Reprinted from JOURNAL OF THE AMERICAN DIETETIC ASSOCIATION
Vol. 42, No. 3, March, 1963
Printed in U.S.A.

Malnutrition and the Health of Children[1]

NEVIN S. SCRIMSHAW, Ph.D., M.D.

Head, Department of Nutrition, Food Science and Technology, Massachusetts Institute of Technology, Cambridge

THE HIGH infant mortality rates in technically underdeveloped areas are widely known and the plight of children under one year of age in these countries is often cited as the major health problem. It is true that infant mortality rates range from three to six times higher in such countries than in the United States and Western Europe. Table 1 gives infant mortality rates for the United States and several western countries compared with some technically underdeveloped countries for which information is available. Of each 1,000 children born live in 1958, over 100 died during their first year of life in Colombia, Ecuador, and Guatemala and over 80 in Mexico and El Salvador, in contrast to 27 in the United States and 30 to 31 for other western countries. While exact figures vary from year to year and are beginning to decrease in some of these countries, they are distressingly high.

Infant deaths are due to a wide variety of causes, including congenital malformations, birth injuries, post-natal asphyxia and atelectasis, and respiratory infections, such as influenza, pneumonia, and bronchitis (2). As the figures for the United States indicate, not all of these infant deaths are preventable by good nutrition or by improved hygiene and medical care.

However, in technically underdeveloped countries, malnutrition is an important factor in the death of children under one year of age, since many receive insufficient breast milk. When weaning occurs before one year of age, a child is often given either milk in very dilute form or a milk substitute, such as sugar water, rice water, or cornstarch solution, all of which are extremely deficient in both protein and calories. The result is usually marasmus, a form of partial starvation. Occasionally, if calories are relatively less deficient, the child under one year will develop kwashiorkor. This is particularly true of some urban centers in Africa and Trinidad where very early weaning is practiced so that the mothers can return to paid jobs.

Malnutrition in Children One to Four Years Old

While the high infant death rates common to most technically underdeveloped areas point to a serious problem and should stimulate strong public health measures directed at their prevention, they do not even begin to indicate either the full magnitude of the problem of high mortality rates among young children in technically underdeveloped areas or to reveal the consequences of malnutrition among such children. These are best shown by examining not only the mortality rates for children under one year but also those for children one to four years old. When data from technically underdeveloped countries are compared with those from the United States, Canada, and Western Europe, the tabulation shows mortality rates not three to six times higher but many which are thirty to sixty times higher (3-5) (Table 2). Moreover, malnutrition is directly or indirectly involved in almost all of the excess mortality in this age group. Unfortunately, for general understanding of the nature of the problem, this fact is not revealed in the usual breakdown of causes of death. A major reason is that deaths in technically underdeveloped areas are largely reported to lay registrars by parents or relatives who, in turn, attribute the death to worms, dysentery, and other infectious diseases, even when they are classic cases of kwashiorkor or marasmus.

In Guatemala, the Pan American Health Organization, Regional Office of the Americas for the World Health Organization in cooperation with the six countries of Central America and Panama, has

[1]Presented at a World Health Conference on Global Research for Global Health, School of Public Health, University of California at Los Angeles, October 6 and 7, 1961. Contribution No. 452 from the Department of Nutrition, Food Science and Technology, Massachusetts Institute of Technology and the Institute of Nutrition of Central America and Panama, Guatemala City, Guatemala.

TABLE 1 Infant mortality per 1,000 live-born in selected countries, 1958 (1)

COUNTRY	MORTALITY RATE
United States	27.1
Canada	30.2
Belgium	31.3
France	31.5
Japan	34.6
Thailand	55.5*
Mexico	80.1
El Salvador	88.7
Colombia	100.0
Guatemala	103.9
Ecuador	105.8
Chile	109.1

*1956 data (1).

TABLE 2 Specific mortality rates per 1,000 population of children one to four years old in selected countries (1955-56) (2-4)

COUNTRIES	MORTALITY RATE
Where Kwashiorkor Is Rare or Unknown	
Sweden	1.0
United States	1.1
Netherlands	1.2
Australia	1.3
Canada	1.5
Belgium	1.6
France	1.6
Argentina	3.8
Japan	3.8
Where Kwashiorkor Is Common	
Venezuela	12.5
Thailand	14.5
Colombia	20.3
Mexico	24.0
El Salvador	22.7
Ecuador	28.8
Guatemala	42.7
Guinea	55.4
Egypt	60.7

established the Institute of Nutrition of Central America and Panama (INCAP), which is studying nutritional problems, training personnel, and conducting educational and applied programs to prevent malnutrition. The mortality rate among children one to four years of age in Guatemala is over forty times that of the United States. While this figure is lower than Egypt's, it is still one of the highest in the world. INCAP personnel selected four villages with an average mortality for children one to four years of 50 per 1,000, a figure over fifty times higher than in the United States, and investigated the cause of each death by visiting the household (6).

