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A STUDY OF LEAN BODY MASS IN ESTIMATING
BASAL METABOLIC RATE

THESIS

Presented to the Graduate Council of the
North Texas State University in Partial
Fulfillment of the Requirements

For the Degree of

MASTER OF SCIENCE

By

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The primary purpose of the study was to determine if measured LBM could be used as a more powerful predictor of BMR than could surface area (SA) as calculated by the formula of DuBois and DuBois (1916). It was also of interest to develop a prediction equation for BMR using multiple regression analysis.

Data from 82 women and 76 men were included in the study. Pearson product-moment correlations indicated that LBM was a better predictor for BMR than SA on either of the principal SA prediction equations, those of Aub and DuBois (1917) and Harris and Benedict (1919). Age, sex, and fat weight were not found to contribute significantly to prediction when included by multiple regression analyses.

Linear equations for BMR as a function of LBM were developed for each sex. Tables based on these equations were also generated as a quick reference for clinicians.

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CHAPTER I

INTRODUCTION

In a series of observations on dogs nearly 100 years ago, Rubner noticed that basal metabolic rate (BMR) varied approximately in proportion to body surface area (Rubner, 1883, cited in Schmidt-Neilsen, 1979, p. 187). The possibility that a surface relationship should exist in homeotherms such as dogs seemed theoretically sound because the rate of heat loss, which is directly proportional to surface area, must be balanced by heat production to maintain constant body temperature. Other early metabolic studies confirmed Rubner's observation (Richet, 1889; Voit, 1901, both cited in Benedict, 1915, p. 265) and supported his hypothesis that the rate of heat loss sets the rate of heat generation. As a result, when BMR norms were later tabulated for humans, body surface area was used as the reference standard.

Though these reference tables are still in use, the surface hypothesis has long been abandoned on theoretical grounds. The most important criticism was raised by the discovery that poikilothermic organisms, for whom heat loss is not a problem, show a similar relationship between BMR

and surface area. Additionally, because these studies were carried over a much greater range of body sizes than was earlier work, it became apparent that BMR was more nearly proportional to (body weight)^{3/4} and not (body weight)^{2/3} as the surface law would require (see Appendix A for complete derivation).

More important than the theoretical problems is the fact that the normative values based on surface area are "only useful when height is in proportion to weight" (Benedict, 1915, p. 277). The unfortunate result is that for those to whom accurate BMR norms are most important, obese and severely underweight patients, there is no suitable reference standard for either comparison or estimation.

While a thyroid problem might be suspected in an emaciated individual, there are no accurate average values for comparison to an actual BMR measurement. A more common problem is the need for reliable BMR estimates when assessing the caloric needs of overweight individuals.

In such cases, the contribution of BMR to the overall energy budget is often underestimated. Obesity, of course, is caused by a prolonged excess of input (feeding) over output (BMR and activity level). Numerous studies have shown that obese people in general eat no more and often less than thin people (Corbin & Pletcher, 1968; Greene, 1939). It is also well documented that the activity level

in this group is significantly below that of the rest of the population (Corbin & Fletcher, 1968; Greene, 1939). Therefore, while BMR accounts for approximately two-thirds of the energy budget of a normal individual (World Health Organization, 1973, cited by Calloway & Zanni, 1980, p. 291), this proportion would generally be well over 70% in sedentary subjects that need to be concerned with weight control. It is therefore not surprising that the single most important factor in predicting both 24 hour expenditure and weight loss on a fixed diet is BMR (Bernstein et al., 1982; Ravussin, Burnand, Schutz, & Jecquier, 1982).

An overestimate of basal requirements would certainly hinder weight loss by exaggerating caloric demands. The surface area tables currently in use tend to err in this direction when applied to overweight patients. An underestimate, on the other hand, could lead one to prescribe an unintentionally austere diet that would actually depress BMR, make the patient lethargic and minimize fat loss while sacrificing muscle tissue (Benedict, 1915; Dauncey, 1979; Forsum, Hillman, & Nesheim, 1981; Warwick & Garrow, 1981). It is clear that any refinement in the accuracy of BMR estimation for a given body can be useful to the clinician either as a comparison to or in place of expensive BMR or thyroid function tests.

Using previously published data, Cunningham (1980) investigated the influence of several variables on BMR in

an effort to find a more generally applicable reference standard. He concluded that lean body mass (LBM), estimated in his sample from sex, age, height, and weight data, was the principal predictor of BMR. Several assumptions were made in this study which would warrant an investigation of direct measures of body composition and BMR to more accurately confirm or disconfirm his conclusions regarding the role of LBM in predicting BMR.

Statement of the Problem

The primary purpose of the study was to determine whether or not measured LBM could be used as a more powerful predictor of BMR than could surface area (SA) as calculated by the formula of DuBois and Dubois (1916). It was also of interest to determine for which populations, if any, reliance upon an LBM reference standard became most important. Further, if other variables such as sex and age were found to contribute significantly to the accuracy of BMR estimation, they would be included in a prediction equation used to generate a set of BMR tables for the clinician.

It was anticipated that the simplified table making reference to LBM only would prove to be superior to the cumbersome tables currently in use. In either case, however, the adoption of LBM as the reference standard for BMR should provide more accurate normative values when

height and weight are not in typical proportions, such as in obesity.

Delimitations of the Study

The tabled BMR data are applicable only to the population sampled. Participants ranged in age from 18 to 60 years, but due to the limited number of subjects beyond age 50, the predictive table should be used for adults between 18 and 50 years of age only.

Definition of Terms

Allometry: The study of the change of various parts of an organism as a consequence of changes in body size (American Heritage Dictionary, 1970). The scaling of body parts with size is most easily expressed as some exponent of body weight.

Basal Metabolic Rate (BMR): The minimal energy expended for the maintenance of respiration, circulation, peristalsis, muscle tonus, body temperature, glandular activity, and the other vegetative functions of the body. BMR is measured directly with a calorimeter or indirectly (via oxygen uptake) under standard basal conditions (see Measurement of Metabolic Rate) and is typically expressed in $\text{Cals/m}^2\text{-hr}$ (Dorland's Illustrated Medical Dictionary, 1965, p. 906).

Homeothermy: The pattern of temperature regulation in which the cyclic variation in core temperature, either

daily or seasonally, is maintained within arbitrarily defined limits ($\pm 2^\circ$) despite much larger variations in ambient temperature. The term is only applied to organisms with relatively high BMR such as birds and mammals (Ostrom, 1978, p. 16).

Lean Body Mass (LBM): Determined by subtracting the fat mass from the total body mass. LBM represents the mass of that portion of the body which is not fat, e.g., bone, muscle, skin, organs, etc. (Wilmore, 1977, p. 255).

Poikilothermy: The wide variation of body temperature of a species such that core temperature varies as a proportional function of ambient temperature (Ostrom, 1978, p. 16). Reptiles and amphibians are poikilotherms, i.e., "cold blooded."

Respiratory Quotient (RQ): The ratio of carbon dioxide produced in the tissues to the oxygen consumed by the tissues. The RQ provides an estimate of the relative proportions of fat and carbohydrate being metabolized (Wilmore, 1977, p. 257).

$\dot{V}O_2$: The rate of oxygen consumption, typically expressed in L/min. VO_2 can be used to closely approximate the metabolic rate (Wilmore, 1977, p. 40).

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CHAPTER II

REVIEW OF LITERATURE

In order to clarify the difficulties involved in establishing a reference standard for basal metabolic rate, a review of literature was conducted. The topics covered include the nature of BMR and its measurement, a review of Surface Law theories, Kleiber's Law, the concept of gravitational metabolic stress and an overview of work suggesting lean body mass as a reference standard.

Basal Metabolism

The entire collection of chemical reactions occurring in all of the cells of the body is referred to as metabolism. As is generally known, most of the energy contained in foodstuffs that are consumed is converted to ATP, a compound that readily liberates the energy stored in its phosphate bonds to promote these cellular reactions.

The cellular process that demands the greatest proportion of ATP under resting conditions is the synthesis of vital cellular components. Probably the most important of these reactions is the formation of peptide linkages between amino acids during protein synthesis. Other compounds that require significant amounts of ATP for

production include hormones, phospholipids, and fatty acids.

A more apparent energy demanding process is muscular contraction. The contractile protein myosin enzymatically cleaves the ATP molecule to initiate the contraction process. In addition to the energy required for motion, a minor amount is needed to maintain skeletal muscle tone and to fuel the contraction of involuntary muscle. Unlike during exercise, muscle metabolism is very low under resting conditions.

Cells also expend significant amounts of energy transporting nutrients and waste products across cell membranes. This includes the selective transport of electrolytes from the renal tubules and of various substances in the gastrointestinal tract. In all these cases, active transport is employed to oppose a diffusion gradient and ATP must be consumed.

Glandular tissue also concentrates materials for storage and subsequent secretion and this concentration process again requires energy. The organic compounds secreted by the glands are also synthesized at some energetic cost to the body.

Finally, ATP fuels the sodium-potassium pump that establishes the concentration differences across the fiber membranes of the nervous system. Though the propagation of the impulse utilizes only the existing potential energy

differences across the membrane, it is the energy which was earlier stored as ATP that serves as the driving force for the impulse (Guyton, 1981, pp. 838-885).

The ATP that drives all of these reactions carries the available energy in its three phosphate bonds. The cleaving of one of these bonds liberates a definite quantity of energy. The fact that this energy is available only in discrete packets is the reason that some energy "change," the fraction of a packet not consumed by a reaction, is generally released. This "change" amounts to approximately half of the energy available in the ATP broken down. A similar efficiency is seen in the earlier conversion of the chemical energy of foodstuffs to ATP. The result is that 25% or less of the chemical energy presented to the body becomes available for cellular function. The balance is released as heat.

With the exception of muscular work performed outside of the body, the fraction of ATP that is used by the cells is eventually converted to heat as well. Because proteins are continuously being turned over, energy stored in synthesis is eventually released during breakdown. Substances that are being actively transported against a concentration gradient are eventually expelled. Even during muscle contraction, the energy expended to overcome the viscosity of the muscle tissue itself causes frictional heating of the muscle. Therefore, when external work is

minimized, it is reasonable to assume that all of the energy released by metabolic processes is eventually converted to heat (Guyton, 1981, p. 881). For this reason, the total quantity of heat produced is used as a measure of the overall metabolic activity of the body at rest.

Measurement of Metabolic Rate

Direct measurement of the heat produced by the body is performed with a calorimeter. This is basically a chamber with multilayered walls to prevent heat exchange between the internal and external environments. As the subject produces heat, the air inside the chamber is warmed slightly. The chamber air is circulated in pipes through a cool water bath to absorb the heat and maintain the chamber at constant temperature. The excess heat released to the water bath can then be quantified by monitoring the increase in water temperature.

The energy expended by the subject in vaporizing water from the lungs and skin surface is determined by trapping the vapor and weighing it. Changes in body temperature are also measured to assess changes in heat content of the body itself. The heat content of any body waste product must also be taken into account (Consolazio, Johnson, & Pecora, 1963, pp. 2-5).

The calorimeter is expensive to build, difficult to operate and limited in use to a narrow range of activities

due to its size. Indirect techniques have come into favor which are nearly as accurate, much less expensive and more versatile (Wilmore, 1977, p. 28).

The most important indirect method makes use of the linear relationship between oxygen consumption and metabolic output. When one liter of oxygen is metabolized with glucose, 5.01 calories are released; with fat, 4.70 calories and with protein, 4.60 calories. Assuming a typical mixed diet, the body averages approximately 4.825 calories per liter of oxygen consumed and thus a measure of oxygen consumption provides an estimate of metabolic rate. If only carbohydrates are burned during the period of gas collection, the resulting estimate of the metabolic rate will be 4% too low; if fats are the only substrate the estimate will be 4% high and if only protein is being consumed, the error will be insignificant (Guyton, 1981, p. 882).

A more exact caloric equivalent for each liter of oxygen can be obtained by calculating the respiratory quotient (RQ) from CO_2 production and O_2 consumption data. At submaximal exertion levels, the RQ serves as a guide to the nutrient mixture being catabolized for energy. By this method, agreements between direct and indirect calorimetry are excellent. One study reports an average difference between the two methods of 0.17% (Gephart & DuBois, 1915, cited in Consolazio et al., 1963, p. 57) and several others

using both animals and humans have consistently demonstrated differences of 1% or less (McArdle, Katch, & Katch, 1981, p. 98).

The equipment used in the present study, the Beckman Metabolic Measurement Cart (MMC), is an advanced indirect calorimeter. It contains a flow meter to measure the volume of expired air as well as oxygen and carbon dioxide analyzers to determine fractional concentrations of these gases in the expired air sample (McArdle et al., 1981, p. 102). The MMC uses these data to perform the necessary calculations and provides an output almost simultaneous to the measurement.

Though there is excellent reason to have confidence in the accuracy of the measurement determined in this way, there seems to be a moderate degree of physiological variation in the basal metabolic rate. Berkson and Boothby (1937) have reviewed variability data from a number of studies. Though the interindividual variability, measured as the standard deviation's percentage of the mean value, was shown to be 6% to 7%, repeated daily measurements on the same individual varied by 2.5% to 5%. Even repeated determinations on the same individual in a single day were found to vary by an average of 3% with fluctuations being slightly higher for females. The problem this presents is that the interindividual variation of interest is not a great deal more than the "noise" of the measurement--the known variation in the measure for a given individual.

The source of such variation over a short time period is probably due to the number of factors known to influence the metabolic rate and the difficulty of controlling all of them. Besides activity, which may elevate metabolism for several hours beyond the activity itself; digestion of protein; thyroid, growth or male sex hormone levels; sympathetic stimulation and environmental temperature all affect metabolic rate (Guyton, 1981, p. 883).

To make both interindividual and intraindividual comparisons meaningful, it has been extremely important to adopt a set of basal conditions under which BMR determinations are to be carried out. These conditions are:

- 1) The subject must not have eaten in the 12 hours immediately preceding the test.

- 2) The test should follow a night of restful sleep. Rest reduces sympathetic nervous activity to a minimal level.

- 3) No strenuous exercise is to be performed following the night of restful sleep and the subject must remain in complete rest lying down for at least 30 minutes prior to the test.

- 4) All psychic and physical factors that cause excitement or disturbance should be eliminated to the greatest extent possible.

- 5) The temperature of the air should be between 68°F and 80°F (20°C to 27°C) to minimize sympathetic efforts

to maintain body temperature (Guyton, 1981, p. 884).

Under these basal conditions, the heat production level is then calculated from the rate of oxygen consumption. The metabolic rate is then expressed as the rate of heat production per square meter of body surface area.

The Surface Law

The convention of using surface area as the reference standard for metabolic rate was set a century ago. Bergmann was the first to hypothesize that the rate of heat production was set primarily by the need to maintain the body at constant temperature (Bergmann, 1852, cited by Benedict, 1915, p. 264). Without experimental data, he argued that heat generation would have to precisely balance heat loss for a body to remain in thermal equilibrium with its surroundings. It was known at the time that the rate of heat flow from a body could be calculated by Fourier's Law:

$$q = \frac{T_i - T_s}{r} (SA) \quad (1)$$

(Kleiber, 1975, p. 187), where

- q = rate of heat flow
- SA = surface area of heat transfer
- T_i, T_s = temperature inside insulative layer and temperature at body surface
- r = specific insulation; equal to the thickness of insulation divided by the heat conductivity of the layer

Thus, for a given temperature difference and insulation area, heat flow is directly proportional to surface area, i.e.,

$$q = \frac{\Delta t}{r} (SA) \quad (2)$$

As noted earlier, Rubner found experimental support for the Bergmann hypothesis (Rubner, 1883, cited by Kleiber, 1975, p. 182). Finding a systematic decrease in relative heat production (Calories/kg-hr) in fasting dogs passing from 3 to 31 kg, he found that the differences almost disappeared when expressed as Calories/m²-hr. From these and similar observations on a variety of species in his lab, he deduced what has come to be known as Rubner's Surface Law: fasting homeotherms produce 1000 Calories per day per square meter of surface area.

The work of Richet (1889, cited in Kleiber, 1975, p. 183) using rabbits of various sizes was accepted as support for Rubner's Surface Law. After measuring heat production, he calculated the surface area of these rabbits by assuming they were spherical in shape with a density of one kg/L. Their daily heat production calculated in this way was 776 Cal/m²-day but, due to the unusual shape of rabbits, he felt justified in subtracting the area of the ears. This brought the calculated BMR up to 917 Cal/m²-day which was acceptably close to Rubner's figure.

Subtracting the ear surface area is certainly questionable, because as long as the ears are maintained at a

temperature greater than that of the environment, heat will be lost from the ears at the rate given by the Fourier Law (equation 1). In fact, ear size in the mammalian order that includes rabbits, hares, and pikas (Lagomorpha) is thought to be strongly determined by the temperature of the environment. The largest ears are found on desert hares and probably aid in heat dissipation while arctic hares and pikas, which live exclusively above timberline, have very small ears. Therefore, if heat loss through the ears is significant enough to impose a physical design constraint, exclusion of the ear SA simply to approximate the 1000 Cal/m²-day figure is probably not realistic.

Similarly, modeling rabbits as spheres underestimates the true surface area because a sphere encloses the maximum volume for a given SA. Such a calculation again underestimates the true animal surface area and thus the real heat production would be well below the initial figure of 776 Cal/m²-day. Nevertheless, the Surface Law became more firmly entrenched in the literature and deviations from it were perceived as errors in SA estimation.

Meeh (1879, cited by Benedict, 1915, p. 274) improved upon Richet's work by deriving a more accurate method of calculating body surface area. From dimensional analysis, SA is proportional to (length)² while volume and weight are proportional to (length)³. Therefore, surface area is proportional to (weight)^{2/3} which is essentially the Meeh

Formula (see Appendix A):

$$SA = kW^{2/3} \quad (3)$$

for k = a constant for a given series of similarly shaped animals

W = mass in kg

The task was then, by anatomical study, to determine a series of k 's to serve as appropriate scaling constants. For example, the constant obtained for humans was 12.3 while the figure for less attenuated species, such as rabbits, would be somewhat less because SA would be less for a given weight. Though the Meeh Formula was used for three decades to calculate surface area, it had the disadvantage of only being valid for "average" body shapes. Thin people have a higher body SA for a given body weight than do stocky people, yet the Meeh Formula calculates SA as a function of weight only. The result was that SA could not be calculated accurately for those to whom this calculation was most important: patients with abnormal body shapes.

Including only normal shaped subjects in their study, Benedict and Emmes (1915) made the first serious attempt to compare basal metabolic rates of men to women. From a sample of 89 men and 68 women, they concluded that the male BMR was 5 to 6% higher than that of the female. They attributed this difference to the males' "greater quantity of active tissue" but apparently did not find this

inconsistent with the hypothesis that SA sets the metabolic rate.

Benedict and Smith (1915) compared BMR of athletes with that of more sedentary individuals. The subjects were divided into 11 groups and paired by weight. The comparison showed that:

- 1) Athletes as a group produce more heat per unit of surface area than do nonathletes.

- 2) The heaviest groups displayed the greatest discrepancy in BMR between athletes and nonathletes.

The same data was analyzed from two additional angles by Benedict in a third publication (1915). Subjects were matched by weight with different heights and in all cases, the taller individual had the greater metabolic rate. He also noted a systematic decline in heat production with age. Both observations were consistent with the conclusion that differences in the quantity of active tissue were responsible for the BMR differences seen. As a further challenge to the Surface Law, he stated that "athletes and those of abnormal shape (could) not rightfully be excluded from complying with a supposed physiological law" (Benedict, 1915, p. 278).

