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Malnutrition and Infection in a Rural Village of Guatemala[1]

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In societies of low socio-economic level, man is affected by the interacting forces of the binomial complex malnutrition-infectious disease. These forces are rarely found associated in the highly industrialized and affluent societies. However, a recent survey in the United States showed that malnutrition is common in special risk groups where infectious disease is known to be frequent. On the other hand, the complex malnutrition-infection is ever present in developing nations where many particular factors favor its appearance.

That infectious disease aggravates malnutrition, and malnutrition accentuates the outcome of infectious disease is a readily accepted concept [24]. It would appear to many that the study of the problem is of mere academic importance. Nevertheless, many facets of the complex interrelations between the host nutrition and invading agents deserve exploration, because such knowledge would be of importance in establishing priorities in the planning and execution of health measures. Research in this field has significance for developing nations, as it shows the consequences of the interaction of malnutrition and infection to health planners. This is badly needed since, due to transplantation of technology from industrial to preindustrial societies, and also as a result of the 'imitation factor', the nutrition approach in less developed nations often does not consider the role of infectious processes and other important environmental variables.

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The present work summarizes observations of a long-term study in a rural highland village of low socio-economic level. Investigations were initiated in 1963 and continued until December 1971, with an emphasis on data collection on dietary intake, morbidity, infection, and growth; no significant interventions were effected in the village in that period [13, 15, 16].

Antenatal Malnutrition and Infection

It has become evident that the interaction of malnutrition and infection begins *in utero*. Owing to the low income and educational level, nutrition and hygiene are deficient. Table I shows the mean daily intakes of pregnant women from the village, in comparison with the recommended allowances [14]. The dietary protein is derived mainly from corn. Vitamin A and riboflavin are also deficient. Iron appears to be adequate, but it is not readily available since it is derived principally from pulses [14]. Despite the controversy regarding the role of maternal nutrition in fetal growth [22], there is good evidence to believe that an improvement of the nutrition during pregnancy results in better fetal growth [6].

Morbidity rates are expectedly high during pregnancy. Data corresponding to virtually all pregnant women of the village, observed during an 8-year period, reveal that respiratory infection and diarrhea are common. Even serious diseases such as bronchopneumonia and dysentery occurred

Table I. Mean daily intake of 33 pregnant women; Santa María Cauqué, Guatemala, 1967

	Trimester			Recommendation
	first	second	third	
	n = 6	n = 9	n = 18	
Calories	1755	2218	2082	2200
Total protein, g[1]	56	65	64	75
Animal protein, g	8	7	7	
Vitamin A, mg	0.9	0.8	0.9	1.6
Riboflavin, mg	0.6	0.7	0.7	1.2
Iron, mg[2]	22	23	21	18

1 80% or more derived from corn.
2 Mainly from pulses.

Table II. Incidence of infectious diseases[1] during pregnancy; Santa María Cauqué, Guatemala, 1964–1971

Trimester of pregnancy	Number of women observed	Urinary tract infection	Diarrhea and dysentery	Respiratory infection		Infectious hepatitis
				superior	inferior	
I	360	5 (1)[2]	22 (6)	65 (18)	7 (2)	0
II	358	7 (2)	20 (6)	85 (24)	14 (4)	1
III	364	5 (1)	31 (9)	109 (30)	13 (4)	1
Total	1082	17 (2)	73 (7)	259 (24)	34 (3)	2
Incidence/100 pregnancies	361	4.9	20.2	71.7	9.4	0.5

1 Diagnosed on clinical grounds.
2 Number of episodes (percentage in parentheses).

with a certain regularity. The commonest were upper respiratory infection, diarrheal disease, and urinary tract infection (table II). Furthermore, asymptomatic infection was exceedingly prevalent; surveys revealed that almost all women are infected with intestinal parasites, while many carry enteroviruses, enteric pathogenic bacteria, bacterial and viral respiratory agents, and vaginal parasites [12, 16].

The significance of increased infection in the mother in terms of fetal damage has not been clearly established. In the present study, a good proportion of the village neonates were born with high levels of immunoglobulin M (IgM), suggesting frequent intrauterine infection; the finding was corroborated in other highland and lowland villages of Guatemala and Peru [7]. A recent inquiry in 4 lowland rural areas of Guatemala, where the child's blood was examined within 3 days of birth, showed that 10% of infants were born with high IgM, a definite evidence of fetal antigenic stimulation (unpublished). Furthermore, preliminary findings from the village of Santa María Cauqué revealed an increased incidence of intrauterine infection [12].

If intrauterine infection is shown to be more frequent in developing nations than in industrialized countries, its significance as a cause of fetal retardation, embryopathies, and other antenatal and postnatal damage will have to be evaluated. This question should receive immediate attention in view of the already known relationship between antenatal infection and fetal growth, infant mortality and postnatal development, both physical and mental [5, 19].

Fetal Growth

Although the significance of poor diets and high rates of infection in the mother cannot be easily demonstrated under field conditions, it can be safely assumed that both factors are causally related to fetal growth retardation and other types of damage. Theoretically, a poor nutritional status and/or inadequate food supply during pregnancy limits fetal nutrition and growth. Furthermore, infections in the mother will have a wasting effect, magnified when malnutrition is present. Still more, the greater the burden of infection during pregnancy, the greater the opportunities for fetal infection, and this may have serious consequences for the fetus and in postnatal life.

