



The associations of long working hours and unhealthy diet with cardiometabolic outcomes and mortality in US workers

Xiang Li^a, Jian Li^{a,b,c}, Xuyuehe Ren^a, Tong Xia^a, Onyebuchi A. Arah^{a,d,e,f}, Liwei Chen^{a,*}

^a Department of Epidemiology, Fielding School of Public Health, University of California, Los Angeles (UCLA), Los Angeles, CA, USA

^b Department of Environmental Health Sciences, Fielding School of Public Health, University of California, Los Angeles (UCLA), Los Angeles, CA, USA

^c School of Nursing, University of California, Los Angeles (UCLA), Los Angeles, CA, USA

^d Department of Statistics & Data Science, UCLA, Los Angeles, CA, USA

^e Practical Causal Inference Lab, UCLA, Los Angeles, CA, USA

^f Research Unit for Epidemiology, Department of Public Health, Aarhus University, Aarhus, Denmark

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ABSTRACT

Objectives: To examine independent and joint associations of long working hours (LWH) and EAT-Lancet diet with cardiometabolic outcomes and mortality in US workers.

Methods: This prospective cohort included US workers from the National Health and Nutrition Examination Survey, with cross-sectional baseline data from 1999 to March 2020. A subsample of participants from 1999 to 2018 was linked to the National Death Index, with mortality follow-up through December 2019. The independent and joint associations of LWH (≥ 55 vs. < 55 h/week) and EAT-Lancet diet scores (low vs. high) with cardiometabolic outcomes and mortality were estimated using multivariable logistic and Cox proportional hazards models, respectively.

Results: LWH was associated with higher odds of obesity (OR = 1.20; 95%CI = 1.07, 1.34) among all workers and higher CVD mortality among workers with high CVD risk at baseline (HR = 1.64, 95%CI = 0.79, 3.12). Low diet scores were associated with higher odds of obesity (OR = 1.34, 95%CI = 1.21, 1.42) and diabetes (OR = 1.33, 95%CI = 1.01, 1.76) among all workers. Working hours and diet scores were jointly associated with obesity and CVD mortality, indicating by the relative excess risk due to interaction greater than zero among all workers.

Conclusions: LWH and unhealthy diet are independent risk factors and may interact to exacerbate adverse cardiometabolic health outcomes.

1. Introduction

Cardiovascular disease (CVD) remains the leading cause of death in the United States (US), with rising healthcare expenses and a growing prevalence, even among the younger working-age population (Roth et al., 2020; Nat. Rev. Cardiol., 2018). A recent meta-analysis of 26 prospective cohorts and 11 case-control studies by the World Health Organization (WHO) and the International Labor Organization (ILO) found that long working hours (i.e., working ≥ 55 h/week, LWH) was associated with a 13% higher risk of ischemic heart disease (IHD) and a 17% higher risk of death from IHD compared to normal working time (i.

e., 35–40 h/week) (Li et al., 2020). Another meta-analysis showed that LWH was also linked to a higher stroke risk (Descatha et al., 2020). However, the majority of studies were conducted in European or Asia-Pacific countries (Ervasti et al., 2021; Wong et al., 2019). In 2022, the average working hours in the US were 36.4 h/week, higher than in Europe and Japan (Employment - Hours worked - OECD Data, 2024). While studies in the US have investigated the association between LWH and outcomes like hypertension and weight gain, only one study in the older workers has examined mortality (Yang et al., 2006; Li et al., 2021; Virtanen et al., 2020; Mercan et al., 2022), and the evidence specifically focused on CVD mortality remains limited.

* Corresponding author at: Department of Epidemiology, Fielding School of Public Health, University of California Los Angeles, Los Angeles, 650 Charles E. Young Drive South, CA 90095, USA.

E-mail addresses: lix@ucla.edu (X. Li), jianli2019@ucla.edu (J. Li), roxyren22@ucla.edu (X. Ren), xiatong@g.ucla.edu (T. Xia), arah@ucla.edu (O.A. Arah), cliwei86@ucla.edu (L. Chen).

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Healthy dietary patterns have well-documented benefits in reducing CVD risk in various populations. Most workers have at least one daily meal at the workplace, where unhealthy drinks and food options are widely provided (Ey et al., 2020). Thus, individuals with LWH are likely exposed to unhealthy foods and dietary patterns. In 2019, the EAT-Lancet Commission of Food, Planet Health introduced the EAT-Lancet diet, characterized as rich in plant-based products as an environment friendly and sustainable healthy dietary pattern (Lancet, 2019). Adherence to the EAT-Lancet diet was associated with lower risks of obesity, type 2 diabetes, and all-cause mortality in European populations (Langmann et al., 2023a; Langmann et al., 2023b; Stubbendorff et al., 2022). However, studies investigating the cardiometabolic health effects of the EAT-Lancet diet in the US, particularly in working populations, are lacking. Differences in diet, lifestyle, and socioeconomic factors between the US and Europe highlight the need for targeted research.

Taken together, LWH is a major adverse working condition in the US, and poor diets may interact with working hours. Therefore, we aimed to examine the independent and joint associations of LWH and EAT-Lancet diet with cardiometabolic outcomes and mortality in a US representative working population.

