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Association of eating duration less than 8 h with all-cause, cardiovascular, and cancer mortality

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ABSTRACT

Aims: To assess the association between eating duration less than 8 h and all-cause and cause-specific mortality.
Methods: Adult participants who reported usual intake from two valid 24-h dietary recalls were included from the National Health and Nutrition Examination Survey in 2003–2018 (n = 19,831). Mortality status as of December 2019 was obtained through linkage to the National Death Index. Average eating duration was categorized as <8, 8–<10, 10–<12, 12–14 h (mean duration), >14–16, and >16 h. Multivariable-adjusted hazard ratios (HRs) were derived.
Results: During a median follow-up of 8.1 years, compared with eating duration of 12–14 h, eating duration <8 h was robustly associated with higher cardiovascular mortality (HR, 2.35 [95 % CI, 1.39–3.98]), but not with all-cause and cancer mortality. The positive association with cardiovascular mortality remained consistent across 8 subgroups stratified by race/ethnicity, socioeconomic factors, and smoking status, and survived 14 sensitivity analyses. However, the association with all-cause mortality did not survive many sensitivity analyses.
Conclusions: Although a positive association was observed between eating duration <8 h and cardiovascular mortality, further research is required to understand whether this risk is attributed to the short eating duration itself or residual confounding resulting from its contributing factors.

1. Introduction

Time-restricted eating (TRE) has gained popularity as a dietary intervention that limits food consumption to a 4- to 12-h window each day. Recent systematic reviews and meta-analyses of intervention studies reported that TRE regimens improved cardiometabolic risk profiles, including improvements in body weight, blood pressure, glycemic control, and inflammatory markers, but did not seem to affect serum lipids [1–3]. Notably, the effect sizes depended on the compared dietary regimen and varied across studies due to the heterogeneities in TRE protocols. Eight-hour TRE is the most commonly studied, but its cardiometabolic benefits may primarily stem from caloric restriction rather than the timing of food intake [4]. Furthermore, emerging concerns about TRE include potential adverse effects such as stress responses in the brain, impaired immune response to infection [5], and unintended muscle mass loss [6].

All published randomized controlled trials (RCTs) studied the effects of TRE regimens on changes in risk factors within 1 year [1–3,6]. Long-term RCTs investigating the effects of 8-h TRE on hard clinical endpoints are currently unavailable and will be challenging to conduct

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in the future. Therefore, it is valuable to assess long-term health consequences of following an 8-h eating window using observational data. We utilized real-world observational data from the National Health and Nutrition Examination Survey (NHANES) in 2003–2018 in the United States to investigate the association of eating duration less than 8 h with all-cause and cause-specific mortality.

2. Methods

2.1. Study design and sample

NHANES is a continuous, multistage, nationally representative survey of the non-institutionalized civilian resident US population. The survey has been conducted annually with data released in 2-year cycles since 1999. In each survey, trained dietary interviewers administer a standardized 24-h dietary recall of foods and beverages at mobile examination centers, and a second dietary recall conducted via telephone 3–10 days post-interview. This analysis included 8 NHANES cycles from 2003–2004 to 2017–2018, when two dietary recalls were conducted. Nonpregnant adults aged 20 years or older who completed two valid 24-h dietary recalls and reported that both days were representative of their usual intake were included. Participants whose daily caloric intake was <50 % of the Mifflin-St. Jeor equation estimated energy requirement were excluded because of extremely low energy intake or incomplete food recalls [7]. Participants reporting extremely high daily energy intake were excluded, defined as >8000 kcal for men and >6000 kcal for women [8,9].

2.2. Eating duration

During the 24-h recall interview, participants were asked to report the clock time when a food or beverage was consumed. An eating occasion was defined as the consumption of foods or beverages exceeding 5 kcal at any given time. The eating duration was calculated daily, measured from the first to the last reported eating occasion. The eating duration was determined by averaging durations across the two recall days and subsequently categorized as: <8, 8–<10, 10–<12, 12–14, >14–16 and >16 h. The mean duration was ~13 h in our sample and therefore, the reference duration in the main analysis was prespecified as 12–14 h.

2.3. Mortality events

Mortality events were ascertained through linkage to the National Death Index through December 31, 2019, using probabilistic matching. Causes of death were ascertained using the International Statistical Classification of Disease and Related Health Problems, Tenth Revision (ICD-10). Cardiovascular mortality was defined as death resulting from heart and cerebrovascular diseases, identified by ICD-10 codes (I00-I09, I11, I13, I20-I51, and I60-I69). Cancer mortality was defined as death resulting from malignant neoplasms, identified by the ICD-10 codes (C00-C97). The follow-up period was calculated from the examination date to either the date of death, or December 31, 2019 for censored participants.

2.4. Covariate assessment

Standardized questionnaires were employed to collect information on age, sex, race/ethnicity, education, income, marital status, employment status, food security, smoking status, drinking status, and physical activity. Body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared. Self-reported race/ethnicity was categorized as non-Hispanic White, non-Hispanic Black, Hispanic, and other. Education level was divided into less than high school, high school graduate, and more than high school. Ratio of family income to poverty was calculated by dividing self-reported family income by the Department of Health and Human Services' poverty guidelines, specific to the family size, appropriate year, and state. Food security was assessed using the 10-item US Household Food Security Survey Module and grouped into 2 levels: food security (<3 affirmative responses) and food insecurity (≥3 affirmative responses), as recommended by the US Department of Agriculture [10]. The Healthy Eating Index 2015 score and total daily energy intake were calculated using data from the 24-h dietary recalls [11]. Participants were asked to self-report their leisure time physical activities, detailing their types, frequency, and duration. Minutes of vigorous-intensity physical activity were multiplied by two and added to minutes of moderate-intensity physical activity to calculate weekly moderate-intensity equivalent physical activity [12]. Participants were asked whether they had ever been told by a health professional that they had any of the following medical conditions: diabetes, hypertension, dyslipidemia, cancer, congestive heart failure, coronary heart disease, angina, heart attack, and stroke. The latter five diseases defined cardiovascular diseases (CVD) as a composite endpoint. Skipping breakfast was coded as 0 (never), 1 (sometimes), and 2 (always), depending on the number of breakfasts skipped in the two recall days.

