Are prediction equations reliable for estimating resting energy expenditure in chronic kidney disease patients?

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Abstract

Background. The determination of resting energy expenditure (REE) is the primary step for estimating the energy requirement of an individual. Although numerous equations have been formulated for predicting metabolic rates, there is a lack of studies addressing the reliability of those equations in chronic kidney disease (CKD). Thus, the aim of this study was to evaluate whether the main equations developed for estimating REE can be reliably applied for CKD patients.

Methods. A total of 281 CKD patients (124 non-dialysis, 99 haemodialysis and 58 peritoneal dialysis) and 81 healthy control individuals were recruited. Indirect calorimetry and blood sample collection were performed after a 12-h fasting. Two most traditionally used equations for estimating REE were chosen for comparison with the REE measured by indirect calorimetry: (i) the equation proposed by Harris and Benedict, and (ii) the equation proposed by Schofield that is currently recommended by the FAO/WHO/UNU.

Results. Schofield's equation exhibited higher REE [1492 ± 220 kcal/day (mean ± SD)] in relation to Harris and Benedict's equation (1431 ± 214 kcal/day; P < 0.001), and both prediction equations showed higher REE in comparison with the reference indirect calorimetry (1352 ± 252 kcal/day; P < 0.001). In patients with diabetes, inflammation or severe hyperparathyroidism, the REE estimated by the Harris and Benedict equation was equivalent to that measured by indirect calorimetry. The intraclass correlation of the REE measured by indirect calorimetry with the Schofield's equation was r = 0.48 (P < 0.001) and with the Harris and Benedict’s equation was r = 0.58 (P < 0.001). According to the Bland and Altman analysis, there was a large limit of agreement between both prediction equations and the reference method. Acceptable prediction of REE (90–110% adequacy) was found in 47% of the patients by using the Harris and Benedict’s equation and in only 37% by using the Schofield’s equation.

Conclusions. The most traditionally used prediction equations overestimated the REE of CKD patients, and the errors were minimized in the presence of comorbidities.

There is a need to develop population-specific equations in order to adequately estimate the energy requirement of these patients.

Keywords: chronic kidney disease; energy requirement; indirect calorimetry; prediction equations; resting energy expenditure

Introduction

Chronic kidney disease (CKD) is recognized as an important public health problem, in which the incidence has increased markedly in the last few years [1]. A number of metabolic disturbances and catabolic conditions related to renal failure and dialysis therapy adversely affect the nutritional condition of CKD patients [2]. Thus, the evidence of high prevalence of protein-energy wasting and, particularly, the recognition of protein-energy wasting as a risk factor for poor clinical outcomes among the CKD population have fostered several strategies for the treatment of these patients [2–4]. An important element for promoting adequate nutritional status relies on the adequate management of energy balance.

The determination of resting energy expenditure (REE) is the primary step for the establishment of the energy requirement of an individual [5]. Few studies on REE in CKD patients have brought important contributions in this field. Previous reports, based on calorimetric methods, indicate reduced REE in non-dialysed CKD patients [6,7] and a normal to increased REE among dialysed patients [8–11] when compared with healthy subjects.

Indirect calorimetry is among the methods that most accurately measure the REE. However, several factors related to subjects (e.g. need of fasting and resting) and device (e.g. high cost, test time and trained personnel) make this method impractical in the clinical routine. Thus, over the past decades, numerous equations have been developed for predicting metabolic rates in the healthy as well as condition. The equation proposed by Harris and Benedict, in
1919, has been the most traditionally used for clinical and research purposes [12]. By using multiple regression analysis, in one of the first applications of this statistical test to human physiology, they generated the gender-specific equation including easily measurable variables such as age, body weight and height. The equation proposed by Schofield, in 1985, is the one recommended by the FAO/WHO/UNU expert consultation on human energy requirement for estimating REE [13]. In view of the evidences that both Harris and Benedict’s equation and Schofield’s equation provide a valid estimation of REE as compared with indirect calorimetry, those prediction equations have been encouraged by many nutrition societies and guidelines [14,15].

