1 The Effect of Malnutrition on the Individual

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In recent years, nutrition as a public policy issue has become a major topic of discussion for an increasing number of persons and bodies concerned with social, political, and economic development. Actually, nobody denies that nutrition is an important factor in the life of the individual from the time of conception to the time of death. Intake of a diet adequate in quantity and quality has been recognized, since the early days of pediatrics, as a prerequisite for the optimal growth and development of the child. Nonetheless, when one moves from the individual to the community as a whole, the need for maximizing insufficient resources forces those responsible for planning and operating development programs to question the priority of malnutrition vis-à-vis other problems. Berg [1] has indicated that the central point for the planner in developing countries is to decide if malnutrition is such an important obstacle to national development as to justify rerouting resources now earmarked for other needs. In the presence of limited resources good planning demands that each disease, each damage, must be ranked against every other need. Some specific aspects of this comparison are (1) the magnitude of the problem; (2) how vulnerable are its proximal and distal causes to the means available for its prevention and treatment; and (3) what are the implications of its presence and its sequelae for the continuous development of the community (historical transcendency).

The intent here is to summarize our knowledge on the physical and mental sequelae of malnutrition with emphasis on how malnutrition affects the capacity of an individual to make a meaningful contribution to society. Because, either alone or more often in combination with infectious disorders, protein-calorie malnutrition constitutes one of the main causes of death and disease in the world population, our review is restricted to this particular form of nutritional inadequacy.

At the community level, protein-calorie malnutrition is a man-made disorder characteristic of the lower segments of society, particularly of the preindustrial societies, where the social system (consciously or unconsciously) creates malnourished individuals generation after generation through a series of social

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mechanisms among which limited access to goods and services, limited social mobility, and restricted experiential opportunities at crucial points in life play a major role.

At the individual level, the term *protein-calorie malnutrition* is a generic name used in the medical literature to group the whole range of mild to severe clinical and biochemical signs present in children as a consequence of a deficient intake and/or utilization of foods of animal origin, accompanied by variable intakes of rich carbohydrate foods. Kwashiorkor and marasmus are the names given to the two extreme clinical varieties of the syndrome. The appearance of one or another of these nutritional disorders is related to the age of the child, time of full weaning, time of introduction of food supplements to breast milk, caloric density and protein concentration of the supplements actually given, and frequency and severity of infectious disorders during weaning.

Although its incidence varies from place to place, the syndrome presents the same basic characteristics of clinical and biochemical pathology. The regional variations observed are generally associated with other concomitant nutritional deficiencies, the pattern of weaning, and the infectious pathology prevalent in the area.

The interaction of the effects produced by the societal factor and the individual factor is what ultimately results in malnutrition. Taking low weight gain in early infancy as the single most characteristic sign of protein-calorie malnutrition, Figure 1 was constructed as a flow diagram to illustrate several pathways through which this complex of interrelations may result in malnutrition in infancy.[2] Starting with a low level of modern technology, which results in limited income and the expenditure of almost all the available energy (time available to be converted to consumption goods) for the procurement of the bare necessities of life, one is confronted with reduced purchasing power and with the absence of reserves and surpluses. At least two pathways can derive from this point to produce low weight gain in infants. The first is direct, and proceeds from insufficient investment in sanitary modifications of the environment to the persistence of traditional conceptions of health and disease. [3,4] These prescientific conceptions include incorrect ideas on the role of food in the production of disease, which translate into practices that, within the limits set by the purchasing power of the family, determine the pattern of distribution of available food within the family; the net result is a reduction in the type and amount of food that the infant is allowed to consume. This ultimate step would be the last link of the

