appropriate child spacing, and health education. Human milk and breast feeding deserve to be recognized as vital components of such programs with profound nutritional, anti-infective, economic, and contraceptive significance.

References and Notes

1. D. B. Jelliffe and E. F. P. Jelliffe, *Human Milk in the Modern World* (Mosby, St. Louis, in press).
2. D. B. Jelliffe, *WHO Monogr. Ser. No. 29*, ed. 2 (1966).
3. J. M. Bengoa and G. Donoso, *PAG (Protein Advis. Group) Bull.* **4**, 24 (1974).
4. D. B. Jelliffe and E. F. P. Jelliffe, *J. Trop. Pediatr. Environ. Child Health*, in press.
5. ———, *J. Am. Diet. Assoc.* **57**, 114 (1970).
6. M. Sadre, E. Emami, E. Donoso, *Ecol. Food Nutr.* **1**, 55 (1970).
7. D. S. McLaren, *Lancet* **1966-II**, 485 (1966).
8. R. R. Puffer and C. V. Serrano, *Pan Am. Health Organ. Sci. Publ. No. 262* (1973).
9. J. A. Sturman, G. Gaull, N. C. R. Raiha, *Science* **169**, 74 (1970).
10. M. A. Crawford, A. J. Sinclair, P. M. Msuya, A. Munhambo, in *Dietary Lipids and Postnatal Development*, C. Galli, J. Jacini, A. Pocale, Eds. (Raven, New York, 1971), pp. 41–56.
11. N. S. Scrimshaw, C. E. Taylor, J. E. Gordon, *WHO Mongr. Ser. No. 57* (1968).
12. P. György, *Am. J. Clin. Nutr.* **24**, 970 (1971).
13. J. W. Gerrard, *Pediatrics* **54**, 757 (1974).
14. M. Bonté and H. Van Balen, *J. Biosoc. Sci.* **1**, 97 (1969); J. K. Van Ginneken, *Stud. Fam. Plann.* **5**, 177 (1974).
15. Berg (*18*) estimated that 5 million births per year are prevented in India as a result of breast feeding.
16. F. Rosa, unpublished data.
17. D. B. Jelliffe, *Clin. Pediatr.* **7**, 2 (1968).
18. A. Berg, *The Nutrition Factor* (Brookings Institution, Washington, D.C., 1973).
19. R. Cook, *J. Trop. Pediatr. Environ. Child Health* **14**, 60 (1968).
20. P. Hwang, H. Guyda, H. Friessen, *Proc. Natl. Acad. Sci. U.S.A.* **68**, 1902 (1971).
21. D. B. Jelliffe, *Nutr. Rev.* **30**, 199 (1972).
22. D. Raphael, thesis, Columbia University (1966).
23. R. E. Brown, *Clin. Pediatr.* **11**, 33 (1972).
24. A. K. Ladas, *J. Trop. Pediatr. Environ. Child Health* **18**, 317 (1972).
25. D. B. Jelliffe and E. F. P. Jelliffe, in *Proceedings, International Conference on Agriculture, Nutrition and Development in Tropical Countries*, Guatemala, December 1974 (Institute of Nutrition of Central America and Panama, Guatemala City, in press).
26. A. S. Rao, *WHO Chron.* **28**, 172 (1974).
27. J. A. Hildes and O. Schaefer, *Hum. Evol.* **2**, 241 (1973).

Nutrition and Infection in National Development

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Why is it that the case fatality rates from measles are often 200 times higher in poor developing countries than in the industrialized countries? The main reason is that the malnourished child is often overwhelmed by the infection whereas the well-nourished child can combat it and survive.

Why is it that so many cases of kwashiorkor develop following an infectious disease and so many cases of nutritional marasmus following gastroenteritis? It is well established that infections result in increased nitrogen loss and that diarrhea reduces the absorption of nutrients from the intestinal tract.