2. Methods

2.1. Study design and participants

The National Health and Nutrition Examination Survey (NHANES) is a series of cross-sectional data with a complex, multi-stage, probability sampling design to obtain a nationally representative sample of the civilian, noninstitutionalized US population. Participants were linked to death certificate records from the National Death Index for prospective follow-up until December 31, 2019. This study analyzed NHANES data from the 1999–2016 and 2017–2020 March (pre-pandemic) cycles (cross-sectional) and their mortality data (prospective).

Among 107,519 eligible participants with paid jobs, those who reported pregnancy ($N = 1\,784$), had history of CVDs (coronary heart disease, myocardial infarction, congestive heart failure, heart attack, angina/angina pectoris, or stroke) ($N = 6802$) or cancers ($N = 4144$), or without reporting working hours ($N = 63,976$) were excluded, leaving a sample of 30,813 workers. Further, workers with missing dietary data ($N = 3329$), extreme total energy intake (< 500 or > 8000 kcal/day) ($N = 200$), or missing survey weights ($N = 3$) were excluded. Based on each of the cardiometabolic outcomes, we obtained subsamples of obesity ($N = 27,114$), hypertension ($N = 26,409$), diabetes ($N = 27,281$), Framingham CVD risk score (FRS) ($N = 17,325$) as illustrated in S Fig. 1.

All NHANES protocols were approved by the National Center for Health Statistics's Research Ethics Review Board. This study followed the Strengthening the Reporting of Observational Studies in Epidemiology reporting guidelines.

2.2. Mortality ascertainment

CVD mortality was the primary mortality outcome, available only until December 31, 2014, as deaths from cerebrovascular diseases were not followed afterward. Secondary outcomes, including all-cause and heart disease mortality, were available for participants from NHANES 1999–2018, followed until December 31, 2019. In addition to the previous exclusion criteria, participants younger than 18 years (no records of death) or not eligible for the mortality linkage were further excluded, leaving a subsample of 19,682 for CVD mortality outcome and 24,630 for all-cause mortality and heart disease mortality outcomes (S Fig. 1b). Cause-specific mortality was identified using the International Classification of Diseases (ICD-10) codes, with heart disease mortality defined by I00–I09, I11, I13, I20–I51, and CVD mortality further including I60–I69.

2.3. Cardiometabolic outcomes assessment

Cardiometabolic outcomes were based on in-person health examinations at the Mobile Examination Center (MEC), laboratory results, or self-reported conditions, including obesity, diabetes, hypertension, and FRS.

Hypertension was determined if previously diagnosed by a physician, or systolic blood pressure (SBP) ≥ 130 mmHg and/or diastolic blood pressure (DBP) ≥ 80 mmHg, or use of any blood pressure-lowering treatment (Whelton et al., 2022). All available blood pressure readings were utilized to calculate the mean blood pressure for each participant (Trends in the Prevalence, Awareness, Treatment, and Control of Hypertension Among Young Adults in the United States, 1999 to 2014 | Hypertension, 2023).

Weight and height were directly measured at the MEC and were used to calculate body mass index (BMI) (kg/m^2). Obesity was defined as a BMI of 30 kg/m^2 or higher. A further checking using lower BMI cutoffs ($\geq 27.5 \text{ kg/m}^2$) (Li et al., 2023) for participants self-reported as non-Hispanic Asian (year 2011 onward) showed negligible differences in obesity prevalence (0.4% higher, data not shown). Thus, the universal cutoff of $\geq 30 \text{ kg/m}^2$ was retained for all participants in this study.

Diabetes was classified as diagnosed (i.e., self-reported previous diagnosis or use of any glucose-lowering medications) and undiagnosed (i.e., glycohemoglobin $\geq 6.5\%$, or fasting glucose $\geq 7 \text{ mmol/L}$, or 2-h oral glucose tolerance test $\geq 11.1 \text{ mmol/L}$) (American Diabetes Association, 2020).

FRS were calculated separately for males and females based on age, total cholesterol, high-density lipoprotein cholesterol, SBP, treatment for hypertension, diabetes, and smoking status (Expert Panel on Detection E and Treatment of High Blood Cholesterol in Adults, 2001). Then, the scores were dichotomized as low CVD risk (score $\leq 20\%$) versus high CVD risk (score $> 20\%$) as commonly applied in primary care (General Cardiovascular Risk Profile for Use in Primary Care | Circulation, 2023).

2.4. Working hours

Working hours were self-reported and obtained from the question, “How many hours did you work at all jobs or businesses last week”.

2.5. EAT-Lancet diet score

Assessment of dietary intake in NHANES has been described previously (Wang et al., 2023). Briefly, a validated 24-h dietary recall was applied during the in-person interview from year 1999 through 2002. Since the year 2003, a second dietary interview was added via telephone 3–10 days after the initial in-person interview. Individual food intakes were averaged based on two-day dietary data for the year 2003 and after.

