Comparison of Predictive Performance of Renal Function Estimation Equations for All-Cause and Cardiovascular Mortality in an Elderly Hypertensive Population

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BACKGROUND
The Modifications of Diet in Renal Disease (MDRD) and Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) are 2 equations commonly used to estimate glomerular filtration rate (eGFR). The predictive performance offered by these equations, particularly in relation to clinical outcomes in elderly hypertensive patients, is not clear.

METHODS
The Second Australian National Blood Pressure Study cohort was used to investigate the predictive performance of these 2 equations for long-term outcomes (median 10.8 years) in elderly treated hypertensive patients. Both equations were used to calculate eGFR in 6,083 patients aged ≥65 years and classified as having chronic kidney disease (CKD) or no CKD (eGFR ≥60 ml/min/1.73 m²).

RESULTS
More patients were classified as having no CKD using the CKD-EPI equation compared with the MDRD equation (72.1% vs. 69.4%; P = 0.001). Both equations performed similarly in risk prediction of all-cause and cardiovascular mortality with decreased eGFR, except for patients with baseline eGFR of 45–59 ml/min/1.73 m², where the CKD-EPI equation predicted higher risk of all-cause mortality compared with those with no CKD. However, the magnitude of difference in risk prediction was too small to be clinically meaningful. Both equations showed similar predictive performance. However, we observed longer survival and no higher risk in those who were reclassified as having no CKD using the CKD-EPI equation, but these patients were classified earlier as having CKD using the MDRD equation.

CONCLUSIONS
There was no clinically relevant difference in predictive performance for long-term survival by eGFR calculated using either of these equations in this elderly hypertensive population.

Keywords: blood pressure; chronic kidney disease; elderly; hypertension; glomerular filtration rate; mortality.

doi:10.1093/ajh/hpu160

Hypertension is a key reason for end-organ damage including chronic kidney disease (CKD) among the elderly.1,2 CKD is clinically characterized by declining glomerular filtration rate (GFR) and increasing albuminuria, usually leading to end-stage renal disease.3 CKD is a global public health concern, not only due to the risk of long-term decline in renal function but also because of the strong association with an increased risk of all-cause and cardiovascular morbidity and mortality.4,5 Early detection of CKD enables the initiation of treatment that may slow the deterioration of renal function and thus its effect on mortality.3 Therefore, use of equations to more easily estimates GFR from serum creatinine measures has been recommended in order to improve the recognition and subsequent treatment of CKD.6,7

Numerous equations have been developed to estimate GFR (eGFR) using readily available indices such as serum creatinine concentration, age, gender and race, and also body weight.8–12 Current evidence suggests that eGFR, similar to measured GFR, is a predictor of all-cause and cardiovascular mortality.13,14 The most widely recommended equation is the Modification of Diet in Renal Disease (MDRD) formula.8,11 More recently, an alternative formula, the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation, has been published.10 The CKD-EPI equation was developed
in an effort to create a formula that is more accurate than the MDRD formula, especially when actual GFR is >60 ml/min/1.73 m², and is considered more accurate in risk prediction across a broad spectrum of population cohorts, particularly those outside the population context from which the MDRD equation was first developed.¹⁵

While there is good evidence to support the notion that the CKD-EPI equation is more accurate and reliable in the measurement of GFR and in clinical risk prediction,¹⁶⁻¹⁸ the notion is challenged in other epidemiological studies of specific patient cohorts.¹⁹,²⁰ There is also existing controversy relating to the use of the CKD-EPI equation in the elderly,²¹,²² with concerns of overestimation of CKD. Of concern is the rising prevalence of hypertension with increasing age and the respective effects of age and hypertension on clinical outcomes, both separately and combined.²³,²⁴ There is a clear need to clarify the predictive performance of eGFR offered by these 2 equations, particularly in relation to clinical outcomes in elderly hypertensive people.

Therefore, we conducted a post hoc analysis of eGFR and long-term all-cause and cardiovascular mortality in a large elderly hypertensive population (aged ≥65 years) who participated in the Second Australian National Blood Pressure (ANBP2) study, all treated with antihypertensive medications. This post hoc analysis was designed to compare the predictive performance of the MDRD and CKD-EPI equations in a hypertensive elderly population.

