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Diabetes, hypertension, obesity, and long-term risk of renal disease mortality: Racial and socioeconomic differences

Maryam Moghani Lankarani[1]*†, Shervin Assari[2,3]

[1]Medicine and Health Promotion Institute, Tehran, Iran, [2]Department of Psychiatry, and [3]Center for Research on Ethnicity, Culture, and Health, School of Public Health, University of Michigan, Ann Arbor, Michigan, USA

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***Correspondence**
Maryam Moghani Lankarani
Tel.: +1-7343-232-0445
Fax: +1-734-763-9265
E-mail address: lankaranii@yahoo.com

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ABSTRACT

Aims/Introduction: Diabetes, hypertension, and obesity increase the risk of chronic kidney disease and associated mortality. Race and socioeconomic status (SES) differences in the effects of these risk factors are, however, still unknown. The current study aimed to investigate whether or not race and SES alter the effects of diabetes, hypertension, and obesity on mortality due to renal disease.

Materials and Methods: Data came from the Americans' Changing Lives Study, 1986–2011, a nationally representative prospective cohort of adults with 25 years of follow up. The study included 3,361 adults aged 25 years and older who were followed for up to 25 years. The outcome was death from renal disease. Diabetes, hypertension, and obesity were the main predictors. Race and SES (education, income, and employment) were moderators. Health behaviors and health status at baseline were covariates. We used Cox proportional hazards models for data analysis.

Results: In separate models, diabetes, hypertension, and obesity at baseline were associated with a higher risk of death from renal disease. From our SES indicators, education and income interacted with diabetes, hypertension, and obesity on death from renal disease. In a consistent pattern, diabetes, hypertension, and obesity showed stronger effects on the risk of death from renal disease among high-SES groups compared with low-SES individuals. Race and employment did not alter the effects of diabetes, hypertension and obesity on the risk of death from renal disease.

Conclusions: Social groups differ in how diabetes, hypertension, and obesity influence health outcomes over long-term periods. Elimination of disparities in renal disease mortality in the USA requires understanding of the complex and non-linear effects of socioeconomic and medical risk factors on health outcomes. Multidisciplinary programs and policies are required to reduce social inequality in renal disease burden caused by diabetes, hypertension, and obesity.

INTRODUCTION

Low socioeconomic status (SES) is a major risk factor for kidney disease[1,2]. Minority status also increases the risk of death from renal disease[3]. In both sexes and all racial groups in the USA, the incidence of end-stage renal disease (ESRD) by all primary renal diseases is greatest in the lowest SES score quartile, and decreases progressively as SES increases[2]. African Americans are three-to-four times as likely to develop kidney failure compared with White Americans[4]. Despite composing just 13% of the population, one-third of all kidney failures in the USA occur among African Americans[4].

In the USA, race and SES operate jointly, as minorities tend to have lower SES[5,6]. For instance, race is closely associated with education, employment, and income[6]. However, a complex association exists between race, SES, risk factors (diabetes [DM], hypertension [HTN], and obesity), and renal disease mortality[7,8].

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Medical risk factors, such as DM, obesity, and HTN, increase the risk of renal disease mortality[9–13]. Diabetes, HTN, and obesity might explain why race and SES predict renal disease mortality[7,8]. Such medical risk factors[9,10] are well-known causes of kidney disease, and at the same time are more common among low-SES groups[14,15]and African Americans[12,16,17].

The medical risk factors of chronic kidney disease (CKD), such as DM[12], obesity[13], and HTN[11], increase the risk of mortality as a result of renal diseases over long periods of time. The effect of DM on the development and burden of chronic kidney disease is well established[18]. In fact, chronic kidney disease is a known complication of DM[19]. Hypertension is also a known etiological risk factor for the development of chronic kidney disease[20], as the risk of end-stage renal disease increases in the presence of HTN[20]. Individuals with obesity also develop kidney disease, partly because of the higher risk of DM and HTN[13].

To build on the existing literature on the complex effects of race, SES, DM, HTN, and obesity on renal disease outcomes[7,8], we examined race and SES (education, income, and employment) differences in the effects of three established medical risk factors (DM, HTN, and obesity) on mortality as a result of renal diseases over 25 years in the USA.

METHODS

Setting

Data came from the Americans' Changing Lives (ACL) Study, a nationally-representative USA cohort study carried out from 1986 until 2011. Detailed information on the study design is available elsewhere[21,22].

Sampling and participants

The ACL enrolled a stratified multistage probability sample of adults aged 25 years or older who lived in the continental USA in 1986. The study included 3,617 non-institutionalized respondents (representing 70% of sampled households and 68% of sample individuals at baseline) with an oversampling of those age 60 years and older, and African Americans. Wave 1 included 70% of sampled households and 68% of sampled individuals. Further interviews were carried out in 1989, 1994, 2001/2 and 2011, but information from those interviews was not relevant for these analyses.