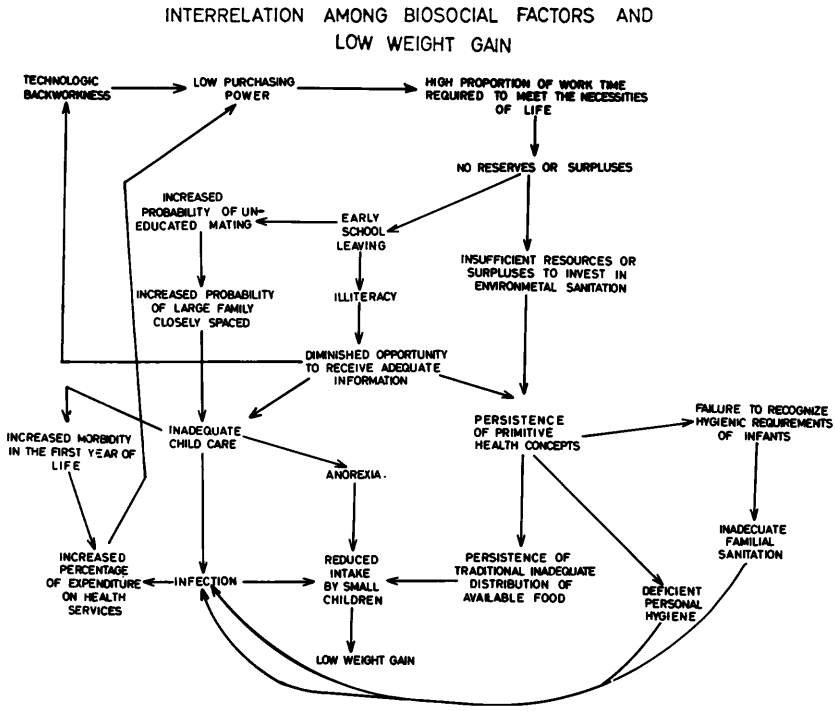

Figure 1. Interrelation among Biosocial Factors and Low Weight Gain

chain producing low weight gain. Indirect branching may occur when primitive conceptions on health result in lack of awareness of the hygienic requirements of the child, and lead through a chain of unsanitary conditions in the home to poor cleanliness of the mother and other members of the household in direct contact with the infant, who is then exposed to a higher risk of infections that either directly or indirectly may produce low weight gain.

A second major pathway proceeds from lack of reserves or surpluses to pressures for early school-leaving as an attempt to increase the purchasing power of the family unit. The consequences of school dropout are high illiteracy and diminished opportunities for obtaining adequate information, which in turn reinforce technological backwardness and the persistence of primitive conceptions of health and disease. Early school dropout also results in the conferral by society of adulthood status and role to a group of individuals at an earlier age than would be the case had they remained students. This situa-

tion may lead to an increased chance of marrying at a younger age and to an equally uneducated spouse, thus multiplying the likelihood of a larger family, inadequate child care, illness, and malnutrition. The increased morbidity in turn would translate into increased expenditures on health services, thus aggravating the effect of low purchasing power.

Clearly many other pathways may be identified, all of them resulting in a reinforcement of opportunities for the persistence of a way of life which drastically reduces the chances of an adequate development of technological and educational competence. Malnutrition becomes just one of the expressions of the interactions of social variables capable of influencing nutritional and health practices.

In examining the possibility that malnutrition may result in suboptimal functioning of the individual it becomes necessary to consider the role of food and feeding along several dimensions. The first one, which may be called a physiological dimension, has as a unit of measurement the *nutrient*, and its function is to provide chemical substances to the organism for its growth, maintenance, and metabolic regulation. The second dimension of food may be considered as psychophysical. Its unit of measurement is the *foodstuff*, which through its organoleptic characteristics would provide the organism with a variety of stimuli (texture, color, aroma, taste, temperature). In this context a foodstuff presented at the table in two different kitchen preparations having the same nutrient and calorie content would in fact be considered as two different foods by the individual. Finally, the third food dimension may be considered as psychosocial in nature, and its unit of measurement is the *mealtime*. The function of food along this line is, on the one hand, to aid in symbol formation through the values that family and society attach to food, such as: a form of reward or punishment; an experience attached to a gratifying or ungratifying person; and an identifying characteristic of an ethnic or subcultural group. On the other hand, the mealtime provides opportunities to demonstrate, clarify, and practice role and status at the family level and at the community level. Who is waited on first? Who sits at the place of honor at the table? Who receives the best part of a dish? Who moderates conversation at the table?

