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Lifelong consequences of poor fetal growth

Susan M Sayers and Gurmeet R Singh

Adaptive responses to a poor intrauterine environment may predispose to obesity and its related chronic diseases in a later, nutritionally enriched, environment

he global burden of death, disability and loss of human capital as a result of impaired fetal development is huge, and affects both developed and developing countries. The Indigenous people of Australia have high rates of low birthweight and chronic non-communicable diseases in adulthood, leading to premature adult mortality, with current life expectancies 17 years less than for other Australians. Improvements are occurring in Aboriginal health, but they are overshadowed by the continuing poor health profile of Aboriginal people. The article by Hoy and Nicol in this issue of the Journal (page 14) is noteworthy in that it highlights the decreases in neonatal and infant mortality in a remote Northern Territory Aboriginal community.

The relationship between birthweight and natural mortality described in the article by Hoy and Nicol³ adds to the body of work showing the influences of early life events on later health and disease. These reports first appeared over 70 years ago,⁴ but it was the influential work of Barker and colleagues from the University of Southampton that gave rise to a whole new paradigm. Now the discipline of the developmental origins of health and disease (DOHaD) is a rapidly growing research area in both basic and clinical sciences, which is supported by an international society and a recently launched journal.⁵

The initial studies showed inverse relationships between surrogates of fetal growth and central distribution of fat, insulin resistance, the metabolic syndrome, type 2 diabetes and ischaemic

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cardiovascular disease.⁶ These studies have been replicated worldwide and later research has focused on the mechanisms underlying these associations. Epigenetic changes in response to the early environmental conditions in fetal development and early infancy are the likely mechanisms; with the effects able to be transmitted to succeeding generations.⁷ In early life, there are critical times when environmental influences have their major effects, mediated through these epigenetic changes that later result in complex physiological mechanisms affecting final health outcomes.^{8,9}

The study by Hoy and Nicol reporting the association of low birthweight with increased mortality in young adult Australian Aboriginal people suggests that the current epidemic of chronic adult diseases seen in this population may be due to improved survival among low birthweight infants.³ A likely mechanism is that the adaptive responses to a poor intrauterine environment that were initially beneficial for fetal survival become detrimental in a later nutritionally enriched (and therefore mismatched) environment.⁸

While early life events set the risk for chronic disease, it is further exposure to multiple risk factors that translates this increased risk to overt chronic disease. The highest risk occurs when low birthweight is coupled with later obesity.^{8,10} This concurrence of low birthweight, infant undernutrition and adult obesity occurs in populations like the Australian Aboriginal population, that are undergoing rapid nutritional transition with the change of traditional diets to energy-enriched carbohydrate diets. This change in diet often occurs in conjunction with decreasing physical exercise.¹¹

A key element in any preventive strategy for chronic disease associated with premature adult mortality is the prevention of overweight and obesity. Obesity is not only a major risk factor on its own, but is an amplifier of other risk factors associated with chronic adult disease.

The findings of Hoy and Nicol suggest that improvements in birthweight will be mirrored by improvements in adult mortality in the Aboriginal population in the future.³ Interventions to prepare the intrauterine environment for the developing fetus need to begin before conception. Aboriginal low birthweight is associated with the known preventable factors of young maternal age, maternal undernutrition, smoking and alcohol consumption.¹² There are recommendations relating to preconception interventions to influence these factors in the United States population, but adaptations for Indigenous populations in general are yet to be determined.¹³ Future research should be directed towards developing and evaluating culturally specific adaptations of this early care for young Aboriginal women of childbearing age.

Debate continues about the optimum growth patterns for children in populations undergoing the nutritional transition. There are clear short-term survival benefits of increasing the speed of growth for low birthweight babies to prevent immediate morbidity and mortality. However, rapid weight gains during some periods of childhood have been shown to predict adult obesity. ¹⁴ The challenge is to balance the benefits of weight gain in early life with the risk of later chronic adult disease. ¹⁵

Currently, there is insufficient evidence to recommend the most favourable pattern of infant growth for preventing adult disease. Recent reports suggest that nutritional intervention with rapid changes of body mass index before the first 2–3 years of life are not predictive of adult obesity. However, considerable work is still needed before firm evidence-based recommendations can be made with confidence. In the meantime, staying with the well known

benefits of breastfeeding is probably the most prudent course of action. 19

Poor fetal growth has immediate and long-term consequences that encompass all aspects of health over the course of an individual's life. As the DOHaD concept and its mechanisms unfold, it is likely to have significant impacts on the direction of public health activities worldwide.

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