Ultraprocessed Foods and the Risk of Cognitive Impairment and Dementia in Older US Adults: 2013–2020 Health and Retirement Study

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Objectives. To examine associations between ultraprocessed food (UPF) consumption and risks of dementia, cognitive impairment with no dementia (CIND), and a composite outcome of CIND or dementia in a nationally representative longitudinal study of older US adults.

Methods. Participants were from the Health and Retirement Study (HRS 2013–2020; n = 5370). We assessed UPF intake via a semiquantitative food frequency questionnaire from the 2013 HRS Health Care and Nutrition Study. We defined cognitive impairment outcomes based on the validated Langa-Weir classification from biennial cognitive assessments (2014–2020). We used weighted Cox regression to examine associations.

Results. Compared with quintile 1, quintile 5 of UPF intake was associated with higher risks of dementia (hazard ratio [HR] = 1.58; 95% confidence interval [CI] = 1.003, 2.48; *P* for trend = .50), CIND (HR = 1.46; 95% CI = 1.13, 1.88; *P* for trend = .03), and CIND or dementia (HR = 1.47; 95% CI = 1.16, 1.87; *P* for trend = .02). Unprocessed or minimally processed food consumption was associated with lower risks of these outcomes.

Conclusions. Increased UPF consumption is associated with higher risks of dementia, CIND, and CIND or dementia among US older adults.

Public Health Implications. Our findings suggest a potential need to reduce UPF consumption for maintaining cognitive health among older adults. (*Am J Public Health*. 2026;116(7):981–992. https://doi.org/10.2105/AJPH.2026.308505)

Ultraprocessed food (UPF) is industrially produced food and beverages to which chemical additives or chemically modified ingredients are added to enhance palatability, extend shelf life, or reduce cost.[1] Characteristics of UPFs, which are often high in saturated fat, sodium, sugar, and energy density and low in dietary fiber, include foods such as sugar-sweetened beverages, breakfast cereals, pizza, and other shelf-stable or ready-to-eat foods.[1,2] Given their widespread availability and convenience, UPF consumption contributes more than 50% of energy intake among US adults, with a concerning increase over time among older adults.[3,4]

Higher UPF consumption has been associated with a range of adverse health outcomes, including obesity,[5,6] diabetes,[7] cardiovascular disease,[8] and a higher risk of mortality.[9] Of particular relevance to healthy cognitive aging, a recent prospective cohort study of Brazilian adults found that UPF consumption was associated with a faster rate of decline in global cognition and executive function.[10] Similarly, higher UPF consumption was potentially associated with executive function impairment among US middle-aged and older adults.[11]

A recent meta-analysis showed that high UPF consumption was associated with an increased risk of all-cause dementia, whereas no association was observed with mild cognitive impairment.[12] Despite accumulating evidence on the link between UPF consumption and cognitive health, evidence from nationally representative studies among US older adults remains limited.[12,13] One US-based longitudinal study reported no significant association between UPFs and a composite outcome of cognitive impairment, combining cognitive impairment with no dementia (CIND) and dementia.[14] However, the previous study did not quantify UPF intake using energy-standardized measures, which may better reflect the overall dietary contribution of UPFs.[15] Evidence regarding the association between UPF consumption and individual cognitive outcomes, including dementia and CIND, remains limited. Diets high in UPFs often reflect poor diet quality, which may contribute to cognitive decline, underscoring the need for further investigation.

Thus, our primary objective was to examine the associations between UPF consumption and the risk of dementia, CIND, and a composite outcome of CIND or dementia using the longitudinal and nationally representative Health and Retirement Study (HRS) among adults aged 50 years or older. As a secondary objective, we examined the associations between unprocessed or minimally processed food (MPF) consumption and cognitive outcomes and assessed the extent to which gender, educational attainment, and social isolation modified the associations between UPF consumption and cognitive impairment. We hypothesized that adults with lower educational attainment and greater social isolation would be more susceptible to cognitive impairment and would show stronger associations. We examined gender differences in an exploratory manner.

METHODS

Participants were from the HRS, a US nationally representative longitudinal survey of adults aged 50 years and older and their spouse or partner. The HRS was initiated in 1992 and has since been conducted biennially (HRS Core).[16] The HRS is conducted by the University of Michigan. Briefly, the HRS adopts a stratified, multistage area probability sampling design, with oversampling of non-Hispanic Black and Hispanic individuals. In addition to its biennial Core survey, the HRS conducted the 2013 Health Care and Nutrition Study (HCNS) as a supplemental off-year survey on food purchases and dietary intake. Measures assessed in the HRS Core survey, such as socioeconomic, health, and lifestyle information, were not collected in the HCNS and were therefore drawn from the most recent previous HRS Core survey (2012).

