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Changing Times, Changing Perspectives: A Relook at Pediatric Nutrition and Growth Status

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Abstract: ***Objectives: Linear growth during early childhood is a potent indicator of healthy growth. India's record of high economic growth over the past decade has not changed its population's nutritional status to the required extent. This article aims to assess various factors leading to stunted growth in pediatrics. Methods: A comprehensive literature search was conducted across multiple databases for the correlation of stunted growth and nutrional status in pediatric population followed by a panel discussion. Results: About 80% of the variation in height occurs due to genetic factors. Children with inadequate intake of protein and essential amino acids may have low levels of IGF - 1, which may result in restricted growth. Of the absolute accumulated height deficit at 60 months, 70% was found to be because of faltering in the first 1000 days (conception to 24months), and 30% was due to continued increases in deficit from the age of 2 to 5 years. The window of opportunity to address stunting does not completely close at the age of 2 years. Conclusion: Early stunting and its influences are majorly irreversible. A judicious combination of nutrition - specific and nutrition - sensitive strategies together can serve as a useful tool in reducing the menace of childhood stunting.***

Keywords: Growth, nutritional status, stunting, nutrition - sensitive strategies

1. Introduction

Linear growth during early childhood is a potent indicator of healthy growth. It is also closely associated with various domains of child development, such as, cognitive, language and sensory - motor capacities.1 Stunting leads to irreversible physical and mental damage to children. Itis an indicator of chronic undernutrition during the most crucial periods of growth and development in early years — a stunted child is too short for their age and does not develop completely. As per WHO child growth standards, stunting is defined as "the percentage of children, aged 0 to 59 months, whose height for age is below minus two standard deviations (moderate and severe stunting) and minus three standard deviations (severe stunting). "[2]

Poor nutrition and illness in childhood lead to growth restriction. As a result, the average height of a population is strongly associated with its standard of living. Over the past two centuries, human height has increased steadily worldwide. On an average, people today are taller compared to their ancestors 100 years ago. This is true for every country across the globe. Human height is a trait which is partly heritable. Nevertheless, the population - wide distribution of height is influenced by non - genetic, environmental factors such as nutrition and health during pregnancy, childhood, and adolescence. Discrepancies in height across the world denote not only genetic differences, but also general differences in the standard of living.[3]

In 2020, amongst the under - five children, 149.2 million were stunted, 45.4 million were wasted, and 38.9 million were overweight globally.[4]

Although we have overcome wasting, stunting is highly prevalent, which may require generations to overcome.[5]

India's record of high economic growth over the past decade has not changed its population's nutritional status to the required extent: latest research shows that although the rate of stunting reduction has doubled in the last 10 years as compared to the previous decade, India continues to have the largest proportion of the world's undernourished population.[6]

India has shown a declining trend in stunting from 48% in 2006 to 35% in 2018—a comparative reduction of 20%. Despite this impressive decline, India has 40.6 million stunted children, accounting for nearly one third of the global burden of childhood stunting.2Moreover, disparities between or within states are quite evident, with four states, namely, Bihar, Madhya Pradesh, Maharashtra, and Uttar Pradesh accounting for more than 50% of stunted children in India.2According toNational Family Health Survey 5 (NFHS - 5) data, nutritional status of children under the age of 5 years is deteriorating. There has been an increase in stunting or chronic malnutrition (i. e., low height with respect to age) in 11 out of 17 states.[7]

Stunting is linked with underdevelopment of the brain, which may have long - lasting harmful consequences, such as, decreased mental ability and learning capacity, poor school performance in childhood, reduced earnings and increased risks of nutrition - related chronic conditions, such as diabetes, hypertension, and obesity in future.[2]

Now identified as a major global health priority, stunting is the focus of several high - profile initiatives like Scaling Up Nutrition, the Zero Hunger Challenge and the Nutrition for Growth Summit. Stunting is also at the center of the six global nutrition targets for 2025, adopted by the World Health Assembly in 2012 (WHO 2012), and it has been proposed as a chief indicator for the post - 2015 development agenda.[8]

Time of growth faltering

Stunting begins from pre - conception when an adolescent girl who becomes a mother in the future is undernourished and anemic, and it aggravates due to poor diet and inadequate sanitation and hygiene of the infant. Often starting in utero, stunting persists for at least the first 2 years of post - natal life. Intrauterine growth faltering is a bigger problem than previously assumed; for example, stunting rates in India are about 20% at birth, and this may contribute tonearly half of growth faltering in Indian children aged under 5 years.[9]

During fetal life and the first 2 years after birth, nutritional requirements to support rapid growth and development are very high. The linear growth rate is highest during fetal life and infancy, gradually slows down during childhood followed by a pubertal spurt, and the adult height is attained by the end of adolescence. Under optimal conditions, the greatest growth rate is in utero (50 cm in 9 months), declining in the first 2 postnatal years (from 25 to 10.5 cm/year) through the childhood to puberty (8.5–5 cm/year), and is followed by a final and relatively short pubertal growth spurt.[10]Thepeculiar stunting pattern in early childhood has established the period from conception to the second birthday (the first 1000 days) as the critical window during which failure to grow is part of an active process of becoming stunted.[8]

It has recently been put forward that growth faltering persists in poor environments beyond the age of 2 years and that there are other windows of opportunity which may offer additional opportunities for intervention to tackle stunting.[11]

