MALNUTRITION AND CONCURRENT INFECTIONS.
COMPARISON OF TWO POPULATIONS WITH
DIFFERENT INFECTION RATES

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Repetitive infections induce malnutrition and growth retardation in children living in impoverished environments, even if food is available. Infection precipitates severe malnutrition in individuals already undernourished. Mechanisms include anorexia, nutrient losses, abnormal synthesis, and diversion from usual metabolic pathways. Infectious diarrheas, malaria, measles, and respiratory infections, all may cause malnutrition, disability, growth retardation, and death.

In two different rural populations of Central America, differences in infant nutrition, health, and survival were more related to the infectious environment than to amount and quality of food consumed.

Exposure to infection was related to living conditions. Thus, the public health goal should be to raise the quality of life in order to diminish the force of infection. Measures recommended are: improvement of environmental sanitation, expansion of primary health care (including oral rehydration therapy and immunoprophylaxis), and promotion of maternal technology (breast-feeding and child-care practice), family planning, and health education. The approach should be holistic.

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I. INTRODUCTION

Epidemiologic studies in tropical and subtropical regions highlighted the interaction between malnutrition and infectious disease, and its contribution to determining much of the morbidity and mortality in developing countries. There is no doubt that infectious diseases cause physiologic and biochemical alterations leading to malnutrition, while a deficient nutritional status worsens the outcome of infection (1). However, the argument persists as to whether concurrent infections or deficient diets are the main contributors to the nutrition, growth retardation and premature death so commonly observed among children in impoverished societies (2). Clarification of this question is fundamental for establishing priorities and policies aimed at prevention and control of infection and malnutrition.

Prospective observations in different parts of the developing world revealed that growth faltering invariably begins at about 3 to 6 months among infants at the breast in traditional societies, or even earlier if infants are prematurely weaned as is the case of populations in transition that adopt bottle-feeding (3). It is not yet clear if onset of stunting is primarily related to supplementation with inadequate foods when mothers' milk becomes insufficient; or to infectious disease; or to an interaction of both. An inadequate milk supply without proper supplementation is commonly the case in developing societies, but the striking even during weaning is the occurrence of infectious diseases (4).

Among the communicable diseases of childhood, infectious diarrhea ranks first in incidence and implications for nutrition and health, followed by acute respiratory disease which is very important as a cause of morbidity and death primarily in infancy. In the third place rank communicable diseases like measles, whooping-cough, chickenpox, rubella, and tuberculosis. Still, malaria, kala-azar, many viral fevers, and other ailments occur in particular ecosystems.

II. FIELD STUDIES ON NUTRITION AND INFECTION

The long term prospective studies to be described here concern two indigenous populations in Central American highlands devoid of malaria, trypanosomiasis, jungle fevers, and other "tropical diseases". In one of them, Santa María Cauqué, the burden of infection is overwhelming because crowding,
poverty, and underdevelopment favor its perpetuation (4). In the other, Puriscal, improved home environment and maternal technology, health services, and scattering of the population result in a very low incidence of infectious disease (5).

At the time of the study most homes in Cauqué had only one room and families slept in one or two beds or mats on the floor; preparation and cooking of food was on a hearth on the dirt floor; water had to be carried from public faucets and reservoirs; few families had intradomiciliary water and only a thiru use latrines. These factors potentiate transmission of infectious agents, especially enteric and respiratory, a condition favored by poverty, illiteracy, cohabitation with domestic animals, and crowding of families within the limited space of dwellings, and of homes within the compact village structure (4), Table I.

In rural Puriscal, living conditions are better as homes are larger; there is greater availability of intradomiciliary water, latrines and water toilets, and widespread use of electricity. This condition, together with a high literacy rate, greater income and availability of health services, improved maternal technology, and ruralism (marked separation of homes from each other), result in a very low incidence of infectious disease (5), Table I.

