Distal, intermediate, and proximal mediators of racial disparities in renal disease mortality in the United States

Shervin Assari1,2*
1Department of Psychiatry, University of Michigan, Ann Arbor, MI, USA
2Center for Research on Ethnicity, Culture and Health, School of Public Health, University of Michigan, Ann Arbor, MI, USA

ABSTRACT

Background: Kidney failure and associated mortality is one of the major components of racial disparities in the United States.

Objectives: The current study aimed to investigate the role of distal (socioeconomic status, SES), intermediate (chronic medical diseases), and proximal (health behaviors) factors that may explain Black-White disparities in mortality due to renal diseases.

Patients and Methods: This is a nationally representative prospective cohort with 25 years of follow up. Data came from the Americans’ Changing Lives (ACL) study, 1986 to 2011. The study included 3361 Black (n = 1156) or White (n = 2205) adults who were followed for up to 25 years. Race was the main predictor and death due to renal disease was the outcome. SES, chronic medical disease (diabetes, hypertension, obesity), and health behaviors (smoking, drinking, and exercise) at baseline were potential mediators. We used Cox proportional hazards models for data analysis.

Results: In age and gender adjusted models, Blacks had higher risk of death due to renal disease over the follow up period. Separate models suggested that SES, health behaviors and chronic medical disease fully explained the effect of race on renal disease mortality.

Conclusions: Black-White disparities in rate of death due to renal diseases in the United States are not genuine but secondary to racial differences in income, health behaviors, hypertension, and diabetes. As distal, intermediate, and proximal factors contribute to racial disparities in renal disease mortality, elimination of such disparities requires a wide range of policies and programs that target income, medical conditions, and health behaviors.

Implication for health policy/practice/research/medical education:
In the United States, racial disparities in mortality due to renal disease are due to distal (socioeconomics), intermediate (hypertension and diabetes), and proximal (health behaviors) factors. Thus, elimination of racial disparities in renal disease mortality in this country requires a wide range of policies and programs that enhance income, promote behaviors, and prevent medical conditions such as hypertension and diabetes.

Please cite this paper as: Assari S. Distal, intermediate, and proximal mediators of racial disparities in renal disease mortality in the United States. J Nephropathol. 2016;5(1):51-59. DOI: 10.15171/jnp.2016.09

1. Background
In the United States, Blacks are at higher risk of mortality due to renal diseases (1,2). This is mainly because compared to Whites, Blacks are 3-4 times more likely to develop kidney failure. While Blacks only make up to 13% of the population, they account for one third of all kidney failures in the United States (1). It is still unknown whether or not racial disparities in renal disease mortality are genuine (i.e. due to biological factors such as genetic predisposition) or they are secondary to Black-White differences in 1) socioeconomic status (SES), 2) chronic medical disease (e.g. hypertension, diabetes, and obesity), or 3) health behaviors (e.g. exercise, smoking, and drinking). First, SES may be distal mediator of the link between race and burden of kidney disease. In the United States, race is a proxy of social class, and a considerable proportion of racial differences in
health is in fact due to class, education, income, or wealth (3-6). Compared to Whites, a lower number of Blacks receive high quality education (7). Blacks are at higher risk of school dropout than Whites (8). Even among highly educated individuals, a large gap exists in income and wealth of Blacks compared to Whites, which is mostly due to structural racism and societal factors (9-12).

Second, chronic medical diseases (e.g. hypertension, diabetes and obesity) may also be intermediate mediators of the association between race and renal disease mortality. Hypertension and diabetes are leading causes of kidney failure among Blacks (1). As a result, at least some of the racial disparities in burden of renal disease may be secondary to higher prevalence of chronic medical conditions among Blacks (13,14). Hypertension (15), diabetes (16), and obesity (17) which are known causes of kidney disease are all more common among Blacks (18-21). The role of diabetes (22,23) and hypertension (24,25) as etiologic factors in development of chronic kidney disease are well known. Obesity, which is more common among Blacks compared to Whites, also increases the risk of chronic renal disease (26). Finally, health behaviors such as exercise, smoking, and drinking may be proximal explanatory factors (i.e. mediators) that explain at least some of the racial disparities in renal disease mortality. We know that the distribution of health behaviors such as physical activity, smoking, and drinking vary across population groups (27). Exercise (28), smoking (29), and drinking (30) all differ between Whites and Blacks. All of these behaviors influence development and progression of renal diseases (31-34).

