HHS Public Access

Author manuscript

Curr Opin Nephrol Hypertens. Author manuscript; available in PMC 2021 March 01.

Published in final edited form as:

Curr Opin Nephrol Hypertens. 2020 March ; 29(2): 227–231. doi:10.1097/MNH.0000000000000587.

Using race in the estimation of glomerular filtration rates: time for a reversal?

Heather Morris[1], **Sumit Mohan**[1,2,3]

[1]Department of Medicine, Division of Nephrology, Columbia University Medical Center, New York, NY

[2]Department of Epidemiology, Columbia University Mailman School of Public Health, New York, NY

[3]The Columbia University Renal Epidemiology (CURE) Group, New York, NY

Abstract

Purpose of Review—Bedside estimates of renal function are essential for clinical practice in the modern era and have largely relied on serum creatinine concentrations despite the known drawbacks associated with this choice of biomarker, including the fact that creatinine clearance overestimates the glomerular filtration rate.

Recent Findings—Initial estimates relied primarily on equations that incorporated factors known to influence creatinine concentrations such as age, gender and anthropometric measures. More recent estimates of glomerular filtration rate have replaced the anthropometric measures with the social construct of race, suggesting that glomerular filtration rates for black individuals are higher at the same concentration of creatinine. This approach has led to large variations in the estimated differences in glomerular filtration rate between black and non-black individuals in the United States that have not been reproducible, resulting in a plethora of population specific formulae across the country.

Summary—The introduction of race in estimated glomerular filtration rate (eGFR) equations may have potential unintended negative consequences for the very population with the greatest burden of kidney disease. These potential disadvantages underscore the need to perhaps return to the replacement of race with more objective anthropometric measures without the loss of precision.

Keywords

Race; black; glomerular filtration rate; kidney function; anthropometry; disparities

Corresponding Author: Sumit Mohan, MD, MPH, Division of Nephrology, Department of Medicine, 622 W 168th St PH4-124, New York, NY, 10032, 212 305 4401, sm2206@cumc.columbia.edu.

Conflicts of Interest

SM is a member of the scientific advisory board for Angion Pharmaceuticals and Deputy Editor of Kidney International Reports. HM has no conflicts of interest.

Introduction

The estimation of the glomerular filtration rate (eGFR) quickly at the bedside has been a fundamental need for clinicians for both the identification of renal disease and the appropriate dosing of medications that are excreted by the kidneys. Recent eGFR equations developed in North American cohorts have utilized black race as a modifying variable. Here we review the historical basis and potential pitfalls of this practice.

Development of Creatinine Clearance Estimation Equations

Early suggestions of the use of endogenous creatinine clearance measurements as being superior to urea date back to 1950's, with Effersoe demonstrating the utility of the relationship of a measured 24-hour creatinine clearance and serum creatinine concentration.[1] The utility of a simple bedside creatinine clearance estimate as a measure of renal function in the absence of a urine collection was suggested by Jelliffe as far back as 1971.[2] Jelliffe's contribution was the development, using hypothetical data, of simplified equations which used reciprocal of the serum creatinine, where creatinine clearance in ml/min was estimated as [(100/SCr) −12] for men and [(80/Cr) – 7] for women.[2] In some ways, his formulae continue to be used in the simplified approach of the reciprocal of creatinine, i.e. 100/creatinine. Although the proposed formulae from Jelliffe underscored the differences between men and women, it failed to account for other factors that would influence endogenous production of creatinine including age and weight. Siesbaek-Nielsen et al, in their response to Jelliffe, included a proposed normogram that accounted for age and gender.[3] Cockcroft and Gault also noted their concern with the absence of age and associated declining excretion of creatinine in the estimation in a letter in 1975 that predated their seminal paper in 1976.[4, 5]

