Metabolic Assessment of the Overweight Patient

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Introduction

Clinicians often see overweight patients seeking weight loss, and those seeking weight loss implore advice from nutrition specialists and dietitians on the quantity of calories they should consume each day. To produce weight loss, a negative energy balance needs to exist whereby the patient consumes less energy than he or she expends in a day. The total energy need of a person is expressed as:

Total daily energy needs (TDEE) = BMR + TEF + TEA + energy needed for growth, reproduction, lactation or healing from injury.

BMR: Basal Metabolic Rate TEF: Thermic Effect of Feeding TEA: Thermic Effect of Activity

The total amount of energy a person expends daily during the waking hours is termed Total Daily Energy Expenditure (TDEE) and is composed of three different components: the Resting Metabolic Rate (RMR), the Thermic Effect of Feeding (TEF), and the Thermic Effect of Activity (TEA) (see Figure 35.1). RMR is the energy expenditure needed to sustain the basic biochemical reactions of the body in a resting state. A resting state is when a person is fasting (not starving), awake in a thermoneutral environment (not sweating or shivering), and lies still without any skeletal muscle movement. The TEF is the energy expenditure attributed to the digestion and absorption of food. The energy needed for the digestion and absorption of foods is greater than that needed for resting, and therefore is designated as TEF. It is the difference in energy use between the fed and fasting states. The TEA is the energy expenditure associated with skeletal muscle movement. TEA is the most influenced component of TDEE because a person can choose to do variable amounts of physical activity that ultimately involve skeletal muscle movement. The RMR accounts for approximately 60 to 75% of TDEE, the TEF constitutes approximately 10%, and the TEA comprises 15 to 30% of energy expenditure.

24-Hour Energy Expenditure

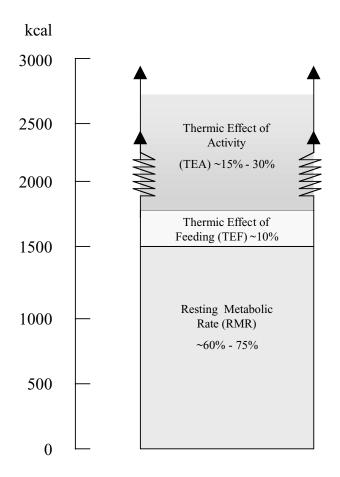


FIGURE 35.1

The three major components of the total daily energy expenditure (TDEE). RMR: Resting Metabolic Rate, TEF: Thermic Effect of Feeding, TEA: Thermic Effect of Activity. Adapted from Poehlman, E.T. *Med Sci Sports Exerc*, 21; 516, 1989.

Definitions of Energy Units and Components of Metabolism

Before introducing the definition of resting metabolic rate, it is important to define the energy unit. This is the "kilocalorie" or "kilojoule." The kilocalorie is the amount of heat content or energy required to raise the temperature of 1 kg of water 1 degree Celsius at 15 degrees Celsius; it is used in measurements of the heat production of chemical reactions including those of biological systems.¹ At any given time, there are continuous biochemical reactions consisting of the breakdown of adenosine triphosphate (ATP) to a smaller molecular of adenosine diphosphate (ADP) + ENERGY to serve the functional element of cells.² This reaction produces the energy that is measurable in kilocalories. The word kilocalorie may be used to define the amount of energy in the food a person consumes; it can also quantify the amount of energy a person expends.

RMR is presented as a measure of energy expressed as the amount of kcalories expended in a day, represented as kcal/d. However, the kilocalorie can also be expressed as the kilojoule to achieve uniformity of SI unit measuring system.¹ One kilocalorie equals 4.184 kilojoules. Although the joule may be a uniform standard unit that scientists use, the layperson will be better served when measurement of energy intake and expenditure is presented as kilocalories in order to make the term relevant to food labels and packaging used in everyday life. Those seeking to lose weight pursue a negative energy balance whereby they expend more kilocalories than consumed. A negative energy balance can be achieved by increasing TDEE or decreasing the amount of kcalories consumed during the weight loss period. Calorie is a word that many people associate with food labels to define the energy richness of a food item; it is often seen on exercise machinery stating the quantity of calories expended for a person of a given body weight for each minute of exercise. When investigating energy balance, it is important to understand the concept that a kcalorie is a measure of energy, and energy that is expended is measurable using different techniques. This section will be devoted to defining RMR and several components that make up TDEE, and it shall provide an overview of the methodology, implementation, and interpretation of the RMR.

TDEE

TDEE in free-living populations can be measured using doubly-labeled water (${}^{2}H_{2}{}^{18}O$). Measurement of TDEE by doubly-labeled water involves using stable isotopes of hydrogen and oxygen. A specimen of urine is collected from the subject at baseline; the administered dosage of doubly-labeled water is determined by body weight. Although this is the gold standard for the measurement of TDEE , doubly-labeled water is very costly and is almost exclusively used by scientists involved in measuring TDEE and various components of the metabolism for clinical research studies.

Usually, clinicians do not have the option of estimating TDEE by doubly-labeled water. Therefore, TDEE is typically estimated by measuring the RMR and estimating the energy expenditure of physical activity. Physical activity patterns may be assessed using accelerometers or by administering a valid questionnaire.

RMR

RMR is one of the three components that comprise total daily energy expenditure.³ RMR can be defined as the energy required to sustain bodily functions and maintain body temperature at rest, and is quantitatively the largest component of energy expenditure in humans. Typically it can account for 60 and 75% of the total daily expenditure.⁴ RMR is often used to estimate the daily energy needs of individuals for population-based studies. It is a useful tool in the clinical management of obesity.⁴ Researchers have defined RMR as being different from that of BMR; BMR is the energy expenditure of a person at rest (not asleep) in a fasting state, at sexual repose in a thermoneutral environment (neither shivering nor sweating). The RMR is defined as the energy expenditure measured on an outpatient basis.⁵ For clinical purposes, the RMR can be assumed to be similar to the BMR.⁵ It is less expensive and intrusive for the participant.

The RMR is the component of metabolism that is difficult to influence. Clinicians working with overweight patients will be quick to point out that their overweight clients often believe that they have a low or sluggish RMR, and consequently feel that this is the reason for their inability to lose or maintain weight. Often, RMR values are not as low as the patient believes. RMR measurements are frequently within the normal range for the patient's age, gender, height, and weight.

TEF

TEF is occasionally called diet-induced thermogenesis, and accounts for 10% of the TDEE of a person. TEF represents the energy expenditure associated with the ingestion, digestion, and absorption of food. There is an increase in the metabolic rate when a person has eaten food. This is why the assessment of RMR typically requires individuals to be fasted for a minimum of 12 hours prior to the assessment of that component of the metabolism. TEF can be measured by taking the measurements of a valid RMR assessment and comparing these to values attained using the same testing procedures after the ingestion of a meal of known energy value and composition. The difference between the RMR and the energy used for digestion and absorption is the TEF (TEF + RMR = energy expended after a meal is consumed).

TEA

The TEA is the only component of the TDEE that we can directly influence. TEA is the amount of energy expended as a direct result of voluntary skeletal muscle movement. TDEE differs between active and sedentary persons. Sedentary persons expend less energy than active persons, and thus their TEA as a percent of their total energy expenditure is less than that of active persons.

Techniques for Measuring RMR

RMR can be measured using two different methods: direct calorimetry and indirect calorimetry. Direct calorimetry is the measurement of overall heat liberated by a body mass. Heat production is proportional to the body surface area available (kilocalories/m²) for the release of heat by radiation or transvection.

Indirect calorimetry involves measuring oxygen consumption (O_2) and carbon dioxide production (CO_2) to determine RMR by using a calculated equation of Weir.⁶ To produce measurement values for indirect calorimetry in kilocalories per day, the measurement of one liter of oxygen consumed generates 3.9 kilocalories, and one liter of carbon dioxide produced generates 1.1 kilocalorie.⁷ The original Weir equation involves measurement of gases that are consumed and produced at rest plus the collection of total 24-hour urine nitrogen during the same day of measurement. However, a second abbreviated Weir equation has been developed that is less than 2% measurement error when compared to the longer equation.⁶ This equation is below.

Abbreviated Weir Formula:

RMR =
$$[3.9(V_{O_2}) + 1.1 (V_{CO_2})]1.44$$

variables: V_{O_2} = oxygen consumption in mL/min

 V_{CO_2} = carbon dioxide production in mL/min

Note: this equation is to determine resting energy expenditure, so for determining RMR the patient must be in a 12-hour fasted state.

