

Malnutrition, Social Inequality and Natural Selection in Human Populations

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INTRODUCTION

The terms “growth” and “development” are used simultaneously because growth generally refers to quantitative aspects of the increase in size or mass, whereas development refers to either one or both quantitative and qualitative aspects of the changes from immature state to a “highly organized, specialized, and mature state” (Bogin, 1999). In this chapter, growth and development of an individual is defined as a regular process of quantitative increase in size or mass together with the progressive changes and canalization of form and function at a different rate from a single fertilized egg to a complex-multicellular individual of mature state. It is an outcome of the complex interaction between genetic and environmental factors. It is considered the best indicator of health and nutritional status because poor growth and development is associated with different factors that are closely linked to the “overall standards of living and the ability of populations to meet their basic needs, such as access to food, housing and health care” (de Onis et al., 2000). On the other hand, growth and development can also be considered a *means* of adaptation because it is associated with climatic conditions, such as temperature, altitude and hypoxia (Baker, 1969; Schell, 1995). Growth and development is, therefore, considered an indicator variable (Norgan, 2000) because of its *plastic* nature in responses to different environmental conditions including nutritional deprivation and socioeconomic conditions.

Despite an overall progress in reducing malnutrition among under-five children for the last 35 years, malnutrition still remains a major health problem in developing countries (Smith and Haddad, 2000; de Onis et al., 2000). Malnutrition, defined by a poor anthropometric status, or growth reduction, is associated with about 50% of the total deaths among under-five children in developing countries (Rice et al., 2000). The underlying cause of malnutrition is poverty compounded by poor environmental conditions and unequal access to material assets and health facilities. The poor or lower socioeconomic

groups have often a higher prevalence of malnutrition than the richer or higher classes (WHO, 2000a), regardless of how we define “lower” and “higher” hierarchies by income, occupation, educational attainment, social position, and so on. Therefore, the question arises whether malnutrition, through social stratification or inequality, acts as a powerful agent of natural selection in developing countries?

This chapter attempts to describe growth and development as a measure of health and nutritional status as well as a good evidence of human plasticity in response to different environmental conditions. An attempt has also been made to describe social inequality as a possible mediator of natural selection that is often responsible for different modes of adaptation.

MALNUTRITION

The term “malnutrition” is no exception to those highly ambiguous terms in scientific literature. It is defined as a pathological condition characterized by inadequate intake of macro- and/or micro-nutrients and by frequent infections or diseases. In the present chapter, the term “malnutrition” is used interchangeably with “under-nutrition” - a pathological condition due to inadequate intake of one or more essential (macro and micro) nutrients, which is synergistically associated with various infections or diseases. Indeed, malnutrition refers not only to inadequate dietary intake or under-nutrition but also to over-nutrition characterized by obesity (i.e., excess accumulation of body fat) and its associated morbidity (or co-morbidities), such as diabetes mellitus, cardiovascular disease, hypertension and stroke, osteoporosis, and some forms of cancer. In addition, malnutrition also includes mal-absorption and imbalance between essential nutrients. According to Jelliffe (1966), malnutrition is a “pathological state resulting from a relative or absolute deficiency or excess of one or more essential nutrients” (Jelliffe, 1966). Thus, the term “malnutrition” refers to under-nutrition, over-nutrition, specific nutrient deficiencies or imbalance

(Chen et al., 2001). However, the major nutritional problems are under-nutrition (including starvation and specific deficiency) and over-nutrition. Obesity along with its co-morbidities is an epidemic in developed countries (WHO/FAO, 2003). Although under-nutrition remains a major health problem in many developing countries, over-nutrition is also emerging because of the rapid economic growth and nutrition transition. Consequently, the double burden of under- and over-nutrition exerts considerable impact on the economy and health system in many developing countries (Khongsdier, 2005b and references therein).

ANTHROPOMETRIC MEASURES OF MALNUTRITION

Malnutrition can be measured by a number of ways including anthropometry, dietary intakes, clinical observations, hematological tests, and other biochemical markers. Anthropometry is the measurement of human morphology or body dimensions and proportions. It is a classical technique used by anthropologists for comparative studies of human forms at a population level. Nowadays, it is widely used for monitoring and assessing the nutritional status of an individual or population. In comparison with other methods, anthropometric measurements and indices are more sensitive to different levels of malnutrition. On the other hand, clinical and biochemical indicators are useful only at the extreme levels of malnutrition. Another advantage of anthropometry is that the methods and techniques of data collection are easier and less expensive as compared to other clinical and biochemical methods. The main drawback of anthropometry is its lack of precision and specificity, especially at the individual level, because body measurements are sensitive not only to inadequate nutrition and/or infections but also to climate, toxicants, psychological stress and genetic factors. The changes in body dimensions and proportions are, therefore, an expression of the interplay between genetic and environmental factors (Eveleth and Tanner, 1990; Bogin, 1999). Nevertheless, anthropometry is the most useful tool for growth monitoring and assessment of nutritional status (WHO Working Group, 1986).

