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A Brief Overview of Resting Energy Expenditure and It's Predictive Equations

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Authors' contributions

This work was carried out in collaboration between Authors KOS and ASA. Author KOS designed the study, performed the statistical analysis, wrote the protocol and first draft of the manuscript. Author ASA contributed immensely to the Genetics and Resting Energy Expenditure Section of the study. Both authors read and approved the final manuscript.

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ABSTRACT

Chronic energy imbalance is a strong predictor of metabolic abnormalities; a sizable number of research has been carried out to investigate how this system of obesity and energy homeostasis are understood, its metabolic consequences, and possible solutions to restore homeostasis. Currently, there is an array of methodologies designed for measurements of various aspects of energy metabolism. It is exigent therefore to understand the relative merits of each methodology in order to choose the most appropriate ones for research and other investigations. It is also important to note that studies of this nature require precision and accuracy. In this review article, we provide information on the utility and limitations of methodologies that are commonly used in energy expenditure research, with a specific focus on its variability, correlations, indications and contraindications.

Keywords: Resting energy expenditure; obesity; cardiometabolic risks.

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1. INTRODUCTION

Obesity, a known major risk factor for a range of chronic diseases including diabetes, cardiovascular diseases and certain cancers have been attributed to the major cause of deaths among adults. It also imposes an array of socio-economic setbacks to individuals and the society as a whole. In 2014, WHO [1] declared an alarming estimation of 1.9 billion adults (or 39% of the world population aged 18 years and over) that are overweight and among which over 600 million are obese. In Nigeria, a systemic review study in 2013 showed that the prevalence of overweight individuals ranged from 20.3%–35.1%, while the prevalence of obesity ranged from 8.1%–22.2% [2]. These epidemic proportions, therefore, call for a closer attention to combating these health disorders. Obesity is a physiological condition of chronic energy imbalance, while the regulation of energy metabolism varies widely among individuals, identifying those who are metabolically prone to weight gain and intervening accordingly is a key challenge for reversing the course of the obesity epidemic [3]. Energy expenditure is a fundamental property of living humans and other animals for the maintenance of cellular homeostasis when evaluated over 24 hours; Total Energy Expenditure (TEE) therefore can be described as the sum of five components: Resting Energy Expenditure (REE), Physical activity- induced Energy Expenditure, Thermic effect of food, Facultative thermogenesis and anabolism/growth [4,5]; Hence, the thermogenic response to food is an increase in REE after ingestion of food [6].

The REE is the energy necessary to run the basic process of the body such as the energy to maintain electrochemical gradients, generate heat and synthesize proteins required by body cell to maintain post absorptive homeostatic functions in resting subjects. REE is also used routinely by clinicians for estimation of energy requirements in patients care as well as by governmental agencies and health organizations in defining population energy requirement and it accounts for approximately 60% to 70% of the TEE [4,5,7]. A minor change in REE could lead to a significant energy imbalance and a huge change of body weight over a long period and REE decreases with muscle wasting, not losing fat alone [8]. For an average adult, the REE is fairly close to 1 kcal/kg body weight/hr or about 1,680 kcal/day for an individual weighing 70kg(5). Weight-controlling can be supported by a proper prescription of energy intake. The individual energy requirement is usually determined through REE and physical activity [9].

2. Genetics and Resting Energy Expenditure

REE is a modestly heritable trait and yet virtually nothing is known about the genetic factors that might influence the familial patterns. Also, despite the paucity of information on the genetics of energy and substrate metabolism, investigations into the genetic susceptibility to obesity suggest that altered energy expenditure and/or preferential substrate utilization are likely to be involved in the etiology of obesity, Such studies are confounded by the fact that both energy expenditure and substrate utilization are influenced predominantly by fat-free mass and likely to be genetically influenced [10]. Other study [11] observed that after adjustment for body size; approximately 11% of the observed variance in REE is due to familial aggregation, suggesting that genetic polymorphisms might influence the level of metabolic activity at rest and that one of the primary aims of energy metabolism research is to understand the inherent relations between REE and body composition. Ageing is associated with a decline in whole body REE at a rate of 1-2% per decade after the second decade of life. And the age-related lowering of REE occurs when body weight remains stable over the same time period. The changes in body composition could partly explain the age-related decrease in REE since Fat-Free Mass (FFM) is the main contributor to REE [7,12]. This could also mean that the age-related decline in the REE is primarily associated with loss of FFM and the loss is partially related to decrements in VO_2 max and nutritional factors [13]. Other study observed that the age-related decline of REE in healthy subjects is not caused by a decreasing organ metabolic rate but is fully accounted for by a reduction in FFM and proportional changes in its metabolically active components [14]. However, change in body composition cannot entirely explain the age-related decrease in REE. Ageing has been shown to be associated with a decrease in β-adrenergically stimulated thermogenesis, and sympathetic nervous system activity is a determinant of facultative thermogenesis. Metabolic active organs like heart, liver, kidney or brain and metabolic less active tissues like muscle, bone or skin could also be responsible for the decline in REE during ageing [7]. Recent findings regarding the existence of functional

