Under- and Over-nutrition with Special Reference to the Significance of Developmental Plasticity in Understanding Obesity and Associated Morbidity in Developing Countries

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INTRODUCTION

The rapid economic development along with an increased urbanization and impact of market globalization over the last decade has brought about considerable changes in diets and lifestyles of the people all over the world. "Food and food products have become commodities produced and traded in a market that has expanded from an essentially local base to an increasingly global one. Changes in the world food economy are reflected in shifting dietary patterns, for example, increased consumption of energy-dense diets high in fat, particularly saturated fat, and low in unrefined carbohydrates. These patterns are combined with a decline in energy expenditure that is associated with a sedentary lifestyle - motorized transport, labour-saving devices in the home, the phasing out of physically demanding manual tasks in the workplace, and leisure time that is preponderantly devoted to physically undemanding pastimes" (WHO/FAO, 2003). It is believed that these changes in dietary and lifestyle patterns are the major factors for the increasing prevalence of obesity associated with non-communicable chronic diseases (NCDs) such as diabetes mellitus, cardiovascular disease, hypertension and stroke, osteoporosis, and some forms of cancer. The problem is more serious in developing countries where undernutrition is also still a major health problem. In this brief overview, an attempt has been made to look into the prevalence of under- and over-nutrition as defined by anthropometric measurements and indices with special reference to the significance of developmental plasticity, or ontogenetic modification, during growth and development in understanding obesity that is associated with NCDs in developing countries.

GROWTH AS A MEASURE/INDICATOR OF UNDER-NUTRITION AND OVER-NUTRITION

Growth is defined as a regular process of quantitative increase in size or mass of different tissues and organs of the body from conception to adulthood. It is generally measured in terms of anthropometric measurements and indices. There are considerable differences between and within populations in the rate of physical growth and attainment of body size at any given age (Eveleth and Tanner, 1990). From the anthropological point of view, these variations may be considered as "adjustment and adaptation to both the nutritional and disease environments; smaller body size may offer an advantage if it adjusts the size of individuals to available nutritional and energetic resources, but it may be disadvantageous in other respects such as greater susceptibility to infectious disease, or lower physical work capacity" (Ulijaszek, 1995).

In general, the variation in growth rate is due to both genetic and environmental factors. Thus growth variation between and within populations is the physical reflection of innumerable biological and environmental factors. From this point of view, growth variation is "an indicator variable" (Norgan, 2000). For example, growth retardation or delay in growth is mainly due to inadequate nutrition and/or infections. Growth retardation is, therefore, considered an indicator of under-nutrition. In a similar manner, growth is considered an indicator of health and conditions of the society because under- and over-nutrition and infections that affect growth are associated with ill health, hygienic and socioeconomic conditions of such society (Eveleth and Tanner, 1990). Also, growth can be considered a means of adaptation or "biological plasticity" (Lasker, 1969). Growth patterns of children in high-altitude populations may be associated with hypoxic stress (Baker, 1969), or the better growth performance of immigrants as compared with non-immigrants may be regarded as an indicator of biological plasticity in response to better environmental quality (Boas, 1912; Lasker, 1952).

The delay or reduction in growth for a given individual or population, as indicated generally by anthropometric measurements and indices, is known as growth retardation. Growth retardation in developing countries is mainly because of

environmental factors, including inadequate nutrition, infections and poor socioeconomic conditions. Empirical evidence shows that under-five children belonging to the higher socioeconomic strata in developing countries have shown similar growth patterns to their coevals in developed or high-income countries (Habitch et al., 1974). Accordingly, growth retardation is generally considered an indicator of poor nutritional status, or a failure in the expression of the "genetic potential" for growth (Gopalan, 1992). Despite criticisms, the growth curves of well-nourished children from high-income countries were widely used to assess or monitor the growth and nutritional status of children all over the world. It is argued that since children in high-income countries are unhindered by nutritional deprivation, thereby enjoying the maximal growth permitted by their genetic potential, they constitute a reference group against which to assess the nutri-tional status of all other groups of children. For this purpose, international standards, or growth references, such as the U.S. National Center for Health Statistics (NCHS) growth references endorsed by the World Health Organization (WHO, 1983, 1995) were widely used for assessing the nutritional status of children all over the world. The children who are below - 2 SD or - 2 Z score of these standards/references are classified as undernourished relative to their sex and age groups. On the other hand, there is lack of consistency in the classification of childhood obesity, a condition of over-nutrition characte-rized by an excess accumulation of fat (see for reviews Shetty, 1999; Reilley et al., 2003). The WHO Expert Committee (WHO, 1995) has recommended the use of > + 2 weight-for-height Z scores of the NCHS/WHO reference as a cut-off for overweight in children all over the world. For adolescents, a cut-off of >85th percentile of body mass index (BMI)-for-age is recommended to classify as overweight, while a >85th percentile of BMI-for-age may be indicative of adolescent obesity. The use of skinfold thickness-for-age along with BMI-for-age is also recommended (WHO, 1995).

