ABSTRACT

Resting Metabolic Rate (RMR) is defined as the amount of energy expended by a person at rest during post absorptive and thermoneutral conditions. Determining an athlete’s RMR is useful to aid in establishing individualized dietary recommendations to maintain an ideal body weight and promote athletic performance. This chapter will review the techniques available to determine RMR and the biological and environmental factors that influence RMR in athletes. Further, it provides comparisons of RMR among different types of athletes.
DEFINITION

Resting Metabolic Rate (RMR) is defined as the amount of energy expended by a person in a resting state under post absorptive and thermoneutral conditions. Basal metabolic rate more precisely represents the RMR measured just after awakening in the morning. Because they differ by less than 10%, these terms are often used interchangeably. This energy requirement reflects the necessary energy needed to sustain the body’s vital functions in the waking state, including respiration, heart rate, and blood pressure. RMR makes up the largest component (~60-75%) of total daily energy expenditure, which also is composed of the Thermic Effect of Meals (TEM) (~10%) and the thermic effect of physical activity (typically 15-30% but may be much higher in highly active individuals).

BENEFITS OF KNOWING RMR

RMR and activity estimates can be used to help establish individualized dietary recommendations in order to develop an optimal weight and energy control program. This may be particularly useful to high caliber athletes to promote athletic performance by achieving body composition goals. It also may be important to the everyday fitness enthusiast as a low RMR has been identified as a predisposing risk factor for the development of obesity. Studies also suggest that the RMR of formerly obese subjects is 3-5% lower than sex-matched adults who have never been obese [1], potentially influencing the risk for weight regain.

WHAT FACTORS CONTRIBUTE TO RMR?

Below we discuss how both biological (inherent) and environmental (behaviors and traits) factors may influence RMR in athletes.

Biological Determinants of RMR

Body size & composition

RMR is proportional to the surface area of the body but when expressed per unit of body mass, RMR is inversely related to body size (i.e. mass specific RMR of a mouse is about six times that of a human). However, the major factor determining RMR is Fat-Free Mass (FFM; the body’s water, bone, organs and muscle content), which is a better predictor of RMR than surface area [2]. Specifically, it is estimated that FFM accounts for 60-85% of the variance in RMR, with the kidneys (7%), heart (10%), skeletal muscle (18%), brain (19%), liver (27%), and miscellaneous lean (i.e. bones, lungs, intestines) sources (19%) contributing to energy consumed at rest [3]. It is estimated that RMR increases by ~50 kcal/day for each kg increase in FFM. Exercise training can increase muscle mass by up to 200%, even in older adults [4]. While clearly important, RMR in individuals with the same FFM may vary by as much as 500 kcal/day, suggesting that other factors also substantially influence RMR. Although not consistently observed [5], some studies suggest that fat mass is an additional contributor to RMR [6, 7].
Age

Age accounts for up to 14% of the variance in RMR. A 2% decrease in RMR per decade is observed throughout adulthood. We studied women athletes aged 18-69 years of age and determined that RMR was similar between the youngest two age groups (18-29 and 30-39 years) but higher in young athletes than young controls [8]. Furthermore, RMR declined with age in the women athletes with the oldest athletes expending almost 240 kcals/day less than the youngest athletes. FFM was highly correlated to RMR in the athletes. In a new analysis of a smaller group (n=33), we measured muscle composition in the mid-thigh by computed tomography (CT) and show that muscle area was associated with RMR across the age span (r=0.61, P<0.0001, Figure 1). While age-associated hormonal changes, including reduced growth hormone, testosterone, and insulin resistance, may be responsible for some of these changes as they affect muscle mass, it appears that the changes in FFM only partly explain changes in RMR over the life span [9,10].

![Figure 1: Relationship between muscle area and Resting Metabolic Rate (RMR) in women athletes (r=0.61, P<0.0001).](image)

Sex

RMR is about 5-10% lower in women than men. This is largely because women possess more body fat, which is metabolically less active than muscle. Further, testosterone and dehydroepiandrosterone, serum concentrations of which are higher in men, stimulate RMR through their effects on FFM. However, when RMR is expressed per FFM, the gender difference is essentially eliminated.
Other Determinants

Other determinants of resting metabolism include genetics, hormone release, and in women, the phase of the menstrual cycle.

Genetic influence

Genetic variants, including ethnicity and body temperature, may affect RMR. After adjusting for fat-free mass, fat mass, and visceral fat, it is suggested that RMR is ~5% higher in Caucasian than African American adults [11]. In mammals, an increase of body temperature by 1°C can increase RMR by 13%.

