RENAL FAILURE
2025, VOL. 47, NO. 1, 2506811
https://doi.org/10.1080/0886022X.2025.2506811

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CLINICAL STUDY

Social determinants of health and all-cause or cardiovascular mortality in chronic kidney disease: insights from 1999-2018 US National Health and Nutrition Examination Survey

Limin Xu[a], Fengling Chen[b], Ying Huang[b], Bing You[b], Xiaopan Zhang[b] and Bo Sun[b]

[a]Department of General Practice, The First Affiliated Hospital of Soochow University, Suzhou, Jiangsu Province, China; [b]Department of Hemodialysis Center, The First Affiliated Hospital of Soochow University, Suzhou, Jiangsu Province, China

ABSTRACT

Objectives: Unfavorable social risk profile has been identified as a fundamental driver for disparate mortality rate. This study aimed to determine the association between social determinants of health (SDOH) and all-cause or cardiovascular disease (CVD) mortality in patients with chronic kidney disease (CKD).

Methods: Data from adult participants with CKD and available information on SDOH were collected from the 1999-2018 US National Health and Nutrition Examination Survey. SDOH was calculated based on eight factors, including employment, poverty-income ratio, food security, education level, access to healthcare, health insurance, housing instability, and marital status. Cox proportional hazard regression analysis, restricted cubic spline analysis and subgroup analysis were performed.

Results: 5,420 participants (mean age 58.13 years, 43.04% men) were analyzed, including 729, 1,713, 1,849 and 1,129 with a SDOH of ≤ 2, 3-4, 5-6, and 7-8, respectively. Over a median follow-up of 92 months, 1,923 (742 CVD-related) deaths occurred. Compared to the reference group (SDOH of 7-8), the hazard ratios and 95% confidence intervals for those with a SDOH of 5-6, 3-4, and ≤ 2 were 1.25 (1.06-1.48), 1.51 (1.26-1.81) and 2.00 (1.54-2.60), respectively, for all-cause mortality, and 1.38 (1.08-1.77), 1.43 (1.09-1.89), and 1.78 (1.15-2.77), respectively, for CVD mortality. Restricted cubic spline analysis indicated linear dose-response relationships between SDOH levels and all-cause or CVD mortality. The association between SDOH and mortality was more pronounced in men than in women.

Conclusion: Lower SDOH levels are independently associated with higher all-cause and CVD mortality rates among US adults with CKD, especially in men.

ARTICLE HISTORY
Received 29 October 2024
Revised 30 April 2025
Accepted 11 May 2025

KEYWORDS
Cardiovascular; Chronic kidney disease; NHANES; mortality; social determinants of health

1. Introduction

Chronic kidney disease (CKD) represents a significant and prevalent healthcare concern with a considerable morbidity and mortality rate globally. Recent epidemiological studies showed that approximately 1.2 million people died from CKD in 2017, with the mortality rate increasing by 41.5% since 1990 [1]. Although the etiologies for CKD are diverse and multifactorial, studies have shown that managing risk factors like diabetes and hypertension appears to be important in mitigating mortality and improving long-term survival [2]. Therefore, identifying critical factors contributing to the high mortality rate would facilitate the formulation of appropriate intervention measures to improve outcomes in CKD patients.

There is growing evidence showing that inequalities in social determinants of health (SDOH) significantly influence individual health outcomes and are fundamental drivers in the disparate mortality rate among CKD patients through various mechanisms, such as limited access to healthcare, unequal utilization of health resources, and increased exposure to environmental hazards and stress [3]. Earlier studies have indicated that CKD is more prevalent in individuals with poor SDOH and racially disadvantaged ethnic groups [4]. In addition, several studies have identified specific components of SDOH, including socioeconomic status, neighborhood factors, and access to healthcare, as critical factors influencing CKD progression and mortality [5,6]. However, the majority of prior research focused on the relationship between individual

CONTACT Bo Sun sunboman86@163.com Department of Hemodialysis Center, The First Affiliated Hospital of Soochow University, Pinghai Road 899, Suzhou 215006, Jiangsu, China.

Supplemental data for this article can be accessed online at https://doi.org/10.1080/0886022X.2025.2506811.

SDOH components and CKD mortality, leaving the overall impact of SDOH on CKD mortality remains largely unexplored. The work by Ozieh et al. demonstrated that a cumulative social determinant score, derived from factors such as family income to poverty ratio, food insecurity, and depression, was inversely associated with all-cause mortality in individuals with CKD [7]. Nonetheless, SDOH encompasses a multitude of domains, such as educational attainment and marital status, which may also exert a profound influence on CKD mortality [8].

Therefore, the aim of this study is to explore the cumulative and individual contributions of SDOH factors in predicting the risk of all-cause and cardiovascular disease (CVD) mortality among CKD patients.

2. Methods

2.1. Data source

Publicly available data were obtained from the US National Health and Nutrition Examination Survey (NHANES), a biennial nationwide cross-sectional survey to collect information on the health status of non-institutionalized US citizens. A comprehensive and detailed description of the survey methodology and related variables can be accessed at the NHANES website and in relevant publications [9]. For this study, we extracted data from 10 continuous cycles of NHANES spanning from 1999 to 2018. Ethical approval for the NAHNES was reviewed by the US National Center for Health Statistics Institutional Review Board. All adult participants provided informed consent.

2.2. Study population

Adult participants with CKD and available information on SDOH were considered for potential inclusion. Among the 46,691 participants aged between 20 and 85 years with data on SDOH, we applied the following exclusion criteria: 1) pregnancy at the time of the survey ($n = 74$); 2) history of cancer ($n = 1,399$); 3) absence of CKD or unknown CKD status ($n = 38,552$); 4) lost to follow-up ($n = 5$); and 5) missing information on other covariates ($n = 1,241$). The process for participant inclusion and exclusion was illustrated in Figure 1. CKD was diagnosed in participants with an estimated glomerular filtration rate (eGFR) < 60 mL/min/1.73m^2 calculated using the 2009 serum creatinine-based Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation and/or a urinary albumin-to-creatinine ratio > 30 mg/g [10].

2.3. Measurement of SDOH

As reported previously [11,12], the SDOH was measured across 5 domains proposed in the Healthy People 2030 initiative, using standardized questionnaires administered through face-to-face interviews. Specifically, 8 measures were collected, including employment status (employed, or student or retired vs unemployed), family poverty-income ratio (≥ 300% vs < 300%), food security (secure vs insecure), educational attainment (college or above vs high school or lower), access to healthcare (routine place to go for healthcare vs no routine place to go for healthcare), health insurance (private insurance vs government insurance or no insurance), housing instability (own home vs rent or other arrangements), and

