

### Energy requirements of obese children and young adults\*

By D. A. SCHOELLER, *Department of Medicine, Box 223, University of Chicago, 5841 S. Maryland Avenue, Chicago, IL 60637, USA* and L. G. BANDINI *Clinical Research Center, Massachusetts Institute of Technology, 50 Ames Street, Cambridge, MA 02142, USA* and L. L. LEVITSKY, *Department of Pediatrics, University of Chicago, 5841 S. Maryland Avenue, Chicago, IL 60637, USA* and W. H. DIETZ, *Department of Pediatrics, Tufts New England Medical Center, 171 Harrison Avenue, Boston, MA 02111, USA*

Estimates of the prevalence of obesity among adolescents in developed countries varies depending on the country and the criteria used to define obesity. There are few, however, who would argue that the prevalence is low. Moreover, the prevalence is increasing. If we define obesity as a tricep skinfold thickness of greater than the 85th percentile from the US Health and Nutrition Evaluation Survey II, then the percentage of US adolescents who are obese has increased from 15% in 1970 to 21% in 1980 (Dietz, 1987). This is of great concern because of the adverse effects of adolescent obesity on social and emotional development (Goodman *et al.* 1963) and because obesity is implicated in the development of hypertension (Rames *et al.* 1978) and maturity-onset diabetes mellitus (Medalie *et al.* 1974). Moreover, approximately 80% of obese adolescents become obese adults (Lloyd *et al.* 1961) and between half and three-quarters of very obese adults (160% ideal-body-weight) were obese as children (Rimm & Rimm, 1976).

As reviewed by Dietz (1983), it is clear that obesity by definition is a state of expanded body energy stores and thus a result of dietary energy intake exceeding energy expenditure, but it is not clear if there is a causal relationship between obesity and excessive energy intake, decreased energy expenditure, or failure to regulate the balance between intake and expenditure.

#### *Energy intake*

Most reports indicate the daily energy intake does not differ between obese and non-obese adolescents. For example, Peckos (1953) monitored intake using a 7 d record in eighty-six children classified as endomorphs (stocky or obese), mesomorphs (muscular), or ectomorphs (lean) and reported that the endomorphs had a 15% lower energy intake than the ectomorphs. Hampton *et al.* (1967) reported similar results after comparing dietary intakes from four non-consecutive 7 d records in 122 children. Children classified as obese on the basis of body fat content had a lower daily energy intake than those classified as lean or average. Durnin *et al.* (1979) also compared the reported energy intakes of children below the 10th percentile (thinnest) and children above the 90th percentile (fattest) from a group of 611 adolescents. The obese girls reported lower intakes than the thin girls, while there was no difference between the obese and thin boys.

Based on the previously-mentioned energy intake values, as well as numerous similar studies (Bandini, 1987), one is led to the conclusion that the obese have lower energy requirements than the lean, either because of greater efficiency in energy metabolism or because of a difference in physical activity.

\*Supported by National Institutes of Health grants DK 26778, DK 30031, HD 17696 and RR 00088 and the Thrasher Fund.

Energy intake values from dietary records, however, are subject to errors. For example, Beaudoin & Mayer (1953) reported that diet records from obese women gave systematically lower values for intake than the women's diet histories, but that the two methods gave similar results in a normal-weight population. It has often been speculated that keeping a diet record alters dietary behaviour or that records may be subject to under-reporting. This may well occur if the obese are embarrassed about the amount and types of food they are consuming.

Direct observation may avoid some of these errors. Waxman & Stunkard (1980) compared intakes by direct observation and food weighing in four sets of brothers in whom one was obese and the other non-obese. From seven to twelve dinners and three to six lunches were observed in each set and it was found that the obese boys consumed more energy than their non-obese brothers.

