Racial disparities in renal function: the role of racial discrimination. The Brazilian Longitudinal Study of Adult Health (ELSA-Brasil)

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ABSTRACT

Background Racial discrimination may play a significant role in higher incidence and poorer prognosis of chronic kidney disease among Black individuals. This study set out to investigate the association between racial discrimination and renal function and to estimate the contribution of racial discrimination to existing racial disparities in renal function.

Methods A cross-sectional analysis using baseline data (2008–2010) of 14 355 participants (35–74 years) in the Brazilian Longitudinal Study of Adult Health cohort study. Renal function was estimated based on estimated glomerular filtration rates (eGFR) obtained by the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation. Racial discrimination was assessed using a modified version of the Lifetime Major Events Scale ; race/colour was self-reported. Covariates included were age, sex, level of education and selected health-related factors.

Results Racial discrimination was reported by 31.6%, 6.3% and 0.8% of Black, Brown and White individuals, respectively. The older the age, the lower the prevalence of racial discrimination among Blacks. Racial discrimination was independently associated with lower mean eGFR (β=−2.38; 95% CI −3.50 to −1.25); however, associations were limited to individuals aged under 55 years. In this age group, eGFR differences between Black and White individuals were reduced by 31% when exposure to racial discrimination was accounted for.

Conclusion Blacks are approximately 40 times more likely to report racial discrimination than Whites. Racial discrimination was associated with lower mean eGFR and explained a significant portion of eGFR differences between Black and White individuals aged under 55 years. Exposure to experiences of racial discrimination should be accounted for in studies investigating racial disparities in renal function.

INTRODUCTION

The impact of racial inequality on chronic kidney disease (CKD) risk and prognosis has been widely recognised, particularly in end-stage renal disease.[1 2] However, the reasons why Black individuals are disproportionately affected by CKD are not completely understood, since this association cannot be fully explained by socioeconomic factors, health-related behaviours, access to healthcare or proximal risk factors for CKD, such as hypertension.[1 2]

Prior experience of racial discrimination has been implicated as a potential contributing factor for higher CKD rates and faster CKD progression among Black people and other minorities, at least in theory.[1–3] Racial discrimination has been associated with proximal risk factors for CKD such as hypertension in North American studies.[4] Moreover, biological changes triggered by exposure to racial discrimination, such as premature ageing and low-grade systemic inflammation,[5–10] are important predictors of incident CKD and kidney function decline.[11–13]

Although plausible, associations between racial discrimination and poor renal function have seldom been subjected to scientific scrutiny,[1 2] with only one recent study published to date.[14] That study involved a representative sample of African Americans and White individuals aged 30–64 years living in Baltimore (USA) and revealed that, while not associated with baseline estimated glomerular filtration rate (eGFR), racial discrimination was weakly associated with a 5-year decline in eGFR among African-American women.[14]

Studies carried out in low/middle-income countries with highly admixed populations and false 'racial democracy' ideology such as Brazil[15] are important to unveil potential relations between perceived racial discrimination and renal function. Brazil has a high rate of miscegenation, with self-reported Black or Brown (or '*pardo*', ie, of mixed race/colour) individuals accounting for 7.6% and 43.1% of the population, respectively.[16] Hence, given historical policies aimed at 'Whitening' a population comprising large numbers of Black people,[17] Brazilian racial classification does not replicate the binary Black-White divide found in other societies, such as the USA. Despite the lack of discriminatory laws after the abolition of slavery, Brazil is far from being a 'racial democracy', since Brazilian social practices and discourse have been strongly permeated by racial discrimination against Black and Brown individuals.[17] Studies that investigated social mobility, income inequalities and educational opportunities among Blacks and Browns in Brazil show that both are in huge disadvantage compared to Whites in all those indexes .[18] These minorities are also disproportionately affected by residential segregation in Brazil,[19] another clear evidence of structural racism. As a consequence, racial disparities in health are mounting for most leading causes of death, risk behaviours and healthcare use in the

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country.[20–22] Yet, studies investigating associations between racial discrimination and health outcomes in the Brazilian population are lacking.

