Proc. Nutt. Soc. (1979), *38, 29*

The malnutrition—infection complex and its environment factors

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Study of the genesis of malnutrition in contemporary traditional and transitional societies clearly reveals that malnutrition is a man-made condition or social disease. Apparently, malnutrition does not occur among wild animals living in small population groups (Van Lawick-Goodhall, 1970 appearing only as a consequence of congenital defects, injury, natural or man-made disaster or abrupt changes in climatic conditions which upset food supply and the ecosystem in general.

It seems probable that ancestral man organized in small groups of hunters and gatherers did not suffer from endemic malnutrition as it is observed today in developing nations (Mata & Mohs, 1976). Feeding practices then ensured a varied diet, while the smallness of the tribe accounted for a rapid extinction of pathogenic organisms entering into the group once susceptibles became immune or died. Breast-feeding was customary as it still is in traditional societies (Mata, 1978), while ablactation probably was accompanied by a significant infant mortality which eliminated the less fit. Exposure to pathogenic micro-organisms depended on intertribal contacts, and experience with some agents was never gained. In fact, the pre-Columbian Amerindians, not knowing measles, smallpox and other debilitating infectious agents, became decimated once exposure to them was provided by Europeans (Dubos, 1959).

The success of man in taming the environment and proliferating wherever he set foot eventually led to the establishment of sedentary societies, often near rivers. As they grew in size, a systematic and progressive elimination of wildlife, destruction of forests, erosion of soil and formation of barren soil and desert was ensured (Eckholm, 1976).

The environment of the malnourished child

In this historic evolution, some environments were more favourable than others to man' s progress and development. The richness of the soil, the existence of marked seasonal variation, and other related features, for instance in Scandinavia, North America and Russia, probably interfered with malnutrition-infection forces and permitted an easier attainment of civilization.

The present concentration of poverty and malnutrition in the tropical and subtropical belts in contemporary times, is clear evidence that the location (latitude) and climate of a given territory have been important determinants of malnutrition. In these regions the environment offers conditions favourable to the endemicity of malnutrition and infection (Mats, 1976).

Man's habitat (macroenvironment) in most developing countries characteristically has soils adequate for development of the infective ova and larvae of debilitating diseases. Vectors proliferate abundantly favouring endemicity and epidemicity of a variety of tropical diseases and fevers. Intense rain and humidity stimulate proliferation of weeds, pests and predators, while dryness and heat enlarge the deserts. Such disadvantageous conditions are accompanied, more often than not, by archaic socio-political and economic systems (the social environment) which perpetuate the status quo.

The home (microenvironment) plays a role in the development of malnutrition and infection. Crowded homes with dirt floors, thatched roofs and cracked walls, favour transmission of respiratory and enteric agents, and proliferation of arthropods and rodents. Deficient sanitation is the most important feature of poor housing. Lack of a safe piped-water supply, inadequate disposal of faeces and garbage, inadequate preparation and storage of food, the presence of animals in the home, and deficient personal hygiene, result in large doses of pathogenic agents on hands, and in food and water. Such environments also may deprive the child of psychological and social stimuli beneficial to nutrition and growth.

In many situations the mother (matroenvironment) is the most important determinant of a child's health and disease. She herself can be malnourished or a carrier of disease (Mata, 1976), and more importantly, the inducer of malnutrition in the child, by gestation of low birth weight and pre term infants (Mata *et al.* 1976), by inadequate or deficient 'maternal technology' affecting postnatal growth and survival, or by both conditions combined.

The concept of maternal technology is derived from observation of contrasting field situations in which protein-energy malnutrition (PEM) (the emphasis of this paper will be on the chromic forms of PEM, generally known as stunting and wasting, and not to the acute forms (marasmus and kwashiorkor)) is absent in extremely-poor homes or present in homes where there seems to be adequate amounts of food and other conveniences. Observation of such situations reveals that mothers in poor homes often have an inadequate level of technology for child feeding and rearing. Maternal technology is dependent on socio-economic factors, education, ethnicity and religion (Aziz, 1978). Positive aspects of maternal technology are an emphasis on breast-feeding, adequate ablactation practices, hand-washing, maintenance of drinking-water in a separate container, avoidance of faeces during meal preparation and eating times, adequate preservation of food, and knowledge of the need for aggressive care of the ill particularly regarding rehydration and feeding in convalescence.

