The Ecological Interdependence of Diet and Disease in Tribal Societies

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Observations among nomads suggest there is a strong ecological interdependence of diet and disease in tribal societies which favors survival of man. This relationship may be disrupted by changes in diet to conform to the highly productive technology of the West. Such changes may result in intensification of indigenous disease and in the transfer of disease characteristics of Western societies. To prevent these consequences, relief feeding and long-term attempts to upgrade nutrition should be carried out with traditional foods wherever possible.

INTRODUCTION

Our purpose in this paper is to show that man's ability to survive disease in his natural environments depends in large part on an ecological balance between himself, his source and supply of food, and his micropredators.

Tribal societies without the advantages of technology afford a unique opportunity to examine closely the interrelationships of diet and disease. Unlike peoples of developing countries, they live intimately with their environment; their lives are frequently punctuated by recurring cycles of famine and plenty; their food comes from limited geographic sources, reflecting local deficiencies or excesses of the soil, and their diet has often been unchanged since the original introduction of their current form of subsistence or agriculture. Over the centuries there has been ample time for host, micropredators, and disease to adapt to both vicissitudes and nature of the local food supply. Furthermore, variables such as smoking, alcohol, processed foods, additives, and lack of exercise which so compound the problem of evaluating diet-disease relationship in our society are conspicuously absent in theirs.

It seems that certain specific patterns of disease and freedom-from-disease occurring in tribal societies may reflect in part human adaptation to local foods over the course of time. This is not true of all tribal societies. In Africa, as in the West, the steady replacement of indigenous food and crops by new and prolific species from other countries may have allowed little time for biological adaptation to occur. Although maize may have first appeared in Africa in pre-Columbian times (1360?) [1], most other imported food plants including rice, guinea corn, sweet potatoes, cassava, taro, ground nuts, beans, and bananas were introduced either by the Portuguese after 1500 or more recently by the colonialists [2]. The only food plants indigenous to Africa are yams, hungry rice, and pigeon peas while bullrush millet, a common crop of the sub-Saharan savannah, appears to have spread early from Asia.

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(1000 B.C.?)[2]. Only the dietary habits of nomads drinking milk as their major
source of energy, of hunter-gatherers in remote corners of the continents, and of
tribes with dietetic constraints imposed by long tradition have remained unchanged.
It is among such groups that our observations have been made.

Those who have worked with nomads cannot fail to recognize their remarkable
resistance to disease in the face of seemingly overwhelming adversity. Famine,
drought, bad water, poor sanitation, exposure to disease-bearing insects, and no
immunization would all be expected to exact a higher toll of life than they do. It is
ture that infant mortality is high among them and would be less with simple measures
of preventive medicine; but infant mortality is a cornerstone of population control in
primitive societies just as it is in the animal world. It prevents expansion of the tribe
beyond the bounds of its food supply. Once past this hurdle, the quality of health
without modern medicine is often surprisingly good and the proportion surviving
into old age not much different from our own[3]. While working in eastern Niger for
a year we were able to observe nomadic Fulani as well as sedentary Kanouri and
Buduma. Our findings there confirmed a frequency of the elderly and their freedom
from malignant and degenerative disease similar to that reported by Truswell in the
Kalahari bushmen[3,4]. Unlike the elderly in Western society, however, the elderly in
these tribal groups were still able to pursue the productive existence essential for
survival in a hostile environment. In more than 16,000 examinations over six years
amongst nomads in Niger, Nigeria, Ethiopia, and Kenya (with 37 percent and 9
percent of this population over the apparent ages of 45 and 65, respectively), we
encountered no stroke, coronary artery disease, hypertension, or diabetes mellitus
and only two malignant tumors. One was a cancer of the bladder in a man heavily
infested with the parasite S. haematobium which predisposes to bladder cancer and
the other a primary cancer of the liver.

EARLY OBSERVATIONS

Observations in eastern Niger during the drought and famine of 1973 first drew our
attention to the possibility of an ecological interdependence of diet and disease.
Despite a difficult period when food and water were in short supply, the undernour-
ished tribespeople in village and nomadic encampments around us seemed unusually
free of malaria (mostly falciparum), common viral illnesses, and malignancies [5].
This freedom seemed contrary to standard teaching of nutritional science which has
repeatedly stressed the adverse effect of malnutrition on host resistance[6,7]. And yet
the phenomenon as we saw it did not seem unreasonable. In fact, a mechanism
ensuring tribal survival in the event of the double adversity of famine and disease
seemed not only a good idea but a biological necessity.