Table III summarizes birth weights of almost all infants born in a 7-year period. Approximately 10% of all newborns were premature by weight and gestational age; an additional 30% were small for date (less than 2,500 g at 37 weeks or more) [8, 27]. The finding that 40% of all newborns could be labelled as premature according to WHO [28] was not anticipated, even

Table III. Fetal growth in 367 live born infants; Santa María Cauqué, 1964–1971

Gestation time, weeks	Classification	Number and relative percentage	Birth weight, g (mean ± SD)
37	Premature	33 (9.0)	1,713 ±368
37	Retarded	116 (31.6)	2,272 ±248
37	Not retarded	218 (59.4)	2,789 ±218

Table IV. Infant mortality by birth weight; Santa María Cauqué, Guatemala, 1964–1972

Weight, g	Number of children	Mortality		Total
		0–28 days	29 days–11 months	
< 2,100	33	6 (18.2)[1]	10 (30.3)[1]	16 (48.5)[1]
2,100–2,499	95	3 (3.1)	5 (5.3)	8 (8.4)
2,500–2,899	137	2 (1.4)	8 (5.8)	10 (7.3)
2,900 +	53	0	0	0
Total	318	11 (34.5)[2]	23 (72.3)[2]	34 (106.9)[2]

1 Number (percent mortality in parentheses).
2 Number and mortality per 1000 live births.

under the poor environmental conditions of the area. But it is expected that this is a more general phenomenon throughout rural areas of the world, with varying rates according to degree of industrialization and welfare of the population. The relationship of birth weight in terms of survival is shown in table IV, revealing an indirect correlation with infant mortality [8, 27]: half of the infants born weighing less than 2,100 g died in the first year of life, while all those weighing 2,900 or more survived the first year.

It is already known that small babies have a poor capacity to thrive, even when environmental conditions are favorable. Notwithstanding that the delivery of medical and nutritional care appropriate and directed to the premature is a utopia in rural areas, the health approach should be oriented toward strengthening maternal and child health programs emphasizing improvement of the nutritional status and prevention and treatment of infectious disease, particularly during pregnancy.

Post-Natal Malnutrition and Infection

It has been noted already that many village babies are born with a deficient weight, and that these babies have a lower survival capacity. However, most infants show an adequate growth rate in the first 3 months of life, regardless of the magnitude of the birth weight deficit. This is likely due to the benefits of maternal milk which, although partially deficient in some nutrients [2], is available in enough quantity for that age group. In addition, there are efficient defence mechanisms operating early in life, namely, passive serum antibodies obtained *in utero*, secretory IgA and lysozyme of breast milk, and the bifidobacterial flora, all of which protect against infection with many common pathogens [17]. Viral, bacterial, and parasitic agents do enter the alimentary tract; many of them are repelled or, if they are established, they become asymptomatic. However, these defence mechanisms are not infallible, and infectious diseases may develop, causing much harm and often death, particularly in infants with low weight.

Weaning begins between 2 and 6 months, when maternal milk is no longer sufficient to meet the nutritional demands of the growing baby, a period coinciding with decay of passive immunity and changes in intestinal flora stimulated by supplementary feeding [16]. The consequences of these events in the child's life are two-fold: on the one hand, there is a progressive deterioration of the nutritional status, since supplementary feeding is far from adequate; secondly, there is a progressive increase of infection and

infectious diseases [16]; malnutrition will become evident by a flattening of the growth curve. Infection will contribute to the deterioration of the nutritional status, an effect that will be evident also in the weight curve [12]. It is thought that both factors act in conjunction and have a synergistic effect [24].

The weaning process consists of the introduction of fluids and gruels, and much later, by the end of the first year of life, of solids [14]. The diet is

Table V. Average daily intake of 45 children studied from birth to age 3 years; Santa María Cauqué, 1964–1970

Age, months	Number	Calories	Total protein[1], g	Vitamin A activity mg
12	45; breast-fed	225	4.6	0.036
24	33; breast-fed	520	13.6	0.125
	12, weaned	614	17.2	0.164
36	11; breast-fed	830	23.7	0.141
	31; weaned	927	25.3	0.272
Recommended	1st	1,100	20	
allowance	2nd	1,250	25	0.600
	3rd	1,400	27	

1 80% or more from corn.

Table VI. Shigella excretion from birth to 3 years of age; Santa María Cauqué, Guatemala, 1964–1967

Age, months	Prevalence, %
0– 2	1.1
3– 5	0.4
6– 8	0.4
9–11	3.2
12–14	8.3
15–17	8.7
18–20	11.1
21–23	16.2
24–26	19.0
27–29	19.6
30–32	21.3
33–35	16.6

based on corn and other vegetables; consumption of meat and milk is negligible; the diet is significantly deficient in calories, proteins, vitamins, and iron (table V). Deficiency is aggravated because most of the protein is derived from corn and iron is of vegetable origin. The diet is deficient throughout the first 2 years of life, and there is relative improvement at the end of the third year and, later on, when the child procures more food by himself [12, 16].

