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Impact of Micronutrient Deficiencies on Growth: The Stunting Syndrome

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Key Words
Child · Nutrition · Growth · Stunting · Diet · Micronutrient deficiencies

Abstract
Stunting is a process that can affect the development of a child from the early stages of conception and until the third or fourth year of life, when the nutrition of the mother and the child are essential determinants of growth. Failure to meet micronutrient requirements, a challenging environment and the inadequate provision of care, are all factors responsible for this condition that affects almost 200 million children under 5 years of age. The timing and duration of the nutritional insult leads to different physiological consequences. Growth retardation is however just one feature of a complex syndrome including developmental delay, impaired immune function, reduced cognitive function and metabolic disturbances leading to increased prospective risk of obesity and hypertension. Prevention is possible by undertaking interventions at all stages of the life cycle, and mainly involves the promotion of exclusive breast-feeding until the age of 6 months and the provision of complementary foods and family foods with adequate micronutrient density. Treatment is possible, at least until the age of 5, and can lead to reversal of all the symptoms, although further research is required to clarify whether accelerating growth velocity might also lead to an increased risk of metabolic syndrome.

A Healthy Child

Achieving and maintaining optimal child health is a challenge for parents, caregivers, paediatricians, nutritionists, public health specialists and for all those who care for the health and well-being of the future generation. A healthy child is not only a child with no clinically apparent illnesses, but a child with adequate physical development, both in terms of achieved size and acquired motor skills, and with adequate neurological, psychological and emotional development. Optimal growth and development therefore encompasses the whole of well-being – physical, psychological and social. Furthermore, as we are increasingly aware of the relationship between current health and nutritional status and its impact on both immediate and future risk of disease, we should define a healthy child as one who is going to be a fit and healthy adult with low morbidity for chronic diseases, adequate physical work capacity and adequate reproductive performance.

It is presently estimated that malnutrition is responsible for 55% of all childhood deaths, but this is probably an

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underestimate. The two main forms of malnutrition among children worldwide are anaemia and stunting (height for age <2 SD below the NCHS/WHO International Growth Reference). In 2000, 32.5% of children under 5 in developing countries were stunted [1] and, despite the fact that in some of the world regions there is a downward trend, in 2005 the estimated global prevalence will still be 29%.

Growth Retardation and Acceleration in the Life Cycle

Stunting of growth can occur at different times in the development of a child. Growth failure can occur as early as the second trimester of gestation, resulting in a proportionate reduction of both skeletal and soft tissue growth. A reduction in nutrient supply to the foetus during the third trimester of pregnancy would instead cause a depletion of the foetal fat stores, with little or no impairment of skeletal growth [2].

Growth impairment can then be experienced from soon after birth, and up until the second or even third year of life. Waterlow [3] showed that height increments of infants and children in developing countries were lower than in developed countries from the third to fourth month of life, even taking into account the difference between breast-fed and formula-fed babies. Martorell et al. [4] observed that in Guatemala the most intense growth faltering takes place between 3 and 18 months of age. The Nutrition Collaborative Research Support Program (CRSP) carried out in Egypt, Mexico and Kenya, showed that growth failure occurred in the first 6 months of life and continued through to 18 months [5]. In a study carried out in the Sahara [6] the prevalence of stunting was already 22% in the 0- to 6-month age group, and it increased up to 56% in the fourth year of life (36–48 months), indicating that the process had been continuing on up to that age. This prolonged duration of stunting has also been shown by Frongillo [7]. Shrimpton et al. [8] have shown that the development of stunting follows the same pattern in all the world regions, although South-East Asia reaches the lowest z score of height-for-age.

It is not usually known what proportion of children stunted at age 5 suffered from growth disturbances during intrauterine life and in what proportion growth disturbance started later. This is an important piece of information, both to understand the significance of the underlying biological processes, and to understand the factors leading to growth impairment. There is a correlation between the prevalence of low birth weight (LBW) and stunting in different countries of the world [9], but the relationship is not linear. Indeed, the slope of the regression line might indicate to what extent the growth problem starts in utero. In South Asian countries, the high stunting rate (50–60%) is parallel to a high prevalence of LBW, while in Sub-Saharan Africa similarly high stunting rates are present in countries where LBW does not exceed 15–20%.