brown adipose tissue in adult humans have suggested its physiological role and the uncoupling protein 1 (UCP1)-linked thermogenesis in energy balance; these was found to be associated with REE and thermoregulatory sympathetic nervous system activity in humans. Diminished REE in G-allele carriers as well as reduced thermoregulatory sympathetic nervous system activity for the G/G genotype, suggest that attenuated UCP1-linked thermogenesis has an adverse effect on the regulation of energy balance [15]. However, variations in human energy expenditure are partly because of an influence of the genotype, even after control for the well-established concomitants of energy expenditure. The existence of a genotype-environment interaction and the emerging nutrient partitioning which is the major determinant of the individual differences in metabolic rate responses to overfeeding or negative energy balance conditions, consistently support the hypothesis that heredity plays a significant role in the various components of energy expenditure in humans [16]. The variability of REE among individuals has been linked to body size, body composition, age, gender, hormones, organ sizes and genetic factors. It was suspected that the relative proportion of high and low metabolically active tissues independent of differences in FFM, significantly add to the residual variance in REE [17]. Other factors like puberty [18], antipsychotic medications [19], menarche [20], ethnicity and race [21], Diabetes [22], work of breathing, sleep and starvation[23] have also been investigated and they showed significant positive correlations.

A positive association was found between REE and weight gain in a lean adult Nigerian population and it was noted that the increased REE in this population was the result, rather than the cause of weight gain [24]. Also, some environmental factors like changes in body temperature in the tropics, increase muscle relaxation induced by the climate, temperature-induced changes to thyroid activity and dietary differences with particular references to protein intake, physiological adaptation to chronic energy restriction or to racial/genetical differences have been suggested to regulate some components of REE [25]. Other study also reported the influence of leptin, sympathetic activities, aerobic activity and resistance training, as well as dietary composition [26]; However, FFM remains the principal determinant of REE across all age ranges [27].

The African- American men and women tend to be more overweight and have lower REE compared with Caucasian men and women of comparable weight, height, age and FFM, the reported observation in those women might be a predisposing risk factor for long term weight gain and obesity but the racial differences in REE were reduced by >50% and were no longer significant when the mass of specific high-metabolic-rate organ was considered, differences in FFM composition may be responsible for the reported REE differences [28,29,30].

REE has been shown to be significantly higher in adult men than in women by an average 50 kcal/day irrespective of differences in body composition and aerobic fitness. It was suggested that the greater thermogenic effect of androgens compared with estrogens might also contribute to the gender differences [31]. Also, Creatine kinase activity was reported to be a determinant of REE, and high activities of this enzyme are particularly described in black people [32]; Creatine kinase increases the cell's capacity to function under high demands, thus greater Creatine kinase activity in cardiovascular muscle and other tissue with high energy demands could increase cardiovascular contractile reserve, enhance tropic responses and increase renal tubular activity to retain salt; and this could facilitate the development of arterial hypertension. On the other hand, studies on sleeping metabolic rate (SMR) have shown that the rate of decline in metabolic rate during sleep is directly related to body weight, BMI and FFM. Average SMR tends to be lower than REE in obese subjects and higher than REE in non-obese subjects [33].