We combined HRS Core data (2012–2020) with 2013 HCNS (n = 8073).[17,18] We included participants aged 50 years or older with UPF consumption data who had no previous Alzheimer's disease, dementia, or memory problems at baseline (2012) and who did not develop cognitive impairment within the first 2 years of follow-up (n = 1173) to mitigate the possibility of reverse causation. Additionally, we excluded participants with implausible energy intake (men: < 800 kcal/day or > 4000 kcal/day; women: < 500 kcal/day or > 3500 kcal/day; n = 348), nonpositive sampling weights (n = 157), or missing covariate information for whom data from earlier waves could not be carried forward (n = 183). This yielded 5370 participants for analysis (Figure A, available as a supplement to the online version of this article at http://www.ajph.org).

Dietary Assessment and Nova Classification

We collected dietary data using a validated semistructured food frequency questionnaire (FFQ), based on the Harvard FFQ, from the 2013 HCNS.[19] We linked dietary data to the Harvard food composition tables to estimate total energy and nutrient intake. UPF classifications followed the Nova system: MPFs (Nova 1), processed culinary ingredients (Nova 2), processed foods (Nova 3), and UPFs (Nova 4).[20] We categorized FFQ items for UPFs based on the approach previously applied in large US prospective cohorts.[21] Slight modifications to the Nova coding scheme included classifying hamburgers and chicken or turkey sandwiches as UPFs, because these foods were typically commercially prepared when the FFQ was administered. UPF and MPF items are listed in Tables A and B (available as a supplement to the online version of this article at http://www.ajph.org). We categorized UPFs into 13 mutually exclusive subgroups:

1. whole grains,
2. grains and derivatives,
3. dairy,
4. fats and oils,
5. processed meats,
6. processed fish,
7. snacks and sweets,
8. sweeteners,
9. sugar-sweetened beverages,
10. other beverages,
11. liquor,
12. sauces, and
13. mixed dishes.

We calculated energy-adjusted UPF or MPF intake (in grams) using the residual method and grouped it into gender-specific quintiles for analysis.[22]

Cognitive Impairment

We used the Langa-Weir classification of cognitive function, which has high sensitivity and specificity,[23] to define cognitive impairment. We included data through 2020, as the summary scores for the classifications were available up to 2020 in the data set distributed via the HRS website. The HRS Core interview included tests of serial 7s, backward counting, and immediate and delayed word recall to assess cognitive impairment at each wave, with a total score of 27. We classified participants as having dementia if they scored 6 or less, as having CIND if they scored 7 to 11, and as having normal cognitive function if they scored 12 to 27. We defined CIND or dementia as a score of 11 or less, encompassing both dementia and CIND as the composite outcome. We calculated person-years from the entry date to the date of first identification of outcome of interest or a censoring event (death, loss to follow-up, or end of follow-up in May 2021), whichever occurred first. For dementia analysis, we treated dementia onset as an event. For CIND, follow-up continued until CIND onset with dementia treated as an additional censoring event. For the composite CIND or dementia outcome, follow-up continued until CIND, dementia, or a censoring event.

Sociodemographic and Lifestyle Covariates

We selected demographic, socioeconomic, lifestyle, and health-related covariates as potential factors related to both UPF consumption and cognitive impairment. For race/ethnicity, the HRS assessed Hispanic/Latino ethnicity first, followed by self-reported race/ethnicity categories including non-Hispanic White, non-Hispanic Black/African American, non-Hispanic American Indian/Alaska Native, non-Hispanic Asian/Native Hawaiian/Pacific Islander, and other. We derived demographic covariates, including age (years, continuous), gender (men, women), and race/ethnicity (non-Hispanic White, non-Hispanic Black, Hispanic, and other), from the 2013 HCNS, and we drew other covariates from the baseline HRS Core (2012). If information on a covariate was missing, we carried data from earlier waves forward.

We used socioeconomic variables, including marital status (never married, married but spouse absent, separated, divorced, widowed; married or living with a partner), education (less than high school, high school graduate, some college/college graduate, postcollege), total net worth (tertiles), and household size (1, 2, ≥ 3). We calculated total net worth as the sum of all wealth components minus debts.[24] To account for cases in which debts exceeded assets, we added a small random constant. We then log-transformed the resulting values and categorized them into tertiles.

Lifestyle and health-related variables included vigorous activity (none, ≤ once/week, > once/week), smoking status (never, ever, current), alcohol consumption (nondrinker, < 5g/day, ≥ 5g/day), total energy intake (in kcal), body mass index (BMI = < 25, 25–29.9, or ≥ 30; defined as weight in kilograms divided by the square of height in meters), depressive symptoms at baseline (yes, no), and chronic disease history (yes, no). Participants reported the frequency of vigorous physical activity as every day, more than once per week, once per week, 1 to 3 times per month, or never. We assessed depressive symptoms using the 8-item Center for Epidemiologic Studies Depression Scale, with scores of 5 or higher indicating the presence of depressive symptoms. We defined chronic disease history based on self-reported physician-diagnosed diabetes, stroke, heart problems, cancer, or hypertension reported in 2 consecutive surveys.

