Reply to W Koo

Dear Sir:

We thank Koo for his comments on our recent article (1) and the opportunity to clarify and expand on certain points. First, we need to make clear that the reported study involved a subset of a calcium intervention study in The Gambia, as yet unpublished, and was not part of the recent World Health Organization (WHO) randomized trial of 8325 pregnant women with low calcium intakes (2).

We agree with Koo that the study was limited by the instrumentation available and by the relatively small number of measurements made with dual-energy X-ray absorptiometry (DXA). We drew attention to this in the methods and discussion sections of our article. However, the observed lack of a significant effect of calcium supplementation during pregnancy on infant length and radial bone mineral content (RadBMC) in all subjects supports the finding of no significant difference in whole-body BMC (WBBMC) between the intervention groups in the subset. The number of infants with DXA measurements at each age (=50) was similar to that in the American study, in which a significant effect of maternal calcium supplementation on infant WBBMC was noted in the subgroup of mothers with a low calcium intake (3). This finding suggests that the sample size was adequate in the Gambian study to detect an equivalent effect of the supplement, if it were present. In addition, in the Gambian study, the tendency toward a lower WBBMC at each age in the infants of supplemented mothers was in the opposite direction of that originally hypothesized, and it is unlikely that, had DXA measurements been made in all infants, it would have materially altered the overall conclusion of the study, ie, that there had been no positive benefit of the supplement on infant bone growth and mineral accretion.

Koo discusses the point that RadBMC increases at a different rate than that of WBBMC during early life; thus, the ratio between them is influenced by gestational and postnatal age. This should not have affected the evaluation of the effect of maternal calcium supplementation on bone mineral accretion, given that the infants in the 2 intervention groups did not differ significantly in gestational age, that the infants were measured at the same postnatal ages, and that the effects on RadBMC and WBBMC were evaluated separately. Similarly, although the DXA software was designed for use in infants weighing >5 kg and tissue depth may have affected bone edge detection and hence absolute DXA values in lighter infants, no significant differences in the weights of the infants in the 2 intervention groups were observed at any age. Thus, potential artifacts caused by the small size of the Gambian infants were unlikely to have affected the between-group comparisons. Furthermore, at 52 wk, the infants had weights within the recommended range, and the DXA data were internally consistent across infancy; very strong intragroup and intradividual correlations were observed (1). This suggests that major problems with the measurements at weights <5 kg that would have obscured a supplement effect at 2 and 13 wk were unlikely.

We stand by our comments about DXA scanning in infants. Although we recognize the valuable studies that Koo and others have conducted to validate the accuracy and precision of DXA instruments and software for use in infants, there continues to be a lack of comparability between systems. In addition, the assessment of accuracy involves comparisons of WBBMC measured by DXA with carcass ash weight in animal models and provides no information about the accuracy of bone size measurements. Comparisons between studies using different instruments are therefore problematic, and it is particularly difficult to draw conclusions about whole-body bone area, and hence bone mineral density (bone mineral content × bone area), as we discussed in our article. In our Table 5 we draw attention to this problem by referencing published data derived by using a variety of different instruments. We acknowledge that Table 5 is not a comprehensive listing of all DXA studies in infants, but, by providing a summary of published DXA data for infants aged <3 wk, we sought to provide comparative data for the Gambian infants at this age while illustrating the difficulties of comparing bone area and bone mineral density obtained with different systems.

Finally, Koo makes an important point about the possibility that an inadequate supply of energy and other nutrients may have limited the beneficial effects of the extra calcium on fetal and infant bone growth and mineralization. The nutritional status of this rural Gambian population has been characterized in many previous studies, and it is well known that energy restriction is common among pregnant and lactating women (4) and children (5), although adult protein intakes tend to be in line with WHO recommendations (6). The pregnancy weight gains recorded for the mothers in the supplement and placebo groups (1), their protein intakes (supplement = 50 g/d, placebo = 60 g/d; P = 0.1; 7), and the growth rates of their infants (1) suggest that this was also the case in our study but that the 2 intervention groups were well-matched for maternal and infant nutritional status.

None of the authors had a conflict of interest.

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Energy intake or energy expenditure?

Dear Sir,

Swinburn et al (1) recently published in the Journal the prediction equation they developed for body weight in children by using cross-sectional data for total energy expenditure (TEE), height, age, and sex. This approach has a high value in predicting mean weight changes in a population as an outcome in response to interventions targeting total energy intake (TEI) or TEE. Swinburn et al assumed that the population was in energy balance, and therefore TEE = TEI = energy flux (EnFlux). They argue that a higher TEI, rather than low physical activity and a low TEE, was the main determinant of high body weight in children. This argument was基于 the positive relation of EnFlux with weight after adjustment for height, age, and sex.

We are not sure whether this conclusion is justified. At a positive energy balance, weight gain has a positive relation with TEI and a negative relation with TEE. However, at a zero energy balance, TEI resembles TEE, and energy intake and energy expenditure above the basal requirement (ie, resting energy expenditure; REE) correspond to each other. The variance in body weight should thus be measured by using REE, which is mainly a function of fat-free mass and age.

Using data for 103 children and adolescents (49 boys and 54 girls aged 4–18 y) and 253 adults (94 men and 159 women aged 28–84 y), we predicted lnWeight from lnREE, height, sex, and age:

\[
\ln \text{Weight} = 0.63(\ln \text{REE}) + 0.017(\text{height})
+ 0.11(\text{sex}) - 4.232 (1)
\]

in children, and

\[
\ln \text{Weight} = 0.79(\ln \text{REE}) - 0.001(\text{age}) (2)
\]

in adults.

In children, the model explained 85% of the variance in lnWeight (SEE = 0.15 kg) and is thus similar to the 86% variance in lnWeight explained by lnTEE, height, age, and sex that was obtained by Swinburn et al (1). The mass of a body, in both children and adults, can thus be described by its basal energy expenditure. The independent contribution of height and sex to the mathematical model for lnWeight prediction in children is explained by a child’s maturity status, which influences the relation between REE and body mass (ie, a higher REE/body mass in younger children). Standardized coefficients (0.71 for height, 0.32 for lnREE, and 0.14 for sex) showed that height was the main predictor of lnWeight in our group of children.

Because lean persons are likely to have a higher TEE per kg body weight (because of a higher REE that results from a higher fat-free mass), the slope of the regression line between TEE and body weight should be steeper in overweight than in normal-weight children (Figure 1). However, under the condition of energy balance, both TEI and TEE should be positively related to body weight in both groups. Alternatively, in obese children, the regression line between weight and TEE may be flatter because a higher specific REE has been found in obese children (2) as well as in obese adults (3–5), possibly as a result of obesity-associated metabolic disturbances such as insulin resistance and elevated sympathetic tone.

Neither of the authors had a personal or financial conflict of interest with respect to the study by Swinburn et al.

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Reply to A Bosy-Westphal and MJ Müller

We thank Bosy-Westphal and Müller for their commentary on our prediction equation for estimating the body weight change in children in response to energy imbalance (1) and for providing additional information on the issue. They have found, as we did, a positive relation between energy expenditure and body weight, although they have used resting energy expenditure (REE) rather than total

![Figure 1. Hypothetical relations between body weight and total energy expenditure (TEE) in lean and overweight children.](Image)