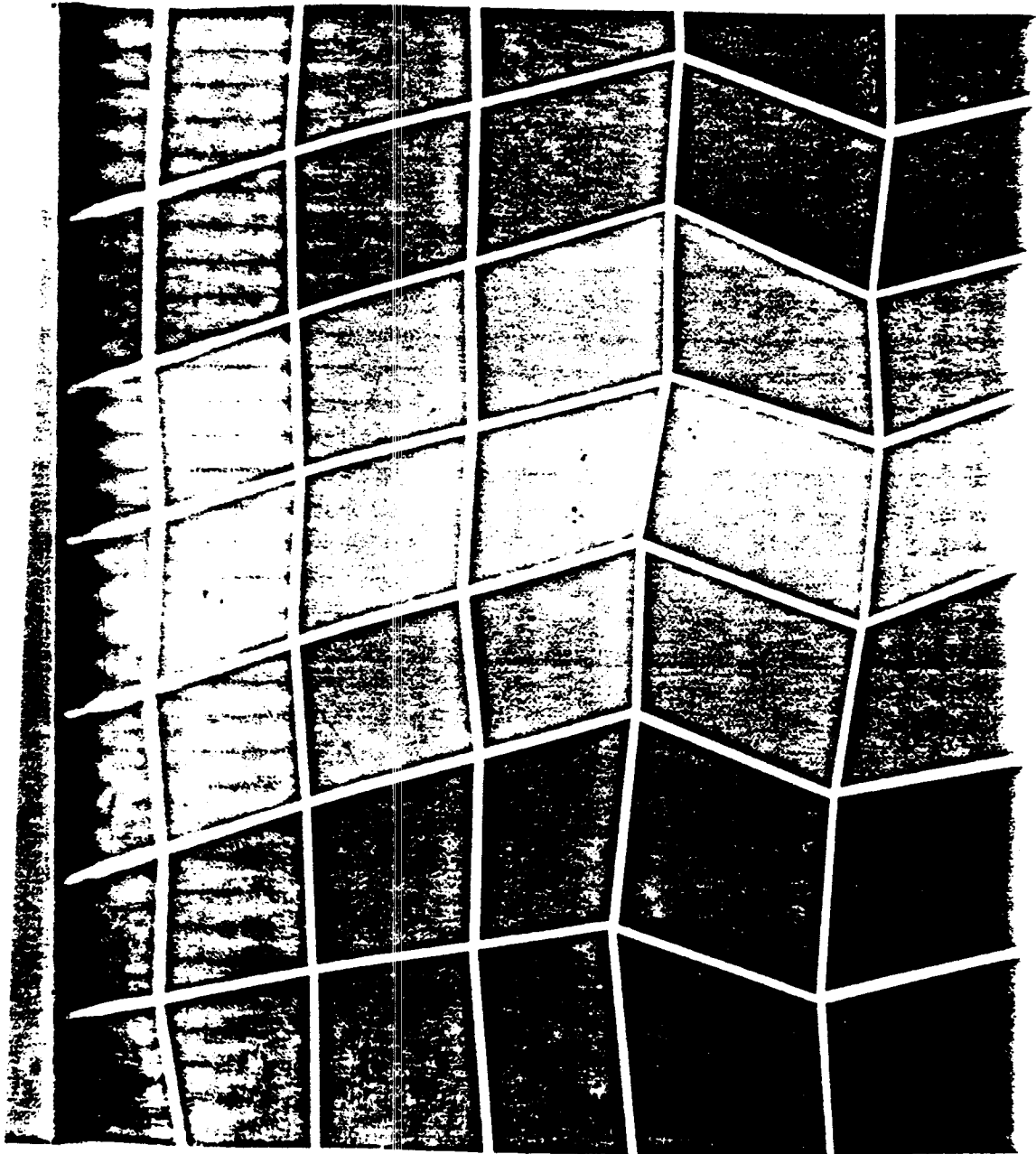


# Confronting Urban Malnutrition

THE DESIGN OF NUTRITION PROGRAMS



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ONE OF THE MOST DISTRESSING PROBABILITIES by the year 2000 is the quadrupling of the urban population of the developing world. Urbanization, the most dramatic and fundamental force affecting developing countries today, proceeds on a vaster scale and at a faster pace in Third World countries than in the industrialized nations of the world. The signs of poverty attendant upon such rapid urban growth—the teeming, center-city slums and peripheral shantytowns of most of the world's million-plus cities—indicate a more serious problem: an insidious wasting away of a developing nation's human capital through malnutrition.

Presupposing a political commitment to meet basic human needs in the urban setting, this study analyzes the systematic planning, design of interventions, program management, and community action that is necessary to translate political will into improved nutrition for the urban poor. Beginning with the first step in nutrition programming—diagnosis of the, and severity, causes, and victims of nutritional deficiencies—the author identifies three categories upon which nutrition planning must be based: the nature of the city under study, its economic behavior, and its nutritional status.

The author also examines nine strategies for nutrition intervention: education, on-site feeding, take-home feeding, nutrient-dense food supplies, ration shops, food-coupon programs, fortification of foods, direct nutrient dosage, and food processing and distribution, plus corollary interventions in health care, water, sanitation, and family planning. Despite the scarcity of empirical data on the performance of various nutrition programs, the author delineates the critical variables of program design and makes judgments that will serve as a guide to the program planner. He assesses methods of financial analysis and evaluation of cost effectiveness and concludes with a discussion of the managerial components critical to effective nutrition programs: control, personnel, publicity, financing, organization, and barriers to direct intervention.

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## Foreword

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I would like to explain why the World Bank does research work and why this research is published. We feel an obligation to look beyond the projects that we help finance toward the whole resource allocation of an economy and the effectiveness of the use of those resources. Our major concern, in dealings with member countries, is that all scarce resources—including capital, skilled labor, enterprise, and know-how—should be used to their best advantage. We want to see policies that encourage appropriate increases in the supply of savings, whether domestic or international. Finally, we are required by our Articles, as well as by inclination, to use objective economic criteria in all our judgments.

These are our preoccupations, and these, one way or another, are the subjects of most of our research work. Clearly, they are also the proper concern of anyone who is interested in promoting development, and so we seek to make our research papers widely available. In doing so, we have to take the risk of being misunderstood. Although these studies are published by the Bank, the views expressed and the methods explored should not necessarily be considered to represent the Bank's views or policies. Rather, they are offered as a modest contribution to the great discussion on how to advance the economic development of the underdeveloped world.

ROBERT S. MCNAMARA  
*President*  
*The World Bank*

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# Urban Malnutrition

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## Problem Assessment & Intervention Guidelines

Submitted to  
The World Bank  
by  
Harvard University

Study Director  
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Dave Wheeler, Joe Wray, Marian Zeitlin.

September, 1976

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D. Infection and Malnutrition: The Use of Public Health Programs as Nutrition Intervention

Since public health programs affect the prevalence of infection and disease in a population, and since the prevalence of infection and disease affects the efficiency with which a human body can utilize the available food, investments in public health programs may be expected to have consequences for the nutritional status of a community. The basic assumption underlying this investigation is that a quantitative understanding of the above relationships is necessary for evaluating the cost-effectiveness of such interventions. To facilitate this understanding we attempt to:

- i) Develop a methodological framework, based on an explicit and consistent set of assumptions, with which to analyse nutrition/infection policy issues;
- ii) Analyse the existing data in terms of this framework;
- iii) Indicate which data deficiencies appear to be paramount; and
- iv) Draw the policy implications which tentatively emerge from this analysis.

The effects of public health programs on health are complex in theory and difficult to measure in practice. While the emphasis in this paper will be on quantifying the relationships between health and nutritional status, this analysis will facilitate a fuller understanding of the role of public health programs as nutrition intervention strategies by indicating the upper bound on the nutritional effectiveness of such programs.

The importance of this question is clear in the 1973 Report of the WHO/FAO Expert Committee on Energy and Protein Requirements.

The nutritional levels suggested in this report are intended to apply to healthy individuals. However, acute and chronic infections of all degrees of severity, including parasitic infestations, are endemic in many regions of the world. Children and elderly people are more frequently ill, and for longer periods, than are young adults in most of these populations. Research is needed on the effects of these disorders on nitrogen and energy metabolism and requirements.

