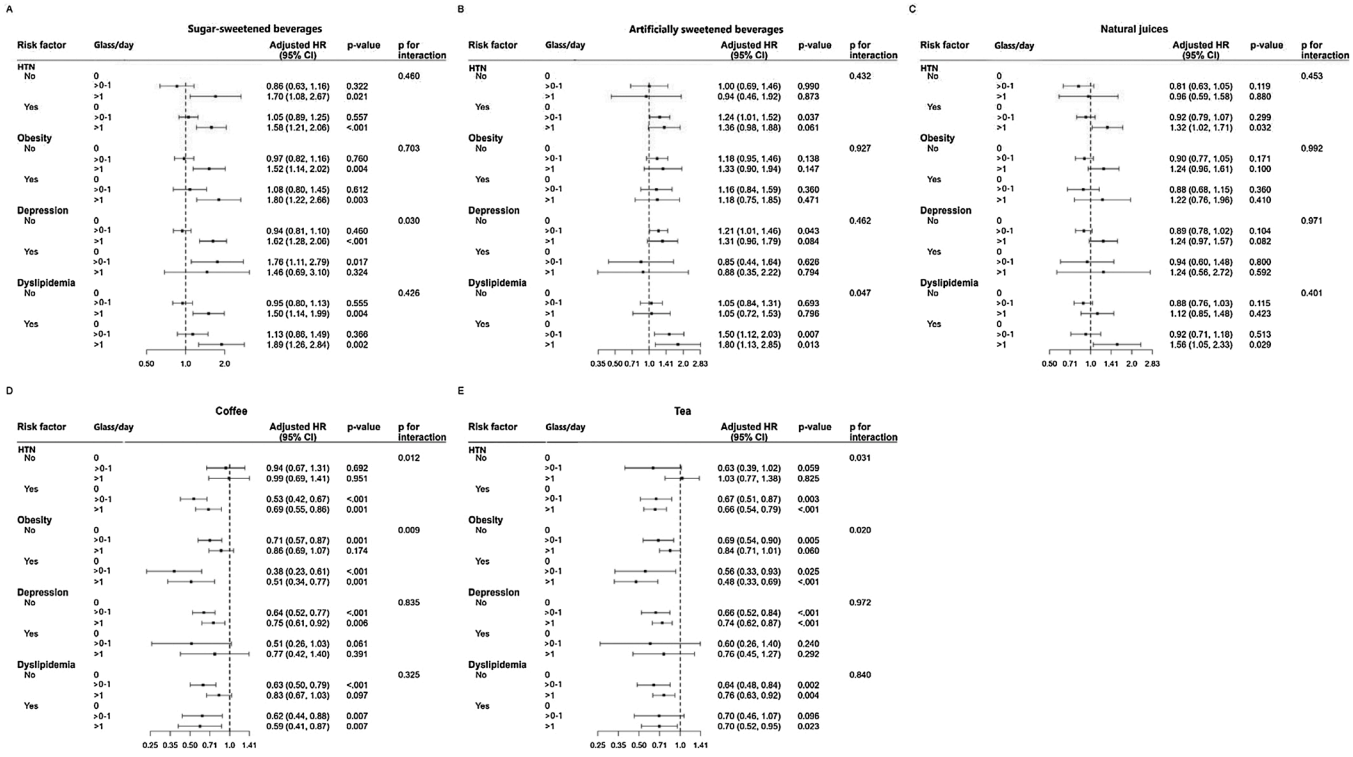

Fig. 2. Forest plots showing interactions between significant modifiable risk factors and beverage intake for all-cause dementia. A: Sugar-sweetened beverages (SSBs), B: Artificially sweetened beverages (ASBs), C: Natural juices (NJs), D: Coffee, E: Tea. Obesity was defined as BMI ≥ 30 kg/m^2. Subgroup analyses were adjusted for age, sex, ethnicity, Townsend deprivation index, household income, education level, smoking status, alcohol intake, physical activity, body mass index, glucose, total cholesterol, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, triglyceride, comorbidities (hypertension, diabetes, and cardiovascular disease), and APOE ε4 carrier status. HR, hazard ratio; CI, confidence interval; APOE, apolipoprotein E.