Generally, the assessment for growth faltering is done using height - for - age z - scores (HAZs). Changes in mean growth deficits over time (as populations age) should be evaluated using the absolute height - for - age difference (HAD) (in centimeters). Growth patterns according to HAZ had a steep decline during18–24 months of age, followed by a leveling off the curves and a lack of additional deterioration up to 60 months of age. The growth curves based on HAD revealed a notably different picture, indicating a constant decline (increase in deficit) from birth to 60 monthswith no sign of improvements or flattening of the curve between ages 24 and 60 months.[11]

Of the absolute accumulated height deficit at 60months, 70% was found to be because of faltering in the first 1000 days (conception to 24months), and 30% was due to continued increases in deficit from the age of 2 to 5 years.[8]

Global stunting at 5 years includes 11·2% in utero, 60·6% between birth and 2 years and 28% between 2 and 5 years.[9]

The first 1000 days of life is the most likely period for growth failure, especially in poor countries. Global evidence shows that growth failure begins in utero, is distinct during the first year of life, and continues, until around 2 years.

Several clinical studies emphasize the need to intervene within the first 1000 days for maximum benefit. Exclusive breastfeeding during the first 6 months, followed by continued breastfeeding for longer period, in addition to other dietary intakes providing adequate amount of energy and nutrients, as well as environments which stimulate physical activity and play are some of the interventions which promote linear growth. Beyond 2 years of life, there may be a modest catch - up growth, owing to delayed maturation and longer growth period.5 Post 2 years of age, growth faltering may occur, due to sustained exposure to poor, unhealthy and unsanitary environment. Further, any nutritional intervention beyond 2 years may result in substantial fat - mass accumulation with increased risk of chronic diseases. Thus, tackling post 2 - year growth faltering necessitates multi - level approach and interventions.[11]

Physiology of linear growth

The linear growth of a child results from bone elongation, which is the primary result of a process known as endochondral ossification at the growth plate of long bones, short tubular bones of hands and feet and the vertebrae. It is a regulated physiological process, where chondrocyte proliferation follows chondrocyte activation. Newly formed chondrocytes are located over their predecessors arrayed in columns, which is essential for bone elongation. Following this, the process of chondrocyte hypertrophy occurs at the edge of the proliferative zone closest to the metaphysis. Hypertrophic chondrocytes excrete extracellular matrix and attract osteoclasts, osteoblast, and formation of new vessels, which are responsible for remodeling of newly formed cartilage into bone. The growth velocity is determined by the velocity of chondrocyte proliferation and their potential to hypertrophy, which depends on an intrinsic mechanism within the growth plate.[12]

The highest growth velocity is observed prenatally and diminishes rapidly after birth because of a programmed process intrinsic to the growth plate cartilage known as senescence, which is an important process for bone development. Senescence leads to maturation of the bone and to growth cessation simultaneously. The process is influenced by several, particularly local, mediators. The expression of these local mediators depends on systemic mediators which include hormones or inflammatory cytokines. As a result, malnutrition, or a chronic inflammatory state not only leads to a decline in linear growth butretards the growth plate senescence. Upon the resolution of growth - inhibiting conditions, the growth rate is accelerated, leading to catch - up growth. Catch - up growth is not because of systemic hormonal changes (e. g., higherlevels of circulating growth hormone) but is a result of accelerated senescence in the growth plate.[12]

Cells have a complex sensing system which ensures that they do not grow in the unavailability of nutrients which supply the energy to support that growth, including glucose, amino acids, lipoproteins, and minerals. The translational machinery of the cells for protein synthesis is activated by mammalian target of rapamycin (mTOR) which is an evolutionarily conserved serine/threonine protein kinase. Activated mTOR stimulates angiogenesis, which leads to an increase in the number of blood vessels through which nutrients can reach the cell. Moreover, it boosts the production of nutrient transporter proteins which improve the ability of the cell to import essential nutrients and

stimulates glycolysis. When nutrient levels are insufficient, mTOR is inactivated, protein synthesis is inhibited, cell growth is arrested and autophagic protein degradation occurs.[13]

mTOR complex 1 (mTORC1) plays the role of a chief molecular connection between nutrient signals and the metabolic processes necessary for cell growth. mTORC1 facilitates cell growth mainly through the activation of key anabolic processes. It also promotes the biosynthesis of macromolecules such as proteins, lipids, and nucleotides through a diverse set of downstream targets, in order to build the biomass underlying cell, tissue, and organismal growth.[14]

Regulation of mTORC1 is done by insulin and nutrients, including glucose and amino acids, especially leucine, along with several cellular stresses. While in some cell types it is activated by amino acids alone, in others, the amino acids collaborate with growth factors such as insulin. If amino acids are not present, the growth factors are helpless.[13]

Growth regulation occurs at multiple levels, including systemic factors such as hormones, along with local factors including miRNAs, enzymes, transcription factors and epigenetic mechanisms, all of which are influencedby variations in the nutritional status. Thus, malnutrition, which includes several nutrient deficiencies, can be considered as a primary cause of failure to thrive and stunting.[13]

Factors

Linear growth faltering is assumed to be of multifactorial etiology. Some of the factors associated with linear growth retardation include maternal malnutrition, low dietary intakes in infants, frequent infections, and poor psychosocial environment. Linear growth is influenced by complex genetic, physiological, and nutrient - sensitive endocrine/ paracrine/ autocrine mediated molecular signaling mechanisms, which may also include sleep adequacy due to its influence on growth hormone secretion.[9]