2.5. Definition of morbidities

CVD was based on self-report. Diabetes was identified as self-reported diagnosis by a health professional or hemoglobin A1c level ≥6.5 %. Hypertension was defined as having blood pressure ≥130/80 mm Hg (mean of all measurements) or self-reported current use of anti-hypertensive drugs. Dyslipidemia was defined by total cholesterol concentration ≥240 mg/dL, low high-density lipoprotein cholesterol concentration (<40 mg/dL in men or <50 mg/dL in women), or self-reported current use of lipid-lowering drugs. Chronic kidney disease was defined as having a urine albumin to creatinine ratio ≥30 mg/g or an estimated glomerular filtration rate <60 mL/min/1.73 m^2.

2.6. Statistical analysis

Multivariable Cox proportional hazard models were used to examine the association between eating duration and all-cause mortality. Cause-specific hazard models were used to analyze cardiovascular and cancer mortality, accounting for competing risks [13,14]. The proportional hazards assumption was tested using the Kolmogorov-type supremum test [15], with no evidence of violations observed. A set of models were constructed sequentially, adjusting for demographics, socioeconomic factors, behavioral and dietary factors, comorbidities, and breakfast skipping. Model 1 was adjusted for age, sex, race/ethnicity, and total energy intake. Model 2 was additionally adjusted for education level, ratio of family income to poverty, marital status, employment status, and food security status. Model 3 was adjusted for model 2 covariates plus smoking status, drinking status, leisure time physical activity, Healthy Eating Index 2015 score, BMI, and BMI squared. Adjusting for BMI squared accounted for the non-linear association of BMI with mortality [16]. Self-reported CVD, cancer, dyslipidemia, hypertension, and diabetes were further adjusted in model 4, as a diagnosis of these diseases may lead to dietary behavior changes. Skipping breakfast was further adjusted in model 5, as previous studies in NHANES reported that breakfast skipping was associated with CVD mortality [17–19]. Missing data in covariates were handled using multiple imputation by chained equations with predictive mean matching across 5 replications [20]. Adjusted hazard ratios (HRs) and 95 % confidence intervals (CIs) were derived. Absolute risk differences (ARDs) for each HR (eating duration <8 vs. 12–14 h) was computed using R *riskRegression* [21] and *pec* [22] packages. The mean value of the covariates, a prespecified length of follow-up (5, 10, or 15 years), and different baseline age (50, 55, 60, or 65 years) were used. The 95 % CIs were derived from 500 bootstrap samples.

To further examine the potential impact of confounding on the robustness of the significant association identified in the overall sample, the association was assessed in 8 relatively more homogenous subgroups with the following characteristics: (1) non-Hispanic White, (2) other races/ethnicities, (3) education <high school, (4) education ≥high school, (5) ratio of family income to poverty ≤1, (6) ratio of family income to poverty >1, (7) never smoking, and (8) current smoking. We also conducted 4 subgroup analyses among people with different morbidities: hypertension, dyslipidemia, chronic kidney disease as well as CVD and diabetes. CVD and diabetes were combined into one composite morbidity to increase the sample size for this subgroup analysis, because the two diseases share many risk factors including dietary ones and have common pathophysiological mechanisms [23].

Fourteen sensitivity analyses were conducted to assess the robustness of the main findings: (1) using self-reported daily energy intake <75 % of the Mifflin-St.Jeor predicted energy requirement to exclude participants; (2) requiring eating duration <8 h on two dietary recall days; (3) requiring eating duration <9 h on two dietary recall days; (4) requiring dietary recalls conducted on one weekday and one weekend day; (5)

Table 1
Baseline characteristics of US adults by eating duration, National Health and Nutrition Examination Survey, 2003–2018.

