Body cell mass: model development and validation at the cellular level of body composition

ZiMian Wang,1 Marie-Pierre St-Onge,1 Beatriz Lecumberri,1 F. Xavier Pi-Sunyer,1 Stanley Heshka,1 Jack Wang,1 Donald P. Kotler,1 Dympna Gallagher,1 Lucian Wielopolski,2 Richard N. Pierson Jr.,1 and Steven B. Heymsfield1

1Obesity Research Center, St. Luke’s-Roosevelt Hospital, Columbia University College of Physicians and Surgeons, New York 10025; and 2Department of Applied Science, Brookhaven National Laboratory, Upton, New York 11973

Submitted 20 May 2003; accepted in final form 13 September 2003

Wang, ZiMian, Marie-Pierre St-Onge, Beatriz Lecumberri, F. Xavier Pi-Sunyer, Stanley Heshka, Jack Wang, Donald P. Kotler, Dympna Gallagher, Lucian Wielopolski, Richard N. Pierson Jr., and Steven B. Heymsfield

Moore et al. (15) developed a BCM estimation formula of the general type

\[
BCM (kg) = k \times K_e (\text{mmol})
\]  

(1)

where \(K_e\) is total body exchangeable potassium measured by \(^{42}\text{K}\), and \(k\) (in kg/mmold) is the reciprocal of the potassium content of BCM. This model assumes that there is negligible extracellular fluid (ECF) potassium, all exchangeable potassium is within cells, and that the average cell potassium content is constant. Moore et al. then used available data to fit their model: an average nitrogen (N) content of BCM (i.e., 0.04 g N per g BCM) and an average K-to-N ratio of 3 mmol/g. A BCM prediction model was then derived as BCM (g) = N/0.04 = (K_e/3)/0.04 = 8.33 \times K_e (\text{mmol}). Assuming \(K_e\) is equivalent to total body potassium (TBK), BCM can also be expressed as

\[
BCM (kg) = 0.00833 \times TBK (\text{mmol})
\]  

(2)

According to the Moore et al. formula, the ratio of TBK to BCM is 1/0.00833, or 120 mmol/kg. The use of \(^{42}\text{K}\) to estimate TBK has two limitations. First, the half-life of \(^{42}\text{K}\) is short (12.4 h), and the radiation exposure is relatively high, making the method impractical for large-scale studies of healthy subjects of all ages. Second, \(^{42}\text{K}\) underestimates TBK by 3–10% (7).

Cohn et al. (4) were the first investigators to experimentally estimate BCM independent of TBK. BCM was derived by application of early fat-free mass (FFM) models on the molecular and cellular body composition levels that incorporated several measured components, including total body nitrogen (TBN), total body water (TBW), total body calcium, and total body chlorine. TBK was measured by whole body \(^{40}\text{K}\) counting. The study by Cohn et al. provided an experiment-based BCM prediction equation

\[
BCM (kg) = 0.0092 \times TBK (\text{mmol})
\]  

(3)

According to this model, the BCM potassium content of BCM is 1/0.0092, or 108.7 mmol/kg. BCM estimates by the models of Moore et al. (15) (Eq. 2) and Cohn et al. (4) (Eq. 3) thus differ by 11% on the basis of their respective \(k\) values (i.e., 0.0083 vs. 0.0092 kg/mmold).

The experimental approach of Cohn et al. (4) has several inherent limitations that are now recognized. Their model did not include several small components, such as soft-tissue minerals (Ms) and glycogen. Cohn et al. also lacked new...
measurement methods; for example, bone mineral (Mo) and ECF were predicted from total body calcium, and total body chlorine was measured by delayed-γ neutron activation analysis, a method unavailable to most investigators (4, 23). However, we can now estimate Mo and ECF by widely available dual-energy X-ray absorptiometry (DEXA) and NaBr dilution methods, respectively.

These new models and methods allowed us in the present report to develop an improved BCM evaluation method. The first aim of the current investigation was to improve the BCM prediction approach of Cohn et al. (4) and, subsequently, to apply experimental data to provide an updated estimate of k as defined by Eq. 1.

\[
\text{BCM} = (\text{TBW} + \text{TBPro} + \text{Mo} + \text{Ms} + \text{glycogen}) - (\text{ECF} + \text{ECS}) \\
= \text{TBW} + 6.25 \times \text{TNB} + \text{Mo} + 0.0129 \times \text{TBW} + 0.275 \times \text{TNB} - 1.02 \times \text{ECW} - 1.732 \times \text{Mo} \\
= 1.0129 \times \text{TBW} + 6.525 \times \text{TNB} - 1.02 \times \text{ECW} - 0.732 \times \text{Mo} 
\]

The classic BCM model of Moore et al. (15) was based on rough body composition knowledge available in the 1960s. Since then, body composition models have greatly improved, and thus the quantity and quality of information available to fit models have also increased. The second aim of the present study was to develop a theoretical BCM prediction model by use of new modeling approaches and then to fit the model with available reference data. This theoretical model was designed to critically evaluate factors influencing BCM prediction and also to provide an updated k value for Eq. 1 on the basis of recent body composition information.