This paper is not the first attempt at addressing these questions. A 1972 conference on malabsorption and nutrition had a similar objective. Rosenberg stated the central question as follows: "If you put money into sanitation, is it going to improve nutrition?" and answered: "Obviously we cannot give a good answer to that yet, knowing as little as we do..."<sup>143</sup>

As will become evident in the course of this analysis we are, four years later, in no position to make a definitive statement on this matter. The simplicity of the questions which are asked, e.g. "How much of the food eaten in, say, India, is wasted due to the presence of infections?" belie the

\*The following team member was involved in the preparation of this section:  
J. Briscoe.

complexity of the underlying mechanisms and the magnitude of the research required.

Since nutrition intervention 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 the body can put that food. However, any statements which are useful for policy makers will severely tax the limited understanding and data at our command and these statements will thus be an inviting target for those who understand the paucity of information in this area. In this analysis we will attempt to deal with this tension 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. We will also attempt to suggest what, from a policy viewpoint, the critical areas for further research on this topic are.

"Infections of almost any degree of severity worsen nutritional status by interfering with food intake and by causing an increased loss of essential nutrients from the body. Conversely, the commonest types of malnutrition, even when subclinical, affect one or more of the mechanisms of resistance to infections or to the resulting infectious disease."<sup>144</sup>

We will focus only on the effect of infection on nutrition and not on the complete interrelationship. While this restriction does alter our ability to interpret the results of clinical studies, we will see that the findings of epidemiological studies which do not take the interrelationships into account are liable to suffer from methodological problems. Studies in Colombia,<sup>145</sup> Indonesia, and Latin America suggest that exclusion of this feedback loop may have particularly important consequences in diarrheal disease studies.

#### 1. Mechanisms by Which Infection Affects Nutrition

Infection can influence nutritional status in an individual by inducing a reduction of food intake, and by causing malabsorptive, catabolic, and internal losses of nutrients. We shall examine the mechanisms underlying these influences briefly.

- a. Reduced Intake of Food:
  - i) Due to anorexia (loss of appetite for food).
  - ii) When a nursing infant gets sick and stops sucking, the mother's milk may decrease<sup>146</sup> or stop completely if the mother does not empty her own breasts.
  - iii) The child may be taken off the regular food supply and put onto a watery gruel.



- b. Reduced Absorption of Nutrients:
  - i) Due to "tropical enteropathy," the malabsorption syndrome in sub-clinical cases.
  - ii) Due to the administration of purgatives during an illness.
  - iii) Due to malabsorption of nutrients during a period of illness, particularly when the illness is accompanied by diarrhea.
- c. Catabolic losses of nutrients:
 

Due to the destruction of body tissue and the excretion of the products of this destruction in sweat, feces, and urine.
- d. Functional wastage:
  - i) Due to nutrient over-utilization during infection.
  - ii) Due to sequestration of nutrients.
  - iii) Due to diversion of nutrients.

## 2. Some Definitions Important for Policy Issues

### a. The meaning of nutrient "loss"

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 over-estimated 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 malabsorption, too, have been overstated by: dividing the sample population into "good" and "poor" absorbers and ascribing the difference between these subgroups to malabsorption, a method which ignores the fact that in any population the absorptive 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 which is required for evaluation of these losses on a community-wide basis do not seem complicated and, yet, is 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:

- i) 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);
- ii) The food intake of the person during the course of the illness; and
- iii) 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:

$$\text{Food Loss (kcal)} = \text{Intake during illness} - \text{"Normal" intake over that period} + \text{Food required to recover weight loss}$$

We note a couple of points about the above:

- i) The implicit assumption is that the food which 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 the assumption is seriously violated, we can look at the food loss in two ways:
  - a) From the point of view of the family as a whole the above equation is applicable.
  - b) From the point of view of the infected individual who is given no additional allowance upon termination of the illness, the food loss is simply equivalent to the weight loss over the course of the illness.
- ii) 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 (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 as different from "food loss" which is what we are investigating in this paper.

In considering subclinical malabsorption, we would take a slightly different position. What we would want to do is evaluate the difference between the amount of energy required to perform the same work and leisure tasks with and without the presence of the malabsorption syndrome. We have to be quite careful in doing this. If we wish to use the result as a correction factor to be applied to the nutrient requirements of a population, then the above method would be correct. If we wish to determine the nutritional value of the administration of an antibiotic, we would have to be much more cautious for, in this case, the nutritional gain (which would be that computed above) may be cancelled out by possible negative effects. If we wish to evaluate the nutritional consequences of, for instance, improved environmental sanitation, we would use the procedure outlined above, in this case comparing the existing situation, not to the situation in which there is no malabsorption, but to the changed malabsorption regime.

## b. The Nutrients Evaluated

In conformity with widely accepted practice, we will assume that the nutrients of major importance are calories and protein and, we will focus our attention on these. Scrimshaw et.al.<sup>147</sup> have compiled the studies which have been done on the effect of infection on other nutrients and we will simply refer to their study in connection with these other effects.

The subject of the relative importance of calories and protein as nutritional inputs for the target groups remains controversial. This controversy is reflected in discussions of the importance of infection on the wastage of calories and protein. Beaton and Swiss<sup>148</sup> note that "major infections and probably recurrent minor infections affect both energy and protein requirements" and add that "there does not seem to be any good reason to assume that the ratio of requirements (for calorie and protein) is seriously disrupted by recurrent minor infections."

Gopalan, Beaton, and Swiss, Sukhatme, and Hegsted<sup>149</sup> reflect current thinking in this area by suggesting that the crucial deficiency in widely different parts of the underdeveloped world is a caloric and not a protein deficiency. Latham,<sup>150</sup> in an article on infection and nutrition, has placed a similar emphasis on the role of calories, while Gopalan<sup>151</sup> has suggested that the role of protein in infection-induced losses has been given undue priority: "Infections may be expected to increase not merely the requirement for protein, but the requirements for other nutrients, including calories, too. The suggestion that diets in the tropics should include a blanket coverage for possible increased protein requirements is unjustifiable on scientific grounds." Other researchers (notably Scrimshaw) have continued to put the major emphasis on protein and the effects of infection on this nutrient.

In this analysis our basis will be towards assessing, where possible, the effects of infection in terms of "food" rather than in terms of the individual nutrients affected. Much of the data available pertain to specific nutrients and this will necessarily continue to be the pattern as scientific investigation of this matter proceeds.

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 assumptions. These are: i) that all of the loss in body mass during an infectious episode is muscular tissue, and ii) that all of the loss is adipose tissue. Data presented by Scrimshaw et. al.<sup>152</sup> suggest that 3.2 kgms of muscular tissue are equivalent to 100 gms of nitrogen. Using the nitrogen: protein ratio of 1:5.95 given for rice by Watt and Merrill,<sup>153</sup> one kg of muscular tissue is equivalent to 190 gms of protein. In terms of rice (parboiled - 33.6 gs protein per pound and NPU of 63%) this amounts to 4.20 kg of rice to replace one kg of muscular tissue. The amount of calories required to build up one kilogram of adipose tissue has been given as 7,700 kilocalories by Woolley.<sup>154</sup> Hegsted<sup>155</sup> confirms

that this is the potential deposition of adipose tissue when excess calories are ingested. Ashworth's<sup>156</sup> data on malnourished children suggest that the efficiency of utilization of calories is greater at extreme malnourishment (9.6 kcals/gm) than it is near recovery (17.4 kcals per gm of tissue). Since the figure given by Woolley is, it seems, a "potential" figure, we take a value from Ashworth's data, of say 12 kcals per gram for children who are undernourished but not severely malnourished. Again expressing this in terms of rice, we see that one lb. of rice (parboiled) provides 1,674 kcals<sup>157</sup> and thus one kg of tissue requires an additional input of 3.3 kg of rice (at 98% caloric absorption.)

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. We thus may be unable to use data both on caloric and nitrogen loss to get some estimate of the food loss.