PREVALENCE OF UNDER NUTRITION

Undernutrition remains a major health problem in developing countries. According to a recent estimate, about 10.9 million children under five years of age die every year, and most of these children live in developing countries (WHO, 2001). It is suggested that more than 50% of these deaths are attributable to undernutrition and its synergetic relation with infections. About 30%, or 161 million, of children under five years of age in developing countries still suffer from stunted growth (defined as height for age = - 2 SD of the NCHS/WHO median). "More than 70% of these children live in Asia, over 25% in Africa, and about 4% in Latin America and the Caribbean. The situation in some parts of Africa is particularly alarming because the numbers of malnourished children are increasing as a result of HIV/AIDS, ecological disasters, armed conflict, civil disturbances, and mass population movements" (WHO, 2001). In Southeast Asia, the prevalence of undernutrition is still alarming particularly in India, Nepal, Bangladesh and Bhutan (WHO, 2002). Table 1 shows that the prevalence of stunting (< - 2 SD of the NCHS/WHO median) among children less than 5 years is highest in Nepal (54.1%), followed by India (45.5%) and Bangladesh (44.7%). On the other hand, the prevalence of underweight (defined as weight-for-age < - 2 SD of the NCHS/WHO median) is highest in Bangladesh (47.7%), followed by Nepal (47.1%) and India (47.0%). In general, it indicates that about 50% of children under five years of age in India, Bangladesh and Nepal are undernourished as per weight-for-age and height-for-age relative to the NCHS/WHO growth references. The prevalence of underweight in many Southeast Asia countries is higher than that of stunting, and the difference is more pronounced in Sri Lanka and Maldives. On the other hand, the prevalence of stunting as compared to underweight is higher in Nepal and Bhutan, and it is more striking in the latter.

Table 1: Prevalence of under-nutrition in children under 5 years of age in selected countries of the Southeast Asian region.

Country	*Prevalence (%) of underweight (Weight for age < - 2 SD NCHS/WHO median)*	*Prevalence (%) of stunting (Height for age < - 2 SD NCHS/WHO median)*
Bangladesh	47.7	44.7
Nepal	47.1	54.1
India	47.0	45.5
Maldives	39.0	30.0
Myanmar	35.3	33.9
Bhutan	18.7	40.0
Sri Lanka	29.4	13.5
Indonesia	20.3	-
Thailand	11.3	-

Source: WHO (2002).

Undernutrition is associated with lower immune system, thereby increasing the risk of mortality from infectious diseases such as diarrhoea, pneumonia, measles, and others (Rice et al., 2000). Chronic under-nutrition in the first two to three years of life may also lead to an increased predisposition to obesity, especially to abdominal obesity (see for review Shetty, 2000) as well as to delays in psychological development that negatively affects social and mental performance (WHO, 1999). Undernutrition among adolescents and adults is also associated with adverse health problems and reduced work capacity (Spurr, 1988; Ulijaszek, 1995), thereby resulting in a decreased productivity which leads in turn to poor economic condition. Thus the whole process consists in a "viscious circle" in terms of the interrelationship of poverty, undernutrition, ill-health, economic returns, work capacity and so forth.

