Clinical Biochemistry 46 (2013) 1423–1429

Contents lists available at ScienceDirect

Clinical Biochemistry

journal homepage: www.elsevier.com/locate/clinbiochem

Comparison between IDMS-traceable Jaffe and enzymatic creatinine assays for estimation of glomerular filtration rate by the CKD-EPI equation in healthy and diabetic subjects

Amanda Veiga Cheuiche [a], Ariana Aguiar Soares [a], Eduardo Guimarães Camargo [a], Letícia Schwerz Weinert [a], Joíza Lins Camargo [a,b], Sandra Pinho Silveiro [a,c,*]

[a] Graduate Program in Medical Science: Endocrinology, Universidade Federal do Rio Grande do Sul (UFRGS), Brazil
[b] Clinical Pathology Department, HCPA, Brazil
[c] Endocrine Unit, HCPA, Brazil

ARTICLE INFO

Article history:
Received 27 November 2012
Received in revised form 23 April 2013
Accepted 25 May 2013
Available online 5 June 2013

Keywords:
Creatinine
Glomerular filtration rate
Enzymatic
Jaffe
CKD-EPI
MDRD
Kidney function
Diabetes mellitus

ABSTRACT

Objectives: The aim of this paper was to compare the agreement between creatinine measured by Jaffe and enzymatic methods and their putative influence on eGFR as calculated by the CKD-EPI (Chronic Kidney Disease Epidemiology Collaboration) equation in healthy and diabetic individuals.

Design and methods: Cross-sectional study conducted in 123 adult southern Brazilians with GFR > 60 mL/min/1.73 m^2 (53 patients with type 2 diabetes, 70 healthy volunteers). Mean age was 49 ± 16 years (range of 19–86). Most were female (55%) and white (83%). Creatinine was measured by a traceable Jaffe method (Modular P, Roche Diagnostic) and by an enzymatic method (CREA plus, Roche/Hitachi 917). GFR was measured by the ^{51}Cr-EDTA single-injection method.

Results: Serum creatinine measured by the Jaffe and enzymatic methods was similar in healthy subjects (0.79 ± 0.16 vs. 0.79 ± 0.15 mg/dL, respectively, P = 0.76), and diabetic patients (0.96 ± 0.22 vs. 0.92 ± 0.29 mg/dL, respectively, P = 0.17). However, the correlation between the two methods was higher in the healthy group (r = 0.90 vs. 0.76, P < 0.001). The difference between Jaffe creatinine and enzymatic creatinine was <10% in 63% of cases in the healthy group and 40% of cases in the diabetes group (P = 0.018). In the subset of patients with diabetes, eGFR based on enzymatic assay results showed better agreement with measured GFR than did eGFR based on Jaffe results.

Conclusion: Jaffe and enzymatic creatinine methods show adequate agreement in healthy subjects, but in the presence of diabetes, the enzymatic method performed slightly better.

© 2013 The Canadian Society of Clinical Chemists. Published by Elsevier Inc. All rights reserved.

Introduction

The National Kidney Foundation (NKF) defines chronic kidney disease (CKD) as the presence of structural or functional renal abnormalities for at least 3 months [1].

Two laboratory parameters, albuminuria and glomerular filtration rate (GFR), are the cornerstone for the diagnosis and classification of CKD [2]. Measured GFR is considered the reference method, but is restricted to special situations [3] because of its complexity and high cost. Hence, in daily clinical practice, estimation of GFR (eGFR) using equations based on serum creatinine levels adjusted for age, gender and ethnicity is the recommended approach for evaluation of renal function. Neither the CKD-EPI (Chronic Kidney Disease Epidemiology Collaboration) nor the MDRD (Modification of Diet in Renal Disease) study equation is optimal across all populations and GFR ranges [4,5], but the CKD-EPI equation appears to be superior for categorization of patients in terms of clinical risk [6]. The National Kidney Disease Education Program (NKDEP) has not made a recommendation on general implementation of this equation, since it is still being validated. While offering some improvement for eGFR between 60 and 120 mL/min/1.73 m^2, it is not clear that implementing CKD-EPI in place of MDRD equation would alter clinical detection or management of patients with CKD. However, a laboratory that reports eGFR numeric values > 60 mL/min/1.73 m^2 should consider using the CKD-EPI equation. (http://nkdep.nih.gov/lab-evaluation/gfr/estimating.shtml accessed January 17, 2013).

At present, it is formally recommended that laboratories routinely report eGFR when serum creatinine testing is requested [1]. An accurate measurement of serum creatinine forms the basis of compliance with these guidelines. Currently, most routine serum creatinine assays are based on the Jaffe reaction, a method first described in 1886 [7]. This method is widely used because of its simplicity and low cost. However,

Abbreviations: eGFR, estimated glomerular filtration rate; CKD-EPI, Chronic Kidney Disease Epidemiology Collaboration; MDRD, Modification of Diet in Renal Disease.

* Corresponding author at: Serviço de Endocrinologia, HCPA, Rua Ramiro Barcelos, 2350, Prédio 12, 4° andar, Porto Alegre, RS, Brazil.
E-mail address: silveirosandra@gmail.com (S.P. Silveiro).

