Relative Contribution of Organs Other Than Brain to Resting Energy Expenditure Is Consistent among Male Power Athletes

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Summary We have previously shown that resting energy expenditure (REE) adjusted by fat-free mass (FFM) in male college athletes remains consistent regardless of FFM. The FFM comprises internal organs with high metabolic activity, such as liver and brain, which account for 60 to 80% of REE in adults. The purpose of the present study is to examine the contribution of internal organs to the REE of the FFM fraction among male power athletes. The study included 37 American male college football players. REE was measured by indirect calorimetry and body composition was measured by dual energy X-ray absorptiometry (DXA). Mass of brain, liver, and kidneys was measured by MRI and mass of heart was estimated by echocardiography. Normal levels of thyroid hormone (triiodothyronine: T3) were confirmed in all subjects prior to the analysis. Multiple regression analysis was used to assess the influence of FFM, fat mass (FM), T3, and mass of organs on variance of REE. Average body weight and FFM were 81.2±11.3 kg and 67.7±7.4 kg, respectively. The relative contributions of liver, kidneys, and heart to REE were consistent regardless of FFM. While the REE of brain was negatively correlated with FFM (r=−0.672, p<0.001). Only FFM and T3 were found to be independent factors influencing REE. These results suggest that a steady contribution of internal organs other than the brain is the major reason for the consistency of the REE/FFM ratio in male power athletes.

Key Words resting energy expenditure, metabolic rate, athletes, fat-free mass, organ mass

Resting energy expenditure (REE) is a basis for estimating energy requirement, and it is an important physiological measurement for athletes. The estimated energy requirement can be predicted by multiplying REE by physical activity level and utilized to determine individual energy needs to provide the energy expended during athletic training and performance. If necessary, REE can also be used to promote body weight gain or reduction, allowing athletes to achieve an ideal body size and composition, since these parameters directly influence performance in a majority of sports. An accurate estimate of REE is fundamental to predicting energy requirement, and this is a significant matter in order for athletes to exploit their maximal ability.

Fat-free mass (FFM) is the sum of a variety of tissue and organs with both high and low levels of metabolic activity (1). Previous studies have shown that FFM accounts for 60 to 80% of the variance in REE in non-athletic populations (2–4). This also seems to be also true of female athletes (5) or women with high physical fitness (6). Typically, athletes will have relatively large FFM compared to non-athletic individuals. Therefore, for athletes, the Japan Institute of Sports Science (JISS) and other study have recommended that REE be estimated by FFM multiplied by the REE to FFM ratio (REE/FFM) (7, 8). In order to use FFM to estimate REE for athletes, another assumption that should be confirmed is that the REE/FFM ratio should be constant regardless of individual variations in FFM. Otherwise, there will be tendencies to over- or under-estimate REE among athletes depending on the value of FFM. In fact, there are numerous studies with non-athletic populations, mostly including the young to elderly and both males and females with wide-ranging FFM values that have reported that the REE/FFM ratio decreases with increasing FFM (1, 9–12). In addition, the cause for the reductions in REE/FFM ratios appears to be a reduced contribution of the components of FFM with high metabolic activity, such as internal organs, as the FFM increases (2).

Our previous study demonstrated the consistency of the REE/FFM ratio in male college athletes (13). FFM values were divided into 3 components, bone mass, skeletal muscle, and residual mass, with specific metabolic rates previously published by Heymsfield et al. (2) used to investigate the relative contributions of these 3 components of FFM. The results suggested that the percent contribution of each component to FFM did not change regardless of different FFM values.