**METHODS**

**Study design and participants**

The ANBP2 study was a prospective randomized open-label blinded endpoint outcome study. In this study, 6,083 hypertensive patients aged 65--84 years were enrolled from family practices across Australia and were randomly allocated to be placed on either an angiotensin-converting enzyme inhibitor--based (n = 3,044) or a diuretic--based (n = 3,039) blood pressure--lowering drug regimen. Details of the study design, methods, and main findings have been published previously.²⁵ Written informed consent was obtained from all ANBP2 participants. The study protocol was approved by the Ethics Committee of the Royal Australian College of General Practitioners and conducted in accordance with the Declaration of Helsinki.

**Measurement and estimation of GFR**

Serum creatinine concentrations were measured on all participants at randomization into the study (baseline). These measures were not taken in a central laboratory but were performed and reported by the commercial laboratory used by the family practice involved. eGFR at baseline was estimated from these serum creatinine measures for all patients using both the MDRD study equation⁸ and the CKD–EPI equation¹⁹ (Supplementary Box 1). As this study was undertaken by a number of laboratories in the pre-creatinine assay standardization era and was not standardized to isotope dilution mass spectrometry (IDMS) values, serum creatinine values were standardized for use in the CKD-EPI equation, reducing them by 5% using the calibration process of the MDRD study laboratory.⁹

Based on the eGFR, patients were categorized as follows: no CKD, eGFR ≥60 ml/min/1.73 m² and CKD, eGFR <60 ml/min/1.73 m². The label of “no CKD” was applied to all participants with an eGFR ≥60 ml/min/1.73 m² as there was no urinary or other structural or pathological information on the study participants to identify those with CKD stage 1 or CKD stage 2. The CKD cohort was further categorized to stage 3A (eGFR 45--59 ml/min/1.73 m²), stage 3B (eGFR 30--44 ml/min/1.73 m²), stage 4 (eGFR 29--15 ml/min/1.73 m²), and stage 5 (eGFR <15 ml/min/1.73 m²), according to the current internationally agreed-to diagnostic framework.³

To keep the categorization comparable to that of the MDRD equation group, patients were further recategorized based on having CKD or no CKD using the CKD-EPI equation. Recategorization included the following subgroups: no CKD, control, patients classified as no CKD using both MDRD and CKD-EPI equations; no CKD, reclassified, patients classified as CKD using MDRD then as no CKD using CKD-EPI; CKD, reclassified, patients classified as no CKD using MDRD then classified as CKD using CKD-EPI; and CKD, both, patients classified using both equations as CKD. This recategorization was done to assess the risk association between those who were reclassified using CKD-EPI with long-term outcome.

**Follow-up and endpoint ascertainment**

Clinical and demographic information was collected for each participant at the time of enrollment into the ANBP2 study. Clinical information included height, weight, body mass index (BMI), blood pressure (systolic/diastolic), serum creatinine concentration, plasma cholesterol (total cholesterol and high-density lipoprotein) concentration, and previous medical condition (presence of diabetes, history of cardiovascular disease, coronary heart disease, or antihypertensive treatment). In the current investigation, we defined the following 2 endpoints: any cause mortality and any cardiovascular mortality. Long-term survival (endpoint) in the ANBP2 study cohort (n = 6,083) was observed over a median of 10.8 years (interquartile range, 9.6--11.4). The endpoints were determined during the ANBP2 clinical trial period (median 4.1 years) by an endpoint committee blinded to drug treatment and after completion of the ANBP2 trial over an additional median 6.9 years by linkage of data to the Australian Institute of Health and Welfare National Death Index (death registry), yielding the International Classification of Disease, version 10, coding for cause of death. The censor date for all analyses was 31 October 2009.

**Statistical analyses**

Statistical analyses were performed using Stata version 11.2 for Windows (College Station, TX).²⁶ We first used Cox regression hazard models to explore the risk relationship for all-cause and cardiovascular mortality for each baseline CKD stage using no CKD (eGFR ≥60 ml/min/1.73 m²) as the reference category obtained from the individual equations separately. Then the predictive performance of the
Cox regression models between the equations were assessed using Akaike information criterion (AIC), Bayesian information criterion (BIC), and Harrell’s concordance statistics (C statistic). A subanalysis was undertaken of the data stratified for patient gender and age at randomization (<75 years or ≥75 years). These analyses were adjusted for clustering of patients within family practices and possible confounders such as age, sex, plasma cholesterol, obesity, pulse pressure, and preexisting medical conditions (diabetes, cardiovascular and coronary heart disease, use of antihypertensive) at baseline and randomized treatment regimen allocation.