Force of infection in poor children

Information on infection was obtained by prospective observation of a group of forty-five children from birth to 3 years of age, in the Guatemalan village of Santa Maria Cauque (Mata, 1978); the Cauque study was conducted from 1963 to 1972. Infection with enteroviruses, bacteria and parasites began at birth. The mothers were frequent carriers of enteric and respiratory pathogens which was reflected in

a high rate of foetal antigenic stimulation and antenatal and perinatal infection (Mats, 1978).

The possibility that maternal and foetal infection induce foetal growth retardation and prematurity has been raised (Mats, 1975). No child was spared from infection with the common enteric agents in the first year of life (Table 1). Prospective weekly observation revealed that although breast-feeding (customary in the study area) confers a good level of protection against intestinal infection (Mats & Urrutia, 1971), infants frequently became infected in the first months of life. Prevalence and incidence of intestinal infection increased progressively with *age* to reach maximum values in the second year and first half of the third year of life (the peak of weaning). Studies elsewhere revealed that a similar phenomenon occurs with the respiratory tract (Ota & Bang, 1972).

Table 1. ***Parasites and viruses in faeces of infants from Santa María Cauqué, Guatemala, 1964–1970***

Period of life (months)	Agent	Prevalence (%)
6–8	*Giardia*	3·7
9–11	*Giardia*	8·0
0–5	Enteroviruses	20·6
6–11	Enteroviruses	41·6
0–5	Adenoviruses	3·1
6–11	Adenoviruses	3·0

The force of infection also relates to dose, expectedly large in unsanitary environments. Studies in another less-developed region showed a high rate of multiple enteroviral infection in children (Parks *et* al. 1967). The large number of carriers in the community provide frequent microbial contamination through food, water and personal contacts. The role of food needs to be emphasized since high concentrations of enteric bacteria are found in 'village' weaning foods (Mata & Capparelli, 1975; Rowland *et al.* 1978).

Incidence of infectious disease

One-third of the total life experience of children during the first 3 years of life was affected by infectious disease. Diarrhoea, lower respiratory infection and the common communicable diseases of childhood, in that order, were the more debilitating illnesses. The average incidence of diarrhoeal disease was 792 episodes/ 100 person-years (approximately eight episodes/child per year) during the first 3 years of life (Mata, 1978). Bronchopneumonia and other lower respiratory illnesses were second in frequency with approximately 2.5 episodes/child per year. The attack rate of the common communicable diseases (measles, chickenpox, rubella, mumps, exanthems, whooping cough) was o•8/child per year.

Rates were lowest during the period of almost exclusive breast-feeding and rose with age to reach the highest value at the peak of weaning, that is, in the second year of life (Table 2). This was particularly true for diarrhoeal disease, and to a lesser extent, lower respiratory infection.

Table 2. *Infectious diseases (/100 person-months) in forty-five children observed from birth to 3 years of age from Santa María Cauqué, Guatemala*

	Age (months)					
	0–5	6–11	12–17	18–23	24–29	30–35
Diarrhoeal diseases	33·3	63·0	77·8	87·4	78·0	55·0
Lower respiratory infections	15·9	23·0	23·7	27·4	24·3	14·0
Common communicable*	1·9	10·0	8·2	9·6	7·7	7·4
Diseases of the mouth	9·3	6·3	8·2	4·1	7·0	3·9

*Measles, chickenpox, rubella, mumps, exanthems, whooping cough.

