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Association between social determinants of health with the all-cause and cause-specific (cancer and cardio-cerebrovascular) mortality among the population with metabolic syndrome: NHANES 2005–2018

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Abstract

Background Social determinants of health (SDOH) and metabolic syndrome (MetS) are related, but their combined effect on mortality risk remains unclear.

Methods We analyzed data from NHANES (National Health and Nutrition Examination Survey) cycles between 2005 and 2018. The composite SDOH score was calculated by summing the weighted scores for each SDOH, categorizing participants into four groups: Q1 (0–1), Q2 (2–3), Q3 (4) and Q4 (≥ 5). Kaplan-Meier survival curves and multivariate Cox proportional hazards models were used to examine the relationship between SDOH and mortality outcome. Restricted cubic spline (RCS) analyses were conducted to explore nonlinear relationships. Subgroup analyses assessed the consistency and robustness of the findings across various demographic and clinical factors.

Results Of the 7,366 patients with MetS, 1,193 died, including 407 from cardiovascular and cerebrovascular diseases and 269 from cancer. Cox regression analyses, using fully adjusted Model 2, revealed that higher SDOH levels had increased hazards for all-cause mortality (HR = 2.41, 95% CI: 1.87,3.12), cancer-related death (HR = 2.45, 95% CI: 1.54,3.89), and Cardio – cerebrovascular disease (HR = 2.62, 95% CI: 1.79,3.84). Kaplan-Meier analyses further supported these findings, demonstrating that participants with higher SDOH scores had lower survival rates. Additionally, RCS

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modeling confirmed a linear relationship between SDOH and mortality, with no indication of a nonlinear relationship (P for nonlinear > 0.05).

Conclusion Our findings indicate that adverse social determinants of health are strongly linked to an increased risk of all-cause mortality in individuals with MetS. However, due to the observational and cross-sectional nature of this study, it is important to interpret these results as associations rather than implying any causal relationships.

Keywords The National health and nutrition examination survey, Social determinants of health, Metabolic syndrome, Mortality

Introduction

Metabolic syndrome (MetS) is a condition defined by a cluster of both metabolic and non-metabolic disorders that include abdominal obesity, insulin resistance, atherogenic dyslipidemia, and hypertension [1]. MetS represents a pathophysiological state that is primarily driven by an imbalance between caloric intake and energy expenditure, where excessive caloric intake, often coupled with inadequate physical activity, creates a fertile ground for the development of this syndrome. However, this imbalance is not the sole contributor. The development of MetS is also influenced by a host of other factors, including genetic and epigenetic predispositions, which can affect the body's response to environmental factors, as well as the increasing prevalence of sedentary lifestyles, where physical activity is often replaced by prolonged periods of inactivity. Furthermore, the quality and composition of an individual's diet—ranging from nutrient deficiencies to the consumption of processed foods high in sugar, fats, and refined carbohydrates—plays a significant role in the manifestation and progression of MetS [2]. The clinical significance of MetS cannot be overstated, as it substantially contributes to the onset and progression of several serious health conditions. These include cardiovascular disease, diabetes, dyslipidemia, stroke, osteoarthritis, various cancers, and premature mortality, all of which are major contributors to global health burdens [3–5].

The growing prevalence of MetS is a cause for concern, as it continues to rise alarmingly, particularly in countries with rapidly aging populations, where the healthcare infrastructure is often under strain [6]. Epidemiological data underscore the alarming scope of this issue, revealing that MetS affects approximately one-third of the adult population in the United States, with the prevalence of this syndrome reaching nearly half of individuals aged 60 years and older [7]. In addition to its health consequences, MetS places a significant financial burden on healthcare systems, often leading to increased medical costs due to the treatment of associated chronic conditions. This burden is compounded by a marked reduction in the quality of life for affected individuals, who may experience physical, emotional, and financial hardships as a result of managing their condition [8, 9].

Social determinants of health (SDOH) refer to the non-medical risk factors that significantly influence health outcomes and overall well-being. These determinants include a wide range of factors, such as income, education, employment, housing, food security, and access to affordable healthcare, all of which play a crucial role in shaping individuals' health outcomes [10]. Each of these domains reflects a set of factors that can either enhance or hinder health, depending on how they are experienced by individuals. Economic stability is one of the core areas and encompasses factors such as employment status, income levels, food insecurity, and housing instability, all of which have profound effects on individuals' ability to lead healthy lives. For instance, individuals facing unemployment or low income may struggle to afford healthy food or housing, leading to long-term health challenges. Education is a powerful determinant of health, as individuals with higher levels of education tend to experience better health outcomes due to greater health literacy, access to resources, and health-promoting behaviors. The social and community context domain includes factors such as social support, social capital, social isolation, loneliness, discrimination, and race and ethnicity. These factors shape individuals' experiences within their communities and influence mental and physical health outcomes, with social isolation and discrimination, for example, being linked to poorer mental health and increased chronic disease risk. The neighborhood and built environment domain focus on the socioeconomic status of the area in which individuals live, as well as environmental factors such as food availability, exposure to violence, and access to safe spaces for physical activity. Finally, the health and healthcare domain addresses access to healthcare services, the quality of care received, health insurance coverage, and health literacy. A lack of access to affordable and quality healthcare can lead to delayed diagnoses, ineffective treatments, and poorer health outcomes overall [11, 12].

SDOH have been shown to have a significant impact on various health conditions, with numerous studies highlighting their strong association with the risk of depression, cardiovascular disease (CVD), cancer, and type 2 diabetes [13–15]. The relationship between SDOH and these diseases emphasizes the importance of addressing

non-medical risk factors in public health interventions. Furthermore, emerging research suggests that other factors, such as alcohol consumption, geographic origin, neighborhood environment, educational disparities, and socioeconomic status, may contribute to the risk of developing MetS. These factors, often intertwined with broader social determinants, can exacerbate the risk of metabolic diseases and are critical to consider in efforts to prevent and manage MetS [16, 17].

However, it is unclear how SDOH affects the risk of death in patients with MetS. Therefore, the aim of this study was to explore the relationship between SDOH and death in patients with MetS by analyzing adult data from the National Health and Nutrition Examination Survey (NHANES). This investigation aims to deepen our understanding of their combined impact on mortality risk, which is essential for the development of effective public health strategies aimed at preventing premature death.

