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World-Wide Occurrence of Protein Malnutrition

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IN 1933 Dr. Cicely Williams, working in the Gold Coast, colony of Western Africa, wrote (1):

"There is a well-marked syndrome...which I have not found described. (It) consists of oedema, chiefly of the hands and feet, followed by wasting; diarrhoea, irritability; sores, chiefly of the mucous membranes; and desquamation of areas of the skin in a manner and distribution which is constant and unique.

The disease attacks children of either sex, between one and four years old. It appears to be due to some dietetic deficiency and to be uniformly fatal unless treated early.

In all of the cases seen there was a history of an abnormal diet. Breast-feeding had been given by an old or else a pregnant woman, and the only supplementary food consisted of preparations of maize.

This defective feeding has gone on for four to twelve months. Then he (the child) begins to get irritable; there are attacks of diarrhoea; and swelling of the hands and feet. At the end of a week to ten days, the skin changes set in. If untreated, the child dies, generally within a month of the skin changes...The hair also...becomes dry, pale and sparse."

Her description of the skin changes is still unsurpassed:

"There suddenly appear on the ankles, on the knees, above the wrists, and on the elbows, some small black patches, first on the extensor surfaces and gradually spreading. .... The number and extent of these patches increase. Their distribution and their character remain distinct from the rash that is typical of pellagra. Soon the legs and forearms, knees and elbows, are covered with a sort of crazy pavement of this thickened epidermis. In a few days as the older patches mature, they strip off very readily, leaving a pink, raw surface exposed underneath. ... The patches of desquamation progress up the thigh and may become severe on the buttocks. Very small patches may also be seen on the face, back and elsewhere. The appearance of the skin condition at this stage is striking. There is the dark or reddish brown of the unaffected skin, the black patches of crumpled and thickened epidermis, and the raw areas where these have peeled off."

Three years and sixty cases later, Dr. Williams amplified her description of the disease and for the first time applied the local native name 'kwashiorkor' to it (2). This, of course, is neither the beginning nor the end of the kwashiorkor story. Although Dr. Williams did not know it at this time, she was merely giving one more name to a condition which had puzzled and disturbed many other persons and which was soon destined to become recognized as a major health problem in the world's technically underdeveloped areas.

Some of the descriptions in colonial times of the consequences of the artificial feeding of infants with cereal pap sound very much like the same condition (3). Nevertheless, as far as we can determine, the first clear description of the disease was published by Czerny and Keller in Germany in 1906 (4) under the name of 'Mehlnarschaden,' literally, 'starch dystrophy.' The syndrome seems to have been next recognized in Yucatan, Mexico in 1908 by Correa (5) as 'Culebrilla' which refers to the 'snake-like skin.' The disease was next described in Africa in 1918 by Jamot (6) who used the French West African name 'Dacaga,' and McConnel (7) in Uganda who called it 'edema disease.' During the 1920's reports were appearing with increasing frequency from almost every part of the globe. The disease was described in 1923 from Japan as 'Chichiko dyspepsia' (8); in 1924 from Kenya as 'edema with ascaris' (9); in 1927 from Italy as 'starch dystrophy' (10); Haiti as 'edema disease' (11) and France in 1927 (12) and Cuba in 1929 (13) as 'nutritional edema.'

Special mention should be made of the description of Normet (14) in Indochina who used the name 'Annam swelling' and wrote in 1926 that the evidence pointed to a disturbance of nitrogen metabolism. He not only considered the disease to be a new clinical entity, but also stated it to be the major cause of death in this country in adolescents and adults as well as young children. In 1928 Normet (15) again rejected completely the prevailing view that various infections and particularly hookworm could be responsible for the disease and reasserted it to be a manifestation of protein deficiency.

In South Africa the syndrome was called 'infantile scurvy' in the early 1930's (16) and 'infantile pellagra' throughout the 1940's (17). An account by Lieurade (18) in French Equatorial Africa, contemporaneous with that of Williams, referred to it as 'red children,' a name which mistakenly came to be considered by some later writers as the meaning of the word kwashiorkor. The literal translation of the 'Ga' word kwashiorkor is revealing of native insight into the epidemiology of the disease since it means 'first-second' and refers to the fact that the disease tends to occur in the first child when a second is born, i.e. when the first child is deprived of mother's milk (19).

Concurrent with and subsequent to the 1932 and 1935 reports of Williams, and generally without knowledge of them, the disease described by her as kwashiorkor was reported for the first time in many more countries—in 1934 in Tanganyika as 'Gillan's edema' (20) and in El Salvador as 'infantile edematous cachexia' (21); in 1937 as 'avitaminosis' in the Belgian Congo (22); in 1938 as 'vitamin deficiency edema' in Costa Rica (23) and as 'multiple dietary deficiency syndrome' in Guatemala (24). This name was also used in reports from Honduras (25) and Venezuela (26) in 1939. In the same year in Egypt (27) and Morocco (28) the name 'nutritional edema' was used.