METHODS

Study design. In the first phase, we developed an improved TBK-independent BCM prediction model on the basis of the earlier model of Cohn et al. (4). The development of this model is presented in METHODS. Available data were then used to derive an estimate of TBK/BCM (i.e., 1/k) in men and women, and we also examined the age dependence of the TBK/BCM ratio.

A theoretical TBK-BCM model was next developed at the cellular level of body composition. The development of this model is presented in RESULTS. Available reference body composition data were then used to derive an estimate of the mean for the ratio of TBK to BCM. The TBK/BCM ratio was then used to derive a value for k as defined in Eq. 1.

The model-derived k values were then compared with the corresponding k values derived by Moore et al. (15) and Cohn et al. (4).

Improved Cohn BCM prediction model. Cohn et al. (4) expressed FFM as the sum of TBW, total body protein (TBPro), and Mo, on the molecular level, and as the sum of BCM, ECF, and extracellular solids (ECS) on the cellular level, respectively

\[
\text{FFM} = \text{TBW} + \text{TBPro} + \text{Mo} \\
\text{FFM} = \text{BCM} + \text{ECF} + \text{ECS} 
\]

Cohn’s BCM model was thus derived from Eqs. 4 and 5

\[
\text{BCM} = (\text{TBW} + \text{TBPro} + \text{Mo}) - (\text{ECF} + \text{ECS}) 
\]

With recent developments in body composition models and techniques, we are now able to expand Cohn’s model. At the molecular level, FFM represents the sum of TBW, TBPro, Mo, Ms, and glycogen (19), such that

\[
\text{FFM} = \text{TBW} + \text{TBPro} + \text{Mo} + \text{Ms} + \text{glycogen} 
\]

where the units of all components are in kilograms. Equation 8 thus expands the BCM model of Cohn et al. (Eq. 6).

Subjects. Healthy subjects (≥20 yr) were recruited from hospital staff and local residents. To be included in the study, subjects had to be ambulatory and have no orthopedic problems or physical handicaps. Each subject completed a medical examination, including medical history, physical examination, and routine blood studies to exclude the presence of underlying diseases. All study participants signed an informed consent. The investigation was approved by St. Luke’s-Roosevelt Hospital’s Institutional Review Board.

Body composition measurements. Each subject completed five studies: DEXA for Mo, \(^3\)H\(_2\)O or \(^2\)H\(_2\)O dilution for TBW, whole body \(^40\)K counting for TBK, prompt-γ IVNA for TBN, and NaBr dilution for ECW. This information was used to solve for TBK/BCM with the improved Cohn model.

Subjects were studied after an overnight fast. Body mass was measured to the nearest 0.1 kg with a digital scale (Weight Tronix, New York) and height to the nearest 0.5 cm with a wall-mounted stadiometer (Holstein, Crosswell, UK).

TBK was measured by whole body \(^40\)K counting at the Body Composition Unit of St. Luke’s-Roosevelt Hospital. The technical error for repeated phantom \(^40\)K counting in our laboratory is 2.4% (8).

Tritium space (\(^3\)H\(_2\)O, in liters) or deuterium space (\(^2\)H\(_2\)O, in liters) was measured with a coefficient of variation (CV) of 1.5 and 1.2%, respectively. The water dilution space was then converted into TBW mass (in kg) by correcting for nonaqueous hydrogen exchange and water density at 36°C (TBW = dilution space × 0.96 ± 0.994) (16).

TBN was quantified using the prompt-γ IVNA facilities at Brookhaven National Laboratory, with a CV of 2.7% as reported by Dilmian et al. (6).

Subjects were scanned with a whole body DEXA system (Lunar DPX, software version 3.6; Madison, WI). Bone mineral content measured by DEXA represents ashed bone. One gram of bone mineral produces 0.9582 g of ash due to loss of labile components including bound water and CO\(_2\) with combustion. BMC was thus converted to Mo as Mo = BMC/0.9582 (10). The DEXA system CV is 1.3% for Mo (9).

