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Number of Chronic Medical Conditions Fully Mediates the Effects of Race on Mortality; 25-Year Follow-Up of a Nationally Representative Sample of Americans

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Abstract

Background—Despite the well-established literature on the effects of race and socioeconomic status (SES) on mortality, limited information exists on mediators of these effects. Taking a life-course epidemiology approach, and using a nationally representative sample of adults in the USA, the current study has two aims: (1) to assess the effects of race and SES at baseline on all-cause mortality over a 25-year follow-up and (2) to test whether the number of chronic medical conditions (CMCs) as a time-varying covariate mediates the effects of race and SES on all-cause mortality.

Methods—Data came from the Americans' Changing Lives (ACL) Study, a nationally representative longitudinal cohort of US adults 25 and older. The study followed 3361 Blacks or Whites for all-cause mortality for up to 25 years from 1986 to 2011. The predictors of interest were race and SES (education and family income) at baseline measured in 1986. Confounders included baseline age and gender. CMC was the potential time-varying mediator measured in 1986, 1989, 1991, 2001, and 2011. We ran Cox proportional hazard models with and without CMC as time-varying covariates.

Author's Contributions

Shervin Assari designed and analyzed this work and drafted the paper. He also confirmed the last version.

Notes

Compliance with Ethical Standard

Ethics

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. University of Michigan Institutional review board (IRB) approved the study protocol.

Conflict of Interest

Shervin Assari declares that he has no conflicts of interest.

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.

Animal Studies

No animal studies were carried out by the authors for this article.

Results—In separate models, race and SES were predictors of all-cause mortality. In the model that tested the combined effect of race and SES, SES but not race was predictive of all-cause mortality. We also found evidence suggesting that CMC fully mediates the effect of race on all-cause mortality. Number of CMC only partially mediated the effect of SES on mortality.

Conclusion—The number of CMC fully mediates the effects of race and partially mediates the effects of SES on all-cause mortality in the USA. Mortality prevention for minority populations will benefit tremendously from elimination of CMC disparities as well as enhancement of CMC management by minority populations. Elimination of the gap due to SES may be more challenging than the elimination of the racial gap in mortality.

Keywords

Race Socioeconomic; status Social; class Education Income African-Americans Mortality Chronic medical conditions

Background

Chronic medical conditions (CMCs) such as obesity, diabetes, arthritis, hypertension, heart disease, stroke, and cancer are among the most common, costly, and preventable of all health problems [1]. As of 2012, one out of every two American adults—117 million individuals—had at least one CMC, and one out of four adults had two or more CMCs [2]. The Medicare Current Beneficiary Survey (MCBS) has shown that among the population over age of 65, 57 % have arthritis, 55 % have hypertension, 38 % have pulmonary disease, 17 % have diabetes, 17 % have cancer, and 16 % have osteoporosis [3, 4]. As CMCs tend to cluster, a considerable proportion of older adults suffer multiple CMCs [5, 6]. Prevalence of multiple CMCs, also known as multi-morbidity, is expected to continue to rise [7, 8, 9].

Poor socioeconomic status (SES) increases risk of CMC such as type II diabetes [10], heart disease, [11], stroke [12], and some types of cancers [13]. According to the life-course epidemiological perspective, racial minority status and low SES increases exposures to a wide range of risk factors over the life course that cumulatively increases risk of CMC, as well as multi-morbidity. In a study using data from Health and Retirement Survey, poor SES early in life was associated with an 8 % increase in number of CMC, and, a lower income was associated with a 5 % increase in number of CMC [14]. Blacks and other minority populations have higher prevalence of CMC [15, 16]. Compared with Whites, Black Americans have greater physical health morbidity and mortality at every age [17]. For example, Blacks develop CMC from an earlier age as a considerable proportion of Blacks suffer CMC at midlife [18]. Blacks are also more likely than Whites to die from hypertension, heart disease, stroke, type II diabetes, and kidney disease [18].