And so the story continues—After 1940 recognizable descriptions of kwashiorkor added many additional countries, among them Chile (29), Colombia (30), China (31), Uruguay (32), Nigeria (33), Brazil (34), Rhodesia (35), Jamaica (36), Hungary (37), Greece (38), India (39), Indonesia (40), the Philippines (41), Fiji (42), Curaçao (43), Spain (44), Ceylon (45). The 1949 monograph by Meneghello (46) of Chile on *Multiple Deficiency Dystrophy* is still one of the most complete discussions of kwashiorkor available.

In 1950 an event occurred which was to tie these scattered studies together and guide the world's physicians and health workers to the cause and prevention of the syndrome, but other developments led to it. The Food and Agriculture Organization of the United Nations (FAO) was founded in 1945 and the World Health Organization (WHO) 3 years later. The first meeting of the Joint Expert Committee on Nutrition of the two organizations, held in Geneva in 1949 (47), had taken cognizance of ". . .The existence of a poorly defined nutritional syndrome of the tropics and subtropics, known by the name of Kwashiorkor, Malignant Malnutrition, Multiple Deficiency, Depigmentation-Edema Syndrome, Infantile Pellagra, etc. . . ."

The Committee recommended a study of the various aspects of kwashiorkor, and a commission made up of John Brock of Capetown representing WHO and Marcel Autret of FAO set out in mid-October of 1950 to visit the 10 countries and territories of Africa South of the Sahara. The result was the now famous Brock-Autret report (48) which firmly established the name kwashiorkor and which made it clear that the various other names referred to a single disease widely prevalent throughout Africa. The subsequent joint WHO/FAO surveys of Autret and Béhar (49) in Central America and Waterlow and Vergara (50) in Brazil made clear the essential identity of protein deficiency disease as it was widely occurring in Latin America with the kwashiorkor of Africa.

In 1953 WHO and FAO, this time with the assistance of the Josiah Macy Jr. Foundation, were once again responsible for a major stimulus in this field by arranging an international round-table conference of persons working with the syndrome, many of them without previous contact with similar workers elsewhere (51). Twenty-six persons from 12 countries and territories met in Jamaica for an entire week to discuss the problem of protein malnutrition and discovered, although a great deal of funda-

FIG. 1. Geographical distribution of kwashiorkor.

mental knowledge was still lacking, that they were unquestionably dealing with a common syndrome. Largely from the inspiration of this meeting and from a series of exchange visits among African, Indian and Latin American workers (financed by WHO) have come the numerous fundamental research studies of the last 5 years.

Progress was so rapid that when a second joint FAO/WHO/Macy Foundation conference was convened on the same subject in June 1955 in Princeton (52), emphasis was no longer on the clinical and biochemical characteristics of kwashiorkor or even its epidemiology, but almost entirely on how it could be prevented.

From this historical review it should be apparent that kwashiorkor is not a rare and exotic disease affecting a few children in a small number of far away tropical countries but, as figure 1 illustrates, is a major public health problem for a very large proportion of the areas and peoples of the world.

Thus far only the amount and distribution of clinical kwashiorkor have been mentioned, but frank cases of the syndrome are only a small part of the total problem of protein deficiency in children. They indicate a very widespread, frequently almost universal, occurrence of protein malnutrition in the general child population of pre-school age in an area. The prevalence of kwashiorkor has been compared with the visible portion of an iceberg which has six-sevenths of its bulk concealed beneath the surface of the water. Such a comparison, although graphic, fails to indicate the true quantitative relationship. For every case of kwashiorkor occurring in a population, there are not six, as suggested by the iceberg simile, but at least a hundred cases of underlying protein malnutrition—sometimes referred to as prekwashiorkor. The simile of the kwashiorkor-prekwashiorkor relationship should not be an iceberg but an underseas mountain with only the tip of its peak protruding above the surface.