Provided a suitable environment is available, catch-up growth in intrauterine growth retardation (IUGR) babies is common. Good and varied nutrition, active parental care, clean water, adequate sanitation and treatment of any pre-existing infections allow an increased growth velocity to be achieved. In developed countries only about one third of the children born with IUGR stay small [10]. In fact there is an inverse relationship between size at birth and potential for catch-up growth. The role of the environment is clear from the remarkable effects of a follow-up study on stunted Indian children adopted in Sweden. Both girls and boys had a rapid catch-up growth in the first 2 years in the new environment, as well as a total remission of the psychomotor development delay [11].

Stunting is accompanied by a development delay that starts very early in life. In healthy populations, such as the Swedish, the onset of the growth spurt in early childhood takes place at 8 months; in deprived populations, such as in depressed areas of Pakistan, it takes place at 13 months [12]. In the Sahara, pubertal staging showed that developmental age lagged 2–3 years behind in both boys and girls [6]. The developmental delay allows the growth period to be extended to age 20–22, but this might not be sufficient to compensate for the initial growth defect.

In children stunted by the age of 5 in developing countries, natural catch-up is not common or at least is not complete. If the underlying environmental factors do not change, the attainment of full growth potential seems unlikely. In a 20-year follow-up study by Satyanarayana et al. [13] the growth rate of stunted children was either normal or slightly accelerated, but as adults the children remained 10 cm smaller than their American counterparts. In the CRSP countries, catch-up growth did not take place. In the Sahara study, some correction occurred and the prevalence of stunting dropped to 25% by age 10. Simondon et al. [14] showed that mean incremental growth of stunted 5- to 16-year-old girls was significantly higher than non-stunted age-matched controls, while boys did not show similar catch-up. In a large sample of stunted Filipino children, 30% had achieved normal height by age 8.5 and 32% by age 12 [15]. More substantial recovery to anticipated adult height has been ob-

served when stunted children have changed their lifestyle as a result of adoption or immigration [16].

The metabolic and health correlates of stunting are probably different in relation to the time when the growth defect started, the duration of the exposure to the factors leading to growth delay and the presence of catch-up growth. According to Barker's Early Origin Hypothesis, early malnutrition should lead to a permanent change in body structure, physiology and metabolism. In proportionate IUGR babies, organ size is different and early catch-up occurs in a smaller proportion of cases.

According to Karlsberg's model of Infant-Child-Puberty [12], the infancy growth pattern is influenced by nutritional factors, while the childhood growth pattern is influenced by growth hormone. Hence, the potential for catch-up growth is probably not indefinite. We do not know whether these sensitive periods are the same in stunted children and in normal children, since development is delayed beyond normal periods of growth.

Causes of Growth Retardation

Short maternal stature, low pre-pregnancy BMI and low pregnancy weight gain are the main recognised determinants of IUGR. There is considerable evidence supporting the role of various micronutrients in determining pregnancy outcomes such as LBW and prematurity but their etiologic role is still not fully demonstrated [17]. The extent to which iron deficiency affects maternal and neonatal health is uncertain. Existing data suggest that iron deficiency anaemia in the mother may be associated with pre-term delivery [18] but supplementation has not been convincingly shown to reduce LBW and prematurity [17]. Folic acid deficiency is another cause of anaemia in pregnant women. Additional folate consumption during periconceptional periods significantly reduces the risk of occurrence of neural tube defects such as anencephaly and spina bifida [19]. However, there is weak evidence for any role of folate in the prevention of LBW and pre-term births. Iodine deficiency during pregnancy causes brain damage to the foetus that results in gross intellectual retardation (cretinism), and in severe cases to pregnancy loss. The evidence linking iodine deficiency to LBW and prematurity is weak. A possible role has been hypothesised for vitamin B complex (B_6 and B_{12}), copper and selenium in improvement of pregnancy outcomes [17]. There is evidence primarily from developed countries suggesting that zinc, calcium and magnesium could improve birth weight and prematurity especially in high-risk groups. An observational study carried out in low income urban women in the USA showed that women using supplements containing iron, folate, zinc, calcium since the second trimester had reduced risk of pre-term delivery, LBW and very LBW [20]. To date, no studies have examined the benefits of multi-vitamin mineral supplements during pregnancy in developing countries where poor dietary intakes and multiple micronutrient deficiencies are common. Further research is needed on the maternal and neonatal benefits of multiple micronutrient supplementation during pregnancy.