Previous study of the Brazilian Longitudinal Study of Adult Health (ELSA-Brasil) revealed 8.9% prevalence of CKD, with higher prevalence among Black (11.1%) and Brown (9.2%) compared with White (7.9%) individuals. Prevalence differences could not be fully explained by socioeconomic conditions and proximal risk factors for CKD. Even when these variables accounted for, Blacks and Browns still had 23% (OR, 95% CI 1.03 to 1.47, p<0.05) and 16% (OR, 95% CI 1.00 to 1.35, p<0.05) more chances of presenting CKD than Whites , respectively.[23] Therefore, the higher risk of CKD among Blacks and Browns remains partially unexplained.

This study set out to investigate cross-sectional associations between racial discrimination and renal function and to estimate the contribution of racial discrimination to existing racial disparities in renal function.

METHODS

Study participants

This study was based on baseline data (2008–2010) from the ELSA-Brasil, a multicentre cohort study involving 15 105 civil servants aged 35–74 years and enrolled in universities and research institutions located at six Brazilian capitals (Belo Horizonte, Porto Alegre, Rio de Janeiro, Salvador, São Paulo and Vitória). This study was approved by the ethics committees of all institutions involved, and volunteers gave written consent to participate. Further study design and cohort profile details have been given elsewhere.[24 25]

Individuals with missing glomerular filtration rate (n=7), discrimination (n=28) or race/colour (n=184) data were excluded from the analysis, along with those self-declaring as Brazilian indigenous (n=157) or of Asian descent (n=374), who constituted a very small group. Therefore, the final samples comprised 14 355 participants.

Outcome assessment

Fasting (12 hours) blood samples were collected by venipuncture. Serum creatinine levels were then determined using the kinetic Jaffé method (Advia 1200 Siemens, USA) following application of a conversion factor derived from calibration samples traceable to an isotope dilution mass spectrometry.[26] eGFRs were estimated by the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI)equation[27] without correction for race/colour.[23]

Classification according to race/colour

Classification according to race/colour was based on the following question: 'The Brazilian Census (IBGE) uses the terms 'Black', 'Brown' ('*Pardo*'), 'White', 'Asian descendent' and 'Brazilian indigenous' to describe people's colour or race. If you were to answer the IBGE census today, how would you declare yourself with regards to colour or race?'

Lifetime racial discrimination

Lifetime discrimination was assessed using a modified version of the Lifetime Major Events Scale.[28] This tool captures unfair treatment on the following domains: at public places (unequal treatment at public places, such as banks, shops, hospitals or government departments); at work (being fired or not recommended for promotion); at police stations (being wrongfully accused, searched or harassed); at schools or colleges (being unfairly discouraged at a given school or college); regarding housing rights (unequal treatment when trying to rent a place or live as part of the community). Respondents reporting unfair treatment in any of the domains on race/colour discrimination grounds were coded as having experienced racial discrimination.

Covariates

Age, sex, level of education (university degree, high school, complete elementary school, incomplete elementary school), household income *per capita* (quintiles), type of occupation/job (manual, not manual but routine work, not manual and not routine work) and several health-related factors were used as covariates in this study. Tobacco smoking (No/Yes), physical inactivity (measured by the leisure time section of the long version of the International Physical Activity Questionnaire), excessive alcohol consumption (≥210 g or ≥140 g of alcohol per week for men and women, respectively), hypertension (systolic blood pressure ≥140 mm Hg, diastolic blood pressure ≥90 mm Hg or use of antihypertensive medication), diabetes (self-reported, use of antidiabetic drugs, fasting glucose ≥126 mg/dL, glucose tolerance test ≥200 mg/dL or glycated haemoglobin ≥6.5%), self-reported medical diagnosis of cardiovascular disease (acute myocardial infarction, myocardial revascularisation, stroke or heart failure), depressive symptoms (measured by Clinical Interview Schedule-Revised) and body mass index were taken into account.

