Malnutrition: Definition, Incidence, and Effect on Growth

With reports from famine-stricken regions appearing almost daily on radio and television, any discussion of energy-protein malnutrition (EPM), its definition, its incidence, and how it affects growth, is particularly timely. An estimated 200 million to 1 billion people in the world suffer from EPM. However, EPM is an ambiguous concept, and it is therefore difficult to determine what, if anything, these prevalence estimates mean.

The basic problem in understanding EPM is that it is defined by three different criteria: (1) *dietary deficiency*, where the intake and/or utilization of energy and protein nutrients are lower than the recommended daily allowances; (2) *substandard anthropometry*, where a person is below the international or local measurement standards of height and weight for age, weight for height, or skinfold thickness; and (3) *functional impairments*, where dietary deficiency results in physical, mental, or emotional changes. High rates of morbidity are linked to functional impairment through decreased immunocompetence, impaired efficiency of physical work performance, impaired mental performance, and emotional stress caused by sensations of hunger.

By utilizing a Venn diagram (Figure), one can identify seven sets of EPM. Dietary deficiency, substandard anthropometry, or functional impairment can occur alone (set 1, 3, or 7) or in various combinations (set 2, 4, 5, or 6). A closer look at these sets reveals the imprecise nature of the definitions given for the three major categories. Many people in set 7 may have functional impairments from disease, emotional stress, social or environmental deprivation, exposure, or other dysfunctional factors of poverty rather than from dietary deficiency. Also of great interest are individuals in set 3: They may have substandard anthropometry of genetic origin and not be functionally impaired. Heredity may be the etiology for the majority of people with substandard anthropometry, who are nevertheless considered to suffer from EPM by the present definitions. If these energy-protein standards and statistical procedures used to estimate dietary deficiency in developing countries were applied to the United States, 67% of males and 80% of females would be considered EPM victims. Do we seriously believe that such a large proportion of the US population is undernourished?

Reexamining the diagram, one sees that only those individuals who fall in set 5 have malnutrition characterized by diminished intake, substandard anthropometry, *and* functional impairment. This set represents a smaller number of EPM victims than would be identified when a broader definition of EPM—for instance, only one of the three standards—is applied.

To reduce the confusion surrounding the definitions of malnutrition, one must consider two alternative theories of the growth process and be acquainted with the "small-but-healthy" hypothesis.

The average person usually thinks of malnutrition in terms of thin, wasted people who have substandard weights for height—in other words, acute EPM. Chronic EPM occurs often when the individual is below standard height-for-age levels. While it may be thought that most people with chronic EPM also have acute EPM, this is not necessarily true. In a study that assessed the nutritional status of children aged 6 to 18 months in 14 developing countries, it was found, on the average, that 90% of the EPM encountered was chronic rather than acute. These children had low height for age, but normal weight for their short stature. Further, only 17% of those with low weight for age had low weight for height according to the classification system used (Gomez). The children who are short but have normal weight typify the small-but-healthy hypothesis. Even though they are not underweight for height, and are thus likely to be considered healthy, they may be nutritionally dwarfed.

Most published reports assume these children are not healthy, but without independent evidence of functional impairment, the meaning of this kind of malnutrition is ambiguous. If, on the other hand, these children are healthy, one must wonder why they are short even though they have appropriate weight for height and other body proportions, body fat, and satisfactory general health. According to the small-but-healthy hypothesis, these children are healthy, but their small size is often the result of decreased caloric and/or protein intake.

Of the two theories of human growth, the *deprivation theory* is the predominant one. It is assumed that every individual is born with a single, genetically determined growth po-

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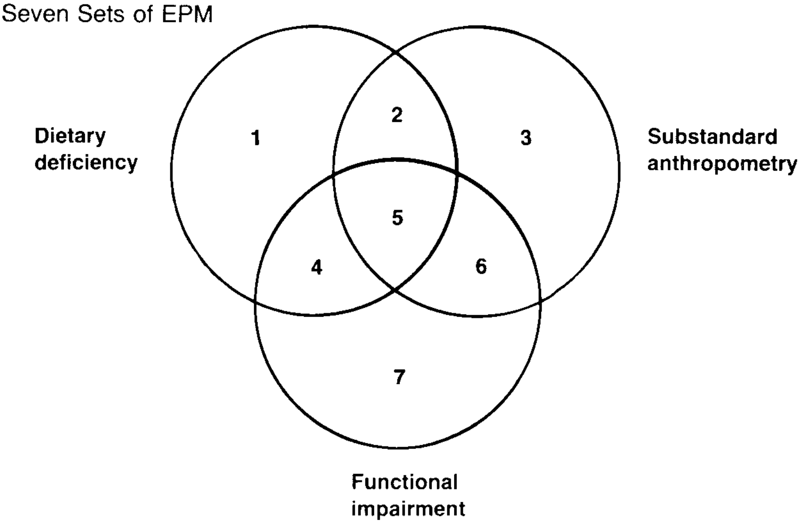
Seven Sets of EPM



