Basal Metabolism of Obese Adolescents: Evidence for Energy Conservation Compared to Normal and Lean Adolescents

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To test if obese adolescents systematically conserve energy, com-ABSTRACT parisons of basal metabolic rate (BMR) of obese, normal, and lean male and female adolescents were made. Obese had elevated values by as much as 23% ($P \le 0.05$) expressed as $kJ \cdot 24 hr^{-1}$ compared to the normal and lean. When indexed to body mass $(kJ \cdot kg \cdot BM^{-1} \cdot hr^{-1})$, the BMR for the obese was depressed by as much as -53% ($P \le 0.01$), and when indexed to fat free mass (kJ·kg-FFM⁻¹·hr⁻¹) it was depressed by -33% compared to normal and lean adolescents. A "theoretical metabolic rate" (TMR), based on the observed fat free mass, fat mass, and their thermal equivalents, was proposed as a theoretical way to properly index basal metabolism, referenced to body composition. Comparisons of the TMR between the obese, normal, and lean revealed that the obese values were depressed by an average -22% ($P \le 0.05$). In comparison, differences in TMR between the normal and lean males and females were no larger than 8% (ns). It was concluded that since both the observed BMR (expressed relative to body composition), and the derived TMR values were depressed for the obese compared to the normal and lean adolescent, the data suggest an energy saving hypothesis for obese adolescents.

While data on basal metabolic rate (BMR) of adolescents are available (Beirring, 1931; Boothby et al., 1936; Harris and Benedict, 1919), there are only a few studies on the obese (Katch et al., 1985, 1988a,b) and lean (Blunt et al., 1921), and no data that directly compare BMRs of adolescents who differ in body size and composition. The existence of a depressed metabolism would help explain increased rates of body mass gain and continued difficulties in achieving ideal body mass for the obese. We have recently suggested for obese adolescents (Katch et al., 1985, 1988a,b), as others have for obese adults (Segal and Gutin, 1983), an energy conservation hypothesis that results in longterm energy conservation.

In the present paper, we compare the BMR of obese adolescents with normal and lean adolescents within the same age range, but who differ with respect to stature and body composition. The present data lend support to a hypothesis of long-term energy conservation for obese adolescents.

METHODS AND PROCEDURES

Data for obese adolescents were collected as part of an obesity intervention study (Katch et al., 1985, 1988a,b; Rocchini and Katch, 1986; Becque et al., 1986, 1988; Hattori et al., 1987; Rocchini et al., 1987, 1988). Subjects were recruited via advertisements in local papers and local physician referral. Normal and lean adolescent data were taken from published reports in the literature, and from subjects tested in our laboratory who differed by no more than ± 2.5 years in age from the obese.

Obese subjects

Of the 67 obese adolescents only 3 (2 girls and 1 boy) were non-white. All of the obese adolescents had a history of being obese (parental reports). The mean birth mass and body length, determined from hospital records, was 4.67 kg (SD = 3.9 kg) and mean

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body length 0.584 m (SD = 0.104 m). At the time of testing the mean percent overweight was 179% of ideal mass for age and stature (Landis, 1988). None of the subjects were under medical care or taking medication. Following written and verbal descriptions of all procedures, parents and children signed informed consent statements.

BMR measurements were made in the Clinical Research Unit of the University Hospital using open circuit spirometry (Katch et al., 1985, 1988b). Following hospital admission, baseline physical examination and an overnight fast, subjects were quietly awakened at 7 A.M. and fitted with a respiratory face mask. A 5 min adjustment period of quiet rest was followed by a 10 min sampling of expired air that was immediately analyzed for volume with a balanced 0.120 m^3 gasometer, percent oxygen with a polargraphic analyzer, and percent carbon dioxide with an infrared detector. The accuracy of measuring minute oxygen uptake $(\dot{VO}_2 \text{ was } \pm 2.0\% \text{ (Katch et al., 1985)})$. The $\dot{VO}_2 \text{ in } \text{m}^3 \cdot \text{min}^{-1} \text{ was converted to kilojoules per min} (kJ \cdot \text{min}^{-1}) \text{ using the calorific}$ equivalent for oxygen (Carpenter, 1939) determined from the whole body respiratory exchange ratio (VCO₂/VO₂).