The need to exclude individuals with disproportionate body shapes was removed the following year when DuBois and DuBois (1916) empirically derived the formula for SA calculation that is still used to express BMR values. By

incorporating both height (L in m) and mass (W in kg) into their formula, a measure of stoutness was included and accuracy increased from $\pm 20\%$ with the Meeh Formula to a maximum error of 5%. The formula:

$$SA = 71.84 W^{0.425} L^{0.725} \quad (4)$$

remains the best method of calculating the surface area of the human body. Employing the new formula to express BMR for a large sample of both sexes, Harris and Benedict (1919, cited by Kleiber, 1975, p. 201) concluded that there was no evidence for the Surface Law within the human species. Other factors appeared to be involved.

Kleiber's Law

In order to find a more suitable unit of metabolic body size, Kleiber (1932, cited by Kleiber, 1975, pp. 201-203) collected results from several American laboratories ranging from a .150 kg dove to a 679 kg steer. Though Voit (1901) had done a similar study using data from Europe and found BMR to be proportional to $(\text{weight})^{2/3}$, Kleiber's results suggested that BMR was more nearly proportional to $(\text{weight})^{3/4}$. As he put it, his data suggested "a trend of modern American animals to take the Surface Law less seriously than did the earlier European animals" (Kleiber, 1975, p. 202). Several authors have followed the same procedure using their own mammalian data ranging from a 4 gram (.004 kg) shrew to a 4 ton (3636 kg)

elephant and arrived at exponents very close to 0.75 (Benedict, 1938, cited by Kleiber, 1975, p. 204; Brody & Proctor, 1935, cited by Kleiber, 1975, p. 204; Galvão, 1948; Schmidt-Neilsen, 1979, p. 180). The same relationship has been seen in birds (Lasiewski & Dawson, 1967, cited by Schmidt-Neilsen, 1979, p. 108) and even reptiles (Bennett & Dawson, 1976, cited by Bauer & Friedl, 1980, p. 257). Hemmingsen (1960) extended the same result to include other poikilothermic and even unicellular organisms. The consistency with which the exponent $3/4$ has appeared has led physiologists to refer to this result as Kleiber's Law:

$$\text{BMR} = k (W)^{3/4} \quad (5)$$

for k = a scaling constant; high k reflects high metabolic rate for a given size organism.

In particular, the observation that non-homeotherms display this same relationship suggests that some underlying principal apart from surface heat loss is at work. Stated another way, "Most people agree that the body usually does not metabolize because it must produce heat but produces heat because it metabolizes (Grande, 1984, p. 11).

Gravitational Metabolic Stress

The surface law should adequately describe the rate of heat loss from two geometrically similar bodies in identical environments. Its failure to account for the metabolic rates seen in mammals highlights a major difference between

the environments inhabited by large and small animals: the influence of gravity.

The simplest demonstration of this fact parallels arguments first stated by Galileo in the 1600s. Consider a limb of diameter d sharing in the static support of a body with specified shape and length l . Because the compression strength of a limb is proportional to its cross-sectional area (hence to d^2) while the weight it must support increases as l^3 , leg diameter must scale as $l^{3/2}$ to maintain a constant ratio of strength to load. Therefore, because large and small animals are designed to withstand different gravitational environments, they are not geometrically similar. This relative thickening of the limbs with increasing body size has been observed in several animal groups with comparable shapes (Bakker, 1980; Bauer & Friedl, 1980; Jolicoeur, 1963; Stahl & Gummerson, 1967) and is generally recognized as a major factor in body design.

The hypergravity conditions experienced by large animals have been imposed on rats, mice and chickens in several centrifugation studies. Oyama and Zeitman (1967), raising rats in a 4.7 G environment for one year, induced both an increase in relative femur weight and an alteration toward the more circular shaft cross-section seen in larger animals. The opposite effect, loss of bone from the femur and the tibia, has been observed in human subjects exposed to zero gravity for 89 days (Van Huss & Heusner, 1979,

p. 26) but similar results have been observed following prolonged bed rest and may in this case be attributable to a lack of lower limb exercise (and consequent loading) during this spaceflight.

Centrifuged animals also display impaired growth rates and reduced size at maturity. Both effects are more pronounced at higher field intensity (Daligcon & Oyama, 1975; Kiel, 1969; Oyama & Platt, 1967; Oyama & Zeitman, 1967) and reflect the cost of growth under excess gravity conditions.

This cost is also reflected in the high basal metabolic rates of animals raised in centrifuges. Following an adaptation period of a month or less, rats have been shown to eat more (Kiel, 1969; Oyama & Chan, 1973; Oyama & Platt, 1967; Oyama & Zeitman, 1967) and have greatly reduced fat reserves (Kiel, 1969; Oyama & Platt, 1967; Oyama & Zeitman, 1967) compared with noncentrifuged control animals. Rats adapted to a 4 G environment also consume 25% more oxygen than weight-matched rats experiencing only the earth's gravitation (Oyama & Chan, 1973). Again, the opposite effect has been observed in astronauts exercising at sub-maximal workloads and requiring less energy compared with the same workload on earth (Van Huss & Heusner, 1979, pp. 14-15).

Even tissue obtained from centrifuged animals appears to metabolize at a higher level. Slices of diaphragm muscle taken from rats exposed to 4.15 G continuously for

20 weeks showed higher rates of glucose uptake and $^{14}\text{CO}_2$ production from [U - ^{14}C] glucose than did tissue from controls (Daligcon & Oyama, 1975). The fact that there was no difference in the rates of ^{14}C storage as glycogen also suggests that the glucose was being broken down for energy production.

Recognition of the contribution of gravity to the metabolic rate led Economos (1979) to propose a theoretical decomposition into surface and gravitational terms:

$$\text{BMR} = \text{SA metabolic cost} + \text{G metabolic cost} \quad (6)$$

In the absence of gravity, the second term is simply given by the surface law (3) and is proportional to $W^{0.67}$. He obtains a value for the gravitational cost by comparing gravitational tolerance data for several body sizes at different centrifugation speeds. His equation:

$$\text{BMR} = 12W^{0.89} + 58W^{0.67} \quad (7)$$

actually fits the data upon which the Kleiber equation is based with a slightly smaller standard error than does the Kleiber equation. It also accurately calculates the approximate percentage of BMR (~30%) expended by humans as gravitational metabolic cost.

Another attempt to theoretically account for the deviation of BMR values from surface law predictions has been advanced by McMahon (1975). This argument is also based on the mechanical adaptations of the body to gravity. Invoking engineering principles, he deduces that any limb

of diameter d must be proportional to $W^{3/8}$ to withstand buckling and bending forces. This approximate relationship has been observed in several anatomical studies (Bakker, 1980; Bauer & Friedl, 1980; Jolicoeur, 1963; Stahl & Gummerson, 1967).

Within a given body, a muscle of cross-sectional area A shortens length Δl against force σA in time Δt where σ is the tensile force developed by the contracting muscle per unit area. The power expended by such a contraction would be:

$$P = \frac{\text{force} \times \text{distance}}{\text{time}} = \frac{(\sigma A)(\Delta l)}{\Delta t} \quad (8)$$

McMahon cites Hill (1950) in claiming that:

a) σ is a constant because the inherent strength of a contracting voluntary muscle fiber is proportional to its cross-sectional area.

b) $\frac{\Delta l}{\Delta t}$, the shortening velocity, is a constant in any particular muscle from species to species. Therefore, he argues, the maximal power output of a muscle is proportional only to its area. Or, $\text{power}_{\max} \propto A$, but $A \propto d^2 = (W^{3/8})^2 = W^{3/4}$. So $\text{power}_{\max} \propto W^{3/4}$.

Now, if maximum power output exceeds BMR by some constant factor ("metabolic scope") which he states is suggested by the work of Hemmingson (1960), then BMR must also be proportional to $W^{3/4}$. Thus, an alternate explanation for the Kleiber law is proposed based only on

the cross-sectional area of muscle required by physical constraints.

Lean Body Mass as the Metabolic Reference Standard

The Kleiber law provides a standard of metabolic body size that is useful for interspecific comparisons. For example, an animal that weighs twice as much as another would be expected to have a metabolic rate $(2)^{0.75} = 1.68$ times as great when expressed simply as Cal/day. The relationship also seems to hold within a given species. Kleiber (1975, p. 200) gives as an example the fact that a 6.0 kg dog and a 5.3 kg rabbit have very similar BMR per kg, both of which differ significantly from that of a 1.5 kg rabbit. This implies that size itself, and not some genetically determined BMR based on the mean size for a species, seems to be the important factor.

The major factor that varies with size, as discussed earlier, is the necessity of structural support. Large animals must contain a greater amount of metabolically inert skeletal and connective tissue. The documented thickening of limb bones with increasing body size (Bakker, 1980; Bauer & Friedl, 1980; Jolicoeur, 1963; Stahl & Gummerson, 1967) suggests that bone weight may scale approximately in proportion to $(\text{body weight})^{1.25}$. Assuming similar percentages of metabolically inert adipose tissue in large and small animals, the active tissue mass must

then scale as body weight to some exponent less than one. It is therefore possible that these skeletal differences and consequent relative amounts of active tissue are largely responsible for the Kleiber relationship.

This point may be expanded a bit by breaking the body down further into several components metabolizing at different rates. As mentioned earlier, fat and bone tissue produce very little heat while skeletal muscle, which accounts for about 40% of the body weight, contributes approximately 25% of the BMR. By contrast, the brain, liver, heart, and kidneys produce 50% of the body's heat while representing only 5% of its mass (Brooks & Fahey, 1984; McArdle, Katch, & Katch, 1981; Stolwijk, & Hardy, 1966). In animals of different sizes, these components seem to scale at different rates. Slowly metabolizing support structures, such as bone, cartilage and presumably muscle mass increase with (body weight)^{1.25} (Bakker, 1980; Bauer & Friedl, 1980; Jolicoeur, 1966; Stahl & Gummerson, 1966). Jerison (1973) found brain weight among mammals to increase only with the 0.67 power of weight, while the heart seems to scale with weight^{0.8} (Stahl & Gummerson, 1966) or weight^{1.0} (Sjöstrand, 1961, cited by Åstrand & Rodahl, 1977). Liver and kidney size, because of their filtering functions, are probably tied directly to the metabolic rate, or weight^{0.75}. Therefore, body components with low metabolic activity occupy a greater proportion of the body

in large animals while metabolically active organs scale at a rate equal to or more slowly than the body weight. The Kleiber relation, the proportionality of BMR to weight^{3/4}, may simply be an overall expression of the allometry of the various tissues.

The other possibility is that the active tissue itself metabolizes at a rate dependent upon the size of the organism from which it originates. Unfortunately, the relationship between tissue metabolism and body size has yet to be established (Kleiber, 1975, pp. 196-202). Though there is some conflict in the data presented, metabolic rates for organ tissue in vitro seem to be independent of body size while the relationship for skeletal muscle is not at all clear. Further complicating the matter is the relationship between in vitro and in vivo metabolic rates in animals of different sizes. For the purposes of this study, this point is only of theoretical interest in determining physiologically what sets the basal metabolic rate.

In human populations, the tremendous variation in the quantity of metabolically inert body fat makes BMR estimates using the Kleiber relation actually less useful than those based on surface area. Galvão (1948) found BMR to be related to a different power of weight depending on body shape. For "lean man," BMR scaled as $W^{0.83}$ while BMR was found to be related to $W^{0.78}$ for "well-proportioned" (fatter) man. Because the "well-proportioned" group would

almost certainly show a positive relationship between body fat percentage and weight that would be absent in the lean group, these results are consistent with the idea that active tissue mass (LBM) is at least partially responsible for observed differences in BMR.

Earlier work also provided evidence for this hypothesis. Benedict and Smith (1915) compared BMR of 11 pairs matched by body weight in athletes and sedentary individuals. In each case, the athlete had the greater metabolic rate and these differences were the greatest between the heavier pairs. Presumably, the latter observation indicated that in the lighter pairs, the sedentary individuals were already relatively lean while the excess weight in the heavier groups was of different composition in the athletes than it was in the nonathletes.

Benedict and Emmes (1915) measured an approximate 6% difference in BMR between men and women. Benedict (1915) rearranged these same data to compare subjects of similar weight, sex and age but different height. Again, the taller (and probably leaner) individuals had higher metabolic rates. Benedict concluded that active tissue mass was primarily responsible for BMR based purely on these qualitative compositional assessments.

An analytical attempt was made by Behnke (1953) to reconcile the apparent dependence of BMR on LBM despite the assumed theoretical necessity that it depend on SA. His

analysis consisted of little more than equating the observed Kleiber law describing BMR as a function of $W^{0.73}$ to the DuBois and DuBois (1916) relation between BMR and SA. He concluded that "contrary to the simplest dimensional reasoning, SA is not always proportional to $W^{0.67}$ (but in this case) to $W^{0.73}$ (Behnke, 1953, p. 13). This argument necessitates the unlikely mechanical event of large animals being relatively more slender than small ones. From McMahon's arguments and anatomical data already presented showing the relative thickening of larger animals, it is clear that SA is more nearly proportional to $W^{0.60}$. In human populations, which Behnke was attempting to describe, the exponent is almost certainly even lower due to the preponderance of obesity, and consequent globular body shapes, in individuals with high body weight.

At the same time, Miller and Blythe (1953) employed a very thorough analysis of original data to examine the same problem. Basal oxygen consumption, weight, LBM and surface area values were acquired from 48 student subjects and the relationships between these variables studied. Their results showed:

$$\text{BMR vs. SA} \quad r = 0.84$$

$$\text{BMR vs. Wt} \quad r = 0.82$$

$$\text{BMR vs. LBM} \quad r = 0.92$$

$$\text{SA vs. LBM} \quad r = 0.79$$

Miller and Blythe then calculated the partial correlation between BMR and SA adjusted for the influence of LBM

and found a correlation of only 0.4. They concluded that LBM was the best reference standard for BMR and that SA derives much of its validity in this role from its relatively high correlation with LBM. No great error results from the use of a surface area standard if body composition is relatively constant between subjects but obesity dissociates LBM from SA and large errors can result.

A multiple regression was also performed with the variables SA, weight, LBM and combinations thereof as predictors of BMR. LBM and a combination of LBM and SA were nearly identical in prediction accuracy while all other combinations produced standard errors at least 50% greater.

Recently, Cunningham (1980) also performed a multiple regression analysis of several variables thought to influence BMR using the data for 223 subjects from the metabolism studies of Harris and Benedict (1919). The factors examined were sex, age, height, body mass and LBM estimated from the other data. Cunningham concluded that LBM was the principal predictor of BMR and presented a best estimate prediction equation ignoring the previously assumed influences of sex and age:

$$\text{BMR (Cal/day)} = 500 + 22 \text{ LBM (kg)}. \quad (9)$$

Several studies have attributed at least the greatest portion of the observed decline in BMR with age to the loss of lean tissue. Calloway and Zanni (1980) compared a group of six older men (average age = 68) to a sample of young

men (average age = 28) and found the 13% difference in BMR to be paralleled by a 12% drop in LBM as measured by underwater weighing. Keys, Taylor and Grande (1973) performed a longitudinal study of the same variables and concluded that, though basal requirements declined at an average rate of approximately 3.2% per decade, this decline was closer to 1% per decade when expressed as Cal/day-LBM.

Longitudinal changes in BMR and LBM were also investigated in a pair of studies by Tsankoff and Norris (Tsankoff & Norris, 1977; Tsankoff & Norris, 1978). Also observing a 3.7% decline in BMR per decade, they went a bit further in attempting to identify the component of LBM responsible for the lessening energy requirements.

The 1977 study used creatinine excretion rate as a measure of muscle mass. After finding the relationship between basal oxygen consumption and creatinine excretion, it was possible to calculate the proportions of $\dot{V}O_2$ consumed by muscle tissue and by nonmuscle tissue. Plotted over time, it was apparent that muscle mass decrement accounted for the decline in basal oxygen consumption that accompanies aging.

It was also pointed out that older subjects have a relatively higher proportion of connective tissue associated with their muscles. Because connective tissue is a metabolically inert component of LBM, this fact may be at least partially responsible for the residual small decline

in BMR per kilogram of LBM. The energy requirement of the nonmuscle component has actually been shown to increase slightly with age, presumably due to metabolic stress associated with heart disease, cancer and other disorders (Tsankoff & Norris, 1978). This implies that the loss of skeletal muscle is fully responsible for the decline in basal requirements and may in fact be understated by the decline in whole body BMR.

One discrepancy between this study and several others makes one view these conclusions with caution. While Tsankoff and Norris attribute 50% of whole body oxygen uptake to skeletal muscle metabolism, other studies have consistently found this figure to be only 15 to 25% (Brooks & Fahey, 1984; Kleiber, 1975; McArdle, Katch, & Katch, 1981; Stolwijk & Hardy, 1966).

Surface Area Prediction Formulas

It appears that the widely used tables of Harris and Benedict (1919) and Aub and DuBois (1917) make concessions to the dissociation of SA and LBM that comes with age. In any population, these two variables show a fairly strong, positive correlation with each other. Before LBM could be measured, then, it is not surprising that SA was found to be the best predictor of BMR.

But because LBM declines with age for a given body size (and SA), BMR does also. This necessitates the

TABLE 1
 BASAL METABOLISM (AUB & DUBOIS)*

Age (yr)	Kilocalories per Hour per Square Meter Body Surface	
	Male	Female
20-30	39.5	37.0
30-40	39.5	36.5
40-50	38.5	36.0
50-60	37.5	35.0
60-70	36.5	34.0

*Source: Aub & DuBois (1917).

incorporation of an age correction factor in the SA prediction formulas. It represents a secondary correction factor to, effectively, "make SA more like LBM." Perhaps it would be simpler and more accurate to base our predictions on LBM.

Summary

Early work suggested basal energy production per unit of body weight declined with increasing size but was nearly constant when expressed as a function of body SA. This observation fit neatly with the physical law stating that heat loss is in direct proportion to SA and the metabolic Surface Law was proposed. As a result, when BMR tables were prepared for humans in the early 1900s, body surface area was used as the reference standard.

Interspecific comparisons showed that BMR was not proportional to $W^{0.67}$ as the surface law would require but more nearly to $W^{0.75}$. Thus, their theoretical validity was in doubt. Additionally, the tables were reasonably accurate only for individuals with typical body shapes and compositions. They were therefore not practical for application to the subjects for whom accurate BMR estimates were most important.

Attempts to theoretically explain the observed deviation from the simple surface law centered on the effects of gravity on organisms of different sizes. Because gravity tends to alter the structural composition of an organism as a function of body size, it seems that BMR is strongly influenced by body composition.

Many studies on human subjects have found body composition to be a primary determinant of BMR. In fact, it appears that the reason surface area has been useful as a metabolic reference standard is its relatively high correlation with LBM. In cases where the two variables dissociate such as obesity or extreme emaciation, LBM retains its predictive value while SA falters. Therefore, LBM is suggested as the standard of both theoretical and practical choice.

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CHAPTER III

METHODS AND MATERIALS

Introduction

In this chapter the experimental procedures followed will be presented in detail. The following sections will include: Subjects; Methods; Pilot Study; Statistical Analysis; and Table Development.