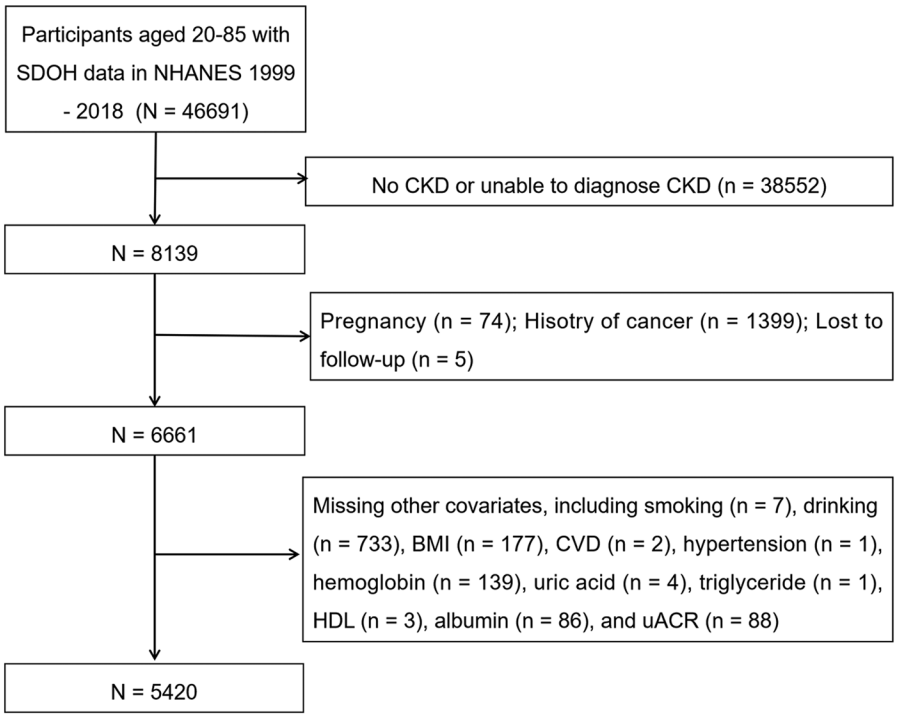

Figure 1. Study flowchart. BMI, body mass index; CKD, chronic kidney disease; CVD, cardiovascular disease; HDL, high-density lipoprotein cholesterol; NHANES, National Health and Nutrition Examination survey; SDOH, social determinants of health; uACR, urinary albumin-to-creatinine ratio.

marital status (married or living with a partner vs not married nor living with a partner). Each measure was dichotomized, with unfavorable and favorable SDOH status assigned scores of 0 and 1, respectively. The methodology employed for the definition and questionnaire of each SDOH measure is the same as reported previously [11,12]. The total SDOH score for each participant ranged from 0 to 8, with a higher score indicating a superior SDOH status.

2.4. All-cause and cardiovascular mortality

Individuals with a linked death record at the National Death Index, irrespective of the underlying causes, were included into the all-cause mortality analysis. Conversely, those without a death record by December 31, 2019 were considered to be alive. For participants who died, those with an International Statistical Classification of Diseases and Related Health Problems, Tenth Revision code I00-I09, I11, I13, and I20-I51, I60-I69 were deemed to have died of CVD [13]. The mortality data were sourced from the National Death Index database.

2.5. Covariates

We considered potential effect modifiers of the SDOH-mortality relationship as covariates. Demographic information (age, sex, and race/ethnicity) and lifestyle factors (smoking, drinking, body mass index and physical activity), were collected through self-report. Race/ethnicity was collected through self-report, and categorized as non-Hispanic white, non-Hispanic black, Mexican Americans, other Hispanic, and other (including multi-racial). Smoking was categorized into never (< 100 cigarettes smoked during lifetime), former (> 100 cigarettes smoked during lifetime but currently not smoking), and current (smoked over 100 cigarettes during lifetime and currently smoking) [14]. Alcohol consumption was categorized into never (< 12 drinks in lifetime), former drinkers (≥12 drinks in lifetime but not in the last year), mild (≤ 1 drink/day for women or ≤ 2 drinks/day for men, on average over the past year), moderate (≤ 2 drinks/day for women, ≤ 3 drinks/day for men, or binge drinking on 2 ~ 5 days per month), and heavy (≥ 3 drinks/day for women, ≥ 4 drinks/day for men, or binge drinking ≥ 5 days per month) [15]. Physical activity levels were defined based on intensity: (1) moderate-intensity: activities causing small increases in breathing/heart rate, and (2) vigorous-intensity: activities causing large increases in breathing/heart rate, performed for ≥ 10 consecutive minutes during work or leisure time in a typical week [16]. In addition to self-report, participants were classified as hypertensive if the blood pressure measured at the Mobile Examination Center ≥ 140/90 mmHg or currently taking antihypertensive medications. Diabetes were determined by physician diagnosis, or a fasting blood glucose ≥ 7.0 mmol/L, or a 2-h post-oral glucose tolerance test plasma glucose ≥ 11.1 mmol/L, or a glycated hemoglobin level ≥ 6.5%, or currently using insulin or oral hypoglycemic medications. CVD history is obtained from the Medical Conditions Questionnaire and defined as those with stroke, congestive heart failure, coronary heart disease, heart attack, or angina. Laboratory tests results collected included hemoglobin, serum albumin, uric acid, triglyceride, total cholesterol and high-density lipoprotein cholesterol.

2.6. Statistical analysis

We stratified participants into 4 groups with SDOH of ≤ 2, 3-4, 5-6, and 7-8 (reference group), and compared the baseline characteristics with one-way analysis of variance or chi-squared test, as appropriate. Kaplan-Meier survival curves were plotted for all-cause and CVD mortality, and the survival status among the 4 groups were compared with the log-rank test. The hazard ratios (HRs) with 95% confidence intervals (CIs) were calculated from the Cox proportional hazards regression model to quantify the risk of all-cause and CVD mortality. The proportional hazards assumption was verified using Schoenfeld residuals test, which confirmed the assumption was met when SDOH was analyzed as a categorical variable. We first fitted a crude model without adjustment for any covariates (Model 1), followed by fitting a partially adjusted model that included participant's age (continuous), sex (men/women), race/ethnicity (non-Hispanic white/non-Hispanic black/Mexican American/Other Hispanics/other), smoking (never/former/current), drinking (never/former/mild/moderate/severe), body mass index (continuous) and physical activity (no/moderate/vigorous) in Model 2. Ultimately, we constructed a fully adjusted model (Model 3) that further included history of CVD (yes/no), hypertension (yes/no), diabetes (yes/no), hemoglobin (yes/no), serum albumin (continuous), uric acid (continuous), triglyceride (continuous), total cholesterol (continuous), high-density lipoprotein cholesterol (continuous), estimated glomerular filtration rate (continuous) and urinary albumin-to-creatinine ratio (continuous). Restricted cubic splines, with knot locations determined by the Akaike information criterion, were utilized to investigate potential nonlinear relationships between SDOH and mortality outcomes using the "MASS" package in R. Specifically, four knots were placed at the 5^{th}, 35^{th}, 65^{th}, and 95^{th} percentiles to flexibly capture the associations between SDOH and mortality outcomes. Subgroup analysis according to participant's age, sex, body mass index, hypertension, diabetes, estimated glomerular filtration rate and urinary albumin-to-creatinine ratio were also conducted to identify any potential interaction effects. To address the complex survey design and oversampling, we applied appropriate weighting to all analyses to obtain nationally representative estimates. A two-sided P value < 0.05 signified statistical significance.

3. Results

3.1. Comparison of baseline characteristics

We included 5,420 participants (mean age 58.13 years, 43.04% men), including 729, 1,713, 1,849 and 1,129 with a SDOH of

≤ 2, 3 – 4, 5 – 6, and 7 – 8, respectively. The mean estimated glomerular filtration rate and urinary albumin-to-creatinine ratio for the 5,420 participants were 76.00 mL/min/1.73m^2 and 181.01 mg/g, respectively. As shown in Table 1, compared to participants with a SDOH of 7-8, those with a SDOH ≤ 2 were significantly younger, more likely to be non-Hispanic black women, more likely to be current smokers and heavy drinkers, more likely to be physically inactive, more likely to have diabetes. In terms of laboratory findings, participants with a SDOH of ≤ 2 had significantly lower hemoglobin, serum albumin, high-density lipoprotein cholesterol, as well as significantly higher estimated glomerular filtration rate and urinary albumin-to-creatinine ratio.