Anthropometric indices, such as weight-for-age, height-for-age and weight-for-age are widely used for assessing the nutritional status of children (WHO, 1995). These indices are expressed as a standard deviation (SD) or Z-score of a child's measurement to the median weight of the reference population. The Z-score of - 2 is generally considered as the cut-off

point for screening the individuals who are likely to be malnourished. The formula for SD or Z-score is as follows:

$$Z = \frac{\text{Child's measurement} - \text{Reference median}}{\text{Reference SD}}$$

where

Child's measurement = height or weight of a given child at age X

Reference median = mean or 50th percentile of the reference population at age X

Reference SD = standard deviation of the reference population at age X

Recently, the National Center for Health Statistics (NCHS) in the Centers for Disease Control (CDC), US Department of Health and Human Services, has given a revised version of the NCHS growth references known as CDC growth charts (Kuczmarski et al., 2000). In this revised growth references, the growth charts are smoothed using the Lambda-mu-sigma (LMS) method (Cole, 1990). The tables contain the L (power in the Box-Cox transformation), M (median) and S (standard deviation) parameters for deriving the exact percentiles and Z-scores. Following is the formula for obtaining the Z-score for a given measurement relative to the CDC revised growth references:

$$Z = \frac{((X/M)**L) - 1}{LS}$$

where, X is the physical measurement (e.g., weight, height, etc.) and L, M and S are the reference values corresponding to a given age in months. The revised CDC growth reference has not been endorsed for international use, although some studies have used it for assessing the growth and nutritional status of children (Khongsdier and Mukherjee, 2003a; Maleta et al., 2003).

For children, anthropometry is basically used as a tool for measuring growth rate at any given age in terms of linear measurements (e.g., height). The delay or reduction in growth, as indicated by anthropometric measurements and/or indices, is known as growth retardation. Growth retardation among under-five children in developing countries is often attributed to environmental factors, including inadequate nutrition, infections and poor socioeconomic conditions. In this sense, growth retardation is synonymous with malnutrition (Beaton et al., 1990; UNICEF, 1998), besides being indicative of poor socioeconomic status. Low height-for-age relative to the reference population is an indicator of growth retardation, or stunting (short stature) relative to a given age. Stunting is a result of slow skeletal growth,

which is generally a long-term response to nutritional and socioeconomic deprivation (WHO Working Group, 1986; WHO, 1995).

In addition to linear measurements, body composition, or different components of the total body mass, is also sensitive to inadequate nutrition and/or infections. Therefore, anthropometric measurements, such as weight, skinfold thicknesses, arm and hip circumferences are commonly used to assess the nutritional status of both children and adults. For example, a reduction in body weight is a common manifestation of inadequate intake of nutrition. Such a decrease in body weight is associated with a reduction in body fat mass (body fat stores) and lean tissues (fat-free mass), besides a disproportionate loss of skeletal tissues and a tendency to overhydration of the body, depending upon the time and levels of malnutrition (Shetty, 1995). This reduction in body weight is, therefore, indicative of malnutrition, after appropriate allowances are made for age, sex, height, etc. For children, weight-for-height index is widely used as a practical measure of fatness or body fat stores relative to height. Low weight-for-height is an indicator of thinness or wasting characterized by a deficit in body fat mass and other tissues. Wasting is generally a short-term response to nutritional and socioeconomic deprivation. It can develop rapidly under poor environmental conditions, but it can also be restored rapidly under favourable conditions. Along with weight-for-age index (i.e., a composite measure of body mass relative to age), weight-for-height index is very important for assessing the current health and nutritional status of a given individual or population (WHO Working Group, 1986). Weight-for-height offers advantage over other indices because it does not take into account the child's age, especially in developing countries where it is often difficult to get accurate information on the age of children (Zere and MacIntyre, 2003).

As for adults, Quetelet or body mass index (BMI = weight in kg divided by height squared in metres) is widely used as a measure of fatness, or the nutritional status of populations in both developed and developing countries. On the basis of data from developed countries, BMI ranges of 25-30 and > 30 kg/m² are considered to be indicative of overweight and obesity, respectively (WHO, 1995). The BMI < 18.5 kg/m² is widely used as a practical measure of chronic energy deficiency (CED), i.e., a "steady" underweight in which an individual is in energy balance irrespective of a loss in body weight, or body energy stores (Khongsdier, 2005a). Such a "steady" underweight is likely to be associated with morbidity,

or other physiological and functional impairments (Shetty and James, 1994; WHO, 1995), despite certain limitations of BMI as an indicator of body energy stores (Khongsdier, 2005a). It may be noted that weight-for-height is similar to BMI as it measures thinness or fatness. It is now recommended that BMI should be used among all age groups from childhood to adulthood (Kuczmarowski and Flegal, 2000).

DEVELOPMENTAL PLASTICITY

Adaptation is a generic term for different modes of responses and adjustments to the environment. According to Baker (1984), "adaptation is simply any biological or cultural trait which aids the biological functioning of a population in a given environment. Thus, it includes such aspects as a population's health, ability to feed itself adequately, functional capability in its physical environment and reproductive performance." Adaptation should not be only necessary but also relatively beneficial (Mazess, 1975). Thus, adaptation may be defined as any necessary or beneficial trait, or strategy, by which an organism fits to its environment for its survival, health, functional capability and reproduction. According to Lasker (1969) there are three modes of adaptation: selection of genotypes, ontogenetic modification, and physiological and behavioural response. The selection of genotypes is concerned with the changes in the genetic constitution of 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*, or *phenotypic plasticity*. The third mode of adaptation, or physiological and behavioural response, refers to short-term reversible changes, or acclimatization to the immediate environment. All these three modes of adaptation refer to responses and adjustments that are relatively necessary or beneficial for survival, health and well-being of an individual or group of individuals, although the last two modes of adaptation may not be necessarily heritable, and they have their limits or trade-offs (Khongsdier, 2005b).

According to Barker (2004), "within the limits imposed by its genes and by mechanical constraints, each individual has a range of options for its life history and final body form and function. The formal definition of developmental plasticity is "the ability of a single genotype to produce more than one alternative form of structure, physiological state or behaviour in response to environmental conditions."