The factors that are most frequently implicated in post-natal growth retardation are nutritional deficiencies; high rates of infections and suboptimal caregiver feeding practices. Growth failure may be caused by inadequate intakes of one or more nutrients including energy, protein, or micronutrients such as iron, zinc and vitamins D, A or C. For some of these nutrients, such as zinc and phosphorus, the sign of deficiency during childhood is specifically growth retardation [21]. The main nutrients can be classified into two types according to their impact on growth (table 1). For the type 1 nutrients, tissue levels are variable and deficiency is often associated with characteristic clinical signs and symptoms. When a type 1 deficiency occurs body stores are depleted, followed by a fall in tissue concentration. Metabolic pathways become compromised leading to clinical symptoms. However, growth is rarely affected. Type 2 nutrients are involved in essential physiological functions, tissue levels are fixed and there is no body store on which to draw upon. In times of deficiency, clinical symptoms tend not to emerge, but growth faltering rapidly occurs. The response to a mild long-standing deficiency of any of these nutrients is progressive stunting. In most cases it is not possible to clarify whether any one of such nutrients, or a combination of these has been responsible for the impairment of growth. For some of the nutrients the evidence about an independent causal role is more solid. A number of studies have demonstrated a causal relationship between zinc deficiency and growth retardation. Some (but not all!) controlled zinc supplementation studies have demonstrated the growth-limiting effect of zinc deficiency among preschoolchildren as well as in older children/adolescents [22]. A recent meta-analysis of zinc supplementation trials has confirmed that in populations where zinc deficiency is common, zinc supplementation may result in significant positive responses in both height and weight [23].

Repeated infections exacerbate pre-existing deficiencies through a reduction in food intake, impaired nutrient

Table 1. Classification of nutrients in relation to their effect on growth

	Type 1: Initially normal growth Decreased tissue concentration Specific deficiency signs	Type 2: Growth retardation Normal tissue concentration Absence of specific deficiency signs
With a compartment	Iodine Iron Copper Calcium Thiamine Riboflavin Ascorbic acid Retinol Tocopherol Cobalamin Vitamin K	Energy
Without a compartment	Selenium	Zinc Nitrogen Carbon skeleton of essential amino acids Lysine Threonine Potassium Sodium Phosphorus Sulphur Magnesium
Specific defect in growth retardation	Manganese Vitamin D	

absorption and increased nutrient needs. In contaminated environments where sanitary conditions are poor and water supplies inadequate, children are very often recurrently infected by parasites or other pathogens that affect nutrient requirements and utilisation, and have a direct pathological action on skeletal metabolism. A critical period is when complementary foods are introduced and children are exposed to food-borne pathogens [24]. Evidence linking intestinal infections and nutritional status (both in terms of anthropometric status and micronutrient deficiencies) are extensive. Helminth infections are known to cause malnutrition through the induction of anorexia [25], while infections of the gastrointestinal tract lead to chronic diarrhoea and nutrient malabsorption [26]. It is thought that *Helicobacter pylori* infections, which are easily transmitted from mother to child, are associated with depressed gastric acid secretion and predispose to such repeated infections [27].

Both nutrient intakes and health are affected by the provision of adequate care, i.e. time, attention and support to meet the physical, mental and social needs of the growing child. Appropriate care practices include breastfeeding, complementary feeding, the use of health care and good hygiene practices. Education, knowledge, beliefs, workload and time availability, health and nutritional status of the caregivers, usually the mothers, are essential [28]. In a study carried out in rural Chad, caregiver decisions on child feeding, actions taken when a child is ill, domestic workload and even caregiver's level of satisfaction with life have shown to have an influence on children's height-for-age [29].

Multiple Micronutrient Inadequacy of Diets in Developing Countries

There are several circumstances that might lead to micronutrient shortages during the lifetime of an individual living in developing countries. Such circumstances usually affect a range of micronutrients simultaneously, some of them clustering in the foods commonly consumed in those countries, i.e. iron, zinc, copper and cal-

cium. Hence, in populations where the major reason for iron deficiency is poor availability of iron from the diet, there is also a risk for marginal zinc status and possibly low calcium intake.