**TABLE I. Differences in Host and Environment between Cauqué (Guatemala) and Puriscal (Costa Rica)**

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Population</td>
<td>1000</td>
<td>24000</td>
</tr>
<tr>
<td>Type of population</td>
<td>Rural, crowded</td>
<td>Rural, sparse</td>
</tr>
<tr>
<td>Literate</td>
<td>56</td>
<td>85</td>
</tr>
<tr>
<td>% use latrine or toilet</td>
<td>32</td>
<td>95</td>
</tr>
<tr>
<td>% has private piped water</td>
<td>7.4</td>
<td>84</td>
</tr>
<tr>
<td>Human milk intake, ml, 1-3 mo.</td>
<td>674</td>
<td>652</td>
</tr>
<tr>
<td>Energy intake, kcal, 6 mo.</td>
<td>400</td>
<td>480</td>
</tr>
<tr>
<td>Protein intake, g, 6 mo.</td>
<td>7</td>
<td>9</td>
</tr>
<tr>
<td>Energy intake, kcal, lactation</td>
<td>2078</td>
<td>2111</td>
</tr>
<tr>
<td>Protein intake, g, lactation</td>
<td>59</td>
<td>53</td>
</tr>
<tr>
<td>Weight, kg, 6 mo.</td>
<td>6.3</td>
<td>7.1</td>
</tr>
<tr>
<td>Height, cm, 6 mo.</td>
<td>60.5</td>
<td>66.0</td>
</tr>
<tr>
<td>Height, cm, pregnant women</td>
<td>143</td>
<td>153</td>
</tr>
<tr>
<td>% low birth weight infants</td>
<td>42</td>
<td>6</td>
</tr>
<tr>
<td>% vaccinated: measles, DPT</td>
<td>0</td>
<td>95</td>
</tr>
<tr>
<td>Infant mortality per 1000 l.b.</td>
<td>93</td>
<td>10</td>
</tr>
</tbody>
</table>
TABLE II. Infection in Cauqué and Puriscal

<table>
<thead>
<tr>
<th>Infection</th>
<th>Cauqué, 1965-72</th>
<th>Puriscal, 1980-81</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mothers</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ascaris, %prevalence</td>
<td>83</td>
<td>10</td>
</tr>
<tr>
<td>Shigella, %prevalence</td>
<td>9</td>
<td>&lt;1</td>
</tr>
<tr>
<td>Bacteriuria, %incidence</td>
<td>27</td>
<td>6</td>
</tr>
<tr>
<td>Neonates</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Shigella, %incidence</td>
<td>3.6</td>
<td>0</td>
</tr>
<tr>
<td>Intestinal protozoa, %incidence</td>
<td>4.7</td>
<td>0</td>
</tr>
</tbody>
</table>

In both field studies the ecosystem and characteristics of the population remained undisturbed, although in Puriscal interventions were established to improve incidence and duration of breast-feeding, making them similar to those in Cauqué, at least during the first six months of life (6). Food consumption by pregnant and lactating women in Puriscal was equal or less than for women of Cauqué. Food consumption by children in both settings was similar during healthy periods.

Fig. 1. Infection in a cohort of 45 Cauqué children, from birth to three years of age. Rates were calculated from weekly examination or culture of feces (7).
While similar dietary conditions prevailed in both rural settings, marked differences were noted in children in both studies regarding incidence of infectious disease, occurrence of malnutrition, and growth retardation and mortality, all of which were markedly greater in Cauqué.

III. INFECTION AND INFECTIOUS DISEASE

Cauqué mothers are common carriers of enteric organisms, and are a source of infection for their infants from birth onwards, Table II. This is favored by traditional delivery in the home in the squatting or kneeling position, and by exposure of neonates to maternal feces (7). Infections occur as early as the first few days or weeks of life, and usually are asymptomatic, probably as a result of protection afforded by human colostrum and milk (6).

By contrast, intestinal infection in Puriscal neonates is virtually absent since mothers are relatively free of infection and almost all deliveries are in clinics or hospitals which thereby reduces the opportunity for fecal contamination at delivery (6).

Customarily, Cauqué infants are exclusively breast-fed until 4 to 9 months. The burden of infection in the environment is so large, however, that diarrheal disease begins in the early months of life, increasing with age as breast milk and transplacental immunities fade away and infants begin consuming contaminated foods (7). Infants are progressively infected with enteric viruses, pathogenic bacteria and parasites, and by one year of age most had been exposed to a wide variety of pathogenic agents, as revealed by weekly examination of feces of cohort children for at least the first three years of life (4). By the third year, more than 50% carry enteroviruses, and more than 25% excrete Shigella and Giardia. Figure 1 illustrates incidence and prevalence of Giardia and Shigella in a cohort of children examined weekly during the first three years of life (7). Prevalences are larger than incidences because some infections last for several weeks. Undernourished children also show abnormalities in intestinal flora and colonization of the upper intestine (9).