Informed consent

All procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) with the Helsinki Declaration of 1975, as revised in 2000. Informed consent was obtained from all participants included in the study.

Measures

Data on demographics, SES, medical risk factors, health behaviors, and health status were measured at baseline during face-to-face interviews in 1986.

Moderators

Race

Race was defined as non-Hispanic Black or non-Hispanic White based on a coding of self-reported items asking about Hispanic ethnicity, nativity, and racial category.

SES

Data on SES were measured with an indicator of education (<12 years of education, ≥12 years), income (10-level categorical variable consisting of <$5,000, $5,000–9,000, $10,000–14,000, $15,000–19,000, $20,000–24,000, $25,000–29,000, $30,000–39,000, $40,000–59,000, $60,000–79,000, $80,000+) and employment (employed, unemployed) at baseline.

Covariates

Demographics

Demographic data included sex (a dichotomous variable with male as the referent category) and age (a continuous variable as number of years since birth).

Health behaviors

We collected data on self-reported smoking (current smoker vs other), drinking (any drinking vs other), and exercise (any exercise vs other) using the following single-item measures. 'Do you smoke cigarettes now?' 'Do you ever drink beer, wine, or liquor?' 'How often do you engage in active sports or exercise – would you say often, sometimes, rarely, or never?' Responses of the first two items were 'yes' and 'no,' and responses of the third item were: (i) often; (ii) sometimes; (iii) rarely; and (iv) never.

Self-rated health

Self-rated health (SRH) was a single-item scale on subjective overall health, with five categories: poor, fair, good, very good, and excellent. SRH was treated as a dichotomous measure, where the original five categories were collapsed to the poor/fair vs good/very good/excellent categories. Categorizing SRH in this way has shown acceptable test–retest reliability and validity[23].

Depressive symptoms

We used the 11-item Center for Epidemiological Studies-Depression scale[24]. In this shortened version of the Center for Epidemiological Studies-Depression scale, a score of 1–3 is given to each item. We used the mean item score as the total score, ranging from 1 to 3. The abbreviated Center for Epidemiological Studies-Depression scale has been used in other studies, and has shown acceptable reliability and validity[25].

Predictors

DM

Self-reported DM was assessed at baseline. All participants were asked whether a healthcare provider had ever told them they had DM[22,26].

HTN

Self-reported HTN was assessed at baseline. All participants were asked whether a healthcare provider had ever told them they had HTN[22,26].

Obesity

The body mass index (BMI) was calculated based on self-reported weights and heights. Weight and height were originally collected in pounds (1 pound = 0.453 kg) and feet (1 foot = 0.3048 m) / inches (1 inch = 0.0254 m), respectively. Obesity was defined as BMI ≥30 kg/m^2 [27]. BMI calculated based on self-reported weight and height is known to be closely correlated with BMI based on direct measures of height and weight[28]. However, using self-reported weight and height could lead to some degrees of underestimation of BMI[29], due to a systematic tendency for humans to underestimate their weight and to overestimate their height[30].

Outcome

Mortality as a result of renal diseases

The main outcome variables were mortality from renal diseases and time of death. Information on all deaths from mid-1986 through 2011 was obtained through the National Death Index, death certificates and also from informants. In most cases, time and cause of death were verified with death certificates. The handful of cases where death could not be verified with death certificates was reviewed carefully, and actual death was certain in all cases. Only in these cases was the date of death ascertained from the informants or the National Death Index report, rather than the death certificate[26,31]. Cause of death was coded as unknown if the death certificate or National Death Index report were unavailable.

We used the International Classification of Diseases (ICD)-9 and ICD-10 codes[32,33], whichever was current at the time the death was recorded, to determine death from renal diseases (kidney–urinary). For ICD-9 codes, we used codes 650 (acute glomerulonephritis and nephrotic syndrome), 660 (chronic glomerulonephritis, nephritis and nephropathy, not specified as acute or chronic, and renal sclerosis, unspecified), 670 (renal failure, disorders resulting from impaired renal function and small kidney of unknown causes), 680 (infections of kidney) and 690 (hyperplasia of prostate). For ICD-10 codes, we used the categorization of 113 selected causes of death provided by the World Health Organization, for which codes 97 (nephritis, nephrotic syndrome and nephrosis), 98 (acute and rapidly progressive nephritic and nephrotic syndrome), 99 (chronic glomerulonephritis, nephritis and nephropathy not specified as acute or chronic, and renal sclerosis unspecified), 100 (renal failure), 101 (other disorders of kidney), 102 (infections of kidney), 103 (hyperplasia of prostate) and 104 (inflammatory diseases of female pelvic organ) were used. Respondents who died of other causes were censored at the time of death. Time of death was registered as the number of months from time of enrollment in the study to time of death, based on the month of death and the month of the baseline interview.