For at least three reasons, more studies are needed to explore whether or not racial disparities in renal disease mortality are genuine or secondary to racial gaps in SES, chronic medical disease, or health behaviors. First, very few studies have focused on mediators of racial differences in renal disease mortality. We know that the distribution of health behaviors such as physical activity, smoking, and drinking vary across population groups (27). Exercise (28), smoking (29), and drinking (30) all differ between Whites and Blacks. All of these behaviors influence development and progression of renal diseases (31-34).

For at least three reasons, more studies are needed to explore whether or not racial disparities in renal disease mortality are genuine or secondary to racial gaps in SES, chronic medical disease, or health behaviors. First, very few studies have focused on mediators of racial differences in renal disease mortality. We know that the distribution of health behaviors such as physical activity, smoking, and drinking vary across population groups (27). Exercise (28), smoking (29), and drinking (30) all differ between Whites and Blacks. All of these behaviors influence development and progression of renal diseases (31-34).

2. Objectives
To better understand mechanisms behind racial disparities in burden of renal disease in the United States, we examined roles of distal (SES), intermediate (chronic medical disease), and proximal (health behaviors) factors that may explain racial inequalities in mortality due to renal diseases between Blacks and Whites.

3. Patients and Methods
3.1. Study design
3.1.1. Setting
Data came from the Americans’ Changing Lives (ACL), a nationally-representative U.S. cohort study conducted from 1986 until 2011. Detailed information on the study design is available elsewhere (39,40).

3.2. Sampling and participants
The ACL enrolled a stratified multistage probability sample of adults ages 25 or above who lived in the continental U.S. in 1986. The study included 3617 non-institutionalized respondents (representing 70% of sampled households and 68% of sample individuals at baseline) with an oversampling of those age 60 and older, and African Americans. Wave 1 included 70% of sampled households and 68% of sampled individuals. Further interviews were conducted in 1989, 1994, 2001-2002 and 2011, but information from those interviews was not relevant for these analyses.

3.2.3. Measures
Information on demographics, SES, chronic medical conditions, and health behaviors was measured at the baseline during face to face interview in 1986.

3.2.4. Race
The predictor was race 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.

3.3. Demographic factors
Demographic indicators included age (a continuous variable as number of years since birth) and gender (a dichotomous variable with male as the referent category).

3.4. Socioeconomic characteristics
Data on SES were measured with an indicator of education (less than 12 years of education, and 12 years or more) and income (10 level categorical variable treated as a continuous measure; <$5000, $5-9K, $10-14K, $15-19K, $20-24K, $25-29K, 30-39K, $40-59K, $60-79K, $80 000+).

3.4.1. Diabetes and hypertension
Self-reported history of hypertension and diabetes
were assessed at baseline. Using separate items, all participants were asked whether a health care provider had ever told them they had hypertension or diabetes (40,41).

3.4.2. 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 equal to or larger than 30 kg/m² (42). 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 (43). However, using self-reported weight and height may lead to some degrees of underestimation of BMI (44), because of a systematic tendency for humans to underestimate their weight and to overestimate their height (45).

3.5. Health behaviors
We collected data on self-reported smoking (current smoker vs. other), drinking (current drinker vs. other), and exercise (frequency of physical activities) 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 1) often, 2) sometimes, 3) rarely, and 4) never.

3.6. Mortality due to renal diseases
The main outcome variable was time of death due to renal diseases. Information on all deaths from mid-1986 through 2011 was obtained through the National Death Index (NDI), 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 were 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 NDI report, rather than the death certificate (41,46). Cause of death was coded as unknown if death certificate or NDI report were unavailable. We used the ICD-9 and ICD-10 codes (47,48), whichever was current at the time the death was recorded, to determine death due to 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 World Health Organization (WHO), 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 due to other causes were censored at the time of death. Time of death was registered as number of months from time of enrollment to the study to time of death, based on the month of death and the month of the baseline interview.