0009-9120/$ – see front matter © 2013 The Canadian Society of Clinical Chemists. Published by Elsevier Inc. All rights reserved.
http://dx.doi.org/10.1016/j.clinbiochem.2013.05.067

Table 1
Clinical and laboratory profile of healthy volunteers and diabetic subjects.

	Healthy subjects (N = 70)	Diabetic subjects (N = 53)	P-value
Male sex	25 (36%)	31 (59%)	0.012
White ethnicity	58 (83%)	44 (83%)	0.98
Age (years)	41 ± 16	59 ± 9	<0.001
BMI (kg/m^2)	26 ± 5	29 ± 4	<0.001
Fasting plasma glucose (mg/dL)	91 ± 9	139 ± 38	<0.001
Microalbuminuria	–	22 (42%)	–
Macroalbuminuria	–	7 (13%)	–
Metformin therapy	–	48 (91%)	–
Low-dose aspirin therapy	–	50 (94%)	–
Fibrate therapy	–	6 (11%)	–

Data expressed as mean ± SD or N (%). BMI, body mass index.

a number of studies have indicated that enzymatic methods tend to be superior to the Jaffe reaction because they are less subject to influence by interfering substances, especially in diabetic subjects [8–11]. Enzymatic assays are considerably more expensive than the Jaffe method, which limits their wider implementation, especially in developing countries. Therefore, the aim of this paper was to compare the agreement between creatinine levels as measured by traceable Jaffe and enzymatic methods and the putative influence on eGFR calculated by the CKD-EPI equation in healthy subjects and type 2 diabetic patients.

Methods

Subjects

This cross-sectional study was conducted in a sample of individuals from Southern Brazil (healthy volunteers and type 2 diabetic patients) with GFR > 60 mL/min/1.73 m^2. Healthy individuals also had normal glucose tolerance and blood pressure levels. All subjects underwent evaluation of family history and personal medical history.

The exclusion criteria were BMI > 35 kg/m^2, history of cancer in the 5 years preceding study enrollment, and, for normal volunteers, use of drugs other than oral contraceptives or thyroid hormone. All participants were invited to take part in the study and provided written informed consent. The study protocol was approved by the Hospital de Clínicas de Porto Alegre Research Ethics Committee with judgment number 07063.

Assessment of renal function

GFR was measured by ^{51}Cr-EDTA single-injection method, and the procedure is described below, following the British Nuclear Medicine Society Guidelines [12].

a) Patient preparation: caffeine drinks were avoided; a low protein meal, exercise restriction, and adequate hydration during the test were prescribed. Body surface area was calculated according to Gehan and George formula: $0.0235 \times ([100 \times \text{height}]^{0.42246}) \times (\text{weight}^{0.51456})$ [13].
b) Syringe preparation: ^{51}Cr-EDTA dose of 5.55 MBq was measured by volume, and the syringe was filled right up to the end of the needle.
c) Injection technique: all activity was injected into the blood stream without extravasation.
d) Blood sampling: samples were collected from the opposite arm, 2, 3 and 4 h post-injection. The blood samples are centrifuged at 1000 *g* for 10 min to separate the red blood cells from the plasma. Two milliliter of plasma was pipetted into counting tubes in duplicate.
e) Preparation of the standard: a predetermined volume of tracer (2 mL), was drawn up using a pipette, and the activity was emptied into the volumetric flask filled with water to the mark of 500 mL.
f) Counting: plasma samples were counted with appropriate standards and blanks for background in a well counter.
g) GFR calculation: the logarithm of the plasma activity was plotted as a function of time and the apparent zero time plasma activity determined by extrapolation of the linear part of the curve. This is a single-compartment approach, and as such, the estimation of the area under the curve will be underestimated. Chantler's correction of the slope–intercept value for the approximation was used, with a constant multiplicative correction factor of 0.87 for the missing area under the curve due to the fast exponential, therefore, GFR = volume of distribution × 0.693 × 0.87 × 1000 / t1/2, according to Chantler, and expressed as mL/min/1.73 m^2 [14]. The mean intra-individual coefficient of variation of GFR at our laboratory is 12% [15].

Serum creatinine was measured by a Jaffe method (Modular P, Roche Diagnostic, Mannheim, Germany) traceable to isotope dilution mass spectrometry (ID-MS) and by an enzymatic method (CREA Plus, Roche Hitachi 917 Roche Diagnostic, Mannheim, Germany). For the Jaffe method, the intra- and interassay coefficients of variation (CV)