Nutritional status thus has an effect on infections, and infections have an effect on malnutrition. These are most important relationships. In developing countries communicable diseases are extremely prevalent and are a major cause of morbidity and mortality, just as they were in Europe and North America at the turn of the century. The majority of children in most developing countries suffer from undernutrition and malnutrition at some time in the first 5 years of life. The problems of infection and malnutrition are closely interrelated (*1*). Yet we tend to introduce, quite independently, programs to control communicable diseases and other efforts to improve nutrition. It would be much more efficient and effective if the twin problems were attacked together. Success in improving the health and reducing the mortality of children is dependent both on control of infectious diseases and improvements in their food intake. It would be beneficial if these were combined with family planning programs. There is increasing evidence to suggest a greater willingness of parents to control their family size when the chances are good that most children born will survive into adulthood. Consideration needs also to be given to providing a stimulating environment for the growing child. The situation in the major industrial cities of Europe and North America 75 years ago was comparable to that in the poorest developing countries today. In New York City in the summer months of 1892, the infant mortality rate was 340 per 1000, and diarrhea accounted for half these deaths (*2*). Improvements in nutrition by the use of milk stations and other means and a reduction in infectious disease served to lower these mortality rates by half in a period of less than 25 years. At the turn of the century in Britain, rickets, combined with infectious diseases, was taking a heavy toll in the insanitary, smoky slums of the industrial cities, and measles was very often a fatal disease among children of poor families, presumably because of poor nutrition (*3*).

The so-called synergistic relationship between malnutrition and infectious diseases is now well accepted, and has been conclusively demonstrated in animal experiments (*4*). The simultaneous presence of both malnutrition and infection will result in an interaction with consequences for the host more serious than the additive effect of the two working independently. Infections make malnutrition worse and poor nutrition increases the severity of infectious diseases.

In experimental animals the interaction between nutrition and infection may not always be synergistic, and at times the reverse situation, known as antagonism, is seen. This occurs in animals with certain severe experimentally induced nutritional deficiencies, in which some infections (especially with certain rickettsiae and viruses) have a less severe effect than in a well-nourished animal. In these cases, the host's poor nutritional status presumably provides an unfavorable environment for the particular organism.

This type of antagonism has not been demonstrated to be of clinical importance in humans, and it is believed to occur rarely if at all. In man, synergism, rather than antagonism, appears to be much more common, especially in relation to the important communicable diseases of childhood and to protein-calorie malnutrition, which is our main concern here.

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Effects of Infection on Nutrition

There are several means by which infection affects nutritional status. Perhaps the most important of these is the fact that bacterial and some other infections lead to an increased loss of nitrogen from the body. This was first demonstrated in serious infections such as typhoid fever (*5*) but has subsequently been shown to be the case in much milder infections such as otitis media, tonsillitis, chicken pox, and abscesses.

Nitrogen is lost by several mechanisms. The principal one is probably increased adrenocortical activity leading to mobilization of amino acids from various tissues and organs, but especially from muscle. The nitrogen is excreted in the urine, and is evidence of a depletion of body protein.

Full recovery is dependent upon the restoration of these amino acids to the tissues once the infection is overcome. To achieve this, increased intake of protein, above maintenance levels, is needed in the postinfection period. Children whose diet is marginal in protein content or those who are already protein-depleted will have a retardation of growth during and after infections. In developing countries children from poor families suffer from many infections in quick succession during the postweaning period, and often have multiple infections.

Anorexia is another factor in the relationship between infection and nutrition. Infections, especially if accompanied by a fever, often lead to loss of appetite and therefore to reduced food intake. Other infectious diseases commonly cause vomiting, with the same result. In many societies the mother, and often the medical attendant as well, consider it desirable to withhold food or to place the child with an infection on a liquid diet. This may be rice water, very dilute soups, water alone, or some other fluid with a low calorie density and usually deficient in protein and other essential nutrients. The old adage of "starve a fever" is of doubtful validity, and this practice may have serious consequences for the child whose nutritional status is already precarious.

The traditional treatment of diarrhea in some communities is to prescribe a purgative or enema. The gastroenteritis may already have resulted in reduced absorption of nutrients from food, and the treatment may further aggravate this situation. These are all examples of how illnesses such as measles, upper respiratory infections, or gastrointestinal infections may contribute to the development of malnutrition.

Intestinal Parasites

With the exception of work done on hookworm and the fish tapeworm, the role of intestinal parasites on nutrition in humans has been inadequately studied. Hookworm disease, due to infections both with *Ancylostoma duodenale* and *Necator americanus*, is still prevalent in many countries. Hookworms cause intestinal blood loss, and although it appears that most of the protein in that blood is absorbed lower down in the intestinal tract, there is a considerable loss of iron, which is mainly absorbed from the proximal jejunum.