Subjects

Subjects for the study were primarily individuals volunteering to announcements made in physical education and in faculty/staff fitness classes at North Texas State University. Several others were respondents to short articles that were placed in both the NTSU Daily and faculty/staff fitness program newspapers. The remainder was made up mostly of individuals that had become interested in the study through friends that had taken part.

The subjects in general had not had any prior experience with the equipment used for determining metabolic rate. There is some evidence that inexperienced subjects tend to give BMR results that are approximately 5% greater than those from trained subjects (DuBois, 1936, pp. 147-148). Although the values obtained from

experienced subjects may have slightly greater physiological significance, DuBois (1936) suggests that measurements made on inexperienced subjects are more valuable for comparison to the majority of clinical patients.

One hundred ninety-five adults participated in the study. They were divided into four age groups as follows: 18 to 29, 30 to 39, 40 to 49, and 50 to 59. A rough balance between sexes was maintained in each category.

Data from 36 subjects were omitted from the analysis because standard basal conditions were not met. The various reasons for omission are outlined in Table 2.

TABLE 2
NUMBERS AND REASONS FOR SUBJECTS OMITTED
FROM ANALYSIS

Reason	Number of Subjects
Equipment Malfunction	5
Physical Pain or Anxiety	6
Food or Caffeine	5
Medications	7
Missing Data	3
Crash Dieting	2
Exercise Pretest	4
Failure of BMR to Stabilize	4
Extreme Deviation from BMR Norm	1
Total	37

Medications affecting the metabolic rate, principally Synthroid, caused the greatest number of tests to be invalid (seven). Synthroid contains synthetic L-thyroxine, the primary hormone secreted by the normal thyroid gland.

Synthroid therefore artificially sets the metabolic rate in the absence of normal thyroid function (Physician's Desk Reference, 1978, p. 874). Another subject was taking Entex. This is a decongestant containing phenylephrine and phenylpropanolamine, compounds with general sympathetic effects that are used to combat congestion (Physician's Desk Reference, 1978, p. 641). Under the standard basal conditions summarized earlier, sympathetic stimulation must be kept to a minimum. Other major causes of invalid tests were excessive anxiety and recent food or caffeine consumption, all of which elevate the metabolic rate, and equipment malfunction.

One female was also omitted from the analysis because the BMR deviated tremendously from the norm. Harris and Benedict (1919) excluded subjects that varied by greater than 25% from their prediction values. This subject was 85% too low according to Harris and Benedict and 36% low by this study's LBM prediction. She was the only subject to deviate by more than 25% in this study.

Altogether, data from 158 subjects were judged to be acceptable. The physical characteristics of the participants are summarized in Tables 3 and 4.

TABLE 3
SEX AND AGE BREAKDOWN OF SUBJECTS

Age	Avg. Age	18-29	30-39	40-49	50-59	Total	%
Female	30.8±8.0	35	31	15	1	82	52.2
Male	32.0±8.9	30	30	11	5	76	47.8
Total in Group		66	61	26	6	158	100.0
% in Group		41.5	38.4	16.4	3.8	100.0	*

TABLE 4
BASIC PHYSICAL CHARACTERISTICS
OF ALL SUBJECTS

Characteristic	Mean ± Standard Deviation
Age (yrs)	31.8 ± 8.3
Weight (kg)	67.7 ± 14.7
Percent Fat (%)	20.1 ± 10.0
Lean Body Mass (kg)	54.1 ± 12.5
SA _(m²)	1.79 ± .21

Methods

Participants were instructed in the procedures to be followed at least 12 hours before arriving at the laboratory. They were also asked during the test session if they had complied with these instructions.

An additional restriction was added to those of the standard basal test to see if the generally poor predictability of the female BMR could be enhanced. DuBois (1936,

pp. 181-184) reviews several studies that report a pre-menstrual BMR elevation of 2 to 6% as well as a similar depression during the week of menstruation. The presence of pain associated with the menstrual period can raise the metabolic rate by 20% or more. For this reason, female subjects were scheduled to avoid both the week before and the week of menstruation.

Subjects arrived to a quiet and thermally comfortable laboratory as dictated by the standard basal conditions outlined earlier. The testing procedure was fully explained and an informed consent form signed to ensure voluntary participation. The subject was then asked to lie down and relax as deeply as possible for at least 30 minutes prior to testing.

Following this period of relaxation, a mouthpiece was inserted and basal oxygen consumption determined with the aid of a Beckman Metabolic Measurement Cart (MMC). The MMC samples expired air continuously and reports averaged metabolic measurements at selected intervals. Once the dead space within the MMC had filled with the subject's expiration gas, metabolic reports were obtained every 4 minutes. After at least three readouts, if the $\dot{V}O_2$ had stabilized with the RQ between .70 and .90, the test was considered acceptable (Consolazio, Johnson, & Pecora, 1963, p. 25).

The MMC was calibrated before and after each test by the Two-Gas Calibration Method described in the MMC Operator's Training Manual (Beckman Instruments, 1980). When readout drift was detected over the course of a test, values were interpolated between initial and final figures. Flowmeter volume calibration, temperature, and pressure entry were performed daily (Beckman, 1980).

Body composition was determined by the skinfold technique using the generalized equations of Jackson and Pollock (1978) and Jackson, Pollock, and Ward (1980) based on age and the sum of three skinfold values. The equation developed for females was shown to have a multiple correlation value [sum of three skinfolds, (sum of three skinfolds)², age] with body density of $R = 0.842$ and a standard error of 0.0086 g/mL body density (Jackson, Pollock, & Ward, 1980). The corresponding values for the equation applicable to males are $R = 0.918$ and a standard error of 0.0072 g/mL (Jackson & Pollock, 1978).

All skinfold measurements were taken by the same investigator using a Harpenden Skinfold Caliper. The following techniques suggested by Katch and Katch (1980) were employed:

- 1) Paying strict attention to the specified anatomical landmarks as reference points for measurement at the precise skinfold locations. These locations were described by Baun, Baun, and Raven (1981).

2) Performing each measurement two to five times and recording the average value.

3) Undertaking a pilot study to gain experience with the technique. For acceptable research data, Katch and Katch recommend a test-retest correlation of 0.85 or better and correlation with an experienced skinfold technician of at least 0.90. These results should be obtained over a population of 100 or more that is similar to the research sample. Results of the pilot study will be discussed in the following section.

Underwater weighing was used in this pilot study to verify the accuracy of the skinfold estimates. It was also used on those extremely overweight subjects from whom accurate skinfold data could not be obtained. This fraction represented approximately 10% of the sample. The procedures for underwater weighing detailed by McArdle, Katch, and Katch (1981, pp. 372-376) were followed.

A Chatillon 250 kg spring scale accurate to ± 0.250 kg was used to measure underwater weight. Residual volumes were measured by the simplified method of Wilmore, Vodak, Parr, Girandola, and Billing (1980). Body density was converted to fat percentage by the Siri formula (1961, cited by Jackson, Pollock, and Ward, 1980, p. 176).

The subjects were weighed in air on an ACME Chair Scale Model 4060 MV with an accuracy level of ± 0.10 kg. A standard physician's scale was used to measure height to

the nearest 1/4 inch (.0064 m) and weight to the nearest pound (.45 kg). From these height and weight data, body surface area was estimated from the chart prepared by DuBois and DuBois (1916).

Pilot Study

A pilot study was undertaken to ensure that testing procedures were both reliable and valid.

Skinfold measurements were obtained on more than 100 subjects that had also been measured by a reliable and valid investigator. The Pearson product-moment correlation coefficient between the two sets of results was 0.99. This comfortably exceeds Katch's minimum figure of 0.90.

Test-retest consistency was also evaluated. Initial scores were related to secondary scores by a reliability coefficient of 0.99. Again, this figure exceeded Katch's threshold for credibility of $r = 0.90$.

Because both underwater and skinfold tests were used to measure body composition, a comparison study was necessary to demonstrate that the values obtained in this lab by either method were interchangeable. The Pearson product-moment correlation between the two methods was 0.90 over a heterogeneous sample of 10 subjects.

Consistency of the BMR measurements was also evaluated. A single subject was tested on five occasions, following an initial exposure to the instrumentation, over a period in

which SA, weight, and LBM remained essentially constant. The results are displayed in Table 5. The mean measurement was 1789 Cal/day compared with predicted values of 1744 Cal/day (Aub & DuBois, 1917) and 1713 Cal/day (Harris & Benedict, 1919). Because the subject was quite lean (4.0% fat), BMR values greater than predicted by standard SA formulas were expected.

TABLE 5
BMR MEASURES ON A SINGLE SUBJECT

Repeated Measured Values (Cal/day)	1580	1726	1839	1801	1818	1760
Mean BMR (Cal/day)	1789					
Standard Deviation (Cal/day)	45					
(% of Mean)	2.54%					
Predicted BMR: Cal/day						
Harris & Benedict (1919)	1713					
Aub & DuBois (1917)	1744					

The standard deviation of these measurements was 2.54% of the mean. This falls well within the range of 2.5 to 5% reported by Berkson and Boothby (1937).

Six subjects were also tested twice within a two week period. The results are reported in Table 6. The Pearson product-moment correlation between the first and second measurements was $r = 0.95$.

TABLE 6
VARIABILITY OF BMR MEASUREMENT

Subject	Test 1	Test 2
1	1488	1451
2	1320	1330
3	1489	1423
4	1619	1635
5	1112	1290
6*	1801	1839

*The two values for subject #6 were selected at random from a series of 5 tests.

$$r = .95$$

Statistical Analysis

Several statistical procedures were performed on the NTSU computer system to analyze the results. All statistics were calculated for the entire sample and for each sex separately.

Means and standard deviations were calculated for all important metabolic variables. These were body weight, fat percentage, LBM, measured BMR, SA, Harris and Benedict (1919)

predicted BMR, Aub and DuBois (1917) predicted BMR and age. Three other variables were calculated to aid in the analyses. These were $10g_{10}$ (BMR), \log_{10} (LBM), and $\frac{BMR}{LBM}$.

The relationships between these variables were assessed in several ways. A Pearson product-moment correlation matrix was prepared to measure the linear associations between the above variables.

Several of these variables were also combined in a multiple regression analysis. The multiple regressions were carried out to determine if the accuracy of BMR estimation could be increased beyond that of the principal predictor alone, LBM. Added variables, suggested by a review of the relevant literature, were percent fat, age, and fat weight. Again, separate equations were calculated for each sex.

Two partial correlations were calculated to remove the effects of confounding variables. Because fat weight and LBM tend to increase together, it was necessary to calculate the correlation between BMR and LBM with fat weight held constant. Similarly, because SA tends to increase with age, the correlation between BMR and SA with age held constant was calculated.

To determine if any statistical associations differed for groups of different body composition, both sexes were divided into thirds. Group one consisted of the leanest 33% of a given sex, group three had the highest body fat

percentage, and group two was composed of all those that fell in between. One-way analyses of variance were used to determine if there were any significant differences in metabolic variables between groups.

Several transformations of the metabolic variables were employed to detect a possible curvilinear relationship between BMR and its principal predictor (see Appendix B). While Pearson product-moment correlations were used to measure the linear relationship between BMR and LBM, they were also used to measure the linear relationship between $\log(\text{BMR})$ and $\log(\text{LBM})$. A high correlation in this case would suggest a power law relationship of the form $\text{BMR} = K(\text{LBM})^x$ for constants K and x . The power law form, therefore, does not allow for a non-zero BMR intercept. Despite this restriction, it was included for comparison because it is a standard method of analyzing metabolic data.

Once LBM had been established as the strong sole predictor, curvilinearity between the two variables was assessed without this limitation. A multiple regression equation for BMR as a function of LBM, LBM^2 etc. was employed to test for significant contributions of nonlinear terms.

All relationships were cross-validated by randomly splitting the sample in half and checking for consistency of these relationships in both halves.

Finally, tables were prepared for BMR using the prediction equations statistically determined to have the greatest validity.

A Tektronix 4052 computer, along with the STAT 50 PLOT curve fitting package was used to determine graphic relationships between certain variables.

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CHAPTER IV

DISCUSSION OF RESULTS

Following a thorough analysis of the results, an attempt was made to compare our findings to those of other researchers. Implications, both practical and for future research, are also discussed.

Descriptive Statistics

Basic descriptive statistics of the 158 subjects from whom acceptable data was obtained are summarized in Tables 2 and 3.

Eighty-two participants were female and 76 were male. Thirty-six of 77 subjects between ages 18 and 29 were female as were 31 of 61 in the 30 to 39 age group. Of those subjects 40 to 49 years of age, 15 were female and 11 were male. One female and five males were between 50 and 59 years of age.

Female participants averaged 30.8 ± 8.0 years of age while the male sample had a mean age of 32.3 and a standard deviation of 8.9 years.

Average height and weight of the female sample is displayed in Table 7. Though slightly taller and lighter than the population norms reported for 1980 (Statistical

TABLE 7
 FEMALE SUBJECTS: SAMPLE HEIGHT AND WEIGHT COMPARED TO POPULATION MEANS

	Age				Overall
	18-20	30-39	40-49	50-59	
Current Study, 1984					
Mean Wt (kg)		(NTSU sample age 30.8 ± 8.0 yrs)			58.7
Ht (cm)					165.0
Statistical Abstracts of U.S. (data 1976-80)	63.2	66.4	68.6	69.5	64.8
Stouidt, Damon, & McFarland, 1960	163.3	163.0	162.3	160.8	163.2
"Reference Woman" Behnke & Wilmore, 1974	57.3	60.4	67.7	67.7	59.0
Sloan, 1962	162.3	161.3	160.5	159.3	161.8
Young, 1962	56.8 (defined to be age 20-24)				56.8
Katch & Michael, 1968	164.0				
Wilmore & Behnke, 1968	55.5 (age 17-25)				
Chen, 1953	165.0				
Young, 1963	59.0 (age 16-30)				
Diaz, 1978	167.5				
	58.4 (age 19-23)				
	165.9				
	58.6 (age 17-29)				
	164.9				
	60.7 (age 31-45)				
	163.3				
	59.6 (sample age 30-39 & 40-49)				
	164.9				
	56.4				
	163.1				
	57.4 (age 18-26)				
	165.0				

Abstracts of the U.S., 1984) the means of 58.7 kg and 165 cm are very similar to the sample means for studies of young men reported in Table 7 (Behnke & Wilmore, 1974; Chen, 1953; Diaz, 1978; Katch & Michael, 1968; Sloan, 1962; Young, 1963; Wilmore & Behnke, 1968).

Body composition values of various samples of females are displayed in Table 8. A brief survey shows that fat percentage means seem to fall between 22 and 28% for women under age 30 and approximately 28 and 32% in samples of women 30 to 50 years of age. The mean in our sample, 24.5%, could be classified as typical for a group of females with a mean age of 30 years. If one combines the 18 to 29 and 30 to 39 age categories, the mean fat percentage values for this expanded group of mean age 30.0 are strikingly similar to our value in several of these studies. This mean was 25.25% for the Aerobics Center population (1984), 24.6% for Pollock, Wilmore, and Fox (1978) and 24.0% in the Parizkova sample (1977). An almost identical 24.7% mean was also found in the most recent study involving body composition performed by members of the NTSU Physical Education Department (Jackson & Gibson, 1984).

The male sample had a mean height of 177.7 cm and mean mass of 77.6 kg (Table 9). Again, if one assumed that the two youngest age categories in the Statistical Abstracts (1984) and Stoudt, Damon, and McFarland (1960) had mean ages of 23.5 (18 to 29) and 34.5 (30 to 39) years respectively,

TABLE 8

FEMALE SUBJECTS: COMPARISON OF SAMPLE BODY COMPOSITION
TO GENERAL POPULATION MEANS
(all numbers indicate body fat percentage)

	Age				Overall
	18-29	30-39	40-49	50-59	
Current Study, 1984					
Mean Fat %	6.1-46.0	10.3-49.0	17.6-40.0	27.3-27.3	24.5±8.6
Range				¹	
N					
Aerobics Center, 1984 ¹	25.5	25.0	27.5	29.3	
Pollock, Wilmore, & Fox, 1978 ¹	25.0	24.2	25.0	26.0	
Parízková, 1977	23.0 ²	25.0	26.8	30.5	
"Reference Woman," Behnke & Wilmore, 1974	27.0 ³				
Sloan, 1962 ⁴	22.9				
Young, 1962 ⁵	28.7				
Jackson & Gibson, 1984 ⁶	24.7				
Chen, 1953 ⁷		28.9			

¹ Figures represent medians rather than means; means would probably be slightly greater.

² Data is from age interval 20-29.

³ "Reference Woman" is defined to be 20-24 years of age.

⁴ Age 17-25 only.

⁵ Age 16-30 only.

⁶ College age females.

⁷ Age 31-45 only.

one could interpolate between these values to our age group mean of 32.3 years. Because size is dependent upon age and date at birth even in an adult population, this age correction is necessary in order to make meaningful comparisons between populations. This is, of course, just a first-order approximation to what is really a weighted average of all age intervals, but probably provides a useful coarse adjustment from the discrete age categories to our population mean age of 32.3 years. Adjusted in this way, the means become 80.2 kg and 176.6 cm (Statistical Abstracts of the U.S., 1984) and 75.0 kg and 174.1 cm (Stoudt, Damon, & McFarland, 1960). The differences between these two figures may be largely the time of sampling; Statistical Abstracts chronicles a 2.5 cm height increase and a 3.7 kg weight increase between 1960 and 1980. Our population was still 1.2 cm taller which may reflect the observed tendency for Texans to be taller than the national average (Tyroler, 1958, cited in Stoudt, Damon, & McFarland, 1960), attributed to British and northwest European ancestry common in this region. A university-based population in 1984, however, probably minimizes this effect. In any case, the height and weight of the male sample appear to be very similar to those found in past studies.

The mean body composition of 15.3% also falls well within the range of 13 to 18% reported elsewhere for males of similar age (Aerobics Center, 1984; Brozek, 1952; Brozek

TABLE 9
 MALE SUBJECTS: SAMPLE HEIGHT AND WEIGHT COMPARED
 TO POPULATION MEANS

	Age			Overall
	18-29	30-39	40-49	
Current Study, 1984				
Mean Wt (kg)				77.6
Ht (cm)				177.7
Statistical Abstracts of U.S., 1984: Data 1976-80				
Mean Wt (kg)	77.3	80.9	81.8	80.9
Ht (cm)	177.0	176.5	175.8	174.5
Stoudt, Damon, & McFarland, 1960				
Mean Wt (kg)	73.2	75.4	75.9	75.0
Ht (cm)	174.5	174.0	172.7	170.9
"Reference Man," Behnke & Wilmore, 1974				
Mean Wt (kg)				70.0
Ht (cm)				174.0

1 "Reference Man" is defined to be 20-24 years of age.

& Keys, 1951; Katch & McArdle, 1973; Myhre & Kessler, 1966; Parizkova, 1977, Pollock, Wilmore & Fox, 1978; Wilmore & Behnke, 1968). Table 10 shows that sample means seem to vary by nearly 10% for a given age so our mid-range figure is very acceptable.

Estimated surface area, calculated by the formula of DuBois & DuBois (1916) from mean height and weight data was 1.64 m^2 for females and 1.96 m^2 for the male sample. Because the mean heights and weights fall within expected ranges for both sexes, it is reasonable to assume that both figures represent typical population means although SA norms are not available.