Methods

Data source and study design

The SDOH score was derived from self-reported data in the NHANES dataset, which utilizes a stratified, multistage probability sampling design. This approach ensures the sample accurately represents the U.S. population across diverse demographic and socioeconomic groups. Data collection follows a standardized process with stringent quality control measures, ensuring the reliability and consistency of the self-reported information. The use of standardized questionnaires and trained interviewers helps minimize measurement errors, thereby strengthening the validity of the SDOH score as a comprehensive measure of social determinants of health.

The NHANES, conducted by the National Center for Health Statistics (NCHS), assesses the health and nutritional status of the U.S. population. All participants provided written informed consent, ensuring their voluntary participation and adherence to ethical research standards. Additionally, the study protocol was rigorously reviewed and formally approved by the NCHS Ethics Review Committee, ensuring compliance with ethical guidelines and safeguarding participant rights and welfare throughout the research process [18].

Study population

In this cohort study, we initially considered 70,190 participants from NHANES between 2005 and 2018. To refine the sample and ensure its relevance, we applied specific exclusion criteria. Participants under the age of 20 were excluded, as were those without MetS or individuals missing key covariate data required for the analysis. After applying these criteria, the final sample consisted of 7,366 eligible participants, with the selection process outlined in Fig. 1.

Definition of SDOH score

Self-reported data on 8 sub-items of SDOH across 5 domains were operationalized according to the criteria outlined in the U.S. Healthy People 2030 initiative and two previous studies, with a cumulative measure of unfavorable SDOH calculated for analysis [11, 19]. These domains include economic stability (employment status, family poverty-income ratio, and food security), education access and quality (education level), healthcare access and quality (health insurance coverage and type of health insurance), neighborhood and built environment (homeownership), and social and community context (marital status). The specific definitions of these SDOH domains and sub-items are provided in Table S1. To simplify the analysis, these SDOH items were dichotomized into favorable or unfavorable levels based on conventional cutoff points. The cumulative number of unfavorable SDOH was determined by summing the 8 dichotomized SDOH items, with a value of 0 representing a favorable level and a value of 1 representing an unfavorable level. To more effectively assess the association between SDOH and mortality in the MteS population, SDOH was categorized into quartiles: Q1 (0–1), Q2 (2–3), Q3 (4), and Q4 (≥ 5) [20]. Additionally, sensitivity analyses were conducted using three-quarter spacing [21].

Definition of MetS

MetS in adults was defined based on the criteria established by the National Cholesterol Education Program's Adult Treatment Panel III report [22], which has been extensively validated in epidemiological studies, including those related to NHANES [23]. A diagnosis of MetS requires the presence of at least three of the following conditions: **1.**Hyperglycemia, fasting blood glucose level ≥ 100 mg/dL (5.6 mmol/L) or the use of antidiabetic medications; **2.**Reduced high-density lipoprotein cholesterol (HDL-C) levels, HDL cholesterol < 40 mg/dL (1.0 mmol/L) for men and < 50 mg/dL (1.3 mmol/L) for women, or the use of lipid-lowering medications; **3.**Hypertriglyceridemia, triglyceride level ≥ 150 mg/dL (1.7 mmol/L).; **4.**Abdominal obesity, waist circumference ≥ 102 cm for men and ≥ 88 cm for women; **5.**Hypertension, systolic blood pressure ≥ 130 mmHg, diastolic blood pressure (DBP) ≥ 85 mmHg, or the use of antihypertensive medications. These criteria provide a standardized approach for identifying MetS in clinical and research settings.

Mortality outcomes

The NCHS provided the Public-Use Linked Mortality Files, which were used to determine mortality outcomes in this study. The primary outcomes assessed included all-cause mortality, as well as mortality due to