Hookworm disease is a major cause of iron deficiency anemia in many countries. The extent of the loss of blood and iron in hookworm infections has been studied (*6*). Daily fecal blood loss per hookworm (*N. americanus*) was reported to be 0.031 ± 0.015 milliliter. It was estimated that about 350 hookworms in the intestine cause a daily loss of 10 ml of blood, or of 2 milligrams of iron. Infection densities much higher than this are not uncommon. In Venezuela, where much of this work was done (*7*), iron losses greater than 3 mg per day often resulted in anemia in adult males, and losses of half this amount frequently produced anemia in women of childbearing age and in young children.

The anemia of hookworm disease can be alleviated by diet alone. In general it is desirable both to cure the anemia and rid the subject of worms. However, in some circumstances it may be cheaper and simpler to control hookworm anemia by provision of iron rather than control the infection by anthelminthic medication, improved sanitation, or other means.

The fish tapeworm (*Diphyllobothrium latum*) has an avidity for vitamin B_{12} and can deprive its host of this vitamin, with resulting megaloblastic anemia. This parasite is common in man in only limited geographic areas where undercooked fish is frequently consumed.

On a worldwide basis, roundworm is among the most prevalent of intestinal parasites. In 1947 it was estimated (*8*) that 644 million people in the world (one-quarter of the world's population) harbored roundworms. With a great increase in world population and few campaigns to control the infection, the worldwide prevalence is probably higher today.

The roundworm (*Ascaris lumbricoides*) is quite large (15 to 30 centimeters long) so its own metabolic needs must be considerable. High parasite densities, particularly in children, are common in poorly sanitated environments. Although complications of ascariasis can develop, such as intestinal obstruction or the presence of the worm in aberrant sites such as the common bile duct, these are relatively rare.

Little research has been done on the effects of larval migration through the liver, the systemic effects of the pneumonitis that results as the parasite passes through the lungs, or the nutritional effects of the parasite in the intestines. Studies have suggested that *Ascaris* may reduce absorption of both protein (*9*) and vitamin A (*10*). However, well-controlled studies in poorly nourished animals and young children are needed before we can conclude that this ubiquitous parasite is an important factor in the poor nutritional status of so many children who have ascariasis. Similarly, the nutritional significance of *Giardia lamblia* infections has been inadequately studied. It too has been shown to reduce the absorption of vitamin A in children (*11*).

Effect of Diarrhea

Many studies have indicated that gastrointestinal infections, and especially diarrhea, are very important in precipitating the onset of both kwashiorkor and nutritional marasmus (*12*). Diarrhea is common in and often lethal to, the young child. In breast-fed infants there is often some protection during the first months of life, and so diarrhea is often a feature of the weaning process. This "weanling diarrhea" (*13*) is extraordinarily prevalent in poor communities throughout the world, both in tropical and temperate zones. The organism responsible varies and often cannot be identified.

Diarrhea was a major cause of mortality in children in industrialized countries up to the beginning of this century, but it now constitutes an infrequent and fairly minor illness. Many factors have contributed to this change, and improved nutrition is one of them.

Several studies have shown that admissions of cases of malnutrition are greatly increased during the season when summer diarrhea is most common. For example, in a report from Iran (*14*), admissions of cases of protein-calorie malnutrition were more than double in the summer than in the winter, and this paralleled the incidence of diarrheal disease.

Xerophthalmia is the major cause of blindness in several Asian countries, and is also prevalent in certain parts of Africa, Latin America, and the Middle East. Hospital and community studies indicate that cases of xerophthalmia and keratomalacia are frequently precipitated by infectious diseases such as measles, chicken pox, and gastroenteritis (*15*). The exact mechanism of this relationship has not been proved, but it is likely that many infections reduce vitamin A absorption and that some result in a decreased consumption of foods containing vitamin A and carotene.

Effects of Malnutrition on Infection

There is considerable literature to demonstrate, both in experimental animals and in man, that dietary deficiency diseases may reduce the body's resistance to infections. A comprehensive review of this work has been published (*16*).

Some of the normal defense mechanisms of the body are impaired and do not function properly in the malnourished subject. For example, children with kwashiorkor were shown to be unable to form antibodies to either typhoid vaccine or diphtheria toxoid, but the capacity to do so was restored after protein therapy (*17*). Similarly, children with protein malnutrition have an impaired antibody response to inoculation with yellow fever vaccine (*18*). An inhibition of the agglutinating response to cholera antigen has been reported in children with both kwashiorkor and nutritional marasmus.