Given thus the fact that human malnutrition is a consequence of a malfunctioning society, and that food is something more than nutrients and calories, it appears as a corollary that the evaluation of its role as a cause of suboptimal performance of the individual can be done only through the longitudinal study of infants at risk and of an appropriate control population over a long

period of time. The information available from cross-sectional studies is not enough to settle the main question. Nevertheless, even in the absence of the final answer, there is plenty of suggestive evidence that individuals who were severely malnourished, particularly in early infancy, have performance levels below those to be expected according to their social class. It is not implicit in this statement that lack of nutrients per se is the responsible factor for the suboptimal functioning.

Size

Since the classical studies of Jackson and Stewart [5] published in 1920, it has been established that malnutrition produced in animals during the suckling period results in permanent reduction of ultimate size. The reports of McCance, [6] Platt, [7] Pratt, [8] and Dobbing [9] among many others leave no doubt that in a variety of animal species malnutrition imposed during the rapid phase of growth, when the rate of protein synthesis and the rate of cell division are at their maximum, decelerates body growth and changes the structure of some organs. The transiency or permanency of the damage is a function of time and duration.

For the human, as so frequently happens, our information on the later effects of early malnutrition is fragmentary and scanty, with as yet no clear-cut conclusions. For animal research, all that is required is the definition of severity of malnutrition, type, duration, and age of onset. Thus adequate controls can be run, and in a relatively short period of observation the problem can be solved. Human malnutrition, on the other hand, does not occur in isolation and many of the nonnutritional factors that accompany or contribute to its appearance may independently influence growth and development. Therefore, the presence of malnutrition conjointly with other potential conditions of risk for maldevelopment makes it difficult to determine the contribution that inadequate nutrient intake per se may be making to the inhibition of growth and development, especially among populations where differences in the level of nutrient consumption are generally associated with other profound differences in familial characteristics and environmental circumstances related directly or indirectly to social disadvantage.

The association between adult stature and socioeconomic status is well known, [10] and recently Naeye et al. found that infants of poor mothers, in New York City, were at delivery 15 percent smaller than infants of nonpoor mothers. [11] Most of the few published studies on the effect of early malnutrition on physical growth in humans are handicapped by our lack of infor-

mation about the genetic potential of the children under observation and by the scarcity of data on the adequacy of the diet and health care received by the children in their home environment after discharge from hospital. The results obtained under these circumstances in several ethnic groups (Bagandan, Peruvian, Chilean, South African Coloured) show that previously malnourished children do not attain, at least for several years, the weight, height, and bone age of children of the same ethnic background but of a higher social class. [12-16]

Considering that children from a higher social class may be nutritionally above average and may also be genetically different, Garrow and Pike [17] decided to compare the growth of previously malnourished Jamaican children with the growth of their siblings or close relatives. Using a matched-pair technique it was found that the index cases examined 2 to 8 years after discharge from hospital were slightly taller and heavier than their siblings, who, in the opinion of the authors, had never been severely malnourished.

Since, on the one hand, no relationship has been found between the severity of malnutrition on admission to hospital and the degree of somatic deficiency years later, or between length of stay in hospital and degree of catch-up in growth; and, on the other hand, there is a weak, not statistically significant relation between age on admission and size at follow-up, the apparent discrepancy of results obtained when comparing previously malnourished children with siblings or with higher class natives can be interpreted as suggesting that the severe acute episode of malnutrition that throws the child into hospital has little influence on subsequent growth after discharge. But chronically malnourished infants, with or without an acute episode of superimposed deficiency, would not reach for many years the norms of growth of their ethnic class.