3.7. Ethical issues
The research followed the tenets of the Declaration of Helsinki. This project was approved by the institutional review board (IRB) of the University of Michigan, Ann Arbor. All participants provided written consent and all data were kept confidential.

3.8. Statistical analysis
Univariate, bivariate, and multivariable analyses were performed using Stata 13.0 (Stata Corporation, College Station, TX, USA). Stratification and clustering in the estimation of standard errors was accounted for by using Taylor series linearization. P < 0.05 was considered statistically significant. Adjusted hazard ratios (HR) with 95% CI are reported. For multivariable analysis, six Cox proportional hazards models were fitted to the data. Cox proportional hazards models require a binary outcome (renal death) and time to the event or to censoring, 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 zero if the respondent did not die, or died from any other causes. Race was the main predictor. Baseline SES (education and income), health behaviors (smoking, drinking and exercise) and chronic medical disease (hypertension, diabetes, and obesity) were potential mediators.

4. Results
Table 1 shows descriptive statistics for the overall sample, and separately for Whites and Blacks. Whites
and Blacks did not differ in age or gender. However, compared to Whites, Blacks had lower education and income, smoked more, drank less, and had more hypertension (HTN), diabetes mellitus (DM) and obesity (All differences were significant at $P<0.05$). Death due to renal disease was also more common among Blacks than Whites.

Table 2 shows a summary of the results of six Cox proportional hazard regression models. According to the results of Model 1, which only adjusted for age and gender, Blacks were at higher risk of death due to renal disease. The adjusted hazard ratio for race did not remain significant in any Models 2 to 6 which controlled for SES, health behaviors, and chronic medical conditions.

5. Discussion
According to our findings, in age and gender adjusted models, race was associated with death due to renal disease over the follow up period. In separate models, SES (income), health behaviors (smoking, drinking, and exercise) and chronic medical disease (diabetes and HTN) fully explained the effect of race on death due to renal disease.

First, this study provided support for SES as a distal mediator of racial disparities in rate of death due to renal disease. It has previously shown that SES is a major determinant of behaviors and health outcomes (49-51). According to the fundamental cause theory (FCT), developed by Link and Phelan, low SES is a root cause of health problems including high-risk behaviors (52-55). Link and Phelan have listed four essential features for SES as fundamental causes of health inequalities (55). First, the effect of SES is not limited to specific health problems as it influences most health outcomes. Second, SES affects health through multiple risk factors and mechanisms. Third, SES involves access to resources that can be used to avoid health risks or to minimize the consequences of health problems once they occur. Finally, SES is continually influencing health inequalities despite radical changes in causes of morbidity and mortality over the past several decades (55).

Second, we found that HTN and diabetes are two intermediate mediators of such disparities. Diabetes and high blood pressure are leading causes of kidney failure among African Americans (1). As a result, at least some of the racial disparity in rate of death due to renal disease may be due to chronic medical conditions (13,14) such as HTN (15), diabetes (16), and obesity (17) which are more common among Blacks (18-21). A well-established literature shows that diabetes is associated with chronic kidney disease (22). In fact chronic kidney disease is one of the complications of diabetes (23). The association between chronic kidney disease and HTN is also bidirectional, and HTN is one of the known etiologic factors in development of chronic kidney disease (25). Longitudinal studies have shown that end stage renal disease is one of many potential outcomes in patients with HTN (18).

Third, health behaviors such as exercise, smoking, and drinking seem to be the most proximal mediators that explain at least some of the racial disparities in rate of death secondary to renal diseases. Distribution of

| Table 1. Descriptive statistics overall and based on race at baseline |
|-------------------------|------------------|------------------|------------------|------------------|
|                         | All              | Whites           | Blacks           |
|                         | 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)      | 4.66-6.16        | 5.57 (0.10)      | 4.88-6.26        | 3.63 (0.05)      | 3.23-4.03        |
| Exercise                | 0.02 (0.03)      | -0.03-0.07       | 0.04 (0.03)      | 0.01-0.07        | -0.22 (0.05)     | -0.33-0.02       |
| 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      |
| Gender                  | % (SE)           | 95% CI           | % (SE)           | 95% CI           | % (SE)           | 95% CI           |
| 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      |
| 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)      | 4.80-6.62        | 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)      | 0.63-2.02        |

Abbreviations: CES-D, Center for Epidemiologic Studies Depression Scale; HTN, hypertension; DM, diabetes mellitus; SE, standard error.