Further analysis was conducted to identify the risk relationship for all-cause and cardiovascular mortality in the recategorized subgroups using the CKD-EPI equation in relation to the no CKD, control group. This analysis was first performed unadjusted and then adjusted for the same covariates used in earlier analysis. Kaplan–Meier survival curves were plotted for all-cause and cardiovascular mortality by recategorized subgroups along with the log-rank test.

RESULTS

Of the 6,083 elderly hypertensive patients in the ANBP2 study, the prevalence of patients with CKD (eGFR <60 ml/min/1.73 m²) that was CKD stage 3 or greater was 30.6% using the MDRD equation and 27.9% using the CKD-EPI equation. Reflective of the ANBP2 study exclusion criteria, there were no patients classified as CKD stage 5 at baseline using either equation (Supplementary Table S1). Use of the CKD-EPI equation resulted in classifying more of the ANBP2 cohort into having no CKD (72.1% vs. 69.4%; P = 0.001) than when using the MDRD equation. This change occurred mostly due to reclassification of patients who were identified as having mild CKD (CKD stage 3A or eGFR between ≥45 and <60 ml/min/1.73 m²) by MDRD estimation. Overall, the CKD-EPI equation reclassified 10% (187/1,862) of patients as no CKD who had been classified CKD with an eGFR <60 ml/min/1.73 m² using the MDRD equation. Conversely, using the CKD-EPI equation, 1% (22/4,221) of patients were reclassified as having CKD (stage 3A) who were initially classified as having no CKD using the MDRD equation.

The baseline demographic and clinical characteristics are summarized in Table 1 for both the overall ANBP2 population and the recategorized subgroups using CKD-EPI equation. In brief, those patients who were reclassified using the CKD-EPI equation as having no CKD (i.e., earlier classified CKD using the MDRD equation) showed that they were younger, more likely to be female, obese (BMI ≥ 30 kg/m²), and with a raised plasma total cholesterol compared with those who were identified as having CKD using both equations. There was no difference detected for previous medical conditions. Conversely, those reclassified to CKD using CKD-EPI were older and more likely to be male compared with those classified as no CKD, control using both equations (Table 1).

CKD-EPI and MDRD equation performance in predicting survival

Over a median 10.8-year follow-up, 1,830 deaths were recorded in the ANBP2 cohort, of which 940 were of cardiovascular origin. In the elderly hypertensive patients, both eGFR equations predicted a similar higher mortality rate (per 1,000 patient-years) and higher hazard ratios (HRs) for both all-cause and cardiovascular mortality in those with CKD stages 3B and 4 compared with those classified as having no CKD (eGFR ≥60 ml/min/1.73 m²) at baseline (Figure 1). However, for those with CKD stage 3A, when compared with use of the MDRD equation, the CKD-EPI equation performed slightly better in risk prediction especially for all-cause mortality (CKD-EPI equation: HR, 1.13; 95% confidence interval [CI], 1.01–1.27; P = 0.03 vs. MDRD equation: HR, 1.10; 95% CI, 0.98–1.23; P = 0.10). A corresponding higher mortality rate was also observed for this stage when the CKD-EPI equation was used compared with use of MDRD. However, all model parameters (AIC, BIC, and C statistic) were identical for the Cox models of both all-cause and cardiovascular mortality predictions between the CKD-EPI and MDRD equations (Figure 1). Further, subanalysis conducted by stratifying for gender (Supplementary Table S2) and age (Supplementary Table S3) showed no significant difference in the predictive capacity of the equations except for CKD stage 3A in females. The CKD-EPI equation resulted in a statistically significant prediction of future all-cause and cardiovascular mortality in females for CKD stage 3A (Supplementary Table S2).

We observed a significantly lower risk for all-cause (HR, 0.46; 95% CI, 0.31–0.68; P < 0.001) and cardiovascular (HR, 0.35; 95% CI, 0.19–0.65; P = 0.001) mortality in the no CKD, reclassified subgroup using the CKD-EPI equation in unadjusted analysis compared with those who were no CKD, control. When these analyses were adjusted for possible confounders, the lower risk persisted for all-cause mortality (HR, 0.66; 95% CI, 0.44–0.99; P = 0.046); however, there was no detectable significance for cardiovascular mortality (HR, 0.54; 95% CI, 0.28–1.04; P = 0.07). Kaplan–Meier survival curves by year for different recategorized subgroups are presented in Figure 2. Significant differences between subgroups regarding the incidence of all-cause mortality (Figure 2A, overall log-rank test P < 0.001) and cardiovascular mortality (Figure 2B, overall log-rank test P < 0.001) were observed.

