

An analysis is presented of the interaction of malnutrition and communicable diseases of childhood in Guatemala. It is shown that nutrition and infection act synergistically. The wider effects of childhood infections are part of a chain reaction which has an important impact on physical and mental growth and development. The need for wide-ranging public health measures is stressed.

MALNUTRITION AND THE COMMON COMMUNICABLE DISEASES OF CHILDHOOD IN RURAL GUATEMALA

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THE world of infectious diseases and malnutrition centers strongly in the developing countries. Physicians of North America and western Europe appreciate that differences in disease behavior exist but tend to view that fact abstractly; or influenced by personal experience, as a concern at least remote and even inconsequential.

Death rates are higher in the developing regions, and infections predominate among leading causes of death. Nutritional surveys show startling differences in intake of calories, protein and of other principal nutrients. What is grossly unappreciated is the result when the two conditions exist together, when malnutrition and infectious disease prevail in high degree in the same general region.

The grandfathers of present-day practitioners would have understood the situation, for three generations ago conditions in the now industrialized countries of the world were much the same as they are today in less privileged areas. An improved environmental sanitation, better medical care, and an expanded economy have contributed to the change.

The interaction of nutrition and infection is far less evident, even insignificant from a community standpoint, and yet in many parts of the world it continues to be of major importance. The combined effect when the two conditions are present in the same individual is more than additive. A synergism exists, with consequences more serious than predictable from either disease occurring alone. Infections precipitate acute nutritional disease in children already malnourished. Infections themselves are rendered more severe by a resistance lessened by malnutrition.¹

The person most affected is the child less than five years old. In most developing countries children are breast-fed well into the second year of life and often during the third year. Weaned from the breast—and of necessity subsisting on a predominantly starchy, adult type of diet—young children proceed in large proportion to varying degrees of malnutrition with an accompanying inhibition of growth and development. Breast milk alone fails to provide sufficient calories and protein beyond six months, and supplements are commonly

inadequate. The severe and fatal calorie-protein deficiency disease, kwashiorkor, often follows with edema, changes in color and texture of hair, pellagroid lesions of the skin, apathy, and anorexia. It usually is precipitated by an acute infectious disease, commonly acute diarrheal disease.²

A usual view of infectious disease, seen in less favored areas of the world, is of exotic conditions such as plague, yellow fever, trypanosomiasis, or onchocerciasis. The present study is of behavior in those regions of the simple diseases that are prevalent wherever one lives: measles, whooping cough, mumps, chickenpox, and rubella—the common communicable diseases of childhood. The aim is to show that they follow the general pattern of enhanced severity, characteristic of infectious disease in developing countries, where host factors are the main determining influence. Host susceptibility is dependent to a large extent on the closely spaced, frequently recurring, ordinary infectious diseases, the diarrheas and the common colds, and on impaired nutritional state.

Clinical case study readily demonstrates a severity of infection beyond the reaction usually seen in a well-ordered environment. Recognition and documentation of the responsible mechanism require long-term prospective field studies, preferably extending from birth to school age. Such studies also are essential to understand the conditions under which an existing malnutrition is precipitated into kwashiorkor by otherwise relatively benign infectious disease. The ultimate effect is interlocking; it is cumulative.

The following examples are from longitudinal studies on synergism of infection and nutrition conducted in rural areas of Guatemala. Village families were visited every two weeks during five years by a resident field worker who recorded all illnesses of children less than

five years old. Reported illnesses were verified by a physician, with the patient revisited each two days until recovery or death. Measurements of height, weight, and skinfold thickness were made every three months, and for a subsample of the population, dietary regimens during a three-day period, twice a year.

Measles

Other than in primitive and highly isolated regions, measles in developing countries is primarily a disease of pre-school children. In rural Guatemala,³ more than half of the cases in a series of epidemics were among children less than three years old. (In industrialized countries the common age is six years, when school begins.) The excessive fatality of measles in developing areas is well established⁴; in Guatemalan epidemics, 6.8 per cent. Several investigations in developing areas have emphasized the capacity of measles to precipitate kwashiorkor in a protein deficient child.^{5,6} A more virulent strain of measles virus is not the answer to the greater severity, because children of middle and upper social class families in regions with high fatality have a wholly ordinary disease. The following case history is from a Guatemalan village epidemic.

Case 238-05: A boy aged two years nine months, the third child of an Indian family, had breast milk as the sole food during the first three months. Weaning started then with the addition of food supplements from the family table. Breast feeding ended in the 22nd month, the child subsisting thereafter on selected foods served by the family, predominantly corn (tortilla), vegetables, and on rare occasions meat broth.