OVERNUTRITION/PREVALENCE OF OBESITY

Although undernutrition remains a major health problem in many developing countries, over-nutrition is also emerging with the improvement in socioeconomic condition and increasing urbanization. Consequently, the double burden of under- and over-nutrition exerts considerable impact on the economy and health system in many developing countries (Popkin, 1998, 2002). In general, many countries in Asia are in this situation due to "changing dietary pattern towards energy-dense and high fat diets, together with a more sedentary lifestyle arising from increasing urbanization" (Florentino, 2002). The increasing urbanization, changes in standards of living, dietary patterns, occupational work patterns are the key factors to risks of the epidemic of obesity and associated morbidity and mortality.

A recent review has revealed that India is also characterized by the development and nutrition transition that may contribute to the risk of overweight and obesity, especially in urban areas (Shetty, 2002). Visweswara Rao et al. (1995) reported that the prevalence of overweight among adults in urban colonies of Hyderabad was 21.8% in males and 27.4% in females, while the prevalence of obesity was 2.1% and 8.9%, respectively. It was also observed that the prevalence of overweight and obesity was higher in the higher income groups for both males and females. A study conducted in urban Delhi by the Nutrition Foundation of India also revealed that the prevalence of overweight (defined as > 25 of BMI) among the "middle class" increased from low- to high-income groups, showing that about 32.2% of males and 50.0% of females in the high-income group suffered from overweight (Gopalan, 1998). Both of these studies indicated that the prevalence of overweight and obesity was higher in females than in males.

Another major concern in developing countries is the increasing risk of obesity and associated morbidity and mortality not only in adults but also in children and adolescents. There are good evidences of the adverse effects of childhood obesity on health in childhood and adolescence. Childhood obesity is associated with childhood hypertension, type 2 diabetes, respiratory disease, orthopaedic and hepatic abnormalities (WHO/FAO, 2003). The psychological consequence of childhood obesity is another aspect of a great concern all over the world. Recently, a systematic review revealed that obese children are more likely to have psychological problems than their non-obese counterparts, and girls are at greater risks than boys (Reilley et al., 2003). Moreover, increasing childhood obesity is likely to be a major contributor of ill health in adulthood. There is increasing evidence that adulthood obesity is associated with childhood obesity and associated morbidity.

Obesity is now widely prevalent not only in high-income countries, but also in lower- and middle-income countries with rapid economic growth and increasing urbanization. Thus, although undernutrition remains a major concern, the increasing prevalence of obesity indicates another health burden in many developing countries. A recent analysis of national data from 79 countries indicated that about 88% of under-five children live in developing countries, and the prevalence of overweight (defined as a weight-for-height > 2 SD of the NCHS/WHO reference) in these children was 3.3% or 17.6 million (De Onis and Blössner, 2000). Although the prevalence of overweight was highest in Latin America and the Caribbean (4.4%), followed by Africa (3.9%) and Asia (2.9%), the highest numbers of overweight children was in Asia because about 60% or 10.6 million of the overweight children lived in Asian developing countries. These estimates merit further studies relating to the assessment, prevention and management of overweight and obesity in children. In India, most of the studies are concerned with undernutrition and associated morbidity and mortality, but there is evidence of the increasing prevalence of overweight and obesity, particularly in urban areas (Yajnik, 2004).

"FETAL ORIGINS" HYPOTHESIS

The main exponent of this hypothesis is Prof. DJP Barker and his colleagues from Southampton General Hospital in the UK. According to this hypothesis, cardiovascular diseases, diabetes, hypertension and stroke originate through development of the fetus in response to undernutrition (Barker and Osmond, 1986; Barker, 1992, 1995). The hypothesis was based on the observation made from a follow-up study in Hertfor-dshire, England, which revealed that lower birth weight was associated with higher risks of later ischaemic heart disease and impaired glucose tolerance or type 2 diabetes. Barker and his group have published several papers to support their hypothesis. The underlying principle of the "fetal origins" hypothesis is that human beings are "plastic" and able to adapt to environmental conditions (Lasker, 1969). This "developmental plasticity", or capacity of the individual or fetus to produce more than one alternative form and function, is universal to all living organisms. Human fetus is able to adapt to a limited supply of nutrients by changing its physiological and metabolic mechanisms that are irreversible after adulthood. It is argued that these changes might be the origins of diabetes and coronary heart diseases in later life (Barker, 1998). In response to maternal undernutrition, the fetus may change by either reducing its size to meet the nutrient requirements, or altering the production of hormones, especially insulin, that regulate growth or redistribute the flow of blood in order to protect vital organs such as the brain at the expense of muscle growth - a phenomenon referred to as "thrifty phenotype." Besides, maternal nutritional intake acts like a signal to the fetus that the after birth environment is likely to be plentiful or harsh. Changes made during early life, or sensitive period of development, might have permanent effects on the body form and function in later life, and the whole phenomenon is sometimes known as "programming". As a whole, developmental plasticity during early life is relatively beneficial for growth and survival but at the expense of longevity because such modes of adaptation has far reaching consequences on health in later life.