Statistical analysis

Univariate, bivariate, and multivariable analyses were carried out using Stata 13.0 (StataCorp, College Station, Texas, USA). We used Taylor series linearization to estimate standard errors as a result of stratification and clustering, as well as non-response. Hazard ratios with 95% confidence intervals (CI) are reported. For main effects, $P < 0.05$ was considered statistically significant. For interactions, we considered $P < 0.1$ as statistically significant.

Cox proportional hazards models require two variables, a binary outcome (renal death) and also time to the renal death event, or to censoring. Time to event was defined as the number of months from baseline to death, loss to follow up, or the end of the year 2011. Renal death was coded as zero if the respondent did not die, or died from any other causes.

For multivariable analysis, 24 Cox proportional hazards models were fitted to the data (six for each moderator). At each step, we ran models with main effects (model 1a, model 2a, and model 3a). Then we ran models with the interaction terms (model 1b, model 2b, and model 3b). The outcome was death from renal diseases and the time that outcome occurred since baseline (time to death from renal diseases). Race and SES indicators (education, income, employment) were the moderators. Chronic medical diseases (DM, HTN, obesity) were predictors. Health behaviors (smoking, drinking, and exercise) and health status (SRH and depressive symptoms) at baseline were potential confounders.

RESULTS

Table 1 shows descriptive statistics for the overall sample, and based on race. Tables 2–5 each show six Cox proportional hazard regression models, with models on the left being the main effect models, and models on the right testing the interactions.

Table 2 shows the results of six Cox proportional hazard regression models with low education as the SES indicator (moderator). In model 1a, DM was associated with a higher risk of death from renal disease, net of health behaviors and other health factors. Model 1b showed an interaction between education and DM, suggesting that the predictive role of DM on risk of death from renal disease is larger for those with high education, compared with those with low education. According to the results of model 2a, HTN was associated with a higher risk of death from renal disease, net of health behaviors and other health factors. Model 2b showed an interaction between education and HTN, suggesting that the predictive role of HTN on risk of death from renal disease is larger for individuals with high education, compared with those with low education. According to the results of model 3a, obesity was associated with a higher risk of death from renal disease, net of health behaviors and other health factors. Model 3b showed an

Table 1 | Descriptive statistics of demographic, socioeconomic, medical conditions, and health behaviors overall and among Blacks and Whites at baseline

	All		Whites		Blacks	
	Mean (SE)	95% CI	Mean (SE)	95% CI	Mean (SE)	95% CI
Age	47.79 (0.53)	46.72–48.86	47.98 (0.60)	46.77–49.19	46.37 (0.71)	44.93–47.81
Income*	5.41 (0.09)	5.22–5.60	5.57 (0.10)	5.36–5.77	4.25 (0.18)	3.88–4.62
Exercise*	0.02 (0.03)	–0.03–0.07	0.06 (0.03)	0.00–0.11	–0.22 (0.05)	–0.33–0.12
	% (SE)	95% CI	% (SE)	95% CI	% (SE)	95% CI
Sex						
Male	47.26 (0.01)	44.86–49.68	47.82 (0.01)	45.12–50.52	43.18 (0.02)	38.79–47.69
Female	52.74 (0.01)	50.32–55.14	52.18 (0.01)	49.48–54.88	56.82 (0.02)	52.31–61.21
Education*	12.53 (0.10)	12.34–12.73	12.69 (0.11)	12.48–12.90	11.37 (0.23)	10.90–11.84
	% (SE)	95% CI	% (SE)	95% CI	% (SE)	95% CI
HTN*	21.37 (0.01)	19.67–23.17	19.77 (0.01)	18.11–21.53	33.17 (0.03)	28.20–38.54
DM*	5.73 (0.00)	4.80–6.82	5.25 (0.01)	4.24–6.50	9.22 (0.01)	7.75–10.95
Obesity*	14.47 (0.01)	12.86–16.24	13.52 (0.01)	11.72–15.54	21.45 (0.02)	17.88–25.52
Smoking*	30.45 (0.01)	27.81–33.23	29.70 (0.01)	26.85–32.72	35.98 (0.03)	30.81–41.49
Drinking*	60.02 (0.02)	56.68–63.26	61.50 (0.02)	58.06–64.83	49.10 (0.03)	43.55–54.68
Renal death	0.52 (0.00)	0.26–1.03	0.44 (0.00)	0.18–1.06	1.15 (0.00)	0.63–02.09

*$P < 0.05$. CES-D, Center for Epidemiologic Studies Depression Scale; CI, confidence interval; CMC, chronic medical conditions; DM, diabetes mellitus; HTN, hypertension; SE, standard error.

interaction between education and obesity, suggesting that the predictive role of obesity on risk of death from renal disease depends on education, with a stronger predictive role for people with high education compared with those with low education (Table 2).