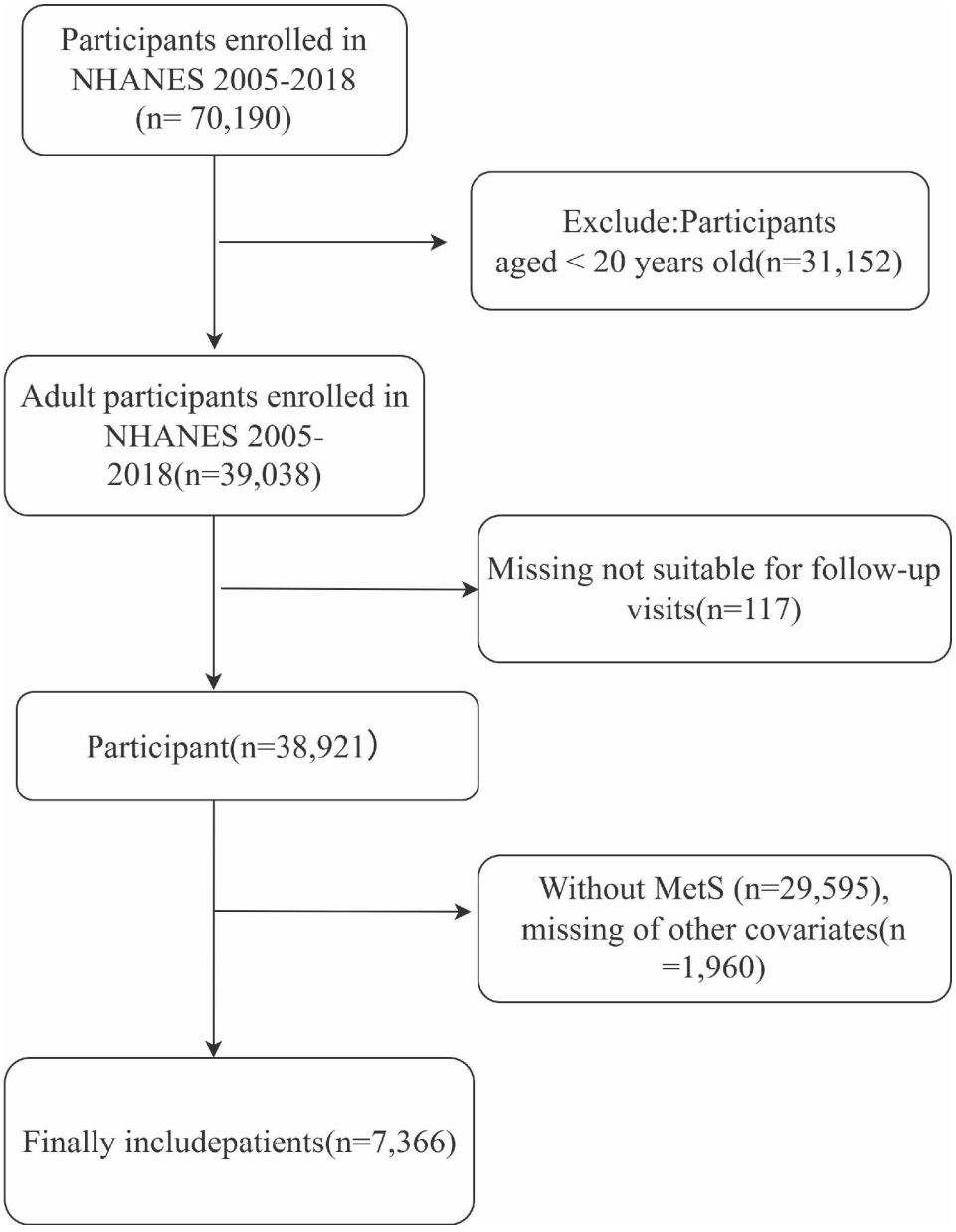

Fig. 1 The flow chart of the included participants in this study

cardio-cerebrovascular diseases (ICD-10 codes I00-I09, I11, I13, I20-I51 and I60-I69) and cancer (ICD-10 codes C00-C97).

Covariables

The variables analyzed in this study included a range of demographic and socioeconomic factors. Age, sex, and ethnicity were recorded, with ethnicity categorized into four groups: Mexican American, Non-Hispanic Black, Non-Hispanic White, and Other. Smoking status was classified based on lifetime cigarette use into three categories: never (fewer than 100 cigarettes), former (more than 100 cigarettes but not currently smoking), and current (more than 100 cigarettes and actively smoking). Alcohol use was classified based on daily and binge drinking frequency: never (consumed < 12 drinks in a lifetime), former (consumed ≥ 12 drinks in one year but not in the last year, or did not drink in the last year but consumed ≥ 12 drinks in a lifetime), mild (≤ 1 drink per day for females and ≤ 2 drinks per day for males), moderate (≤ 2 drinks per day for females and ≤ 3 drinks per day for males), or heavy (≥ 3 drinks per day for females and ≥ 4 drinks per day for males) [24, 25]. Body mass index (BMI) (kg/m^2) was calculated by dividing weight (kg) by height squared (m^2) and categorized into three groups: <25 kg/m^2, 25–29.9 kg/m^2, and > 29.9 kg/m^2 [26]. Hyperlipidemia

was defined as having elevated triglyceride levels of 150 mg/dL (1.7 mmol/L) or higher, total cholesterol levels of 200 mg/dL (5.18 mmol/L) or higher, low-density lipoprotein (LDL) levels of 130 mg/dL (3.37 mmol/L) or higher, or high-density lipoprotein levels below 40 mg/dL (1.04 mmol/L) in men and below 50 mg/dL (1.30 mmol/L) in women [27]. Additionally, individuals taking cholesterol-lowering medications were classified as hyperlipidemic. Hypertension was diagnosed using a combination of factors, including the use of antihypertensive medication, self-reported history of hypertension, and the average of three blood pressure measurements. A diagnosis of hypertension was made if the average systolic blood pressure was ≥ 130 mmHg or the average diastolic blood pressure was ≥ 80 mmHg, ensuring that both clinical measurements and self-reported data were considered in the diagnosis of hypertension [28]. Type 2 Diabetes mellitus (DM) was determined by self-reported diagnosis or current use of antihyperglycemic medications [29]. A diagnosis of cardio-cerebral vascular disease (CCVD) was indicated by any affirmative answer to having been informed of congestive heart failure, coronary heart disease, angina, a heart attack, or a stroke [30].

Statistical analyses

Sample weighting, clustering, and stratification were systematically applied throughout all analyses, which is essential for ensuring the results accurately represent the national population. This methodology accounts for the multi-stage probability sampling of NHANES. In the baseline analysis, the MetS population was categorized according to SDOH levels. Continuous variables were expressed as mean ± standard error (SE), and differences between groups were assessed using weighted anova. Categorical variables were presented as numbers (percentages) and compared using weighted chi-square tests. Kaplan-Meier (KM) survival analyses were conducted to estimate survival probabilities over time for all-cause and cause-specific mortality, stratified by SDOH status in the MetS population. Differences between groups were tested using log-rank tests. Multivariate Cox proportional hazards regression models were used to examine the relationship between SDOH and all-cause and disease-specific mortality in the MetS cohort. Multiple models were constructed with varying levels of adjustment: the crude model did not adjust for any covariates; model 1 adjusted for age, sex, ethnicity, alcohol use, smoking, and BMI; and model 2 (fully adjusted) further included DM, hypertension, CCVD, hyperlipidemia, cancer, and depression. To assess the stability of the model, we conducted sensitivity analyses by categorizing the SDOH into tertiles: Q1 (0–2), Q2 (3–4) and Q3 (≥ 5). A fully adjusted Restricted Cubic Spline (RCS) model was used to evaluate the non-linear associations between SDOH and mortality outcomes. The RCS analysis tests for non-linearity by comparing the fit of a linear model to one that includes spline terms. Specifically, a P-value for non-linearity < 0.05 suggests that the relationship between SDOH and mortality is non-linear and cannot be adequately represented by a linear model. Conversely, a P for non-linearity > 0.05 indicates that the relationship is linear and requires further testing.

In addition to the RCS analysis, we conducted a trend test (P for trend) to evaluate the linear relationship between mortality outcomes and increasing levels of SDOH. A significant P for trend < 0.05 suggests a consistent linear relationship, indicating that mortality risk increases progressively with higher SDOH scores. This combination of RCS and trend analysis enables a comprehensive evaluation of both non-linear and linear trends in the data.

In addition to evaluating the main effects of SDOH on mortality outcomes, we conducted subgroup analyses to identify potential effect modifiers. These analyses aimed to determine whether specific demographic and clinical factors—such as age, sex, race, alcohol use, BMI, and hyperlipidemia—might influence the relationship between SDOH and mortality risk. This approach is commonly used in epidemiological studies to understand how various factors may modify the impact of exposures on health outcomes.

All statistical tests were two-sided and P values less than 0.05 were considered statistically significant. Analyses were performed using R software.

