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School health and community nutrition

Early nutrition and risk of disease in the adult

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Abstract

The differentiation of key metabolic systems that occurs during intrauterine life is greatly influenced by environmental nutritional conditions, which in turn are related to maternal nutritional status. In postnatal life, childhood exposure to slow-acting environmental factors, primarily through the diet, will begin to condition adult susceptibility to diseases. Examples of these dietary factors are intake of saturated fat, sodium, calcium, etc. For example, bone calcium accumulation during childhood and adolescence will be a major determinant of risk of osteoporosis later in life. Similarly, a high intake of saturated fat during childhood may promote the process of atherosclerosis in persons with genetic susceptibility, thus accelerating the clinical manifestations of coronary heart disease in adult life.

These findings, although still not completely clarified, constitute a significant opportunity for preventive intervention. While preventive intervention in adult life may reduce risk, this is usually difficult and results are often limited. One example would be obesity. In contrast, interventions early in life, aimed at reducing these early risk factors, could potentially result in major reductions in the incidence of several diseases of adults.

Keywords
Children
Nutrition
Preventive interventions
Risk factors

Scientific research on the ageing process and its consequences to health has increased dramatically over the past decade, primarily due to evidence indicating that in the next few decades people above 60 years of age will comprise a major segment of the world population. Therefore, a better understanding of factors that may reduce the adverse effects of ageing on health is particularly important. Among these, diet is undoubtedly one of the most significant.

Early nutrition and adult health

It is well recognised that nutritional deficiencies early in life can have a direct and immediate impact on the health of the individual. Protein-energy malnutrition, and specific micronutrient deficiencies, if not corrected promptly, usually lead to growth retardation and structural and functional alterations that are frequently irreversible[1–3]. In this context, it is evident that nutrition during childhood can have a significant impact on the health of the adult. These effects are related to *impaired* structure, size and/or function that develops over variable periods of time, and may be expressed only after the child has reached adult age.

A different type of effect of early nutrition is one mediated not by obvious structural alterations but by subtle changes in the process of *differentiation*. This type of effect has been termed 'metabolic programming', and appears to result from the host response to the nutritional environment predominant at a specific stage of development[4,5]. By altering patterns of differentiation of tissue and metabolic pathways during foetal and early postnatal development, this process can increase (or reduce) the risk of disease that a given individual may exhibit later in life. For example, ambient glucose concentration during foetal development has an important effect on the differentiation of centres in the central nervous system (CNS) that control energy balance. A low glucose supply (for example, due to maternal undernutrition) enhances the development of energy-sparing metabolic pathways, thus maximising energy efficiency. This adaptation, although favourable for the prevailing conditions in foetal life, may increase the risk of excess energy accumulation (i.e. obesity), later in extrauterine life, if the individual is exposed to abundant dietary energy availability, as is common in developed societies. Similarly, a restricted oxygen supply to the foetus may increase placental vascularity, leading to an increased susceptibility to high blood pressure[6–8]. Indeed, several of these events have been experimentally demonstrated in animal models[9].

One of the early descriptive epidemiological studies that highlighted the association between foetal growth

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and adult disease was that of Barker[10]. Examining the medical records of thousands of adults in poor regions of the UK, he found, contrary to what was expected, a higher prevalence of cardiovascular disease, which is usually associated with affluence. Further analysis revealed a significant inverse correlation between birth weight and risk of cardiovascular disease; i.e. a lower birth weight increased the risk of cardiovascular disease in adulthood. Similar associations were subsequently described for diabetes, glucose intolerance, high blood pressure and pulmonary diseases[8,11,12].

The association between low foetal and postnatal growth and disease is not consistent for all chronic diseases, nor across all studies[13,14]. Adult adiposity and obesity appear less susceptible to early nutrition impairment, whereas high blood pressure and diabetes have been more consistently associated with low birth weight[4,15]. In populations with a high prevalence of childhood undernutrition, Schroeder *et al.* found a positive, not inverse, correlation between body weight in childhood and adult body mass index (BMI)[16]. Similarly, breast-feeding in healthy babies, although associated with lower growth rates relative to formula-fed infants, appears to protect against the development of excess adiposity later in life[17].

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