For subgroup analyses by social isolation, we derived a social isolation index score based on the 2012 HRS Core items administered to approximately half of the participants:

1. unmarried or living alone;
2. contact with children less than once a month;
3. contact with family members other than children less than once a month;
4. contact with friends less than once a month; and
5. participation in groups, clubs, or other social organizations less than once a month.

We assigned 1 point for each condition met and summed the points across the 5 items (range = 0–5). We classified scores of 2 or more as socially isolated.[25,26]

Statistical Analysis

We applied attrition-adjusted sampling weights to account for the multistage area probability sampling design of the HRS and the analytic subsample. We estimated propensity scores using logistic regression to model the probability that HCNS respondents were eligible for the analytic subsample, adjusting for age,

gender, race/ethnicity, and years of education. We multiplied decile-based reciprocals of the mean propensity score by the original HCNS full-sample weight to compute the adjusted sampling weight. For participant characteristics, we present continuous variables as weighted means and SEs and categorical variables as frequencies and weighted percentages.

We used weighted Cox proportional hazards models to examine the associations of UPF and MPF consumption with the risks of cognitive impairment, estimating hazard ratios (HRs) and 95% confidence intervals (CIs). The proportional hazards assumption was not violated based on Schoenfeld residuals. We adjusted models for age, gender, and race/ethnicity (Model 1). Model 2 additionally adjusted for marital status, educational attainment, total net worth, household size, total energy intake, vigorous physical activity, smoking, alcohol consumption, depressive symptoms, and chronic disease history. We assessed linear trends by including the median value of UPF consumption in each quintile as a continuous variable in the model. We used restricted cubic splines (4 knots) to assess possible nonlinearity between UPF consumption and cognitive impairment risk, after truncating the top 1% of UPF intake. In sensitivity analyses, we further adjusted for BMI and diet quality. We assessed diet quality using the Mediterranean diet score (Table C),[27] given its relevance to cognitive health, and we included BMI as a potential mediator. Because UPF intake units might affect the UPF–disease associations, we further modeled UPFs as percentage of total grams and percentage of total energy intake.

To test for effect modification by gender, education, and social isolation, we included multiplicative interaction terms in the model and assessed their statistical significance using the Wald test. We did not examine effect modification by social isolation for dementia owing to small case numbers in some strata. We also estimated the associations between each UPF category and the risk of cognitive impairment. We simultaneously included each UPF category in fully adjusted models and excluded total UPF intake from the models. We conducted all statistical analyses using SAS 9.4 (SAS Institute Inc., Cary, NC; we set significance level at $P < .05$).

RESULTS

In 2013, the weighted mean age was 64.5 years (SE = 0.3); 55.2% were women, and 81.7% were non-Hispanic White. Over 8.7 years of median follow-up, we identified 266 (5.0%) new cases of dementia, 1191 (22.2%) cases of CIND, and 1310 (24.4%) cases of CIND or dementia. On average, UPFs (Nova 4) accounted for 21.5% (SD = 13.8%) of total food intake (in grams), whereas MPF (Nova 1) accounted for 71.8% (SD = 15.0%). By energy, UPFs contributed 42.4% (SD = 12.3%) and MPF contributed 43.1% (SD = 11.7%) of total energy intake. The largest contributors to total UPF intake were sugar-sweetened beverages (31.2%), other beverages (22.2%), dairy (11.2%), snacks and sweets (9.7%), and grains and derivatives (6.2%) (Table D). Participants with higher UPF consumption, compared with those in the lowest quintile, were more likely to be men, non-Hispanic Black, never married, living in a single-person household, high school graduates, current smokers, nondrinkers, physically inactive and to have lower total net worth, obesity, depressive symptoms, history of chronic disease, and higher energy intake (Table 1).

Ultraprocessed Food and Cognitive Outcomes

The associations between UPF consumption and incident cognitive impairment are shown in Table 2. UPF consumption (quintile 5 vs quintile 1) was associated with a higher risk of dementia (HR = 1.58; 95% CI = 1.003, 2.48) after adjustment for socioeconomic, health, and lifestyle factors (model 2), although the linear trend across quintiles was not statistically significant (P for trend = .50). For CIND, compared with quintile 1, UPF consumption was associated with higher risk of CIND (quintile 5: HR = 1.46; 95% CI = 1.13, 1.88), with a significant linear trend (P for trend = .03). Greater UPF consumption was associated with a higher risk of CIND or dementia (quintile 5 vs quintile 1: HR = 1.47; 95% CI = 1.16, 1.87), with a significant linear trend (P for trend = .02). We observed no significant linear or nonlinear associations for dementia, CIND, or the composite outcome of CIND or dementia in spline models (Figure B, available as a supplement to the online version of this article at http://www.ajph.org).

