# The quantitative effect of infection on the use of food by young children in poor countries[1,2]

*John Briscoe,*[3] *Ph.D.*

ABSTRACT    A framework based on a consistent and explicit set of assumptions is developed for evaluating the effect of infection on the intake and efficiency of use of food by children under 5 years old in poor countries. A variety of data is used to obtain quantitative estimates of catabolic losses, clinical and subclinical absorbtive losses, changes in food intake patterns, and losses due to high infant and childhood mortality. Although additional data are required before these estimates can be considered reliable, it appears that about 9% of the food available to a cohort of children under 5 years old in Bangladesh is not used for maintenance, growth, or activity of those children who survive to their 5th birthday. The amount of food that is not used effectively may be reduced to about 3% in a (hypothetical) situation where all sources of infection are eliminated but other conditions remain unchanged. The estimates suggest that the most important factors contributing to this inefficiency are reduced intake through food withdrawal and anorexia, and high mortality in young childhood. The potential nutritional effects of various public health programs are tentatively assessed.    *Am. J. Clin. Nutr.* 32: 648–676, 1979.

While much has been written on the importance of the interaction of infection and nutrition, a satisfactory basis for the design of integrated health and nutrition programs has not yet been established. In this paper, we develop a framework for evaluating the effect of infection on the intake and efficiency of use of food by children under 5 years old in poor countries. A variety of sources is used to obtain quantitative estimates of this effect and to suggest critical deficiencies in available information. It is our hope that this analysis will contribute to an understanding of the nutritional effects of public health programs and thus, assist health planners in the effective use of scarce resources.

The objective of this study is proscribed in two important ways. The first proscription emanates from the fact that public health programs are never exclusively nutrition intervention programs, but are designed to meet several other legitimate health-related objectives. This analysis is not undertaken with the intention of assessing the overall efficacy of such programs in meeting their multiple objectives, but in the belief that clarification of the relationship between these programs and one of the important objectives is a significant step in developing a rational basis for such an overall assessment.

The question addressed in this study is further limited in that the issue of causation in the infection/malnutrition cycle is largely bypassed. We will consider a cohort of children from birth to 5 years old and estimate the nutritional effects of a hypothetical eradication of all causes of infection. This estimate gives an upper bound on the nutritional effect

of the elimination of any disease or group of diseases. Our analysis also enables us to estimate a lower bound on the effect of a reduction in the incidence of any specific disease of known prevalence. We are unable, however, to specify the "multiplier effect" of such a reduction since we have insufficient understanding of the degree to which an attack of one disease predisposes the child to subsequent attacks by other diseases. Unfortunately, the range covered by these upper and lower limits is wide in all cases. In sidestepping the intricacies of the infection/malnutrition cycle, we rely primarily on clinical studies in conjunction with population-based prevalence data for the derivation of the upper and lower bounds. Field studies of infection and nutrition are useful in assessing whether the actual effect of any program is likely to be closer to the upper or lower bound. These studies suggest, for instance, that the multiplier effect is particularly important in measles (1) and diarrhea (2, 3, 4).

This analysis is undertaken in the belief that our inability to give definitive answers to questions concerning the nutritional impact of health programs does not relieve nutritionists and health scientists of the responsibility for making judgments on the basis of the imperfect information that is available. Since nutrition intervention policies and other public health policies are going to be formulated, these policies will, implicitly or explicitly, assume some relationship between the provision of food and the use to which that food will be put. Our approach is to: *1)* develop a methodological framework, based on an explicit and consistent set of assumptions, with which to analyze the effect of infection on the intake and use of food by young children in poor communities; *2)* analyze the existing data in terms of this framework; *3)* indicate which data deficiencies appear to be paramount; and, *4)* draw the policy implications that tentatively emerge from this analysis.

We will attempt to reconcile the tension between the demand for policy guidance, on the one hand, with the demand for scientific validity, on the other, by being clear about how the "best estimates" are arrived at, what the implicit assumptions are, which of these are well-supported by existing knowledge and which are no more than educated guesses.

## Mechanisms by which infection affects nutrition

The effects of subclinical infections are that a large proportion of apparently healthy people in tropical countries appear to absorb nutrients poorly due to morphological changes in the gut.

The effects of disease are that the food intake of a sick child may be reduced due to anorexia, due to a reduction in the supply of mother's milk or due to substitution of the child's regular food supply with a watery gruel. The sick child may absorb nutrients less efficiently due to the administration of purgatives, due to the rapid transit of food through the gut during diarrhea, or as a result of morphological changes in the gut. The child may also suffer from catabolic losses of nutrients; body tissue may be destroyed and the products of this destruction excreted in sweat, feces, and urine. Functional wastage may result from nutrient overuse during infection, the sequestration of nutrients or the diversion of nutrients.

The effects of mortality are that the process of human reproduction in poor countries is nutritionally inefficient, since the high infant and child mortality rates mean that substantial quantities of food are consumed by children who do not survive childhood.

## Some definitions with implications for the formation of policy

### A semantic issue in describing host response to infection

Discussions of the effect of infection on nutrition are usually confined to an analysis of the alterations in the ways in which infected and diseased human beings use nutrients. In this paper we expand the terms of reference to include the nutritional costs of high mortality resulting from frequent invasion of the human host by pathogenic organisms. Our analysis will show that in nutritional terms the mortality associated costs are significant relative to the infection and disease related costs.

Without taking teleological speculation to an extreme, it seems plausible that many host responses to infection may be purposeful activities to ensure the survival of the host. At

least part of the muscle protein that is sacrificed during febrile illnesses is used for host defense purposes (5). Fever per se has been shown to confer protection against death in rats and it has been suggested that it may fulfil a similar function in man (6). The use of nutrients for purposes other than body maintenance, growth, and activity may appear to be inefficient when we assume that the host would survive in the absence of such reactions. When we admit the possibility that further infections and even the biggest nutritional loss of all, death, may be averted, such changes are no longer obviously wasteful.

### The measurement of nutrient "losses"

In the literature, there are numerous ways in which the effects of infection on nutrition are exaggerated. The caloric costs of an acute infectious attack have been overestimated by: counting some of the caloric losses from the body twice (by measuring the same loss in two different ways and then adding the effect of these two losses); not taking into account possible decreases in caloric requirements for activity during an attack of acute illness; and double-counting the effects of the caloric loss by adding the direct cost of the caloric loss to the caloric cost of recouping the loss. The effects of subclinical malabsorbtion, too, have been overstated by: dividing the sample population into "good" and "poor" absorbers and ascribing the difference between these subgroups to malabsorbtion, a method that ignores the fact that in any population the absorbtive capacity of a population is distributed around the mean value; and adding the monetary drain due to food wastage to the economic loss due to further limitation of food calories available for work activities.

The type of data that are required for evaluation of these losses on a community-wide basis do not seem complicated yet are not available. If we are working with caloric loss only (this choice will be discussed later), and, if we know, for a given illness, the following: the food intake of the person "at balance" before the attack (i.e., the sum of the person's requirements for maintenance and for activity); the food intake of the person during the course of the illness; and the loss of weight during the course of the illness, corrected, if necessary, for weight losses due

to dehydration; then we would be able to estimate the caloric loss associated with that particular illness in that particular person as:

Food loss (kcal) = intake during illness − "normal" intake over that period + food required to recover weight loss

We note a couple of points about the above:

1) The implicit assumption is that the food that is withheld (or refused) during the course of an illness is "stored" for that person and is available for that person during the recovery period. This is unlikely to be the case. If this assumption is seriously violated, we can look at the food loss in two ways. From the point of view of the family as a unit, the above equation remains applicable. From the point of view of the infected individual who is given no additional allowance upon termination of the illness and who receives no additional food when other family members are ill, the food loss is equivalent to the weight loss over the course of the illness.

2) In theory, it is quite possible that there will be a "negative food loss" (a food saving) due to an attack of illness when the loss equation is evaluated at the family level. This is not to say, of course, that the illness was a blessing to either the individual or family. We have considered the illness from one perspective only. The other costs associated with the illness (suffering, reduced participation in work and leisure activities, increased susceptibility to further illness of both the patient and the family as a whole) are not included. The argument is not that these are unimportant, but that they should logically be considered different from food loss.

This perspective is important in computing the increased nutrient requirements as a result of infection (7). Food that is diverted during infections should not be included when nutrient requirements are increased to take account of infection. A corollary of the fact that diverted food is different to lost food is that increased per capita availability of food during illness will not affect the intake of the sick individual.

### The nutrients evaluated

The relative importance of caloric and protein deficiencies in poor countries remains

controversial. Most recent analyses (8–11) suggest that it is calories that are of prime importance in most poor countries. There are others, particularly Scrimshaw (12), who believe that present nutrition standards (7) seriously underestimate the protein requirements of most populations. One of the major arguments of the latter group has been that the protein losses as a result of infection have not been adequately considered in setting these standards. Beaton and Swiss (11), Gopalan (10), and Latham (13) have argued that, because both energy and protein requirements are affected by major and recurrent minor infections, there does not seem to be any good reason to assume that the ratio of protein to calorie requirements should be increased. Recent data from Guatemala (14) on the effect of infection on the absorbtion of calories and protein support this argument for this particular component of the effect of infection on nutrition. The Institute of Nutrition for Central America and Panama (INCAP) data show that during mild to moderate episodes of diarrhea, additional fecal losses in children fluctuated between 58 to 210 kcal and 25 to 175 mg of nitrogen per day. If, in the absence of data on the distribution of these losses, we assume that the mean losses are the averages of the extremes given, then approximately 220 additional kcal are lost for each additional gram of protein that is lost. This ratio is much higher than the calorie:protein ratio in rice and wheat, which contain 46 and 29 kcal/gram of protein, respectively (15). These limited data on this one effect of infection on nutrition suggest that any correction to the ratio of calorie to protein requirement would further emphasize the importance of calories vis-à-vis protein.

In attempting to evaluate the effects on infection in terms of the standard diet of the region, we have attempted to assess the food required to build up one kilogram of body mass under two extreme assumptions. These are: *1)* that all of the loss in body mass during an infectious episode is muscular tissue, and *2)* that all of the loss is adipose tissue. Data presented by Scrimshaw et al. (16) suggest that 3.2 kg of muscular tissue are equivalent to 100 g of nitrogen. Using a nitrogen:protein ratio of 1:5.95 for rice (17), 1 kg of muscular tissue is equivalent to 190 g of protein. In terms of rice (parboiled—33.6 g protein per pound and NPU of 63%) this amounts to 4.20 kg of rice to replace 1 kg of muscular tissue. The number of calories required to build up 1 kg of adipose tissue has been estimated as 7700 kcal by Woolley et al. (18). Hegsted (9) confirms that this is the potential deposition of adipose tissue when excess calories are ingested. Ashworth's (19) data on malnourished children suggest that the efficiency of caloric use is greater at extreme malnourishment (9.6 kcal/g) than it is near recovery (17.4 kcal/g of tissue). Because the figure given by Woolley et al. (18) is, it seems, a "potential" figure, we take a value from Ashworth's data (19), of, say 12 kcal/g for children who are undernourished, but not severely malnourished. Again expressing this in terms of rice, we see that 1 pound of rice (parboiled) provides 1674 kcal (17) and thus, 1 kg of tissue requires an additional input of 3.3 kg of rice (at 98% caloric absorbtion).

These calculations suggest that if we look at the effect of infection on the weight of a child in a rice-eating community, it is immaterial whether we think of the weight loss as resulting from caloric depletion, a protein depletion, or a combination of the two. Thus, we may be able to use data both on caloric and nitrogen loss to get some estimate of the food loss.

Arguments over the primacy of calories or protein may not only be irrelevant when we look at the losses in terms of food, but exclusive attention to either of these obscures important interactions between the two nutrients. The work of Hegsted (9) and Miller and Mumford (20) suggests that:

Caloric requirement = f (caloric intake, protein intake, body weight)

$$\text{where } \frac{\partial CR}{\partial CI} > 0, \frac{\partial CR}{\partial PI} < 0, \frac{\partial CR}{\partial BW} > 0$$

Conversely, Scrimshaw (12) has pointed out that protein requirements are related to caloric intake (even when caloric intake exceeds caloric requirement) and has suggested that "the quantitative interrelation between protein and energy intakes must be better understood before recommended allowances for protein can be revised with any confidence."

## Some methodological issues

*Absorbtion studies*: Many data concerning malabsorbtion in human populations have been based on the D-xylose absorbtion test. A recent conference on malabsorbtion discussed the xylose malabsorbtion issue at some length. The sense of this discussion is exemplified by the comments of Baker (21) at this conference: "It is quite obvious from the studies performed that the degree of xylose malabsorbtion we have been discussing does not necessarily parallel malabsorbtion of other foods. I don't think you can say that because you have such-and-such a prevalence of xylose malabsorbtion, you also have such-and-such a prevalence of overall malabsorbtion of ordinary foods. After all, who eats xylose in the normal course of events?" While no published data giving individual absorbtion characteristics for xylose, protein, fat, and calories are available, it is apparent that the relationships are unlikely to be simple and well defined.