When the causes of death as reported to the National Department of Vital Statistics were compared with those assigned by the INCAP workers, only one case attributed to malnutrition was found in the official figures. This was a child who died soon after being brought to the regional hospital and whose death was the only one to be medically certified. The INCAP workers, on the other hand, found that 38 per cent, or over one-third, of all of the deaths occurred in children with full blown symptoms of severe protein deficiency or kwashiorkor.

Infection and Its Concomitants

Kwashiorkor is characterized by edema, pigmented skin lesions, changes in the color and texture of the hair, apathy, anorexia, growth failure, serious biochemical imbalances, and pathologic tissue changes (7). The syndrome is the result of inadequate protein relative to calories and is fatal unless the quantity and quality of protein in the diet is improved. It is important to note that kwashiorkor is nearly always precipitated in an already chronically malnourished child by an infectious episode which is most commonly either dysentery or a childhood disease, such as measles or chickenpox.

Infection exerts its adverse influence on nutritional status in several different ways. Inevitably, a decrease in food consumption due to anorexia and even actual intolerance of food results. Even more importantly, in technically underdeveloped areas, the quality of food offered to the child is changed because mothers and often physicians, too, believe that solid food should be taken away from the sick child, especially from a child with diarrhea, and liquids which are generally low in protein are substituted. Similarly, infections result in an increased loss of cellular protein which is manifest by extra nitrogen excreted in the urine and frequently by a significant nitrogen imbalance.

One quarter of the deaths in the four communities investigated appeared to be a direct result of infectious diarrhea. Few well nourished children ever die of this cause and almost none within 24 to 48 hr. after onset as occurs frequently in malnourished populations. Most of the remaining deaths, 37 per cent, seemed to result from other infections secondary to childhood disease, but which again would rarely be fatal in a well nourished child. Thus, even the deaths attributed to infection were really due to the combined effects of infection and malnutrition—a synergism, so to speak, in which the consequences of the combination were far worse than would be predicted for either one alone.

For example, in 1956 the death rate from measles in Mexico was 164 times higher per 100,000 population than in the United States. In Guatemala, it was 228 times higher, and in Ecuador, 368 times higher. This was not because the measles virus was more virulent or prevalent in these countries, or even because of the differences in medical care, but because the resistance of the host (the child) was lower. We believe that this lower resistance is due

primarily to malnutrition. Measles is a good example for the purpose because it occurs almost universally in children, regardless of social and economic status and even without medical care is not usually a fatal disease in well nourished populations.

It should be emphasized that the synergism works both ways. As mentioned previously, most of the deaths attributable to severe protein malnutrition were really attributable to a combination of dietary deficiency and infectious stress. Of course, the relative proportion of deaths due to the synergism between malnutrition and infection varies from one technically underdeveloped country to another; nevertheless, the principle remains the same. Nearly all of the great excess of mortality among children one to four years old, as compared with developed countries, is associated directly or indirectly with malnutrition acting synergistically with infection. In some technically underdeveloped areas, half of the children born alive are dead before age five, as a result of both a high infant mortality and a high mortality in the one-to-four-year-age group.

Relation of Mortality to Morbidity

These mortality statistics are striking, but they tell only a small part of the sad story. Because for every child in the one-to-four-year-age group who dies, many more suffer seriously from malnutrition during this age period. In fact, growth and maturation studies among the preschool children in the lower income groups in many technically underdeveloped countries reveal that nearly all children pass through a period of several years following weaning in which growth and development are almost at a standstill. The evidence indicates that this is due primarily to protein malnutrition. The death of so many children during this preschool age period, which is characterized by delayed growth and development, is due to either a lowering of resistance to infection by the malnutrition or exaggeration of malnutrition by infection and its sequelae. These consequences include not only loss of appetite and a tendency to withdraw solid food because of the illness, but also to the loss of protein from damaged or destroyed cells.