consumption. Although recent UK Biobank data suggest that moderate coffee or tea intake may reduce the risk of dementia [25]—a finding consistent with our results—meta-analyses indicate that the overall certainty of the evidence is low, and causality cannot be established [26]. These inconsistencies may reflect differences in study design, exposure measurement, and participant characteristics. Notably, prior studies rarely examined substitution effects between beverage types. This study contributes to the literature by evaluating substitution models and identifying stronger associations in individuals with modifiable risk factors, using a large, well-characterized cohort with long-term follow-up and stratified interaction analyses.

The biological mechanisms underlying these associations are multifactorial. Excessive intake of added sugars from SSBs has been linked to hyperglycemia, insulin resistance, oxidative stress, and neuroinflammation, all of which are implicated in cognitive decline and dementia [27,28]. Notably, in our subgroup analyses, even moderate SSB intake was associated with an increased risk of dementia among participants with depression [29]. This finding aligns with evidence suggesting that SSB consumption stimulates the hypothalamic-pituitary-adrenal axis and increases cortisol secretion, which can exacerbate depressive symptoms and impair cognitive resilience [30]. Furthermore, SSBs have been shown to disrupt hippocampal neurogenesis and promote amyloid-β accumulation in animal models, mechanisms relevant to AD pathology [31].

While ASBs are widely marketed as healthier alternatives [32], emerging research suggests potential adverse metabolic effects. In the present study, ASB intake was associated with an increased risk of dementia in individuals with dyslipidemia, potentially reflecting interactions between sweetener-induced lipid dysregulation and cognitive decline. Consistent with this finding, data from the NHANES study reported a link between high artificial sweetener consumption and an increased risk of hypercholesterolemia [33]. Similarly, a 2022 World Health Organization report noted worsened lipid profiles, including unfavorable total-to-HDL cholesterol ratios [34]. Moreover, the NutriNet-Santé cohort study found that aspartame intake was associated with increased risk of cerebrovascular disease [35]. These findings align with mechanistic studies showing that artificial sweeteners, such as aspartame and sucralose, may alter gut microbiota, impair lipid metabolism, and induce oxidative stress [36,37]. Additionally, phenylalanine in aspartame may disrupt monoamine neurotransmitter synthesis and increase neuronal excitability, potentially contributing to neurodegeneration [38].

NJs, often considered nutrient-rich, contain flavonoids, vitamins, and antioxidants that may support cognitive health [39,40]. However, their relatively high fructose content, especially when consumed in large quantities, may counteract potential benefits [41]. Our findings suggest a non-linear relationship: moderate NJ intake showed a weak protective association, whereas higher intake tended to be associated with increased risk. This pattern highlights the need for portion control and clear differentiation between 100% juice and variants with added sugar in future research.

Coffee and tea were consistently associated with a lower risk of dementia in all models, with strong protective effects observed in participants with hypertension and obesity. These beverages are rich in bioactive compounds, including caffeine, chlorogenic acid, and catechins, which possess antioxidant, anti-inflammatory, and neuroprotective properties [42–44]. Caffeine has been shown to increase brain-derived neurotrophic factor levels [45] and improve vascular function [46]. At the same time, polyphenols may reduce insulin resistance and systemic inflammation, pathways relevant to both cardiometabolic and cognitive health [47]. Prior studies have linked coffee and tea intake to a reduced incidence of hypertension, obesity, and depression, which are key dementia risk factors [48–50]. Our findings suggest that coffee and tea may offer dual benefits, particularly in individuals with these modifiable conditions, by targeting both vascular and neurodegenerative processes.

Beyond these cognitive benefits, the potential implications of coffee and tea consumption may extend to broader aspects of healthy aging. Although our study did not directly examine frailty, the observed protective associations of coffee and tea intake may suggest broader implications for healthy aging beyond dementia risk. Previous studies have reported that higher coffee, tea, and caffeine consumption is associated with a reduced risk of physical frailty in later life [51]. For instance, a large cohort study among older American adults demonstrated that regular coffee intake was inversely associated with frailty [52]. Therefore, these findings collectively indicate that coffee and tea consumption may contribute to both cognitive and physical resilience in aging populations. Future research should further explore how specific beverage choices concurrently influence cognitive decline and physical frailty, fostering a more comprehensive understanding of healthy longevity.