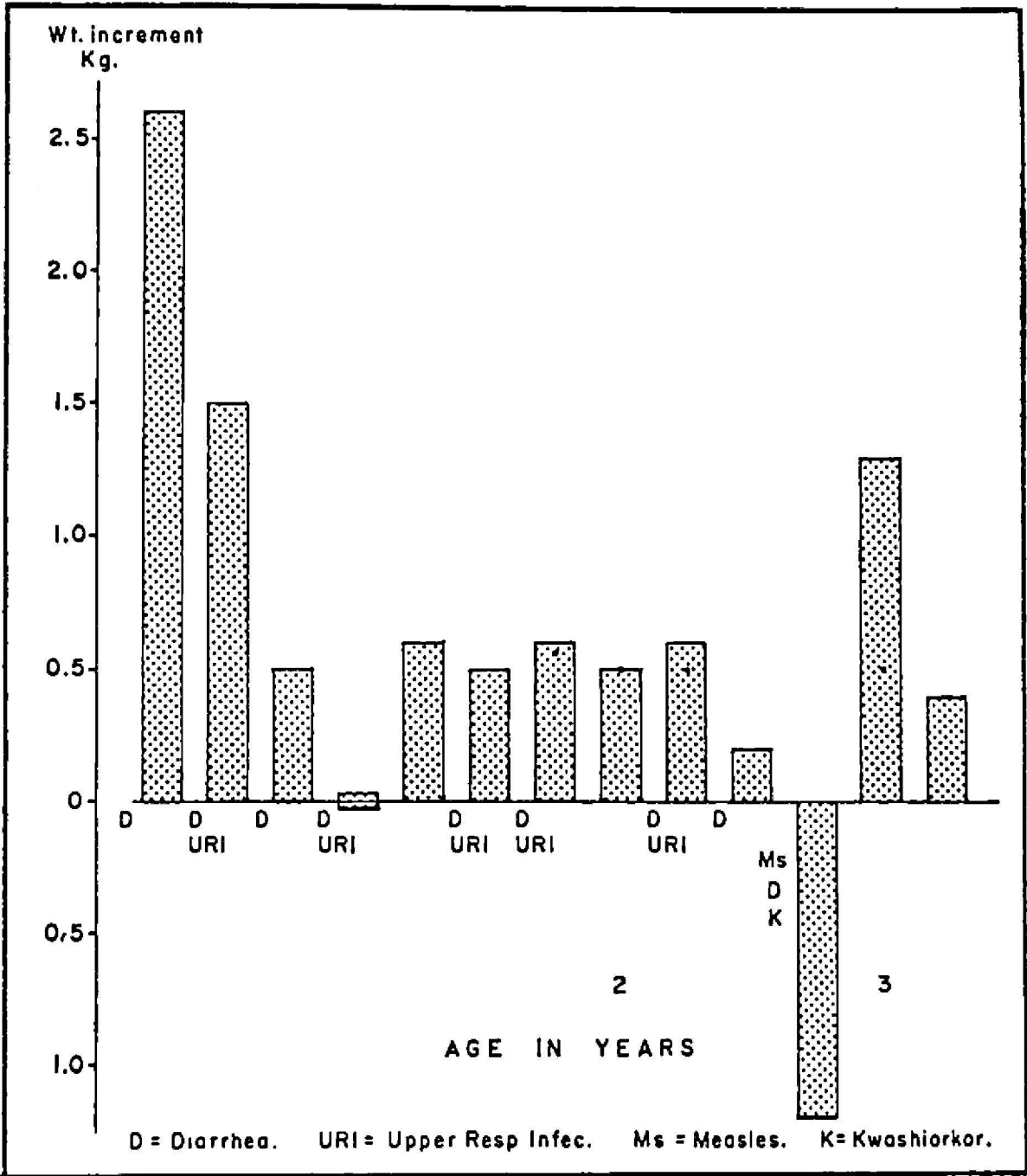
During the first year the child had the usual medical history of infants born in this environment: four attacks of acute

diarrheal disease, totaling 37 days, and 23 days of upper respiratory disease in two episodes; in all 60 days of illness or the equivalent of 17 per cent of his first year, distributed by quarterly periods as shown in Figure 1. The first and fourth quarters of the second year were free of illness, but 26 days of diarrheal disease and 28 of respiratory infection were accumulated during the second and third quarters, again 15 per cent of the second year, and four separate illnesses. The third year had provided a similar experience when measles

developed at 33 months. The disease was associated with a severe diarrhea of nine days' duration, first evident in the prodromal period preceding the rash, and kwashiorkor occurred three weeks later.