The original analysis from Cockcroft and Gault was limited to a study of 249 white male patients that were derived from a cohort of 534 patients with 2 creatinine clearance measurements at the Queen Mary Veterans Hospital – and notably excluded anyone with a creatinine excretion rate of < 10mg/Kg.[5] They noted that a 24 hour creatinine excretion could be estimated at [28 –(0.2*age)]*wt (in Kg) which could then be transformed to estimate the creatinine clearance in mL/min to the Cockcroft-Gault equation that we all now know.[5] However, their analysis also made 2 important observations: 1)changes in muscle mass influenced the estimate; and 2) it was not unreasonable to assume that women may have lower creatinine concentrations, but acknowledged their inability to study this directly given their study cohort.[5] Effersoe's cohort of patients in Copenhagen included both men and women, and the observed differences in creatinine concentrations for the same clearance per 1.73m^2 body surface area had already been attributed to differences in muscle mass.[1] As a result, in a rather crude approximation, Cockcroft and Gault suggested a 15% reduction for women as a midpoint of prior recommendations of 10–20% from prior studies – and hence the 0.85 multiplier in the Cockcroft-Gault equation for women.[5] It should also be noted that the mean difference between the measured and estimated creatinine clearance was 20.9 mL/min and that 95% of estimates were within 35% of the measured value – a degree of precision that has not really been improved on much in the intervening half century with the newer equations. The authors also acknowledged the need for special considerations for

patients with low muscle mass and liver disease as well as the impact of diet, but notably made no mention of race - perhaps a reflection of the era and their patient population. Despite the many limitations of the original study as described in detail by others, this became the most widely used estimate of renal function and continues to be used in clinical practice even today.[5, 6]

Glomerular Filtration Rate Estimation Equations: The Introduction of Black Race as a Modifying Variable

While multiple other efforts have been made to improve on the bedside estimate of renal function, the most notable next step in the wide adoption of estimates came with the introduction of the Modification of Diet in Renal Disease (MDRD) formula in 1999, which leveraged the precise measurements of GFR performed as part of the MDRD study.[7] Levey et al used a stepwise regression approach using data from 1628 individuals to develop an equation to estimate GFR rather than the creatinine clearance, which overestimates GFR.[7] In this cohort that was 80% white, black men and women, who made up only 12% of the cohort, had significantly higher creatinine excretion rates than their white counterparts. (Of note, the study cohort included 87 Hispanics, 17 Asian Americans and 23 others, all of whom are referred to as “white” in the text).[7, 8] Superficially, this finding was consistent with a report in the preceding year from NHANES that also suggested that non-Hispanic black individuals had higher serum creatinine concentrations, but given the absence of any objective data on the glomerular filtration rate and known differences in the prevalence of CKD, the value of this finding was at best unclear and potentially meaningless.[9] The higher serum creatinine concentrations at the same GFR for the black patients in the MDRD cohort meant that, in addition to age and sex, black race was also an independent predictor of the GFR. It should be noted, however, the 95% confidence intervals for the difference between the measured and the estimated GFR using the MDRD formula ranged from approximately −20 to +30mL/min for at a measured GFR of 60mL/min – a margin of error not unlike that seen with the Cockcroft Gault estimate of creatinine clearance.[10]

Around the time of the development of the MDRD equation were efforts from the African American Study of Kidney Disease (AASK). In the AASK pilot study, the creatinine index (i.e. the creatinine excretion rate in mg/Kg/day) demonstrated a wide range of 8.6 – 46 for men and 3.3 – 30.8 for women across all ages – approximating the original creatinine excretion rates reported by Cockcroft and Gault in a white cohort.[11] Subsequent to the release of the MDRD equation, an eGFR formula was developed specifically for African Americans using the full AASK cohort. While having different coefficients, it performed similarly to the MDRD formula and may have contributed to the notion that we would need specific formulae tailored to specific populations.[8] Also notable was that the Cockcroft Gault equation for creatinine clearance *underestimated* the GFR in the AASK cohort of patients.[8]

Black patients have a multiplicative factor of 1.18 in the MDRD formula resulting in 18% increased eGFR at a given creatinine than non-black patients. The simpler 4 variable MDRD formula was subsequently developed and reported which included 21.2% increase in eGFR

for blacks.[12] A decade after the introduction of the MDRD came the development of the CKD-EPI equation from a pooled cohort of 8254 participants.[10] In addition to resulting in an equation with better (though suboptimal) precision and lower bias, the development cohort had a sizable (32%) proportion of blacks, and resulted in a smaller increase (15.9%) in the eGFR for blacks compared to non-blacks.[10] Further refinements in the estimating questions led to the inclusion of cystatin C, a biomarker whose concentration is independent of diet and muscle mass.[13] Notably, equations that excluded race and used only cystatin C did not provide a more or less accurate measure of renal function and equations that used both creatinine and cystatin C suggested only an 8% higher eGFR for blacks compared to non-blacks with the same serum creatinine concentration.[13] The variations in the relative adjustment provided for blacks in these related formulae is perhaps an early sign that race, which is a social rather than a biological construct, may not belong in our estimation equations. We should also note that the mere introduction of another marker of renal function that is not based on muscle mass may not provide necessary clarity, as evidenced by the dramatic discordance that has been reported between Cystatin C-based and creatinine-based estimates of GFR in certain populations.[14, 15]