Body mass (BM) was measured to the nearest 0.01 kg with a beam balance scale, stature (HT) to the nearest 0.0001 m with a stadiometer, and total body volume to the nearest 0.0001 m³ using hydrostatic weighing (Katch et al., 1967). A minimum of 8 to 10 underwater weighing trials were performed and an average of the last 2 to 3 used to represent true underwater weight (Katch, 1969). Residual lung volume (RLV) was determined in duplicate just prior to underwater weighing in the same bent forward seated position, by use of an oxygen dilution procedure (Wilmore, 1969). The average RLV was used in the calculations of total body volume (TBV). Body density (D_b) was converted to percent body fat (%BF) with the Siri equation (1956), and fat free mass (FFM) was calculated by subtraction, FFM = BM - FM, where FM is fat mass (BM * %BF). BSA was calculated using the formula of Dubois and Dubois (1916).

Normal subjects

BMR (closed circuit method), BM, stature, and BSA data for 65 male adolescents (age range 10–15 yr) reported by Bierring (1931), and 39 female adolescents (age range 10.1–14.3 yr) reported by MacLeod (1924) were used. These studies are unique since complete individual values as well as estimates of measurement error were reported. In all instances reliability exceeded r = 0.92. Body composition estimates were made using the Mellits and Cheek (1968) and Moore et al. (1963) technique that involves the prediction of total body water (TBW) using gender, age, and stature specific equations. FFM was calculated from TBW, and % fat determined by subtraction. The validity of these procedures has been previously presented (Sheng and Huggins, 1979; Lukaski and Johnson, 1985).

Additional data on 9 male and 11 female subjects similar in age to the obese, and in body composition to the Beirring and Mac-Leod subjects were collected in our laboratory and included in the data set. The procedures for BMR and body composition data acquisition were the same as for the obese, with the exception that 6 of the subjects (3 male and 3 female) reported to the laboratory at 7 A.M. following an overnight fast. They rested in a bed for 2 hours prior to data collection. With the addition of these subjects the total N for normal males was 74, and 50 for normal females.

Lean subjects

BMR (closed circuit method), BM, stature, and BSA for 14 male and 12 female lean adolescents (age range 7.6-12.0 yr) were taken from the study by Blunt et al. (1921). The classification of underweight was based on mass for age and stature (Landis, 1988), and body composition was estimated as described above. Validity of the BMR exceeded r=0.90 (Blunt et al., 1921). In addition, data on 6 lean males and 6 lean females of the same age as the obese, and with a similar body composition as the Blunt subjects were included in the data set. We used the same data collection methods and procedures as with the obese subjects. Five of the lean subjects (3 males and 2 females) did not spend the night in the Clinical Research Unit, but reported to the lab at 7 A.M. and rested for 2 hours prior to data collection. With the addition of these subjects the total N for the lean group was 20 males and 18 females.

Statistical analyses

Comparisons between samples were made using a two-way analysis of variance (gender-by-group). Sheffee's post hoc analysis was used to identify specific mean differences (McNemar, 1969). The relationship between BMR and estimates of body composition were made using linear and polynomial regressions. Comparisons between the slopes were made using analysis of variance. In all cases, data are presented as mean \pm SD. Statistical significance was set at $P \leq 0.05$.

RESULTS

Table 1 presents the data for males and females for each group. There was a significant gender-by-group interaction ($P \le 0.05$) for all variables except age. Since the lean subjects were slightly younger (ns) than the obese and normal subjects, and since the normal and lean data for the most part were taken from studies conducted many years ago, the possibility of secular effects of growth and nutrition needs to be addressed. There is no way of knowing the extent of this effect on BMR per se; nevertheless, comparisons of the BMRs for the data taken from the literature with the data collected in our laboratory revealed no appreciable or statistical differences in BMR. The greatest difference was 3% between the BMR data for the 6 lean female adolescents collected in our laboratory, and the female data collected by Blunt et al. (1921). These data suggest that perhaps any secular trends in nutrition and or growth are limited with respect to effects on BMR. Thus, we believe this permits comparisons between groups.