To find a simple method able to predict accurately the REE of CKD patients would be of relevant importance for the routine care of these patients. Since there is a lack of studies addressing the applicability of REE prediction equations in CKD, we aimed to evaluate whether the most traditionally used equations of REE are reliable for estimating REE in a CKD population.

Materials and methods

Patients

A total of 281 CKD patients (124 non-dialysis, 99 haemodialysis and 58 peritoneal dialysis) were included in the present study. We studied patients from the renal outpatient clinic and the dialysis unit of the Federal University of São Paulo (São Paulo, Brazil) who had participated in previous studies (10, 30–33) by following the same protocol for the assessment of REE.

Exclusion criteria were age <18 years, amputation, pregnancy, altered thyroid function, presence of malignancy, and hospitalization in the month prior to the study. Dialysis patients underwent haemodialysis or peritoneal dialysis therapy for at least 3 months. Peritoneal dialysis patients with episodes of peritonitis within 3 months prior to the study were not included. Of the entire group, 89% was taking diuretics and/or antihypertensive medications, and 33% was using β-blockers. The majority of the patients were under use of iron saccharate and renal-specific vitamin supplementation, and none was under use of corticosteroid or immunosuppressive drugs. Thirteen percent of the patients were taking calcitriol. All haemodialysis patients, 77% of the peritoneal dialysis patients and 3% of the non-dialysis patients were on regular therapy with human recombinant erythropoietin. Haemodialysis patients were dialysed for 4 h thrice a week, and the predominant vascular access was arteriovenous fistula (92% of the patients). A diet containing 30–35 kcal/kg/day and 0.6–0.8 g of protein/kg/day (non-dialysis patients) or 1.2–1.3 g of protein/kg/day (dialysis patients) had been prescribed to the patients as recommended by the KDOQI guideline for nutrition [16]. Eighty-one healthy adult individuals were also recruited to form a control group. They were clinic employees or relatives of the patients. All control subjects had normal renal and thyroid function, and none of them were taking any medication.

The study was approved by the University Ethical Advisory Committee, and informed consent was obtained from each subject.

Study design and protocol

In this cross-sectional study, the indirect calorimetry test, nutritional assessment and fasting blood tests were all performed on the same day. In haemodialysis patients, the indirect calorimetry and blood tests were carried out on an interdialytic day, and the nutritional assessment was performed post-dialysis session of the same week. Peritoneal dialysis patients underwent nutritional assessment and indirect calorimetry test with emptied peritoneal cavity. Two prediction equations using easily measurable variables were compared with the reference indirect calorimetry for the estimation of REE.

Nutritional evaluation

Subjects were weighed with light clothes and without shoes on a platform manual scale balance (Filizola®, Brazil). Weight was measured to the nearest 0.1 kg and height to the nearest 0.1 cm with a stadiometer. Body mass index (BMI) was calculated as body weight divided by squared height.

Body composition was assessed by bioelectrical impedance analysis using a single frequency tetrapolar technique with an electrical current of 800 μA at 50 kHz (BIA 101 Quantum, RJL Systems, USA). With the subject in the supine position, the electrodes were placed in the standard positions (two electrodes placed on the hand and wrist and another two positioned on the foot and ankle) on the right side of the body or in the opposite side of the vascular access for haemodialysis patients. The software Fluids & Nutrition (version 3.0) provided by the manufacturer was used to estimate body composition. Energy intake was assessed by means of 3-day food record and protein intake by calculating protein equivalent of nitrogen appearance (PNA) according to the KDOQI guideline for nutrition [16].

Laboratory data

Blood samples were drawn after an overnight fast of 12 h just before the indirect calorimetry test. Serum creatinine, urea and glucose were determined by a standard autoanalyser. Bicarbonate (normal range: 23–27 mmol/L) was measured by an automated potentiometer, thyroid-stimulating hormone (TSH, normal range: 0.3–4.0 μIU/L) by immunofluorometric assays and albumin (normal range: 3.4–4.8 g/dL) by bromresol green technique. Intact parathyroid hormone (PTH) (normal range: 10–65 pg/mL) and high-sensitivity assay for C-reactive protein (CRP) (inflammatory state: >0.5 mg/dL) were determined by immunochemiluminescence. Glomerular filtration rate was evaluated using standard creatinine clearance (CrCl) corrected for body surface area. Single-pool Kt/V was calculated for haemodialysis and peritoneal dialysis patients according to the KDOQI guidelines for dialysis (2006) [17].