By then, the intestinal mucosa has been damaged by pathogenic infectious agents, particularly those that invade the mucosa, i. e., *Shigella* [9]. Infection increases progressively with age, attaining high incidence and prevalence rates by the end of the first year and particularly during the second and third years. The situation with *Shigella* (table VI), illustrates the point. This organism replicates in the epithelial cell and the *lamina propria*, resulting in associated symptomatology in more than two thirds of the cases. A similar picture can be depicted for other enteric infections, particularly viruses and protozoa [11, 12].

Effect of Infectious Disease on Growth

The high frequency of infection and infectious disease is demonstrated by the history of a village child, considered to be a not unusual case (fig. 1). The child was born with a weight significantly above the village average and showed an adequate growth rate up to the age of 6–8 months, when the weight curve deviated from the desired standard. Cellulitis and upper respiratory infection occurred during a period characterized by a negative weight increment. Weight losses were observed in association with bouts of diarrhea, complicated measles, and bronchopneumonia. During a whole year, the weight of the child was faltering. Eventually, by 18 months, the child grew again but the deficit in weight was already considerable and no tendency to catch up with the standard was observed. Even at 29 months of age, more infectious diseases were noted, followed by significant weight loss and further nutritional deterioration [16]. In addition to disease episodes, many infections go unrecognized, as illustrated in figure 1, for enteric infection with viruses, pathogenic bacteria, and parasites. The overwhelming infection just noted has a counterpart in the upper respiratory tract and the skin.

Diarrhea, bronchopneumonia, cellulitis, and the common communicable diseases of childhood are often associated with weight loss. Figure 2

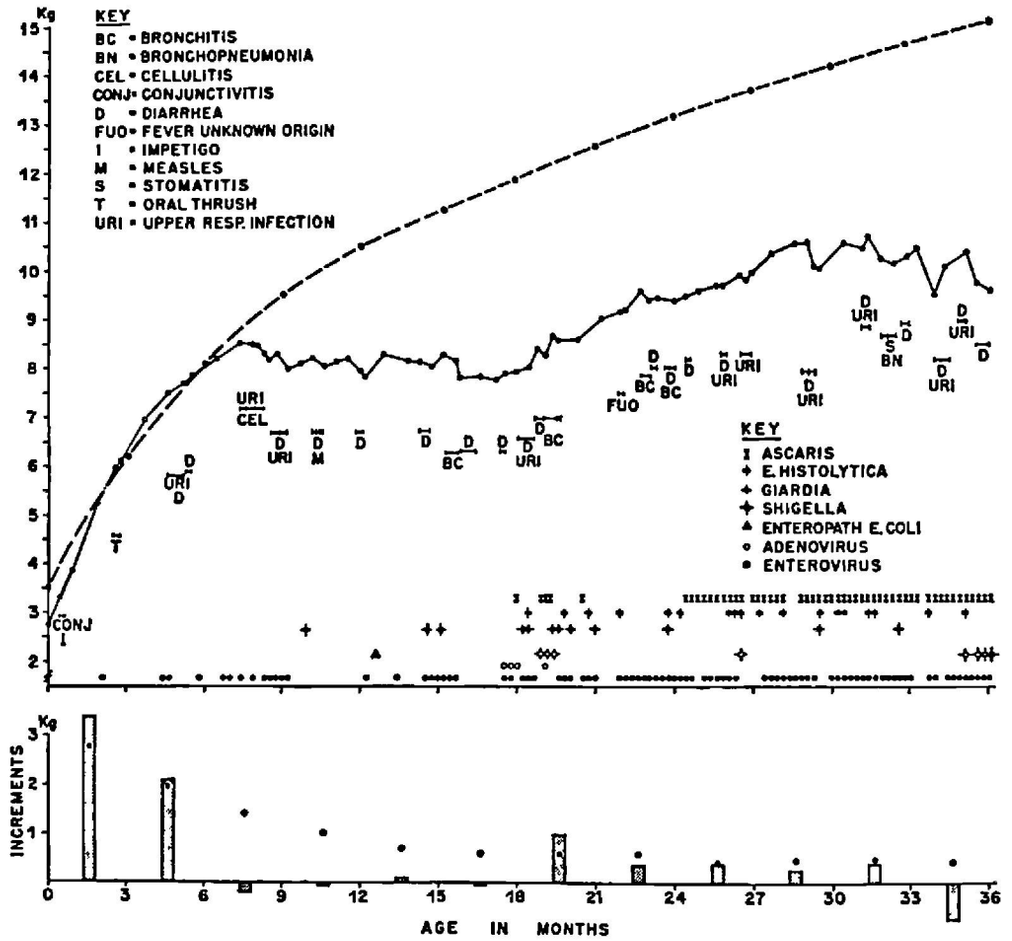

Fig. 1. Weight, infections, and infectious diseases in male child No. 12. *Top:* solid line represents weight of child; broken line is median of the standard. Length of each horizontal line indicates duration of infectious disease. Each mark shows a weak positive for the particular infectious agent. *Bottom:* observed weight increments (vertical bars) and expected median increments of the standard.

shows a child with marked weight loss associated with measles complicated by diarrhea and bronchopneumonia. Weight loss started at 9 months of age, and recovery was not observed until the 15th month. In the ensuing interval, other infectious diseases appeared. Comparison of this child's curve with the village average is an indication that most children have similar histories [12].