There were significant differences in body composition between the 3 groups for all variables. Moreover, there were gender differences between and within each group for all variables except BM for the obese males and females, and for % BF and FM for the normal males and females. For all variables, the obese had significantly larger values than the normal and lean, and the normals had larger values than the lean.

Figure 1 presents the BMR data expressed in absolute and relative terms. When expressed as kJ · hr⁻¹ (Fig. 1A), obese males had a nearly 12% higher BMR compared to normal males ($P \le 0.05$), and 22% compared to lean males ($P \le 0.05$). When indexed to BM (Fig. 1B) obese males had a significantly depressed BMR of -38% compared to normal males ($P \le 0.01$), and -50% compared to lean males ($P \le 0.01$). Similarly, when indexed to FFM (Fig. 1C) and BSA (Fig. 1D), obese males had significantly depressed values of -16.6% ($P \le 0.05$) and -14% ($P \le 0.05$) compared to normal males, and -33% ($P \le 0.01$) and -22.5% ($P \le 0.01$) compared to lean males, respectively.

Obese females' BMR expressed as kJ hr⁻¹ (Fig. 1A) were elevated by 9.5% ($P \le 0.05$) compared to normal females, and 23% compared to lean females ($P \le 0.05$). As with the males, when indexed to BM (Fig. 1B) obese females had significantly depressed BMRs of -34% compared to normal females ($P \le 0.01$), and -53.8% compared to lean females ($P \le 0.01$). When indexed to FFM (Fig. 1C), obese female values were depressed by -11.4% compared to normal females ($P \le 0.05$), and -30% compared to lean females ($P \le 0.01$). Indexed to BSA (Fig. 1D), it was less by -14% compared to normal females ($P \le 0.05$), and -26% compared to lean females ($P \le 0.05$).

There were no significant differences in BMR between males and females (Fig. 1). BMR $(kJ \cdot hr^{-1})$ for normal males and females was greater than for lean males and females $(P \le 0.05)$. However, when indexed relative to BM, FFM, and BSA, normals had values that were significantly $(P \le 0.05)$ depressed in comparison to lean males and females, respectively.

Table 2 presents the correlations between BMR $(kJ \cdot hr^{-1})$, age, BM, stature, BSA, and body composition. For females, BSA, BM, and stature accounted for approximately the same amount of variance in $kJ \cdot hr^{-1}$ as either FM or FFM. The correlations for males were very similar for obese and lean, while the correlations for normals were much higher for 6 of the 7 comparisons. The correlations with BM, BSA, FFM, and stature were remarkably high for normal males.

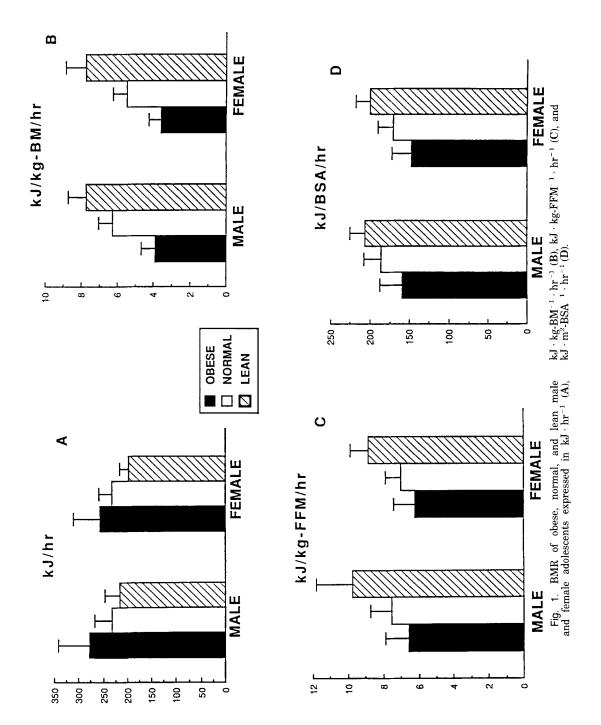
DISCUSSION

The major finding of the present study was the difference in basal metabolic rate between obese, normal, and lean adolescents (Fig. 1). When expressed as $kJ \cdot hr^{-1}$ the obese were *hypermetabolic*. When indexed relative to BM, FFM, or BSA they were *hypometabolic*. These findings offer conflicting theoretical perspectives in terms of understanding the relationship between obesity, metabolism, and body composition.