Sensitivity Analysis

Further adjustment for BMI yielded similar results, and adjustment for the Mediterranean diet scores somewhat attenuated HRs for dementia (quintile 5: HR = 1.53; 95% CI = 0.96, 2.42), for CIND (quintile 5: HR = 1.39; 95% CI = 1.08, 1.80), and for CIND or dementia (quintile 5: HR = 1.43; 95% CI = 1.11, 1.83; Table E, available as a supplement to the online version of this article at http://www.ajph.org). When we used UPF percentage of

TABLE 1— Baseline Characteristics for Dementia Analysis by Quintile of Energy-Adjusted Ultraprocessed Food Intake: United States, Health and Retirement Study, 2013–2020

Baseline Characteristic	Energy-Adjusted Ultraprocessed Food Intake, No. (%) or Mean ±SE					
	Total	Quintile 1	Quintile 2	Quintile 3	Quintile 4	Quintile 5
Age, y	64.5 ±0.3	65.0 ±0.6	65.9 ±0.5	65.4 ±0.5	64.7 ±0.3	62.0 ±0.3
Gender = women	3185 (55.2)	637 (58.3)	637 (54.9)	637 (54.2)	637 (54.9)	637 (53.9)
Race/ethnicity						
Non-Hispanic White	3961 (81.7)	793 (80.5)	813 (82.4)	803 (82.5)	759 (79.0)	793 (83.6)
Non-Hispanic Black	721 (7.9)	102 (5.1)	140 (8.8)	135 (7.3)	190 (11.3)	154 (7.4)
Hispanic	540 (7.3)	130 (9.1)	98 (6.3)	111 (7.8)	94 (7.3)	107 (6.3)
Other	148 (3.1)	49 (5.3)	23 (2.6)	25 (2.4)	31 (2.4)	20 (2.7)
Marital status						
Never married[a]	1601 (29.2)	295 (26.9)	325 (29.8)	320 (29.3)	336 (31.2)	325 (29.1)
Married or living with a partner	3769 (70.8)	779 (73.1)	749 (70.2)	754 (70.8)	738 (68.9)	749 (70.9)
Total net worth						
Tertile 1 (low)	1789 (32.7)	298 (26.6)	310 (28.4)	308 (26.8)	394 (35.8)	479 (44.1)
Tertile 2 (medium)	1791 (32.9)	349 (31.7)	363 (33.0)	360 (34.5)	368 (34.5)	351 (31.0)
Tertile 3 (high)	1790 (34.5)	427 (41.7)	401 (38.6)	406 (38.7)	312 (29.7)	244 (25.0)
Household size						
1	1110 (20.9)	204 (19.8)	231 (21.5)	214 (20.0)	237 (21.5)	224 (21.5)
2	2964 (53.8)	629 (57.1)	587 (52.1)	620 (57.7)	585 (52.6)	543 (50.1)
≥3	1296 (25.3)	241 (23.2)	256 (26.4)	240 (22.3)	252 (26.0)	307 (28.4)
Education level						
<high school	676 (10.8)	125 (9.8)	128 (9.4)	130 (10.4)	136 (11.3)	157 (12.8)
High school graduate	1739 (31.4)	315 (26.8)	337 (31.8)	341 (30.4)	361 (32.7)	385 (35.1)
Some college/college graduate	2172 (42.3)	454 (44.4)	435 (43.1)	432 (41.8)	429 (41.5)	422 (40.8)
Postcollege	783 (15.5)	180 (19.0)	174 (15.7)	171 (17.5)	148 (14.6)	110 (11.4)
Smoking status						
Never smoker	2489 (46.8)	519 (48.3)	505 (45.5)	491 (47.5)	502 (48.3)	472 (44.6)
Ever smoker	2179 (38.4)	461 (41.1)	453 (42.0)	462 (40.1)	412 (35.1)	391 (34.3)
Current smoker	702 (14.8)	94 (10.6)	116 (12.5)	121 (12.4)	160 (16.6)	211 (21.2)
Alcohol consumption, g/d						
Nondrinker	1973 (34.2)	327 (27.3)	361 (30.4)	373 (32.3)	417 (36.9)	495 (43.0)
<5	1912 (35.3)	361 (32.7)	369 (33.9)	414 (36.9)	399 (38.2)	369 (35.0)
≥5	1485 (30.5)	386 (40.1)	344 (35.8)	287 (30.8)	258 (25.0)	210 (22.0)
Vigorous activity						
None	2011 (35.5)	335 (30.5)	362 (32.1)	401 (33.9)	430 (38.3)	483 (42.0)
≤once/wk	1258 (24.1)	215 (19.8)	240 (23.1)	288 (27.7)	269 (26.1)	246 (24.0)
>once/wk	2101 (40.4)	524 (49.7)	472 (44.8)	385 (38.4)	375 (35.6)	345 (34.0)
Body mass index,[b] kg/m^2						
<25	1126 (21.3)	292 (29.7)	262 (25.0)	210 (19.2)	198 (18.3)	164 (14.9)
25–29.9	1839 (34.3)	397 (35.2)	365 (34.5)	407 (38.8)	333 (30.2)	337 (33.0)
≥30	2405 (44.4)	385 (35.2)	447 (40.5)	457 (42.1)	543 (51.5)	573 (52.1)