While these eGFR equations would appear to represent a forward march in the ability to determine the renal function at the bedside without a urine collection with reasonable accuracy, the relatively recent introduction of race/ethnicity in the equations developed in North America has resulted in researchers in other parts of the world feeling the need to do the same for their patients. As a result, there are now separate equations for individuals of Chinese, Japanese, and South Asian descent.[16–20] Studies from the African continent including locations as disparate as Ghana and South Africa suggest that GFR estimates are in fact more accurate without the race/ethnicity adjustment used for blacks derived using cohorts in the United States, although this has not been a consistent finding.[21–24] Moreover, these equations do not appear to be adequate for individuals with non-African ancestry living in these countries.[25] This is similarly true for African Europeans, where the CKD-EPI formula resulted in an overestimation of renal function when using the adjustment for blacks. [26]

Race-based eGFR Reporting: Perils and Pitfalls

The MDRD formula remains the most widely reported GFR estimate in the United States – a function of the important, widespread and successful efforts spearheaded by the National Kidney Disease Education Program (NKDEP) to improve the recognition of kidney disease and facilitate an understanding that the same creatinine concentration in different individuals can imply very different levels of kidney function. As a result, by 2017, 86% of laboratories were reporting an eGFR with every creatinine [27] but in 2013 only 54% were using IDMS-traceable creatinine calibration – with a surprising 34% of labs using the inappropriate formula.[28] Despite the limited data to which laboratories have access, or perhaps because of it, the NKDEP recommends eGFR reporting accompany every creatinine measurement. As a result, laboratories report the eGFR without evidence of whether it is appropriate to do so, i.e, is there a steady state creatinine or not or if there is a clinical circumstance that makes the eGFR less reliable. Laboratories do not have access to the patients self-reported race either, which forces them to report the eGFR for both black and non-black patients together

and allowing clinicians to make the determination of which result is appropriate. This approach has led to the introduction of the social construct of race in a laboratory report while simultaneously doing away with more objective anthropometric measures that are easier for patients and providers in the estimating equations. This choice is particularly perplexing for patients who are often surprised if not confused by the sharp dichotomization of race. There is also the conundrum faced by individuals who are left out of this binary categorization when they encounter their laboratory test results.

The potential negative implications of reporting higher eGFR for all black patients in a vacuum of additional information needed to inform the estimate is immediately obvious in certain situations. Clinicians are also confronted with the challenge of attempting to understand how to interpret the estimates for individuals who are black but perhaps not African Americans given data from other parts of the world. While one could argue that at the individual patient level what matters most over time is the progression of disease and that the slope of decline in renal function would be the same regardless of which category one picked, that is not particularly reassuring to patients, especially when clinical decisions are often based on eGFR thresholds. Important clinical decisions including medication dosing, screening for metabolic complications of CKD, timing of vascular access placement, pre-emptive listing for and access to transplantation, acceptance as potential living donors or even initiation of renal replacement therapy are predicated on achieving eGFR thresholds.[29] Higher GFR estimates may result in a delay in these events occurring. For example, the increased prevalence of metabolic complications at higher eGFR among blacks may in fact be the result of inappropriate GFR inflation.[30] In addition, the resulting higher eGFR may potentially underestimate the true prevalence of chronic kidney disease among black individuals in the United States and to a greater extent, in other parts of the world, resulting in inadequate attention and resources provided to address this public health challenge.[31–33] Given the evidence that racial differences in creatinine production are related to objective anthropometric measures such as height, weight and lean body mass this particular choice to invoke race in reports of kidney function seem odd.[23]

Variations in dietary composition are often cultural but also influenced by socioeconomic status which make these likely confounders of direct comparisons of serum creatinine concentrations across racial groups. Families with low socioeconomic status In the United States tend to consume diets poor in fruits and vegetables but rich in prepared foods that include large amounts of phosphorus and animal protein.[34] In contrast, access to animal protein is considerably diminished for those living in poverty in the rest of the developing world and the resultant lower serum creatinine concentrations likely contributes to the differences in eGFR formula performance, even among cohorts that might share ancestry, especially when direct anthropometric measures are replaced with surrogates such as race.