In predominantly rural areas of low income countries the diet is based on cereal staples, that account for 60–70% of total energy intake and includes about 12% proteins, most of which is vegetable proteins. The micronutrient density of such diets is low, and the high phytate content impairs micronutrient absorption, resulting in a high risk of inadequacy. In periods of increased need, such as pregnancy, such a risk is even higher and frank deficiencies develop. Iron deficiency increases among pregnant women of most world populations. Zinc deficiency in pregnant women has been reported in Egypt [30], Nigeria [31] and Malawi [32]. Folic acid deficiency has also been described in pregnant women in South Africa [33]. Low micronutrients in mothers' diet could potentially result in low nutrient stores at birth [34].

After birth and in the first 6 months of life, exclusive breast-feeding should allow complete coverage of energy and nutrient needs, although maternal deficiency can lead to low levels of water-soluble vitamins and possibly zinc in breast milk [35]. A comparison of predominantly breast-fed babies with children given complementary foods earlier than 6 months indicates that linear growth is better in the former [36]. However, in many world populations few children are exclusively breast-fed, particularly as a result of early introduction of liquids or other foods. According to the 4th Report on the World Nutrition Situation [1], exclusive breast-feeding rates in many countries of Sub-Saharan Africa can be as low as 1–3% and only two of the surveys reported found rates greater than 50%. Breast milk is usually replaced with fruit juice or other sweet drinks or cereal-based gruels, all of which are micronutrient-poor items, thus leading to high risk of inadequacy.

After the age of 6 months, complementary foods should be introduced in addition to breast milk. Nutrient density, frequency of feeding and factors related to the palatability and ease of consumption of the foods (viscosity, flavour, variety) are all determinants of child micronutrient intake. A comparison of the diets of Peru and Mexico with the diets of US children indicate that the density of iron, zinc and calcium in complementary foods is low and inadequate coverage of nutrient requirements is likely [37]. In order for the requirements of these 'vulnerable nutrients' to be met, the presence of animal foods such as beef, pork or chicken liver would be required. If foods with low iron bioavailability such as beans are used, more than two thirds of the total dietary energy should be provided by that food, which is totally non-feasible. Where animal products are not available, the provision of fortified foods or supplements may be necessary. Mineral bioavailability can also be enhanced by reducing the food factors limiting absorption, e.g. by reducing hexa- and penta-inositol phosphate content with soaking, fermentation and germination [38].

Older children are fed the family diet based on cereals and pulses, in which again animal products are only occasionally present, and hence again, zinc, iron and calcium intakes are likely to be inadequate. Data from the Nutrition CRSP supports the existence of multiple micronutrient deficiencies in developing countries. The Nutrition CRSP was a longitudinal study of the impact of marginal malnutrition on the function of infants, pre-schoolchildren, schoolchildren and adults in Mexico, Kenya and Egypt [39–41]. Multiple food intake measures on these individuals made it possible to explore the relationships between the intake of specific foods, nutrients and growth. Table 2 presents an elaboration of the data reported in the literature for pre-schoolers, comparing the intake of energy, protein and some key micronutrients with the average intake desired for those micronutrients in that age group. Since raw data have not been used, a proper calculation of dietary adequacy cannot be done, but an overall understanding can be obtained of the nutritional value of such diets. Cereals (maize, wheat, rice) provided, respectively, 62, 68 and 71% of total energy intake; dairy products, meat and eggs were eaten in very small quantities in all three locations, especially in Mexico and Kenya. All micronutrients were below desired intake in Mexico and Kenya, and they were marginally sufficient for iron and copper in Egypt. Micronutrients were also largely obtained from cereals, which are sources with low bioavailability. It is therefore easy to see how lack of sufficient micronutrient intakes fail to sustain increased growth rates for catch-up towards the normal growth curve.

This concept of micronutrient clustering in foods is well known to scientists trying to selectively induce deficiencies of individual nutrients, particularly zinc: when trying to induce zinc deficiency, copper deficiency is also induced.