* $P<0.05$. 
of a wide range of social, medical, and behavioral factors. Distribution of other risk factors also differs between Whites and Blacks (19). Similar findings in other chronic conditions have suggested that not race, per se, but SES and other risk factors explain racial differences in outcomes (59-61).

Multiple studies have decomposed the effects of race, SES, and risk factors on health. Our study is, however, different from those who measure additive effects of race, SES, and medical conditions on outcomes (62-64). For instance, a number of studies have shown that race has a residual main effect above and beyond SES and other confounders on self-care and knowledge in HTN, diabetes, and other conditions (63,64). The real association between race, SES, and health is, however, complex and multiplicative rather than additive (65,66). The same risk factors including SES may not have the same effects across race groups (67). Some of these studies have shown that Blacks and Whites would show similar outcomes if they could have similar SES (68). Other studies have shown that SES interacts with race on outcomes (69). Farmer and Ferraro, for instance, showed that racial disparity in self-rated health was largest at the higher levels of SES, providing some evidence for the “diminishing returns” hypothesis. As education levels increases, Blacks may not have the same improvement in health as Whites (69).

In a study, Perneger et al conducted a case-control study to compared 716 patients with end-stage renal disease (ESRD) with 361 population controls. Race, indicators of SES, and indicators of access to health services were used as confounders. The results showed that race had a residual main effect above and beyond SES and other confounders on self-care and knowledge in HTN, diabetes, and other conditions (63,64). The real association between race, SES, and health is, however, complex and multiplicative rather than additive (65,66). The same risk factors including SES may not have the same effects across race groups (67). Some of these studies have shown that Blacks and Whites would show similar outcomes if they could have similar SES (68). Other studies have shown that SES interacts with race on outcomes (69). Farmer and Ferraro, for instance, showed that racial disparity in self-rated health was largest at the higher levels of SES, providing some evidence for the “diminishing returns” hypothesis. As education levels increases, Blacks may not have the same improvement in health as Whites (69).

In a study, Perneger et al conducted a case-control study to compared 716 patients with end-stage renal disease (ESRD) with 361 population controls. Race, indicators of SES, and indicators of access to health services were used as confounders. The results showed that race had a residual main effect above and beyond SES and other confounders on self-care and knowledge in HTN, diabetes, and other conditions (63,64). The real association between race, SES, and health is, however, complex and multiplicative rather than additive (65,66). The same risk factors including SES may not have the same effects across race groups (67). Some of these studies have shown that Blacks and Whites would show similar outcomes if they could have similar SES (68). Other studies have shown that SES interacts with race on outcomes (69). Farmer and Ferraro, for instance, showed that racial disparity in self-rated health was largest at the higher levels of SES, providing some evidence for the “diminishing returns” hypothesis. As education levels increases, Blacks may not have the same improvement in health as Whites (69).

In a study, Perneger et al conducted a case-control study to compared 716 patients with end-stage renal disease (ESRD) with 361 population controls. Race, indicators of SES, and indicators of access to health services were used as confounders. The results showed that race had a residual main effect above and beyond SES and other confounders on self-care and knowledge in HTN, diabetes, and other conditions (63,64). The real association between race, SES, and health is, however, complex and multiplicative rather than additive (65,66). The same risk factors including SES may not have the same effects across race groups (67). Some of these studies have shown that Blacks and Whites would show similar outcomes if they could have similar SES (68). Other studies have shown that SES interacts with race on outcomes (69). Farmer and Ferraro, for instance, showed that racial disparity in self-rated health was largest at the higher levels of SES, providing some evidence for the “diminishing returns” hypothesis. As education levels increases, Blacks may not have the same improvement in health as Whites (69).
care were assessed. Authors showed that adjustment for SES partially explained the odds ratio for Blacks. The proportions of ESRD incidence that could be attributed to race and income were 46% and 53% respectively. Authors showed that limited access to health care also explains some of the excess of ESRD in Blacks (70). Other studies have explored the role of the health care system or relation between patient and system as possible mediators of such disparities (71).