The injection of vaccines in this manner is a good simulation of an attack on the body by an organism, and it can be done ethically, whereas experimental infections with pathogenic organisms can seldom be justified in human research. These studies provide a fairly clear indication that the malnourished body has a reduced ability to defend itself against infection.

Another defense mechanism that has been studied in relation to nutrition is that of leukocytosis and phagocytic activity. Children with kwashiorkor show a lower than normal leukocyte response in the presence of an infection. Perhaps of greater importance is the reduced phagocytic efficiency of the polymorphonuclear leukocytes in malnourished subjects. These cells appear to have a defect in their intracellular bactericidal capacity (*19*). The mechanism for this phenomenon has now been postulated, and involves lowered adenosine triphosphate levels in the leukocytes of malnourished infants, combined with decreased activity of reduced nicotinamide adenine dinucleotide phosphate oxidase in response to phagocytic stimulation (*20*).

Although malnourished children frequently have increased immunoglobulin levels (presumably related to concurrent infections), they also may have depressed cell-mediated immunity. In a recent study, the extent of this depression was directly related to the severity of the protein-calorie malnutrition (*21*). Serum transferrin levels are also low in those with severe protein-calorie malnutrition, and often take considerable time to return to normal even after proper dietary treatment (*22*).

An interaction of nutrition and infection of quite a different kind results from the effect of some deficiency diseases on the integrity of the tissues. By reducing the integrity of certain epithelial surfaces, notably the skin and mucosa, resistance to invasion is decreased and an easy avenue of entry exists for pathogenic organisms. Examples of this are the cheilosis and angular stomatitis in riboflavin deficiency, the bleeding gums and capillary fragility in ascorbic deficiency, the flaky paint dermatosis and atrophic intestinal changes of severe protein deficiency, and the serious eye lesion of vitamin A deficiency.

Fatality Rates for Measles and Other Infectious Diseases

A dramatic illustration of the effect of malnutrition on infection is seen in fatality rates of common childhood diseases such as measles. Measles is a severe disease with a case fatality rate around 15 percent in many poor countries, because the young children who develop it have poor nutritional status, lowered resistance, and poor health. In Mexico the fatality rates from measles is 180 times higher than that in the United States; in Guatemala, 268 times higher; and in Ecuador, 480 times higher. The decline in case fatality rates from measles in North America, Europe, and other industrialized countries has been dramatic over the last century. Differences in the clinical severity and the fatality rates from measles in developed and developing countries are due not to differences in virus virulence but to differences in the state of host nutrition (*23*). In my experience working for several years in African hospitals, it was extremely uncommon to have fatalities from measles in the children of Tanzanian families of moderate income, such as those of hospital employees, during a measles epidemic that was causing considerable mortality among the children of poorer families.

Other common infectious diseases such as whooping cough, diarrhea, and upper respiratory infections also have much more serious consequences in malnourished than in well-nourished children. Mortality statistics from most developing countries show that communicable diseases such as these are the major causes of death. In a visit this year to several African countries in the Sahel, I was told that very few children were now dying of starvation or malnutrition, but that deaths from measles, respiratory infections, and other infectious diseases were still very much above prefamine levels. It is clear that many, perhaps the majority, of these deaths are due to malnutrition. This may seem a moot point for a grieving parent. But for the policy planner and the public health official, it is important to know to what extent morbidity and mortality rates are due, or related, to undernutrition.

The inter-American investigation of mortality in childhood (*24*) showed that of 35,000 deaths of children under 5 years of age in ten countries, 57 percent had malnutrition as either the underlying or associated cause of death. Nutritional deficiency was the most serious health problem uncovered, and was frequently associated with common infectious diseases.

Studies in Central America and South Africa (*25*) have shown that the poorer the state of nutrition of the child, the more frequent and the more severe his attacks of diarrhea. A report from Colombia showed that the age of the child is also an important factor (*26*).

Intervention Studies

There have been relatively few well-controlled intervention studies to demonstrate either the effects of improved diets on infection, or the nutritional effects of control of infectious diseases. Research in the village of Candelaria in Colombia showed that diarrhea declined sharply as a result of supplementary feeding of children. A similar study in a Guatemalan village illustrated a significant decline in morbidity and mortality from certain common illnesses following the introduction of a nutritious daily supplement for preschool children (*27*).