Genetics factors: About 80% of the variation in height is estimated to be due to genetic factors. Individual growth potential in terms of height and overall shape, which is mainly a function of bone growth, is determined genetically and every individual will follow a growth curve canalized in terms of both extent and time course under favorable conditions. The genetic programming of the time courses of linear growth, particularly with respect to events in the growth plate which mediate the slowing down of the initial speedy fetal, early infancy growth phase with ultimate cessation of linear growth after puberty, is particularly complex.9It was observed that children belonging to different ethnic backgrounds grow at very similar speeds in the initial years when the environment is affluent and conducive. Children belonging to different geographical areas grow at a notably similar rate during fetal life and the initial few years of postnatal life if born to mothers whose nutritional and health needs are met and if raised in unrestricted conditions.[15]

Hormones and inflammation: The conducive conditions include a diet which can exert a proper regulatory anabolic drive on growth and provide necessary substrates, in an environment with minimal inflammatory challenges. Inflammation, which follows most infections and environmental enteric dysfunction (EED), leads to inhibition of endochondral ossification through the action of mediators including proinflammatory cytokines, the activin A - follistatin system, glucocorticoids, and fibroblast growth factor 21.[9]

Growth factors: Growth hormone secretion from the pituitary gland is stimulated by physical activity. This stimulates insulin - like growth factor - 1 (IGF - 1) production, directly at the level of the growth plate and indirectly at the level of the liver.

Physical activity: Torun and Viteri (1994) observed that in children aged 24–48 months old, who were recovering from protein - energy malnutrition, linear growth was more intense in physically active children despite similar dietary intake. As per current guidelines, 60 minutes per day of moderate - to - vigorousphysical activity for children and youth is recommended to promote health and prevention of diseases (WHO, 2010).[16]

Nutrition: Adequate nutrition and right balance of both macronutrients and micronutrients ensure physiological growth along with prevention of diseases. Apart from themain macronutrients like proteins, carbohydrates, and lipids, fibers and liquid intake are important. Micronutrients are also essential: insufficiency of even one of them can have important consequences, which can lead to impaired growth, delayed maturation, or deficiency diseases such as rickets, scurvy, cretinism.[17]

Growth faltering is linked to overall poor quality of diet, which includes reduced intake of animal foods and increased intake of foods which contain inhibitors that lower the bioavailability of nutrients essential for growth. It is well established that severe protein malnutrition in children mechanistically causes linear growth retardation through a reduction of IGF - 1.[21]

Recent evidence suggests that stunted children might begetting inadequate amount of essential amino acids through diet, thereby having low circulating amino acids. IGF - 1, a protein hormone that mediates the effects of growth hormone reportedly has numerous anabolic effects on skeletal muscles and other tissues. Children with inadequate intake of protein and essential amino acids may have low levels of IGF - 1, which may result in restricted growth.[18]

When it comes to the consumption patterns in India, as far as intake of milk and milk products is concerned, only 8.7% in rural and 14.3% of the population in urban areas reportedly consumed as per the recommended intakes. Vegetable consumption was done by around 8.8% of the population in rural and 17% in urban areas as per the recommended intake, and consumption of recommended intake of nuts and oil seeds was 22 % in rural and 27 % in urban areas. Importantly, other foods (chips, biscuits, chocolates, sweets, juices, etc.,) accounted for 11% per day in urban areas *versus* 4% in rural areas. When compared with the '**My Plate**' recommendations, a large proportion of the

population consumed lower than recommended levels of pulses, legumes, milk, nuts, and vegetables, while cereals and millets were consumed higher than recommended levels. Percentage of people consuming more than recommended amount of cereal was 97.1% in rural and 68.8% in urban region.[19]

Classification of nutrients can be done according to their influence on linear growth. A child responds to essential nutrient deficiency either by continuing to grow and consuming body stores with eventual lowering of the bodily functions (Type I) or by decreasing growth and avidly conserving the nutrient to maintain its concentration in the tissues (Type II). Type 1 deficiency includes conditions such asanemia (iron deficiency), beri - beri (thiamin deficiency), pellagra (niacin or nicotinic acid deficiency), scurvy (vitamin C or ascorbic acid deficiency), xerophthalmia (vitamin A or retinol deficiency) and iodine deficiency disorders. Diagnosis is comparatively simple through clinical symptoms and measurement of the concentration of the nutrient itself. Type II nutrient deficiency causes preservation of plasma and tissue levels even at the cost of growth, repair, and immune system. Therefore, it is necessary to note that the deficiency of type II nutrient does not show any physical signs or clinical symptoms and is difficult to identify. In this type of deficiency, growth ceases, the body starts to conserve the nutrient, and its excretion falls to very low levels. Examples of type II nutrients include protein, zinc, magnesium, phosphorus, and potassium.[20]

Insufficient intake of dietary protein may restrict the availability of amino acids necessary for growth. Poor nutrition may not only limit the intakes of essential amino acids but may also limit the synthesis of other vital conditionally essential amino acids, including arginine, further restricting growth and development. A recent study assessed serum amino acids levels in a cohort of young children in rural Malawi, concluded that compared to non - stunted children, stunted children have reduced serum concentrations of not only all nine essential amino acids, but also arginine and two other conditionally essential amino acids. This is suggestive of inadequate dietary intake of amino acids in stunted children.[22]