As somewhat of an aside we note that arguments over the primacy of calories or protein may not only be irrelevant when we look at the losses in terms of food (as shown above) but that exclusive attention to either of these obscures important interactions between the two nutrients. The work of Hegsted and Miller<sup>158</sup> would suggest that:

Caloric Requirement = f (Caloric Intake, Protein Intake, Body Weight),

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

Conversely, Scrimshaw<sup>159</sup> 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."

### 3. Some Methodological Issues

#### a. Absorption Studies:

Much of the data concerning malabsorption in human populations has been based on the D-xylose absorption test. A recent conference on malabsorption discussed the xylose malabsorption issue at some length. The sense of this discussion is exemplified by comments made by Baker<sup>160</sup> at this conference: "It is quite obvious from the studies performed that the degree of xylose malabsorption we have been discussing does not necessarily parallel malabsorption of other foods. I don't think you can say that because you have such-and-such a prevalence of xylose malabsorption, you also have such-and-such a prevalence of overall malabsorption of ordinary foods. After all, who eats xylose in the normal course of events?" While no published data giving individual absorption 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 absorption studies it is necessary to specify the "norm" for absorption of the particular nutrient in a population. If Lindebaum's (1973)<sup>161</sup> finding in Bangladesh, viz. that mal-absorption 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.<sup>162</sup> (using Atwater's data) report that about 92% of protein, 95% of fat and 99% of carbohydrate are normally absorbed (not passed in feces) in young American men. Since the digestibility of different types of foods are quite different, we used Revelle's<sup>163</sup> data on the average Indian and American "food baskets" in conjunction with the digestibility factors for different foods<sup>164</sup> to estimate the average absorption of protein, fat, carbohydrate and calories in a person who is absorbing "normally" and who is eating each of these diets.

Table 2.71

	<u>American Diet</u> (Atwater's data)	<u>(From Watt and Merrill)</u>	<u>Indian Diet</u> (From Watt and Merrill)
Protein	92%	91%	79%
Fat	95%	95%	93%
Carbohydrate	<u>99%</u>	<u>97%</u>	<u>97%</u>
Total Calories	-	95.4%	94.4%

These calculations demonstrate the importance of taking the actual diet consumed by the population 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 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.

If the nutritive value of the food intake is calculated from tables similar to those given by Watt and Merrill then these values include losses for normal metabolic and digestive losses and thus the % of "available intake" in the feces is:

$$\frac{100 - \% \text{ digested}}{\% \text{ digested}}$$

The percentage of available intake which is expected in the fecal matter of these two cases is thus:

Table 2.72

	<u>American diet</u>	<u>Indian diet</u>
Protein	9.9%	26.6%
Fat	5.3%	7.5%
Calories	4.8%	5.9%

Finally, we note a reservation expressed by Bayless<sup>165</sup>, concerning the findings from fat absorption tests which is similar in nature to Baker's comment that "no one eats xylose." Bayless has pointed out that estimates of fat malabsorption 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.

b. Population-Wide Balance Studies:

One of the only estimates of the % of food which is lost through malabsorption and infection is that given by Revelle.<sup>166</sup> 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 population is, literally speaking, neither expanding nor contracting. On the basis of the 1965 FAO/WHO Caloric Requirements (these are slightly higher than the 1973 requirements), Revelle estimates that the intake is about 2,000 calories per capita per day and the requirement only about 1,800 calories per capita per day, and thus the amount of food "malabsorbed" is about 10% of the intake. While this approach is both instructive and seems more useful than virtually any of the alternatives, the computation involves estimating the difference between two numbers which cannot be specified with much precision, which are both large and which are not much different from each other, and ascribing a definiteness to the relatively small difference. Some crude calculations which we have carried out (estimating the standard error in the measurement of caloric intakes from the two estimates-- by the USDA and ICNND -- which are given by Frisch and Revelle<sup>167</sup> for a number of Asian and Latin American countries; and assuming that the requirements are known with certainty (which they clearly are not); and assuming that intakes and requirements are uncorrelated) suggest that the standard error of the estimate of caloric malabsorption may be about the same size as the mean of this estimate. While the figure of 10% which 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 "malabsorption" 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 malabsorption, wastage due to the destruction of body tissue during febrile episodes.)

c. Longitudinal and Cross Sectional Growth Studies:

From cross-sectional studies <sup>168</sup> we usually observe correlations between incidence of disease and low weight gain. There are two important problems with drawing inferences from these data - those of multicollinearity in the "explanatory" variables, and those of distinguishing cause and effect. The first is exemplified in the ICNND study of Pakistan, in which it can be seen that the nutritional status of the population is highly correlated with the seasonal production of pulses (the relationship which was focussed on in the ICCN study) but also with the seasonal incidence of diarrhea. With the type of analysis done (tabulation) it is simply not possible to evaluate the significance of the contributing factors. Given that the relationship between infection and nutrition is reciprocal we are left, in cross sectional studies, without any means for evaluating whether it is the nutritional status which has increased susceptibility and/or decreased resistance to infection, whether the presence of infection has determined nutritional status, or whether both of these have been operative. In numerous instances this correlation is observed and the correlation taken an evidence to support the hypothesis which the particular investigator is supporting.

In a series of brilliant papers <sup>169</sup> Mata has presented longitudinal data on the relationships between infective episodes and nutritional status in individual Guatemalan children. From the resulting plots we get 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 looking at 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. While multivariate analytic techniques are by no means a panacea, and while the data requirements for a cross-sectional-cum-time-series analysis (which would make the most efficient use of data collected by Mata) are considerable, the use of these techniques is both interesting heuristically and offers some possibility for sorting out the relative strengths of the infection-nutrition and nutrition-infection relationships. It is recommended that an analysis of this sort be undertaken.

4. Data on the Effect of Infection on Nutrition

a. The Effect on Food Intake:

Infection may interfere with food intake through loss of appetite and withholding of food.<sup>170</sup> Some investigators <sup>171</sup> believe this mechanism to be crucial in the infection-nutrition relationship while others <sup>172</sup> discount this factor. There are few data available on the quantitative effect of these behavioral changes.

The practice of withholding of foods during an episode of diarrhea has been widely observed and almost universally condemned. In the context of the issue which this paper purports to address - viz. the relationships between public health programs and nutrition and nutrition programs -- it is important to understand one of the major reasons underlying this seemingly irrational practice.

In the Punjab, Wyon<sup>173</sup> found that "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." Cravioto<sup>174</sup> has observed a similar phenomenon in rural Mexico: "The mothers were reluctant to give their babies milk because many of them died of diarrhea. Proteins such as milk, eggs, and meat are excellent breeding grounds for bacteria while protein-poor substance such as gruel, are not. Cravioto concludes that the price of survival for the marginales in a society unable or unwilling to improve sanitation is malnutrition."

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

While Mata et. al.<sup>175</sup> believe restriction of food intake to be of key importance in the deterioration of the nutritional state, their data do not facilitate estimation of the effect of infection on food intake. They do report the food intake for one Guatemalan child between the age of 24 and 40 months. While a series of overlapping illnesses (otitis externa, cellulitis, and bronchitis) led to a marked reduction in food intake, two attacks of diarrhea, each lasting for about a week, did not appear to markedly affect food intake. In another publication, Mata (1976)<sup>176</sup>, gives data for Guatemalan pre-school children who have whooping cough. These data indicate that there is a 9% decrease in intake during the first month of whooping cough, that this decrease declines to 7% in the second month and that in the third month the intake is increased by an average of 9% over the normal consumption. (We note that the average duration of whooping cough in the U.S. is one to two months)<sup>177</sup> and that the increased intake during the third month may well indicate increased intake during the recovery period. If this finding is true in general, 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 which is postponed until the recovery period. Far more data are required before such an assumption could be reasonably considered accepted or rejected.)

#### b. The Effect on Absorption

##### i) Subclinical Malabsorption:

Bayless<sup>178</sup>, has noted that "almost every study involving a general population in a tropical area has shown a prevalence rate (of D-xylose malabsorption) between 30% and 50%." This is confirmed for India, for Thailand and Bangladesh.<sup>179</sup> Clinical examination of the villus height in the jejunum of these populations confirms that there is a morphological change in those populations which malabsorb D-xylose.<sup>180</sup> While Lindenbaum<sup>181</sup> reports that in Bangladesh "observations suggested that small intestinal injury was occurring in the entire population rather than in a subgroup of abnormals," Rosenberg et. al.<sup>182</sup> indicate that "tropical enteropathy is found in virtually the entire



low income adult population in tropical less developed countries." Lindenbaum<sup>183</sup> has found that there is little, if any, seasonal variation in xylose malabsorption in Bangladesh.

