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Long-term effect of eating duration on all-cause mortality under different energy intake and physical activity levels

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Abstract

The long-term impact of eating duration on the risk of all-cause mortality remains unclear, with limited exploration of how different levels of energy intake and physical activity might influence this impact. To investigate, 24 484 American adults from the National Health and Nutrition Examination Survey spanning 1999–2018 were included. Eating duration was assessed via 24-h dietary recall, and all-cause mortality data were sourced from the National Death Index. The relationship between eating duration and all-cause mortality was analysed using Cox proportional hazards regression models, restricted cubic splines and stratification analysis with complex weighted designs. The median (IQR) of eating duration for participants was 12·5 (11·0, 14·0) h. In this study, 2896 death events were observed, and the median follow-up time (IQR) was 125 (77, 177) months. After multivariable adjustment, compared with Q1, Q2, Q3 and Q4 had reduced risks of all-cause mortality by 17, 15 and 13 %, respectively. Furthermore, each additional hour of eating duration was correlated with a 2 % decrease in the risk of all-cause mortality. Additionally, a non-linear dose–response relationship was observed between eating duration and the risk of all-cause mortality, showing a *U*-shaped relationship from 8·9 h to 15·3 h (P for non-linearity < 0·05). Interestingly, the non-linear dose–response relationship was observed exclusively among individuals with high energy intake or a lightly active physical activity level. These findings suggest potential health benefits from adjusting eating duration, though further prospective studies are needed for validation.

Keywords: **Eating duration: All-cause mortality: Energy intake: Physical activity: National Health and Nutrition Examination Survey**

Poor dietary habits and a sedentary lifestyle are prominent risk factors for non-communicable diseases, including type 2 diabetes, CVD and cancers[1]. American adults who adhere to national recommendations for a healthy diet and regular physical activity tend to have significantly lower rates of cardiovascular morbidity and mortality compared with those who do not[2,3]. Hence, adopting a healthy lifestyle, such as maintaining healthy dietary habits and engaging in regular physical activity, may effectively decelerate the progression of these non-communicable diseases, curtail mortality and thereby mitigate the socio-economic burden associated with these ailments[4,5].

In the realm of chrononutrition, the intricate interplay among food intake, circadian rhythms and metabolism is a focal point, underscoring the significance of suitable eating schedules for human health[6]. Factors such as meal frequency, regularity, the temporal distribution of energy or nutrient intake and the timing of daily eating periods are considered in chrononutrition[7]. Recently, the influence of eating duration on the development of non-communicable diseases has garnered increasing attention. As a promising method for ameliorating metabolic disorders, time-restricted eating denotes a dietary strategy that restricts eating duration to a specific window of 8–12 h or less within a 24-h period[8,9]. Recent human studies have demonstrated that time-restricted eating may confer diverse metabolic benefits, primarily in body weight loss and adipose tissue reduction[10–12]. Additionally, experiments on *Drosophila* and mice further revealed that reducing feeding duration can have a long-term

Abbreviations: HR, hazard ratios; MET, metabolic equivalents; NHANES, National Health and Nutrition Examination Survey.

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impact on health, such as extending lifespan, especially when combined with restricted energy intake within a specific time frame[13–15]. Nonetheless, the evidence from human studies with extended durations and larger sample sizes remains rare to definitively ascertain the long-term impact of eating duration on all-cause mortality.

Aside from eating duration, the daily level of physical activity is also intricately linked to health. A sedentary lifestyle is regarded as the fourth major risk factor for global mortality, following only hypertension, smoking and elevated blood glucose levels[16]. There is compelling evidence indicating that a sedentary lifestyle could increase susceptibility to numerous chronic ailments and mortality[17,18]. However, consistent results have not been observed across previous studies regarding the association between physical activity and the risk of mortality[19–24]. Multiple studies have demonstrated that participating in vigorous physical activity, regardless of its intensity, duration or type, is associated with a dose-dependent reduction in the risk of mortality[20–22]. However, a 5-year human trial from Norway found no association between high- or moderate-intensity exercise and decreased all-cause mortality among the elderly[23]. Furthermore, the understanding of how eating duration and physical activity jointly contribute to the risk of all-cause mortality is limited, with only a few prospective studies exploring the combined effects of diet and physical activity on this outcome[25–27]. The findings indicated that following either a physically active lifestyle or a healthy diet could reduce the risk of all-cause mortality, but the greatest potential for risk reduction lies in combining both healthy behaviours[27]. Notably, it is still largely undetected whether different degrees of physical activity have a diverse impact on the effect of eating duration on all-cause mortality.