Direct Calorimetry

The measurement of energy need of an adult who is neither gaining nor losing weight can be made using a whole body calorimeter. This instrument measures the heat released by the body as a result of its metabolism.⁷⁻⁹ It is a composite value in that it is not only the result of baseline metabolic reactions that produce heat, but also the heat that results from the ingestion, digestion, and absorption of foods, plus that which results from muscular activity. Subjects must remain in the whole body calorimeter for hours at a time so that sufficient data can be accumulated. This technique is extremely expensive and has limited clinical potential.⁷

Indirect Calorimetry

For most, a viable alternative is the indirect calorimeter or metabolic cart. Indirect calorimetry measures the gas exchange of an individual.^{8,10} The gases detected by the metabolic cart are compared to the environmental conditions of the surrounding room's gases at standard temperature, pressure, and humidity (STPD). STPD is a symbol indicating that a gas volume has been expressed as if it were at standard temperature (0° Celsius), standard pressure (760 mm Hg absolute), and 0% humidity; under these conditions, a mole of gas occupies 22.4 liters. The testing environment should be controlled for temperature, barometric pressure, and humidity. Depending on the instrumentation, the measurement conditions are entered prior to beginning the assessment, and correction factors are applied to standardize the results. The room temperatures should also be kept between 68 and 74° Fahrenheit. Furthermore, the room should be dimly lit, and spare blankets should be offered to individuals who may experience coldness when sitting for prolonged periods of time.

The measurement of gas exchange allows for the calculation of the respiratory quotient (RQ). (RQ = CO_2/O_2). The RQ reflects cellular metabolism and is a reflection of heat production (direct calorimetry). The RQ indicates the fuel mixture being oxidized.⁸ Different fuels such as fats, carbohydrates, and proteins require different amounts of oxygen for oxidation to CO_2 and water. Thus, the RQ varies depending on the ratio of fat to carbohydrate in the fuel mixture.¹¹ In starvation, the major fuel is fat, and the RQ is 0.70. Usually a mixture of fuels (carbohydrate and fat) is oxidized. The various substrates and their RQ values are shown in Table 35.1.

Respiratory Quotient and Energy Content of Various Substrates					
Fuel (Substrate)	Energy Content (Kcal · g ⁻¹)	Respiratory Quotient (RQ)			
Carbohydrate	4.1	1.00			
Fat	9.3	0.70			
Protein	4.3	0.80			

TABLE 3	85.1
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Respiratory Quotient and Energy Content of Various Substrates

Adapted from American College of Sports Medicine. *Guidelines for Exercise Testing and Prescription*. Malvern, PA: Lea & Febiger, page 14, 1991.

Instrumentation Available

There are two indirect calorimetry systems: open-circuit and closed-circuit systems. Both techniques require devices to measure the concentration of O_{2} , CO_{2} , gas volume or flow

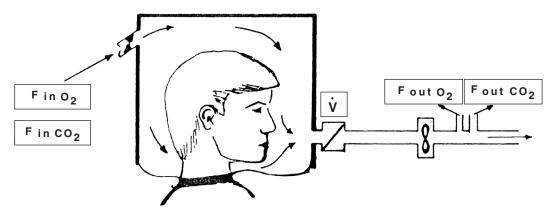


FIGURE 35.2

Open Circuit technique of Indirect Calorimetry using a canopy hood. From Ferrenini E. *Metabolism*, 37; 296, 1988. With permission.

Labels: FinO₂: Forced Inspiratory Oxygen; FinCO₂: Forced Inspiratory Carbon Dioxide; : Gas Flow; FoutO₂: Forced Expiratory Oxygen; FoutCO₂: Forced Expiratory Carbon Dioxide.

rate, temperature, and time. In the closed-circuit system, the patient breathes from a reservoir (a mixture of gases resembling ambient air), and the decrease in oxygen over time is used to calculate \dot{V}_{O_2} . Closed-circuit systems are usually simpler in design and less costly than open systems. Open-circuit systems are more versatile and can be more easily used in the clinical setting.⁷ The patient breathes from a reservoir of air of known composition in the closed-circuit system; the depletion of oxygen, $\dot{V}C_{O_2}$ and \dot{V}_{O_2} are calculated.⁷ In the open-circuit technique (see Figure 35.2), the patient breathes room air and expires into a gas sampling system which eventually vents the expired air back into the room. Open-circuit systems are more commonly used to measure RMR in the clinical setting, since they are more versatile and can be used in a variety of clinical conditions. The techniques described in this section will therefore focus on the open-circuit indirect calorimetry system.

Types of Collection Systems

Many types of accessories allow for the collection of consumed and expired gases of the person being tested, including face masks, mouth pieces, chambers, and ventilated hoods.

Face Masks

Similar to the face masks used by firefighters and military personnel, face masks provide a sealed environment around the nose and mouth in order to collect all gases. The face mask has an elastic head harness which encompasses the back of the head. These work well for collection of gases; however, they may be more awkward for some patients than other collection systems, and it is important to have several sizes of masks on hand to optimize fit. Although not as comfortable as some of the other collection devices, the face mask is very easily used with portable gas analyzers and is useful for field settings or where exercise-induced energy expenditure is measured.¹²

Mouthpieces

These are similar to the snorkel that is used to allow breathing underwater. The mouthpieces used for RMR measurement are usually identical to mouthpieces used for maximal V_{O_2} testing. In order for the mouthpiece to work correctly, the subject being tested needs to maintain a tight seal around the mouthpiece and have a nose clip sealing off the nasal passageway. A certain amount of discomfort may be experienced from a static contraction of the jaw muscles to keep a tight seal around the mouthpiece. Therefore, this collection system is not often used to assess RMR.

Ventilated (Canopy) Hood

The ventilated hood is the most widely used collection system. It is advantageous for a number of reasons — it allows for easy spontaneous breathing in apparently healthy individuals, there is no error associated with facial features such as beard or facial hair of the test subject, it has been found to be accurate in long term measurement of RMR, and it is a relatively noninvasive gas collection system. The hood drapes over the entire head of the subject in a semirecumbent position.

Comparison of Collection Devices

There are advantages and disadvantages for each collection device. In the clinical setting, the ventilated hood may offer a more relaxed and unobtrusive measurement environment. However, for patients that may be claustrophobic, the lights of the laboratory may need to be dimmed to reduce feelings of being confined. The face masks work well for collection of gases; however, structural differences in the size of the face may make it necessary to use different sized masks for the variety of structural differences found in patients. Furthermore, face masks are expensive, and for valid measurements a variety of masks is necessary. Finally, although mouthpieces may seem to have no limitations, they are difficult for patients when the measurements last for long periods of time. Moreover, a nose clip must be placed on the nose during measurement to prevent any escape of non-measured gas, and this can be extremely invasive for the patient as well.

Clinical Applications and Usefulness of RMR

Understanding the energy requirement of an individual can be useful in prescribing a personal dietary intake. The interpretation of the values attained by RMR measurements should be done by an experienced clinician. Typically, university hospitals and established university weight loss centers will have access to metabolic carts to perform RMR assessments. An accurate measure of RMR will allow the clinician to tailor the energy intake of the individual (and increase overall energy expenditure via the thermic effect of activity) in order to produce a negative energy balance.

This information may also be important in the estimation of TDEE. The values of RMR may be multiplied by an activity factor to produce best estimates of TDEE, as outlined in Table 35.2. It may be helpful get detailed recent exercise histories from patients to help assess the appropriate level of general activity. Bear in mind that most people overestimate their activity levels.

TABLE 35.2

Factors for Estimating Total Daily Energy Needs of Activities for	
Men and Women (Age 19 to 50)	

Level of General Activity	Activity Factor (Multiplied by REE ⁺)	Energy Expenditure (Kcal/kg/day)
Very light		
Men	1.3	31
Women	1.3	30
Light		
Men	1.6	38
Women	1.5	35
Moderate		
Men	1.7	41
Women	1.6	37
Heavy		
Men	2.1	50
Women	1.9	44
Exceptional		
Men	2.4	58
Women	2.2	51

From Food and Nutrition Board, National Research Council, NAS: Recommended Dietary Allowances, 10th ed. Washington, DC. National Academy Press, 1989, p. 29 (with permission). See Heshka S., Feld K., Yang M.U. et al. Resting energy expenditure in the obese: A cross-validation and comparison of prediction equations. *J. Am. Diet. Assoc.* 93, 1031-1036, 1993 for a comparison of various prediction equations.

⁺ REE = resting energy expenditure.

Helping Patients Gain Weight

RMR values attained from indirect calorimetry can also be used for certain anabolic circumstances. Often, hospital clinicians will measure RMR in patients suffering from severe burns or frail, elderly patients as a result of the onset of disease. For these instances, the values attained at bedside are important in tailoring meal plans to facilitate weight gain in life-threatening medical situations.