6. Conclusions
To conclude, we found that Black-White differences in rate of death due to renal diseases over a 25-year period are due to a complex network of distal, intermediate, and proximal factors, namely SES (income), chronic medical disease (diabetes and HTN), and health behaviors (smoking, drinking, and exercise), respectively. Our findings extend the existing knowledge on racial disparities in renal disease and associated mortality. This is particularly important as Black-White disparity in chronic kidney disease is a major challenge in the United States (72).

7. Limitations of the study
Despite the unique contribution that the current study makes to the literature, the results should be interpreted with consideration of the study limitations. The first limitation was lack of any measure of kidney disease at baseline or over course of the follow up. In addition, measurement of chronic medical conditions (HTN, diabetes, and obesity) was based on self-reported data, which is subject to recall bias (73). Further research can use multiple sources regarding the history of medical conditions. Future studies should assess how race, SES, behaviors, and clinically-diagnosed HTN and diabetes explain disparities in decline in kidney function and developments of end stage renal disease among Blacks and Whites. Despite these limitations, the study had major strengths, including a long term follow up, a nationally representative U.S. sample and a large sample of Blacks.

Author’s contribution
SA is the single author of the manuscript. SA designed the study, analyzed the data, drafted the paper, and revised the manuscript.

Conflicts of interest
SA declares that he has no conflicts of interest.

Funding/Support
The Americans’ Changing Lives (ACL) study was supported by Grant # AG018418 from the National Institute on Aging (DHHS/NIH), and per the NIH Public Access Policy requires that peer-reviewed research publications generated with NIH support are made available to the public through PubMed Central. NIH is not responsible for the data collection or analyses represented in this article. The ACL study was conducted by the Institute of Social Research, University of Michigan. Shervin Assari is supported by the Heinz C. Prechter Bipolar Research Fund and the Richard Tam Foundation at the University of Michigan Depression Center.