It is not clear to what extent chronic PEM influences susceptibility to infection. Although it has been repeatedly stated that malnutrition predisposes to infection (Scrimshaw *et al.* 1968) there is no clear evidence to support the concept. The high rate of diarrhoea in well-nourished individuals travelling to developing countries (Lee & Kean, 1978) indicates that a contaminated environment and immunity, but not the nutritional state, determine infection. However, a deficiency in local secretory immunity in severe PEM may facilitate intestinal invasion and colonization by pathogens. On the other hand, infection in malnourished children often follows a protracted course with prolonged shedding (Mata *et al. 1*969; Scheifele & Forbes, 1972).

Effects of infection on nutrition

Controlled studies in adult volunteers demonstrated the profound metabolic alterations during the course of experimental infection (Beisel, 1972, 1977).

Diarrhoea is a good example to illustrate the deleterious effect of infection on the nutritional state, (Mata & Villegas, 1978). Food consumption is reduced in part due to an ethnic or traditional response of the mother or attendants to disease occurrence. On the other hand, infection alters nutrition in a direct way; anorexia, vomiting and increased motility reduce the volume of food consumed. Digestion is impaired through decrease in enzyme secretion or action (Scrimshaw *et* al. 1968). Increased peristalsis reduces the time available for digestion. Absorption is altered by epithelial lesion or by enterotoxins that stimulate release of cAMP inhibiting absorption of sodium and water. Certain bacteria have the capacity to deconjugate bile salts reducing the capacity for micelle formation, while some parasitic and bacterial agents are capable of competing with the host for nutrients. The result of these alterations is impaired absorption, loss of tissue and spoilage of nutrient supplies (Gorbach, 1972). Secretion is increased in diarrhoea by imbalance of digestive-absorptive processes, hydrostatic pressure due to vascular obstruction, stimulation of cAMP or increased cell permeability (Field, 1977). The over-all result is loss of fluid, electrolytes and protein.

Generalized infection induces a variety of metabolic alterations generally referred to as nutrient wastage (Beisel, 1972) (Table 3). Such alterations occur even with subclinical infections. Gross manifestations of host catabolic response are negative nitrogen balance, weight loss, and growth retardation.

Table 3. ***Nutrient wastage (metabolic alterations induced by generalized infection, Beisel (1972))***

Nutrient over-utilization	
	Increased expenditure of energy sources
	Increased synthesis of cholesterol and triglycerides
	Over-utilization of vitamins
Nutrient sequestration	
	Sequestration of iron in liver
	Increased intake of zinc by hepatocyte
Nutrient diversion	
	Uptake of plasma AA for 'acute phase reactants' synthesis
	Enzyme synthesis by liver
	Synthesis of foreign protein, lipids and carbohydrates