A wide range of temperatures are considered normal in adults (i.e. 33.2-38.2 °C), depending on the gender and location (i.e. oral vs. rectal) measured and normal body temperature may differ by as much as 0.5°C from one day to the next [12].

Hormonal influence

The release of triiodothyronine (T3; thyroid hormone) or catecholamines increases RMR [13-15], while the effects of circulating leptin on RMR are inconclusive with studies finding a positive [16,17], negative [18], or no [19,20] association. However, the addition of Thyroid Stimulating Hormone (TSH) to RMR predictive equations does not appear to improve the accuracy of predicting RMR [21].

Menstrual cycle influence

It is estimated that during the luteal phase of the menstrual cycle, there may be up to a 5-10% increase in RMR [22], while pregnancy can increase RMR by up to 35% [23]. In women with amenorrhea, which may be present in as much as 45-50% of highly active athletes [24], RMR is significantly depressed [25]. However, RMR is not significantly different in premenopausal than postmenopausal distance runners [26]. The effect of menstrual dysfunction on RMR was examined in young (~25-30 years) women runners who were amenorrheic, eumenorrheic, and eumenorrheic sedentary controls [27]. Runners had similar age, percent body fat, VO₂max, and training and competition times. RMR adjusted for body weight or FFM was significantly lower in amenorrheic runners than eumenorrheic runners and sedentary controls. T3 levels were lower in the amenorrheic group but dietary intake was similar across groups. This study suggests that the reduced RMR in amenorrheic runners could be an adaptive mechanism for energy conservation.

Environmental Determinants of RMR

Physical activity& fitness

Although RMR is measured at rest, the carry over effect of exercise on metabolic rate strongly influences RMR. Those who perform habitual exercise (both aerobic and resistive) have ~5-10% higher RMR than their sedentary counterparts, despite similar FFM and lower body fat [28,29].
We report in women athletes that RMR is associated with VO\textsubscript{2}\text{max} (l/min) \( (r=0.56, P<0.0001, \text{Figure 2}) \) demonstrating the importance of fitness in resting energy metabolism. In a study of women who exercise regularly, VO\textsubscript{2}\text{max} accounts for 35% of the individual variance in RMR [26]. Habitual exercise [26] and the initiation of exercise appear to slow down age-related declines in RMR, independent of its effects on body composition [30,31].

![Figure 2: Relationship between VO\textsubscript{2}\text{max} and Resting Metabolic Rate (RMR) in women athletes \( (r=0.56, P<0.0001) \).]

**Detraining effects on RMR**

Although exercise training can increase RMR, other studies have shown that RMR is unaffected by training [32]. It is possible that the timing of the RMR measurement could explain the discrepant results. Examination of the cessation of training on RMR may offer insight into whether RMR is modified by exercise training. However, only a limited number of studies have examined the effects of detraining on RMR. With a three-day interruption of training in a small sample \( (n=8) \) of trained individuals, RMR significantly decreased by \(~6.5\%\) [33]. In a sample of highly trained female athletes (runners, triathletes, \( n=9 \)), RMR was measured 15 h post exercise, and 39, 63, and 87 hours after suspension of training with control of timing during the menstrual cycle [34]. The results indicate that RMR dropped 8% by 39 hour post-exercise and is maintained at this reduced level at 87 hour post-exercise. In another study of competitive collegiate swimmers who detrained for \(~5\) weeks, RMR decreased 7% which coincided with increased body fat mass and reductions in VO\textsubscript{2}\text{peak} during the 35-to 42-day detraining period [35]. These studies suggest that the training-induced increase in RMR, partially represent an acute effect of repeated exercise bouts and that detraining may adversely affect metabolic rate.
Weight loss & Weight cycling in athletes

Many athletes desire to alter their body composition by practicing energy restriction to enhance athletic performance. Weight loss may have a negative impact on RMR, resulting in an adaptive decrease in RMR by as much as 20%, predisposing individuals to weight regain [36]. It has been questioned whether the decline in RMR is directly related to the loss of FFM observed with weight loss or truly reflects metabolic adaptations below that which can be explained by losses in lean tissue. The decline in RMR with weight loss may be less in athletes, as we [37] and others [38] show that the addition of exercise to weight loss preserves FFM.

There is some thought that weight cycling can influence resting energy expenditure. Weight cycling is defined as repeated cycles of weight loss and weight regain. In a study of women rowers who had a history of dieting and weight fluctuation, RMR was measured prior to the beginning of the season, at the peak of the season and during the off-season over one year [39]. Compared to controls, women rowers lost and regained ~ 4 kg over the seasons. RMR and T3 changed over time in both groups and were not due to changes in body weight but changes in FFM. This would suggest that it is not the weight fluctuation; rather it is the composition of the weight change that is important.