Sodium bromide dilution space (in liters) was measured with a CV of 1.4%. The dilution space was then converted into ECW (in kg) by correcting for the weight fraction of water in plasma (0.94), the Gibbs-Donnan effect (0.95), and the penetration of bromide into the intracellular space of erythrocytes (0.90) (16).

FFM was calculated from TBW, TBN, and Mo according to Eq. 7.
Statistical analysis. Results are expressed as the group mean ± SD, with P < 0.05 considered statistically significant. Statistical comparisons of TBK/BCM ratios between men and women and BCM predictions by various models were made by Student’s t-test. Simple linear regression analysis was applied to describe the relationships between BCM estimates by the various developed models. The differences in BCM between models were examined using Bland-Altman analysis (2). Statistical calculations were carried out using Microsoft Excel for Windows.

RESULTS

Cellular level model TBK/BCM estimates. Because almost all body potassium exists in intracellular fluid (ICF) and ECF, TBK is equal to the sum of potassium in ICF (K in ICF) and potassium in ECF (K in ECF). The ratio of TBK to BCM can be expressed as

\[ \frac{\text{TBK}}{\text{BCM}} = \frac{(\text{K in ICF}) + (\text{K in ECF})}{\text{BCM}} \]  (9)

BCM can be expressed as a function of intracellular water (ICW), BCM = ICW/\( a \), where \( a \) is the fraction of BCM as ICW. The K in ICF can be expressed as a product of ICW and the potassium concentration in ICF ([K]ICW), K in ICF = ICW \times [K]ICW. Similarly, K in ECF is a product of ECW and the potassium concentration in ECW ([K]ECW), K in ECF = ECW \times [K]ECW. In addition, ECW can be expressed as a function of ICW, ECW = (E/I) \times ICW, where E/I is the ratio of ECW to ICW. Equation 9 can be converted to

\[ \frac{\text{TBK}}{\text{BCM}} = \frac{[\text{K}]_{\text{ICW}} \times \text{ICW} + [\text{K}]_{\text{ECW}} \times \text{ECW} \times (E/I)}{\text{ICW}/a} \]  (10)

Equation 10 reveals that TBK/BCM can be calculated from four determinants: the BCM fraction as ICW (\( a \)), the potassium concentration in ICF ([K]ICW), the potassium concentration in ECW ([K]ECW), and the ratio of ECW to ICW (E/I). The physiological aspects and mean magnitudes of the four determinants have been discussed previously (13, 18): \( a = 0.70 \), [K]ICW = 152 mmol/kgH₂O, [K]ECW = 4 mmol/kgH₂O, and E/I = 0.97. The mean TBK/BCM ratio (109.1 mmol/kg) can therefore be predicted for healthy adults according to Eq. 10.

Table 1. Physical characteristics and body composition of the 112 healthy subjects

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Mean ± SD</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>43.0±10.6</td>
<td>22–74</td>
</tr>
<tr>
<td>Body mass, kg</td>
<td>73.8±15.0</td>
<td>42.1–105.7</td>
</tr>
<tr>
<td>Ht, m</td>
<td>1.64±0.08</td>
<td>1.42–1.87</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>27.4±5.2</td>
<td>16.8–34.7</td>
</tr>
<tr>
<td>TBK, mmol</td>
<td>2,690±487</td>
<td>1,672–4,365</td>
</tr>
<tr>
<td>TBN, kg</td>
<td>1.4±0.26</td>
<td>0.94–2.15</td>
</tr>
<tr>
<td>TBW, kg</td>
<td>33.3±5.3</td>
<td>22.4–47.6</td>
</tr>
<tr>
<td>Mo, kg</td>
<td>2.56±0.42</td>
<td>1.25–3.48</td>
</tr>
<tr>
<td>ECW, kg</td>
<td>16.2±2.9</td>
<td>10.3–25.6</td>
</tr>
<tr>
<td>FFM, kg</td>
<td>45.5±6.4</td>
<td>32.5–64.7</td>
</tr>
<tr>
<td>BCM, kg</td>
<td>24.7±4.1</td>
<td>17.2–39.0</td>
</tr>
</tbody>
</table>

BCM, body cell mass measured by the improved evaluation method (Eq. 9); BMI, body mass index; ECW, extracellular water; FFM, fat-free mass; Ht, height; Mo, bone mineral; TBK, total body potassium; TBN, total body nitrogen; TBW, total body water.