Instead of relying on mortality statistics, the severity of protein malnutrition among preschool children in a community can be judged by the frequency of clinical cases of kwashiorkor, but this requires a house-to-house canvas. Some idea can be obtained, however, by visiting hospital and outpatient clinics. By this means, kwashiorkor has been shown to be a public health problem in nineteen of the twenty-one countries of the Americas, all of the countries and territories of Africa south of the Sahara, in India, and in most countries of the Middle and Far East (8). Clinical cases, whether estimated from mortality figures or surveys of hospital and clinic patients, represent only part of the problem. As suggested, clinical cases of kwashiorkor are more like the tip of an iceberg, protruding above the ocean surface but with its hidden vast bulk corresponding to the concealed cases of underlying protein malnutrition.

If the almost universal occurrence of underlying protein malnutrition among children of lower income groups in technically underdeveloped areas meant only increased mortality due to a synergism of nutrition and infection, this would be bad enough. If the children who survived suffered nothing more than permanent stunting of their growth and development, this might be considered a useful adaptation. Unfortunately, recent studies from Mexico suggest that intelligence is impaired as well. Ramos-Galván and co-workers are finding a significant correlation between retardation in height and weight and the score of children reaching school age on the standard Goodenough "Draw-a-Man" test and on the standard Gesell tests (9, 10).

Retardation in weight-for-age may reflect to some extent current nutritional status, but it is largely a reflection of impaired nutritional status during several of the formative years. Retardation in height is more clearly due to previous malnutrition. If malnutrition during preschool years affects the intellectual performance of children, it is essential to prevent malnutrition in preschool children if educational measures are to have a fair chance of contributing to the improvement of the productivity, prosperity, and democracy of a country. In other words, malnutrition during the preschool years is serious both for the children who survive and the many who die as a direct or indirect consequence of it.

Other Deficiencies in Children

While protein malnutrition is the most universal nutritional deficiency among children in the world today and is a problem likely to become worse in many areas as population growth continues to match or even outstrip increases in food supply, there are other nutritional deficiencies which also seriously affect many of the world's children.

One is infantile beriberi. This condition is characterized clinically by pallor, insomnia, restlessness, anorexia, vomiting, and terminally by cyanosis, dyspnea, tachycardia, and sudden death a few hours after onset (11). The disease is actually increasing in some parts of Southeast Asia, notably Thailand, because the introduction of gasoline-driven rice mills in small villages has displaced home-pounding which left a greater proportion of hull and thiamine-containing germ. As a consequence, the mothers receive less thiamine in their diet and secrete less thiamine in their milk. If this vitamin is sufficiently inadequate, the child is likely suddenly to develop symptoms at two or three months of age.

In Yugoslavia, Egypt, Basutoland, and a number of other countries, pellagra is still a problem in those portions of the population consuming a predominantly corn diet (12). Corn is deficient in both niacin and tryptophan, the amino acid precursor of niacin. Pellagra, due to niacin deficiency, affects children and adults alike and is exacerbated by exposure to strong sunlight. Its occurrence tends to be seasonal because the time of poorest diet coincides with greatest exposure to sunlight during work in the fields.

VITAMIN A DEFICIENCY

Even more widespread is vitamin A deficiency. In mild form, at least, this deficiency almost parallels the occurrence of kwashiorkor and is responsible for occasional cases of xerophthalmia and keratomalacia superimposed on kwashiorkor and marasmus. In Indonesia, it is responsible annually for many thousands of cases of keratomalacia and much preventable blindness. Like kwashiorkor, it is often precipitated by measles or some other infection in a child whose diet is borderline in vitamin A. A recent report from the World Health Organization (12) estimates that 5 per cent of all children in Indonesia have impaired vision or are blind as a consequence of vitamin A deficiency.

One indication of the extent to which the child population of Indonesia subsists on a borderline status with respect to vitamin A is indicated by the experience with free distribution of a large quantity of skim milk a few years ago. The augmented protein intake increased the frequency of xerophthalmia and keratomalacia sharply by increasing the requirement for vitamin A, and importation of the skim milk had to be prohibited until arrangements could be made for distribution of supplemental vitamin A capsules (13). If whole milk with its natural vitamin A content had been used, this would not have occurred. Economic factors, however, make it convenient to use skim milk to prevent protein malnutrition in many parts of the world.