Another essential variable that was calculated from anthropometric data was LBM. Mean lean body mass was 44.3 kg for women and 65.2 kg among men. Again, because body weight and composition are in agreement with previously established norms, LBM must also approximate values found elsewhere on similar populations.

Mean BMR measurement for the entire sample was 1568 Cal/day. The mean Aub and DuBois estimate (1917) for our population was 1635 Cal/day, a 5.8% overestimate. The Harris and Benedict (1919) prediction was 1580 Cal/day, a 2.0% overestimate.

When separated by sex, it was apparent that both prediction formulas were more accurate for men and that Aub

TABLE 10

MALE SUBJECTS: COMPARISON OF SAMPLE BODY COMPOSITION
TO GENERAL POPULATION MEANS
(all numbers represent body fat percentage)

	Age				Overall
	18-29	30-39	40-49	50-59	
Current Study, 1984					15.3±9.3
Mean Fat%, St. Dev.	3.9-15.7	2.5-35.0	8.6-34.8	10.0-39.0	
Range					
N					
Aerobics Center, 1984 ¹	18.1	22.0	23.5	23.8	
Brozek, 1952	13.5	18.6	22.7	24.6	
Pollock, Wilmore, & Fox, 1978 ¹	21.0	22.2	23.9	24.8	
Myhre & Kessler, 1966 ²	15.3	19.6	22.2	27.0	
Parízková, 1977 ³	11.4 ³	18.0	18.9	21.2	
"Reference Man," Behnke & Wilmore, 1974 ⁴	15.0 ⁴				

¹ Figures represent medians rather than means; means would probably be slightly greater.

² Means were determined by graphic methods pooling different age intervals.

³ Data is from age interval 20-29.

⁴ "Reference Man" is defined to be 20-24 years of age.

and DuBois in general produced a greater overestimate than did Harris and Benedict.

Mean female BMR was 1335 Cal/day compared to 1444 Cal/day predicted by Aub and DuBois (a 9% overestimate) and 1376 Cal/day by Harris and Benedict (4.0% overestimate). Among men, measured values averaged 1824 Cal/day. Aub and DuBois overestimated this figure by 1.1% (1844 Cal/day). Harris and Benedict gave a general 1.0% underestimate with a mean prediction of 1803 Cal/day.

Primary Correlations

Pearson product-moment correlation coefficients were calculated between basal metabolic rate and the major predictive variables indicated by previous research. These are displayed in Table 11.

For the entire sample, the correlation between BMR and LBM was 0.91. Over 83% of the variability in BMR can therefore be predicted by variation in LBM, as illustrated in Figure 1. The linear relationship between BMR and SA was 0.75 which implies that surface area accounts for approximately 56% of the variation of BMR.

When the sexes were viewed separately, similar correlations with LBM were found in both the male and female samples. Among males, the correlation with LBM was 0.79 compared with 0.77 for females. Using SA as the predictor, the correlations were 0.62 for men and 0.51 for women.

TABLE 11
 PEARSON CORRELATION COEFFICIENTS BETWEEN BMR AND SEVERAL
 HYPOTHESIZED PREDICTORS: COMPARISON TO OTHER STUDIES

	Weight	LBM	SA	% Fat	Fat Weight
Present Study, 1984					
All subjects (N=159)	.7547	.9137	.8006	-.4898	
Females only (n=83)	.4405	.7726	.5115	-.1115	
Males only (n=76)	.4367	.7907	.6242	-.3158	
Miller & Blythe, 1953	.82	.92	.84		
Keys et al., 1973	.417		.447		
Johnstone & Bernstein, 1955		.94	.92		
Cunningham, 1980		.84			
Bernstein et al., 1983	.655	.650		.597	
James et al. 1978		.829		.512	
Ravussin et al., 1982	.741	.822	.787	.419	
Bray et al., 1970	.615	.563	.540	.723	

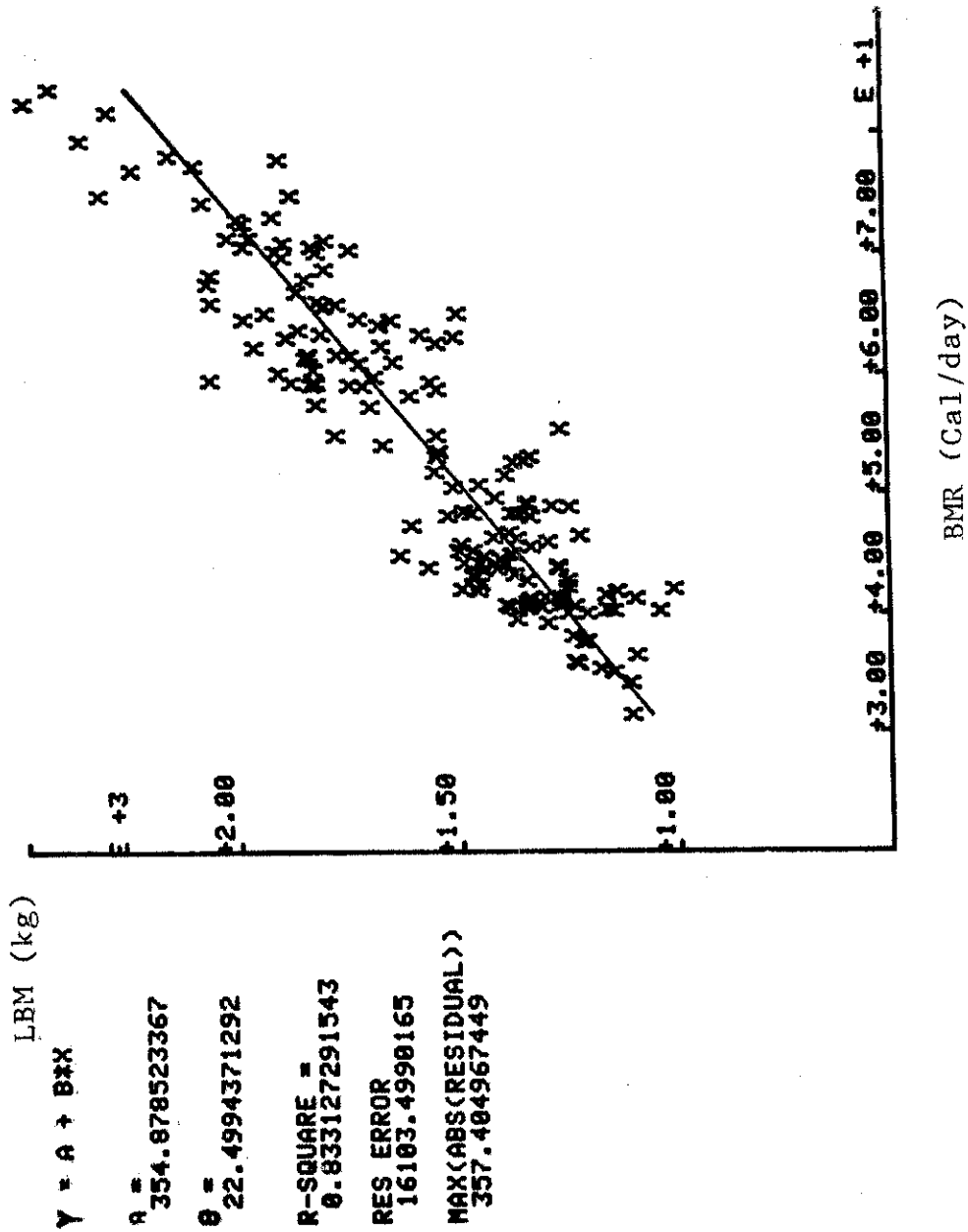


Fig. 1--Linear regression for all subjects: BMR vs LBM.
 [BMR (Cal/day) = 22.5 (LBM, kg) + 355 Cal/day, $r^2 = .833$]

The predictive value of body weight was much less than that for either LBM or SA. This was the case for both sexes individually as well as for the entire sample. Overall, this correlation was 0.75 while it was 0.44 among both males and females viewed separately.

Because the relationships between metabolic rate and the selected physical variables are dependent upon the body types of the populations studied, it is not surprising that the results of the present study are similar to those found elsewhere on samples with heights and weights in typical proportions.

Most studies have shown the strongest correlate of BMR to be LBM. As one would expect, the highest correlations have been found when fat weight is minimized because the LBM itself is then generating the vast preponderance of metabolic energy. This explains the relatively high ($r = 0.92$) correlation between BMR and LBM found in population of 48 lean young men by Miller and Blythe (1953). Correlations in this study were also fairly high using weight ($r = 0.84$) and SA ($r = 0.82$). Presumably, this was because these variables correlated highly with LBM in this group. In lean males, it is intuitively clear that weight and LBM associate closely. The less obvious relationship between LBM and SA, however, showed a correlation of 0.79. When the partial correlation between BMR and SA was adjusted for this influence of LBM, r fell to only 0.40.

All correlation coefficients in the Miller and Blythe study were similar to though slightly higher than those found in the present study. The only divergence between our results was the BMR relationship with weight. This correlation was 0.75 in our study compared with 0.82 found by Miller and Blythe. This was apparently due to the much stronger relationship between LBM and weight in the leaner, earlier sample.

Johnston and Bernstein (1955) also had slightly higher values than those found in our research. Studying a sample of 17 females ranging in mass from 39 to 186 kg, they demonstrated correlations with BMR of 0.92 and 0.94 for LBM and SA respectively. Keys, Taylor, and Grande (1973) point out, however, that a very small sample distributed over a wide range of the independent variable (LBM) vastly inflates the linear correlation by "creating a line". Removing the five grossly obese subjects (over 117 kg) and one emaciated woman (40% underweight), the correlations with LBM and SA drop to 0.17 and 0.03. The remaining sample, though much smaller, would be more comparable to the normal population sampled in the present study.

The only study to assess the relationship between BMR and LBM on a normal population was the classic study of Cunningham (1980). Focusing on the 223 subjects of the original Harris and Benedict study (1919), Cunningham obtained correlation of 0.84 between BMR and LBM estimated

from height, weight, sex, and age data. Though similar to the 0.9131 of the current study, Cunningham's value may have been slightly deflated by the unavailability of actual measured LBM information.

Keys et al. (1973) also studied BMR in a very large, general population ranging in age from 19 to 77. The Pearson product-moment correlation coefficient between BMR and body weight was a low 0.417. The value for BMR vs SA was an only slightly higher 0.447. Unfortunately, LBM was not determined. Because the average individual loses approximately 0.25 kg of LBM while gaining 0.50 kg of adipose tissue each year after age 30 (Burmeister & Bingert, 1967; Forbes & Welle, 1983; Pollock, Wilmore, & Fox, 1978), there is a continual dissociation of LBM and weight throughout adulthood. Surface area is also calculated partially from body weight so that SA and LBM must also diverge with advancing age. Assuming BMR is tracking with LBM as it generally seems to do, SA and weight become poor predictors over such a wide range of ages. These aging trends may explain a great deal of the difference between the results of this study and the higher values obtained in our research.

Metabolic studies on obese groups have usually had less success predicting BMR from LBM alone. This may be because the heat generated by adipose tissue is no longer negligible. Bernstein et al. (1983), Ljunggren, Ikkos, and

Luft (1961), and others have concluded that adipose tissue respire at a rate 1/8 to 1/3 that of lean tissue per unit mass. It has also been noted (Bernstein et al., 1983) that the univariate regression equations for BMR as functions of LBM developed by Cunningham (1980) and Tsankoff and Norris (1977) both have positive intercepts on the BMR axes. Because this intercept represents the expected BMR for an LBM of zero, this can be interpreted as the heat generated by the non-LBM compartment, i.e., adipose tissue. This value is 501 Cal/day using the equation from the Cunningham study. The Tsankoff and Norris equation reduces to 99.1 ml O₂/min in the absence of LBM which, if one assumes a typical basal value of 4.801 Cal/L O₂ (RQ = 0.80), gives a very similar figure of 476 Cal/day. Though the magnitude of this contribution is approximately twice that calculated from bloodflow and oxygen extraction data (Brooks & Fahey, 1984, pp. 332-333; McArdle, Katch, & Katch, 1981, p. 108, 225-226), it does suggest that the heat generated by fat tissue may not be negligible.

Several of the BMR studies summarized in Table 11 were carried out on groups with mean body composition in excess of 40% fat. The actual metabolic contribution of this fat tissue may be modified as adipocytes appear to vary their heat production in inverse proportion to their size. A recent Swedish study (Sörbris, Monti, Nilsson-Ehle, & Wadsö, 1982), for example, found that although an obese

group carried five times as much fat as a normal weight group, the heat generated by this tissue was only 1.65 times as great. This moderating influence probably explains why LBM is still generally the best predictor of BMR even in populations in which LBM may represent no more than 50% of the body weight. Nevertheless, the sheer bulk of fat tissue creates an additional energy expenditure that cannot be accounted for by LBM alone. As a result, though LBM may be the best predictor, the accuracy of prediction is lowest among the group for whom estimates are most often necessary: obese subjects.

It has also been suggested that the varied mass distribution in these obese subjects, particularly females, may differentially affect the work of respiration and hinder attempts at BMR estimation. Ljunggren, Ikkos, and Luft (1961) have raised the possibility that the extra work performed in raising the weighted chest wall may effectively preclude an obese subject's reaching a truly basal state. In their study, an overweight group expended approximately 25% more work in ventilation than controls did. Figures presented by Guyton (1981, p. 479) however, indicate that the work of ventilation typically accounts for only 2 to 3% of basal energy expenditure. This suggests an overall metabolic elevation of less than 1%, approximately half the expected variation in measurement on a single subject on consecutive days (Berksen & Boothley, 1937). On the basis

of these figures, the work of respiration seems unlikely to be a source of significant variation even among obese subjects.

Though secondary to LBM, the thickness of the fat blanket does seem to have some effect on BMR. In fatter populations, fat weight correlates reasonably well with BMR. Unfortunately, total fat weight was not tested as a predictive variable in any of the previously mentioned work on normal populations.

James, Bailes, Davies, and Dauncey (1978) attribute the increased correlation of BMR and fat weight to the often observed increase in LBM associated with obesity. Though fat weight correlated at $r = 0.512$ with BMR in an obese group, once the co-correlation between fat weight and LBM ($r = 0.582$) had been removed, the contribution of body fat to the variability in BMR was only 0.2%. If increased LBM is the culprit, one would expect increased correlations with fat weight as fat increased but still much lower than the correlation with LBM. James et al. data, collected from a moderately overweight sample, does fit this expectation: BMR vs fat weight, $r = 0.512$; BMR vs LBM, $r = 0.829$.

Ravussin, Burnard, Schutz, and Jecquier (1982) had similar results on a heterogeneous sample that could also be characterized as moderately obese. The relationship between BMR and LBM ($r = 0.822$) was slightly lower than was found in our study while the associations with weight

($r = 0.741$) and SA ($r = 0.787$) were slightly stronger. On this sample, the correlation with fat weight was also a moderate $r = 0.419$.

Bernstein et al. (1983) investigated the same relationships on a population of 202 subjects averaging 77.4% overweight by the Fogarty Conference Tables. Significant correlations were found between BMR and LBM ($r = 0.650$) and weight ($r = 0.655$). These were much lower than we found in the current study, again demonstrating the lowered predictability of BMR for obese populations. Fat weight, however, showed a relatively high 0.597 correlation with BMR. Apparently, the contribution of fat metabolism, which increases BMR slightly faster than LBM increases, is picked up more and more by correlations with body weight and fat weight in obese groups.

An extreme example of this phenomenon may be seen among the results of a study by Bray, Schwartz, Rozin, and Lister (1970). The subjects were 18 extremely obese females: one was listed at 3'8" (112 cm), 264 lb (119.4 kg) and another at 5'6" (168 cm) and 469 lb (213 kg). Overall, the subjects averaged 330 lb (150 kg) and 60% body fat. In this case, the highest correlate with BMR was fat weight ($r = 0.723$) while body weight ($r = 0.615$), LBM ($r = 0.563$), and SA ($r = 0.540$) showed much lower values than one would have expected from a normal, leaner population.

Although the great differences between this sample and the group that we used in the present study make comparison difficult, they underline apparent population differences in the major predictors of BMR. In summary, LBM appears to be the best for all but grossly obese populations with surface area slightly behind in accuracy of estimation. The actual correlations with LBM are highest in populations of lean young men. Surface area and body weight also show their highest correlation on populations of this sort. With increasing body fat, correlations with body fat and body weight seem to increase and in extreme cases, may exceed the value for LBM as the single best predictor of BMR.

For the NTSU sample, the utility of the LBM prediction is compared with that for the primary SA formulas in Table 12. Both Harris and Benedict (1919), $r = .8664$ and Aub and DuBois (1917), $r = .8524$, predict BMR quite well overall. The simple BMR vs SA correlation is $.8006$, indicating that the age correction factors which are introduced into the SA formulas (as an unknowing attempt to help track LBM) do aid in BMR prediction. They fail to reach the level of accuracy achieved by LBM alone ($r = .9128$) however.

The same is true when the sexes are analyzed separately. The correlation between LBM and BMR among males is $.7907$. The Harris and Benedict ($r = .7172$) and Aub and DuBois ($r = .7027$) estimates also do quite well. The

TABLE 12
BMR PREDICTION: CORRELATIONS WITH NTSU SAMPLE

	Male n = 76	Female n = 82	Overall N = 158
LBM Linear Regression NTSU, 1984	.7907	.7726	.9128
BMR vs SA	.6242*	.5115*	.8006*
Harris & Benedict, 1919	.7172	.5528*	.8664
Aub & DuBois, 1917	.7027	.5606*	.8524

*Indicates that r is significantly less than BMR vs LBM correlative coefficient ($\alpha = 0.5$).

correlations produced for the female sample (.5528, Harris & Benedict; .5606, Aub & DuBois) both fall well short of the LBM correlation of $r = .7726$.

Prediction Accuracy: Sex Differences

One obvious group breakdown that has minor effect on the accuracy of BMR estimation is sex. Referring to Table 13, it should be noted that the relationships with all variables are higher for men than they are for women. Similarly, the Aub and DuBois prediction tables (1917) produced an average 9.0% overestimate for females while Harris and Benedict (1919) gave an average 4.0% estimate. The figures for males were 1.0% high (Aub & DuBois) and 0.1% too low (Harris & Benedict).

These major overestimates relative to the present female sample might suggest a lessened LBM compared to SA

TABLE 13
 MEAN BMR VALUES, STANDARD DEVIATIONS, AND COMPARISON
 TO PREDICTED VALUES

	All Subjects N=158	Females n=83	Males n=76
Present Study, 1984 Cal/day \pm St. Dev.	1568 \pm 310	1335 \pm 140	1824 \pm 234
Aub & DuBois, 1917 Cal/day \pm St. Dev.	1635 \pm 240	1444 \pm 137	1844 \pm 127
<u>Aub. & DuBois (predicted)</u> BMR (measured)	1.058 \pm .127	1.090 \pm .129	1.011 \pm .115
Harris & Benedict, 1919 Cal/day	1580 \pm 259	1376 \pm 127	1803 \pm 155
<u>Harris & Benedict (predicted)</u> BMR (measured)	1.020 \pm .120	1.040 \pm .129	0.999 \pm .108

between the original study and the present. This is another way of saying that these earlier female populations may have been slightly leaner than those today while the excellent agreement between SA prediction and measured BMR among males suggests little change for men.