Because of the potential errors in diet records (Bandini *et al.* 1987a), it is difficult to come to any conclusion about the role of intake in adolescent obesity, however, the findings have led to the suggestion that obese adolescents are more energy efficient than the non-obese. Because of this suggestion, there have been a number of energy expenditure studies in obese adolescents. Most of these studies can be categorized according to the major components of energy expenditure that were investigated: basal metabolic rate, dietary-induced thermogenesis or physical activity.

#### *Basal metabolic rate*

Basal metabolic rate is the rate of energy expenditure of an individual at rest and in the postabsorptive state that is measured under standardized conditions. It generally comprises more than half the total daily energy expenditure and represents the amount of energy necessary to maintain life processes.

Comparisons of basal metabolic rate between obese and non-obese adolescents show that the obese have higher metabolic rates than the non-obese, but then the obese are physically larger than the non-obese. To account for the larger body size, values have been normalized by any one of a number of factors. The method that gives the smallest inter-individual variation in most studies and thus the method recently used by many investigators is normalization according to fat-free mass. Unfortunately, most studies of basal metabolic rate in adolescents tend to predate the use of fat-free mass for normalization. A number of studies, however, do use factors that correlate with fat-free mass. In studies by Blunt *et al.* (1926) and Mossberg (1948) there was no difference in the basal metabolic rate per unit surface area between obese and non-obese adolescents. Talbot & Worcester (1940) compared basal metabolic rates on the basis of creatinine excretion and reported that obese children had higher basal metabolic rates than non-obese children. Creatinine excretion, however, is an index of muscle mass which is one of the least-metabolically-active tissues in the fat-free mass of individuals at rest and thus may not be a good basis for comparison.

Recently, we have begun studies in sixty-one obese and non-obese adolescents. As in previous studies, we found a higher basal metabolic rate expressed in absolute terms in obese subjects (mean) than in non-obese (boys: 9427 (SD 1548) v. 7293 (SD 766) kJ/d (2253 (SD 370) v. 1743 (SD 183) kcal/d); girls: 7945 (SD 1443) v. 6029 (SD 561) kJ/d (1899 (SD 345) v. 1441 (SD 134) kcal/d)). When the values were expressed per unit fat-free mass as calculated from total body water, however, there were no differences between groups (boys: 163 (SD 16.7) v. 159 (SD 20.9) kJ/kg fat-free mass per d (39 (SD 4) v. 38 (SD 5) kcal/kg fat-free mass per d); girls: 151 (SD 12.6) v. 146 (SD 12.6) kJ/kg fat-free mass per d (36 (SD 3) v. 35 (SD 3) kcal/kg fat-free mass per d)) (Bandini *et al.* 1987b).

From the previously-mentioned evidence, it can be concluded that obese adolescents have higher unnormalized basal metabolic rates than the non-obese. The obese, however, have a larger fat-free mass than the non-obese. No difference is observed when the values are expressed on a per kg fat-free mass basis.

#### *Dietary-induced thermogenesis*

A broad definition of dietary-induced thermogenesis is the increment in energy expenditure associated with feeding. This includes the obligatory energy costs of digestion, transport, and storage of nutrients, and a non-obligatory component that may serve a compensatory function for weight maintenance. The latter component dates in part to the concept of 'luxuskonsumption' that was proposed by Neumann (1902) and later Gulick (1922) who both reported that weight could be maintained at different levels of dietary intake and proposed that excess dietary intake was dissipated as heat.

Small-animal studies have provided strong evidence for a numerically-significant non-obligatory dietary-induced thermogenesis that is mediated by brown adipose tissue (Himms-Hagen, 1976). Brown adipose tissue, however, does not constitute a large tissue mass in humans other than newborns (Heaton, 1972). Moreover, a re-evaluation of Neumann's (1902) and Gulick's (1922) findings by Forbes (1984) reported that weight did indeed vary with intake and the weight gain during overfeeding was consistent with storage of the excess energy rather than a large non-obligatory dietary-induced thermogenesis. Indeed, a well-controlled measurement of 24 h energy expenditure in adults found no evidence for 'luxuskonsumption' during overfeeding (Ravussin *et al.* 1986).