Adherence to the EAT-Lancet was evaluated by calculating an EAT-Lancet diet score based on the consumption of 14 components, including seven emphasized components (vegetables, fruits, unsaturated oils, legumes, nuts, whole grains, fish) and seven limited components (beef and lamb, pork, poultry, eggs, dairy, potatoes, added sugar) (Stubbendorff et al., 2022). A total score was calculated by summing the 14 component scores, ranging from 0 (worst) to 42 (best), with higher scores indicating higher diet quality.

2.6. Covariates

Sociodemographic information in NHANES was collected during in-person interviews, including age, sex, race/ethnicity, education, marital status, and ratio of family income to poverty. Smoking status was self-reported based on lifetime and current cigarette use. Alcohol drinking status was defined according to the self-reported number of drinks in a lifetime and in a week (Drinking Levels Defined, 2024). Total physical activity was measured using the Global Physical Activity Questionnaire

and leisure time physical activity was defined as vigorous and moderate recreational activity. Metabolic equivalent (MET) minutes were calculated to quantify the weekly energy expenditure for leisure time physical activity (Doerrmann et al., 2020).

2.7. Statistical analysis

To account for the complex survey design, sampling weights were applied to all analyses per NHANES guidelines (National Center for Health Statistics, 2020). Weighted means (standard errors (SE)) were estimated for continuous variables, and counts (weighted percentages) were calculated for categorical variables.

In primary analysis, LWH (i.e., ≥ 55 h/week) was defined according to WHO report (Li et al., 2020), and was dichotomized into two categories (yes vs. no). The EAT-Lancet diet score was grouped as high vs. low using a median cut-off of 24. Multivariable logistic regression models were used to assess the cross-sectional associations with cardiometabolic outcomes, adjusting for age (years), sex, race/ethnicity (Mexican American, other Hispanic, non-Hispanic white, non-Hispanic black, other race including multi-racial), education (less than high school, some high school, high school or more), poverty income ratio (<1 or ≥ 1), marital status (married/living with a partner, widowed/divorced/separated, never married), smoking status (non-smoker, former smoker, current smoker), alcohol drinking (non-drinker, light-to-moderate drinker, heavy drinker), leisure time physical activity ($<$ or ≥ 500 MET min/week), and total energy intake (kcal/day). Multivariable Cox proportional hazards models were used to assess the prospective associations with mortality outcomes, adjusting for the same covariates. Stratified analyses for the association with CVD mortality were performed by baseline FRS, as previous studies have shown that the impact of LWH may be more greater in individuals with preexisting CVDs (Trudel et al., 2021). Models assessing LWH were adjusted for diet scores, and models assessing diet scores were adjusted for working hours, both in original continuous form to optimize confounding control and statistical precision. Joint associations of LWH (no vs. yes) and EAT-Lancet diet (high vs. low) were analyzed using four categories: (i) no + high (reference), (ii) no + low, (iii) yes + high, (iv) yes + low.

Multiplicative interaction was evaluated by adding an exposure interaction term in the model. The relative excess risk due to interaction (RERI) was used to examine additive interaction, calculated as $RERI_{OR} = OR_{11} - OR_{10} - OR_{01} + 1$ (or HRs), where subscripts 1/0 indicate exposure presence/absence: OR_{11} (both present), OR_{10} and OR_{01} (each alone). $RERI > 0$ indicates a synergistic interaction, 0 indicates additivity, and < 0 indicates antagonism (Andersson et al., 2005).

Multiple imputation was carried out to impute covariates with missing in the primary analyses. Several sensitivity analyses were performed. First, participants working <35 h/week were excluded because they were assumed to have health issues with reduced work ability for full-time jobs and higher risk of sequent health outcomes. Second, self-reported occupations, which were only available before NHANES 2015, were further adjusted in the multivariable models. Third, missingness in covariates was coded as a separate category to repeat analyses. Lastly, repeated regression analyses were conducted to further adjust for or excluding shift work.

Multiple imputations and forest plots were conducted in R (version 4.2.2), and all other analyses were performed using SAS 9.4 (SAS Institute Inc., Cary NC).

3. Results

3.1. Baseline characteristics

Among all participants, the weighted mean (SE) age was 39.5 (0.2) years, 55% were male, 87% had high school or more education, and 89% had income above the poverty level. In total, 14% worked long hours (i.e., ≥ 55 h/week). LWH workers were more likely to be male, married or

living with partners, smokers, light-to-moderate drinkers, higher educated, and had higher incomes (Table 1). Similar characteristics were observed in the CVD mortality subsample.

Overall, the weighted mean (SE) of the EAT-Lancet score was 24 (0.06), ranging from 8 to 42. Workers with low diet score were more likely to be younger, male, never married, smokers, heavy drinkers, less educated, less active and had lower income. Among all participants, 6.8% were in the LWH + low diet score group, 8.1% in the LWH + high diet score group, 49.8% in the working hours <55 h/wk + high diet score group, and 35.3% in the working hours <55 h/wk. + low diet

Table 1

Characteristics of US workers by working hours and EAT-Lancet diet score, the National Health and Nutrition Examination Survey 1999–2020 March ($N = 25,717$).