It is important to remember that the United States is among a small minority of countries that collects racial classification data in the census which may reflect why innate racial differences are invoked more often even in the presence of evidence to the contrary in medicine.[35] The inclusion of race, in estimation formulae raises complex questions around identity (cultural and/or biological) and ancestry. Thresholds and criteria that are developed using these measures can be difficult to eliminate once entrenched.[36, 37] With the recent

discovery of the APOL1 genetic variants and their strong association with sub-Saharan African ancestry, difficult questions about identifying individuals at risk are being asked in nephrology as the limits of self-identification of race are becoming increasingly evident.[38] In the case of a genetic variant like APOL1, it's clear that the primary determinant of the likelihood of having 2 risk variants (but not risk of overt disease) is one of biological ancestry, as difficult as that might be to ascertain.

Thus the value of "self-reported race" is being called into question in circumstances where what is actually being sought is an individual genetic ancestry rather than how one might identify culturally. However, those limitations also likely apply in a situation such as estimation of GFR where race is a surrogate for body composition, dietary choices, cultural preferences, socioeconomic status and perhaps genetics - all intertwined in a manner that teasing them apart is likely impossible. The use of the social construct of race is even more problematic when one considers that the race recorded in the medical record is often not that reported by the patient but rather inferred, often erroneously, by the medical staff or the provider using visual cues.[39, 40]

Conclusion

Given the value of having a bedside tool to estimate renal function, abandoning estimation formulae is both unrealistic and undesirable. Instead, a return to formulae that reflect known biological factors that impact creatinine production rather than regression formulae that include social constructs that may be poor surrogate measures of factors that directly influence creatinine production such as diet and body composition appears to be the way forward.[41] In fact, analyses have already demonstrated the ability of existing equations to perform reasonably well without accounting for race.[42] Estimating equations that include urea nitrogen or even phosphorus as a marker of dietary protein intake (and thus indirectly creatinine) as well as anthropometric measurements (height and weight being the simplest and most widely available), with the removal of race will probably result in similar precision and bias as existing formulae. Given the ubiquity of creatinine assays and the growing standardization globally, our patients deserve estimates of renal function that are easily accessible, accurate, reproducible that rely on our understanding of the underlying biology while also being free of potentially harmful social constructs.

Acknowledgements

SM is supported by National Institute of Diabetes and Digestive and Kidney Diseases (R01 DK114893 and U01 116066) and by the National Institute of Minority Health and Disparities (R01 MD014161)

Financial Support and Sponsorship

None.

References

1. Effersoe P Relationship between endogenous 24-hour creatinine clearance and serum creatinine concentration in patients with chronic renal disease. Acta Med Scand. 1957;156(6): 429–434. [PubMed: 13402411]

2. Jelliffe RW, Jelliffe SM. Estimation of creatinine clearance from changing serum-creatinine levels. Lancet (London, England). 1971;2(7726): 710–710.

3. Siersbaek-Nielsen K, Hansen JM, Kampmann J, Kristensen M. Rapid evaluation of creatinine clearance. Lancet. 1971;1(7709): 1133–1134.

4. Gault M, Cockcroft D. Creatinine clearance and age. Lancet (London, England). 1975;2(7935): 612–613.

5. Cockcroft DW, Gault MH. Prediction of Creatinine Clearance from Serum Creatinine°. Nephron. 1976;16: 31–41. [PubMed: 1244564]

6. Perrone RD, Madias NE, Levey AS. Serum creatinine as an index of renal function: new insights into old concepts. Clin Chem. 1992;38(10): 1933–1953. [PubMed: 1394976]

7. Levey AS, Bosch JP, Lewis JB, et al. A More Accurate Method To Estimate Glomerular Filtration Rate from Serum Creatinine: A New Prediction Equation. Annals of Internal Medicine. 1999;130(6): 461–470. [PubMed: 10075613]

8. Lewis J, Agodoa L, Cheek D, et al. Comparison of cross-sectional renal function measurements in African Americans with hypertensive nephrosclerosis and of primary formulas to estimate glomerular filtration rate. Am J Kidney Dis. 2001;38(4): 744–753. [PubMed: 11576877]

9. Jones CA, McQuillan GM, Kusek JW, et al. Serum creatinine levels in the US population: third National Health and Nutrition Examination Survey. Am J Transplant. 1998;32(6): 992–999.