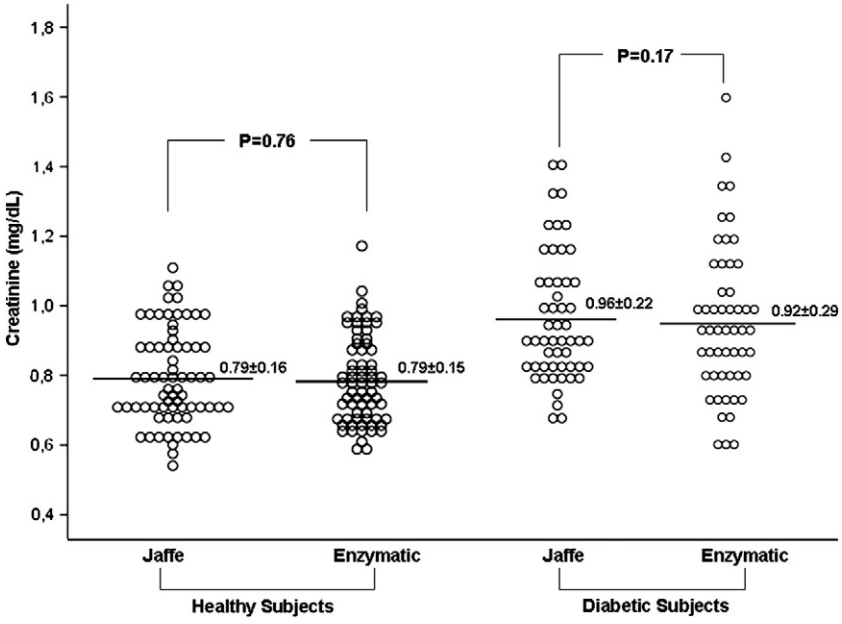

Fig. 1. Individual serum creatinine values (mean ± SD) as measured by IDMS-traceable Jaffe and enzymatic methods in healthy (N = 70) and diabetic (N = 53) subjects.

were 1.5% and 2.5% respectively for a creatinine level of 1.32 mg/dL. For the enzymatic method, CVs were 0.9% (within-run) and 2.1% (between-run) at a creatinine level equal to 1.54 mg/dL. To convert serum creatinine in mg/dL to μmol/L, multiply by 88.4.

eGFR was calculated by the CKD-EPI equation, as follows: GFR (mL/min/1.73 m^2) = 141 · min (Scr / k, 1)$^{\alpha}$ · max (Scr / κ, 1)$^{-1.209}$ · 0.993^{age} · 1.018 (if female) · 1.159 (if black), where Scr is serum creatinine, κ is 0.7 for women and 0.9 for men, α is −0.329 for women and