Data analysis

Descriptive statistics were used to describe study population features according to race/colour. Categorical variables and means were compared using the X^2 test and one-way analysis of variance, respectively. Age and sex-adjusted means of eGFR according to race/colour and racial discrimination were performed using linear regression.

Associations between racial discrimination and eGFR were investigated using linear regression models. After the crude analyses, we gradually added the following variables into the multivariable models: race/colour, age, sex and education (model 1); perceived racial discrimination (model 2); tobacco smoking, physical activity, excessive alcohol consumption, body mass index, hypertension, diabetes and self-reported cardiovascular disease (model 3). Income, type of occupation/job and depressive symptoms did not remain significantly associated with eGFR and were therefore not included in the models.

Whether associations between racial discrimination and eGFR would be modified by sex was tested by adding a bivariate interaction term to fully adjusted regression models. Given linear regression was used, statistically significant regression coefficients of the product term indicate interaction on an additive scale.[29] Finally, given the differences in prevalence of self-reported racial discrimination according to age among Black individuals in this study (figure 1), we tested if the association between racial discrimination and eGFR varied by age also including a bivariate interaction term in the fully adjusted regression models. As we found evidence for additive interaction (p-value: age*racial discrimination=0.008), therefore, analyses were stratified by age (<55 and ≥55 years). This cut-off for age was chosen because after 54 years the association between racial discrimination and eGFR changed the pattern as can be seen in the online supplementary figure 1.

Analyses were conducted using Stata V.14.0 (StataCorp, College Station, USA) and the level of significance set at 5%.

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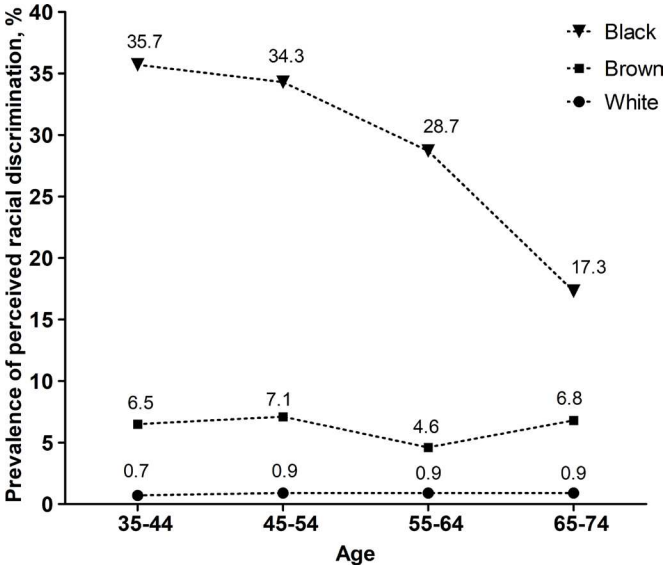

Figure 1 Prevalence (%) of lifetime racial discrimination among Black (n=2393), Brown (n=4191) and White (n=7771) individuals according to age. The Brazilian Longitudinal Study of Adult Health (2008–2010).

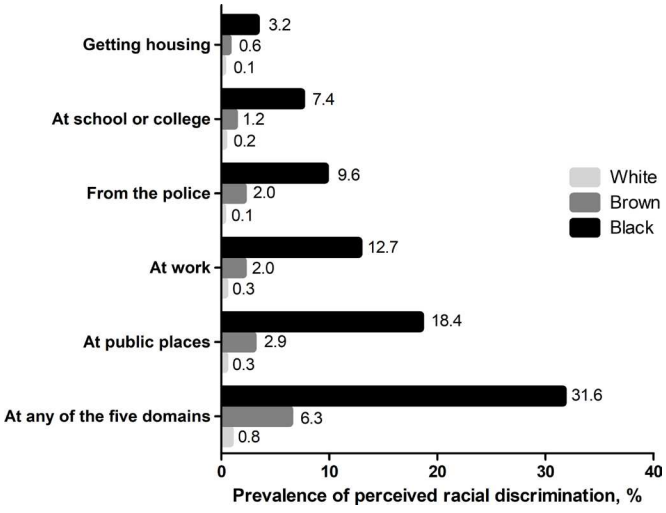

Figure 2 Prevalence (%) of lifetime racial discrimination in five different domains and all domains combined among Black (n=2393), Brown (n=4191) and White (n=7771) individuals. The Brazilian Longitudinal Study of Adult Health (2008–2010).