10. Levey AS, Stevens LA, Schmid CH, et al. A new equation to estimate glomerular filtration rate. Ann Intern Med. 2009;150(9): 604–612. [PubMed: 19414839]

11. Toto RD, Kirk KA, Coresh J, et al. Evaluation of serum creatinine for estimating glomerular filtration rate in African Americans with hypertensive nephrosclerosis: results from the African-American Study of Kidney Disease and Hypertension (AASK) Pilot Study. Journal of the American Society of Nephrology : JASN. 1997;8(2): 279–287. [PubMed: 9048347]

12. Stevens LA, Coresh J, Greene T, Levey AS. Assessing Kidney Function — Measured and Estimated Glomerular Filtration Rate. New England Journal of Medicine. 2006;354(23): 2473–2483. [PubMed: 16760447]

13. Inker LA, Schmid CH, Tighiouart H, et al. Estimating glomerular filtration rate from serum creatinine and cystatin C. New England Journal of Medicine. 2012;367(1): 20–29. [PubMed: 22762315]

14. Husain SA, Willey JZ, Park Moon Y, et al. Creatinine- versus cystatin C-based renal function assessment in the Northern Manhattan Study. PloS one. 2018;13(11): e0206839–e0206839. [PubMed: 30427947] * Demonstrates the dramatic variation in the prevalence of CKD using creatinine and cystatin based estimates of GFR when applied to older patient populations.

15. Peralta CA, Lee A, Odden MC, et al. Association between chronic kidney disease detected using creatinine and cystatin C and death and cardiovascular events in elderly Mexican Americans: the Sacramento Area Latino Study on Aging. J Am Geriatr Soc. 2013;61(1): 90–95. [PubMed: 23252993]

16. Ma Y-C, Zuo L, Chen J-H, et al. Modified glomerular filtration rate estimating equation for Chinese patients with chronic kidney disease. Journal of the American Society of Nephrology. 2006;17(10): 2937–2944. [PubMed: 16988059]

17. Matsuo S, Imai E, Horio M, et al. Revised equations for estimated GFR from serum creatinine in Japan. Am J Kidney Dis. 2009;53(6): 982–992. [PubMed: 19339088]

18. Jessani S, Levey AS, Bux R, et al. Estimation of GFR in South Asians: a study from the general population in Pakistan. Am J Kidney Dis. 2014;63(1): 49–58. [PubMed: 24074822]

19. Mulay A, Gokhale S. Comparison of serum creatinine-based estimating equations with gates protocol for predicting glomerular filtration rate in Indian population. Indian journal of nephrology. 2017;27(2): 124. [PubMed: 28356664]

20. Kumar V, Yadav AK, Yasuda Y, et al. Existing creatinine-based equations overestimate glomerular filtration rate in Indians. BMC nephrology. 2018;19(1): 22. [PubMed: 29390980]

21. van Deventer HE, George JA, Paiker JE, Becker PJ, Katz IJ. Estimating glomerular filtration rate in black South Africans by use of the modification of diet in renal disease and Cockcroft-Gault equations. Clinical chemistry. 2008;54(7): 1197–1202. [PubMed: 18487286]