Built on the Link and Phelan [19] theory of fundamental causes, which suggests SES influences a wide range of health outcomes through access to social resources [20], we conducted the current study with two aims: (1) to assess whether race and SES (education and family income) at baseline have separate and additive effects on all-cause mortality over 25-year follow-up and (2) to test whether the number of CMC—as a time-varying factor—mediates the effects of race and SES on risk of all-cause mortality. In this study, we

hypothesized that the number of CMC over time mediates the effects of race and baseline SES on mortality, particularly because race and SES increase CMC risk [10, 11, 12, 15, 16, 21] and seven of the top ten causes of death are due to chronic diseases, with two particular CMC—heart disease and cancer—accounting for almost half of all deaths [22].

Methods

Data came from the Americans' Changing Lives (ACL), 1986–2011. ACL is a nationally-representative longitudinal study of US adults [15]. More information on the sampling and data collection has been published elsewhere [23, 24].

Ethics

All procedures were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) with the Helsinki Declaration of 1975. All participants provided informed consent. The study protocol received approval from the University of Michigan Institutional review board (IRB).

Sampling and Participants

The ACL has enrolled a stratified multistage probability sample of American adults. All participants were living in the continental US in 1986 (representing 70 % of sampled households and 68 % of sample individuals at baseline), were noninstitutionalized, and were adults 25 years old and older. The study oversampled older adults (age > 60) and Blacks. The overall sample size was 3617.

Table 1 presents descriptive statistics for all variables. Participants were only non-Hispanic Whites ($n = 2205$) or non-Hispanic Blacks ($n = 1156$). Population was composed of 53 % women and 47 % men, with a mean age of 42.5 years at baseline (1986). Individuals had 13.18 (0.10) on average education years at baseline. A 74.5 % of population had at least 12 years of education. Chronic medical conditions showed an increase from wave 1 in 1986 to wave 4 in 2001.

Process

Face to face interviews were used for data collection at baseline. Some information were collected using telephone interviews in the next waves. Death certificates and National Death Index (NDI) information were used to assess date of death.

Measures

Demographic Factors

Demographic characteristics included gender (male as the referent category) and age (a continuous variable).

Socioeconomic Status

Socioeconomic characteristics included educational attainment (11 years of schooling or more) and family income, measured in 1986. While education was conceptualized as a dichotomous variable, income was operationalized as a continuous variable.

Race

Information on participant's race at baseline were collected in 1986 using multiple survey items. Self-identified race was defined as non-Hispanic Whites ($n = 2205$) or non-Hispanic Blacks ($n = 1156$). Non-Hispanic White was the referent category. Participants were asked an open-ended response to the question, "In addition to being American, what do you think of as your ethnic background or origins?" and then were asked: "Are you white, black, American Indian, Asian, or another race?" and allowed to answer with multiple categories. Individuals who responded with more than one non-White group were asked to identify which "best described" their race. ACL also assessed the state or foreign country of birth. Together, these items were used to construct race categories of "Non-Hispanic White," "Non-Hispanic Black," "Non-Hispanic Native American," "Non-Hispanic Asian," and "Hispanic".

Number of CMC

Conceptualized as a time-varying covariate, the number of CMC was measured using self-reports by respondents of whether a health care provider had ever told them they had each of seven focal conditions (hypertension, type II diabetes, chronic lung disease, heart disease, stroke, cancer, and arthritis) in 1986, 1989, 1994, 2001, and 2011. A sum score was calculated, ranging from 0 to 7. Higher scores suggest more CMCs [15, 24].

Mortality

Mortality data (cause and time) was obtained through the National Death Index (NDI), death certificates, and informants. In almost all cases, time and cause of death were verified with death certificates. Mortality data were collected for all participants regardless of their retention in the longitudinal study. With a handful of exceptions, we were able to determine death status of all our participants. In a handful of cases, the date of death was ascertained from informants or the NDI report, rather than the death certificate. Overall, 1737 Black or White participants were deceased, while 1624 Black or White individuals survived [25, 26].