Continued

TABLE 1— Continued

Baseline Characteristic	Energy-Adjusted Ultraprocessed Food Intake, No. (%) or Mean ±SE					
	Total	Quintile 1	Quintile 2	Quintile 3	Quintile 4	Quintile 5
Has depressive symptoms[c]	363 (7.1)	66 (6.2)	52 (5.8)	72 (6.2)	80 (7.6)	93 (9.3)
Has history of chronic disease[d]	3882 (67.0)	740 (62.9)	785 (66.8)	774 (66.9)	806 (70.5)	777 (67.8)
Total energy intake, kcal/d	1629.8 ±9.6	1581.4 ±23.1	1642.9 ±23.6	1669.7 ±21.6	1658.8 ±21.3	1602.0 ±26.9

Note. Total participants = 5370; n = 1074 per quintile. Characteristics are presented for the baseline analytic sample used in the dementia analysis. We weighted all values using attrition-adjusted sampling weights. We defined quintiles as gender-specific categories of energy-adjusted ultraprocessed food intake.

[a]We included never married, married but spouse absent, separated, divorced, and widowed in this category.

[b]Body mass index is defined as weight in kilograms divided by the square of height in meters.

[c]We defined depressive symptoms as a Center for Epidemiologic Studies Depression Scale–8 items score of ≥ 5 at baseline.

[d]Chronic diseases included diabetes, stroke, heart problems, cancer, and hypertension.

total grams, the associations did not substantially change (Table F). Using UPF percentage of total energy intake, the associations were attenuated, with the association for dementia no longer statistically significant (Table G, available as a supplement to the online version of this article at http://www.ajph.org). Across UPF subgroups modeled simultaneously, only processed meat consumption was associated with a higher risk of dementia (quintile 5: HR = 2.25; 95% CI = 1.31, 3.87), for CIND (quintile 5: HR = 1.32; 95% CI = 1.02, 1.72), and for CIND or dementia (quintile 5: HR = 1.38; 95% CI = 1.09, 1.75; Table H, available as a supplement to the online version of this article at http://www.ajph.org). By contrast, MPF consumption was associated with lower risks of dementia (quintile 5: HR = 0.59; 95% CI = 0.32, 0.98), for CIND (quintile 5: HR = 0.76; 95% CI = 0.62, 0.93), and for CIND or dementia (quintile 5: HR = 0.74; 95% CI = 0.61, 0.90; Table I).

Subgroup Analysis

In subgroup analyses, higher UPF consumption was associated with increased risk of CIND (*P* for trend = .02) and CIND or dementia (*P* for trend = .02) among socially isolated participants (n = 525; 22.8%), whereas we observed no significant trends among nonsocially isolated participants (Table 3). However, we observed no statistically significant interactions, although results were suggestive (*P* for interaction = .08 for both outcomes). There was no significant interaction by gender (*P* for interaction = .99 for dementia, .71 for CIND, and .75 for CIND or dementia) or educational attainment (*P* for interaction = .87 for dementia, .62 for CIND, and .54 for CIND or dementia).

DISCUSSION

Increased UPF consumption was associated with higher risks of dementia, CIND, and CIND or dementia in a nationally representative longitudinal cohort of US older adults. Greater MPF consumption was associated with lower risks of these outcomes. Processed meat was the only UPF subgroup associated with increased risks of dementia, CIND, and CIND or dementia. The UPF-cognitive outcome associations did not differ by gender, education level, or social isolation.

Our findings build on previous research on UPF consumption and cognition. A meta-analysis[12] and prospective findings from the UK Biobank have reported that UPF consumption is associated with an increased risk of dementia,[28] and the Framingham Heart Study observed a higher risk of Alzheimer's disease.[29] Despite these studies, the research on UPF consumption and cognition remains mixed. A meta-analysis found no significant associations between UPF consumption and mild cognitive impairment.[12] Some included studies focused on selected UPF subgroups, such as processed meat and sugar-sweetened beverages, which may not capture the breadth of UPF intakes. Similarly, a study using the HRS observed no significant associations of UPF consumption with incident cognitive impairment, which encompassed both dementia and CIND.[14]

Extending previous research, our study quantified UPF intake using energy-adjusted measures (in grams) rather than daily servings (i.e., number of servings per day). Gram-based metrics may be particularly informative for certain UPF categories, such as artificially sweetened beverages, which can contribute substantial weight (in grams) but little or no energy.[30] Of note, previous FFQ validation studies of UPFs have shown that energy-adjusted values are

TABLE 2— Association Between Energy-Adjusted UPF Intake and Incident Dementia, CIND, and CIND or Dementia: United States, Health and Retirement Study, 2013–2020