In analyzing data from absorbtion studies, it is necessary to specify the "norm" for absorbtion of the particular nutrient in a population. If Lindenbaum's (22) finding in Bangladesh, viz., that malabsorbtion affected the whole population and not just a small and unusual subgroup who are highly infected, is true for other situations, too, then we cannot determine the norm from any group within the population.

Davidson et al. (23) (using Atwater's data) report that about 92% of protein, 95% of fat, and 99% of carbohydrate are normally absorbed by young American men. Because the digestibility of different types of foods are quite different, data on the average Indian and American "food baskets" (24) are used in conjunction with the digestibility factors for different foods to estimate the average absorbtion of protein, fat, carbohydrate, and calories in a person who is absorbing "normally" and who is eating each of these diets (Table 1).

These calculations show that the actual diet consumed by the population should be taken into account when estimating the absorption norm, and suggest that empirical studies on groups from similar racial stocks living under different environmental conditions (such as, Indian villagers, on the one hand, and Indian

TABLE 1
Normal nutrient absorbtion on American and Indian diets

|  | American diet[a,b] | Indian diet[a] |
|---|---|---|
|  | % | |
| Protein | 92    91 | 79 |
| Fat | 95    95 | 93 |
| Carbohydrate | 99    97 | 97 |
| Total calories | 95.4 | 94.4 |

[a] Absorbtion data from Atwater (quoted from Reference 23).   Absorbtion data from Watt and Merrill (Reference 17).

TABLE 2
Normal nutrient losses on North American and Indian diets

|  | American diet | Indian diet |
|---|---|---|
|  | % | |
| Protein | 9.9 | 26.6 |
| Fat | 5.3 | 7.5 |
| Calories | 4.8 | 5.9 |

professionals and students living in the United States, on the other hand), but fed the same diet, are an essential component of the research required in this area.

When the nutritive value of the food intake is calculated using standard tables (17), then this value includes normal metabolic and digestive losses and thus the percentage of the "available intake" in the feces is: $(100 - \%$ digested$)/(\%$ digested$)$. The percentage of available intake expected in the fecal matter of Americans and Indians is given in Table 2.

Finally, we note a reservation expressed by Bayless (25) concerning the findings from fat absorbtion tests which is similar in nature to Baker's comment (21) that "no one eats xylose". Bayless has pointed out that estimates of fat malabsorbtion in populations on indigenous diets which are based on the loading test are likely to be misleading since the purpose of the test is to magnify differences between individuals and thus, the fat loads used are much greater than those encountered in the normal diet.

*Population-wide balance studies.* One of the only estimates of the percentage of food that is lost through malabsorbtion and infection is that given by Revelle (24, 26). The basis of Revelle's calculations is the assumption that the useful caloric intake of the population considered (in this case, East Pakistan, in the

1960's) must match the caloric requirements of the population since the individuals in the population are, literally, neither expanding nor contracting. On the basis of the 1965 Food and Agriculture Organization/World Health Organization caloric requirements (27), which are slightly higher than the 1973 requirements, Revelle estimates that the intake is about 2000 kcal per capita per day and the requirement only about 1800 per capita per day, and thus, the amount of food "malabsorbed" is about 10% of the intake. Although this approach is both instructive and seems more useful than virtually any of the alternatives, the computation involves estimating the difference between two numbers that cannot be specified with much precision, that are both large and that are not much different from each other, and ascribing a definiteness to the relatively small difference. We have carried out some crude calculations to estimate the standard error associated with this estimate of caloric malabsorbtion. We estimated the standard error in the measurement of caloric intakes from the two estimates, by the United States Department of Agriculture and the Interdepartmental Committee on Nutrition and National Defense (ICNND), which are given by Frisch and Revelle (28) for a number of Asian and Latin American countries; we assumed that the requirements are known with certainty (which they certainly are not); and we assumed that intakes and requirements are not correlated. This calculation suggests that the standard error of the estimate of caloric malabsorbtion may be about the same size as the mean of this estimate. While the figure of 10% that arises from this model is interesting, and may well be of the right order of magnitude, we should be cognizant of the fact that the data base on which it has been derived is quite crude and that considerable uncertainty attaches to the estimate. (We should note, too, that the figure emerging from an analysis of this sort does not, strictly speaking, measure food malabsorbtion alone, but measures losses during food preparation and total food wasted through inefficiencies in the body. This figure would include, in addition to subclinical and overt malabsorbtion, wastage due to the destruction of body tissue during febrile episodes.)

*Longitudinal and cross-sectional growth studies.* Cross-sectional studies have been useful in showing that certain types of diseases do not affect nutritional status. These studies have shown, for instance, that although children in poor countries may suffer from respiratory illness as much as one third of the time, these illnesses are not associated with malnutrition (29). Unfortunately, it is far more difficult to draw clear inferences from cross-sectional studies (2, 30, 31) that show correlations between the incidence of certain types of diseases and low weight or low weight gain. Interpretation of these results is difficult due to multicollinearity in the "explanatory" variables and due to problems in separating cause from effect. Multicollinearity is a problem in the ICNND study of East Pakistan (31), for instance. While the authors of this study focussed on the high correlation between nutritional status and the seasonal production of pulses, their data show an equally strong relationship between nutritional status and the seasonal incidence of diarrhea. Cross-sectional studies that show a correlation between infection and nutritional status do not elucidate the relative importance of poor nutritional status in increasing susceptibility and decreasing resistance to infection, on the one hand, and infection in decreasing food intake and the efficiency of food use, on the other. In numerous instances this correlation is observed and taken as evidence in support of the particular hypothesis that the investigator is advancing.

In a series of brilliant papers Mata and his colleagues (32–34) have presented longitudinal data on the relationships between infective episodes and nutritional status in individual Guatemalan children. The resulting plots give a graphic indication of the importance of infection as a determinant of nutritional status. In the analysis of the data emerging from these studies, however, much of the richness of the data and the qualitative description have been lost by pooling the data and examining correlations. As a result, we are not able to draw clear inferences on the effect of a single infection, or an infective regime, on food wastage. Mata's cross-section and time-series data could be pooled and the parameters of a simultaneous equation regression model estimated. Estimation of such a model is by no means a routine task; the data requirements are considerable and

the methodological issues complicated. The use of multivariate techniques is no panacea, but this analysis would be interesting heuristically and offers some possibilities for sorting out the relative strengths of the infection-nutrition and nutrition-infection relationships.

## Data on the effect of infection on nutrition

### The effect of subclinical infections

*Prevalence of subclinical malabsorbtion.* Studies in India (35), Thailand (36), Bangladesh (37), and Guatemala (14) confirm Bayless' (25) observation that "almost every study involving a general population in a tropical area has shown a prevalence rate (of D-xylose malabsorbtion) between 30% and 50%." Clinical examination of the villus height in the jejunum in these populations confirms that there is a morphological change in those who absorb D-xylose poorly (37). Lindenbaum (22) reports that in Bangladesh "small intestinal injury was occuring in the entire population rather than in a subgroup of abnormals" and Rosenberg et al. (38) indicate that "tropical enteropathy is found in virtually the entire low income adult population in tropical less-developed countries." In Guatemala, Schneider et al. (14) found the prevalence to be much lower (7 to 10%) in a group of healthy soldiers living under hygienic conditions than in the general population. There is little, if any, seasonal variation in xylose malabsorbtion in Bangladesh (37).

*Etiology of subclinical malabsorbtion.* The etiology of "tropical enteropathy," the subclinical malabsorbtion syndrome, remains unclear. Indications are that the cause is neither subclinical malnutrition nor a ubiquitious food toxin, but that is "(probably) related to exposure to a contaminated environment" (35), since "all areas in which this enteropathy occurs are those where there is a high contamination of the environment" (21). In his studies in Thailand, Keusch (36) found that all adults "have an abnormal flora in the jejunum" and suggests that this is "probably related to continuous environmental contamination". Lindenbaum et al. (37) have suggested the following mechanism whereby environmental contamination is related to subclinical malabsorbtion:

The high incidence of recurrent diarrheal episodes in Bengali infants and children as well as in Peace Corps Volunteers shortly after arrival in Pakistan may indicate that early in life, as a result of fecal contamination of food and water, there are repeated episodes of symptomatic small intestinal infection, such as the traveller's diarrhea syndrome that may be caused by certain strain of *E. coli*. Such mild enteric infections may be associated with profound impairment of absorbtive function. It is possible that, as Indians and Pakistanis grow to adulthood and are exposed repeatedly to such agents they no longer show clinical symptoms with each episode of infection, but the process of subclinical small bowel injury is perpetuated.

Similar inferences can be drawn from the INCAP studies in Guatemala (14) and the work of Einstein et al. (30) in Bangladesh, who showed that "xylose malabsorbtion (in children) was associated with growth retardation, increased incidence of severe weanling diarrhea, and increased incidence of live born sibling death."

These associations are not found in all of the studies that have investigated these relationships. In his study of Bengalee children, Harper (39), found that xylose malabsorbtion was not correlated with serious diarrheal episodes, amebiasis, giardiasis, or shigellosis, while Lindenbaum et al. (37) found xylose malabsorbtion to be uncorrelated with either diarrheal episodes or nutritional status.

In a careful recent review, Rosenberg et al. (38) weigh up the conflicting evidence and conclude that "the contribution of recurrent intestinal infections (to tropical enteropathy) is very likely".

*The quantitative importance of subclinical malabsorbtion.* The high prevalence of xylose malabsorbtion in populations of tropical areas coexists with a general, if unsubstantiated, belief that "it is likely that xylose malabsorbtion indicates nutrient malabsorbtion in the long run" (21). Subclinical malabsorbtion has thus been posited as an important source of food loss in these populations. Recent data from INCAP permit an estimate of the quantitative importance of this factor for the first time. Schneider et al. (14) briefly report the results of absorbtion studies on rural adult males, 30% of whom malabsorbed D-xylose. Individuals suffering from diarrhea were excluded from these studies. Because those who are recovering from diarrhea are included in the study population,

the effect of recent diarrhea on absorbtive capacity, that Schneider and his colleagues have postulated to be important, is included under the rubric of "subclinical malabsorbtion" in this study. "(These) absorbtion studies have shown that 40–51% of rural adult males have a decreased capacity to absorb nitrogen, calories and fat from their regular diet, losing through feces 200–300 kcal/day/individual above the average fecal excretion observed in a reference group of Guatemalans with normal absorbtion". The conclusion drawn from this data, "that subclinical malabsorbtion represents a serious health problem in rural Guatemala" (14), does not appear to be warranted.

We may expect the absorbtive capacities of the individuals to be distributed approximately symmetrically around the mean absorbtion for the population. Therefore, in the "control" population of normal xylose absorbers we would expect approximately 50% of the individuals to absorb less calories and nitrogen than the average for that population (and would expect approximately 50% of the individuals to absorb more calories and nitrogen than the average). The fact that 41 to 50% of the individuals in the population which absorbs D-xylose poorly absorb less calories and protein than the average absorbtion in the control population suggests that there is very little difference in the mean caloric and protein absorbtive capabilities of the two populations.

The interpretation of these data on the basis of previously available research would be as follows. The INCAP data indicate that two populations that have different D-xylose absorbtion characteristics show little difference in their abilities to absorb protein and calories. Studies on the D-xylose absorbtive capacity of individuals who move from the tropics to industrialized societies (37) show that these individuals change from poor to normal absorbtion within 6 months of residence in the more sanitary environment. The selection of "Guatemalan soldiers who had been living for the last two years under far better conditions of sanitation than those prevailing in rural areas" (14) as a control group, thus appears to be appropriate and consequently, subclinical malabsorbtion does not appear to be a significant nutritional problem in rural adult males in Guatemala.

The INCAP findings on the poor association between absorbtion as measured by D-xylose and caloric and protein absorbtion however, throws much of the accepted wisdom on absorption into doubt and, paradoxically, makes interpretation of their own data difficult.

Rosenberg (personal communication) has suggested that the capacity of the soldiers to absorb proteins and calories may not have improved in 2 years despite the changes in living conditions and despite their changed D-xylose absorbtion capacity. If this is so, the choice of the control group is inappropriate. The INCAP team have recognized this problem and are conducting absorbtion studies on Guatemalans and Americans living in California to determine the normal absorbtion of this diet. Before these results become available the normal absorbtion of calories and protein can be estimated by applying standard food absorbtion factors (17) to the diet fed to the study populations. A comparison of these values with the observed absorbtive capacity of the soldiers should help clarify this question.

While the INCAP study relates to the absorbtive capacity of adults, the focus of this study is on young children. Baker and Mathan (35) report the prevalence of xylose malabsorbtion to be highest in preschool children in South India. Keusch (personal communication), however, found the same degree of xylose malabsorbtion in children and adults in Thailand, and the preliminary observations of Schneider et al. (14) suggest that children and adults in the study population present similar gastrointestinal alterations. The findings on subclinical malabsorbtion in adults thus appear to be transferable to the under-5 age group.