Statistical Analysis

As ACL has used a complex sampling strategy, Stata version 12 was used to account for the survey design. Standard errors were recalculated based on the strata, clusters, and nonresponse weights (at baseline), using Taylor series linearization.

Cox proportional hazards models were used for data analysis. These models require two outcomes: (1) a binary outcome and (2) time to event (since baseline). Mortality was coded as 1 if death occurred between 1986 and 2011, and 0 otherwise. Time to death, or to censoring, was defined as the number of months from baseline to time of death or the end of

the year 2011. We used -estat phtest- in Stata to evaluate the proportional hazard assumptions of our Cox proportional hazard models, using Schoenfeld residual analysis [27].

The main predictors of interest were race and SES at baseline (education and family income) measured in 1986. The main outcome was time to the event of all-cause mortality over up to 25 years of follow-up. The numbers of CMCs—conceptualized as time-varying covariates—were the potential mediators, measured at four initial waves (1986, 1989, 1991, and 2001). Covariates included baseline demographic factors (age, gender) measured in 1986, used to adjust for potential confounding. Although race and SES were associated, there was no strong evidence regarding multi-linearity between education, family income, and race. In addition, age was positively correlated with CMC, and there was no source of bias due to collinearity in this study.

First, we ran models to test if race and baseline SES predict time to death over up to 25 years of follow-up. In the next step, we added CMC as a time-varying covariate at each wave to evaluate the mediating effects of CMC on the effects of race and SES on mortality, while age and gender were controlled. Hazard ratios with 95 % confidence intervals (CI) are reported.

Results

Table 2 shows correlations between study variables. Table 3 presents the results for two Cox proportional hazards models with and without CMC as covariates. *Model 1* only included race, age, and gender. *Model 2* also controlled for CMC as time-varying covariates. The results of *Model 1* show that race was related with risk of mortality, as Blacks and other race groups were at higher risk than Whites. *Model 2* shows that CMCs at 1994 and 2001 but not race were associated with mortality, suggesting that CMC—as a time-varying covariate—fully mediates the effect of race on mortality.

Table 4 shows the results for two Cox proportional hazards models. *Model 1* only included SES (education and family income), age, and gender. *Model 2* also controlled for CMC, as time-varying covariates. The results of *Model 1* show that lower education and family income were associated with higher risk of mortality. *Model 2* shows that CMCs at 1994 and 2001 and baseline education but not baseline family income were related with mortality, suggesting that CMC partially mediated the effect of SES on mortality (Table 4).

Figure 1 shows hazard ratios (HRs) associated with race, education, and income on all-cause mortality using Cox proportional hazards models. This figure shows HRs before (*Model 1*) and after (*Model 2*) adjusting for time-varying chronic medical conditions (CMCs). This figure suggests that CMC fully mediates the effect of race and partially mediates the effects of education and income on mortality.

Discussion

Using a nationally representative sample, our study showed at least three main findings. First, race and SES at baseline had separate effects on all-cause mortality. Second, when we tested the combined effect of race and baseline SES, SES but not race predicted all-cause

mortality risk. Third, CMC fully mediated the effect of race and partially mediated the effect of baseline SES on mortality.

Among individuals with low SES, disadvantage and adversities accumulate and increase the risk of multiple CMCs across the life course [28, 29]. Although socioenvironmental exposures differently impact disease etiology [30], several CMC types share common risk factors [31]. It has been shown that SES in childhood and adulthood has combined effects on health outcomes such as CMC [32, 33].

In our study, education had a residual effect on mortality over CMC across time. The protective effect of education on health is one of the most robust findings in the sociology and epidemiology literatures [34, 35]. Mirowsky and Ross (2003) described the effect of education on health as “enduring, consistent, and growing” [36]. Confirmed by longitudinal studies such as the Health and Retirement Study (HRS) [37], Americans’ Changing Lives Study [38, 39], British Cohort Study (BCS) [40], and Survey of Health, Ageing and Retirement in Europe (SHARE) [41], education has been shown to protect against disability, illness [38], and mortality [42, 43, 44, 45].