REE

REE was measured by indirect calorimetry using an open circuit ventilated computerized metabolic system (Vmax series 29n; SensorMedics Corp, Yorba Linda, CA, USA). The oxygen and carbon dioxide sensors were calibrated before each REE measurement with the use of mixed reference gases of known composition. All subjects were previously instructed to refrain from any unusual physical activity (24 h prior) to the test and to sleep at the same time as usual in the night before the REE measurement. They were admitted to the clinic at 8:00 a.m. after an overnight fast of 12 h. After resting for 30 min in a recumbent position, subjects breathed for 30 min through a clear plastic canopy over their heads in a quiet dimly lit thermoneutral room. They were instructed to avoid hyperventilation, fidgeting or falling asleep during the test. Oxygen consumption and carbon dioxide production were measured at 1-min intervals, and the mean of the last 20 min was used to calculate the REE according to the following Weir’s equations, without using urinary urea nitrogen [18]:

\[
\text{Basal metabolic rate (kcal/min)} = 3.9 \times [\text{VO}_2(\text{L/min})] + 1.1 \times [\text{VO}_2(\text{L/min})]
\]

where \(\text{VO}_2\) is the volume of oxygen, and \(\text{VCO}_2\) is the volume of carbon dioxide. The intra-individual variation coefficient for REE by indirect calorimetry obtained in two consecutive occasions was 5%.

The equations used for the predicting REE are as follows:

(i) Harris and Benedict’s equations [12]:

\[
\begin{align*}
\text{Men} & = 66 + (13.7 \times \text{weight}) + (5 \times \text{height}) - (6.8 \times \text{age}) \\
\text{Women} & = 655 + (9.6 \times \text{weight}) + (1.7 \times \text{height}) - (4.7 \times \text{age})
\end{align*}
\]

(ii) Schofield’s equations reported by the World Health Organization [13]:

<table>
<thead>
<tr>
<th>Age Range</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>18–30 years</td>
<td>$15.057 \times \text{weight} + 692.2$</td>
<td>$14.818 \times \text{weight} + 486.6$</td>
</tr>
<tr>
<td>30–60 years</td>
<td>$11.472 \times \text{weight} + 873.1$</td>
<td>$8.126 \times \text{weight} + 845.6$</td>
</tr>
<tr>
<td>&gt;60 years</td>
<td>$11.711 \times \text{weight} + 587.7$</td>
<td>$9.082 \times \text{weight} + 658.5$</td>
</tr>
</tbody>
</table>
Results

Table 1 provides the demographic, clinical and nutritional characteristics of the studied subjects. CKD patients were older, had higher serum concentrations of glucose and C-reactive protein, and had reduced body fat and energy intake in comparison with the healthy control. The aetiology of CKD was undetermined in 30% of the patients—hypertensive nephrosclerosis accounted for 26% of the causes, followed by chronic glomerulonephritis (15%) polycystic kidney (10%) and others (19%). The duration on therapy was similar between haemodialysis patients (24 (2–162) months) and peritoneal dialysis patients (23 (3–109) months). Single-pool Kt/V was 1.3 ± 0.2 in haemodialysis patients and 2.2 ± 0.6 in peritoneal dialysis patients. Twenty-six patients (9%) had diabetes, 25% had inflammation (defined as CRP ≥ 1.0 mg/dL) and 20% had severe hyperparathyroidism (defined as PTH ≥ 700 pg/mL).