A classical study conducted in the town of Narangwal in the Punjab of India (*28*) demonstrated the value of combining nutritional and health care in one program. Children were divided into four groups. One group was given both dietary supplements and health care, two other groups received one of the care programs, and the fourth group served as control. As far as nutritional status and certain other health parameters were concerned, the combined treatment gave the best results, with nutritional supplementation alone also having a major impact. There was no improvement in nutritional status of the group receiving only medical care but no dietary supplements.

Nutrition, Infection, and National Development

Malnutrition and infections combine to pose an enormous hazard to the health of the majority of the world's population who live in poverty. The ever-present threat is particularly to children under 5 years of age. Many of the children who suffer from both malnutrition and a series of infections succumb and die. They are continually replaced by parents who have a strong desire, and often a real need, to have surviving children. The children who live beyond 5 years of age are not mainly those who have escaped malnutrition or infectious diseases, but those who have survived. They seldom are left without the permanent sequelae or scars of their early health experiences. They are often retarded in their physical, psychological, or behavioral development (*29*), and they may have other abnormalities that may contribute to a reduced ability to function optimally as adults and possibly to a shortened life expectation. Other factors influencing the development of these children include a lack of environmental stimulation and a host of deprivations related to poverty.

The challenge to health workers and to development economists, to governments and to international agencies, is how best to reduce the morbidity, mortality, and permanent sequelae that result from this synergism of malnutrition and infection. The answer is not fancy hospitals such as those erected in the capital cities and provincial centers of developing countries, and it does not lie in elaborate manufactured foods such as spun soy protein or expensive infant formulas (*30*). The need is not for overtrained doctors nor for advanced food technology. A huge dent could be made in a huge problem by relatively simple means, if we in the affluent nations could summon up the resolve to make the reduction of deprivation our number one goal, and if the governments of developing countries could accept this as a number one priority. The politicians must be persuaded that attention to these problems is not only highly desirable but will have a political payoff.

The governments in both the developed and developing countries must first decide what "development" really means. Too often in the past, development has been viewed mainly in terms of industrialization, and measured in terms of productive capacity and material output of a country. Indicators of development were gross national product or mean per capita incomes.

Economists have tended to view improved nutrition and health as welfare questions. But it is now clear that these classical patterns of development often contribute very little to the quality of life of the majority of citizens of any country, and may sometimes aggravate the problems of the poor. We need therefore to ask, What is the purpose of economic development? Who is it for? If improved health and better nutrition for people are not considered in development plans, then one might seriously question whether this really is development.

Developing countries should, of course, strive for overall economic development, and especially for improvements in agricultural development. Support, however, should be given largely to those projects and that type of development that will benefit a large segment of the population, will help reduce inequalities in income distribution, and are likely to improve the nutrition, health, and quality of life of those currently deprived. For example, labor-intensive projects are often preferable to capital-intensive ones, and support for small farmers is more important than that to large estates (*31*).

The control of infectious diseases and projects aimed at providing more and better food for people are fully justified and important components of a development plan. By themselves they may contribute to increased productivity and better lives. But an improved infant or toddler mortality rate, a lowered disease incidence, and a better nourished population are perhaps better indicators of development than national averages of telephones or automobiles per 1000 families, or even than dollars or pesos per capita. Who would claim that development is far behind in China when childhood malnutrition has apparently been almost eliminated, major communicable diseases controlled, and the population increase stabilized? Yet China has very many fewer private telephones or automobiles per family and a much lower per capita income than many countries where malnutrition is common, health is poor, and the population is rising rapidly.

Conclusions

It is not possible here to provide a blueprint for development that includes the objectives of reducing infectious diseases and improving food intake. But it is increasingly realized that health care can be effectively delivered by persons with considerably less training than doctors. There are advantages in having many auxiliary medical workers in widely scattered dispensaries and health centers in villages and rural areas rather than having a few doctors and a hierarchy of supporting staff in hospitals in the principal towns. The majority of common illnesses can be successfully treated by health auxiliaries with simple equipment and a quite limited range of medicines. A health system using auxiliaries can be devised to place much more attention on preventive rather than curative medicine. These same auxiliaries can give immunizations, provide maternal and child health services including family planning, administer simple nutrition programs, and direct local public health measures.