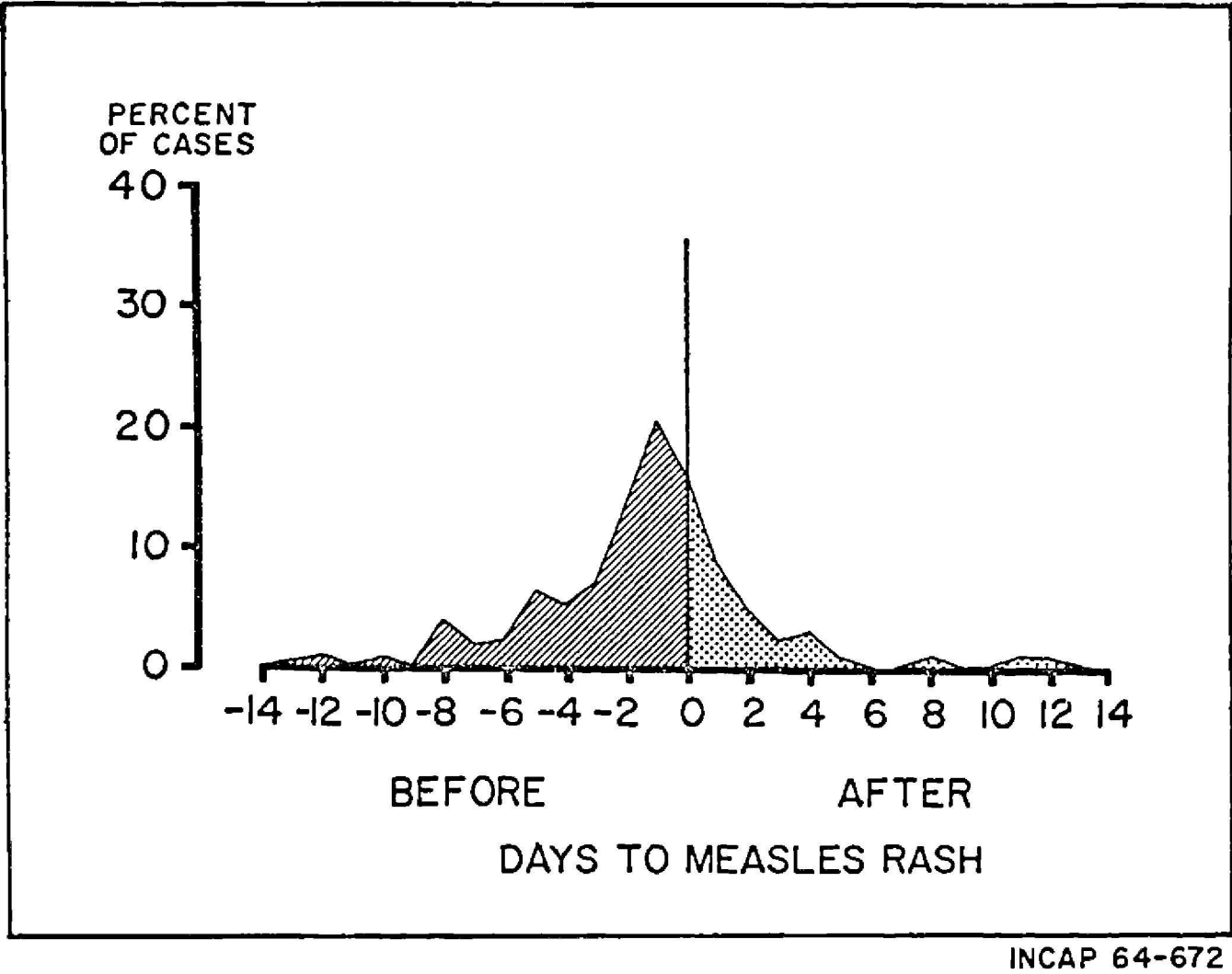
The effect of this succession of illnesses on growth and development and the ultimate nutritional collapse precipitated by measles is shown in Figure 1 according to measurements of weight increase by quarterly periods. Increments of weight, indicative of growth rate, are judged more expressive of the effect of

Figure 1—Quarterly increments of body weight in kilograms, and preceding illnesses of a Guatemalan boy who at 33 months of age contracted measles, a complicating diarrhea and subsequent kwashiorkor.



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Figure 2—Per cent distribution of 127 cases of diarrhea complicating measles in pre-school children, by day of onset before and after measles rash, Santa Cruz Balanya, Guatemala, 1960-1963.



infection than gross weight at a prescribed time. The mean in kilograms for well-nourished children of this region is shown by the dotted line of the graph.

The rate of growth in the first three months was better than the INCAP standard. Subsequently, in the face of ensuing infections, rates of growth decreased progressively until at the end of the first year there was no demonstrable increment, a circumstance which paralleled the occurrence of acute infectious disease. With no illness during the first three months at the second year, a normal growth pattern resumed and, despite minor illnesses, was maintained until the second quarter of the third year. A 12-day attack of diarrheal disease at that time inhibited weight increment by half. Shortly thereafter the patient had measles and a second epi-

sode of diarrhea. The result was an inversion of the growth pattern, a marked loss of weight, and ultimately kwashiorkor. With hospitalization and a high protein diet, the weight rebound was gratifying and in the absence of further immediate illness a normal growth potential resumed.

Measles and a complicating diarrhea were clearly the precipitating causes of kwashiorkor. The real origin was more remote, primarily in the long series of acute infectious illnesses, especially acute diarrheal disease, each with its inhibiting effect on growth and development. Measles decided the issue.

Although diarrhea is not a feature of measles in present-day well-nourished populations of temperate regions, it was a common accompaniment in the Britain of a hundred years ago⁷; in de-

veloping countries of today, diarrhea complicates measles with much frequency. Morley recorded its occurrence in more than a fourth of hospital patients in Nigeria,⁸ and Scrimshaw et al.,⁹ in better than half the cases under field conditions in Guatemala.