The extremely tentative conclusion is that subclinical malabsorbtion in young children is not an important source of food wastage in poor countries. The conclusions are tentative since they rely exclusively on the preliminary results of one uncorroborated study, there remains a serious question about the control group that was assumed to be absorbing calories and protein normally, and the present interpretation of the results is quite different from that of the investigators. The uncer-

tainty notwithstanding, this estimate is our only, and therefore our best, estimate.

### The effect of clinical infections

*The effect on food intake.* Infection may interfere with food intake through loss of appetite and withholding of food (40). Some investigators, including Mata et al. (41), believe this mechanism to be crucial in the infection-nutrition relationship while others (H. Pollack, personal communication), discount this factor. There are few data available on the quantitative effect of these behavioral changes.

The practice of withholding food during an episode of diarrhea has been widely observed and almost universally condemned. Behavior in subsistence agrarian societies is certainly different from that which is the norm in Western industrial societies, but it is seldom capricious. A few investigators have outlined some reasons why foods are withheld from young children who are ill.

In the Punjab, "a mother, recognizing the increased incidence of diarrheal infection when supplemental foods are given, and recognizing the incidence of mortality from the infection, makes a conscious, seemingly sensible decision to withhold such foods" (42). Similar behavior has been observed among Indian mothers in rural Mexico. Recognizing that milk, eggs, and meat are excellent breeding grounds for pathogens, while substances such as gruel are not, these mothers were reluctant to give cow's milk to their babies (43).

These observations suggest that an improvement in environmental sanitation may be required for the successful implementation of nutrition programs that are designed to be implemented by the mothers of preschool children.

Mata et al. (41) have studied the effect of illness on the food intake of a cohort of 45 Guatemalan children during the first 3 years of life. While their analysis shows that food intake is negatively correlated with the prevalence of certain illnesses, they do not report regression coefficients and we are, thus, not able to estimate the effect of a particular type of illness on the reduction in food intake. Data on two children are presented in more

detail. In the first child, caloric intake fell by about 40% during an episode of diarrhea that lasted 11 days. In the second child, the effect of diarrhea was less clear. During an untreated 8-day episode, intake was reduced by 25%, but during a treated episode that lasted 7 days, reported food intake was normal. In this child, a series of overlapping illnesses (otitis externa, cellulitis, and bronchitis) lasting for 19 days, led to a reduction of about 50% in the intake of the child.

Elsewhere, Mata (44) gives data for preschool children who have whooping cough. These data indicate that there is a 9% decrease in intake during the 1st month of whooping cough, that this decrease declines to 7% in the 2nd month and that in the 3rd month the intake is increased by an average of 9% over the normal consumption. Because the average duration of whooping cough is 1 to 2 months (45), the increased intake during the 3rd month may well indicate increased intake during the recovery period. If this finding has general validity, there are important implications in assessing the effect of infection on nutritional status. As a result of delayed consumption, the child would fall below the weight profile for some time. While this growth pattern may have adverse health effects, these data suggest that the appropriate accounting procedure would not consider the decreased intake by the child during the course of an illness to be a loss to the child, but would inventory this food as consumption that is postponed until the recovery period. Far more data are required before such an assumption could be reasonably considered accepted or rejected.

Because the seriousness of this issue is alluded to in most discussions of infection and nutrition, it is somewhat surprising that these observations of three Guatemalan children provide the only published quantitative data on the effect of different infections on the food intake of children. A variety of perspectives suggests that a reduction in food intake is most important in the case of diarrhea.

The quantitative effect of metabolic losses due to fever appear to be greater than the losses due to malabsorbtion of nutrients during diarrhea, yet field studies consistently show that diarrheal diseases exert the greatest effect on nutritional status. On the basis of

such observations, it has been hypothesized (46) that reductions in food intake are more pronounced in the case of diarrhea than in the case of fevers or respiratory infections.

Later in this paper, we will present data on weight losses associated with sickness-days due to malaria and diarrhea in a group of Gambian children (47). The actual weight losses will be compared with the losses that would be expected from metabolic effects alone. This comparison suggests that, while there is no implied food reduction during malaria, about 75% of the weight loss during diarrhea can only be explained by changes in food intake. Children suffering from diarrhea appear to consume about 22% less food than their healthy counterparts.

Anthropological investigations support this hypothesis that the effect of diarrhea on intake is most important. In Bangladesh, for instance, restriction of nutrient intake during infantile diarrhea is believed to be widely practiced. "An infant suffering from loose motions is believed to have a 'hot' stomach, so it is given a diet of 'cool' foods—(such as) barley water or glucose water with a little lemon—which lessen the heat and allow the stool to harden." (S. Lindenbaum, unpublished observations).

A recent small sample study of Muslim mothers in rural Bangladesh by Ahmed, Chakraborty, and Briscoe suggests that, while notions regarding hot and cold foods are pervasive, there is no such a thing as a "standard practice" with regard to the nutritional intake of children with diarrhea. Our data show that the response of a child's appetite to an episode, as well as the range and quantities of food which are made available to the child during an episode of diarrhea, vary widely. Because food intake during diarrhea seems to be such an important factor in the infection-malnutrition relationship, and because an episode of diarrhea may affect the food intake of different children quite differently, it is plausible that the infection/food-intake factor may explain a substantial part of the variance in the health of individuals in an otherwise homogeneous group of children. For the small population which we studied, the average caloric intake of children was reduced by 30% and the average protein intake 35% during an episode of diarrhea. Food

availability was not increased during convalescence for these children.

In the next section, we present data that suggest that the prevalence of diarrhea in young children of poor countries is about 8%. Considering the Guatemalan, Gambian, and Bangladesh data, we assume that, on the average, the food intake of children suffering from diarrhea is reduced by 30%, implying a reduction of 2.4% in the average caloric intake of the group of children.

The effect of illness on children is highly dependent on exactly what happens to the food that is not consumed during an illness. If the unconsumed food is used to supplement the child's diet during recovery when appetite returns to supernormal levels, this "deferred consumption" represents no food loss to either the individual or the group of under five children. If the unconsumed food is consumed by other preschool children in the family, this redistribution within the group represents no loss to the group of under-5 as a whole. Because infection is unevenly distributed among these children (L.J. Mata, personal communication) the effects of such infection-related food redistribution on the individuals who suffer most from illness may be serious. If the food is consumed by family members other than preschool children, or if the saving is used for nonfood items, this reduction in intake is an absolute loss to the under-5 group. (The situation is, of course, symmetrical, with under-5s receiving some additional food input as a result of illness in other age groups. Since illness in older people is seldom accompanied by so great a reduction in food availability, and since the prevalence of illness is much lower in these other age groups, the quantity of this "return flow" to under-5s may be neglected.)

Intensive study of intrafamilial food consumption patterns is a high-priority nutrition research area. In designing such studies, it would be extremely useful to incorporate an investigation of the effect of infection on consumption habits.

*The effect of manifest infection on malabsorbtion.* The absorbtion of nutrients may also be affected by both clinical and subclinical infestation with intestinal helminths. Rao and Sen (48) quote a study done in West Bengal which shows that hookworm infesta-

tion of a mild nature interferes with the digestion and absorbtion of protein. Scrimshaw et al. (16) report a case where human adults with heavy hookworm infection had an average protein absorbtion of 62.5% compared with 73.3% in worm-free subjects on the same diet, and a case in which there was no difference in fecal nitrogen in West African children before and after treatment for ascariasis and hookworm disease. Young and Scrimshaw (49) quote an Indian study that showed that fecal nitrogen in children with moderately heavy ascariasis was reduced by half as a result of deworming.

Taylor and de Sweemer's (50) suggestion that the adverse effects on nitrogen balance of helminthic disease such as hookworm are roughly proportional to the parasite load is supported by animal studies in Cambridge (16) which showed fluctuations in protein absorbtion to parallel changes in worm burdens. In a study of the nutritional effects of hookworm in China, Crowley et al. (51) found the effect to be proportional to the hookworm load, while Tripathy et al. (52) drew similar inferences from their study of ascariasis in Colombia.

On the basis of Crowley's data (51), Briscoe (53) and Pollack (personal communication) independently concluded that each adult hookworm causes the loss of about 1 kcal/ day. Using Stoll's (54) estimate of the world average hookworm load of 100 worms (which is the load found in West Bengal in a careful study (55)) about 5% of human caloric intake is lost due to hookworm infestation alone. Given the high loads of other intestinal parasites, and the existence of other, apparently equally important, mechanisms relating infection to malnutrition, this effect of hookworm alone seems unreasonably high.

This unrealistic assessment of the effect of hookworm infestation that emerges from the Chinese data is reported here since it exemplifies problems that arise in drawing general conclusions from the results of isolated uncorroborated micro-level studies. The present analysis is undertaken in the belief that a synthesis of a large number of micro studies in conjunction with macro-level consistency checks is the appropriate approach in assessing the relative and cumulative effects of different components of the effect of infection on nutrition.

In their comprehensive review of the relationships between infection and nutrition Scrimshaw et al. (16) concluded that "the relative importance of (helminthic) infections in contributing to protein malnutrition is often overemphasized, for the simple reason that intestinal helminths are visible whereas other infectious agents can be recognized only through complicated technical procedures." In this analysis, we will follow an emerging concensus (27, 38, 56) and assume that the effect of manifest infections on food absorbtive capacity is insignificant unless the infection is accompanied by diarrhea. We therefore need estimates of the prevalence of diarrhea and the metabolic consequences of an attack of diarrhea to determine the importance of this factor for children under 5 years old.

In a study of a cohort of children from birth to age 2 years in rural Guatemala, Mata et al. (32) found a weekly diarrheal incidence of 6.4%. A comparison with other data (see Table 3) suggests that this figure represents the experience of a wide range of poor countries reasonably well. Mata's data indicate an average duration of an attack of diarrheal disease of 8 days, implying a prevalence of about 7% in this age group. A subsequent Guatemalan study (46) found the prevalence

TABLE 3
Weekly incidence of diarrhea
in young children

| Country | Age group of children | Weekly incidence of diarrhea |
|---|---|---|
| | | % |
| Sudan (57) | Preschool | 5.0–6.1 |
| United Arab Republic (57) | Preschool | 2.3–8.7 |
| Iran (57) | Preschool | 8.3–11.2 |
| East Pakistan (57) | Preschool | 4.6 |
| Venezuela (57) | Preschool | 9.0–9.9 |
| Colombia (Candelaria)(in (29)) | 0–35 mo | 7.5–8.1 |
| Gambia (rural) (in (29)) | 6–23 mo | 4.8 |
| South India (Vellore) (in (29)) | 6–11 mo | 14.0 |
| | 1 yr | 9.0 |
| North India (Punjab) (in (29)) | 0–5 yr | 9.4 |
| Indonesia (Jakarta) (in (29)) | 0–1 yr | 1.7 |
| | 1–2 yr | 1.2 |
| Indonesia (Yogjakarta) (in (29)) | 1–3 yr | 5.2 |
| Jamaica (in (29)) | 6–11 mo | 5.8–7.7 |
| | 1 yr | 3.8–5.8 |

of diarrhea in a group of low-income, rural children under 2 years old to be 10.0%, while for the under-5 group the diarrheal prevalence was 5.8%. Similar prevalence figures have been recorded in Africa and Asia. In Gambia, the prevalence of gastroenteritis, diagnosed on the basis of diarrhea, was found to be 14% in children under 3 years old (47), in the Punjab, the prevalence of diarrhea in a group of children under 3 years old was 7.4% (58).

These data thus yield consistent estimates of the incidence of diarrhea in preschool children. While the corresponding duration (and thus, prevalence) data are less rich, the diarrheal prevalence appears to average about 8% in this group.

Few data are available on the effect of diarrhea on the absorption of food. Scrimshaw et al. (16) report that, "even in severe gastroenteritis, such as typhoid fever, only a small part of the adverse nitrogen balance is apparently attributable to decreased absorption of nitrogen, (and) even with severe diarrhea the absorption of nitrogen rarely fell below 75%." Beisel's (59) observation that during an episode of diarrhea, stool nitrogen may double or triple accords with this figure, because this observation implies that the absorption of nitrogen on an American diet would drop from about 91% to between 73 and 82%. INCAP has recently published data on the malabsorption of calories and protein in children 3 to 5 years old suffering from mild to moderate diarrhea (14). In their preliminary report of these data, they do not report the food intake of these children. If we assume that the calorie and protein intakes were at the requirement level for this age group as specified by the 1973 Food and Agriculture Organization/World Health Organization standards, then the additional absorptive losses of 58 to 210 kcal and 25 to 175 mg of nitrogen per day translate into malabsorption of 3.5 to 12.5% of calories and 0.5 to 3.0% of protein.

Because about 8% of preschool children have diarrhea at any one time, and because these children appear to malabsorb about 8% of their caloric intake (and much less of the protein intake), overt malabsorption accounts for about 0.7% of the caloric intake of young children in tropical countries. Further data on the malabsorption of nutrients during

diarrhea are needed before this estimate can be considered reliable.

