Brief Report

CARDIAC RESEARCH

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Life's Essential 8 and mortality in US adults with clinical obesity

A cohort study

Zhenyang Cao, BS[a], Qian Lu, BS[a], Jiaxin Shao, BS[a], Youxin Deng, BS[a], Huping Gong, PhD[b,*], Jianghua Zhou, PhD[c,*], Pan Huang, PhD[a,*]

Keywords: cardiovascular health, clinical obesity, life's essential 8, mortality, NHANES

1. Introduction

Obesity has become a major global public health challenge and a key contributor to adverse cardiometabolic outcomes. In 2025, the Lancet Diabetes and Endocrinology Commission redefined obesity as a chronic, multisystem condition and introduced clinical obesity, characterized by excess adiposity leading to impaired tissue, organ, or whole-body function.[1] This framework emphasizes the transition from excess body fat to clinically significant functional impairment.

Obesity is also a modifiable driver of cardiovascular disease (CVD), promoting cardiometabolic dysfunction through metabolic disturbances, hemodynamic strain, and chronic inflammation.[2] To assess cardiovascular health (CVH), the American Heart Association developed Life's Essential 8 (LE8),[3] an integrated metric of health behaviors and physiological factors. LE8 has been associated with cardiovascular events and mortality across diverse populations.[4,5] However, whether LE8 is associated with mortality among individuals with clinical obesity remains unclear.

Although clinical obesity is high-risk, cardiovascular risk remains heterogeneous. Identifying risk variation is essential for clinical management. As a multidimensional and modifiable measure, LE8 may serve as a pragmatic tool for risk stratification and targeted intervention. Therefore, this study investigated temporal trends in clinical obesity and evaluated the associations between LE8 and mortality in a representative US population.

2. Methods

We analyzed data from the National Health and Nutrition Examination Survey (NHANES) cycles (2005–2018), with mortality follow-up through December 31, 2019. NHANES uses a complex, multistage probability sampling design to produce nationally representative estimates of the US civilian population. Analyses accounted for survey stratification, clustering, and sampling weights according to National Center for Health Statistics guidelines.

Clinical obesity was defined according to the Lancet Diabetes & Endocrinology framework using available NHANES variables (Table S1; Figure S1, Supplemental Digital Content, https://links.lww.com/CARES/A11). Briefly, excess adiposity was identified using body mass index (BMI) and confirmed by anthropometric or body fat indicators when required. Clinical obesity was defined as excess adiposity accompanied by evidence of organ-system functional impairment or limitations in activities of daily living.

Among 13,532 adults aged ≥ 20 years with clinical obesity, 8,767 were included after excluding incomplete data (Figure S2, Supplemental Digital Content, https://links.lww.com/CARES/A11). Baseline characteristics of included and excluded participants were compared using standardized mean differences (Table S2, Supplemental Digital Content, https://links.lww.com/CARES/A11).

ZC, QL, and JS contributed equally to this work.

Supplemental Digital Content is available for this article.

[a] School of Nursing, Wenzhou Medical University, Wenzhou, China, [b] School of Nursing, Gannan Medical University, Ganzhou, China, [c] Department of Cardiology, the First Affiliated Hospital of Wenzhou Medical University, Wenzhou, China.

* Correspondence: Huping Gong, School of Nursing, Gannan Medical University, Ganzhou 343000, China (e-mail: 1025272457@gmu.edu.cn); Jianghua Zhou, Department of Cardiology, The First Affiliated Hospital of Wenzhou Medical University, Wenzhou 325000, China (e-mail: zhoujianghua@wmu.edu.cn); Pan Huang, School of Nursing, Wenzhou Medical University, Wenzhou 325000, China (e-mail: hpzyqzjh@wmu.edu.cn).