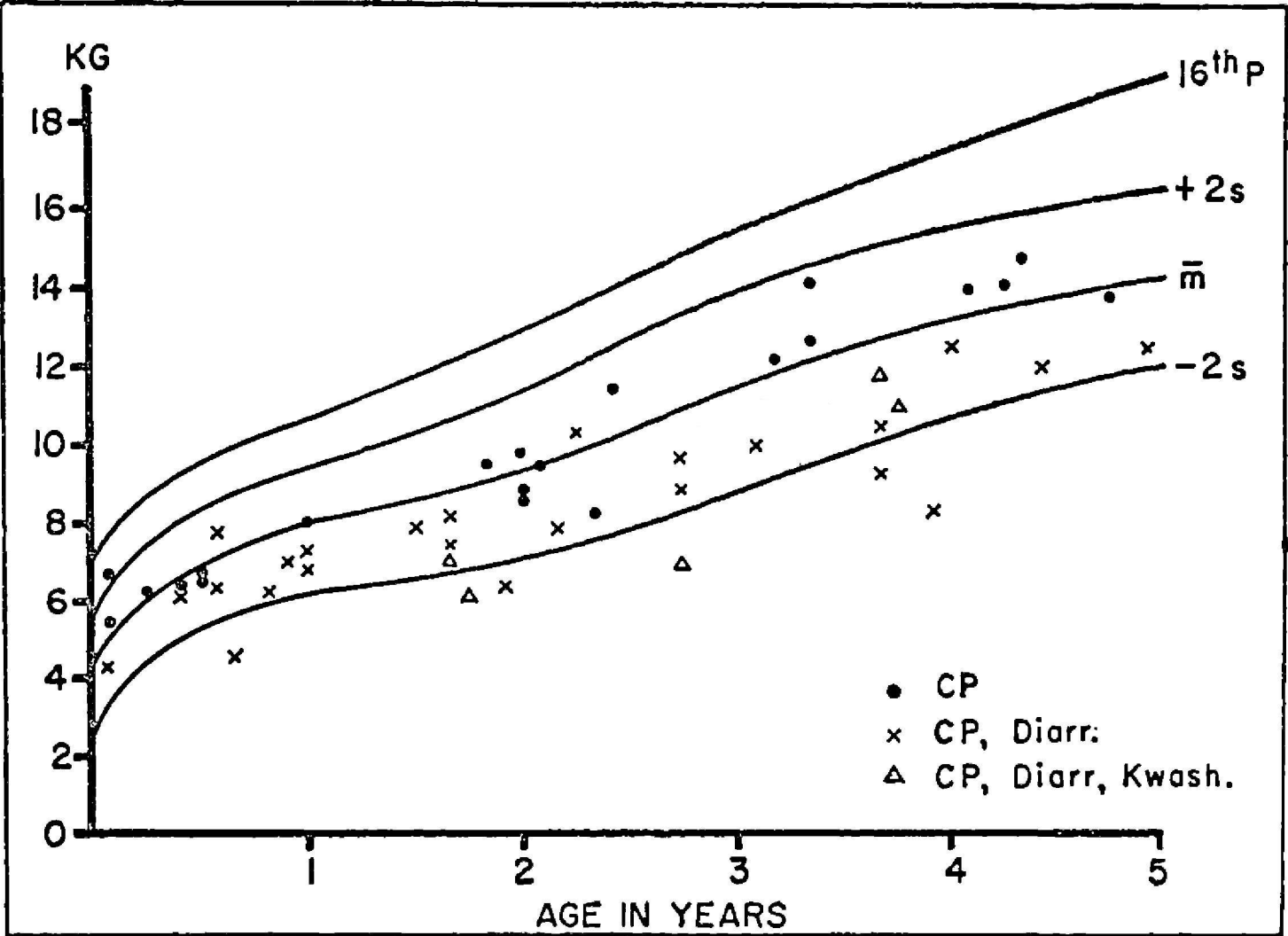
The onset of diarrhea was concentrated in the prodromal stage of measles (Figure 2). Frequency was also a function of degree of malnutrition; diarrhea in patients less than 75 per cent of average normal weight for age was three times that for patients of average normal weight. Vega, et al.,¹⁰ also in Guatemala, found that the frequency and severity of bacterial complications in children (less than five years of age) with measles increased in proportion to intensity of malnutrition.

Chickenpox

Diarrheal disease as a complication of chickenpox was almost as frequent as in measles in 58 per cent of 50 preschool children in a Guatemalan epidemic. It was not observed in older children with chickenpox.¹¹ Onset likewise was in the prodromal stage of the disease. Incidence among well-nourished preschool patients was 12 per cent, among the malnourished 75 per cent, and it occurred in all of six patients with third degree malnutrition. Kwashiorkor involved five of 50 preschool children, primarily those most severely malnourished, and without exception after an accompanying diarrhea (Figure 3). A case of the outbreak is presented.

Case No. 3-63-G09: A boy aged three

Figure 3—Chickenpox in 50 patients less than 5 years old, by nutritional status at time of onset, as judged by body weight.



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Figure 4—Increments of body weight in kilograms, by three-month intervals, and preceding diseases of a boy who contracted chickenpox, diarrheal disease, and kwashiorkor.



