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 >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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Jonathan CK Wells
Childhood Nutrition Research Centre
UCL Institute of Child Health
30 Guilford Street
London WC1N 1EH
United Kingdom
E-mail: j.wells@ich.ucl.ac.uk

Philip Treleaven
Department of Computer Science
University College London
New Engineering Building
Malet Place
London WC1E 7JE
United Kingdom

Tim J Cole
MRC Centre of Epidemiology for Child Health
UCL Institute of Child Health
London WC1N 1EH
United Kingdom
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—ie, nutrients or biomarkers—that have known relations with a disease outcome of interest (2–4). This has the advantage of building on nutrients or biomarkers—that have known relations with a disease outcome of interest (2–4). This has the advantage of building on
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Gina L Ambrosini  
David J Johns  
Susan A Jebb  
Medical Research Council Human Nutrition Research  
120 Fulbourn Road  
Cambridge CB1 9NL  
United Kingdom  
E-mail: gina.ambrosini@mrc-hnr.cam.ac.uk

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**LETTERS TO THE EDITOR**

**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—ie, nutrients or biomarkers—that have known relations with a disease outcome of interest (2–4). This has the advantage of building on

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5. Tucker KL. Dietary patterns, approaches, and multicultural perspective.


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**Reply to GL Ambrosini et al**

Dear Sir:

We appreciate the comments of Ambrosini et al regarding our use of reduced rank regression (RRR) to explore dietary patterns associated with fat and bone mass in young children (1). RRR is not strictly an a priori approach nor a purely exploratory approach, as indicated in the RRR methods article by Hoffmann et al (2) and as addressed in their subsequent response to a letter to the editor (3). In our analyses, RRR was used to intentionally identify dietary pat-
terns correlated with fat and bone mass. We could then control for
other factors expected to be related to fat and bone mass. The expectation that both dietary intake and the selected covariates
would be related to fat and bone was based on a priori knowledge.
Our intention was to determine linear functions of the food groups
(predictors) that maximally explained the variation in fat and bone
mass (outcomes). Importantly, our approach implies that high fat
mass and low bone mass serve as markers for similar high fat
mass and low bone mass later in life.

Finding single macronutrient, micronutrient, and vitamin compo-
nents of the food groups that influence the outcomes was not our goal,
because much is already known about such relations. We wanted to
determine what young children are actually eating to gain insight on
potential target foods. For example, let us assume that protein intake
is related to body mass accrual in children. A primary aim was to find
a practical approach for encouraging children to consume foods con-
taining protein. Unfortunately, lean protein foods were not com-
monly consumed to the degree that they were a significant factor
in predicting fat mass and bone mass in our cohort. The fact that di-
etary patterns are based only on foods that were actually consumed,
not necessarily ideal foods, might be considered a limitation of the
RRR method. However, the strength of RRR is that it allows for iden-
tification of practical approaches to promote low fat mass and high
bone mass accrual. Our finding that processed meats (although not
historically condoned) are a significant protein source suggests that
such foods are a real factor to consider when addressing children’s

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