In addition, the E/I ratio is equal to (152 × TBW – TBK)/(TBK – 4 × TBW). Equation 11 can be simplified by using the above reference values for each determinant, and a BCM prediction formula is thus derived

\[ \frac{\text{TBK}}{\text{BCM}} = \frac{0.70 \times [(152 + 4) \times (TBW – TBK)/(TBK – 4 \times TBW)]}{(TBK – 4 \times TBW)} \]  (12)

where BCM and TBW are in kilograms, and TBK in millimoles.

We further simplified this model into a TBK-only version. Because the values of \( a \), [K]ICW, and [K]ECW (i.e., 0.70, 152 mmol/kgH₂O, and 4 mmol/kgH₂O, respectively) can be as-

![Fig. 1. Body cell mass (BCM, in kg), measured by the approach of Cohn et al. (4), improved here, on the ordinate and total body potassium (TBK), measured by whole body counting, on the abscissa. BCM (kg) = 0.0071 × TBK (mmol) + 5.75; \( r = 0.84 \), P < 0.001; SEE = 2.28 kg; \( n = 112 \) healthy subjects.](http://ajpendo.physiology.org/)

![Fig. 2. TBK/BCM ratio (in mmol/kg) on the ordinate vs. age (in yr) on the abscissa. TBK/BCM = 110.7 – 0.039 × age; \( r = –0.037 \), P > 0.05; \( n = 112 \) healthy subjects.](http://ajpendo.physiology.org/)
identity is shown.

5.5 showed that TBK is the strongest predictor of BCM (P = 0.001; SEE = 2.24 kg; n = 112 healthy subjects. Line of identity is shown.

sumed to be highly stable across subjects, Eq. 10 can be simplified to

\[
\text{TBK/BCM} = 106.4 + 2.8 \times (E/I)
\]  

(13)

A feature of Eq. 13 is that changes in water distribution (i.e., E/I) have only a small effect on the magnitude of the TBK/BCM ratio. When the E/I ratio increases by 50% (e.g., from 0.80 to 1.20), the corresponding TBK/BCM ratio increases by only 1.1% (e.g., from 108.6 to 109.8 mmol/kg). Hence, although the E/I ratio is variable across subjects, the impact of this variability on the TBK/BCM ratio is small. If we assume that the mean value of the E/I ratio for the healthy adult human is 0.95 (18), Eq. 13 can be further simplified to

\[
\text{TBK/BCM} = 109.1 \text{ mmol/kg}
\]  

or

\[
\text{BCM (kg)} = 0.0092 \times \text{TBK (mmol)}
\]  

(14)

Improved Cohn model estimates of TBK/BCM. A total of 112 healthy subjects, 14 men and 98 women, ranging in age from 22 to 74 yr, in body mass from 42.1 to 105.7 kg, and in body mass index (BMI) from 16.8 to 34.7 kg/m2, were evaluated (Table 1).

The measured BCM and TBK estimates were highly correlated (Fig. 1), and the corresponding average BCM/TBK ratio is 0.00918 ± 0.00091 kg/mmol. Multiple regression analysis showed that TBK is the strongest predictor of BCM (P = 5.5 × 10^-22). Age did not influence the prediction of BCM, whereas BMI had a significant negative influence on the prediction of BCM (P = 0.0025): BCM (kg) = 0.00723 × TBK - 0.125 × BMI + 8.72; r = 0.85, P < 0.001; standard error of the estimate (SEE) = 2.2 kg.

The measured TBK/BCM ratio was 109.0 ± 10.9 mmol/kg. There was no significant sex difference in the TBK/BCM ratio (109.4 ± 6.9 mmol/kg for men vs. 108.9 ± 11.3 mmol/kg for women, P > 0.05), and there was also no significant effect of age on the TBK/BCM ratio (Fig. 2).

\[
\text{TBK/BCM} = 110.7 - 0.039 \times \text{age};
\]  

\[
r = -0.037, P > 0.05
\]  

(15)

Model comparisons. BCM measured with the improved Cohn model (Eq. 8) and BCM estimates by the TBK-TBW (Eq. 12) and simplified TBK (Eq. 14) models were highly correlated (both r = 0.84, P < 0.001; Fig. 3). BCM measured by the improved Cohn model was 24.7 ± 4.1 kg (Table 2). BCM predicted by both the TBK-TBW and simplified TBK models was 24.7 ± 4.5 kg. The three BCM estimates did not differ significantly from each other (P > 0.05).