The etiology of "tropical enteropathy," the subclinical malabsorption syndrome, remains unclear. Indications are that the cause is neither subclinical malnutrition nor a ubiquitous food toxin but that it "is (probably) related to exposure to a contaminated environment,"<sup>184</sup> since "all areas in which this enteropathy occurs are those where there is a high contamination of the environment."<sup>185</sup> In his studies in Thailand, Keusch<sup>186</sup> found that all adults "have an abnormal flora in the jejunum" and suggested that this is "probably related to continuous environmental contamination." Lindenbaum et. al.<sup>187</sup> have suggested the following mechanism whereby environmental contamination is related to subclinical malabsorption:

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 strains of *E. coli*. Such mild enteric infections may be associated with profound impairment of absorptive 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 INCAP studies in Guatemala and the work of Einstein, et. al.<sup>188</sup> in Bangladesh, who showed that "xylose malabsorption (in children) was associated with growth retardation increased incidence of severe weaning diarrhea and increased incidence of live born sibling death."

These associations, however, are not found in all of the studies which have investigated these relationships. In his study of Bengali children, Harper<sup>189</sup> found that xylose malabsorption was not correlated with serious diarrheal episodes, amebiasis, giardiasis, or shigellosis, while Lindenbaum<sup>190</sup> found xylose malabsorption to be uncorrelated with either diarrheal episodes or nutritional status.

In a careful recent review, Rosenberg et. al.<sup>191</sup> weigh the conflicting evidence and conclude that "the contribution of recurrent intestinal infections (to tropical enteropathy) is very likely."

#### ii. The Effect of Manifest Infection on Malabsorption

Several studies on the effect of infections on the ability of a subject to absorb food conclude that the effect of manifest infection on absorption is likely to be insignificant unless the infection is accompanied by diarrhea.<sup>192</sup> Keusch et. al.<sup>193</sup> found that of two groups of Thai infants, that group which had more diarrhea had more rapid appearance of lactose intolerance and suggested that lactose intolerance may be a manifestation of recurrent diarrhea.

Data on the prevalence of diarrhea in poor countries are surprisingly difficult to find. Mata<sup>194</sup> observed diarrhea in a cohort of children from birth to age two years in rural Guatemala. In interpreting Mata's data, and in comparing these data with those of other researchers, it is important to understand clearly the definitions of "case" and "duration" used by Mata:

A new case of diarrhea dated from the first recognized abnormality of stools after at least 15 preceding days of normal bowel habit. One or more days without diarrhea but not more than 15 days, were included in the episode (case) when they came between bouts of clinically definite diarrhea. A diarrheal attack was considered terminated when stools were three or less per day and no recurrence was noted within the succeeding two weeks.

Duration of diarrhea was calculated as actual days during which diarrhea was present except that periods of one or two days with less than four bowel movements, but more than the usual number, and followed again by diarrhea, were included. When a period of normality extended for two to 14 days with again a recurrent diarrhea, only the initial days or days of diarrhea in that period were considered in the computation. Diarrhea lasting from one to 14 days was classed as acute diarrheal disease; chronic diarrheal disease was of 15 or more days duration.

Mata's data indicate that there was an average of 6.4 cases of diarrheal disease per 100 persons weeks in the population, and that the average duration of an attack of diarrheal disease was about 8 days. These data imply that on a given day we may expect a prevalence - the average proportion of the population suffering from diarrhea - of about 7%.

In their work in the rural Punjab, the Johns Hopkins team<sup>195</sup> collected data on the prevalence of diarrhea in children from birth to three years of age. Their data yielded a figure quite similar to that emerging from the Guatemalan data, with the prevalence of the symptom "abnormal frequency of stool" being 7.4%.

In their work in Candelaria, Colombia, Wray et. al.<sup>196</sup> "(asked) the mothers. . . whether the child had had diarrhea during the last week." On the average about 40% of the children were reported to have had diarrhea (four or more loose stools on any one day). While these data are reported under the heading of "Reported prevalence of acute diarrheal disease" we should note that what has been reported is not a prevalence figure but a figure reporting the incidence of diarrhea in the last week. To convert this figure into a prevalence we need to know the average number of days during the given week that the diarrhetic child actually had diarrhea. (We note that the definition of diarrhea given by Mata is not applicable to these data). If the prevalence of diarrhea is similar to that found in the Punjab and Guatemala, this would imply that those children who reported diarrhea in the last week had loose stools on the average of two days of the week. This does not seem to be an unreasonable figure and we, therefore, conclude that Wray's data do not appear to be incompatible with the above estimate of the prevalence of diarrhea.

The WHO Diarrheal Diseases Advisory Team<sup>197</sup> reported monthly diarrheal incidence rates for preschool children in seven countries.

Table 2.73

<u>Country</u>	<u>Incidence of diarrhea</u> (%)	
	<u>Area with water supply</u>	<u>Area without water supply</u>
Mauritius	-	2
Sudan	21.8%	26.6%
UAR	10	38
Ceylon	2	2
Iran	36	48.7
East Pakistan	-	20
Venezuela	39	43

If we disregard the data for Mauritius and Ceylon (which the WHO considered unreliable), we see that the equivalent weekly incidence rates in areas without water supply varied from about 4% to 12%. Mata's figure of 6.4% is within this range, and we conclude that the estimate of diarrheal prevalence of about 8% in preschool children of poor countries is not obviously inconsistent with any of the data presented.

"Even in severe gastro-enteritis, such as typhoid fever, only a small part of the adverse nitrogen balance is apparently attributable to decreased absorption of nitrogen. Even with severe diarrhea the absorption of nitrogen fell below 75%"<sup>198</sup> Beisel's<sup>199</sup> observation that in the presence of diarrhea the nitrogen in the stool may double or triple accords with these data.

In considering the impact of diarrhea on a population, we should bear in mind that diarrhea gives rise to two separate phenomena. "One is the fatal syndrome in young children where diarrhea is more frequent and has more serious consequences among children with malnutrition and where diarrhea turns marginal nutritional deficiencies into severe malnutrition. The other phenomenon is where diarrhea causes malabsorption of nutrients in all ages, leading to food wastage and its consequent economic loss, but seldom resulting in fatality in school-age children and adults" (Wall and Kieve, 1974).<sup>200</sup>

Both clinical and subclinical infestation with intestinal helminths may affect the absorption of nutrients. Hookworm infestation of a mild nature may interfere with the digestion and absorption of protein. Taylor and deSweemer<sup>201</sup> have suggested that the adverse effects on nitrogen balance of helminthic diseases such as hookworm, are roughly proportional to the parasite load. Animal studies at Cambridge<sup>202</sup> corroborate this suggestion in that they found that fluctuations in protein absorption parallel changes in worm burdens. In a study of the nutritional effects of hookworm in China, Crowley et. al.<sup>203</sup> found the effect to be proportional to the hookworm load, while Tripathy et. al.<sup>204</sup> drew similar inferences from their study of the effect of ascariasis in Colombia.

Scrimshaw et. al.<sup>205</sup> have summarized the findings of many studies concerning effects of the helminthic infestations without coming to any definite conclusions other than that "the relative importance of these 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 by complicated technical procedures." The quantitative data which they do present include a case where human adults with heavy hookworm infection had an average absorption 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 quote the result of an Indian study which showed the fecal nitrogen in children with moderately heavy ascariasis was reduced by half as a result of deworming.

On the basis of Crowley's study, Briscoe and Pollack<sup>206</sup> have independently arrived at the conclusion that each adult hookworm causes the loss of about 1 kcal per day. Given the high hookworm loads in many parts of the tropical world<sup>207</sup> gives a "moderate world average figure of 100 worms per person," which is about what the load is in rural West Bengal<sup>208</sup> this implies that about 5% of the human consumption of calories is lost due to hookworm alone. Given the high loads of other intestinal parasites, the high infection rates and the widespread existence of sub-clinical malabsorption, this effect of hookworm seems unrealistically high. (This points up an important problem in all analyses of this sort. When we try to build up from the micro-level data, we have to use isolated uncorroborated studies and extrapolate the results of these studies a good deal. It seems essential to have some macro-level types of consistency checks if we are ever to hope that these micro-studies will be fitted together in a reasonable way.)