Those individuals seeking to gain weight for performance purposes can also benefit from accurate measures of RMR. Individuals who seek to increase overall lean body mass (or fat-free mass) may wish to understand how much energy is required above maintenance to produce a safe rate of weight gain. The values attained from RMR in conjunction with counseling on an appropriate activity and exercise program may be helpful to individuals training for body-building or sports performance-related events. Two case studies depicting the usefulness of values attained from assessment or prediction of RMR are described below.

Case Studies

Person Seeking Weight Loss

A 40-year-old woman with a height of 5'6" and weight of 185 pounds (BMI of 30 kg/m²) seeks to lose 20 pounds. This person seeks treatment from a dietician in a hospital that has no metabolic cart. Therefore, an equation to predict resting metabolic rate will be used. Using a prediction equation that has a table with value of kcals based on age and gender multiplied by body surface area,¹³ the RMR is predicted to be 1498 kcal/d. Furthermore,

the woman participates in 30 minutes of vigorous aerobic exercise 4 days per week. Therefore, to determine her predicted TDEE, we multiply her RMR by an activity factor that is equal to a moderately active person. This activity factor is 1.5. Multiplying 1498 by 1.5 yields a TDEE of 2247 kcal/d. To predict a safe rate of weight loss of 1.5 pounds per week, the energy intake needs to be restricted by 750 kcals per day, equaling a consumption of approximately 1447 kcal/d (if 3500 kcal equals one pound of weight loss).

Person Seeking Weight Gain

A 20-year-old man who is weight training and agility training over a 15-week period seeks to gain 15 pounds for the start of fall football season. He is 6'5," weighs 270 pounds, and has 18% body fat. Fortunately, he lives near a university hospital that has dieticians who specialize in sports nutrition and have access to a metabolic cart. When assessed for RMR, the man has a baseline RMR of 2653 kcal/d. The man's training habits, which involve two hours of strength and agility training each day, warrant that his RMR be multiplied by an activity factor of 1.7. His determined TDEE is therefore 4510 kcal/d. However, he seeks to gain weight and not remain weight stable. Therefore, in order to predict an average of 1 pound of weight gain per week, the man needs to ingest 500 kcal/d more than his predicted TDEE. This value is equal to 5010 kcal/d.

Predicting RMR

Often, technology for direct measurement of RMR is not readily available to healthcare professionals who provide treatment plans based on RMR values. As an alternative, clinicians frequently use prediction equations to estimate RMR. These prediction equations have mostly been developed using regression equations to fit functions according to gender, age, height, weight, and other available clinical variables.¹⁴ A majority of these equations were developed using normal-weight persons who were relatively sedentary. Unfortunately, this poses a problem when predicting RMR in the obese population, considering that RMR is directly related to the fat-free mass (FFM) of the individual,¹⁵⁻¹⁸ and the obese person has a larger distribution of adipose tissue and a decreased proportion of FFM when compared to their normal-weight counterparts.¹⁹ Equations are available for the prediction of RMR for the normal weight and overweight populations (Table 35.3).

Harris-Benedict Equation

For normal-weight individuals (determined by BMI or body composition analysis), the prediction equation of Harris and Benedict²⁰ offers an appropriate equation:

Men: kcal/d = 66.4730 + 13.751W + 5.0033L - 6.750A Women: kcal/d = 655.0955 + 9.563W + 1.8496L - 4.6756A

where W = weight (kg); L = height (cm); A = age (years)

These equations were developed in 1919 and are currently widely used by clinicians. However, given the increased prevalence of overweight in industrialized countries like the U.S.,²¹ it may be necessary for the clinician to use prediction equations that have been validated using overweight persons.

TABLE 35.3

Reference	Equations		
Bernstein et al.	W: 7.48(kg) – 0.42(cm) – 3.0(yr) + 844	81	
	M: $11.0(kg) + 10.2(cm) - 5.8(yr) - 1,032$		
Cunningham	501.6 + 21.6(LBM); where	82	
	for W: LBM = $[69.8 - 0.26(kg) - 0.12(yr)] \times kg/73.2$		
	for M: LBM = $[79.5 - 0.24(kg) - 0.15(yr)] \times kg/73.2$		
Harris and Benedict	W: $655 + 9.5(kg) + 1.9(cm) - 4.7(yr)$	20	
	M: $66 + 13.8(kg) + 5.0(cm) - 6.8(yr)$		
Fleisch ^a	W/M: kcal's/m ² of BSA from Fleisch table × [[0.007184 × (kg)] ^{0.425} × (cm) ^{0.725}]] × 24	22	
James	W: 18 – 30yr: 487 + 14.8(kg)	83	
	30 - 60 yr: $845 + 8.17$ (kg)		
	>60yr: 658 + 9.01(kg)		
	M: 18 – 30yr: 692 + 15.1(kg)		
	30 – 60yr: 873 + 11.6(kg)		
	>60yr: 588 + 11.7(kg)		
Mifflin et al.	W: $9.99(kg) + 6.25(cm) - 4.92(yr) - 161$	23	
	M: $9.99(kg) + 6.25(cm) - 4.92(yr) + 5$		
Owen et al.	W: $795 + 7.18$ (kg)	84	
	M: 879 + 10.2(kg)		
Pavlou et al.	M: -169.1 + 1.02(pRMR)	85	
Robertson and Reid ^a	W/M: kcals/m ² of BSA from Robertson and Reid table × [[0.007184 × (kg)] ^{0.425} × (cm) ^{0.725}]] × 24	13	

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Equations	tor Estima	tino Restinc	r Metabolic	Rate (RMR) kcal/24 hours ^a
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Key. W = Women, M = Men, pRMR = predicted RMR from the Harris and Benedict equation, ^athis equation uses tabled values for kcals/m². From Heshka S., Feld K., Yang M.U. et al. Resting energy expenditure in the obese: A cross-validation and comparison of prediction equations. Copyright The American Dietetic Association. Reprinted with permission from *J. Am. Diet. Assoc.* 93, 1031-1036, 1993.

Robertson-Reid and Fleisch Equations

Three prediction equations may potentially offer reasonable predictions of RMR for the overweight patient. The prediction equations of Robertson and Reid¹³ and the equation of Fleisch²² have been recommended for obese populations.¹⁴ The Robertson and Reid and Fleisch equations will be presented in this section as viable prediction equations for the obese patient. The Robertson and Reid equation was derived from the actual measurement of RMR of 987 men and 1323 women age 3 to 80. The equation requires that the clinician find a value for heat output (in table form) based on the patient's age and gender. Subsequently, this value is multiplied by the body surface area of the person (in m²) to determine kcal/hr; this number is then multiplied by 24 to yield daily RMR. The basis of this prediction equation is that there is constant heat output that corresponds to surface area within people who are the same gender and age:

RMR in kcal/d = heat output in kcal × body surface area in $m^2 \times 24$ hours heat output = value derived for men and women from a table developed by Robertson and Reid¹³ body surface area (BSA) = (([0.007184 × (wt in kgs)]^{0.425} × (ht in cms)^{0.725})) time = 24

The equation of Fleisch uses the same concept as the Robertson and Reid equation. However, the values of heat output differ, and Fleisch provides a separate table to calculate the predicted RMR.²²

Mifflin et al. Equation

A third prediction equation for the obese population comes from Mifflin et al.²³ This equation was derived using linear regression analysis on a subset of patients (247 females, 251 males) who had their RMRs measured using indirect calorimetry. In an unpublished research study at the Johns Hopkins School of Medicine, the equation has provided predicted RMR values in obese patients at a university weight loss center that are not different than those produced by actual measurement. The equation is as follows:

Men (kcal/d) = 9.99(kg) + 6.25(cm) - 4.92(years of age) + 5Women (kcal/d) = 9.99(kg) + 6.25(cm) - 4.92(years of age) - 161

Pretesting Procedures for Measurement of RMR

The testing procedures for determining RMR necessitate that a strict protocol be followed to ensure that the measurement is accurate. Individuals should be provided with pretesting requirements for the RMR estimate. The subject should be questioned about his/her adherence to pretest procedures prior to the test. If one or more of these procedures are not followed, the individual should be rescheduled at a later date to reduce measurement error.

Weight Stable

If the subject has experienced recent weight loss, measurement of RMR may not be valid. Measurement should be avoided if the person being tested is on a weight loss program or has lost or gained more than one pound in the past week. To reduce error associated with physiological responses to weight loss, a period of weight stabilization of two weeks is necessary prior to an RMR assessment.

Well Rested

RMR measurement should be administered as close to the time a person awakes as possible. Additionally, the individual being tested should get a restful night's sleep prior to coming to the clinic or hospital for the RMR assessment. If an individual has had an uneasy night's rest prior to RMR assessment, confounding environmental influences may unduly increase the metabolic expenditure of the individual. Measurement should occur before 10:00 am; measurements taken in the late morning may be suspect to increased metabolic activity.