To date, most human studies on the impact of eating duration on health are of relatively limited sample sizes and/or short follow-up periods[8], leaving us with a knowledge gap on the long-term impact of eating duration on the risk of all-cause mortality. In this study, a representative sample of American adults from the National Health and Nutrition Examination Survey (NHANES) was utilised to investigate the hypothesis that eating duration may have long-term effects on the risk of all-cause mortality, as well as the potential impact of different levels of energy intake and physical activity on these effects.

Materials and methods

Study population

This study was conducted utilising data from NHANES, a nationally representative survey conducted using a stratified and multistage sampling method, with detailed descriptions available elsewhere[28]. The survey was approved by the Ethics Review Board of National Center for Health Statistics, and all participants provided written informed consent[29]. Study participants underwent standardised face-to-face interviews, followed by physical examinations and laboratory tests, to obtain data on socio-demographics, dietary intake, health-related inquiries and physiological measurements and laboratory results. The procedures were conducted in a mobile examination centre under the supervision of trained medical personnel.

A total of 101 316 participants were enrolled in this study, including NHANES data from 1999 to 2018. To ensure the stability of the results, exclude those with missing follow-up data on all-cause mortality status (*n* 42 252), missing 24-h dietary recall data (*n* 6662), unreliable total energy consumption (<600 or >5000 kcal/d, *n* 1606)[29–31], unusual eating duration (<1 h or >23 h, *n* 221), currently pregnant (*n* 1473) and missing one or more covariates data (*n* 24 618). The final number of participants included in our study was 24 484 (Fig. 1).

Collection of dietary information

From 1999 to 2002, only one 24-h dietary review interview was held, while a second dietary review interview was held about 3–10 d after the first dietary review interview between 2003 and 2018. During the interview, participants were asked to report the type, quantity and intake time of each food. To ensure the consistency of the data from the two investigation periods, data from the first dietary review interview were adopted in this study[32].

As the primary exposure variable, eating duration was defined as the duration spanning from the initial time of the first meal to the finished time of the last meal[33,34]. To obtain the eating duration, we simply subtracted the finished time of the last food from the initial time of the first food. For instance, if the first food was consumed at 8:00 and the last food was consumed at 20:00, the final eating duration would be 12 h. To maintain uniformity of the analysis, all times were converted into hours (i.e. intake at 7:30 h was recorded as 7·5 h). The study focused on eating duration within a single wake-up period, so we established the beginning of a behavioural day at 5.00 hours.

Main outcomes

The primary outcome variable of this study was all-cause mortality, which was derived from the National Death Index by 31 December 2019. The National Death Index is a centralised database managed by the National Center for Health Statistics and contains death record information from across the country, utilising identifying details such as social security numbers to ensure accuracy. The link between NHANES and the National Death Index was established through a rigorous process that involved probability matching and a comprehensive review of death certificates.

Covariates

A standardised household interview questionnaire was used to collect socio-demographic and lifestyle information and potential covariates including age, sex, race, education, family income: poverty ratio, intake day, energy intake, physical activity, drinking status and smoking status. Race was divided into five groups: Mexican American, other Hispanic, non-Hispanic white, non-Hispanic black and other races. Education was categorised into five groups: less than 9th grade, 9–11th grade, high school grade or general educational development, some college or associate of arts degree and college graduate or above. Family