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years and five months, born in the village where the epidemic occurred and the 12th child of a family, was wholly breast-fed for five months. The diet was then supplemented with gruels, usually of corn but sometimes of rice, and with occasional vegetables and other food from the family table. Weaning at 24 months was to the ordinary adult diet of corn tortillas, beans, and other vegetables.

In the first year of life, the child had one episode of severe diarrheal disease lasting four days and two attacks of acute upper respiratory infection totaling 27 days. He had two long-continued bouts of impetigo for a sum of 125 days. The second year was still more eventful, with no less than six attacks of diarrheal disease, two of them severe; two respiratory illnesses, one long-continued;

and a further indefinite febrile illness of 30 days to give a total of 204 days sickness within the 365. The effect on gain in weight is seen in Figure 4. The third year was more favorable with four diarrheal attacks, only one of them severe, and a single noteworthy respiratory illness. Total days of sickness were 70. The fourth year was initiated by two diarrheal attacks of short duration, and three months later the patient contracted chickenpox. Weight at the time was 11 kg. Acute diarrhea began three days before the eruption. Appetite was lost as the rash appeared and the mother gave no food. As the rash resolved, a slight edema of the feet appeared at about the sixth day, and within the next 15 days had extended to the legs and face. Serious loss of weight developed and the general clinical condi-

tion deteriorated. About three weeks after onset of chickenpox, the child was admitted to hospital with kwashiorkor, and placed on a high protein diet. Clinical improvement was prompt, a highly satisfying gain in weight followed, and recovery was eventually complete.

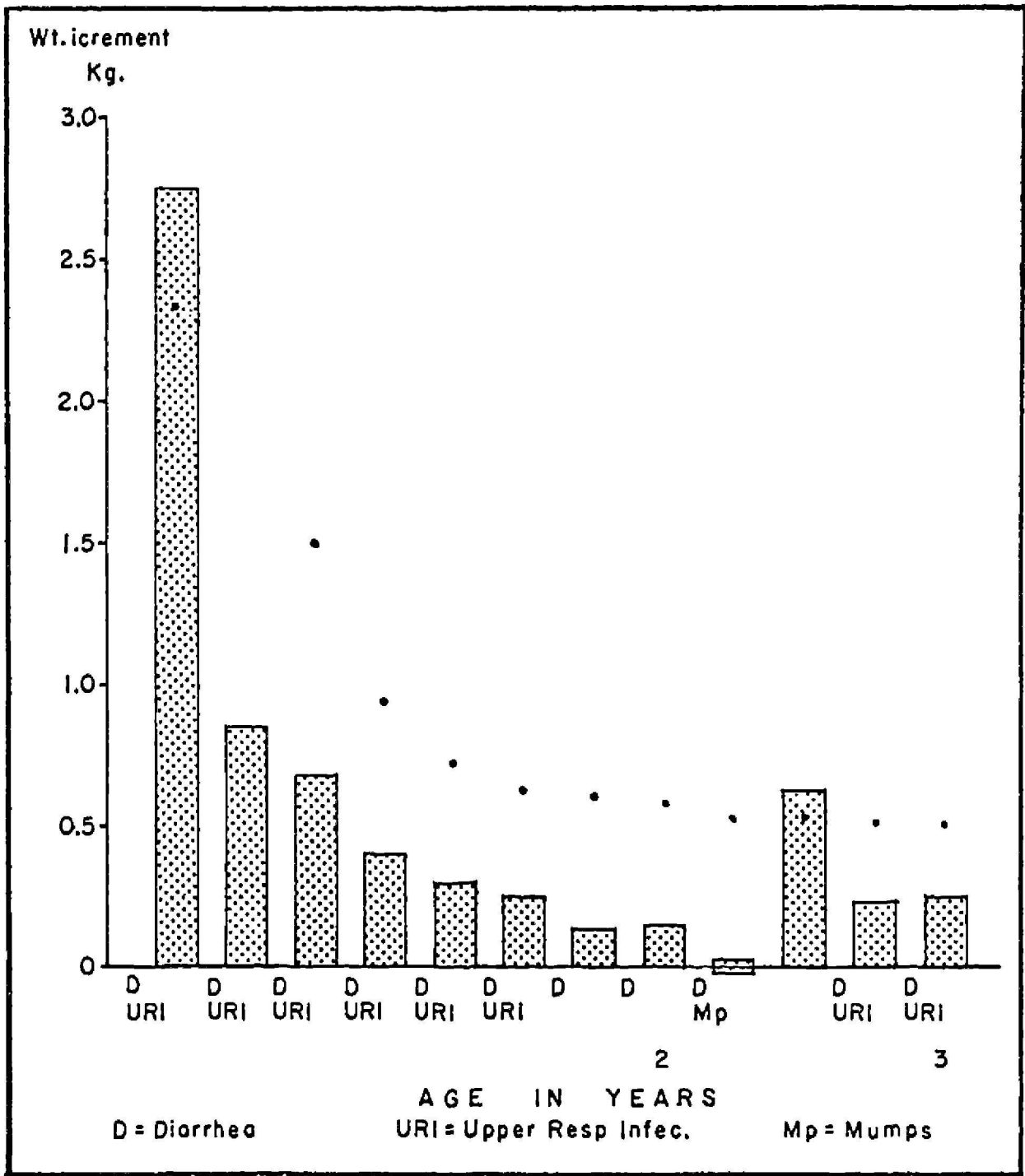
Mumps

The clinical potentialities of mumps, as with others of the common communicable diseases of childhood, are not

to be determined by observations restricted to the brief period when the disease is active. They rest in knowledge of host nutrition, presumed general resistance when the disease is acquired, and in continued observation for late effects. Figure 5 shows weight increments by three-month intervals for a village child of the Guatemalan highlands.