22. Eastwood JB, Kerry SM, Plange-Rhule J, et al. Assessment of GFR by four methods in adults in Ashanti, Ghana: the need for an eGFR equation for lean African populations. Nephrology Dialysis Transplantation. 2010;25(7): 2178–2187.
23. Glaser N, Deckert A, Phiri S, Rothenbacher D, Neuhann F. Comparison of various equations for estimating GFR in Malawi: how to determine renal function in resource limited settings? PLoS One. 2015;10(6): e0130453. [PubMed: 26083345]
24. Matsha TE, Yako YY, Rensburg MA, Hassan MS, Kengne AP, Erasmus RT. Chronic kidney diseases in mixed ancestry south African populations: prevalence, determinants and concordance between kidney function estimators. BMC Nephrol. 2013;14: 75. [PubMed: 23547953]
25. Adeniyi AB, Laurence CE, Volmink JA, Davids MR. Prevalence of chronic kidney disease and association with cardiovascular risk factors among teachers in Cape Town, South Africa. Clinical kidney journal. 2017;10(3): 363–369. [PubMed: 28621342]
26. Flamant M, Vidal-Petiot E, Metzger M, et al. Performance of GFR Estimating Equations in African Europeans: Basis for a Lower Race-Ethnicity Factor Than in African Americans. American Journal of Kidney Diseases. 2013;62(1): 182–184.
27. Laboratories Reporting eGFR with creatinine results by year. Vol 2019 Chronic Kidney Disease Surveillance System.
28. Miller G. Practices and recommendations for reporting estimated glomerular filtration rate (eGFR). Vol 2019.
29. Ibrahim HN, Wang C, Ishani A, Collins AJ, Foley RN. Screening for chronic kidney disease complications in US adults: racial implications of a single GFR threshold. Clinical Journal of the American Society of Nephrology. 2008;3(6): 1792–1799. [PubMed: 18784208]
30. Foley RN, Wang C, Ishani A, Collins AJ. NHANES III: Influence of Race on GFR Thresholds and Detection of Metabolic Abnormalities. Journal of the American Society of Nephrology. 2007;18(9): 2575. [PubMed: 17656476]
31. Delanaye P, Mariat C, Maillard N, Krzesinski J-M, Cavalier E. Are the Creatinine-Based Equations Accurate to Estimate Glomerular Filtration Rate in African American Populations? Clinical Journal of the American Society of Nephrology. 2011;6(4): 906. [PubMed: 21441133]
32. King KL, Husain SA, Jin Z, Brennan C, Mohan S. Trends in Disparities in Preemptive Kidney Transplantation in the United States. Clinical journal of the American Society of Nephrology : CJASN. 2019;14(10): 1500–1511. [PubMed: 31413065]
33. Schold JD, Navaneethan SD, Jolly SE, et al. Implications of the CKD-EPI GFR Estimation Equation in Clinical Practice. Clinical Journal of the American Society of Nephrology. 2011;6(3): 497. [PubMed: 21115622]
34. Gutiérrez OM, Anderson C, Isakova T, et al. Low socioeconomic status associates with higher serum phosphate irrespective of race. Journal of the American Society of Nephrology. 2010;21(11): 1953–1960. [PubMed: 20847142]
35. Braun L Theorizing race and racism: preliminary reflections on the medical curriculum. American journal of law & medicine. 2017;43(2–3): 239–256. [PubMed: 29254464]
36. Braun L. Spirometry, measurement, and race in the nineteenth century. J Hist Med Allied Sci. 2005;60(2): 135–169. [PubMed: 15737956]
37. Braun L. Race, ethnicity and lung function: A brief history. Can J Respir Ther. 2015;51(4): 99–101. [PubMed: 26566381]
38. Neugut YD, Mohan S, Gharavi AG, Kiryluk K. Cases in Precision Medicine: APOL1 and Genetic Testing in the Evaluation of Chronic Kidney Disease and Potential Transplant. Annals of internal medicine. 2019: 10.7326/M7319-1389.
39. Mohan S, Iltis AS, Sawinski D, DuBois JM. APOL1 Genetic Testing in Living Kidney Transplant Donors. American journal of kidney diseases : the official journal of the National Kidney Foundation. 2019;74(4): 538–543. [PubMed: 30982552] * Discusses the challenges of using race as a surrogate for genetic ancestry and its potential implications for nephrology in the context of APOL1 testing
40. Polubriaginof FCG, Ryan P, Salmasian H, et al. Challenges with quality of race and ethnicity data in observational databases. J Am Med Inform Assoc. 2019;26(8–9): 730–736. [PubMed:

31365089] * Describes the limitations of race and ethnicity data that are currently available in observational datasets.

41. Rule AD, Bailey KR, Schwartz GL, Khosla S, Lieske JC, Melton LJ III. For estimating creatinine clearance measuring muscle mass gives better results than those based on demographics. Kidney international. 2009;75(10): 1071–1078. [PubMed: 19177154]
42. Zanocco JA, Nishida SK, Passos MT, et al. Race adjustment for estimating glomerular filtration rate is not always necessary. Nephron extra. 2012;2(1): 293–302. [PubMed: 23243414]

Key Points

1. The accurate estimation of renal function is a necessary cornerstone of clinical medicine in the modern era.
2. Race is a social construct and likely a poor surrogate for known factors that influence the production of creatinine
3. Similarly precise estimates of glomerular filtration rate are possible with the elimination of race and reintroduction of simple objective anthropometric measures