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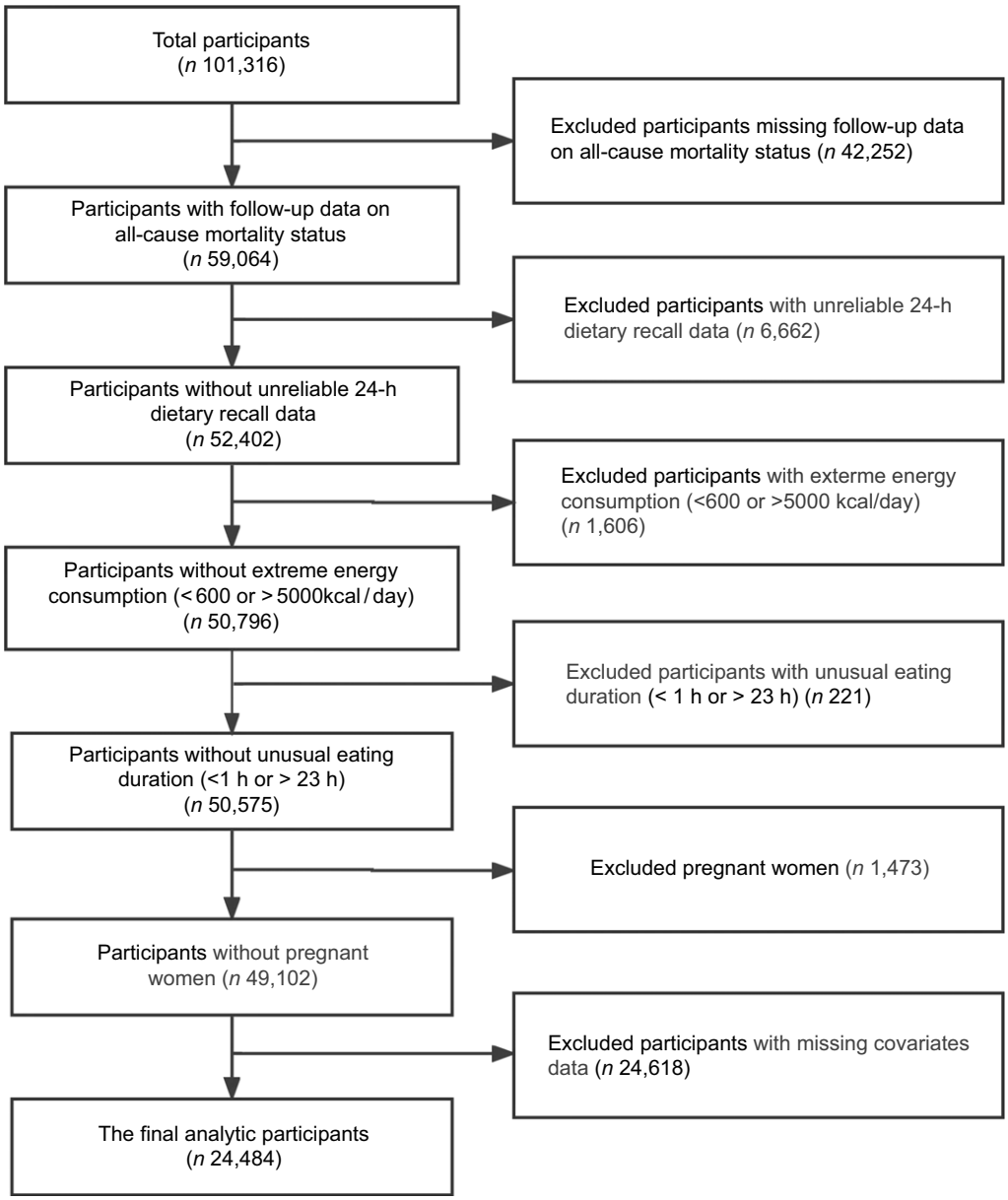

Fig. 1. Flow chart of participants inclusion from the National Health and Nutrition Examination Survey 1999–2018.

income uses the poverty ratio to classify as <1·3, 1·3–2·4, ≥2·4. According to the question: 'Intake day of the week?', intake day is divided into weekends and workdays. According to the calorie level ranges that are estimated based on sex and age, energy intake was classified into three groups: low, reference and high energy intake levels[35]. Physical activity was categorised into three levels based on tertiles: 'lightly active' for less than 600 metabolic equivalents (MET)/week, 'moderately active' for 600–2400 MET/week and 'highly active' for more than 2400 MET/week. According to the question: 'Had at least 12 alcoholic drinks a year?', participants were divided into drinkers and non-drinkers. According to the question: 'Smoked at least 100 cigarettes in life?', participants were classified as smokers and non-smokers. The professionals measured participants' standing body height and body weight at the mobile examination centre. BMI was calculated as body weight (kg) divided by height squared (m^2).

Chronic metabolic diseases such as diabetes, hyperlipidaemia, hypertension and obesity were defined by medicinal history, relative physiological measurements and laboratory data.

Statistical analyses

Data are presented as the medians (IQR) for the continuous variables and unweighted numbers (weighted percentages) for the categorical variables. Continuous baseline characteristics were compared using general linear models, while x^2 tests were employed to assess categorical variables. Cox proportional hazards regression models, accounting for complex weighted

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designs, were utilised to estimate hazard ratios (HR) and their corresponding 95 % CI for all-cause mortality across quartiles of eating duration. Three statistical models were fitted to comprehensively describe the association between eating duration and all-cause mortality. In model 1, we adjusted for age, sex and race. In model 2, we incrementally adjusted for BMI, education level, family income:poverty ratio, drinking status and smoking status. In model 3, we further adjusted for intake day, energy intake and physical activity level. Moreover, a restricted cubic spline regression with five knots was utilised to examine the potential non-linear relationship between eating duration and all-cause mortality based on Cox proportional hazards regression analysis. Stratified analyses were conducted to estimate the relationship between eating duration and all-cause mortality by energy intake and physical activity levels. The statistical significance of interactions was assessed by examining the *P*-values associated with the product terms between eating duration and the stratification variables.