The *genotype* is the genetic-make up of an individual, whereas *phenotype* refers to any observable characteristic of an individual such as morphological, physiological and behavioural traits. In this chapter, *developmental plasticity* is defined simply as any observable modification or phenotypic variation in response to a sequence of environments during growth and development or throughout life.

The relationship between genotype and phenotype is very vital for understanding developmental plasticity. We know that the phenotype is the developmental outcome of the complex interaction between genotype and environment. The development of any phenotype depends on the genotype that determines a range or set of alternative phenotypes in response to a sequence of environments. This range is known as the range of reaction, or *norm of reaction*, of the genotype (Dobzhansky, 1970). It is also defined as the mapping function of environment into phenotype for a given genotype (Lewontin, 2004). The norm of reaction of each genotype is highly unpredictable because it encompasses a set of alternative modifications, or outcomes (phenotypes), depending upon a sequence of environments in relation to that genotype. Experimental works on plants show that a given genotype produces different phenotypes according to different elevations from sea level. In addition, the relative height of different plants was unpredictable from one environment to another. For example, the genotype of a given plant that grew tallest at low elevation was the shortest at medium elevation and the second tallest at high elevation (Zuzuki et al., 1981; Lewontin, 2004).

LIMITS OF DEVELOPMENTAL PLASTICITY

It is suggested that there are two levels of ontogenetic modification or developmental plasticity. The first level is concerned with a “growth response” mainly to the “human-made-environment”, while the second level is related to a “plastic adaptation” that promotes survival, functional capacity and reproduction in relation to physical environment (Schell, 1995; Bogin, 1995). Most of the present evidence for 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. The better growth performance of immigrant children as compared to non-immigrant children is often cited as good evidence of human “growth response” to better environmental quality. The same is true with respect

to differences between urban and rural children, or between children belonging to the higher and lower socioeconomic groups. Evidence for “plastic adaptation” needs long-term studies of irreversible changes in the adult phenotype that may be related to survival, functional capacity and reproduction (Lasker, 1969; Bogin 1995).

There is considerable evidence that human fetus is able to adapt to a limited supply of nutrients by changing its physiological and metabolic mechanisms that are irreversible after adulthood. However, such a mode of adaptation or developmental plasticity is not “plastic adaptation” that promotes a long-term survival and reproduction. It is suggested that these changes are the origins of diabetes and coronary heart diseases in later life (Barker, 1992, 1998). In addition, it is also argued that smaller body size may offer an advantage to individuals under nutritional deprivation because smaller body size requires less energy and nutrients, but it may be disadvantageous with respect to greater susceptibility to infectious disease, lower physical work capacity, poor cognitive development (Spurr, 1988; Ulijaszek, 1995). Thus, although individual genotype has the ability or capacity to develop and express an array of alternative phenotypes, such capacity has some limits or trade-offs set mostly by stabilizing natural selection. In addition, some genotypes, such as those for the ABO-blood groups are rigid, while other genotypes for polygenetic traits, such as height, weight, etc., are more flexible or plastic, which are the subject matter of the present chapter.

Several studies have revealed that children in high-income countries are growing taller, but at the same time they are developing obesity (Gortmaker et al., 1987; Chin et al., 1998) that is associated with non-communicable chronic diseases. The finding of a recent study in Taiwan is a good example of such a U-shaped relationship between birth weight and risk of type 2 diabetes (Wei et al., 2003). In addition, studies in the USA have indicated that high birth weight is also associated with an increased risk of obesity among adult women (Leong et al., 2003) and children (Gillman et al., 2003). It is also reported that high birth weight is associated with a number of childhood cancers (Yeazel et al., 1997; Schüz et al., 2001; Hjalgrim et al., 2003) and rheumatoid arthritis (Jacobsson et al., 2003). In short, there is considerable evidence of the limits of developmental 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.

CANALIZATION

The other aspect of developmental plasticity is canalization, the concept introduced by Waddington (1943, 1961). Canalization is defined differently by different authors (Gibson and Wagner, 2000; Debat and David, 2001). According to Debat and David (2001), canalization refers to “a set of processes historically selected to keep the phenotype constant” despite genetic and/or environmental variations. With respect to the phenotypes of polygenic traits that are commonly used in nutritional and medical fields, it may be subjectively assumed that neither too big nor too small is necessary good for health and well-being (Khongsdier, 2005b). The phenotypes of weight and height, for example, vary considerably within and between human populations depending upon genetic and environmental conditions (Eveleth and Tanner, 1990). On the other hand, the variation in these phenotypes is constantly maintained mostly by stabilizing selection in spite of the variation in genetic and/or environmental conditions. Thus, the reaction norm of genotypes (the potentiality of genotypes to produce a range of phenotypes, depending upon environments) may be assumed to operate in accordance with the process of canalization. The significance of canalization is its reduction in variability of a trait that is associated with a complex genetic basis (Gibson and Wagner, 2000). Canalization, however, may not be necessarily static, rather it is considered to be adaptive not only because of stabilizing selection, but also because of directional selection. For example, secular trend in height is a common phenomenon in populations with improved living conditions, thereby canalizing a new pathway. Nevertheless, the success or failure of a given phenotype is conditioned by the reaction norm of genotype. A better understanding of developmental plasticity and canalization is of considerable importance to many aspects of organic life, especially to the health and well-being of human populations.

MEDICAL AND ADAPTABILITY MODELS OF DEVELOPMENTAL PLASTICITY

Medical model of developmental plasticity is the most common model used in public health, nutritional science and pediatrics. According to this model, growth retardation is an indicator of poor nutritional status, or a failure in the expression of the so-called *genetic potential for growth* (Gopalan, 1992). On the other hand, the full expression of *genetic potential for growth* is indicative of good

health and nutritional status. 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, the growth curves of well-nourished children in high-income countries are 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 nutritional status of all other groups of children. For this purpose, international standards, or growth references, such as the NCHS growth references, endorsed by the World Health Organization (WHO, 1983, 1995), are 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. Thus, the medical model uses growth “as a measure of health and of adaptive success” (Schell, 1995).