An extreme case is that illustrated in figure 3 [7], which shows a girl born with a birth weight within the average for the village and who exhibited an adequate growth rate in the first 9 weeks of life. Subsequently, the child had upper respiratory infection and bronchitis with diminishing breast-feeding, when meningitis to *Diplococcus pneumoniae* was diagnosed. Despite the consequences of this disease and the appearance of diarrhea and varicella immediately thereafter, there was a significant growth spurt and the child reached the average weight for the village at 40 weeks of age. Interference with proper feeding was the main reason for deterioration of weight from

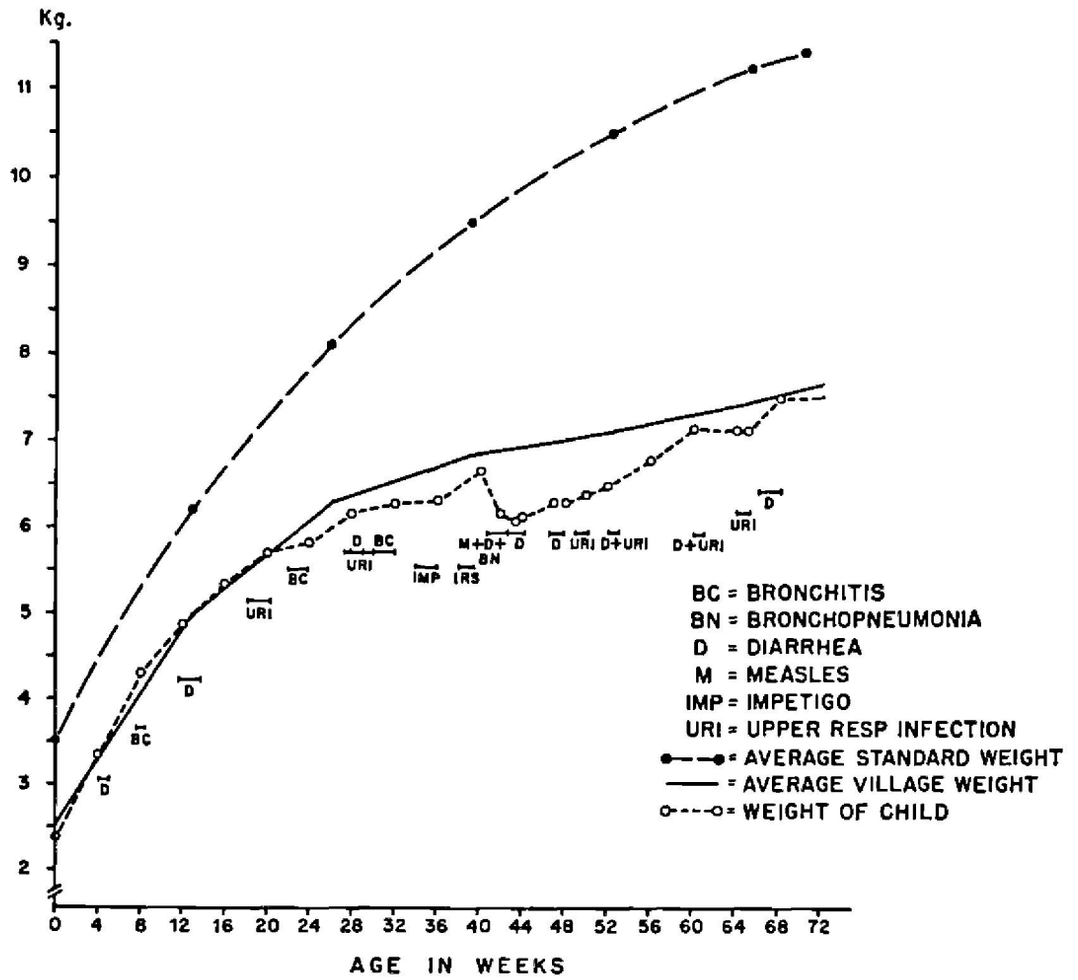

Fig. 2. Weight curve and infectious diseases in male child No. 84. Dotted line represents weight of child; solid line is average village weight curve; upper interrupted line is standard for male children.

there on, and, at 1 year of age, there began a sequence of recurrent infectious diseases, ending in death several months later. An evaluation at 1 year of age indicated a psychomotor development (Gesell) of 5 months, although the clinical evaluation did not show any sequela indicative of an infectious etiology of the psychomotor retardation [11].

The impact of infectious disease on weight becomes evident in a series of cases of whooping cough in which the time needed to recover the weight loss was evaluated (table VII). Whooping cough is a severe disease characterized by a high mortality and significant weight loss. Most children took more than 9 weeks to regain the loss, and 25% took more than 6 months.

It should be pointed out that weight abnormality becomes more serious if the span of weight stagnation occurs during a significant period of growth, namely, at an early age.