Fasted

Measurements should be taken first thing in the morning after a 12-hour overnight fast.²⁴ A light meal the night before measurement should be encouraged; RMR is the energy expended at rest in a fasted state, and therefore any lasting effects of food or drink would contribute additional energy expenditure from diet-induced thermogenesis. Early morning coffee or tea should be avoided, and only water should be ingested prior to mea-

surement. Clinicians can determine whether the patient is fasting by examining the RQ values during testing.

Measurement in Relation to Last Exercise Bout

Testing should be performed at least 24 hours after rest from exercise to eliminate any residual effects from the most recent training session.²⁵ Hence, the person being testing should be instructed to abstain from programmed exercise for at least one day prior to measurement of RMR.

Location and Acclimation to the Testing Environment

Studies have shown that there are no differences in measurements of RMR performed with or without an inpatient overnight stay.⁵ Therefore, to avoid excessive costs associated with inpatient stays, an outpatient procedure is usually recommended. Upon arrival to the lab where the RMR assessment will occur, the subject should be instructed to rest in a sedentary supine or semirecumbent position for at least 30 minutes prior to the assessment. During this time, the subject should remain still. The subject should be asked if they need to void, since it is necessary that they are comfortable during the entire test. Some laboratories will further acclimatize the individual by placing the collection system (canopy hood) over him/her to ensure that he/she is comfortable with the measurement setting.

Analysis

The metabolic cart will often express resting metabolic rate as the mean of multiple minute measurements. However, the cart will also provide continuous measurements of V_{O_2} and V_{CO_2} . Many researchers suggest that the calculation of RMR be the average of five of these continuous minute measurements of steady state.^{18,26} Steady state is 5 minutes of measurement of V_{O_2} and V_{CO_2} that possess an intravariability of 5% or less.²⁶

Table 35.4 is a simple checklist to ensure that the assessment of RMR is valid.

Factors Affecting RMR

A variety of factors have been shown to influence RMR, including genetics, age, gender, total body weight, fat-free weight, aerobic fitness level, total energy flux through the body, body and/or environmental temperature, hormonal factors, drugs, and stress.²⁷ Of these factors, the strongest correlation exists between an individual's FFM and RMR,²⁷ and collectively, fat-free mass, age, sex, and physical activity account for 80 to 90% of the variance in resting metabolic rate.²⁸

Exercise

Since exercise training has been associated with increases in fat-free mass, this is one factor which can be manipulated to potentiate resting metabolism. Cross-sectional studies have

TABLE 35.4

Checklist for RMR Testing

Pre-Testing Subject Requirements

- ▶ 12 hour fast. Water is allowed ad libitum.
- ▶ Refrain from strenuous physical activity/exercise for 48 hours prior to testing.
- Well Rested. Make sure subject has at least 8 hours of sleep.
- Minimize activity the morning of test. Light grooming allowed. Shower the night prior.
- ✓Keep food diaries for 48 hours prior to testing. Dinner meal should be <1000 kcals night prior.

Laboratory Requirements

- ✓ Room should be isolated to reduce any external noise.
- ✓ Room should be dimly lit, but not dark.
- ✓ Temperature should be controlled and ideally at 22° to 24° Celsius. Blankets should be used if subject is cold.
- The bed or comfortable chair should be semi-recumbent and not flat; having a slight incline of approximately 10 degrees.

Testing Procedures

Monitor subject during testing. Direct subject to avoid any: talking, fidgeting, and sleeping.

✓ Acclimate patient to test. Possibly perform practice test prior to actual procedure.

Rest subject in semi-recumbent position for at least 30 minutes prior to testing.

✓ If able to, use HR monitor to track HR the day before, morning of and during test.

Authors would like to thank Dr. Jack Wilmore for the helpful suggestions for the RMR checklist.

demonstrated that aerobically trained individuals have higher resting metabolic rates for their metabolic size than their untrained counterparts.²⁹⁻³²

Age

Age is another variable that has been found to have a significant impact on an individual's resting metabolism. In fact, the decline in resting metabolic rate is one of the most consistent physiological changes that occurs with age.³³ Recent studies have suggested a curvilinear reduction in RMR with advancing age that is accelerated beyond middle-age and post-menopausal years.³³ Several studies attribute the age-related decline in RMR primarily to the loss of fat-free mass that often accompanies aging; however, there remains uncertainty whether other physiological factors may also contribute to the reduction of RMR.³³

Gender

Gender differences in resting metabolism have also been reported, with males having a higher RMR than females by approximately 50 kcal/day.²⁸ This difference is independent of the gender difference in fat-free mass, and is consistent across the life span.²⁸ Menopausal status has also been pinpointed as an influence on RMR in women. Studies have found lower RMR in postmenopausal women relative to premenopausal women, which was again primarily attributable to reductions in lean mass and a decline in aerobic fitness.³³⁻³⁵

Environment

Environmental factors also influence RMR, with the resting metabolism of people in tropical climates typically 5 to 20% higher than that of their counterparts living in more

temperate areas.³⁶ Cold climates also have a significant impact on resting metabolic rate that is dependent on an individual's body fat content and the amount and type of clothing worn.³⁶ During extreme cold stress at rest, metabolic rate can double or triple with shivering as the body attempts to maintain a stable core temperature.³⁶

Cigarette Smoking

Some studies have documented the influence of such substances as cigarettes, caffeine, alcohol, and certain medications on resting metabolism. Many lay persons believe that cigarette smoking may be helpful in maintaining body weight,³⁷ and many smokers are unwilling to quit because of their fear of weight gain. Over time, studies have demonstrated that the increase in metabolic rate resulting from cigarette smoking is transient.³⁷ One study found no effect in habitual smokers when assessment of metabolic rate did not begin until 25 to 30 minutes after smoking. Thus, it is thought that the acute metabolic effects of cigarettes are not significant beyond 30 minutes after smoking. Yet, given the typical ~30 minutes between cigarettes for most smokers, RMR may remain slightly elevated throughout the day as a result of these "acute" effects.³⁷

Caffeine

Caffeine has been identified as a substance that elevates metabolic rate, and caffeine ingestion has also been shown to increase work performance and promote lipid oxidation during prolonged exercise.³⁸⁻⁴⁰ In a study investigating the influence of caffeine on the resting metabolic rate of exercise-trained and inactive subjects it was found that metabolic rate was increased in response to a stimulus of approximately two cups of coffee (300 mg).³⁸ This study also compared regular and non-regular caffeine consumers to investigate the effects of consumption levels on metabolic response. This investigation confirmed previous findings that with regular consumption, the physiological and stimulatory effects of caffeine were not diminished.³⁸

Alcohol

Alcohol is another substance that has been found to influence resting metabolic rate. Alcohol is decidedly the most commonly consumed psychoactive drug in the U.S., and because of its energy density, it is widely believed to be a causal factor in the development and maintenance of obesity.⁴¹ However, in a study utilizing data gathered in two national cross-sectional surveys — the Second National Health and Nutrition Examination Survey (NHANES II; n = 10929) — and the Behavioral Risk Factor Surveys (BRFS; n = 18388), it was found that alcohol consumption had a slight negative effect on the body weights of men, and a profound negative effect on the body weights of women.^{41,42} This negative effect was not a result of lowered dietary intake among drinkers. In fact, in controlled isoenergetic dietary studies, subjects tended to lose weight on alcohol-containing regimens.^{43,44} This has lead to the hypothesis that alcohol intake may increase resting energy expenditure.⁴¹ Early studies found inconsistent evidence regarding the effects of alcohol on resting energy expenditure.^{41,45} However, recent studies have found evidence in support of the hypothesis that alcohol may increase resting energy expenditure,^{41,46} although further investigations are needed to explain the mechanism by which alcohol suppresses body weight.41

Medications

Medications are also known to impact resting metabolism. Beta-blocking medications, for example, are prescribed to several million Americans with cardiovascular disease to treat conditions such as hypertension and angina.⁴⁷ Unfortunately, despite their widespread use in medical practice, beta-blocking medications have many side effects. One such side effect is the influence on resting energy expenditure. Research indicates that resting energy expenditure and perhaps the energy needs of individuals treated with beta-blockers are reduced.⁴⁷ The magnitude of this reduction in resting metabolic rate has been found to vary between 8 and 17%.⁴⁷ One study reported a reduction in resting metabolism of approximately 17% or 4 kcal/kg/day in a group of healthy subjects taking 80 mg of propranolol twice daily for 5 days.⁴⁷ This could result in significant weight gain in a patient receiving beta-blockers long-term if no changes were made to both dietary and exercise habits.⁴⁷