DISCUSSION

This study of the use of 2 eGFR equations in a prospective trial of an elderly treated hypertensive cohort showed that use of the CKD-EPI equation resulted in a greater number of patients having no CKD (eGFR ≥ 60 ml/min/1.73 m²) than when the MDRD equation was used. The CKD-EPI equation reclassified approximately 10% of the CKD patients (eGFR <60 ml/min/1.73 m² by MDRD) as having no CKD. The performance of eGFR using the CKD-EPI equation for future all-cause and cardiovascular mortality prediction did not show any significant superiority over the MDRD equation. Both equations predicted a similar higher risk for CKD stage 3B or greater (eGFR <45 ml/min/1.73 m²) with respect to all-cause and cardiovascular mortality; however, the CKD-EPI equation performed marginally better for all-cause mortality prediction for those with CKD stage 3A (eGFR 45–59 ml/min/1.73 m²). Better survival or no increased risk was observed among those who were recategorized to no...
CKD, reclassified using the CKD-EPI equation compared with those classified as no CKD using both equations.

In our study, approximately one quarter of elderly hypertensive patients were classified as having CKD stage 3A using either eGFR equation. Studies assessing the usefulness of GFR equations to predict mortality risk in elderly populations (aged ≥65 years) have shown that a moderate decline in renal function (CKD stage 3A) is common in this age group. Even though such decline is related to higher risk of mortality among younger and middle aged populations, often that is not the case among the elderly.27,28 However, the classification based only on use of the CKD-EPI equation predicted a statistically significant and slightly higher future risk for all-cause mortality for this CKD stage compared with the no-CKD group, especially in females. Nevertheless, the magnitude of difference between the all-cause mortality HRs calculated using these 2 equations was quite small and thus unlikely to be clinically significant. Those with a more marked level of renal function loss (classified as having CKD stage 3B and worse), calculated using either equation, had a clearly associated and similarly higher risk for all-cause and cardiovascular mortality, with risk increasing as renal function worsened. The quantitative estimates for fitness of the model to predict future risk such as a lower AIC and BIC measurement worsened. The quantitative estimates for fitness of the 2 equations in predicting future risk.

Our results are consistent with the mixed results observed by others who compared the predictive performance of the 2 equations. A study conducted in the elderly showed that the 2 equations perform similarly in renal function classification and in long-term risk prediction.29 In contrast, in younger and middle aged populations, the more recently developed CKD-EPI equation showed improved accuracy when