### iii. Some Field Studies Concerning the Quantitative Importance of Malabsorption

While there are, as discussed earlier in this report, important problems associated with the use of D-xylose malabsorption as an index of food malabsorption, the sense of discussions on this issue is captured by Baker,<sup>209</sup> who suggests that "it is likely that xylose malabsorption indicates nutrient

malabsorption in the long run." Interesting data pertaining to the relationship between xylose malabsorption and nutritional status have emerged from two studies on children in Bangladesh. Harper <sup>210</sup> found a positive correlation between xylose excretion (a measure of the individual's absorptive capacity) and weight gain. Einstein et. al. <sup>211</sup> found that the presence of xylose malabsorption was lowest in the group of children who dropped below the expected weight profile. These data suggest that the effect of malabsorption on weight gain is more marked than the effect on nutritional status on absorptive capacity.

The most comprehensive field study of food wastage due to malabsorption which had been undertaken to date is presently being conducted by the University of North Carolina and the Institute of Nutrition for Central America and Panama (INCAP) in rural Guatemala. The results of this study, which are yet to be published, will almost certainly facilitate reasonable estimates of the food losses due to subclinical malabsorption and the losses accompanying episodes of diarrhea. These results will contribute greatly to an understanding of the quantitative effect of infection on nutrition.

c. 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 defense responses and the heightened requirements of metabolizable energy!"<sup>212</sup>  
The most important principles emerging from the pioneering work of William Beisel are: that there is a stereotypical (i.e., not dependent on the etiological agent) catabolic response to infection<sup>213</sup>; that fever is the major stimulus for initiating catabolic losses during an acute infectious episode 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.<sup>214</sup>

To estimate the overall effect of catabolic losses on a population, we need an estimate of the prevalence of fever and an estimate of the average temperature in febrile individuals. The only available data pertaining to the prevalence of fever in children in a poor country are those reported at Narangwal in the Punjab (Kielmann).<sup>215</sup> The prevalence of fever is 4.2% (implying an average of 15 symptom days per child annually.) No data on the average temperature recorded during a fever are reported.

i. Evaluated in Terms of Nitrogen Loss:

Shaffer and Coleman <sup>216</sup> observed 202 typhoid fever patients and found that in those who received no more than 40 kcals/kg/day - these patients received on an average 33 kcal/kg/day and 12 gms N/day - the average net losses of nitrogen were 6.5 gms per day. This figure is in agreement with Scrimshaw et. al.'s <sup>217</sup> statement that "there is a two to three fold increase in excretion of nitrogen, mostly through urine, associated with typhoid fever." Beisel's<sup>218</sup> data on artificially induced tularemia, sandfly fever and Q fever suggest typical total negative nitrogen balances during the course of these fevers of 50, 15, and 35 gms N respectively. (The data emerging from the Schaffer and Coleman study and those emerging from Beisel's work will be compared again later in this paper when we attempt to derive a quantitative relationship between the "fever index" and catabolic losses.)

Particularly important is the "interference with compensatory mechanisms which permit the body to minimize nitrogen losses in under-nutrition." 219 "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." 220 Thus, if a normal person lowers his or her 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 tend to be increased or maintained at close to normal rates. 221

The difference in the nitrogen excreted as a result of an infectious episode in a well-nourished person and in a malnourished person is not known. 222 It has been proposed that "there are 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." 223

While the validity of this theory of a "labile nitrogen reserve" in the body remains unclear, 224 it is known that in a malnourished subject there would be a smaller nitrogen loss for a given infection than there would be in a comparable well-nourished subject. As has been pointed out by the FAO/WHO 225 since the negative nitrogen balance is lower in malnourished individuals, the consequences of the extra nitrogen loss may be more serious."

#### ii. Evaluated in Terms of Caloric Loss

While little quantitative information is available on the effect of infections on fats and carbohydrates 226 or on total caloric loss 227 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. 228 Pollack 229 has examined some clinical data collected in the early part of this century on the increased energy requirements arising from infections with tuberculosis, malaria, and typhoid. His findings are summarized on the table below:

Table 2.74

Metabolic Requirements of Disease

Disease	Increase in Metabolic Requirements		% Increment in Basic Annual Caloric Requirements per Casa
	Acute Phase (Duration)	Prodromal or Postdromal Phase (Duration)	
Tuberculosis respiratory	+22% (3 months)	7% (9 months)	11.0
Malaria	+18% (2.5 months)	10-kg (22 lb) weight loss 5-kg (11 lb) weight loss	8.8 3.8 4.4
Dysentery	+40% (20 days)	4-kg (8.8 lb) weight loss	2.2

We digress briefly to discuss some methodological problems relating to the above accounting for the "costs" of disease.

The way in which these metabolic requirements were determined is of importance. On the basis of Shaffer and Coleman's typhoid fever data, Pollack<sup>230</sup> found that "extrapolating such losses to the usual 20 day course of the disease would give total losses approximately 130 gm. of body nitrogen, roughly equivalent to 4 kg of body mass." (We note that the implicit factor converting nitrogen to body mass [3.2 kg of tissue = 100 gm nitrogen] is the same as that given by Scrimshaw et. al.<sup>231</sup> Pollack then assumes that it takes 7,700 kcals to replace a kilogram of body mass (see the earlier discussion of this factor) and thus determines a "caloric equivalent" to the amount of body mass lost during the infection.

In 1915 Coleman and duBois 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 in the above table) Pollack assumes that the increased nitrogen excretion is the result of protein breakdown while 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, 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."<sup>232</sup> When protein is used as an energy source, amino (NH<sub>2</sub>) groups are split from the constituent amino acids and formed into simple nitrogen-containing substances (chiefly urea) that are excreted by the kidneys. The non-nitrogenous fragments of amino acids, the carbon chains, are then oxidized in the same way that carbohydrate and fats are metabolized.

It thus appears that Pollack's method overestimates the caloric cost of an acute illness, since 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.<sup>233</sup> for an indirect estimate of the relative importance of protein gluconeogenesis.

Beisel presents data on the cumulative nitrogen and body weight losses in febrile patients. Assuming (as before) that 100 gms. of nitrogen loss corresponds to 3.2 kgms. of muscular tissue, we find that the average muscle losses were: tulameria - 3.5 lbs., sandfly fever - 1.1 lbs. and Q-fever - 2.6 lbs. The corresponding body weight losses of 5.9 lbs., 3.2 lbs., and 4.6 lbs. imply that 60%, 35%, and 56% of the loss is due to protein destruction. Since no dehydration took place, we estimate that 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.

In using the above data it is also important to note that no allowance has been made for the possibility that, while 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 duBois found a stereotypical response to the severity and duration of fever which is remarkably similar to the more recent finding of Beisel<sup>234</sup> 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 indicate 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."<sup>235</sup>

In the table below, we have calculated the equivalent caloric costs of the diseases investigated by Beisel et. al.<sup>236</sup> on the assumption that one kilogram of body mass is equivalent to 7,700 kcals and that basal metabolism accounts for 2,000 kcals daily. We see that these values are remarkably similar to those which may be expected by application of the duBois formula.