RMR and Weight Loss

America currently has a preoccupation with weight loss and as a result, for many years scientists have been interested in identifying interventions that might potentiate RMR to facilitate weight loss in overweight and obese patient populations.²⁷ Factors causing a decrease in resting metabolic rate would make weight maintenance or weight loss difficult, or possibly result in weight gain. Conversely, anything that increases resting metabolic rate would potentially facilitate weight loss and maintenance of the weight lost.⁴⁸

Energy Restriction and RMR

Over the past decade, there has been a dramatic increase in the prevalence of overweight and obesity in adults as well as children and adolescents. Using data from the National Health and Nutrition Examination Survey (NHANES III) it has been reported that more that 33.4% of U.S. adults are overweight,^{21,49} representing an increase of 8% over the past 10 years;^{50,51} paradoxically, dieting has become a way of life for many Americans. In a study utilizing data from the 1996 state-based Behavioral Risk Factor Surveillance System, it was reported that 28.8 and 43.6% of men and women, respectively, trying to lose weight at any given time.52 Researchers have been exploring the consequences of dieting particularly those related to changes in the resting metabolic rate.²⁶ Several investigators have found that a restrictive diet depresses resting metabolic rate, which may contribute to the regaining of weight often observed after treatment. One such study found that the RMR of obese individuals decreased during a protein-sparing modified fast, and remained depressed for two months after treatment despite increased energy consumption to a level that allowed body weight stabilization.⁵³ Similar findings were reported by Heshka et al.¹⁹ in participants of a conservative weight-loss program. It was found that resting metabolic rate declined to a greater degree than would be expected from loss of lean mass alone.¹⁹ Other investigators have found no adverse effects on RMR, and have concluded that any decline in resting metabolism is fully explained by an anticipated reduction in fat-free mass accompanying weight loss.²⁶ A study examining the short-term and long-term effects of very low-calorie diets (VLCDs) observed a 17.3% decrease from baseline of resting energy expenditure after patients consumed 500 kcal/d for just 2 weeks.⁵⁴ This reduction

in resting metabolism was associated with a weight reduction of only 5.8%. There was, however, an observed rebound in RMR accompanying the patients' return to a 1000 to 1200 kcal/d balanced diet, and the 11% end-of-treatment decline in RMR was paralleled by a 12% reduction in body weight.⁵⁴

It appears that RMR declines rapidly in response to energy restriction. Reductions as great as 30% have been reported in some individuals.²⁷ Very low-energy diets, in particular, have been found to be associated with substantial short-term reductions in RMR.²⁶ This decline, however, appears to be attributable primarily to the caloric restriction, and is largely reversed when dieting is stopped.²⁶ With weight stability, reductions in RMR have been found to be modest, and are highly related to the changes in fat-free mass.²⁶ It is thought that physical activity, energy deficit, macronutrient distribution, and rate of weight loss may be key factors in the retention of fat free mass, and by extension, RMR.

Exercise and RMR

Many effects of exercise are thought to be beneficial to weight loss and weight maintenance. A single bout of exercise produces an increase in energy expenditure, the magnitude of which is dependent on the intensity, duration, and type of exercise.¹⁵ Weight-bearing activities such as walking, jogging, and cross-country skiing lead to energy expenditure that is directly related to body weight, and may be of particular benefit to obese individuals.¹⁵ Muscle-strengthening exercises may produce an added advantage by maintaining or increasing muscle mass. Some investigators have proposed a carryover effect of exercise on metabolic rate; however, if any long-term effect exists, it is thought to only occur after very vigorous and sustained physical activity.¹⁵

Both resistance and endurance training have therefore been proposed as interventions that might potentiate RMR to facilitate weight loss in overweight and obese patient populations. Findings from several cross-sectional studies have indicated that athletes demonstrate a higher RMR than sedentary individuals, and training studies indicate that sedentary individuals who are not restricting energy can increase their RMR by beginning a regular exercise program.⁴ Despite these findings, the research literature regarding the effects of resistance and endurance training, separately or in combination, on elevating the RMR is mixed, and whether exercise training enhances RMR remains a controversial question.²⁵

Resistance Training and RMR

Resistance training is thought to have the potential to increase RMR by increasing fat-free mass.²⁵ This belief is founded on the significant relation between fat-free mass and RMR. Heavy resistance training promotes skeletal-muscle development, which could have a favorable impact on a person's RMR by increasing the total amount of metabolically active tissue.²⁷ However, the extent to which resistance training is able to increase RMR has not been well documented, and studies evaluating the impact of high-intensity resistance exercise on body composition and other physiological adaptations during weight loss have reported inconsistent findings. In one longitudinal study comparing the effects of strength training and aerobic exercise on body composition, body weight, and RMR in healthy, non-dieting young men, the resistance training was associated with increased strength and FFM, but body weight and RMR did not change significantly.²⁷ These findings were corroborated by a similar study with untrained female subjects in which a statistically significant increase in RMR was not observed despite favorable alterations in body composition.¹⁸ Further studies of longer duration are needed to determine whether a significant increase in RMR would be observed with a longer resistance training program.¹⁸

Endurance Training and RMR

Physical activity, especially in the form of endurance exercise, significantly affects energy intake and expenditure, and is therefore a key regulatory component in the energy balance equation.

After exercise, oxygen consumption decreases rapidly but may remain above resting levels for several hours or even days after the bout of activity.⁴ The repair of damaged muscle tissue and the resynthesis of substrates such as CP, ATP, and glycogen partially account for the excessive post-exercise oxygen uptake (EPOC) in the exercised muscles, and may be the cause of the elevated muscle oxygen uptake in recovery. Bullough⁵⁵ reported that RMR was greater in trained than untrained subjects only when trained subjects were in a state of recovery from vigorous exercise. Their data indicate that RMR is influenced by exercise, energy intake, and their interaction, and suggest that higher RMR in trained versus untrained individuals results from acute effects of high-intensity exercise rather than from a chronic adaptation to exercise training.

Phelain et al.⁵⁶ have examined the effects of low- and high-intensity aerobic exercise of similar energy output on post-exercise energy expenditure and substrate oxidation in fit eumenorrheic women. They used continuous indirect calorimetry performed during cycle ergometry exercise and for three hours after low-intensity exercise (500 kcal at 50% VO₂ max) or high-intensity exercise (500 kcal at 75% VO₂ max). Mean EPOC for the three-hour post-exercise period for high-intensity exercise (9.0 \pm 1.7 L, 41 kcals) was significantly greater than that for the lower intensity activity $(4.8 \pm 1.6 \text{ L}, 22 \text{ kcals})$. Oxygen consumption (VO₂) following the higher intensity exercise remained elevated at the end of the three-hour post-exercise period, but was not with the low intensity group. Quinn et al.⁵⁷ reported that exercise duration increases EPOC significantly, and that a 60-min bout of aerobic exercise yields approximately twice the EPOC than either 20 or 40 min in trained younger women. However, Almuzaini et al.⁵⁸ examined the effects of splitting a 30-min exercise bout of cycling into two equal sessions versus a single uninterrupted session. They compared the effects of these two exercise trials on EPOC and resting metabolic rate (RMR) and concluded that dividing a 30-min exercise session into two parts for these individuals significantly increases magnitude of EPOC but does not affect RMR.

Short and Sedlock⁵⁹ also found that fit individuals have faster regulation of post-exercise metabolism when exercising at either the same relative or absolute work rate than their untrained counterparts. Gillette and colleagues⁶⁰ also compared strenuous resistive exercise to steady-state endurance exercise of similar estimated energy cost. They found that the resistance training resulted in a greater excess post-exercise V_{O2} compared to the aerobic exercise.

Energy Restriction Combined with Exercise Training: The Effects on RMR

Dietary restriction without exercise does not appear to be an optimal strategy to promote weight loss and weight maintenance.⁶¹ Additionally, in clinical practice, exercise when used alone has not been viewed as an optimal means of weight reduction.⁶¹ This may be attributed in part to the difficulty many patients have in maintaining an appropriate program of physical activity.