Some studies have shown that health effect of education is over and above other socioeconomic factors [46, 47, 48]. The effect of education on health is due to multiple mechanisms [49]. Education credentials such as a university degree affect attained employment and income [50]. Certain education levels operate as “gatekeepers” to jobs that are more rewarding and have higher pay [51, 52]. Each additional year that individuals spend at school improves their human cognitive resources and behaviors as well [53]. Such psychosocial and cognitive resources help individuals to avoid risk of CMC and minimize the CMC consequences once they occur [19, 54]. As education operates in multiple mechanisms such as increasing income [51, 52] as well as a wide range of personal and social resources [19, 55, 56], CMC may not capture all effects of education on mortality. Herd et al. used data from the ACL (1986 through 2001/2002) and showed that education is more predictive than income on the onset of both functional limitations and chronic conditions, while income is more strongly associated than education with the progression of both [38].

There is a need for future studies on how SES gradients influence health via differential exposure or vulnerability to stress and other risk factors [57, 58, 59]. In a landmark study, Lantz and colleagues used ACL data and showed that (1) socioeconomic position reflects exposure to life events and other types of stressors, and (2) negative lifetime events predicted mortality. Authors showed that differential exposure to stress and negative life events is one of many ways in which socioeconomic inequalities in health are produced in society [58]. In another study, exposure to stressors across several domains explained the effect of SES on self-rated health outcomes. The study suggested that exposure to psychosocial stressors may be one of the mediators underlying the higher prevalence of poor health within lower SES groups [59].

When we tested the combined effect of race and SES, SES but not race predicted all-cause mortality. Some studies have shown that race and SES are independently and significantly

related to poor health outcomes [60]. Whites have better health status than racial-ethnic minorities, especially Blacks, and a substantial part of these disparities has been attributed to the link between race and SES [60]. We found that the racial disparity we normally see in national samples was attenuated or completely erased when White and Black Americans live under similar conditions. The sole exception was smoking, where we found that white residents in the Baltimore sample were more likely to smoke than black residents. When social factors and medical care are equalized, racial disparities are minimized. [61].

As expected, later years of CMC in 1994 and 2001, and not earlier years in 1986 and 1989, were important as mediators in the relationship between race to mortality and social class to mortality. This is because of the cumulative nature of CMC which results in strong correlation between CMC across time points (Table 2). For instance, CMCs in 1994 and 2001 cover CMC in 1986 and 1989, as CMC never cures and only accumulates. As a result, while CMCs in 1994 and 2001 are in the model, CMCs in 1986 and 1989 are also included.

Socioeconomic status may confound the effect of race on health [62]. Socioeconomic status may also mediate the effect of race on health [63]. Race may also moderate the effect of SES on health [64, 65]. SES may also moderate the effect of SES on health [66, 67]. Health status varies by both race and socioeconomic status, and socioeconomic status tends to be lower among black Americans than among white Americans. Consequently, the overlap between these two factors complicates efforts to determine whether it is “race and class” or “race or class” that produces disparities in health status [61, 68].

Our findings have policy and public health implications. There are state-of-the-art effective interventions for prevention of CMC in various settings [3, 69, 70]. Promotion of healthy lifestyle is one of the main strategies for prevention of CMC [71]. CMC prevention policies and programs that particularly enhance exercise and healthy nutrition should particularly target low SES and minorities.