The possible effect on growth of subtle or asymptomatic infections has been suggested, since every replicating agent theoretically wastes a portion

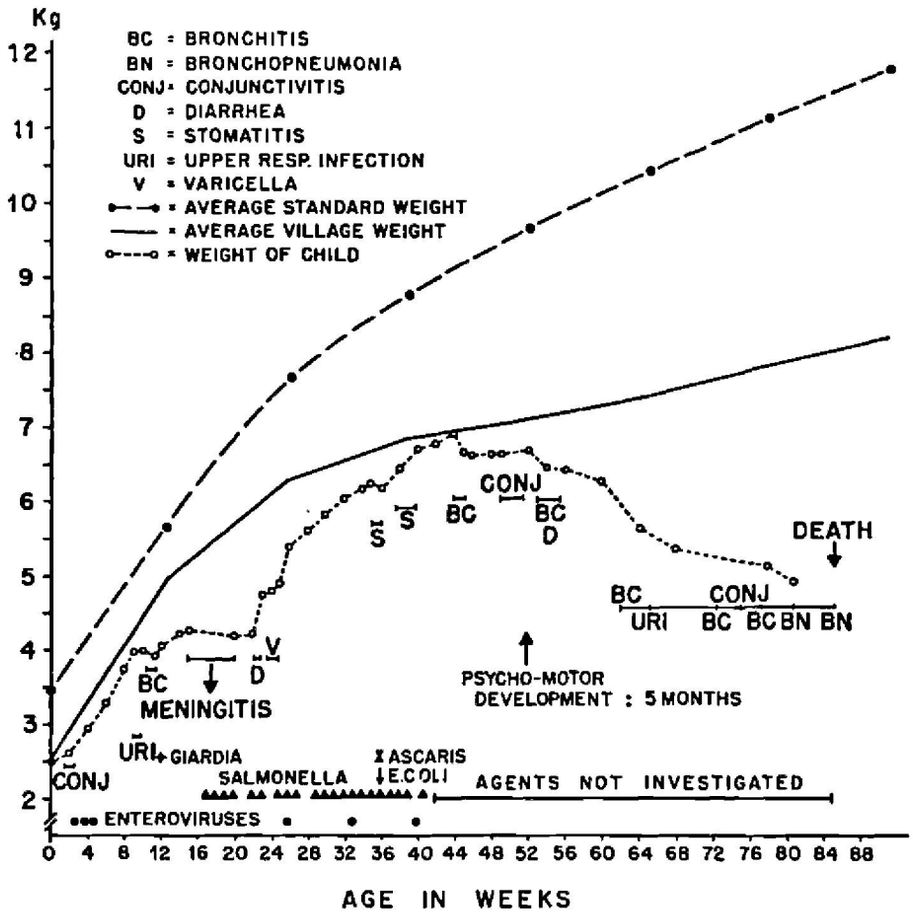

Fig. 3. Weight curve and infectious diseases in female child No. 19. Bottom two lines as in figure 2; upper interrupted line is standard for female children.

Table VII. Time needed to recover from weight loss after whooping cough; Santa María Cauqué, Guatemala, March–August, 1968

Time, weeks	Number of cases
0– 4	6 (14)[1]
5– 8	8 (18)
9–12	8 (18)
13–16	4 (9)
17–24	7 (16)
25 +	11 (25)
Total	44 (100)

1 Values in parentheses are percentages

Table VIII. Enteric viruses in a cohort of 45 infants; Santa María Cauqué, Guatemala, 1964–1966

	First 3 months of life			First 6 months of life		
Group	A	B	C	A	B	C
Number of infants	15	15	15	15	15	15
Total viral isolates	6	31	71	50	93	146
Infection rate/child/period	0.40	2.27	4.73	3.33	6.20	9.73

