

NUTRITION AND INFECTION IN NATIONAL DEVELOPMENT

Mohammad Hussain,

Department of Biochemistry, Ayub Medical College, Abbottabad

Introduction

Why is it that the 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. 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. 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 related.¹ Yet we tend to introduce, quite independently, programmes to control communicable disease and other efforts to improve nutrition. It would be much more efficient and effective if the twin problems were attacked together. Success in improving depends both on control of infectious diseases and improvements in their food intake.

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.²

Nitrogen is lost by several mechanisms. The principal one is probably increased adrenocortical activity leading to mobilization of amino acids from various tissues especially muscles. The nitrogen is excreted in the Urine and is evidence of a depletion of 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 reduce 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 very dilute soups, water alone, or some other fluid with a low colour density and usually deficient in protein and other essential nutrients. The old proverb of "Strave 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 diarrhoea 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 illness such as measles, as 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 the *Ankylostoma doude-nale* and *Necator americanas*, is still prevalent in many countries. Hookworm causes intestinal blood loss and is a major cause of iron deficiency anaemia in many countries. The extent of the loss of blood and iron in hookworm infections has been studied.³

Daily faecal blood loss per hookworm was reported to be 0.031 ± 0.015 millilitre. It was estimated that about 350 hookworms in the intestine cause a daily loss of ml of blood or of 2 milligrams of iron.

The fish tapeworm (*DIPHYLLOBOTHRIUM LATUM*) has an avidity for vitamin B₁₂ and can deprive its host of this vitamin, with resulting megaloblastic anaemia. This parasite is common in man in only limited geographic areas where undercooked fish is frequently consumed.

Roundworm is the most prevalent of intestinal parasites.⁴ The roundworm is quite large (15-30 cm). So its own metabolic needs must be considerable.

Effect of Diarrhoea

Many studies have indicated that gastrointestinal infections and especially diarrhoea, are very important in precipitating the onset of both kwashiorkor and nutritional marasmus.⁵ Diarrhoea is common and often lethal to the young child. In breast-fed infants there is often some protection during the first months of life, and so diarrhoea is often a feature of weaning process. This weaning diarrhoea⁶ is extraordinarily prevalent in poor communities through out the world.

Diarrhoea was a major cause of mortality in children in industrialized countries upto 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.

Effects of Malnutrition on Infection

There is considerable literature to demonstrate, both in experimental animal and in man that dietary deficiency diseases may reduce the body's resistance to infections. Some of the normal defence mechanisms of the body are impaired and do not function properly in malnourished subjects.⁷ For example, children with kwashiorkor were shown to be unable to form antibodies to either typhoid vaccine or diphtheria toxin but the capacity to do so was restored after protein therapy.⁸ Similarly children with protein malnutrition have an impaired antibody response to inoculation with yellow fever vaccine.⁹

Another defence 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 greater importance, is the reduced phagocytic efficiency of the polymorphonuclear leukocytes in malnourished subjects. The cells appear to have a defect in their intracellular bactericidal capacity.¹⁰

The mechanism for this phenomenon have now been postulated and involves lowered adenosine triphosphate levels in the leucocytes of malnourished infants, combined with decreased activity of reduced nicotinamide adenine dinucleotide phosphate oxidase in response to phagocytic stimulation.¹¹

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.¹² 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.¹³

Discussion

It is now widely accepted that most of the malnutrition in the third world is due simply to inadequate intake of food. Most cereals contain 8 to 12 per cent protein and 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.

Fairly simple, relatively inexpensive nutrition programmes can be used to control specific nutritional problems, the most important of which are vitamin A deficiency, a major cause of blindness, iodine deficiency leading to goitre and endemic cretinism, and anaemias due to iron or folate deficiency.

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.

REFERENCES

1. Scrimshaw, N.A., JAMA, 1970; 212: 1985.
2. Shaffert P.A. and Coloman, W., Arch. Intern. Med: 1909; 4: 538.
3. Martinez Tomex, C., M. Roche, M. Lacyrisse, Trans. R. Soc: Trop. Med. Hyg: 1967; 61: 373.
4. Stoll, N.S., Parasitol, J. 1947; 33: 1.
5. Salemon, J.B., Mate, L.T., J.E. Gordon, Am: J. Public Health, 1968; 58: 505.
6. Gordon, J.E., Chitkara, I.D., Wyon, J.B., Am: J. Med: Sci: 1963; 245: 345.
7. Scrimshaw, N.S., Taylor, C.E., Gordon, J.E., W.H.O. Monorg. Ser: No. 57, 1968; Geneva.
8. Pretori P.J. 4 L.S. de villiers, Am: J. Clin: Nutr: 1962; 10: 379.
9. Brownx, R.E., Katz, M., Trop: Geogr: Med: 1966; 18: 129.
10. Seth, V. and Chandra, R.K., Arch. Dis: Child: 1972; 47: 282. Tejada, C., Argueta, V., Sanchez, M., Albertazzi, C., Pediate, J., 1964; 64: 753. Balch, H.H., Spencer, M.T., Am: J: Clin: Innest. 1954; 33: 1321.
11. Rathman, S.J., Bhat, K.S., Biochem: J. 1972; 127: 255.
12. Neumann Et. al. Am: J: Clin' Nutr: 1975; 28: 89.
13. Coovadia, H.M., Parent, M.A., Loening, W.E.M., Wesley, A., Burgess, B. and Hallett, F., Ibid, 1974; 27: 665.