Outcome	Quintile 1	Quintile 2	Quintile 3	Quintile 4	Quintile 5	*P* for Trend
Median (IQR) UPF intake, g/d[a]	222.7 (182.5–253.8)	328.4 (304.1–354.0)	434.0 (403.8–468.4)	596.1 (549.0–656.7)	972.6 (826.8–1284.4)	
Dementia						
No. of cases/person-years	39/8227	70/8167	56/8185	62/8193	39/8259	
Model 1,[b] HR (95% CI)	1 (Ref)	2.17 (1.38, 3.41)	1.80 (1.11, 2.93)	1.97 (1.33, 2.93)	1.86 (1.19, 2.91)	.07
Model 2,[c] HR (95% CI)	1 (Ref)	2.16 (1.42, 3.29)	1.74 (1.07, 2.83)	1.80 (1.20, 2.71)	1.58 (1.003, 2.48)	.5
CIND						
No. of cases/person-years	220/7571	264/7520	237/7563	230/7536	240/7586	
Model 1,[b] HR (95% CI)	1 (Ref)	1.44 (1.17, 1.78)	1.31 (1.04, 1.64)	1.28 (1.02, 1.61)	1.80 (1.40, 2.33)	≤ .001
Model 2,[c] HR (95% CI)	1 (Ref)	1.41 (1.13, 1.74)	1.28 (0.97, 1.67)	1.15 (0.90, 1.45)	1.46 (1.13, 1.88)	.03
CIND or dementia						
No. of cases/person-years	238/7571	292/7520	261/7563	260/7536	259/7586	
Model 1,[b] HR (95% CI)	1 (Ref)	1.46 (1.19, 1.78)	1.32 (1.07, 1.62)	1.34 (1.07, 1.66)	1.81 (1.42, 2.30)	≤ .001
Model 2,[c] HR (95% CI)	1 (Ref)	1.42 (1.16, 1.75)	1.28 (1.00, 1.64)	1.20 (0.95, 1.50)	1.47 (1.16, 1.87)	.02

Note. CI = confidence interval; CIND = cognitive impairment with no dementia; HR = hazard ratio; IQR = interquartile range; UPF = ultraprocessed food. Total participants = 5370.

[a]Values are presented as the energy-adjusted UPF intake (g/day) estimated using residual method, using gender-specific quintile.

[b]We adjusted models for age (continuous, years), gender (men, women), and race/ethnicity (non-Hispanic White, non-Hispanic Black, Hispanic, and other [Alaska Native/American Indian, Asian/Pacific Islander]).

[c]We further adjusted models for marital status (never married, married but spouse absent, separated, divorced, widowed; married or living with a partner), education (< high school, high school graduate, some college/college graduate, postcollege), total net worth (tertile), household size (1, 2, ≥ 3), energy intake (continuous, kcal/d), vigorous physical activity (none, ≤ once/week, > once/week), smoking (never smoker, ever smoker, current smoker), alcohol consumption (nondrinker, < 5g/d, ≥ 5g/d), baseline depressive symptom (yes or no; Center for Epidemiologic Studies Depression Scale–8 items score ≥ 5), and chronic disease history (yes or no; diseases included diabetes, stroke, heart problems, cancer, and hypertension). All models incorporated attrition-adjusted sampling weights and accounted for the complex sampling design.

TABLE 3— Associations Between Energy-Adjusted UPF Intake and Incident Dementia, CIND, and CIND or Dementia Stratified by Sociodemographic and Social Factors: United States, Health and Retirement Study, 2013–2020