Table 3 shows a summary of the results of six Cox proportional hazard regression models with employment as the SES indicator (moderator). According to the results of model 1a, DM was associated with a higher risk of death from renal disease, net of health behaviors and other health factors. Model 1b did not show an interaction between employment and DM, suggesting that the effect of DM on risk of death from renal disease does not depend on employment status. According to the results of model 2a, HTN was associated with a higher risk of death from renal disease, net of health behaviors and other health factors. Model 2b did not show an interaction between employment and DM, suggesting that the effect of DM on risk of death from renal disease does not depend on employment status. According to the results of model 3a, obesity was associated with a higher risk of death from renal disease, net of health behaviors and other health factors. Model 3b did not show an interaction between employment and obesity, suggesting that the effect of obesity on risk of death from renal disease does not depend on employment status (Table 3).

Table 4 shows a summary of the results of six Cox proportional hazard regression models with income as the SES indicator (moderator). According to the results of model 1a, DM was associated with a higher risk of death from renal disease, net of health behaviors and other health factors. Model 1b showed an interaction between income and DM, suggesting that the predictive role of DM on risk of death from renal disease depends on income, with a better prediction role for those with high income, compared with those with low income. According to the results of model 2a, HTN was associated with a higher risk of death from renal disease, net of health behaviors and other health factors. Model 2b showed an interaction between low income and HTN, suggesting that the effect of HTN on the risk of death from renal disease depends on income, with HTN better predicting renal disease mortality for those with a high income, compared with their low-income counterparts. According to the results of model 3a, obesity was associated with a higher risk of death from renal disease, net of health behaviors and other health factors. Model 3b showed an interaction between income and obesity, suggesting that the effect of obesity on risk of death from renal disease depends on income, with a better predictive role of obesity for high- vs low-income individuals (Table 4).

Table 5 shows a summary of the results of six Cox proportional hazard regression models with race as the moderator. According to the results of model 1a, DM was associated with a higher risk of death from renal disease, net of health behaviors and other health factors. Model 1b did not show an interaction between race and DM, suggesting that the effect of DM on risk of death from renal disease does not depend on race. Similarly, although HTN and obesity were associated with the outcome (model 2a and model 3a), these associations did not depend on race (model 2b and model 3b; Table 5).

DISCUSSION

Using 25-year data, the current study showed that DM, HTN, and obesity interact with SES on renal disease mortality. We found that the predictive role of DM, HTN, and obesity are

Table 2 | Summary of Cox regressions on the interaction between low education and medical risk factors of death from renal disease in the USA

	HR (SE)	95% CI Model a (main effects)	*P*	HR (SE)	95% CI Model b (interactions)	*P*
Model 1						
Race (Black)	2.20 (0.96)	0.92–5.29	0.076	2.13 (0.92)	0.89–5.08	0.088
Age (years)	1.09 (0.02)	1.06–1.13	0.000	1.09 (0.02)	1.06–1.12	0.000
Sex (women)	0.40 (0.25)	0.12–1.39	0.146	0.51 (0.28)	0.17–1.51	0.220
SES (low education)	0.56 (0.30)	0.19–1.66	0.292	1.45 (0.93)	0.40–5.26	0.566
Smoking	3.46 (1.86)	1.17–10.24	0.026	3.32 (1.61)	1.25–8.81	0.017
Drinking	0.13 (0.08)	0.04–0.42	0.001	0.16 (0.08)	0.06–0.44	0.001
Exercise	0.78 (0.17)	0.51–1.21	0.269	0.85 (0.17)	0.57–1.26	0.414
SRH (poor)	2.26 (1.03)	0.90–5.68	0.082	2.20 (0.99)	0.89–5.44	0.085
Depressive symptoms	1.52 (0.27)	1.06–2.17	0.024	1.60 (0.28)	1.12–2.29	0.011
DM	7.57 (4.02)	2.60–22.07	0.000	18.90 (14.83)	3.89–91.81	0.001
DM × SES	–	–	–	0.12 (0.12)	0.02–0.92	0.042
Model 2						
Race (Black)	1.84 (0.87)	0.71–4.76	0.202	1.99 (0.92)	0.78–5.07	0.144
Age (years)	1.08 (0.01)	1.05–1.11	0.000	1.08 (0.01)	1.05–1.11	0.000
Sex (women)	0.36 (0.25)	0.09–1.49	0.155	0.41 (0.28)	0.11–1.60	0.195
SES (low education)	0.55 (0.35)	0.15–1.95	0.346	7.89 (7.49)	1.17–53.37	0.035
Smoking	3.73 (2.13)	1.18–11.78	0.026	3.68 (2.05)	1.20–11.32	0.024
Drinking	0.12 (0.08)	0.03–0.47	0.003	0.13 (0.08)	0.03–0.47	0.003
Exercise	0.81 (0.18)	0.52–1.26	0.341	0.87 (0.19)	0.57–1.34	0.524
SRH (poor)	2.40 (1.14)	0.92–6.27	0.073	2.42 (1.08)	0.98–5.95	0.055
Depressive symptoms	1.54 (0.22)	1.15–2.06	0.005	1.58 (0.25)	1.15–2.17	0.006
HTN	4.25 (2.69)	1.19–15.18	0.027	29.42 (28.03)	4.32–200.48	0.001
HTN × SES	–	–	–	0.03 (0.03)	0.00–0.24	0.002
Model 3						
Race (Black)	1.93 (1.00)	0.68–5.50	0.213	1.97 (0.96)	0.74–5.24	0.168
Age (year)	1.10 (0.01)	1.07–1.13	0.000	1.10 (0.02)	1.07–1.13	0.000
Sex (women)	0.36 (0.25)	0.09–1.42	0.141	0.42 (0.28)	0.11–1.62	0.204
SES (low education)	0.59 (0.35)	0.17–1.97	0.378	1.48 (0.91)	0.43–5.11	0.524
Smoking	3.41 (1.96)	1.07–10.86	0.039	3.57 (2.04)	1.12–11.32	0.032
Drinking	0.13 (0.08)	0.04–0.42	0.001	0.15 (0.08)	0.05–0.41	0.001
Exercise	0.82 (0.16)	0.55–1.23	0.332	0.87 (0.17)	0.58–1.29	0.473
SRH (poor)	2.63 (1.16)	1.08–6.38	0.033	2.38 (0.92)	1.09–5.17	0.030
Depressive symptoms	1.51 (0.19)	1.17–1.95	0.002	1.59 (0.24)	1.17–2.15	0.004
Obesity	2.88 (1.71)	0.87–9.51	0.082	7.14 (5.42)	1.54–32.97	0.013
Obesity × SES	–	–	–	0.11 (0.11)	0.02–0.79	0.029