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Cardiac Research (2026) 2:2

Received: 24 November 2025 / Received in final form: 2 March 2026 / Accepted: 3 March 2026

http://dx.doi.org/10.1097/re9.0000000000000015

CVH was measured using LE8, which integrates four health behaviors (diet, physical activity, nicotine exposure, and sleep) and four health factors (BMI, blood lipids, blood glucose, and blood pressure). Each component was scored 0 to 100, with higher scores reflecting more favorable CVH. Following prior studies,[5] total LE8 scores were categorized as low (0–49) or moderate-to-high (50–100) CVH (Tables S3 and S4, Supplemental Digital Content, https://links.lww.com/CARES/A11), as few participants had high CVH.

The primary outcomes were all-cause mortality (ACM) and cardiovascular mortality (CVM). Mortality status and causes of death were ascertained by linkage to the National Death Index. CVM was defined using International Classification of Diseases, Tenth Revision codes.

Covariates included age, sex, race/ethnicity, marital status, educational level, poverty income ratio (PIR), alcohol use, depression, and serum creatinine. All were measured at baseline and included in fully adjusted models.

Baseline characteristics were summarized as means with standard deviations or counts with percentages. Temporal trends in clinical and traditional obesity were evaluated. Weighted ACM rates were calculated per 1000 person-years. Survival distributions were compared using Kaplan–Meier curves with log-rank tests. Associations between CVH and mortality were examined using Cox proportional hazards regression models, reported as hazard ratios (HRs) with 95% confidence intervals (CIs). Nonlinear associations were assessed using restricted cubic spline models. Subgroup and sensitivity analyses were conducted to evaluate the robustness of the findings. Additional details on survey-weighted analyses, clinical obesity definition, outcome ascertainment, covariate assessment, and statistical analyses are provided in the Supplemental Materials.

3. Results

A total of 8,767 adults with clinical obesity were included in the analysis. The mean age of the study population was 48.74 years; 53% were female, and 65% were non-Hispanic White. Overall, 32% had low CVH and 68% had moderate-to-high CVH. Participants with higher CVH were more likely to be younger, married, to have a higher educational level and poverty income ratio PIR, and report less alcohol use or depression, reflecting more favorable baseline health profiles (Table S6, Supplemental Digital Content, https://links.lww.com/CARES/A11). From 2005 to 2018, the weighted prevalence of clinical obesity increased steadily but remained lower than that of traditional BMI-defined obesity (Fig. 1A). Clinical obesity was more common among younger adults, women, non-Hispanic Black individuals, and those with a lower educational level (Figure S3, Supplemental Digital Content, https://links.lww.com/CARES/A11). Among adults with low CVH, ACM rates were higher in men than in women (10.75 vs. 9.13 per 1,000 person-years), whereas mortality rates were lower among participants with moderate-to-high CVH in both sexes (Fig. 1B).

During a median follow-up of 78 months, 758 deaths occurred, including 256 cardiovascular deaths. Individuals with low CVH showed poorer survival (Figure S4, Supplemental Digital Content, https://links.lww.com/CARES/A11). In fully adjusted Cox models, moderate-to-high CVH was associated with a lower risk of ACM compared with low CVH (HR = 0.68, 95% CI: 0.58–0.78; $p < 0.001$). Moderate-to-high CVH