In a similar way it is now widely accepted that most of the malnutrition in the Third World is due simply to inadequate intake of food. Protein deficiency

is not the most important nutrition problem in the world. In most populations where the staple food is a cereal such as rice, wheat, corn, or millet, serious protein deficiencies, although common, seldom occur except where there is also a calorie or overall food deficiency (*32*). There has been an overemphasis on the protein problem and too much stress on protein deficiencies with a relative neglect of the first goal of a food policy, which should be to satisfy the energy needs of the population. Most cereals contain 8 to 12 percent protein and are often consumed with moderate quantities of legumes and vegetables. If calorie requirements are met on these diets, protein deficiencies usually become uncommon, and are certainly confined mainly to very young children with increased nitrogen losses due to frequent infections. The situation in those whose staple food is plantain, cassava, or some other low protein food may be very different.

The lesson to be learned is that commercial production of relatively expensive protein-rich foods, the amino acid fortification of cereal grains, the production of single cell protein, and several other ventures that a few years ago were offered as panaceas for the world's nutrition problems can only reduce the problem of protein-calorie malnutrition to a very small degree. Similarly, genetic efforts to change by small amounts the amino acid pattern of cereal grains are much less important than increasing the yields per acre of these cereals, and of other food crops.

A modest increase in cereal, legume, and vegetable consumption by children will greatly reduce the prevalence of protein-calorie malnutrition and growth deficits of children in the Third World, especially if combined with control of infectious diseases. Breast feeding during the first few months of life can assure an adequate diet, whereas bottle feeding as discussed elsewhere in this issue (*33*) is a major cause of diarrhea and nutritional marasmus.

Fairly simple, relatively inexpensive nutrition programs can be used to control specific nutritional problems, the most important of which are vitamin A deficiency, which is a major cause of blindness; iodine deficiency leading to goiter and endemic cretinism; and anemias due to iron or folate deficiency. For example, xerophthalmia is being controlled in some areas by the provision to all young children every 6 months of a capsule containing 200,000 international units of vitamin A and in other areas by fortification of commonly eaten foods with the vitamin (*15*). Vitamin A is a relatively cheap nutrient, and the major cost is in the delivery system. There is no good reason why blindness due to vitamin A deficiency cannot be almost totally eliminated in the world in the next 10 years. Mandatory iodization of salt has been remarkably successful in reducing goiter and the mental deficiency associated with cretinism in several countries. Nutritional anemias could be controlled by using similar rather simple methods.

The control of infectious diseases and the improvement of nutrition both deserve a high priority in development plans and in international or bilateral assistance to low income countries. They should be instituted together because they will be mutually reinforcing and more economical if provided in a coordinated manner rather than separately. Allied to this is the need to provide a stimulating environment for the growing child.

Historical and epidemiological evidence suggest that reductions in infant and child mortality and improvements in health and nutritional status may be prerequisities to successful family planning efforts. In the overpopulated countries of the world, population control deserves a high priority, and parents in all countries should receive assistance to help them achieve their desired family size.

There seems to be clear logic in recommending coordinated programs that have three objectives: namely, to control infectious disease, to improve nutrition, and to make family planning services widely available. These three types of services may themselves be synergistic.