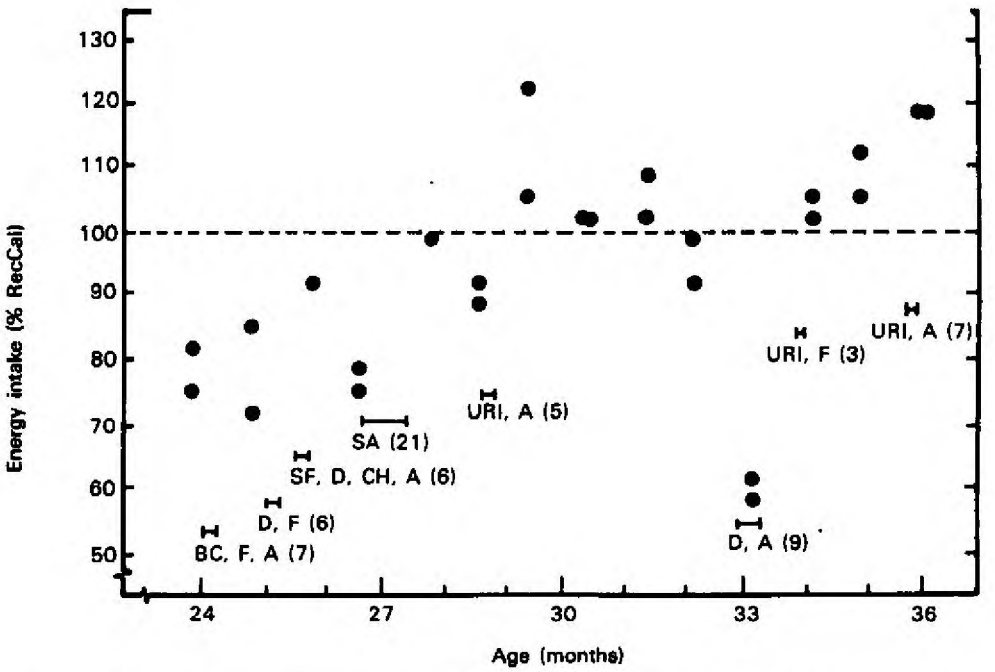

Fig. 1. Energy intake (% recommended energy; % RecCal) and morbidity of male child no. 37 from the Cauqué study (Mata, 1978); definitive weaning was at 24 months. Each point represents one weekly dietary study. Horizontal bars represent disease episodes; duration (d) is shown in parentheses. This child was among those with higher energy consumption (Mata & Villegas, 1978). A, anorexia; BC, bronchitis; CH, cheilitis; D, diarrhoea; F, fever; SA, skin allergy; SF, scarlet fever; URI, upper respiratory infection.

Anorexia. Reduced food consumption during infectious disease becomes evident by graphic display of dietary and morbidity histories of individual children, as shown in Fig. i for male child no. 37 (Mata & Villegas, 1978). Results extended from onset of full weaning to 3 years of age. Energy intake was measured by the recall method *(Mata et al.* 1978), and expressed as percentage of recommended energy (%RecCal) (Mata, Kronmal, Urrutia *et al.* 1976).

The 'village' diet consists of maize and beans in an approximate proportion of 7:3 complemented with green leaves, fruits and small amounts of animal protein (Mata *et al.* 1977). Furthermore, the percentage energy from net dietary protein for fully-weaned 2-year-old children was 5.6 (Mata *et al.* 1978), which indicate that protein is not a problem in the 'village' diet. Energy, however, appeared significantly deficient as previously reported *(Mata et aL* 1967, 1977; Mata, Kronmal, *Garcia et al.* 1976).

Energy deficits in child no. 37 were noted during the first 6-month period after weaning, characterized by frequent episodes of infectious disease. During the following 5 months which were free of disease, energy consumption improved and approached the t00% recommended level. An episode of diarrhoea and anorexia of 9 d duration was associated with a marked energy restriction to a level of 6o% of the recommendation; consumption improved thereafter. Histories of the other children in the group exhibited a similar phenomenon.

Table 4 summarizes energy consumption values belonging to seventeen of the forty-five children of the Cauque study (Mata, 1978) on whom %RecCal values were available for weeks with disease or free of disease; %RecCal values were averaged to obtain means for weeks with diarrhoea, with non-diarrhoeal illnesses, or free of disease.

Values for the a weeks following illnesses were not computed on the assumption that anorexia was still present, although it is often the situation with *Shigella* and rotavirus infection. It is probable that children remained anorexic for longer periods and therefore the results for healthy periods are underestimated.

To illustrate, child no. 76 had three episodes of diarrhoea and the mean %RecCal value was 83.7; for 8 weeks with other illnesses, the mean %RecCal value was 94.4; and for 9 weeks free of illnesses 95.7. The reduction in energy intake due to diarrhoea was 12%. Three children had slightly higher intakes during diarrhoea than in healthy periods, and fourteen had reduced energy consumption, as much as 56% less than the usual level of intake. A summary of all values gave a mean %RecCal for all 'diarrhoea' weeks of 79-9 (based on 90 weeks) and of 92.9 for disease-free weeks (based on 140 weeks) (Mata & Villegas, 1978).

Summarizing, the results reveal that ten of the seventeen children had adequate intakes during periods free of disease (the value could be larger if mean %RecCal values for healthy periods were underestimated). Furthermore, infectious disease frequently resulted in marked reduction in energy consumption, equivalent to an average of 0,84 MJ (zoo kcal)/d.

