Early nutrition and adult outcomes: pieces of the puzzle

The association between nutrition in early life and long-term health has been of interest for decades. Since the articulation of the fetal origins hypothesis by David Barker and colleagues,1 there has been debate about the implications of fetal undernutrition and early childhood growth on outcomes of importance in adult health and risks of chronic diseases. Both epidemiological and animal studies have shown that the risk of metabolic syndrome is significantly increased after exposure to suboptimum nutrition during crucial periods of development.1 The importance of these findings greatly increased after reports about the global burden of non-communicable diseases and risk factors were published in December, 2012.2

Evidence for the importance of early nutrition for adult outcomes was derived initially from observational cohort studies3 and was reaffirmed by analysis of outcome data from several cohort studies in 2008.4 This analysis4 was focused on a meta-analysis of coefficients from different sites: birthweight, weight and length Z scores, and stunting at age 2 years. In The Lancet, Linda Adair and colleagues5 report findings from a study in which they pooled data from five birth cohorts and investigated how linear growth and relative weight gain in several age ranges affected adult outcomes. They report that higher birthweight was associated with an adult body-mass index of greater than 25 kg/m² (odds ratio 1·28, 95% CI 1·21–1·35) and a reduced likelihood of short stature (0·49, 0·44–0·54) and of not completing secondary school (0·82, 0·78–0·87). Faster linear growth was also strongly associated with reduced likelihood of short adult stature (age 2 years: 0·23, 0·20–0·52; mid-childhood 0·39, 0·36–0·43) and of not completing secondary school (age 2 years: 0·74, 0·67–0·78; mid-childhood 0·87, 0·83–0·92). Faster relative weight gain was associated with an increased risk of adult overweight (age 2 years: 1·51, 1·43–1·60; mid-childhood 1·76, 1·69–1·91) and elevated blood pressure (age 2 years: 1·07, 1·01–1·13; mid-childhood: 1·22, 1·15–1·30).

Notwithstanding the key findings, several limitations of this pooled analysis should be recognised. The authors had to make do with disparate information about socioeconomic status and income, and impute some information that was missing. Although they adjusted for maternal education and socioeconomic status (largely assets rather than income), other potential confounding factors (eg, household and learning environment) could not be assessed in relation to attained schooling. Several additional limitations preclude firm conclusions. Little or no information was available about maternal nutrition and micronutrient status. Additionally, Adair and colleagues do not report any outcomes related to intrauterine growth retardation or gestational age at birth, and merely report association with birthweight, which might be oversimplified. Being small for gestational age at term, and especially preterm, has now been recognised as a major risk factor for excess newborn and infant mortality6 and accounts for a substantial proportion of child stunting (unpublished). Prematurity is associated with increased risks of metabolic syndromes in later life.7 Potential variations in body composition of newborn babies might not be captured by mere measurement of birthweight or size. So-called thin-fat infants—ie, small newborn babies that have elevated body fat content—have been described8 and could be associated with increased risks of insulin resistance in childhood.9 These limitations aside, Adair and colleagues’ findings5 are some of the most important from existing cohorts linking early childhood nutrition—especially birthweight and improved patterns of linear growth—with long-term outcomes. They have clear implications...
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for public health policy and nutrition interventions. As shown by an analysis of evidence-based interventions, a focus on improvements in nutrition in pregnancy and linear growth in the first 2 years of life could lead to substantial reductions in stunting and improved survival. These improvements form the basis for the emphasis on the first 1000 days of life, which has been used effectively to scale up nutrition activities.

However, this tenet could be too simplistic, because it focuses on care during pregnancy and ignores the vital contribution of maternal health and nutrition in the periods before and just after conception to intrauterine and postnatal growth. Evidence supports an association between micronutrient supplementation around the time of conception and DNA methylation and increased methylation of the IGF2 genes in childhood, indicating that these factors could affect linear growth postnatally. Although Adair and colleagues’ analysis of birth outcomes in the international cohorts does not shed light on the importance of maternal health and nutrition before conception, these factors might be just as important as postnatal factors and should be investigated.

What is the way forward? Although the evidence emerging from observational studies such as Adair and colleagues’ is important for policy, well designed prospective studies with appropriate interventions and follow-up are clearly needed. The outcomes should include elements of child development, education, employment, and earnings, which would allow improved estimation of effect on human capital. Although expensive and difficult to organise and implement, such cohort studies are a crucial investment for the future and, in view of the interest in human development in the post-2015 era, should be prioritised for funding.

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I declare that I have no conflicts of interest.