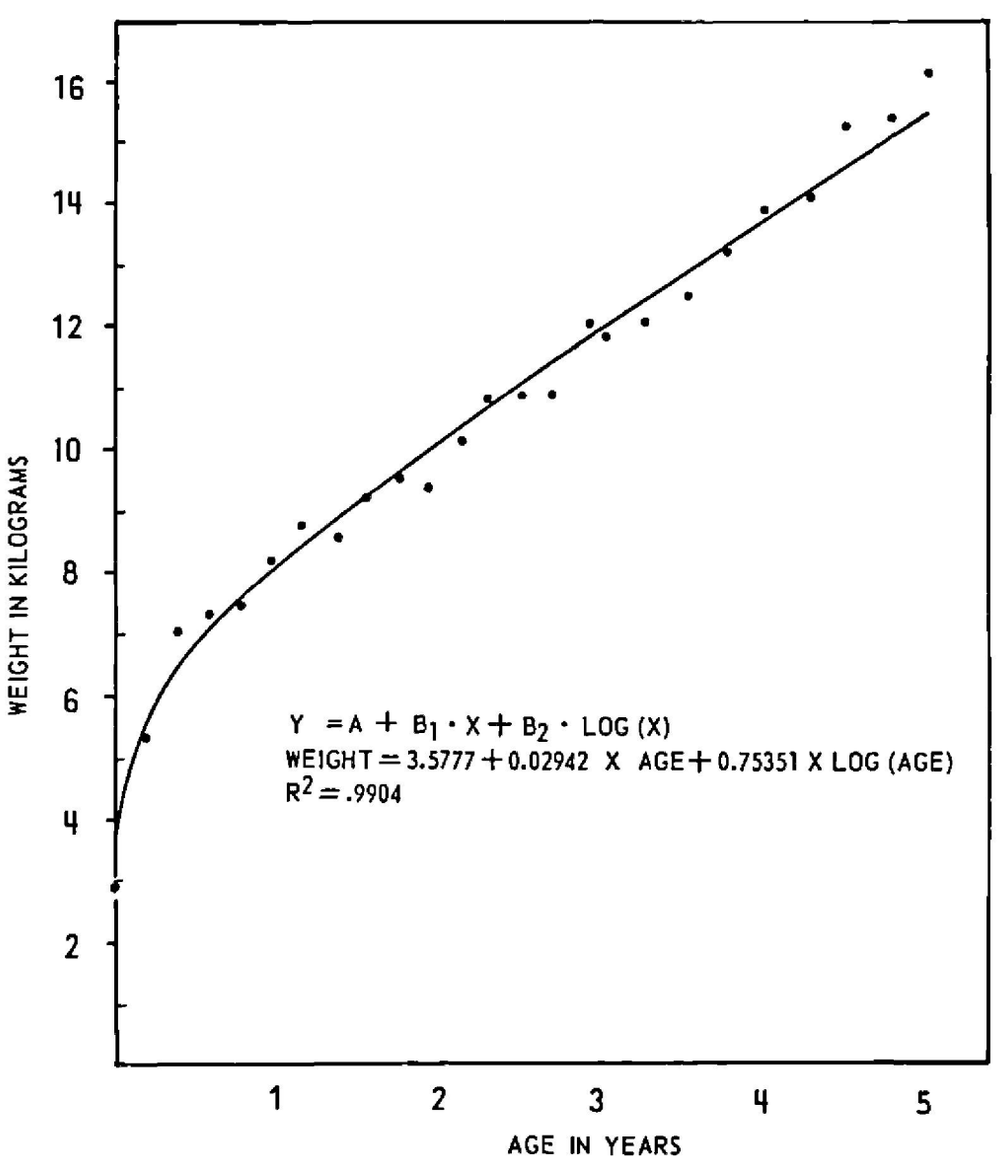

Fig. 4. Weight values (points) of male child No. 35 during first 5 years of life, and fitting by model (solid line).

Table IX. Weight (g) and length (cm) for a cohort of 45 infants estimated at 3-month intervals by virus infection rate 0–6 months

Group[1]	Infection rate/ child	Age, months				Total
		3	6	9	12	
		Weight				
A	3.33	3,840	4,405	4,787	5,094	4,532
B	6.20	3,838	4,385	4,756	5,056	4,509
C	9.73	3,409[2]	3,909[2]	4,253[2]	4,533[2]	4,026[2]
		Length				
A	3.33	50.13	52.71	54.49	55.89	53.31
B	6.20	50.05	52.75	54.52	55.92	53.31
C	9.73	48.68[2]	51.16[2]	52.84[2]	54.20[2]	51.72[2]

1 15 children/group
2 $p < 0.01$.

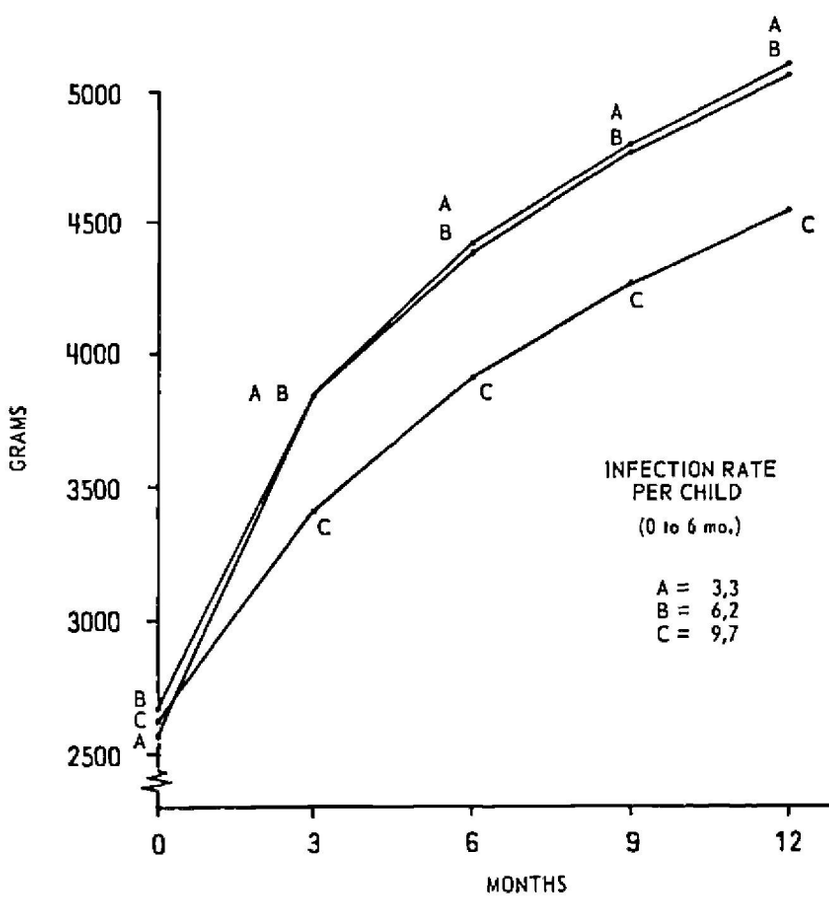

Fig. 5. Estimated weight curves for 3 groups of 15 children each, with different virus infection rates. Weights at 3-month intervals were obtained from individual fitting of curve of each child for the first 5 years of life, as in figure 4. Individual values in each group of 15 children were averaged to produce curves A, B, and C.

