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Combined stunting and overweight in young children – a paradox?

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The paper by Mamabolo and co-workers1 in this issue of the *Journal* raises some important questions around the pathogenesis and significance of stunting and apparent obesity in young children in rural South Africa, and the long-term consequences related to these. It has been established clearly in a number of studies conducted in South Africa (including the current one) that stunting is the major clinical form of undernutrition present in children in many parts of the country, with nearly 20% being stunted by 2 years of age. The current study found a very high prevalence of stunting (48%) at 3 years of age, rising from 35% at 1 year, in a longitudinal sample of children living in villages in the central region of Limpopo province.2 This compares with a figure of 16% stunting at 2 years of age in children from the Birth to Twenty cohort in Johannesburg.3 The possibly more surprising finding in the Limpopo children was the high prevalence of overweight and obesity in the stunted children at 3 years of age, which depending on the methods and cutoffs used (either body mass index (BMI) or weight-for-height z-score (WHZ)) ranged from 31% to 46%, despite the high prevalence of stunting and therefore presumed chronic undernutrition. Furthermore some 19% of the total cohort were both stunted and overweight and 40% of the overweight children were stunted. In this study children at 3 years of age were six times more likely to be overweight if they had a low WHZ score at birth. Unlike the Limpopo study, although stunting at 2 years of age in the Birth to Twenty cohort was a predictor of higher BMI, it did not predict subcutaneous fat values or fat distribution.3 Because of the lack of higher subcutaneous fat values or greater centripetal fat ratios, it was suggested that the higher BMI values were spurious and not a reflection of increased overweight or obesity, but rather due to the effect of a reduction in height on the BMI as the denominator is height squared – any reduction in height therefore dramatically increases the BMI. Support for this conclusion was also provided by finding a difference in the relationship between height and BMI in the nonstunted and stunted 2-year-old children; in the former group there was no relationship between height and BMI, while in the stunted group there was a significant inverse relationship between the two.3

The above discussion raises issues about the best method to categorise and define overweight and obesity in stunted young children. Despite this, however, it will be both interesting and important to follow the Limpopo cohort through childhood and adolescence as there is considerable concern that low birth weight and stunting in young children predisposes them to obesity and the metabolic syndrome in later life.4 Nevertheless, in the Johannesburg Birth to Twenty cohort we have been unable to find evidence that stunting by itself in early life predisposes to obesity during the prepubertal years, as children who were stunted at 2 years of age remained at 9 years shorter and lighter with similar percentage lean and fat masses as the non-stunted children.3 These latter findings are supported by data from a longitudinal study conducted in Jamaica, which did not show a relationship between early stunting and later obesity in prepubertal children.5

However, it appears that early rapid postnatal weight gain (within the first 2 years of life) is important in predisposing children and adults to central adiposity, insulin resistance and the

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metabolic syndrome.6 One study from the UK found an association between insulin resistance by 8 years of age and early rapid weight gain.7 It is therefore of great concern that 84% of the young children in the Limpopo study had shown catch-up weight gain (a gain of > 0.67 SD score in weight for height was used in this study, rather than the usual > 0.67 SD score in weight for age) by 3 years of age.2 In a review of the subsequent risk for obesity in infants with rapid weight gain, Ong and Loos8 suggest that the greatest risk is for those infants who gain rapidly in the first week of life. Further, the risk increases by 60% if the duration of rapid weight gain is increased from 1 to 2 years. It is interesting to note that the influence of rapid weight gain in infancy on later obesity risk is not related to birth weight, the effect being similar in both average and low-birth-weight infants. The odds ratio associated with rapid infant weight gain for developing obesity in later life is estimated to be between 2 and 3 in most studies.8 We have assessed the relationship between early rapid weight gain and obesity and skeletal maturity during childhood in the Birth to Twenty cohort.9 Some 20% of the cohort with normal birth weights could be defined as having rapid weight gain during the first 2 years of life. At 9 years of age, these children were 3.8 cm taller and 3.8 kg heavier than their peers and had 3.7% greater body fat. After controlling of differences in BMI between the two groups, the rapid weight gain children still had significantly greater fat and lean tissue mass.

Mamabolo *et al.*1 suggest that the high prevalence of stunting in association with overweight and obesity in the cohort of Limpopo children at 3 years of age might be due to the poor quality of the diet the children were consuming. The diet was typically low in animal protein, high in carbohydrate, low in fat and micronutrient deficient. The authors hypothesise that the lack of animal protein inhibited linear growth, while the high carbohydrate content of the diet allowed for fat deposition and obesity. This is an interesting hypothesis, which needs to be tested further in animal and human studies; however, it might be possible to get supportive evidence for the hypothesis if further analyses of the current data set were done. For example, is there any association between the highest and lowest protein intakes and the prevalence of overweight/obesity?

The prevalence of obesity and the metabolic syndrome is likely to continue to rise in South Africa as lifestyle and dietary patterns change, so considerable effort must be made to understand the pathogenesis of these conditions if we are to develop appropriate preventive strategies. The current longitudinal cohort in Limpopo province is an important asset and should be maintained throughout childhood, adolescence and early adulthood, so that we can learn more about the outcomes and pathogenesis of these major chronic diseases of lifestyle in rural children living in very deprived circumstances.

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