Health Consequences of Stunting

Stunting is the result of repeated insults to the growth plate, with reduced chondrocyte proliferation and maturation. A stunted child will have a lower height than her/

Table 2. Average daily nutrient intake by Egyptian, Mexican and Kenyan pre-schoolers

Foods consumed	kcal/day	Protein g/day	Fe mg/day	Cu mg/day	Zn mg/day	Ca mg/day	Vitamin A µg/day
Egypt							
Maize	169	4.6	–	–	1.2	7.5	30.8
Wheat	292	10.8	3.1	0.6	3.9	41.2	3.1
Rice	126	2.5	0.1	0.07	–	2.5	–
Legumes	43	2.9	1.0	–	–	27.5	35.0
Dairy	59	2.8	0.4	–	0.3	102.0	28.2
Meat	85	5.2	0.8	–	0.9	2.7	3.6
Fats	162	–	–	–	–	–	–
Total	936	28.8	5.4	0.7	6.3	183	100.7
Average desired			4.0	0.6	6.0	450	400
Mexico							
Maize	551	15	–	–	4.0	24.4	100.5
Wheat	75	2.7	0.8	0.1	1.0	10.5	0.8
Rice	26	0.5	0.02	0.01	–	0.5	–
Legumes	80	5.4	0.7	–	–	16.4	35
Dairy	66	3.1	0.4	–	0.3	114	30.3
Meat	60	3.7	0.6	–	0.7	1.9	2.5
Fats	98	–	–	–	–	–	–
Total	956	30.4	2.5	0.1	6.0	185	199.1
Average desired			4.0	0.6	6.0	450	400
Kenya							
Maize	369	10.0	–	–	2.7	16.3	67.3
Wheat	24	0.9	0.2	0.05	0.3	3.3	0.26
Rice	41	0.8	0.04	0.02	–	0.8	–
Legumes	83	5.6	1.9	–	–	53	57.5
Dairy	59	2.8	0.25	–	0.3	102	28.2
Meat	6	0.37	0.06	–	–	0.19	0.25
Fats	29	–	–	–	–	–	–
Total	611	20.4	2.4	0.07	3.3	175.6	163.5
Average desired			4.0	0.6	6.0	450	400

Note: Food intake data are taken from reference 58, nutrient content has been calculated by estimating food quantities from energy intake and by applying food composition data contained in reference 59, and average requirements are taken from reference 60.

his peers and will resemble a younger child, usually 2–3 years younger. Stunting is also associated with a developmental delay, with retarded achievement of the main child development milestones, such as walking. This might create an overall comparative disadvantage in an already difficult environment. In Filipino children, severe stunting at age 2 was associated with later deficits in cognitive ability [42] and reduced school performance has been observed in stunted children in Guatemala [43]. Berkman et al. [44] showed that stunted growth caused by chronic malnutrition during the first 2 years of life had an adverse affect on a child's cognitive ability later in childhood. Stunting is also associated with increased child morbidity and mortality [45]. A very low height-for-age is the single strongest predictor of childhood mortality in the first 5 years of life [46].

A stunted child also has a higher risk of developing chronic diseases, impaired fat oxidation such as occurs in

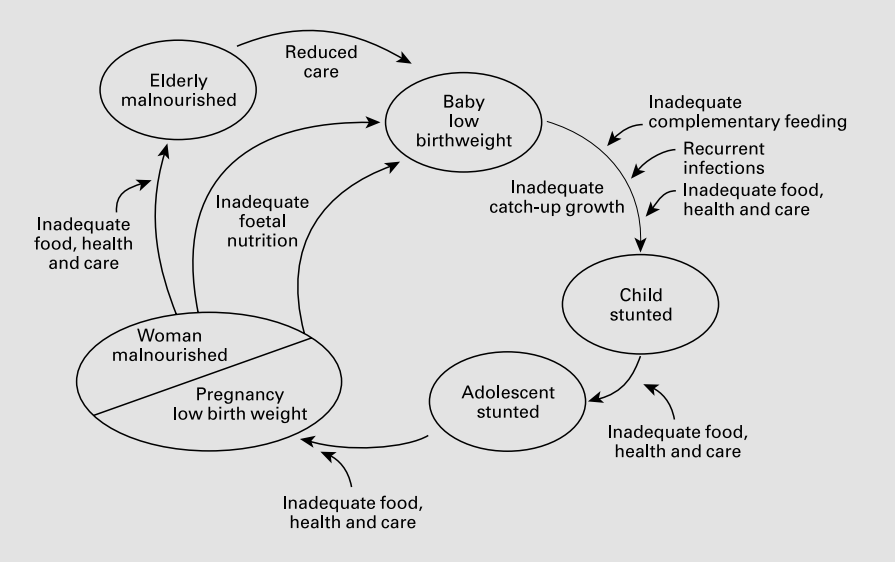

Fig. 1. The poor nutrition cycle.