*The effects on body losses (catabolic losses).* "Clinically the most prominent metabolic response during a febrile infection is the wasting of body tissues ... The body seems able to sacrifice large quantities of muscle protein in order to provide precursor materials necessary for meeting both the anabolic requirements for key host defence responses and the heightened requirements of metabolizable energy" (5). The most important principles emerging from the pioneering work of Biesel and his colleagues are: that there is a stereotypical (i.e., not dependent on the etiological agent) catabolic response to infection (61); that fever is the major stimulus for initiating catabolic losses during an acute infectious episode (5); and that the magnitude of the catabolic losses during an acute infection is related to the severity (as measured by body temperature) and duration of the fever (5).

To determine the effect of catabolic losses on a population, we need estimates of the prevalence of fever and the average temperature in febrile individuals. In the rural Punjab, 4.2% of the children under 3 years old had fever at any one time (58). Martorell et al. (46) report a fever prevalence of 3.3% in children under 3 years old and 2.9% in under-5 in a poor Guatemalan community. Neither of these studies report the temperatures recorded during these fevers. In the village of Santa Maria Cauque, Mata et al. (41) found the average rectal temperature of febrile children under 3 years old to be about 38.6 C (101.5F).

1. Febrile losses evaluated in terms of nitrogen: Nitrogen losses during typhoid fever have been studied since the early part of this century (62). These studies have shown that "there is a two- to three-fold increase in excretion of nitrogen, mostly through urine, associated with typhoid fever" (16). Careful studies have also documented the relationship between fever and nitrogen losses for artificially induced tularemia, sandfly fever, and Q fever (61).

"If a normal person lowers or temporarily discontinues his intake of dietary protein, the body responds quickly by reducing urinary nitrogen losses. This compensatory response does not generally occur during febrile infections. Rather, urinary losses of nitrogenous

compounds such as urea, ammonia, creatinine and amino acids tend to be increased or maintained at close to normal rates" (5). Infections, thus, interfere with the mechanisms which enable the body to minimize nitrogen loss in undernutrition (50). "Despite severe protein malnutrition, infection-stimulated control mechanisms within the human or animal host continue to be potent enough to divert scarce amino acids from essential body needs into the synthesis of apparently non-specific serum glucoproteins" (63).

Although the consequence of an additional nitrogen loss due to infection is most serious in malnourished individuals, for a given infection the quantity of nitrogen lost is smaller in a malnourished than a wellnourished subject. Pollack and Halpern (64) explain this finding in terms of "two types of protein in the body: a) a protein in equilibrium with the nitrogenous pool at a very slow velocity and b) a protein in dynamic equilibrium with this pool at a comparatively high velocity. Undernourished individuals who fail to respond to stress with increased nitrogen excretion still have large quantities of muscle protein. This muscle protein is not available rapidly enough for injury stimulus response. The location of the labile protein pool is presumably in the liver, kidneys, pancreas, circulating blood, and to some extent in the extracellular fluids of the large striated muscle masses. . . . The concept of a body protein source which is only slowly available and of a separate source of labile, readily metabolizable protein, may be consistent with experimental observations. This labile protein is synthesized by well nourished people and may be proportional to the level of protein intake".

The validity of this theory of a "labile nitrogen reserve" in the body remains unclear (W. R. Beisel, personal communication).

2. Febrile losses evaluated in terms of calories: Although little quantitative information is available on the effect of infections on fats and carbohydrates (16) or on total caloric loss (50), it is well known that there is an increased expenditure of metabolizable fuels to meet body energy requirements during the infectious process and especially during fever (63).

An interesting analysis of the importance of disease in the world food problem by Pollack and Sheldon (62), draws on clinical data on the increased metabolic requirements arising from infections with tuberculosis, malaria, and typhoid. Pollack and Sheldon's (62) interpretation of these data is presented in Table 4.

The method followed in deriving these estimates will be discussed in some detail here. Shaffer and Coleman (65) observed typhoid patients who were receiving an average of 33 kcal/kg per day and 12 g nitrogen per day and measured net nitrogen losses of 6.5 g/day. Pollack (62) found that "extrapolating such losses to the usual 20 day course of the disease would give total losses approximating 130 grams of body nitrogen, roughly equivalent to 4 kg of body mass." (The implicit factor for the conversion of nitrogen to body mass, 3.2 kg of tissue = 100 g of nitrogen, is the same as that given by Scrimshaw et al. (16).) Pollack then assumes that it takes 7700 kcal to replace 1 kg of body mass, a figure discussed earlier in this paper, and thus, determines a "caloric equivalent" to the amount of body mass lost during the infection.

TABLE 4
Metabolic requirements of disease

| Disease | Increase in metabolic requirements | | |
|---------|------------------|-----------------|----------------------------------|
|         | Acute phase (duration) | Procromal or postcromal phase (duration) | Increment in basic annual caloric requirements/case |
|         |                  |                 | % |
| Tuberculosis (respiratory) | + 22% (3 mo) | 7% (9 mo) (10 kg (22 lb) weight loss) | 11.0 / 8.8    19.8 |
| Malaria | + 18% (2.5 mo) | 5 kg (11 lb) weight loss | 3.8 / 4.4    8.2 |
| Dysentery | + 40% (20 days) | 4 kg (8.8 lb) weight loss | 2.2 / 3.5    5.7 |

In 1915, Coleman and du Bois (66) reported metabolic rate increases during the acute phases of typhoid fever. These observations provide the basis for the 40% increase in metabolic requirements during the acute phase. On the assumption of a basal metabolic requirement, Pollack was able to determine the caloric equivalent of this metabolic increase.

In determining the total caloric cost of an episode of typhoid (or dysentery), Pollack (62) assumes that the increased nitrogen excretion is the result of protein breakdown whereas the increased caloric expenditure comes from carbohydrate and fat stores in the body, and, thus, adds the caloric equivalent of the excess nitrogen loss and the increased metabolic requirement to estimate the total caloric loss.

Recent work by Beisel (5), however, suggests that at least some of the excess calories derive from muscle protein and not from carbohydrate stores in the body: "In the face of the increased energy demands of acute infection or severe trauma, amino acid utilization serves the immediate need despite its potentially wasteful consequences. An increased flux of gluconeogenic amino acids such as alanine from muscle-to-plasma-to-liver supports this activity" (5). Bogert et al. (67) have described what happens when protein is used as an energy source: "Amino (NH₂) groups are split off from the constituent amino acids and formed into simple nitrogen-containing substances (chiefly urea) that are excreted by the kidneys. The nonnitrogenous fragments of amino acids, the carbon chains, are then oxidized in the same way that carbohydrates and fats are metabolized."

Pollack's (62) method appears to overestimate the caloric cost of an acute illness, because that muscle protein which is going to gluconeogenesis is being counted twice. The degree of overestimation of the caloric cost of an acute illness is dependent on the relative caloric contributions of muscle protein and body carbohydrate and fat stores. We turn to the studies of Beisel et al. (61) for an indirect estimate of the relative importance of protein gluconeogenesis.

The studies of Beisel et al. (61) show total negative nitrogen balances of 50, 15, and 35

g of nitrogen in artificially-induced tularemia, sandfly fever, and Q fever. Because 100 g of nitrogen loss correspond to 3.2 kg of muscular tissue, the implied average muscle losses are: tularemia, 3.5 pounds; sandfly fever, 1.1 pounds; and Q fever, 2.6 pounds. The corresponding body weight loss of 5.9, 3.2, and 4.6 pounds imply that 60, 35, and 56% of the loss is due to protein destruction. Because no dehydration took place, we estimate about 50% of the body mass loss is due to protein destruction and about 50% to the destruction of adipose tissue. The implication is that Pollack's method would overestimate the caloric cost of the disease by about 50% in the cases examined by Beisel et al. (61).

In using the above data, it is also important to note that no allowance has been made for the possibility that, although there is an increase in the basal metabolic rate, there may be a fall in energy requirements for activity. When computing caloric losses from data of this sort, this reduction in caloric requirements for activity should be accounted for as a "negative loss" during the fever.

In their examination of the caloric costs of disease in the early part of this century, Barr, Cecil, and du Bois (cited in Reference 62) found a stereotypical response to the severity and duration of fever which is remarkably similar to the more recent finding of Beisel et al. (61) with respect to nitrogen losses. "Barr and his associates calculated the relationship of basal metabolism to temperature in six patients with fevers of differing origin: typhoid, pulmonary tuberculosis, erysipelas, arthritis, malaria and intravenously administered vaccines. These data indicated.... that the metabolic rate increases approximately 7.2% for each degree (Fahrenheit) of fever, a statistic which.... has surprisingly little dispersion ... and which appears in quite good agreement with other independent observations" (62). This is known as "the duBois formula" (62).

In Table 5, we have calculated the equivalent caloric costs of the diseases investigated by Beisel et al. (61), on the assumption that 1 kg of body mass is equivalent to 7700 kcal and that basal metabolism accounts for 2000 kcal daily. We see that these values are remarkably similar to those which may be expected by application of the du Bois formula.

TABLE 5
Metabolic increase due to fever

| Infection | Weight loss | Fever index | Implied metabolic increase for one degree of fever |
|-----------|-------------|-------------|----------------------------------------------------|
| | *lbs* | *F days* | *%* |
| Tularemia | 5.9 | 156 | 6.6 |
| Sandfly fever | 3.2 | 111 | 5.0 |
| Q fever | 4.6 | 99 | 8.2 |

Roe and Kinney (68) have found that "surgical operations, burns, severe sepsis and loss of functional tissue mass can effect changes in the rate of both heat production and heat loss independently of temperature change" and conclude that the du Bois formula is of limited utility in the treatment of patients suffering from these conditions. "In fever uncomplicated by (these severe traumas, however) the temperature is 'reset' by the hypothalamic thermoregulatory mechanism, and after a period of equilibrium (sic), heat production and heat loss are balanced again at a higher rate of expenditure. Under these circumstances, a rise in temperature of 1°F brings about an increase in energy expenditure of 7% of the basal value" (68). Use of the du Bois formula therefore appears to be appropriate in this analysis.

We now have estimates of all elements which are required for estimating catabolic losses in young children. The prevalence of fever in this group is about 4%, with the average temperature elevation during fever being about 3 F (1.67 C). Thus about 0.9% of calories ingested by preschool children are accounted for by catabolic losses. The components of this estimate are well established and thus the estimate is reliable for the areas studied. In areas where malaria is important, the effect of this factor would be substantially increased because a malaria prevalence of 2.5% implies a 1% caloric loss.

*The effect on "functional wastages."* "In addition to negative balances during infection, many nutrients are wasted within the body by pathophysiological processes which may lead to an excessive utilization of these substances, their sequestration in relatively inaccessible body pools, or their diversion from the usual pathways of metabolism. In contrast to the stereotyped patterns of absolute catabolic wastage, functional forms of wastage appear to vary in pattern and severity with different forms of infections . . . " (63).

Beisel (personal communication) has suggested that these functional wastages may be neglected for the purposes of this quantitative analysis.

*The effect of infection on overall weight changes.* Longitudinal data from Africa (47), Central America (33), and Asia (58) have shown clear causal relationships between episodes of infection and nutritional status.

Numerous other field studies have reported an association between the morbidity history and nutritional status of children. For example, in the Oxford Child Health Survey, "growth retardation in children is directly proportional to the amount of morbidity" (50); in Latin America, "clinical records show that nutritional deficiency resulted from repeated episodes of diarrheal diseases" (4); in Indonesia, children growing at rates slower than the Harvard standards had 50% higher illness rates than those children who grew faster (29); in Guatemala, there are more episodes of illness in children with lower growth (69) and in Columbia, similar results have been reported (2). The considerable difficulties which arise in attempting to separate cause from effect are discussed earlier in this paper.

A recent study of infection and nutrition in the Gambia (47) uses multiple regression techniques to evaluate the quantitative relationship between the prevalence of different categories of disease and weight and height gain of 152 children between 0.6 and 3 years old. Of the nine disease categories (upper respiratory tract infections, lower respiratory tract infections, gastroenteritis, infectious fever, malaria, giardiasis, superficial infections, deep infections, and nonspecific disorders) only gastroenteritis and malaria showed significant negative relationships with weight gain in the children. With weight gain measured in grams per month and disease prevalences being used as the independent variables, the regression coefficients for gastroenteritis and malaria were − 746 and − 1072, respectively.

On the basis of our estimates we would expect an undernourished, but not severely malnourished, population with a mean intake of 1000 kcal per capita per day and suffering from diarrhea throughout a month to malabsorb 2400 kcal and, thus, to grow about 200 g less than a comparable population with-

out diarrhea. Similar calculations for malaria show the 40% increase in metabolic requirement (62) to translate into a weight differential of 1000 g/month between populations having malaria every day and those not having any malaria.

These calculations suggest that the regression coefficient for malaria can be accounted for almost entirely by the catabolic losses accompanying the fever, whereas the absorptive losses during diarrhea account for only about one-fourth of the weight loss resulting from diarrhea. This finding is not unexpected, because it has been hypothesized (46) that anorexia and food withholding are particularly marked in diarrheal episodes. Assuming, as we implicitly have throughout these calculations, that activity remains constant, the implied reduction in food intake to account for a loss of 546 g/month is 220 kcal/day or 22% of the usual intake of the child.