Puriscal infants also are exclusively breast-fed, although for a shorter interval than Cauqué infants, but the rate of infection is markedly lower.

Diarrhea among Cauqué children averaged seven or eight episodes per child per year in the first three years of life, a contrasting figure with the low rates for Puriscal children,
The maximum incidence and severity of diarrhea and other infections in Cauqué were observed during and after weaning (4). The etiology of diarrhea is similar in both settings with rotaviruses being predominant, followed by enterotoxigenic Enterobacteriaceae, Shigella, Campylobacter, Giardia, and other parasites.

The enormous infection burden observed in Cauqué can be accounted for by deficient sanitary conditions and traditional practices which potentiate communicability of enteric and respiratory agents. Children excrete as many as $10^6$-$10^9$ shigellae per gram of wet feces during bouts of diarrhea, and carriers may excrete bacilli for several months (4, 10). Thus, children in poor villages are at disadvantage when compared to their industrial counterparts, due to exposure to larger doses of virulent organisms. This might account for the greater severity of disease among children and adults in tropical areas regardless of their nutritional state, if sanitation and hygiene are deficient.

Few comprehensive studies on the etiology of respiratory disease in poor traditional societies have been conducted (11) but on clinical and epidemiological grounds, responsible viruses and bacteria are the same as described for industrial nations. Mortality due to respiratory disease is high among infants, especially if they are feeble or undernourished.

Measles, whooping-cough, chickenpox, rubella and mumps, induce severe manifestations among indigenous populations, and although they normally attack once in a lifetime, they are of great public health importance due to their malnourishing effect, disability, and mortality (4, 7, 9, 12).

**Table III. Incidence of Infectious Diseases in Cauqué and Puriscal Infants, per 100 Person-Months**

<table>
<thead>
<tr>
<th>Age, months</th>
<th>Number of infants</th>
<th>Person-months</th>
<th>Enterica</th>
<th>Respiratoryb</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-5</td>
<td>45</td>
<td>270</td>
<td>33.3</td>
<td>15.9</td>
</tr>
<tr>
<td>6-11</td>
<td>45</td>
<td>270</td>
<td>63.0</td>
<td>23.0</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0-5</td>
<td>115</td>
<td>690</td>
<td>4.2</td>
<td>5.5</td>
</tr>
<tr>
<td>6-11</td>
<td>114</td>
<td>684</td>
<td>7.5</td>
<td>7.7</td>
</tr>
</tbody>
</table>

aDiarrhea and dysentery  
bLaryngotracheobronchitis, bronchitis and bronchopneumonia
IV. NUTRITIONAL IMPLICATIONS

The negative effects of infectious disease on the nutritional state are reduced food consumption, nutrient losses, metabolic alterations, hormonal imbalances, and alterations in immune function. They are manifested as wasting, stunting, reduced activity, acute malnutrition, and death.

A. Reduced food consumption

This is best illustrated by enteric disease, in which agents adhere, colonize or invade mucosal cells, initiating pathophysiologic processes in susceptible individuals. Anorexia, febrile infections, diarrhea, and vomiting were the main cause for reduced food consumption in Cauqué children. This was complicated by food withdrawal and inappropriate treatment and feeding during convalescence. Marked reduction in calorie intake ("dips") coincided with infectious diseases (13, 14) in all fully weaned two-year-old children, among a cohort of 45 studied by weekly surveys, Figure 2.

