Commentary: Early ‘catch-up’ growth is good for later health

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The importance of events before birth for lifetime health has been observed and confirmed in many populations. In the past when infectious diseases were even more common than today it was self-evident that non-optimal early growth affected health later in life. Recent findings point towards the importance of events during critical periods of growth and development in the pathogenesis of many non-communicable diseases, e.g. coronary heart disease (CHD) and type 2 diabetes. It is now well established that the development of a fetus in an abnormal intrauterine environment implies structural and functional adaptations with long-lasting consequences for the metabolism of that offspring in later life. These consequences are thought to be caused by biological programming.

The original ‘fetal origins of adult disease hypothesis’ postulates that impaired fetal growth may predispose individuals to heart disease in later life. In the original observations from Hertfordshire, UK, death from ischaemic heart disease was more common in men who had been small at birth and who were small at one year of age.
Individuals exposed to undernutrition in utero seem to be more susceptible to CHD and type 2 diabetes if they ‘catch-up’ in weight and body mass index during childhood. This means that the risk associated with small size at birth is modified by later growth.5–8 Those having the highest risk for CHD and type 2 diabetes are those who were small at birth but changed ‘channels of growth’ during childhood.

The paper by Victora and coworkers in this issue of *International Journal of Epidemiology* focuses on the interesting and controversial role of ‘catch-up’ growth with regard to long-term health outcomes in individuals born small.10 In the study by Victora et al. there are obvious short-term advantages of ‘catch-up’ growth among small-for-gestational-age (SGA) infants. There was less hospitalization for SGA children with fast growth than for those with intermediate or slow growth during infancy. This observation is of extreme importance since malnutrition in early life is a widespread health problem and promoting weight gain in infancy is standard medical practice. The impact of the problem is easy to understand knowing that approximately one-third of the world’s children suffer from protein-energy malnutrition.

The early patterns of growth that predispose to adult diseases are complex. Previous studies have shown that ‘catch-up’ growth might well have detrimental long-term consequences. The reason for this is however not known. Those studies showing a negative effect of rapid childhood growth have focused mainly on growth from 7 years onwards.5–7

The importance of distinguishing between early and late ‘catch-up’ growth is nicely stressed by Victora et al.10 Early ‘catch-up’ growth appears to be beneficial based upon the Brazilian study. In line with this, rapid weight gain in infancy reduced later CHD risk among Finnish men thus supporting the notion of long-term positive health benefits of early ‘catch-up’ growth.8

Many previous studies have not been able to distinguish between early and late ‘catch-up’ in growth—probably the main underlying cause of the controversy regarding long-term effects of ‘catch-up’ growth. The most unfavourable growth pattern seems to be small body size or thinness at birth, continued slow growth in early childhood/infancy and thereafter acceleration in growth. The present findings add to the evidence that protection of fetal and infant growth is a key area in strategies for the prevention of many non-communicable adult diseases.

It is easy to agree with the authors that early catch-up growth is beneficial but the other side of the coin is obesity in childhood and later life.10 Those most vulnerable seem to be those with fast growth in childhood. Further health benefits will therefore come from preventing rapid increase in weight after infancy.

The thrifty phenotype hypothesis suggests that the fetal nutritional environment has a programming effect on such things as glucose and lipid metabolism and blood pressure and consequently health in adult life.11 The mismatch between the relatively poor intrauterine environment and a nutritionally rich environment in later life is supposed to increase the risk of type 2 diabetes and many other related non-communicable diseases. Adaptation to undernutrition in utero may limit the extent of dietary change to which a generation can be exposed without adverse effects.

However, in most cases adult non-communicable diseases are not programmed per se but the tendency towards disease is programmed. Therefore it is important to consider impaired early growth as one risk factor for adult disease—not as a causative factor. These early risk factors are to a large degree modified by both biological and social factors during childhood and adult life.

If fetal and maternal nutrition are important determinants of future disease this area has major implications in the prevention of non-communicable diseases. Presently we do not know what the effects would be of providing adequate nutrition to pregnant women. Only future research will tell us whether improving the body compositions and diets of young women is to be one of the strategies for preventing type 2 diabetes and closely related non-communicable diseases. This is a very complex area and one must always bear in mind that fetal growth is also regulated by hormones, growth factors, and placental function—not only by availability of food—and this again introduces an array of other factors responsible for fetal growth.

However, lifestyle from the cradle to the grave matters. It has recently been shown that a lifestyle intervention programme (diet and exercise intervention) among adults with impaired glucose tolerance reduced the 6-year cumulative incidence of type 2 diabetes by 58%.12 The public health implications of these results are wide.

References