### Table 1. Baseline demographic and clinical characteristics of the study participants

<table>
<thead>
<tr>
<th>Variable</th>
<th>No CKD Control</th>
<th>Reclassified by CKD-EPI A</th>
<th>Reclassified by CKD-EPI B</th>
<th>CKD C</th>
<th>Both D</th>
</tr>
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<tbody>
<tr>
<td>Number</td>
<td>6,083</td>
<td>4,199</td>
<td>187</td>
<td>22</td>
<td>1,675</td>
</tr>
<tr>
<td>Male, %</td>
<td>48.9</td>
<td>53.9</td>
<td>12.3&lt;sup&gt;a,b&lt;/sup&gt;</td>
<td>90.9&lt;sup&gt;a,b&lt;/sup&gt;</td>
<td>39.7</td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Mean ± SD, y</td>
<td>71.9±4.9</td>
<td>71.2±4.6</td>
<td>67.8±3.0&lt;sup&gt;a,b&lt;/sup&gt;</td>
<td>77.1±2.9&lt;sup&gt;a,b&lt;/sup&gt;</td>
<td>74.0±5.0</td>
</tr>
<tr>
<td>- Age ≥ 75 y, %</td>
<td>30.2</td>
<td>24.3</td>
<td>5.9&lt;sup&gt;a,b&lt;/sup&gt;</td>
<td>100 &lt;sup&gt;a,b&lt;/sup&gt;</td>
<td>46.9</td>
</tr>
<tr>
<td>Body mass index</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Mean ± SD, kg/m²</td>
<td>27±4</td>
<td>27±4</td>
<td>28±4</td>
<td>27±3</td>
<td>27±4</td>
</tr>
<tr>
<td>- ≥30 (obese), %</td>
<td>21.7</td>
<td>21.2</td>
<td>27.8&lt;sup&gt;a&lt;/sup&gt;</td>
<td>22.7</td>
<td>22.1</td>
</tr>
<tr>
<td>Increase waist, %</td>
<td>46.9</td>
<td>45.0</td>
<td>55.6&lt;sup&gt;a&lt;/sup&gt;</td>
<td>36.3</td>
<td>50.6</td>
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<tr>
<td>Blood pressure</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Systolic (mean ± SD)</td>
<td>168±13</td>
<td>167±12</td>
<td>166±13&lt;sup&gt;b&lt;/sup&gt;</td>
<td>168±10</td>
<td>170±13</td>
</tr>
<tr>
<td>- Diastolic (mean ± SD)</td>
<td>91±8</td>
<td>91±8</td>
<td>92±7&lt;sup&gt;b&lt;/sup&gt;</td>
<td>93±9</td>
<td>90±9</td>
</tr>
<tr>
<td>Previous medical condition</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>- Treated with antihypertensive, %</td>
<td>62.2</td>
<td>60.0</td>
<td>64.2</td>
<td>59.1</td>
<td>67.6</td>
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<tr>
<td>- Diabetes, %</td>
<td>7.3</td>
<td>7.5</td>
<td>6.4</td>
<td>4.5</td>
<td>6.7</td>
</tr>
<tr>
<td>- Cerebrovascular disease, %</td>
<td>4.5</td>
<td>4.3</td>
<td>2.1</td>
<td>9.1</td>
<td>5.2</td>
</tr>
<tr>
<td>- Coronary heart disease, %</td>
<td>7.9</td>
<td>7.1</td>
<td>3.7&lt;sup&gt;b&lt;/sup&gt;</td>
<td>4.5</td>
<td>10.1</td>
</tr>
<tr>
<td>Laboratory values</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Raised total cholesterol (&gt;6.5 mmol/L), %</td>
<td>22.2</td>
<td>20.2</td>
<td>29.8&lt;sup&gt;a&lt;/sup&gt;</td>
<td>30.0</td>
<td>26.4</td>
</tr>
<tr>
<td>- Low high-density lipoprotein (&lt;1mmol/L), %</td>
<td>13.3</td>
<td>13.3</td>
<td>9.9</td>
<td>5.0</td>
<td>13.9</td>
</tr>
<tr>
<td>- eGFR MDRD (ml/min/1.73 m²)</td>
<td>63.6±13.9</td>
<td>74.±11.8</td>
<td>58.3±0.9&lt;sup&gt;a,b&lt;/sup&gt;</td>
<td>60.4±0.4&lt;sup&gt;a,b&lt;/sup&gt;</td>
<td>51.2±6.4</td>
</tr>
<tr>
<td>- eGFR CKD-EPI (ml/min/1.73 m²)</td>
<td>64.9±13.6</td>
<td>75.8±9.4</td>
<td>61.2±0.7&lt;sup&gt;a,b&lt;/sup&gt;</td>
<td>59.7±0.4&lt;sup&gt;a,b&lt;/sup&gt;</td>
<td>51.6±6.8</td>
</tr>
</tbody>
</table>

Abbreviations: CKD, chronic kidney disease; CKD-EPI, Chronic Kidney Disease Epidemiology Collaboration; eGFR, estimate glomerular filtration rate; MDRD, Modifications of Diet in Renal Disease; SD, standard deviation.

<sup>a</sup>Difference exist (<i>P</i> < 0.05) with no CKD, control.

<sup>b</sup>Difference exist (<i>P</i> < 0.05) with CKD, control.
estimating GFR and when predicting subsequent higher mortality risk compared with the MDRD equation.\textsuperscript{18,31–33} These observed mixed results could be due to sample variability as well as population differences. Clearly, the ongoing development in GFR estimate equations has resulted in improved accuracy and identification of CKD, though the debate on choosing the more appropriate CKD classification framework in the elderly continues.

Even though both equations performed similarly in predicting fatal outcome in the present study, the CKD-EPI equation categorized a higher proportion of people into the category of eGFR at around 60 ml/min/1.73 m\(^2\), which is an important threshold for diagnosing CKD. We found that those who were reclassified based on the CKD-EPI estimate to the no-CKD group (i.e., 10% of patients who were classified as CKD stage 3A using MDRD) were not at higher risk; rather they experienced longer survival for all-cause and cardiovascular mortality. This finding is supportive of the use of the CKD-EPI equation in this older population group, particularly where hypertension is a feature. The patients in this elderly cohort who were reclassified as no CKD using the CKD-EPI estimate of GFR were relatively younger and more likely to be women. These findings reinforce earlier findings\textsuperscript{10} and suggest that the use of estimates of GFR with the MDRD equation would significantly increase the estimated prevalence of CKD at the population level,\textsuperscript{17,34} which could consequently adversely affect health resource allocation and policy if such estimates are used.