## The effect of mortality

The process of human reproduction in poor countries is nutritionally inefficient because the high infant and child mortality rates mean that substantial quantities of nutrients are consumed by children who do not survive childhood. In this section, we estimate these indirect nutrient costs under the different mortality schedules prevailing in typical communities in a poor country (Bangladesh), a middle income area (Latin America), and a wealthy country (United States). Our method is to follow a synthetic cohort from conception to age 5 years. The caloric intake of those who live to the age of 5 is determined and the additional intake required to feed children who do not survive to this age estimated. The nutritional efficiency of the reproductive process in the three cases will be compared and the role of infection in explaining the differences assessed. Because economists are interested in those who can become productive adults, the efficiences of producing 15-year-olds will also be assessed.

*1. Fetal wastage data.* Studies of fetal wastage are few and are plagued by methodological problems. In the most careful study to date, French and Bierman (70) report a fetal wastage rate of 237/1000 live births in a high-income Hawaiian community. In the rural Punjab, Potter et al. (71) estimated a rate of 136/1000 live births. This appears to be a considerable underestimate. During the first 4 months of pregnancy, when accurate data are most difficult to obtain, the Punjab rates were only ⅓ of those found in Hawaii, whereas in the latter half of pregnancy, the Punjab rates were 3 times the Hawaiian rates.

Because "combined malnutrition and infection can produce synergistic negative effects on fetal growth" (60), in poorer communities, a higher proportion of pregnancies probably end in fetal death. Sheps (72) has suggested that "while we may estimate the probability of spontaneous fetal death as 24–30% of recognizable pregnancies in relatively healthy populations, for populations living under poorer health conditions this probability may be of even greater magnitude, such as 35% or perhaps even as high as 50% (and that) the reduction in fetal wastage (under improved economic conditions) would probably apply to fetal deaths occurring relatively late in pregnancy more than to early deaths".

In this analysis, we use the Hawaiian data for the United States cohort, assuming a fetal wastage rate of 237/1000 live births with a mean gestation of fetal death being 10.3 weeks (70). For Bangladesh, we assume a fetal wastage of 450/1000 live births with a mean gestation of 15 weeks. The sensitivity of the results of these assumptions will be tested.

*2. Infant and child mortality data.* Mortality in young children has been intensively studied in the three communities chosen. Matlab Thana in rural Bangladesh is the field study area for the Cholera Research Laboratory (73); Monterrey, Mexico, and suburban San Francisco, United States, were studied during the 1969/1971 Pan American Health Organization (PAHO) Study of Mortality in Childhood (4). Matlab Thana and San Francisco represent extremes of high and low mortality whereas Monterrey was chosen as the community whose childhood mortality rates most closely represented the median rates for the Central and South American communities studied by PAHO (Table 6).

*The nutrient costs of different mortality schedules.* From these data, life tables are constructed to represent the experience of a synthetic cohort from conception to age 15 years. For each period the number of person-years lived by the cohort is broken down into the number of person-years lived by those

TABLE 6
Childhood mortality data (1970)

| | Matlab Thana, Bangladesh | Monterrey, Mexico | San Francisco, United States |
|---|---|---|---|
| Infant mortality rate (per 1000 live births) | 117.0 | 60.7 | 17.2 |
| Neonatal | 79.0 | 26.0 | 12.7 |
| Postneonatal | 38.0 | 34.8 | 4.5 |
| Death rates/1000 persons per yr | | | |
| 1–4 yr | 27.7 | 4.3 | 0.7 |
| 5–14 yr | 3.0 | 0.5[a] | 0.4[a] |

[a] Extrapolated on the basis of the appropriate regional model life table (74).

who survive past age 15 and the number lived by those who die in each of the periods before the survivors of the cohort reach the age 15. The person-years are converted into caloric "intakes" by assuming an additional intake of 200 kcal/day during pregnancy (75) and intakes of 950, 1050, 1375, and 1940 kcal per capita per day for the other age groups. The latter are the estimated intakes of Bangladeshis during the early 1960's (76). The cumulative caloric consumption of those who live and those who have died at different ages are determined. These calculations are presented for the three cohorts in Appendix Tables 1 to 3 and the results summarized on Table 7.

*The role of infection in mortality and the consequent nutrient losses.* Because the purpose of this paper is to assess the effect of infection on nutrient loss, the contribution of infection to the nutritional cost of mortality needs to be established. While it is not possible to do this with any precision, the contribution of infection to fetal, infant, and child mortality in the three populations will be discussed.

*1. Fetal wastage.* Assuming a fetal death rate of 45% in Bangladesh, fetal attrition increases the caloric intake required by the cohort in the first 5 years by 1.1%, accounting for 15% of the caloric cost of under-5 mortality. In the United States cohort, fetal wastage is absolutely less and relatively more important: fetal attrition increases the caloric intake required by the cohort up to the age of 5 years by 0.24% accounting for 51% of the caloric cost of under-5 mortality.

The hypothesized differences in fetal wastage in the Bangladeshi and United States

populations are a consequence of differential quality of obstetrical care (72) and differential malnutrition and infection rates during pregnancy.

Data on the effect of malnutrition on fetal survival come largely from studies of acute starvation. Prenatal exposure to famine in early gestation appeared to produce a rise in stillbirth ratios in the Dutch famine during the winter of 1944 to 1945, but no such association was found as a consequence of famine exposure in late gestation (77). In Leningrad in 1942, where famine continued for much longer, fetal wastage was doubled (78). No increase in still births was observed during the 1975 famine in Bangladesh (73).

Maternal infections are more common in poor communities and probably contribute to the high frequencies of congenital infections and intrauterine growth retardation recorded in such communities (41) and to fetal wastage.

*2. Infant mortality.* Elimination of all infant deaths in Matlab Thana would reduce the excess caloric intake in the birth-to-5-year period from 5.77 to 4.94%. For Monterrey, the comparable reduction would be from 1.04 to 0.77%.

*3. Neonatal mortality.* The neonatal death rate in Matlab Thana is 3 times that of Monterrey and more than twice that of the highest mortality areas (such as rural El Salvador) studied by PAHO. Preliminary analysis of data from administration of diptheria-tetanus toxoid to women of reproductive age in Matlab Thana indicates that about 50% of neonatal deaths are due to tetanus. Tetanus is relatively unimportant in Latin America where neonatal deaths are largely due to congenital abnormalities, complications of

TABLE 7
The ratio of food intake by those who die to food intake of those who live

| | 5 Yr | 15 Yr |
|---|---|---|
| | % | |
| Bangladesh (Matlab Thana) | 7.43 (5.76)[a] | 3.46 (3.00)[a] |
| Latin America (Monterrey) | (1.04)[a] | (0.54)[a] |
| United States (San Francisco) | 0.46 (0.18)[a] | 0.32 (0.25)[a] |

[a] Calculated with the prenatal period excluded.

pregnancy, difficult labor and birth injury, and anoxic and hypoxic conditions. Infective and parasitic diseases, pneumonia, and influenza directly accounted for about ¼ of neonatal deaths in the PAHO study areas. In San Francisco, the neonatal death rate is about ½ of the Monterrey rate, but the relative contributions of the above broadly-defined groups of causes are about the same.

The effective administration of tetanus vaccine in Bangladesh would thus halve the neonatal mortality rate, bringing it to the levels found in Latin America. Whereas "the context of neonatal mortality is highly complex, involving the action of multiple factors that may go back into pregnancy or even earlier (including) the health status of the mother before and during pregnancy" (4) other infectious disease control programs would probably have little effect because neonatal deaths are "largely congenital, developmental and obstetrical" (79).

*4. Postneonatal mortality.* The relatively low postneonatal death rate in Matlab Thana (the rate in Matlab Thana is less than ½ that of rural El Salvador and similar to that of the much higher income community of Monterrey) is probably largely due to the universal practice of prolonged breast-feeding in Bangladesh. In Monterrey and El Salvador only about ½ of the children are breast-fed for 1 month or longer.

In the PAHO investigations, both "underlying" and "associated" causes of death are reported. An underlying cause is "the disease or injury which initiated the train of morbid events leading directly to death". Associated causes include "any other significant condition which unfavourably influenced the course of the morbid process and thus contributed to the fatal outcome, but which was not related to the disease or condition directly causing death (and) those morbid conditions that are usually included in the chain triggered by the underlying cause" (4).

The PAHO data (Table 8) are used to assess the relative importance of infections as a cause of death and to assess the role of malnutrition in predisposing the children to the final cause of death.

In both Monterrey and Matlab Thana, reductions in infectious diseases, and particularly diarrhea, would substantially reduce postneonatal mortality. Because reduction in

TABLE 8
Mortality in Monterrey and San Francisco

| Rates/1000/yr | Monterrey | San Francisco |
|---|---|---|
| Post neonatal mortality | 34.80 | 4.50 |
| Infective and parasitic diseases (diarrhea) | 21.55 (15.47) | 0.55 (0.09) |
| Pneumonia and influenza | 5.21 | 1.00 |
| Other respiratory diseases | 1.74 | 0.09 |
| Percentage of deaths in which malnutrition was an associated cause | 49 | 9 |

these death rates has been slow in relatively affluent Latin America, and because malnutrition is an even more important constraint in Bangladesh, the prognosis for substantially reducing infections in this group in Bangladesh is poor.

*5. Mortality in young childhood.* Elimination of all deaths in the 1-to-5 age group in Matlab Thana would reduce the excess caloric intake in the birth-to-5-year period from 5.77 to 0.82%. For Monterrey, the comparable reduction would be from 1.04 to 0.26%.

In our examination of infant mortality in Bangladesh, we found that the postneonatal rates were relatively low for such a poor community. This phenomenon continues into the second year of life. "(In developing countries deaths) of the second year ordinarily account for 50% of those in the four years and sometimes as much as 70%" (79); in Bangladesh, the 1-year-olds account for only ⅓ of the mortality of the 1-to-5 group (80). Diarrhea and other gastrointestinal diseases accounted for 27% and measles 10% of the deaths in this group in Matlab Thana in 1968 to 1969 (73). The importance of malnutrition as an associated cause of death was graphically illustrated during the famine of 1971, when death rates in this age group rose by about 50% with acute gastrointestinal diseases responsible for most of the increase.

Comparisons between Latin American communities and Bangladesh are difficult because of the difference in the type of mortality data available. Some insights into the role of infections in different mortality regimes may be gained by comparing the PAHO mortality data for rural El Salvador, an area with mortality rates comparable to those in Matlab Thana, with the data for Monterrey and suburban San Francisco (Table 9).

In both El Salvador and Monterrey we see

TABLE 9
Cause of death in El Salvador,
Monterrey and San Francisco

| Rates/1000/yr | Rural El Salvador | Monterrey | San Francisco |
|---|---|---|---|
| 1–5 yr mortality | 26.22 | 4.30 | 0.70 |
| Infective and parasitic diseases | 17.82 | 2.50 | 0.03 |
| (Diarrhea) | (13.77) | (0.65) | (0.00) |
| (Measles) | (2.09) | (1.03) | (0.00) |
| Pneumonia and influenza | 0.84 | 0.39 | 0.02 |
| Other respiratory diseases | 0.65 | 0.30 | 0.00 |
| Percentage deaths in which malnutrition was an associated cause | 54 | 52 | 22 |

the familiar patterns with death in the 2nd year accounting for over 50% of the deaths in the 4-year period. Diarrhea accounts for over ½ of the deaths in El Salvador and 15% of the deaths in Monterrey. Measles, pneumonia, influenza, and other respiratory diseases account for twice as many deaths in El Salvador as in Monterrey, but the relative importance of these causes is greater in Monterrey. Malnutrition is associated with over half of the deaths in both communities.

As expected, the death pattern in San Francisco is quite different. Deaths are more uniformly distributed across the age group, infectious diseases account for less than 7% of the deaths, and malnutrition is implicated in less than ¼ of the deaths.

Thus, while the pattern of mortality in this age group is quite variable, there is no doubt about the fundamental role of acute infectious diseases and malnutrition.

*6. Summary.* Relative to Monterrey, Mexico, the nutritional efficiency of human reproduction in Matlab Thana, Bangladesh is very low. The major part of this difference can be attributed to the higher toll taken by infectious diseases and malnutrition in a low-income, high-mortality community.

Rohde (29) has presented an interesting method for estimating the effect of malnutrition on diarrhea rates. Using data on the relative risk of diarrhea attacks with nutritional status, he assumed that, in the absence of malnutrition, the attack rates of all groups would have been those of the well-nourished groups. On this basis, he computed the number of cases of diarrhea attributable to malnutrition.