Fig. 2. Energy intake by Cauqué child 24, as % of recommendation by weight. Dots are daily means; numbers are illnesses; bars duration. Episodes 27, 29 (diarrheas), 30 (rubella), 31 (fever, respiratory disease), and 32 (cellulitis, bronchitis) were associated with reduced consumption (13).
Energy and protein consumption was measured during weeks of diarrhea and during healthy periods; convalescence weeks were excluded from the analysis. Energy consumption was reduced by 21% and protein by 24% during bouts of diarrhea, Table IV. It is estimated that as much as 16% of dietary calories and 18% of protein were not consumed by Cauqué children as a result of infections since children were ill during one fourth to one third of their entire first three years of life (4). The impact is considerable for children at risk of becoming malnourished or already undernourished, as food consumption is often below recommended levels (4, 13, 14). Similar results were obtained in other societies of different ethnic background (15-17). Anorexia also occurs in acute respiratory disease and it is noteworthy during attacks of whooping-cough and measles, but reduced food consumption is observed even with mild infections (7, 14, 17, 18).

B. Altered digestive - absorptive processes

Diarrhea accelerates transit through the intestinal tract, and this restricts consumption and interferes with digestion. The surface epithelium can be coated and altered by Giardia or bacteria or it may be damaged by invading viruses, bacteria and parasites. Microscopic mucosal lesions concurrent with bacterial colonization are seen in persons living in the tropics (9, 19); jejunitis and the accompanying malabsorption of sugars, fats, and vitamins disappear spontaneously after the person settles in an environment with better sanitation (20). Enterotoxins stimulate formation of cAMP and cGMP impairing absorption of sodium and water. Certain bacteria split bile

<table>
<thead>
<tr>
<th>Health condition</th>
<th>Weekly measurements</th>
<th>Mean daily consumption</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Energy, kcal(MJ)</td>
</tr>
<tr>
<td>Well</td>
<td>105</td>
<td>914(3.82)</td>
</tr>
<tr>
<td>Diarrhea</td>
<td>84</td>
<td>721(3.02)</td>
</tr>
<tr>
<td>% Difference</td>
<td></td>
<td>21</td>
</tr>
<tr>
<td>Well-Diarrhea</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

salts reducing micelle formation and fat absorption, and increasing the bile acids pool which, in turn, irritates the mucosa. Bacteria and parasites are capable of sequestering nutrients that are needed by the host.

The intestine can be regarded as a secreting organ, diarrhea being an abnormal hypersecretory state, with losses of fluid, electrolytes and protein. Hypersecretion is caused by imbalances in digestive-absorptive processes; changes in hydrostatic pressure; synthesis or increase in enterotoxins, bile and fatty acids, hormones and neurotransmitters; and greater calcium permeability (21).

C. Metabolic alterations

Practically all metabolic pathways become altered during the course of infection (22). Abnormalities also occur with mild or asymptomatic infections. The most important alterations are negative nitrogen balance, protein-losing enteropathy, and abnormal nutrient metabolism. The stress of fever, cramps, tenesmus, anxiety, and the need to satisfy gluconeogenesis, result in corticosteroids release and mobilization of amino acids from muscle (1). Protein losses probably relate to thinning and alterations of the intestinal wall (18) and occur in diarrheas of various etiologies, as with Shigella, enterotoxigenic E.coli and rotaviruses (23). There are important losses of sodium, potassium, bicarbonate, chloride and phosphate during enteric and systemic infections. There is an increased synthesis of liver enzymes; of foreign protein, lipid, and carbohydrate (in viral replication); and of acute-phase reactant proteins (nutrient diversion). Also, there is a decrease of circulating trace elements (zinc, iron and copper) and concentration in hepatic cells (nutrient sequestration); and an increased expenditure of energy sources and vitamins (nutrient over-utilization) (22, 24).

D. Wasting and Stunting Effect

Repetitive infections and infectious processes, particularly in children who normally do not consume an optimal amount of food, conduce to progressive deterioration of the nutritional status, manifested as weight stagnation or weight loss, and impaired linear growth. These effects are evident in individual growth curves of Cauqué children observed prospectively throughout preschool age.
Fig. 3. Growth of a Cauqué breast-fed child born with adequate weight. Periods of growth arrest were associated with diarrhea of various etiologies (5).

Fig. 4. Growth of a Cauqué breast-fed child who had fetal growth retardation. Diarrheas were related to growth arrest. Marked stunting was evident at one year of age (5).
In Cauqué there was about 40% low birth weight infants; breast-feeding was universal; and the association of diarrhea with weight faltering and stunting was clear. There was no adequate knowledge of oral rehydration, while other forms of handling severe dehydration were not accepted by the community during the study period, contributing to the severity of the disease (4).