Case 175-08: A boy aged two years three months, the fifth child of the fam-

Figure 5—Weight increments in kilograms and previous diseases of a child developing mumps at 27 months of age.



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ily. Breast milk was the only food during the first five months, with progressively increased supplements thereafter, primarily the starchy foods characteristic of the region. Weaning was completed in the 34th month. The child then subsisted on the adult diet of the country.

In infancy the child had eight illnesses totaling 160 days or almost half of that year. The second year was much the same, with 107 days of illness in six episodes. Mumps developed in the first quarter of the third year, immediately preceded by acute diarrheal disease of seven days' duration. No illness occurred in the next quarter year, but by the end of the third year the child had had in all 315 days of illness, a time close to a third of his lifetime.

In the initial three months the growth pattern was much better than demanded by INCAP standards. Gain in weight thereafter slowed progressively in accord with days of illness due to acute infections. Mumps in the 27th month brought weight increase to a dead stop. With no recorded illness in the next quarter the child had a weight rebound exceeding the expected increment. With returning infections the previous growth pattern resumed, namely a failure to gain satisfactorily. Although mumps did not lead to kwashiorkor, the induced stress was sufficient to halt all weight gain. With the brake removed the rebound was wholly satisfying, although short-lived, for recurring infections resulted in growth deficits as in preceding periods.

Whooping Cough

For more than 50 years, deaths from whooping cough have declined consistently in industrialized areas, particularly in the United States, Canada, and Great Britain. In developing countries whooping cough, along with measles, remains a killing disease. The

remarkable decline in mortality in technically advanced countries came long before vaccines were in general use. Although further aided later by immunization programs, the main influence was seemingly a steadily improved host resistance brought about by an advancing medical practice, a better nutrition and a more efficient environmental sanitation, with the result reflected mainly in fewer complications. The importance of these associated illnesses is illustrated by the following case report.

Case 175-08: A boy aged 24 months, the fourth child of his family, was breast-fed until the fifth month when the customary supplements were added. Weaning was completed in the 27th month. During weaning a nutritional survey of the supplement given at seven months of age showed 28 per cent of protein and 28 per cent of the calories recommended by INCAP dietary standards. At 21 months food intake had improved only fractionally. After weaning, at 34 months, the survey reported a daily consumption of 38 per cent of recommended protein and 74 per cent of calories, protein intake thus being at a decidedly low level, and caloric intake from a predominantly carbohydrate diet.

The illnesses of the child matched the deficiencies in diet. During infancy, days of illness were 170 in six episodes distributed as in Figure 6. The second year had 157 days' illness and six attacks, altogether the equivalent of 10.9 of the 24 months of life. Whooping cough appeared in the 24th month. With the attendant complications it lasted 90 days, for the patient had repeated attacks of diarrhea, experienced a pneumonia, and ended up with kwashiorkor.

During the first quarterly period the weight increment in kilograms was greater than INCAP standards. The boy had a stormy time in the second and third quarters, with repeated severe diar-