Some studies report that arginine stimulates the release of growth hormone, and it has been proposed to play a role in linear growth. A preclinical data model suggests that oral administration of arginine may improve linear growth of long bones, in part, by inducing the secretion of growth hormone, an important modulator of linear growth. The data from the observational study by Van Vught *et al.*, suggests that dietary consumption of protein with higher levels of arginine may be linked with improvement in linear growth velocity in normally growing children. An assessment of the association between arginine intake and growth velocity was performed in pre - pubertal children. The results revealed a dose - dependent physiological role of protein intake, and more particularlyarginine intake, on linear growth velocity in normally growing children. Children with an arginine intake between 2.8 and 3.2 g/d grew 0.33 cm/year faster compared to those with an arginine intake below 2.2 g/d.[23]

Of the animal - source foods, only milk has been specifically and repeatedly shown to have an important impact on linear growth in both undernourished and well - nourished children.[9]

Micronutrients: The micronutrients related to linear growth are those believed to be nutritionally significant or `essential'; persistently low availability of these micronutrients below a certain threshold leads to a reduction in one or more physiologically important functions. As per a WHO Expert Committee, these ‘essential’ micronutrients include iodine, zinc, selenium, copper, molybdenum, chromium, vitamin A and calcium.[24]

Iodine: The effect of severe iodine deficiency on human growth and development is clear in terms of delayed physical development through growth restriction and delayed bone maturation, as well as impaired mental function. Deficiencies of Se, Fe and vitamin A worsen the effects of iodine deficiency. Owing to the importance of the T4/T3 axis for growth and development, hypothyroidism reduces circulating IGF - 1 and IGFBP - 3 levels, and thyroid hormonereplacement thereby increasing them; severe iodine deficiency leads to severe retardation of linear - growth.[9]

Zinc: Zinc is an essential transition metal that plays a vital role in normal linear growth via mechanisms that involve release of growth hormone and IGF - 1, chondrogenesis, collagen synthesis, osteoblast function, and calcification of bone. Thus, it is no surprise that moderate - to - severe zinc deficiency in children inhibits growth and skeletal maturation.[21]

Calcium: Bone is the chief reservoir for body calcium, where it is stored in the form of hydroxyapatite component. Calcium deficiency can induce bone resorption, which may lead to linear growth impairment.2[1]Decreased calcium intake during growth may result in growth retardation and suboptimal peak bone mass.[25]

Vitamin A: Vitamin A is necessary for growth, and clinical vitamin A deficiency has been associated with poor growth performance.2[1]Vitamin A maintains the epithelium integrity in the respiratory and gastrointestinal tracts, and its deficiency is associated with increased risk of diarrhea, *Plasmodium falciparum* malaria, measles, and overall mortality.[26]

Iron: Among tissues, bone marrow requires large amount of iron for hemoglobin synthesis. Iron deficiency manifests itself with lowered physical performance due reduced amount hemoglobin and myoglobin. Other consequences of the deficiency state include impaired immune response with reduced function of macrophages and neutrophils along with a decrease in T lymphocytes. Brain iron deficiency leads to reduced synthesis of myelin and neurotransmitter with impaired movement, memory, and perception control.[17]

Vitamin D: Optimal vitamin D status, which is often evaluated by measuring calcifediol (i. e.25 [OH]D), facilitates calcium absorption and growth to support active vitamin D (i. e. calcitriol [1, 25{OH}2D3. Prolonged

insufficiency of vitamin D impairs transcriptional regulation of skeletal homeostasis and linear growth, which may lead to stunting.[27]

Vitamin K2: Vitamin K2 is required for activation of osteocalcin, an important protein secreted by osteoblasts, which are the body's bone - building cells. Activated osteocalcin promotes the transfer of calcium into bones and regulates calcium homeostasis. When osteocalcin is completely activated, it attaches to calcium and transports it into bone, supportingbone mineralization, thereby helping in building a strong skeletal system. Insufficiency of vitamin K has been found to be associated with low bone mass, osteoporosis, and fracture risk.[28]

Circulating osteocalcin measurement (uncarboxylated and carboxylated, is used as a biomarker for vitamin K status in bone. Increased levels of inactive uncarboxylated osteocalcin indicate suboptimal vitamin K status and has been related to decreased bone mineral content in children. Better vitamin K status has been associated with improvement in bone mineral density and content.[29]

Vicious cycle

The combined and interactive influences of infections, environmental factors, and malnutrition as possible determinants of child stunting have been believed to be of great importance but were first articulated in a systematic way by Scrimshaw *et al.*[9]

Both serious acute infections, particularly those involving the gastrointestinal tract, and chronic infections lead to linear growth impairment.[9]

The interplay of inadequate nutrition and frequent infections results in a vicious cycle of worsened nutritional status and increased vulnerability to infections. Infection compromises the nutritional status through decreased appetite, impaired intestinal absorption, increased catabolism, and direction of nutrients away from growth and towards immune response. In turn, undernutrition leads to increased risk of infection by its negative effect on the epithelial barrier function and altered immune response.[8]

Stunting, a chronic state of undernutrition, is related to increased morbidity and mortality due to infections, particularly pneumonia and diarrhea, as well as sepsis, meningitis, tuberculosis, and hepatitis. This is suggestive of a generalized immune disorder in children with severely stunted growth.[8]