CI, confidence interval; DM; diabetes, HR, hazard ratio; HTN; hypertension; SE, standard error; SES, socioeconomic status; SRH; self-rated health.

higher for individuals with high education and high income, compared with their low-education and low-income counterparts. This finding expands the current literature on how DM, HTN, obesity, and SES predict renal disease mortality[7,8]. In a recent study by Assari[7], SES (income) and medical disease (DM and HTN), as well as health behaviors (smoking, drinking, and exercise) explained the effect of race on death from renal disease. In another study by Assari[8], DM, HTN, and other chronic medical conditions explained the effects of race and SES on all-cause mortality. None of these studies tested the differential effects of DM, HTN, and obesity on mortality based on race or SES, which is the unique contribution of the present study.

Diabetes and HTN are leading causes of kidney failure in the USA, particularly among minorities and low SES individuals[3]. Thus, the presence of risk factors, such as HTN[11], DM[12], obesity[13], and other chronic medical conditions[9,10] convey information on future risk of mortality from renal disease in the USA. It was previously known that these medical risk factors are more common among racial minorities and low-SES individuals[12,16,17]. The current study shows that role of these conditions as a predictor of mortality from renal disease is not universal across all subpopulations.

How DM increases the risk of chronic kidney disease is well-established[18], as studies have shown that chronic kidney disease is a complication of DM[19]. The association between chronic

Table 3 | Summary of Cox regressions on the interaction between unemployment and medical risk factors of death from renal disease in the USA