Unlike the medical model, the human adaptability model views growth as the mechanism of developmental plasticity for “achieving an adapted state rather than a result of that adaptation” (Schell, 1995). In other words, the human adaptability model considers growth as a means of adaptation. It has long been established that human weight and height are correlated with climatic conditions, thereby confirming the applicability of Bergmann’s and Allen’s rule to humans (Roberts, 1953, 1978; Ruff, 1994; Bindon and Baker, 1997). There is also considerable evidence of developmental plasticity to high-altitude hypoxia (for reviews see Greksa, 1991; Schell, 1995). According to Frisancho (1976), the pattern of accelerated growth of the oxygen transport systems as well as the retarded growth in high altitude regions may be part of the functional adaptation. Schell (1995) summarizes, “the examples from studies of developmental responses to thermal stress and to high-altitude hypoxia are sufficient to indicate that plasticity must be recognized as a possible mode of adaptation. Thus, the adaptability model of growth cannot be dismissed in favour of the medical model.”

Each model of developmental plasticity is compelling when considered alone. Schell (1995) has suggested that although it is difficult to use growth and development simultaneously as a means and a measure of adaptation, one may “apply the medical

model to studies of adaptation to a feature of the human-made environment, and the adaptability model to studies of adaptation to a feature of the physical environment.” He has also pointed out the lack of progress in testing and refining the human adaptability model as compared to the medical model due to lack of funding rather than to demerits of the model. One of the problematic tests of adaptability model is the “small but healthy” hypothesis proposed by Seckler (1982).

On the basis of his observation on the populations of India and Nepal, Seckler (1982) suggested that most of the children treated as mild and moderate undernourished, according to height for age relative to international growth references, should be regarded as “small but healthy.” According to his observation, “about 90% of all the malnutrition found in these countries are those people with low height for age but *with proper weight for height ratio* (author’s italics). Now, if one thinks of malnutrition in the conventional imaginary of thin, wasted bodies, rather than in terms merely of short people, the incidence of malnutrition must be considerably reduced. Of course, since short people with proper weight to height ratio will also be light people, their consumption requirements will also be less than conventionally estimated.” Seckler was of the opinion that there were no functional impairments in the range between mild and moderate malnutrition as defined by the international growth references, “because this range represents an adaptive response of body size to adverse conditions *in order to avoid these impairments*” (author’s italics). Accordingly, he suggested that appropriate population references for the assessment of malnutrition should be lower than the recommended reference values predicted under the concept of genetic potential for growth. Payne (1992), though in a different way, also supported that the scientific concept of nutrition should concern not with the failure of meeting some normative targets, but with the failure of maintaining the functional capabilities relative to nutritional intake. On the contrary, most of the individuals below the reference values as proposed under the concept of genetic potential for growth do not show such functional impairment. Payne (1992) criticized the genetic potential theory for supporting the armpit of obesity and associated risks of non-communicable chronic diseases.

The “small but healthy” hypothesis has been severely criticized by many scholars (Gopalan, 1992; Bogin, 1995, 1999), but its significance lies with the concept of small body size. Tanner (1978) warned

against assuming that being small is necessarily bad. “Though rate of growth remains one of the most useful of all indices of public health and economic well-being in developing and heterogeneously developed countries, it must not be thought that bigger, or faster, is necessarily better.” The advantage of small body size is that it enables a person to survive and sustain his level of activity in a given habitat of nutritional constraint, because a smaller body requires less energy. However, if small body size predisposes to infections, and is associated with low level of productivity and poor cognitive development; it proves to be disadvantageous (Spurr, 1988; Bogin, 1995; Ulijaszek, 1995). On the basis of their study of three Gurung villages in Nepal, Strickland and Tuffrey (1997) pointed out, “Were Seckler’s hypothesis to be reworded ‘small individual, but healthy household’, this study would have gone some way towards supporting it. Although “short people are less fit, less productive, and likely to be less reproductively successful, . . . these disadvantages to the short individual did not appear clearly and simply to translate into social economic disadvantage for the household.” Thus, Strickland’s and Tuffrey’s study suggests the possible existence of human plasticity to minimize the lineage extinction, but at the expense of individual physical disadvantages.

Although Seckler’s hypothesis has many weaknesses, it cannot also be denied that the main focus of the hypothesis is on short stature, which is related to the conflicting interpretations between medical and adaptability models of developmental plasticity. Is short stature (or height) really a matter of concern? On the basis of their review on the advantages and disadvantages of short stature, Strickland and Tuffrey (1997) summarized, “disregarding their significance and cause, ‘costs’ associated with short stature outnumber ‘benefits’”. However, reported outcomes seem often to be population and environment specific, possibly because in situations where the environment is ‘improving’, the trend is for secular increase in height, while in deteriorating environmental circumstances, the secular trend is in the opposite direction. This would imply that some indices of health cannot be universally employed except at the extremes of survival.” Recently, a review by Samaras et al. (2004) has revealed that “shorter people have substantially lower rates of CHD (coronary heart disease) mortality and moderately lower levels of stroke mortality.” Developmental plasticity has, therefore, its limits or trade-offs, and the reaction norm of genotypes, or in the present context, the range of height and weight

phenotypes determined by multi-genetic factors in response to different environments, does look as though they were stabilized by the mechanisms of canalization.