This study has several strengths. Using a large, prospective cohort with detailed baseline and follow-up data, we conducted robust multivariable adjustments and comprehensive stratified analyses. The long follow-up period (median 13.45 years) reduces the risk of reverse causation. Notably, this study incorporated substitution modeling to estimate the effects of replacing one beverage with another, a method rarely employed in prior research. Additionally, we conducted interaction analyses to assess whether beverage-related associations vary among individuals with different modifiable dementia risk factors, providing insights into population-specific vulnerabilities. These methodological strengths underpin practical implications for both individualized dietary counseling and broader dementia prevention strategies.

Nonetheless, some limitations should be acknowledged. First, although this observational study identified statistically significant associations, it cannot be conclusively stated that beverage substitution directly reduces dementia risk. Future well-designed randomized controlled trials and mechanistic studies are warranted to confirm these associations and clarify the underlying biological mechanisms. Second, beverage intake was self-reported, which may have introduced recall bias. Third, dementia diagnoses were based on clinical records, potentially underestimating the true incidence by missing undiagnosed or mild cases. Fourth, the UK Biobank cohort is not fully representative of the general population, as participants tend to be healthier and better educated, limiting the generalizability of our findings. Fifth, although water intake was not a primary focus of this study, additional analyses and covariate adjustments showed no significant association between water intake and dementia risk, and its inclusion did not materially alter the main findings. In addition, the substitution model used in this study, although designed to compare beverage types under energy-adjusted conditions, does not ensure a strictly isocaloric replacement. Therefore, the observed associations may partly reflect differences in energy intake or total beverage volume rather than beverage type alone. This limitation has been acknowledged, and future studies should employ more rigorously isocaloric-controlled substitution models to confirm these findings. Sixth, beverage intake was categorized into broad groups (0, >0 to ≤1, and >1 serving/day), which may limit dose–response precision because serving sizes vary across beverage types. Moreover, grouping sugar-sweetened and artificially sweetened beverages without differentiating subtypes (e.g., colas vs. energy drinks, or diet soda vs. stevia-sweetened tea) may have obscured potential differences in health effects. Nevertheless, this categorical approach has been widely applied in large-scale epidemiological studies and remains practical for public health interpretation. Finally, despite extensive adjustment for covariates, residual confounding cannot be ruled out entirely. Our findings provide valuable evidence in this field; however, they should be interpreted with caution and further validated across more diverse populations and study designs.

5. Conclusion

In this large prospective cohort study, replacing SSBs or ASBs with coffee or tea was associated with a lower risk of dementia, particularly

among individuals with modifiable risk factors such as obesity, hypertension, depression, and dyslipidemia. These findings highlight the potential of targeted beverage substitution as a simple and practical strategy for mitigating the risk of dementia. Given the global rise in prevalence of dementia and the modifiable nature of beverage habits, this strategy may have meaningful public health implications. Further research is warranted to confirm these associations and elucidate the underlying mechanisms.

CRediT authorship contribution statement

JHK, YJK, and JWL contributed to the conception and design of the study. YL and SJH performed the statistical analysis. JHK wrote the first draft of the manuscript. YJK, YL, THH, MYL, SJH, and JWL critically reviewed and improved the drafts of the manuscript. The authors read and approved the final manuscript.

Consent for publication

Not applicable.

Ethics approval and consent to participate

All participants provided informed consent, and ethical approval was obtained from the National Health Service North West Centre for Research Ethics Committee (Ref: 11/NW/0382).

Declaration of Generative AI and AI-assisted technologies in the writing process

This paper was written without the use of AI or AI assistive technology.

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Data availability

Researchers can register and apply for data access at https://www.ukbiobank.ac.uk/.

Declaration of competing interest

The authors declare no competing interests.

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Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.jnha.2025.100740.

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