Although the evidence for a large non-obligatory dietary-induced thermogenesis in humans is weak at best, there is indeed an increment in energy expenditure that occurs after a meal. This increment is of the order of 10% and a number of studies have suggested that there are a significant number of obese adults who have a dietary-induced thermogenesis that is reduced to about half that observed in lean subjects (Schutz *et al.* 1985; Segal *et al.* 1985). There is an absence of information on dietary-induced thermogenesis in adolescents.

#### *Physical activity*

The second-largest component of total energy expenditure is the energy expended in physical activity. This includes mild activities, such as sitting, as well as rigorous exercise and thus is a component that is quite variable between individuals. Few values exist for the energy expended during physical activity. There is, however, a body of literature regarding the types of activities and amount of time spent in these activities by obese and non-obese adolescents.

In the Hueneman *et al.* (1967) study, 120 obese and non-obese boys kept activity records. Both groups were reported to be generally inactive and there was no significant difference between groups. Using a more-sophisticated time-sampled activity record, Waxman & Stunkard (1980) reported that obese boys were less active than non-obese boys at home, but there were no differences in their activities when they were in a playground.

Self-recorded activity records suffer from the same potential compliance problems as self-reported dietary intake and thus should be compared with more direct measures of activity. Stunkard & Pestka (1962) used pedometers to actually record the distances walked by adolescent girls. They found that obese girls did not walk as far as non-obese girls, but the difference was not significant. Bullen *et al.* (1964) kept a filmed record of

Table 1. *Total energy expenditure in adolescents determined by the doubly-labelled-water method*

(Mean values and standard deviations for nine subjects per group)

Group	kJ/d		kcal/d		kJ/kg FFM per d		kcal/kg FFM per d	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Prader-Willi	8660	2175	2070	520	280	50	67	12
Exogenous obese	15480	3180	3700	760	263	54	63	13
Non-obese	12090	2930	2890	700	272	42	65	10

FFM, fat-free mass.

girls at summer camp and found that the level of activity of obese girls was less than that of non-obese girls.

Taken together, the previously-mentioned evidence suggests that obese adolescents are less active than non-obese adolescents. When one considers, however, that the obese have a greater body mass than the non-obese then it is likely that the difference may not actually translate into a lower mean daily energy expenditure due to physical activity.

#### *Total energy expenditure*

Taking all the previous findings together leads to a paradox. The intake values suggest that obese adolescents may be energy efficient because they have the same self-reported intakes as the non-obese, but larger body sizes. Summing the components of energy expenditure from multiple studies, however, fails to reveal this significant discrepancy. Obviously, this comparison is fraught with potential errors because it spans multiple studies. Until recently, scientists have had to rely on these types of investigations because there has not been a method for measuring total energy expenditure in free-living subjects.

We have recently begun to use the doubly-labelled-water method to measure total daily energy expenditure in free-living adolescents and young adults and thus avoid the problems of trying to sum the components of energy expenditure and of indirect measures such as dietary intake. The theory and details of this method have been discussed by others during this symposium and have been published elsewhere (Schoeller, 1983; Schoeller *et al.* 1986). We have initiated studies in three groups of adolescents: a genetically-obese group of children and young adults with Prader-Willi syndrome, a group of adolescents with exogenous obesity, and a non-obese group (L. G. Bandina, D. A. Schoeller and W. H. Dietz, unpublished results).

There were nine subjects in each group with either four or five boys and girls in each group. Mean values for the Prader-Willi group were: age 17 (SD 4) years, height 1.44 (SD 0.11) m, weight 64 (SD 23) kg, body fat 48 (SD 7) % as calculated from  $^{18}\text{O}$  measurement of total body water and an assumption of 73% hydration of fat-free mass. The corresponding values for the exogenous-obesity group were: age 16 (SD 2) years, height 1.69 (SD 0.07) m, weight 108 (SD 25) kg, body fat 45 (SD 6) %. The values for the non-obese group were: age 14 (SD 1) years, height 1.69 (SD 0.12) m, weight 59 (SD 9) kg, body fat 21 (SD 10) %.