3.2. Kaplan-Meier survival curves

A total of 1,923 (27.91%) participants died during a median follow-up time of 92 (interquartile range 52 – 142) months, of which 742 were CVD mortality. There were 226 (27.37%), 629 (30.74%), 744 (32.17%) and 324 (20.82%) all-cause mortality, and 80 (8.99%), 241 (11.18%), 293 (13.28%) and 128 (7.36%) CVD mortality in the SDOH of ≤ 2, 3 – 4, 5 – 6, and 7 – 8 groups, respectively. The Kaplan-Meier survival curves for all-cause mortality, as shown in Figure 2A, showed that SODH of 7-8 and 5-6 exhibited the highest and lowest survival probability, respectively. Analysis of CVD mortality (Figure 2B) indicated that the survival rate from CVD mortality was highest for the SDOH of 7 – 8 group, followed by SDOH of 5 – 6, 3 – 4 and ≤ 2, in order of decreasing CVD-free survival probability.

3.3. Associations between SDOH and participant survival

The risks of all-cause and CVD mortality in CKD participants, as denoted by the HR and 95% CI, were summarized in Table 2. In the crude analysis, the SDOH 5 – 6 group had the highest all-cause and CVD mortality rate, followed by SDOH 3 – 4 and SDOH ≤ 2 group. When SDOH were analyzed as categorical variables, the HRs (95% CIs) for those with a SDOH of 5 – 6, 3 – 4, and ≤ 2, in comparison to the reference SDOH of 7 – 8 group, were 1.25 (1.06 – 1.48), 1.51 (1.26 – 1.81) and 2.00 (1.54 – 2.60), respectively, for all-cause mortality, and 1.38 (1.08 – 1.77), 1.43 (1.09 – 1.89), and 1.78 (1.15 – 2.77), respectively, for CVD mortality. Restricted cubic spline analysis (Figure 3) indicated a linear dose-response relationship between SDOH and all-cause or CVD mortality.

3.4. Associations between individual SDOH component and mortality

We then analyzed the associations of individual component of SDOH and all-cause or CVD mortality. As shown in Table 3, unemployment, poverty-income ratio < 3, food insecurity, government health insurance or no insurance, and not married or no partner were independent risk factors for increased all-cause mortality, and unemployment, poverty-income ratio < 3, and not married or no partner were independent risk factors for increased CVD mortality.

3.5. Subgroup analysis

Subgroup analysis (Tables 4 and 5) showed that participant's race, body mass index, hypertension, diabetes, estimated glomerular filtration rate, urinary albumin-to-creatinine ratio and CVD status did not appear to modify the associations between SDOH and all-cause or CVD mortality. Notably, the associations between SDOH and all-cause or CVD mortality were stronger in men than in women. In addition, the association between SDOH and CVD mortality was more pronounced in male participants aged < 60 years.

3.6. Sensitivity analysis

In the sensitivity analysis, we excluded participants with follow-up less than 24 months to minimize the risk of reverse causation. The results (Supplementary Table 1) still showed a lower SDOH category is related to a higher risk of all-cause and CVD mortality in the fully adjusted model 3.

4. Discussion

In the current analysis of 5,420 CKD with a median follow-up period of 92 months, we found that compared to individuals with an SDOH score of 7 – 8, those with an SDOH score of ≤ 2 had a 1.0-fold increased risk of all-cause mortality and a 0.78-fold increased risk of CVD mortality. In addition, the associations between SDOH and all-cause or CVD mortality were linear and remained consistent regardless of participant's race, body mass index, hypertension, diabetes, estimated glomerular filtration rate, urinary albumin-to-creatinine ratio, and CVD status. Notably, the associations between SDOH and all-cause or CVD mortality were more pronounced in men compared to women.

Our study found that CKD participants with higher SDOH scores were more likely to be men and less likely to be non-Hispanic Blacks, current smokers, or heavy drinkers, implying significant sex and racial disparities in the social risk profile. This observation may also explain the shift in the trend for the association between SDOH categories and mortality rates from the unadjusted Model 1 to partially adjusted Model 2. Moreover, this study also revealed that compared to those with an SDOH score of 7-8, participants with SDOH scores of 5 – 6, 3 – 4 and ≤ 2 exhibited a 25%, 51%, and 100% increased risk of all-cause mortality, respectively, and a 38%, 43%, and 78% increased risk of CVD mortality. We are aware that there is a growing body of evidence suggesting that poor SDOH had a detrimental impact on mortality in the general population, and some studies have explored the association between SDOH and CKD outcomes using different SDOH measurements. In an analysis of data from the 2006-2018 National Health Interview Survey, Javed et al. found that the highest quintile of SDOH, representing the most unfavorable SDOH, had approximately 181% and 190% increased risk of all-cause and CVD mortality in the general population [17]. Similarly, in the general population from NHANES, Bundy et al. observed that individuals with 1, 2, 3,

Table 1. Comparison of baseline characteristics of participants with chronic kidney disease from the 1999–2018 NHANES stratified by the social determinants of health (SDOH).