Current evidence indicates that environmental enteric dysfunction (EED) plays a central role in the pathogenesis of stunting. Small intestinal inflammation in EED is associated with high levels of C - reactive protein and accompanied by release of cytokines. These lower the appetite and food intake and hinder the production and action of chondrocyte growth factors. Inflammation is also associated with increased levels of cortisol, which inhibits linear growth directly with respect to chondrocyte proliferation, hypertrophy and cartilage matrix production.[30]

Recent studies reported that stunted, malnourished Ugandan infants and children (age 6 months–5 years) had high levels of interleukin 6 (IL - 6). IL - 6 blocks growth hormone induction of IGF - 1 production and inhibits its action at the growth plate. Furthermore, in EED, insufficient intake and malabsorption of zinc may decrease IGF - 1 production and action, thereby leading to impaired linear growth. Also, growth failure in EED may be exacerbated by the development of acute malnutrition, which is often seen in sepsis as well as gastrointestinal and pulmonary infections.[30]

Diseases and malnutrition demonstrate interaction across the spectrum. Chronic Malnutrition is associated with increased incidence of respiratory infections, diarrhea, malaria, measles, and other communicable diseases at one end of the spectrum, and increased risk of non - communicable diseases due to overnutrition and unhealthy diet at the other end. Moreover, the poor population, typically exposed more to communicable diseases because of their living and working conditions, have an additional handicap vis - à - vis non - communicable diseases as low birth weight and undernutrition during early childhood are related to increased risk of diabetes and cardiovascular diseases later in life.[6]

Analysis based on NFHS - 4 revealed that childhood undernutrition contributes to 45% of under - 5 mortality rate alone and remains a key public health challenge in India. An undernourished child is at increased risk of death due to respiratory infections, diarrhea, malaria, measles, and other infectious diseases. Earlier studies have shown that more than half of child deaths from malaria (57%), diarrhea (61%) and pneumonia (52%), as well as 45% of deaths from measles are related to protein - energy malnutrition and micronutrient deficiencies in India, where child malnutrition is accountable for 22% of the country's overall disease burden.6

In developing countries, on an average each child has five episodes of acute respiratory infection (ARI) per year, contributing to 30% - 50% of the total pediatric outpatient visits and 20% - 30% of the pediatric admissions. As per recent community - based estimates from a prospective study, ARI accounts for 70% of morbidities among children under 5 years of age. In developing country, a child is likely to have around 0.3 episodes of pneumonia annually, while in developed countries, the likelihood is 0.03 episodes per child per year. Based on this, India is anticipated to have over 700 million episodes of ARI and over 52 million episodes of pneumonia annually.[31]

Apart from this, infections also contribute to existing burden of undernutrition. In the case of anemia, malaria is one of thechief causative factors, along with iron, folic acid, and vitamin B12 deficiencies. As per a report on India's state level disease burden, child and maternal malnutrition contributes to disease through increased risk of neonatal disorders, nutritional deficiencies, diarrheal diseases, lower respiratory infections, and other common infections.[6]

Helminth infections in school - aged children are linked to cognitive deficits. Numerous worm infections, including hookworm, schistosomes, and *Giardia*, are linked with iron -

deficiency anemia and a considerable loss of micronutrients. Blood loss may be as high as 45 mL/day, or the equivalent of 9.9 mg of iron. Children unaffected by parasites have better nutritional status, grow faster, learn more, and are freer of infections than are children with parasites.[26]

Stunting syndrome

Stunting begins in - utero. The neonates' nutritional status is directly affected by that of the mother before, during and after pregnancy. A malnourished mother is more likely to give birth to smaller babies which can continue the stunting cycle in future generations. The stunting syndrome recognizes interactions between malnutrition and infection throughout the maternal, infant and child life cycle which inhibit growth.[9]

Indeed, in 20% to 25% of infants and children considered "stunted", the growth failure begins in utero: prematurity and intrauterine growth restriction, especially in combination, are associated with increased risk of postnatal stunting by twofold to sevenfold.[30] In a study on five countries including India, maternal short stature was related to child stunting. Mothers with height less than 150.1 cm were 3.2 times more prone to have stunted offspring as against taller mothers. Decreased maternal stature is linked with increased odds of child stunting. Compared to the children born to very tall women having a height greater than 156 cm, children born to tall mothers were 1.4 times more prone to be stunted; those born to mothers with average height were twice more prone to be stunted; those born to short mothers were 2.5 more prone to be stunted and children with very short mothers of <147 cm height were 3.7 times more prone to be stunted.[32]

Deficiency of macro - or micronutrients during the prenatal period could potentially direct the fetus towards slower linear growth during postnatal life or lead to low nutrient stores at birth. Low intake of micronutrient in young infants in developing countries may be due to low levels of various micronutrients in breast milk due to maternal deficiency, and from inadequate complementary feeding practices during the second half of infancy. Energy as well as micronutrient intakes of infants are generally low in many of these settings. Despite adequate energy intakes, diets are often vegetarian and are low in content and poor in bioavailability of nutrients like iron, calcium, zinc, and vitamin A. Infants and children in developing countries suffer frequent infections resulting in decreased intake, impaired absorption, increased nutrient losses, hypercatabolism, and other metabolic perturbations, all of which contribute to linear growth faltering.[24]

Monitoring

Growth monitoring is widely acknowledged and supported by health professionals.3[0] It can be stated that "A GROWING CHILD IS A HEALTHY CHILD", and equally true that, "A CHILD WHOIS NOT GROWING IS NOT HEALTHY".[34]

Growth monitoring helps detect: [33]

a) feeding difficulties, especially inthe younger child
b) chronic ill health from any cause, including respiratory infection, malaria, tuberculosis, and growth hormone deficiency
c) social deprivation, where poverty and home circumstances are such that poor nutrition is one of the outcomes
d) "non-organic failure to thrive", where poor weight gain is assumed to be because of psychosocial causes

Growth monitoring includes anthropometric measurements, biochemical investigations, clinical assessment, and dietary assessment (ABCD).