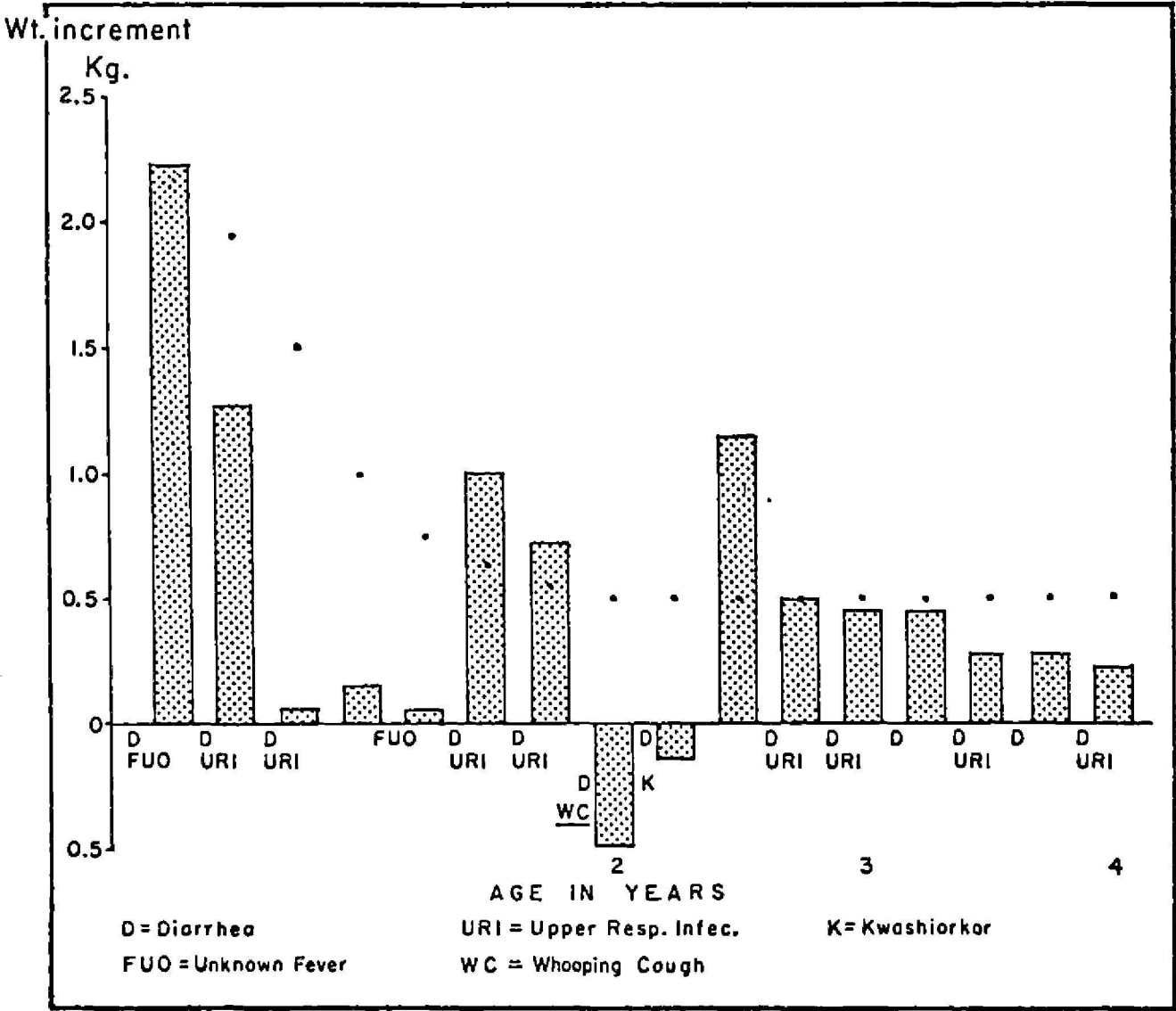
rheal attacks and several respiratory infections. Weight defect was prolonged into the fourth and fifth quarters. The sixth and seventh quarterly periods showed some progress despite a return of acute infections. Weight at the end of two years was eight kilograms, in contrast to the standard of 14 kilograms, a deficit of 43 per cent, or the average weight for a normal nine-month-old child.

Whooping cough and its complications had a profound effect. The child not only failed to gain weight but experienced weight loss during six months, with the end result of kwashiorkor. With hospital care and an adequate diet the child eventually made up some of

the lost ground, as the record of the tenth quarter shows. However, the growth pattern reverted to its previous characteristics when he returned home, with a weight increment about half of the expected values.

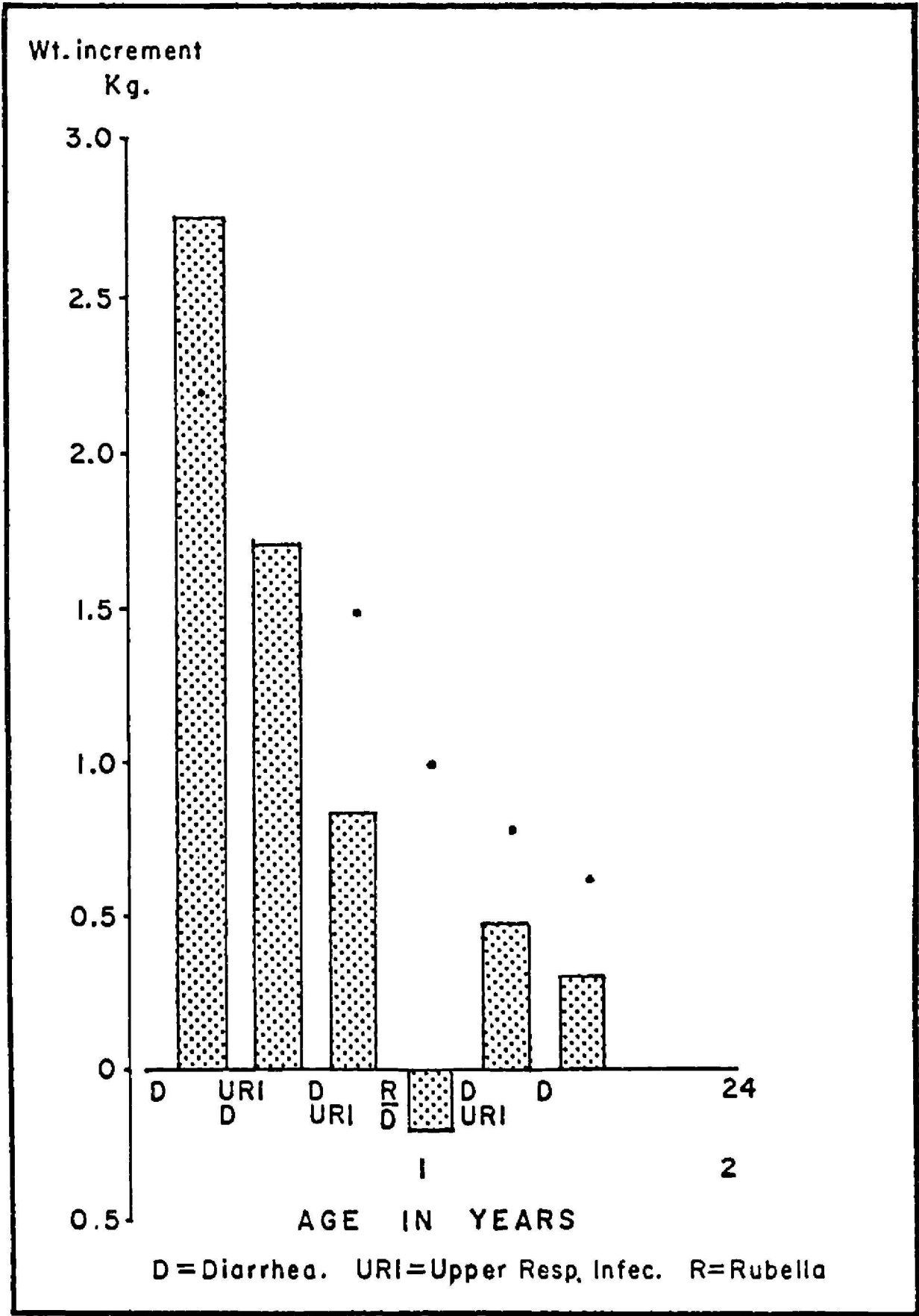
The potential of kwashiorkor had existed in this case for many months as the weight curve demonstrates; malnutrition had developed in high degree. Whooping cough in severe form turned the scale. During hospitalization and shortly thereafter growth rate was good, but under the impact of an environment characterized by a deficient diet and repeated infections, the child soon returned to an existence of weight deficits. No better example is to be had of