In contrast to the apparent freedom from disease around us, the reverse was true in
our desert hospital. Malaria was frequent there whatever the primary reason for
admission. More impressive still was its occurrence in apparently healthy but
undernourished relatives accompanying patients to hospital. It seemed to occur
within a week of their arrival. Since there had been neither rain nor mosquitoes for
over a year and the patients and relatives denied any symptoms resembling malaria
for the preceding six months, these attacks could only have resulted from activation
of pre-existing quiescent disease. Famine seemed to have suppressed the disease while
refeeding with relief grain, mostly sorghum and wheat imported from Western
countries, had permitted its reactivation. A prospective study confirmed these
suspicions and pointed to a peak occurrence of infection on the fifth day after the
start of refeeding (Fig. 1) [5]. Although we never were able to carry out similar
prospective studies on viral infections such as measles, poliomyelitis, and hepatitis, those illnesses were extraordinarily mild even in our most severely marasmic children and seemed of low infectivity—findings at variance with hospital-based studies of sporadic malnutrition [9] but not out of keeping with observations made on measles and malnutrition in the Warsaw ghetto during World War II [10] nor with our later controlled observations.

Feeding with relief grain had one further complication. We were faced with an abrupt but lethal outbreak of cerebral malaria in children of non-nomadic tribespeople. Only one of 23 (from a total village population of 1,500) survived this complication. The children of a tribe of 1,100 milk-drinking nomads in the same area were not similarly affected except for three (included in the 23) who had access to relief grain as a major replacement of their all-milk diet [11]. The implications seemed clear; famine protected from cerebral malaria whereas refeeding with grain but not milk favored its occurrence. Were nomads by virtue of their all-milk diet more resistant to the complications of falciparum malaria than their grain-eating counterparts, and were we dealing with our first example of the ecological interdependence of diet and disease? We thought so. Curiously enough, neither of the observations on malaria was entirely new. In the Bengal famine of 1943 Ramakrishnam observed that deaths from malaria were least in famine and greatest during relief feeding [12], while Edington in Ibadan, Nigeria, had commented on the striking resistance of the undernourished child and the easy susceptibility of the well-nourished child to cerebral malaria [13]. From these and similar observations on the occurrence of malaria, brucellosis, and tuberculosis in milk-drinking Somali nomads refed with grain rather than milk in the feeding camps of the Ethiopian Ogaden [14], we hypothesized that famine tended to suppress infections favoring an intracellular habitat while refeeding (especially with a food alien to tribal culture!) tended to activate them [15] (Fig. 2). This sequence, we believe, evolved as a biological system for protecting man from extinction or overpopulation. It is our further belief that a similar mechanism operates continuously as an ecological compromise on a less grand scale—local deficiencies or certain unusual nutrients in the diet tending to suppress some indigenous diseases and local excesses or other unusual nutrients to promote them.
LATER OBSERVATIONS

Somali nomads obtain nearly all their energy from the milk of goats and camels [16]. As a consequence of the low content of iron in milk, the nomads are frequently iron-deficient [17]. During a pilot study in the feeding shelters of the Ethiopian Ogaden, we observed that iron-deficient nomads entering the camp seemed to have less intracellular infections than their iron-replete counterparts. A prospective controlled study of iron repletion revealed a distinct increase in intracellular infections in repleted nomads over untreated controls [17](Table 1). Since the Somalis have presumably been iron-deficient for centuries, it struck us that iron deficiency may have permitted them to adapt favorably to the intracellular infections of their own region. Iron deficiency was protecting them against the more serious complications of potentially fatal infection and permitting an ecological compromise.

TABLE 1
Observations during the course of treatment of two matched groups of iron-deficient Somali nomads treated with either placebo (P) or iron as ferrous sulfate (F)

|                              | Iron Depleted (P) | Iron Repleted (F) |
|------------------------------|-------------------|-------------------|
| Number of cases              | 66                | 71                |
| Episodes of fever            | 6                 | 29                |
| Attacks of malaria           | 1                 | 13                |
| Malarial parasites seen      | 2                 | 21                |
| Clinical brucellosis         | 0                 | 5                 |
| Tuberculosis                 | 0                 | 3                 |
| Enlarged liver               | 0                 | 6                 |
| Enlarged spleen              | 0                 | 11                |
in which both host and parasite could co-survive harmoniously at the price of their combined iron deficiency. Curiously, this observation is not new either. Armand Trousseau, in his textbook of clinical medicine of 1872, describes vividly the salutary effect of iron deficiency on the course of pulmonary tuberculosis and the inexorable deterioration of the disease that follows iron repletion [18].