	Overall	Eating duration, hours					
		<8	8–<10	10–<12	12–14	>14–16	>16
Number of participants, n	19,831	383	1411	4821	8162	3573	1481
Age, mean (SE), y	51.7 (0.1)	48.0 (1.1)	50.8 (0.5)	52.0 (0.3)	52.2 (0.2)	52.3 (0.3)	49.2 (0.4)
Sex, n (%)							
Men	10,182 (51.3)	214 (55.9)	687 (48.7)	2277 (47.2)	4165 (51.0)	1986 (55.6)	853 (57.6)
Women	9649 (48.7)	169 (44.1)	724 (51.3)	2544 (52.8)	3997 (49.0)	1587 (44.4)	628 (42.4)
Race/ethnicity[a], n (%)							
Non-Hispanic White	10,173 (51.3)	121 (31.6)	580 (41.1)	2275 (47.2)	4368 (53.5)	2035 (57.0)	794 (53.6)
Non-Hispanic Black	3276 (16.5)	143 (37.3)	351 (24.9)	861 (17.9)	1184 (14.5)	472 (13.2)	265 (17.9)
Hispanic	4472 (22.6)	87 (22.7)	383 (27.1)	1243 (25.8)	1818 (22.3)	692 (19.4)	249 (16.8)
Other	1910 (9.6)	32 (8.4)	97 (6.9)	442 (9.2)	792 (9.7)	374 (10.5)	173 (11.7)
Education, n (%)							
Less than high school	4370 (22.0)	129 (33.7)	449 (31.8)	1250 (25.9)	1693 (20.7)	616 (17.2)	233 (15.7)
High school graduate	4519 (22.8)	105 (27.4)	364 (25.8)	1145 (23.8)	1809 (22.2)	772 (21.6)	324 (21.9)
More than high school	10,942 (55.2)	149 (38.9)	598 (42.4)	2426 (50.3)	4660 (57.1)	2185 (61.2)	924 (62.4)
Ratio of family income to poverty, median (IQR)	2.3 (3.1)	1.5 (1.9)	1.8 (2.3)	2.1 (2.7)	2.5 (3.3)	2.8 (3.5)	2.6 (3.2)
Marital status, n (%)							
Married or living with partner	12,498 (63.0)	179 (46.7)	769 (54.5)	2938 (60.9)	5386 (66.0)	2357 (66.0)	869 (58.7)
Others	7333 (37.0)	204 (53.3)	642 (45.5)	1883 (39.1)	2776 (34.0)	1216 (34.0)	612 (41.3)
Employment status, n (%)							
Employed	10,435 (52.6)	178 (46.5)	619 (43.9)	2170 (45.0)	4389 (53.8)	2187 (61.2)	892 (60.2)
Others	9396 (47.4)	205 (53.5)	792 (56.1)	2651 (55.0)	3773 (46.2)	1386 (38.8)	589 (39.8)
Food security status, n (%)							
Food security	16,917 (85.3)	299 (78.1)	1144 (81.1)	4034 (83.7)	7060 (86.5)	3134 (87.7)	1246 (84.1)
Food insecurity	2914 (14.7)	84 (21.9)	267 (18.9)	787 (16.3)	1102 (13.5)	439 (12.3)	235 (15.9)
Smoking status, n (%)							
Never	10,886 (54.9)	211 (55.1)	823 (58.3)	2755 (57.1)	4527 (55.5)	1855 (51.9)	715 (48.3)
Former	5366 (27.1)	82 (21.4)	336 (23.8)	1280 (26.6)	2291 (28.1)	992 (27.8)	385 (26.0)
Current	3579 (18.0)	90 (23.5)	252 (17.9)	786 (16.3)	1344 (16.5)	726 (20.3)	381 (25.7)
Drinking status, n (%)							
Never	2631 (13.3)	69 (18.0)	223 (15.8)	741 (15.4)	1045 (12.8)	415 (11.6)	138 (9.3)
Former	3941 (19.9)	80 (20.9)	339 (24.0)	1020 (21.2)	1588 (19.5)	661 (18.5)	253 (17.1)
Current	13,259 (66.9)	234 (61.1)	849 (60.2)	3060 (63.5)	5529 (67.7)	2497 (69.9)	1090 (73.6)
Leisure time physical activity, median (IQR), min/week	28 (240)	0 (180)	0 (180)	0 (240)	40 (240)	60 (300)	35 (270)
HEI-2015 score, mean (SE)	55.2 (0.1)	50.0 (0.6)	51.9 (0.3)	55.0 (0.2)	55.9 (0.1)	56.0 (0.2)	54.5 (0.4)
Energy intake, mean (SE), kcal/d	2087.0 (5.4)	1721.2 (29.8)	1858.1 (18.0)	1946.3 (10.1)	2104.7 (8.3)	2230.2 (12.8)	2414.3 (22.9)
Skipping breakfast, n (%)							
Always	873 (4.4)	144 (37.6)	196 (13.9)	213 (4.4)	163 (2.0)	73 (2.0)	84 (5.7)
Sometimes	2487 (12.5)	134 (35.0)	358 (25.4)	684 (14.2)	737 (9.0)	334 (9.3)	240 (16.2)
Never	16,471 (83.1)	105 (27.4)	857 (60.7)	3924 (81.4)	7262 (89.0)	3166 (88.6)	1157 (78.1)
Meal frequency, median (IQR) per day	3.0 (0.5)	2.0 (0.5)	2.5 (1.0)	3.0 (0.5)	3.0 (0.5)	3.0 (0.5)	3.0 (0.5)
Snack frequency, median (IQR) per day	2.5 (2.0)	1.0 (1.0)	1.5 (1.0)	2.0 (1.5)	2.5 (1.5)	3.0 (1.5)	3.5 (2.5)
BMI, mean (SE), kg/m^2	28.7 (0.0)	28.8 (0.4)	29.2 (0.2)	28.7 (0.1)	28.6 (0.1)	28.6 (0.1)	29.4 (0.2)
Diabetes[b,c], n (%)	2522 (12.7)	34 (8.9)	198 (14.0)	653 (13.5)	1037 (12.7)	403 (11.3)	197 (13.3)
Hypertension[b,c], n (%)	7191 (36.3)	129 (33.7)	493 (34.9)	1748 (36.3)	2986 (36.6)	1284 (35.9)	551 (37.2)
Dyslipidemia[b,c], n (%)	6902 (34.8)	94 (24.5)	415 (29.4)	1652 (34.3)	2930 (35.9)	1307 (36.6)	504 (34.0)
Cardiovascular disease[b,c], n (%)	2144 (10.8)	48 (12.5)	194 (13.7)	612 (12.7)	912 (11.2)	383 (10.7)	151 (10.2)
Cancer[b,c], n (%)	2185 (11.0)	31 (8.1)	134 (9.5)	553 (11.5)	890 (10.9)	411 (11.5)	166 (11.2)
Systolic BP, mean (SE), mm Hg	124.9 (0.1)	125.8 (1.1)	125.7 (0.5)	125.7 (0.3)	124.9 (0.2)	123.9 (0.3)	123.6 (0.5)
Diastolic BP, mean (SE), mm Hg	70.4 (0.1)	70.1 (0.7)	69.4 (0.3)	70.1 (0.2)	70.5 (0.1)	70.7 (0.2)	71.5 (0.3)
Fasting plasma glucose, mean (SE), mg/dL	106.2 (0.2)	101.5 (1.1)	108.5 (1.1)	106.2 (0.5)	106.3 (0.4)	105.0 (0.5)	106.9 (0.9)
Hemoglobin A1c, mean (SE), %	5.7 (0.0)	5.6 (0.0)	5.7 (0.0)	5.7 (0.0)	5.7 (0.0)	5.7 (0.0)	5.8 (0.0)
HDL-C, mean (SE), mg/dL	53.2 (0.1)	53.6 (0.9)	52.6 (0.4)	52.8 (0.2)	53.3 (0.2)	53.4 (0.3)	53.3 (0.4)
LDL-C, mean (SE), mg/dL	113.4 (0.3)	114.2 (1.8)	111.4 (0.9)	113.0 (0.5)	114.0 (0.4)	114.1 (0.6)	111.0 (0.9)