	Total ($n=5420$)	SDOH ≤ 2 ($n=729$)	SDOH 3-4 ($n=1713$)	SDOH 5-6 ($n=1849$)	SDOH 7-8 ($n=1129$)	P value
Age, years	58.13 ± 0.34	50.82 ± 0.77	56.16 ± 0.72	61.00 ± 0.56	58.90 ± 0.49	<0.001
Sex (n, %)						<0.001
Male	2590 (43.04)	295 (38.07)	745 (38.93)	922 (43.09)	628 (48.07)	
Female	2830 (56.96)	434 (61.93)	968 (61.07)	927 (56.91)	501 (51.93)	
Race/ethnicity (n, %)						
Non-Hispanic White	2506 (67.96)	160 (36.19)	620 (54.67)	1009 (72.82)	717 (84.01)	<0.001
Non-Hispanic Black	1220 (12.82)	243 (26.92)	426 (17.58)	367 (10.91)	184 (6.39)	
Mexican American	907 (7.68)	180 (17.44)	395 (12.56)	246 (5.71)	86 (2.62)	
Other Hispanic	407 (5.36)	100 (12.62)	143 (7.46)	112 (4.51)	52 (2.17)	
Other	380 (6.18)	46 (6.83)	129 (7.73)	115 (6.05)	90 (4.81)	
Smoking (n, %)						<0.001
Never	2704 (50.63)	316 (40.79)	822 (46.42)	937 (51.34)	629 (56.60)	
Former	1719 (30.74)	155 (18.37)	503 (26.90)	668 (33.78)	393 (34.55)	
Current	997 (18.63)	258 (40.84)	388 (26.68)	244 (14.88)	107 (8.85)	
Drinking (n, %)						<0.001
Never	962 (15.30)	150 (18.49)	362 (19.02)	329 (16.96)	121 (9.22)	
Former	1424 (22.01)	179 (22.39)	518 (27.01)	526 (24.33)	201 (15.01)	
Mild	1677 (34.31)	121 (16.53)	396 (23.08)	607 (33.45)	553 (50.55)	
Moderate	587 (13.54)	71 (10.40)	184 (12.54)	174 (12.49)	158 (16.64)	
Heavy	770 (14.84)	208 (32.19)	253 (18.35)	213 (12.77)	96 (8.57)	
Physical activity (n, %)						0.003
No	3350 (56.13)	494 (61.61)	1121 (59.51)	1127 (55.84)	608 (51.84)	
Moderate	1235 (25.01)	125 (19.96)	355 (23.12)	458 (27.04)	297 (25.93)	
Vigorous	835 (18.86)	110 (18.43)	237 (17.38)	264 (17.11)	224 (22.23)	
Hypertension (n, %)	4254 (73.63)	570 (72.87)	1343 (72.91)	1484 (76.38)	857 (71.32)	0.082
Diabetes (n, %)	2094 (32.98)	318 (39.44)	740 (37.52)	679 (32.72)	357 (27.35)	<0.001
CVD (n, %)	1339 (20.96)	242 (17.17)	191 (23.33)	438 (23.47)	468(21.75)	0.003
CVD subtypes						
Coronary heart disease	525 (8.82)	135 (9.95)	55 (6.01)	132 (7.14)	203 (10.04)	0.014
Congestive heart failure	478 (7.03)	65 (3.79)	78 (9.08)	175 (9.34)	160 (7.67)	<0.001
Heart attack	576 (8.68)	108 (7.56)	78 (7.89)	178 (9.29)	212 (9.49)	0.295
Stroke	470 (7.27)	67 (4.76)	71 (8.84)	171 (9.41)	161 (7.46)	<0.001
Angina	328 (5.73)	69 (5.42)	35 (5.24)	113 (6.80)	111 (5.42)	0.564
BMI, kg/m^2	30.25 ± 0.15	30.94 ± 0.38	30.48 ± 0.30	29.93 ± 0.21	30.19 ± 0.29	0.080
Dialysis (n, %)	58 (0.77)	5 (0.75)	11 (4.59)	25 (5.40)	17 (2.75)	0.002
Hemoglobin, g/dL	14.01 ± 0.04	13.85 ± 0.09	13.85 ± 0.07	14.04 ± 0.06	14.18 ± 0.05	<0.001
Albumin, g/dL	4.19 ± 0.01	4.13 ± 0.02	4.16 ± 0.01	4.19 ± 0.01	4.24 ± 0.01	<0.001
Uric acid, mg/dL	5.93 ± 0.03	5.80 ± 0.09	5.93 ± 0.05	5.98 ± 0.05	5.91 ± 0.06	0.344
Triglycerides, mg/dL	177.47 ± 3.51	184.61 ± 7.64	176.37 ± 4.62	185.26 ± 7.82	167.14 ± 5.20	0.108
Total cholesterol, mg/dL	197.68 ± 0.91	202.72 ± 2.43	197.43 ± 1.33	199.02 ± 1.58	194.70 ± 1.83	0.058
HDL, mg/dL	52.27 ± 0.38	51.27 ± 0.81	52.63 ± 0.65	51.14 ± 0.46	53.60 ± 0.88	0.044
eGFR, ml/min/1.73m^2	76.00 ± 0.55	87.24 ± 1.48	79.02 ± 1.07	72.69 ± 0.85	73.58 ± 0.95	<0.001
uACR, mg/g	181.01 ± 9.62	244.49 ± 26.54	215.87 ± 18.04	186.71 ± 19.37	124.36 ± 12.43	<0.001
CKD stages						<0.001
Stage 1	1700 (35.35)	343 (51.60)	570 (40.75)	478 (30.77)	309 (30.74)	
Stage 2	1293 (22.41)	164 (22.10)	423 (21.51)	431 (21.96)	275 (23.80)	
Stage 3	2188 (39.12)	191 (23.16)	632 (33.02)	853 (44.08)	512 (43.79)	
Stage 4	168 (2.22)	14 (1.45)	62 (3.18)	67 (2.41)	25 (1.42)	
Stage 5	71 (0.90)	17 (1.69)	26 (1.54)	20 (0.78)	8 (0.25)	
Individual component of SDOH						
Employment (n, %)						<0.001
Employed	4170 (79.43)	229 (33.42)	1178 (65.40)	1670 (89.01)	1093 (95.35)	
Unemployed	1250 (20.57)	500 (66.58)	535 (34.60)	179 (10.99)	36 (4.65)	
Poverty-income ratio, (n, %)						<0.001
≥3	1510 (38.94)	4 (0.71)	62 (4.76)	475 (30.90)	969 (89.44)	
<3	3910 (61.06)	725 (99.29)	1651 (95.24)	1374 (69.10)	160 (10.56)	
Food security (n, %)						<0.001
Secure	3909 (77.41)	140 (19.78)	1027 (60.28)	1634 (87.96)	1108 (98.60)	
Insecure	1511 (22.59)	589 (80.22)	686 (39.72)	215 (12.04)	21 (1.40)	
Education attainment (n, %)						<0.001
College or above	2205 (49.53)	73 (11.53)	420 (29.05)	777 (44.59)	935 (84.86)	
High school or lower	3215 (50.47)	656 (88.47)	1293 (70.95)	1072 (55.41)	194 (15.14)	
Healthcare (n, %)						<0.001
Routine place to go for healthcare	4797 (88.65)	464 (59.04)	1470 (83.11)	1752 (93.75)	1111 (97.18)	
No routine place for healthcare	623 (11.35)	265 (40.96)	243 (16.89)	97 (6.25)	18 (2.82)	
Health insurance (n, %)						<0.001
Private insurance	2591 (57.67)	20 (2.95)	376 (26.97)	1182 (66.57)	1013 (91.24)	
Government insurance or no insurance	2829 (42.33)	709 (97.05)	1337 (73.03)	667 (33.43)	116 (8.76)	
Housing instability (n, %)						<0.001
Own a home	3568 (70.75)	108 (13.99)	860 (48.73)	1506 (80.26)	1094 (97.00)	
Rent or other arrangements	1852 (29.25)	621 (86.01)	853 (51.27)	343 (19.74)	35 (3.00)	

(*Continued*)

Table 1. Continued.

	Total ($n=5420$)	SDOH ≤ 2 ($n=729$)	SDOH 3-4 ($n=1713$)	SDOH 5-6 ($n=1849$)	SDOH 7-8 ($n=1129$)	P value
Marital status (n, %)						<0.001
Married or partnered	2957 (59.15)	155 (22.28)	697 (40.25)	1108 (57.15)	997 (89.43)	
Not married or no partner	2463 (40.85)	574 (77.72)	1016 (59.75)	741 (42.85)	132 (10.57)	

BMI, body mass index; CVD, cardiovascular disease; eGFR, estimated glomerular filtration rate; HDL, high-density lipoprotein cholesterol; SDOH, social determinants of health; uACR, urinary albumin-to-creatinine ratio. P denotes the comparisons among the 4 SDOH subgroups.

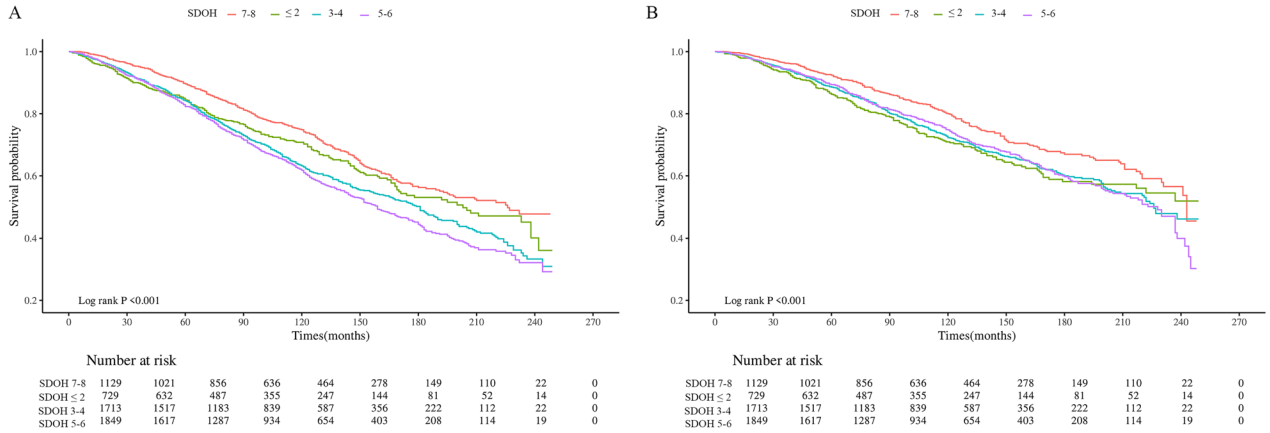

Figure 2. Kaplan-Meier curves for all-cause (A) and cardiovascular (B) mortality in participants with chronic kidney disease stratified by social determinants of health.