In support of the above suggestion are the findings of Ashworth, [18] who studied for a sufficiently long period the growth rates of 8 children recovering from protein-calorie malnutrition. Compensatory growth was very rapid at the beginning of recovery, but when the expected weight for height was reached, food intake fell abruptly by 30 percent and growth rates dropped to a level comparable with those of normal children of that height and weight. Accordingly it can be expected that, if height for age was markedly low on admission, stunting will be present at least for several years, even if home conditions are favorable to meet the requirements for rapid growth. Since it has been calculated that a child needs 81 Kcal to form 10 g of new tissue containing 1.7 g/protein and 2.5 g/fat, a reasonable conclusion is that under

present conditions it may be very difficult for children to receive at home a diet adequate to enable them to reach their full growth potential.

It seems clear that the extent of permanent stunting due to early malnutrition depends on many factors. The importance of attaining full physical growth can be illustrated by Thomson's study, [19] which documented that in primigravidae in Aberdeen (excluding those over 29 years of age, in whom the matter is more complex) short stature is associated with a greatly increased liability to delivery by caesarean section, and of perinatal deaths due to birth trauma. There were 43.6 caesarean sections per thousand mothers of less than 60 inches of stature, and only 4.2 per thousand when stature was equal to 62 inches. Similarly, perinatal deaths due to birth trauma rose from 2.7 per thousand in infants from mothers with heights around 64 inches to 8.6 in infants from mothers of only 60 inches or less.

Mental Performance

The association between malnutrition in preschool children and low levels of mental performance has been amply documented in several regions of the world where malnutrition is highly prevalent. A direct association between deficits in height and weight of malnourished children and retardation in psychomotor, adaptive, language, and social-personal behavior, as measured by the Gesell, Cattell, or Bayley techniques, has been reported. [15, 20-23]

Studies of mental performance of kwashiorkor patients during the period of rehabilitation have shown that, as children recover from malnutrition, developmental quotients increase in most cases. The magnitude of the increment varies in direct relation to the age at which the children suffered the disease. Therefore, with successful treatment, the difference between chronological and mental age progressively diminishes in all children except those who are sticken by severe malnutrition below the age of 6 months. [24] Research conducted in infants recovering from nutritional marasmus has also disclosed that basal intelligence and psychomotor activity, as judged by the Bayley scales, remain severely retarded despite apparent somatic recovery. [25] These studies extend the results found in children recovered from kwashiorkor and point out the fact that both extremes of chronic severe protein-calorie malnutrition behave in similar ways, giving a marked retardation in mental development, which is present even after physical and biochemical rehabilitation have occurred. [26, 27]

The effect of added stimulation during the initial recovery has been recently reported. [28] Two groups of severely malnourished children were evaluated

during their recovery by the Griffiths scale. One group, paired for sex and age, was situated in an environment where there were pictures, drawings, toys, and music, and where nurses played with the children and sang to them, establishing a good emotional relationship. The nonstimulated group stayed in a place with similar dimensions but with no decorations, and without toys and music. The medical and dietetic treatment was the same for both groups. The initial difference in developmental quotients was not significant. Both groups increased their mental performance in a significant and almost parallel form, and only toward the end of the observation period (4 months) did the stimulated group show higher quotients, resulting basically from a drop in the performance level of the nonstimulated group. Both groups remained below the values expected for their age; the interesting fact was that the greater deficit occurred, as was the case among Roblés et al.'s Mexican children, in the area of language and communication.

Another approach to assessing the persistence of mental lags in malnutrition has been the study of survivors several years after discharge from a hospital. Four such studies have been published. The first is a report on the follow-up of 36 Serbian children who had been hospitalized for severe malnutrition when they were between 4 and 24 months of age. [29] Their level of intelligence was evaluated when they were between 7 and 14 years old, using a modification of the Binet-Simon scale. The mean IQ level was 88, which is significantly lower than the figure of 93 found in a group of normal children of unskilled workers. It is interesting that one-third of the fathers of the children rehabilitated from malnutrition were either professionals or army officers, and the rest were skilled or unskilled workers. In relation to the IQ distribution, one-half of the 39 rehabilitated children showed IQs below 90, with 6 children not scoring above 70. These frequencies are in contrast with those found in the general population of Serbian children in which 32 percent had IQ scores above 110. The weight deficit on admission to the hospital and the IQ at school age showed a significant association.