Birth weight is widely used as an indicator of fetal growth and nutritional status, although the same birth weight might be the result of different growth trajectories. But there is increasing evidence of the inverse relationship between birth weight and increased risk of developing coronary heart disease, hypertension, type 2 diabetes and the metabolic syndrome in later life (See for review Fall, 2001). With respect to catch-up growth after birth, a cohort study of men born in Helsinki during 1934-1944 showed that low birth weight or thinness at birth combined by a rapidly weight gain at 3 years of age was associated with an increased risk of coronary heart disease (Eriksson et al., 2001). It is known that catch-up growth during early childhood is associated with factors relating to intrauterine restraint of fetal growth. The greatest variation in weight velocity or weight gain is generally observed in the first-two years of life, and a rapidly catch-up growth during this period is likely to be a risk factor for childhood obesity (Ong et al., 2000).

Although data on the inverse relationship between birth and NCDs came mostly from the high-income countries, there is also evidence from other countries such as India, China and Taiwan. A study in South India revealed that the prevalence of coronary heart disease decreased from 11% for men and women whose birth weights were 2·5 kg or less to 3% for those whose birth weights were more than 3·1 kg (Stein et al., 1996). High rates of the disease were also observed in the individuals whose mothers had a low body weight during pregnancy. Also, short length and small head circumference at birth were associated with an increa-sed prevalence of the disease. In another report, they (Fall et al., 1998) observed that the prevalence of type 2 diabetes was higher in men and women who were short at birth and high in ponderal index. Short, fat babies of heavier mothers during pregnancy were likely to develop type 2 diabetes, with a lower 30-minute insulin increment, a marker of reduced beta cell function. Barker (1999) explained these findings in relation to the increasing epidemic of type 2 diabetes in urban and migrant Indian populations. Fetal undernutrition is widespread in the Indian population, and this may predispose people to insulin resistance After moving to urban areas, young women gain more weight and become more insulin resistant due to decrease in physical activity levels. "These women would be less able to maintain glucose homeostasis during pregnancy, even at relatively low levels of obesity, and could become hyperglycemic (although not necessarily diabetic). Their children, who would be exposed to high circulating glucose concentrations in utero, may have impaired pancreatic b-cell development and become insulin deficient. These ideas need to be confirmed by further studies in India" (Barker, 1999).

Evidence of the time of insulin resistance has also been reported from India. In their study of

children born in the King Edward Memorial Hospital, Pune, Bavdekar et al. (1999) reported that children with lower birth weight were more insulin resistant at 8 years of age. The highest levels of insulin resistance and LDL cholesterol were in children of low birth weight but high fat mass at 8 years. There is evidence also good accounts of increased insulin levels or hyperinsulinemia among Indian babies at birth (Yajnik et al., 2002). Further, studies have shown that the body composition of Indians with type 2 diabetes is different from that of the white Caucasians. Indians have more central fat and higher percentage of body fat for a given BMI, and they are more insulin resistant as compared to white Caucasians. This characteristic is referred to as "thin-fat phenotype" which origins in fetal life (Yajnik et al., 2002; Yajnik, 2004). All studies stressed the need for improving fetal growth which is directly associated with maternal factors. Accordingly, the obvious strategy is to improve the nutritional and health condition of mothers during reproductive age.