		Long working hours		EAT-Lancet diet score	
		No (< 55 h/week)	Yes (≥ 55 h/week)	High (≥ 24)	Low (< 24)
Baseline characteristics, N (%)	Overall	N = 23,672	N = 3609	N = 15,924	N = 11,357
Age (years), mean (SE)	39.5 (0.2)	39.3 (0.2)	40.7 (0.3)	40.5 (0.2)	38.6 (0.2)
Male	14,943 (55.0)	12,287 (51.6)	2656 (74.4)	8474 (53.2)	6469 (57.5)
<i>Race and ethnicity</i>					
Mexican	5489 (9.5)	4792 (9.5)	697 (9.4)	3463 (10.4)	2026 (8.3)
Americans	2390 (6.0)	2103 (6.2)	287 (5.2)	1501 (6.3)	889 (5.7)
Other Hispanics	10,683 (66.1)	9191 (66.9)	1492 (67.3)	5935 (65.1)	4748 (67.5)
Non-Hispanic white	5592 (10.9)	5223 (11.0)	769 (10.3)	3118 (9.7)	2874 (12.6)
Non-Hispanic black	2727 (7.5)	2363 (7.4)	364 (7.8)	1907 (8.6)	820 (5.9)
Other race					
<i>Education</i>					
Less than high school	2190 (3.7)	1942 (3.7)	248 (3.2)	1451 (4.0)	739 (3.1)
Some high school	3278 (8.7)	2876 (8.8)	402 (8.1)	1711 (7.4)	11,567 (10.4)
High school or more	21,813 (87.6)	18,854 (87.5)	2959 (88.7)	12,762 (88.5)	9051 (86.5)
<i>Marital status</i>					
Married/living with partner	15,706 (61.3)	13,326 (60.4)	2380 (66.8)	9506 (62.3)	6200 (59.9)
Widowed/divorced/separated	3685 (13.0)	3166 (12.8)	519 (14.1)	2180 (13.0)	1505 (13.1)
Never married	7890 (25.7)	7180 (26.8)	710 (19.1)	4238 (24.7)	3652 (27.1)
<i>Poverty income ratio</i>					
≥ 1.0	22,784 (89.2)	19,581 (88.6)	3203 (92.1)	13,403 (89.6)	9381 (88.5)
<i>Smoking status</i>					
Non-smoker	16,242 (58.1)	14,178 (58.8)	2064 (54.7)	9955 (61.1)	6287 (54.1)
Former smoker	5297 (21.0)	4526 (20.5)	771 (23.8)	3218 (21.8)	2079 (19.8)
Current smoker	5742 (20.9)	4968 (20.8)	774 (21.6)	2751 (17.1)	2991 (26.1)
<i>Alcohol drinking</i>					
Non-drinker	5739 (17.6)	5086 (18.0)	653 (15.5)	3588 (18.5)	2151 (16.4)
Light to moderate	10,217 (38.5)	8693 (37.6)	1524 (43.7)	6210 (40.1)	4007 (36.3)
Heavy	11,325 (43.8)	9893 (44.4)	1432 (40.8)	6126 (41.3)	5199 (47.3)
<i>Leisure time physical activity</i>					
≥ 500 MET min/week	13,377 (52.8)	11,638 (53.1)	1739 (51.5)	8056 (55.2)	5321 (49.5)

Mean, SE, and percentages are survey weighted.
MET, metabolic equivalent.

score group.

3.2. Cross-sectional associations of working hours, EAT-lancet score and cardiometabolic outcomes

Compared to those working <55 h/wk., LWH was associated with higher odds of obesity in both crude (OR = 1.20, 95%CI = 1.08, 1.34) and adjusted models (OR = 1.20, 95%CI = 1.07, 1.34). LWH was also associated with higher odds of hypertension only in crude model (OR = 1.20 (1.08, 1.34), but not the adjusted model (OR = 1.01, 95%CI = 0.90, 1.13). No associations were found between LWH and diabetes, or FRS (Table 2).

Compared to the high diet score group, a low EAT-Lancet diet score was associated with a higher odds of obesity (crude: OR = 1.29, 95%CI = 1.20, 1.39; adjusted OR = 1.34, 95%CI = 1.21, 1.42) and diabetes (crude OR = 1.17, 95%CI = 1.04, 1.32; adjusted OR = 1.34, 95%CI = 1.17, 1.53) in both crude and adjusted models, but only crude model for FRS (OR = 1.20, 95%CI = 1.03, 1.40). A low diet score was also associated with higher odds of hypertension in adjusted model (adjusted OR

= 1.12, 95%CI = 1.03, 1.20) and borderline statistically significant in crude model (crude OR = 1.07, 95%CI = 0.99, 1.15).

The joint associations of working hours and the EAT-Lancet diet score with cardiometabolic outcomes are presented in Fig. 1(A–D). Compared to the group of working <55 h/week. + high diet score, participants in the group of LWH + low diet score had increased odds of obesity (adjusted OR = 1.61, 95%CI = 1.36, 1.92), diabetes (adjusted OR = 1.33, 95%CI = 1.01, 1.76), and hypertension (adjusted OR = 1.16, 95%CI = 0.96, 1.39), adjusting for confounders. No association was observed for FRS. The RERI for obesity outcome was greater than 0 (RERI = 0.15, 95%CI = 0.10, 0.21), indicating a synergistic interaction between LWH and low diet scores. The RERIs for diabetes (RERI = -0.27, 95%CI = -0.56, 0.02), hypertension (RERI = 0.15, 95%CI = 0.10, 0.26), and FRS (RERI = -0.14, 95%CI = -0.28, 0.00) were more uncertain and included positive, zero and negative interaction between LWH and low diet scores.