Ballor and Poehlman⁶² performed a meta-analysis to examine how exercise training and gender influence the composition of diet-induced weight loss. They found that diet-plus-exercise training (DPE) groups did not differ from dietary-restriction-only (DO) groups with respect to either the amount of body weight lost (mean = -10 ± 1.4 kg) or fat mass lost (mean = -8 ± 1.1 kg). Exercise training, however, attenuated the amount of body weight lost as fat-free mass compared to DO for the same sex. The percentage of weight

□ Men **■** Women

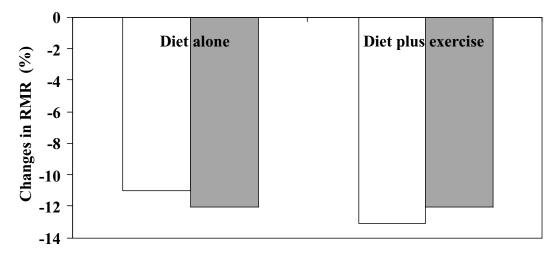


FIGURE 35.3

Changes in resting metabolic rate following interventions of diet plus exercise training or dietary restriction alone. Data adapted from Ballor, D.L. and Poehlman, E.T. *Eur J Appl Physiol*, 71; 535-42, 1995.

lost as fat-free mass for DPE subjects was approximately half (P <0.05) of that for DO subjects of the same sex. The DO males lost $28 \pm 4\%$ of weight as fat-free mass, while DPE males lost $13 \pm 6\%$. The DO females lost $24 \pm 2\%$ of their weight from lean mass compared to the DPE females, who only lost $11 \pm 3\%$ of their weight from the FFM. These data provide evidence that exercise training reduces the amount of fat-free mass lost during diet-induced weight loss. In addition, gender differences do not seem to exist with respect to body composition changes of weight reduction.

The decline of RMR in response to energy restriction has been well documented, and is suspected to decrease the rate of weight loss during periods of energy restriction.⁶³ Exercise is frequently advocated in the treatment of obesity as a means of increasing energy expenditure and potentially counteracting the negative effects of dietary restriction.¹⁶ Several studies based on the addition of a component of exercise to dietary restriction have been published.^{15,16,49,61,64} Some studies have continued to report similar decreases in RMR, whereas others have shown an attenuation of the decrement, or an increase in RMR when an element of exercise was added. Ballor and Poehlman conducted a meta-analysis to examine the independent and interactive effects of dietary restriction, endurance exercise training, and gender on RMR.⁶⁵ Collectively, weight loss was greater (P < 0.05) for men (18 kg) than for women (12 kg). They found no exercise training or gender effects on RMR during weight loss. Collectively, dietary restriction resulted in a -0.59 kJ min⁻¹ (approximately -12%) decrease in RMR (P <0.05). When normalized to body weight, RMR was reduced by less than 2% (P < 0.05). These data suggest that exercise training does not differentially affect RMR during diet-induced weight loss. In addition, decreases in resting metabolism appear to be proportional to the loss of the metabolically active tissue (Figure 35.3).

Energy Restriction Combined with Resistance Training: The Effects on RMR

In theory, strength training should attenuate the decline of RMR if it preserves fat free mass by preventing atrophy of skeletal muscle. Skeletal muscle contributes more than

50% of the fat free mass of the body.⁶⁴ It is for this reason that resistance training was initially added to weight-loss programs: to reduce or prevent the loss of muscle during energy restriction, which, in theory, should attenuate the drop in RMR typically seen with weight loss.

Few studies have been conducted that combined diet with heavy resistance exercise, and studies evaluating the impact of high-intensity resistance exercise on body composition and other physiological adaptations during weight loss have reported inconsistent findings.⁶¹ Conflicting results regarding the impact dietary restriction combined with resistance training has on lean body mass have been reported. An additional problem in evaluating the impact of resistance training is the fact that many investigators fail to examine all the physiological variables of interest simultaneously. Two studies incorporating strength training during energy restriction found contradictory results. One study reported an increase in fat free mass (RMR was not measured),^{64,66} whereas the second found no effect of strength training on fat free mass or RMR, indicating that there are no advantages of a resistance training program to maintenance of lean body mass and attenuating reductions in resting metabolic rate.^{64,67} The lack of an effect on fat free mass in this study may have been attributable to the relatively low energy intake of 522 kcal/d overriding the potential benefits of strength training.⁶⁴

In the case of VLCDs, a limited number of studies have combined resistance training with a VLCD.⁶⁸ Most studies have found that incorporating resistance training into the very low energy diet regimen does not attenuate the loss of fat free mass or decrease in RMR.⁶⁸ It has, however, been reported that significant muscle hypertrophy is possible in an individual undergoing severe energy restriction.^{68,69} Hypertrophy was observed only in the exercised muscles, and the resistance training was unable to prevent the loss of overall fat free mass any better than diet alone.^{68,69} In a study comparing the benefits of aerobic and resistance training when combined with an 800 kilocalorie liquid diet, it was found that the addition of an intensive, high-volume resistance training program resulted in preservation of fat free mass and RMR during weight loss.⁶⁸

The results of studies examining both moderate and severe dietary energy restriction have lead to the following hypothesis: there may be a minimum level of dietary intake necessary for significant muscle hypertrophy to occur with resistance training.⁶⁸ Researchers have reported that a dietary intake of at least 1000 to 1500 kcal/day is required to attain the positive benefits that exercise training can have on RMR and fat free mass.^{68,70,71} Further studies are therefore necessary to determine whether a diet adequate in protein, fiber, and vitamins and minerals, but low in total energy, can help mediate the expected chronic adaptations to heavy resistance training.⁶¹

Caloric Restriction Combined with Endurance Training: The Effects on RMR

Aerobic exercise not only increases energy expenditure, but may also minimize the reductions in resting energy expenditure (REE) that accompany dieting by potentially increasing sympathetic nervous system activity.^{66,70,72} It has also been found to attenuate the loss of fat-free mass.^{66,73} In turn, this should prevent reduction in REE. Several studies have been undertaken to examine the effects of incorporating endurance training into weight loss regimens, with the hypothesis that its addition would attenuate losses of FFM and, by extension, reductions in RMR.⁷² Studies have documented favorable effects of aerobic activity on REE in participants who consumed diets providing 1200 to 1500 kcal/day.^{72,74-76} In a study designed to examine the effects of diet and exercise training on resting metabolic rate, participants were placed on a program combining moderate energy restriction and supervised aerobic exercise.⁷⁴ It was found that REE, when adjusted for body weight, increased 10% in this group of obese women.⁷⁴ In another study examining the effects of exercise on weight, body composition, REE, appetite, and mood in obese women, it was found that participants who consumed diets providing 1200 to 1500 kcal/day and engaged in aerobic activity experienced favorable changes in REE.⁷² In addition, the study confirmed the findings of previous investigators regarding the effect of aerobic training in participants consuming VLCDs.^{77,78} When participants were prescribed a 925 kcal/day diet, there was no effect of aerobic training on REE. However, when participants terminated their marked dietary energy restriction, a positive effect was observed.⁷² In contrast, there have been studies that have found no attenuation of the reduction in RMR in patients consuming a balanced deficit diet consisting of 1200 to 1500 kcal/day. In a study examining the physiologic changes after weight reduction with vigorous exercise and moderate intensity physical activity, there were no differences between groups in decreases in RMR.⁷⁹ In this study, vigorous aerobic activity did not attenuate reductions in RMR in patients consuming a self-selected diet.⁷⁹ There have also been studies which have failed to find any positive effect on RMR of aerobic training in participants consuming VLCDs. In fact, one study found that participants who exercised vigorously while consuming 720 kcal/ day had significantly greater reductions in REE than did nonexercising dieters.⁷⁷ Similar findings were reported by Heymsfield et al.⁷⁸ This reduction in REE appears to be a consequence of compounding the marked caloric deficit introduced by the VLCD with that introduced by exercise.⁷² It has therefore been suggested that the most favorable findings for weight loss are obtained when exercise was combined with diets of 1000 to 1500 kcal/day rather than with VLCDs providing 400 to 800 kcal/day.⁷²

Summary

The research literature regarding the effects of dieting resistance and endurance training, separately or in combination, on RMR is mixed. (Table 35.5 shows a collection of studies.) Despite the numerous reported benefits of exercise training, many studies have failed to show significant benefits of exercise on changes in weight and body composition or RMR. Consequently, the significance of many of the effects of exercise remains questionable.¹⁶

The precise cause of the discrepancy among longitudinal studies investigating the effects of exercise training on RMR is unknown. There are, however, several factors which have been suggested as playing a role in the inconsistent findings. The timing of the RMR measurement in regard to the last bout of exercise training, as well as differences in training mode, exercise intensity, duration, frequency, and total training load have been highlighted as potential factors which may account for some of the discrepancies among studies.²⁷ Thus, more rigorous, well-controlled longitudinal studies are needed to elucidate the impact of exercise training, both resistance and endurance as well as combined strength and endurance, on RMR.

Despite the equivocal findings regarding the impact of exercise training on resting metabolic rate of dieting individuals, exercise appears to be the single most important behavior for long-term weight control in obese individuals.⁸⁰ In addition to the well-known benefits of both resistance and endurance training, it has been found, almost universally, that persons who maintain their weight loss report that they exercise regularly, whereas weight regainers do not. Exercise should therefore remain a cornerstone in the treatment of overweight and obesity.