Sociodemographic or Social Factor	No. Cases/ Person-Years	Quintile 1, HR (95% CI)	Quintile 2, HR (95% CI)	Quintile 3, HR (95% CI)	Quintile 4, HR (95% CI)	Quintile 5, HR (95% CI)	*P* for Trend	*P* for Interaction
Gender (n = 5 370)								
Dementia								.99
Men	113/16 574	1 (Ref)	2.10 (1.02, 4.34)	1.79 (0.80, 3.99)	1.53 (0.74, 3.19)	1.50 (0.68, 3.32)	.86	
Women	153/24 457	1 (Ref)	2.15 (1.15, 4.01)	1.75 (0.91, 3.36)	2.08 (1.25, 3.47)	1.70 (0.88, 3.28)	.4	
CIND								.71
Men	512/15 210	1 (Ref)	1.44 (1.03, 2.02)	1.37 (0.90, 2.09)	1.15 (0.78, 1.70)	1.55 (1.04, 2.32)	.11	
Women	679/22 566	1 (Ref)	1.37 (1.07, 1.75)	1.19 (0.87, 1.63)	1.15 (0.86, 1.53)	1.38 (1.03, 1.86)	.17	
CIND or dementia								.75
Men	563/15 210	1 (Ref)	1.47 (1.04, 2.06)	1.41 (0.95, 2.08)	1.23 (0.84, 1.79)	1.55 (1.05, 2.29)	.11	
Women	747/22 566	1 (Ref)	1.37 (1.10, 1.72)	1.18 (0.88, 1.58)	1.18 (0.89, 1.55)	1.41 (1.07, 1.87)	.11	
Educational level (n = 5 370)								
Dementia								.87
< high school	78/4 876	1 (Ref)	3.21 (1.62, 6.39)	2.95 (1.38, 6.32)	2.13 (1.03, 4.40)	2.62 (1.09, 6.32)	.34	
High school graduate	83/13 022	1 (Ref)	2.66 (1.17, 6.07)	1.57 (0.64, 3.81)	1.46 (0.63, 3.40)	1.29 (0.48, 3.46)	.5	
≥ college	105/23 133	1 (Ref)	1.66 (0.80, 3.45)	1.43 (0.70, 2.90)	2.39 (1.37, 4.17)	1.40 (0.64, 3.04)	.31	
CIND								.62
< high school	286/4 096	1 (Ref)	1.57 (0.99, 2.49)	0.94 (0.58, 1.53)	0.79 (0.47, 1.33)	1.37 (0.81, 2.32)	.5	
High school graduate	435/11 777	1 (Ref)	1.42 (0.98, 2.06)	1.14 (0.73, 1.77)	1.04 (0.70, 1.54)	1.19 (0.78, 1.81)	.99	
≥ college	470/21 903	1 (Ref)	1.24 (0.87, 1.75)	1.54 (1.12, 2.13)	1.38 (0.98, 1.93)	1.65 (1.05, 2.60)	.03	
CIND or dementia								.54
< high school	313/4 096	1 (Ref)	1.63 (1.06, 2.51)	1.08 (0.65, 1.78)	0.86 (0.52, 1.42)	1.46 (0.86, 2.48)	.4	
High school graduate	465/11 777	1 (Ref)	1.45 (1.03, 2.04)	1.17 (0.76, 1.78)	1.07 (0.74, 1.54)	1.20 (0.81, 1.78)	.98	
≥ college	532/21 903	1 (Ref)	1.26 (0.90, 1.78)	1.46 (1.10, 1.95)	1.45 (1.07, 1.97)	1.65 (1.07, 2.55)	.02	
Social isolation (n = 2 303)[a]								
CIND								.08
Nonsocially isolated	379/12 785	1 (Ref)	1.81 (1.30, 2.54)	1.49 (1.04, 2.14)	1.19 (0.77, 1.86)	1.08 (0.72, 1.61)	.22	
Socially isolated	151/3 450	1 (Ref)	0.57 (0.29, 1.13)	1.33 (0.62, 2.89)	0.61 (0.30, 1.21)	1.84 (0.90, 3.74)	.02	

Continued

TABLE 3— Continued

Sociodemographic or Social Factor	No. Cases/ Person-Years	Quintile 1, HR (95% CI)	Quintile 2, HR (95% CI)	Quintile 3, HR (95% CI)	Quintile 4, HR (95% CI)	Quintile 5, HR (95% CI)	*P* for Trend	*P* for Interaction
CIND or dementia								.08
Nonsocially isolated	418/12 785	1 (Ref)	1.80 (1.33, 2.44)	1.55 (1.09, 2.21)	1.23 (0.82, 1.85)	1.12 (0.77, 1.62)	.27	
Socially isolated	165/3 450	1 (Ref)	0.66 (0.35, 1.27)	1.38 (0.66, 2.86)	0.74 (0.38, 1.43)	1.82 (0.94, 3.53)	.02	

Note. CI = confidence interval; CIND = cognitive impairment with no dementia; HR = hazard ratio; UPF = ultraprocessed food. We incorporated attrition-adjusted sampling weights and accounted for the complex sampling design in all models. We adjusted models for age (continuous, years), gender (men, women), and race/ethnicity (non-Hispanic White, non-Hispanic Black, Hispanic, and other [Alaska Native/American Indian, Asian/Pacific Islander]), marital status (never married, married but spouse absent, separated, divorced, widowed; married or living with a partner), education (< high school, high school graduate, some college/college graduate, postcollege), total net worth (tertile), household size (1, 2, ≥ 3), energy intake (continuous, kcal/d), vigorous activity (none, ≤ once/week, > once/week), smoking (never smoker, ever smoker, current smoker), alcohol consumption (nondrinker, < 5g/day, ≥ 5g/day), baseline depressive symptom (yes or no; Center for Epidemiologic Studies Depression Scale–8 items score ≥ 5), and chronic disease history (yes or no; diseases include diabetes, stroke, heart problems, cancer, and hypertension). When testing effect modification by social isolation, we excluded marital status and household size because they were components of the social isolation measure.

[a]Because we observed fewer than 5 dementia cases in some UPF–social isolation subgroups (n = 525 socially isolated participants), we did not examine effect modification by social isolation in relation to dementia.

less biased in examining disease associations than are absolute intake measures.[15] Associations with dementia, CIND, and CIND or dementia combined were attenuated when UPFs were modeled as a percentage of energy intake, suggesting there should be careful consideration of UPF intake measures in cognitive health analyses.

Several possible mechanisms may underlie these associations, including gut microbiota alterations that may affect brain-derived neurotrophic factor signaling cascades and neuroplasticity.[31] UPFs may disrupt the gut–brain axis and reward system, leading to compulsive eating behaviors and biological dysfunctions. These effects may increase oxidative stress and chronic inflammation. Moreover, food additives (e.g., dietary emulsifiers and nonnutritive sweeteners) may alter antimicrobial activity, potentially increasing cognitive risks.[31–35] However, this evidence is largely based on animal models, and further studies in humans are needed.