ENDEMIC GOITER

Another very common nutritional deficiency in which children are seriously involved is endemic goiter. Unless the diet includes iodine-containing foods or iodized salt, this condition appears wherever the soil lacks iodine, thus rendering water supplies and locally-grown vegetables deficient in this element. The requirements for iodine may be increased by a variety of goitrogenic factors, including a chemical substance found in plants of the family *Brassica* to which Brussels sprouts and the common cabbage belong (14), and also possibly by vitamin A deficiency (15, 16) and by water supplies rich in lime (17). Adequate iodine intake will always prevent the condition.

Some idea of the widespread occurrence of endemic goiter may be gained from the realization that it is, or has been until control measures were introduced, a public health problem in every country and territory on the mainland of the western hemisphere, in much of Africa, in the mountainous areas of Europe, in parts of India, and in some countries of the Far East (18). It is readily surveyed by examining school children whose soft diffuse goiters reflect the current status of iodine nutriture of the population. Administration of iodine causes the majority of goiters in school children to disappear in as short a period as twelve weeks even in areas where over 50 per cent of the children have goiters (19). When the goiter persists, it gradually enlarges, becomes fibrous, and turns into the irreversible large visible goiters so common in adults in goitrous areas.

Usually, the hypertrophy of the gland is a successful compensatory mechanism for more efficient extraction of iodine from the blood stream and is not associated with metabolic changes in children. When goiter occurs in pregnant women, it appears responsible for cases of endemic cretinism and may be associated with an increase in still-births, deaf mutes, and congenital abnormalities.

RICKETS

Rickets, once a scourge of northern countries because of the wintertime shortage of sunlight needed for the conversion of ergosterol in the skin to active vitamin D, has all but disappeared in these countries because of the use of vitamin D concentrates. Rickets still occur in children in the tropics who, for special reasons, are not exposed to the sunlight. In the slums of tropic cities, children are sometimes deprived of sunlight because they are ill, live in tenements with little opportunity for exposure to the sun, or stay with the working mother whose occupation keeps her inside during the daylight hours.

How WHO Helps to Solve These Problems

The remaining section of this presentation will be devoted to describing some of the ways in which The World Health Organization (WHO) is making an important and effective contribution to the solution of these problems.

In 1945, the Food and Agriculture Organization (FAO) was founded, followed three years later by the establishment of WHO. The two organizations convened the first meeting of the Joint Expert Committee on Nutrition (20) in Geneva in 1948, where recognition was given to the existence of an ill-defined nutritional syndrome observed previously in the tropics and subtropics. Through the initiative of Dr. Frederick Clements, the first Chief of the Nu-

trition Section of WHO, Dr. John Brock on behalf of WHO and Dr. Marcel Autret representing FAO were sent to survey the problem in ten countries and territories of Africa in 1950. The now famous Brock-Autret report (21), adopting the name *kwashiorkor*, clarified the fact that a variety of local names referred to a single disease which was prevalent throughout Africa and led to the realization of the problem of kwashiorkor in other parts of the world. Similar joint WHO/FAO surveys (22, 23) of kwashiorkor in Central America and Brazil followed.

In 1953, the Third Joint Expert Committee met in Gambia and viewed the kwashiorkor problem first hand (24). Earlier, WHO initiated a program of travel grants which enabled the leading workers in the field in Africa, India, Latin America, and other regions (where kwashiorkor was a major clinical and public health problem) to visit other laboratories and field projects. WHO also financed the sending of consultants to many different countries to assist local workers and to collect information. Their work resulted in the development of more uniform criteria and a better understanding of the characteristics, epidemiology, treatment, and prevention of the disease. In addition, several research projects were supported which seemed likely to contribute importantly to the solution of the problem.

With the assistance of the Josiah Macy, Jr. Foundation, WHO and FAO sponsored three international round-table conferences for persons working with the syndrome. In 1953, twenty-six persons from twelve countries and territories assembled in Jamaica to discuss the problem of protein malnutrition (25). The second and third such conferences, one held at Princeton, New Jersey in 1955 (26) and one at Cuernevaca, Mexico in 1960 (27), brought together active workers from all over the world. These meetings served to familiarize the participants with current studies and to stimulate further research and applied programs for the prevention of the disease.

THE PROTEIN-RICH FOODS PROGRAM

That a partial answer at least lay in the development of new foods, including products not previously deemed suitable for human consumption, became increasingly evident. In 1955, the Director General of WHO appointed a Protein Advisory Group which recommended criteria for the acceptance of new food products for human consumption and stimulated a broad program of research and development of protein sources. This group, whose name was soon abbreviated to PAG, influenced strongly the policies not only of WHO but also of FAO and the United Nations Children's Fund (UNICEF) and has now been established as a joint advisory group to the three organizations.