IMPLICATIONS AND CONCLUDING REMARKS

Growth as a quantitative increase in size or mass variation is the total reflection of the interaction between genetics and environment; thereby it may be considered an indicator-variable of either genetics or environment, or a combination of the two. It is measured in terms of anthropometric measurements and indices. In medical and nutritional fields, anthropometric measurements and indices are widely used as indicators of the nutritional status of the population. Universal anthropometric standards and references have also been developed for the purpose of measuring the health and nutritional status of children across populations. The underlying principle is that children especially under five years of age are by and large similar in the so-called "genetic potential" for growth if they are provided with adequate nutrition. Thus the nutritional status of children all over the world is assessed in terms of anthropometric measurements and indices relative to the universal growth standards or references. The results indicate that a large proportion of under-five children in developing or low-and middle-income countries is undernourished. For example, the prevalence of undernutrition in India, as indicated by weight-for-height and weight-for-age, ranges between 45% and 47% of children under five years of age.

In general, this brief review reveals that undernutrition still remains a major concern in developing countries "mainly because of poverty that contributes to house-hold food insecurity, poor maternal and child care, unhealthy environments and poor access to health services" (WHO, 2001). About 50% of deaths among under-five children in developing countries are attributable to undernutrition and its synergetic relation with infections. There is good evidence that undernutrition is associated with lower immune system, thereby increasing the risk of mortality from infectious diseases. Chronic undernutrition in the first two to three years of life may also lead to an increased predisposition to obesity, especially abdominal obesity, and to delays in psychological development. Under- nutrition among adolescents and adults is also associated with adverse health problems and reduced work capacity, thereby reducing a productivity output. Thus the whole process consists in a "viscious circle" in terms of the interrelationship of poverty, undernutrition, ill-health, economic returns, work capacity and so forth.

While the problem of undernutrition is still raging in many developing countries, the epidemic of over-weight and obesity is also spreading at alarming rate especially in those countries with rapid economic development compounded by increasing urbanization. Thus many lower and middle-income countries are facing the double burden of undernutrition and over-nutrition. A major concern in developing countries is the increasing risk of obesity and associated morbidity and mortality not only in adults but also in children and adolescents. There are good evidences of the adverse effects of childhood obesity on health in childhood and adolescence. Moreover, increasing childhood obesity is likely to be a major contributor of ill health in adulthood. Thus the spread of obesity needs to be monitored and prevented, but it should not be done at the expense of the efforts to alleviate undernutrition. Most nutrition programmes in developing countries pay more attention to alleviating undernutrition, especially in providing food complements, without much attention to monitor and prevent the epidemic of obesity that may create more harm in the future generations (Uauy and Kain, 2002).

Understanding of the factors/determinants of childhood obesity is essential for the development of programmes for the prevention and intervention of obesity and associated morbidity and mortality. These include: 1) understanding of the changes in body composition during different phases of growth and development that

may have far consequences later in life, 2) increasing urbanization and impact of market globalization on changes in diets and lifestyles of the people should be taken into consideration.

The applicability of the "fetus origins hypothesis" for understanding the health and nutritional problems in developing countries may not be ruled out. Further studies to test the hypothesis may be needed, especially at a population level taking into consideration different ethnic groups of different ecological settings. There is evidence of the increasing epidemic of obesity, coronary heart disease, hypertension, and type 2 diabetes in India, and it is more common in urban areas. Whether or not this trend is an evidence of the "fetus origins" hypothesis that predicts more NCDs in populations that are undergoing nutrition transition? The higher prevalence of obesity and type 2 diabetes in urban areas as compared to rural areas in India need to be examined carefully. In general, it suggests the importance of both prenatal and postnatal factors. Prenatal factors are mostly related to intrauterine restraints that are directly related to maternal factors. Thus the need for improving reproductive health is obvious. But the question as to what would be the ideal weight gain during pregnancy for women in developing countries is yet to be clearly understood. There are limited studies of the relationship between body size at birth and maternal nutritional intake. A study in Gambia showed that energy supplement increased weight gain in pregnancy as well as birth weight, but the supplementation had no significant effect on birth length (Cessay et al., 1997). In India, it was reported that energy and protein intakes were not associated with birth weight (Rao et al., 2001). Instead, micronutrients might be more important in reducing fetal growth retardation. In fact, the energy needs for women appears to vary enormously depending on nutritional status of the mothers and gestational age (Prentice and Goldberg, 2000).