3. Cardiometabolic Risks and Resting Energy Expenditure

Obesity-related cardiometabolic risk factors contribute to inter individual variation in REE, with hypertension, insulin resistance and T2DM been associated with higher REE. There are also significant associations of REE with systolic and diastolic blood pressure, FPG, insulin concentrations, and HOMA-IR. Similarly, increase REE is associated with hyperglycemia and glycemic intolerance and positively correlated with fasting insulin concentrations in non-diabetics [14]. Evidence suggests that a low REE may be due to genetic variation involving sympathetic activity, thyroid activity, β-receptor sensitivity, sodium, potassium and adenosine triphosphatase (Na+, K+-ATPase) enzyme

activity [34,35]. The etiology of a greater REE in diabetics has been suggested to be the result of abnormal protein metabolism and high insulin resistance. However, the exact mechanism still remains unclear [8]. Studies have also shown linear relationships among T2DM patients, REE, rates of hepatic glucose production, lipid oxidation, urinary albumin loss and anaemia accompanied by diabetic nephropathy [36,37].

A significant link between serum adiponectin concentrations and low REE was also reported [38], with the speculation that protection by adiponectin against obesity-related disorders is especially important for subjects with low REE. Again, subjects with low REE are at increased risk of developing these disorders because larger portion of their daily food intake is stored as fat. These findings, together with the well-known inverse relation between adiponectin and insulin resistance, fit in the same framework and confirm the important interplay between adiponectin and the pathogenesis of the Metabolic Syndrome.

In theory, higher energy expenditure should promote a negative energy balance and thereby weight loss in obese T2DM patients. Together with urinary glucose, this may serve as a defense mechanism against further weight gain. However, T2DM patients are often more resistant than matched non-diabetic individuals to losing weight In weight management programs, independent of whether the intervention is conventional or pharmaceutical assisted [39]. In contrast, acquired insulin resistance as a consequence of obesity may lead to a higher REE by increasing protein turnover, futile cycling, gluconeogenesis, and the activity of the sympathetic nervous system; as a result an increased REE at an impaired glucose tolerance is a metabolic consequence of obesity that is directed against further weight gain [14].

4. Measurements of Resting Energy Expenditure

The gold standard for measurement of REE is Calorimentry (Direct or Indirect). The equipment required to measure respiratory exchange make this procedure time consuming, costly and often unavailable. It requires extensive subject cooperation as well as accurate and precise flow and concentration measurement, using sophisticated flow and gas analyzers.

Direct Calorimetry can be used for the assessment of energy expenditure by measurement of the body's heat production in a Calorimeter, but the most commonly used method is the Indirect Calorimetry, a ventilated open circuit system by which the rate of energy expenditure is estimated in vivo from total body respiratory gas exchange measurements rather than directly from heat. It allows air (gas) volumes to be measured, and CO_2 and O_2 gas analyzers to determine the volumes of individual gases being produced or used. The respiratory quotient (RQ), which provides information about metabolic substrate utilization (lipid or carbohydrate), is calculated by dividing the volume of CO_2 produced by the volume of O_2 consumed ($RQ = VCO_2/VO_2$). It collects and mixes the expired air, measures the flow rate, and analyzes the gas concentration of the incoming and outgoing air for both O_2 and CO_2 [40]. Other methods, though less common, are the Refractometry, Mass Spectrometry, The Doubly Labeled Water method, metabolic carts and non-calorimetric techniques like physical activity log and kinematic measurements, heart rate and ventilation monitoring etc. However, using indirect calorimetry to compare energy expenditure among individual subjects that differ in body weights has inherent inaccuracies. Differences in body weight are usually associated with differences in tissue distribution; Since Energy expenditure of different tissues varies over a broad range and it is not possible to calculate the contribution of each organ, there is no clear-cut agreement about how Energy Expenditure is best expressed. To control for this confounder, some researchers perform pair feeding experiments which on its own has flaws possibly owing to a relative state of semi-starvation that is perceived by experimental animals [41]. Likewise, the impractical direct measurement of REE and characterized two perspectives from which a prediction of REE could be approached, i.e, clinical and physiologic has been supported [42]. The corresponding variables of interest are weight, height and gender for the clinical and FFM for the physiologic perspective.

5. Equations for Predicting Resting Energy Expenditure

A number of recognized prediction equations to calculate REE of individuals have been developed and recommended in clinical practice. They are estimates of how many calories an individual will burn if he/she were to do nothing but rest for 24 hrs [43]. These can provide the basis for prescribing an individualized energy

intake to attain a desired level of energy deficit and serve as the basis from which daily energy needs are established for the prescription of the meal plan with computation of macronutrients for weight control [9,37]. These prediction formulas were originally important in diagnosing thyroid disease, although today their primary role is in estimating subject's energy requirements [44,45].

