This was proposed in the report of the original Modification of Diet in Renal Disease equation to achieve accurate drug dosing.4

Another possible problem is that a Modification of Diet in Renal Disease or CKD-EPI eGFR is an estimate of a size-indexed GFR, not a measured value. A patient with a CKD-EPI eGFR of 70 ml/min per 1.73 m² could well have a measured, size-indexed GFR of 60 or 80 ml/min per 1.73 m², or—less likely—55 or 85 ml/min per 1.73 m², etc.2,4 This introduces unappreciated heterogeneity into a small, carefully assembled database. In other words, the study assessed the relationship of individually determined patient characteristics with Pglom, but used estimates for glomerular filtration. “Unindexing” the eGFRs would still leave estimates, not measured values.

DISCLOSURES

The author has nothing to disclose.

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REFERENCES


Authors’ Reply

Steiner1 poses an interesting question related to the interpretation of eGFR. In our study, eGFR, on the basis of the Chronic Kidney Disease Epidemiology Collaboration formula, was primarily used to exclude patients at increased risk for contrast nephropathy. The main goal of the regression analysis, as reported in our article, was to determine whether the estimated glomerular pressure (Pglom) had correlations consistent with current knowledge about glomerular hyperfiltration.2 As described in the supplemental material, we found that an increased estimated Pglom was associated with the prevalence of diabetes, renal perfusion pressure, and body mass index, but not with eGFR. We agree with Steiner that use of eGFR may complicate the interpretation of our results for multiple reasons, including the notion that eGFR is indexed for body surface area (BSA).

Indexation of GFR to the individual patient’s body size is appropriate when using estimates based on serum creatinine in the identification of patients with CKD.

We agree with Steiner that, in case of obesity, indexation of GFR for BSA can conceal obesity-related hyperfiltration.3 However, (e)GFR and other surrogates used for Pglom are affected by other factors, such as kidney mass and the loss of functional nephrons. This makes it cumbersome to identify patients with hyperfiltration on the basis of (e)GFR.2

We reanalyzed our data using an eGFR unindexed for BSA; this did not change our findings. In our population, we did find a large spread in the renal fractional flow reserve and estimated Pglom; suggesting there are meaningful intraindividual differences between renal function in patients with normal eGFR.4 To acquire a better understanding in the fundamental relation between GFR, filtration fraction, and Pglom, we plan to perform iodine-125–iothalamate and iodine-131–hippuran clearance measurements in combination with intrarenal hemodynamic measurements in patients undergoing percutaneous renal revascularization.

DISCLOSURES

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Robert W. Steiner
University of California San Diego Center for Transplantation and Division of Nephrology, University of California at San Diego School of Medicine, San Diego, California

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Correspondence: Dr. Bert-Jan H. van den Born, Department of Vascular Medicine, Amsterdam University Medical Centers, University of Amsterdam, PO Box 22660, 1100 DD, Amsterdam, The Netherlands. Email: b.j.vandenborn@amsterdamumc.nl
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Didier Collard,1 Lennart van de Velde,1,2 Liffert Vogt,3 and Bert-Jan H. van den Born1
1Department of Vascular Medicine, Amsterdam University Medical Centres, University of Amsterdam, Amsterdam, The Netherlands
2Multi-Modality Medical Imaging Group, Technical Medical Centre, University of Twente, Enschede, The Netherlands
3Department of Nephrology, Amsterdam University Medical Centres, University of Amsterdam, Amsterdam, The Netherlands
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