Does energy expenditure affect changes in body fat in children? 1–3

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The search for the phenotypic expression of the genetic susceptibility to obesity has stimulated several investigations of energy expenditure that have used various study designs. These investigations have included longitudinal studies of the effects of baseline energy expenditure on weight gain in susceptible populations such as Pima Indians (1), comparisons of rates of weight gain among overfed monozygotic twin pairs (2), comparisons of energy expenditure after changes in body weight (3), and comparisons of energy expenditure in nonoverweight children of obese and nonobese parents (4). This issue of the Journal includes the publication of a study by Goran et al (5) that determined whether reduced baseline energy expenditure increased susceptibility to increased changes in fatness in young children.

Longitudinal studies have shown that lower metabolic rates occurred in overweight Pima Indians who gained ≥7.5 kg over a 2-y period (1). Additional studies of Pima Indians showing that resting metabolic rate had a familial component failed to show an increase in the prevalence of obesity among individuals from families with low metabolic rates compared with individuals from families with high metabolic rates (6). In overfed identical twins (2), the observation that the variance of rates of weight gain within twin pairs was lower than the variance between twin pairs suggested that there was a significant genetic component to the response to overfeeding. Studies of previously obese individuals after weight-reduction surgery suggested that energy expenditure may be reduced in the postobese, and by implication, in the preobese state (3).

The observation that obesity occurs more frequently in children of obese parents led to a variety of studies in which energy expenditure of children of obese and nonobese parents was compared. In 1976, Griffiths and Payne (4) extrapolated energy expenditure from resting metabolic rate and heart rate in 4–7-y-old nonoverweight children and suggested that children of overweight parents had lower energy expenditures than did children whose parents were not overweight (4). Although these authors speculated that the reduced energy expenditure would enhance susceptibility to obesity, a subsequent follow-up study failed to show an increased prevalence of obesity among the children of overweight parents (7). Nonetheless, the strategy to examine the effects of energy expenditure at baseline on subsequent changes in body fat remains a sound approach to exploring the phenotypic expression of genetically susceptible individuals.

Goran et al (5) report that baseline energy expenditure did not predict changes in body fatness over a 4-y period in male and female children aged 3.5–7.0 y at study entry. Although reduced energy expenditure may not predict changes in body fat, the average reader of this article may not be able to draw the same conclusion from Goran et al’s data. First, the data published elsewhere raise substantial questions about the validity of the method. Second, the outcome measures of body composition depend on anthropometric analyses that may have been flawed. Third, the data were so extensively adjusted that these adjustments may have obscured any differences that may have existed. I will consider each of these problems in turn.

In this study, energy expenditure was measured with the doubly labeled water method. The cohort examined included 30 children whose energy expenditure was described previously (8). Resting metabolic rate was measured 2–3 h after a meal. Both meal size and the time at which resting energy expenditure is measured after a meal can affect the magnitude of the thermic effect of a meal. In > 25% of these 30 children, resting energy expenditure was either higher than or within 418 kJ of total energy expenditure (8). Although energy expenditure was measured a second time in the same subjects, no other raw data are presented that allow us to assess the reproducibility of this finding in the original 30 children studied or the frequency of this unlikely possibility in the other subjects included in this report.

Rates of change in body fat mass, the primary outcome measure, were assessed through a complex series of measurements. Total body fat was calculated from an equation that included triceps and subscapular skinfold thicknesses, weight, total body water estimated from bioelectrical impedance analysis, and sex (9). The authors do not make clear whether all of the measurements were performed by the same individual. By my calculations, 3-mm differences in skinfold thickness measurements, a difference well within the range of interobserver variability, would introduce a difference of 25% in the calculation of body fat mass at the 4-y follow-up. Because the error of the method may exceed the magnitude of the difference in body fatness anticipated, the conclusion that differences in body fat did not exist is questionable. Even if we accept the validity of the anthropometry, the range of percentage body weight as fat shown

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in Table 1 suggests that several of the children were already overweight. The development of obesity may eliminate preexisting differences in energy expenditure (1).

The rate of change in body fat mass was adjusted for the rate of change in fat-free mass by calculating the residual fat mass derived from a regression of fat mass and fat-free mass for the entire cohort at each of the ages examined. The residual fat mass was then plotted as a function of age to obtain an individual slope that reflected the rate of change in residual fat mass. This approach generates several problems. First, the equation used to calculate fat mass included the term $h^2/R$ derived from impedance (where $R$ is resistance), and the same term was used to estimate fat-free mass. Regression of two variables that include the same term may introduce a spurious correlation. The same problem affects the data in Figure 2, on which the conclusions of the paper depend. In this figure, adjusted residual fat mass was not significantly related to total energy expenditure adjusted for fat-free mass. Again, the effect of the impedance term on both sides of the relation remains unclear, but the term introduces a potential error. In both analyses, it would have been helpful to see analyses that included simple direct anthropometric measures before these measurements were obscured with adjustments of questionable validity. A final problem with these data is that the oldest subjects at the end of the study were 11 y old. At this age, some girls were likely pubertal whereas all the boys were likely still prepubertal. In both pubertal girls and prepubertal boys, increases in body fat are expected (10, 11). Therefore, the negative slopes for the changes in body fat in boys shown in Figure 1 are unexpected and suggest that some of the adjustments may have introduced another source of error.

The difficulties posed by the interpretation of these data emphasize several principles. First, reviewers and editors should insist that they receive the primary data on which the conclusions of a manuscript are based. In this report, the methods and data were included in three other publications, all of which were referenced (8, 9, 12), but publication of essential information elsewhere requires the reader to retrieve the data essential to understand the current publication. Second, the validity of the mass-spectroscopy analyses (13) on which the calculations of energy expenditure depend must be included in the methods. Third, anthropometry is a poor substitute for more refined measures of body composition. Finally, call me old-fashioned. I prefer measurements to statistical manipulations.

REFERENCES