UPFs represent a heterogeneous group, with health effects varying across UPF subcategories.[8,36] We observed adverse associations between processed meat consumption and cognitive outcomes. Our findings align with previous studies reporting that UPF animal products were associated with higher risk of cognitive impairment.[14] However, these subgroup-specific findings should be interpreted cautiously given potential limitations, including a limited range of intake levels and exposure misclassification.[37]

We found that the associations between UPF consumption and cognitive impairment tended to be stronger among socially isolated older adults, with a marginally significant interaction. Given that social isolation is a risk factor for dementia,[38] our findings indicate

that greater attention is warranted in this subgroup. Screening for social isolation has been encouraged in the United States through initiatives from the Administration for Community Living,[39] and integrating such efforts into public health or clinical settings may help identify high-risk individuals, who could benefit from reducing UPF intake for healthier cognitive aging.

Limitations

This study has several strengths, including the use of a nationally representative longitudinal cohort, which allowed us to examine temporal associations. Assessing UPF intake in different units and sensitivity analyses demonstrated the robustness of our findings.

Several limitations should also be acknowledged. The FFQ was not specifically validated for UPF consumption and may have underestimated UPF intake. We observed that the average UPF consumption was 42% in our study population, lower than the 57% reported in the Centers for Disease Control and Prevention's National Health and Nutrition Examination Survey, which used 24-hour dietary recalls.[4] As FFQs are prone to underestimating UPF intake compared with 24-hour recalls,[40] such underestimation may have attenuated the associations toward the null. However, the Nova categorization applied to this FFQ has been associated with outcomes such as cardiovascular disease and type 2 diabetes,[7,8] supporting its use.

Second, dementia and CIND were not clinically diagnosed, which may have led to misclassification of cognitive outcomes. However, we defined the cognitive outcomes based on the validated Langa-Weir classification, which has been shown to have high sensitivity and specificity and incorporates proxy assessments that may improve ascertainment of cognitive impairment or dementia in population-based studies.[23]

Third, although we excluded cognitive impairment cases occurring within the first 2 years, we cannot entirely rule out reverse causation, as cognitive changes may influence dietary behaviors. Fourth, the potential for misclassification should be acknowledged. For example, yogurt was assessed as a single FFQ item in the HRS, grouping plain and artificially sweetened yogurt, and thus classifying it as a UPF in our analysis. However, a previous study using the Harvard FFQ reported that such ambiguous items accounted for less than 5% of the total FFQ items.[8,21]

Fifth, the observed associations may partly reflect poorer overall diet quality rather than ultraprocessing per se. However, adjustment for diet quality did not fully explain the higher risks of CIND and CIND or dementia observed with greater UPF intake. Lastly, we cannot rule out potential residual or unmeasured confounding. Future studies with longer follow-ups are needed to replicate our findings.

Public Health Implications

UPF consumption has increased among US older adults, and the intake of MPF has simultaneously decreased.[4,41] We observed that, among US older adults, UPF consumption was associated with higher risks of incident dementia, CIND, and CIND or dementia combined, whereas MPF consumption was associated with lower risks of these outcomes. These findings underscore the potential importance of lower UPF consumption and greater intake of MPF for healthy cognitive aging. Consistent with this perspective, the Dietary Guidelines for the Brazilian Population recommend avoiding UPFs while emphasizing a diet based on MPFs.[42] In addition, the 2025 US Dietary Guidelines Advisory Committee highlighted the need for further research on cognitive decline.[43] The Dietary Guidelines for Americans, 2025–2030 recommend "[avoiding] highly processed foods", although they do not explicitly reference UPFs.[44]

From a public health perspective, strategies to reduce UPF intake among older adults could include expanding community-based meal programs prioritizing MPFs and promoting simple food preparation skills to reduce reliance on convenient UPFs. However, policy- and industry-level action should be prioritized. Given UPFs' low cost, taxation may be warranted, as illustrated by Colombia's recent UPF tax.[45] Front-of-package labeling, warning labels, and advertising restrictions also merit consideration.[45] Regulation of health- or environment-related marketing claims is needed, as such messaging may lead to a misperception that all UPFs are acceptable.[39] As seen in tobacco control, these measures are most effective when implemented simultaneously,[46] suggesting that a similarly coordinated regulatory approach may be necessary to mitigate the growing burden of UPF consumption. Our findings may provide evidence to inform future dietary guidance aimed at limiting UPFs while encouraging greater intake of MPFs to support cognitive health. Future studies examining food-to-food replacement across different levels of food processing may help clarify the potential cognitive benefits. AJPH

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HUMAN PARTICIPANT PROTECTION

This study was approved by the University of Michigan institutional review board. All participants provided verbal or written informed consent. The study data are publicly available at the Health and Retirement Study websites (https://hrsdata.isr.umich.edu).

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