3.3. Prospective associations of working hours, EAT-lancet score, and mortality

The median (interquartile) follow-up time for all-cause and heart disease mortality was 10.3 (5.5–15.2) years, and 7.4 (3.6–11.5) years for CVD mortality. During the follow-up period, 1060 all-cause deaths, 221 heart disease deaths, and 257 CVD deaths were documented. The overall mortality rates were 43.0 per 1000 for all-cause, 9.0 per 1000 for heart disease, and 13.1 per 1000 for CVD mortality.

No association was observed between LWH and CVD mortality in crude (HR = 1.02, 95%CI = 0.63, 1.63) and adjusted models (adjusted HR = 0.97, 95%CI = 0.60, 1.56) among all workers. In workers with high CVD risk (i.e., high FRS scores) at baseline, LWH was associated with a 64% higher hazard of CVD mortality compared to those working <55 h/week. in the adjusted model (adjusted HR = 1.64, 95%CI = 0.79, 3.12), although it was not statistically significant. No association was found between LWH and CVD mortality in workers with low FRS scores (adjusted HR = 0.90, 95%CI = 0.44, 1.83). No associations were found for LWH with all-cause mortality (adjusted HR = 0.93, 95%CI = 0.69, 1.25), or heart disease mortality (adjusted HR = 0.86, 95%CI = 0.51, 1.46) among all workers (Table 3).

A low Eat-Lancet diet score was associated with higher hazard of CVD mortality in both crude (HR = 1.34, 95%CI = 0.91, 1.97) and adjusted models (adjusted HR = 1.45, 95%CI = 0.96, 2.19) among all workers, whereas increased CVD mortality risk only among participants with low FRS scores (adjusted HR = 1.98, 95%CI = 1.10, 3.57). Additionally, a low diet score was associated with higher hazard of all-cause mortality (adjusted HR = 1.25, 95%CI = 1.05, 1.50) and heart disease mortality (adjusted HR = 1.62, 95%CI = 0.98, 2.69) among all workers, after adjusting for confounders (Table 3).

The joint associations on mortality are shown in Fig. 1(E–G). The LWH + low diet score group had higher hazards for CVD mortality (adjusted HR = 1.73, 95% CI = 0.60, 3.09), heart disease mortality (adjusted HR = 1.50, 95% CI = 0.65, 3.48), and all-cause mortality (adjusted HR = 1.13, 95% CI = 0.60, 1.73) compared to no LWH + high diet score group. The RERI for CVD mortality indicated a synergistic interaction (RERI = 0.21, 95% CI = 0.03, 0.39), while RERIs for all-cause (RERI = -0.08, 95% CI = -0.16, 0.01) and heart disease mortality (RERI = 0.23, 95% CI = -0.72, 1.18) suggested an additive interaction.

3.4. Sensitivity analyses

Results for several sensitivity analyses are presented in S Tables 1–4. Overall, the results remained unchanged when 1) excluding participants working <35 h/week, 2) including occupation types in the multivariable models, and 3) coding missing values as a separate category, 4) adjusting shift work status or excluding shift worker.

Table 2

Independent cross-sectional associations between working hours, EAT-Lancet diet score, and cardiometabolic outcomes in US workers, the National Health and Nutrition Examination Survey 1999–2020 March.

	Long working hours		EAT-Lancet diet score	
	No (< 55 h/week)	Yes (≥ 55 h/week)	High (≥ 24)	Low (< 24)
	OR (95%CI)	OR (95%CI)	OR (95%CI)	OR (95%CI)
Obesity (N = 27,114)				
No. cases / Total number	7762 / 23,529	1348 / 3585	4970 / 15,821	4140 / 11,293
Crude model	1.00	1.20 (1.08, 1.34)	1.00	1.29 (1.20, 1.39)
Fully adjusted model	1.00	1.20 (1.07, 1.34)	1.00	1.31 (1.21, 1.42)
Diabetes (N = 27,281)				
No. cases / Total number	2127 / 23,672	367 / 3609	1484 / 15,924	1010 / 11,357
Crude model	1.00	1.12 (0.94, 1.34)	1.00	1.17 (1.04, 1.32)
Fully adjusted model	1.00	1.08 (0.90, 1.30)	1.00	1.34 (1.17, 1.53)
Hypertension (N = 26,409)				
No. cases / Total number	7219 / 22,922	1261 / 3487	4865 / 15,403	3615 / 11,006
Crude model	1.00	1.20 (1.08, 1.34)	1.00	1.07 (0.99, 1.15)
Fully adjusted model	1.00	1.01 (0.90, 1.13)	1.00	1.12 (1.03, 1.20)
Framingham 10-year CVD risk: High (N = 17,325)				
No. cases / Total number	1651 / 14,687	275 / 2638	1103 / 10,520	823 / 6805
Crude model	1.00	0.99 (0.81, 1.21)	1.00	1.20 (1.03, 1.40)
Fully adjusted model ^a	1.00	0.98 (0.80, 1.19)	1.00	1.16 (0.99, 1.36)

CVD, cardiovascular disease.