On the island of Anjouan in the Comorran archipelago, which lies between Madagascar and Mozambique, both ascariasis and malaria are very common. But strangely children heavily infected with the roundworm, *Ascaris lumbricoides*, and showing the unusual syndrome of bilateral painless enlargement of the parotid glands and central forehead edema were relatively free of malaria (Fig. 3). Children from the same island without severe ascariasis or the syndrome were highly susceptible to malaria [19]. When the former were treated successfully for ascariasis with the ascaricide piperazine, the parotid enlargement and the forehead edema disappeared but they were afflicted with malaria. We suggested that this phenomenon represented an ecological compromise of a host and two parasites through a nutritional mechanism—the ascariasis led to an as yet unidentified nutritional deficiency which resulted in enlarged parotids and forehead edema but simultaneously suppressed the maturation and multiplication of the plasmodium [20]. The price of protection from the dangerous complications of malaria was the discomfort of ascariasis and the small risk of its complications. We like to think of such a relationship as a microecological one favoring the survival of man—"Promethean microecology." It is an interesting exercise based on current knowledge to speculate on the nature of this mechanism. The Ascaris cleverly survives the proteolytic medium of the intestine by fabricating an array of antienzymes which block the tryptic activity of the host's gut [21]. It is possible that the enzymic suppression prevents the intestinal release and absorption of a nutrient essential for the maturation and multiplication of the malarial parasite.

There is an additional observation which may have implications for the amplification of this mechanism. A major dietary staple on the island is the pigeon pea (*Cajanus cajan*) which like many legumes contains a trypsinhibitor but one which is not readily destroyed by heat [22]. In fact, the pigeon pea has a reputation for indigestibility probably based to some extent on the presence of the inhibitor [23]. The pea may not only enhance the antitryptic effect of the *Ascaris lumbricoides* and help prevent malaria but may be partly responsible for the parotid hypertrophy so frequent in the children. In animal studies the antitryptic effect of the legumes is often

![FIG. 3. These boys from the Comorran island of Anjouan look healthy but the one on the right has an exceptionally round face from enlargement of both parotid glands. This finding in Anjouan was associated with heavy infestation with the roundworm *Ascaris lumbricoides* and remarkable freedom from the malaria so common on the island. Following treatment with the ascaricide piperazine, the parotids subsided but the boy had an attack of malaria.](image-url)
associated with hypertrophy and hypersecretion of the pancreas through excessive feedback stimulation of the pancreas by the intestinal hormones, cholecystokin-pancreozymin (CCK-PZM) [24]. The many similarities of the pancreas and the parotid glands make it likely that the CCK-PZM feedback also has a hypertrophic effect on the parotid. In fact when CCK-PZM is injected to test pancreatic function, parotid secretion is increased [25].

Relations of this sort are not always an advantage to the host. In countries where ascariasis abounds, tubers such as sweet potatoes, taro, and cassava (often not indigenous!) may form the main dietary staples. Some of the tubers also contain relatively heat-stable factors which can inhibit the trypic enzymes in the gut of the host [22]. When these effects are added to those of antitryptic factors released by the ascaris to prevent its own proteolytic dissolution [21], the survival and growth of the roundworms is encouraged further. Since the tubers are frequently a poor source of protein, kwashiorkor is common in the same countries. In fact a vicious cycle intensifying protein-calorie malnutrition (PCM) may ensue. Tubers providing calories but little protein encourage heavy infestation with ascaris; the combined antitryptic activities of the ascaris and the tubers added to the existing pancreatic dysfunction of PCM [26] further decrease the digestion and absorption of what little protein there is in the diet, creating more protein calorie malnutrition. And so the well-intentioned introduction of exotic, rapidly growing tubers to improve the supply of food may have intensified both ascariasis and kwashiorkor. The powerful effect of a tuber antienzyme may be seen in pig-bel, a serious and often fatal necrotizing enteritis of children in the New Guinea highlands [27]. The normal proteolytic destruction of the B toxin of type C Clostridium welchii released during meat meals may be inhibited by the high content of antienzymes in the local sweet potatoes eaten with the meat. The toxin is then free to exert its destructive effects on the intestinal wall [28].