Sensitivity analyses were conducted to evaluate the robustness of the results. First, participants with <2 years of follow-up were excluded to mitigate potential bias from reverse causality. Second, early-morning eaters (eating duration >20 h) and participants with unusual first mealtimes (<6.00 hours or >11.00 hours) were excluded. Third, given the potential influence of other eating times, we further adjusted for the first and last mealtime to account for their potential confounding effects on the association of interest. Finally, we verified the results among participants with any of the following metabolic diseases: diabetes, hyperlipidaemia, hypertension and obesity.

All statistical analyses were conducted using R software (version 4·3·2). A two-tailed *P*-value < 0·05 was considered statistically significant for all analyses.

Results

The median (IQR) eating duration for the final analytic participants was 12·5 (11·0, 14·0) h. Table 1 provides a summary of the baseline characteristics of the final adult participants (*n* 24 484) sorted according to quartiles of eating duration. Participants with a shorter eating duration tended to be younger, female and of Mexican American descent and consumed lower amounts of dietary energy. On the other hand, those with a longer eating duration were predominantly non-Hispanic white Americans, who tended to have higher levels of education and family income and were fond of physical activity, drinking and smoking. Additionally, participants were more likely to have shorter dietary periods on weekends. Notably, no significant differences were observed in BMI and drinking status across the four groups.

The median follow-up time was 125 (77, 177) months, during which 2896 death events were observed. Using Cox proportional hazards regression analyses in complex weighted designs, we found a significant association between longer eating duration and lower all-cause mortality (Table 2). For each additional hour of eating duration, there was a 2 % lower risk of all-cause mortality in the full multivariable-adjusted model (HR 0·98; 95 % CI 0·96, 1·00; $P < 0·05$). The full multivariable-adjusted HR (95 % CI) across quartiles of eating duration were 1·00 (reference), 0·83 (0·73, 0·94), 0·85 (0·74, 0·97) and 0·87 (0·75, 1·00) for all-cause mortality. Figure 2 shows the non-linear dose–response relationship between eating duration and all-cause mortality (*P* for non-linearity < 0·05). Within the eating duration range of 8·9 h to 15·3 h, a distinct *U*-shaped relationship was observed between HR and eating duration, with the lowest HR at 12·6 h. Beyond this range, the HR was likely to exhibit a negative correlation with eating duration. Furthermore, an interaction analysis was conducted to investigate the effect of different levels of energy intake and physical activity on the relationship between eating duration and all-cause mortality. However, the results of the interaction test were not significant for energy intake and physical activity levels ($P_{\text{for interaction}} > 0·05$). To further explore this, stratified analyses were conducted. Figure 3 shows the dose–response relationship of eating duration with all-cause mortality in different energy intake and physical activity groups. The non-linear dose–response relationship can still be found among participants with high energy intake and lightly active physical activity levels (*P* for non-linearity < 0·05). The restricted cubic spline curve in the lightly active physical activity group was similar to that of the integrated population (Fig. 3(d)). When the eating duration was below 12·4 h, the HR was negatively correlated with eating duration. However, when the eating duration was between 12·4 h and 14·1 h, HR was positively correlated with eating duration and then negatively correlated again (Fig. 3(c)). In contrast, the associations between eating duration and the risk of all-cause mortality were not statistically significant among participants with low or reference energy intake levels, as well as moderately or highly active physical activity levels (*P* for non-linearity > 0·05).

In sensitivity analyses, the non-linear dose–response relationship of eating duration with all-cause mortality was more pronounced when excluding participants with a follow-up time of <2 years (online Supplementary Fig. 1). However, the dose–response relationship exhibited a *U*-shaped curve after excluding early-morning eaters (eating duration >20 h) (online Supplementary Fig. 2). The non-linear dose–response relationship remained even after excluding participants with unusual first mealtimes (<6.00 hours or >11.00 hours), although the shape of the curve was slightly altered (online Supplementary Fig. 3). Similar results were also observed when we further adjusted for the first mealtime or the last mealtime (online Supplementary Figs. 4–5). Finally, the non-linear dose–response relationship was more pronounced among a total of 19 659 participants who had any of the metabolic diseases (diabetes, hyperlipidaemia, hypertension and obesity), with the curve remaining largely unchanged compared with the entire population (online Supplementary Fig. 6).