Weight loss and weight cycling also is practiced among wrestlers for performance reasons. It is thought that weight cycling may result in a loss of FFM and a decline in RMR. In an early study of how cycles of weight loss and weight regain affect RMR, Melby et al. [40] reported that wrestlers have a significantly higher RMR than controls preseason. Although there is a reduction in RMR during the season when the wrestlers reduce their weight for competition, both absolute RMR and RMR expressed per body weight and per FFM are significantly higher for the wrestlers than non-wrestlers for pre and postseason. These results suggest that one season of weight cycling does not increase energy efficiency as the postseason RMR values were not different than the preseason RMR values. This same group followed up with another paper showing that no difference in RMR was observed after a second year of follow-up or two competitive seasons [41]. Of note, many wrestlers dehydrated to lose weight and not necessarily used food restriction, which could confound interpretations as dehydration can lead to an elevation in RMR. In another study, RMR was compared over a one-year competitive season between collegiate wrestlers who were “non-cyclers” and “cyclers” [42]. Cyclers was defined as those who underwent rapid weight loss/regain cycles, specifically wrestlers who were “often” or “always” dieting, cut weight to meet a specific weight category of at least 3.0 kg, and did so 6-9 times or 10+ times per competitive season. RMR did not change throughout the season over time or by group, whether expressed in absolute values, per body weight, or per FFM. Serum total T3 levels also were similar between groups and over the one-year period. There is one review which covered weight cycling and effects on RMR which concluded that the suppressed RMR with weight loss in wrestlers was transient but needed further confirmation [43]. Although this review was published over 20 years ago, no further research could be found on this topic in wrestlers.
Professional jockeys also are prone to chronic weight cycling through severe energy restriction and dehydration to achieve a required racing weight. RMR was measured in older retired male jockeys (~59 years of age) and values were described as being somewhat lower than a BMI matched but slightly older group of men [44]. The retired jockeys were however, either overweight or obese with an increase in total body fat and high cholesterol, placing them at risk for cardiovascular disease. The effect of retirement from competition among other sports on RMR is unknown.

**Dietary factors**

Studies examining the effects of dietary intake on RMR in athletes and non-athletes alike are scant. There is evidence to suggest that an increased food intake can increase RMR. In a study of female athletes, it is suggested that energy intake may be a significant predictor of measured RMR, accounting for ~36% of the variance [45]. Further, in trained subjects, a high energy diet results in an increased RMR, even in periods of energy balance [46].

There also is evidence that the TEM may be affected by physical activity. TEM refers to the fact that oxygen consumption increases after ingestion of food. In a critical review of almost 50 studies that evaluated TEM, TEM was reduced in obese individuals compared to lean subjects [47]. TEM was noted to be related to the degree of insulin resistance, physical activity, meal size, meal composition, and aging. It is controversial whether physical activity changes TEM as an acute bout of physical activity preceding a meal has been shown to enhance TEM, as well as have no effect on postprandial thermogenesis. Thus, it is reasonable to hypothesize that TEM may be altered in highly trained athletes and yet, there is very little known about the TEM in athletes. Metabolic rate was measured in swimmers under three conditions to test the calorigenic effect of a liquid meal [48]. There was a 22% increase in metabolic rate over RMR at 20 minutes post exercise but this returned to pre-exercise level by 40 min post exercise. Caloric expenditure increased ~25 kcal/h when swim exercise preceded the meal compared to ~20 kcal/h without the preceding swimming. The authors suggest that the difference of ~18 kcal over four hours may not be that important for short term energy balance but could be significant in long-term energy balance. TEM was not different between a small sample of collegiate wrestlers matched for age, weight, and FFM with collegiate swimmers [49]. Additionally, TEM does not differ between endurance trained male athletes receiving adequate nutrition versus those on caloric restriction (1,490 kcal/d less than adequate group) [50]. Further research is needed to identify whether TEM is altered in highly trained individuals and the factors that may regulate such an effect.