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The main component of FFM is skeletal muscle, which is about 40 to 60% of body weight according to previous studies (1,3). However, skeletal muscle accounted for only 20 to 30% of REE because the metabolic rate of skeletal muscle is 13 kcal·kg$^{-1}$·d$^{-1}$, whereas the metabolic rates of liver, brain, heart, and kidney mass were determined to be 200, 240, 440, and 440 kcal·kg$^{-1}$·d$^{-1}$ (1). Thus, these 4 organs account for 60 to 70% of REE in adults, even though their combined weight is less than 6% of total body weight (1). As a consequence, small relative differences in fractional contributions of organ mass to FFM may significantly influence REE/FFM ratios. Moreover, one of the distinct characteristics of power athletes is that their attempts to build an ideal body composition are focused particularly on building skeletal muscle by a combination of diet and daily physical training. Therefore, it is not assured that relationship between FFM and detailed body composition including internal organs is the same in power athletes as in non-athletes.

The purpose of this study was to examine the contribution of the internal organs to the REE/FFM ratio among male power athletes. We hypothesized that the reason athletes maintain a constant REE/FFM ratio regardless of FFM is that the relative contribution of the internal organs to the FFM remained unchanged, which proved true for all of the organs of interest, except the brain.

METHODS

Subjects. The study included 37 male American football players from a National Collegiate Athletic Association Division 1 team. The subjects played in a variety of different positions in the sport and heavily trained for speed and agility as well as muscle strength and power, which are the characteristic of power-type athletes. The energy and food intake were assumed to be adequate for all the subjects due to the intentional weight gain to increase muscle mass. All of the subjects had participated in some type of sports team since they were in elementary school; hence they were very active from a young age according to the exercise history survey. The measurements were taken during their off-season to avoid any physical and/or mental exhaustion since the amount of training in the off-season is relatively low compared to the on-season. All of them voluntarily agreed to participate in the study. None had a history of cardiovascular, endocrine, or orthopedic disorders, nor had any of them been taking any medication when the measurements were taken. Each subject was fully informed about the research study by verbal and written descriptions, each gave informed consent before testing, and the study was approved by the Human Research Committee of Waseda University for use of human subjects in accordance with the Declaration of Helsinki.

Body composition. Body weight (BW) was measured to the nearest 0.1 kg by bioelectrical impedance analysis (Inner Scan BC-660, Tanita Co., Tokyo, Japan) and standing height was measured to the nearest 0.1 cm with a stadiometer (YL-65, Yamagi, Inc., Nagoya, Japan) with subjects wearing minimal clothing and no shoes. Body mass index (BMI) (kg/m$^2$) was calculated by dividing BW by the square of standing height. Dual-energy X-ray absorptiometry (DXA) (Hologic QDR-4500, DXA Scanner, Hologic Inc., Waltham, MA) was used to measure the appendicular lean soft tissue mass (ALST) (g), and % body fat. The subjects were asked to wear loose-fitting light clothes without any metal objects and were in a supine position on the scanning table during the total body scan. FFM and fat mass (FM) were then calculated based on BW and % body fat.

Skeletal muscle (SM) mass was estimated using the sum of appendicular lean soft tissues with age in a prediction model established by Kim et al. (14) as follows: SM (kg) = 1.13×ALST (kg)−0.02×age (y)+(0.61×sex: male=1)+0.97. Adipose tissue is specialized loose connective tissue which contains not only fat but also fat-free components such as protein, minerals and water, and the FM calculation was performed with the assumption that 85% of the adipose tissue was considered as FM calculated from the percent body fat. Thus, the remaining 15% of the adipose tissue included the remaining calculated fat-free component (FM/adipose tissue=0.85) (2). Hence, adipose tissue can be calculated as follows: adipose tissue (kg)=FM (kg)×1.18.