	HR (SE)	95% CI Model a (main effects)	*P*	HR (SE)	95% CI Model b (interactions)	*P*
Model 1						
Race (Black)	1.99 (0.95)	0.76–5.21	0.156	1.99 (0.96)	0.75–5.27	0.160
Age (years)	1.08 (0.02)	1.03–1.12	0.001	1.08 (0.02)	1.03–1.13	0.001
Sex (women)	0.35 (0.25)	0.08–1.51	0.154	0.35 (0.25)	0.08–1.50	0.152
SES (unemployed)	2.29 (1.35)	0.70–7.50	0.166	2.43 (1.93)	0.50–11.98	0.266
Smoking	3.26 (1.72)	1.13–9.42	0.030	3.24 (1.79)	1.07–9.83	0.039
Drinking	0.15 (0.08)	0.05–0.44	0.001	0.15 (0.08)	0.05–0.43	0.001
Exercise	0.76 (0.17)	0.48–1.19	0.220	0.76 (0.17)	0.49–1.18	0.217
SRH (poor)	1.80 (0.71)	0.81–4.00	0.142	1.81 (0.71)	0.82–3.98	0.137
Depressive Symptoms	1.42 (0.26)	0.98–2.06	0.065	1.42 (0.27)	0.97–2.07	0.072
DM	6.83 (3.55)	2.39–19.45	0.001	7.98 (9.84)	0.67–95.68	0.099
DM × SES	–	–	–	0.83 (1.20)	0.05–15.10	0.900
Model 2						
Race (Black)	1.70 (0.93)	0.57–5.12	0.335	1.67 (0.92)	0.55–5.05	0.359
Age (years)	1.06 (0.02)	1.02–1.11	0.009	1.06 (0.02)	1.02–1.11	0.008
Sex (women)	0.32 (0.25)	0.06–1.57	0.155	0.31 (0.25)	0.06–1.55	0.150
SES (unemployed)	2.71 (1.74)	0.74–9.90	0.127	5.22 (4.98)	0.76–35.65	0.090
Smoking	3.39 (1.78)	1.18–9.74	0.024	3.34 (1.78)	1.14–9.78	0.028
Drinking	0.15 (0.08)	0.05–0.45	0.001	0.15 (0.08)	0.05–0.46	0.001
Exercise	0.78 (0.18)	0.49–1.24	0.288	0.78 (0.18)	0.49–1.24	0.284
SRH (poor)	1.89 (0.76)	0.84–4.25	0.118	1.92 (0.75)	0.87–4.24	0.103
Depressive symptoms	1.46 (0.22)	1.08–1.96	0.015	1.46 (0.21)	1.09–1.94	0.011
HTN	3.82 (2.21)	1.19–12.24	0.025	8.67 (8.90)	1.10–68.59	0.041
HTN × SES	–	–	–	0.36 (0.46)	0.03–4.89	0.433
Model 3						
Race (Black)	1.81 (1.09)	0.54–6.08	0.330	1.84 (1.10)	0.55–6.14	0.314
Age (years)	1.08 (0.02)	1.04–1.12	0.000	1.08 (0.02)	1.04–1.12	0.000
Sex (Women)	0.31 (0.25)	0.06–1.54	0.147	0.31 (0.24)	0.06–1.52	0.144
SES (unemployed)	2.99 (2.14)	0.71–12.62	0.133	2.19 (1.25)	0.69–6.91	0.176
Smoking	3.16 (1.72)	1.06–9.43	0.039	3.19 (1.73)	1.07–9.52	0.038
Drinking	0.16 (0.08)	0.06–0.43	0.001	0.15 (0.08)	0.06–0.43	0.001
Exercise	0.80 (0.17)	0.52–1.21	0.278	0.79 (0.16)	0.53–1.20	0.270
SRH (poor)	2.05 (0.74)	0.99–4.25	0.052	2.04 (0.75)	0.97–4.28	0.059
Depressive symptoms	1.45 (0.21)	1.09–1.93	0.011	1.45 (0.21)	1.08–1.95	0.014
Obesity	2.75 (1.64)	0.83–9.13	0.097	1.29 (0.98)	0.28–5.98	0.736
Obesity × SES	–	–	–	2.41 (2.46)	0.31–18.87	0.394

CI, confidence interval; DM; diabetes, HR, hazard ratio; HTN; hypertension; SE, standard error; SES, socioeconomic status; SRH; self-rated health.

kidney disease and HTN is also bidirectional, as HTN is listed as a known etiological factor for the development of chronic kidney disease[20]. Longitudinal studies have consistently shown that baseline HTN causally contributes to the subsequent risk of end-stage renal disease[12]. Based on the current study, DM and HTN better predict renal disease mortality for high SES people.

Several studies have tried to deconstruct the effects of race, SES, and medical risk factors on health[34–36]. These associations are, however, shown to be multiplicative rather than additive[37,38]. There is still a debate on whether HTN, DM, and other conditions explain the effects of race and SES on health or not[35,36]. Another remaining question is whether race and SES have parallel or overlapping effects[39,40], and whether their effects are simply because of increasing exposure to different risk factors[41,42] or also altering vulnerabilities to the effects of risk factors[43]. Race and SES interact with health outcomes[44–46], as racial differences in health depend on SES[44], and SES effects on health depend on race[45,46]. Thus, race, SES, and other risk factors might have multiplicative rather than additive effects on health[44]. Findings of the current study support such an argument.

According to our findings, SES differences in death from renal disease are not exclusively a result of SES gaps in exposures to medical risk factors (DM, HTN, and obesity). Instead, the same risk factors can have different roles across social groups, which is in line with the differential effect hypothesis[47,48]. Thus, understanding the causal paths between social factors, medical risk

Table 4 | Summary of Cox regressions on the interaction between low income and medical risk factors of death from renal disease in the USA