Enhancing CMC self-management among minority populations should be also regarded as a main strategy for increasing life expectancy of Blacks and low SES populations with multiple CMCs [72, 73, 74]. Due to the presence of multiple chronic conditions, self-management of CMC is more challenging among Blacks and other minorities, as they face barriers such as limited resources, poor access to healthcare, and lack of insurance [74]. As culturally sensitive tailored interventions higher efficacy and acceptability [75, 76], interventions should be specifically designed for the target populations. Enhancement of physical activity and improvement of nutrition at the community level may contribute to CMC prevention as well as enhanced CMC management for those who have already developed CMC. Policies that alter fundamental causes such as neighborhood segregation, unemployment, equal pay, walkability, healthy food access, and access to health care should consider minorities and low SES communities a priority.

Some programs should focus on behavioral changes of minorities and individuals with low SES. Public health programs in economically deprived and majority minority communities should facilitate walking, and to reduce obesity, smoking, excessive drinking, and sedentary life style [77, 78, 79, 80]. A major proportion of CMC and associated burden is caused by

smoking, drinking, low exercise, and other behaviors that are more common in low SES and high minority communities. As behavior risk factors are clustered, interventions should target multiple risk factors in low SES and minority individuals [53, 81].

Our study has major implications for improving life expectancy by race and socioeconomic status and also elimination of disparities in life expectancy based on race and SES. The number of CMC fully mediates the effects of race and partially mediates the effects of SES on all-cause mortality in the USA. Mortality prevention for minority populations will benefit tremendously from elimination of CMC disparities as well as enhancement of CMC management by minority populations. Elimination of the gap due to SES may be more challenging than the elimination of the racial gap in mortality and would require covering factors other than CMC as well.

Findings of this study should be interpreted in the context of USA. We already know that countries differ in how SES and CMC influence health [21, 82, 83]. In a cross-country comparison of the effects of SES and CMC on morbidity, only in the USA, number of CMC fully explained the effect of income on health. Authors showed that CMC does not similarly explain the effect of SES on health outcomes in several countries such as China, India, Russia, Costa Rica, Puerto Rico, Mexico, Argentina, Barbados, Brazil, Chile, Cuba, Uruguay, Ghana, and South Africa [84]. It is also unknown whether or not the results hold for both genders or not.

This study had at least four major limitations. First, only our mediator (CMC) was allowed to vary over time (time-varying covariate), SES was considered fix. This approach was taken because we were interested in comparing the long-term effects of race and SES at baseline, through CMC as the only mediator, using a life-course epidemiology approach, which suggests that baseline SES will have late effects. Over the 25-year follow-up from 1986 to 2011, deep globalization and dramatic economic pattern change, and the social class of study cases could vary significantly in these 25 years. Future research should also collect SES information over multiple times for more accurate modeling. Second, we used self-reported data to measure CMC. Self-reported CMC is subjected to measurement error (recall bias or miss-classification) [77]. Differential validity of CMC across racial and SES groups may be another source of bias in this study [85, 86, 87]. Third, CMC type was not entered into the analysis. This is particularly important because various CMC types have differential effects on mortality. Fourth, this study did not measure access and use of health services that may confound or mediate the associations of interest in this study. Fifth, socioeconomic characteristics were only measured in 1986.

In summary, in a nationally representative study in the USA, the number of CMC fully mediates the effects of race and partially mediates the effects of SES on all-cause mortality. We believe that elimination of CMC disparities as well as enhancement of CMC management among minority populations should be considered as core elements of policies and programs that aim to eliminate or at least reduce the mortality gap due to race and SES in the USA. Elimination of the gap due to SES may be, however, more challenging than elimination of the racial gap in mortality.

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Fig. 1.
Hazard ratios before (*Model 1*) and after (*Model 2*) adjusting for time-varying chronic medical conditions (CMC) for race, education, and income on all-cause mortality using Cox proportional hazards models

Table 1.