INEQUALITY/SOCIAL STRATIFICATION

Inequality is defined herein as the difference between social groups or classes in access to food, health, education, wealth and other opportunities that often makes the disadvantaged to become more disadvantaged. It is often used as synonymous with the term "disparity" that is commonly used in the USA. The concept of equality/inequality is different from that of equity/inequity because the latter has a moral and ethical dimension. Equality means the absence of inequality, while equity refers to fairness or social justice. Identifying health inequities is more difficult because it involves normative judgement, which varies across individuals and societies. Nonetheless, nutritional or health inequity can be broadly defined as a disparity in nutrition and health due to the absence of fairness or social justice in the society (Kawachi et al., 2002).

Conventionally, anthropologists have recognized three major categories of human society with respect to inequality: egalitarian, rank and stratified societies. In *agrarian societies* (hunting and gathering societies), there is an absence of social groups with greater access to economic resources, power or prestige; but there exists an individual variation in age, sex and abilities such as health, hunting skill, intelligence, and physical prowess. *Rank societies* are characterized by social groups with unequal access to prestige or status but not to economic resources or power (Ember & Ember, 1990). In *stratified societies* or class societies, there are group differences in access to economic resources, health, education, power and prestige; although there are different categories of stratified societies ranging from open to more or less closed class or caste systems. In open societies, there are no hindrances for the individuals to move from one class to another. On the other hand, in societies with caste system, membership to a particular caste, generally associated with occupation, is determined and sanctioned by religion at birth and marriage is often restricted to members of one's own caste, although it is not as rigid as before in the context of the present Indian society (Béteille, 2002).

The development of social stratification is not fully understood. It is suggested that most of the prehistoric human societies were by and large

egalitarian, despite the persistence of certain hunting and gathering societies in modern times. Rank societies were more common during 10,000 to 15,000 years ago. The emergence of stratification is more or less synonymous with the advent of civilization and the state formation beginning about 5000 years ago (Cohen, 1998). Although individual inequalities could influence the health, nutrition and survival of persons in the egalitarian societies, the change from egalitarian to civilized and industrialized societies has brought about both negative and positive progresses in terms of improved health and nutrition. It is suggested that social stratification, which is an inherent part of civilization, exacerbates different health problems by reducing the resources available to the poor far below the level and by allowing the rich to exert disproportionate demand for luxury goods (Cohen, 1998).

SOCIAL INEQUALITY AND MALNUTRITION

Malnutrition affects all sexes and ages. What makes the situation more serious is that children under 5 years of age are the most vulnerable victims. Malnutrition predisposes an individual to infection and vice versa. It is one of the major risk factors for infections and diseases (WHO, 2000a). About 50% of the total annual deaths in children under 5 years of age are associated with malnutrition in developing countries (Rice et al., 2000; WHO, 2000b). Malnutrition is attributable not only to poor access to food but also to other poor environmental conditions, such as poor housing and hygienic conditions, unsafe drinking water, heavy workloads, lack of preventive and control measures of locally endemic diseases and infections (Khongsdier, 2002). These poor environmental conditions are the common characteristics of population groups belonging to the lower socioeconomic strata of the society, especially in developing countries (Onis et al., 2000). In other words, the major cause of malnutrition is poverty compounded by other poor environmental conditions that predispose an individual to morbidity and mortality. There is considerable evidence that children in the lower socioeconomic groups especially in developing countries are often the victims of malnutrition and its associated morbidity and mortality (WHO, 2000a). Malnutrition is, therefore, one of the important indicators of health inequality in developing countries.

The tragic consequences of malnutrition include death, disability, stunted or retarded physical growth, thereby affecting the national socioeconomic

development (WHO, 2000b). Children with poor socioeconomic conditions are more undernourished than their counterparts in the higher socioeconomic strata of societies. Despite improvement in agricultural productivity in the 20th century, millions of people in developing countries still remain poor and undernourished because food is “neither produced nor distributed equitably” (WHO, 2000a). This problem remains a major setback to the “recognized fundamental human right to adequate food and nutrition, and freedom from hunger and malnutrition, particularly in a world that has both the resources and knowledge to end this catastrophe” (WHO, 2000b).

SOCIAL STRATIFICATION AS A MEDIATOR OF NATURAL SELECTION

Natural selection acts primarily at the individual level. The simple definition of natural selection given by Darwin (1859) is the “preservation of favourable individual differences and variations, and the destruction of those which are injurious.” He further clarified that “under the term of “variations,” it must never be forgotten that mere individual differences are included.” Thus, natural selection operates primarily at the individual level through differential survival and reproduction. The aggregate or average differential survival and reproduction of a given number of individuals may be considered its action at a group or population level.

The moot question is that whether being poor is also indicative of being victims of natural selection? There is considerable evidence that the health and nutritional status of the poor is worse than is the rich. Mortality rates due to malnutrition, infections and other causes of deaths are much higher in the lower socioeconomic classes. The significance of these inequalities also influenced the writings of Malthus (1803) and Darwin (1871, 1859). According to Malthus (1803), the “constant tendency in all animated life to increase” would prevent any permanent amelioration of poverty in the lower classes. In Central and South Asia, the positive checks including epidemics and consequences of “indigence and bad nourishment” would fall heavily on those in the lowest socioeconomic strata “before any considerable degree of want had reached the middle classes of the society” (Malthus, 1803). Acknowledging this important observation of Malthus, Darwin (1871) wrote, “As all animals tend to multiply beyond their means of subsistence, so it must have been with the progenitors of man; and this would inevitably

lead to a struggle for existence and to natural selection.” Although Darwin did not say that natural selection is stronger among the poor, he also observed the “greater death-rate of infants in the poorest classes... as well as the greater mortality, from various diseases, of the inhabitants of crowded and miserable houses, at all ages” (Darwin, 1871). It was Franz Boas (1938) who argued that natural selection in humans operates primarily through social stratification. In addition, malnutrition, associated with poor environmental conditions in the lower socioeconomic strata, is suggested to be a strong force of natural selection especially among children and reproductively-active women (Segraves, 1977). Thus, the view that social inequality mediates the process of natural selection in human populations seems to have originated with Darwin himself (Strickland and Tuffrey, 1997).