Results

Baseline characteristics

Table 1 The baseline characteristics of the study population, categorized by SDOH score tertiles, are summarized as follows. The weighted mean age of participants was 55.3 ± 0.3 years. Significant differences were observed across the SDOH tertiles for demographic variables, including age, sex, race, smoking status, alcohol consumption and the presence of hypertension, CCVD, depression and cancer ($P < 0.05$). However, no statistically significant differences were found in the prevalence of DM, BMI or hyperlipidemia ($P > 0.05$). These findings suggest that certain demographic and lifestyle factors may influence SDOH, which in turn may affect the health status of this population.

RCS analysis

In a fully adjusted RCS model that accounted for confounding factors, a higher SDOH score in individuals with MetS was associated with an increased risk of all-cause, cancer, and cardio-cerebrovascular mortality. However, all three outcomes followed linear patterns,

Table 1 Baseline characteristics of participants with MetS according to SDOH in NHANES

Variable	Total	Q1	Q2	Q3	Q4	Pvalue
Age	55.3 ± 0.3	57.0 ± 0.4	56.8 ± 0.5	52.3 ± 0.7	49.8 ± 0.5	< 0.0001
Follow-up Time (months)	87.2 ± 1.3	90.7 ± 2.0	86.4 ± 1.7	83.0 ± 2.0	82.4 ± 1.6	0.01
Follow-up Status, *n* (%)						< 0.0001
Alive	6173(87.3)	1905(90.4)	1912(84.0)	876(85.2)	1480(86.8)	
Deceased	1193(12.7)	275(9.6)	478(16.0)	178(14.8)	262(13.2)	
Sex						< 0.0001
Female	3984(52.2)	956(43.2)	1303(56.6)	613(60.2)	1112(62.5)	
Male	3382(47.8)	1224(56.8)	1087(43.4)	441(39.8)	630(37.5)	
Ethnicity, *n* (%)						< 0.0001
Mexican American	1172(7.7)	186(3.1)	324(7.0)	245(13.3)	417(17.9)	
Non-Hispanic Black	1510(10.1)	349(5.3)	505(10.5)	241(15.1)	415(18.7)	
Non-Hispanic White	3469(72.4)	1342(85.0)	1197(72.6)	407(59.2)	523(46.7)	
Other	1215(9.8)	303(6.6)	364(9.8)	161(12.4)	387(16.7)	
Employment, *n* (%)						< 0.0001
Employed, student, retired	5308(77.4)	2071(94.5)	1978(80.6)	656(59.1)	603(36.5)	
Not employed	2058(22.6)	109(5.5)	412(19.4)	398(40.9)	1139(63.5)	
PIR, *n* (%)						< 0.0001
<3	4904(53.0)	392(13.9)	1803(70.5)	989(92.0)	1720(98.1)	
≥3	2462(47.0)	1788(86.1)	587(29.5)	65(8.0)	22(1.9)	
Food security, *n* (%)						< 0.0001
Full food security	5009(75.3)	2125(97.7)	1878(78.7)	600(54.5)	406(21.6)	
Marginal, low, or very low	2357(24.7)	55(2.3)	512(21.3)	454(45.5)	1336(78.4)	
Education, *n* (%)						< 0.0001
High school or more	5311(82.1)	2109(96.7)	1852(82.5)	632(69.3)	718(50.7)	
Less than high school	2055(17.9)	71(3.3)	538(17.5)	422(30.7)	1024(49.3)	
Access to healthcare, *n* (%)						< 0.0001
No routine place, or ER/hospital/other	814(10.0)	35(1.8)	152(7.6)	157(19.2)	470(30.7)	
Routine place to go for healthcare	6552(90.0)	2145(98.2)	2238(92.4)	897(80.8)	1272(69.3)	
Health insurance, *n* (%)						< 0.0001
Government or no insurance	3623(38.1)	211(9.2)	977(37.6)	800(74.6)	1635(93.1)	
Private insurance	3743(61.9)	1969(90.8)	1413(62.4)	254(25.4)	107(6.9)	
Housing instability, *n* (%)						< 0.0001
Own home	4958(73.9)	2102(96.9)	1832(74.0)	573(53.4)	451(25.3)	
Rent or other arrangement	2408(26.1)	78(3.1)	558(26.0)	481(46.6)	1291(74.7)	
Marital status, *n* (%)						< 0.0001
Married or living with a partner	4481(66.0)	1917(88.4)	1461(58.1)	539(49.6)	564(31.9)	
Not married nor living with a partner	2885(34.0)	263(11.6)	929(41.9)	515(50.4)	1178(68.1)	
Smoke, *n* (%)						< 0.0001
former	2273(31.8)	760(35.1)	804(34.4)	306(28.0)	403(20.3)	
never	3719(49.8)	1208(54.3)	1207(48.8)	514(47.2)	790(41.5)	
now	1374(18.3)	212(10.6)	379(16.8)	234(24.8)	549(38.2)	
Alcohol user, *n* (%)						< 0.0001
former	1696(19.9)	383(15.8)	595(22.6)	277(24.6)	441(22.5)	
never	1193(12.1)	237(8.5)	376(12.8)	203(14.5)	377(19.1)	
now	4477(68.0)	1560(75.7)	1419(64.6)	574(60.9)	924(58.4)	
BMI, *n* (%)						0.2
<25	437(5.3)	125(4.9)	149(5.1)	54(5.8)	109(6.2)	
>29.9	4891(68.2)	1430(67.1)	1542(68.3)	722(69.5)	1197(70.2)	
25-29.9	2038(26.5)	625(28.0)	699(26.6)	278(24.7)	436(23.6)	
Hypertension, *n* (%)						< 0.001
no	1913(28.3)	536(25.7)	584(28.1)	288(32.8)	505(32.9)	
yes	5453(71.7)	1644(74.3)	1806(71.9)	766(67.2)	1237(67.1)	
DM, *n* (%)						0.5

Table 1 (continued)