	HR (SE)	95% CI Model a (main effects)	*P*	HR (SE)	95% CI Model b (interactions)	*P*
Model 1						
Race (Black)	2.03 (0.86)	0.87–4.75	0.099	2.04 (0.86)	0.87–4.74	0.097
Age (years)	1.09 (0.02)	1.06–1.13	0.000	1.09 (0.02)	1.05–1.12	0.000
Sex (women)	0.40 (0.26)	0.11–1.50	0.169	0.45 (0.27)	0.14–1.49	0.186
SES (Low Income)	0.80 (0.49)	0.23–2.74	0.717	1.84 (1.45)	0.38–9.03	0.442
Smoking	3.42 (1.90)	1.11–10.50	0.033	3.46 (1.78)	1.23–9.75	0.020
Drinking	0.13 (0.08)	0.04–0.44	0.002	0.15 (0.07)	0.06–0.41	0.000
Exercise	0.78 (0.17)	0.50–1.19	0.239	0.81 (0.16)	0.54–1.20	0.283
SRH (poor)	2.14 (1.00)	0.83–5.48	0.112	1.96 (0.85)	0.82–4.69	0.125
Depressive symptoms	1.46 (0.25)	1.04–2.06	0.031	1.46 (0.26)	1.02–2.08	0.040
DM	7.35 (3.88)	2.53–21.30	0.000	19.57 (17.64)	3.18–120.25	0.002
DM × SES	–	–	–	0.20 (0.19)	0.03–1.29	0.090
Model 2						
Race (Black)	1.75 (0.80)	0.70–4.41	0.227	1.80 (0.82)	0.72–4.51	0.202
Age (years)	1.08 (0.02)	1.05–1.11	0.000	1.08 (0.01)	1.05–1.11	0.000
Sex (women)	0.39 (0.25)	0.11–1.44	0.154	0.42 (0.26)	0.13–1.43	0.162
SES (low income)	0.60 (0.46)	0.13–2.77	0.508	3.56 (3.19)	0.59–21.58	0.163
Smoking	3.66 (2.06)	1.18–11.38	0.026	3.77 (2.09)	1.24–11.49	0.021
Drinking	0.13 (0.08)	0.03–0.48	0.003	0.13 (0.08)	0.03–0.48	0.003
Exercise	0.80 (0.17)	0.53–1.23	0.304	0.80 (0.17)	0.52–1.23	0.295
SRH (poor)	2.33 (1.11)	0.89–6.09	0.083	2.29 (1.02)	0.94–5.60	0.068
Depressive Symptoms	1.54 (0.23)	1.15–2.07	0.005	1.60 (0.26)	1.16–2.20	0.005
HTN	4.28 (2.83)	1.13–16.23	0.033	19.30 (18.48)	2.81–132.72	0.003
HTN × SES	–	–	–	0.09 (0.10)	0.01–0.81	0.033
Model 3						
Race (Black)	1.82 (0.91)	0.67–4.98	0.235	2.03 (0.90)	0.83–4.95	0.117
Age (years)	1.10 (0.02)	1.07–1.13	0.000	1.10 (0.02)	1.07–1.13	0.000
Sex (women)	0.38 (0.25)	0.10–1.40	0.143	0.46 (0.25)	0.15–1.40	0.166
SES (low income)	0.76 (0.48)	0.21–2.72	0.662	2.40 (2.22)	0.37–15.49	0.350
Smoking	3.34 (1.89)	1.07–10.41	0.038	3.75 (2.17)	1.17–12.01	0.027
Drinking	0.14 (0.08)	0.04–0.42	0.001	0.16 (0.07)	0.06–0.41	0.000
Exercise	0.82 (0.16)	0.55–1.22	0.322	0.85 (0.16)	0.59–1.24	0.391
SRH (poor)	2.56 (1.17)	1.02–6.44	0.046	2.26 (0.85)	1.06–4.82	0.035
Depressive symptoms	1.49 (0.18)	1.17–1.91	0.002	1.50 (0.19)	1.17–1.93	0.002
Obesity	2.80 (1.60)	0.89–8.82	0.078	11.33 (11.68)	1.42–90.38	0.023
Obesity × SES	–	–	–	0.10 (0.11)	0.01–0.91	0.041

CI, confidence interval; DM; diabetes, HR, hazard ratio; HTN; hypertension; SE, standard error; SES, socioeconomic status; SRH; self-rated health.

factors, and health outcomes requires understanding the existing complex heterogeneity in the effects of the same risk factors on outcomes[43]. We argue that such heterogeneity might be a result of contextual factors, such as race, ethnicity, sex, and place, that shape vulnerabilities and resilience to the effects of the same risk factors on outcomes[49–52].

Thus, it is not SES or medical risk factors, such as DM, HTN, and obesity, *per se* that cause renal disease[20]. As the distribution of risk factors depends on racial and SES groups[16], differential exposure might ultimately result in differential vulnerability to the effects of risk factors[43]. Thus, disparities in kidney disease mortality in the USA are the result of complex interactions between social and medical risk factors, such as DM and HTN[12,53–55].