The periodicity of malaria in the tropics is another interesting phenomenon; while many factors may be operating in its genesis, the autumnal rise in frequency of the disease in India clearly preceds the appearance of mosquitoes [29]. We suspect that this is an effect of the monsoon on the availability of food. With the dry months and the scarcity of food, malaria is suppressed, but with the arrival of rains and the abundance of the harvest that follows, the parasite thrives to the point of clinical disease. Edwards observed this characteristic seasonal pattern in the occurrence of foot and mouth disease in the central provinces of India in the 1930s [30]. This type of periodicity may be an example of a regular cyclical interdependence of diet and disease.

MECHANISMS

We have implied that undernutrition and specific nutrient deficiency may be responsible in tribal societies for an ecological balance favoring increased host resistance to certain infections. But how does such a system work? Usually immunocompetence as measured in vitro is depressed during famine [31]. But host resistance is not simply the sum of a series of in vitro tests; in fact, experimental studies in animals point to increased resistance to viral and other intracellular infections during malnutrition [32] and in some specific deficiencies [33]. It seems likely that intracellular infective agents require normally functioning host cells at the time they take over and often a cell capable of replication.

By chance Maegraith et al. observed that an all-milk diet (cow's and human)
prevented clinical malaria in mice following their inoculation with Plasmodium berghei [34]. A similar effect was observed with P. cynomolgi infection in monkeys [35]. Hawking showed this effect was most likely due to the well-known deficiency of paraminobenzoic acid (PABA) in milk. Certain plasmodia cannot use preformed folate and must synthesize it from PABA so that when PABA is added to milk its protective effect against malaria is abolished [36]. Later Kretschmar was able to precipitate malaria in suckling infants exposed to infection (suckling infants are known to have high resistance to malaria) by administering PABA [37]. It is interesting to speculate why milk is deficient in PABA when the latter is so ubiquitous in most animal diets and knowing the ease with which most dietary additives are secreted by the lactating breast. We are tempted to suggest that the inability of the lactating breast to secrete PABA is another of its many defense mechanisms against infection of the suckling offspring—in this case against organisms requiring PABA for their replication.

There are other nutritional possibilities for this phenomenon in nomads. An unusual but plausible one has been suggested by Eaton and his colleagues. Milk-drinking nomads may be depleted of the antioxidant vitamin E through its deficiency in milk [38]. The already considerable oxidant stress posed by the plasmodium on the red cell is amplified by the low host levels of vitamin E so the red cells disrupt before the parasite can mature, multiply, and infect further red cells [39]. Whatever the mechanism, the result would be the same—the defective diet playing an important role in moderation of the infection.

Iron is an important biocatalytic agent involved in a variety of intracellular functions including DNA synthesis [40] so that it would be surprising if these functions were not disturbed in iron depletion. Dormancy of infection as we saw it in iron-deficient Somalis may be related then to an abnormal intracellular milieu or to reduced replication of cells. Although transferrin, the iron-binding protein in the serum, has a bacteriostatic effect on many organisms proportionately related to its degree of iron unsaturation [41], we do not believe that this is the major mechanism of these ecological compromises. The plasmodium, located as it is in the red cell, and surrounded by iron, ought never to be in short supply. The mechanism is likely to be more fundamental.

During active phases of growth or mitosis a number of factors are operating which restrain the cell mass from proliferation beyond predetermined dimension [42]. Growth-inhibiting or antimitotic factors almost certainly originate within the target cells themselves and operate by direct effect on the G1 and G2 phase of their own cell cycle [43]. During phases of reduced growth and mitosis, the stimulatory factors are overwhelmed by the inhibitory ones while during active growth the position is reversed. Applying this general principle to the diet-disease interrelationship, we envision the following sequence: as external energy sources are curtailed or specific nutrient deficiencies develop, the cells automatically turn down certain intracellular functions and their own replication in an effort to eke out the dwindling resources of nutrients. Fortuitously (or perhaps not so fortuitously) replication of intracellular organisms is synchronously impaired or the intracellular milieu so deranged that the microorganisms cannot thrive or remain dormant, much as they seem to do in hibernating animals [30], until energy supplies and active growth are restored. During the accelerated catch-up growth which characteristically accompanies refeeding after undernutrition [44] restoration of cell function or increased cell replication may permit the disease to erupt with even greater violence.
A FIELD STUDY