Though admittedly a tenuous extrapolation, height and weight data from the last 20 years (Statistical Abstracts of the U.S., 1984) does suggest that the ponderal index ($[\text{weight}]^{1/3}/\text{height}$), a measure of "stockiness," does suggest that women have thickened somewhat while men have not. This thickening, of course, implies an increase in body fat but unfortunately this trend cannot be documented back to this period of earlier BMR study. Further, the variation between sample populations of nearly 10% fat (McArdle, Katch, & Katch, 1981, p. 389) may minimize the need for an explanation based on a change in the general population.

One conclusion that is definitely supported by recent research is that the male BMR is simply easier to predict from anthropometric data than is the female BMR. Bernstein et al. (1983) presented seven multivariate equations for BMR and in each case, the coefficient of variation was much higher for males than for females. Controlling for periodic hormonal changes thought by Benedict (1915, pp. 263-288) to contribute to these errors may have helped in our study. This conclusion should be accepted with caution,

however, because the failure to exclude one extreme subject judged to be abnormal metabolically (by our 25% deviation criterion; she was labelled "low normal" by a physician when tested by other means) lowered r^2 from 0.77 to 0.52. The latter value would be typical of other studies.

Multiple Regression Analysis

Much research has focused on the possibility of using more than one predictor for basal energy expenditures. Because results differ for each sex, men and women were analyzed separately. Inspection of the initial correlation matrix between all variables highlighted only three major variables correlated highly with BMR but showed lesser associations between each other. These were LBM, fat weight, and age.

The predictive utility of both LBM and fat weight have already been discussed and seems physiologically sound. The effect of age may be a bit more subtle in that it appears to alter the relative proportions of the various components of LBM. Tsankoff and Norris (1977) have demonstrated that nearly all of the loss of LBM with age is from the skeletal muscle compartment while the changes in relative proportions of the various organs with time are not altogether clear (Parízková, 1968). In any case, because the proportions of LBM made up of different tissues change with age and, because the metabolic rates of these tissues differ, the overall metabolic rate of LBM per unit

mass must change with age. It is therefore not unreasonable to hypothesize an increased accuracy of estimation by adding fat weight and age to LBM in the multivariate equation.

When entered into the equation, however, neither can contribute significantly to the estimate provided by LBM alone. The best multivariate equations are thus simply functions of LBM:

$$\text{Males: BMR (Cal/day)} = 23.78 \text{ LBM (kg)} + 272.7 \quad (10)$$

$$\text{SE} = 143 \text{ Cal/day}$$

$$r^2 = .7907$$

$$r^2 = .6254$$

$$\text{Females: BMR (Cal/day)} = 18.43 \text{ LBM (kg)} + 531.6 \quad (11)$$

$$\text{SE} = 89 \text{ Cal/day}$$

$$r = .7725$$

$$r^2 = .5968$$

$$\text{Overall: BMR (Cal/day)} = 22.5 \text{ LBM (kg)} + 355 \quad (12)$$

$$\text{SE} = 127 \text{ Cal/day}$$

$$r = .9127$$

$$r^2 = .8331$$

The overall equation is quite similar to the Cunningham equation (1980) of

$$\text{BMR (Cal/day)} = 22.0 \text{ LBM (kg)} + 501$$

The only difference concerns the BMR intercept for an LBM of zero. It should be recalled that this has been hypothesized to represent the basal energy output not attributable to LBM, i.e., to fat metabolism. Actual tissue studies

suggest that fat metabolism contributes 5 to 10% to basal energy expenditure (Bernstein et al., 1983; Ljunggren et al., 1961) while both Cunningham's (1980) and Tsankoff and Norris' (1977) equations both suggest over 25%. The intercept in this study, though lower, still represents 22% of the mean BMR.

Past studies employing multivariate analysis have generally shown LBM to be either the sole predictor or at least to account for the vast majority of variability in BMR. Cunningham (1980) showed BMR and LBM only to associate with an $r = 0.84$ while inclusion of age, height, sex, and age only increased r to 0.86. James et al. (1978) and Ljunggren et al. (1961) reached the same conclusion while focusing on obese populations. The correlation coefficients were slightly lower. Bernstein et al. (1983) concluded that both age and total fat mass could add somewhat to the prediction of BMR provided by LBM. Because their correlations are not reported in a stepwise fashion, however, the magnitude of this additional contribution cannot be reported.

The fact that other variables emerge only slightly and inconsistently among BMR studies strongly suggests that LBM is the best known predictor of BMR in the normal population. The results of our study certainly support this contention.

Partial Correlations

To separate the contributions of certain associated variables, three important partial correlations were calculated for each sex.

Body surface area and LBM are two variables that tend to track together in human populations. As an illustration, one should consider a pair of 220 lb (100 kg) individuals, one of whom stands 5 feet (1.52 m) tall while the height of the other is 7 feet (2.13 m). The shorter, more globular shaped subject would have a lower body surface area than the tall, attenuated individual. He would also almost certainly be much fatter and, since the weights are equal, have a smaller lean body mass. Therefore, LBM and SA tend to track together over a wide range of body types, i.e., subjects with lesser LBM generally have smaller SA. As mentioned earlier, it is partially the relationship between LBM and SA that has allowed SA to serve for 60 years as the reference standard for BMR.

Age, however, tends to break down this association between lean body mass and surface area. Both males and females tend to gain weight slowly with advancing age and this tends to increase the surface area of the body. At the same time, the lean body mass declines at a rate depicted in Appendix C (Burmeister & Bingert, 1967). A simple correlation between BMR and SA over a sample heterogeneous in age would therefore underestimate the relationship between BMR and SA with age held constant.

Because our sample was made up of subjects from 18 to 59 years of age, it was necessary to calculate this partial correlation between BMR and SA with age held constant.

Among females, while BMR and SA are related by $r = 0.5115$, the partial correlation with age held constant increases only to $r = 0.5205$. This is still far less than the BMR vs LBM correlation of 0.7726. In the male sample, the correlation coefficient increased from 0.4581 to 0.5970 for the partial correlation. Despite the sizable increase, SA again failed to challenge BMR ($r = 0.7907$) as the dominant predictor.

The reason that the age correction had a much greater effect on the male correlation can readily be understood with reference to Appendix C. Males lose LBM much more rapidly with age than females do as measured by the slopes of the two LBM curves through adulthood. In our sample, at least, males also gain more weight with advancing age. This is suggested by the moderate and direct relationship found between SA and age (0.1929) as opposed to -0.0370 among females. This greater increase in weight along with the steeper decline in LBM intensifies the age dissociation between SA and LBM among males.

Because LBM and fat weight tend to increase together in obesity, it was necessary to compute the partial correlation between BMR and LBM with fat weight held constant. Otherwise, the metabolic contribution of this fat tissue could distort the true variance in BMR directly attributable to changes in LBM. (See Appendix D for a more thorough graphic explanation.)

The partial correlation between BMR and LBM with fat weight held constant was 0.7816 for males compared with

0.7907 for the simple correlation. The slightly greater simple correlation indicates that the fat tissue did enhance the BMR vs LBM correlation slightly.

For females, BMR and LBM at constant fat weight gave a correlation of $r = 0.8260$. Without removing the influence of fat, the correlation was $r = 0.7726$. The additional fat therefore did have a tendency to lower the BMR-LBM curve at higher LBM.

A third partial correlation, BMR vs fat weight at constant LBM, assesses the effect of fat tissue on metabolic rate apart from the effects of the increased LBM at high fat levels. This partial correlation was essentially zero for males, implying that fat tissue, either by metabolic activity or insulation, had only a very minute effect on the BMR-LBM curve. For women, this same partial correlation was $r = -0.2960$. This suggests that as fat weight increases, BMR decreases slightly when the metabolic effect of the additional LBM is ignored. The two most obvious explanations for this observation might be:

1) subjects with higher fat content tended to have lower BMR in the first place, or 2) extra fat increases insulation and depresses heat production to maintain body temperature. Though admittedly the observed trend is very moderate, there is nothing in either of these explanations that would necessarily apply more to females than to males.

Apparently this is simply another curious example of differences in BMR between males and females.

Curvilinearity

Up to this point, only the linear relationships between variables have been discussed. It is possible that BMR might bear a curvilinear relationship to its major predictor, LBM.

By essentially the same argument given algebraically in Appendix A, it seems reasonable to expect increases in LBM to be associated with smaller and smaller increments in BMR. In other words, BMR would be proportional to LBM raised to some power less than one.

If BMR and LBM were related linearly, each gram of lean tissue would produce the same quantity of heat. Because heat can only be dissipated from the surface which increases more slowly than weight and LBM do, larger bodies would have a problem with heat buildup unless the rate of heat generation was lower than in smaller bodies.

Because obesity increases surface area without increasing LBM, one could argue that SA may not increase much more slowly with weight than LBM does in human populations. However, not only does fat tissue add some heat generation of its own, it presents an insulative barrier to heat loss that should further add to heat buildup problems if BMR per unit LBM was a constant value (Jecquier, Gygas, Pittet, & Vanotti, 1974).

Most of the studies that have addressed nonlinear models for BMR have focused on weight. These include the series of animal studies summarized by Kleiber's Law: $BMR = k (\text{weight})^{0.73}$ and Galvão's (1948) work on human populations. Galvão found BMR to be proportional to $(\text{weight})^{0.78}$ generally and to $(\text{weight})^{0.83}$ when he restricted his focus to leaner subjects. The lowered exponent in the fatter population is probably caused by the failure of BMR to keep pace with weight increases because LBM and weight diverge with higher weight in obese groups. The extreme example of this trend may be seen in the results of Bray et al. (1970). His sample contained several grossly obese subjects in excess of 60% fat and the exponent was 0.5824.

Miller and Blythe (1953) did try a curvilinear regression between BMR and LBM. This resulting equation, $BMR = 17.35 (\text{LBM})^{0.64}$, was based on a sample of 48 lean young men. The exponent is surprisingly low considering Galvão's (1948) work on a lean male population. One would expect a higher exponent for LBM than for weight because the obesity effect already discussed would no longer be a factor.

Using the same technique, a linear regression between $\log(BMR)$ and $\log(LBM)$, our data suggested a proportionality of BMR to $LBM^{0.75}$. A polynomial regression between BMR and LBM, LBM^2 etc. to test curvilinearity was also performed and the relationship was shown to be strictly linear.

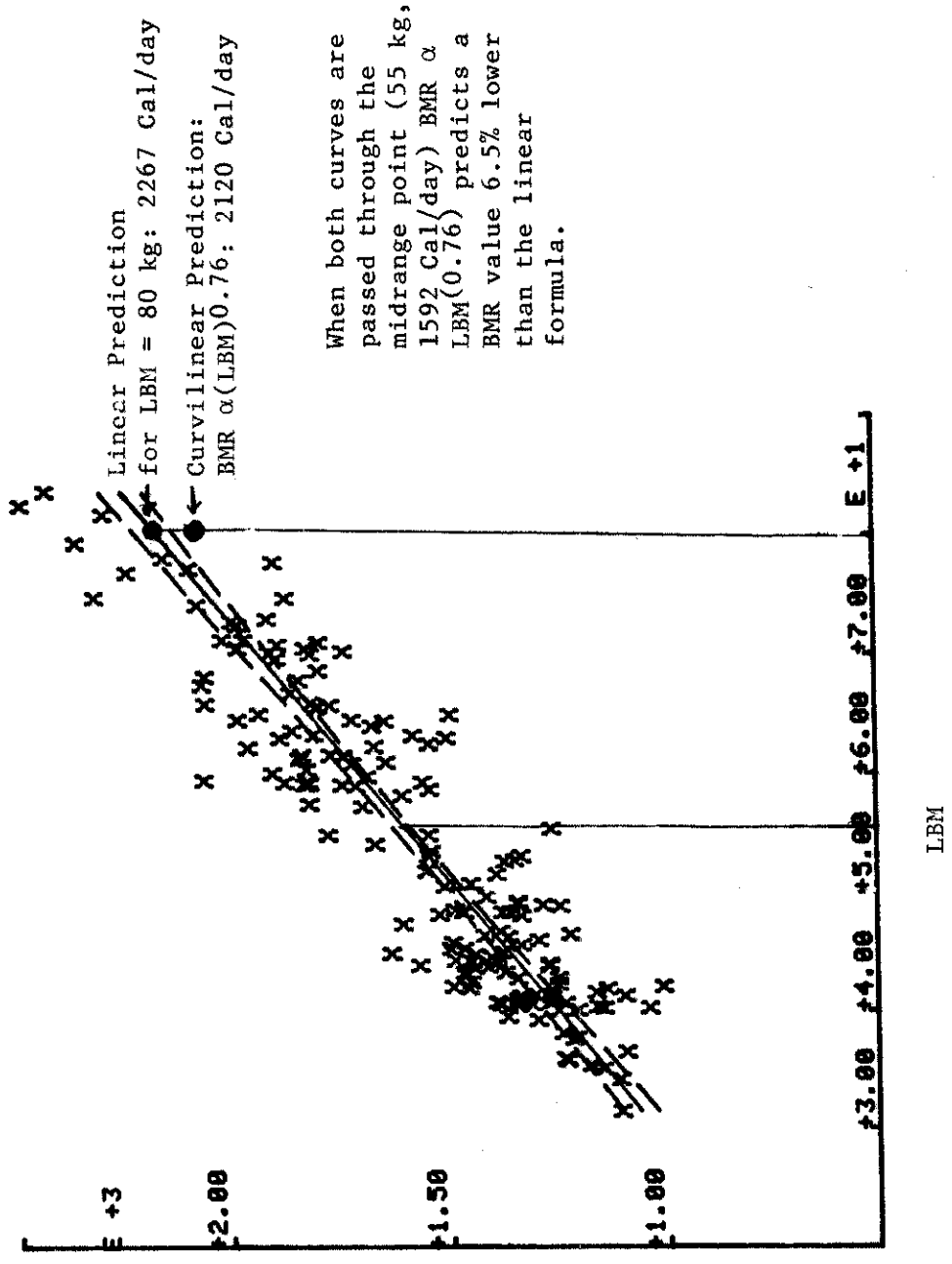
The apparent inconsistency is probably explained by the difficulty with the logarithmic regression discussed in Appendix B, Part 2. A power law regression must include the point 0,0 and plots of BMR vs LBM or BMR vs weight generally seem to have positive intercepts. Fitting a curve to include the body of the data envelope as well as the point 0,0 requires a curve that is convex upward and an exponent of less than one. This may explain the discrepancy between our linear dependence of BMR on LBM and the lower exponents obtained by previous authors, as well as in the present study when the power law form is used.

It should also be noted that the difference between the two is very slight over the range of body sizes for any given species. Taking a mid-range human LBM of 55 kg and a high value of 80 kg, one can calculate predicted BMR for both by the linear model. These values are 1592 and 2267 Cal/day. Passing the curvilinear model through the low value results in a fall-off only to 2120 Cal/day at 80 kg. This is only slightly below the 95% confidence interval for the positioning of the linear regression line (see Figure 2). The difference between the two, approximately 6.5%, is not much greater than the variation expected between repeated determinations on the same individual. Whether the relationship is generally linear within humans is really only of theoretical interest.

```

Y = A + B*X
A = 354.878523367
B = 22.4994371292
R-SQUARE = 0.833127291543
RES ERROR 16183.4998165
MAX<ABS(RESIDUAL)> 357.484967449

```



When both curves are passed through the midrange point (55 kg, 1592 Cal/day) BMR \propto LBM(0.76) predicts a BMR value 6.5% lower than the linear formula.

Fig. 2--Comparison of linear and curvilinear estimates.

Tables of expected BMR levels for adult males and females based on LBM (by linear models) are presented in Appendices F and G.

Groupings

Because many of the basic results of metabolic studies differ according to the population studied, we divided our sample into six groups to focus on specific populations. The two variables used for the group divisions were sex and body composition.

The total population was first divided into male and female samples. Each sex was then broken at both the 33rd and 67th percentiles yielding three groups of approximately equal numbers. Data for the various groups are presented in Tables 14 and 15.

The leanest third of the female sample (group LF) consisted of females between 16.1 and 19.3% fat. The middle group (MF) covered the range of 19.4% to 28.2% and the fattest group (FF) contained all females in the study carrying from 28.3% fat to our sample maximum of 53%. The male breakdown was as follows: 2.5% to 9.5% (LM), 9.6% to 17.1% (MM) and 18.1% to 39.0% fat (FM). As one would expect, the sample means of each sex were squarely within but slightly beyond the midpoint of the middle thirds.

The first notable result is the general LBM decline from the leanest to the fattest groups in both sexes. This

TABLE 14

COMPARISON OF PHYSICAL CHARACTERISTICS AND METABOLIC RATE BETWEEN
GROUPS OF FEMALES SEPARATED BY BODY FAT PERCENTAGE
(N=83)

	Leanest (L) (n=27)	Middle (M) (n=28)	Fattest (F) (n=28)
Fat % : Range	6.1 - 19.3	19.4 - 28.2	28.3 - 49.0
Mean \pm St. Dev.	15.4 \pm 3.3	23.8 \pm 2.8	33.8 \pm 5.8
LBM (kg)			
Mean \pm St. Dev.	44.7 \pm 4.1	44.2 \pm 4.7	43.0 \pm 6.8
Fat Wt (kg)			
(Mean \pm St. Dev.)	8.13 \pm 1.74	13.6 \pm 1.60	22.2 \pm 3.80
SA (m ²)			
(Mean \pm St. Dev.)	1.57 \pm .08	1.63 \pm .08	1.71 \pm .21
Age (yrs)			
(Mean \pm St. Dev.)	27.2 \pm 5.6	31.7 \pm 7.7	34.5 \pm 8.3
BMR (Cal/d)			
(Mean \pm St. Dev.)	1346 \pm 116	1362 \pm 148	1295 \pm 147
<u>Aub & DuBois (est.)</u> BMR (measured)	1.041 \pm .081	1.065 \pm .097	1.162 \pm .163
<u>Harris & Benedict (est.)</u> BMR (measured)	.999 \pm .079	1.012 \pm .104	1.106 \pm .163
<u>BMR</u> Wt Cal/kg-d (Mean \pm St. Dev.)	25.49 \pm 2.20	23.76 \pm 2.58	19.73 \pm 2.24
<u>BMR</u> LBM Cal/kg (Mean \pm St. Dev.)	30.27 \pm 2.54	31.00 \pm 3.08	30.52 \pm 3.53

TABLE 15
 COMPARISON OF PHYSICAL CHARACTERISTICS AND METABOLIC RATE BETWEEN
 GROUPS OF MALES SEPARATED BY BODY FAT PERCENTAGE
 (N=76)

	Leanest (L) (n=25)	Middle (M) (n=26)	Fattest (F) (n=25)
Fat % : Range (Mean \pm St. Dev.)	2.5 - 9.5 6.8 \pm 1.9	9.6 - 17.1 12.6 \pm 2.5	18.1 - 39.0 26.7 \pm 6.4
LBM (kg) (Mean \pm St. Dev.)	67.9 \pm 8.2	66.9 \pm 7.7	60.9 \pm 5.5
Fat Wt (kg) (Mean \pm St. Dev.)	4.96 \pm 1.38	9.64 \pm 1.91	22.2 \pm 5.33
SA (m ²) (Mean \pm St. Dev.)	1.91 \pm 1.38	1.94 \pm .13	2.02 \pm .13
Age (yrs) (Mean \pm St. Dev.)	25.9 \pm 5.3	31.2 \pm 7.7	40.9 \pm 7.6
BMR (Cal/d) (Mean \pm St. Dev.)	1899 \pm 251	1845 \pm 211	1726 \pm 213
<u>Aub & DuBois (pred.)</u> BMR (measured)	.963 \pm .087	1.005 \pm .084	1.101 \pm .127
<u>Harris & Benedict</u> BMR (measured)	.951 \pm .081	.976 \pm .082	1.069 \pm .122
<u>BMR</u> Wt Cal/kg (Mean \pm St. Dev.)	26.05 \pm .344	24.12 \pm 2.76	20.72 \pm 2.56
<u>BMR</u> LBM Cal/kg (Mean \pm St. Dev.)	28.02 \pm 1.98	27.71 \pm 2.62	28.36 \pm 2.41

trend is particularly pronounced among males for whom LBM drops from 67.9 kg to 66.9 kg between LM and MM groups and falls of to 60.9 kg in the fattest group. Females showed only a modest 0.5 kg drop between the LF and MF groups but a more dramatic 1.4 kg decrease between the MF and FF groups. No two female groups were statistically different at the .05 level while the male trend reached significance only between the MM and MF groups.