obesity, and reduced glucose tolerance. Hoffman et al. [47] tested the hypothesis that nutritional stunting is associated with impaired fat oxidation in poor Brazilian children. Stunted children were more likely to have problems oxidising fat and, as a result, stored more fat in their adipose tissues. The mechanisms behind this relation are unclear, but the researchers speculated that long-term undernutrition might have damaged the enzymes and hormones responsible for optimal lipid oxidation. Stunting can also lead to increased risk of hypertension. In a study performed in Jamaica, Gaskin et al. [48] found that stunting in the first 2 years of life was associated with elevated systolic blood pressure at age 7–8 years.

As previously stated, the stunted child is going to be an adult of small stature. A small adult has some functional limitations compared to a taller one, such as reduced working capacity. In societies where manpower is essential for subsistence this may have further consequences on the health and well-being not only of the individual, but also of his/her dependants [49]. Stunted individuals often remain in a state of poverty throughout their lives, as they are not able to produce the extra income that might allow them to escape the cycle of mere subsistence. Reproductive performance may also be affected by stature: a small woman will usually deliver a small child. The occurrence of IUGR is higher in stunted girls [50] and this creates an inter-generational cycle of stunting (fig. 1).

The combined presence of growth retardation, developmental delay, defects in cognitive function, defects in substrate metabolism, increased morbidity and mortality indicates that stunting is by no means a condition affecting just the skeletal system, although the most apparent and easily diagnosable feature is small stature. In order to advocate for a wider understanding of the problem we would like to propose the denomination of 'stunting syndrome', since a syndrome is a disease entity with multiple systemic features that are sometimes maintained throughout life. Stunting cannot be considered a form of cost-free adaptation or just an indicator of socio-economic status. Small is not 'beautiful' and a stunted child is by no means a healthy child, nor is going to be a fit and healthy adult.

Are the features of the stunting syndrome just a coincidence or is there a common basis for their origin? We have discussed that stunting might result from past exposure to multiple micronutrient deficiencies that may still be present. In a few cases the simultaneous presence of stunting and micronutrient deficiencies has been demonstrated. A study among pre-schoolchildren in Brazil documented an association of stunting, diarrhoea and anaemia [51], and another study among schoolchildren in Tanzania showed high rates of both stunting and anaemia [52]. In most cases the micronutrient status of stunted children has not been investigated, both because of the technical difficulties and because of the failure to identify stunting as an active condition of poor health. Poor zinc status would compromise immunity and neurological function; iron and copper deficiency would produce anaemia and affect development of cognitive function, and inadequate vitamin A status would also lead to increased susceptibility to infections (table 3). In other words, the outcome typ-

ical of the stunting syndrome, i.e. retarded growth, developmental delays, poor cognitive function, increased morbidity and mortality could be caused by poor status of such micronutrients.

Table 3. Conditions associated to stunting in children and adults

Children	Adults
Developmental delay	Obesity
Depressed immune function	Reduced glucose tolerance
Defects of cognitive functions	Coronary heart disease
Impaired fat oxidation	Hypertension
	Osteoporosis

The Prevention and Treatment of the Stunting Syndrome

Prevention should be carried out by tackling the factors responsible at the different times of their occurrence, and therefore only a life cycle approach can effectively reduce the incidence of stunting. Intrauterine growth should be protected by providing adequate maternal nutrition. Good infant growth will be ensured by exclusive breast-feeding until the age of 6 months, and by the provision of complementary foods of adequate micronutrient density up to the age of 2 years. A healthier environment, with sufficient care and good hygiene, would also be necessary. The availability of a diversified family diet with adequate micronutrient content will then be necessary throughout childhood and adolescence. All these factors are needed at the same time in order to produce a substantial effect.