In India, there is evidence of higher birth weight in urban areas, probably due to better health and nutritional status of mothers, on average, in urban areas as compared with those in rural areas. There is also evidence that heavier mothers during pregnancy tend to have short, fat babies who are likely to develop type 2 diabetes in later life. Thus the available evidence suggests that we are facing the "paradox of increased adiposity at both ends of the birth weight spectrum-higher BMI with higher birth weight and increased central obesity with lower birth weight" (Oken and Gillman, 2003).

There is good account of literature that developmental plasticity originates in fetus and continues throughout different stages of growth and development. It may be recalled that according to Lasker (1969) there are three modes of adaptation: selection of genotypes, ontogenetic modification, and physiological and behavioral response. The selection of genotypes is concerned with the changes in the genetic constitution of the population, while ontogenetic modification refers to a mode of adaptation during growth and development, which is "essentially irreversible after adulthood" and is denoted as 'plasticity" or developmental plasticity. The third mode of adaptation, or physiological and behavioral response, refers to short-term reversible changes, or acclimatization to the immediate environment. All these three modes of adaptation refer to changes that are relatively advantageous or beneficial for the survival, health and well-being of an individual or a group of individuals. However, the last two modes of adaptation may not be heritable, and they have their limits. While acclimatization is helpful for short-term benefits of the individual, developmental plasticity in response to undernutrition during prenatal period may be beneficial for growth and survival of the fetus but at the expense of longevity because it has far reaching consequences on health and survival in later life. Conversely, the better growth performance of immigrant children as compared to non-immigrant children is often cited as good evidence of human plasticity in response to better environmental quality (Boas, 1912; Lasker, 1952; Bogin, 1995). The same is true with regard to the differences between urban and rural children, or the differences between children belonging to the higher and lower socioeconomic groups. The present review acknowledges the significance of the increasing epidemic of obesity and associated morbidity in response to socio-economic growth compounded by increasing urbanization and sedentary lifestyles. Thus, in the present context, under- and over-nutrition look as though they were the limits of develop-mental plasticity. To what extent of developmental plasticity should be considered as relatively beneficial for the long-term survival and well-being is the moot question of future researches.

Schell (1995) suggested two levels of ontogenetic modification or plasticity in growth and development. The first level is concerned with a plastic or "growth response" mainly to the "human-made-environment", while the second level is related to a "plastic adaptation" that promotes survival, functional capacity and repro-

duction. Most of the present evidence of growth patterns is related to improved environmental quality, or human-made-environment, which may not be completely sufficient for promoting survival, functional capacity and reproduction. Evidence for plastic adaptation needs long-terms studies of irreversible changes in the adult phenotype that may be related to survival, functional capacity and reproduction (Lasker, 1969; Bogin 1995). The "fetus origins" hypothesis predicts that developmental plasticity in the utero is not "plastic adaptation" that promotes long-term survival and reproduction.

The question then arises how is to measure plastic adaptation and/or selection of genotypes that promote survival and reproduction? There has been no evidence so far that any scientific method or model could tell the accuracy of the magnitude of the operation of natural selection in natural population. This is perhaps due to the fact that both genetics and environment of our species are constantly changing, although the nature and rates of changes in the latter may be much faster. This does not mean that the concept of adaptation is not important. The concept of adaptation in terms of selection and "relative fitness" (reproductive success) has been undoubtedly beneficial for promoting health and well-being. As Little et al. (1991) suggested, "Scientific interests can be directed towards understanding mechanisms of adjustments or pattern of variation, including processes that contribute to or limit variation." Current evidence for the nutritional status of human population suggests that there must be a "delicate balance" between individual or population and the environment, including the resources and challenges of life (Roberts, 1991). The present review reveals that the rapid changes in dietary patterns and lifestyles associated with economic development, industrialization and market globalization are the immediate determinants of obesity, especially in urban areas. This indicates the existence of imbalance between our genetic make-up and the so-called "man-made environment." Our genetic make-up is the result of many generations through the action of natural selection. Thus the degree or extent of changes in the environment surpasses the changes in the genetic-make up, or genetic capacity to response to such changing environmental conditions, thereby disturbing the delicate balance mentioned above (Eaton et al., 1988). Further researches are expected to throw much more light in this context. Recently, a joint WHO/FAO Expert Consultation Group (WHO/FAO, 2003) has stressed the importance of "life-course approach" in order to prevent and control obesity and associated morbidity and mortality. It suggests that a major concern is relating to changing patterns in diets and lifestyles. Thus the need to change these environments and their associated components, which affect health and well-being, is deserved to receive more attention from all sections of the human society. An effort to change the genetic composition of the population is more difficult than any other effort to change the environment.