The $kJ \cdot hr^{-1}$ data suggest that factors other than a depressed metabolism, perhaps hormonal disturbances (Vinik, 1983), increased caloric consumption (Curtis and

	Ob	Obese	Normal	mal	Lean	u	
	$\begin{array}{l} \mathbf{Male} \\ \mathbf{N} = 30 \end{array}$	Female $N = 37$	$ Male \\ N = 74 $	Female $N = 50$	$\begin{array}{l} \text{Male} \\ \text{N}=20 \end{array}$	Female N = 18	Significance ²
	$\overline{X}\pm SD$	$\overline{\mathbf{X}}\pm\mathbf{S}\mathbf{D}$	$\overline{\mathbf{X}} \pm \mathbf{SD}$	$\overline{\mathbf{X}} \pm \mathbf{SD}$	$\overline{X} \pm SD$	$\overline{\mathbf{X}}\pm\mathbf{SD}$	4
Age, yr BM, kg	12.7 ± 1.7 76.92 ± 22.8	12.8 ± 1.7 74.3 ± 17.8	13.0 ± 1.7 38.0 ± 9.0	12.3 ± 1.1 43.0 ± 7.6	11.9 ± 1.9 28.0 ± 4.3	10.8 ± 1.8 26.1 ± 4.9	NS OBM > OBF,NM,NF,LM,LF, OBF > NM,NF,LM,LF, NM > LM,LF;
Stature, m	1.595 ± 0.108	1.596 ± 0.109	1.492 ± 0.117	1.490 ± 0.082	1.390 ± 0.084	1.390 ± 0.098	NF > LM,LF OBM > NM,NF,LM,LF, OBF > NM,NF,LM,LF; NM > LM,LF;
BSA, m ²	1.789 ± 0.30	1.747 ± 0.24	1.270 ± 0.19	1.370 ± 0.14	1.063 ± 0.12	1.000 ± 0.12	OBM > NM,NF,LM,LF; OBF > NM,NF,LM,LF; OBF > NM,NF,LM,LF; NM > LM,LF;
% fat	39.85 ± 8.1	42.30 ± 5.6	15.90 ± 4.6	20.08 ± 2.7	13.45 ± 6.4	12.25 ± 3.1	OBM > NM.NF.LM.LF. OBF > NM.NF.LM.LF. OBF > NM.NF.LM.LF. NM < NF. NM < NF.
FM, kg	32.17 ± 16.3	31.99 ± 11.0	6.04 ± 1.6	8.63 ± 2.7	3.67 ± 1.3	3.19 ± 1.2	OBM NR NM.NFLMLF. OBF > NM.NFLMLF. OBF > NM.NFLMLF. NM < NF. NM > LMLF. NF > LMLF.
FFM, kg	45.02 ± 9.6	42.46 ± 8.5	31.96 ± 8.7	34.37 ± 4.5	24.24 ± 4.3	22.90 ± 3.8	NF > LM,LF OBM > NM,NF,LM,LF; OBF > NM,NF,LM,LF; NM > LM,LF; NF > LM,LF;
FFM/FM	1.399 ± 0.45	1.344 ± 0.33	5.291 ± 0.66	3.982 ± 0.74	6.4477 ± 0.65	7.178 ± 0.64	OBM > NM,NF,LM,LF; OBF > NM,NF,LM,LF; NM > LM,LF; NF > LM,LF;

²Lists specific mean differences. For example, LF < NF,NM should be interpreted as lean female value less than normal female and normal male. The other comparisons should be interpreted similarly (OBM = obese male; OBF = obese female; NM = normal male; NF = normal female; LM = lean male; LF = lean female) All values are X ± SD.