Perhaps the ecological effects of a recent change in the nature of diet can best be seen in a study of the Turkana of northern Kenya. On the western shore of Lake Rudolph a number of Turkana have taken to eating fish as a supplement to their all-milk diet. Other Turkana living in the same area have a strong dislike for fish and have adhered to their traditional diet of milk. We examined the incidence of infection and disease in two matched groups living under identical conditions in the same geographical area—the only difference between the two groups was the consumption of cooked fish to 150 grams a day or more by the fish eaters. Fish eaters had significantly more infections (malaria, brucellosis, and diarrhea) than milk drinkers. Common warts which are rare in the Cushite nomads were found only in the former (Table 2) [45]. Although the milk drinkers were mildly iron-deficient, we did not believe that this was the prime reason for the difference. We suspect the fish was providing some nutrients which were deficient in milk and was changing the ecological balance in favor of the pathogenic microorganisms. Impressive among our findings was the remarkable infrequency in milk drinkers but high frequency in fish eaters of serological evidence for infection with *Entamoeba histolytica* (the cause of amebic dysentery) [45]. Milk, beside being a poor source of iron, contains partly saturated iron-binding proteins such as lactoferrin which may compete in the gut with *E. histolytica* for the relatively large amounts of iron it requires for maturation and development of invasive qualities. Where the iron content of the diet is exceptionally high, as it is among the Bantu, amebiasis is frequent and severe [46].

DEGENERATIVE DISEASE

We have spent considerable time discussing infection as a model for our hypothesis but it seems likely that both degenerative disease and malignancy are also involved in ecological interrelationships with diet. Arteriosclerosis and coronary artery disease are remarkably uncommon in many tribal societies and especially milk-drinking nomads who have low levels of serum cholesterol despite their high intake of energy as fat [47]. Cow's milk has been observed to contain agents capable of lowering serum cholesterol of humans—currently called milk factors. Mann suggested that hydroxymethyl glutarate in milk could turn off the rate-limiting enzyme of cholesterol

|                        | Milk Drinking Only (M) | Milk Drinking Plus Fish Eating (F) |
|------------------------|------------------------|-----------------------------------|
| Numbers of cases       | 230                    | 231                               |
| Episodes of fever      | 4                      | 27                                |
| Attacks of malaria     | 1                      | 19                                |
| Malarial parasites seen| 1                      | 24                                |
| Episodes of diarrhea   | 6                      | 31                                |
| Clinical brucellosis   | 0                      | 6                                 |
| Tuberculosis           | 1                      | 3                                 |
| Molluscum contagiosum  | 1                      | 9                                 |
| Common warts           | 0                      | 5                                 |
| Positive serum test for infection with *E. histolytica* | 3 | 39 |
synthesis, 3-hydroxy-3-methyl glutarate coenzyme A reductase (HOMG reductase) if milk was drunk in sufficient quantity [48]. So far hydroxymethyl glutarate has not been isolated from milk but orotic acid which has a hypocholesterolemic effect has been [49]. In rats, orotic acid appears to exert its effect by inhibiting acetyl coenzyme A synthetase but its administration unfortunately produces fatty liver [50]. The nature of the milk factor remains uncertain but it has been found in the milk of many species and may be produced by bacteria. *Pseudomonas fluorescens* will increase the concentration of milk factors in milk many-fold [51]. It is possible that the nature of animal feed may play an important role in the production of milk factors and in turn in the prevention of coronary artery disease. Perhaps a sequence may be: the pregnant mother drinks the milk, transmits the factor or factors in breast milk to her infant, in doing so sets the enzymatic mechanisms of the infant liver to deal with the high fat intake and to prevent atherogenesis in later life [52]. Milk-drinking nomads amongst others are also singularly free of gallstone disease. The same mechanisms may operate to protect them from stone-forming (lithogenic) bile. Inhibition of HOMG reductase or acetyl coenzyme A synthetase may lower cholesterol in bile; without a simultaneous reduction in the bile salt chenodeoxycholic acid the tendency for cholesterol to precipitate in bile and form stones would be reduced [53].