Discussion

To our knowledge, rare prior studies have explored the association between eating duration and all-cause mortality among US adults. Using data from NHANES and the National Death Index, our hypothesis was confirmed by demonstrating a negative association between eating duration and all-cause

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Table 1. Baseline characteristics of participants according to eating duration quartiles in the National Health and Nutrition Examination Survey (1999–2018)†

Characteristic	Quantile of eating duration (x, h)								
	Q1 ($x \leq 11$), N 7101	%	Q2 ($11 < x \leq 12{\cdot}5$), N 5271	%	Q3 ($12{\cdot}5 < x \leq 14$), N 6011	%	Q4 ($x > 14$), N 6101	%	P*
Age, y	40	28, 55	44	31, 58	46	34, 58	47	35, 57	<0·001
BMI, kg/m²	27	24, 32	27	24, 32	27	24, 31	27	24, 31	0·8
Sex, (%)									<0·001
Female	3406	50 %	2604	52 %	2819	48 %	2549	43 %	
Male	3695	50 %	2667	48 %	3192	52 %	3552	57 %	
Race, (%)									<0·001
Mexican American	1255	9·0 %	887	7·2 %	851	5·9 %	765	5·7 %	
Non-Hispanic black	1640	13 %	908	8·4 %	1078	8·7 %	1032	7·7 %	
Non-Hispanic white	3133	66 %	2665	73 %	3188	76 %	3327	77 %	
Other Hispanic	565	5·6 %	404	5·3 %	442	3·7 %	415	3·6 %	
Other race	508	6·3 %	407	5·7 %	452	5·7 %	562	6·0 %	
Education level, (%)									<0·001
Less than 9th grade	690	4·7 %	453	4·0 %	445	3·2 %	358	2·7 %	
9–11th grade	993	11 %	620	8·7 %	713	8·5 %	730	8·9 %	
High school grad/GED	1708	24 %	1215	22 %	1284	21 %	1398	23 %	
Some college or AA degree	2176	34 %	1580	33 %	1820	32 %	1935	33 %	
College graduate or above	1534	26 %	1403	33 %	1749	35 %	1680	33 %	
Family income:poverty ratio, (%)									<0·001
<1·3	2252	25 %	1373	19 %	1501	16 %	1453	15 %	
1·3–2·4	1634	20 %	1176	18 %	1215	17 %	1255	17 %	
≥2·4	3215	56 %	2722	63 %	3295	67 %	3393	67 %	
Intake day, (%)									<0·001
Weekend	3188	35 %	2064	30 %	2226	27 %	1899	22 %	
Workday	3913	65 %	3207	70 %	3785	73 %	4202	78 %	
Energy intake, kcal	1883	1403, 2513	2012	1517, 2632	2111	1606, 2759	2230	1704, 2908	<0·001
Energy intake level, (%)									<0·001
Low energy	3816	51 %	2330	43 %	2420	38 %	2138	34 %	
Reference energy	1858	28 %	1639	32 %	1876	33 %	1932	33 %	
High energy	1427	21 %	1302	25 %	1715	29 %	2031	33 %	
Physical activity, MET	1078	360, 3360	1200	440, 3251	1199	400, 3360	1440	480, 4200	<0·001
Physical activity level, (%)									<0·001
Lightly active	2545	36 %	1758	33 %	2045	34 %	1818	30 %	
Moderately active	2338	33 %	1806	36 %	2045	34 %	1986	34 %	
Very active	2218	31 %	1707	31 %	1921	31 %	2297	36 %	
Drinking status, (%)									0·050
Non-drinker	1665	19 %	1175	18 %	1257	17 %	1213	17 %	
Drinker	5436	81 %	4096	82 %	4754	83 %	4888	83 %	
Smoking status, (%)									<0·001
Non-smoker	3920	56 %	2907	54 %	3157	52 %	2956	49 %	
Smoker	3181	44 %	2364	46 %	2854	48 %	3145	51 %	

†Data are presented as the medians (IQR) for the continuous variables and unweighted numbers (weighted percentages) for the categorical variables.
*P-values were derived from general linear models for continuous variables and x^2 tests for categorical variables.
GED, general educational development; AA, associate of arts; MET, metabolic equivalent.

