

# Racial and socioeconomic disparities in end-stage renal disease in the United States

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## Abstract

### Introduction

African Americans are disproportionately burdened by advanced chronic kidney disease and end-stage renal disease. Low socioeconomic status has been shown to be associated with an increased risk of end-stage renal disease and mortality. Low socioeconomic status may affect end-stage renal disease risk and outcomes through social and psychosocial factors, which in turn manifest as unhealthy choices and encumbrance to access health care or health information. Individuals of low socioeconomic status may also have negative health consequences from factors related to the physical environment such as overexposure to toxins or pollutants. Race and socioeconomic status are highly correlated in the United States, and certain ethnic minority groups, namely African Americans, Hispanics and Native Americans, are more likely to experience poverty and may do so for their entire lifespan, thus leading many to surmise that socioeconomic status-related factors might explain the disparities in end-stage renal disease incidence and outcomes observed in African Americans. However, low socioeconomic status appears to have a stronger association with end-stage renal disease risk among African Americans than among whites

intimating that socioeconomic influences may be different or cumulative among African Americans. This review discusses the role of socioeconomic status in explaining racial disparities in end-stage renal disease incidence and outcomes and illuminates gaps in our current knowledge and potential areas of future research.

### Conclusion

The interplay between race and socioeconomic status in end-stage renal disease risk and outcomes is complex and not well understood. Socioeconomic factors, both community and individual level, are likely to contribute to racial disparities in end-stage renal disease risk through many different mechanisms.

### Introduction

Advanced chronic kidney disease (CKD) and, to a much greater extent, end-stage renal disease (ESRD) are two of the most striking examples of health disparities. Both advanced CKD and ESRD are marked by inequalities in the incidence and prevalence, risk factors and disease treatment across different races and ethnicities and socioeconomic status (SES). In the United States, the burden of advanced CKD disproportionately affects African Americans/blacks despite similar rates among racial/ethnic groups in early stages of CKD<sup>1-3</sup>. The incidence of ESRD is 3.4 times higher in blacks compared with whites<sup>3</sup>. The high incidence of ESRD among blacks has been attributed to the high prevalence of major CKD risk factors including hypertension<sup>4,5</sup>, diabetes<sup>6,7</sup> and obesity<sup>8</sup>; genetic predisposition<sup>9-11</sup>; low SES<sup>12</sup> and inequities in access to and quality of CKD care<sup>13</sup>.

Race and SES are strongly correlated in the United States, and certain racial/ethnic minorities are more likely to be classified as having low SES. Income (or poverty status) is one of the most commonly used metrics to ascertain SES. In 2011, 27.6% of blacks lived below the US federal poverty level compared with 9.8% of non-Hispanic whites<sup>14</sup>. Other measures of SES commonly used in research include education attained and occupation. However, it is important to recognise the complexity of measuring an individual's SES, and none of these metrics may be sufficient to fully capture it. Community-level SES factors such as neighbourhood poverty and residential segregation have also been used in research<sup>15,16</sup>. These factors may be equally or more important social determinants of health, especially in African Americans, than individual-level factors such as income. Low SES individuals/families tend to cluster, creating areas of high poverty density<sup>17</sup>. Poverty density directly impacts the community resources available to an individual. An individual with a low income living in or in close proximity to higher SES neighbourhoods may still have access to better schools, healthy foods, sanitary housing conditions, safe parks for recreation and quality health care as opposed to someone with higher income who is living in a poverty dense area<sup>17</sup>. It is also conceivable that individual-level SES factors may be modified by community-level SES factors.

Low SES is independently associated with ESRD incidence<sup>18,19</sup>. SES may influence ESRD risk through a number of mechanisms. Individuals

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living in poverty have a high burden of acute and chronic social and psychosocial stressors<sup>20,21</sup>, which may in turn lead to unhealthy behaviours and impact their ability to access health information or services. Individuals living in poverty are also more likely to be exposed to toxins or pollutants from their physical environment<sup>22</sup>. Additionally, the negative health consequences of being persistently disadvantaged may be cumulative<sup>21</sup>. The aim of this review was to discuss racial and socioeconomic disparities in ESRD in the United States.