In general, growth during exclusive breast-feeding exhibited a velocity comparable to that of the NCHS-CDC reference curves. At about 3 to 6 months there was a tendency to growth faltering with onset of supplementary feeding. Often, foods were poorly prepared, too bulky, and contaminated. Inadequate supplementation after mother’s milk became insufficient (four to six months) resulted in subtle starvation of infants remaining at the breast. However, marked inflections in growth curves during weaning corresponded, more often than not, to concurrent infectious diseases, particularly diarrhea.

Figure 3 shows the growth curve (body length) and diarrheal episodes of a Cauqué child who had normal intrauterine growth. Diarrheas were associated with weight loss or weight stagnation regardless of their etiology. Obviously, losses of water, electrolytes, plasma, and cells during diarrhea had a negative effect on nutrition. Acute weight loss of the order of 5-10% or more was common. Furthermore, wasting (deficit of weight for height) persisted for weeks or months, contributing to the genesis of marasmus. This was complicated in many instances by inadequate feeding during convalescence. The wasting and stunting phenomena occurred also in conjunction with other diseases, very notoriously with measles and whooping-cough. The malnourishing capacity of illnesses that attack only once can be more pronounced that that of diarrhea.

In general, impaired growth was more evident if infants had fetal growth retardation, or were prematurely weaned (5). Figure 4 depicts the growth curve (body length) and diarrheal episodes of another Cauqué child who had fetal growth retardation. Several diarrheas associated with a variety of agents occurred concomitantly with periods of arrest in linear growth. One double infection with Shigella and Salmonella occurred without recognized diarrhea, and yet was concurrent with marked growth retardation lasting for more than two months. At the age of two years the child was, as the previous case, considerably stunted. Cauqué infants with normal fetal growth can also be affected by infection and become stunted, although not as severely.
Fig. 5. Growth of a Puriscal weaned child born with adequate weight. A bout of diarrhea caused significant weight loss.

Fig. 6. Growth of a Puriscal weaned child who became undernourished due to neglect, and resulted very ill after bouts of diarrhea requiring hospitalization.
The situation of Puriscal children studied more recently (1979-81), was markedly different in that only 6% of them had low birth weight. Sanitary conditions, education, and hygiene were significantly better than those in Cauqué. There had been a deterioration of breast-feeding in Puriscal evident at the beginning of the study, but hospital and field interventions commencing in 1977 resulted in more than 90% of infants being breast-fed for at least one to two months. Puriscal infants usually had fewer infections which were also milder than in Cauqué, probably due to smaller inocula and fewer sources of infection resulting from improved hygiene, and from isolation of dwellings (ruralism).

Most Puriscal children are vaccinated against measles, whooping-cough and other communicable diseases. A very low rate of diarrhea was observed in Puriscal breast-fed infants (see Table III). Furthermore, diarrhea generally did not show a marked negative impact in breast-fed children as it did in Cauqué. However, weaned Puriscal children experienced weight loss and growth retardation as a result of infectious diseases, particularly diarrhea. To illustrate, Figure 5 depicts a prematurely weaned child with normal fetal growth who developed diarrhea associated with weight loss. Another child prematurely weaned and suffering from neglect and several bouts of diarrhea, exhibited marked weight loss and required hospitalization (Figure 6). No marked inflection in the growth curve of breast-fed infants could be attributed to infections.

Thus, the main environmental difference between the two settings is the considerably greater risk of infection and infectious morbidity in Cauqué as compared to Puriscal. The notorious biologic difference was the exceedingly higher frequency of prematurity and fetal growth retardation in Cauqué, as contrasted with Puriscal. No marked differences were evident regarding food consumption among mothers and children of the two populations. In fact, breast milk intake by Cauqué infants was slightly greater than in Puriscal. Because Cauqué women consume about one half kilogram of corn per day, their calorie and protein consumption is similar, or greater than those of Puriscal women.