Food loss due to anorexia and fever were estimated as 4 and 2% respectively, calculated on morbidity figures for the Guatemalan children. Other sources of food

Table 4. *Energy intake (mean % recommended energy) mean %RecCal* by village children from Santa María Cauqué, Guatemala, during illness and periods free of symptoms (after Mata & Villegas (1978))*

(No. of weekly measurements is given in parentheses)

	Mean %RecCal			
Child no.	Diarrhoea†	Other illness‡	Healthy§	Change in mean %RecCal‖
63	82·6 (5)	60·3 (3)	80·1 (12)	2·5
54	82·4 (12)	—¶	80·3 (8)	2·1
46	97·5 (2)	103·3 (7)	96·8 (9)	0·7
23	88·3 (4)	85·7 (3)	90·3 (9)	−2·0
35	74·3 (9)	84·5 (6)	78·0 (2)	−3·7
79	102·0 (6)	98·5 (2)	107·6 (8)	−5·6
15	68·5 (2)	72·0 (2)	75·3 (12)	−6·8
80	72·8 (4)	75·0 (8)	82·0 (5)	−9·2
76	83·7 (3)	94·4 (8)	95·7 (9)	−12·0
49	70·0 (3)	64·0 (2)	83·7 (9)	−13·7
24	85·5 (2)	61·8 (5)	101·9 (17)	−16·4
82	65·0 (3)	61·9 (7)	82·2 (5)	−17·2
34	71·2 (11)	69·8 (5)	93·4 (13)	−22·2
91	70·0 (1)	112·0 (1)	99·4 (5)	−29·4
37	69·3 (3)	86·7 (3)	101·1 (11)	−31·8
88	106·6 (5)	—	143·0 (4)	−36·4
69	65·8 (15)	86·8 (14)	122·5 (2)	−56·7

*Mata, Kronmal, Urrutia *et al.* (1976).
†Diarrhoea, and diarrhoea associated to other illnesses.
‡Respiratory, exanthematic, febrile, skin, eye, ear.
§Includes convalescence, except two week periods following illnesses.
‖Consumption in total 'healthy' weeks minus consumption in 'diarrhoea' weeks.
¶Food consumption not recorded.

loss (wastage) are malabsorption due to subclinical small bowel injury (2.5% loss), malabsorption due to diarrhoea (o- 5% loss), and of loss due to nutrient diversion and sequestration (4%) (Briscoe, 1976). In total, food losses due to infection are of the order of 8%. Actually losses are greater because food not consumed by the child often is not diverted to other members of the family.

Physical growth. The effect of infection on the nutritional state is easily noted in growth curves of individual children. Fig. 2 depicts the life history on male child no. 35 during the first 3 years of life (Mats & Villegas, 1978). For clarity, only diarrhoeic episodes were recorded in Fig. 2. Histories containing complete morbidity records have *been* published elsewhere (Mata, 1978). During the period of intensive breast-feeding, a 14 d episode of diarrhoea did not alter the weight curve but affected height. The other diarrhoeal attacks occurred after supplemental foods were introduced; they were clearly related to acute weight loss and height stagnation. The periods of growth arrest consequent to infections may extend for weeks or months.