Natural selection is a blind natural force that preserves the beneficial variations and eliminates the injurious ones. The process of preserving the beneficial variations is also known as the *survival of the fittest* in the *struggle for existence*. According to Malthusian and Darwinian points of view, the struggle for existence, or competition for survival, is due to the increase in population beyond the means of subsistence. The short supply of resources, therefore, increases competition in different forms including social stratification in which “members of the privileged class may own even up to or over 10,000% of what a poor person owns” (Cohen, 1998). The high prevalence of malnutrition and infections is a clear evidence of poor access to adequate nutrition and health amenities among the lower socioeconomic classes. From this point of view, one may argue that social stratification mediates natural selection in human populations in the form of malnutrition and infections, which ultimately lead to higher morbidity and mortality in the lower strata of social stratification. However, this argument is based simply on differential survival or *survival of the fittest* due to limited resources mediated by social stratification.

Natural selection or *survival of the fittest* also occurs whenever two or more individuals of distinct genotypes transmit their genes to the succeeding generations at different rates, despite the absence of limited resources (Birch, 1957). Any population is capable of increasing in number only when the progeny are able to survive and reproduce from generation to generation. Thus, according to the genetic point of view, natural selection operates in human population through differential reproduction (fecundity, fertility and mortality) among individuals

of distinct genotypes. Considering both differential fertility and mortality, one may argue that reproductive success is lower in the higher socioeconomic groups than in the lower socioeconomic groups because fertility and mortality rates are lower in the former than in the latter. Indeed, there might not be a large difference between low and high socioeconomic classes in differential reproduction because a higher mortality rate among the low socioeconomic class is compensated for by a higher fertility rate. There is considerable evidence that fertility rates are higher in the lower socioeconomic groups than in the higher ones. "This situation is undesirable, irrespective of any genetic considerations. People who should be able to provide the best environment for the physical and mental development of their children produce fewest progeny" (Dobzhansky, 1962). To understand the implications of these empirical evidences, we may take into consideration adaptation as an explanatory paradigm (Little et al., 1991).

PLASTICITY AND SOCIAL INEQUALITY

From the genetic point of view, natural selection often results in adaptation when genotypes of superior adaptability have a Darwinian fitness, or reproductive fitness, higher than do the genotypes of lesser adaptability (Dobzhansky (1972). Thus, adaptation is closely related to the concept of Darwinian fitness, which is measured in terms of reproductive performance of the individuals or populations. It is, however, difficult to detect genetic adaptation in human populations, except in some cases, such as the coincidence of abnormal hemoglobins and other red cell anomalies with the distribution of malaria (Allison, 1964; Smith, 1998). As for social stratification, there is no clear evidence that social classes of modern civilization are genetically maintained even in closed classes or castes (Dobzhansky, 1962; Holtzman, 2002).

According to adaptive systems theory, parents living in risky and uncertain environment maximize the current reproduction in terms of the *quantity* of offspring to minimize the risk of lineage extinction because of high mortality; while parents living in good environmental conditions maximize the *quality* of their offspring by reducing the quantity of the current reproduction (Chisholm and Burbank, 2001). Consequently, the future reproductive success of the parents under good environmental conditions is higher than that of the parents under poor environmental conditions because the high quality offspring are

more likely to survive and reproduce from generation to generation. More studies are needed to know whether the higher fertility and mortality rates in the lower classes are a form of plasticity to minimize the lineage extinction at the cost of high mortality? There is also a possibility of minimizing the lineage extinction at the expense of individual physical disadvantages (Strickland and Tuffrey, 1997). However, such a form of plasticity, if any, is because of necessity rather than for long-term benefits of populations.

The point to be noted here is that humans have the capacity to adapt to a wide range of environmental stresses. Malnutrition and infections are those environmental stresses by which natural selection is operating in human populations. In the process, natural selection is often responsible for different modes of adaptation. All the three modes of adaptation suggested by Lasker (1969) refer to modifications or traits that are relatively necessary or beneficial for survival, health and well-being, but the last two modes of adaptation have their limits. While acclimatization is helpful for short-term benefits of the individual, developmental plasticity in response to under-nutrition 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 (Barker, 1992, 1995). A smaller body size may also offer an advantage to individuals under nutritional deprivation because smaller individuals require less energy and nutrients, but it may be disadvantageous in other respects such as greater susceptibility to infectious disease, or lower physical work capacity (Ulijaszek, 1995; Norgan, 2000). Similarly, in view of the adaptive systems theory, although physiological capacity to reproduce is necessary for minimizing the lineage extinction in the lower class, such a mode of adaptation is at the expense of ill health and higher mortality. Natural selection that operates in the lower strata of social stratification does not result in a long-term beneficial adaptation. As for the upper class, Harrison (1998) points out, "any physiological ability facilitating access to better environments will be strongly favoured through the greater success, reproduction and offspring survival which the better environments are likely to promote . . . Darwinian fitness will tend to be highest in the upper class, especially in the absence of contraception, and physiological ability can influence the probability of being in those classes" through its effects on health and functional capability.