Variable	Total	Q1	Q2	Q3	Q4	Pvalue
DM	2852(32.3)	780(31.3)	925(32.7)	431(33.3)	716(33.9)	
no	4514(67.7)	1400(68.7)	1465(67.3)	623(66.7)	1026(66.1)	
Hyperlipidemia, *n* (%)						0.1
no	436(5.3)	150(5.8)	151(5.8)	55(4.7)	80(3.7)	
yes	6930(94.7)	2030(94.2)	2239(94.2)	999(95.3)	1662(96.3)	
Depression, *n* (%)						< 0.0001
no	6510(89.9)	2097(95.7)	2177(90.6)	916(85.7)	1320(75.4)	
yes	856(10.1)	83(4.3)	213(9.4)	138(14.3)	422(24.6)	
Cancer, *n* (%)						< 0.0001
no	6365(85.3)	1802(82.2)	2043(85.9)	934(87.4)	1586(91.2)	
yes	1001(14.7)	378(17.8)	347(14.1)	120(12.6)	156(8.8)	
CCVD, *n* (%)						< 0.001
no	5838(81.9)	1794(84.2)	1889(81.8)	821(77.3)	1334(78.7)	
yes	1528(18.1)	386(15.8)	501(18.2)	233(22.7)	408(21.3)	
Heart attack, *n* (%)						0.4
no	6738(92.7)	2014(93.0)	2192(93.0)	959(91.5)	1573(91.9)	
yes	628(7.3)	166(7.0)	198(7.0)	95(8.5)	169(8.1)	
Stroke, *n* (%)						< 0.0001
no	6855(94.4)	2091(96.4)	2203(93.8)	966(91.5)	1595(92.2)	
yes	511(5.6)	89(3.6)	187(6.2)	88(8.5)	147(7.8)	
Congestive heart failure, *n* (%)						< 0.0001
no	6848(94.1)	2070(95.7)	2223(94.1)	969(91.1)	1586(91.7)	
yes	518(5.9)	110(4.3)	167(5.9)	85(8.9)	156(8.3)	
Coronary heart disease, *n* (%)						0.4
no	6750(91.8)	1975(91.2)	2182(91.9)	981(92.7)	1612(93.0)	
yes	616(8.2)	205(8.8)	208(8.1)	73(7.3)	130(7.0)	
Angina, *n* (%)						0.2
no	6967(94.8)	2071(95.3)	2265(95.0)	997(93.8)	1634(93.8)	
yes	399(5.2)	109(4.7)	125(5.0)	57(6.2)	108(6.2)	

Abbreviations: CI, confidence interval; BMI, body mass index; DM, diabetes mellitus; CCVD, cardio-cerebral vascular disease; SDOH, Social determinants of health; MetS, Metabolic syndrome; NHANES, National Health and Nutrition Examination Survey

Weighted Data: All data presented in this table are weighted to reflect the complex survey design of the NHANES dataset, ensuring that the sample is representative of the U.S. population

Data are presented as weighted mean (SE) or weighted frequencies (weighted percentages)

with no evidence of non-linearity (P for non-linearity = 0.071, 0.323, and 0.062, respectively), as illustrated in Fig. 2**(A-C)**.

Association of SDOH with mortality in MetS

Kaplan-Meier survival analyses further confirmed that survival probabilities for all-cause mortality, cancer mortality, and cardiovascular disease were significantly associated with SDOH levels ($P < 0.05$). The data demonstrated that higher SDOH scores were linked to decreased survival probabilities (Fig. 3).

Table 2 illustrates that within the study population, higher SDOH scores were associated with an increased risk of all-cause and cause-specific mortality. This relationship remained consistent across all three models, with all trends being statistically significant (P for trend < 0.05). In the fully multivariable-adjusted analyses, the categorization of SDOH revealed that individuals with high SDOH had an 141%, 145%, and 162% higher risk of all-cause, cancer, and cardio-cerebrovascular mortality, respectively, compared to those in the low-SDOH group.

In our analysis of SDOH and mortality outcomes, we identified significant associations across various domains **(Table S2-S4)**. For all-cause mortality, significant predictors included unemployment (HR = 1.41, 95% CI: 1.13, 1.75, $P = 0.002$), a PIR of ≥ 3 (HR = 0.78, 95% CI: 0.64, 0.95, $P = 0.01$), less than high school education (HR = 1.19, 95% CI: 1.02, 1.38, $P = 0.02$), and being unmarried or not living with a partner (HR = 1.20, 95% CI: 1.04, 1.40, $P = 0.01$). For cancer mortality, unemployment was a significant predictor (HR = 1.79, 95% CI: 1.19, 2.68, $P = 0.005$). Regarding cardio-cerebrovascular mortality, significant associations were found for less than high school education (HR = 1.39, 95% CI: 1.08, 1.78, $P = 0.01$) and being unmarried or not living with a partner (HR = 1.56, 95%