of the host economy. To explore this interaction, a cohort of 45 children was distributed among 3 groups according to the average number of virus infections per child during the first 3 months or the first 6 months of life (table VIII). Weekly fecal cultures were carried out in 3 cell culture systems. Weight and height data for every child during the first 5 years of life were fitted by a formula that produced excellent R2 values in most cases (fig. 4). The curves for every group of 15 children were averaged, and values at 3-month intervals during the first year of life were predicted [11]. It was shown that children with more infections during the first 3 or 6 months of life had a lesser growth in weight and height than those with fewer infections (table IX and fig. 5). The differences disappeared, after adjusting by analysis of covariance for weight or height of the preceding trimester, thus pointing to an association of growth with infection in the early months of life. On the other hand, the differences were not determined by birth weight or height, since adjusting by analysis of covariance for the birth values did not remove the difference.

Many avenues where infection and infectious disease affect the nutritional status and growth have been described [24]. Under field conditions, a significant decrease in food intake is frequently observed, attributable to anorexia, fever, and vomiting. Furthermore, interference with possible digestion and absorption has been noted. However, the most significant effect could be related to metabolic alterations; practically all biosynthetic and enzymatic pathways can be altered during infection, even under adequate nutritional conditions [1]. Nitrogen losses, alterations in amino acid metabolism and enzymatic systems, deviation of biosynthetic pathways, and synthesis of toxins or abnormal proteins can all be important mechanisms [23]. At the community level, the interaction of social factors and cultural characteristics often leads to poor management of the sick, such as the supression of food, inadequate manipulation of the diet, and administration of equivocal treatments. It should be emphasized that, in areas of low socio-economic level, the direct and indirect effects of infection on the nutritional status are aggravated by the prevailing malnutrition, thus effecting a vicious circle [12, 16, 24].

Influence of Malnutrition on the Outcome of Infection

The fact that malnutrition and infection are always prevalent in less developed areas of the world has made it difficult to determine if malnutrition

Table X. Duration of *Shigella* infections in children studied from birth to age 3 years; Santa María Cauqué, 1964–1967

Shigella	Number of infections	Duration, weeks					
		1	2–4	5–8	9–12	13–16	17–38
dysenteriae	29	7	10	4	3	2	3
flexneri	75	18	21	18	9	5	4
boydii	21	14	6	1			
sonnei	7	7					
Total	132	46 (35)[1]	37 (28)	23 (17)	12 (9)	7 (5)	7 (5)

1 Values in parentheses are percentages.

Table XI. Frequency of complications in measles, by age; Santa María Cauqué, Guatemala, 1965–1971

Age, years	Number of cases	Diarrhea[1]	Broncho-pneumonia	Diarrhea and broncho-pneumonia
< 1	50	39 (78)[2]	20 (40)	14 (28)
1	37	30 (81)	23 (62)	20 (54)
2–4	64	47 (73)	41 (64)	30 (47)
5 +	23	13 (57)	14 (61)	7 (30)
Total	174	129 (74)	98 (56)	71 (41)

1 Excluding dysentery.
2 Number of cases (percentage in parentheses).

predisposes to infection; this has not yet been clarified. On the other hand, infectious diseases tend to follow a more severe course, are of longer duration, present more complications, and result in death more often when there is underlying malnutrition. For example, the duration of infections such as *Shigella* is longer (table X) as compared to its behavior in well-nourished populations [9]. Table XI shows how frequently complications appear in measles as observed in a highland village [26]. Chronic malnutrition and the accompanying alterations in gastrointestinal functions result in abnormal colonization of the small intestine; such alterations are readily corrected