References
1. U.S. Renal Data System, USRDS 2010 Annual Data Report: Atlas of Chronic Kidney Disease and End-Stage Renal Disease in the United States. Bethesda, MD: National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases; 2010.
2. Assari S, Burgard S. Black-White differences in the effect of baseline depressive symptoms on deaths due to renal diseases: 25 year follow up of a nationally representative community sample. J Renal Inj Prev. 2015;4(4):127-34.
3. Navarro V. Race or class versus race and class: mortality differentials in the United States. Lancet. 1990;336(8725):1238-40.
4. Schulz AJ, Mullings L. Gender, race, class, and health: Intersectional approaches. San Francisco, CA: Jossey-Bass; 2006.
5. Williams DR. Race, socioeconomic status, and health the added effects of racism and discrimination. Ann N Y Acad Sci. 1999;896(1):173-88.
6. Williams DR, Lavizzo-Mourey L, Warren RC. The concept of race and health status in America. Public Health Rep. 1994;109(1):26.
7. Crichlow W, ed. Race, identity, and representation in education. Routledge; 2013.
8. Rumberger RW. Dropping out of high school: The influence of race, sex, and family background. Am Educ Res J. 1983;20(2):199-220.
9. Conley D. Being black, living in the red: Race, wealth, and social policy in America. University of California Press; 1999.
10. Shapiro TM. Race, homeownership and wealth. Wash. Journal of Law and Policy. 2006;2:53.
11. Altonji J, Blank R. Race and gender in the labor market. Handbook of Labor Economics. 1999; 3:3143-59.
12. Gee GC, Ford CL. Structural racism and health inequities: old issues, new directions. Du Bois Rev. 2011;8(1):115-32.
13. Cabassa LJ, Humensky J, Druss B, Lewis-Fernández R, Gomes AP, Wang S, et al. Do race, ethnicity, and psychiatric diagnoses matter in the prevalence of
multiple chronic medical conditions? Med Care. 2013;51(6):540-7.
14. Johnson-Lawrence VD, Griffith DM, Watkins DC. The effects of race, ethnicity and mood/anxiety disorders on the chronic physical health conditions of men from a national sample. Am J Men Health. 2013;7(4):58S-67.
15. Lindhorst J, Alexander N, Blignaut J, Rayner B. Differences in hypertension between blacks and whites: an overview. Cardiovasc J Afr. 2007;18(4):241-7.
16. Link CL, McKinlay JB. Disparities in the prevalence of diabetes: is it race/ethnicity or socioeconomic status? Results from the Boston Area Community Health (BACH) survey. Ethn Dis. 2009;19(3):288-92.
17. Jackson CL, Szklo M, Yeh HC, Wang NY, Dray-Spira R, Thorpe R, Brancati FL. Black-white disparities in overweight and obesity trends by educational attainment in the United States, 1997-2008. J Obes. 2013;140743. doi:10.1155/2013/140743.
18. Signorello LB, Schlundt DG, Cohen SS, Steinwandel MD, Buchowski MS, McLaughlin JK, Hargreaves MK, Blot WJ. Comparing diabetes prevalence between African Americans and Whites of similar socioeconomic status. Am J Public Health. 2007;97(12):2260-7.
19. Abate N, Chandalia M. The impact of ethnicity on type 2 diabetes. J Diabetes Complications. 2003;17:39-58.
20. Harris MI, Flegal KM, Cowie CC, Eberhardt MS, Goldstein DE, Little RR, et al. Prevalence of diabetes, impaired fasting glucose, and impaired glucose tolerance in US adults: the Third National Health and Nutrition Examination Survey, 1988–1994. Diabetes Care. 1998;21(4):518-24.
21. Sampson UK, Edwards TL, Jahangir E, Munro H, Wariboko M, Wassef MG, et al. Factors associated with the prevalence of hypertension in the southeastern United States: insights from 69,211 blacks and whites in the Southern Community Cohort Study. Circ Cardiovasc Qual Outcomes. 2014;7(1):33-54.
22. Laville M, Lengani A, Serme D, Fauvel J, Ouandaogo B, Zech P. Epidemiological profile of hypertensive disease and renal risk factors in black Africa. J Hypertens. 1994;12(7):839-43.
23. Seedit YK. Hypertension in black South Africans. J Hum Hypertens. 1999;13(2):96-103.
24. Richardson AD, Piepho RW. Effect of race on hypertension and antihypertensive therapy. Int J Clin Pharmacol Ther. 2000;38(2):75-9.
25. Weisstuch JM, Dworkin L.D. Does essential hypertension cause end-stage renal disease? Kidney Int. 1992;36:S33-7.
26. Norris KC, Agodaa IY. Unraveling the racial disparities associated with kidney disease. Kidney Int. 2005;68(3):914-24.
27. Sorkin DH, Billimek J. Dietary behaviors of a racially and ethnically diverse sample of overweight and obese Californians. Health Educ Behav. 2012;39(6):737-44.
28. August KJ, Sorkin DH. Racial/ethnic disparities in exercise and dietary behaviors of middle-aged and older adults. J Gen Intern Med. 2011;26(3):245-50.
29. Kandel DB, Kiros GE, Schaffran C, Hu MC. Racial/ethnic differences in cigarette smoking initiation and progression to daily smoking: a multilevel analysis. Am J Public Health. 2004;94(1):128-35.
30. Gilman SE, Breslau J, Conron KJ, Koenen KC, Subramanian SV, Zaslavsky AM. Education and race-ethnicity differences in the lifetime risk of alcohol dependence. J Epidemiol Community Health. 2008;62(3):224-30.
31. Knap B, Buturović-Ponikvar J, Ponikvar R, Bren AF. Regular exercise as a part of treatment for patients with end-stage renal disease. Ther Apher Dial. 2005;9(3):211-3.
32. Koning SH, Gansevoort RT, Mukamal KJ, Rimm EB, Bakker SJ, Joosten MM; PREVEND Study Group. Alcohol consumption is inversely associated with the risk of developing chronic kidney disease. Kidney Int. 2015;87(5):1009-16.
33. Orth SR, Hallan SI. Smoking: a risk factor for progression of chronic kidney disease and for cardiovascular morbidity and mortality in renal patients—absence of evidence or evidence of absence? Clin J Am Soc Nephrol. 2008;3(1):226-36.
34. Orth SR, Stöckmann A, Conradt C, Ritz E, Ferro M, Kreusser W, et al. Smoking as a risk factor for end-stage renal failure in men with primary renal disease. Kidney Int. 1998;54(3):926-31.
35. Hall YN. Racial and ethnic disparities in end stage renal disease: access failure. Clin J Am Soc Nephrol. 2012;7(2):196-8.
36. Peralta CA, Katz R, DeBoer I, Ix J, Sarnak M, Kramer H, et al. Racial and ethnic differences in kidney function decline among persons without chronic kidney disease. J Am Soc Nephrol. 2011;22(7):1327-34.
37. Gómez-Puerta JA, Feldman CH, Alarcón GS, Guan H, Winkelmayer WC, Costenbader KH. Racial and ethnic differences in mortality and cardiovascular events among patients with end-stage renal disease due to lupus nephritis. Arthritis Care Res (Hoboken). 2015;67(10):1453-62.
38. Norris KC, Kalantar-Zadeh K, Kopple JD. The role of race in survival among patients undergoing dialysis. Nephrol News Issues. 2011;25(13):13-4.
39. House JS, Lepkowski JM, Kinney AM, Merlo RP,
Assari S