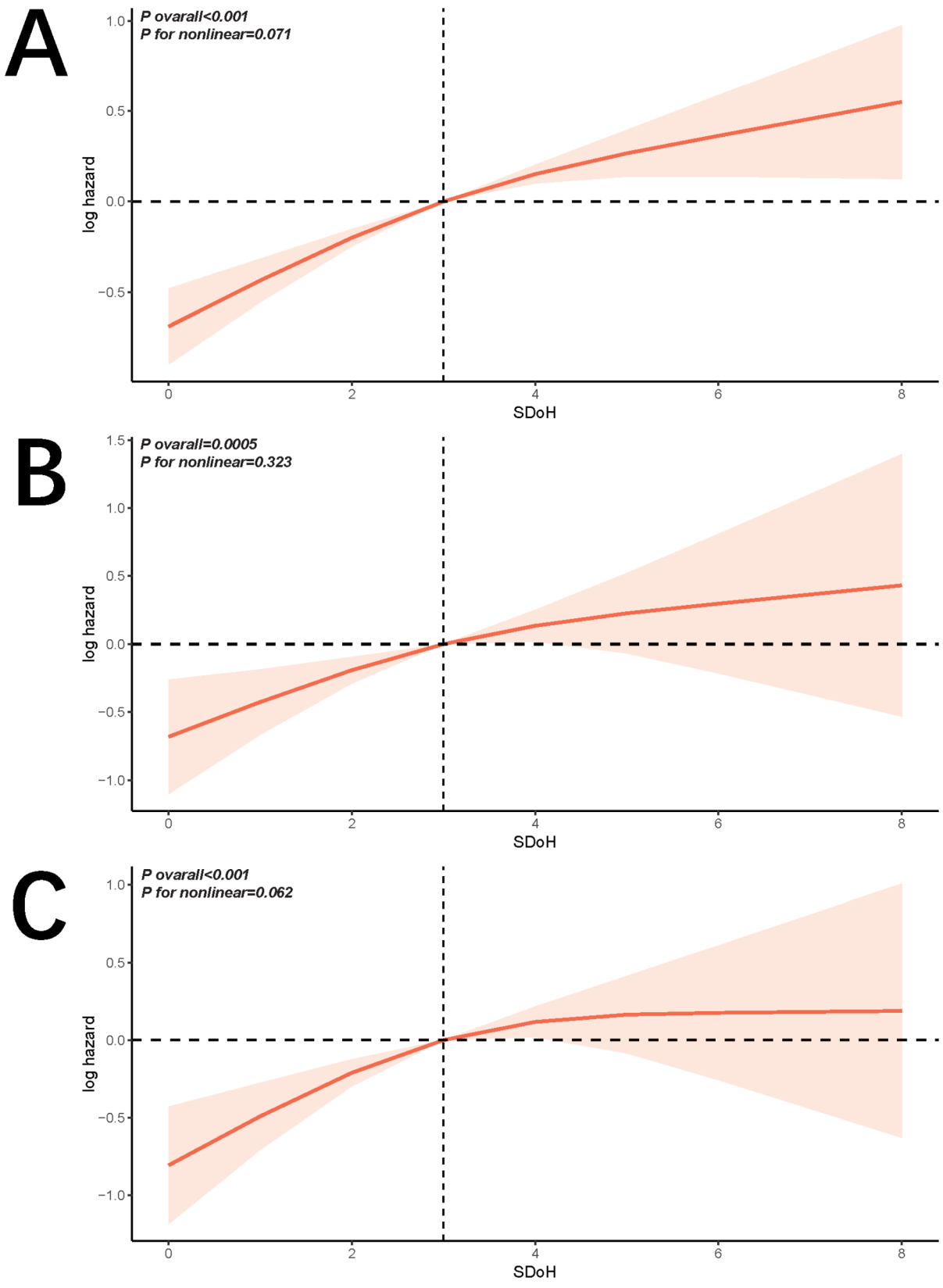

Fig. 2 Association between SDOH and all-cause mortality **(A)**, cancer mortality **(B)** and cardio–cerebrovascular mortality **(C)**. Adjusted for age, sex, ethnicity, alcohol user, smoke, BMI, DM, Hypertension, CCVD, Hyperlipidemia, cancer, depression

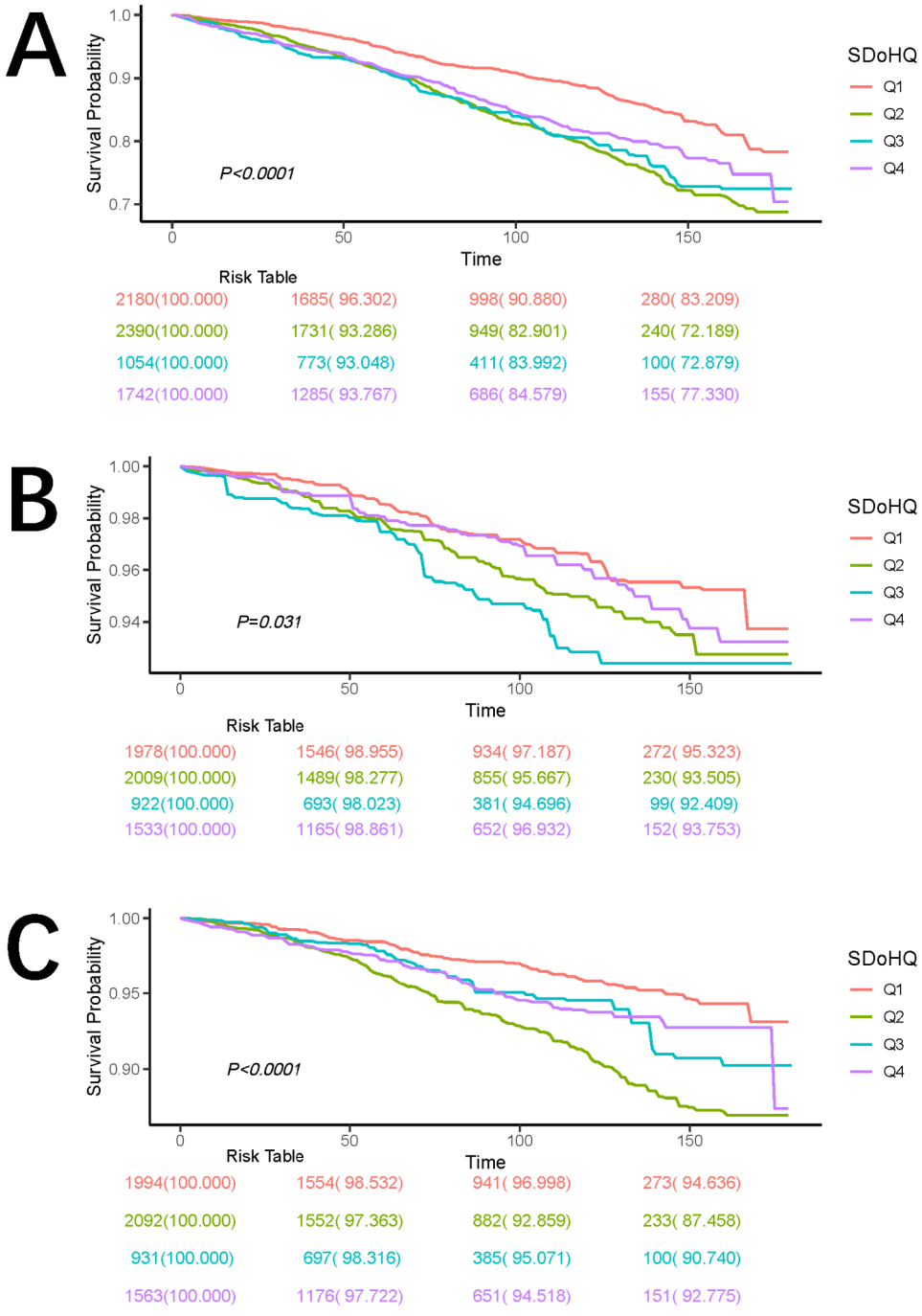

Fig. 3 Kaplan-Meier survival curve of all-cause mortality **(A)**, cancer mortality **(B)**, and cardio–cerebrovascular disease mortality **(C)** by SDOH

CI: 1.24, 1.95, $P<0.001$). These findings underscore the influence of specific SDOH domains on different mortality outcomes.

