Reply to AM Nevill et al

Dear Sir:

We read with interest the letter from Nevill et al, which discusses possible explanations for our recent report that height was positively correlated with some body girths but negatively correlated with other girths, and that these correlations differed between the 2 sexes (1).

Given widespread interest in the etiology of obesity, we emphasized in particular the inverse correlation between height and waist girth, which was substantially stronger in men ($t$ ratio = 23.9) than in women ($t$ ratio = 3.6) (1). We suggested that such an association in men might derive from genetic factors or from a developmental link between growth and adiposity. We considered the latter a likely important explanation, as evidenced by work among Brazilian children in whom stunting has been associated with impaired fat oxidation and predisposition to central adiposity (2).

Nevill et al suggest a simpler explanation, on the basis of allometry. In their work, they found that skinfold thickness increased faster in relation to body size than would be expected on the assumption of geometric similarity. They therefore suggest that shorter men must, if heavier, increase disproportionately in abdominal girth, because there is less capacity in other body depots to store adipose tissue. They suggest a similar explanation for inverse associations between height and arm or thigh girths in women.

That allometry accounts for variability in adipose tissue deposition is undisputed. An elegant analysis by Pond and Ramsay (3) showed that the high amounts of body fat in polar bears simply continue allometric trends in fat distribution evident in carnivore species inhabiting tropical or temperate climates, and thus offered little support for the hypothesis that increased adiposity functions as insulation in this arctic species.

Nevertheless, an allometric explanation for our findings is oversimplistic and fails to acknowledge an increasing body of work relating growth patterns in early life to adult fat distribution. One limitation of allometric analyses is that they treat body size as a simple physical trait, whereas we argue that it is more appropriate to interpret size and shape as the consequences of growth strategy. If short men tend to have high central adiposity, we suggest these 2 characteristics are likely to have developed together from early life onward, whether under genetic or environmental influence. A developmental approach also offers a more powerful explanatory framework for sex differences in associations between body composition and stature, because fat distribution diverges most strongly between males and females during puberty.

In the large Avon Longitudinal Study of Parents and Children (ALSPAC) cohort, Ong et al (4) investigated early growth patterns and subsequent fat distribution at age 5 y. They identified a subset of children who had shown rapid growth (gaining $\geq 0.67$ wt $z$ scores) in the first 2 y of life. They showed that these fast-growing children had smaller mean birth size (weight, length, and ponderal index) and had indications of reduced fetal growth such as maternal smoking or primiparity. To some extent, these postnatal growth patterns reflect regression to the mean; however, they also generate long-term effects, as reflected by the variability in phenotype evident at age 5 y. At this age, the fast-growing infants had greater height, weight, body mass index, and waist girth. In this population of 5-y-olds, those born small and short therefore developed greater central fat through catch-up growth.

However, growth retardation at later ages may increase the predisposition to central adiposity without also inducing catch-up growth in height. Similar to the findings from Brazil (2), early stunting has been associated with a central fat distribution in Guatemalan adults (5) and Jamaican children (6), although not in children from South Africa (7). In Siberia, shorter Buryat women had a lower fasting fat oxidation rate than did taller women, were heavier and fatter, and had higher serum lipid concentrations (8). Associations between early growth retardation and fat distribution have been reported in numerous studies, as reviewed previously (9), and the fact that these associations often depend on adjustment for current size strongly implicates postnatal growth patterns as an important mediating factor.

The variability between studies is therefore likely to reflect different opportunities between populations in the potential to catch up after early growth retardation (reflecting variability in dietary quantity and quality) and variability in the period when growth retardation occurred. In the ALSPAC cohort, growth retardation occurred in fetal life, and substantial catch-up in height was possible in infancy. In the other populations, poor height growth continued into early childhood, and catch-up does not seem to have occurred. Whether variability in fat oxidation rate translates into variability in central adiposity may likewise depend on childhood or adult dietary composition.

Although existing evidence on the association of height and central adiposity is therefore inconsistent between populations, it is clear that both linear growth and fat distribution respond to early nutritional experience (9). We suggest that allometric explanations as suggested by Nevill et al ignore important issues that are relevant to understanding the etiology of obesity. Such an approach offers no explanation for why short men and women might differ in their regional fat distribution or why populations might show varying associations between stature and fat distribution, as discussed above. Growing and storing energy are best considered as strategies that have complex relations with life-course experience. It is precisely because adult fat distribution tends to be linked with early life experience that efforts to reduce the prevalence of obesity should further investigate this issue.

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Identifying dietary patterns by using reduced rank regression

Dear Sir:

In an article published in a recent issue of the Journal, Wosje et al (1) applied reduced rank regression (RRR) to identify dietary patterns related to fat mass and bone mass in young children. RRR is an emerging statistical method in nutritional epidemiology that identifies dietary patterns associated with selected response variables—i.e., nutrients or biomarkers—that have known relations with a disease outcome of interest (2–4). This has the advantage of building on a priori knowledge of biological relations, by including plausible intermediates between diet and the outcome of interest. However, one of the criticisms of RRR is that observed relations between the dietary patterns are forced to correlate with fat mass and bone mass later in life.

Wosje et al (1) have used their outcome measures, fat mass and bone mass, as the response variables in their RRR analysis and then used multivariable linear regression to evaluate the relations between their dietary patterns and their outcome measures. Accordingly, the dietary patterns are forced to correlate with fat mass and bone mass, undermining the inherent value of this approach. Instead, it would be more appropriate to select response variables hypothesized to be on the pathway between dietary intake and bone and fat mass—for example, nutrient intakes such as calcium, protein, or fat. This would give better insight into the role of dietary factors in shaping the changes in body composition during growth and development.

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REFERENCES