Abbreviations: BMI, body mass index; BP, blood pressure; HDL-C, high-density lipoprotein cholesterol; HEI-2015, healthy eating index 2015; LDL-C, Low-density lipoprotein cholesterol; IQR, interquartile range; SE, standard error.

[a] Race/ethnicity was self-reported and grouped into 4 categories: non-Hispanic White, non-Hispanic Black, Hispanic, and other. The “other” group included other non-Hispanic races and multiple races.

[b] Based on self-report.

[c] All p values for the differences in the characteristics across different eating duration subgroups were <0.001, except for diabetes (0.45), hypertension (0.29), cardiovascular disease (0.15), and cancer (0.08).

using daily energy intake <800 or >8000 kcal for men and <600 or >6000 kcal for women to exclude participants with implausible caloric intake, a commonly used exclusion criterion in nutritional epidemiology; (6) using eating duration of 10–12 h for reference; (7) using eating duration of 11–13 h for reference; (8) using eating duration of 13–15 h for reference; (9) removing mortality events during the first year of follow-up to address reverse causation; (10) removing mortality events during the first two years of follow-up; (11) additionally adjusting for measured cardiovascular risk factors including systolic blood pressure, diastolic blood pressure, fasting plasma glucose, hemoglobin A1c, low-density lipoprotein cholesterol, and high-density lipoprotein cholesterol, but these may be mediators, available in a subset of participants only; (12) additionally adjusting for meal frequency; (13) additionally adjusting for snack frequency; and (14) additionally adjusting for both meal frequency and snack frequency. The energy-adjusted Pearson correlation coefficients among eating duration, meal frequency, and snack frequency were estimated to assess the possibility of overadjustments due to strong correlations. Additionally, a post-hoc analysis was conducted to explore the association between eating duration and lean body mass.

All analyses were conducted following the NHANES analytic guidelines and accounted for the unequal probability of selection, oversampling of certain subpopulations, and nonresponse to ensure obtaining nationally representative estimates. A 2-tailed P value < 0.05 was used to determine statistical significance. Results from subgroup analyses were treated as exploratory. All analyses were performed using SAS version 9.4 and R version 4.4.1.

2.7. Patient and public involvement

This is a secondary analysis of de-identified data from a national survey that was not specifically designed for the current study. Therefore, no patients were involved in the study design, study implementation, setting the research question, or the outcome measures.

3. Results

3.1. Characteristics of study sample

Among the 19,831 participants included (Fig. S1), the weighted mean (SE) age was 51.7 (0.1) years at baseline, 10,182 (51.3 %) were men, and 10,173 (51.3 %) were non-Hispanic White (Table 1). The mean eating duration was 12.80 h and the median was 12.75 h. Compared to those with the reference eating duration of 12–14 h, participants with eating duration <8 h: (1) were younger, and more likely to be men, non-Hispanic Black, current smokers, and to have food insecurity; (2) were more likely to have < high school education, lower income, lower total energy intake, lower diet quality score, and less leisure-time physical activity, and were less likely to be current alcohol consumers; (3) had lower prevalence of dyslipidemia, hypertension, diabetes, and cancer, all based on self-reports; and (4) were more likely to always skip breakfast and had lower meal frequency and snack frequency. The percent of missing data for each covariate is shown in Table S1. Ratio of family income to poverty had the highest percent of missing data (7.5 %).

3.2. Association of eating duration <8 h with mortality

During a median follow-up of 8.1 years (interquartile range, 4.4 to 11.6; maximum 17), 2794 all-cause deaths occurred, including 833 cardiovascular deaths and 646 cancer deaths. Based on the fully adjusted model 5 in the overall sample, compared with eating duration of 12–14 h, eating duration <8 h was significantly associated with all-cause mortality (hazard ratio [HR], 1.40 [95 % CI, 1.01 to 1.94], P = 0.04), but not cancer mortality (HR, 1.27 [95 % CI, 0.66 to 2.47], P = 0.47). Eating duration <8 h was significantly associated with a higher risk of cardiovascular mortality (HR, 2.35 [95 % CI, 1.39 to 3.98], P = 0.002; 15-year absolute risk difference (ARD) at baseline age of 60 years, 1.95 % [95 % CI, 1.64 % to 2.97 %]) (Fig. 1 and Table 2). Other eating durations were not associated with all-cause, cardiovascular, or cancer mortality, compared with eating duration of 12–14 h (all $P > 0.05$).

3.3. Association of eating duration <8 h and cardiovascular mortality by subgroups

Among relatively homogeneous subgroups, compared with eating duration of 12–14 h, eating duration <8 h was still associated with cardiovascular mortality in the subgroups with the following characteristics: non-Hispanic White (HR, 2.32 [95 % CI, 1.17 to 4.59]), minority race/ethnicity (HR, 2.45 [95 % CI, 1.23 to 4.91]), <high school (HR, 2.97 [95 % CI, 1.22 to 7.22]), ≥high school (HR, 2.10 [95 % CI, 1.14 to 3.85]), ratio of family income to poverty ≤1 (HR, 3.23 [95 % CI, 1.37 to 7.63]), ratio of family income to poverty >1 (HR, 2.48 [95 % CI, 1.36 to 4.52]), and current smoking (HR, 7.94 [95 % CI, 3.18 to 19.85]) (Table 3). Although eating duration <8 h was not statistically significantly associated with cardiovascular mortality in never smokers (HR, 2.03 [95 % CI, 0.83 to 4.92]), the HR estimate was qualitatively similar to that in the overall sample (HR, 2.35 [95 % CI, 1.39 to 3.98]).