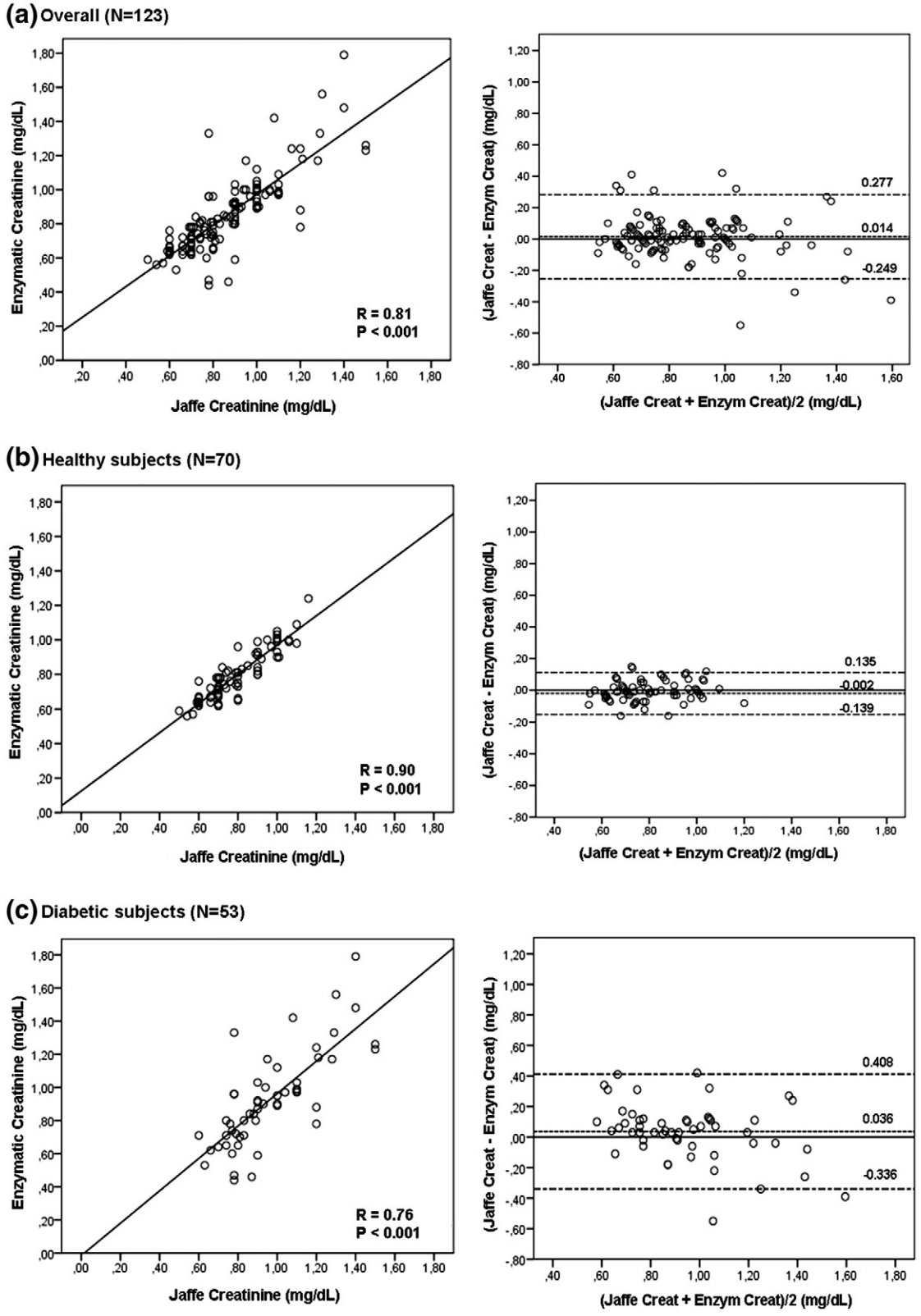

Fig. 2. Pearson's correlation coefficients between Jaffe and enzymatic creatinine (panels at left) and plots of mean Jaffe and enzymatic creatinine vs. the difference between them (panels at right) in the sample as a whole (panel a), among healthy subjects (panel b) and in diabetic subjects (panel c). Bias (dotted lines) and limits of agreement (dashed lines) are depicted in the right panel plots.

−0.411 for men, min indicates minimum Scr / κ or 1, and max indicates maximum Scr / κ or 1 [16].

Statistical analyses

Results were presented as mean ± standard deviation or median (range), unless otherwise stated. The Kolmogorov–Smirnov test was employed to assess the distribution of variables. Bias was calculated as the mean difference between creatinine methods and between measured and estimated GFR. Accuracy was calculated as the percentage of estimates of eGFR within 30% (P30) of measured GFR and of Jaffe creatinine within 10% of enzymatic creatinine values. The agreement between creatinine methods and between measured GFR and eGFR was evaluated by Bland–Altman analysis, with calculation of agreement limits (bias ± 2 SD) and confidence intervals (CIs) [17]. Correlation coefficients were employed to evaluate the determinants of serum creatinine measured by each method. The sample size for this study was calculated as 48 pairs of measurements, based on a 0.1 mg/dL difference in creatinine between methods, 90% statistical power, and $\alpha = 0.05$. All analyses were performed in the PASW Statistics 18 software environment and the level of significance adopted was 5%.

Results

The clinical and laboratory characteristics of the subjects at the time of enrollment are shown in Table 1. Diabetic patients were generally older and more predominantly male.

Serum creatinine levels measured by the Jaffe and enzymatic methods were similar in both the healthy (0.79 ± 0.16 vs. 0.79 ± 0.15 mg/dL, respectively, P = 0.76), and the diabetic (0.96 ± 0.22 vs. 0.92 ± 0.29 mg/dL, P = 0.17; Fig. 1) groups, and was significantly higher in the diabetic patients (P < 0.001), despite similar measured GFRs between groups (115 ± 24 vs. 107 ± 29 mL/min/1.73 m^2, respectively in healthy and diabetic patients, P = 0.097). The correlation between the two creatinine methods was strong and statistically significant in both groups. However, it was higher in the healthy group (r = 0.90 vs. 0.76, P < 0.001; Fig. 2, panels at left). The agreement between Jaffe and enzymatic creatinine methods in the sample as a whole is shown in Fig. 2 (panels at right).

The difference between Jaffe creatinine and enzymatic creatinine was less than 10% (i.e. within the acceptable range of variability) in 63% of cases in the healthy group, versus only 40% of cases in the diabetic group (P = 0.018). Enzymatic method results correlated borderline and weakly with blood glucose levels (r = 0.18, P = 0.05), whereas Jaffe creatinine was significantly correlated with blood glucose (r = 0.30, P = 0.001).

Fig. 3 depicts measured and estimated GFR for each group. In diabetic patients, both creatinine methods largely underestimated measured GFR. In this group, eGFR using enzymatic creatinine was higher, and therefore closer to measured GFR than was Jaffe creatinine based equation. Accuracy (the number of cases in which eGFR differed less than 30% from measured GFR) was 87% for CKD-EPI_{enz} and 86% for CKD-EPI_{Jaffe} in healthy subjects. In the diabetic patients, accuracy was significantly lower: 64% for CKD-EPI_{enz} and CKD-EPI_{Jaffe} alike (P < 0.01). Fig. 4 shows Bland–Altman plots for the agreement between eGFR and measured GFR in the sample as a whole and in the healthy and diabetic groups.

Discussion

In this study, we demonstrated adequate agreement between creatinine levels measured by Jaffe and enzymatic methods in healthy subjects. However, in the diabetic patients, whose GFR was >60 mL/min/1.73 m^2, the enzymatic method performed slightly better, and both creatinine methods largely underestimated measured GFR. Some authors have obtained reasonably good comparisons between the two creatinine methods [18–20], but a recent study highlights the greater interference of the presence of diabetes with the Jaffe method [11].

In his landmark 1886 paper, Jaffe described the reaction that occurs between creatinine and picrate ions formed in an alkaline medium, producing a red–orange adduct [21]. At that time, Jaffe already noticed that the alkaline picrate reaction could also be observed with other organic compounds, such as acetone and glucose [21]. The nonspecific nature of the Jaffe reaction is widely known, and interference is quite common with a number of substances. The compounds that are known to produce Jaffe-like chromogens include protein, glucose, ascorbic acid, ketone bodies, pyruvate, guanidine, cephalosporins and bilirubin [7,20], with protein having the greatest influence [22]. Conversely, enzymatic methods—as their name implies—rely on a cascade of enzymatic reactions. Hydrogen peroxide ammonium is the end product for some of these systems, ultimately reacting with dye for photometric determination of the concentration of creatinine [23]. Recent studies stress that this method is highly accurate and is less subject to interference than the Jaffe reaction [10,11,15]. However, it is also considerably more expensive—about 10 times the price of Jaffe methods, which limits its wider implementation, especially in developing countries.