DELICATE BALANCE/TRADE-OFF

Social inequality is not the only mediator of natural selection in human populations. As a blind natural force, natural selection may act even at the slightest variation in physical forms, physiological function and genetic constitution due to interaction with different factors including climate, nutrition, social stratification and other challenges of life. Thus, there should be a “delicate balance” or a trade-off between our genetic/biological make-up and the environment to maintain good health and functional capability (Khongsdier, 2005b). Any deviation from some threshold of such delicate balance is a predisposition to ill health and ultimately to mortality or action of natural selection. Current evidence for health inequalities according to social stratification are indicative of huge differences between the rich and the poor in degree of attaining a delicate balance, or a harmonious relationship between genotypes and environments, that everyone has the fundamental rights to achieving it. Physical, cultural and social environmental factors are often documented to be responsible for health and nutritional inequalities between the rich and the poor, the educated and the uneducated, the disadvantaged and the advantaged, the upper caste and the lower caste, and so on.

There is no biological justification for social inequality in health and nutritional status. The individual capacity for intellectual development are genetically conditioned, and is “one of our biological traits of our species essential for its survival” (AAPA, 1996). It has been suggested for the last 30 years that about 85% of human genetic diversity is due to individual differences between and within the same population, whereas differences among major human groups account for only 5 to 10% of the overall genetic variance (Lewontin, 1972; Jorde et al., 2000; Rosenberg et al., 2002). Recent studies of DNA markers have also confirmed, although they do not rule out the possibility of certain genetic discontinuity between continental groups of mankind (Romauldi et al., 2002; Edwards, 2003; Hinds et al., 2005). Each individual has a unique genotype qualifying him or her for learning or carrying out a number of roles and functions in the society. Equality of economic opportunity in a society, for example, enables a man to choose any occupation, which is most suited for him by his abilities and willingness to strive. A person of outstanding ability in music or sports has no doubt a unique genotype, but such a genotype is not inherited from his or her parents. In fact, “we inherit genes, not genotypes, of our parents, and we transmit our genes,

not our genotypes, to our children” (Dobzhansky, 1962). Thus, what we inherited from our parents is an array of “alternative pathways of development” being contingent for their outcomes upon the environments (Lewontin, 2004). In short, individual phenotypes such as morphological, physiological and behavioural characteristics are a unique outcome of the *norm of reaction* of the genotypes with its environment during growth and development and throughout life. Knowing such a norm of reaction is a remarkable task for the Human Genome Project especially in determining who will get what disease and in finding out ways of choosing the appropriate medication for an individual patient. Indeed, the biggest task is to show the mapping of a unique genotype into a set of complex-alternative behaviours, irrespective of progress in identifying single-gene disorders that result in mental retardation such as phenylketonuria, etc.

Each individual has a unique genotype, and each phenotype is a unique outcome of the *norm of reaction* of the genotype with the environments (including socio-environments) that is experienced differently by different individuals. The genotype or descriptor of the individual genome is formed as a matter of chance depending upon the assortment, recombination and interaction of a constellation of genes or DNA molecules inherited from the parents. In addition, the developmental outcome of any genotype in the form of phenotype is “a unique consequence of the interaction between genome and environment” throughout life (Lewontin, 2004). More than 40 years ago, Dobzhansky (1962) wrote, “my phenotype at this moment has been determined by the norm of reaction of my genotype to the succession of environments that I have met in my lifetime; my phenotype tomorrow, or a year hence, will be determined by its present state, as modified by my responses to the environments that I shall have encountered in the meantime.”

SUMMARY

Malnutrition is often measured by a poor anthropometric status, i.e., poor anthropometric indices are used as indicators of malnutrition. Anthropometric indices such as weight-for-age, height-for-age and weight-for-height, relative to a recommended population reference, are widely used in assessing the nutritional status of children. On the other hand, BMI is frequently used for the assessment of the adult nutritional status. Anthropometric measurements and indices are basically used for

quantitative analysis of body forms as well as growth and development. In this sense, growth and developmental retardation is also considered as synonymous with malnutrition.

Growth and development of an individual is a regular process of quantitative increase in size or mass together with the progressive changes and canalization of form and function at a different rate from a single fertilized egg to a complex-multi-cellular individual of mature state. It can be considered as an outcome of the complex interaction between genetic and environmental factors. One of the commonly observable characteristics of growth and development is its plasticity in response to the environment. This plasticity is known as *phenotypic plasticity* or *developmental plasticity*, which is the outcome of the *norm of reaction* in which a given genotype could produce a range of alternative phenotypes in response to a sequence of environments. Thus, developmental plasticity refers to any observable modification or phenotypic variation in response to a sequence of environments during growth and development or throughout life. Such phenotypic variation or developmental plasticity may be reversible in one aspect, irreversible in other, and may also have serious health consequences in later life in some other aspects. Accordingly, it is suggested that there are two levels of developmental plasticity. The first level is concerned with a "growth response" mainly to the "human-made-environment", while the second level is related to a "plastic adaptation" that promotes survival, functional capacity and reproduction in relation to physical environment. 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-term studies of irreversible changes in the adult phenotype that may be related to survival, functional capacity and reproduction.

There is considerable evidence that developmental plasticity has its own limits. In other words, although individual genotype has the ability or capacity to develop and express an array of alternative phenotypes, such capacity has some limits or trade-offs set mostly by stabilizing natural selection. 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.

The other aspect of developmental plasticity is canalization, or "a set of processes historically

selected to keep the phenotype constant" despite genetic and/or environmental variations. Subjectively, neither too big nor too small is necessary good for health and well-being. The success or failure of a given phenotype is conditioned by the reaction norm of genotype. The reaction norm of genotypes may be assumed to operate in accordance with the process of canalization that has a complex genetics. A better understanding of developmental plasticity and canalization is of considerable importance to many aspects of organic life, especially to the health and well-being of human populations.