RESULTS

Black and Brown individuals had lower socioeconomic status (education, income and occupation) and higher prevalence of proximal risk factors for CKD compared with White participants (table 1).

Experience of racial discrimination was reported by 31.6% (n=755), 6.3% (n=264) and 0.8% (n=64) of Black, Brown and White individuals, respectively, mostly at public places (figure 2). Prevalence of racial discrimination decreased significantly with advancing age among Blacks, but not among Browns or Whites (figure 1).

Age and sex-adjusted mean eGFR varied according to race/colour. Blacks had the lowest adjusted mean eGFR, followed by Browns and Whites (figure 3). In addition, in spite of overlapping 95% CI, racial discrimination appeared to be associated with lower mean eGFR in Black, Brown and the 64 White individuals reporting such experiences (figure 3).

Black individuals aged <55 years had lower mean eGFR compared with White individuals in the same age group in the

Table 1 Descriptive characteristics of participants in the baseline of the Brazilian Longitudinal Study of Adult Health (ELSA-Brasil) according to race/colour, n=14 355 (2008–2010).

Variables	White n=7771	Brown n=4191	Black n=2393	P values*
Age, mean (SD)	52.5 (9.3)	51.2 (8.7)	51.8 (8.7)	<0.001
Sex, women (%)	53.8	51.7	60.8	<0.001
Education (%)				
University degree	66.8	40.2	27.2	<0.001
High school	26.0	42.7	50.9	
Complete elementary school	4.1	8.7	12.2	
Incomplete elementary school	3.1	8.4	9.7	
Monthly per capita household income in US$, mean (SD)	1123.0 (819.2)	704.3 (591.7)	580.4 (500.3)	<0.001
Occupation (%)				
Manual	11.4	24.0	28.7	<0.001
Not manual but routine work	23.4	33.2	40.1	
Not manual and not routine work	65.3	42.8	31.2	
Smoking (%)	12.3	13.6	14.5	<0.001
Physical inactivity (%)	73.8	79.1	82.6	<0.001
Excessive alcohol consumption (%)	7.1	8.1	7.9	0.08
Hypertension (%)	31.1	37.1	48.4	<0.001
Diabetes (%)	16.5	19.9	27.4	<0.001
Self-reported cardiovascular disease (%)	4.4	4.8	6.0	0.01
Body mass index, mean (SD)	26.7 (4.6)	27.0 (4.6)	28.0 (5.1)	<0.001
Depressive symptoms (%)	11.3	15.2	16.0	<0.001

*The p values were obtained using X^2 test (categorical variables) and one-way analysis of variance (ANOVA) (continuous variables).

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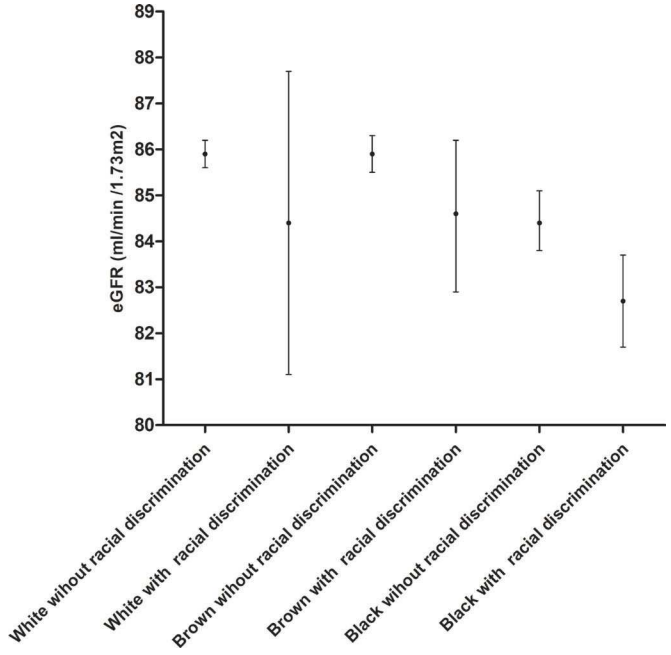