TABLE 35.5

Collection of Some of the Studies Investigating Changes in Physiological Variables Associated with Treatment

Study	Intervention	# of Participants	Weight Change (kg)	Fat Change (kg)	FFM Change (kg)	RMR Change (kcal/day)
			0	-	0	
Belko et al., 1987 ⁸⁶	diet only diet + aerobic	5 6	-7.78 -5.70	-5.1 -4.7	-2.68 -0.96	-109.9 -10.0
Broeder et al.,	control	19	-3.70	-4.7	-0.90	-10.0 -17.2
1992 ⁸⁷	strength only	13	0.00	-2.1	2.10	58.5
1992	aerobic only	15	-1.10	-2.1 -1.4	0.30	30.9
van Dale et al.,	diet only	6	-12.2	-9.4	-2.80	-533.66
1987 ⁸⁸	diet + aerobic	6	-13.2	-10.9	-2.30	-371.53
van Dale et al.,	diet only (no yo-yo)	7	-15.20	-11.9	-3.30	-389.0
1989 ⁸⁹	diet + aerobic (no yo-yo)	7	-18.90	-15.2	-3.70	-374.0
	diet + aerobic (no yo-yo)	6	-19.40	-15.4	-4.00	-302.0
Frey-Hewitt et al.,	control	41	0.38	-0.27	0.64	27.1
1990%	diet only	36	-6.68	-5.52	-1.16	-149.0
	aerobic only	44	-4.10	-4.12	0.01	-22.8
Hill et al., 198791	diet only	3	-7.90	-4.48	-3.44	-211.2
	diet + aerobic	5	-8.30	-6.13	-2.13	-252.0
Keim et al., 199092	diet + aerobic	5	-13.08	-7.30	-4.70	-139.0
	aerobic only	5	-5.61	-3.90	-1.30	18.0
Lennon et al.,	diet only	22	-6.90	-6.01	-0.89	
198574	diet + aerobic (self-	23	-6.70	-6.40	-0.30	
	selected)	20	-9.70	-8.60	-1.10	
	diet + aerobic (prescribed)					
Wadden et al.,	diet (BDD) + aerobic	9	-18.20	-16.3	-0.70	-203.3
1990 ²⁶	diet (VLCD) + aerobic	6	-21.60	-15.6	-1.90	-166.5
Geliebter et al.,	diet only	22	-9.50	-6.80	-2.70	-88.2
199764	diet + strength	20	-7.80	-6.70	-1.10	-127.2
	diet + aerobic	23	-9.60	-7.20	-2.30	-148.9
Pavlou et al., 1989 ¹⁵	diet + aerobic	15	-8.30	-6.91	-1.30	1.0
	diet only	16	-6.40	-5.47	-0.80	-176.0
Mathieson et al.,	diet + aerobic (high carb	5	-6.70	na	na	-333.0
1986 ⁹³	VLCD) diet + aerobic (low carb VLCD)	7	-8.00	na	na	-207.0
Svendson et al.,	control	20	0.50	0.50	0.60	63.0
1993 ⁹⁴	diet only	50	-9.50	-7.80	-1.20	-86.25
	diet + aerobic	48	-10.30	-9.60	0.00	-45.95
Wilmore et al., 1998 ²⁵	aerobic only	77	0.00	-0.60	0.70	16.72
Kraemer et al.,	control	6				132.0
199761	diet only	8	-6.2			-75.0
	diet + aerobic	9	-6.8			-30.0
	diet + aerobic + strength	8	-7.0			-143.0
Wadden et al.,	diet only	29	-14.40	-11.6	-2.80	-106.0
1997 ⁷²	diet + aerobic	31	-17.20	-10.6	-3.20	-46.0
	diet + strength	31	-13.70	-14.0	-3.10	-20.0
	diet + aerobic + strength	29	-15.20	-13.4	-1.80	-7.0
Racette et al.,	diet only	13	-8.30	-6.1	-2.2	-129.0
1995 ¹⁶	diet + aerobic	10	-10.50	-8.8	-1.7	-129.0
Sum et al., 1994 ⁹⁵	aerobic + strength	42	-16.10	1.70	-17.8	109.3
Donnelly et al.,	diet only	26	-20.4	-4.70	-16.1	-138.3
1991%	diet + aerobic	16	-21.4	-4.80	-16.6	-158.6
	diet + strength	18	-20.9	-4.70	-16.1	-186.9
	diet + aerobic + strength	9	-22.9	-4.10	-18.0	-217.0

TABLE 35.5 (Continued)

Collection of Some of the Studies Investigating Changes in Physiological Variables Associated with Treatment

Study	Intervention	# of Participants	Weight Change (kg)	Fat Change (kg)	FFM Change (kg)	RMR Change (kcal/day)
Kraemer et al.,	control		-0.35	-0.80	-4.84	-93
1999 ⁶¹	diet only	8	-9.64	-6.68		-80
	diet + aerobic	11	-8.99	-7.00		-122
	diet + aerobic + strength	10	-9.90	-9.57		-136
Henson et al., 1987 ⁹⁷	diet + aerobic	7	-9.50	-8.59	-1.1	-247.0
Heymsfield et al.,	diet only	5	-7.0	-4.4	-2.60	-115.2
198978	diet + aerobic	6	-7.5	-5.3	-2.20	-278.4
Bryner et al., 199968	diet + aerobic	10	-18.1	-12.8	-4.1	-210.7
	diet + strength	10	-14.4	-14.5	-0.8	63.3
Doucet et al.,	diet only (phase 1)	10	-11.9	-10.7	-1.2	-304
199998 (men)	diet + aerobic (phase 2)	9	-2.0	-3.3	1.3	134
Doucet et al.,	diet only (phase 1)	7	-7.6	-5.8	-1.8	-148
199998 (women)	diet + aerobic (phase 2)	7	-1.2	-2.2	1.0	-199
Franckowiak et al.,	diet + aerobic	18	-7.03	-5.99	-1.03	-185.4
1999 ⁷⁹	diet + lifestyle	21	-5.42	-4.71	-0.72	-170.5

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References

- 1. Stedman's Medical Dictionary. Baltimore, MD: Williams & Wilkins; 1995, pg 262.
- 2. Moffett DF, Moffett SB, Schauf CL. *Human Physiology: Foundations and Frontiers*. St. Louis, MO: Mosby-Yearbook, 1993, ch 4.
- 3. Poehlman ET. Med Sci Sports Exerc 21: 515; 1989.
- 4. Andersen, RE. In: *Lifestyle and Weight Management Consultant Manual*. Cotton RT, Ed. San Diego, CA: American Council on Exercise; 1996, pg 95.
- 5. Bullough RC, Melby CL. Ann Nutr Metab 37: 24; 1993.
- 6. Weir JBdV. J Physiol 109: 1; 1949.
- 7. Matarese LE. J Am Diet Assoc 97: 154S; 1997.
- 8. Webb P. In: *Obesity* (Björntorp P, Brodoff BN, Eds). Philadelphia, PA: J.B. Lippincott; 1992, pg 91.
- 9. Jéquier E. In: *Substrate and Energy Metabolism*, Garrow JS, Halliday D, Eds. London: Libbey; 1985, pg 82.
- 10. Ferrannini E. Metabolism 37: 287; 1988.
- 11. American College of Sports Medicine. *Guidelines for Exercise Testing and Prescription*. Malvern, PA: Lea and Febiger; 1991, pg 14.
- 12. National Institutes of Health. Consensus conference on physical activity and cardiovascular health. 276, 241-246. 1996.
- 13. Robertson JD, Reid DD. Lancet 1: 940; 1952.