Physicians working with children in hospital or outpatient clinics in areas in which kwashiorkor is endemic recognize severe protein malnutrition to be a serious problem. There is, however, a notable lack of appreciation of its magnitude and importance on the part of public health administrators and vital statisticians, who usually consider diarrheal disease a far more serious problem in this age group. Furthermore, the vital statistics gathered from these regions seem to confirm this latter point of view. The explanation lies in the fact that deaths of children with kwashiorkor are almost invariably listed as due to causes which are later coded within the category of diarrheal and parasitic diseases and hence are not identifiable in the official vital statistics of a country as deaths due to severe protein malnutrition. Furthermore, those deaths occurring in children with underlying protein malnutrition which are the immediate result of diarrheal and other infections are quite

TABLE 1. DEATHS 1–4 AGE GROUP 4
GUATEMALAN HIGHLAND VILLAGES

CAUSES	CIVIL REGISTER	INCAP STUDY
RESPIRATORY INFECTIONS	15	15
INFECTIOUS DISEASES	11	14
PARASITIC DISEASES	45	0
DIARRHEA	15	25
SEVERE MALNUTRITION (MOSTLY KWASHIORKOR)	1*	40
OTHER	22	15

* DIED IN A HOSPITAL INCAP-1958

naturally not listed as due to malnutrition, even though a well-nourished child would not have died from the infection alone.

These points are well illustrated by a recent 2-year study carried out in 4 representative villages in the Guatemalan highlands (53). Each death was personally investigated and the causes determined in this manner compared with those given in the Civil Register. With a single exception, the latter were based on the statements of the parents as interpreted by the town Secretary. The results are shown in table 1. Of the 109 child deaths 1–4 years of age investigated over a 2-year period, 40 occurred in children with the signs and symptoms of clinical kwashiorkor, yet only one was officially listed as dying from malnutrition and this was the only child in the group to die in a hospital and to have his death medically certified. The deaths of most of the children dying with kwashiorkor had been ascribed in the town records to 'parasites' or to 'worms.' Twenty-five children died of infectious diarrhea and an additional number of other infectious diseases which would not ordinarily have been fatal to a well-nourished child.

It is not exaggerating the role of protein malnutrition in this situation to point out that the difference between the mortality rate for children 1–4 in these villages of 42/1000 during the study compared with 1.1/1000 in the United States in 1956 could largely be eliminated if protein malnutrition could be prevented. Elimination of the infectious disease might also prevent the majority of these deaths, but it would not restore normal growth and development to children in this area.

The main characteristics of kwashiorkor are now familiar: retarded growth and development, apathy and anorexia, edema, pellagroid skin lesions, alterations in the color, texture or implantation of the hair, fatty liver, and diarrhea. Not every one, however, realizes that protein malnutrition is found in a continuous range from the classical forms in which calories are adequate, even abundant, as in the 'sugar-baby' type described from Jamaica (54), to those in which there has been a severe calorie deficit and considerable wasting has ensued.

In the sugar-baby type of kwashiorkor in which calories have been abundant and tissue wasting has not occurred, the child has all of the characteristic clinical, biochemical and pathological lesions of kwashiorkor, recovers readily when fed protein of good quality, and once the edema has disappeared, looks relatively healthy.

In a child with marasmus tissue wasting occurs as a result of partial starvation. Such a child has suffered protein deficiency but with an equally severe calorie deficiency, so that he has been to some extent living on his own tissues. For these reasons he does not develop the clinical, pathological or biochemical stigmata of kwashiorkor. The kwashiorkor syndrome as it is most often seen in nearly all endemic areas is combined with a varying degree of marasmus and is often spoken of as marasmic kwashiorkor.

The age at which weaning takes place and its abruptness largely determine the extent to which tissue wasting occurs before the signs of kwashiorkor are superimposed. In Latin America many cases are so close to marasmus that after treatment has been initiated and the edema has disappeared, they are indistinguishable from it except for the possible persistence of traces of the former skin lesions and of discolored bands in the hair. Clinical history can often be read in hair; in the simplest case an outer band of light brown corresponds to a period

of underlying protein malnutrition preceding the development of clinical kwashiorkor; a pale whitish band in the middle corresponds to the episode of kwashiorkor, and a black band at the base is normal hair growing out during treatment in the hospital. Depending upon the past history, additional bands may be seen.

Once the range of possible relationships between protein and calorie deficiency is understood and the different vitamin deficiencies which may be associated with kwashiorkor in various degrees are recognized, regional differences are easier to understand and it becomes clear that the basic syndrome is the same the world over. Most authorities working in the field now believe that the basic cause of kwashiorkor has been established conclusively to be a deficiency of protein relative to calories. It has been repeatedly demonstrated that the syndrome responds promptly and completely to skim milk or to vegetable mixtures of good protein quality (55). These preparations are, of course, also sources of other essential nutrients, but it has been further shown that the basic signs of kwashiorkor disappear or are greatly improved when vitamin-free casein (56) or even suitable mixtures of synthetic amino acids (57) are given.