Total daily energy expenditure is summarized in Table 1. Each group differed significantly from the others (*t* test,  $P < 0.05$ ) with the exogenous obese having the

greatest expenditure and the Prader–Willi subjects having the lowest expenditure. If the total expenditure is expressed on a per kg fat-free mass basis, these differences disappear.

Expression of the values on a per kg fat-free mass basis, however, provides little information on physical activity and is not very informative. We have therefore calculated the expenditure in activity using the multiple-regression equations of Ravussin *et al.* (1986), i.e. total daily energy expenditure = 667 + 20.5 fat-free mass + activity. In the exogenous-obese and the non-obese groups, we measured basal metabolic rate and showed that it was very similar to that predicted from fat-free mass (Cunningham, 1982). In a second group of six Prader–Willi subjects, not included in the total energy expenditure study, we showed that this prediction averaged 97 (SD 7) % of the measured values. The energy expended in activity was statistically different between all groups. Again, the expenditure was greatest in the exogenous obese and least in the Prader–Willi subjects. When expenditure in activity was expressed as a percentage of total daily energy expenditure, the pattern was the same, but the difference between the non-obese and exogenous obese was not significant. Finally, we expressed the calculated expenditure in activity on a per unit body-weight basis because the rate of expenditure for a given physical activity is largely related to the mass (Givoni & Goldman, 1971). When expressed on this basis, we found that the expenditure was similar for the exogenous obese and non-obese, but significantly lower for the Prader–Willi subjects.

#### *Conclusions and remarks*

The implication from dietary-intake records that obese adolescents are more energy efficient than non-obese is not supported by measurement of energy expenditure. The obese adolescents actually have a greater energy expenditure than the non-obese. It is only in the small-statured obese such as the Prader–Willi subjects that the expenditure is less than that of the non-obese. Thus, neither our small study nor reported studies have identified large energy efficiencies in obese adolescents and raises grave doubts about the validity of the intake values.

Although there is evidence in the literature for reductions in components of energy expenditure such as dietary-induced thermogenesis, it should be remembered that these reductions are smaller than the 3347 kJ (800 kcal)/d difference in the total expenditure in our obese and non-obese adolescents. Because these exogenous-obese individuals maintain their obesity in the presence of this large expenditure it is hard to imagine that obesity can be explained from differences in expenditure alone. Perhaps it will be more important, but obviously more difficult, to formulate research questions about obesity by looking at energy balance rather than intake or expenditure alone.

The nine exogenous-obese subjects in our preliminary studies had an energy fat store of 36 kg or about 1422 MJ (340 000 kcal) more than the non-obese subjects. If we assume that this excess storage was amassed gradually over 5 years, then we are dealing with an imbalance between intake and expenditure of slightly less than 837 kJ (200 kcal)/d. Does the imbalance actually occur on a daily basis or does it represent periods of gross imbalance that are not compensated for by reduced intake for the next few days? Does it represent the influence of biochemical failure in appetite or energy storage and retrieval? Is there a significant psycho-social influence that results in excessive intake? Evidence suggests that many of these factors may be influential in causing obesity, but definitive answers will require a research approach that considers the balance between intake and expenditure.

