

# Early nutritional influences on obesity, diabetes and cardiovascular disease risk. International Workshop, Université de Montréal, June 6–9, 2004

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### Importance of the issue

The burden of chronic diseases is rapidly increasing globally, owing in particular to cardiovascular disease, obesity and diabetes. In developing countries and countries in transition, non-communicable diseases have rapidly become an epidemic. Adult chronic disease 'reflects cumulative differential lifetime exposures to damaging physical and social environments' (Darnton-Hill *et al.*, 2003). Not all individuals are equally susceptible to non-communicable chronic diseases, and nutrition is a major underlying factor. The more susceptible are the poor, the uneducated, and the minority or ethnic groups in industrialized countries.

There is increasing evidence of an inverse association of fetal growth and nutrition with later chronic disease. In contrast, much less is known of the long-term impact of early infancy growth and nutrition, because birthweight has tended to be the focus of research on early origins of chronic disease. Prenatal and post-natal influences may interact, and indeed they need to be considered jointly, rather than in isolation. The theory of the early origins of chronic disease and the twin theory of early programming by nutrition have important public health implications as they represent potentially modifiable risk factors, in addition to, or in interaction with, other environmental and genetic risk factors.

Data from developed and developing countries suggest that not only small size at birth, but also post-natal growth retardation and rapid catch-up growth in infancy or childhood, are independent factors of increased risk of adulthood obesity, diabetes, hypertension and cardiovascular disease. This is of concern for developing countries, because 17% of the neonates born in developing countries have a low birthweight, that is, nearly three times as many as in developed countries (Bale *et al.*, 2003). Additionally, individuals who were born small and who grow rapidly during childhood are the most at risk. This is a growth pattern that is becoming more common in developing country populations with the nutrition transition that is underway, particularly in urban areas. This is another matter for concern, as suboptimal fetal and postnatal growth and nutrition may exacerbate the chronic disease risks associated with diet and lifestyle changes typical of nutrition transition. Hence, developing countries may be particularly vulnerable to nutrition transition because of recent or concurrent undernutrition, as it may compound the chronic disease risk associated with higher fat diets, lack of physical activity, and other lifestyle patterns such as tobacco smoking. In other words, as is so well put by Adair & Prentice (2004), prenatal factors contribute to a phenotype that may be more sensitive to lifestyle factors associated with the development of obesity and the chronic diseases. However, more than one growth phenotype may show an increased risk of chronic disease. Both thin children and obese children may be at higher risk of central obesity, insulin resistance and diabetes in adulthood. Different paths or growth trajectories need to be considered. The long-term impact of early growth, independent of or in conjunction with the feeding mode, may not be the same according to nutritional and growth status at birth.

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## Objectives of the workshop

The purpose of the workshop was to appraise and share the current knowledge on the links between nutrition in early life and subsequent chronic disease risk and more specifically, on the impact of growth, feeding mode and diet in infancy. The objectives were: (1) to assess the existing evidence for long-term effects of the early nutritional environment of the child on chronic disease risk; (2) to examine the implications of the existing evidence for public health/nutrition policy and programmes for the prevention of chronic disease in developing countries undergoing rapid nutrition transition; and (3) to determine priority research needs to better understand the impact of early growth and nutrition on later disease.

Specific questions that the workshop was to address were the following:

- What is the scientific evidence for an impact of growth and nutrition in infancy on chronic disease risk, independent from, or in interaction with fetal growth?
- Are breastfeeding and other infant feeding practices associated with chronic disease risks in later life?
- For children born small, is the target of maximum child growth still relevant, and if so, for what age range and what type of catch-up growth?
- Are there evidence-based policy and programme options to address the double burden of obesity and undernutrition in countries undergoing rapid nutrition transition?

Six themes were successively developed over the 2-day workshop:

1. Early nutritional influences in perspective;
2. Growth in infancy and childhood and chronic diseases;
3. Early diet and chronic disease risk;
4. Early nutritional influences in a nutrition transition context;
5. Animal models of nutritional programming; and
6. Implications for research, policies and programmes.

Following the individual articles, a concluding paper summarizes the evidence and unanswered questions, and the implications for further research and for policy.

## References

- Adair L.S. & Prentice A.M. (2004) A critical evaluation of the fetal origins hypothesis and its implications for developing countries. *Journal of Nutrition*, **134**, 191–193.
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- Darnton-Hill I., Nishida C. & James W.P.T. (2003) A life course approach to diet, nutrition and the prevention of chronic diseases. *Public Health Nutrition*, **71** (1A), 101–121.