An effort was made to use Matlab Thana

data on death rates in groups whose nutritional status was different (81) to derive a similar estimate for the mortality attributable to malnutrition. This effort ran into two problems. The first was that undernutrition is so widespread in rural Bangladesh that no data were available on a group which would be considered well nourished by international standards. The second problem is that children who have a high susceptibility to disease for reasons other than nutritional deficiencies would tend to fall into the lower nutritional category as a result of the effect of infection on nutrition. The assumption that all of the excess diarrhea (or death) in the community may be attributed to nutritional deficiencies may be inappropriate.

An examination of the data presented here suggests that if we were able to separate out the intimately-connected effects of infection and malnutrition on death in young children in Bangladesh, then a hypothetical eradication of all infections may reduce mortality by about 50% in the neonatal period (due primarily to elimination of tetanus) and by about 50% in the postneonatal and 1 to 5 age group (primarily due to eradication of diarrhea). A hypothetical eradication of infections in young Bangladeshi children may thus approximately halve the nutritional inefficiency due to premature mortality.

Turning to our "intermediate" and "rich" populations, mortality rates among infants and among children between 1 and 5 years old are 3 times and 6 times higher in Monterrey than San Francisco. In nutritional terms, however, the reproductive efficiency of both populations is high (see Table 7) and the effect of substantially reducing the differential mortality would not be great.

*An estimate of the overall effect of infection on food utilization by young children in a very poor country like Bangladesh*

On Table 10, the estimates of the various components of food loss are used to estimate the effect of an hypothetical eradication of all infections on the efficiency with which Bangladeshi children use food available to them. This table shows that 9% of the food available to the cohort is not used for the maintenance, growth, or activity of those who survive to age 5. Because deaths due to causes other than infections (particularly malnutrition)

TABLE 10
Effective food use by young children in Bangladesh

| | Under existing conditions | | With the (hypothetical) elimination of all infections |
|---|---|---|---|
| Food available to a cohort of children from birth to age 5 | | 100.00 | 100.00 |
| Food that is consumed by other age groups because of anorexia and food withdrawal associated with diarrhea in young children | $(30\% \times 8\% \times 100) =$ | $2.40^b$ | 0.00 |
| Food that is consumed by those who die before they reach age 5 | $(100 - 2.4) \times \dfrac{5.76}{105.76} =$ | $5.31^a$   $100.0 \times \dfrac{2.88}{102.88} =$ | $2.80^b$ |
| Food lost due to subclinical malabsorption | | $0.00^c$ | 0.00 |
| Food lost due to malabsorbtion associated with diarrhea in those who reach age 5 | $(100 - 30.0 - 5.31) \times 0.007 =$ | $0.45^c$ | 0.00 |
| Food not used for maintenance, growth, or activity by those who reach age 5 because of catabolic effects | $(100 - 2.40 - 5.31) \times 0.009 =$ | $0.83^a$ | 0.00 |
| Therefore, % of available food that is used by those who survive to age 5 for growth, maintenance, and activity | | 91.01 | 97.20 |

[a] High confidence associated with this estimate.  [b] Fair confidence associated with this estimate.  [c] Low confidence associated with this estimate.

would still occur if all infections were eliminated, in such a hypothetical situation nutritional inefficiency of the population would not be absent. The amount of food that would not be used effectively may be reduced to about 3% if all infections were eradicated.

## Policy implications of the interaction of infection and nutrition

Recognition of the interactions of infection and nutrition has given rise to a concern for dealing with health and nutrition problems in a holistic manner (13, 44, 82–84). Two major field studies have not yielded clear answers concerning the relative effectiveness of combined versus separate programs. In the Guatemalan study (85), the results of the feeding program were inconclusive, the integrated health care program was difficult to evaluate and the evidence on the interaction of infection and nutrition was weak or non-existent. The Narangwal study in India (58), showed that the effect of nutrition and infection care programs on reductions in infant mortality were not synergistic, but that combined programs may be appropriate because the unit costs of services were lower when services were combined. Our analysis does not provide a basis for assessing the relative merits of combined versus separate programs, but provides a quantitative measure of the potential effect of health improvements on nutrition and indicates which infection interventions may have the greatest nutritional impact.

Although confirming the widely-held belief that infections contribute substantially to malnutrition in children in Third World countries, our estimates suggest that the importance of some factors has been assessed incorrectly. Research has focussed largely on the metabolic effects of infection and has led policy-makers to assume that the inefficient use of food energy and nutrients is solely due to absorptive and catabolic effects (86). Key planners in two of the largest international agencies have suggested that malabsorption may account for food losses of 20 to 25% (86) and 30% (A. Berg, personal communication). These figures vastly exaggerate the importance of this particular component. A striking implication of Table 10 is that health programs that reduce anorexia and food withdrawal and affect mortality will have the greatest nutritional impact.

### The nutritional consequences of some public health programs

*Vaccinations:* These may play an important role despite the fact that there are no vaccinations available for "the conditions that mostly damage children (which) are gastroen-

teritis, respiratory disease, dyspepsia, worms and maternal inadequacy" (87). Administration of a tetanus vaccine in Bangladesh would reduce neonatal mortality by 50%, but would have little effect on the nutritional efficiency of the population because these deaths occur in such young children. The effects of a measles vaccine would be more impressive. The direct food losses from measles which are probably largely due to food withdrawal, account for between 0.19 and 0.45% of the food intake of those who survive to 5 years of age[4]. In Bangladesh, the caloric intake per survivor to age 5 in the cohort is increased by 0.6% as a consequence of measles deaths[5]. For the higher death rates from measles reported from Africa, this figure would rise to over 2%[6]. Furthermore, the prevention of measles plays a role in preventing the familiar infection-malnutrition spiral: "Anergy following measles is the classical example resulting in a temporary depression in cell-mediated immunity and increased susceptibility to tuberculosis and perhaps other bacterial infections" (29). The future diseases and associated nutritional losses which are averted should be included in determining the benefits of the measles vaccine. This analysis strongly supports Morley's (1) contention that a measles vaccination is among the most important of public health measures now available to improve the health of young children.

Recent advances in the understanding of viral diarrheas (90) give hope that a vaccination may be available against these diseases. Whereas field data are being collected for the first time at present, it seems likely that these agents will prove to be responsible for a substantial portion of diarrheas. The health and nutrition benefit of such a vaccine would be enormous.

*Water supply and sanitation*: Environmental changes apparently have been more important than medical advances in reducing mortality in 18th century Europe (91) and in developing countries in the 20th centruy (92). Because health changes emanating from improvements in water supply and sanitation affect most of the spectrum of infections that influence nutritional status, these are attractive options in attempting to reduce malnutrition through the control of infections.

Understanding of the health effects of both water supply and sanitation programs is ru-

dimentary because of our limited knowledge of behavior, etiology, and epidemiology. Because more research has been done on the health effects of water supply programs and because these programs are receiving high priority in most countries, we will discuss the nutritional effects of water supply programs only here.

To assess the nutritional impact of an improved water supply, we need several pieces of information: we need to know the relationship between provision of the infrastructure (e.g., a hand-pumped tubewell) and use of that facility by different groups in the community; we need to know the relationship between use of the facility (in terms of both

---

[4] African children have been shown to lose an average of 7% of their body weight as a result of an attack of measles (1), while Guatemalan children under 2 years old lose 3% of their body weight (32). The median age of attack by measles in poor countries is about 2 years (1), an age at which the weight of a typical group of African children was 10 kg (1). Few unimmunized children escape measles (1). Children who are undernourished, but not severely malnourished need about 12 kcal to gain 1 g of body weight (19). Using Morley's (1) measles data, these children would, thus, require about 8400 additional kcal after the episode to regain their previous weight. Using data on food intakes in Bangladesh in 1962 to 1964 (76) and the age structure in India in 1961 (88), the average food intake by the under-5 population is 1027 kcal/day, and the average food consumption by a child before his 5th birthday is about 1.9 million kcal. The upper bound on the estimate of the direct food lost as a result of measles is thus 0.44%, using Morley's (1) data, or 0.19%, using Mata's (32) data, of the food consumption of the under-5 group. Given that fasting children lose from 1 to 2% of their body weight per day (29) and that children with measles are put onto a restricted diet in many poor countries (1), most of the weight change is probably not due to food "wastage", but due to the withdrawal of food.

[5] In Bangladesh in 1967/68, 1.8% of deaths under age 1 and 9.0% of 1 to 4 year old deaths were due to measles (89). Using the usual life table and food consumption data and assuming that, on the average, these deaths occur at 6 months and 3 years respectively, the group who dies from measles consumes: $(0.018 \times 117 \times 950 \times 365/2) + (0.09 \times 90.9 \times ((1 \times 950 \times 365) + (2 \times 1050 \times 365))) = 9.47 \times 10^6$ kcal. Because the total intake of the survivors in the life table is $792.1 \times 1.9 \times 10^6$ kcal, the caloric intake of the cohort is increased by 0.63% due to measles deaths.

[6] African data (1) show a death rate of 5% due to measles. Using the Bangladesh life table and an average age of death of 2 years, the intake of those who die from measles is:
$0.05 \times 883 \times (950 + 1050) \times 365 = 32.2 \times 10^6$ kcals, or over 2.1% of the caloric intake of those who survive to age 5.

quantity and quality of water used) for different purposes and the nutritionally significant infections in the group of interest; and we need to know the relationship between these infections in that group and nutritional status.

The role of behavioral and institutional factors in the successful implementation of a water supply program have been underestimated. "Improved" water supply systems are frequently poorly maintained (93) and, where the new system is working, traditional water sources are often preferred (94). Thus, the correlation between investment in water supplies and use of these supplies is often poor, particularly in rural areas. The effects of different water use patterns on health are not well known. Some studies have shown that the use of increased quantities of better quality water reduce the incidence of fecal-oral diseases (95, 96); other studies apparently have shown that the use of uncontaminated drinking water has not affected the incidence of classic water-borne diseases (97, 98). The classificatory work of Bradley (94) is an important advance, but understanding of the reasons for the success of some water interventions and the failure of others remains largely speculative (99).

For a variety of political and welfare considerations (including the expected health benefits), international agencies and the governments of Third World countries are committing large amounts of resources to the improvement of water supplies. Evaluation of the mortality and morbidity effects of such investments is a difficult task that should not be further complicated by attempting to simultaneously estimate the nutritional consequences. Analysis along the lines presented in this paper could be used in conjunction with the measured mortality and morbidity effects to assess the nutritional impact of such investments.

*Low-level antibiotic feeding.* The low-level feeding of antibiotics to animals has been commercially successful in reducing morbidity and mortality and increasing weight gains. The long term use of antibiotics in man has commonly been associated with improved weight gain, probably primarily as a result of reduced malabsorbtive losses and reduced nonviral and nonprotozoal diarrhea rates. The feeding of low doses of antibiotics to children has been proposed as a possibly effective way of breaking the diarrhea-malabsorbtion-malnutrition cycle. The potential role of such programs cannot be assessed until more information is available on the morbidity and nutritional effects, on the one hand, and the emergence of antibiotic-resistant strains and toxic or allergic side effects, on the other. Further pilot studies appear to be justified using antibiotics that are not first-line drugs for use in intestinal infections and in which plasmid transmission of resistance factor is nonexistent (100).

*Oral rehydration.* The World Health Organization has recently urged the use of oral therapy as the major weapon to control morbidity and mortality from acute diarrheal diseases. The successful implementation of rehydration therapy would markedly affect the two factors, namely, mortality and reduced food intake, that play the greatest role in the efficiency of food use by young children. Studies in the Punjab (101) and in Indonesia (29) show that acute diarrheal death rates among children can be halved when mothers are trained to give oral rehydration mixtures to their children. A hospital-based study of Apache children (102) suggested that early rehydration would prevent the negative nutritional impact of diarrhea primarily through the early restoration of appetite.

*Convalescent care programs.* Food that is withheld from or refused by a sick child may be fed to that child when appetite returns to supernormal levels during the early convalescent and recovery phases. Studies in Uganda and Jamaica show that during this hyperphagic period children consumed approximately twice their normal intake until the weight-for-height ratio of the child had returned to a normal value (103). Programs which encourage this practice (29, 83, 103) require neither an increase in the amount of food available to the family nor the control of infections, yet may have a marked impact on child health and nutrition. The makeup food presumably would not be seen as a drain on the food supplies of the family if the mother was told: "If you want your child to regain his health, feed him the food he missed when he was sick" (29). Furthermore, where immune competency is compromised by undernutrition, mortality may be reduced by

the effect of refeeding on the rapid restoration of normal immune functioning (29).

Such convalescent programs, therefore, directly address the two most important mechanisms relating infection to the poor use of available nutrients by young children. Rohde's (29) enthusiastic advocacy of convalescent programs as effective means for intervening in the malnutrition-infection interaction appears to be appropriate.

*Maternal nutrition and health programs.* Improved maternal health and nutrition may play an important role in improving the nutritional efficiency of reproduction by promoting better fetal growth (44, 60), higher birth weight, and lower child mortality. The importance of birth weight has been stressed by Mata et al. (34) who found that "half of the infants born weighing less than 2100 g died in the 1st year of life, while all those weighing 2900 g or more survived the 1st year". In the Punjab, children who had weight less than 70% of the Harvard standards for 3 or more consecutive months during the 4 to 35 month period of life had death rates 9 times higher than other children (58).