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Table 2. Hazard ratios (HR) (95 %CI) for all-cause mortality were estimated based on eating duration among participants

Group	Characteristic	HR	95 % CI	*P*
Model 1	Eating duration			
	Q1(x ≤ 11)	–	–	
	Q2(11 < x ≤ 12·5)	0·82	0·72, 0·93	**0·002**
	Q3(12·5 < x ≤ 14)	0·82	0·72, 0·94	**0·005**
	Q4(x > 14)	0·83	0·72, 0·96	**0·012**
	Per-hour increment	0·97	0·95, 0·99	**0·005**
Model 2	Eating duration			
	Q1(x ≤ 11)	–	–	
	Q2(11 < x ≤ 12·5)	0·82	0·72, 0·94	**0·003**
	Q3(12·5 < x ≤ 14)	0·84	0·73, 0·96	**0·011**
	Q4(x > 14)	0·85	0·74, 0·98	**0·026**
	Per-hour increment	0·98	0·96, 1·00	**0·014**
Model 3	Eating duration			
	Q1(x ≤ 11)	–	–	
	Q2(11 < x ≤ 12·5)	0·83	0·73, 0·94	**0·004**
	Q3(12·5 < x ≤ 14)	0·85	0·74, 0·97	**0·017**
	Q4(x > 14)	0·87	0·75, 1·00	**0·049**
	Per-hour increment	0·98	0·96, 1·00	**0·035**

Model 1: adjusted for age, sex and race.
Model 2: adjusted (from model 1) for BMI, education level, family income:poverty ratio, drinking status and smoking status.
Model 3: adjusted (from model 2) for intake day, energy intake and physical activity levels.

mortality. Furthermore, a non-linear dose–response association was observed between eating duration and all-cause mortality, and stratified analysis revealed differences in this association among populations with varying levels of energy intake and physical activity. Interestingly, the non-linear dose–response association between eating duration and all-cause mortality was observed only among participants with high energy intake or lightly active physical activity.

Previous studies have suggested that a shorter eating duration is associated with a lower risk of cardiometabolic diseases[36–38]. There is compelling evidence demonstrating that time-restricted eating could extend the healthy lifespan of animals[13,14], but sparse evidence exists regarding the associations between eating duration and all-cause mortality in humans. This nationally representative study, to the best of our knowledge, is the first to explore the associations between eating duration and all-cause mortality among adults. Therefore, there are no directly comparable studies available to provide corroborative assessments. In this study, Cox proportional hazards regression analyses revealed a negative correlation between eating duration and the risk of all-cause mortality. Some possible reasons have been speculated to explain this finding. First, individuals with a shorter eating duration might lack synchronisation for biological and social times. A vital study reported that the lifespan-extending effect of intermittent time-restricted feeding on *Drosophila* was closely related to enhanced transcription of specific genes regulated by circadian. It was shown that the induction of autophagy specifically during nighttime is essential for the lifespan extension effect of intermittent time-restricted feeding in flies, but this beneficial effect was absent in autophagy specifically induced during daytime[14]. Second, many individuals shortened their eating duration by reducing the amount of food they consumed and shortening intervals between meals; the above two dietary behaviours were reported to be associated with a higher risk of all-cause mortality[39]. Additionally, it was observed that individuals with shorter eating duration consumed relatively less daily energy in our study. Third, in an observational study, a shorter eating duration was often accompanied by a delayed first mealtime (after 8.00 hours), which was associated with an increased risk of all-cause mortality[40,41]. A randomised controlled trial study also showed that early time-restricted eating had greater benefits for metabolic health than midday fasting[42]. Hence, a longer eating duration may be linked to a reduced risk of all-cause mortality in natural conditions, which needs to be confirmed by additional studies. Moreover, a *U*-shaped dose–response relationship between eating duration and the risk of all-cause mortality was observed, with the lowest

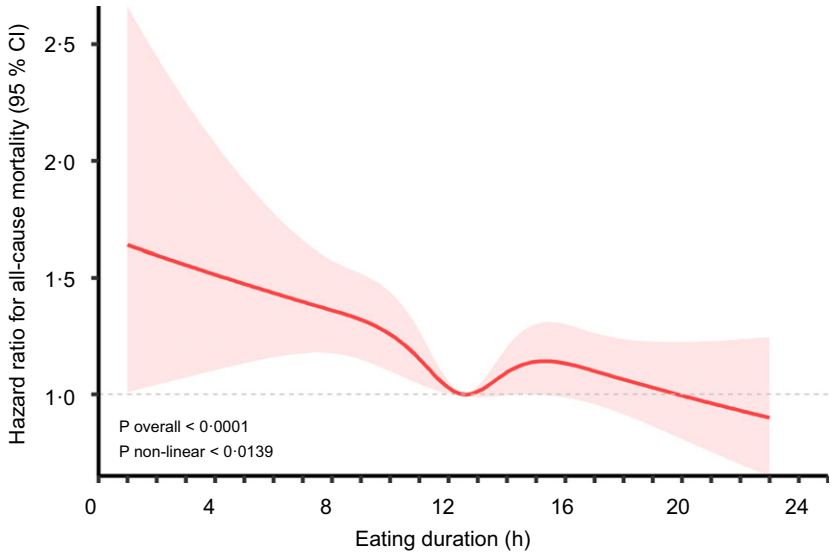

Fig. 2. Associations between eating duration and all-cause mortality among participants. Hazard ratios were adjusted for age, sex, race, BMI, education level, family income:poverty ratio, drinking status, smoking status, intake day, energy intake and physical activity levels. *P* non-linearity < 0·05.