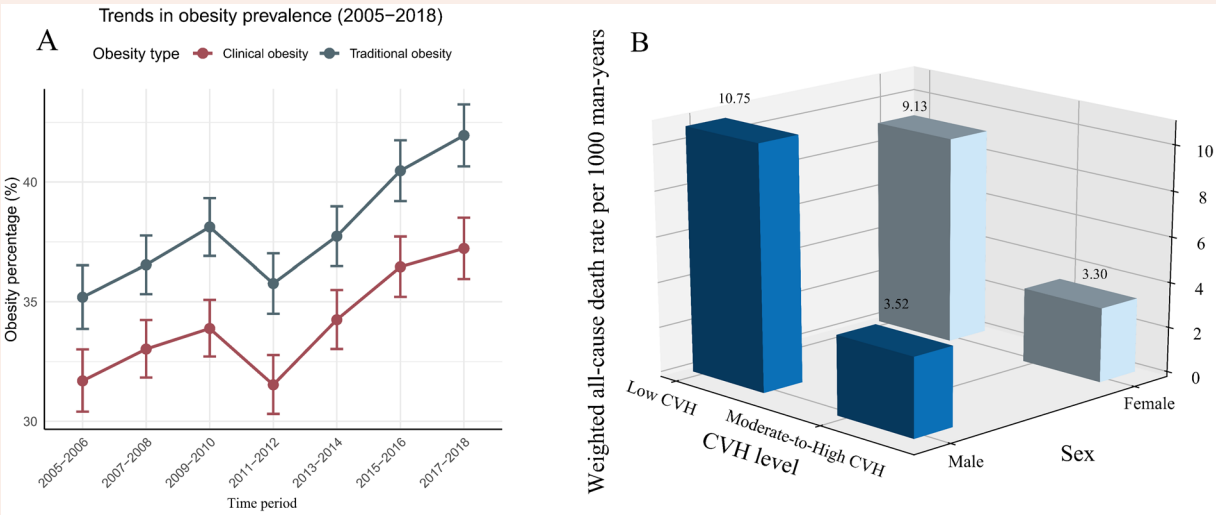


Figure 1. Obesity prevalence trends and weighted mortality by cardiovascular health. Panel A depicts trends in the prevalence of clinical and traditional obesity over the period from 2005 to 2018. Panel B shows weighted all-cause mortality rates per 1,000 person-years across cardiovascular health levels, stratified by sex. CVH = cardiovascular health.

Table 1

Associations between cardiovascular health levels and mortality among individuals with clinical obesity.

		Model 1	Model 2	Model 3
	Case/participants	HR (95% CI) *p*	HR (95% CI) *p*	HR (95% CI) *p*
All-cause mortality				
Per 10 points increase	758/8,767	0.66 (0.63, 0.70) < 0.001	0.74 (0.70, 0.79) < 0.001	0.78 (0.73, 0.83) < 0.001
Low CVH	413/2,840	Reference	Reference	Reference
Moderate-to-High CVH	345/5,927	0.41 (0.36, 0.48) < 0.001	0.60 (0.52, 0.69) < 0.001	0.68 (0.58, 0.78) < 0.001
p for trend		< 0.001	< 0.001	< 0.001
CVD mortality				
Per 10 points increase	256/8,767	0.65 (0.60, 0.72) < 0.001	0.73 (0.66, 0.81) < 0.001	0.78 (0.70, 0.87) < 0.001
Low CVH	141/2,840	Reference	Reference	Reference
Moderate-to-High CVH	115/5,927	0.40 (0.31, 0.51) < 0.001	0.60 (0.47, 0.76) < 0.001	0.68 (0.53, 0.88) 0.004
p for trend		< 0.001	< 0.001	< 0.001

Model 1: unadjusted;
Model 2: adjusted for age, sex;
Model 3: adjusted for age, sex, race/ethnicity, educational level, marital status, PIR, alcohol use, depression, and SCr.
CVD = cardiovascular disease; CVH = cardiovascular health; PIR = poverty income ratio; SCr = serum creatinine.

was also associated with lower CVM risk (HR = 0.68, 95% CI: 0.53–0.88; $p = 0.004$) (Table 1). Additional component-specific associations are shown in Figure S5, Supplemental Digital Content, https://links.lww.com/CARES/A11. Restricted cubic spline analyses further showed that higher LE8 scores were consistently associated with lower risks of both ACM and CVM, supporting a dose–response relationship between better CVH and lower mortality (Figure S6, Supplemental Digital Content, https://links.lww.com/CARES/A11).