In order to account for the sensitivity of alkaline picrate methods to non-creatinine chromogens, manufacturers have adjusted calibration to minimize interference by introducing a negative offset to compensate for the positive intercept [24]. However, the trueness of such

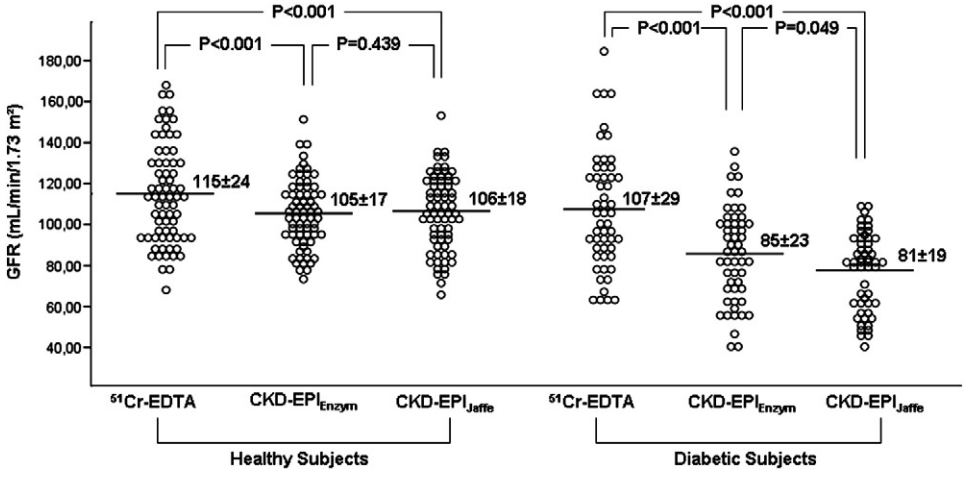

Fig. 3. Glomerular filtration rate (GFR) as measured by ^{51}Cr-EDTA and estimated by the CKD-EPI equation (mean ± SD) in healthy (N = 70) and diabetic (N = 53) subjects.