For subgroups living with different medical conditions, eating duration <8 h, compared to eating duration of 12–14 h, was significantly associated with a higher risk of cardiovascular mortality in individuals with CVD or diabetes (HR, 3.24 [95 % CI, 1.80 to 5.83], P < 0.001; 15-year ARD at baseline age of 60 years, 7.45 % [95 % CI, 4.81 % to 10.10 %]). However, such a positive association was not seen in individuals with hypertension, dyslipidemia, and chronic kidney disease.

3.4. Sensitivity analyses

Fourteen sensitivity analyses were conducted in total. For cardiovascular mortality, using self-reported daily energy intake <75 % of the Mifflin-St.Jeor equation predicted energy requirement to exclude participants yielded results similar to those of the primary analyses (Table 4). Results also did not change qualitatively when using daily energy intake <800 or >8000 kcal in men and <600 or >6000 kcal in women for exclusion, requiring eating duration <8 or <9 h on both dietary recall days, requiring dietary recalls conducted for one weekday and one weekend day, using eating duration of 10–12, 11–13, or 13–15 h as the reference, and excluding mortality events within the first one or two years of follow-up did not materially change the results. Further adjusting for objectively measured cardiovascular risk factors, meal frequency, snack frequency, and the latter two combined did not alter the observed association. However, the association between eating duration <8 h and all-cause mortality did not survive the majority of sensitivity analyses (Table S2). The energy-adjusted Pearson correlation coefficient between eating duration and meal frequency was 0.22 and for snack frequency, it was 0.45 (both P < 0.001).

3.5. Post-hoc analysis

The post-hoc analysis in a subsample (n = 8319) showed that eating duration <8 h, compared to eating duration of 12–14 h, was significantly associated with lower total lean body mass (−2.7 kg) and lower appendicular lean mass (−1.3 kg) assessed using dual-energy X-ray absorptiometry, both overall and relative to BMI (Table S3).

4. Discussion

Among 19,831 US adults who were followed for up to 17 years, eating duration <8 h vs 12–14 h was significantly associated with a higher risk of cardiovascular mortality in the overall sample and among individuals with CVD or diabetes. The positive association between eating duration <8 h and cardiovascular mortality remained robust in 8