Figure 3 Age and sex-adjusted mean (95% CI) estimated glomerular filtration rates (eGFR) according to race/colour and racial discrimination. The Brazilian Longitudinal Study of Adult Health (2008–2010).

order of 2.55 mL/min/1.73 m^2 (model 1) even considering the effect of age, sex and education (table 2). Adjustment for the racial discrimination attenuated this association by 31% (model 2), and this proportion achieved 40% if the racial discrimination was considered together with other health-related factors (model 3). Brown colour was not associated with eGFR (table 2).

We also found that mean eGFR was also lower (eGFR differences of 2.38 mL/min/1.73 m^2; table 2) in all participants aged <55 years reporting experience of racial discrimination, regardless of all adjustments. This association was not modified by sex (p-value for additive interaction: women*racial discrimination=0.343).

After all adjustments, race/colour and racial discrimination were not associated with eGFR among participants aged ≥55 years (table 2).

DISCUSSION

In a large multiracial cohort of Brazilian civil servants, race/colour and racial discrimination were associated with lower mean eGFR in this study, regardless of sex, socioeconomic status or health-related factors. However, this association was limited to individuals aged under 55 years. The fact that eGFR differences between Black and White individuals in this age group were substantially reduced when racial discrimination was accounted for suggests exposure to racial discrimination plays a major role in racial disparities in renal function.

The magnitude of the association between racial discrimination and eGFR was not trivial because individuals who reported racial discrimination presented a lower mean of eGFR in the order of 2.38 mL/min/1.73 m^2. Mean difference in eGFR per year of age in ELSA-Brasil corresponded to 0.77 mL/min/1.73 m^2. It can therefore be argued that the impact of self-reported racial discrimination on eGFR is equivalent to approximately three additional years of life among Black individuals. However, this effect was limited to individuals aged under 55 years. The ELSA-Brasil study revealed that the proportion of Black individuals reporting racial discrimination decreased with advancing age (36% and 17% among individuals aged 35–44 and ≥65 years, respectively). These findings are consistent with previous Brazilian studies[18] and may reflect a cohort effect of cultural world views on racial discrimination in the Brazilian society over time. The recent darkening phenomenon in the Brazilian society is a most compelling evidence of this effect: 3 million Brazilians changed their racial identity from non-Black to Black between 2000 and 2010, that is, one in every three Black individuals

Table 2 Associations between race/colour, racial discrimination and estimated glomerular filtration rate (eGFR) in adult individuals participating in the baseline of the Brazilian Longitudinal Study of Adult Health (ELSA-Brasil) according to age (2008–2010).

Age		Model 1 β (95% CI)	Model 2 β (95% CI)	Model 3 β (95% CI)
<55 years	Race/colour			
	White	Ref	Ref	Ref
	Brown	−0.54 (−1.19 to −010)	−0.40 (−1.04 to 0.25)	−0.30 (−0.96 to 0.35)
	Black	**−2.55 (−3.35 to −1.74)*** **	**−1.77 (−2.67 to −0.88)*** **	**−1.54 (−2.44 to −0.63)** **
	Racial discrimination			
	No		Ref	Ref
	Yes		**−2.20 (−3.32 to −1.08)*** **	**−2.38 (−3.50 to −1.25)*** **
≥55 years	Race/colour			
	White	Ref	Ref	Ref
	Brown	0.56 (−0.34 to 1.46)	0.57 (−0.34 to 1.48)	0.85 (−0.05 to 1.76)
	Black	**−1.50 (−2.59 to 0.40)** **	**−1.46 (−2.64 to −0.28)* **	−0.87 (−2.06 to 0.31)
	Racial discrimination			
	No		Ref	Ref
	Yes		−0.14 (−1.78 to 1.48)	−0.00 (−1.63 to 1.63)