The observed inverse relation between fat percentage and LBM seems at first to be inconsistent with the generally confirmed elevated LBM associated with obesity (Forbes & Welle, 1983; James et al., 1978, Ravussin et al., 1982; Sörbus et al., 1982). In our sample, however, age is directly related to fat percentage and since the groups have different mean ages, it may be that the expected drop in LBM with age obscures any potentially discernible increase in LBM with fat percentage. When mean ages of each group were plotted on curves for expected decline in LBM with age (see Appendix C; data from Burmeister & Bingert, 1967), it is apparent that the LBM differences between groups are just what would be expected from age differences alone. Specifically, both the very minor differences in female groups and the greater differences between male groups, particularly after age 40, match the age effect demonstrated in the Burmeister and Bingert study.

To focus on the relationship between LBM and fat weight more clearly, a partial correlation was calculated between these two variables with age held constant. The partial correlation was 0.38 for women and approximately zero for men. The female value fits expectations but the independence of LBM and fat weight may reflect a mild sampling bias in the male sample. Assuming LBM would normally have increased with fat weight in our sample as has been shown repeatedly, perhaps training-induced LBM elevation in the leanest group has obscured this effect. This explanation is consistent with the observation that many of the leaner subjects were highly trained, athletic individuals with moderately elevated LBM in addition to lean composition.

The progressive decline observed in LBM from lean to fat groups is matched by a parallel decline of BMR in both sexes. Group means for males were (LM) 1889 Cal/day, (MM) 1844 Cal/day, (FM) 1726 Cal/day. Mean BMR figures for the female groups were (LF) 1346 Cal/day, (MF) 1362 Cal/day, (FF) 1295 Cal/day. Not only do the rates decline in general following LBM means, the magnitudes of these BMR differences correspond with the size of the LBM changes between groups. Most striking are the BMR decrements after age 30, particularly among males.

Because body surface area is calculated partially from body weight, it increases as one passes from the leaner to

the fatter groups of both sexes. The increase in SA is associated with a decrease in BMR. Again, this would appear to oppose the theorized proportionality between BMR and SA were it not for the age differences between groups. Surface area formulas for BMR allow for decreasing levels of heat production per meter squared as age increases, a correction factor instituted 60 years ago to allow for the (unrecognized) age-related LBM drop.

To compare the utility of the currently-used prediction formulas between groups of different body composition, the ratios of predicted to measured BMR were calculated. Prediction standards used were Aub and DuBois (1917) and Harris and Benedict (1919). A ratio of 1.00 indicates exact agreement between actual and predicted values. If the ratio is less than 1.00, the formula has given an underestimate for BMR while greater than 1.00 represents an overestimate.

As indicated in Table 14, the predictive accuracy of the Harris and Benedict equation is excellent for both the lean and midrange groups for which the mean errors are -0.1% and 1.2%, respectively. When applied to the fattest third, however, the error is nearly 11% as LBM and SA have become dissociated. This 11% compares with the 15% deviation from the norm proposed by Benedict (1915) to diagnose abnormal BMR. Subtle variations within normal limits could therefore lead one to conclude that a large proportion of

obese females are abnormal by reference to the Harris and Benedict standards. The Aub and DuBois standards yield even higher estimates than those of Harris and Benedict. The lean group is overestimated by 4.1%, the middle group by 6.5%, and the fattest group by 16.2%. The magnitude of this overestimation by Aub and DuBois strongly suggests their sampling of a leaner population of females for whom LBM would have been greater for a given SA.

The male metabolic rates are estimated quite well overall by the Harris and Benedict tables. Lean and middle range groups were underestimated by 4.9% and 2.4% while the fat group was overestimated by 6.9%. Overall, the mean ratio of predicted to actual BMR was 0.999 suggesting that the body composition between the Harris and Benedict sample and ours was probably very similar. But despite this generally excellent agreement, the errors with these standards become apparent for certain groups. Aub and DuBois again produced acceptable estimates for lean and normal weight groups but gave a 10.1% overestimate for the fattest third.

The general tendency toward overestimation of BMR in fat subjects along with the converse is illustrated in Figure 3. This plots actual BMR vs predicted BMR by the Aub and DuBois formula. Solid points, which cluster below the prediction line, represent actual BMR values for fat subjects. Circles represent data from lean subjects and

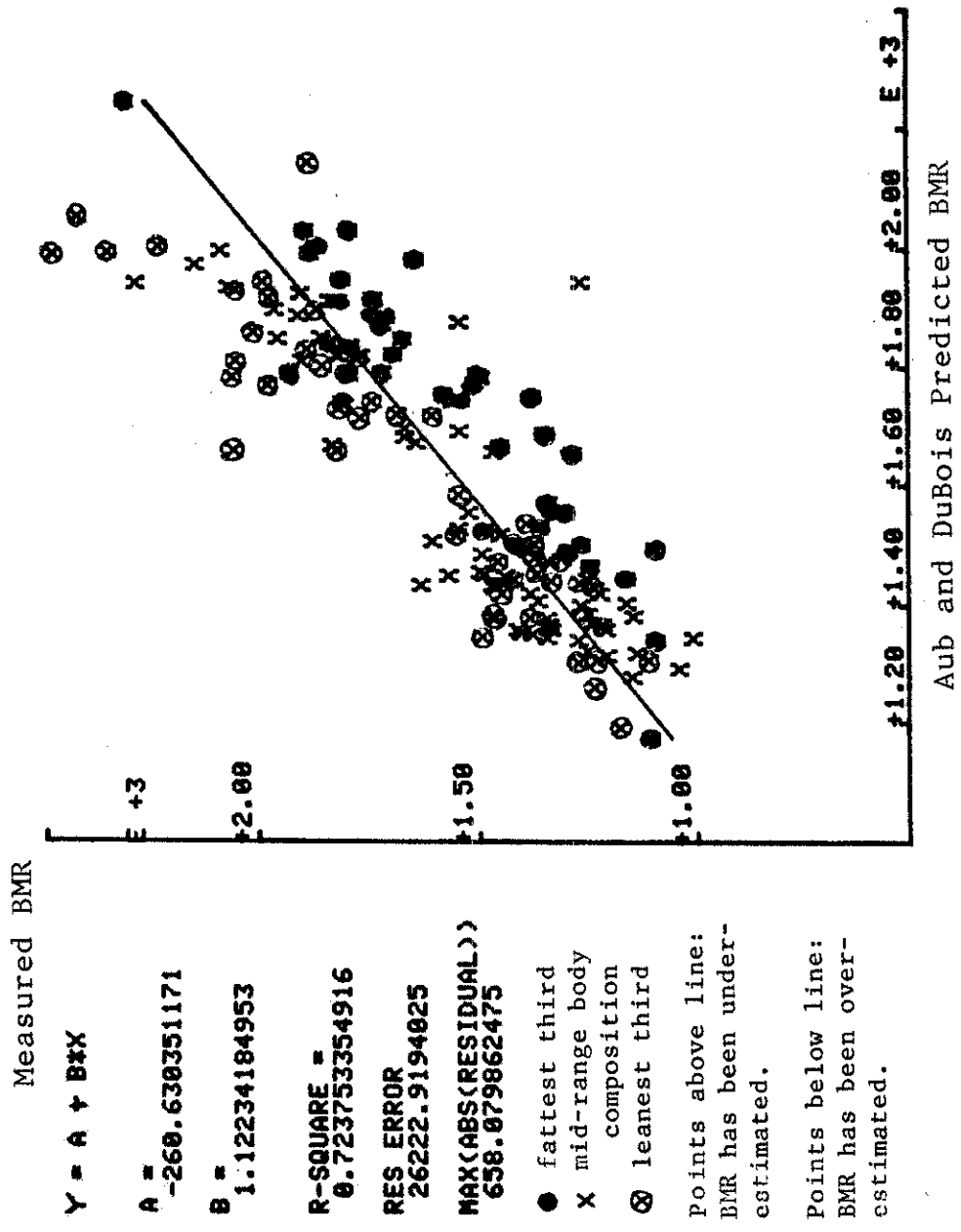


Fig. 3--Measured BMR vs predicted BMR: The effect of body composition. The clustering of fat subjects below the line illustrates the tendency to overestimate BMR of fat subjects using SA prediction.

Xs are used for subjects with average body composition. The circles tend to cluster above the regression line, indicating that BMR is underestimated in lean subjects.

The magnitude of these overestimates has practical significance. If one were to use the Aub and DuBois estimates for the average NTSU participant from the fattest groups, the excess predicted BMR would amount to approximately 200 Cal/day for either sex. In practical situations, this error is often magnified because caloric needs are calculated by multiplying some factor, say 1.25, by the estimated BMR. An error of this size would hinder weight loss (or cause a weight gain) of approximately 1 kg every 2 months. Further, estimates for patients that are hospitalized with weight problems are probably much greater overestimates than those for the fattest third in our study. Several of the subjects in our fatter groups did not even qualify as obese (Males: 20% fat; females: 30% fat).

Although not a major result, BMR/weight was calculated to demonstrate the effect on overall metabolic rate of body composition. Because LBM metabolizes several times more rapidly than fat tissue, one would anticipate that a body with less fat would have a higher BMR per unit mass. This trend is indicated by the progressive decline in BMR/weight from 25.49 Cal/day-kg in the lean female group to 19.73 Cal/day-kg among fat females. The same trend is observed

between the male groups but all the values are higher because males are leaner in a group-by-group comparison.

The differences between groups disappear and the comparison between males and females is reversed when correction is made for differences in body fat. This correction is made by focusing on BMR/LBM. The value of this variable is essentially the same in all groups among males with a mean of 28.02 Cal/kg. Female values were also similar in all groups with a mean value of 30.60 Cal/kg. The lowest value among female groups was nearly 2.0 Cal/kg greater than the greatest male group mean.

The fact that heat generation is not constant per kg of lean tissue raises at least two possibilities. First, because males are generally larger than females, the lowered BMR/LBM in males may reflect an intrinsic depression of heat generation associated with larger body size to aid the problem of heat buildup.

The correlations between BMR/LBM and LBM are moderate and negative in both sexes: $r = -0.5770$ for females, $r = -0.2424$ for males. Both fit this hypothesis; as LBM increases, metabolic rate per gram of lean tissue decreases. The correlations within each sex show that this size relationship holds not only between but within sexes.

Another interesting possible explanation concerns the varying metabolic rates of the different components of LBM. Data from several sources (Brooks & Fahey, 1984; Kleiber,

1975; McArdle, Katch, & Katch, 1981; Miller & Blythe, 1953; Stolwijk & Hardy, 1966; Tsankoff & Norris, 1977) indicate that resting muscle tissue metabolizes at a rate two and one-half to three times more slowly than LBM does as a whole per unit mass. This means that LBM with a high proportion of muscle tissue should show a lower BMR/LBM than a less muscular lean body.

A subgroup of nine male weight-trainers subjectively judged to have a high proportion of muscle averaged 27.1 Cal/kg. This figure was nearly 1.0 Cal/kg below the male group mean. Only three females with significant muscle mass were included in the female sample. They had values of 29.5, 29.0, and 28.8 Cal/kg of lean tissue, all of which were also well below the female mean of 31.0 Cal/kg. A quick survey of several conspicuously undermuscled subjects appeared to give the opposite result. Although a survey such as this would be better left to a more objective means of muscle mass assessment such as creatinine excretion, these results are consistent with the hypothesis that excess skeletal muscle elevates BMR less than would be predicted from LBM measurement alone.

Quantitatively, the difference between male and female heat generation per unit lean tissue can be viewed by reference to the body composition figures presented for "Reference Man" and "Reference Woman" (Behnke & Wilmore, 1974). The LBM of Reference Man consists of 52% muscle tissue

while the LBM of Reference Woman is said to include 47% muscle. If this extra 5% muscle metabolizes at one-third the rate of LBM as a whole, this would account for approximately one-half of the sex differences in BMR/LBM. Among other possibilities, the remaining difference could be caused by the size factor just discussed or perhaps by the increased metabolic contribution of fat although average total fat weight is probably no more in women than in men.

BMR/LBM and Aging

Tsankoff and Norris (1977) have attributed the entire decline in BMR with age to the loss of skeletal muscle. With the loss of slowly metabolizing muscle tissue, one would expect BMR/LBM to increase with age unless non-muscle LBM components showed reduced metabolic activity. In our study, we did not see this. Among women, the correlation between BMR/LBM and age was essentially zero while for men it was -0.1665. The male correlation suggests a very moderate decline in BMR per gram of lean tissue with age. Keys et al. (1973) also found a small decline from 3.90 ml O₂/kg LBM to 3.77 ml O₂/kg LBM over a 20 year period. Keys et al. attributed this drop to the increased proportion of metabolically inert connective tissue associated with skeletal muscle with advancing age. This may also explain our results: perhaps this increased connective tissue is the component of LBM that lowers BMR/LBM with age

despite the loss of muscle tissue. Apparently, while decreased muscle mass at a given age elevates BMR/LBM by increasing the proportion of LBM composed of metabolically active organs, across ages this effect is obscured. Because the lost muscle tissue is replaced by connective tissue of essentially zero metabolic activity, the average metabolic rate of the lean compartment may be depressed.

LBM Changes and Weight Control

Because the BMR decline with age is often singled out as the primary cause of creeping obesity with age, LBM maintenance is sometimes advocated as a means of weight control. Though this would certainly aid one's efforts to create an energy deficit, it is important that the magnitude of this contribution not be overestimated. The relatively low metabolic rate of resting skeletal muscle, along with the minor LBM changes that generally accompany exercise programs, combine to minimize BMR elevation. A quick calculation in Appendix E compares energy expended in a four-month weight training program including expected BMR elevation to the energy expended in a reasonable jogging program over the same period. Although the calculation is dependent upon the assumptions employed, it would be difficult to select reasonable assumptions that would suggest that the LBM factor is likely to tilt the energy balance in favor of weight training when the primary goal is weight control.

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CHAPTER V

SUMMARY AND CONCLUSIONS

The purpose of this study was to develop an equation (or set of equations if necessary) to estimate basal metabolic rate for adults of both sexes. Eighty-two females and 76 males between the ages of 18 and 59 volunteered and participated in the study.

All subjects were tested for BMR under standard basal conditions using a calibrated Beckman MMC. Height, weight, age, and percent fat by the skinfold technique were also recorded. Several secondary variables were also calculated with these primary data. They were LBM, fat weight, SA, Harris and Benedict (1919) predicted BMR, Aub and DuBois (1917) predicted BMR, BMR/LBM, BMR/weight, log (BMR), and log (LBM). Pearson product-moment correlations were computed between all variables and LBM was shown to be the highest correlate ($r = 0.9127$ overall; males $r = 0.7907$; females, $r = 0.7726$) of BMR. Surface area was the second best predictor ($r = 0.7517$), much of which was probably due to the often-demonstrated correlation between LBM and SA. Multivariate analysis, introducing such variables as age, sex, and fat weight in addition to LBM, failed to enhance the predictive value of the equation.

Logarithmic variable transformations were employed to test for a simple power law relationship between BMR and LBM. The power law obtained, $BMR = k (LBM)^{0.76}$ was similar to those found in previous studies. A polynomial regression, however, revealed that the data were essentially linear. The lower exponent determined for the power law equation was thought to be a result of forcing the prediction curve through the origin. It was suggested that this may also have been a misleading result of earlier studies.

Each sex was also divided into thirds based on body composition. Possible relationships between all variables in all groups were investigated. It was noted that SA prediction formulas currently in use tend to overestimate BMR by the greatest margin in the population for whom those estimates are most important: the obese. The magnitude of those overestimates for a group carrying approximately 10% more body fat than the mean was approximately 210 calories/day or nearly 1/2 lb. (0.25 kg) of fat storage per week.

The practicality of increasing skeletal muscle mass for weight control was also discussed. Because LBM changes with exercise are generally minor and the muscle tissue added respire at a very low rate at rest, it was concluded that LBM-induced BMR elevation is not enough to cause weight training to compete with aerobic exercise as a means of weight control.

Male BMR was predicted with success similar to that found in past studies. The linear equation below is presented for BMR estimation from LBM data. Variation in common is 62.5% and $r = .7907$. A table of values generated from the equation appears in Appendix F.

$$\text{BMR (Cal/day)} = 23.78 \text{ LBM (kg)} + 272.2$$

$$\text{SE} = 143 \text{ Cal/day}$$

$$r = .7907$$

$$r^2 = .6252$$

Female BMR may be predicted with similar accuracy from the linear equation below. Tabulated values appear in Appendix G.

$$\text{BMR (Cal/day)} = 18.43 \text{ LBM (kg)} + 531.6$$

$$\text{SE} = 89 \text{ Cal/day}$$

$$r = .7725$$

$$r^2 = .5968$$

Both LBM-based regression formulas predict BMR with greater success than the widely used tables of Harris and Benedict (1916) and Aub and DuBois (1919) when applied to our sample.

Conclusions

1. In normal populations, LBM is the best predictor of BMR. It typically accounts for 60 to 70% of the variance of basal energy expenditure. The great disparity between lean and fat tissue metabolic rates gives a sound physiological explanation for the tracking of BMR with LBM.

Surface area owes its utility as a reference standard for BMR to its fairly strong ($r = 0.8691$ overall in the present study) association with LBM. The partial correlation between BMR and SA adjusted for the influence of LBM was less than 0.40.

2. The surface area tables currently in use (Aub & DuBois, 1917; Harris & Benedict, 1919) tend to underestimate BMR slightly for lean subjects and significantly overestimate BMR for obese individuals. These errors are in the direction that would cause the greatest harm for atypical subjects. The magnitude of the overestimate on the fattest third of our female sample (mean fat percentage = 33.8%) was over 200 Cal/day. The overestimate also becomes greater with increasing fat percentage and may be of little value for grossly obese patients.

3. In obese populations, LBM is generally a good predictor of BMR but fat weight seems to gain predictive value as the population becomes fatter. The literature suggests that fat weight may actually become the best predictor of BMR in certain grossly obese populations. Its accuracy is still insufficient, however. BMR was predicted as accurately for females as it was for males in the present study. This has not generally been the case in past studies. Controlling for periodic hormonal fluctuations may have aided this effort.