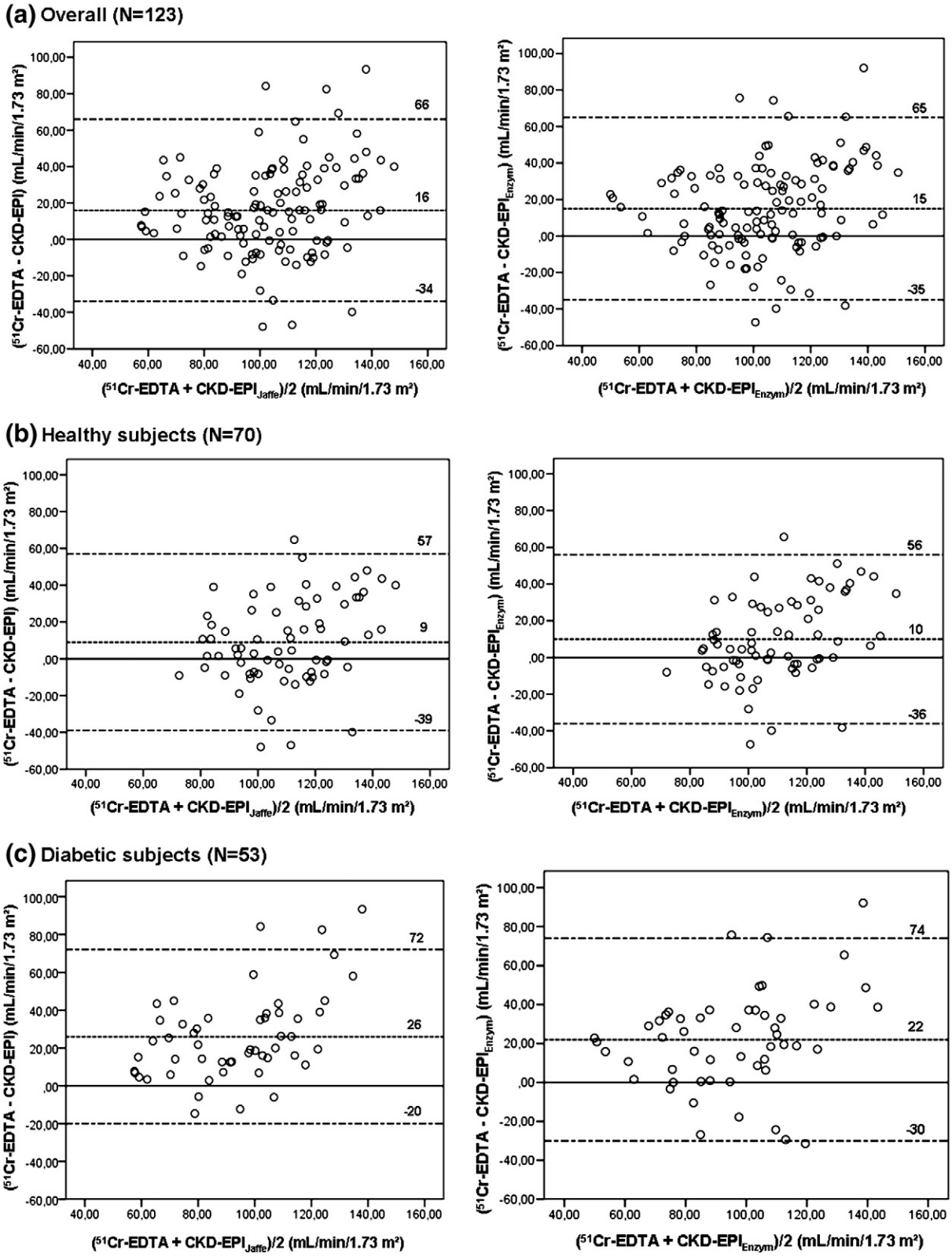

Fig. 4. Plots of the mean of estimated and measured GFR vs. the difference between them, using Jaffe (left) and enzymatic (right) methods, in the sample as a whole (a) and among healthy (b) and diabetic (c) subjects. Bias (dotted lines) and limits of agreement (dashed lines) are shown.

analytical methods may be poor for children (who generally present higher concentrations of chromogens and low serum creatinine levels) and individuals with low serum protein and creatinine levels, such as older adults, pregnant women and patients with cancer [25,26].

The enzymatic method is also not completely specific for creatinine [18] and is subject to interferences of its own, especially from bilirubin [27]. A recent study found that the influence of interfering substances was less frequent with enzymatic than with Jaffe methods,

but none of the creatinine measurement procedures was unaffected. The influence of the interfering substance was greater at creatinine concentrations within the reference range, and in patients with diabetes, the influence of interfering substances seems to be substantially more frequent with Jaffe-based procedures than with enzymatic assays [11]. Our data confirm these observations.

In our study, both the Jaffe and enzymatic methods yielded significantly higher creatinine levels in diabetics as compared with healthy volunteers, and this finding could not be ascribed to worse renal function in the diabetic group, since measured GFRs were similar. The proportion of males was greater in the diabetic group, which may suggest a muscle origin for higher creatinine levels. We have previously demonstrated that, even when matched by age and gender, diabetic patients tend to exhibit higher serum creatinine than nondiabetic controls, probably by analytical interference of elevated blood glucose levels [28]. Another possible interference with creatinine levels in diabetic subjects could be the effect of medications. Previous studies have demonstrated that high-dose aspirin use could elevate creatinine levels without changing measured GFR [29]. Nevertheless, other studies have failed to confirm these findings [30,31], and the low-dose aspirin of 200 mg/day used by our patients makes it an implausible explanation, as we have previously described no such an effect of aspirin in the renal function of diabetic subjects [32]. Another putative influence of drugs would be the use of fibrates by diabetic patients. In one study, fenofibrate was associated with increases in serum creatinine, probably unrelated to a decrease in GFR [33]. However, analysis of our data showed that the 6 diabetic individuals who were on fenofibrate therapy did not have significantly higher levels of creatinine. Whatever the reason for the higher creatinine levels in the diabetic group, it does not affect our results, because comparisons between the two creatinine methods were performed separately in each group.

Two recent studies indicate that the enzymatic method is more suitable for diabetic patients, since it is less subject to influence from interfering substances, particularly increased serum glucose, which has a significant effect on Jaffe methods [11,34]. Indeed, we have demonstrated that glucose was only significantly correlated with Jaffe but not with enzymatic creatinine. Although those studies showed that the enzymatic method correlates more closely with the reference method in sera from patients with increased glucose, it is not possible to affirm that glucose was the causal agent of the discrepancies observed with the Jaffe method. Other interferences specific to this patient population may have caused the disagreement. In our study, GFR estimated with the CKD-EPI equation using the enzymatic method for creatinine was closer to measured GFR in diabetic subjects, even though it could not bridge the entire gap between estimated and measured GFR. We have previously shown that CKD-EPI eGFR based on Jaffe creatinine measurements underestimated measured GFR both in healthy [35] and in normal range-GFR diabetic subjects, more markedly in the latter [36]. We now confirm that use of an enzymatic method for creatinine measurement decreases, but does not fully correct, the GFR underestimation in the presence of diabetes.

In this study, only individuals with GFRs > 60 mL/min/1.73 m^2 were evaluated. Previous studies have reported discordant findings depending on serum creatinine levels: one found less interference at high creatinine levels (mean, 3.02 [2.15–4.24] mg/dL) [11], whereas another one showed significant underestimation of pre-dialysis serum creatinine levels of patients receiving regular hemodialysis [34]. In the latter study, the negative bias was due to a different action of albumin in uremic serum as compared to normal serum (where it acts as a positive bias), binding with a negative-bias substance and exerting a negative effect. Since our results are directly applicable only to individuals with measured GFR in the normal range, further studies evaluating lower GFRs and therefore higher creatinine values are still warranted.