Table 2. Associations of social determinants of health and all-cause or cardiovascular mortality in participants with chronic kidney disease from the 1999–2018 NHANES.

	Model 1		Model 2		Model 3	
	HR (95% CI)	P	HR (95% CI)	P	HR (95% CI)	P
All-cause mortality						
SDOH 7-8	1 (Reference)	/	1 (Reference)	/	1 (Reference)	/
SDOH 5-6	1.69 (1.43–1.99)	<0.001	1.31 (1.12–1.53)	<0.001	1.25 (1.06–1.48)	0.008
SDOH 3-4	1.64 (1.39–1.93)	<0.001	1.61 (1.37–1.90)	<0.001	1.51 (1.26–1.81)	<0.001
SDOH ≤2	1.49 (1.22–1.83)	<0.001	2.19 (1.73–2.78)	<0.001	2.00 (1.54–2.60)	<0.001
Cardiovascular mortality						
SDOH 7-8	1 (Reference)	/	1 (Reference)	/	1 (Reference)	/
SDOH 5-6	1.97 (1.53–2.53)	<0.001	1.463 (1.15–1.87)	0.002	1.38 (1.08–1.77)	0.011
SDOH 3-4	1.69 (1.28–2.23)	<0.001	1.59 (1.23–2.07)	<0.001	1.43 (1.09–1.89)	0.011
SDOH ≤2	1.38 (0.96–1.98)	0.08	2.04 (1.34–3.09)	<0.001	1.78 (1.15–2.77)	0.01

Model 1 was unadjusted; Model 2 was adjusted for participant's age, sex, race, smoking, drinking, body mass index and physical activity; Model 3 was adjusted for Model 2, plus hypertension, diabetes, hemoglobin, serum albumin, uric acid, triglycerides, total cholesterol, high-density lipoprotein cholesterol, cardiovascular disease, estimated glomerular filtration rate and urinary albumin-to-creatinine ratio.
CI, confidence interval; HR, hazard ratio; SDOH, social determinants of health.

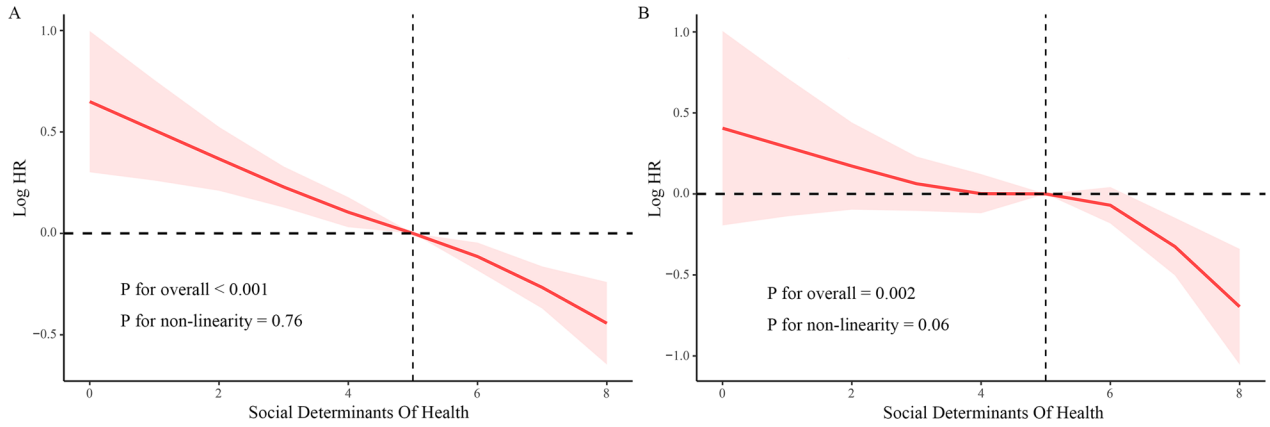

Figure 3. Restricted cubic spline analysis results showing the dose-response relationship between social determinants of health (SDOH) and all-cause (A) or cardiovascular (B) mortality in participants with chronic kidney disease.

Table 3. Associations of individual components of social determinants of health and all-cause or cardiovascular mortality in participants with chronic kidney disease from the 1999-2018 NHANES.

	All-cause mortality		Cardiovascular mortality	
Individual components of social determinants of health	HR (95% CI)	P value	HR (95% CI)	P value
Employment (Unemployed *vs* Employed)	1.66 (1.40–1.96)	<0.001	1.61 (1.19–2.20)	0.002
Poverty-income ratio (< 3 *vs* ≥ 3)	1.39 (1.22–1.60)	<0.001	1.40 (1.12–1.75)	0.003
Food security (Insecure *vs* Secure)	1.36 (1.15–1.60)	<0.001	1.06 (0.84–1.33)	0.648
Education (High school or lower *vs* College or above)	1.06 (0.95–1.17)	0.315	1.08 (0.90–1.28)	0.423
Healthcare (No routine place *vs* Routine place to go)	1.15 (0.88–1.51)	0.305	0.91 (0.59–1.40)	0.657
Health insurance (Government or no insurance *vs* Private insurance)	1.18 (1.05–1.33)	0.005	1.16 (0.94–1.42)	0.173
Housing instability (Rent or other arrangements *vs* Own a home)	1.12 (0.97–1.30)	0.118	1.09 (0.87–1.36)	0.480
Marital status (Not married or no partner *vs* Married or partnered)	1.34 (1.17–1.52)	<0.001	1.35 (1.11–1.64)	0.002

The HR was adjusted for participant's age, sex, race, smoking, drinking, body mass index and physical activity, hypertension, diabetes, hemoglobin, serum albumin, uric acid, triglycerides, total cholesterol, high-density lipoprotein cholesterol, cardiovascular disease, estimated glomerular filtration rate and urinary albumin-to-creatinine ratio. CI, confidence interval; HR, hazard ratio.

Table 4. Subgroup analysis for the associations between social determinants of health (SDOH) and all-cause mortality in US adults with chronic kidney disease.