4. Because skeletal muscle metabolizes more slowly than the remainder of LBM, increasing muscle mass in an attempt to significantly elevate BMR may approach futility. It is calculated in Appendix E that the expected LBM-induced BMR elevation over a four-month progressive resistance training program may be less than 10 Cal/day.

5. The data from the present study strongly suggest a linear relationship between BMR and LBM for both sexes. Simple power law models from previous work indicating exponents much less than one may be the result of an inappropriate curve-fitting technique for their data.

6. The age effect on the composition of LBM seems not to change the heat generation per kilogram of active tissue. This is consistent with the findings of Tsankoff and Norris (1977) and Keys et al. (1973) who suggested that the loss of skeletal muscle with age is accompanied by a gain of metabolically inert connective tissue. This apparently cancels out the expected gain in $\frac{\text{BMR}}{\text{LBM}}$ one would anticipate with the loss of slowly metabolizing muscle tissue. The result is that the equations generated here should be applicable to the adult population regardless of age.

7. It is suggested that these equations be used to estimate BMR on normal to moderately obese individuals. Estimates for clinically obese patients should be based on studies of more seriously obese populations.

Recommendations for Further Study

One could argue that refinements in the accuracy of BMR estimates are not nearly as important as

- 1) Refining estimates for the entire daily budget;
- 2) Finding ways of keeping individuals within these budget estimates.

For the general population, this statement is probably true. A major use of this information, however, is for individuals confined to institutional settings for whom input may be controlled. And while BMR is the easiest component of the energy budget to estimate, among sedentary individuals, it is also the largest. This proportion is typically two-thirds of the total output but can exceed 80% in obese, hospitalized, or otherwise inactive populations. Hospital nutritionists often estimate needs by multiplying the Harris and Benedict estimates by 1.25 which multiplies any error in the tabulated estimate (Mercer, 1984). In confined settings, therefore, a simple and inexpensive method of accurately estimating BMR is a valuable tool.

Practical Work

LBM appears to be the most useful anthropometric variable for predicting BMR but still accounts for only 60% of the variance in common for either sex. Further refinements may still be possible by anthropometric correlation but many factors such as hormonal fluctuation and

nutritional state have been shown to have significant effects on BMR. We may be approaching a limit for simple, anthropometric BMR estimation among normal populations. Refinements in accuracy may also be lost in the error of estimating expenditures beyond BMR in the daily budget.

1) Prediction among extreme subjects may be enhanced by further study. Further multiple regression studies on obese subjects may refine prediction in this important but somewhat unpredictable group. In particular, a combination of LBM and fat weight may prove useful.

2) A further study testing curvilinearity of the BMR-LBM relation may show the linear relationship seen in our study to be specific to our data. Animal studies seem to suggest that the fit should be curvilinear if size alone is the predominant factor in setting BMR/kg. Our linear relationship suggests this rate may be set for a given species (human) independent of size. This is inconsistent with the observation by Kleiber (1975, p. 200) that a 6.0 kg dog and a 5.3 kg rabbit had more similar BMR/kg than the large rabbit had with a 1 kg rabbit.

A more involved curve-fitting problem of the form $BMR = c_1 LBM^x + c_2$ may prove slightly superior on another data set.

Theoretical

1) Can BMR be quantified more accurately by determining the composition of the LBM itself? Different inherent metabolic rates for each tissue suggest this may be the

case. A simple starting point for this series of investigations might be the introduction of a third body component for which the resting metabolic rate is known: skeletal muscle mass. This can be quantified by creatinine excretion and, because it is a highly variable component of the LBM, should increase the ability to predict BMR. This is obviously not suggested as a practical means of BMR estimation.

2) Does the allometric scaling of these tissues account for the Kleiber relation or are the metabolic rates of the individual tissues centrally-tuned depending upon the size of the organism they reside in? Kleiber (1975) states that these rates seem to be independent of animal size but that more work needs to be done.

Over a wide range of body sizes, it would be interesting to determine the relative proportions of the main metabolic contributors (CNS, heart, liver, kidneys, skeletal muscle) and that of relatively inert tissues such as fat, bone, and connective tissue.

A separate study, focusing on the metabolic rates of these tissues in vivo would highlight any systematic variation related to body size from which these tissues originate.

Combining both studies would clarify the source of the observed decline in heat production per gram with increasing body size.

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APPENDICES

APPENDIX A

The Surface Law requires that BMR be proportional to (body weight)^{2/3} not (body weight)^{3/4} as is observed. The dependence on (body weight)^{2/3} can be derived as follows:

Consider a very simplified body shaped like a cube with side length ℓ . To remain at constant temperature, the heat generated inside the cube (BMR) exactly balances heat loss through the surface,

$$\begin{aligned} \text{BMR} &= \text{heat loss} \\ &= (\text{SA}) \frac{T_i - T_s}{r} \\ &= (6\ell^2) \frac{T_i - T_s}{r} \end{aligned} \quad (6)$$

By the Fourier Law where $T_i - T_s$ is the temperature difference across the surface and r an insulation constant as before.

because the surface area of a cube, $\text{SA} = 6\ell^2$. For any other body shape, the constant 6 would be different but the proportionality to ℓ^2 would remain.

The weight of this cube would simply be:

$$\begin{aligned} \text{volume} \times \text{density} &= \text{weight} \\ \ell^3 \times D &= \text{weight} \\ \ell &= \left(\frac{\text{weight}}{D}\right)^{1/3} \end{aligned}$$

which can be substituted back into equation (6) to give:

$$\begin{aligned} \text{BMR} &= 6 \left(\frac{\text{weight}}{D}\right)^{2/3} \frac{T_i - T_s}{r} \\ &= (\text{weight})^{2/3} \underbrace{6 \frac{T_i - T_s}{D^{2/3} r}}_{\text{(a constant)}} = k (\text{weight})^{2/3} \end{aligned} \quad (3)$$

APPENDIX B

LOGARITHMIC VARIABLE TRANSFORMATION

Part 1

If BMR and LBM were related by a simple power law, log transformations of both variables should produce a straight line on a log (BMR) vs log (LBM) plot. The slope of this line would be the exponent of the independent variable, LBM.

$$\text{Say } \text{BMR} = a (\text{LBM})^n$$

if $a = \text{constant}$, $n = \text{exponent}$

$$\begin{aligned} \text{then } \log (\text{BMR}) &= \log [a (\text{LBM})^n] \\ &= n \log (\text{LBM}) + a \end{aligned}$$

which is the familiar form of a line:

$$y = m x + b$$

if $y = \text{dependent variable (log BMR)}$

$m = \text{slope (n)}$

$x = \text{independent variable (log LBM)}$

$b = \text{dependent variable intercept}$

(theoretical BMR for LBM = 0 : a)

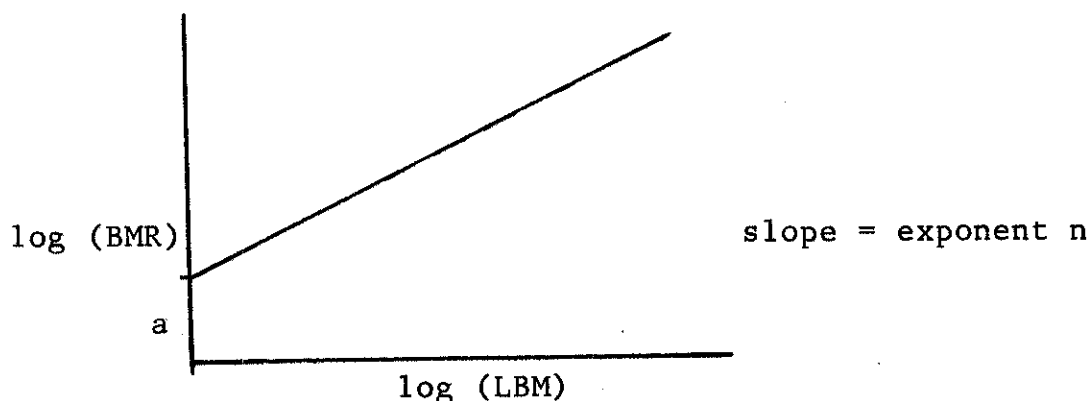


Fig. 4--Logarithmic variable transformation

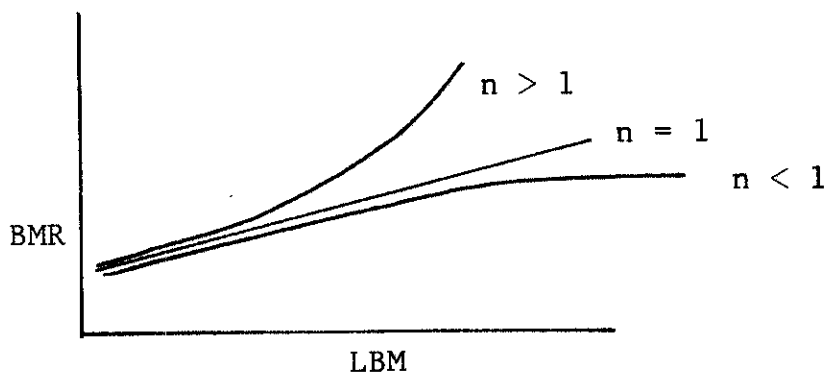


Fig. 5-- $BMR = a LBM^n$: Illustration of curvature for various exponents.

If the exponent $n = 1$;

$$BMR = a LBM^1 \text{ which is simply linear.}$$

All other exponents are curvilinear. $n > 1$ is concave upward while $n < 1$ is concave downward. The linear model is simply a special case of a more general curvilinear model when this exponent happens to equal one.

Part 2

The simple power law model does, however, not allow for a non-zero y-intercept. Therefore attempts to fit curves by this technique "force" the curve through the point (0,0) which may be deceptive. Consider the envelope (1) of data points, similar to those found in the present study, shown below. The linear regression gives a positive

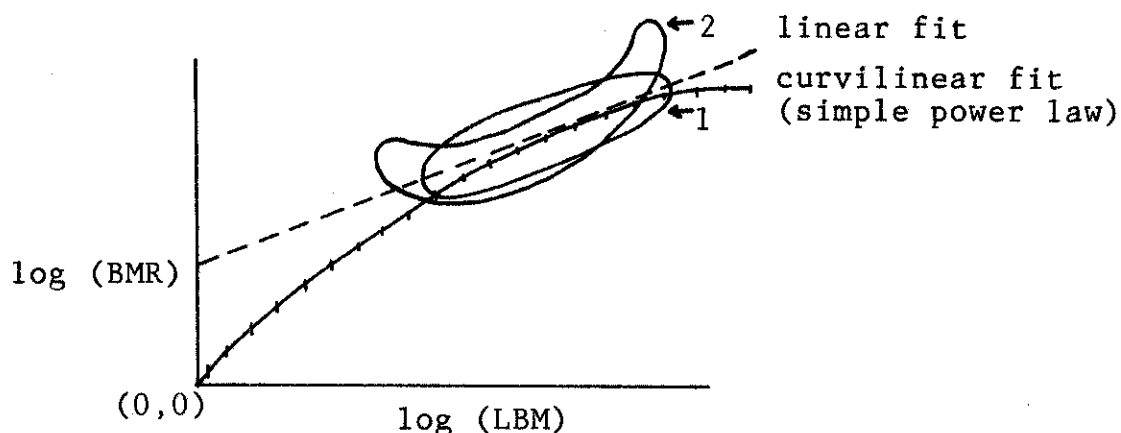


Fig. 6--Log (BMR) vs Log (LBM): Comparison of simple power law with linear regression.

intercept (BMR). Forcing the curve through zero, however, produces a negative curvature (exponent less than one) even though the envelope itself does not show this. One could even conclude that the data showed a negative curvature when in fact it was positive (envelope 2).

Despite this pitfall, this technique was employed because it is a standard method of analyzing data of this type and may explain the discrepancy between results obtained here and elsewhere (Galvão, 1948; Miller & Blythe, 1953).

APPENDIX C

CHANGE IN LBM WITH AGE IN BOTH SEXES

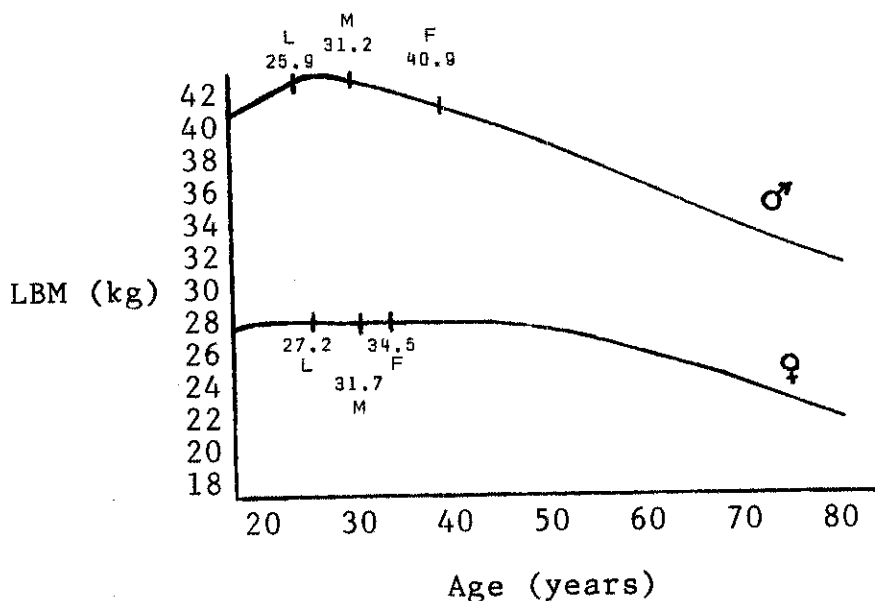


Fig. 7--Change in LBM with age in both sexes: Mean age of groups can be used to explain observed LBM differences (data from Burmeister & Bingert, 1967).

These curves above are used to determine expected LBM for groups with a given mean age. In the present study, the lean, middle, and fat groups of both sexes all had different mean ages. Therefore, LBM would be expected to vary between groups. The mean ages for the lean (L), middle (M), and fat (F) groups are shown on the upper curve for males and the lower curve for females. The mean age then indicates an expected LBM level. The LBM decline illustrated here explains the LBM differences noted in the present study between groups of different body composition that also happened to have differing age means.

APPENDIX D

Miller and Blythe (1953) presented a nonlinear relationship between BMR and LBM based on a population of lean young men. Graphically, their relation looked something like the figure below.

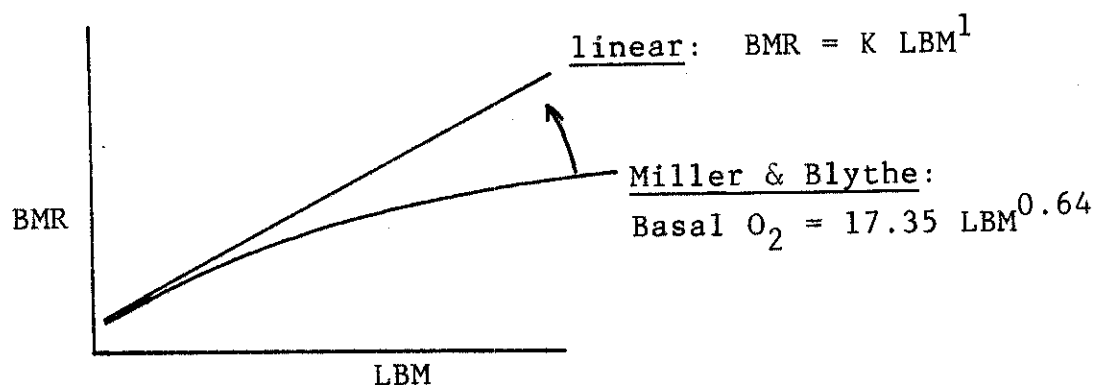


Fig. 8--BMR vs LBM: Possible metabolic contribution of fat at high LBM "linearizing" the curve when obese subjects are included.

The metabolic contribution of fat at higher LBMs (because LBM and fat weight tend to track together) could elevate the curve and increase the linear tendency of the curve. It would also explain why a curvilinear model is appropriate for a lean male population but becomes less so with increasing body fat.

Elevating the curve would require a BMR vs fat weight partial correlation with LBM held constant to be positive. For males this relationship is essentially zero and for women $r = -0.216$, effectively lowering the curve.

APPENDIX E

Contributions to the energy budget made by two exercise programs requiring equal 120 minutes per week commitments. The subject is assumed to weigh 70 kg.

Estimates of Caloric expenditure are made for:

- 1) energy consumed by the activity itself
- 2) energy consumed by LBM-induced BMR elevation
- 3) energy consumed by post-exercise resting metabolic rate are neglected because they are assumed to be similar for both activities. If there is a difference, it would almost certainly favor the jogging program and therefore not affect the basic conclusion arrived at by this calculation:

Program #1 Weight training: 9 stations x 3 sets

3 workouts/week, 40 minutes/workout,
4 months duration

Induced LBM change: 2% of LBM.

(Gettman, 1981)

Program #2 Jogging for 40 minutes, 3 runs/week,
4 months duration

Expenditure

#1: (40 minutes/workout)(3 workouts/week)(18 weeks)
(5.0 Cal/min)

= 10,800 Calories

(Wilmore, 1977)

LBM increase: Although a 2% LBM increase might be achieved at the conclusion of the training program, the average LBM over the four-month period is the important factor in the calculation. For a linear increase, the average would be one-half the final figure but LBM has been shown to increase almost logarithmically. A figure of 0.7 x final increase would be reasonable.

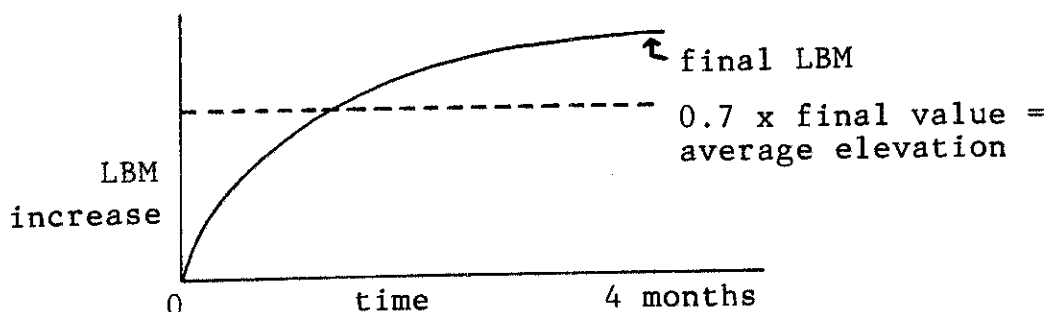


Fig. 9--Curve factor: Average LBM over training period

Therefore

$$(2\% \text{ LBM increase})(0.7 \text{ curve factor})(1800 \text{ Cal/day}) \\ (1/3 \text{ muscle met. rate/LBM met. rate})$$

$$= (8 \text{ Cal/day})(120 \text{ days}) = 960 \text{ Calories}$$

Total expenditure attributable to weight program:

$$10,800 \text{ Cal} + 960 \text{ Cal} = 11,760 \text{ Calories}$$

Program #2 9 minute jogging pace expends 13.7 Cal/min for
a 70 kg man of average running efficiency
(McArdle, Katch, & Katch, 1981)

$$(40 \text{ min/bout})(13.7 \text{ Cal/min})(3 \text{ bouts/week}) \\ (18 \text{ weeks})$$

$$= 29,000 \text{ Calories}$$

Equal time commitments strongly favor the aerobic program for weight control purposes despite the inclusion of LBM-induced BMR elevation. The recently recognized contribution of LBM changes to energy expenditure associated with progressive resistance training accounts for very roughly 10% of the energy burned in such a program. The contribution would probably be less for more advanced lifters for whom LBM has leveled off.