## Summary

This brief and incomplete survey suggests that convalescent care, oral rehydration, and measles vaccination programs may be the most cost-effective methods available for reducing the effect of infections on the intake and use of food by young children in poor countries. Whereas our ability to assess the effects of water supply and sanitation programs is poor, it is likely that these programs have an important role to play in countries where behavioral and institutional constraints have been overcome through social and economic change. If and when vaccines are developed against viruses that cause diarrhea, these may significantly reduce the effect of infections on nutrition in young children.

## References

1. MORLEY, D. Pediatric Priorities in the Developing World. London: Butterworths, 1973, p. 470.
2. WRAY, J., AND A. AGUIRRE. Protein-calorie malnutrition in Candelaria, Colombia: I: Prevalence, social and demographic causal factors. J. Trop. Pediat. 15: 76, 1969.
3. PRAWIRANEGARA, D. D., AND D. KARJADI. Some studies on the association of nutrition and infection in Indonesia. In: Proceedings of the 9th International Congress of Nutrition, edited by A. Chavez. New York; S. Karger, 1975, vol. 2, p. 148.
4. PUFFER, R. R., AND C. V. SERRANO. Patterns of Mortality in Childhood. Washington, D.C.: Pan American Health Organization, 1973, p. 470.
5. BEISEL, W. R. Metabolic response to infection. Ann. Rev. Med. 26: 9, 1975.
6. KEUSCH, G. T. The consequences of fever. Am. J. Clin. Nutr. 30: 1211, 1977.
7. WORLD HEALTH ORGANIZATION. Energy and Protein Requirements: Report of WHO/FAO Ad Hoc Expert Committee. Geneva: World Health Organization, 1973, p. 118.
8. SUKHATME, P. V. Size and nature of the protein gap. Nutr. Rev. 28: 223, 1970.
9. HEGSTED, D. M. Energy needs and energy utilization. Nutr. Rev. 32: 33, 1974.
10. GOPALAN, C. Protein versus calories in the treatment of protein calorie malnutrition: Metabolic and population studies in India. In: Protein-Calorie-Malnutrition, edited by R. E. Olsen. New York: Academic Press, 1975, p. 329.
11. BEATON, G. H. AND L. D. SWISS. Evaluation of nutritional quality of food supplies: Prediction of "desirable" or "safe" protein:calorie ratios. Am. J. Clin. Nutr. 27: 485, 1974.
12. SCRIMSHAW, N. S. Strengths and weaknesses of the committee approach: An analysis of past and present recommended dietary allowances for protein in health and disease. New Engl. J. Med. 294: 136, 1976.
13. LATHAM, M. C. Nutrition and infection in national development. Science 188: 561, 1975.
14. SCHNEIDER, R. E., M. SHIFFMAN AND J. FAIGENBLUM. The potential effect of water on gastro-intestinal infections prevalent in developing countries. Am. J. Clin. Nutr. 31: 2089, 1978.
15. CHEN, L. C., AND R. H. CHAUDHURY. Demographic change and food production in Bangladesh 1960-1974. Pop. Dev. Rev. 1: 201, 1975.
16. SCRIMSHAW, N. S., C. E. TAYLOR, AND J. E. GORDON. Interactions of Nutrition and Infection. Geneva: World Health Organization, 1968, p. 329.
17. WATT, B. K., AND A. L. MERRILL. The Composition of Foods. Washington, D.C.: United States Department of Agriculture, Handbook no. 8, 1963, p. 189.
18. WOOLLEY, P. O., C. A. BERRY AND R. N. ECCLES. Syncrisis: the Dynamics of Health: Panama. Washington D.C.: United States Agency for International

Development, 1970, p. 236.

19. ASHWORTH, A. Regulation of weight and height during recovery from severe malnutrition. In: Proceedings of the 9th International Congress of Nutrition, edited by A. Chavez. New York: S. Karger, 1975, vol. 2, p. 280.

20. MILLER, D. S., AND P. M. MUMFORD. Gluttony: low or high protein diets. Am. J. Clin. Nutr. 20: 1212, 1967.

21. BAKER, S. J. Discussion: session IV. Am. J. Clin. Nutr. 25: 1243, 1972.

22. LINDENBAUM, J. Nutrition: the role of malabsorbtion. In: Disaster in Bangladesh, edited by L. C. Chen. New York: Oxford University Press, 1973, p. 52.

23. DAVIDSON, S., R. PASSMORE, J. F. BROCK AND A. S. TRUSWELL. Human Nutrition and Dietetics (6th Ed.) Edinburgh: Churchill Livingston, 1975, p. 756.

24. REVELLE, R. Food and population. Sci. Am. 231: 160, 1974.

25. BAYLESS, T. Discussion: session I. Am. J. Clin. Nutr. 25: 1074, 1972.

26. REVELLE, R. Comment. In: Nutrition, National Development and Planning, edited by A. Berg, N. S. Scrimshaw, and D. L. Call. Cambridge: M.I.T. Press, 1973, p. 91.

27. WORLD HEALTH ORGANIZATION. Nutrition and Infection: Report of a W.H.O. Expert Committee. Geneva: World Health Organization, 1965, p. 30.

28. FRISCH, R. AND R. REVELLE. Variations in body weights among different populations. In: The World Food Problem: Report of the Panel on the World Food Supply, Washington D.C.: President's Science Advisory Committee, The White House, 1967, vol III, p. 5.

29. ROHDE, J. E. Preparing for the next round: convalescent care after acute infections. Am. J. Clin. Nutr. 32: 2258, 1978.

30. EINSTEIN, L. P., D. M. MCKAY AND I. H. ROSENBERG. Pediatric xylose malabsorbtion in East Pakistan: correlation with age, growth retardation and weanling diarrhea. Am. J. Clin. Nutr. 25: 1230, 1972.

31. INTERDEPARTMENTAL COMMITTEE ON NUTRITION AND NATIONAL DEFENSE. Pakistan: Nutrition Survey of East Pakistan, March 1962–January 1964. Washington D.C.: United States Department of Health, Education and Welfare, 1966, p. 426.

32. MATA, L. J., J. J. URRITIA AND J. E. GORDON. Diarrheal disease in a cohort of Guatemalan children observed from birth to age two years. Trop. Geograph. Med. 19: 247, 1967.

33. MATA, L. J., J. J. URRITIA AND A. LECHTIG. Infection and nutrition of children of a low socio-economic rural community. Am. J. Clin. Nutr. 24: 249, 1971.

34. MATA, L. J., J. J. URRITIA AND B. GARCIA. Malnutrition and infection in a rural village of Guatemala. Proceedings of the 9th International Congress of Nutrition. 2: 175, 1975.

35. BAKER, S. J., AND V. I. MATHAN. Tropical enteropathy and tropical sprue. Am. J. Clin. Nutr. 25: 1047, 1972.

36. KEUSCH, G. T. Subclinical malabsorbtion in Thailand: I: Intestinal malabsorbtion in developing countries. Am. J. Clin. Nutr. 25: 1062, 1972.

37. LINDENBAUM, J., J. W. HARMON AND C. D. GERSON. Subclinical malabsorbtion in developing countries. Am. J. Clin. Nutr. 25: 1056, 1972.

38. ROSENBERG, I. H., N. W. SOLOMONS AND D. M. LEVIN. Interaction of infection and nutrition: Some practical concerns. Ecol. Food Nutr. 4: 203, 1976.

39. HARPER, G. P. Xylose malabsorbtion and growth retardation in East Pakistani children. Am. J. Clin. Nutr. 25: 1227, 1972.

40. GOPALAN, C., AND S. G. SRIKANTIA. Nutrition and disease. World Rev. Nutr. Dietet. 16: 97, 1973.

41. MATA, L. J., R. A. KRONMAL, J. J. URRITIA AND B. GARCIA. Effect of infection on food intake and the nutritional state: perspectives as viewed from the village. Am. J. Clin. Nutr. 30: 1215, 1977.

42. CALL, D. L., AND J. LEVINSON. A systematic approach to nutrition intervention programs. In: Nutrition, National Development and Planning, edited by A. Berg, N. S. Scrimshaw, and D. L. Call. Cambridge: M.I.T. Press, 1973, p. 165.

43. BARNETT, T., AND R. MULLER. Global reach. New Yorker, Dec. 4, 1974.

44. MATA, L. J. The Children of Santa Maria Cauque: a Prospective Field Study of Health and Growth. Cambridge, Mass: MIT Press, 1978, p. 395.

45. AMERICAN PUBLIC HEALTH ASSOCIATION. Control of Communicable Diseases in man. Washington D.C.: American Public Health Association, 1975, p. 413.

46. MARTORELL, R., J. P. HABICHT, C. YARBROUGH, A. LECHTIG, R. E. KLEIN AND K. A. WESTERN. Acute morbidity and physical growth in rural Guatemalan children. Am. J. Diseases Children 129: 1296, 1975.

47. ROWLAND, M. G. M., T. J. COLE AND R. G. WHITEHEAD. A quantitative study into the role of infection in determining nutritional status in Gambian children. Brit. J. Nutr. 37: 441, 1977.

48. RAO, M. N., AND P. C. SEN. Singur Health Centre: its Achievements and Lessons. Calcutta: All Indian Institute of Hygiene and Public Health, 1969, p. 45.

49. YOUNG, V. R., AND N. S. SCRIMSHAW. Working paper on the effects of environmental factors on the nutritional requirements of underprivileged populations. Washington, D.C.: Committee on International Nutrition Programs, Food and Nutrition Board, National Research Council, p. 28.

50. TAYLOR, C. E., AND C. DE SWEEMER. Nutrition and infection. World Rev. Nutr. Dietet. 16: 203, 1973.

51. CROWLEY, L. V., H. POLLACK AND J. E. BROCKETT. Studies on nutrition in the Far East, XII. Metabolism 5: 297, 1956.

52. TRIPATHY, K., E. DUQUE, O. BALANOS, H. LOTERO AND L. G. MAYORAL. Malabsorption syndrome in ascariasis. Am. J. Clin. Nutr. 25: 1276, 1972.

53. BRISCOE, J. Public Health in Rural India: the Case of Excreta Disposal. Cambridge: Harvard University, Ph.D. dissertation, 1976, p. 414.

54. STOLL, N. R. On endemic hookworm: where do we stand today? Exptl. Parasitol. 12: 241, 1962.

55. JOHNS HOPKINS CENTER FOR MEDICAL RESEARCH AND TRAINING. Annual Report. Baltimore: Johns Hopkins University, 1970, p. 108.

56. BEISEL, W. R., R. S. PEKAREK AND R. W. WANNEMACHER. The impact of infectious disease on trace

element metabolism of the host. In: Trace Element Metabolism in Animals, edited by W. G. Hoekstra. Baltimore: University Park Press, 1974, Vol 2, p. 217.

57. VAN ZIJL, W. J. Studies on diarrheal diseases in seven countries by the W.H.O. Diarrheal Diseases Advisory Team. Bull. World Health Organ. 43: 249, 1966.

58. KIELMANN, A., AND I. S. UBEROI. Interactions of nutrition and infection: a perspective field study on children in selected villages of Punjab. In: Final Report to the Indian Council of Medical Research. Narangwal, Punjab: Rural Health Center, April, 1972, p. 58.

59. BEISEL, W. R. Discussion: session V. Am. J. Clin. Nutr. 25: 1287, 1972.

60. BEISEL, W. R. Synergistic effects of maternal malnutrition and infection on the infant. Am. J. Diseases Children 129: 571, 1975.

61. BEISEL, W. R., W. D. SAWYER, E. RYLL AND E. CROZIER. Metabolic effects of intracellular infections in man. Ann. Internal. Med. 67: 744, 1967.

62. POLLACK, H., AND D. R. SHELDON. The factor of disease in the world food problems. J. Am. Med. Assoc. 212: 598, 1970.

63. BEISEL, W. R. Nutrient wastage during infection. Proceedings of the 9th International Congress of Nutrition, 2: 160, 1972.

64. POLLACK, H., AND S. L. HALPERN. Therapeutic Nutrition. Washington D.C.: Natl. Acad. Sci. Natl. Res. Council Publ. 234, Washington, D.C., 1952, p. 94.

65. SHAFFER, P., AND W. COLEMAN. Protein metabolism in typhoid fever. Arch. Internal Med. 4: 538, 1909.

66. COLEMAN, W., AND E. DU BOIS. Calorimetric observations on the metabolism of typhoid patients with and without food. Arch. Internal Med. 15: 887, 1915.

67. BOGERT, L. J., G. M. BRIGGS AND D. H. CALLOWAY. Nutrition and Physical Fitness. Philadelphia: W. B. Saunders Co., 1973, p. 598.

68. ROE, C. F., AND J. M. KINNEY. Caloric equivalent of fever: II. Influence of major trauma. Ann. Surg. 161: 140, 1965.

69. SALOMON, J. B., L. J. MATA AND J. E. GORDON. Malnutrition and the common communicable diseases of childhood in rural Guatemala. Am. J. Public Health 58: 505, 1968.