Table 2.75

Infection	Equivalent Caloric Loss for Disease		
	Weight Loss (lbs)	Fever Index (°F days)	Implied metabolic increase for one degree of fever
Tulameria	5.9	156	6.6%
Sandfly Fever	3.2	111	5.0%
Q Fever	4.6	99	8.2%

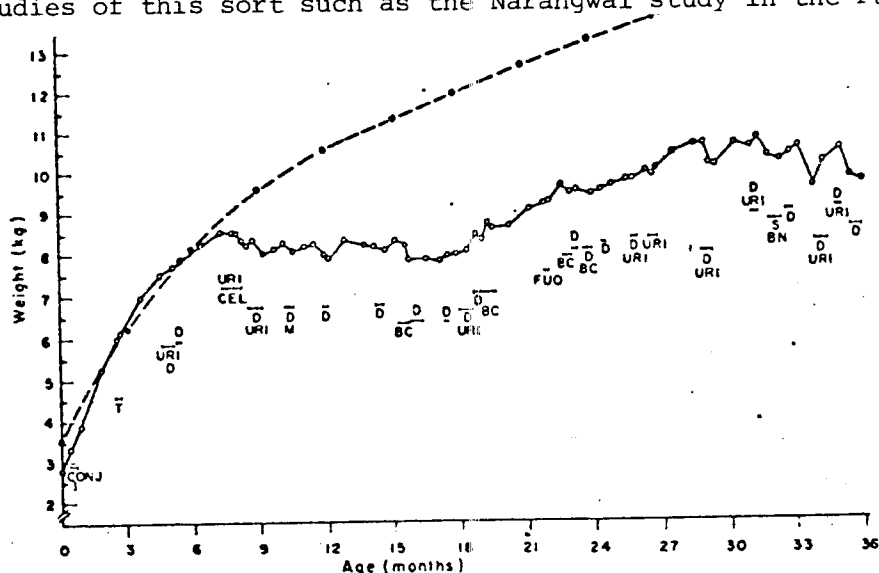
Roe and Kinney<sup>237</sup> have found that "surgical operations, burns, severe sepsis and loss of functional tissue mass can effect changes in the rate of both heat production and heat loss independently of temperature change" and conclude that the duBois formula is of limited utility in the treatment 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" (Roe and Kinney (1965)).<sup>238</sup> Use of the duBois formula, therefore, appears to be appropriate in this analysis.

d. The Effect of Infection 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.."<sup>239</sup> Beisel<sup>240</sup> has suggested that these functional wastages may be neglected for the purposes of this analysis.

e. The Effect of Infection on Overall Weight Changes

In field studies of populations in underdeveloped areas, the relationship between episodes of infection and the nutritional status of the infected individuals is extremely clear. This has been illustrated most graphically in Mata's studies (see figure below) in Guatemala, but is also evident in other studies of this sort such as the Narangwal study in the Punjab.<sup>241</sup>



Weight, Infections, and Infectious Diseases in a Male Child. The solid line represents the weight of the child; the broken line is the median of the standard. The length of each horizontal line indicates duration of infectious disease. BC, bronchitis; BN, bronchopneumonia; CEL, cellulitis; I, impetigo; M, measles; S, stomatitis; T, oral thrush; URI, upper respiratory infections; CONJ, conjunctivitis; D, diarrhea; FVO, fever of unknown origin.

This correlation between infection and change in weight has been observed in the Oxford Child Health Survey, in which "growth retardation in children is directly proportional to the amount of morbidity;"<sup>242</sup> in Latin America where "clinical records show that nutritional deficiency resulted from repeated episodes of diarrheal diseases;"<sup>243</sup> in China where Crowley et. al.<sup>244</sup> found that a hookworm load of 45 adult worm per capita resulted in a weight loss of 0.9 kg over a 120 day period. In Guatemala Saloman and Mata<sup>245</sup> have found that there are more episodes of infectious diseases in children with lower growth, while Wray<sup>246</sup> has made similar observations in Columbia.

(It is important to note the different "information content" of these different forms of reporting of the above results. The form of presentation used by Mata maintains the richness of the longitudinal data which has been collected and leaves no doubt that declines in nutritional status are causally related to infectious episodes. Data such as those collected by Wray and the Oxford Child Health Survey are, since they are cross-sectional data, much less informative since they give not sense of causation but only a knowledge of association. The problem of causation remains even with Mata's data, however, while it is quite clear that infection does affect nutritional status, we are unable, on an examination of these diagrams, to determine the degree to which poor nutritional status predisposes subjects to infection and to what degree the severity and the duration of established infections is affected by the nutritional status of the subject.)

Other data pertinent to this issue are given by Gordon and Scrimshaw<sup>247</sup> who state that in active typhoid fever a weight loss of 10 lbs. in 10 days is common, and Mata<sup>248</sup> who found that children lost an average of about 3%<sup>248</sup> of their body weight during an attack of measles.

Rosenberg et. al.<sup>249</sup> have examined the effects of infection on weight gain in animals. "The effect (of antibiotics) is greatest when growth is otherwise poor, morbidity and mortality is high, birth weight is low, diet quality is poor, the environment is dirty and stresses (e.g., unfavorable temperature) are present. When any of the adverse conditions are present, feed antibiotics result in greater than the 10% average increased meat production. Conversely, under ideal animal husbandry conditions, antibiotics may provide little or no advantage."

## 5. Some Policy Issues

### a. Interactions between Nutrition and Infection Interventions

From all of the evidence presented above it is clear that the presence of high rates of infection and malnutrition are intimately connected. Out of this recognition has emerged a concern for dealing with these problems in a holistic manner.

Scrimshaw and Gordon:<sup>250</sup> "... Where both malnutrition and infection are serious, success in control of either condition commonly depends on the other. Problems of malnutrition and infectious disease are interdependent in clinical management of patients and in public health programs;"

Mitra:<sup>251</sup> "The unfinished public health revolution deserves to be completed to confer the full benefits of positive nutrition programs. Incidentally, it is on the instrumentality of the public health and sanitation machinery and the immunization network that the worthwhile nutrition programs of the country can be made to ride piggy-back with the greatest economic efficiency and the maximum economy of human effort;"

Latham:<sup>252</sup> "The problems of infection and malnutrition are closely interrelated, yet we tend to introduce, quite independently, programs to control communicable diseases and other efforts to improve nutrition. It would be much more efficient and effective if the twin problems were attacked together. Success in improving the health and reducing the mortality of children is dependent on both control of infectious diseases and improvements in their food intake."

Mata:<sup>253</sup> "All too often the significance of high infection rates among poor children has been discounted by health workers and nutritionists.... (This) has discouraged needed research on the interrelation of infection and nutrition and has prompted the recommendation of applied nutrition programs in total disregard of principles of sanitation and prevention of infection." "...recent evidence has brought out that improved diets of children with high morbidity and mortality rates with no action towards infection control and prevention, do not result in a beneficial effect on nutritional status; programs aimed at education, hygiene and control of infection have produced dramatic results in better health and nutritional status of the child."

Bengoa:<sup>254</sup> "What should be emphasized here is the need for a combined approach to malnutrition and infectious diseases in young children, and this should be a matter of urgency. For instance, rehydration programmes for children with severe diarrhea require a follow-up with nutritional rehabilitation. The development of low-cost weaning foods also requires to be complemented with action to prevent infectious disease, particularly by immunization and environmental sanitation. The above programmes, given just as examples, will be able to produce a much more significant impact on the health of children when they are implemented simultaneously."

While there is no question regarding the interaction of infection and nutrition in a clinical sense, and while the above statements represent the distilled wisdom of some of the most thoughtful people concerned with this range of problems, the question of the relative effectiveness of combined versus separate programs, and, indeed, the over-riding question of how limited resources in this area should be allocated, has yet to be satisfactorily addressed. To say that both nutrition and infection are important determinants of health does not enable one to judge how resources should be allocated.

There have been two major efforts at elucidating the separate and combined effects of nutrition and infection control programs in "field experiments." In the Guatemalan study reported by Scrimshaw<sup>255</sup> there were inconclusive results on the effects of the feeding program, the integrated health care program was difficult to evaluate and the evidence on the interaction of nutrition and infection was weak or non-existent.

The data of a subsequent study in India, the Narangwal study,<sup>256</sup> are still being analyzed. Some interesting results have been reported. In the table below we see the effects of three year infection care (including immunization but not water supply or sanitation) and nutrition care (for pre-school children) programs on mortality:

Table 2.76

India: Narangwal Study

	Experimental Groups:			
	Nutrition & Infec. Care	Nutrition Only	Infection Care Only	Control
Infant mortality/ 1,000 live births	81	92	80	107
In 13-35 months (per 1,000 population in this group)	11	16	14	23

Source: Taylor et al. (1975).