Subgroup analysis

Sensitivity analyses, performed after partitioning the SDOH into tertile spacing, are presented in Table S5. In patients with MetS, the associations between SDOH and the risk of all-cause, cardio – cerebrovascular disease mortality, and cancer mortality were consistent with the overall findings. In the subgroup analyses, the associations between depression, SDOH, and mortality remained similar across different age, sex, race, alcohol user, bmi and hyperlipidemia subgroups (all P for interaction > 0.05) (Table S6). These subgroup analyses aim to assess the consistency of the associations between SDOH and mortality across various demographic and clinical subgroups. However, we acknowledge that relying solely

Table 2 Cox regression analysis of SDOH and long-term mortality in MetS participants

	Crude model		Model 1		Model 2	
All-cause mortality	HR (95%CI)	P	HR (95%CI)	P	HR (95%CI)	P
Q1	ref		ref		ref	
Q2	1.77(1.46,2.16)	< 0.0001	1.51(1.27,1.80)	< 0.0001	1.55(1.30,1.84)	< 0.0001
Q3	1.74(1.36,2.22)	< 0.0001	2.08(1.67,2.58)	< 0.0001	1.98(1.59,2.46)	< 0.0001
Q4	1.56(1.22,1.99)	< 0.001	2.51(1.96,3.23)	< 0.0001	2.41(1.87,3.12)	< 0.0001
P for trend		< 0.0001		< 0.0001		< 0.0001
Cancer mortality						
Q1	ref		ref		ref	
Q2	1.43(1.02,2.00)	0.04	1.35(0.97,1.88)	0.08	1.45(1.05,1.99)	0.02
Q3	1.82(1.14,2.89)	0.01	2.38(1.49,3.82)	< 0.001	2.51(1.57,4.02)	< 0.001
Q4	1.17(0.76,1.81)	0.47	2.18(1.38,3.45)	< 0.001	2.45(1.54,3.89)	< 0.001
P for trend		0.11		< 0.0001		< 0.0001
Cardio – cerebrovascular disease mortality						
Q1	ref		ref		ref	
Q2	2.25(1.67,3.03)	< 0.0001	1.97(1.46,2.64)	< 0.0001	1.98(1.47,2.66)	< 0.0001
Q3	1.52(1.02,2.27)	0.04	2.05(1.41,2.98)	< 0.001	1.78(1.22,2.61)	0.003
Q4	1.54(1.04,2.27)	0.03	2.89(1.96,4.25)	< 0.0001	2.62(1.79,3.84)	< 0.0001
P for trend		0.01		< 0.0001		< 0.0001

Crude model: No adjustment for any potential influence factors

Model 1: Adjusted for age, sex, ethnicity, alcohol user, smoke, BMI

Model 2: Adjusted for age, sex, ethnicity, alcohol user, smoke, BMI, DM, Hypertension, CCVD, Hyperlipidemia, cancer, depression

Abbreviations: HR, hazard ratio; CI, confidence interval; BMI, body mass index; DM, diabetes mellitus; CCVD, cardio-cerebral vascular disease; SDOH, Social determinants of health

on P for interaction tests may lead to misleading conclusions, and caution is advised when interpreting these results. The potential for chance effects must be considered, and further studies with larger sample sizes or randomized controlled trials are necessary to validate these findings.

Discussion

To our knowledge, this is the first retrospective study to examine the relationship between SDOH and mortality in patients with MetS, providing new insights into the role of health factors in influencing survival outcomes. Our findings suggest that higher SDOH scores were significantly associated with an increased risk of all-cause, cardio – cerebrovascular, and cancer mortality.

Our study and Bundy et al.'s both highlight the SDOH-mortality link [19]. However, our research focuses on MetS patients, who face a higher mortality risk due to metabolic issues, whereas Bundy et al. examined the general US population, allowing broader application of their findings. We clarify in the manuscript that, despite these population differences, both studies confirm the significant impact of SDOH on mortality risk. Unlike Bundy et al.'s cumulative SDOH score approach, we categorized SDOH into quartiles (Q1-Q4) to explore its dose-response relationship with mortality, offering a more detailed analysis than their method. Our analysis adjusted for multiple confounders like age, sex, ethnicity, BMI, smoking, alcohol use, diabetes, hypertension, CCVD, hyperlipidemia, cancer, and depression. Sensitivity analyses confirmed that the SDOH-mortality association remained significant after these adjustments, indicating the reliability of our results. We used Cox regression and RCS to analyze the relationship between SDOH and mortality. The results showed a linear relationship without nonlinear effects. Separate Cox regressions for each SDOH subdomain revealed that unemployment, lower income-to-poverty ratios, and less than a high school education were significantly linked to increased mortality risk. For instance, unemployment was tied to higher all-cause mortality (HR = 1.41, 95% CI: 1.13, 1.75, $P = 0.002$) and cancer mortality (HR = 1.79, 95% CI: 1.19, 2.68, $P = 0.005$). Not married nor living with a partner and low education levels were particularly risky for cardio-cerebrovascular disease mortality.

Lifestyle and environmental factors, play an important role in the development of MetS. Among these, behaviors such as overeating and physical inactivity have emerged as major contributors. Specifically, high caloric intake, especially when sustained over time, may serve as a primary causative factor. Visceral obesity, which often results from excessive calorie consumption, has been shown to trigger a cascade of metabolic pathways that are central to the development of MetS [31, 32].This understanding is crucial because it highlights how dietary habits, particularly those leading to increased body fat, can be linked to the activation of multiple MetS-related mechanisms. Additionally, this may provide valuable

insight into why individuals with high BMI experience significantly poorer outcomes in relation to SDOH within the context of MetS. These insights are essential for understanding the broader impacts of obesity, which not only affect metabolic health directly but also influence social and environmental factors that contribute to health disparities.

As observed in our study, SDOH was significantly associated with cancer-specific mortality risk. This observation is consistent with a substantial body of evidence that highlights the critical role SDOH play in shaping health outcomes for cancer patients. Numerous studies have demonstrated that social and environmental factors, such as socioeconomic status, access to healthcare, and neighborhood conditions, substantially influence the prognosis and treatment effectiveness in cancer patients. In particular, socioeconomic disparities have been closely linked to differences in cancer care, with those from lower socioeconomic backgrounds often facing significant barriers to accessing high-quality treatment. Financial insecurity, lack of insurance, and limited access to essential resources like transportation have all been identified as major obstacles that prevent cancer patients from achieving optimal health outcomes [33]. These issues create a cascade of challenges that disproportionately affect patients in lower-income groups, complicating their ability to manage the disease effectively. For instance, individuals living in lower socioeconomic neighborhoods generally have lower rates of cancer screening, which often results in diagnoses occurring at more advanced stages of cancer, when treatment options are limited and prognosis is poorer [34, 35]. The lack of early detection is particularly problematic, as it significantly impacts the chances of successful treatment and survival. Additionally, cancer patients who reside in disadvantaged areas tend to experience higher rates of rehospitalization and mortality, which are often attributed to factors such as limited access to healthcare services, inadequate follow-up care, and the inability to adhere to treatment regimens due to financial and logistical difficulties [36, 37]. Taken together, these factors underscore a robust and multifaceted relationship between SDOH and cancer risk, highlighting the urgent need to address these determinants in both cancer care and prevention efforts. Increasing awareness of the role that SDOH play in cancer outcomes is crucial for developing policies and interventions aimed at reducing health disparities and ensuring more equitable access to cancer care.