PAG was instrumental in obtaining support from the Rockefeller Foundation totaling over half a million dollars. The sum has been invested in research efforts to develop new protein sources for human feeding and has been spent mainly for work in technically underdeveloped areas. The program led to a conference held in Washington in August 1960 to summarize the results of this and related research sponsored largely by a grant from the U. S. National Institutes of Health. The proceedings of the conference have been published in a book, *Progress in Meeting Protein Needs of Infants and School Children* (28). The tremendous significance of the program for the people of technically underdeveloped areas is manifest on perusal of this report. The report describes work with: soya protein in Brazil; fish flour in Mexico, Peru, and Chile; a cereal-cottonseed mixture (Incaparina) in Central America; leaf protein concentrates in Jamaica; peanut-cereal mixtures in Uganda and Nigeria; a protein-supplemented maize in South Africa; peanut flour in Senegal; various oil seed meals in the Congo; a chick pea-peanut flour mixture for India (Indian Multipurpose Food); fermented soybean products, such as "natto" and "tofu" for Japan and "tempeh" for Indonesia; plus many more promising developments.

The total protein-rich foods program sparked by the PAG and the staffs of WHO, FAO, and UNICEF has resulted in the construction of UNICEF-equipped plants to produce fish flour in Chile and Morocco and a soya milk preparation called "Saridele" in Indonesia, and the local production of powdered milk in many countries. Commercial production of Incaparina, a low-cost, protein-rich vegetable mixture has begun in several countries of Latin America. The Indian Multipurpose Food mixture, consisting mainly of a combination of peanut flour and chick pea, is now being used in India, and a mixture of peanut flour and corn with added skim milk is being distributed in Uganda on a pilot basis. Many other research developments reported at the Washington conference are expected to reach practical application soon.

The most extraordinary and hopeful development in international nutrition has been the way in which in the last decade WHO, in cooperation with FAO and UNICEF, has led a vigorous and effective assault on the widespread problem of protein malnutrition and on the accompanying high prevalence of kwashiorkor and marasmus in young children in technically underdeveloped areas. This effort has resulted in not only a better understanding of the problem, but also in the development of practical measures for prevention. It is an inspiring record of accomplishment with the full effects only beginning to be felt.

WHO is now rapidly expanding its sphere of

activity with an extensive and intensive program to coordinate and stimulate research on many major health problems. Areas of research to be supported include the development of simplified methods for determining nutritional status and their application where serious malnutrition is occurring; further studies of kwashiorkor and marasmus; the epidemiology and prevention of hypovitaminosis A; the prevalence and prevention of nutritional anemia; and the epidemiology and prevention of infantile beriberi. Add to these: the medical and public health significance of endemic goiter, the effect of intestinal parasites on the nutritional status of children, interrelationships of malnutrition and infection, patterns of growth and development in underdeveloped areas, and the effect of acute and chronic malnutrition on them.

The results of the attack on malnutrition and its related disorders under the aegis of WHO, FAO, and UNICEF will assure infants of the food needed for normal growth and development and result in healthy, productive adults better able to develop fully their mental and physical potential.

References

(1) Epidemiological and Vital Statistics Report. Vol. 11. Geneva, Switzerland: World Health Organ., 1958, p. 98.

(2) Epidemiological and Vital Statistics Report. Vol. 13. Geneva, Switzerland: World Health Organ., 1960, p. 527.

(3) Summary of Four-Year Reports on Health Conditions in the Americas. Pan Am. Sanitary Bur. Regl. Off. WHO, Scientific Pub. No. 40, 1958.

(4) Demographic Year Book 1957. 9th ed. N.Y.: United Nations, 1957.

(5) Enquête demographique 1954-1955. Haut Commissariat de la République Française.

(6) BEHAR, M., ASCOLI, W., AND SCRIMSHAW, N. S.: An investigation into the causes of death in children in four rural communities in Guatemala. WHO Bull. 19: 1093, 1958.

(7) SCRIMSHAW, N. S., BEHAR, M., ARROYAVE, G., TEJADA, C., AND VITERI, F.: Kwashiorkor in children and its response to protein therapy. JAMA 164: 555, 1957.

(8) SCRIMSHAW, N. S., AND BEHAR, M.: World-wide occurrence of protein malnutrition. Fed. Proc. 18: 82 (Suppl. 3, Pt. 2), 1959.