The second study was done in Indonesia [30] in a group of children 5 to 12 years old whose nutritional status at the age of 2 to 4 years was known. When tested with the Goodenough and Wechsler techniques, the children who had been previously malnourished and who had shown signs of vitamin A deficiency in the preschool years had significantly lower intelligence scores than the children who were regarded as healthy during the 2-year to 4-year age period. The IQs derived from the total Wechsler scale were 77 ± 2.3 and 68 ± 2.7, respectively, for 33 children considered as healthy in the preschool

period and the 12 children who at that time were diagnosed as malnourished and showing vitamin A deficiency. The group of 19 school-age children whose diagnosis at the preschool age was malnutrition without vitamin A deficiency had a mean IQ of 73 ± 3.3. The difference between normal and malnourished, with or without vitamin A deficiency, is significant at the 0.01 level of confidence. According to the authors, the intellectual development as well as the physical development of the children could be predicted with a high degree of accuracy on the basis of their nutritional status during the preschool years.

The third study is the follow-up of a group of Indian children who had been treated for kwashiorkor. [31] The ages at the time of admission into the hospital were between 18 and 36 months. At the time of follow-up the children were between 8 and 11 years of age. Each index case was matched for age, sex, religion, caste, socioeconomic status, family size, birth order, and educational background of the parents, with three children who had never been hospitalized for malnutrition. All control children also belonged to the same class in school as the index case. The results of the mental tests showed a significant difference between groups not only in intelligence but also in level of intersensory adequacy. The younger age group gave the maximal differences. Subsequently, at the 1971 Asian Nutrition Conference (report in press) the author (Srikantia) showed that the mothers of kwashiorkor children were less competent than those of the controls.

The fourth report describes a study carried out in a group of Mexican school-age children who had suffered severe protein-calorie malnutrition before their 30th month of life. [32] In an attempt to reduce the influence of environmental variables, a group of siblings of similar age and sex were also examined; the difference in age between the index child and his sibling never was more than 3 years. The Wechsler Intelligence Scale for Children was administered to both groups. As expected, the results showed that the environment in which children at risk of malnutrition live is highly effective in reducing mental competence. Children developing in this milieu have a high probability of scoring in the low range of values in intelligence tests as well as in other types of tests related to basic mechanisms for learning. It is important to emphasize that the presence of an episode of severe malnutrition early in life increased the chances of scoring in the very low range of values. The distribution of total IQ illustrates the differences found between rehabilitated children and siblings. Thus, while 9 of the 37 siblings had quotients below 70, 18 survivors of malnutrition were in this range. In contrast only 4 survivors scored above 90, while 10 siblings obtained these values.

Learning

In further assessment of the mental development of school-age children who experienced severe malnutrition early in life, a series of investigations compared the performance of children who were hospitalized because of the severity of the syndrome before the age of 30 months, and a group of their siblings. It was assumed that selecting a comparable group from within the sibship to which the malnourished child belongs is probably the best procedure, in a cross-sectional study, to control familial and social circumstances which in themselves may be conducive to impaired development. In order to minimize the differences found between index cases and controls, which can represent the residual effect of recent long-term hospitalization, only children who had been out of the hospital at least 30 months were included. The sibling closest in age to the index case was included in the comparison group.

Mental performance on a variety of tests related to basic learning mechanisms has been evaluated in index cases and siblings in a series of sessions, while maintaining high motivation during the administration of the stimuli.