Subgroup analyses showed generally consistent associations across demographic strata, with significant interactions observed for age and educational level (Figure S7, Supplemental Digital Content, https://links.lww.com/CARES/A11). Sensitivity analyses excluding deaths within the first 2 years of follow-up or participants with baseline CVD yielded similar results (Tables S7 and S8, Supplemental Digital Content, https://links.lww.com/CARES/A11). Component-level analyses showed inverse associations of both health behavior and health factor domains with ACM and CVM (Table S9, Supplemental Digital Content, https://links.lww.com/CARES/A11). When CVH was further categorized into low, moderate, and high levels, a graded inverse association with mortality was observed in Cox proportional hazards models (Table S10, Supplemental Digital Content, https://links.lww.com/CARES/A11). Competing-risk analyses treating noncardiovascular death as a competing event further confirmed the association between moderate-to-high CVH and lower CVM risk (Table S11; Figure S8, Supplemental Digital Content, https://links.lww.com/CARES/A11).

4. Discussion

This study demonstrates a clear association between CVH and mortality among adults with clinical obesity. Despite increasing clinical obesity prevalence, mortality risk varied substantially by CVH status, with lower ACM and CVM among individuals with higher CVH. These findings highlight prognostic heterogeneity within clinical obesity and suggest that CVH provides valuable risk stratification information.

A key strength is the use of LE8, which integrates multiple interrelated risk factors into a single score. By capturing cumulative CVH burden rather than isolated risk factor control, LE8 provides a holistic cardiometabolic risk assessment. The persistent inverse association between LE8 and mortality supports its clinical relevance. LE8 encompasses health behaviors and factors that influence blood pressure regulation, insulin sensitivity, lipid metabolism, and systemic inflammation. Improvements across these domains may reduce cardiovascular burden and limit obesity-related organ damage.[6,7]

Our results reveal demographic and socioeconomic variation in clinical obesity. Higher prevalence was observed among younger adults, women, non-Hispanic Black individuals, and those with lower education, reflecting known demographic patterns in obesity risk.[8,9] The association between CVH and mortality in this clinical obesity population varied by age and education. Stronger associations among younger adults may reflect lower competing noncardiovascular mortality and greater sensitivity to modifiable cardiometabolic factors earlier in life, whereas multimorbidity and survivorship bias among older adults may attenuate risk estimates.[4] Differences in health literacy, preventive care access, and barriers to risk factor control may explain why the association was stronger among individuals with higher educational attainment.[10] Together, these findings suggest that the prognostic value of CVH differs by demographic and socioeconomic context.

Several limitations should be acknowledged. First, clinical obesity and some LE8 components were partially based on self-reported data, which may have caused misclassification. Second, limited organ-function measures in NHANES may have constrained the precision of clinical obesity classification. Third, overlap between clinical obesity and LE8 physiological components may have

attenuated associations. Finally, the observational design precludes causal inference, and residual confounding cannot be excluded.

Overall, our results emphasize the relevance of CVH for prevention and clinical management in adults with clinical obesity. Early identification of individuals with unfavorable CVH profiles may facilitate tailored lifestyle, behavioral, and cardiometabolic interventions. As clinical obesity becomes increasingly common, improving CVH may help reduce mortality and narrow health inequities in this vulnerable population.

Acknowledgements

We thank NHANES participants and staff for their contributions, and Mingjie Chen (Shanghai NewCore Biotechnology Co., Ltd.) for providing visualization support for the heatmap.

Ethical statement

NHANES protocols were approved by the NCHS Ethics Review Board; all participants provided written informed consent. This study complied with the Declaration of Helsinki. Details: www.cdc.gov/nchs/nhanes/about/erb.html.

Conflicts of interest

The authors have no conflicts of interest to disclose.

Funding source

Supported by Zhejiang Provincial Philosophy and Social Sciences Planning Project (25NDJC112YBMS).

Data availability statement

Data are available via the NHANES at: www.cdc.gov/nchs/nhanes.

Author contributions

Zhenyang Cao: Formal data analysis and writing—review & editing.

Qian Lu: Writing—review & editing.

Jiaxin Shao and Youxin Deng: Curation and investigation.

Huping Gong: Drafted the original manuscript.

Jianghua Zhou and Pan Huang: Conceptualized and designed the study.

All authors approved the final manuscript.

AI statement

Generative AI-assisted technology was used only for language polishing. The authors reviewed all content and take full responsibility for the manuscript accuracy, integrity, and originality.

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