Regression coefficients β represent the difference in eGFR in mL/min/1.73 m^2.
Model 1: adjusted for age, sex and education.
Model 2: model 1+perceived racial discrimination.
Model 3: model 2+tobacco smoking, physical activity, excessive alcohol consumption, body mass index, hypertension, diabetes, self-reported cardiovascular disease.
Bold text indicates a statistically significant difference with a p-value <0.05.
*P<0.05; **P<0.01; ***P<0.001.

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counted in the 2010 census was a newly reclassified Black, particularly among youth and young adults.[30]

The historical lack of legal segregation after the end of slavery in Brazil and the 'racial democracy myth' have led to a unique scenario of 'prejudice against discrimination'.[22 31] Discrimination in Brazil became somewhat natural and was ostensibly denied and hidden. Although the 'racial democracy myth' still prevails in the Brazilian society, racial discrimination is now more widely acknowledged. These world view changes may have translated into a 'minimization bias' among older Black individuals. The 'minimization bias' occurs when individuals might see and report less racial discrimination than what actually exists.[32 33] Minimization bias may have interfered with results of this study in two ways: (1) the perception of being discriminated among older birth cohorts of Black individuals is lower, and therefore the stress response and its associated effect on eGFR might also be lower; (2) less accurate reporting of exposure to racial discrimination in older birth cohorts of Blacks introducing a null bias in associations between racial discrimination and renal function in older individuals.

The findings of this study suggest the experience of racial discrimination per se has an impact on eGFR. However, racial discrimination is mostly directed towards Black individuals (and to a lesser extent to Brown individuals), since personally experienced racial discrimination is far more common among Blacks than Whites. Therefore, if this association is truly causal, the population impact of exposure to racial discrimination (population attributable fraction) would be much greater among Blacks. Future longitudinal studies are warranted to test this hypothesis. In this cohort, racial discrimination explained nearly one-third of the associations between Black race/colour and eGFR. Hence, racial discrimination may in fact be a major contributing factor to racial inequities in renal function.

Several biological mechanisms may play a role in associations between racial discrimination and renal function. Racial discrimination may promote engagement in health risk behaviours for stress relief purposes,[34] which could increase the probability of declining eGFR. Moreover, the incidence and progression of CKD are related to chronological ageing[13 15] and racial discrimination has been related with increased oxidative stress[9] and with shortening of telomere length which is an emerging marker of biological age, and accelerated shortening is related to ageing-related diseases.[7 35] Racial discrimination has also been associated with forecast greater epigenetic ageing in adolescents living in less supportive family environments in two different African-American cohorts.[36] Experimental studies manipulating perceptions of discrimination (eg, via watching racist video clips, racially noxious image scenes, and so on) revealed associations with cardiovascular reactivity and psychological responses to stress (anger, self-esteem, depression, negative emotions, and so on).[34] According to the allostatic load theory, physiological changes in the nervous, endocrine and immune systems triggered by ongoing exposure to stress have proinflammatory effects.[37] In fact, associations between discrimination and higher levels or dysregulation of inflammatory markers such as cortisol,[8] C-reactive protein [10] and cytokines[6] have been reported and low-grade inflammation has been associated with higher CKD incidence and progression.[11 12]