Anthropometry: Families and health workers often fail to identify childhood stunting in communities where short stature is so frequent that it is considered normal. To a great extent, this is because linear growth is not routinely measured as part of community health programs, along with lack of awareness of the devastating health consequences of stunting. Assessment of linear growth is not difficult, but it requires adherence to the chief principles and attention to detail. The accuracy and dependability of length and height measurements highly rely on the robustness, precision, maintenance, and calibration of the anthropometric equipment, the techniques of measurement and the establishment of data quality procedures. Variation in length and height measurements can occur due to a variety of influences, including the setting where measurements are taken, the child's behavior and co - operation, the accuracy and precision of the instruments, the anthropometrist's technical capability and data recording methods. Proper training and adherence to standardized methods and procedures are therefore essential for reducing measurement error and minimizing bias.[8]

Growth monitoring is routinely practiced in most settings, but it has not been evaluated adequately, probably because intuitively it does not appear harmful. However, this assumption may not be correct as growth monitoring can lead to anxiety in the caregiver (usually the mother) and considerable amount of valuable health worker and caregiver's time is expended in carrying it out.[35]

Clinical assessment

Physical examination helps in identifying signs of specific nutrient deficiency that may be visible in the form of changes in skin, nails, mouth, eyes, hair, and musculature or a macronutrient deficiency by observing muscle wasting, subcutaneous fat loss, and edema.[36]

Micronutrient deficiencies have an impact on child survival, growth, brain development, educational achievement, and disease resistance. Micronutrient deficiencies are caused due to inadequate dietary intake, poor nutrient absorption, excessive losses, increased requirement, or a combination of these factors.[37]

Evaluation of iron deficiency is performed using history of easy fatigability, pica, passage of worms in stools and by looking for pallor in the lower palpebral conjunctiva, palms, nail beds and tongue. History of easy fatigability is elicited from the caregiver through comparison of activities of the child with his peers of the same age and sex group. Pica is the constant ingestion of non - nutritive substances like plaster, charcoal, paint, and soil for a month in a manner

which is not appropriate for the developmental level and not a part of culturally sanctioned practice.[37]

Iodine deficiency is evaluated by obtaining history of delayed gross developmental milestones, delayed dentition and examining for goiter. Clinical evaluation of zinc using clinical features is difficult, but an attempt was made to assess the burden by using stunting as proxy for zinc deficiency. Deficiency of vitamin A is assessed by history of night blindness, appearance of Bitot'sspots and conjunctival xerosis. Vitamin B and C deficiency is estimated by examining for angular stomatitis, cheilosis, glossitis, mouth sores, bleeding gums, perifollicular hemorrhages and history of prolonged bleeding and delayed healing. Deficiency of vitamin is estimated by examining for features suggestive of rickets, such as frontal bossing, bow legs, rickety rosary etc.[37]

Biochemical measures

These are used for identification of any evidence of inflammation, such as C - reactive protein, fluid imbalance, and specific nutritional deficiency (such as iron, vitamin A, D, zinc). Inflammatory markers indicateincreased nutrient requirements related to stress metabolism.[38]

Dietary assessment

It is essential to evaluate the quality and quantity of the child's food intake. Deficient or unbalanced food intake may be due to poor socioeconomic circumstances, the child's food preferences, mechanical or physical feeding challenges, or inflammatory conditions which increase calorie expenditure. This helps in identification of the cause of growth deficit, if any.[39]

Intervention

Undernutrition develops during the first two years of life, a periodduring which 85% of the total brain development occurs. The United Nations Children's Fund has time and again called attention to the critical windows of opportunity for preventing undernutrition: the first two years of a child's life; the adolescence years for girls; and pregnancy and lactation periods for mothers. These are the periods when validated nutritional interventions offer children the best survival chance and an opportunity to reach their optimal growth and development. Optimal infant and young child feeding practices include commencement of breastfeeding within one hour of birth; exclusive breastfeeding for the first six months of the infant's life; and continued breastfeeding for two years or more, along with safe, adequate, and appropriate complementary foods starting at the age of six months.[6]

Interventions for promoting linear growth can be broadly classified as nutrition - specific and nutrition - sensitive interventions. Both nutrition - specific and nutrition - sensitive strategies should be combined to lower linear growth faltering.[40]

Nutrition - specific interventions: The most common nutritional intervention strategy is using nutrition - specific interventions for improving maternal and child nutrition. These interventions are important not only in mothers and children but should be started right from puberty in girls (would - be mothers) to break the intergenerational stunting cycle. Dietary diversification and increased dietary intake of nutrient - rich foods needs to be achieved for optimum nutrition to promote the linear growth. It may however be difficult to achieve this in developing countries owing to lack of resources. The nutrition - specific strategies usually involve some form of nutritional supplementation and/or nutritional education to address this. Several studies have shown benefit with these strategies. An analysis by Bhutta and colleagues as a part of Lancet nutrition series found that if the coverage of nutrition - specific interventions was scaled up to 90%, stunting would be lowered by a mean 20.3%.[40]