Descriptive statistics

All		
	% (SE)	95 % CI
Gender		
Men	47.09 (0.01)	44.86–49.34
Women	52.91 (0.01)	50.66–55.14
Education		
11 years and less	25.49 (0.01)	26.00–28.62
12 years and more	74.51 (0.01)	71.38–77.40
	Mean (SE)	95 % CI
Age (years)	42.56 (0.42)	41.71–43.41
Education (years)	13.18 (0.10)	12.98–13.38
Income (USD 10,000)	6.01 (0.11)	5.78–6.24
Chronic medical conditions		
CMC 1986	0.57 (0.03)	0.51–0.62
CMC 1989	0.62 (0.02)	0.57–0.66
CMC 1994	0.80 (0.03)	0.74–0.86
CMC 2001	1.23 (0.04)	1.15–1.31

CMC chronic medical conditions

Table 2.

Correlation between race, socioeconomic status, and chronic medical conditions

	1	2	3	4	5	6	7	8	9
Blacks	1.00								
Women	0.06	1.00							
Age	−0.03	0.17 *	1.00						
Education	−0.25 *	−0.07	−0.24 *	1.00					
Income	−0.27 *	−0.21 *	−0.16 *	0.42 *	1.00				
CMC 1	0.09	0.16 *	0.45 *	−0.30 *	−0.23 *	1.00			
CMC 2	0.09	0.19 *	0.45 *	−0.27 *	−0.22 *	0.71 *	1.00		
CMC 3	0.09	0.15 *	0.43 *	−0.25 *	−0.19 *	0.63 *	0.66 *	1.00	
CMC 4	0.05	0.16 *	0.40 *	−0.19 *	−0.16 *	0.53 *	0.56 *	0.61 *	1.00

* $P < 0.05$

Table 3.

Mediating effect of chronic medical conditions on the effect of race on all-cause mortality using Cox proportional hazards models

	HR (SE)	95 % CI	P	HR (SE)	95 % CI	P
	Model 1			Model 2		
Race						
Non-Hispanic Whites Ref						
Non-Hispanic Blacks	1.32 (0.07)	1.20–1.46	<0.001	1.18 (0.14)	0.94–1.49	0.146
Age	1.08 (0.00)	1.08–1.09	<0.001	1.08 (0.00)	1.07–1.09	<0.001
Gender						
Men	Ref			Ref		
Women	0.66 (0.03)	0.60–0.72	<0.001	0.85 (0.09)	0.68–1.05	0.133
Time-varying CMC						
CMC 1986	–	–	–	1.00 (0.00)	1.00–1.00	0.964
CMC 1989	–	–	–	1.00 (0.00)	1.00–1.00	0.648
CMC 1994	–	–	–	1.01 (0.00)	1.01–1.02	0.049
CMC 2001	–	–	–	1.01 (0.00)	1.01–1.02	0.001

CMC chronic medical conditions, HR hazard ratio, SE standard error, CI confidence interval

Table 4.

Mediating effect of chronic medical conditions (CMC) on the effect of socioeconomic status (SES) on all-cause mortality using Cox proportional hazards models

	HR (SE)	95 % CI	<i>P</i>	HR (SE)	95 % CI	<i>P</i>
	Model 1			Model 2		
Education						
11 years and less	Ref			Ref		
12 years and more	0.86 (0.05)	0.77–0.95	0.005	0.76 (0.09)	0.60–0.96	0.020
Income	0.91 (0.01)	0.89–0.93	<0.001	0.97 (0.02)	0.93–1.02	0.201
Age	1.08 (0.00)	1.07–1.08	<0.001	1.07 (0.00)	1.07–1.08	<0.001
Gender						
Men	Ref			Ref		
Women	0.60 (0.03)	0.54–0.66	<0.001	0.82 (0.09)	0.65–1.03	0.083
Time-varying CMC						
CMC 1986	–	–	–	1.00 (0.00)	1.00–1.00	0.814
CMC 1989	–	–	–	1.00 (0.00)	1.00–1.00	0.771
CMC 1994	–	–	–	1.01 (0.00)	1.01–1.02	0.036
CMC 2001	–	–	–	1.01 (0.00)	1.01–1.02	0.001

CMC chronic medical conditions, HR hazard ratio, SE standard error, CI confidence interval