kJ∙hr ^{−1} versus	Male			Female		
	Obese	Normal	Lean	Obese	Normal	Lean
Age	0.47*	0.58*	0.52*	0.53*	0.04	0.63*
BM	0.63^{*}	0.92^{*}	0.52*	0.75*	0.56*	0.74*
Stature	0.56*	0.89*	0.58*	0.48*	0.48*	0.77*
BSA	0.63*	0.99*	0.58*	0.69*	0.56*	0.76*
% BF	0.53*	0.54*	0.37	0.43*	0.55*	0.18
FM	0.59*	0.19	0.10	0.71*	0.53*	0.50
FFM	0.41*	0.92^{*}	0.57*	0.64*	0.57*	0.77*

TABLE 2. Correlations between $kJ \cdot hr^{-1}$ versus body composition variables

*Significantly different than zero, $P \leq 0.05$.

Bradfield, 1971), decreased energy expenditure (Bradfield et al., 1971), or a decreased dietary induced thermogenic response to food (Kaplan and Leveille, 1976; Felig and Cunningham, 1983) may be responsible for increased body mass gain in obesity.

In contrast, the ratio expressions of BMR suggest that body mass gain may be more a function of long term energy conservation. The underlying assumption for use of ratio expressions of metabolism have been previously reviewed and shown to be rarely justified on statistical grounds (Tanner, 1949; Katch, 1973). Nevertheless, their use is widespread because they are believed to perform a statistical "adiposectomy," that allows for comparisons of metabolism among individuals or groups that differ in body composition (Cunningham, 1981, 1982; Katch, 1973). The use of ratio expressions is based on the assumption that animals of the same species are comparable, and thus more or less similar. This assumption has been stated as the "Law of Similarity" (Kleiber, 1975), and supposedly offers the basis for comparing build and function of animals of different sizes. The most likely terms for comparing build and function are those that remain constant for changing sizes, such as length volume⁻¹, surface area vol ume^{-1} volume volume⁻¹, and volume mass⁻¹. These ratio expressions have long been used in comparative physiology (see Kleiber, 1950, 1975).

There are certain assumptions made when using ratio expressions. In case of the BMR, for example, the assumption is that the energy expenditure is directly proportional to BM (or whatever relative indexing term is used). In fact, the ratio terms assumes the following (Tanner, 1949):

$$\mathbf{kJ} \approx \mathbf{F} \cdot \mathbf{BM} \tag{1}$$

where the constant \mathbf{X} is determined by the mean body mass and mean energy expenditure in kJ. This ratio, by virtue of its form, implies that it passes through the origin as well as through the mean kJ and BM. Stated another way, the ratio assumes a perfect linear relationship between kJ and BM, and that the two variables are directly proportional. In actual practice, correlations between kJ and $\dot{B}M$ (or any other body composition component) are less than r = 1.0 (see for example, Cunningham, 1982; Tanner, 1949; Katch, 1973). In the present data, no one body composition variable is particularly good as an indexing variable across the 3 groups (Table 2). For example, while BSA is highly correlated to BMR for normal males (r = 0.99), it is only moderately correlated $(r \le 0.76)$ for the other groups. The same is true for the correlations with FFM.

As early as 1919 Harris and Benedict were well aware of this ratio "fallacy" and so established the norms of BMR per surface area based on regressed scores (BMR vs. BSA) rather than simple ratio scores (BMR/BSA). In 1938 Benedict stated, "It is believed that far greater progress will be made by discarding all thoughts of a uniformity in heat loss and emphasizing the non-uniformity in heat production" (Benedict, 1938, p. 194). This argues against the use of simple ratio expressions.

Now it is obvious that the daily rate of metabolism (heat production) of a large (obese) individual is greater than that of a smaller (lean) individual. If this metabolic difference is an inherent function of the metabolic requirements of different body tissues (e.g., fat free mass, fat mass), this suggests the need to reference energy metabolism to some measure of composition (Kleiber, 1950). This then is the rationale for use of a metabolic-body composition indexing expression. This expression must be a sensitive measure of metabolism per se, and also be able to reflect inherent differences in the energy metabolism of individuals with markedly different fat and fat free components (e.g., males and females, obese and lean, or even the fit and unfit) by considering the specific energy expenditure (thermal equivalents) of both the fat and fat free body mass.