The most frequently used formula for predicted energy expenditure are the Harris – Benedict equations which was established in 1919 and took into account gender, age, height and weight; although the non consideration of weight history and ethnicity of the individuals has been questioned [46].

The comparative studies for accuracy of some of the prediction equations by [43,47,48,49], showed that the Harris – Benedict equation is the most accurate of all the equations studied, considering the clinical variables, i.e. Age, gender, Weight and Height. The prediction equations of REE is highly dependent on the methodology employed to compare the various formulas, and Harris- Benedict equations have been supported to yield reasonable REE predictions for normal sized and obese subjects[50]. Harris - Benedict derived their equation using data from healthy, non-obese infants and other subjects in the age range 18-70yrs old, thus excluding a large group including the pediatric obese population [47,51].

Harris-Benedict equation is correct 80-90% of the time in healthy and normal volunteers. In obese volunteers, the equation predicts REE correctly only 40- 64% of the time. In critically ill patients the equation is correct only 50% of the time. The total energy expenditure of a hospitalized patient can however be calculated by multiplying the REE with the injury or activity factor and the thermic effect (if they are digesting and absorbing food). The activity factor for such patient is simplified to a factor of 1.2 if the patient is confined to bed, or 1.3 if allowed out of bed [23].

A major investigative focus of energy metabolism research over the past four decades is the development of REE prediction formulas based on FFM. Although investigations have expressed an increasing interest in REE – FFM relationships, several fundamental questions remain unanswered. Zimian et al. [52] showed The linear REE- FFM relationship long observed in adult humans is qualitatively consistent with the curvilinear REE – Body Mass relationship observed in mammals as a whole. The experimental data also suggest that mammals exhibit a decrease in the proportion of FFM as high metabolic rate organs with greater FFM. FFM may thus not be a "metabolically homogenous" compartment across mammals generally, and humans specifically, varying widely in Body Mass. The derived whole body level and tissue/organ level REE – FFM models are general and unsuitable for individual REE prediction [52].

The use of prediction equations has been recommended for calculating energy expenditure more importantly among the populations from which they were derived. This is due to individuals not within a particular population falling outside the parameters set forth in the equation [47,53]. In addition, some predicted equations were proposed based on whole body level, tissue organ level, cellular level and molecular level [54]. Despite several other proposed predicted equations, the most frequent used formula for predicted energy expenditure are the Harris – Benedict equations. FAO/WHO/UNU [55] advocated the use of multiples of REE to estimate energy expenditure and then proposed a revised equation which has also been investigated. Besides, most clinicians rely on REE prediction equations that incorporate easily measurable variables, such as body weight and height. Of these, the Harris-Benedict equations gave the lowest bias and narrowest limits of agreement, followed by the Owen equations. Therefore if weight and height are available, Harris- Benedict equation is highly recommended [46]. There are now many published methods and equations (Tables I and II) for predicting REE from measured body mass and body composition. Although these published reports extend back almost a century, new related studies appears on a regular basis, It then remains unclear what the similarities and differences are between these many methods and what, if any, advantages the newly introduced REE prediction models offer [56].

Studies comparing predictive equations with indirect calorimetry in critically ill patients showed a poor agreement between calculated and measured energy expenditure [57,58]. Indirect calorimetry allows for accurate determination of Energy expenditure, but widespread adoption of the technique has been limited due to the technical demands of measurements [59]. More so, even when Indirect Calorimetry is not

available, there is no consensus about which equation to use in hospitalized patients and critically ill children and this is has presented as a huge challenge for clinicians. Hence, nutrition for critically ill should be provided according to measurement of REE to avoid the consequences of overfeeding or malnutrition [60]. Several guidelines recommend a calorie delivery targeted at energy expenditure and suggested that lower energy targets may be acceptable during the first weeks of ICU stay, but there is still uncertainty regarding optimal targets for patients with preexisting malnutrition or a prolonged course of critical illness. Regardless of feeding strategy, setting individual caloric goals requires an estimation of EE either by use of equations with inputs from various patient characteristics, or indirect calorimetry [61].