Fully adjusted model: adjusted for age, sex, race/ethnicity, marital status, poverty income ratio, education level, smoking status, alcohol drinking status, leisure time physical activity, total energy intake Diet scores were further adjusted in models assessing long working hours, and working hours were further adjusted in models when assessing diet scores. Covariates (diet score/working hours) were retained in their original continuous form when adjusted for in models to maximize confounding control, while exposures were dichotomized for primary analyses.

^a Adjusted for race/ethnicity, marital status, poverty income ratio, educational level, alcohol drinking status, leisure time physical activity, total energy intake. Diet scores were further adjusted in models assessing long working hours, and long working hours was further adjusted in models when assessing diet scores.

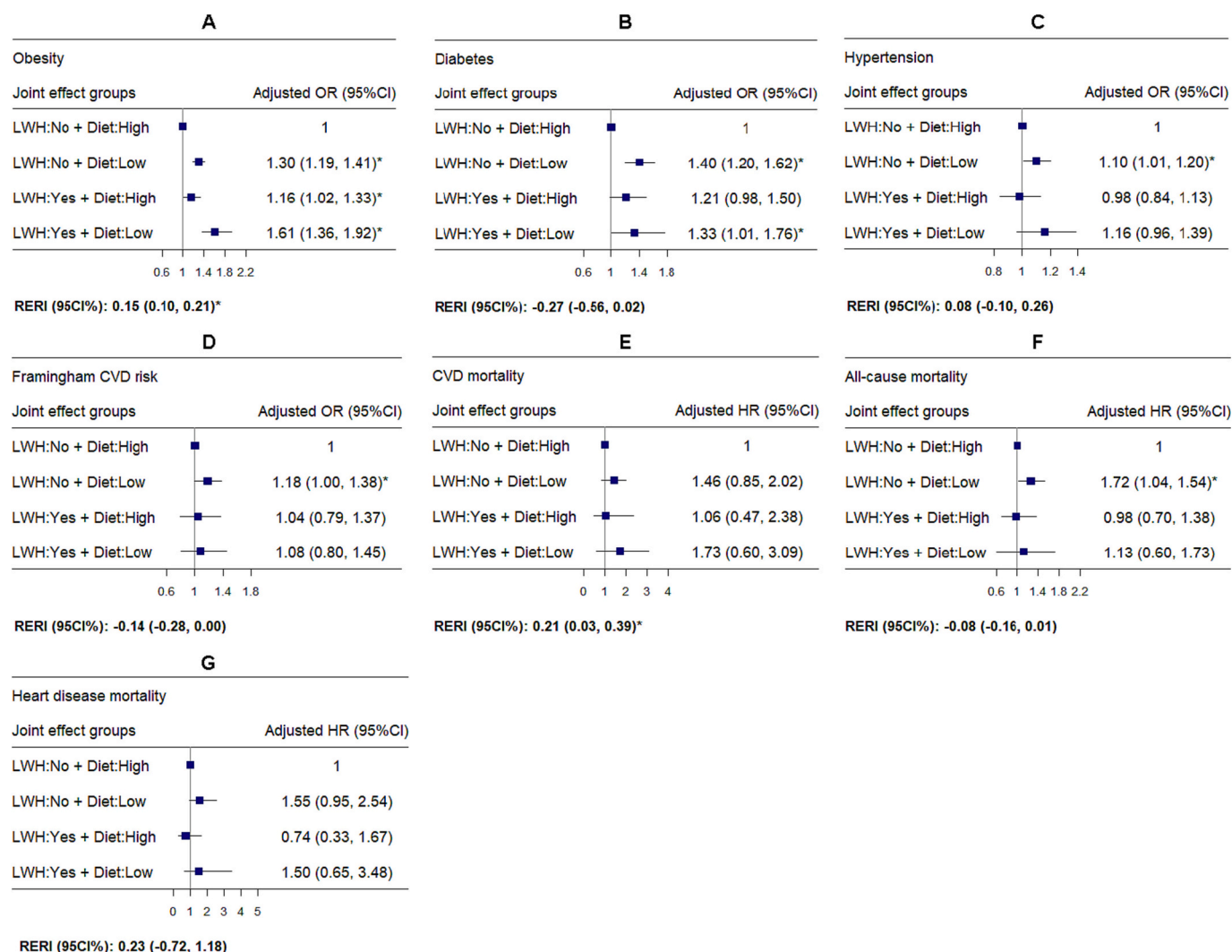


Fig. 1. Joint associations of working hours and EAT-Lancet diet score with cardiometabolic outcomes and mortality outcomes in US workers, the National Health and Nutrition Examination Survey 1999–2020.

Abbreviation: LWH: No, working hours <55 h/week.; LWH: Yes, working hours ≥55 h/week.; Diet: High, EAT-Lancet diet score ≥ 24; Diet: Low, EAT-Lancet diet score < 24; OR, odds ratio; HR, hazard ratio; RERI, relative excess risk due to interaction.