Organ mass. The volumes of liver, brain, and kidneys were measured using magnetic resonance imaging (MRI) (Signa 1.5T; General Electric Co., Milwaukee, WI). The images were taken by a T1-weighted spin-echo and axial-plane sequence with 10 mm slice thickness, 500 ms repetition time, and 13.1 ms echo time. Brain images for some of 1st-year players were measured using a T1-weighted spin-echo with head coil, while the method used in others measured the anterior commissure-posterior commissure plane sequence with a slice thickness of 5 mm, 1.5 mm inter-gap, a repetition time of 500 ms, and an echo time of 14 ms. The coefficient of variation was 0.2% between the 2 brain scanning techniques. The subjects were in the supine position with the hands placed on the abdomen and the legs extended during both MRI techniques. During the scans of the trunk region, in order to minimize blur in the images, the subjects were asked to inhale and hold their breath for about 28 s and to breathe when prompted by an announcement. The MRI cross-sectional images of the liver, kidneys, and brain were analyzed using image analysis software (Slice-o-matic; Tomovision, Montreal, QC), and automatic segmentation of an image was performed using watersheds of the gradient magnitude using the Mathematical Morphology (“Morpho”) mode. Cross-sectional areas (cm$^2$) were determined after assigning areas of interest different color codes for the tissues to be analyzed. The volume of each organ was determined from the sum of its cross-sectional areas multiplied by the 1 cm slice thickness. Volumes (cm$^3$) of the organs were then converted to mass in kg using densities of 1.060 kg/cm$^3$ for liver, 1.036 kg/cm$^3$ for brain, and 1.050 kg/cm$^3$ for kidneys, which were reported previously (15). All analyses were performed by the same investigator to minimize analysts’ variation, and the intra-observer coefficients of variation were 5.2% for...
liver, 0.6% for brain, and 5.5% for kidneys.

Left ventricular mass (LV mass) was measured using echocardiography (Titan, SonoSite, WA) with 2.8 MHz probe (C15/4-2 MHz, SonoSite). Subjects were asked to lie in a partial left decubitus or supine position during the measurement. At least 5 M-mode end-diastolic phase images of the left ventricle in the parasternal long axis view were captured for the measurements. The dimensions and the wall thickness were evaluated at or below the tips of the mitral valve leaflets. The LV mass was calculated using the 2-dimensional linear formula suggested by the American Society of Echocardiography as follows: LV mass=0.8×(1.04 [(LV internal dimension at end diastole+LV wall thickness at the inferolateral walls+LV wall thickness at the cardiace base for the anteroseptum)3−(LV internal dimension at end diastole)]+0.6 g (16, 17). This was multiplied by a factor of 1.50 to obtain a mass of total heart (18). The intra-observer coefficient of variation for LV mass was 5.2%.

Measured REE. REE was measured by open-circuit indirect calorimetry using a Douglas bag. Subjects came to the testing facility in the early morning after at least 12 h of fasting (i.e., no food or drink other than water was allowed) (19). The subjects were asked to minimize any exertion prior to the laboratory visit for the measurement of REE. The laboratory room was kept at a neutral temperature (20 to 25°C) according to the previous report (19), and noise was kept to a minimum. After a rest period of 30–40 min in the supine position with a mask (Rudolph mask; Hans Rudolph Inc., Kansas City, MO), two 10-min samples of expired gas were collected in separate Douglas bags. Resting heart rate and body temperature were measured during the rest period to confirm an adequate duration of the resting period. The subjects were instructed to remain awake, quiet, and motionless without bending knees or arms in order to minimize contraction of skeletal muscles before and throughout the measuring periods.

An expiration gas analyzer (Minato AE-300S, Minato Medical Science, Tokyo, Japan) was used to assess the oxygen and carbon dioxide concentrations of the collected gas samples. The volume of expired air was determined by a dry gas volume meter (DC-5, Shinagawa, Japan). Gas exchange results were converted to REE in kcal/d using Weir’s equation (20). The mean of the 2 collected values was used for analysis.

Biochemical parameters. Whole blood was sampled from a cephalic vein in the morning after at least 12 h of fasting and immediately after REE determination. The blood samples were collected with the volunteers in sitting positions by a certified nurse using 21 or 22 gauge butterfly needles (Terumo Corp., Tokyo, Japan) with Luer adapter and tube holder (Terumo Corp.). All of the blood sample analyses were conducted in the blood analysis laboratory (BML Co. Ltd., Tokyo) including red blood cell count, hemoglobin, hematocrit, mean corpuscular volume, mean corpuscular hemoglobin concentration, total and HDL cholesterol, triglyceride, and fasting plasma glucose. Because thyroid hormone influences metabolic rate (21), the blood level of thyroid hormone (triiodothyronine: T3) was also evaluated. T3 is an activated form of thyroid hormone that is necessary for the thyroid hormone action. The T3 levels of all of the subjects were confirmed to be within the normal range.