Comparisons of REE predicted by the equations with REE measured by indirect calorimetry are presented in Figure 1. As can be seen, Schofield’s equation exhibited higher REE [1492 ± 220 kcal/day (mean ± SD)] in relation to Harris and Benedict’s equation (1431 ± 214 kcal/day; P < 0.001), and both prediction equations showed higher REE in comparison with the indirect calorimetry (1352 ± 252 kcal/day; P < 0.001) in the CKD group. The same pattern was found among non-dialysis and haemodialysis patients, as well as among controls. In peritoneal dialysis patients, the Harris and Benedict’s equation exhibited similar REE in relation to the reference. The intraclass correlation of the REE measured by indirect calorimetry with that predicted by Harris and Benedict’s equation (r = 0.58; P < 0.001) was stronger than with that by Schofield’s equation (r = 0.48; P < 0.001) among patients. In the control group, the correlations between measured and predicted REE were similar (Harris and Benedict r = 0.65; P < 0.001 and Schofield r = 0.62; P < 0.001). REE by both prediction equations as well as by indirect calorimetry correlated negatively with age and positively with lean body mass, BMI, PNA, and energy intake in CKD patients. C-reactive protein correlated positively only with measured REE (r = 0.22; P < 0.001). The REE error (predicted minus measured) correlated inversely with serum glucose (Harris and Benedict r = −0.15; P = 0.01 and Schofield r = −0.17; P = 0.004), parathyroid hormone (Harris and Benedict r = −0.17; P = 0.004 and Schofield r = −0.14; P = 0.02) and C-reactive protein (Harris and Benedict r = −0.24; P < 0.001 and Schofield r = −0.22; P < 0.001). When the analyses were performed according to the presence of comorbidities, we found that in patients with diabetes, inflammation or severe hyperparathyroidism (n = 137), the REE estimated by the Harris and Benedict’s equation (1374 ± 263 kcal/day)

Table 1. Demographic, clinical and nutritional characteristics of the patients and controls.

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>CKD</th>
<th>Non-dialysis</th>
<th>Haemodialysis</th>
<th>Peritoneal dialysis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n = 81</td>
<td>n = 281</td>
<td>n = 124</td>
<td>n = 99</td>
<td>n = 58</td>
</tr>
<tr>
<td>Male</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>42.7 ± 12.6</td>
<td>50.1 ± 15.9**</td>
<td>54.2 ± 16.0**</td>
<td>44.2 ± 13.8</td>
<td>51.4 ± 16.2**</td>
</tr>
<tr>
<td>Serum creatinine (mg/dL)</td>
<td>0.87 ± 0.21</td>
<td>7.1 ± 4.6**</td>
<td>3.0 ± 1.6**</td>
<td>11.2 ± 3.3**</td>
<td>9.0 ± 3.3**</td>
</tr>
<tr>
<td>CrCl (mL/min/1.73 m²)</td>
<td>–</td>
<td>–</td>
<td>30.2 ± 14.3</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Proteinuria (g/day)</td>
<td>84.6 ± 9.7</td>
<td>96.1 ± 60.3*</td>
<td>90.9 ± 9.1**</td>
<td>89.5 ± 60.2</td>
<td>120.3 ± 103.3*</td>
</tr>
<tr>
<td>Serum glucose (mg/dL)</td>
<td>–</td>
<td>345 ± 413</td>
<td>220 ± 248</td>
<td>345 ± 404</td>
<td>603 ± 565</td>
</tr>
<tr>
<td>Serum albumin (g/dL)</td>
<td>–</td>
<td>4.0 ± 0.4</td>
<td>4.0 ± 0.4</td>
<td>4.1 ± 0.4</td>
<td>3.8 ± 0.5</td>
</tr>
<tr>
<td>C-reactive protein (mg/dL)</td>
<td>0.31 (0.01–2.48)</td>
<td>1.11 (0.01–21.5)**</td>
<td>0.68 (0.01–8.5)**</td>
<td>1.42 (0.01–21.5)**</td>
<td>1.51 (0.05–16.1)**</td>
</tr>
<tr>
<td>Body mass index (kg/m²)</td>
<td>25.2 ± 3.8</td>
<td>24.9 ± 4.3</td>
<td>26.1 ± 4.5</td>
<td>23.5 ± 3.7**</td>
<td>24.9 ± 3.8</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>28.6 ± 7.9</td>
<td>26.0 ± 10.1*</td>
<td>27.4 ± 10.7</td>
<td>24.4 ± 9.3**</td>
<td>25.6 ± 9.8*</td>
</tr>
<tr>
<td>Lean body mass (kg)</td>
<td>49.0 ± 10.4</td>
<td>48.9 ± 10.4</td>
<td>50.0 ± 10.7</td>
<td>48.2 ± 10.4</td>
<td>47.7 ± 10.0</td>
</tr>
<tr>
<td>nPNA (g/kg/day)</td>
<td>–</td>
<td>0.92 ± 0.23</td>
<td>0.94 ± 0.19</td>
<td>1.10 ± 0.22</td>
<td>0.71 ± 0.15</td>
</tr>
<tr>
<td>Energy intake (kcal/kg/day)</td>
<td>34.2 ± 7.1</td>
<td>28.1 ± 8.4**</td>
<td>25.8 ± 7.4**</td>
<td>28.8 ± 11.0</td>
<td>30.8 ± 5.9</td>
</tr>
</tbody>
</table>