Researches are yet to generate more information on the prenatal and postnatal growth processes especially the determining mechanisms that are related to obesity and morbidity. For example, it is suggested that age at "adiposity rebound" may be considered a good indicator of the subsequent risk of obesity. The earlier the rebound, the higher is the adiposity at the end of growth" (Rolland-Cachera, 1998). An accelerated growth during the first-two year of life might be a biological response to a high protein-energy intake (Rolland-Cachera et al., 1999). This is characterized by an early adiposity rebound with early maturation probably due to early production of insulin-like-growth factor I (IGF-I). There is considerable evidence of the importance of dietary intake in raising IGF-1 levels, and protein content of the diet may have a significant effect in fetal and early postnatal growth. Whether an excess protein intake in the first two years of life has long-term effects is an interesting question under active researches. There is good evidence that protein intake during the first two years of life is high in high-income countries. Whether an excess protein intake may also contribute to an early adiposity rebound and subsequent over-weight and obesity merits further researches.

The role of protein intake during the early growth has recently been the focus of attention due to observations linking prolonged breast-feeding with the prevention of overweight and obesity. Protein content of infant formula is much higher than breast-milk, and its widespread use in high-income countries during the period between 1950s and 1980s could be one of the factors for faster growth and early adiposity rebound. Many studies have revealed the significance of breast-feeding in improving the health of both infants and mothers especially in lower and middle-income countries (Kramer, 1981; WHO Study Team, 2001; Kramer and Kakuma, 2002).

The role of nutritional factors in genomic imprinting or epigenetic mechanism that determines growth patterns through "genetic assimilation" may also be taken into consideration in order

to have a better understanding of biological plasticity (Waddingdon, 1967; Pritchard, 1995). A recent study of three cohorts of an isolated community in northern Sweden born in 1890, 1905 and 1920 showed that an excess food intake during the "father's slow growth period" in his childhood was significantly associated cardiovascular morbidity and mortality of his grandchildren (Kaati et al., 2002). It was observed that mortality rate due to diabetes "increased if the paternal grandfather was exposed to surfeit of food during his slow growth period." This may indicate the possibility of epigenetic inheritance through male line that may be associated with cardiovascular morbidity and mortality (Pembrey, 2002).

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KEYWORDS Growth. Developmental Plasticity. Obesity. Morbidity

ABSTRACT This brief review deals with the prevalence of under-and over-nutrition with special reference to the significance of developmental plasticity during growth and development in understanding obesity that is associated with non-communicable chronic diseases (NCDs) in developing countries. Although undernutrition remains a major health problem in many developing countries, over-nutrition is also emerging because of rapid economic development and nutrition transition. The changing dietary patterns compounded by increasing urbanization and sedentary lifestyles are the key factors to increasing risks of the epidemic of obesity and NCDs. Consequently, the double burden of under- and over-nutrition is likely to exert considerable impact on the economy and health system in many developing countries. There is a need for the prevention and intervention of obesity and associated morbidity that may originate

through developmental plasticity in response to the environmental quality during growth and development. Most nutrition programmes in developing countries pay more attention to alleviating undernutrition without much attention to monitor and prevent the epidemic of obesity. Researches are yet to generate more information on the prenatal and postnatal growth processes especially the determining mechanisms that are related to obesity and NCDs. Areas of further studies have been suggested, and it seems that developmental plasticity during growth and development is one of the important aspects concerning the development of the epidemic of obesity especially in developing countries.

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