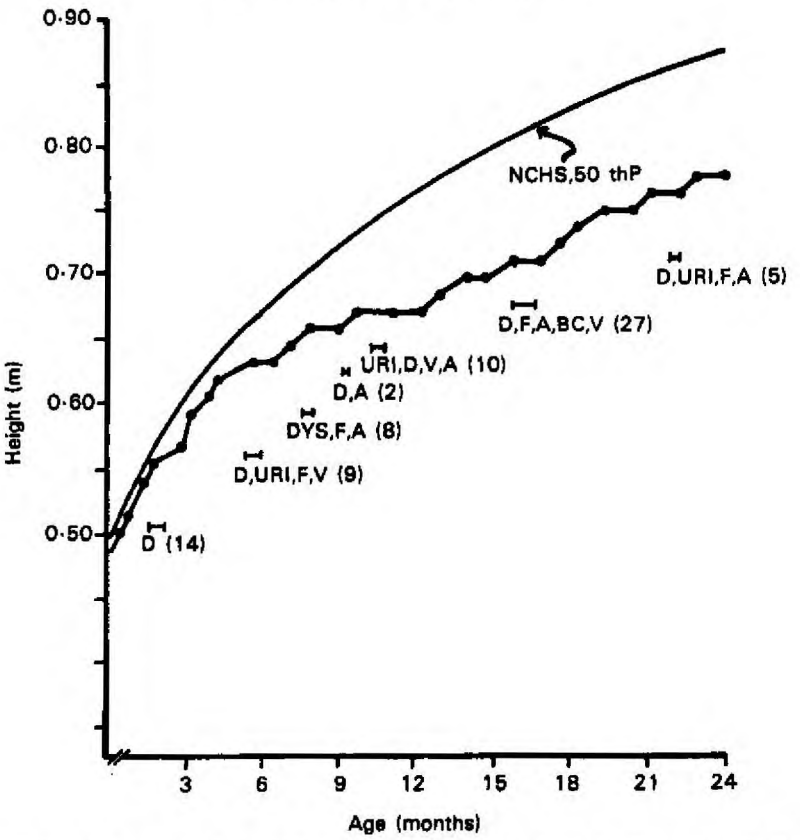

Fig. 2. Height (m) curve and morbidity of male child no. 35 from the Cauqué study (Mata, 1978). The child was fairly representative of the group in terms of experience with diarrhoeal disease. Only episodes of diarrhoea and vomiting, either alone or in association with other illnesses, are shown. Except for a period of decceleration coinciding with an episode of diarrhoea of 14 d, growth was fairly adequate during the first 4 months of exclusive breast-feeding. Most episodes of diarrhoea during the weaning period were associated with a decceleration or arrest in growth. (—), The 50th percentile of the (US) National Center for Health Statistics (1977) is shown for comparison. Horizontal bars represent disease episodes; duration (d) is shown in parentheses; A, anorexia; BC, bronchitis; D, diarrhoea; DYS, dysentery; F, fever; V, vomiting; URI, upper respiratory infection.

Other infectious diseases such as measles and whooping cough may be related to an even greater effect as measured by weight loss (Table 5) or by direct study of the growth curve; diarrhoea, however, has greater relevance in view of its much greater frequency.

Two facts become evident from the previous presentation. One is the high frequency of weight loss and growth arrest consequent to infectious disease. The other concerns the observed adequate growth velocity during periods of health, also reported for Gambian children living under stressful conditions (Rowland *et al.* 1977). These considerations, coupled with the often recorded adequate energy intake in the absence of illness, are a strong argument against an emphasis on

supplementary feeding programmes in rural settings where food availability is not the limiting factor and an intervention in the sanitary environment is not concomitantly made.

Table 5. *Weight loss (% weight at onset) of children (0–3 years old) from Santa María Cauqué, Guatemala surviving attacks of measles or whooping cough*

(No. of cases (% total) is given in parentheses)

Disease	No. of children	Wt loss 2	Wt loss 2–4	Wt loss >5
Measles	108	39 (36)	39 (36)	30 (28)
Whooping cough	44	13 (30)	19 (43)	12 (27)

Intervention to interrupt the malnutrition–infection cycle

Table 6 summarizes interventions and the expected effect on nutrition and health at the village level. Provision of water supply results in a marked reduction in transmission of *Shigella* (Hollister *et al.* 1955); immunoprophylaxis sharply reduces morbidity and mortality, particularly when practiced at the national level (Mata & Mohs, 1976). However, interventions affecting maternal technology population growth and agricultural practices require a considerable input in education and social development.

