**Invited Commentary**

**Shorter adults, yet taller children: what’s up?**

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We know that obesity tracks steadily from childhood into young adulthood, and then onward into middle age. We know that tall children tend to become tall adults. Yet, the study in this issue of the *British Journal of Nutrition* by Bosy-Westphal et al. (1) finds that heavier children are taller than average and heavier adults are shorter than average. What’s up with that?

Although obesity is truly excessive body fat in relation to lean mass, the definition of ‘excessive’ has not been standardized. Given the difficulty of accurately measuring body fatness, the assessment and treatment of obesity are most commonly based on the measure of BMI, defined as weight (kg) divided by the height (m²). This formula, originally known as the Quetelet index, was described in 1832 by Adolphe Quetelet, a Belgian statistician. Quetelet noted that beyond the first few years of life, the weights of individuals increase proportional to the square of their heights. While critics accurately point out the failure of BMI to correlate with body fatness in certain individuals (for example, in very muscular athletes), it has proved generally accurate as a measure for the study of populations.

A tenet of both the Quetelet index and our sense of fairness is that obesity is independent of height. With the exception of environments where nutrition is inadequate, it is felt that height is genetically determined, and an individual’s weight is due to a combination of their height and behaviours determining energy balance (energy-in minus energy-out). Obesity, it is believed, is an equal opportunity condition. However, some past research, confirmed by this large cross-sectional analysis by Bosy-Westphal et al. (1), has questioned this assumption.

Using cross-sectional data from a variety of weight-management facilities (183 176 women and 30 628 men) and the Kiel Obesity Prevention Study (6240 girls and 6171 boys), Bosy-Westphal et al. (1) assessed the relationship between obesity and height in adults and in children. For the adults, they found a higher prevalence of obesity among those who were shorter. This observation has been noted in a few past studies, with the association strongest when evaluating obesity-related cardiovascular risk factors and the shortness of leg length specifically (rather than total length)(2–6). However, most studies on obesity have not commented on any association between adult height and adult obesity, perhaps assuming that no relationship exists.

If we are to combat the adult obesity epidemic, it is imperative to decrease the rate of childhood obesity. Childhood BMI has a strong correlation with adult BMI. Although we may have a mental image of the short and overweight child, in fact, studies from the 1930s began documenting that obese children are taller than their peers(7,8). More recent investigations, and the childhood cohort studied by Bosy-Westphal et al. (1), confirm this association(9–12).

In summary, using very large cohorts, Bosy-Westphal et al. (1) have provided us with an analysis documenting the height–obesity relationship of two generations, and left us with a conundrum. Since BMI in childhood is directly correlated with BMI in adulthood, and taller children tend to be taller adults, the positive association of height and obesity in children seems to contradict the inverse association of height and obesity in adults. Why? Some of the answer appears to be that the excess energy intake that promotes obesity in children also speeds up the body maturation process. An overweight 8-year-old child may have the anatomy and physiology of a normal-weight 10-year-old child. This concept should not be used as an excuse for the child’s weight by the argument that they are not overweight, but just measured at the wrong age cut-off. In fact, it seems that these tall and overweight children have an even higher risk of becoming overweight young adults compared with their overweight peers who are shorter(12,13). Using BMI as a continuous variable, for younger children (aged 2–8 years) childhood height may have an independent effect (i.e. even after adjusting for childhood BMI) on adult BMI(13). Thus, increased height in children may be a surrogate marker for higher risk of obesity in later life. On the surface, this may suggest that these overweight and tall children become overweight and short adults. The authors pose a possible explanation for this whereby the ‘preterm skeletal maturity in obese children may limit [subsequent linear] growth and final height in adults’. As support, they cite articles noting that ‘the countries with the tallest adults have the lowest rates of obesity’(14,15).

However, beware of conclusions regarding longitudinal associations derived from ecological cross-sectional studies done simultaneously on different generations! The adults in the study by Bosy-Westphal et al. (1) had a mean age of approximately 43 years and were assessed between the years of 1996 and 2006. Thus, many grew up before the childhood obesity epidemic. Additionally, the study found an inverse relationship between age and height (with a range from the shortest to the tallest of approximately 15 years). Since individuals tend to gain weight with age, the inverse association of height and BMI in the adults may be a function of the older subjects being heavier. Given the recent gains in adult height (especially in Europe), these older subjects may also have been shorter due to generational, and not individual reasons.
Since the evidence seems overwhelming that obese children are taller than their non-obese peers, what other possibilities might explain the authors’ findings that the obese adults were shorter than average? The authors offer one very interesting possibility: food serving sizes. Serving sizes of foods bought at a market or a restaurant are similar for tall and short individuals. Tall individuals have higher BMR of energy expenditure and thus can better tolerate larger intakes. Since many individuals consume food based on visual cues, it is reasonable that shorter individuals would take in relatively more energy than their body needs. Put another way, the very large serving sizes offered in many countries may be adversely affecting short individuals more than tall individuals.

Another possible explanation for obesity having different associations with height in the adult and child cohorts may have been due to recruitment issues. The children came from a diverse sample of the population. However, the adults came from weight-loss programmes. Since cultural issues of body morphology may be a driving force for someone to enter a weight-loss programme, heavy individuals who are also short may have been oversampled.

Still, this study and others evaluating the relationship of obesity with stature leave us with some fascinating issues for research.

First and foremost, more longitudinal studies are needed to assess the tracking of both BMI and height from pre-pubertal children into adulthood. This is especially important to follow children who grew up in the recent era of the childhood obesity epidemic.

Until longitudinal studies are available, obesity researchers need to include analyses that are stratified by height, even if the findings are null. If adult obesity is consistently associated with shortness, and if serving sizes are a potential explanation, then perhaps this relationship would be stronger in more recent cohorts as compared with cohorts from the pre-’super-size me’ days.

We should seek to find any easily measured markers that could distinguish the child who is tall because he/she is destined to be tall from the one who is tall from excessive energy intake.

Even with these research questions outstanding, some clinical changes can be justified.

Do not use advanced childhood height as an excuse for excessive childhood weight. When evaluating a child who is overweight and also tall, one may hope that the child’s weight growth will slow, that his/her height growth pattern will continue, and his/her BMI will normalise. From this perspective, tallness may be interpreted as protective against shortness, and if serving sizes are a potential explanation, then perhaps this relationship would be stronger in more recent cohorts as compared with cohorts from the pre-’super-size me’ days.

We should seek to find any easily measured markers that could distinguish the child who is tall because he/she is destined to be tall from the one who is tall from excessive energy intake.

Follow childhood growth closely beyond the early immunisation years. The medical profession is very good at following children through their first few years of life. However, we miss the changes in adolescence, which is where the action seems to lie for the issue of skeletal growth patterns. We must pay special attention to the height trajectories of overweight children, many of whom enter puberty taller than their peers.

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