The prevalence of CKD increases markedly with advancing age;\textsuperscript{35} however, this is often underrecognized when serum creatinine concentration alone is used to determine GFR. The introduction of automated eGFR reporting together with serum creatinine measurements has allowed for improved recognition of reduced and declining renal function in all age

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**Figure 1.** Risk prediction (hazard ratio) and number of events (event rate) by chronic kidney disease stage for all-cause and cardiovascular mortality for Modifications of Diet in Renal Disease (MDRD) and Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equations. Hazard ratio adjusted for patient sex, baseline age, diabetes status, being obese, high cholesterol, pulse pressure, antihypertensive drug use, preexisting cardiovascular condition, and in-trial antihypertensive drug and effect of family practices. Chronic kidney disease stage estimate glomerular filtration rate in mL/min/1.73 m\(^2\). Abbreviations: AIC, Akaike information criterion; BIC, Bayesian information criterion; C statistic, Harrell’s concordance statistics; eGFR, estimate glomerular filtration rate.
coholes, particularly the elderly where renal function can be dramatically reduced before serum creatinine rises to abnormal levels. As the presence of CKD is associated with worse outcome, particularly from cardiovascular disease, early diagnosis of CKD is critical for allowing optimal treatment and cardiovascular risk reduction, with the aim of improving the quality of life and survival in the elderly population.

Our study has several limitations. First, serum creatinine of ANBP2 study participants was measured before the time when creatinine estimates were standardized to IDMS values. As a result, for the CKD-EPI equation, the creatinine levels were calibrated by a 5% reduction. Though this method of calibration is widely accepted, it might have introduced some systematic bias in GFR estimation. Second, only serum creatinine measures were available to determine the stage of CKD. No other information was available on other renal indices such as albuminuria, proteinuria, urine sediment, or renal structural changes. Absence of this information may have caused some patients with CKD stage 1 and 2 to be labeled as having no CKD. In addition, the most recent diagnostic framework for the staging of CKD includes urinary albumin, which this study was not able to provide. Availability of this information could have allowed more robust analysis of the predictive capacity of the 2 equations. Third, all study participants included in the analysis had treated hypertension; therefore, from this perspective, they had an increased risk of fatal outcome. Thus, caution should be taken in generalizing the results of this study to all elderly people. Finally, those patients reclassified to no CKD using the CKD-EPI equation compared with the classification using the MDRD equation constitute only 3% of the total study population. This small sample size could be one reason why the results lost statistical significance when adjusted for other variables for cardiovascular outcome.

In conclusion, our study suggests that in elderly treated hypertensive patients, estimation of GFR using the CKD-EPI equation resulted in similar risk prediction as when the MDRD equation was used. However, we showed that estimation of GFR using the CKD-EPI equation in this elderly group resulted in a reduction in the number of patients classified as CKD, more appropriately categorizing no CKD, with the potential to reduce subsequent investigations and treatments without added risk. Finally, these findings could provide useful contribution to future metaanalysis for decision making and for providing a consistent picture on the use of an GFR estimation equation in the elderly population.

SUPPLEMENTARY MATERIAL

Supplementary materials are available at American Journal of Hypertension (http://ajh.oxfordjournals.org).

ACKNOWLEDGMENTS

The ANBP2 was supported by the Australian Commonwealth Department of Health and Aging; the National Health and Medical Research Council (NHMRC) of Australia (grant 546272); and Merck Sharp & Dohme (Australia) Pty Ltd. The long-term ANBP2 cohort follow-up study was supported by a NHMRC Program grant (546272) awarded to C.M.R., who is supported by a NHMRC Research Fellowship (1045862). We are indebted to the participants, family practitioners, study staffs, data management centers, and ANBP2 Management Committee. The Management Committee consists of the following members: Lindon M.H. Wing (chair), Christopher M. Reid, Lawrence J. Beilin, Mark A. Brown, Garry L.R. Jennings, Collin I. Johnston, John J. McNeil, John E. Marley, Trefor O. Morgan, Philip Ryan, John Shaw (deceased), Malcolm J. West, and Graham J. MacDonald.

DISCLOSURE

The authors declared no conflict of interest.

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