Table 1. Equations for predicting REE derived from FFM in adult humans

Author	Descriptive equation
Owen et al. (1986)	REE = 19.7 × FFM + 334
Mifflin et al. (1990)	REE = 19.7 × FFM + 413
Luke & Schoeller (1992)	REE = 20.0 × FFM + 585
Jensen et al. (1988)	REE = 20.0 × FFM + 662
Ravussin et al. (1982)	REE = 20.82 × FFM + 471
Ravussin et al. (1986)	REE = 20.93 × FFM + 478.7
Elia 1992)	REE = 21.11 × FFM + 450
McNeil et al. (1987)	REE = 21.5 × FFM + 329
Heymsfield et al. (1988)	REE = 21.6 × FFM + 302
Cunningham (1980)	REE = 21.6 × FFM + 501.6
Ravussin & Bogardus (1989)	REE = 21.8 × FFM + 392
Owen et al. (1987)	REE = 22.3 × FFM + 290
Heshka et al. (1990)	REE = 22.94 × FFM + 356.7
Owen (1988)	REE = 23.6 × FFM + 186
Kashiwazaki et al. (1988)	REE = 24.5 × FFM + 304

FFM, fat-free mass (kg); Sex, male=0; female=1.REE, whole body resting energy expenditure (kcal/day). [52]

Table 2. The predictive equations for REE derived from age, weight, height, and gender

Authors	Equations
Kleiber (1932)	REE= 73.3xBM$^{0.74}$ (kcal/day)
Kleiber (1961)	REE= 73.3xBM$^{0.75}$ (kcal/day)
Harris & Benedict (1919)	REE= 66.437+13.752 Wt+5.003 Ht-6.755 Age (male)
	REE= 655.096+9.563 Wt+1.85 Ht-4.676 Age (female)
	[kcal/day (18-30 years)]
Gallagher et al. (1998)	REE= 689 brain+27.5 skeletal muscle-210 (kcal/day)
WHO/FAO/UNU (1985)	REE= 679+15.3 Wt (male)
	REE= 496+14.7 Wt (female) [kcal/day (18-30 years)]
Schofield et al. (1985)	REE= 688.5+15.1Wt (male)
	REE= 603.2+13.1Wt (female) [kcal/day (18-30 years)]
Henry & Rees (1991)	REE= 672+13.4 Wt (male)
	REE= 614.8+11.5 Wt(female) (kcal/day)
Maffies (1993)	REE= 28.6 Wt+23.6 Ht-69.1 Age+1287(male)
	REE= 35.8 Wt+15.6 Ht-36.3 Age+1552(female) (kj/day)
Mifflin et al. (1990)	REE= 15.1 Wt+371 (kcal/day)
Molner (1995)	REE= 50.2 Wt+29.6 Ht-144.5 Age-550 Sex+594.3 (kj/day)

Wt-Weight (kg); Ht-Height (cm); FM-Fat mass (gm); FFM-Fat free mass (gm); Age (years); gender: male=0; female=1 [4,62]

When indirect Calorimetry is not available, predictive equations are used to estimate REE, However many researchers have investigated the validity of most of these equations; and comparative studies on different equation and indirect Calorimetry have been carried out and have also presented their opinions. There is no consistency regarding a more accurate equation with a closer estimate to that of Indirect Calorimetry, or a specific and common equation either for obese, non-obese, children, adult or critically ill patients. Although, no predictive equations had the same values of REE as compared to those of Indirect Calorimetry, WHO and Harris-Bennedict equations were recommended because they least underestimated REE [63]. Also, validity studies [57,64,65] have shown that there is a wide variation in the accuracy of REE predictive equations; and there is no consensus about which equation should be used in hospitalized patients(60). Akin to these, it was recommended that clinical judgment should be adhered to, regarding when to accept estimated REE using predictive equations in any given individual. Indirect calorimetry may be an important tool when, in the judgment of the clinician, the predictive methods fail an individual in a clinically relevant way. For members of groups that are greatly underrepresented by existing validation studies of predictive equations, a high level of suspicion regarding the accuracy of the equations is warranted [66].

6. CONCLUSION

The developed prediction equations for REE has been skirmished with a lot of inconsistencies and criticisms, the general opinion is that indirect calorimetry remains the most reliable method for REE estimation, be it in obese, non-obese, children, adults or the critically ill, regardless of gender, ethnic or racial background. However, the importance of REE estimation cannot be overemphasized and researchers have shown severally how it influences the development of obesity and cardiometabolic risks. There is a need for a more accurate, precise and non-ambiguous prediction equation for a dietary prescription, clinical and research purposes.

CONSENT

It is not applicable.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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