Statistical analysis. The data were expressed as mean ± standard deviation (SD) with the normal range provided in parentheses. SPSS ver. 20.0 was used for statistical analysis (SPSS Inc., Chicago, IL). Multiple regression analysis with forced entry was used to determine whether FFM, FM, T3 level, and internal organs independently influenced REE. The level of significance for all statistical analyses was defined as p<0.05.

RESULTS

Subject characteristics

Table 1 shows the characteristics of the subjects. The average body height was 174.7 ± 5.9 cm. BW was 81.2 ± 11.3 kg, and % body fat was 16.3 ± 4.1%. The largest internal organ was the liver, followed by brain, kidneys, and heart (1.74 kg, 1.40 kg, 0.40 kg, and 0.31 kg). The sum of the 4 internal organs was 3.8 ± 0.4 kg, which was about 4.8% of the total body weight on average. Furthermore, the average relative contribution of the sum of internal organs was 5.7% of FFM. On the other hand, skeletal muscle had the largest contribution to FFM, which was 52.6%. The measured REE was 1,869 ± 230 kcal/d and REE/FFM ratio was 27.7 ± 1.9 kcal/kg/d, and showed a significant correlation with FFM (r=0.825, p<0.001).

Based on the biochemical parameters none of the subjects were anemic. The average values of total cholesterol, HDL cholesterol, triglyceride, and FPG were all within normal range. The average T3 was 128 ± 19 ng/
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dL and T3 levels for all of the subjects fell in the normal range (Table 1).

Organ mass and FFM
There were significant association between all 4 organs and FFM (Fig. 1 and Table 2). Liver showed the highest correlation of all 4 internal organs with FFM ($r=0.712$, $p<0.001$). Only the brain showed relatively low correlation with FFM ($r=0.333$, $p=0.044$). Furthermore, unlike the other 3 organs, the brain did not show a significant association with skeletal muscle. FM was strongly associated with both FFM ($r=0.637$, $p<0.001$) and skeletal muscle ($r=0.522$, $p<0.01$). The liver, heart, and kidneys significantly related to each other, while the brain did not correlate with any of the other organs (Table 2).

Organ mass contribution to REE
The masses of liver, heart, kidneys, and skeletal muscle all contributed consistently to REE regardless of different FFM (Fig. 2). On the other hand, the relative rate of brain contribution to measured REE became significantly smaller as the FFM became larger ($r=-0.672$, $p<0.001$). The averages of the relative contribution to REE from each tissue and organ were: skeletal muscle 24.8±1.8%, liver 18.7±2.1%, brain 18.1±2.1%, heart 7.3±1.2%, and kidneys 9.4±1.8%. The average percentage of the sum of liver, brain, heart, and kidneys to total REE was 53.5±3.9%. The measured adipose tissue also increased as FFM increased, and thus, the relative contribution of adipose tissue to REE became larger as FFM increased ($r=0.399$, $p<0.05$). There was also a significantly positive relationship between T3 and REE, even after adjusting for FFM ($r=0.457$, $p<0.01$). Multiple regression analysis was used to assess the effects of FFM, FM, and T3 on variance of REE, and also the associations between REE and the mass of each organ (kg) after adjustment for FFM, FM, and T3 (Table 3). The results showed that FFM alone explained 68.1% of the variance in REE, and T3 explained an additional 7.8% of the variance of REE. FM was not an independent factor influencing the variance of REE. None of the organs significantly influenced REE as independent variables, but the liver showed a positive trend ($p=0.068$) as an independent factor and accounted for an additional 2.5% of variance of REE (Table 3).