SDOH is also strongly associated with an elevated risk of cardiovascular mortality, a relationship that has been widely documented in the literature. Research has consistently shown that individuals residing in economically disadvantaged neighborhoods in high-income countries face a significantly higher risk of developing coronary heart disease, even after controlling for individual-level factors such as income, education, and occupation [38]. This suggests that the neighborhood context, which includes factors like access to healthy food, safe spaces for physical activity, and environmental exposures, plays a critical role in shaping cardiovascular health outcomes. In addition to these environmental factors, individuals from lower socioeconomic backgrounds experience a greater burden of CCVD, which contributes to an increased risk of mortality. This heightened risk is primarily due to a combination of factors, including a higher prevalence of unhealthy behaviors, such as poor diet, physical inactivity, and smoking, along with greater psychosocial stress and limited access to quality healthcare [39]. These factors interact in complex ways, exacerbating health inequalities and leading to worse health outcomes in economically disadvantaged populations. Similarly, large-scale epidemiological studies comparing urban and rural populations have shown that individuals with lower levels of education are at a significantly higher risk for CVD events and related mortality. This risk is particularly pronounced in low- and middle-income countries, where access to healthcare and preventive services is often limited and where educational attainment is strongly correlated with socioeconomic status [40]. The lack of access to quality education, which typically leads to poorer employment opportunities and financial instability, contributes to a cycle of disadvantage that affects cardiovascular health. Individuals facing these challenges often experience a range of health adversities, including limited access to healthcare resources, fewer support networks, and difficulty in managing chronic diseases. These barriers are compounded by unhealthy behaviors, such as poor dietary habits and lack of exercise, and insufficient financial resources to address health-related needs or to access preventive care [41].

These results highlight the significant association between adverse SDOH and increased mortality risks in individuals with metabolic syndrome. To confirm the true causal efficacy of these interventions, rigorous research, including randomized controlled trials and comprehensive policy evaluations, is required. Such studies will provide stronger evidence on whether changes in social conditions directly lead to improved health and reduced mortality, or if the observed associations are influenced by other underlying factors.

The strength of this study lies in its utilization of data from the NHANES dataset. The reliability and validity of the SDOH score are supported by the robust design of the NHANES dataset, which incorporates a well-established methodology for data collection. The stratified, multistage probability sampling design ensures that the sample is highly representative of the U.S. population, allowing the SDOH score to effectively capture a wide

range of experiences from individuals across various demographic, socioeconomic, and geographical backgrounds. This design not only enhances the generalizability of the results but also provides a more comprehensive understanding of how social determinants of health affect diverse groups. Furthermore, the standardized data collection process, combined with rigorous quality control measures implemented throughout the NHANES survey, serves to minimize potential measurement errors. This attention to detail ensures that the SDOH score accurately reflects the true social determinants of health within the study population. The combination of these features—careful sampling, high-quality data collection, and stringent quality control—provides a solid foundation for the validity of our findings and reinforces the utility of the SDOH score in evaluating the impact of social determinants on mortality outcomes.

Limitations

The present study has several limitations that should be acknowledged. First and foremost, the observational and cross-sectional design of this study limits the ability to draw causal inferences. As a result, the observed associations between SDOH and mortality may be influenced by unmeasured confounding factors. Although we made efforts to adjust for key covariates, factors such as healthcare access dynamics and psychosocial stressors were not fully captured in our analysis, which could potentially impact the validity of the associations observed. These unmeasured variables may introduce biases that affect the interpretation of the results. Second, while we identified significant associations between SDoH and mortality outcomes in patients with MetS, we did not employ formal causal mediation models to explore the pathways through which SDoH might influence these outcomes. The lack of a mediation framework leaves us uncertain about whether the observed associations represent a direct biological effect of SDoH on mortality or whether they are mediated through intermediate factors, such as disease progression, psychosocial factors, or disparities in healthcare access. Without a formal mediation analysis, the precise mechanisms linking SDoH to mortality outcomes remain unclear, and future studies utilizing such models would be beneficial in better understanding these complex relationships. Furthermore, some important SDOH domains, such as experienced racism, discrimination, and social support, were not extensively assessed in NHANES, which could have influenced the outcomes. Therefore, replication of these findings in larger prospective cohort studies is necessary. Finally, the study's results may be most applicable to the U.S. population, and further research is required to assess whether these findings are generalizable to other populations or settings.

Conclusions

In conclusion, our study emphasizes the significant association between SDOH and mortality risk in patients with MetS. These findings highlight the critical importance of considering SDOH in clinical and public health practices, as they may play a substantial role in determining health outcomes. However, given the observational and cross-sectional design of this study, the ability to draw definitive causal conclusions is limited. As such, further research is necessary to confirm these associations and explore potential causal mechanisms. Longitudinal and experimental studies, including causal mediation models, could provide deeper insights into the pathways through which SDOH influence mortality risk and inform interventions aimed at reducing health disparities.

Supplementary Information

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Supplementary Material 1

Supplementary Material 2

Supplementary Material 3

Supplementary Material 4

Supplementary Material 5

Supplementary Material 6

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Data availability

The data supporting the findings of this study are publicly available from the National Health and Nutrition Examination Survey (NHANES) database.

NHANES datasets can be accessed at https://www.cdc.gov/nchs/nhanes/index.htm.

Declarations

Consent for publication
All participants agreed to publish.

Competing interests
The authors declare no competing interests.

Conflict of interest
The authors declare no conflict of interest regarding the publication of this.

Ethics statement
NHANES was implemented by the National Center for Health Statistics of the US Centers for Disease Control and Prevention (CDC) and approved by the National Center for Health Statistics Institutional Review Board. All participants have provided written informed consent.

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