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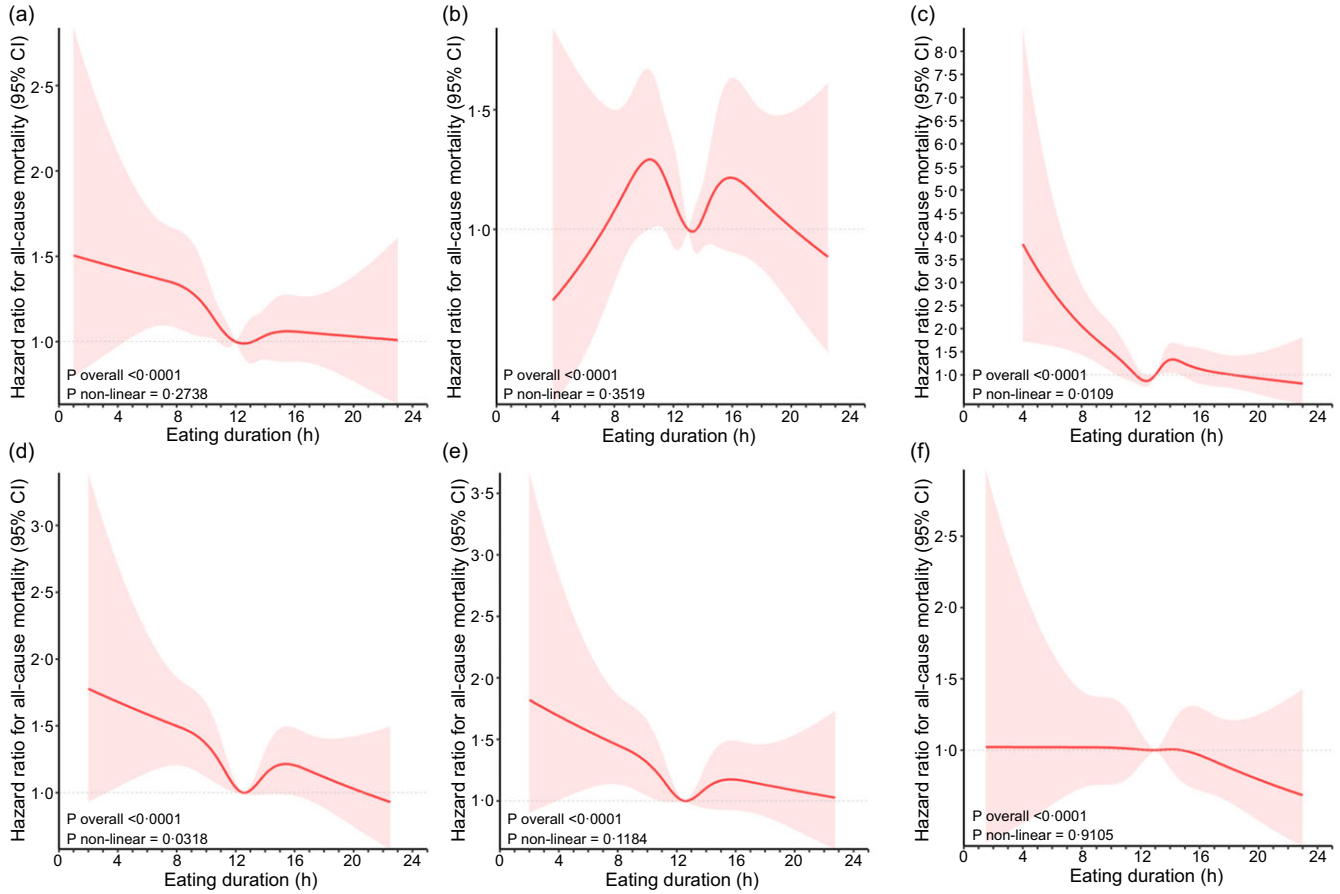

Fig. 3. Associations between eating duration and all-cause mortality in different levels of energy intake and physical activity. Low energy intake (a), reference energy intake (b) and high energy intake (c). Model adjusted for age, sex, race, education level, family income:poverty ratio, drinking status, smoking status, intake day and physical activity levels. Lightly active physical activity (d), moderately active physical activity (e) and highly active physical activity (f). Model adjusted for age, sex, race, education level, family income:poverty ratio, drinking status, smoking status, intake day and energy intake levels. Different levels of energy intake and physical activity.