	SDOH ≤ 2	P	SDOH 3–4	P	SDOH 5-6	P	SDOH 7-8	P for interaction
Age, years								0.18
< 60	2.27 (1.13–4.58)	0.02	1.44 (0.78–2.67)	0.25	1.44 (0.81–2.57)	0.22	Reference	
≥ 60	1.68 (1.27–2.22)	<0.001	1.38 (1.14–1.68)	0.001	1.12 (0.95–1.31)	0.17	Reference	
Sex								0.003
Male	2.45 (1.69–3.55)	<0.001	1.45 (1.10–1.91)	0.009	1.56 (1.25–1.94)	<0.001	Reference	
Female	1.56 (1.08–2.25)	0.02	1.36 (1.03–1.78)	0.03	0.92 (0.70–1.20)	0.52	Reference	
Race/ethnicity								0.36
Mexican American	0.84 (0.37–1.92)	0.68	0.55 (0.26–1.14)	0.11	0.46 (0.21–0.99)	0.05	Reference	
Non-Hispanic Black	2.01 (1.31–3.09)	0.001	1.42 (0.92–2.18)	0.11	1.30 (0.83–2.04)	0.25	Reference	
Non-Hispanic White	2.00 (1.32–3.03)	0.001	1.53 (1.23–1.89)	<0.001	1.23 (1.02–1.47)	0.03	Reference	
Other Hispanic	1.39 (0.47–4.10)	0.55	1.69 (0.65–4.36)	0.28	0.96 (0.36–2.59)	0.94	Reference	
Other	5.48 (1.28–23.54)	0.02	2.06 (0.61–6.88)	0.24	1.57 (0.48–5.13)	0.45	Reference	
Body mass index, kg/m^2								0.26
< 25	3.06 (1.84–5.08)	<0.001	1.65 (1.16–2.36)	0.006	1.22 (0.89–1.67)	0.21	Reference	
≥ 25, < 30	1.41 (0.92–2.17)	0.12	1.21 (0.87–1.69)	0.26	1.08 (0.81–1.45)	0.59	Reference	
≥ 30	1.98 (1.22–3.21)	0.006	1.57 (1.12–2.20)	0.009	1.45 (1.06–1.98)	0.02	Reference	
Hypertension								0.14
No	3.14 (1.46–6.77)	0.003	1.91 (1.13–3.24)	0.02	1.14 (0.77–1.70)	0.51	Reference	
Yes	1.86 (1.41–2.46)	<0.001	1.42 (1.17–1.72)	<0.001	1.24 (1.02–1.49)	0.03	Reference	
Diabetes								0.99
No	1.84 (1.34–2.52)	<0.001	1.41 (1.13–1.76)	0.003	1.18 (0.96–1.44)	0.11	Reference	
Yes	1.97 (1.25–3.11)	0.003	1.54 (1.13–2.10)	0.007	1.28 (0.94–1.73)	0.12	Reference	
Estimated glomerular filtration rate, mL/min/1.73 m^2								0.30
< 30	2.95 (1.32–6.62)	0.009	1.90 (1.00–3.61)	0.05	1.88 (1.10–3.23)	0.02	Reference	
30–59	1.64 (1.17–2.28)	0.004	1.33 (1.05–1.69)	0.02	1.14 (0.94–1.37)	0.19	Reference	
60–89	2.48 (1.56–3.92)	<0.001	1.74 (1.21–2.49)	0.003	1.66 (1.16–2.39)	0.006	Reference	
≥ 90	1.29 (0.69–2.39)	0.43	0.85 (0.46–1.57)	0.60	0.90 (0.50–1.64)	0.74	Reference	
Urinary albumin-to-creatine ratio, mg/g								0.20
≤ 300	2.08 (1.57–2.75)	<0.001	1.50 (1.25–1.81)	<0.001	1.23 (1.04–1.45)	0.01	Reference	
> 300	1.29 (0.62–2.68)	0.50	1.29 (0.71–2.36)	0.41	1.36 (0.78–2.38)	0.28	Reference	
Cardiovascular disease								0.84
No	2.09 (1.49–2.93)	<0.001	1.61 (1.25–2.07)	<0.001	1.26 (1.00–1.57)	0.05	Reference	
Yes	1.87 (1.22–2.88)	0.004	1.35 (0.98–1.86)	0.07	1.26 (0.99–1.61)	0.06	Reference	

4, 5, and ≥ 6 unfavorable SDOH components had a 93%, 124%, 298%, 378%, 508% and 682% increased risk of premature all-cause mortality compared to those without unfavorable SDOH [18]. In another study, Hundermer and colleagues observed that education below high school, unemployment and single status were independently associated with an increased likelihood of inpatient dialysis initiation and lower odds of preemptive access creation [19]. Additionally, Ozieh et al. showed that in adults with diabetic kidney disease, each one-point increase in SDOH, as measured by family income-to-poverty ratio, depression and food insecurity, was associated with a 41% increased risk of all-cause mortality [7].

It should be noted that the impact of SDOH on mortality may differ significantly between the general population and individuals with chronic disease [20]. The results of the current study not only further corroborated findings in the general population, but also uncovered that the contribution of individual components of SDOH to mortality varied substantially. Specifically, we showed that unemployment, poverty-income ratio < 3, and being unmarried or without a partner were independently associated with both all-cause and CVD mortality, whereas food insecurity, and having government health insurance or no insurance were only linked to elevated all-cause mortality. The impact of income, as exemplified by the poverty-income ratio, on the health outcomes of CKD patients can be both direct (limited healthcare access) and indirect (food insecurity) [21]. For instance, the Reasons for Geographic and Racial Differences in Stroke study showed

Table 5. Subgroup analysis for the associations between social determinants of health (SDOH) and cardiovascular mortality in US adults with chronic kidney disease.

	SDOH ≤ 2	P	SDOH 3-4	P	SDOH 5-6	P	SDOH 7-8	P for interaction
Age, years								0.02
< 60	3.99 (1.03–15.39)	0.05	2.50 (0.71–8.76)	0.15	3.22 (1.10-9.39)	0.03	Reference	
≥ 60	1.23 (0.77–1.97)	0.38	1.20 (0.91–1.59)	0.20	1.15 (0.89-1.49)	0.27	Reference	
Sex								0.006
Male	1.76 (1.04–2.98)	0.04	1.15 (0.74–1.80)	0.53	1.63 (1.17-2.27)	0.004	Reference	
Female	1.53 (0.80–2.92)	0.19	1.41 (0.90–2.22)	0.13	1.05 (0.68-1.62)	0.83	Reference	
Race/ethnicity								0.17
Mexican American	2.07 (0.43–9.94)	0.36	2.43 (0.56–10.54)	0.24	0.68 (0.14-3.23)	0.63	Reference	
Non-Hispanic Black	2.11 (1.14–3.91)	0.02	1.80 (0.97–3.34)	0.06	1.45 (0.76-2.77)	0.26	Reference	
Non-Hispanic White	1.36 (0.64–2.86)	0.42	2.00 (1.44–2.78)	<0.001	2.27 (1.74-2.96)	<0.001	Reference	
Other Hispanic	1.04 (0.18–5.88)	0.97	0.84 (0.17–4.15)	0.83	0.70 (0.12-4.05)	0.69	Reference	
Other	10.99 (1.05–115.50)	0.05	9.22 (0.95–89.95)	0.06	8.89 (0.95-83.24)	0.06	Reference	
Body mass index, kg/m^2								0.18
< 25	3.07 (1.33–7.05)	0.008	1.77 (0.95–3.30)	0.07	1.48 (0.81-2.71)	0.20	Reference	
≥ 25, < 30	1.04 (0.51–2.10)	0.92	0.94 (0.57–1.55)	0.81	1.04 (0.69-1.57)	0.84	Reference	
≥ 30	1.85 (0.84–4.05)	0.13	1.76 (1.05–2.96)	0.03	1.81 (1.19-2.76)	0.006	Reference	
Hypertension								0.45
No	1.01 (0.29–3.50)	0.98	1.75 (0.76–4.06)	0.19	1.47 (0.74-2.92)	0.27	Reference	
Yes	1.94 (1.24–3.02)	0.003	1.40 (1.06–1.84)	0.02	1.38 (1.07-1.77)	0.01	Reference	
Diabetes								0.60
No	1.42 (0.81–2.51)	0.23	1.35 (0.93–1.94)	0.11	1.17 (0.84-1.63)	0.34	Reference	
Yes	2.03 (1.04–3.97)	0.04	1.50 (0.93–2.41)	0.09	1.79 (1.19-2.69)	0.005	Reference	
Estimated glomerular filtration rate, mL/min/1.73 m^2								0.93
< 30	1.16 (0.39–3.45)	0.79	1.05 (0.44–2.54)	0.91	1.15 (0.57-2.30)	0.69	Reference	
30-60	2.08 (1.15–3.76)	0.02	2.04 (1.42–2.94)	<0.001	2.00 (1.47-2.72)	<0.001	Reference	
60-90	1.79 (0.97–3.31)	0.06	2.31 (1.48–3.60)	<0.001	2.12 (1.27-3.54)	0.004	Reference	
≥ 90	2.06 (0.74–5.79)	0.17	1.44 (0.55–3.82)	0.46	1.52 (0.51-4.60)	0.46	Reference	
Urinary albumin-to-creatinine ratio, mg/g								0.77
≤ 300	1.77 (1.12–2.80)	0.02	1.43 (1.06–1.92)	0.02	1.34 (1.01-1.78)	0.04	Reference	
> 300	1.92 (0.71–5.19)	0.20	1.03 (0.40–2.66)	0.95	1.74 (0.82-3.69)	0.15	Reference	
Cardiovascular disease								0.20
No	2.50 (1.42–4.39)	0.001	1.73 (1.19–2.53)	0.004	1.34 (0.94-1.92)	0.104	Reference	
Yes	1.10 (0.61–2.00)	0.75	1.10 (0.71–1.71)	0.66	1.45 (1.02-2.06)	0.04	Reference	