DISCUSSION
The present study has determined that the major reason that the REE/FFM ratio was steady in the male power athletes was because of the consistency of the contributions of internal organs to REE with high meta-

Table 2. Pearson correlation coefficients among body compartment sizes.

<table>
<thead>
<tr>
<th></th>
<th>n=37</th>
<th>FFM</th>
<th>FM</th>
<th>SM</th>
<th>Liver</th>
<th>Brain</th>
<th>Heart</th>
</tr>
</thead>
<tbody>
<tr>
<td>FFM</td>
<td></td>
<td>0.637**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>FM</td>
<td></td>
<td>0.917**</td>
<td>0.522**</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SM</td>
<td></td>
<td></td>
<td>0.646**</td>
<td>0.628**</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Liver</td>
<td></td>
<td>0.712**</td>
<td>0.646**</td>
<td>0.628**</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Brain</td>
<td></td>
<td>0.333*</td>
<td>0.050</td>
<td>0.250</td>
<td>0.493**</td>
<td>-0.106</td>
<td></td>
</tr>
<tr>
<td>Heart</td>
<td></td>
<td>0.538**</td>
<td>0.469**</td>
<td>0.470**</td>
<td>0.709**</td>
<td>0.105</td>
<td>0.494**</td>
</tr>
<tr>
<td>Kidneys</td>
<td></td>
<td>0.683**</td>
<td>0.606**</td>
<td>0.610**</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

FFM: fat-free mass, FM: fat mass, SM: skeletal muscle.
* $p<0.05$, ** $p<0.01$.  

Fig. 1. Relationship between fat-free mass (FFM) (kg) and organ mass of liver, brain, kidneys and heart (kg).
The mass of liver, heart, and kidneys correlated significantly with FFM, and thus the relative contribution of these three organs did not change, regardless of FFM. Similarly, Illner et al. (4) and Sparti et al. (22) found significant correlation between FFM and the liver, heart, and kidneys. Heymsfield et al. (23) also reported significant correlation between liver mass and FFM. Furthermore, previous studies have reported correlation between FFM and the liver, heart, and kidneys in both young adult men and women, as measured by the same method as used in the current study (3, 4, 22). It is well known that left ventricular hypertrophy with resistance training is associated with increases in left ventricular mass (24, 25). Therefore, it may be possible that the relatively lower correlation between FFM and the heart in the present study as compared to the liver or kidneys is due to the characteristics of the athletes, who were involved in daily physical training, including resistance training. In any case, our results have supported the results of previous research in which the liver, heart, and kidneys typically become large correspondently with FFM, no matter whether the subjects are athletes or non-athletes.

As for the brain, there have been other studies that also supported a significant relationship between brain mass and FFM (3, 23). Heymsfield et al. (23) reported that the human brain positively correlated with FFM, even though the relationship was much weaker than that of the liver. They also found that FFM accounted for 55% of the variation of liver mass in men, but only 6% of the mass of the brain after adjusting for age (23). Illner et al. (4) also found a relatively lower correlation between the brain and FFM compared to the liver, heart, and kidneys. Based on these findings, it seems that the brain does correlate positively with FFM, but the relationship is not as strong as for the other internal organs.

In the present study, the inclusion of subjects with much larger FFM compared to the previous study (23) may be another reason why the percentage contribution to REE from brain mass was negatively correlated with FFM.

Because of the reduced relative contribution of the brain to the REE, REE/FFM could be expected to become smaller as FFM become larger, but in fact, in our subjects, the REE/FFM remained constant, regardless of the FFM. The influence of adipose tissue was considered one of the reasons for this. FFM and adipose tissue were significantly correlated ($r=0.637$, $p<0.001$) in the present study, and thus the fractional contribution of adipose tissue to REE increased as FFM became larger ($r=0.399$).