Table 6. *Interventions that will improve nutrition and health*

Intervention	Effect
Water supply and control of excreta	Prevention of food loss and nutrient wastage due to diarrhoea and parasitism
Immunoprophylaxis	Prevention of anorexia and nutrient wastage due to measles, whooping cough and other childhood diseases
Maternal technology	Prevention of infection in children; improvement of child nutrition; increase in child survival
Family planning	Delayed conception; increase in child-spacing; improvement of maternal and foetal nutrition
Agricultural measures	Increased food production; prevention of food loss

Familiarity with field situations in less-developed countries indicate that much could be advanced if methods to raise the level of maternal technology were available. Table 7 shows relevant factors for protecting the child against infection and promoting better nutrition. To fully understand the importance of the mother and the immediate environment of the child in terms of opportunity for infection, one must realize not only the role of infection in the genesis of malnutrition but the origin of diarrhoeal disease itself. For instance, the inability to identify an infectious cause in most diarrhoeas in the recent past, coupled with the common

occurrence of diarrhoeas during weaning (Gordon *et* al. 1963), led to belief in a nutritional cause of weanling diarrhoea. The situation changed with demonstration of pathogenicity of classically non-pathogenic *Escherichia soli* (De et *al.* 1956) and of enterotoxin-producing bacteria in the small intestine of diarrhoeic children (Gorbach *et al. 1*970, and of new diarrhoea viruses (Kapikian *et al.* 1972; Bishop *et al. 1*973; Flewett *et* al. 1973). Now it is possible to find infectious etiologic agents in approximately 60-70% of the diarrhoea cases in tropical countries. Since the agents are excreted in the stools, dissemination in the community occurs by direct or indirect contact with faeces.

Table 7. ***Maternal technology and traditions, beliefs and taboos***

Food handling, preparation, storage
Food consumption in health and disease
Habits relating to occurrence of illness
Habits of sleeping
Hand-washing
Handling of drinking-water
Habits of defaecation
Ablution
Bathing and personal hygiene
Pattern of socializing

Hence, there is a need to recognize the relevance of maternal technology particularly as it relates to food, water and contacts with other children, animals and objects in the surroundings of the child. Unfortunately, not much quantitative information is available on the influence of the various facets of maternal technology on disease occurrence, and much less on systems to boost such technology in rural and slum populations. Mission-oriented research is needed to correct present ignorance, focusing on interruption of transmission, oral rehydration and feeding during weaning, particularly in convalescence (Rohde & Northrup, 1976).

These actions should develop in conjunction with other interventions in a holistic approach to health intervention. The cost of improving sanitation should not be questioned so much as enormous sums are being spent in programmes of doubtful effectiveness. For instance, an investment in water supply in Costa Rica to cover a given population for many years, is comparable to a food distribution programme for preschool children during x year. On the other hand, people are entitled to have, sooner or later, adequate housing, education, health and food. In fact, values given by the World Health Organization reveal a progressive improvement in sanitation in many less-developed countries.

Comment

The role of infection in the causation of malnutrition has been increasingly recognized (Mata *et al.* 1967; Wittmann *et al.* 1967; McGregor *et al. 1968;* Taylor & DeSweemer, 1973; Rowland *et al. 1*977). Unfortunately, the emphasis has been on biomedical aspects of the malnutrition—infection complex, with less regard for its psychosocio-cultural factors.

Such studies, however, were important in demonstrating that infection is one of the main determinants (and often the most important one) of malnutrition in regions where food availability is not the limiting factor. Occurrence of both adequate energy consumption and physical growth during disease-free periods, and deleterious effects caused by infectious diseases (particularly diarrhoea) cast doubt on the justification of food-supplementation programmes for less-developed countries.

The ultimate cause of infection and malnutrition in underprivileged societies is poverty and low socio-economic development. Thus, the goal of interventions should be an over-all improvement in the quality of life, which will demand significant social changes quite incompatible with political systems prevailing in most countries in need. However, several measures could be readily implemented to diminish the force of infection and improve food utilization under the present world situation. These are, the classical interventions on environmental sanitation and the provision of health services and immunoprophylaxis, which should be accompanied by programmes to strengthen the level of maternal technology. The aim is to reduce disease transmission and to improve child-feeding practices. Interventions should be concomitant with measures affecting family size, education, agricultural practices, and other features of community development.

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