that individuals with lower household incomes (annual income < 20,000 dollars) had approximately 58% increased risk of all-cause mortality than those with annual incomes > 20,000 dollars [22]. With regard to unemployment, studies have indicated a reciprocal association between CKD and employment, with CKD patients had 110% increased likelihood of unemployment and even temporary unemployment could increase the risk of all-cause mortality by 76% in the general population [23,24]. Indeed, in addition to the biological pathways linked to socioeconomic deprivation, SDOH components have also been shown to expose individuals to chronic psychosocial stressors and inflammatory states [25,26], which accelerate CKD progression and increase mortality rates.

We observed sex-specific associations between SDOH categories and all-cause or CVD mortality, which were more pronounced in men than in women. Previous research has indicated that US men and women exhibit distinct social risk profiles, with women demonstrating greater vulnerability to poverty, depression, and lack of partnership compared to men [27]. Although men generally have better SDOH profiles, a prior meta-analysis of 68 studies showed that men with CKD of various etiologies experience a faster decline in renal function over time than women [28]. This discrepancy suggests that a broader range of factors beyond SDOH, such as the extent of risk factor control and treatment effectiveness, may interact in a synergistic manner to determine mortality risk in patients with CKD [2]. Although race and ethnicity have also been identified as factors influencing mortality rates in CKD patients [22], and some researchers advocate for their inclusion in SDOH measures [29], we did not observe significant interactions among participants from different racial/ethnic groups in the current study. Notably, the study by Derose's group suggested that White individuals may even experience higher mortality rates than Black individuals among patients with stage 3 to 4 CKD [30]. Therefore, further research is needed to conclusively determine the impact of race and ethnicity on the prognosis of CKD patients.

Disparities exist between age subgroups with regard to SDOH and CVD mortality. Specifically, younger patients with CKD were more significantly impacted by adverse SDOH in terms of CVD mortality compared to older individuals. The exact mechanisms underlying this age difference remains poorly understood. Available literature suggests that compared to the non-elderly, elderly patients with CKD tend to have worse management of risk factors, such as hypertension and diabetes, compared to younger patients [31], which may be a more important factor for determining CVD mortality than SDOH. This finding highlight the importance of appropriate SDOH screening and interventions targeting the non-elderly population to reduce CVD mortality.

Another interesting observation is that the associations between SDOH with all-cause and CVD mortality were statistically insignificant in participants with an estimated

glomerular filtration rate ≥ 90 mL/min/1.73 m^2. The attenuation of SDOH impact in this subgroup may be attributed to several potential mechanisms. First, these individuals likely possess greater physiological reserve, rendering them less vulnerable to SDOH-related stressors compared to those with more severe renal impairment. Second, the detrimental effects of poorer SDOH may require longer exposure duration to manifest clinically, potentially exceeding our study's follow-up period. Third, their preserved kidney function may enable better homeostatic adaptation to SDOH challenges than patients with advanced renal disease.

We believe that our findings have important implications for both healthcare providers and policymakers. It is imperative for clinicians to be aware of this association and proactively screen for SDOH and provide contextualized care to fit specific social needs. Correspondingly, policymakers should make appropriate policies to motivate clinicians to actively screen SDOH and addresses broader issues like food insecurity at the population level.

The strengths of this study include use of nationally representative data and a detailed analysis of the impact of individual SDOH components on CKD mortality. However, the findings of this study should be interpreted in recognition of its potential limitations. First, the SDOH measures assessed in the current study are not exhaustive as other measures, such as social support, geographic remoteness, interpersonal relationships, religious beliefs, health literacy, air pollution, and community resources, may also influence CKD progression or mortality [6,32,33]. Second, this study represents a mortality follow-up analysis of a cross-sectional survey. The availability of longitudinal data on SDOH and its dynamic changes over time limit our ability to capture more nuanced understanding of its relationship with mortality. Third, our study lacked data on CKD-specific treatments that may also influence mortality. Specifically, participants who progressed to end-stage kidney disease, requiring dialysis or kidney transplantation, may have received different treatment regimens and exhibited distinct SDOH profiles. However, we speculated that this potential bias would be minimal, given that the majority of included participants had only mild to moderate CKD (mean estimated glomerular filtration rate of 76 mL/min/1.73 m^2, with only 3.12% at stage 4 or 5). Finally, given the wide heterogeneity of SDOH across countries and ethnic groups, the findings of this study are specific to the US population and external extrapolation to other countries or races should be cautious.

5. Conclusion

In conclusion, this nationwide, representative study showed that lower levels of SDOH are associated with an increased risk of all-cause and CVD mortality among patients with CKD, especially in men. In addition, the contributions of each component of the SDOH measures to mortality vary significantly. The association between SDOH and CVD mortality was more pronounced in participants under 60 years of age. It is imperative that clinicians are aware of this relationship and mitigating SDOH inequalities may help to improve survival.

Disclosure statement

No potential conflict of interest was reported by the author(s).

Ethical approval

This study uses data from a publicly available database, in which all study participants gave informed consent in accordance with the Institutional Review Board and study ethic guidelines at the Centers for Disease Control and Prevention

Funding

The author(s) reported there is no funding associated with the work featured in this article.

Data availability statement

The dataset was based on the NHANES, which is publicly available and could be found at https://www.cdc. gov/nchs/nhanes/.

References

[1] GBD Chronic Kidney Disease Collaboration. Global, regional, and national burden of chronic kidney disease, 1990-2017: a systematic analysis for the Global Burden of Disease Study 2017. Lancet. 2020;395(10225):709–733. doi: 10.1016/S0140-6736(20)30045-3.

[2] Geng T, Xu W, Gao H, et al. Relationship between control of cardiovascular risk factors and chronic kidney disease progression, cardiovascular disease events, and mortality in Chinese adults. J Am Coll Cardiol. 2024;84(14):1313–1324. doi: 10.1016/j.jacc.2024.06.041.

[3] Deng K, Xu M, Sahinoz M, et al. Associations of neighborhood sociodemographic environment with mortality and circulating metabolites among low-income black and white adults living in the southeastern United States. BMC Med. 2024;22(1):249. doi: 10.1186/s12916-024-03452-6.