**Table 3. Multiple regression analysis with REE as the dependent variable and FFM, FM, $T_3$, and liver as the independent variables.**

<table>
<thead>
<tr>
<th>REE (kcal/d)</th>
<th>$R^2$</th>
<th>$\beta$</th>
<th>$p$ value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model 1</td>
<td>0.681</td>
<td>0.825</td>
<td>$&lt;0.001$</td>
</tr>
<tr>
<td>FFM (kg)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Model 2</td>
<td>0.683</td>
<td>0.782</td>
<td>$&lt;0.001$</td>
</tr>
<tr>
<td>FFM (kg)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>FM (kg)</td>
<td>0.067</td>
<td>0.597</td>
<td></td>
</tr>
<tr>
<td>Model 3</td>
<td>0.761</td>
<td>0.697</td>
<td>$&lt;0.001$</td>
</tr>
<tr>
<td>FFM (kg)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>FM (kg)</td>
<td>0.078</td>
<td>0.484</td>
<td></td>
</tr>
<tr>
<td>$T_3$ (ng/dL)</td>
<td>0.289</td>
<td>0.003</td>
<td></td>
</tr>
<tr>
<td>Model 4</td>
<td>0.786</td>
<td>0.579</td>
<td>$&lt;0.001$</td>
</tr>
<tr>
<td>FFM (kg)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>FM (kg)</td>
<td>0.002</td>
<td>0.989</td>
<td></td>
</tr>
<tr>
<td>$T_3$ (ng/dL)</td>
<td>0.283</td>
<td>0.002</td>
<td></td>
</tr>
<tr>
<td>Liver (kg)</td>
<td>0.236</td>
<td>0.068</td>
<td></td>
</tr>
</tbody>
</table>

REE: resting energy expenditure, FFM: fat-free mass, FM: fat mass, $T_3$: plasma triiodothyronine.
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$p<0.05$), unlike previous studies, which did not show a relationship between FFM and FM ($4, 22, 26$). Therefore, adipose tissue may contribute more to increase the REE/FFM ratio in athletes with larger FFM. Male athletes commonly have relatively low percentages of body fat compared to non-athlete males with similar body weight; however, for power athletes in particular with large FFM, FM may be relatively large, and this may result in a marked impact on raising the REE/FFM ratio.

Multiple regression analysis revealed FFM as the major determinant of variance of REE ($68.1\%$) in the present study. However, FM was not an independent factor, and only an additional $0.2\%$ of variability was explained by FM, compared to the model of FFM alone. One of the reasons for the low contribution of FM to the variance of REE is the low metabolic rate of adipose tissue ($4.5\text{ kcal} \cdot \text{kg}^{-1} \cdot \text{d}^{-1}$). T$_3$ explained additional $7.8\%$ of variability in REE after FFM and FM were adjusted, and its contribution was quite high compared to that in other studies. For example, a study by Taguchi et al. ($27$) found that T$_3$ accounted for $5.3\%$ of REE after adjusting for FFM ($R^2=0.503$), similar to the findings of Svendsen et al. ($28$), in which T$_3$ explained an additional $2\%$ of REE after adjusting FFM, FM, and androstenedione (a precursor of male and female sex hormones) ($R^2=0.46$). Thyroid hormone has been found to alter the behavior of many metabolic pathways that are possibly relevant for the basal metabolic rate ($29$). Strong candidates for the underlying mechanisms are uncoupling of cellular metabolism from adenosine triphosphate (ATP) synthesis, and/or changes in the efficiency of metabolic processes downstream from the mitochondria ($29$-$31$). Even though the underlying pathways of thyroid hormone are not fully understood, the energy available for conversion into fat is reduced by increasing REE. Thus, alterations of thyroid hormones are thought to be an adaptation process to prevent obesity ($32$). In the present study, the level of T$_1$ had a significant association with REE/FFM ($r=0.463$, $p<0.05$), despite the fact that T$_3$ concentrations of all the subjects were within the normal range. Based on these results, although the true factors influencing the level of T$_1$ in the present cross-sectional study were unknown, thyroid hormone was considered another independent factor that significantly influences REE/FFM in male athletes. Finally, none of the organs independently influenced the variance of REE after adjusted FFM, FM, and T$_3$, although there is some suggestion that the liver alone may contribute to the variance of REE ($p=0.068$) after adjustment for FFM and T$_3$. Since the brain was not an independent factor which influenced REE, the reduction in brain contribution to REE as FFM became larger did not seem to have a large impact on REE/FFM ratio. Consequently, the results indicate that even though organ mass is considered, it may not significantly improve the accuracy of predicting REE for male athletes. Hence, the FFM alone should be an adequate component among body composition to determine REE for power athletes.