HR being observed at 12·6 h within the range of 8·9 h to 15·3 h. Previous studies on the dietary regimes of Americans revealed that the typical daily eating duration was between 9 h and 14 h, with only a small percentage extending it to 16 h or longer[43–45]. Therefore, the *U*-shaped relationship observed in our study provided valuable insight into the optimal range of eating duration for minimising the risk of all-cause mortality.

Several cohort studies have shown a correlation between consuming energy intake beyond an individual's needs and an elevated risk of mortality[46,47]. Similarly, our findings demonstrated a significant association between consuming a high-energy diet within a short duration and an elevated risk of all-cause mortality. Furthermore, the non-linear association between eating duration and all-cause mortality was solely found in individuals with a high energy intake. Therefore, our study highlighted the importance of avoiding excessive energy intake within a short eating window. Given that physical activity is widely recognised as one of the most important strategies for individuals to improve their health[48–50], stratified analyses were conducted to assess the correlation between eating duration and all-cause mortality by physical activity level. Ample evidence demonstrates that increased physical activity, both recreational and non-recreational, is linked to a reduced risk of mortality[51,52]. The physical activity guidelines in the USA show that as individuals increase their levels of physical activity, there is a corresponding decrease in the relative risk of all-cause mortality[49]. In this study, the association between shorter eating duration and increased risk of all-cause mortality was only found in individuals engaged in light physical activity. This finding could potentially offer additional evidence supporting the significant and positive impact of regular physical activity in reducing the risk of mortality. However, further studies are required to elucidate the underlying mechanisms.

The main results of our study were verified by sensitivity analyses. However, it is worth noting that after excluding early-morning eaters, a clear *U*-shaped relationship between eating duration and all-cause mortality was observed. This finding further supports our previous notion that there may be an optimal eating duration that may significantly reduce the risk of all-cause mortality. Interestingly, a majority of the participants in our study had one or more metabolic diseases (diabetes, hyperlipidaemia, hypertension and obesity). Upon conducting a detailed analysis of these individuals, we found that the dose–response relationship between eating duration and all-cause mortality followed a similar pattern to that observed in the integrated analytical population. Several studies have indicated

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that metabolic disorders could profoundly influence dietary habits and preferences, necessitating significant changes in eating patterns to effectively manage their health[53,54]. For example, individuals with diabetes will adjust their mealtimes and restrict carbohydrate intake to control postprandial blood glucose levels, resulting in natural changes in their eating duration. Therefore, it is still unclear whether the observed association is due to the changed dietary regime induced by metabolic diseases or whether the changes in eating duration fundamentally contribute to the development of these diseases and subsequently influence all-cause mortality. Thus, more comprehensive investigations and analyses are needed to better understand the relationship between metabolic diseases, eating duration and the risk of all-cause mortality.

This study has several strengths. First, a nationally representative sample of US adults with long-term follow-up data was used in our study, which may facilitate the generalisability of our findings to the broader population of US adults. Second, the employment of a standardised and comprehensive laboratory and physical examination protocol by NHANES successfully minimised measurement errors. Third, a wide range of potential confounders in the multivariable analyses were taken into account in the study, which enhanced the reliability and validity of the results. However, the following limitations should be fully considered when interpreting the results of the study. First, eating duration was calculated based on a single recall over the past 24 h, which could not fully represent the participant's habitual dietary behaviour, and possible effects of random measurement errors were likely present. Second, despite our efforts to control major confounders, we cannot entirely dismiss the possibility of residual confounding from unknown or unmeasured variables in the correlation between eating duration and all-cause mortality. Lastly, it is crucial to recognise that the observational nature of our study limits our ability to establish causality, highlighting the need for future prospective large cohort studies to provide more robust evidence regarding the association between eating duration and all-cause mortality.

Conclusion

A negative association was identified between the risk of all-cause mortality and eating duration, and a non-linear dose–response relationship between eating duration and all-cause mortality was observed exclusively among participants with high energy intake or low levels of physical activity. These findings offer valuable insights for managing daily dietary habits, especially in terms of eating duration. However, further research is necessary to confirm and explore these results more comprehensively.

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F. G. and M. L. conceptualised the study question and designed the research; M. L. and J. H. analysed the data; S. D., J. C. and K. S. provided technical support; F. G. and M. L. wrote the paper. All authors contributed to the interpretation of the results, reviewed and approved the final manuscript.

The authors declare no conflict of interest.

Supplementary material

For supplementary material/s referred to in this article, please visit https://doi.org/10.1017/S0007114524001739.

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