Energy expenditure in obesity 1,2

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A few years ago, obesity seemed to be a problem that was almost solved. Molecular studies were highlighting the underlying causes of overeating and weight gain, and novel drugs were promising new ways to permanently treat America’s greatest health problem. With these exciting developments being pursued, gathering basic information on such issues as energy expenditure (EE) seemed of dubious long-term value.

Unfortunately, the prevalence of obesity has continued to increase, and at the same time it has become clear that pharmacologic agents are not effective solutions for every weight problem. Long-term studies of currently available antiobesity medications showed that medication use can augment weight loss modestly (by 3–8% of initial weight) beyond that attributable to diet and exercise (1). However, medications are not appropriate for all obese persons, and some of the more effective drug treatments for obesity also proved to be unsafe and are no longer available (2, 3). Combined with the reports suggesting that persons who are highly active may be more able to both lose weight and maintain weight loss than are sedentary persons (4, 5), these findings emphasize the need for further studies of energy intake and expenditure that allow for improved definitions of usual energy requirements and the uncertain role of low EE. Placed in this context, observational studies like those of Delany et al (6) provide us with essential information.

Delany et al (6) conducted repeated measures of total EE (TEE) and resting metabolic rate in a large group of children over a 2-y span in order to examine the determinants of EE. They used the doubly labeled water method to determine the TEE and hence the weight-maintenance energy intake in these subjects, thus obtaining reliable measurements that were not influenced by subject reporting. One of the important findings of their study was that the mean rate of EE for physical activity in this preadolescent population was low relative to the standard EE and did not increase over the 2-y span between the original and follow-up studies. Physical activity level values (TEE divided by resting metabolic rate) for the population averaged only 1.5, which is within the second-to-lowest activity category among the activity categories defined in the new dietary reference intakes (7). Although population survey data indicate that physical activity has not decreased over time in parallel with the rising national prevalence of obesity (8), previous doubly labeled water research raised the possibility that mean physical activity level values of ≥1.89 may be needed to reliably prevent obesity (4). Because the subjects in the study of DeLany et al were all schoolchildren at the time of the study, questions should be raised about the appropriate role of schools in particular and society in general in facilitating suitable levels of physical activity for healthy physical development in childhood.

DeLany et al (6) also reported that there was no difference in physical activity level values between lean children and obese children, but the obese group had higher values of TEE. Whereas EE for physical activity was low in the population as a whole, it was not lower in the obese children than in the lean children. Instead they had higher absolute TEE and therefore higher usual energy intakes. This research clearly justifies a practical emphasis on combined strategies that focus both on increasing EE broadly in the population and on decreasing energy intake specifically among the obese. These findings and recommendations are consistent with the results in recent reports showing parallel increases in both energy intake per capita and reported energy intake in national surveys over the period when the prevalence of obesity increased (9). However, per capita information is developed on the basis of many assumptions, and food surveys are susceptible to an uncertain bias introduced by subject reporting. Thus, unbiased information from studies like that of DeLany et al is an important adjunct to survey data from the larger normative populations.

DeLany et al also showed that the absolute mean TEE of the African American children was similar to that of the white children, despite the fact that the African American children weighed more, whereas they had lower TEE than did the white children after adjustment for differences in body size. Previous studies suggested that African American children may have lower resting metabolic rates that might increase their risk of obesity (10, 11). The study by DeLany et al documented low resting metabolic rates along with the lower TEE in the African American children, which highlighted the potential for low EE to increase the African American children’s susceptibility to obesity. The finding of lower TEE among the African American children than among the white children suggests that dietary recommendations for African American children should be modified to prevent overeating. In relation to improving dietary recommendations to help especially susceptible

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groups, definitions of obesity may need to be race specific. In the group of children studied by DeLany et al (6), the mean body mass index was significantly higher but the percentage body fat determined by DXA was significantly lower in the African American children than in the white children. The prevalence of obesity will be falsely overestimated if body mass index is used to define obesity in populations with low body fat.

In summary, if we are to appropriately prevent and treat most cases of human obesity with diet and exercise, we need accurate information on the usual energy requirements of different groups of nonobese and obese persons and on the effects of low EE on the risk of weight gain in each group. The study by DeLany et al provides one piece of this information. We hope that many further such studies will be planned, and we also hope that the inexpensive isotope needed to make such studies possible will become (and remain) more widely available.

REFERENCES