References and Notes

1. N. S. Scrimshaw, *J. Am. Med. Assoc.* **212**, 1685 (1970); M. C. Latham, *N.Y. State J. Med.* **70**, 558 (1970).
2. G. F. McLeary, *Infant Mortality and Infant Milk Depots* (King, London, 1905).
3. M. C. Latham, in *Nutrition, National Development and Planning*, A. Berg, N. S. Scrimshaw, D. L. Call, Eds. (MIT Press, Cambridge, Mass., 1973), pp. 313–328.
4. WHO Expert Committee, *WHO Tech. Rep. Ser. No. 314* (Geneva, 1965).
5. P. A. Shaffer and W. Coleman, *Arch. Intern. Med.* **4**, 538 (1909).
6. C. Martinez-Tomes, M. Roche, M. Layrisse, *Trans. R. Soc. Trop. Med. Hyg.* **61**, 373 (1967).
7. *Nutr. Rev.* **26**, 47 (1968); M. Layrisse and R. Roche, *Am. J. Hyg.* **79**, 279 (1964).
8. N. S. Stoll, *J. Parasitol.* **33**, 1 (1947).
9. P. S. Venkatachalam and V. N. Patwardhan, *Trans. R. Soc. Trop. Med. Hyg.* **47**, 169 (1953); K. Tripathy, E. Duque, O. Balanos, H. Lotero, L. G. Mayoral, *Am. J. Clin. Nutr.* **25**, 1276 (1972).
10. V. Reddy, personal communication.
11. C. P. Katsampes, A. B. McCoord, W. A. Philips, *Am. J. Dis. Child.* **67**, 189 (1944).
12. J. B. Salcmon, L. J. Mata, J. E. Gordon, *Am. J. Public Health* **58**, 505 (1968).
13. J. E. Gordon, I. D. Chitkara, J. B. Wyon, *Am. J. Med. Sci.* **245**, 345 (1963).
14. H. Hedayat, M. Gharib, M. Sadre, *J. Trop. Pediatr. Environ. Child Health* **14**, 124 (1968).
15. H. A. P. C. Oomen, *Nutr. Rev.* **32**, 161 (1974).
16. N. S. Scrimshaw, C. E. Taylor, J. E. Gordon, *WHO Monogr. Ser. No. 57* (Geneva, 1968).
17. P. J. Pretorius and L. S. de Villiers, *Am. J. Clin. Nutr.* **10**, 379 (1962).
18. R. E. Brown and M. Katz, *Trop. Geogr. Med.* **18**, 129 (1966).
19. V. Seth and R. K. Chandra, *Arch. Dis. Child.* **47**, 282 (19
20. C. Tejada, V. Argueta, M. Sanchez. C. Albertazzi, *J. Pediatr.* **64**, 753 (1964); H. H. Balch and M. T. Spencer, *Am. J. Clin. Invest.* **33**, 1321 (1954).
21. S. J. Ratham and K. S. Bhat, *Biochem. J.* **127**, 255 (1972).
22. C. G. Neumann *et al. Am. J. Clin. Nutr.* **28**, 89 (1975).
23. H. M. Coovadia, M. A. Parent, W. E. M. Loening, A. Wesley, B. Burgess, F. Hallett, P. Brain, J. Grace, J. Naidoo, P. M. Smythe, G. H. Vos, *ibid.* **27**, 665 (1974).
24. D. Morley, *Paediatric Priorities in the Developing World* (Butterworth, London, 1973).
25. R. R. Puffer and C. V. Serrano, *Patterns of Mortality in Childhood* (Pan American Health Organization–World Health Organization, Washington, D.C., 1973).
26. J. E. Gordon, W. Ascoli, L. J. Mata, M. A. Guzman, N. S. Scrimshaw, *Arch. Environ. Health* **16**, 424 (1968); W. Wittman, A. D. Moodie, S. A. Fellingham, J. D. C. Hansen, *S. Afr. Med. J.* **41**, 664 (1967).
27. M. C. Latham, L. F. Cobos, R. Rueda-Williamson, F. J. Stare, in *Proceedings of the Eighth International Congress of Nutrition*, Prague, 1969.
28. N. S. Scrimshaw, in *Preschool Child Malnutrition* (National Academy of Sciences–National Research Council, Washington, D.C., 1966), pp. 63–74.
29. C. E. Taylor and C. DeSweemer, in *Food, Nutrition and Health*, M. Rechsigl, Ed. (Karger, Basel, 1973), pp. 204–226.
30. M. C. Latham, *Physiol. Rev.* **54**, 541 (1974); J. Cravioto, E. R. DeLicardie, H. G. Birch, *Pediatrics* **38**, 319 (1966).
31. D. B. Jelliffe, *Food Technol.* **25**, 55 (1971); B. M. Popkin and M. C. Latham, *Am. J. Clin. Nutr.* **26**, 1015 (1973).
32. J. Mellor, in *Nutrition, National Development and Planning*, A. Berg, N. S. Schrimshaw, D. L. Call, Eds. (MIT Press, Cambridge, Mass., 1973), pp. 70–74; R. McNamara, address to the Board of Governors of the World Bank, Washington, D.C., 27 September 1971.
33. P. V. Sukhatme, *Indian J. Agric. Econ.* **22**, 7 (19
34. D. S. McLaren, *Lancet* **1974-II**, 93 (1974).
35. D. B. Jelliffe and E. F. P. Jelliffe, *Science* **188**, 557 (1975).