Compared with the improved Cohn model, BCM predicted by the Moore model (22.4 ± 4.1 kg) underestimated BCM by 2.3 ± 2.4 kg (P < 0.001) (Table 2).

Bland-Altman analysis indicated that the differences between BCM estimates by the improved Cohn model and by the three other models were not significantly associated with the mean BCM estimates (r = -0.15 for the TBK-TBW model, -0.17 for the simplified TBK model, and 0.03 for Moore model, all P > 0.05) (Table 3).

**DISCUSSION**

Although custom has enshrined the k value of 0.0833 kg/mmol to predict BCM, Moore and Boyden (14) suggested that 0.0833 kg/mmol is only approximate and that the true BCM/TBK value probably lies between 0.0075 and 0.0095 kg/mmol. In the present study, we critically reviewed and refined earlier approaches for estimating BCM. Our findings show close concord among three approaches for estimating the central BCM prediction model component k [i.e., 1/(TBK/BCM)] value: 0.00918 ± 0.00091 kg/mmol for experimental data, combined with an improved version of Cohn’s BCM

**Table 2. Body cell mass assessed by 4 BCM models**

<table>
<thead>
<tr>
<th>Method</th>
<th>Equation</th>
<th>Mean ± SD</th>
<th>Ratio*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Improved Cohn</td>
<td>BCM=1.0129×TBW+6.525×TBW-1.02×ECW-0.732×Mo</td>
<td>24.7±4.1</td>
<td>1.000±0.101</td>
</tr>
<tr>
<td>TBK-TBW</td>
<td>BCM=0.00965×TBK-0.0386×TBW</td>
<td>24.7±4.5</td>
<td>1.003±0.100</td>
</tr>
<tr>
<td>Simplified TBK</td>
<td>BCM=0.0092×TBK</td>
<td>24.7±4.5</td>
<td>1.003±0.100</td>
</tr>
<tr>
<td>Moore</td>
<td>BCM=0.00833×TBK</td>
<td>22.4±4.1†</td>
<td>0.908±0.090</td>
</tr>
</tbody>
</table>

*Ratio of BCM estimated by evaluated model to BCM measured by the improved Cohn evaluation method. †P < 0.001 by paired Student’s t-test for comparisons vs. BCM measured by the improved Cohn evaluation method.

**Table 3. Intermethod comparison of the improved Cohn evaluation method and 3 other methods by Bland-Altman analysis**

<table>
<thead>
<tr>
<th>Model</th>
<th>Mean Δ</th>
<th>SD</th>
<th>r</th>
</tr>
</thead>
<tbody>
<tr>
<td>TBK-TBW</td>
<td>0.06</td>
<td>2.53</td>
<td>−0.17</td>
</tr>
<tr>
<td>Simplified TBK</td>
<td>−0.01</td>
<td>2.49</td>
<td>−0.15</td>
</tr>
<tr>
<td>Moore</td>
<td>2.33</td>
<td>2.35</td>
<td>0.03</td>
</tr>
</tbody>
</table>

Δ, Mean difference (in kg) between the improved Cohn evaluation BCM method and the evaluated model; r, Bland-Altman analysis correlation coefficient (i.e., the difference between BCM estimates by the improved Cohn evaluation method and evaluated model vs. the mean of the two BCM estimates); SD, standard deviation.
estimation model (0.0092 kg/mmol) for a theoretical TBK-TBW approach fitted with available reference data; and 0.0092 kg/mmol for a simplified version of the theoretical model. In contrast, the $k$ value in the Moore model (0.00833 kg/mmol) is substantially lower, leading to an underestimation of BCM.

**Improved Cohn BCM prediction method.** The improved BCM approach is based on a limited number of model assumptions that are formulated on body composition principles. The model error of the improved BCM approach is thus small. However, the improved BCM prediction model (Eq. 8) has measurement error resulting from TBW, TBN, ECW, and Mo estimates. The measurement error caused by any one of these estimates can be evaluated in healthy subjects by assuming an average body composition, as shown in Table 1, and measurement precisions as described in methods

\[
(\sigma_{\text{BCM}})^2 = (1.0129 \times 33.3 \times 0.015)^2 + (6.525 \times 1.42 \times 0.027)^2
+ (1.02 \times 16.0 \times 0.014)^2 + (0.732 \times 2.56 \times 0.013)^2
= 0.2560 + 0.0626 + 0.0522 + 0.0244 = 0.3952
\]

This calculation reveals that the TBW estimate is the major source of measurement error. The propagated BCM measurement error ($\sigma_{\text{BCM}}$) for the improved model is 0.63 kg for healthy subjects.