### Discussion

The authors have referenced some of their own studies in this review. These referenced studies have been conducted in accordance with the Declaration of Helsinki (1964), and the protocols of these studies have been approved by the relevant ethics committees related to the institution in which they were performed. All human subjects, in these referenced studies, gave informed consent to participate in these studies.

### Race and ESRD

Blacks have a disproportionately high burden of CKD in the United States<sup>3</sup>. Racial disparities in access to care and quality of care in advanced CKD and treated ESRD have also been well documented. Blacks are less likely than whites to receive pre-ESRD care<sup>23</sup> and are more likely to be referred late for nephrology care<sup>24</sup>, both of which are associated with worse survival<sup>25,26</sup>. Blacks are also less likely to utilise home dialysis therapies<sup>27</sup> and have significantly lower rates of kidney transplantation<sup>28,29</sup>. Furthermore, blacks with ESRD are more likely to start haemodialysis without an arteriovenous fistula<sup>30</sup>, have untreated and higher intact parathyroid hormone levels<sup>31</sup> and receive inadequate dialysis<sup>32</sup> – all indices associated with poorer outcomes.

Despite the higher rates of progression to ESRD and inequalities in care, blacks with treated ESRD have a paradoxically better survival when compared with whites – a robust observation as elucidated by numerous studies<sup>33–46</sup>. The reason for this survival paradox is not well understood. Biological theories include a more favourable nutritional and/or inflammatory profile<sup>43</sup>, resilience to inflammation in blacks<sup>47</sup> and differential sensitivity to dialysis dose<sup>33</sup>. Others postulate that improved access to health care afforded by the US Centres for Medicare and Medicaid Services (CMS) ESRD insurance coverage program may confer a survival benefit to black patients who are more likely to be uninsured prior to dialysis initiation<sup>16</sup>. This provision of health insurance coverage improves access to care and medication as well as facilitates nutrition and social work counselling, which may have been previously lacking in these patients. A recent study by Kucirka et al.<sup>48</sup> challenged the robustness of this racial survival paradox by demonstrating that it was not present across all age strata. In their study, 18- to 30-year-old dialysis patients had a nearly twofold increased risk of death compared with whites. The authors postulated that this younger dialysis population in the United States might be particularly vulnerable to racial and socioeconomic disparities in CKD care and ESRD management, which may be offset by Medicare entitlement in the older adult population.

### Race, SES and ESRD incidence

Both community-level (such as neighbourhood poverty, residential segregation and access to quality foods and health care) and individual-level (such as education, income and health literacy) socioeconomic factors have been associated with risk of CKD<sup>49</sup>, incidence of ESRD<sup>18,19</sup> and treatment of advanced CKD and ESRD<sup>50,51</sup>. Whittle et al. in an ecological analysis

of the Maryland Network 31 ESRD Regional Registry demonstrated that the relative risk of hypertensive ESRD comparing African Americans with whites was attenuated from 5.6 to 4.5 (95% CI, 3.2–6.2) after accounting for education, hypertension, severe hypertension, diabetes and population age in their analysis<sup>52</sup>. Perneger et al. examined the role of low SES (household annual income and years of education) and limited access to care (health insurance status, number of missing teeth, usual source of care and use of preventative services) in ESRD incidence in a case-control study of 716 patients with ESRD and 361 population controls (age 20–64) from Maryland, Virginia, West Virginia and Washington DC<sup>53</sup>. They found that low SES and limited access to health care were independently associated with ESRD risk, and adjusting for these factors partially reduced the odds of ESRD in blacks. The proportion of ESRD incidence attributed to black race, low SES and poor access to care was 46%, 53% and 33%, respectively. Using National Health and Nutrition Examination Survey data from 1976 to 1980, Tarver-Carr et al. reported that socioeconomic factors, including poverty status and education, accounted for 12% of the excess risk of ESRD experienced by African Americans<sup>54</sup>. Similar findings have been demonstrated by other studies<sup>7,19,55</sup>. In 2008, Volkova et al. extended these prior studies by assessing whether the SES influence was different in African Americans and whites<sup>15</sup>. In a study of three southeastern US states, they showed that increasing neighbourhood poverty was associated with increasing risk of ESRD for both African Americans and whites, but the effect was greatest for African Americans<sup>15</sup>. This may indicate that African Americans and whites have differential exposure to the negative health consequences of low SES. Low SES may also potentiate differences in underlying biology between African Americans and