Heart disease mortality, defined by the International Classification of Diseases codes I00–I09, I11, I13, I20–I51.

CVD mortality, cardiovascular disease mortality, defined by the International Classification of Diseases codes I00–I09, I11, I13, I20–I51, and I60–I69.

Results were weighted and adjusted for age, sex, race/ethnicity, marital status, poverty income ratio, education level, smoking status, alcohol drinking status, leisure-time physical activity, and total energy intake.

*, P-value <0.05.

4. Discussion

In this large study of nationally representative samples of US workers, LWH and diet quality were independently and jointly associated with cardiometabolic outcomes and mortality. LWH and low diet quality exhibited a super-additive interaction on obesity and elevated CVD mortality risk.

Our findings on a positive association between LWH and obesity align with previous research. A meta-analysis of 10 cross-sectional studies reported a 13% increased odds of obesity (pooled OR = 1.13, 95%CI = 1.01, 1.26) associated with LWH (Li et al., 2021). Another meta-analysis of 19 cohort studies supported the association with being overweight or obese (RR = 1.17, 95%CI = 1.08, 1.27) (Virtanen et al., 2020). Our estimates remained unchanged after lifestyle and occupational adjustments, suggesting alternative explanatory pathways. Some studies suggested that psychological stress and glucocorticoid release through the hypothalamic pituitary adrenal axis may contribute to LWH-induced obesity by inhibiting fat breakdown and muscle growth

(Chrousos, 2000). Unfortunately, there is no such data to confirm in the current study.

Some of our findings align with the literature on the relationship between LWH and cardiometabolic outcomes, while others differ. Our results indicating no association between LWH and hypertension was consistent with a meta-analysis of twelve cohorts and seven cross-sectional studies (pooled OR = 1.09, 95%CI = 0.88, 1.35) (J. Epidemiol. Community Health, 2024). While LWH was not associated with diabetes in our study, it was linked to higher diabetes risk in individuals with low socioeconomic status (RR = 1.29, 95%CI = 1.06–1.57) (Kivimäki et al., 2015). LWH was not associated with FRS in our analysis, differing from the positive association among Korean workers (OR = 1.42, 95%CI = 1.06, 1.89) (Kang et al., 2014), possibly due to the longer working hours (over 61 h/week) in the Korean study. These discrepancies may stem from variations in study design, outcome measurement, population characteristics, and confounder adjustment.

In this study, we did not observe the association between LWH and CVD mortality among all participants. However, there was suggestive

Table 3

Independent associations between working hours, EAT-Lancet diet score, and all-cause mortality, heart disease mortality, and cardiovascular disease mortality in US workers, overall and by baseline cardiovascular risk¹, the National Health and Nutrition Examination Survey 1999–2019.

		Long working hours		EAT-Lancet diet score	
		No (< 55 h/week)	Yes (≥ 55 h/week)	High (≥ 24)	Low (< 24)
		HR (95% CI)	HR (95% CI)	HR (95% CI)	HR (95% CI)
CVD mortality					
All participants	No. CVD death/Total Mortality (1/1000)	168 / 10,871	28 / 2008	87 / 6716	107 / 6171
		15.5	13.9	13.0	17.3
	Crude model	1.00	1.02 (0.63, 1.63)	1.00	1.34 (0.91, 1.97)
	Fully adjusted model	1.00	0.97 (0.60, 1.56)	1.00	1.45 (0.96, 2.19)
Low CVD risk at baseline	No. CVD death/Total Mortality (1/1000)	100 / 9694	14 / 1811	46 / 5913	68 / 5592
		10.3	7.7	7.8	12.2
	Crude model	1.00	0.99 (0.49, 2.02)	1.00	1.93 (1.09, 3.39)
	Fully adjusted model ^a	1.00	0.90 (0.44, 1.83)	1.00	1.98 (1.10, 3.57)
High CVD risk at baseline	No. CVD death/Total Mortality (1/1000)	68 / 1177	12 / 197	41 / 803	39 / 579
		57.8	60.9	51.1	67.4
	Crude model	1.00	1.16 (0.51, 2.67)	1.00	1.30 (0.70, 2.43)
	Fully adjusted model ^a	1.00	1.64 (0.79, 3.12)	1.00	1.21 (0.65, 2.26)
All-cause mortality					
All participants	No. all-cause death/Total Mortality (1/1000)	930 / 21,244	130 / 3386	464 / 12,084	596 / 12,546
		43.8	38.4	38.4	47.5
	Crude model	1.00	0.98 (0.74, 1.30)	1.00	1.23 (1.03, 1.47)
	Fully adjusted model	1.00	0.93 (0.69, 1.25)	1.00	1.25 (1.05, 1.50)
Heart disease mortality					
All participants	No. heart disease death/Total Mortality (1/1000)	192 / 21,244	29 / 3386	90 / 12,084	131 / 12,546
		9.0	8.5	7.4	10.4
	Crude model	1.00	0.93 (0.56, 1.56)	1.00	1.48 (0.92, 2.39)
	Fully adjusted model	1.00	0.86 (0.51, 1.46)	1.00	1.62 (0.98, 2.69)

CVD mortality, cardiovascular disease mortality, defined by the International Classification of Diseases codes I00–I09, I11, I13, I20–I51, and I60–I69. Since deaths from cerebrovascular diseases (I60–I69) were not tracked after December 31, 2014, in the National Health and Nutrition Examination Survey, a subsample of participants from the 1999–2014 cycles was examined for CVD mortality only.