Nutritional supplementation during pregnancy and lactation: During pregnancy and lactation, the nutrient requirements are very high because of the need to support fetal growth and milk production. As per the latest Indian Council of Medical Research recommendations, the energy requirements are 18% greater during pregnancy (350 kcal more) and 31% greater during lactation (600 kcal more), and protein requirements are 40% greater during both periods. The micronutrient requirements are also increased by 50% during this period. In developing countries, it may be challenging to supply these needs through the diet alone. Nutritional supplementation strategies during this period include:

- fortification of staple foods with micronutrients,
- multiple micronutrient supplements,
- fortified food products.[41]

Complementary food supplements are fortified food - based products that can either be consumed alone or can be added to other foods. Lipid-based nutrient supplements (LNS) and fortified full-fat soy flour are included in such food products. These products can provide both macro - and micronutrients including fatty acids, vitamins, and proteins. No preparation is needed for the complementary food supplements and there is no nutrient loss during cooking. However, they are more costly as compared to micronutrient powders.[41]

In the Women First Preconception Maternal Nutrition Trial, nutritional supplement in the form of a daily lipid - based micronutrient supplement three months prior to conception was found to have 44% reduction in stunting in newborn children at birth.[42]A systematic review by Lassi et al. reported reduced stunting in newborns with balanced energy protein (BEP) supplementation and food distribution programs in pregnant women.[43]

Nutrition education: Maternal nutrition education may lead to improvement in dietary habits and may help in lowering stunting, especially in communities having adequate food resources. Studies of nutrition education in food secure populations demonstrated a notable increase in height and HAZ. Studies of nutrition education in food insecure populations have also revealed remarkable influences on linear growth. In a trial conducted in Peru, stunting prevalence was reduced to 8% with nutrition education as compared with 15% in the control group.[44]A randomized trial by Vazir and colleagues evaluated the efficacy of complementary feeding and complementary feeding plus education about complementary feeding. Improvement in

length with education to caregivers about feeding was reported.4[5] A study conducted in Haryana to evaluate the effectiveness of health education intervention to mothers of children aged below 18 months on complementary feeding practices and on the nutritional status of children revealed that the mean energy intake from complementary foods was notably higher in the intervention group children at 9 months and 18 months.[46]

Sunguya *et al.*, evaluated the effect of nutrition training for health workers on the improvement of feeding practices of children aged between 6 months and 2 years by caregivers. It was noted that there was improvement in mean daily energy intake, feeding frequency, and dietary diversity among the children in the intervention group as against the control group.[47]

Promotion of breastfeeding: Exclusive breastfeeding till 6 months of age has been recommended by WHO. Combined individual and group counseling are helpful in encouraging breastfeeding.[48]

Strategies to improve complementary feeding: In order to meet the energy and nutrient requirements after six months of age, complementary feeding in optimum quantity and content is needed. There is great risk of nutrient deficiency during this period as the complementary foods many times are diluted and are in insufficient quantities. Ideally, complementary feeding should comprise of diverse foods which are rich in energy density, contain adequate amounts of proteins with all essential amino acids and adequate quantity of micronutrients as well as should be palatable. It is used to estimate the quality and adequacy of protein in complementary foods. The recommended protein - energy (PE) ratio, the ratio of energy from protein to total energy in dietary foods varies from 8 to 15 depending on the quality of proteins in food and age.[48]

The complementary foods can be general household food items or specially prepared foods. Sattu - like preparations are commonly consumed in India and are also used for complementary feeding. They consist of equal amount of cereal (rice, wheat, or ragi), pulse (moong, chana, or arhaara), and half parts of groundnut. Small amount of ghee or sugar can be added to address the energy deficiency. In developing countries, ideal complementary feeding to achieve good nutrition is challenging. Complementary feeding challenges include delayed introduction of complementary foods, inadequate frequency, and quantity of foods, and poor quality and diversity of diet. As per a study, only 7% infants met the acceptable criteria in complementary feeding. Fortified food products may help to tide over nutrient deficiencies in complementary feeding. A randomized controlled trial by Dhingra *et al.*, reported linear growth improvement with fortified milk supplements. Fortified food products used for complementary feeding can be categorized into three groups as under: [48]

Fortified blended foods: These are made from cereals and fortified with micronutrients. They are available in the ready - to - use foods which requires little preparation and ensure quality. The drawback of fortified blended foods is that they may displace milk as they provide sufficient energy. Over - dependence on fortified blended foods may reduce dietary diversity.[48]

Evidence for complementary foods: Dhingra *et al.*, observed greater weight and height gain in children who were given fortified milk powder. Many studies have reported advantageous effects of complementary foods on linear growth.[49]