In a first study, [33] the developmental course of auditory-visual equivalence was studied in 39 index cases and in 39 siblings. One of the reasons for selecting auditory-visual competence was the report of Birch and Belmont [34] who have shown that this form of intersensory integration—besides having a clear-cut developmental course in normal children during the first school years—can be effectively used to differentiate good readers from poor readers. The child's ability to integrate auditory and visual stimuli was studied by an equivalence method. The children were asked to identify visual dot patterns corresponding to rhythmic auditory patterns; that is, the task explored the ability to equate a temporally structured set of auditory stimuli with a spatially distributed set of visual ones.

The siblings in the low urban social class had scores below those of children of the same age but of a better socioeconomic status. The improvement in auditory-visual competence with age was obvious for all social classes and for urban and rural environments. The difference in performance is shown by the slope of the lines relating achievement score to age.

When the performance age-by-age of siblings and index cases is contrasted it becomes apparent that the children recovered from severe malnutrition were well below their siblings in auditory-visual integration, and well below the expected values for their social class. To illustrate that this difference in ability was not due to a few extreme cases affecting the mean value of the group, the cumulative percentage of 7-year-old index cases and siblings was

compared. The lag in development of auditory-visual competence of the index cases was evident.

In a second study, [32] the visual-kinesthetic intersensory integration, an ability closely related to learning to write, was explored by a method of equivalence in the perception of geometric forms. The kinesthetic sense modality in this context refers to the sensory inputs obtained through passive arm movement. Such motion entails sensory input from the wrist, elbow, and shoulder joints and from the arm and shoulder muscles as its principal components. In this test, kinesthetic information is provided by placing the child's preferred arm behind a screen and, with the arm out of sight, passively moving it through a path describing a geometric form. [35]

It was evident that, age by age, the children recovered from severe early malnutrition had significantly lower performance levels than their siblings. With respect to the proportions of index cases and siblings making errors in the identification of either identical or nonidentical geometric forms at ages 5 to 7, significant differences in accuracy of judgment always are in favor of the siblings. Similar findings have been reported by Champakam and coworkers [31] in India. The data from the siblings in our study and in the matched controls of the Indian group show that the lag in a mechanism basic for learning to write that is present in the children rehabilitated from early severe malnutrition is greater than the lag which could be expected as an effect of the low social class.

There is a tendency to view the human organism as an agent that processes information. Humans live primarily in a visual world, and, logically, we expect more elaboration and more uses of visual information than of information from other sense modalities. Consequently, reading and writing have become primary tools in our society.

Learning to read has as an essential prerequisite the ability to distinguish simple visually presented figures. However, the ability to make gross discriminations among visually perceived figures, although it is a necessary component ability, does not constitute a sufficient refinement of perceptual skill for the task of reading. In addition to making gross discriminations, if a child is to learn to read he must also respond to more differentiated aspects of the figural percept such as angular properties and spatial orientation.

The child's failure to respond to the spatial orientation of a visual form can result in his confusing a number of letters in the Roman alphabet that are identical in form but distinguishable by their spatial positioning. Letters such as b, p, d, and q, or N and Z, W and M, all represent equivalent shapes, with

the distinction among them depending upon the child's ability to respond simultaneously to shape and to orientation in visual space.

Birch and Lefford, [36] in order to obtain information on features of increased differentiation in visual perceiving, have constructed a visual discrimination task that provides information not only on gross discrimination ability but also on response to spatial position and to differences in angular symmetry. It is known that children with significant mental subnormality are incapable of making a discrimination requiring that they (1) respond selectively to aspects of the whole figure; (2) take into account spatial orientation with respect to a coordinate system; (3) separate complex visual wholes into their component subwholes; or (4) reconstruct a pattern from its elements. For this reason, visual perception of forms was explored in children recovered from severe malnutrition as a means of assessing their readiness to learn to read. Their achievement was compared with that of siblings who had not suffered severe malnutrition.

The performance of both groups on the recognition of geometric two-dimensional forms showed that as age increased from 5 to 10 years the mean number of errors committed progressively diminished. Again the performance level, although low for both groups of children, was significantly lower for the previously malnourished children until age 9, when both siblings and recovered children achieved similar levels of performance. When the children were tested for their ability to analyze geometric forms, the mean number of errors committed also decreased as age advanced. A sharp difference was found again to be in favor of the siblings.