From this table we note that there is a considerable reduction in mortality, particularly among the weaning age population, as a result of each of the programs. (On the basis of his extensive work in Guatemala, Mata <sup>257</sup> has stated that "...the high infant mortality was not dramatically altered by the medical care provided." Since Mata provided essentially the same sort of medical care that was provided in Narangwal, with the notable exception of the extensive immunization program which was undertaken in Narangwal, it seems likely that the mortality changes due to the infection care program in Narangwal were largely the result of the preventive rather than the curative measures taken. This finding reinforces the emerging consensus that mortality reductions in the developed world were not primarily due to medical advances, and that curative medicine has a limited role to play in the improvement of the health status of the poor countries.) From the Narangwal data above we note, too, that the effects of the different programs were not synergistic with respect to mortality reduction. If the programs were synergistic the respective death rates in the two populations would be below 65 and 7 in the case where both nutrition and infection care are provided. (This suggests that the word "synergism" is used rather loosely. While there may still be a justification in combining nutrition and infection care programs to reduce mortality on the basis of the above data, this would be because the costs of providing infection care in one village and nutrition care in a similar village may be greater than the provision of both services in a single village.)

Further analysis of the Narangwal data reveals that the effect of these programs on morbidity "appears to be less than the effect on mortality," and that "...the combination of medical care and nutritional supplements over 2-3 years could not do more than reduce the proportion of malnourished children from 25% to 17.5% <sup>258</sup> and there was no effect on the proportion severely malnourished".

These findings make it clear that the objective of nutrition intervention programs should be clearly specified and the efficiency of different programs in meeting these objectives clearly delineated.

### 6(b) Interactions among Target Groups

In this study we are concerned with the nutritional status of those in the target groups in poor countries, namely pregnant and lactating women and preschool children. While the medical profession in the very definition of the area of "maternal and child health" has recognized the primacy of the mother-child relationship, until recently nutrition planners have paid little attention to the way in which the health and nutritional status of mothers affect the nutritional status of their children.

It is increasingly clear that both maternal nutrition (Mata<sup>259</sup> "There is good evidence to believe that an improvement of nutrition during pregnancy results in better fetal growth rate") and maternal health (Rosenberg<sup>260</sup> stresses that "low birthweight is associated with a high incidence of maternal infection and the likelihood of transmission of infection to the fetus," while Biesel<sup>261</sup> emphasizes that "certain infections in the pregnant women may be accompanied by deleterious effects upon the fetus, regardless of the presence of fetal infection") are important determinants of the nutritional status and health of the child.

The vital role played by infant nutritional status is clear from both Narangwal and Santa Maria Cauque. Classifying those children who have had weight less than 70% of the Harvard standard for three consecutive months as malnourished, the Narangwal team<sup>262</sup> found that "the death rate for malnourished children in the ages 4-35 months was almost nine times that for the corresponding well-nourished groups." In Mata's<sup>263</sup> analysis of Guatemalan children "half of the infants born weighing less than 2100 gms died in the first years of life, while all those weighing 2900 or more survived the first year."

In recent analysis of some practical issues emanating from the concern with the interaction of nutrition and infection Rosenberg *et al.*<sup>264</sup> have come out very strongly in favor of giving prime attention to this interaction between the target groups: "Intervention during the post natal period is sure to be much more costly and of less certain success than prevention of fetal growth retardation by increasing attention to nutrition and prevention of infection during pregnancy." Beisel<sup>265</sup> has stressed that "carefully controlled trials are necessary to determine the efficacy of various prophylactic or therapeutic measures in preventing or diminishing fetal growth retardation."

### 6(c) Towards an Estimate of the Quantitative Effects of Infection on Nutrition

In this chapter we have attempted to lay out a coherent framework in which to understand the implications of the effects of infection on nutrition, and have manipulated the available data into a form which is in consonance with this framework. In this section we wish to draw these data together, to indicate what the critical data deficiencies may be and to suggest approaches for obtaining the required information.

In assessing the impact of infection on the community as a whole, we need to estimate the following:

- (i) Catabolic Losses: We can assume that the magnitude of catabolic losses is related to the severity and duration of fever during an acute illness. We therefore need data on the prevalence of fever in the group, the average temperature of those who have fever, and the caloric loss associated with a degree of fever.

The only available data indicate that about 4.2% of children may be expected to have fever at any particular time. While we have no data available on the degree of fever in these children an estimate of an average temperature of 4° F above normal seems reasonable. We have good data which indicate that there is a 7% rise in caloric requirement for each degree of fever and it therefore appears that about 1.2% of the calories ingested by preschool children in a poor country may be wasted due to catabolic losses. We note that more data on the prevalence of fever and the associated temperatures are necessary before this figure can be considered reliable.

- (ii) Overt Malabsorption Losses: We can assume that this affect is significant only when the infection is accompanied by diarrhea. Data are required on the prevalence of diarrhea and the caloric loss associated with a day of diarrhea. We have good data on the incidence of diarrhea in preschool children in a poor country. While the corresponding duration (and thus prevalence) data are less rich, it appears that a prevalence of 8% is unlikely to be drastically wrong. We essentially have no data on the effect of diarrhea on the absorbtive capacity of a child. The only piece of data pertaining to this issue suggests that a child with acute diarrhea is unlikely to malabsorb more than 15% of his or her food intake. It would thus appear that this factor would account for no more than about 1.2% food wastage. The University of North Carolina/ INCAP study is likely to yield satisfactory data on this issue.

- (iii) Subclinical Malabsorption Losses: We can assume that subclinical malabsorption is caused by intestinal abnormalities resulting from environmental assaults. Data are required on the absorptive capacities of populations who are of the same ethnic stock and who are fed the same diet but who live in different environmental conditions. No such data are presently published but, again, it is likely that data will soon be emerging from the UNC/INCAP study.

In assessing the impact of infection on affected individuals it is necessary to make assumptions about the relationship between an episode of disease and food which is either withheld or refused. It is also necessary to be explicit about the effect of disease on intrafamilial food consumption patterns. We need to know whether food "released" as a result of sickness in a preschool child is distributed to other preschool children in the family, whether adults consume this food or whether the food is "stored" for consumption by the affected child during recovery from the illness. That this issue is important can be illustrated by the following calculation using numbers which have no basis in the literature but which do not seem unreasonable. If we assume that 75% of the normal caloric intake of a child is withheld or refused during episodes of diarrhea, a diarrhea prevalence of about 8% implies a reduction of 6% in the aggregate intake of this group.

The effect of diarrhea prevalence on children is thus highly dependent on exactly what happens to the food which is not consumed during an episode of diarrhea. There are virtually no data pertaining to this important issue. Elsewhere in this report it is suggested that intensive study of intra-familial food consumption patterns is an area of high research priority. In designing such studies it would be extremely useful to incorporate an investigation of the effect of infection on consumption basis.

Many analyses of food and nutrition in poor countries suffer seriously from the fact that data are presented on "average deficits" where it is well known that these deficits are highly concentrated in certain groups within the population and that for these groups the situation is far worse than is indicated by the aggregate figures. The present study is no exception, for the same reasons which are so frequently presented in defense of similar shortcomings in other papers: there are inadequate data on the distribution of food intake which emerges from this framework - "x% of the food eaten by pre-school children is lost as a result of infection" - is of considerable interest in revising estimates of the nutritional requirements of a population and in delineating the aggregate nutritional effect of changes in the infectious regime. This figure, however, should be interpreted with caution. The distribution of infection and malnutrition in the population is likely to be highly skewed. If we are concerned with "the bottom 20%", for instance, it seems quite conceivable that 80% of the total caloric loss in the population could take place in this group which would thus have an average percentage loss four times higher than the population average.

(d) The Nutritional Consequences of Some Public Health Program

The basic approaches which we consider here for intervening in the infection side of the infection/nutrition interaction are:

- i) nutrition and health surveillance along the model of the pilot program in Narangwal for which results are reported in Table 2.76;
- ii) an improvement in environmental sanitation and water supply;
- iii) immunoprophylaxis; and iv) the use of antimicrobials.<sup>266</sup>

We will consider the efficacy of these interventions.