CKD, chronic kidney disease; CrCl, creatinine clearance; nPNA, normalized protein equivalent of nitrogen appearance.

*P < 0.05, **P < 0.01 patients vs control, independent Student’s t-test or Mann–Whitney’s test.
was equivalent to the REE measured by indirect calorimetry (1402 ± 222 kcal/day; P = 0.13).

The agreement of the REE prediction equations with the indirect calorimetry is shown in Figure 2. There was a large limit of agreement between both prediction equations and the reference indirect calorimetry among non-dialysis patients (Figure 2A) and, particularly, among dialysis patients (Figure 2B and C). The individual variability of the prediction equations against the reference method was also evident in the control group (Figure 2D). Acceptable REE prediction, from 90% to 110%, was found in 47% of the patients by using the Harris and Benedict’s equation and in only 37% by using the Schofield’s equation (Figure 3). As can be seen, the overestimation of REE (adequacy >110%) by the predictive equations was highly prevalent in both CKD and control groups.

In order to estimate the energy requirement of the CKD patients, we calculated the total energy expenditure (TEE) as the REE multiplied by 1.55 (the mean physical activity factor for sedentary adults according to the World Health Organization). The results from the Harris and Benedict’s equation [36.9 (34.9–39.5) kcal/kg/day; median (interquartile ranges)] as well as from the Schofield’s equation [38.4 (36.0–41.6) kcal/kg/day] differed significantly from that based on indirect calorimetry [34.3 (31.4–38.2) kcal/kg/day; P < 0.001]. In addition, the analyses by quartiles of TEE exhibited kappa statistic of 0.15 between Harris and Benedict’s equation and indirect calorimetry, and 0.12 between Schofield’s and indirect calorimetry, indicating the lack of agreement between the predicted and measured methods for estimating energy requirement of these patients. By using Harris and Benedict’s equation, 64% of the patients and, by using Schofield’s equation, 66% of the patients did not meet the same quartile of TEE estimated by using indirect calorimetry.

Discussion

The present study demonstrated that the most frequently used REE prediction equations proposed by Harris and Benedict and by Schofield overestimated REE measurements in CKD patients. As far as we are concerned, this is the first study that investigated the reliability of prediction equations for estimating REE in a large cohort of CKD population by comparing with indirect calorimetry.

The knowledge of REE is essential for determining energy requirement since REE is the predominant component of TEE [19]. Due to the limitations of sophisticated calorimetric methods for evaluating REE in the clinical settings, simple prediction equations of REE are valuable to guide a correct dietary energy supply. Actually, a number of equations have been developed for such a purpose. Recently, when Wejs et al. performed a systematic search for publications of REE prediction equations for use in adults, they found a total of 18 equations developed based on gender, age, body weight and/or height. The authors observed that the equation proposed by Schofield had the smallest prediction error against indirect calorimetry, followed by the Harris and Benedict’s equation in outpatients as well as inpatients. However, in agreement with the present study, the errors of both equations were relatively large. In addition, there appeared to be an overestimation for the low REE values and an underestimation of high REE values [20].