- 14. Heshka S, Feld K, Yang MU, et al. JADA 93: 1031; 1993.
- 15. Pavlou KN, Whatley JE, Jannace PW, et al. Am J Clin Nutr 49: 1110; 1989.
- 16. Racette SB, Schoeller DA, Kushner RF, et al. Am J Clin Nutr 61: 486; 1995.
- 17. Seefeldt VD, Harrison, GG. In: *Anthropometric Standardization Reference Manual*, Lohman TG, Roche AF, Martorell R, Eds. Champaign, IL, Human Kinetics Books, 1988: pg 111.
- 18. Cullinen K, Caldwell M. JADA 98: 414; 1998.
- 19. Heshka S, Yang M-U, Wang J, et al. Am J Clin Nutr 52: 981; 1990.
- Harris JA, Benedict FG. Biometric Studies of Basal Metabolism in Man. Washington, DC: Carnegie Institute of Washington; 1919.
- 21. Kuczmarski RJ, Flegal KM, Campbell SM, et al. JAMA 272: 205; 1994.
- 22. Fleisch A. Helv Med Acta 1: 23; 1951.
- 23. Mifflin MD, St.Jeor ST, Hill LA, et al. Am J Clin Nutr 51: 241; 1990.
- 24. Berke EM, Gardner AW, Goran MI, et al. Am J Clin Nutr 55: 626; 1992.
- 25. Wilmore JH, Stanforth PR, Hudspeth LA, et al. Am J Clin Nutr 68: 66; 1998.
- 26. Wadden TA, Foster GD, Letizia KA, et al. JAMA 264: 707; 1990.
- 27. Broeder CE, Burrhus KA, Svanevick LS, et al. Am J Clin Nutr 55: 795; 1992.
- 28. Goran MI. Med Clin N Am 2: 347; 1984.
- 29. Poehlman ET, Gardner AW, Ades PA, et al. Metabolism 41: 1351; 1992.
- 30. Poehlman ET, McAuliffe TL, Van Houten DR, et al. Am J Physiol 259: E66; 1990.
- 31. Poehlman ET, Melby CL, Badylak SF. Am J Clin Nutr 47: 793; 1988.
- 32. Poehlman ET, Melby CL, Badylak SF, et al. Metabolism 38: 85; 1989.
- 33. Poehlman ET, Arciero PJ, Goran MI. Exerc Sport Sci Rev 22: 251; 1994.
- 34. Arciero PJ, Goran MI, Poehlman ET. J Appl Physiol 75: 2514; 1993.
- 35. Poehlman ET, Goran MI, Gardner AW, et al. Am J Physiol 264: 450E; 1993.
- McArdle WD, Katch FI, Katch VL. Exercise Physiology: Energy, Nutrition, and Human Performance. Philadelphia: Lea & Febiger; 1991, ch 9.
- 37. Perkins KA. J Appl Physiol 72: 401; 1992.
- 38. Poehlman ET, Despres JP, Bessette H, et al. Med Sci Sports Exerc 17: 689; 1985.
- 39. Costill DL, Dalsky GP, Fink WJ. Med Sci Sports 10: 155; 1978.
- 40. Ivy JL, Costill DL, Fink WJ, et al. Med Sci Sports Exerc 11: 6; 1979.
- 41. Klesges RC, Mealer CZ, Klesges LM. Am J Clin Nutr 59: 805; 1994.
- 42. Williamson DF, Forman MR, Binkin NJ, et al. Am J Publ Health 77: 1324; 1987.
- 43. McDonald JT, Margen S. Am J Clin Nutr 29: 1093; 1976.
- 44. Pirola RC, Lieber CS. Pharmacology 7: 185; 1972.
- 45. Lieber CS. Nutr Rev 46: 241; 1988.
- 46. Suter PM, Schutz Y, Jequier E. N Engl J Med 326: 983; 1992.
- 47. Lamont LS. J Cardiopulm Rehabil 15: 183; 1995.
- 48. Connolly J, Romano T, Patruno M. Fam Pract 16: 196; 1999.
- 49. Andersen RE, Wadden TA, Bartlett SJ, et al. JAMA 281: 335;1 999.
- 50. Coulston AM. JADA 98: 6S; 1998.
- 51. Mokdad AH, Serdula MK, Dietz WH, et al. JAMA 282: 1519; 1999.
- 52. Serdula MK, Mokdad AH, Williamson DF, et al. JAMA 282: 1353; 1999.
- 53. Elliot DL, Goldberg L, Kuehl KS, et al. Am J Clin Nutr 49: 93; 1989.
- 54. Foster GD, Wadden TA, Feurer ID, et al. Am J Clin Nutr 51: 167; 1990.
- 55. Bullough RC, Gillette CA, Harris MA, et al. Am J Clin Nutr 61: 473; 1995.
- 56. Phelain JF, Reinke E, Harris MA, et al. J Am Coll Nutr 16: 140; 1997.
- 57. Quinn TJ, Vroman NB, Kertzer R. Med Sci Sports Exerc 26: 908; 1994.
- 58. Almuzaini KS, Potteiger JA, Green SB. Can J Appl Physiol 23: 433; 1998.
- 59. Short KR, Sedlock DA. J Appl Physiol 83: 153; 1997.
- 60. Gillette CA, Bullough RC, Melby CL. Int J Sport Nutr 4: 347; 1994.
- 61. Kraemer WJ, Volek JS, Clark KL, et al. Med Sci Sports Exerc 31: 1320; 1999.
- 62. Ballor DL, Poehlman ET. Int J Obes Relat Metab Disord 18: 35; 1994.
- 63. Henson LC, Poole DC, Donahoe CP, et al. Am J Clin Nutr 46: 893; 1987.
- 64. Geliebter A, Maher MM, Gerace L, et al. Am J Clin Nutr 66: 557; 1997.
- 65. Ballor DL, Poehlman ET. Eur J Appl Physiol 71: 535; 1995.

- 66. Ballor DL, Katch VL, Becque MD, et al. Am J Clin Nutr 47: 19; 1988.
- 67. Donnelly JE, Pronk NP, Jacobsen DJ, et al. Am J Clin Nutr 54: 56; 1991.
- 68. Bryner RW, Ullrich IH, Sauers J, et al. J Am Coll Nutr 18: 115; 1999.
- 69. Donnelly JE, Sharp T, Houmard J, et al. Am J Clin Nutr 58: 561; 1993.
- 70. Poehlman ET, Melby CL, Goran MI. Sports Med 11: 78; 1991.
- 71. Sweeney ME, Hill JO, Heller PA, et al. Am J Clin Nutr 57: 127; 1993.
- 72. Wadden TA, Vogt RA, Andersen RE, et al. J Consult Clin Psychol 65: 269; 1997.
- 73. King AC, Haskell WL, Taylor CB, et al. *JAMA* 266: 1535; 1991.
- 74. Lennon D, Nagle F, Stratman F, et al. Int J Obes Relat Metab Disord 9: 39; 1985.
- 75. Tremblay A, Fontaine E, Poehlman ET, et al. Int J Obes Relat Metab Disord 10: 511; 1986.
- 76. Nieman DC, Haig JL, de Guia ED, et al. J Sports Med 28: 79; 1988.
- 77. Phinney SD, LaGrange BM, O'Connell M, et al. Metabolism 37: 758; 1988.
- 78. Heymsfield SB, Casper K, Hearn J, et al. Metabolism 38: 215; 1989.
- 79. Franckowiak SC, Andersen RE, Bartlett SJ, et al. Med Sci Sports Exerc 31: 345S; 1999.
- 80. Kayman S, Bruvold W, Stern JS. Am J Clin Nutr 52: 800; 1990.
- 81. Bernstein RS, Thornton JC, Yang M-U, et al. Am J Clin Nutr 37: 595; 1983.
- 82. Cunningham JJ. Am J Clin Nutr 33: 2372; 1980.
- 83. James WPT. Postgrad Med J 60: 50; 1984.
- 84. Owen OE, Kavle E, Owen RS, et al. Am J Clin Nutr 44: 1; 1986.
- 85. Pavlou KN, Hoeffer MA, Blackburn GL. Ann Surg 203: 136; 1986.
- 86. Belko AZ, Van Loan M, Barbieri TF, et al. Int J Obes Relat Metab Disord 11: 93; 1987.
- 87. Broeder CE, Burrhus KA, Svanevick LS, et al. Am J Clin Nutr 55: 802; 1992.
- 88. van Dale D, Saris WHM, Schoffelen PFM, et al. Int J Obes Relat Metab Disord 11: 367; 1987.
- 89. van Dale D, Saris WHM. Am J Clin Nutr 49: 409; 1989.
- 90. Frey-Hewitt B, Vranizan KM, Dreon DM, et al. Int J Obes Relat Metab Disord 14: 327; 1990.
- 91. Hill JO, Sparling PB, Shields TW, et al. Am J Clin Nutr 46: 622; 1987.
- 92. Keim NL, Barbieri TF, Belko AZ. Int J Obes Relat Metab Disord 14: 335; 1990.
- 93. Mathieson RA, Walberg JL, Gwazdauskas FC, et al. Metabolism 35: 394; 1986.
- 94. Svendsen OL, Hassager C, Christiansen C. Am J Med 95: 131; 1993.
- 95. Sum CF, Wang KW, Choo DCA, et al. Metabolism 43: 1148; 1994.
- 96. Donnelly JE, Pronk NP, Jacobsen DJ, et al. Am J Clin Nutr 54: 56; 1991.
- 97. Henson LC, Poole DC, Donahoe CP, et al. Med Sci Sports Exerc 46: 893; 1987.
- 98. Doucet E, Imbeault P, Almeras N, et al. Obes Res 7: 323; 1999.