A derived standard with the above characteristics can be written as the sum of the contribution from different tissues (in this example, only fat and fat free mass are considered), each with a specific mass and energy expenditure per unit time, as follows:

$$\mathbf{kJ} \cdot \mathbf{hr}^{-1} = (k_1 \cdot FM) + (k_2 \cdot FFM); \quad (2)$$

where *FM* and *FFM* are the fat mass and fat free mass in kilograms, and k_1 and k_2 are the energy constants for the fat and fat free mass compartments expressed in kJ · kg-FM⁻¹ · hr⁻¹, and kJ · kg-FFM⁻¹ · hr⁻¹, respectively. Equation 2 can be rewritten as follows:

kJ · kg-BM⁻¹ · hr⁻¹ = (%BF
$$[k_1 - k_2] + k_2)$$
(3)

$$\mathbf{kJ} \cdot \mathbf{hr}^{-1} = FM \cdot (k_1 + FFM/FM \cdot k_2) \quad (4)$$

Equation 3 gives the metabolic rate referenced to body mass, based on the decimal expression of %BF. Equation 4 considers the metabolic contributions of the fat free mass to fat mass ratio (FFM \cdot FM⁻¹). The metabolic rates calculated using these deriva-

tions should provide a means of comparing the metabolic rates for individuals of different body masses and compositions taking into consideration the specific energy expenditures of the fat and fat free mass components. For the sake of clarity, these derived values can be referred to as a "theoretical metabolic rate" (TMR) and calculated for each individual (and group) in the present study. To illustrate this derivation, for example, we used a k_1 constant of 1.117 kJ · kg-FM⁻¹ · hr⁻¹ and a k_2 constant of 4.864 kJ · kg-FFM⁻¹ · hr⁻¹. These values were recently derived for adults by Garby et al. (1988), and are similar to the values reported by Nakamura and Abe (1975) for adults and more recently by Marks and Katch (1986) for adolescents. It should be understood that these energy constants need to be validated and their use here is only illustrative.

Figure 2 presents the observed BMR and derived TMR values expressed in kJ kg- $BM^{-1} \cdot hr^{-1}$ (Eq. 3). The large differences between obese, normal, and lean males and females for BMR (solid bars) are obvious (Fig. 1). The differences in TMR (open bars) revealed that the obese male and female values were depressed by an average 22% compared to the normal and lean males and females ($P \leq 0.05$), respectively. The differences in TMR between the normal and lean males and females was only 8% (ns). Thus, the TMR, like the BMR expressed relative to BM, BSA, and FFM, was depressed for the obese compared to the normal and lean adolescent. These data suggest an energy conservation hypothesis for the obese adolescent compared to normal and lean adoles-

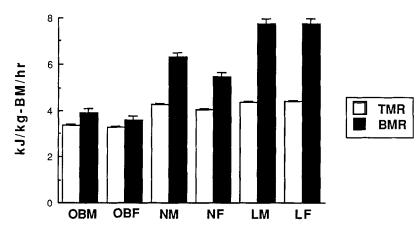


Fig. 2. Comparisons of BMR (filled bars) and TMR (open bars) for obese males (OBM), obese females (OBF),

normal males (NM), and normal females (NF), lean males (LM), and lean females (LF).

cents. A depressed metabolic rate of 20% is substantial when converted to energy (fat) storage over time, and could help explain long-term body mass gain for the obese. In summary, the data from the present

study indicate that obese male and female adolescents have depressed resting metabolic rates, and hence conserve energy, compared to normal and lean adolescents whether expressed in traditional ratio terms, or expressed as a theoretical metabolic rate (TMR) that considers the specific thermal equivalents of the fat and fat free body mass. Further research needs to focus on specific mechanisms and factors that contribute to energy conservation for the obese. Specifically, the role that body composition, and in particular the fat free mass, plays in determining individual differences in metabolism appears particularly important, as do changes in the metabolic-FFM relationship.

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