The present recommendation is that GFR should be automatically reported using the CKD-EPI or MDRD equation whenever a creatinine measurement is ordered [1]. Primary care physicians recommended subspecialty referrals earlier when using eGFR versus creatinine for assessment of kidney function, thus improving clinical outcomes for patients with CKD [37,38]. It is therefore of the utmost importance that available methods for measurement of creatinine be reliable, allowing correct identification of kidney disease.

Although our sample size was limited, it comprised the recommended number of individuals for identification of differences between methods, assuming 90% statistical power. One strength of our paper was the use of a reference method for GFR measurement, therefore enabling comparison of the performance of creatinine-based GFR estimation equations to a reference method. Although the clearance of ^{51}Cr-EDTA is about 5–10% less than that of inulin (possibly due to activity bound by plasma proteins), and also if an allowance of about 10% is made for the arteriovenous concentration difference (higher venous concentration), a composite correction factor applied to the slope clearance provides a reasonable estimate of the GFR [14,39]. This simplified one-compartment approach of GFR measurement is, nevertheless, less reliable than a two-compartment system (double slope analysis) [40].

Conclusion

In conclusion, we demonstrated adequate agreement between creatinine measured by Jaffe and enzymatic methods in healthy subjects, but in the presence of diabetes, the enzymatic method performed slightly better.

Conflict of interest statement

The authors stated that there are no conflicts of interest regarding the publication of this article.

Acknowledgments

The authors would like to thank the Fundo de Incentivo à Pesquisa (FIPE) do Hospital de Clínicas de Porto Alegre for its financial support. AVC and AAS are supported by National Council for Scientific and Technological Development (CNPq) grants. We thank Vania Naomi Hirakata and Luciano Santos Guimarães for statistical analyses supervision.

References

[1] Levey AS, Jong PE, Coresh J, El Nahas M, Astor BC, Matsushita K, et al. The definition, classification and prognosis of chronic kidney disease: a KDIGO Controversies Conference report. Kidney Int 2011;80:17–28.
[2] Soares AA, Eyff TF, Campani RB, Ritter L, Camargo JL, Silveiro SP. Glomerular filtration rate measurement and prediction equations. Clin Chem Lab Med 2009;47(9):1023–32.
[3] Stevens LA, Levey AS. Measured GFR as a confirmatory test for estimated GFR. J Am Soc Nephrol 2009;20:2305–13.
[4] Earley A, Miskulin D, Lamb EJ, Levey AS, Uhlig K. Estimating equations for glomerular filtration rate in the era of creatinine standardization: a systematic review. Ann Intern Med 2012;156(11):785–9 [5].
[5] Delanaye P, Cavalier E, Mariat C, Maillard N, Krzesinski JM. MDRD or CKD-EPI study equations for estimating prevalence of stage 3 CKD in epidemiological studies: which difference? Is this difference relevant?BMC Nephrol 2010;11:8 [http://www.biomedcentral.com/1471-2369/11/8].
[6] Matsushita K, Tonelli M, Levey AS, Coresh J, Hemmelgarn BR. Clinical risk implications of the CKD Epidemiology Collaboration (CKD-EPI) equation compared with the Modification of Diet in Renal Disease (MDRD) study equation for estimated GFR. Am J Kidney Dis 2012;60(2):241–9.
[7] Perrone RD, Madias NE, Levey AS. Serum creatinine as an index of renal function: new insights into old concepts. Clin Chem 1992;38(10):1933–53.
[8] Crocker H, Shephard MD, White GH. Evaluation of an enzymatic method for determining creatinine in plasma. J Clin Pathol 1988;41:576–81.
[9] Fossati P, Ponti M, Passoni G, Tarenghi G, d'Eril GV, Prencipe L. A step forward in enzymatic measurement of creatinine. Clin Chem 1994;40:130–7.
[10] Blijenberg BG, Brouwer HJ, Kuller TJ, Leeneman R, van Leevwen CJ. Improvements in creatinine methodology: a critical assessment. Eur J Clin Chem Clin Biochem 1994;32:529–37.
[11] Greenberg N, Roberts WL, Bachmann LM, Wright EC, Dalton RN, Zakowski JJ, et al. Specificity characteristics of 7 commercial creatinine measurement procedures by enzymatic and Jaffe method principles. Clin Chem 2012;58(2):391–401.