Behaviors among iranian drug injectors; a national status determines risk of receptive syringe sharing

Assari S, Ahmadi K, Rezazade M. Socio-economic factors on health-related quality of life in adolescents after kidney transplant. Exp Clin Transplant. 2011;9(1):50-5.

Mero RP, Chen J. Socioeconomic factors, health behaviors, and mortality: results from a nationally representative prospective study. Arch Gen Psychiatry. 2006;63(7):824-30.

Taylor AW, Dal Grande E, Gill TK, Chittleborough CR, Wilson DH, Adams RJ, et al. How valid are self-reported height and weight? A comparison between CATI self-report and clinic measurements using a large cohort study. Aust N Z J Public Health. 2006;30(3):238-46.

Lantz PM, House JS, Lepkowski JM, Williams DR, Mero RP, Chen J. Socioeconomic factors, health behaviors, and mortality: results from a nationally representative prospective study of US adults. JAMA. 1998;279(21):1703-8.

Anderson RN, Minino AM, Hoyert DL, Rosenberg HM. Comparability of cause of death between ICD-9 and ICD-10: preliminary estimates. Natl Vital Stat Rep. 2001;18(2):49:1-32.

Centers for Disease Control and Prevention, National Center for Health Statistics. Instruction manual, part 9. ICD-10 cause-of-death lists for mortality statistics year 2003). National Center for Health Statistics. Instruction manual, part 9. ICD-10 cause-of-death lists for mortality statistics year 2003).

Malekahmadi MR, Rahimzadeh S, Dezfuli Nejad MI, Lankarani MM, Einollahi B, Assari S. Importance of socioeconomic, clinical, and psychological factors on health-related quality of life in adolescents after kidney transplant. Exp Clin Transplant. 2011;9(1):50-5.

Assari S, Ahmadi K, Rezazade M. Socio-economic status determines risk of receptive syringe sharing behaviors among iranian drug injectors; a national study. Front Psychiatry. 2015;23:194.

Assari S, Rezazade M, Ahmadi K, Sehat M. Socio-economic status may suppress the effect of knowledge on sexual risk among female sex workers. Int J Health Allied Sci. 2014; 3(2): 84.

Phelan JC, Link BG, Tehranifar P. Social conditions as fundamental causes of health inequalities: theory, evidence, and policy implications. J Health Soc Behav. 2010;51 (Suppl):S28-40.

Freese J, Lutfey K. Fundamental causality: challenges of an animating concept for medical sociology; Handbook of the Sociology of Health, Illness, and Healing. Springer; 2011:67-81.