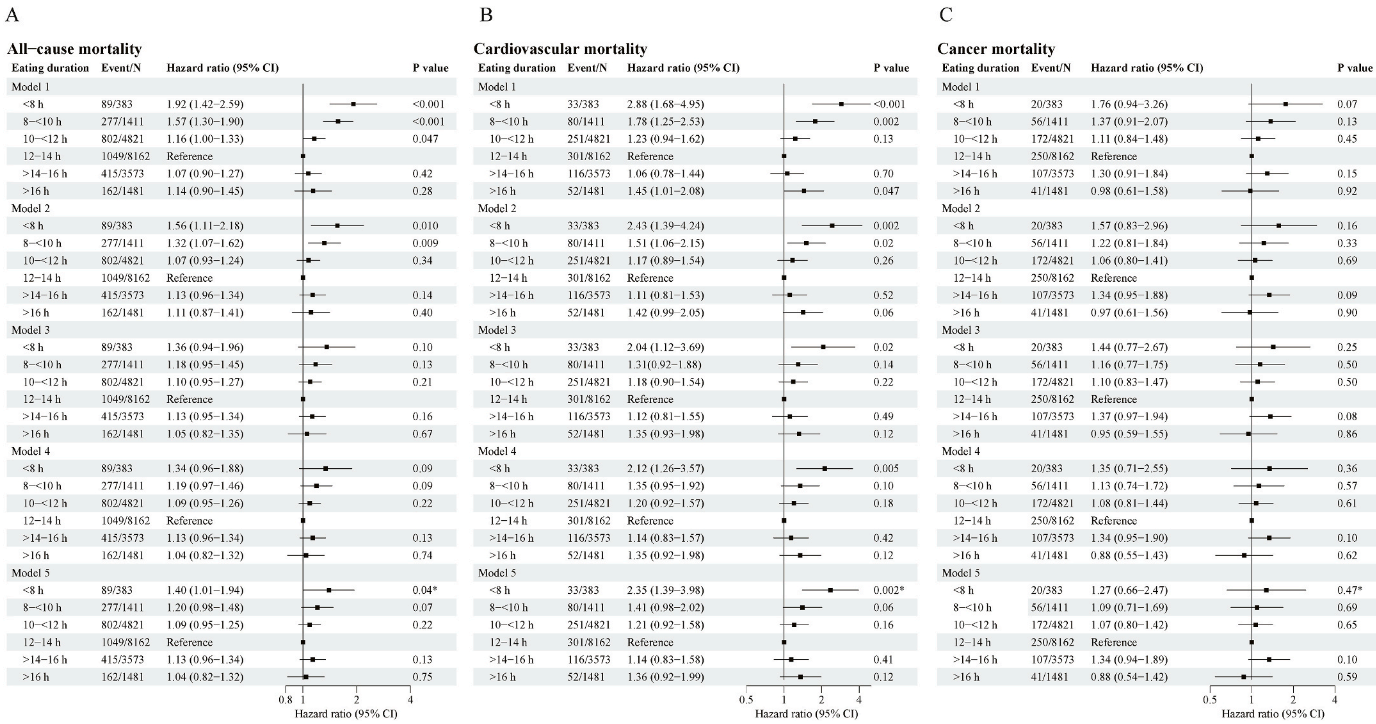

Fig. 1. Associations of eating duration with all-cause, cardiovascular, and cancer mortality.
Cox proportional hazard models were used for all-cause mortality. Cause-specific hazard models were used for cardiovascular and cancer mortality. Model 1 was adjusted for age, sex, race/ethnicity, and total energy intake. Model 2 was additionally adjusted for education level, ratio of family income to poverty, marital status, employment status, and food security status. Model 3 was adjusted for model 2 covariates plus smoking status, drinking status, leisure time physical activity, Healthy Eating Index 2015 score, body mass index, and body mass index squared. Model 4 was adjusted for model 3 covariates plus self-reported diabetes, hypertension, dyslipidemia, cardiovascular disease, and cancer. Model 5 was adjusted for model 4 covariates plus skipping breakfast. *After applying the Benjamini-Hochberg method to control the false discovery rate, the adjusted P value was 0.06 for all-cause mortality, 0.006 for cardiovascular mortality, and 0.47 for cancer mortality.

subgroups stratified by race/ethnicity and socioeconomic factors, as well as survived 14 sensitivity analyses. Eating duration <8 h was not associated with all-cause and cancer mortality.

No clinical trials in humans have studied the effect of TRE on clinical endpoints such as CVD and mortality, due to short intervention times, usually ranging from 1 month to 1 year [1–3]. Animal studies found that different forms of time-restricted feeding (TRF) promoted health and longevity in multiple species, with caloric restriction showing the strongest lifespan extension effect [24–27]. However, emerging evidence suggests that might come with health trade-offs, such as weakened immune function and accelerated atherosclerosis progression and loss of lean mass when significant caloric restriction is simultaneously applied [5,24,28]. Whether lessons learned from animal experiments are translatable to humans for assessing TRE's effect on survival remains unknown. To our knowledge, only two prospective cohort studies have investigated the association between eating duration and clinical events [29,30], but neither assessed eating duration <8 h specifically. A study conducted in 2431 women with breast cancer reported that in comparison to fasting <13 h per night, fasting ≥13 h per night (converted to an eating duration ≤11 h) was significantly associated with lower risk of breast cancer recurrence, but not of all-cause or breast cancer-specific mortality [29]. The other study using data from the NutriNet-Santé cohort did not find an association between nighttime fasting duration >13 h (i.e., eating duration <11 h) and CVD [30]. This finding is largely in line with the results of our study that eating duration of 8–<10 h or 10–<12 h was not associated with cardiovascular mortality. To our knowledge, ours is the first investigation of all-cause and cause-specific mortality risk associated with eating duration <8 h.

Careful adjustment for confounding is essential to identify an independent association between eating duration <8 h and mortality. Although both 8-h TRE in the interventional setting and eating duration less than 8 h in the real world focus on an 8-h eating window, their underlying contributing reasons and thus the related health implications are likely not the same. Eating duration can be affected by intentional adoption of TRE practice for health or weight management, or by a wide range of other factors, including socioeconomic status, food insecurity, social risk factors, dietary behaviors, and lifestyle choices [31,32]. Unfortunately, our study was not able to distinguish whether the short eating duration was due to intentional or unintentional reasons. In this study, a set of demographic, economic, social, and lifestyle factors were adjusted to minimize confounding effects. Importantly, total energy intake was adjusted to obtain an energy-independent association, because caloric restriction is a major contributor to the health effects observed in RCTs [4,25]. Skipping meals, in particular breakfast, was considered as it may contribute to a shorter eating window and has been associated with an increased risk of cardiovascular mortality [17–19]. Eating duration and meal/snack frequency are expected to be strongly correlated, which was seen in our data. Short eating duration may be a cause as well as an effect of lower meal and snack frequencies. Similarly, the associations between eating duration and measured risk factors such as blood pressure, glycemic measures, and serum lipids may be bidirectional. However, sensitivity analyses revealed a robust association between eating duration <8 h and cardiovascular mortality even after adjusting for meal frequency, snack frequency, and clinical risk factors. Although overadjustment cannot be ruled out, it would contribute to weakening the association. Subgroup analysis can be treated as a complementary confounding control strategy in addition to multivariable adjustment, due to the examination of the association in more homogenous subgroups. The consistent results observed from subgroup analyses in this study provide support for the existence of a positive association between eating duration <8 h and cardiovascular mortality.

The NHANES is not designed to answer mechanistic questions, but

Table 2
Absolute risk difference for the association between eating duration <8 vs 12–14 h and cardiovascular mortality.

	Absolute risk difference (95 % CI), at different length of follow-up[a]		
	5-year	10-year	15-year
Overall sample[b]			
Starting age at 50 years	0.19 (0.15–0.33)	0.46 (0.37–0.79)	0.70 (0.60–1.24)
Starting age at 55 years	0.33 (0.25–0.54)	0.78 (0.63–1.26)	1.17 (1.00–1.92)
Starting age at 60 years	0.57 (0.43–0.87)	1.32 (1.04–1.98)	1.95 (1.64–2.97)
Starting age at 65 years	0.99 (0.72–1.41)	2.21 (1.69–3.13)	3.16 (2.58–4.57)
People with cardiovascular disease or diabetes[c]			
Starting age at 50 years	0.96 (0.47–1.51)	2.37 (1.28–3.62)	3.79 (2.12–5.59)
Starting age at 55 years	1.44 (0.77–2.16)	3.47 (2.00–5.07)	5.38 (3.26–7.58)
Starting age at 60 years	2.14 (1.20–3.12)	5.00 (3.02–6.99)	7.45 (4.81–10.10)
Starting age at 65 years	3.15 (1.83–4.42)	7.05 (4.45–9.54)	9.99 (6.67–13.15)

[a] Absolute risk difference was estimated using R *riskRegression* and *pec* packages. The 95 % CI was derived from 500 bootstrap samples. Baseline age was set at 50, 55, 60, and 65 years. The follow-up length was prespecified at 5, 10, and 15 years. The mean values of the included covariates were used. Cause-specific hazard models were adjusted for age, sex, race/ethnicity, total energy intake, education level, ratio of family income to poverty, marital status, employment status, food security status, smoking status, drinking status, leisure time physical activity, Healthy Eating Index 2015 score, body mass index, body mass index squared, and self-reported diabetes, hypertension, dyslipidemia, cardiovascular disease, cancer, and skipping breakfast, where relevant.