[12] Fleming JS, Zivanovic MA, Blake GM, Burniston M, Cosgriff PS. Guidelines for the measurement of glomerular filtration rate using plasma sampling. Nucl Med Commun 2004;25:759–69.
[13] Gehan EA, George S. Estimation of human body surface. Cancer Chemother Rep 1970;54:225–35.
[14] Chantler C, Barratt TM. Estimation of glomerular filtration rate from plasma clearance of ^{51}-chromium edetic acid. Arch Dis Child 1972;47:613–7.
[15] Gross JL, Friedman R, de Azevedo MJ, Silveiro SP, Pecis M. Effect of age and sex on glomerular filtration rate measured by ^{51}Cr-EDTA. Braz J Med Biol Res 1992;25:129–34.
[16] Levey AS, Stevens LA, Schmid CH, Zhang YL, Castro 3rd AF, Feldman HI, et al. A new equation to estimate glomerular filtration rate. Ann Intern Med 2009;150:604–12.
[17] Bland JM, Altman DG. Measuring agreement in method comparison studies. Stat Methods Med Res 1999;8:135–60.
[18] Peake M, Whiting M. Measurement of serum creatinine — current status and future goals. Clin Biochem Rev 2006;27(4):173–84.
[19] Badiou S, Dupuy AM, Descomps B, Cristol JP. Comparison between the enzymatic vitros assay for creatinine determination and three other methods adapted on the Olympus analyzer. J Clin Lab Anal 2003;17(6):235–40.
[20] Chromý V, Rozkosná K, Sedlák P. Determination of serum creatinine by Jaffe method and how to calibrate to eliminate matrix interference problems. Clin Chem Lab Med 2008;46(8):1127–33.
[21] Delanghe JR, Speeckaert MM. Creatinine determination according to Jaffe — what does it stand for? Nephrol Dial Transplant Plus 2011;4(2):83–6.
[22] Wuyts B, Bernard D, Van Den Noortgate N, Van de Walle J, Van Vlem B, De Smet R, et al. Reevaluation of formulas for predicting creatinine clearance in adults and children, using compensated creatinine methods. Clin Chem 2003;49:1011–4.
[23] Skurup A, Kristensen T, Wennecke G. New creatinine sensor for point-of-care testing of creatinine meets the National Kidney Disease Education Program guidelines. Clin Chem Lab Med 2008;46(1):3–8.
[24] Lamb EJ, Wood J, Stowe HJ, O'Riordan SE, Webb MC, Dalton RN. Susceptibility of glomerular filtration rate estimations to variations in creatinine methodology: a study in older patients. Ann Clin Biochem 2005;42:11–8.
[25] Panteghini M. Enzymatic assays for creatinine: time for action. Clin Chem Lab Med 2008;46(4):567–72.
[26] Delanghe JR, Cobbaert C, Harmoinen A, Jansen R, Laitinen P, Panteghini M. Focusing on the clinical impact of standardization of creatinine measurements: a report by the EFCC working group on creatinine standardization. Clin Chem Lab Med 2011;49(6):977–82.
[27] Owen LJ, Keevil BG. Does bilirubin cause interference in Roche creatinine methods? Clin Chem 2007;53:370–1.
[28] Camargo EG, Soares AA, Detanico AB, Weinert LS, Veronese FV, Gomes EC, et al. The Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation is less accurate in patients with type 2 diabetes when compared with healthy individuals. Diabet Med 2011;28(1):90–5.
[29] Burry HC, Dieppe PA. Apparent reduction of endogenous creatinine clearance by salicylate treatment.Br Med J 1976;2(6026):16–7 [3].
[30] Nielsen IL, Rasmussen S, Hilden T. Acetylsalicylic acid and renal function.Br Med J 1980;280(6214):610 [1].
[31] Rasmussen S, Petersen J, Nielsen IL, Christensen P, Hilden T. Effect of acetylsalicylic acid on renal function in systemic lupus erythematosus. Eur J Clin Pharmacol 1982;23(6):505–8.
[32] Camargo EG, Weinert LS, Soares AA, Ferreira MN, Araujo GN, Silveiro SP. Low-dose aspirin does not affect the renal function of microalbuminuric type 2 diabetic patients. Rev HCPA 2011;31(4):402–6.
[33] Keech A, Simes RJ, Barter P, Best J, Scott R, Taskinen MR, et al. Effects of long-term fenofibrate therapy on cardiovascular events in 9795 people with type 2 diabetes mellitus (the FIELD study): randomised controlled trial. Lancet 2005;26;366(9500):1849–61.
[34] Liu WS, Chung YT, Yang CY, Lin CC, Tsai KH, Yang WC, et al. Serum creatinine determined by Jaffe, enzymatic method, and isotope dilution-liquid chromatography–mass spectrometry in patients under hemodialysis. J Clin Lab Anal 2012;26(3):206–14.
[35] Soares AA, Eyff TF, Campani RB, Ritter L, Weinert LS, Camargo JL, et al. Performance of the CKD Epidemiology Collaboration (CKD-EPI) and the Modification of Diet in Renal Disease (MDRD) study equations in healthy south Brazilians. Am J Kidney Dis 2010;55(6):1162–3.
[36] Silveiro SP, Araujo GN, Ferreira MN, Souza FD, Yamaguchi HM, Camargo EG. Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation pronouncedly underestimates glomerular filtration rate in type 2 diabetes. Diabetes Care 2011;34(11):2353–5.
[37] Greer RC, Powe NR, Jaar BG, Troll MU, Boulware LE. Effect of primary care physicians' use of estimated glomerular filtration rate on the timing of their subspecialty referral decisions.BMC Nephrol 2011;12:1 [14].
[38] Jones GR, Mathew T, Johnson D, Peake M. Implementation of the routine reporting of eGFR in Australia and New Zealand. Scand J Clin Lab Invest Suppl 2008;241:23–9.
[39] Rehling M, Nielsen LE, Marqversen J. Protein binding of 99Tcm-DTPA compared with other GFR tracers. Nucl Med Commun 2001;22(6):617–23.
[40] Cohen ML. Radionuclide clearance techniques. Semin Nucl Med 1974;4(1):23–38.