If infection is an important determinant of malnutrition, marked differences in growth are to be expected among the two cohorts. To explore this possibility, mean weight curves were computed for 133 cohort Cauqué infants born in the period 1964-67 (4), and were compared with mean curves for 277 cohort Puriscal infants born during 1979-81. Children in both cohorts were consecutively born during the stated periods. Cohort infants were classified according to fetal maturity.
Term-adequate-for gestational age Cauqué infants were notoriously smaller than their Puriscal counterparts which matched the 50th P of the NCHS-CDC reference curve, Figure 7. Furthermore, term-small-for gestational age Cauqué infants exhibited an even greater growth retardation, while the comparable Puriscal infants followed the corresponding track of the reference curve. Also, it should be noted that no large-for-gestational age infants were observed in Cauqué, a reflection of the marked fetal growth retardation observed in this village (4). It is rather evident that in Cauqué, infection together with fetal growth retardation, appear as the main determinants of postnatal growth failure, both of body weight and length.

E. Severe malnutrition and death

Repetitive infectious diseases lead to chronic malnutrition. This is clearly evident in village children observed longitudinally, as progressive wasting and stunting develops. The marasmic state persists for months and it is similar to that resulting from food shortage, or child abuse. Marasmic children become critically ill under the stress of acute watery diarrhea, dysentery, measles and other infectious diseases,

![Graph](image)

**Fig. 7.** Mean weight curves of Cauqué and Puriscal cohort infants observed prospectively from birth to nine months of age. TLGA= term-large, TAGA= term-adequate, and TSGA= term-small for gestational age.
as they do after episodes of social and psychologic perturbation of the home environment, for instance, the loss of a parent, violence in the family, or child neglect (25).

Sudden falls in serum albumin concurrent with infections (measles, hookworm, diarrhea) may precipitate kwashiorkor. This possibility would explain the etiology of edematous malnutrition which could result from hypoproteinemia in marasmus after severe diarrhea (18). When serum albumin falls slowly and an attack of measles or diarrhea develops, kwashiorkor may occur. It has been known for decades that severe energy-protein malnutrition appears in the community a few weeks or months after outbreaks of diarrhea, measles, and malaria (26, 27), sometimes quite independent of the availability of food (4, 28).

Children in poor villages may suffer infectious diseases for a fourth to a third of their entire infancy and preschool age. Thus, they represent a main determinant of acute malnutrition, and are responsible for elimination of children who are weak, born premature of with fetal growth retardation, or suffering immunologic, biochemical or organic deficiencies. Figure 8 illustrates the life history of a Cauqué girl who grew well in the first two months of life until she developed mild respiratory infection and bronchitis with marked growth deceleration. After several weeks of weight stagnation, and

![Fig. 8. Weight curve, enteric infections and morbidity of Cauqué female child 19. Two distinct episodes of malnutrition developed in connection with infectious diseases. The girl died from acute lower respiratory disease (4).](image-url)
despite being at the breast, she developed meningitis due to *Streptococcus pneumoniae*. Although growth improved significantly thereafter, a series of mouth, respiratory, eye, and gastrointestinal infections, and malnutrition, ended in death (4).

In the absence of adequate family technology, health services, and oral rehydration, infectious diseases represent the main factor generating malnutrition and death in malnourished children in developing countries.

V. EFFECT OF NUTRITION ON RESISTANCE

The most important host factor curtailing or ameliorating infection is breast-feeding in the first months of life (3). Other mechanisms of host defense against infection are gastric acidity, intestinal motility, intestinal microflora, and specific immune response and its amplification. Adequately nourished individuals can cope better with dehydration, nutrient losses, and other consequences of infection; they also exhibit a competent cell-mediated immunity, a good amplification of the immune response, and an integrity of natural barriers (1).

Intrauterine growth retardation results in impaired immunocompetence, and small-for-gestational age infants exhibit a higher incidence of infection and mortality in the first years of life (4, 29). Postnatal undernutrition, whether of a nutritional origin (as in famine) or due to nutrition-infection interaction (the commonest form in Central America) is associated with alterations in resistance. Alterations in integrity of skin and mucosae, hypochlorhydria, and changes in intestinal microbiota, all favor infection. Furthermore, severely undernourished children may have a decreased lymphoid cell mass, an impaired function of T and B immunocytes, and diminished synthesis of complement and secretory IgA (30, 31).