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tential. It is further assumed that a healthy and well-nourished child will grow to his or her genetic potential. In contrast, by definition, growth that is significantly below genetic potential indicates functional impairment. Of course, some individuals are normally small, but this may be difficult to determine. Nevertheless, in large populations, a skew of the distribution curve of height toward lower stature is perceived as functional impairment within that population.

In contrast to this view, the *homeostatic theory* of growth holds that the genetic endowment of the organism interacts with the environment in a system of cybernetic control to maintain homeostasis. According to this theory, the genetic growth potential of the deprivation theory is replaced by a broad array of potential growth curves in several anthropometric dimensions—a potential growth space, in other words. Within the bounds of this potential growth space, the growing organism may be mapped through various paths of size and shape in response to nutrition, disease, climate, activity, emotional stress, and other environmental influences.

The homeostatic theory promulgates that a major control instrument is the regulation of growth rate with respect to the height of the child. If, for example, nutrient constraints are encountered at a given rate of growth, the rate is slowed to bring nutrient demand into equilibrium with supply. Similarly, the growth rate may be accelerated in response to overconsumption. According to this theory, neither nutrient constraint nor overconsumption is necessarily abnormal; they are merely adaptations. Through regulation of the speed of internal physiologic "clocks," short-term equilibrium can be established and the ultimate size and shape of the adult molded to the environment. Of course, there are bounds to these adaptive possibilities. If deprivation is severe, acute EPM may be superimposed on the expected short stature that may result from a modest decrement in caloric intake or utilization.

Thus, while the deprivation theory postulates a continuous relationship between small size and the functional impairments of EPM, with the incidence and severity of deficiencies increasing as the size of the organism decreases, the homeostatic theory essentially postulates a discontinuous, threshold relationship. According to the latter theory, smallness is not necessarily correlated with functional impairment, although there is a high incidence and severity of functional impairment as the lower boundaries of size are transgressed. Thus, children with mild to moderate chronic EPM are likely to remain small but healthy. With regard to acute EPM and moderate to severe chronic EPM, the two theories are concordant.

If the homeostatic theory is valid, it explains why many small persons are not functionally impaired. In a study of Bangladeshi children, Chen et al found a high incidence of mortality in those with severe EPM, but no difference in mortality between children with mild to moderate EPM and normal children. Once some difficult statistical problems due to aggregation of individual variation in thresholds are better sorted out, similar threshold effects are likely to become apparent.

In summary, the essential difference between the deprivation and homeostatic theories is the difference between maximum and optimum. The deprivation theory states that the optimal size must be in accord with the maximum genetic potential. Hence, it follows that smallness is bad per se. The homeostatic theory, on the other hand, defines optimal size in terms of a functionally stable growth space that may be considerably below maximum height-for-age ratios. The lower boundary of the growth space, where significant functional impairments begin to occur, can be determined only by empirical research. Thus, while the deprivation theory deduces functional impairment from the premise that maximum growth is necessary to health, the homeostatic theory requires evidence of functional impairment to define EPM. To date, the incidence of malnutrition has been defined primarily in relation to the deprivation theory.

The differences between the deprivation and homeostatic theories open important fields of research in pediatrics, genetics, endocrinology, and even economics. If it is found that the growth, and perhaps the shape, of the human body are controlled by factors other than nutrition and disease—genetic and environmental interactions, for example—then pinpointing the control mechanism and what it responds to becomes of great scientific interest.

In terms of nutritional policy, it seems clear that nutritional resources now being devoted to accelerating the linear growth of children should be reallocated to those children in clear and present danger of functional EPM: namely, those with serious to severe acute EPM (underweight for height). This approach would reduce the target population of nutritional programs to less than 10% of the children who are conventionally defined as having EPM. With proper management, the resources now largely being spent on accelerating linear growth in small but relatively healthy children could eradicate functional EPM. In fact, attempts to get children on higher growth curves before their poverty has been alleviated may put them out of equilibrium with their environment and do irreparable harm.

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