[b] The number of cases of cardiovascular mortality and all participants in the <8-h subgroup were 33 and 383 and in the subgroup with eating duration of 12–14 h were 301 and 8162, respectively.

[c] Cardiovascular disease was a composite endpoint, including self-reported congestive heart failure, coronary heart disease, angina, heart attack, and stroke. Diabetes was identified through self-reported diagnosis by a health professional or hemoglobin A1c ≥ 6.5 %. The number of cases of cardiovascular mortality and all participants in the <8-h subgroup were 21 and 79 and in the subgroup with eating duration of 12–14 h were 180 and 1889, respectively.

excess cardiovascular mortality after following a short eating window might not likely be attributed to blood pressure, serum lipids, or blood glucose, because adjusting for these factors did not attenuate the association and these factors did not differ considerably between people with eating duration <8 h and those with the reference eating duration. A possible mechanism contributing to excess cardiovascular mortality might be loss of lean body mass following prolonged fasting. A meta-analysis found that a short eating window of 6–8 h, but not longer, reduced lean mass [6]. Furthermore, such loss in lean mass was seen in the intervention studies only restricting eating window, but not in the studies additionally including caloric restriction and/or exercise as a composite strategy. Low lean mass and loss of lean mass have been associated with an increased risk of CVD and mortality [33,34]. Consistently, our post-hoc analysis using dual-energy X-ray absorptiometry data revealed that individuals with eating duration <8 h had lower lean mass, both overall and relative to BMI, compared with those with eating duration of 12–14 h. Other potential mechanisms are from animal studies. In mice, prolonged fasting and the following re-feeding can trigger hormonal stress responses in the brain and lead to a surge of monocytes post-fasting, impairing the host's immune response to

Table 3
Subgroup analysis for the association between eating duration <8 vs 12–14 h and cardiovascular mortality.

Subgroup characteristics	Events/N in <8 h	Event/N in 12–14 h	Hazard ratio[a] (95 % CI)	P value
Race/ethnicity				
Non-Hispanic White	13/121	216/4368	2.32 (1.17–4.59)	0.02
Others[b]	20/262	85/3794	2.45 (1.23–4.91)	0.01
Socioeconomic status				
<High school	17/129	85/1693	2.97 (1.22–7.22)	0.02
≥High school	16/254	216/6469	2.10 (1.14–3.85)	0.02
Ratio of family income to poverty ≤1[c]	11/121	43/1326	3.23 (1.37–7.63)	0.008
Ratio of family income to poverty >1[c]	22/262	258/6836	2.48 (1.36–4.52)	0.003
Smoking status				
Never smoking	15/211	132/4527	2.03 (0.83–4.92)	0.12
Current smoking	7/90	33/1344	7.94 (3.18–19.85)	<0.001
Comorbidity				
Hypertension[d]	22/182	242/4197	1.74 (0.99–3.07)	0.056
Dyslipidemia[e]	18/184	189/4173	1.87 (0.96–3.65)	0.07
Chronic kidney disease[f]	21/85	175/1444	1.55 (0.91–2.63)	0.10
Cardiovascular disease or diabetes[g]	21/79	180/1889	3.24 (1.80–5.83)	<0.001

[a] Cause-specific hazard models were adjusted for age, sex, race/ethnicity, total energy intake, education level, ratio of family income to poverty, marital status, employment status, food security status, smoking status, drinking status, leisure time physical activity, Healthy Eating Index 2015 score, body mass index, body mass index squared, and self-reported diabetes, hypertension, dyslipidemia, cardiovascular disease, cancer, and skipping breakfast, where relevant.

[b] The "Others" group included non-Hispanic Black, Hispanic, other non-Hispanic races, and multiple races.

[c] Ratio of family income to poverty was calculated by dividing self-reported family income by the Department of Health and Human Services' poverty guidelines, specific to the family size, appropriate year, and state.

[d] Hypertension was defined as having blood pressure ≥130/80 mm Hg or self-reported current use of anti-hypertensive drugs.

[e] Dyslipidemia was defined based on total cholesterol ≥240 mg/dL, low high-density lipoprotein cholesterol (<40 mg/dL for men or <50 mg/dL for women), or self-reported current use of lipid-lowering drugs.

[f] Chronic kidney disease was defined as having a urine albumin to creatinine ratio ≥30 mg/g or an estimated glomerular filtration rate <60 mL/min/1.73 m^2.

[g] Cardiovascular disease was a composite endpoint, including self-reported congestive heart failure, coronary heart disease, angina, heart attack, and stroke. Diabetes was identified through self-reported diagnosis by a health professional or hemoglobin A1c ≥ 6.5 %.

Table 4
Sensitivity analysis for the association between eating duration <8 h and cardiovascular mortality.