A systematic review and meta - analysis evaluated the impact of nutrition - based interventions on linear growth in children aged >2 years. Particularly, interventions containing iron, calcium, or iodine or those providing food showed no improvement in linear growth, whereas interventions containing zinc (0.15; 95% CI: 0.06, 0.24), vitamin A (0.05; 95% CI: 0.01, 0.09), multiple micronutrients (0.26; 95% CI: 0.13, 0.39), or protein (0.68; 95% CI: 0.30, 1.05) showed a significant positive effect on height. Nutrient deficiencies are not likely to occur in isolation; thus, multiple micronutrient supplementation has been promoted as a more holistic strategy to address malnutrition as well as stunting. Therefore, the nutritional interventions, namely zinc, vitamin A, multiple micronutrients, and protein, delivered to children aged >2 years have the potential to improve linear growth, especially in those who have experienced growth failure. The results suggest that the window of opportunity to address stunting does not completely close at the age of 2 years.[21]

Treatment and prevention of childhood illnesses: Prompt treatment of common childhood infections may be helpful in breaking the vicious cycle of infection and stunting.[26]

During illness, children require additional protein and essential amino acids to recover. The protein and essential amino acid requirements are higher in the presence of chronic or acute infections. Inflammation leads to increased requirement of amino acidsby three - fold. Further, evidence suggests that energy deficit increases protein and essential amino acid requirement.[18]

Nutrition sensitive interventions: Nutrition - specific interventions are by themselves inadequate to tackle childhood stunting. The water supply, sanitation, hygiene needs to improve to reduce the incidence of childhood infections. These water, sanitation, and hygiene (WASH) interventions are also important in tackling childhood stunting. The improvement in social and economic conditions may bring food security in household and may improve dietary diversity. Maternal education may bring about increase in marital age and proper spacing of births. Good antenatal care also has a bearing on birth length and weight. All these are termed as nutrition sensitive interventions. The important nutrition sensitive interventions are: [50]

- WASH programs
- Food security
- Social security
- Maternal education
- Child protection
- Schooling
- Family and health planning services to achieve proper spacing of births

- Immunization

A study on rural indigenous communities of Jharkhand and Odisha, Eastern India reported that birth spacing, handwashing, cooking outdoors were positively associated with linear growth whereas later birth order and repeated episodes of diarrhea were significantly associated with faltering of linear growth.[50]

Nutritional supplementation programs: Nutritional supplementation is part of several government schemes in order toaddress the issue of maternal and child malnutrition. These include integrated child development scheme, special nutrition program, Balwadi nutrition program, and mid - day meal program.[51]

POSHAN Abhiyaan: Government interventions are needed to give impetus for improving the nutritional status of the community. The Prime Minister's Overarching Scheme for Holistic Nourishment (POSHAN Abhiyaan), or the National Nutrition Mission, is the Government of India's flagship program for improving nutritional outcomes in children, pregnant women and lactating mothers. The objective of POSHAN Abhiyaan is to reduce stunting in Indian districts identified with the highest malnutrition burden by improving utilization of key Anganwadi Services and improving the quality of Anganwadi Services delivery. It aims to ensure holistic development and adequate nutrition for pregnant women, mothers, and children.[51]

WASH strategies comprise of improved water quality, sanitation, and handwashing strategies which can help in reducing stunting. In a randomized controlled trial in Zimbabwe, Humphrey et al. studied the impact of improved water, sanitation, and hygiene (WASH) and Improved infant and young child feeding (IYCF). The IYCF intervention resulted in an increase in mean length - for - age Z score by 0·16. However, the WASH intervention showed no effect on these outcomes.[51]

Applied nutrition program and various programs by food and nutrition board are run by Government of India to boost food production. However, only nutrition - specific interventions are not adequate for reducing the prevalence of childhood stunting. To achieve optimal results, a combination of nutrition - specific and nutrition - sensitive interventions is required.[51]

The available evidence suggests that a single strategy is not likely to have universal success in combating childhood stunting. There should be a strong political will and appropriate co - ordination between government, and non - governmental organizations, and community participation in the fight against stunting. A judicious combination of nutrition - specific and nutrition - sensitive strategies together with improvement in food availability and better living conditions is required for reducing the menace of childhood stunting.[51]

Peru's experience offers useful lessons on how to address the problem of stunting. Thousands of Peruvian children who were babies and toddlers during the terrible food crisis in 2008 were much smaller and had a negative impact on their cognitive skills development.[52]

Peru reduced child stunting with improved socioeconomic determinants, sustained implementation of out - of - health - sector and within - health - sector changes, and implementation of health interventions. These efforts were propelled by a multisectoral approach, strong civil society advocacy, and keen political leadership. Findings from Peru suggest that children with early recovery from stunting performed better in vocabulary and mathematics tests than children who remained stunted.[52]

Recovery from stunting after the first thousand days may also result in the reversal of developmental setbacks such as cognitive impairment. Factors like household income, maternal education and health, local water, sanitation, and health infrastructure, which are important in preventing stunting, are also important for recovering from stunting. Growth monitoring helps in early identification of growth faltering as well as in early intervention. Combined use of nutrition - specific and nutrition - sensitive strategies seem to be effective.[52]

2. Conclusion

Childhood stunting is the best overall measure of children's well - being and a precise reflection of social inequalities. Affecting millions of children globally, stunting is the most prevalent form of child malnutrition. Growth faltering often begins *in utero* and persists for the first 2 years of post - natal life and beyond. Early stunting and its influences are majorly irreversible. The first 1000 days being the critical period, if pregnant mothers and babies miss out on adequate nutrition, it can lead to physical stunting and cognitive impairment. So, while the first thousand days are very important, the rest of a child's life is also important. It now seems clear that children can recover from stunting after their first thousand days.

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