The studies using matched controls or siblings as comparison groups suggest that it is not only general environmental deprivation but also factors closely related to the event of early severe malnutrition that are contributing to a further depression of intellectual performance and learning.

Based on the demonstration that the adequate integration of information deriving from the sense avenues constitutes one of the major functions of the cerebral cortex, the development of intersensory liaisons in the kinesthetic-visual, kinesthetic-haptic, haptic-visual, and auditory-visual modalities have been explored by the equivalence method in Guatemalan, Mexican, Indian, and Philippine school-age children who have had variable degrees of risk of malnutrition during their preschool-age period. [31, 37-40]

In communities whose children had been at great risk of malnutrition, those of shorter stature, age by age, showed poorer intersensory development than taller children. Analysis of various factors related to environmental and

familial background supported the view that height differences among these school-age children can be regarded as an indirect indicator of previous malnutrition. In a comparison group of urban upper-class school children, intersensory development was more advanced and no relationship was found between neurointegrative adequacy and height.

These studies of neurointegrative adequacy in four different cultural groups may be significant because they seem to indicate that functional lags can occur at the mild-moderate degrees of protein-calorie malnutrition associated with stunted growth, and are not limited to the extremely severe cases represented by kwashiorkor and marasmus.

All the information available leads one to conclude that the existence of an association between protein-calorie malnutrition in infancy and retardation in mental development has been established beyond reasonable doubt. However, it must be emphasized that the fact of such an association provides strongly suggestive but by no means definite evidence that the lack of nutrients per se directly affects intellectual competence.

At least two possibilities should be considered in an effort to define a causal linkage between insufficient nutrient intake and subnormal mental functioning. The simplest hypothesis would be that nutrient deficiency directly affects intellect by producing central nervous system (CNS) damage. In favor of this explanation is the fact that increase of cell cytoplasm with extension of axons and dentrites (one of the processes associated with the growth of the brain in early life) is largely a process of protein synthesis. From the microspectrographic investigation of the regenerating nerve fibers it has been estimated that protein substance increases more than 2,000 times as the apolar neuroblast matures into the young anterior horn cell. In experimental animals specific amino acid deficiencies can cause structural and functional lesions of the CNS. [41] Inhibition of protein synthesis in the brain, produced by puromycin, is accompanied by loss of memory in mice. [42] Delays in myelination, and reductions in cell number and in cell distribution in the brain, caused by interference with adequate nutrition in early life have been amply documented. [9, 43-48] Preliminary findings of reduction in brain size, and even in cell number, in children who died with severe malnutrition have been reported from Chile, [49] Mexico, [50] and Uganda. [51]

The second hypothesis states that malnutrition in human infants may contribute to intellectual inadequacy through at least three possible indirect mechanisms:

1. Loss of learning time. Since the child was less responsive to his environ-

ment when malnourished, at the very least he had less time in which to learn and had lost a certain number of months of experience. On the simplest time basis, therefore, he would be expected to show some developmental lags.

2. Interference with learning during critical periods of development. Learning is by no means simply a cumulative process. A considerable body of evidence indicates that interference with the learning process at specific times during its course may result in disturbances in function that are both profound and of long-term significance. Such disturbance is not merely a function of the length of time the organism is deprived of the opportunities for learning. Rather, what appears to be important is the correlation of the experiential opportunity with a given stage of development—the so-called critical periods of learning. It is possible that exposure to malnutrition at particular ages may in fact interfere with development at critical points in the child's growth course and so cause either abnormalities in the sequential emergence of competence or a redirection of developmental course in undesired directions.