Some reasons for the inaccuracy of the REE prediction equations can be discussed. Firstly, the error of such equations might be attributed to the fact that they were developed for estimating basal metabolic rate and not REE. Then, an underestimation of REE by the equations would be expected since basal metabolic rate is ~10–20% lower than the REE [19]. However, both equations overestimated REE against the reference indirect calorimetry in the present study (mean error by Harris and Benedict was 5.8% and by Schofield was 10.4%). Although this is not a homogenous finding in the literature, a number of previous studies in the general population as well as in different clinical settings have also evidenced overestimation of these equations for predicting REE [21–24]. In accordance, overestimation of REE by the equations was noticeable not only among non-dialysed and dialysed CKD patients but also in our healthy control group (7.9% by...
Harris and Benedict and 9.3% by Schofield). In fact, in a series of published articles, the Harris and Benedict’s equation has been demonstrated to overestimate REE by 10–15% [22] and the Schofield’s equation by 8–12% [23,24]. In CKD patients, REE might be reduced due to the diminished lean body mass as a result of a cluster of...
catabolic conditions to which these patients are commonly exposed. And, as known, lean body mass is the main determinant of the energy expenditure accounting for 73% of the variations in REE and 80% of the variations in TEE [25]. Thus, the use of total body weight by the prediction equations could be a potential source of overestimation of REE in these particular patients. This rationale is consistent with the findings that overestimation by such equations is systematically greater among obese individuals [26,27]. Another reason for the overestimation of the REE equations in CKD patients could be related to the lack of energy expended by the kidneys, since in healthy individuals, the kidneys are thought to account for ~7% of the resting energy expenditure [28]. However, the overestimation by the equations was noticeable also among healthy controls. Finally, a potential explanation for an inaccurate estimation of REE by the prediction equations in the present study can be the differences between the study population and the population from which the equations were originally derived. For instance, the equation developed by Harris and Benedict was based on a sample of young and healthy non-obese men and women and the equation by Schofield was derived mainly from Italian men with relatively high REE values. Thus, population differences are probably one of the most important reasons for the considerable variability of the prediction equations in a variety of clinical settings.

Previous studies using indirect calorimetry have documented the important role of the comorbidities such as diabetes, hyperparathyroidism and inflammation increasing significantly the REE of CKD patients [29–33]. Therefore, we cannot exclude the possibility that in such circumstances, an overestimation of REE by the prediction equation could be favourable for CKD patients by providing enough energy replacement to ensure their increased energy needs. Accordingly, in the present study, the REE error (predicted minus measured) by both equations correlated inversely with serum glucose, parathyroid hormone and C-reactive protein, suggesting that the higher the concentration of these markers, the lower the error of the REE prediction equations. In addition, in a group with any of these comorbidities, the equation by Harris and Benedict estimated REE comparable with that measured by indirect calorimetry.

In conclusion, this study showed that the main available prediction equations overestimated the REE of CKD patients. Overestimation was greatest with the equation proposed by Schofield followed by the one by Harris and Benedict. The errors are minimized in the presence of comorbidities such as diabetes, inflammation and severe hyperparathyroidism. Dietitians should be aware of the limitations of the REE equations when prescribing energy intake for CKD patients. Since the establishment of energy recommendation in CKD patients is still a matter of debate, to pursue a more thorough analysis of existing information, there is the need to promote further studies with geographic and ethnic representative sample in order to confirm our findings and also to develop CKD population-specific equations for estimating REE.

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Conflict of interest statement. None declared.

References

Fig. 3. Percentage of subjects according to the adequacy of REE [(REE predicted by the equations × 100) / REE measured by indirect calorimetry].
Estimated glomerular filtration rate in the nephrotic syndrome

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Abstract

Background. Plasma creatinine concentration and creatinine-based equations are most commonly used as markers of glomerular filtration rate (GFR). The abbreviated MDRD formula is considered the best available formula. Altered renal handling of creatinine, which may occur in the nephrotic syndrome, will invalidate creatinine-based formulas.

We have evaluated the abbreviated MDRD formula in a large cohort of patients with proteinuria.

Methods. Data on a cohort of patients with glomerular diseases were available from a large database. We have studied the relationship between estimated GFR (MDRD formula), and plasma cystatin C (CysC) and plasma beta-2-microglobulin (β2m) as markers of GFR.

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