(9) ROBLES, B., RAMOS-GALVAN, R., AND CRAVIOTO, M. J.: Evaluation of the behavior of the child with advanced malnutrition and of its modification during recovery (preliminary report). Bol. Med. Hosp. Infant. (Mex.) 16: 317, 1959.

(10) RAMOS-GALVAN, R., PEREZ NAVARRETE, J. L., AND CRAVIOTO, M. J.: Various aspects of growth and development of Mexican children. Bol. Med. Hosp. Infant. (Mex.) 17: 455, 1960.

(11) BURGESS, R. C.: Beriberi. 6. Special problems concerning beriberi. B. Infantile beriberi. Fed. Proc. 17: 39 (Suppl. 2, Pt. 2), 1958.

(12) Joint FAO/WHO Expert Committee on Nutrition, Fourth Report. WHO Tech. Rep. Ser. 97, July 1955.

(13) OOMEN, H. A. P. C.: Clinical experience on hypovitaminosis A. *In* Nutritional Disease—Proceedings of a Conference on Beriberi, Endemic Goiter and Hypovitaminosis A. (Kinney, T. D., and Follis, R. H., Jr., eds.) Fed. Proc. 17: 162 (Suppl. 2, Pt. 2), 1958.

(14) ASTWOOD, E. B., GREER, M. A., AND ETTLINGER, M. G.: L-5-vinyl-2-thiooxazolidone, antithyroid compound from yellow turnip and from Brassica seeds. J. Biol. Chem. 181: 121, 1949.

(15) GREER, M. A.: Nutrition and goiter. Physiol. Rev. 30: 513, 1950.

(16) HAUBOLD, H.: Der Einfluss des Carotinoidmangels auf die Nachkriegsstrumen der Erwachsenen. Verhandl. deutsch. Gesellsch. inn. Med., Kong. 57: 112, 1951.

(17) MURRAY, M. M., RYLE, J. A. SIMPSON, B. W., AND WILSON, D. C. Thyroid Enlargement and Other Changes Related to the Mineral Content of Drinking Water (with a Note on Goitre Prophylaxis.) Med. Res. Council Mem. No. 18. London: H. M. Stationery Off., 1948, p. 39.

(18) KELLY, F. C. AND SNEDDEN, W. W.: Prevalence and geographical distribution of endemic goitre. WHO Bull. 18: 5, 1958.

(19) SCRIMSHAW, N. S., CABEZAS, A., CASTILLO, F., AND MENDEZ, J.: Effect of potassium iodate on endemic goitre and protein-bound iodine levels in school-children. Lancet 2: 166, 1953.

(20) Joint FAO/WHO Expert Committee on Nutrition. Report on the First Session. WHO Tech. Rep. Ser. 16, 1950.

(21) BROCK, J. F., AND AUTRET, M.: Kwashiorkor in Africa. FAO Nutrition Studies No. 8, 1952.

(22) AUTRET, M., AND BEHAR, M.: Síndrome Policarencial Infantil (Kwashiorkor) and Its Prevention in Central America. FAO Nutrition Studies No. 13, 1954.

(23) WATERLOW, J., AND VERGARA, A.: Protein Malnutrition in Brazil. FAO Nutrition Studies No. 14, 1956.

(24) Joint FAO/WHO Expert Committee on Nutrition Report on the Third Session. WHO Tech. Rep. Ser. 72, 1952.

(25) Protein Malnutrition. Proceedings of a Conference in Jamaica (1953) Sponsored Jointly by the Food and Agriculture Organization of the United Nations, the World Health Organization and the Josiah Macy, Jr., Foundation. (Waterlow, J. C., ed.) London: Cambridge Univ. Press, 1955.

(26) Human Protein Requirements and Their Fulfillment in Practice. Proceedings of a Conference in Princeton, New Jersey, 1955, Sponsored Jointly by the Food and Agriculture Organization of the United Nations, the World Health Organization and the Josiah Macy, Jr., Foundation. (Waterlow, J. C., and Stephen, J. M. L., eds.). London: H. M. Stationery Off., 1957.

(27) Proceedings of conference held in Cuernavaca, Mexico, 1960, sponsored jointly by the Food and Agriculture Organization of the United Nations, the World Health Organization, and the Josiah Macy, Jr., Foundation. In press.

(28) Meeting Protein Needs of Infants and Children. Natl. Acad. Sci.-Natl. Research Council, Pub. No. 843, 1961.