Heart disease mortality, defined by defined by the International Classification of Diseases codes I00–I09, I11, I13, I20–I51.

Fully adjusted model: adjusted for age, sex, race/ethnicity, marital status,

poverty income ratio, education level, smoking status, alcohol drinking status, leisure time physical activity, and total energy intake. Diet scores were further adjusted in models assessing long working hours, and working hours were further adjusted in models when assessing diet scores. Covariates (diet score/working hours) were retained in their original continuous form when adjusted for in models to maximize confounding control, while exposures were dichotomized for primary analyses.

¹ Defined by Framingham 10-year cardiovascular disease risk score (low: < 20% vs. high: ≥ 20%).

^a Adjusted for race/ethnicity, marital status, poverty income ratio, educational level, alcohol drinking status, leisure time physical activity, and total energy intake. Diet scores were further adjusted in models assessing long working hours, and working hours were further adjusted in models when assessing diet scores.

evidence that LWH increased CVD mortality risk among workers with high baseline CVD risk. The WHO/ILO reported a pooled RR of 1.17 (95%CI = 1.05, 1.31), which may be driven by individual studies with greater weights showing positive associations among male or semi-routine workers (Li et al., 2020). European studies focusing on fatal CVD reported an increased (HR = 1.68, 95%CI = 1.08, 2.61) risk of death with LWH (Ervasti et al., 2021). The lack of significant associations of LWH and mortality in this U.S. study may be due to higher job turnover deriving from discrepancies in history and cultural attitude (Bureau of Labor Statistics, 1983), limiting the ability of a single-time LWH measurement to capture long-term exposure. However, LWH increased CVD mortality among participants with higher baseline CVD risk, aligning with European findings linking work stress to a 1.68-fold higher mortality risk in those with cardiometabolic disease, possibly through transient endothelial dysfunction, myocardial ischemia, and cardiac arrhythmia (Kivimäki et al., 2018). While direct evidence linking LWH to mortality is lacking, its strong association with stressful experience at work provides a compelling basis for expecting similar patterns in LWH-related mortality (Lee et al., 2017). Notably, the effect size remained robust even after occupation adjustment.

Our study is pioneering in assessing the EAT-Lancet diet's relationship with cardiometabolic outcomes and CVD mortality in the US workforce. Our results on the diet's beneficial association with diabetes and all-cause mortality align with European studies (Langmann et al., 2023a; Langmann et al., 2023b; Stubbendorff et al., 2022). While our high diet quality group's score range was similar to the quintile reported by Stubbendorff et al., we observed smaller effect sizes for all-cause and CVD mortality, possibly due to differences in reference groups (Stubbendorff et al., 2022). Our work addresses knowledge gaps regarding the interaction of LWH and poor diet quality. Despite identifying only 7% of participants with both LWH and poor diet quality, the joint association on obesity, hypertension, and mortality is significant, especially given the large sample size. Moreover, the reduced or null joint association seen in those with LWH and a high diet score underscores the potential of this diet to counteract LWH's adverse impacts. As a health promotion strategy, individual actions like a healthy diet are critical, given the longer timeline of organizational job redesign.

This study has several strengths. First, our analyses were based on a nationally representative sample of US workers, including a diverse range of occupations, increasing the generalizability of the results. Second, considering the limited evidence on the joint associations, our findings could expand current knowledge. Third, we conducted a comprehensive examination of various cardiometabolic disorders and mortality outcomes. Last, potential confounders, including socioeconomic and lifestyle factors, were controlled.

We acknowledged several limitations. Although self-reported working hours showed high validity (Spearman correlation = 0.89 with annual records) (Imai et al., 2016), potential misclassification of working hours and diet exists due to a lack of repeated measurements, which may not capture changes over time. This misclassification is likely nondifferential, biasing mortality outcomes toward the null. Despite excluding participants with a history of CVD or cancer to remove reverse

causation, the nature of the cross-sectional design prohibits us from establishing causality regarding the association with cardiometabolic outcomes. The potential protective effects involving mechanisms of voluntary LWH and/or overtime payment that may counterbalance the adverse effects of LWH remain unknown. Lastly, even though adjusting for major confounders, residual confounding cannot be completely ruled out.

CRediT authorship contribution statement

Xiang Li: Writing – review & editing, Writing – original draft, Visualization, Validation, Software, Resources, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization. **Jian Li:** Writing – review & editing, Resources, Methodology, Investigation, Conceptualization. **Xuyuehe Ren:** Writing – review & editing, Validation. **Tong Xia:** Writing – review & editing, Formal analysis. **Onyebuchi A. Arah:** Writing – review & editing, Methodology. **Liwei Chen:** Writing – review & editing, Resources, Project administration, Methodology, Investigation, Funding acquisition, Conceptualization.

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.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.jpmed.2025.108275>.

Data availability

Data will be made available on request.

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