(i) Nutrition and Health Surveillance

More than 90% of national health programs in developing countries have a nutrition component, <sup>267</sup> which consists at the minimum of an attempt to provide face to face nutrition, health, and hygiene education to mothers in health centers which may or may not be further specialized to care for vulnerable groups. Increasingly, the use of weight charts which provide a system of monthly surveillance of the nutritional status of each child is being introduced. In developed countries, six-weekly surveillance during the first half year, three-monthly during the second half, biannually during the second year, and annually thereafter is sufficient. In developing countries monthly surveillance at least for some children should probably take place during the most vulnerable 6 - 24 month age period. Surveillance can be targeted by categorizing infants for "at risk" factors. A long list of these factors is given in Table 2.77a.<sup>268</sup> A corresponding list for pregnant women, designed to prevent fetal malnutrition is shown in Table 2.78b.<sup>269</sup> In slum areas lacking clinic facilities mobile units can provide surveillance, nutrition education, and limited curative facilities during monthly visits. During these visits local community health aides should



Table 2.6

	<u>India: Narangwal Study</u>			
	Experimental Groups:			
	Nutrition & Infec. Care	Nutrition Only	Infection Care Only	Control
Infant mortality/ 1000 live births	81	92	80	107
In 13-35 months (per 1000 population in this group)	11	16	14	23

Source: Taylor et al. (1975).

Table 2.77

Reasons for Considering Children "At Risk" and in Need of  
Special Care

A child is considered "at risk" if one or more of the following factors apply:

1. He or she happens to be a twin;
2. He weighs less than 1.5 kgs.;
3. If, when less than 6 months old, he is not breast fed and comes from a poor family;
4. If he has no surviving parents;
5. If he is looked after by his siblings while his mother is away at work;
6. If, during the first 3 months, he gained less than 1/2 kg a month or between 4 and 6 months failed to gain more than 1/4 kg a month;
7. Whether he lost more than one of his brother or sisters during their infancy;
8. If he has chronic diarrhea, whooping cough or measles;
9. If he is the 4th child or above in number;
10. If his weight is below 65% of the Harvard Standard;
11. If he was born after his parents had been married many years;
12. If he was born of parents who had been operated on for family planning;
13. If he has a step-mother.

Table 2.78b

Reasons for Considering Pregnant Woman "At Risk" and in Need  
of Special Care to Prevent Foetal Malnutrition

A pregnant woman is "at risk" when one of the following factors apply:

1. a. If she weighs 38 kgs or less before pregnancy;  
b. If she weighs 42 kgs or less at 34th week of pregnancy;  
c. If she gains less than 1 kg a month after the 5th month of pregnancy;
2. If she is less than 145 cms tall;
3. If, when she has her first baby, she is below 18 or above 30 years old;
4. If she is 35 years old or above at the time of pregnancy;
5. If she has a history of abortion or still-birth during a previous pregnancy;
6. If, during a previous pregnancy, she lost her child within a month;
7. If her previous delivery was a caesarean;
8. If it's her 5th or more child;
9. If she had swollen legs while pregnant or has swelling now;
10. Or bleeding;
11. Or jaundice;
12. If, during her pregnancy there was a doubt about her having twins;
13. If she has got high blood pressure;
14. If a child from the previous delivery had a birth weight of 2 kgs or less.

assist the surveillance team by assembling mothers and children at the mobile unit those targeted for care. A low cost surveillance system in rural Jamaica succeeded between 1970 and 1972 in decreasing young child mortality rates by one half. The incidence of cases of malnutrition did not decrease over this period. This is not an uncommon finding during the initial phases of surveillance and may reflect both a lag in community acceptance of nutrition and education and the fact that malnourished children who would otherwise have died are being saved so that their relatively low status brings the averages down to previous levels. However, children who were "treated" for malnutrition by regular monthly home visits by a community-health auxiliary who reinforced nutritional, sanitary, and hygienic practices in the home, showed significant improvement. Children weighing less than 75% of standard were assigned to the community-health auxiliary living nearest to them for such visits. Over two years 45% of these children improved, 11% remained the same, 7% declined, and only 1% died. The auxiliaries were literate community of women and men who were given three months of training at the Elderslie health center. 270

(ii) Environmental Sanitation, Water Supply

Analyses of historical declines of mortality in Europe<sup>271</sup> and similar analyses of the modern decline of death rates in developing countries<sup>272</sup> focus on these environmental changes as primary causes of the declines which took place. These changes affect most of the spectrum of infections which are believed to exert an influence on the nutritional status of the host and are thus especially attractive options in attempting to interfere with the infection factor in the nutrition/infection relationship.

In attempting to assess the nutritional impact of environmental improvements (e.g. improved water supply) we need several pieces of the information: We need to know the relationship between provision of the infrastructure (e.g. a public standpipe) and use of that facility; we need to know the relationship between use of the facility (e.g. in terms of quantity and quality of water consumed) and the infections of interest in the group of interest; and we need to know the relationship between infection in that group and nutrient loss. In this chapter we have indicated some of the difficulties in quantifying the last of these links and have suggested what research is required for a more complete understanding of the quantitative effect of infection on nutrition. The information available on the other links, too, is limited. In the words of White, Bradley and White,<sup>273</sup> those who wish to promote public works to improve domestic water supplies invariably employ as their chief justification the expected gains to public health. These confident and vigorous claims have often been extrapolated beyond the evidence on which they are based." The significant empirical problems notwithstanding, the available studies "...provide a significant amount of evidence that more and better water is associated with better health".<sup>274</sup> The question: "Exactly how much improvement in health can be expected from a specific water supply and sanitation-related improvement in a particular area and its given characteristics, however, remains unanswered."<sup>275</sup>

It is, thus, simply not possible to specify the effects of improved water supply and sanitation on food wastage due to infection. When the data on the relationships between infection and nutrition are more complete we will have an estimate on the upper bound of the amount of food which could be saved as a result of these improvements. In a particular context (rural East Africa) White *et al.*<sup>276</sup> have made "guesstimates" of the reductions in specific disease which may result from improved water supplies. The cautious use of

these estimates in conjunction with the other data on infection and nutritional wastage will indicate whether or not these environmental improvements are likely to have significant nutritional effects.

The standard argument against the implementation of these programs is that they are too costly to be able to reach a significant proportion of the population in the short run.<sup>277</sup> While any attack on these deficiencies undoubtedly will require a large commitment of resources, the costs involved have been exaggerated by the imposition of standards which may be suitable for developed countries but which are unlikely to be appropriate in poor countries.<sup>278</sup> In addition, the technologies which have been used in poor countries have frequently been imposed by foreign "experts" who have little knowledge of local conditions and, frequently, powerful vested interests in promoting imported, expensive, technologies. In this regard, it is encouraging that innovative, less costly forms of water supply and sanitation are being examined as viable alternatives to the conventional choices.

If the data suggest that water supply and sanitation programs are not promising nutrition intervention strategies, it should not be inferred that investments in these programs are inappropriate in developing countries, but merely that the justification for these investments may not be made exclusively in terms of the amount of food "saved" as a result of decreased infection and disease in the population. These programs have other extremely important objectives, namely reduction in mortality and morbidity and these objectives, too, should be considered when allocating available resources.

(iii) Immunoprophylaxis

The major drawback associated with this approach is that the infections which can be affected through the use of presently available immunizations generally only account for a small proportion of the morbidity in these areas.<sup>279</sup> In particular there are no vaccines for the common diarrheal diseases which affect infants in underdeveloped countries. In addition the administration of, say, a measles vaccination imposes nutritional losses on the children who are immunized. In analyzing the efficacy of immunization programs from a nutritional perspective, the certainty of these losses should be balanced against the probability of the larger losses which may be incurred if the disease itself is contracted.

(iv) Antibiotic Feeding

Rosenberg et al.<sup>280</sup> have surveyed the evidence on the potential use of using low level antibiotics to reduce intestinal infection and thus food wastage. Serious problems may arise from the extensive use of antibiotics. There may be a directly toxic effect on the host as a result of the selection of antibiotic resistant pathogens and as a result of unknown effects from alterations in the normal intestinal flora. At present there is far too little knowledge of these ecological effects to recommend even larger scale field trials and this is an option which can be ruled out in the formulation of policy at present.