whites. The discovery of the association of apolipoprotein L1 (APOL1) risk variant status with hypertensive and HIV-related kidney disease suggest that a gene-environment interaction is very plausible<sup>9-11</sup>.

### Race, SES and ESRD survival

Few studies have examined the interaction of race and SES in ESRD survival. Earlier population-based studies in the United States found an association between low SES and higher mortality and indicated that this relationship may be limited to blacks. Port et al. in a cohort of 20- to 59-year-old haemodialysis patients in Michigan between 1980 and 1987 found a significant inverse relationship between the adjusted mortality risk for blacks and the average household income in their residential area; however, the trend for whites was not significant<sup>56</sup>. Garg et al. in a prospective cohort study of 3165 incident ESRD patients in the early 1990s confirmed the relation between income and ESRD mortality and showed that increasing neighbourhood income was associated with better survival<sup>51</sup>. However, more recent studies have not indicated that African Americans of low SES have worse survival on haemodialysis. The Eisenstein et al.'s study reported in 2009 used Zip Code level median income to stratify patients into low, middle and high income<sup>38</sup>. They found no difference in survival by income level, and black patients had better survival compared with whites across all income groups. Rodriguez et al., in 2007, using the racial composition of Zip Codes as a marker of neighbourhood SES, also found no difference in survival for blacks compared with whites living in Zip Codes where  $\geq 75\%$  of residents were black<sup>46</sup>. A more recent study by Kimmel et al. in 2013, using race-specific Zip Code median income, demonstrated that the lowest income category was associated with worst survival for blacks and whites; however, blacks

maintained a survival advantage over whites when they adjusted for all income categories<sup>16</sup>. They also examined the race-survival association by the dissimilarity index, which measures the severity of residential segregation, and demonstrated that blacks who were living in more racially segregated counties had a higher hazard of death while the risk of death for whites was unchanged. This finding suggests that community-level SES factors, such as neighbourhood of residence and living conditions, may play a more important role in African Americans than individual-level factors such as income. It is, however, important to note that in these latter studies the average age of the incident dialysis cohorts was 60 years or above, and none of these studies stratified by age group. This could possibly mask the effects of SES and access to care factors, which could be more racially disparate in a young adult dialysis cohort because of Medicare eligibility among older patients<sup>48</sup>.

### Conclusion

In summary, the interplay between race and SES in ESRD risk and outcomes is complex and not well understood. Socioeconomic factors, both community and individual level, are likely to contribute to racial disparities in ESRD risk through many different mechanisms. Low SES also appears to have a differential effect in African American compared with whites, possibly through a dose relationship (years of exposure) and/or by potentiating racial differences in biology. Studies are needed to address whether there may be a cumulative effect on the chronically disadvantaged. Future studies examining the role of SES in ESRD risk and outcomes should also include children and young adults who may be particularly vulnerable to the negative health consequences of low SES. Furthermore, studies should also include the pre- and perinatal

periods, where exposure to psychosocial and physical factors may be particularly relevant; and further research is also needed to advance our understanding of gene-environment interactions in CKD.

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