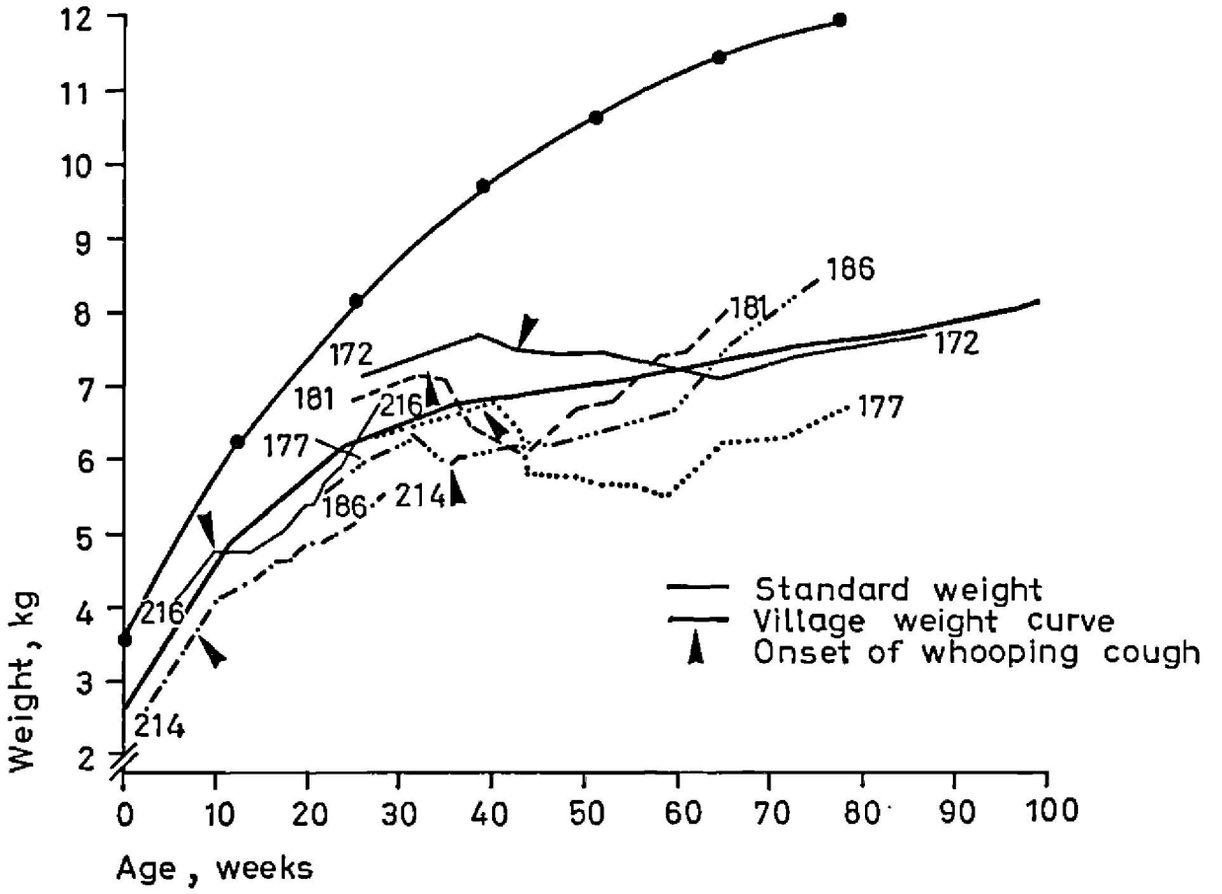

Fig. 6. Weight curves of 6 children that suffered whooping cough. Lower solid line is average village weight curve; upper solid line is standard for both sexes.

during nutritional therapy and without the aid of concomitant antimicrobials [10]. Finally, the mortality rate of many diseases is higher in malnourished children [3, 18, 20, 21].

That malnutrition affects the outcome of infection is clearly evident from the weight curves of children suffering from whooping cough (fig. 6). When whooping cough has its onset during the period of absolute breast-feeding, practically no effect on weight is observed (case 214); later on, still within the first 6 months of life, and when milk intake is probably adequate, the growth rate may be altered, but catch-up can be noted (case 216); however, at a later age, when children have a deteriorated nutritional status (as a result of progressive malnutrition and recurrent infections), the effect of disease becomes more marked, with very serious weight loss and a prolonged course of recuperation, as in cases 172, 181, and 177.

The mechanisms by which malnutrition affects the outcome of infection are several [24]. Cellular immunity seems to be defective even when malnutrition is not severe, resulting in an impaired capacity of the host to react

to viruses and intracellular bacterial infection [4, 24, 25]. Alterations in intestinal microflora have been recorded in malnourished children; these aggravate the existing malabsorption and chronic diarrhea [10]. Other factors related to the amplification of the immune response have been reported altered in malnutrition. The capacity of the host for tissue repair and the diminished supply for intermediary metabolites and other elements important for recuperation undoubtedly play a role [24].

Comments

The studies briefly summarized here attempt to clarify the importance of the binomial complex malnutrition-infection in rural communities of low socio-economic level. During the course of this research, about 4 million numbers were collected and are being analyzed by computers, with the hope to better elucidate the interactions and their significance for human growth. Presently, the complex malnutrition-infection emerges as the most significant biologic interacting variable influencing growth, health, and survival during childhood. Its relevance to mental development awaits further scrutiny, but it is expected to be important. Present knowledge indicates that neither the provision of an adequate diet nor the prevention and control of infection (as utopian as these measures may be in developing nations) will yield the expected results if implemented independently. Although more information on the various facets of the epidemiology of the complex malnutrition-infection is needed, current knowledge can be judiciously applied following rational and priority schemes. For example, the nutrition effort should be directed to the pregnant woman and to the child in the first two years of life; prevention and control of disease should be concerned with the pregnant woman and with the infant and pre-schooler, emphasizing vaccination in childhood and rational treatment of infectious disease in the mother and child. This will lead to better fetal and postnatal growth and to a reduction in infant mortality.

A survey of conditions prevailing in the developing nations of the world may show that these recommendations could encounter serious difficulties in their acceptance and application, owing to the prevailing political and economic conditions. The intellectuals possessing the knowledge should press to create a conscience among those responsible for the leadership of the nations, an attitude that should have the understanding and support of the societies of more advanced countries.

Conclusions

A long-term prospective study showed that, in an area of low socio-economic development, the interaction malnutrition-infection begins *in utero*. Mothers were malnourished and showed a high prevalence of infection; there was an indication of a high frequency of fetal antigenic stimulation. Post-natal infections have a marked effect on the weight curve; malnutrition aggravates the outcome of infection, as is shown by a prolonged course of infection, frequent complications, and higher death rates. The end result is a marked growth retardation beginning early in life.

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