The Global Epidemic of Noncommunicable Disease: The Role of Early-Life Factors

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Although meeting the Millennium Development Goals and the prevention of undernutrition remains a major problem in many populations, the rapid increase in noncommunicable disease (NCD) has now become the highest priority for global public health. According to the WHO, in 2008 approximately 63% of all deaths (36 out of 57 million per year) were due to NCDs, comprising cardiovascular disease (CVD), diabetes, cancers and chronic respiratory disease [1]. Of these, nearly 80% (29 million) occurred in low- or middle-income countries, with CVD being the most common. In fact, contrary to popular belief, most deaths from CVD (80%) occur in low- or middle-income countries rather than in richer populations [1].

Whilst modifiable behavioral factors such as tobacco use, insufficient physical activity, the harmful use of alcohol, and unhealthy diets are the major risk factors, research over the last 10 years has highlighted the key role of early life factors in the development of NCD [1, 2]. Factors in utero and early postnatal life such as breastfeeding [3] have been shown to affect the risk of CVD by influencing the propensity to obesity, diabetes, hypertension and dyslipidemia – the so-called developmental origins of adult disease hypothesis. Many of these factors, such as infant nutrition, are modifiable, raising the possibility that interventions in early life could help stem the current global epidemic of NCD. For instance, epidemiological data linking low birthweight and prenatal nutrition (e.g. famine during pregnancy, antenatal protein and energy intake, and maternal micronutrient supplementation) with later obesity and CVD suggest that nutrition during pregnancy is a modifiable risk factor for NCDs [2, 3]. However, currently there is little experimental evidence to suggest that modifying prenatal nutrition can affect long-term health.

In contrast, the concept of early postnatal nutrition, and particularly slower infant weight gain (the growth acceleration hypothesis [4]), is
strongly supported by data from experimental studies that can establish a causal link between infant nutrition and future health. For example, there are now 5 randomized, double-blind, controlled trials which show that a higher plane of nutrition, which promotes faster weight and length gain in the first year of life, predisposes to later risk factors for CVD such as obesity and hypertension. Along with many observational studies from low-, middle- and high-income countries, these adverse effects of faster infant growth have been seen in trials in infants born preterm, and in both healthy and low-birthweight infants born at term [4]. The size of these effects, although smaller in cohorts from low- and middle-income countries, is substantial in a Western environment, where, for example, approximately 20% of the population risk of overweight in childhood can be attributed being in the highest quintile for weight gain in infancy [as reviewed in 4]. The mechanisms involved are poorly understood, but include epigenetic changes and resetting of endocrine systems that affect energy metabolism and appetite. These early life factors may interact with and exacerbate the detrimental effects of a sedentary lifestyle and energy-dense diets later in life. As a consequence, the impact of early life factors on long-term health may be particularly important in low- and middle-income countries, which face the fastest increase in urbanization and greatest changes to lifestyle. Strategies to optimize infant nutrition/growth in these countries could therefore help stem the global epidemic of NCDs [5].

References