In general, there are two major models of the study of growth and development: medical and adaptability models, which are at times conflicting each other because growth and development can be used as an indicator of health and nutritional status as well as a means of adaptation. One good example of the conflicting interpretations between medical and adaptability models is the debate on the "small but healthy" hypothesis especially with respect to the question whether short stature is a matter of concern? Nevertheless, medical model stresses more on developmental plasticity in relation to "human-made-environment" or environmental quality especially unequal access to nutrition and health care, whereas adaptability model stresses more on adaptation to a specific feature of physical environment, such as temperature, altitude and hypoxia.

Malnutrition is considered a major risk factor of under-five mortality in developing countries. The underlying cause of malnutrition is poverty compounded by poor access to material assets and health amenities. The difference in malnutrition between the rich and the poor or higher and lower classes, is the common phenomenon in developing countries, although inequality in the overall health status persists even in developed countries. Thus, it looks as though the poor or the lower classes were often the victims of natural selection.

Natural selection is operating in human population through many agents including social stratification, which is socially inherent part of modern societies. Malnutrition and infections, which ultimately lead to higher morbidity and mortality, are common phenomena in the lower strata of societies in developing countries. Natural selection often results in different modes of adaptation that are relatively beneficial for survival, health, functional capability and reproduction. Malnutrition may result in smaller body size that may offer an advantage to the individuals under nutritional deprivation, but it may be disadvantageous in respect of greater susceptibility

to infectious disease, or lower physical work capacity. Higher fertility in the lower strata of social stratification may offer advantage to minimize the risk of lineage extinction, but at the expense of higher malnutrition, infections and mortality. Thus, natural selection in the lower strata of social stratification does not result in a long-term beneficial adaptation. On the other hand, Darwinian fitness will tend to be highest in the higher strata of social stratification through its effects on health and functional capability.

Social inequality is not the only mediator of natural selection in human populations. As a blind natural force, natural selection may act at any deviation from some threshold of a delicate balance or trade-off between our biological make-up and the environment. In other words, any deviation from some threshold of such a delicate balance is a predisposition to ill health and ultimately to mortality. Empirical evidence for health inequalities between the rich and the poor is indicative of a large difference in degree of attaining a delicate balance, or a harmonious relationship between genotypes and environments, that each individual has the fundamental rights to achieving it.

Natural selection acts primarily at the individual level. Each individual has a unique genotype, and each phenotype is a unique outcome of the *norm of reaction* of the genotype with its environment during the process of growth and development. Each individual, irrespective of sex, class and caste, has the biological capacity to develop and learn a vast set of alternative ideas, behaviours and skills, which are essential for survival, health and well-being. Equality of economic opportunity in a society, for example, enables each person to choose any occupation, which is most suited for him or her according to his or her abilities and willingness to strive. In short, there is no biological justification for social inequality in health and nutritional status.

REFERENCES

- Allison, A. C.: The distribution of the sickle-cell in East Africa and elsewhere and its apparent relationship to the incidence of subtertian malaria. *Tans. R. Soc. Trop. Med. Hyg.*, **48**: 312-318.
- American Association of Physical Anthropologists (AAPA): AAPA statement on biological aspects of race. *Am. J. Phys. Anthropol.*, **101**: 569-570 (1996).
- Baker, P. T.: Human adaptation to high altitude. *Science*, **163**: 1149-1156 (1969).
- Baker, P. T. : The adaptive limits of human populations. *Man*, **19**: 1-14 (1984).
- Barker, D.J.P. (ed.): *Fetal and Infant Origins of Adult Disease*. BMJ Books, London (1992).
- Barker, D.J.P.: Fetal origins of coronary heart disease. *Brit. Med. J.*, **311**:171-174 (1995).
- Barker, D.J.P.: Developmental origins of adult health and disease. *J. Epidemiol. Comm. Health*, **58**:114-115 (2004).
- Beaton, G.H., Kelley, A., Kevany, J., Martorell, R. and Mason, J.: *Appropriate Uses of Anthropometric Indices in Children*. ACC/SCN Nutrition Policy Discussion Paper No. 7. United Nations, Geneva (1990).
- Béteille, A. *Caste, Inequality and Affirmative Action*. International Institute for Labour Studies, Geneva (2002).
- Bindon, J. R. and Baker, P. T.: Bergmann's rule and the thrifty genotype. *Am. J. Phys. Anthropol.*, **104**: 201-210 (1997).
- Birch, L. C. : The meaning of competition. *Am. Nat.*, **91**: 5-18 (1957).
- Boas, F.: *The Mind of Primitive Man*. Macmillan, New York (1938).
- Bokin, B.: Plasticity in the growth of Mayan refugee children living in the United States. pp. 46-74. In: *Human Variability and Plasticity*. C.G.N. Mascie-Taylor and B. Bogin (eds.). Cambridge University Press, Cambridge (1995).
- Bokin, B.: *Patterns of Human Growth*, 2nd Edition. Cambridge University Press, Cambridge (1999).
- Chen, C.H.C., Schilling, L.S. and Lyder, C.H.: A concept analysis of malnutrition in the community. *J. Adv. Nurs.*, **36**:131 – 142 (2001).
- Chinn, S., Hughes, J. M., Rona R. J.: Trends in growth and obesity in ethnic groups in Britain. *Arch. Dis. Child.*, **78**:513–517 (1998)
- Chisholm, J.S. and Burbank, V. K. : Evolution and inequality. *Int. J. Epidemiol.*, **30**: 206-211 (2001).