Not all factors are considered here. The cost of moving this increased LBM throughout the day might also be taken into account but might be partially offset by an increased post-exercise elevation associated with jogging. In any case, it would be difficult to select a set of reasonable assumptions which would lead one to conclude that weight training could rival jogging as a weight control program. The point is very simply that LBM-induced BMR changes are not significant enough to use as a selling point for a progressive resistance training program. It would be unfortunate to see this BMR-LBM association abused or misunderstood simply because the effect had not been addressed quantitatively.

APPENDIX F

ESTIMATED BMR IN Cal/day FOR MALES
BETWEEN AGES 18 AND 50

$$\text{BMR (Cal/day)} = 23.78 \text{ LBM (kg)} + 272.7$$

$$\text{SE} = 143.3 \text{ Cal/day}$$

$$R^2 = 0.625 \text{ S1}$$

<u>LBM</u>		<u>BMR</u>	<u>LBM</u>		<u>BMR</u>
<u>lb</u>	<u>kg</u>	<u>Cal/day</u>	<u>lb</u>	<u>kg</u>	<u>Cal/day</u>
105	27.7	1407	160	72.7	2002
110	50.0	1462	165	75.0	2057
115	52.3	1516	170	77.3	2111
120	54.5	1570	175	79.5	2165
125	56.8	1624	180	81.8	2219
130	59.1	1678	185	84.1	2273
135	61.4	1733	190	86.4	2327
140	63.6	1787	195	88.6	2381
145	65.9	1841	200	91.0	2435
150	68.2	1894	205	93.2	2489
155	70.4	1947	210	95.4	2543

APPENDIX G

ESTIMATED BMR IN Cal/day FOR FEMALES
BETWEEN AGES 18 AND 50

$$\text{BMR (Cal/day)} = 18.43 \text{ LBM (kg)} + 531.6$$

$$\text{SE} = 110 \text{ Cal/day}$$

$$r = .7725$$

$$r^2 = .5968$$

<u>LBM</u>		<u>BMR</u>	<u>LBM</u>		<u>BMR</u>
<u>lb</u>	<u>kg</u>	<u>Cal/day</u>	<u>lb</u>	<u>kg</u>	<u>Cal/day</u>
70	31.8	1118	115	52.3	1495
75	34.1	1160	120	54.5	1537
80	36.4	1201	125	56.8	1579
85	38.6	1242	130	59.1	1621
90	40.9	1286	135	61.4	1663
95	43.2	1328	140	63.6	1705
100	45.4	1370	145	65.9	1747
105	47.7	1412	150	68.2	1789
110	50.0	1453	155	70.5	1831

RAW DATA

MALES (n=76)

% Fat	LBM (kg)	BMR (cal/d)	SA (m ²)	HB (cal/d)	ADB (cal/d)	Age*	ADB BMR	HB BMR	Wt (kg)	BMR LBM
11.3	79.4	2345	2.10	2047	1991	1	.85	.87	89.50	29.53
15.7	70.3	1730	2.00	1938	1896	1	1.10	1.12	83.40	24.61
10.5	65.9	2046	1.96	1877	1928	1	.94	.92	73.60	31.05
11.0	69.6	1881	1.93	1861	1829	1	.97	.99	78.20	27.03
8.0	65.6	1787	1.89	1785	1787	1	1.00	1.00	71.00	27.24
9.1	48.0	1330	1.67	1558	1558	1	1.17	1.17	54.10	27.71
6.4	59.5	2051	1.75	1646	1659	1	.81	.80	63.60	34.47
9.5	65.0	1923	1.89	1750	1789	1	.93	.91	71.80	29.58
4.1	66.0	1800	1.84	1711	1739	1	.97	.95	68.80	27.27
6.4	73.0	1901	1.99	1844	1886	1	.99	.97	78.00	26.04
3.9	82.5	2473	2.10	1983	1986	1	.80	.80	85.70	29.98
7.5	78.1	2137	2.08	1980	1971	1	.92	.93	84.10	27.36
7.6	81.8	2277	2.04	2003	1939	1	.85	.88	88.60	27.84
9.6	65.7	1760	1.92	1776	1820	1	1.03	1.01	72.70	26.79
9.8	74.2	2060	2.04	1755	1933	1	.94	.85	82.30	27.76
6.3	83.7	2413	2.16	2144	2050	1	.85	.89	89.30	28.83
7.8	61.6	1760	1.81	1678	1715	1	.97	.95	66.80	28.57
14.4	70.1	1896	2.03	1963	1924	1	1.01	1.04	81.90	27.05
11.2	64.9	1489	1.88	1723	1782	2	1.20	1.16	73.20	22.94
16.2	72.8	1981	1.90	1903	1943	2	.98	.96	87.00	27.21
30.0	64.4	1637	2.09	1952	1981	2	1.21	1.19	92.00	25.42
11.2	70.0	1806	2.05	1866	1948	2	1.08	1.03	78.80	25.80
10.6	77.8	1886	2.16	1962	2029	2	1.08	1.04	87.00	24.24
9.4	63.6	1848	1.90	1743	1801	2	.97	.94	70.20	29.06
27.2	59.2	1860	2.00	1829	1896	2	1.02	.98	77.00	31.42
4.3	70.6	1816	1.92	1734	1820	2	1.00	.95	73.90	25.72
15.9	57.5	1811	1.83	1623	1730	2	.96	.90	68.20	31.50
35.0	55.4	1262	2.05	1894	1943	2	1.54	1.50	85.20	22.78

RAW DATA--Continued

MALES (n=76)

% Fat	LBM (kg)	BMR (cal/d)	SA (m ²)	HB (cal/d)	ADB (cal/d)	Age*	ADB BMR	HB BMR	Wt (kg)	BMR LBM
21.1	59.4	1813	2.01	1821	1910	2	1.05	1.00	75.20	30.52
25.4	59.2	1811	1.92	1851	1820	2	1.00	1.02	79.50	30.59
17.1	54.1	1661	1.78	1610	1682	2	1.01	.97	65.30	30.70
15.8	59.3	1553	1.84	1684	1742	2	1.12	1.08	70.50	26.19
15.8	62.2	1948	1.95	1742	1848	2	.95	.89	73.90	31.32
35.0	63.0	1875	2.10	2002	1991	2	1.06	1.07	94.30	29.76
20.4	60.1	1894	1.91	1744	1810	2	.96	.92	75.50	31.51
13.4	74.8	1860	2.11	1960	2000	2	1.08	1.05	86.40	24.87
2.5	71.2	2004	1.96	1783	1858	2	.93	.89	72.70	28.15
10.1	65.4	1791	1.87	1698	1791	2	1.00	.95	72.70	27.39
5.1	60.4	1814	1.75	1570	1659	2	.91	.87	63.60	30.03
27.1	70.8	1879	2.26	2121	2142	2	1.14	1.13	97.00	26.54
21.4	61.3	1833	1.92	1759	1839	2	1.00	.96	78.00	29.90
22.9	53.5	1535	1.84	1643	1744	2	1.14	1.07	69.50	28.69
12.5	64.0	1665	1.95	1751	1848	2	1.11	1.05	73.20	26.02
21.9	60.9	1712	1.98	1796	1872	2	1.09	1.05	77.90	28.11
10.9	76.9	2223	2.11	1935	2000	2	.90	.87	86.30	28.91
35.0	74.8	2302	2.36	2333	2242	2	.97	1.01	115.00	30.78
10.6	61.0	1635	1.81	1595	1672	3	1.02	.98	68.10	28.80
18.2	58.2	1600	1.86	1633	1718	3	1.07	1.02	70.90	27.49
8.6	63.2	1575	1.90	1639	1755	3	1.11	1.04	69.10	24.92
18.3	59.1	1736	1.89	1633	1741	3	1.00	.94	72.30	29.37
14.5	77.3	2079	2.16	1945	1995	3	.96	.94	90.40	26.90
28.9	59.0	1703	2.04	1798	1844	3	1.11	1.06	83.00	28.86
18.1	63.0	1497	1.91	1723	1787	3	1.19	1.15	76.80	23.76
30.1	58.7	1539	2.03	1853	1875	3	1.22	1.20	84.10	26.22
18.1	64.5	1970	1.94	1685	1769	3	.90	.86	78.60	30.54
34.5	61.5	1733	2.07	1892	1913	3	1.10	1.09	93.90	28.18
34.8	59.1	1819	2.06	1916	1909	3	1.05	1.05	90.70	30.78

RAW DATA--Continued

MALES (n=76)

% Fat	LBM (kg)	BMR (cal/d)	SA (m ²)	HB (cal/d)	ADB (cal/d)	Age*	ADB BMR	HB BMR	Wt (kg)	BMR LBM
39.0	50.5	1505	1.97	1700	1773	4	1.14	1.10	97.90	26.02
20.7	67.9	1829	2.12	1857	1908	4	1.04	1.02	85.50	26.94
10.0	62.5	1537	1.88	1565	1692	4	1.10	1.02	69.50	24.59
29.9	68.6	1785	2.26	1964	2029	4	1.14	1.10	97.90	26.02
27.5	52.7	1345	1.88	1508	1687	4	1.25	1.12	72.70	25.52
9.5	71.2	1955	2.00	1913	1896	1	.97	.98	79.00	27.46
7.3	70.6	1969	2.02	1919	1915	1	.97	.97	78.20	27.89
5.2	59.8	1678	1.81	1691	1720	1	1.03	1.01	63.10	28.06
7.2	67.5	2053	1.88	1760	1782	1	.87	.86	72.70	30.41
7.3	72.5	1969	2.02	1919	1915	1	.97	.97	78.20	27.16
4.4	62.3	1660	1.79	1692	1697	1	1.02	1.02	65.20	26.65
6.0	71.0	1784	1.96	1842	1835	1	1.03	1.03	75.50	25.13
14.9	66.7	1850	1.95	1867	1849	1	1.00	1.01	78.20	27.74
8.0	64.5	1711	1.89	1777	1792	1	1.05	1.04	70.20	26.53
13.3	47.3	1598	1.59	1481	1507	1	.94	.93	54.50	33.78
9.8	68.1	2045	1.91	1810	1810	1	.89	.89	75.40	30.03
26.4	57.3	1688	1.97	1817	1822	2	1.08	1.08	77.90	29.46
7.4	63.3	1797	1.71	1716	1735	1	.97	.95	68.40	28.39
10.3	61.6	1825	1.76	1591	1668	2	.91	.87	68.60	29.63

*Age: 1 = 18-29 3 = 40-49
2 = 30-39 4 = 50-59

RAW DATA
FEMALES (n=82)

% Fat	LBM (kg)	BMR (cal/d)	SA (m ²)	HB (cal/d)	ADB (cal/d)	Age*	ADB BMR	HB BMR	WT (kg)	BMR LBM
33.8	42.5	1447	1.71	1457	1518	1	1.05	1.01	63.40	34.05
6.1	45.6	1330	1.53	1332	1358	1	1.02	1.00	48.60	29.17
17.9	48.4	1464	1.62	1414	1434	1	.97	.97	59.00	30.25
19.4	48.8	1245	1.65	1409	1465	1	1.18	1.13	60.50	25.51
32.0	39.5	1365	1.64	1400	1451	1	1.06	1.03	58.10	34.56
19.4	45.8	1488	1.66	1418	1485	1	1.00	.95	56.80	32.49
16.7	49.6	1410	1.69	1410	1500	1	1.06	1.00	59.70	28.43
26.1	53.1	1540	1.72	1454	1527	1	.99	.94	63.20	29.00
18.4	36.0	1233	1.42	1248	1260	1	1.02	1.01	44.20	34.25
22.5	48.3	1460	1.73	1481	1656	1	1.13	1.01	62.30	30.23
16.1	40.3	1039	1.46	1300	1292	1	1.24	1.25	47.50	25.78
13.3	51.5	1386	1.69	1430	1490	1	1.08	1.03	59.40	26.91
22.8	40.8	1386	1.56	1430	1496	1	1.08	1.03	52.80	33.97
19.0	39.2	1296	1.54	1334	1489	1	1.15	1.03	48.50	33.06
36.0	48.3	1375	1.97	1603	1749	1	1.27	1.17	75.50	28.47
19.0	46.0	1293	1.68	1394	1491	1	1.15	1.08	56.80	28.11
25.7	43.1	1459	1.61	1400	1430	1	.98	.96	57.90	33.85
12.0	51.8	1545	1.71	1415	1518	1	.98	.92	58.90	29.83
18.9	41.0	1340	1.55	1333	1376	1	1.03	.99	50.60	32.68
20.0	44.7	1435	1.63	1394	1447	1	1.01	.97	55.90	32.10
32.0	50.7	1446	1.87	1554	1660	1	1.15	1.07	74.50	28.52
22.6	41.2	1265	1.58	1353	1398	1	1.11	1.07	53.20	30.70
11.3	42.0	1488	1.51	1315	1345	1	.90	.88	47.30	35.43
25.3	35.8	1229	1.47	1270	1305	1	1.06	1.03	47.90	34.33
12.6	45.3	1460	1.52	1347	1381	1	.95	.92	52.10	32.23
18.9	49.2	1388	1.66	1422	1474	1	1.10	1.06	60.80	27.20
15.8	43.7	1268	1.51	1316	1340	1	1.06	1.04	51.90	29.02
20.9	48.1	1517	1.75	1433	1554	1	1.02	.94	60.70	31.54
27.0	49.0	1338	1.80	1454	1572	2	1.17	1.09	67.10	27.31

RAW DATA--Continued

FEMALES (n=82)

% Fat	LBM (kg)	BMR (cal/d)	SA (m ²)	HB (cal/d)	ADB (cal/d)	Age*	ADB BMR	HB BMR	Wt (kg)	BMR LBM
25.2	43.3	1368	1.69	1372	1480	2	1.08	1.00	57.90	31.59
23.2	44.2	1444	1.62	1337	1419	2	.98	.93	57.40	32.67
25.8	40.4	1158	1.60	1316	1401	2	1.21	1.14	54.50	28.66
28.8	37.6	1215	1.56	1314	1366	2	1.12	1.08	52.80	32.31
28.2	37.7	1208	1.55	1296	1357	2	1.14	1.07	52.50	32.04
16.6	43.3	1461	1.57	1303	1375	2	.94	.89	51.90	33.74
14.3	44.0	1268	1.49	1288	1302	2	1.03	1.02	51.40	28.82
22.2	40.5	1340	1.54	1288	1344	2	1.00	.96	51.90	33.09
15.0	35.4	1174	1.36	1177	1191	2	1.01	1.00	41.70	33.16
27.0	40.0	1248	1.58	1336	1384	2	1.11	1.07	54.80	31.20
28.4	42.8	1250	1.51	1270	1322	2	1.06	1.02	52.40	29.21
25.2	40.3	1143	1.58	1314	1380	2	1.21	1.15	53.90	28.36
26.6	43.9	1564	1.66	1375	1449	2	.93	.88	59.80	35.63
29.6	41.9	1451	1.68	1382	1472	2	1.01	.95	59.50	34.63
37.5	48.9	1281	1.89	1539	1655	2	1.29	1.20	77.70	26.20
25.8	42.8	1341	1.57	1358	1375	2	1.03	1.01	57.70	31.33
22.1	40.8	1337	1.56	1292	1366	2	1.02	.97	52.30	32.77
31.2	41.6	1161	1.65	1366	1445	2	1.24	1.18	60.50	27.91
16.6	43.7	1408	1.55	1296	1358	2	.96	.92	52.30	32.22
32.7	41.3	1334	1.68	1350	1471	3	1.10	1.01	61.40	32.30
33.4	40.7	1232	1.66	1351	1430	3	1.16	1.10	61.10	30.27
29.0	40.6	1379	1.60	1306	1378	3	1.00	.95	57.20	33.97
29.1	35.1	1147	1.48	1218	1278	3	1.11	1.06	49.50	32.68
17.6	44.5	1398	1.57	1284	1352	3	.97	.92	54.00	31.42
29.2	36.5	1092	1.56	1253	1340	3	1.23	1.15	52.10	29.92
24.7	43.6	1437	1.65	1329	1435	3	1.00	.92	57.70	32.96
28.3	52.6	1370	1.74	1355	1503	3	1.10	.99	64.50	26.05
40.0	41.4	1300	1.80	1430	1555	3	1.20	1.10	69.00	31.40
36.8	40.6	1308	1.71	1376	1477	3	1.13	1.05	64.00	32.22

RAW DATA--Continued

FEMALES (n=82)

% Fat	LBM (kg)	BMR (cal/d)	SA (m ²)	HB (cal/d)	ADB (cal/d)	Age*	ADB BMR	HB BMR	Wt (kg)	BMR LBM
27.3	42.0	1006	1.60	1350	1344	4	1.34	1.34	57.90	28.95
**53.0	63.9	1180	2.41	2185	2198	1	1.86	1.85	136.00	18.47
19.3	44.2	1386	1.60	1398	1536	1	1.11	1.01	54.80	31.36
11.0	48.6	1483	1.63	1386	1451	1	.98	.93	54.50	30.51
15.3	46.3	1415	1.62	1374	1442	1	1.02	.97	54.50	30.56
28.5	38.1	1238	1.54	1367	1367	1	1.10	1.10	53.20	32.49
13.7	46.2	1361	1.58	1363	1407	1	1.03	1.00	53.60	29.46
30.5	48.3	1357	1.75	1459	1533	2	1.13	1.08	69.50	28.10
10.3	44.9	1375	1.54	1295	1349	2	.98	.94	50.00	30.62
19.6	54.9	1540	1.81	1463	1585	2	1.03	.95	68.30	28.05
14.8	46.7	1379	1.62	1330	1419	2	1.03	.96	54.30	29.53
35.0	44.3	1482	1.73	1465	1525	2	1.03	.99	68.20	33.45
33.5	41.4	1267	1.64	1368	1436	2	1.13	1.08	61.90	30.60
19.0	41.8	1136	1.51	1281	1318	2	1.16	1.13	51.50	27.18
17.2	46.4	1220	1.62	1332	1419	2	1.16	1.09	56.10	26.29
27.1	53.1	1331	1.64	1418	1440	2	1.08	1.07	64.10	25.07
24.0	45.2	1493	1.68	1336	1455	3	.97	.89	59.50	33.03
30.2	42.3	1250	1.67	1346	1443	3	1.15	1.08	60.50	29.55
21.2	44.8	1627	1.66	1320	1434	3	.88	.81	56.70	36.32
37.0	31.5	1105	1.36	1228	1175	3	1.06	1.11	50.00	35.08
34.7	41.3	1095	1.73	1335	1494	3	1.36	1.22	63.60	26.51
19.8	40.0	1206	1.50	1259	1314	2	1.09	1.04	50.00	30.15
31.1	34.2	1108	1.47	1316	1306	1	1.18	1.19	51.10	32.40
34.9	40.8	1263	1.69	1442	1500	1	1.19	1.14	62.70	30.96
49.0	54.9	1769	2.07	1830	1814	2	1.03	1.03	107.70	32.22

*Age: 1 = 18-29 3 = 40-49
2 = 30-39 4 = 50-59

**Subject omitted for abnormally low BMR.

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