There are some limitations to this study. An assumed metabolic rate of organs and tissues was used to calculate the metabolic contributions to REE. However, the metabolic rates were determined based on a Western population ($1$). Hence, racial variations in metabolic rates may have been an influence. Gallagher et al. ($33$) have indicated that differences of REE between Caucasians and African Americans were due to the difference in the size of internal organs, but not due to the changes in the metabolic rates ($33$). For that reason, it is unlikely that the metabolic rates of organs and tissue are significantly different between races. The present study only measured T$_3$ to test the level of thyroid hormone because T$_3$ is known to have strong correlation with REE. It was also reported to reflect the influence of overfeeding and underfeeding better than the other thyroid hormone markers ($34$). However, measuring other markers such as Thyroxin (T$_4$) may be necessary in order to understand the accurate thyroid hormonal condition. The other limitation is the limited number of subjects from different types of sports. All of the subjects of the present study were American football players; consequently, there are unknown possibilities for bias in the results. American football is a unique sport with about 8 positions ($35$), and in order to carry out the tasks of each position on the field, body composition and physical talents of players varies widely ($35$). This relatively wide range of body composition, including FFM, was expected to minimize the bias in comparison with male athletes in general. Tatsuta et al. ($36$) reported that a group of elite athletes with relatively small FFM (average $54.1\pm3.9$ kg) had significantly higher metabolic rates of FFM ($31.9\pm2.6$ kcal·kg$^{-1}$·d$^{-1}$) compared to a group with larger FFM ($27.6\pm2.6$ kcal·kg$^{-1}$·d$^{-1}$). Therefore, additional research with different types of athletes with leaner body compositions would certainly be helpful in order to confirm the appropriate application of FFM metabolic rates to estimate REE for all types of athletes with wide-ranging body compositions.

Based on the findings from the present study, the adequacy of estimating REE based on an individual’s FFM has been established, at least among male athletes ranging between $57$ kg and $84$ kg. However, the risk of under- or over-estimation of REE in athletes with lower or higher FFM values should not be completely disregarded. Our results also suggested that considering internal organs did not significantly increase the accuracy of estimating REE in this group. It is ideal when individual REE can be measured by indirect calorimetry. Where it is difficult to measure REE directly, FFM can be used as a dependable factor to estimate REE, as long as FFM is accurately measured. In the present study, FFM was measured by DXA and the mass of internal organs was estimated by MRI and echocardiography, which are commonly used in similar studies examining body composition and internal organs with high accuracy. Therefore, the findings of these modalities in regards to the influence of internal organs on FFM or REE should be able to provide comparable results, and thus a further understanding of REE for athletes.

In conclusion, the study examined the contribution of the internal organs to the REE/FFM ratio among male
power athletes and found that the consistency of the REE/FFM ratio regardless of FFM was maintained by the steady relative contribution of internal organs to REE, except for the brain.

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REFERENCES

16) Lang RM, Bierig M, Devereux RB, Flachskampf FA, Foster E, Pelliikka PA, Picard MH, Roman MJ, Seward J, Shanewise JS, Solomon SD, Spencer KT, Sutton MS, Stewart WJ. 2005. Recommendations for chamber quantification: A report from the American Society of Echocardiography’s Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. J Am Soc Echocardiogr 18: 1440–1463.
29) Bianco AC, Maia AL, da Silva WS, Christoffolete MA.