The deviations from such an ideal situation are obvious in the countries showing high stunting rates. An overall improvement in the quality of life would be required and therefore a resolution of the problem cannot be achieved in absence of economic development. There is a relationship between GNP and prevalence of stunting, and in the world regions where such economic progress has been made, such as South-East Asia, stunting rates have decreased in the past 15 years. Meanwhile, the enormous socio-economic cost produced by the world diffusion of the stunting syndrome warrants specific preventive actions, supported by the international community, and targeting the highest risk groups. Since in most countries the largest incidence is in the first 2 years of life, such actions could concentrate on the promotion of exclusive breast-feeding and on incentives for the preparation of complementary foods. Increase in the micronutrient density of complementary foods can most effectively be achieved by the introduction of small quantities of animal products. Technological improvements, such as the use of germinated grains to reduce phytate content, and therefore enhance micronutrient bioavailability, might also help. Fortification should be considered as an option when other means of providing micronutrients are not there. This can also be achieved with community-based projects so that the cost of the foods is affordable even to poor families. Issues such as convenience and safety of preparation could indeed be tackled by small-scale village production, which might perform better than the mothers themselves, overburdened by different productive and care tasks in the family.

Once stunting has developed, there still is scope for correction. Treatment will be beneficial for the different aspects of the stunting syndrome, although in most circumstances growth has been the main outcome used to evaluate the effectiveness of the interventions. Research in Guatemala, which provided children with a high-energy, low-protein supplement (fresco) or a high-energy, high-protein supplement (atole), reported improvements in linear growth for both groups [53]. The provision of a multiple micronutrient supplement daily over a 12-month period has been found to help correct growth in Mexican children initially aged <12 months [54].

In a study carried out in the Sahara, catch-up growth was shown in stunted children aged 30–60 months who were given a high micronutrient density peanut paste for 6 months [55]. The study also showed reduced morbidity. Cognitive development was not objectively evaluated, but the mothers reported an increase in the responsiveness and in the liveliness of their children.

The defects of cognitive development can also be reversed. Grantham-McGregor et al. [56] showed that micronutrient supplementation could improve cognitive indices, as well as psychosocial stimulation, although full recovery was only possible with a combination of the two. There is a limit to the possibility to recover cognitive function, and this is up to the age of 3.

The presence of disturbances in substrate metabolism should raise a concern about stunting management. There is no information on whether the induction of catch-up growth might further increase the prospective risk of chronic diseases. Recent epidemiological data have suggested that postnatal accelerated growth patterns – catch-up growth – could alter the risk for adult disease in IUGR

infants. It is not known whether an increased risk is limited to infants born with IUGR or is extended to children who develop stunting during infancy. Evidence has been obtained that nutritional supplementation could alter the pattern of growth, having greatest effects in IUGR infants. Cianfarani et al. [57] maintain that catch-up growth and not just the condition of IUGR is responsible for the increased lifetime risk of metabolic syndrome.

In many populations with high incidence of stunting, life expectancy has been very short. Improved life expectancy in those countries will allow observation of the increasing occurrence of chronic diseases. Long-term studies of morbidity in stunted children would be required to clarify the issue, as well as intervention trials using biomarkers able to point out metabolic disturbances at their early stages.

Conclusions

Good nutrition and a healthy lifestyle are essential throughout the whole life cycle to ensure optimal health both of the individual and future offspring. When a child misses these, and his mother's loving attention, he may develop stunting. Stunting is considered an indicator of poverty, and this reflects the fact that in poor families the quality of diet is poor, the environment is challenging, health care is less accessible and psychosocial stimulation and parents' care are not provided. However, stunting is not just short stature, but is the second most important form of malnutrition in the world after anaemia, and a truly invalidating clinical condition. Stunting is the most evident manifestation of a complex syndrome also involving reduced immune function, retarded development and impairment of cognitive function, as well as other metabolic disturbances that might affect the individual either immediately or in the long term.

The stunting syndrome is one of the main forms of the widespread shortage of micronutrients in the diet, the so-called 'hidden hunger'. Elimination and control of hidden hunger and its health-related consequences is an important public health priority that can only be achieved by ensuring adequate micronutrient intakes in all population groups. Overall improvement of the living standards will be needed and dietary diversification should be pursued in the long term. Immediate actions might involve intensive promotion of exclusive breast-feeding up to 6 months, as well as timely introduction of complementary foods and, in situations of forcedly poor dietary patterns, food fortification or even the distribution of pharmaceutical preparations of micronutrients. In practice, a phased combination of the different types of actions should be designed.

Interventions to correct growth and development are possible at least until the age of 5 years and are justified, although the extent to which catch-up is possible and the long-term implication of this remain to be clarified.

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