**TBK-dependent models.** Although Moore’s model is the first suggested BCM estimation approach and is still widely applied, there remain fundamental questions related to this classic model. First, Moore’s model assumes a stable K/N ratio of 3 mmol/g. However, individual tissues and organs vary widely in their K/N ratios, from a low of 0.45 mmol/g in skin to a high of 5 mmol/g in the brain (22). The corresponding whole body K/N ratio is $\sim2.0$ mmol/g (i.e., 3.580 mmol/1,800 g) in Reference Man (17). Second, Moore’s model assumes a BCM potassium concentration of 1/0.0083, or 120 mmol/kg BCM. If we assume a mean cell water content of 70% (1), this yields an ICF [K] of 171 mmol/kgH2O, well above the commonly accepted average of 152 mmol/kgH2O (13). Third, Moore’s model ignores the potassium content of ECF, and this assumption may cause a small error in BCM estimates.

In the present study, we examined two TBK-related strategies for predicting BCM, one based on a TBK-TBW model and the other based on a TBK/BCM ratio. Both approaches appear to provide good means of predicting BCM in healthy subjects. For the TBK-TBW model (Eq. 12), the measurement error caused by both TBK and TBW assessments can be evaluated by assuming a mean subject body composition, as shown in Table 1, and TBK and TBW measurement precisions of 2.4 and 1.5%, respectively

\[
(\sigma_{\text{BCM}})^2 = (0.00965 \times 2.690 \times 0.024)^2 + (0.0386 \times 33.3 \times 0.015)^2
= 0.3881 + 0.0193 = 0.4074
\]

This calculation shows that the TBK estimate is the major source of measurement error in the TBK-TBW model. The BCM measurement error ($\sigma_{\text{BCM}}$) in the TBK-TBW approach is 0.64 kg in healthy subjects.

**TBK assessment is the only source of measurement error in the simplified TBK model (Eq. 14).** The error caused by TBK assessment can be calculated as

\[
\sigma_{\text{BCM}} = 0.0092 \times 2.690 \times 0.024 = 0.59 \text{ kg}
\]

Therefore, the measurement errors of the two TBK-dependent models (0.64 kg for the TBK-TBW model and 0.59 kg for the simplified TBK model) are comparable to the error of the improved Cohn BCM evaluation method (0.63 kg).

The model-derived TBK/BCM ratio (109.1 mmol/kg) is almost identical to the measured TBK/BCM value (109.0 ± 10.9 mmol/kg). Our observations indicate that the TBK/BCM ratio is relatively stable by sex across groups. The measured $E/I$ ratio in our earlier study was 0.82 ± 0.16 for adult men and 1.07 ± 0.22 for adult women (18). According to Eq. 13, the model-predicted TBK/BCM ratio is 108.7 mmol/kg for adult men and 109.4 mmol/kg for adult women, respectively. Therefore, the influence of sex on the TBK/BCM ratio is small. Accordingly, the present study does not support a significant difference in the measured TBK/BCM ratio between healthy adult men and women (109.4 ± 6.9 vs. 108.9 ± 11.3 mmol/kg, $P > 0.05$).

However, the simplified TBK model may include considerable model error. The suggested constancy of the TBK/BCM ratio (109.1 mmol/kg) is based on assumptions that all four determinants are stable, $a = 0.70$, $[\text{K}]_{\text{ICW}} = 152$ mmol/kg, $[\text{K}]_{\text{ECW}} = 4$ mmol/kg, and $E/I = 0.95$. Although these assumptions are necessary for deriving the TBK-only prediction method, any variations in the four determinants across subjects may cause a corresponding model error for the simplified TBK method. Future validation studies are needed to evaluate the relationship between TBK and BCM in populations outside of healthy adults, including children and patients with underlying diseases.

**Conclusion.** On the basis of the cellular-level body composition model, a BCM prediction equation was derived from TBK and TBW measurements. The model-derived magnitude of the TBK/BCM ratio was 109.1 mmol/kg, identical to the value measured in healthy subjects with an improved version of Cohn’s BCM prediction model (109.0 ± 10.9 mmol/kg). A simplified model was thus suggested as BCM (kg) = 0.0092 × TBK (mmol).

**GRANTS**

This study was supported by National Institute of Diabetes and Digestive and Kidney Diseases Grant DK-42618.

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