**HOW IS RMR DETERMINED?**

**Measured**

Direct calorimetry is considered the gold standard for measurement for RMR. It measures energy expenditure through heat loss by placing a subject in a thermally isolated metabolic chamber and measuring dissipation of heat. While highly accurate, it is technically difficult and
expensive, thus, indirect calorimetry is the most frequently used technique for measuring RMR. The underlying principle for indirect calorimetry is that oxygen is needed for the production of energy and carbon dioxide is release as an end-product of metabolism. Thus, the rates of oxygen (VO$_2$) consumed and carbon dioxide (VCO$_2$) production is measured in breath samples. Samples can be measured in a respiration chamber or by an open circuit ventilated hood system, which is the most common. For this assessment, breath samples are collected from a subject lying in a supine position in a comfortable environment for about 30-40 minutes. At least 12 hours of fasting is required so that there is no energy required for digestion and absorption of ingested food. Athletes also should avoid exercise for 48 hours prior to the measurement to eliminate the effects of acute activity because exercise can increase RMR up to 39 hours post-exercise [34]. Steady state VO$_2$ and VCO$_2$ measurements can be used to estimate energy expenditure using various published formulas, the most common being the modified Weir equation [51]: RMR (kcal/d) = [(3.9 x VO$_2$) + (1.1 x VCO$_2$) x 1.44], with VO$_2$ and VCO$_2$ in L/min. Disadvantages of indirect calorimetry are the expense, measurement time, clinical expertise, and specialized equipment required to collect and analyze respiratory gases.

**Predicted**

Several equations are available to predict RMR from factors such as age, height, weight, and FFM. The majority of these predictive equations are derived from sedentary adult populations, which may underestimate RMR in athletes. In fact, it is estimated that these clinical predictive equations do not estimate metabolic rates in about 50% of athletes [45]. However, several predictive equations are validated in physically active individuals. Predictive equations that best predict RMR measured by indirect calorimetry in athletes of various ages and sports may be viewed in Table 1. Of note, predictive equations in physically active individuals are focused on adolescent and young adults and are lacking in middle aged and older adults. Overall, it appears that the equation proposed by Cunningham *et al.* [52], where RMR (kcal/d) = 500 + [22 x FFM], most accurately reflects measured RMR in young athletes.
Table 1: Validation of RMR predictive equations in athletes.

<table>
<thead>
<tr>
<th>Athlete Validation Study</th>
<th>Validated Population</th>
<th>RMR Reference and Equation (kcal/d)</th>
<th>Kcal/d Variation (measured RMR-predicted RMR)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Males</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>De Lorenzo et al. [56]</td>
<td>Soccer players (N=25; age: 16.8±1.0 years)</td>
<td>Cunningham et al. [52] 500 + [22 x FFM]</td>
<td>4</td>
</tr>
<tr>
<td>De Lorenzo et al. [57]</td>
<td>Various sports (N=51; age range: 16-18 years)</td>
<td></td>
<td>59</td>
</tr>
<tr>
<td>Loureiro et al. [58]</td>
<td>Pentathlon athletes (N=17; age: 15±2 years)</td>
<td></td>
<td>-21</td>
</tr>
<tr>
<td>Thompson et al. [45]</td>
<td>Highly trained endurance athletes (N=24; age:</td>
<td></td>
<td>158</td>
</tr>
<tr>
<td>Kim et al. [55]</td>
<td>Soccer players (N=30; age 16.7±1.0 years)</td>
<td>Cunningham et al. [59] 370 + [21.6 x FFM]</td>
<td>-30</td>
</tr>
<tr>
<td>De Lorenzo et al. [57]</td>
<td>Various sports (N=51; age range: 16-18 years)</td>
<td>De Lorenzo et al. [57] -857 + [9.0 x Weight] + [11.7 x Height]</td>
<td>91</td>
</tr>
<tr>
<td>Wong et al. [60]</td>
<td>Elite athletes of various sports (N=92; age: 21.4±3.0 years)</td>
<td>FAO/WHO/UNU [61] [0.064 x Weight] + 2.84</td>
<td>25</td>
</tr>
<tr>
<td>Wong et al. [60]</td>
<td>Elite athletes of various sports (N=92; age: 21.4±3.0 years)</td>
<td>Institute of Medicine of the National Academies [64] 189 – [17.6 x Age] + [625 x (Height/100)] + [7.9 x Weight]</td>
<td>-23</td>
</tr>
<tr>
<td>De Lorenzo et al. [56]</td>
<td>Soccer players (N=25; age: 16.8±1.0 years)</td>
<td>Schofield et al. [63] [16.24 x Weight] + [1.37 x Height] + 515.3</td>
<td>-32</td>
</tr>
<tr>
<td><strong>Females</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Loureiro et al. [58]</td>
<td>Pentathlon athletes (N=11; age: 14±3 years)</td>
<td>Cunningham et al. [52] 500 + [22 x FFM]</td>
<td>12</td>
</tr>
<tr>
<td>Thompson et al. [45]</td>
<td>Highly trained endurance athletes (N=13; age:</td>
<td></td>
<td>103</td>
</tr>
<tr>
<td>Wong et al. [60]</td>
<td>Elite athletes of various sports (N=33; age: 20.4±2.1 years)</td>
<td>Institute of Medicine of the National Academies [64] 189 – [17.6 x Age] + [625 x (Height/100)] + [7.9 x Weight]</td>
<td>-3</td>
</tr>
<tr>
<td>Kim et al. [55]</td>
<td>Soccer players (N=20; age 16.4±1.1 years)</td>
<td>Mifflin et al. [65] [9.99 x Weight] + [6.25 x Height] – [4.92 x Age] - 161</td>
<td>-23</td>
</tr>
<tr>
<td>Kim et al. [55]</td>
<td>Soccer players (N=20; age 16.4±1.1 years)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Genders Combined</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ten Haaf et al. [66]</td>
<td>Recreationally active athletes (N=90; age: 23.2±4.8 years)</td>
<td>Cunningham et al. [52] 500 + [22 x FFM]</td>
<td>17</td>
</tr>
<tr>
<td>Carsohn et al. [67]</td>
<td>Rowers and canoeists from German national team (N=17)</td>
<td></td>
<td>133</td>
</tr>
<tr>
<td>Kim et al. [55]</td>
<td>Soccer players (N=50; age 16.6±1.0 years)</td>
<td>Kim et al. [55] 730.4 + [15 x FFM]</td>
<td>-2</td>
</tr>
<tr>
<td>ten Haaf et al. [66]</td>
<td>Recreationally active athletes (N=90; age: 23.2±4.8 years)</td>
<td>ten Haaf et al. [66] [11.936 x Weight] + [587.728 x Height] – [8.129 x age] + [191.027 x sex(M=1,F=0)] + 29.279</td>
<td>13</td>
</tr>
</tbody>
</table>