	Event/N in <8 h	Event/N in 12–14 h unless specified otherwise	Hazard ratio[a] (95 % CI)	P value
Refining exposure classification				
Excluding reported energy intake <75 % of Mifflin-St.Jeor equation predicted energy requirement	20/243	257/7115	2.58 (1.29–5.12)	0.008
Using total energy intake <800 kcal/day for men and <600 kcal/day women to exclude participants	31/414	304/8261	2.24(1.36–3.68)	0.002
Requiring eating duration <8 h on two dietary recall days	8/116	157/3876	1.78 (0.85–3.74)	0.13
Requiring eating duration <9 h on two dietary recall days	24/264	157/3876	2.02 (1.05–3.87)	0.03
Requiring dietary recalls conducted on one weekday and one weekend day	21/211	158/4166	3.14 (1.64–6.00)	<0.001
Using other eating durations for reference				
10–12 h	33/383	272/5631	2.06 (1.25–3.40)	0.005
11–13 h	33/383	345/7660	2.15 (1.30–3.54)	0.003
13–15 h	33/383	237/6758	2.21 (1.30–3.78)	0.004
Addressing reverse causation				
Excluding death events within the first year of follow-up	30/374	282/8099	2.36 (1.35–4.14)	0.002
Excluding death events within the first two years of follow-up	24/366	264/8010	2.08 (1.07–4.04)	0.03
Additional adjustment				
Cardiovascular risk factors[b]	17/161	141/3734	3.83 (1.91–7.67)	<0.001
Meal frequency	33/383	301/8162	2.22 (1.29–3.82)	0.004
Snack frequency	33/383	301/8162	2.06 (1.18–3.58)	0.01
Meal and snack frequency	33/383	301/8162	1.87 (1.05–3.34)	0.04

[a] Cause-specific hazard models were adjusted for age, sex, race/ethnicity, total energy intake, education level, ratio of family income to poverty, marital status, employment status, food security status, smoking status, drinking status, leisure time physical activity, Healthy Eating Index 2015 score, body mass index, body mass index squared, and self-reported diabetes, hypertension, dyslipidemia, cardiovascular disease, cancer, and skipping breakfast, where relevant.
[b] Cardiovascular risk factors including systolic blood pressure, diastolic blood pressure, fasting plasma glucose, hemoglobin A1c, low-density lipoprotein cholesterol, and high-density lipoprotein cholesterol.

infection [5]. Another small experiment conducted in apolipoprotein E-deficient mice reported that alternate-day fasting aggravated both early and advanced atherosclerotic lesion formation [28]. It should be clear that all possible mechanisms discussed are speculative before more concrete mechanistic evidence is available.

This study utilized highly standardized data derived from a large nationally representative sample with a maximal follow-up of 17 years, adjusted for potential confounding factors, implemented a number of sensitivity analyses, accounted for competing risks, and provided both relative and absolute risk estimates. However, limitations need to be acknowledged to appropriately interpret the study findings. First, dietary measurements in our study were derived from two 24-h recalls, which could be subject to recall bias. Furthermore, participants' habitual eating pattern may not be accurately assessed, although we excluded people who reported an atypical diet on either day. However, the results were robust when limited to those with eating duration <8 or <9 h on two dietary recall days and retaining participants who reported dietary intake on one weekday and one weekend day. Second, there is no established duration for normal eating. The reference duration of 12–14 h in primary analyses was arbitrarily centered around the mean eating duration of the study sample. Nevertheless, using eating duration of 10–12, 11–13, or 13–15 h as the reference yielded similar results. Third, due to the sample size limitation, early and late 8-h eating window could not be studied separately, although evidence from short-term RCTs suggests differential effects between early and late 8-h TRE [35]. Fourth, self-reported dietary and other data are prone to errors, which may lead to overestimations or underestimations of associations. Fifth, residual confounding is still likely despite the confounding adjustment in primary analyses, robust subgroup results, and thorough sensitivity analyses. However, even if the association between eating duration <8 h and cardiovascular mortality were null when all confounding was perfectly eliminated, the absence of a beneficial association would still not support the long-term application of a short eating window. Sixth, the associations may have suffered from reverse causation. However, excluding mortality events during the first one or two years of follow-up did not materially alter the results. Further excluding events that occurred during longer follow-up could not be performed due to the small size of the eating duration <8 h subgroup. Seventh, the reliability of subgroup estimates is compromised due to the limited sample size in the <8 h eating duration group (~2 % of the whole sample), resulting in wide confidence intervals. This suggests that certain subgroup analyses may be underpowered. Caution should be taken when interpreting these results. Nonetheless, subgroup results were consistent with those of primary analyses. Eighth, the data used are representative only of US adults; therefore, caution should be taken when generalizing the study findings to other countries and regions. Ninth, the observational nature of this study precludes establishing causality. Last but not least, eating duration <8 h in the real world may not be comparable to 8-h TRE for the intentional pursuit of health, warranting cautious interpretation and generalization of the conclusions drawn from this study, in comparison with the 8-h TRE literature.

In conclusion, eating duration <8 h was significantly associated with an increased risk of cardiovascular mortality. Further research is required to understand whether this risk is attributed to the short eating duration itself or residual confounding resulting from its contributing factors.

Contributions

Concept and design: Chen, Xu, Rong, and Zhong.
Acquisition, analysis, or interpretation of data: All authors.
Drafting of the manuscript: Chen, Xu, Rong, and Zhong.
Critical revision of the manuscript for important intellectual content: All authors.
Statistical analysis: Chen, Xu, and Zhong.
Administrative, technical, or material support: Rong and Zhong.

IRB approval

NHANES procedures and protocols were approved by the National Center for Health Statistics Ethics Review Board. Written informed consent was obtained from each participant. Shanghai Jiao Tong University School of Medicine Public Health and Nursing Research Ethics Review Committee approved this study.

Data availability

All data used in this study are publicly available. The National Health and Nutrition Examination Study (NHANES) is a major program of the National Center for Health Statistics, assessing the health and nutritional status of adults and children in the United States. NHANES data can be download at https://www.cdc.gov/nchs/nhanes/about_nhanes.htm. The mortality status and underlying cause of death of adult participants can be found in the public-use linked mortality files, which can be download at https://www.cdc.gov/nchs/data-linkage/mortality-public.htm.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.dsx.2025.103278.

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