Figure 6—Weight increments in kilograms and previous infectious diseases of a patient who at 24 months of age had a sequence of whooping cough, diarrheal disease, and kwashiorkor.



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Figure 7—Weight increments and preceding illnesses of an 11-month-old patient with rubella.



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the ecologic interplay of man, environment, and health.¹²

Rubella

After more than a century of relative obscurity among the common communi-

cable diseases of childhood, rubella attained a new respect through a demonstrated relation to congenital anomalies. Actual numbers of cases nevertheless remain highly indefinite because the usual mildness of the disease results in grossly

incomplete reporting. The next case shows that rubella in developing areas has further significance than a relation to developmental abnormalities; that in a nutritionally depleted host, complications ensue and growth processes change within a magnitude comparable to that of others of this group of diseases.

Case 079-08: A boy aged 11 months, still breast-fed, had 20 days of diarrheal disease during the first three months, in the second three months 22 days of diarrhea in two episodes, and 18 days of conjunctivitis. The subsequent history was of much the same order so that by the time rubella appeared as a part of a small village epidemic, the child at 11 months had had six bouts of acute infectious disease and 50 days of illness. The clinical indisposition accompanying rubella lasted for nine days, largely by reason of diarrheal disease of five days' duration, first manifest as the rash of rubella appeared. Weight increment, Figure 7, always deficient except for the first three months, was then negative, with a loss of 0.2 kilograms. Weight gain resumed in the second year, but in the presence of a similar chain of infections has never reached expected values.

Summary and Conclusions

The effect of the common communicable diseases of childhood—measles, whooping cough, mumps, rubella, and chickenpox—on nutritional status of the patient scarcely rests in the episode itself, but rather in the extent to which the attack is a part of a sequence of repeated, often nonspecific infections, most often the ordinary infectious syndromes of intestinal and upper respiratory tracts. The effect is additive and cumulative. Conversely, these usually benign diseases derive their enhanced fatality, greater frequency of complications and exaggerated clinical course through attack on a less resistant host, to which nutritional deficiency contributes importantly. To know the full potentiality

of these diseases in developing countries, information is necessary on preceding numbers and duration of these other infections, so largely the diarrheas and the common colds. That is to be had by long-term prospective field studies of childhood populations.

That all five diseases behave in exaggerated fashion supports the existence of a common mechanism: nutrition and infection acting synergistically, malnutrition decreasing host resistance to infectious disease, and infectious disease demanding of a nutritionally depleted host greater requirements of basic nutrients.

The ultimate effect of childhood infection suggestively extends beyond the immediate event. A substantial action on physical growth and development has been demonstrated. Increasing evidence suggests a concomitant effect on mental growth and development, with both tracing back to these repeated insults in early childhood. The remedy is in public health measures directed toward both nutrition and infection, not individually but rather as elements within a single complex.

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This paper was presented before the Food and Nutrition Section of the American Public Health Association at the Ninety-Fourth Annual Meeting in San Francisco, Calif., November 2, 1966.

These studies were supported by the Pan American Health Organization, the World Health Organization, and Grants SR01-00492 and AI05405-05 from the National Institutes of Health (INCAP Publication I-389).

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