## REFERENCES

- Bandini, L. G. (1987). Energy expenditure in obese and nonobese adolescents. PhD Thesis, Massachusetts Institute of Technology, Cambridge, Mass.
- Bandini, L. G., Schoeller, D. A., Cyr, H., Young, U. R. & Dietz, W. H. (1987a). *International Journal of Obesity* **11**, 437A.
- Bandini, L. G., Schoeller, D. A., Young, V. R. & Dietz, W. H. (1987b). *American Journal of Clinical Nutrition* **45**, 868, Abstr.
- Beaudoin, R. & Mayer, J. (1953). *Journal of the American Dietetic Association* **29**, 29–33.
- Blunt, K., Tilt, J., McLaughlin, L. & Gunn, K. B. (1926). *Journal of Biological Chemistry* **67**, 491–503.
- Bullen, B. A., Reed, R. B. & Mayer, J. (1964). *American Journal of Clinical Nutrition* **14**, 211–223.
- Cunningham, J. J. (1982). *Journal of the American Dietetics Association* **80**, 335–338.
- Dietz, W. H. (1983). *Journal of Pediatrics* **103**, 676–686.
- Dietz, W. H. (1987). *American Journal of Diseases in Children* **141**, 535–540.
- Durnin, J. V. G. A., Lonergan, M. E., Good, J. & Ewan, A. (1979). *British Journal of Nutrition* **32**, 169–179.
- Forbes, G. B. (1984). *American Journal of Clinical Nutrition* **39**, 349–350.
- Givoni, B. & Goldman, R. F. (1971). *Journal of Applied Physiology* **30**, 429–433.
- Goodman, N., Dornbusch, S. M., Richardson, S. M. & Hastorf, A. H. (1963). *American Sociological Review* **28**, 429–435.
- Gulick, H. (1922). *American Journal of Physiology* **60**, 371–395.
- Hampton, M. C., Hueneman, R. L., Shapiro, L. R. & Mitchell, B. W. (1967). *Journal of the American Dietetic Association* **50**, 385–396.
- Heaton, J. M. (1972). *Journal of Anatomy* **112**, 35–39.
- Hueneman, R. L., Shapiro, L. R., Hampton, M. C. & Mitchell, B. W. (1967). *Journal of the American Dietetic Association* **18**, 433–440.
- Himms-Hagen, J. (1976). *American Review of Physiology* **38**, 315.
- Lloyd, J. K., Wolff, O. H. & Wheland, W. S. (1961). *British Medical Journal* **ii**, 145–148.
- Medalie, J. H., Papier, C., Herman, J. B., Goldbourt, U., Tamer, S., Neufeld, H. M. & Riss, E. (1974). *Israel Journal of Medical Science* **10**, 681–697.
- Mossberg, H. (1948). *Nordisk Medicin* **39**, 1718–1720.
- Neumann, R. O. (1902). *Archives für Hygiene* **45**, 1–87.
- Peckos, P. S. (1953). *Science* **117**, 631–633.
- Rames, L. K., Clarke, W. R., Connor, W. E., Reiter, M. A. & Lauer, R. M. (1978). *Pediatrics* **61**, 245–251.
- Ravussin, E., Lillioja, S., Anderson, T. E., Christin, L. & Bogardus, C. (1986). *Journal of Clinical Investigation* **78**, 1568–1578.
- Rimm, I. J. & Rimm, A. A. (1976). *American Journal of Public Health* **66**, 479–481.
- Schoeller, D. A. (1983). *American Journal of Clinical Nutrition* **38**, 999–1005.
- Schoeller, D. A., Ravussin, E., Schutz, Y., Acheson, K. J., Baertschi, P. & Jequier, E. (1986). *American Journal of Physiology* **150**, R823–R830.
- Schutz, Y., Acheson, K. & Jequier, E. (1985). *International Journal of Obesity* **9**, Suppl. 2, 111–114.
- Segal, K. R., Gutin, R., Nyman, A. M. & Pi-Sunyer, F. X. (1985). *Journal of Clinical Investigation* **76**, 1107–1112.
- Stunkard, A. & Pestka, J. (1962). *American Journal of Diseases of Children* **103**, 812–817.
- Talbot, N. B. & Worcester, J. (1940). *Journal of Pediatrics* **16**, 146–150.
- Waxman, M. & Stunkard, A. J. (1980). *Journal of Pediatrics* **96**, 187–193.