70. FRENCH, F. E., AND J. M. BIERMAN. Probabilities of fetal mortality. Public Health Repx. 77: 835, 1962.

71. POTTER, R. G., J. B. WYON, M. NEW AND J. E. GORDON. Fetal wastage in eleven Punjab villages. Human Biol. 37: 262, 1965.

72. SHEPS, M. C. Pregnancy wastage as a factor in the analysis of fertility data. Demography 1: 111, 1964.

73. CURLIN, G. T., L. C. CHEN AND S. B. HUSSAIN. Demographic crisis: The impact of the Bangladesh Civil War (1971) on births and deaths in a rural area of Bangladesh. Pop. Studies 30: 87, 1976.

74. COALE, A. J. AND P. DEMENY. Regional Model Life Tables and Stable Populations. New Jersey: Princeton University Press, 1966, p. 871.

75. NATIONAL ACADEMY OF SCIENCE. Maternal Nutri-tion and the Course of Pregnancy. Washington D.C.: National Academy of Sciences, 1970, p. 241.

76. REVELLE, R., AND H. A. THOMAS, JR. Population and food in East Pakistan. Indust. Trop. Health 7: 27, 1970.

77. STEIN, Z., M. SUSSER, G. SAENGER AND F. MAROLLA. Famine and Human Development. New York: Oxford University Press, 1975, p. 284.

78. ANTONOV, A. N. Children born during siege of Leningrad in 1942. J. Pediat. 30: 250, 1947.

79. GORDON, J. E. Social implications of nutrition and disease. In: Food, Science and Society, a Symposium. New York: Nutrition Foundation, 1968, p. 3.

80. ROSENBERG, I. H. Nutrition: food production, dietary patterns and nutritional deficiencies. In: Disaster in Bangladesh: Health Crises in a Developing Nation, edited by L. C. Chen. New York: Oxford University Press, 1973, p. 31.

81. SOMMER, A., AND M. S. LOEWENSTEIN. Nutritional status and mortality: a prospective validation of the QUAC stick. Am. J. Clin. Nutr. 28: 287, 1975.

82. SCRIMSHAW, N. S., C. E. TAYLOR AND J. E. GORDON. Interactions of nutrition and infection. Am. J. Med. Sci. 237: 367, 1959.

83. BENGOA, J. M. Recent trends in public health nutrition: Protein-calorie malnutrition. In: Proceedings of the 8th International Congress of Nutrition, edited by J. Masek. Amsterdam: Excerpta Medica, 1970, p. 14.

84. MITRA, A. The nutrition movement in India. In: Nutrition, National Development and Planning, edited by A. Berg, N. S. Scrimshaw, and D. L. Call. Cambridge: M.I.T. Press, 1973, p. 357.

85. SCRIMSHAW, N. S. Synergism of malnutrition and infection: evidence from field studies in Guatemala. J. Am. Med. Assoc. 212: 1685, 1970.

86. UNITED STATES AGENCY FOR INTERNATIONAL DEVELOPMENT. The A.I.D. Nutrition Program Strategy. Washington D.C.: U.S. A.I.D., 1973, p. 38.

87. WILLIAMS, C. Health services in the home. Pediatrics 52: 773, 1973.

88. KEYFITZ, N., AND W. FLIEGER. World Population: An Analysis of Vital Data. Chicago: University of Chicago Press, 1968, p. 659.

89. MOSLEY, W. H., A. K. M. A., CHOWDHURY, K. M. A. AZIZ, M. S. ISLAM AND M. FAHIMUDDIN. Demographic Studies in Rural East Pakistan, May 1966–April 1967. Dacca: Pakistan S.E.A.T.O. Cholera Research Laboratory, 1968, p. 26.

90. KAPIKIAN, A. Z. Viral diarrhea. Etiology and control. Am. J. Clin. Nutr. 31: 2219, 1978.

91. MCKEOWN, T., AND R. G. BROWN. Medical evidence related to English population in the eighteenth century. Pop. Studies, 9: 119, 1955.

92. PANIKAR, P. G. K. Fall in mortality rates in Kerala: An explanatory hypothesis. Econ. and Pol. Weekly. 10: 1811, 1975.

93. CAIRNCROSS, A., AND R. G. FEACHEM. Operation and maintenance of rural water supplies. Courier. 43: 57, 1977.

94. WHITE G. F., D. J. BRADLEY AND A. U. WHITE. Drawers of Water: Domestic Water Use in East Africa. Chicago: University of Chicago Press, 1972, p. 306.

95. SCHLIESMAN, D. J. Diarrheal disease and the envi-

ronment. Bull. World Health Organ. 21: 381, 1959.

96. HOLLISTER, A. C., D. BECK, A. M. G. GITTELSOHN AND E. C. HEMPHILL. Influence of water availability on Shigella prevalence in children of farm labor families. Am. J. Public Health. 45: 354, 1955.

97. CURLIN, G. T., K. M. A. AZIZ AND M. R. KHAN. The influence of drinking tubewell water on diarrhea rates in Matlab Thana, Bangladesh. Dacca: Cholera Research Laboratory, 1977, p. 18.

98. LEVINE, R., M. R. KHAN, S. D'SOUZA AND D. R. NALIN. Failure of sanitary wells to protect against cholera and other diarrheas in Bangladesh. Lancet 2: 82, 1976.

99. BRISCOE, J. The role of water supply in improving health in poor countries (with special reference to Bangla Desh). Am. J. Clin. Nutr. 31: 2100, 1978.

100. ROSENBERG, I. H., AND N. W. SOLOMONS. The potential for antidiarrheal and nutrient-sparing effects of oral antibiotic use in children—a position paper. Am. J. Clin. Nutr. 31: 2202, 1978.

101. KIELMAN, A. A., AND C. McCORD. Home treatment of childhood diarrhea in Punjab villages. J. Trop. Pediatr. 23: 197, 1977.

102. HIRSCHHORN, N., AND K. M. DENNEY. Oral glucose-electrolyte therapy for diarrhea: a means to maintain or improve nutrition? Am. J. Clin. Nutr. 28: 189, 1975.

103. BEISEL, W. J. Resumé of discussion concerning the nutritional consequences of infection. Am. J. Clin. Nutr. 30: 1294, 1977.

## Appendix

### TABLE 1
Caloric consumption by a cohort of Bangladeshi children[a]

| Age | Probability of dying in the period | No. dying/ 1000 live births | No. living | Person-yr lived in the period | Person-yr lived by those who survived the period | Person-yr lived and calories consumed (kcal × 10^6) in the given period by those dying between | | | | | | Total calories | Cumulative calories |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | Conception-birth | birth-1 yr | 1 yr-4 yr | 5 yr-9 yr | 10 yr-14 yr | Over 15 yr | | |
| −9 mo | | | 1818.0 | | | | | | | | | | |
| | 0.4500 | 818.0 | | 928.3 | 692.3 | 236.0 (17.2) | 81.0 (5.9) | 62.9 (4.6) | 10.9 (0.8) | 5.4 (0.4) | 532.2 (32.9) | 67.8 | 67.8 |
| 0 | | | 1000.0 | | | | | | | | | | |
| | 0.1170 | 117.0 | | 918.1 | 883.0 | | 35.1 (12.2) | 90.9 (31.5) | 15.7 (5.4) | 7.8 (2.7) | 768.7 (266.5) | 318.3 | 386.1 |
| 1 yr | | | 883.0 | | | | | | | | | | |
| | 0.1030 | 90.9 | | 3278.6 | 3168.4 | | | 110.2 (42.2) | 62.8 (24.1) | 31.2 (12.0) | 3074.8 (1178.4) | 1256.7 | 1642.8 |
| 5 yr | | | 792.1 | | | | | | | | | | |
| | 0.0198 | 15.7 | | 3921.3 | 3882.0 | | | | 39.3 (19.7) | 39.0 (19.6) | 3843.5 (1929.0) | 1968.3 | 3611.1 |
| 10 yr | | | 776.4 | | | | | | | | | | |
| | 0.0100 | 7.8 | | 3862.8 | 3843.5 | | | | | 19.3 (13.7) | 3843.5 (2721.6) | 2735.3 | 6346.4 |
| 15 yr | | | 768.7 | | | | | | | | | | |
| Calories consumed by those dying between the given ages | | | | | | 17.2 | 18.1 | 78.3 | 50.0 | 48.4 | 6134.4 | | |
| Cumulative | | | | | | 17.2 | 35.3 | 113.6 | 163.6 | 212.0 | 6346.4 | | |

[a] Sample calculation:

$$\frac{\text{Intake of those who die between 0-5}}{\text{Intake up to 5 of those who live past 5}} = \frac{12.2 + 31.5 + 42.2}{318.3 + 1256.7 - 12.2 - 31.5 - 42.2} = 5.77\%$$

**TABLE 2**
Caloric consumption by a cohort of Latin American children[a]

| Age | Probability of dying in the period | No. dying/ 1000 live births | No. living | Person-yr lived in the period | Person-yr lived by those who survived the period | Person-yr lived and calories consumed (kcal × 10^6) in the given period by those dying between | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | Birth–1 yr | 1 yr–4 yr | 5 yr–9 yr | 10 yr–14 yr | Over 15 yr | Total calories | Cumulative calories |
| 0 yr | 0.0589 | 58.9 | 1000.0 | 954.5 | 941.1 | 13.4 (4.6) | 16.0 (5.5) | 2.9 (1.0) | 2.0 (0.7) | 920.2 (319.0) | 330.8 | 330.8 |
| 1 yr | 0.0170 | 16.0 | 941.1 | 3721.2 | 3700.4 | | 20.8 (8.0) | 11.6 (4.4) | 8.0 (3.1) | 3680.8 (1410.6) | 1426.1 | 1756.9 |
| 5 yr | 0.0031 | 2.9 | 925.1 | 4618.2 | 4611.0 | | | 7.2 (3.6) | 10.0 (5.0) | 4601.0 (2309.2) | 2317.8 | 4074.7 |
| 10 yr | 0.0022 | 2.0 | 922.2 | 4606.0 | 4601.0 | | | | 5.0 (3.5) | 4601.0 (3258.0) | 3261.5 | 7336.2 |
| 15 yr | | | 920.2 | | | | | | | | | |
| Calories consumed by those dying between the given ages: | | | | | | 4.6 | 13.5 | 9.0 | 12.3 | 7296.8 | | |
| Cumulative: | | | | | | 4.6 | 18.1 | 27.1 | 39.4 | 7336.2 | | |

[a] Sample calculation:

$$\frac{\text{(Intake of those who do not reach age 5)}}{\text{(Intake, during the first 5 yr of life, of those who survive beyond 5)}} = \frac{18.1}{1756.9 - 18.1} = 1.04\%$$

TABLE 3
Caloric consumption by a cohort of United States children[a]

| Age | Probability of dying in the period | No. dying/ 1000 live births | No. living | Person-yr lived in the period | Person-yr lived by those who survived the period | Person-yr lived and calories consumed (kcal × 10^6) in the given period by those dying between: | | | | | | Total calories | Cumulative calories |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | Conception–birth | Birth–1 yr | 1 yr–4 yr | 5 yr–9 yr | 10 yr–14 yr | Over 15 yr | | |
| −9 mo | 0.2372 | 311.0 | 1311.0 | 753.8 | 692.3 | 61.5 (4.5) | 11.1 (0.8) | 1.9 (0.1) | 1.4 (0.1) | 1.2 (0.1) | 676.8 (49.5) | 55.1 | 55.1 |
| 0 | 0.0161 | 16.1 | 1000.0 | 985.4 | 983.9 | | 1.5 (0.5) | 2.8 (1.0) | 2.0 (0.7) | 1.7 (0.6) | 977.5 (338.9) | 341.7 | 396.8 |
| 1 yr | 0.0028 | 2.8 | 983.9 | 3929.7 | 3924.8 | | | 4.9 (1.9) | 8.0 (3.1) | 6.8 (2.6) | 3910.0 (1498.5) | 1506.1 | 1902.9 |
| 5 yr | 0.0020 | 2.0 | 981.2 | 4900.8 | 4896.0 | | | | 4.8 (2.4) | 8.5 (4.3) | 4887.5 (2453.0) | 2459.7 | 4362.6 |
| 10 yr | 0.0018 | 1.7 | 979.2 | 4891.5 | 4887.5 | | | | | 4.0 (2.8) | 4887.5 (3460.9) | 3463.7 | 7826.3 |
| 15 yr | | | 977.5 | | | | | | | | | | |
| Calories consumed by those dying between the given ages | | | | | | 4.5 | 1.3 | 3.0 | 6.3 | 10.4 | 7800.9 | | |
| Cumulative | | | | | | 4.5 | 5.8 | 8.8 | 15.1 | 25.5 | 7826.3 | | |

[a] Sample calculation:

$$\frac{\text{(Intake from conception of those who do not reach age 15)}}{\text{(Intake from conception of those who live beyond age 15)}} = \frac{25.5}{7826.3 - 25.5} = 0.32\%$$