Undernourished Cauqué children experienced a higher attack rate of diarrhea, a greater severity and mortality due to infectious diseases, and a greater difficulty in controlling infection and recovering from illness (4, 32, 33). In fact, duration of infectious disease and of the carrier state (as in shigellosis and measles) was generally longer in undernourished than in well nourished individuals (4). Severely malnourished children show an absent or diminished T cell response, and children may die of measles without manifestation of the exanthem.

Severe undernutrition observed under conditions of extreme food deprivation, may result in depletion of the immune system
which may rather antagonize infection. Thus, starved individuals may develop acute infectious disease upon nutritional recuperation (34, 35). On the other hand, infection may alter immune function in a variety of ways including exacerbation of immune function, secondary immunodeficiency and immunological paralysis (36).

COMMENTS

It may be equivocal to assume that malnutrition predisposes to infection since infection rather depends on exposure of the host to the agent. This is substantiated by the high acquisition rate of enteric infection and diarrhea among well-nourished travelers to tropical regions (37), thus incriminating the environment with its high potential for host contamination, much more than the nutritional status as the crucial factor for inducing infectious disease.

In traditional societies breast-feeding is a part of the culture, and nutrition and growth are adequate for the first three to six months of life. During this period, the child is much protected from infection and from its negative nutritional impact by maternally-derived nutrition and immunity. Also, dehydration is corrected or ameliorated by a continuous supply of breast milk. As weaning begins, continuous exposure to infection results in concurrent diseases and a deterioration of the nutritional state. In transitional societies, many infants are not breast-fed at all, or are weaned early in life, and the deleterious effects of infection may be evident shortly after birth if infants live in a deprived environment. If this is the case, recurrent disease episodes induce weight loss and growth arrest, leading to chronic or acute malnutrition.

The nutritional damage is worse if oral rehydration and other forms of treatment are not available, a common situation in villages throughout the world. Recurrence of diarrhea, anorexia, and fever, coupled with diets of low biological value and unhygienically prepared (38), induce marasmus, or precipitate kwashiorkor. Death is common among dehydrated children not receiving fluid therapy. The risk of death is greater for preterm and small-for-gestational age infants, or for those with chronic or severe malnutrition. Well nourished children, however, may also die from diarrhea, bronchopneumonia and other infectious diseases.

Prospective community studies emphasize the importance of environmental sanitation and home technologies (4, 14) for promotion of child nutrition and health, as opposed to food
distribution programs and isolated nutritional interventions. However, populations suffering from acute food shortages constitute an exception in that food distribution should receive immediate consideration.

A practical way to know whether infection or diet should be the target for intervention is to examine community prevalence of undernutrition and mortality behavior. When food supply is limited, as a result of natural or man-made disasters or war, undernutrition and excess mortality appear in all ages and sometimes in all social classes. The usual situation in the developing world, however, does not seem to be a limitation in diet, as children in Asia, Africa and Latin America consume more than 80% of the calories and protein recommended by WHO/FAO; this level seems compatible with normal growth of children in industrial nations (39). Furthermore, undernutrition in most developing countries is circumscribed to infants and very young children and to the very old who live in isolation (4). Then, a logic explanation for the malnutrition observed in children of developing countries is the continuous stress resulting from infection and social pathology. In this regard, observations in Uganda and Costa Rica revealed that psychosocial factors lead to voluntary and involuntary child abuse and neglect, and precipitate severe undernutrition (25, 40). Deficient maternal technology (14) or maternal incompetence (41) in many cases promote infection and potentiate its sequelae.

Epidemiologic evidence indicates that prevention and control of infection, particularly enteric disease, correlate with improved nutrition. For instance, a decrease in diarrheal disease deaths in Costa Rica was highly correlated with a decline in infant mortality and a secular positive trend in height of children (42, 43). During the observation period, drastic changes in individual food consumption were recorded that could account for the significant gains in nutrition and infant mortality. Nevertheless, there was a dramatic reduction in deaths due to diarrheal and respiratory diseases, measles, whooping-cough and other communicable diseases. These changes were concurrent with a marked increase in per capita income, sanitation, immunizations, and availability of health services.

It is then expected that widespread application of oral rehydration, and current trends in improved water supplies, primary health services (including immunizations, family planning), and education, will result in a generalized betterment of the world nutritional situation, without necessarily requi-
ring an increase in food consumption. This comment, however, does not apply to areas where there is a proven limitation in food availability.

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