3. Motivation and personality changes. It should be recognized that the mother's response to the infant is to a considerable degree a function of the child's own characteristics of reactivity. One of the first effects of malnutrition is a reduction in the child's responsiveness to stimulation and the emergence of various degrees of apathy. Apathetic behavior in its turn can reduce the value of the child as a stimulus and diminish the adult's responsiveness to him. Thus, apathy can provoke apathy and so contribute to a cumulative pattern of reduced adult-child interaction. If this occurs it can have consequences for stimulation, for learning, for maturation, and for interpersonal relations, the end result being significant backwardness in performance on later more complex learning tasks. It has been reported in experimental animals that small, but statistically significant, differences in the size of the cerebral cortex can be obtained by manipulation of the stimulatory aspects of the environment. [52]

Regardless of whether or not insufficient nutrient intake per se can cause mental subnormality, it is evident that children who have survived the severe forms of malnutrition show alterations in intellectual performance and learning ability which clearly imply a higher risk of failure to profit from school exposure. The child who lags in the performance of basic mechanisms related to fundamental skills such as reading and writing will be ill prepared for the learning tasks required of him when he enters school. If he is behind when he enters, he may never have an opportunity to match the performance of his mates. If the initial impression he gives is of a child who cannot fully benefit

from the learning experiences provided by the school, the behavior of his teachers toward him will reflect their expectations of his performing below par, thus reinforcing the probability of inadequate performance.

In a preindustrial society where staying in school imposes a real sacrifice on the parents and other members of the household, the demand for leaving school to contribute to the familial purchasing power may be a social mechanism that prevents the child from being classed as backward, giving him instead the role of a victim whose sacrifice is necessary, almost indispensable, for the survival of the family group. It is conceivable that through this mechanism the self-esteem of these individuals may be sustained, since the self concept—the individual as he is known to himself—is the result of the reactions that other persons have to his behavior, and of the expectations that those others hold about the ways he will behave. To remain in school can lead to a series of failures that will create a negative self-image, which in turn will lead the individual to define himself as incompetent. To abandon school, on the other hand, is to conform to the expected pattern of behavior, to take the role and status of a victim, avoiding without trying a series of continuous failures. Motivation to complete the number of school years constituting the national norm would be markedly reduced under these circumstances.

It has been observed that children who are malnourished in infancy, or who belong to families where food is not abundant, tend to develop anxiety about food. It is understandable that if a child is worried about what or when will he eat next time, his attention and motivation for learning will be reduced, limiting his probabilities for profiting from the school experience. Even if the child has good mental equipment, if his motivation is low he will not learn early what the school expects of him, and he may be forever handicapped or he may be another member who could not progress because his sacrifice was needed by his society.

It is apparent that children who survived a severe episode of chronic malnutrition run a higher risk of failing to profit from the cumulative knowledge available to their socioeconomic group. Survival from severe malnutrition may constitute the event that starts a developmental path characterized by psychologically defective functioning, school failure, and subsequent subnormal adaptive functioning. At the familial and societal levels the ultimate result of this chain of events is what in an ecological sense could be called a "spiral" effect. A low level of adaptive functioning, lack of modern knowledge, social custom, infection, or environmental insufficiency of foodstuffs produces malnutrition, resulting in a large pool of survivors who come to

function in suboptimal ways. Such survivors themselves run more risk of being the victims of their poor socioeconomic environment, since they are less effective than otherwise would be the case in their social adaptations. In turn, they will choose mates of similar characteristics and may rear children under conditions and in a fashion fatally programmed to produce a new generation of malnourished individuals.

It is obvious from the unequal distribution of malnutrition among the various socioeconomic groups of society that its consequences interact with the negative impact of all the other factors, present in those subsegments of the population, that interfere with the optimal functioning of the individual. It is apparent that many questions, particularly those related to a causal relationship between nutrient deficiency and mental development, remain to be answered, but the available knowledge leaves no doubts about the strong association between the antecedent of severe malnutrition in infancy and suboptimal performance at school age. It is also obvious that the consequences of early malnutrition will be greatest when, after rehabilitation, the child continues to live in an environment in which both social and nutritional circumstances are poor and hostile to his growth and development.

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