[4] Chu CD, Powe NR, McCulloch CE, et al. Trends in chronic kidney disease care in the US by race and ethnicity, 2012-2019. JAMA Netw Open. 2021;4(9):e2127014. doi: 10.1001/jamanetworkopen.2021.27014.

[5] Anandh U, Meena P, Karam S, et al. Social, political and legal determinants of kidney health: perspectives from lower- and middle-income countries with a focus on India. Front Nephrol. 2022;2:1024667. doi: 10.3389/fneph.2022.1024667.

[6] Motairek I, Sharara J, Makhlouf MHE, et al. Association between particulate matter pollution and CKD mortality by social deprivation. Am J Kidney Dis. 2023;81(4):497–499. doi: 10.1053/j.ajkd.2022.09.015.

[7] Ozieh MN, Garacci E, Walker RJ, et al. The cumulative impact of social determinants of health factors on mortality in adults with diabetes and chronic

kidney disease. BMC Nephrol. 2021;22(1):76. doi: 10.1186/s12882-021-02277-2.
[8] Grant CH, Salim E, Lees JS, et al. Deprivation and chronic kidney disease-a review of the evidence. Clin Kidney J. 2023;16(7):1081–1091. doi: 10.1093/ckj/sfad028.
[9] Kong X, Wang W. Prevalence of individuals with diabetes and chronic kidney disease indicated for finerenone treatment in the United States: a National Health and Nutrition Examination Survey-based estimation. Diabetes Obes Metab. 2025;27(1):435–437.
[10] Levey AS, Stevens LA, Schmid CH, et al. A new equation to estimate glomerular filtration rate. Ann Intern Med. 2009;150(9):604–612. doi: 10.7326/0003-4819-150-9-200905050-00006.
[11] Li J, Lei L, Wang W, et al. Social risk profile and cardiovascular-kidney-metabolic syndrome in US adults. J Am Heart Assoc. 2024;13(16):e034996. doi: 10.1161/JAHA.124.034996.
[12] Liang JH, Liu ML, Pu YQ, et al. Contribution of individual and cumulative social determinants of health underlying gender disparities in periodontitis in a representative US population: a cross-sectional NHANES study. J Clin Periodontol. 2024;51(5):558–570. doi: 10.1111/jcpe.13941.
[13] Kong X, Wang W. Association between Life's Essential 8 and all-cause or cardiovascular-specific mortality in patients with rheumatoid arthritis. Clin Exp Rheumatol. 2024;42(7):1459–1466. doi: 10.55563/clinexprheumatol/ppsp71.
[14] Du R, Tang X, Yang C, et al. Association between the duration of smoking cessation and α-Klotho levels in the US middle-aged and elderly population. Heliyon. 2024;10(19):e38298. doi: 10.1016/j.heliyon.2024.e38298.
[15] Zhou Q, Zhou L, Chen X, et al. Composite dietary antioxidant index is associated with reduced prevalence of metabolic syndrome but not mortality in metabolic syndrome: results from NHANES 2001-2018. Prev Med Rep. 2024;41:102704. doi: 10.1016/j.pmedr.2024.102704.
[16] Soini E, Rosenström T, Määttänen I, et al. Physical activity and specific symptoms of depression: a pooled analysis of six cohort studies. J Affect Disord. 2024;348:44–53. doi: 10.1016/j.jad.2023.12.039.
[17] Javed Z, Valero-Elizondo J, Cainzos-Achirica M, et al. Race, social determinants of health, and risk of all-cause and cardiovascular mortality in the United States. J Racial Ethn Health Disparities. 2024;11(2):853–864. doi: 10.1007/s40615-023-01567-9.
[18] Bundy JD, Mills KT, He H, et al. Social determinants of health and premature death among adults in the USA from 1999 to 2018: a national cohort study. Lancet Public Health. 2023;8(6):e422–e431. doi: 10.1016/S2468-2667(23)00081-6.
[19] Hundemer GL, Ravani P, Sood MM, et al. Social determinants of health and the transition from advanced chronic kidney disease to kidney failure. Nephrol Dial Transplant. 2023;38(7):1682–1690. doi: 10.1093/ndt/gfac302.
[20] Embury J, Tsou MH, Nara A, et al. A spatio-demographic perspective on the role of social determinants of health and chronic disease in determining a population's vulnerability to COVID-19. Prev Chronic Dis. 2022;19:E38. doi: 10.5888/pcd19.210414.
[21] Chang TI, Lim H, Park CH, et al. Association between income disparities and risk of chronic kidney disease: a nationwide cohort study of seven million adults in Korea. Mayo Clin Proc. 2020;95(2):231–242. doi: 10.1016/j.mayocp.2019.09.028.
[22] Fedewa SA, McClellan WM, Judd S, et al. The association between race and income on risk of mortality in patients with moderate chronic kidney disease. BMC Nephrol. 2014;15(1):136. doi: 10.1186/1471-2369-15-136.
[23] Choi S, Jang SY, Choi E, et al. Association between prevalence and severity of chronic kidney disease and employment status: a nationwide study in Korea. BMC Public Health. 2024;24(1):216. doi: 10.1186/s12889-023-17338-4.
[24] Nie J, Wang J, Aune D, et al. Association between employment status and risk of all-cause and cause-specific mortality: a population-based prospective cohort study. J Epidemiol Community Health. 2020;74(5):428–436. doi: 10.1136/jech-2019-213179.
[25] Baumer Y, Pita MA, Baez AS, et al. By what molecular mechanisms do social determinants impact cardiometabolic risk? Clin Sci (Lond). 2023;137(6):469–494. doi: 10.1042/CS20220304.
[26] Vasunilashorn SM, Wolfson E, Berger M, et al. Multi-level social determinants of health, inflammation, and postoperative delirium in older adults. J Am Geriatr Soc. 2025;73(1):279–282. doi: 10.1111/jgs.19159.
[27] Byhoff E, Tripodis Y, Freund KM, et al. Gender differences in social and behavioral determinants of health in aging adults. J Gen Intern Med. 2019;34(11):2310–2312. doi: 10.1007/s11606-019-05225-x.
[28] Neugarten J, Acharya A, Silbiger SR. Effect of gender on the progression of nondiabetic renal disease: a meta-analysis. J Am Soc Nephrol. 2000;11(2):319–329. doi: 10.1681/ASN.V112319.
[29] Coots M, Linn KA, Goel S, et al. Racial bias in clinical and population health algorithms: A critical review of current debates. Annu Rev Public Health. 2025;46(1):507–523. doi: 10.1146/annurev-publhealth-071823-112058.
[30] Derose SF, Rutkowski MP, Crooks PW, et al. Racial differences in estimated GFR decline, ESRD, and mortality in an integrated health system. Am J Kidney Dis. 2013;62(2):236–244. doi: 10.1053/j.ajkd.2013.01.019.
[31] Liang S, Wang Y, Wang WL, et al. Characteristics of hospitalized elderly patients with CKD: a comparison between elderly and non-elderly CKD based on a multicenter cross-sectional study. Int Urol Nephrol. 2024;56(2):625–633. doi: 10.1007/s11255-023-03675-1.
[32] Slaven A, Hsu J, Schelling JR, et al. Social support in older adults with CKD: A report from the CRIC (Chronic Renal Insufficiency Cohort) study. Kidney Med. 2021;3(5):776–784.e1. doi: 10.1016/j.xkme.2021.04.025.
[33] Nair D, Cavanaugh KL, Wallston KA, et al. Religion, spirituality, and risk of end-stage kidney disease among adults of low socioeconomic status in the Southeastern United States. J Health Care Poor Underserved. 2020;31(4):1727–1746. doi: 10.1353/hpu.2020.0129.