Link BG, Phelan J. Social conditions as fundamental causes of health inequalities. Handbook of Medical Sociology; 2010:3-17.

Link BG, Phelan J. Social conditions as fundamental causes of disease. J Health Soc Behav. 1995;35:80-94.

Kong G, Singh N, Krishnan-Sarin S. A review of culturally targeted/tailored tobacco prevention and cessation interventions for minority adolescents. Nicotine Tob Res. 2012;14(12):1394-406.

Resnicow K, Baranowski T, Akuwula JS, Braithwaite RL. Cultural sensitivity in public health: defined and demystified. Ethn Dis. 1999;9(1):10-21.

Lengani A, Laville M, Serde M, Faufel JP, Ouandaogo Bj, Zech P. Renal insufficiency in arterial hypertension in black Africa. Presse Med. 1994;23(17):788-92.

Drake KA, Galanter JM, Burchard EG. Race, ethnicity and social class and the complex etiologies of asthma. Pharmacogenomics. 2008;9(4):453-62.

Krause JS, Broderick LE, Saladin IK, Broyles J. Racial disparities in health outcomes after spinal cord injury: mediating effects of education and income. J Spinal Cord Med. 2006;29(1):17-25.

Brown SA, Saunders LI, Krause JS. Racial disparities in depression and life satisfaction after spinal cord injury: a mediational model. Top Spinal Cord Inj Rehabil. 2012;18(3):232-40.

Hertz RP, Unger AN, Cornell JA, Saunders E. Racial disparities in hypertension prevalence, awareness, and management. Arch Intern Med. 2005;165(18):2098-104.

Zell JA, Rhee JM, Ziegas A, Lipkin SM, Anton-Culver H. Race, socioeconomic status, treatment, and survival time among pancreatic cancer cases in California. Cancer Epidemiol Biomarkers Prev. 2007;16(3):546-52.

Heisler M, Smith DM, Hayward RA, Krein SL, Kerr EA. Racial disparities in diabetes care processes, outcomes, and treatment intensity. Med Care. 2003;41(11):1221-32.
W, McKenzie S, et al. An interaction of race and ethnicity with socioeconomic status in rectal cancer outcomes. Ann Surg. 2011;253(4):647-54.

66. O’Connell MJ, Kasprov WJ, Rosenheck RA. Differential impact of supported housing on selected subgroups of homeless veterans with substance abuse histories. Psychiatr Serv. 2012;63(12):1195-205.

67. Hudson DL, Puterman E, Bibbins-Domingo K, Matthews KA, Adler NE. Race, life course socioeconomic position, racial discrimination, depressive symptoms and self-rated health. Soc Sci Med. 2013;97:7-14.

68. LaVeist T, Pollack K, Thorpe R Jr, Fesahazion R, Gaskin D. Place, not race: disparities dissipate in southwest Baltimore when blacks and whites live under similar conditions. Health Aff (Millwood). 2011;30(10):1880-7.

69. Farmer MM, Ferraro KF. Are racial disparities in health conditional on socioeconomic status? Soc Sci Med. 2005;60(1):191-204.

70. Saha S, Arbelaez JJ, Cooper LA. Patient-physician relationships and racial disparities in the quality of health care. Am J Public Health. 2003;93(10):1713-9.

71. Perneger TV, Whelton PK, Klag MJ. Race and end-stage renal disease. Socioeconomic status and access to health care as mediating factors. Arch Intern Med. 1995;155(11):1201-8.

72. Kochanek KD, Arias E, Anderson RN. How did cause of death contribute to racial differences in life expectancy in the United States in 2010. NCHS Data Brief. 2013;125:1-8.

73. Boyer GS, Templin DW, Goring WP, Cordonni-Huntley JC, Everett DF, Lawrence RC, et al. Discrepancies between patient recall and the medical record. Potential impact on diagnosis and clinical assessment of chronic disease. Arch Intern Med. 1995;155(17):1868-72.

Copyright © 2016 The Author(s); Published by Society of Diabetic Nephropathy Prevention. This is an open-access article distributed under the terms of the Creative Commons Attribution License (http://creativecommons.org/licenses/by/4.0), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.