FFM= fat-free mass; WHR= waist to hip ratio. FFM in kg, Weight in kg, Height in cm, Age in years.

**COMPARISONS OF RMR AMONG DIFFERENT TYPES OF ATHLETES**

Given the variety of sports and the distinct nutritional and training regimens, it is interesting to examine whether RMR varies among athletic groups. Male collegiate wrestlers and collegiate swimmers were tested for differences in RMR, VO₂max, and maximal anaerobic capacity [49].
Swimmers had higher VO$_2$max but lower maximal anaerobic capacity than wrestlers but RMR adjusted for FFM was not different between sports. RMR and total energy expenditure determined by the doubly labeled water method of elite Japanese national team synchronized swimmers was similar to other athletes including competitive swimmers during training [53]. In a study of endurance athletes, there were no RMR differences in postmenopausal runners and postmenopausal swimmers [26], which suggest that the type of endurance training is not as important as regular exercise. In a study that compared gymnasts and figure skaters to soccer players and controls, there was no difference in RMR and FFM between the two athlete groups and controls [54] but energy intake was lowest in the gymnast/figure skater group. However, there were differences in age and BMI between the groups, factors that could influence their results. A literature search on Pubmed (http://www.ncbi.nlm.nih.gov/pubmed) on RMR in other sports (rowing, football, tennis, lacrosse, etc.) did not yield any papers on this subject. Thus, there is limited research comparing RMR among different athletes with appropriate controls. Overall, the literature suggests that for male athletes, RMR ranges between 1788-1868 kcal/d and for eumenorrheic females between 1,328-1,486 kcal/d [45], with variations likely due to differences training volume and intensity. Reporting RMR relative to body weight or FFM may minimize the perceived differences that are observed when reported as kcal/day, with RMR ~25-28kcal/kg body weight and 28-31kcal/kg FFM in athletes [50, 55].

**CONCLUSION**

Assessing resting energy expenditure can be particularly useful to athletes whose athletic goals may be directly influenced by body composition. For athletes interested in assessing their energy needs, use of the Cunningham predictive equation [52] may result in the most accurate estimate of RMR, if direct or indirect calorimetry is not available. Habitual exercise appears to help maintain RMR with aging and even in periods of active weight loss, mainly through its effects on FFM. Thus, athletes should strive to optimize energy expenditure by maintaining lean mass with regular exercise throughout life.

**ACKNOWLEDGEMENT**

This work was supported by: VA Senior Research Career Scientist Award (ASR), VA Career Development Award (MCS), National Institutes of Health (R01-AG030075, Mid-Atlantic Nutrition and Obesity center, P30 DK072488, and the Claude D. Pepper Older Americans Independence Center, P30AG028747), and the Baltimore VA Geriatric Research, Education, and Clinical Center (GRECC).

**References**