The present study had a few limitations that should be considered before the results are interpreted. The first limitation was a lack of measurement of kidney disease at baseline or over the course of the follow up. The second limitation was the measurement of HTN, DM, and obesity using self-reported data, which is subject to measurement bias[56]. Self-rated medical conditions should always be validated by other sources of information, such as medical records. The third limitation was that the present study did not measure decline in kidney function over time. The fourth limitation was the lack of information on the diabetes type. Type 1 and type 2 diabetes have different pathogenesis mechanisms, and the patterns of renal disease development and progression might also be different. Fifth, we did not have enough data to explore the role of other races

Table 5 | Summary of Cox regressions on the interaction between race and medical risk factors of death from renal disease in the USA

	HR (SE)	95% CI Model a (main effects)	*P*	HR (SE)	95% CI Model b (interactions)	*P*
Model 1						
Race (Black)	1.95 (0.93)	0.74–5.10	0.169	2.41 (1.16)	0.91–6.36	0.075
Age (years)	1.09 (0.02)	1.06–1.13	0.000	1.09 (0.02)	1.06–1.13	0.000
Sex (women)	0.37 (0.27)	0.09–1.61	0.182	0.38 (0.27)	0.09–1.58	0.178
Smoking	3.38 (1.85)	1.12–10.20	0.031	3.35 (1.80)	1.14–9.86	0.029
Drinking	0.13 (0.08)	0.04–0.45	0.002	0.14 (0.08)	0.04–0.44	0.001
Exercise	0.77 (0.17)	0.50–1.19	0.232	0.77 (0.16)	0.50–1.19	0.229
SRH (poor)	2.09 (0.91)	0.87–5.03	0.097	2.05 (0.87)	0.87–4.81	0.097
Depressive symptoms	1.44 (0.26)	1.00–2.07	0.048	1.43 (0.26)	0.99–2.06	0.054
DM	7.46 (4.07)	2.48–22.40	0.001	8.51 (5.64)	2.24–32.30	0.002
DM × race	–	–	–	0.62 (0.52)	0.12–3.38	0.576
Model 2						
Race (Black)	1.61 (0.92)	0.51–5.06	0.409	1.89 (1.22)	0.51–6.93	0.330
Age (years)	1.08 (0.02)	1.04–1.11	0.000	1.08 (0.02)	1.04–1.11	0.000
Sex (women)	0.35 (0.27)	0.07–1.69	0.186	0.35 (0.27)	0.07–1.69	0.186
Smoking	3.57 (1.93)	1.20–10.60	0.023	3.55 (1.86)	1.24–10.19	0.020
Drinking	0.13 (0.08)	0.04–0.48	0.003	0.13 (0.08)	0.04–0.47	0.003
Exercise	0.80 (0.17)	0.52–1.24	0.310	0.80 (0.17)	0.52–1.24	0.311
SRH (poor)	2.22 (0.96)	0.92–5.32	0.074	2.22 (0.98)	0.92–5.40	0.076
Depressive symptoms	1.49 (0.20)	1.14–1.96	0.005	1.50 (0.20)	1.13–1.97	0.005
HTN	4.14 (2.65)	1.14–15.00	0.032	4.32 (3.22)	0.97–19.36	0.055
HTN × race	–	–	–	0.81 (0.77)	0.12–5.50	0.828
Model 3						
Race (Black)	1.71 (1.05)	0.49–5.89	0.389	2.38 (1.28)	0.80–7.05	0.115
Age (years)	1.09 (0.02)	1.06–1.13	0.000	1.10 (0.02)	1.07–1.13	0.000
Sex (women)	0.35 (0.26)	0.08–1.57	0.167	0.36 (0.26)	0.08–1.57	0.168
Smoking	3.32 (1.85)	1.08–10.22	0.037	3.44 (2.11)	1.00–11.84	0.050
Drinking	0.14 (0.08)	0.04–0.43	0.001	0.14 (0.08)	0.05–0.43	0.001
Exercise	0.82 (0.16)	0.55–1.22	0.321	0.82 (0.16)	0.55–1.23	0.325
SRH (poor)	2.48 (1.03)	1.07–5.74	0.035	2.45 (1.01)	1.06–5.63	0.036
Depressive symptoms	1.47 (0.19)	1.13–1.92	0.005	1.46 (0.21)	1.09–1.95	0.012
Obesity	2.86 (1.77)	0.82–9.96	0.096	3.48 (2.84)	0.67–17.98	0.133
Obesity × race	–	–	–	0.49 (0.57)	0.05–5.08	0.545

CI, confidence interval; DM; diabetes, HR, hazard ratio; HTN; hypertension; SE, standard error; SES, socioeconomic status; SRH; self-rated health.

and sex in the modification of the effect of medical risk factors on renal disease mortality in the present study. Despite these limitations, the current study made a unique contribution to the literature. The major strengths of the present study were long-term follow up, large sample size, and a nationally representative USA sample.

To conclude, we found that SES (education and income) alters how medical risk factors (DM, HTN, and obesity) increase the risk of deaths from renal diseases over a 25-year period, above and beyond health behaviors (smoking, drinking, and exercise) and other health factors (SRH and depressive symptoms). Thus, the effects of social and medical determinants of health in shaping the burden of chronic kidney disease are not additive, but multiplicative. These findings extend the existing knowledge on the complex link between race, SES, chronic disease, and renal disease mortality in the USA. Thus, the attribution of racial and SES differences in renal disease mortality to racial and SES differences in exposure to risk factors might be oversimplistic[57].

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DISCLOSURE

The authors declare no conflict of interest.

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