In highly racialised societies such as Brazil, experiences of racial discrimination cannot be separated from those of race/colour. The combined effect of race/colour and discrimination on health outcomes justifies the need to test for additive interactions (synergism). Still, we chose not to investigate interactions between racial discrimination and race/colour in this study for two reasons: (1) small numbers of White individuals reporting perceived racial discrimination (37 individuals aged <55 years; 84% university degree holders) and (2) question aimed at investigating racial discrimination may capture different experiences in Whites compared with Black and Brown individuals. For example, according to Cunningham *et al*,[38] Whites may see affirmative action policies aimed at attenuating racial inequities as a form of institutional racism against Whites. In Brazil, slavery lasted longer than in any other American society and, despite impressive race/colour gaps on almost all measurable life circumstances indicators, affirmative action policy implementation dates from the beginning of 21st century and elicited appalling reactions (against racial quotas in universities, for example).[39]

This study has some potential limitations. First, the cross-sectional nature of the analysis precluded the establishment of temporal relations. However, it is unlikely that silent alterations in eGFR affect the report of racial discrimination or self-report of race/colour. Second, the role of unmeasured or unknown confounding variables must be considered, even though several potential confounders were accounted for. Finally, the sample in this study comprised university and research institute employees with stable jobs and high educational achievements for the most part, which is by no means a representative sample of the Brazilian population. Individuals with higher levels of education may be more aware of race-related discrimination.[17] It is however worth pointing out that the prevalence of racial discrimination that we found in ELSA-Brasil is lower than the one found in a population survey representative of the Brazilian population aged 16 years or over (6% of them with university degree) carried out in 2008 (White: 7.5%, Brown: 14.7%, Black: 41.1%).[18] The prevalence of racial discrimination may therefore have been underestimated in this study compared with the general population.

In conclusion, our findings extend prior research by suggesting that prevalence of racial discrimination is a problem among Blacks and Browns in a Brazilian cohort dominated by highly educated individuals. These findings are consistent with previous

What is already known on this subject

- Experience of racial discrimination has been implicated as a potential mechanism for higher incidence and faster progression of chronic kidney disease among Black individuals and other minorities.
- Scientific studies investigating associations between racial discrimination and renal function or the contribution of such discriminations to race/colour disparity in renal function are scarce.

What this study adds

- Racial discrimination was associated with lower mean estimated glomerular filtration rate (eGFR) in men and women aged under 55 years.
- Impacts of exposure to racial discrimination on renal function are far greater among Blacks, as the prevalence of racial discrimination is substantially higher in this group. Therefore, Black people are a true target of racial discrimination.
- Mean eGFR differences between Black and White individuals were reduced by 31% when exposure to racial discrimination was taken into account.

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social science studies reporting racialised social structures in Brazilian society. Racial discrimination was independently associated with lower eGFR in this study. The fact that this association was limited to individuals aged under 55 years suggests a minimization bias among older Black individuals. Further studies are warranted to investigate the complex associations between racial discrimination and renal function because as public health professionals, we have a duty to provide robust evidence to support imperative actions aimed at promoting social justice in health.

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Competing interests None declared.

Patient consent Not required.

Ethics approval ELSA-Brasil research protocol was approved by the Research Ethics Committee of Universidade de São Paulo (USP), Research Ethics Committee of Universidade Federal de Minas Gerais (UFMG), Research Ethics Committee of Fundação Oswaldo Cruz (FIOCRUZ), Research Ethics Committee of Universidade Federal do Espírito Santo (UFES), Research Ethics Committee of Universidade Federal da Bahia (UFBA), Research Ethics Committee of Universidade Federal do Rio Grande do Sul (UFRGS) and also by the National Research Ethics Committee (CONEP).

Provenance and peer review Not commissioned; externally peer reviewed.

Data sharing statement The data used in this study are available for research proposal on request to the ELSA's Datacenter and to the ELSA's Publications Committee (publiELSA). Additional information can be obtained from the ELSA's Datacenter (estatisticaelsa@ufrgs.br) and from the ELSA Coordinator from the Research Center of Minas Gerais (sbarreto@medicina.ufmg.br).

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