That vitamin deficiencies become limiting and vitamins are required for further recovery beyond approximately 2 weeks, in no way weakens the argument for protein deficiency as the basic cause of kwashiorkor, since no animal organism will do well for any length of time on a diet providing protein, calories and minerals but no vitamins. The argument that kwashiorkor should be traced to the deficiency of one or more individual amino acids is not a very practical one. Even though certain of the signs of kwashiorkor may be due to deficiencies of specific amino acids, all of our growing knowledge of amino acid requirements suggests that the adequate utilization of any single essential amino acid requires that a balanced mixture of all be administered.

Kwashiorkor does occur in adults and has been so described from a variety of countries (58, 59). It occurs most often in pre-school children, however, because the diet after weaning is particularly likely to be relatively deficient in protein and because the protein requirements of the child per kilogram of body weight are higher at this time than at any later period in life. It is also an age when children are particularly vulnerable to the infections which precipitate kwashiorkor, since exposure is heavy and they have had little opportunity to build immunity.

The influence of infection on the development of kwashiorkor has been frequently recognized, but the extent to which it is an almost universal factor has only recently been appreciated. Infection reduces net nitrogen retention, due largely to increased nitrogen excretion in the urine, and is a direct precipitating factor in kwashiorkor. It also has an indirect effect, since in areas in which kwashiorkor occurs, the mother often treats the infection by withdrawing what little solid food the child may be getting and administering only thin cereal or starch gruels of negligible protein content. If the infection is enteric, as is very commonly the case, she may even administer a strong purgative to help get rid of the worms she believes to be responsible.

Even though there is no doubt as to the contribution of infection to mortality from protein deficiency, uncertainty remains as to the mechanism whereby protein deficiency increases mortality from infection. Although there may be little effect on resistance to initial infection, the capacity of the body to withstand the effects of infections and infestations and to resist secondary complications is reduced. This is an area in which additional clinical and epidemiological research is badly needed.

A discussion of world-wide protein malnutrition would not be complete without mention of general measures for its prevention. To help prevent deaths from kwashiorkor in endemic areas, professional and lay people alike should be taught to recognize the disease and to know that its true cause is the failure to consume foods which will supply protein of good quality. The application of modern knowledge to the prevention of infectious diseases for which effective immunization is available, and the improvement of environmental sanitation for the control of enteric disease will also reduce kwashiorkor. The primary goal of prevention, however, is not merely the elimination of kwashiorkor, but the restoration of normal

growth and development and good resistance to the complications of infection. This requires that adequate supplies of protein-containing foods be available at reasonable cost for the supplementary and mixed feeding of infants and young children.

Much is being done to increase milk production and conservation in tcchnically underdeveloped areas and to make surplus skim milk available for relief purposes. Nevertheless, for many areas—for agricultural, economic or cultural reasons—there is no reasonable expectation of solving the problem with milk alone.

One important result of the recommendations of the 1955 Princeton conference (52) is that WHO and FAO, joined by UNICEF, have initiated intensive efforts to stimulate the production of protein-rich foods in areas in which kwashiorkor is endemic. The Rockefeller Foundation has also made funds available to the National Research Council to support research in this field. As a consequence progress is being made in many centers in the development of inexpensive and effective combinations of foods of vegetable origin of good protein content and quality. These centers include the University of Capetown in South Africa, the Institute for Scientific Research in the Belgian Congo, the University of Dakar in French West Africa, the Group for Research in Infantile Malnutrition in Uganda, the Health Department of Nigeria, the Central Food Technological Research Institute in Mysore and the Institute of Nutrition in Coonoor—both in India, the Children's Hospital in Mexico and the Institute of Nutrition of Central America and Panama (INCAP) in Guatemala.

In the latter Institute several all-vegetable formulae have been developed with a protein quality comparable to that of milk. These have proved entirely satisfactory even as the sole protein source for the treatment of severe kwashiorkor. The latest of the formulae, INCAP Mixture 9B, contains only corn, sorghum, cottonseed meal, yeast and added synthetic vitamin A, yet provides all of the requirements of a child at weaning, except for calories and vitamin C, for an ingredient cost of 2 or 3 cents a day. On such vegetable mixtures and the increased availability of milk, fish and other animal protein sources, together with intensive educational efforts, rest the hopes for eliminating kwashiorkor and the underlying protein malnutrition which precedes it and which contributes to mortality from infections.

The objective of the many national and international efforts to achieve this goal is to ensure that the ordinary children in the areas in which kwashiorkor is endemic, whose growth and development are now retarded, whose resistance to the consequences of disease is lowered and whose vitality is sapped by protein malnutrition, are converted into healthy youngsters. Only then can they be expected to absorb the knowledge and learn the skills and attitudes required to build a stable, prosperous and peaceful world.

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