Most of our observations and remarks on the ecological interdependence of diet and disease have been confined to warm climates but there is no reason why similar relationships should not exist in cold environments. In fact Greenland Eskimos may owe their singular freedom from myocardial infarction to an unusual long chain polyunsaturated fatty acid in their diet of fish, whale, and seal meat. Their serum lipid pattern which favors reduced risk from myocardial infarction is clearly the result of dietary rather than genetic factors [54]. While their total intake of polyunsaturates, especially linoleic and linolenic acids, is lower than their Danish counterparts, their intake and plasma level of eicosapentaenoic acid, a C-20 fatty acid with five double bonds, are much higher [55]. This fatty acid has unusual effects on the function of blood platelets. On a mixed diet a balance for optimal hemostasis is struck between the platelet-aggregating factor thromboxane A2 in platelets and a factor in the vessel wall which inhibits platelet aggregation—prostacyclin. The common precursor of both of these factors is arachidonic acid. In Greenland Eskimos the platelet microsomes convert the eicosapentaenoic acid (which largely replaces arachidonic acid in their diet) into thromboxane A3 which unlike A2 has no capacity for aggregating platelets. The hemostatic balance is shifted in favor of endothelial prostacyclin which still retains its ability to inhibit the aggregation of platelets [55]. Acute myocardial infarction is believed to be often precipitated by the formation of microthrombi on areas of endothelial damage in the coronary arteries [56]. Thus eicosapentaenoic acid, by discouraging the formation of microthrombi in arteries, may lessen the likelihood of myocardial infarction but at the price of the mild bleeding tendency which Eskimos have [54]. The net effect is much like that of aspirin. The less effective hemostatic plug of platelets may not be so important, however, in a cold climate where vasoconstriction occurs readily.

**MALIGNANCIES**

The type and frequency of malignancies vary from country to country but, as our Western style of life pervades other cultures, the nature of their malignancies more approximates our own. Primary hepatoma, a malignant tumor of the liver, is common in Africa and Asia, an incidence which has been suggested is due in part to aflatoxin produced by contamination of ground nuts (as well as corn and sorghum)
in their diet by the fungus *Aspergillus flavus* [57]. Ground nuts (*Arachis hypogaea*) native to Brazil were only introduced into Africa and Asia as a food crop this century. It might be argued that we are witnessing there the disruptive effect of the dietary change—in this case contaminated ground nuts—on a long-standing ecological balance. It may take hundreds of years for the host to adapt to the new strain.

There is another aspect to be considered here. An unusually high frequency of hepatitis-B virus (HBV) associated antigens and antibodies occur in the serum of Africans and Asians [58], with the highest frequency occurring in those with established hepatomas. Prince and coworkers have shown, however, that the risk of hepatomas is no greater in carriers of HBV in Mozambique where the incidence of hepatomas is high than in carriers in the United States [59]. The difference in frequencies of hepatoma reported from both countries (98.2 and 2 per 100,000 males per year, respectively) would then relate best to the HBV carrier rates in the two populations. But the clear relationship between increasing amounts of dietary aflatoxin and the rising frequency of hepatomas in African and Asian societies reaching both peaks in Mozambique [60] cannot be ignored. Lutwick suggest that aflatoxin suppresses cell-mediated immunity enough to allow persistence of HBV and an increased carrier rate which in turn favors more hepatomas [61]. In support of his hypothesis he cites the many immunosuppressive effects of even low doses of aflatoxin which have been observed in animal models. They include reduced resistance to injected pathogenic bacteria, impaired antibody synthesis, diminished complement activity, defective phagocytosis, less production of interferon, and depressed T cell function.

**IMPLICATIONS**

It is important in a study like this to be aware of differences other than diet which may influence occurrence of disease amongst nomadic and sedentary people living in the same area. There are remarkably few. Nomads live in small groups more widely dispersed than villagers and spend most of their time on the move tending herds. Their exposure to infection may be both more and less than villagers. Nomads live mostly outdoors but at night some may be packed closely into small portable huts. In marginally productive lands like the Sahel and the Ogaden, population density is low both with nomads and villagers. Finally, all are exposed to the same risks of infection from contact, water, biting insects, poor sanitation, and lack of public health facilities.

What are the implications of these observations not only for us but for tribal societies? Our Western diet is so varied in source and nature and so subject to new additions that we have had, and always will have, little time to adapt to the changes. Offsetting this disadvantage, however, we have the benefits of Western medical technology including clean water, adequate sewage disposal, immunization programs, and antibiotics, all of which have reduced infant mortality strikingly in this country. But our old age is still beset with diabetes, degenerative arterial disease, and cancer. While it is clear that we are unlikely to be able to modify our dietary programs to achieve the same control of infection, malignancy, and degenerative disease enjoyed by tribal societies, we can investigate the mechanisms by which these diets work and attempt to apply the knowledge to the solution of our own problems. At the same time (and we believe this is of the utmost importance), no effort should be spared to prevent hasty modification of tribal diets by changing patterns of agriculture to conform to the highly productive patterns of our own hemisphere if we are to spare tribal societies the synchronous transfer of Western disease.
REFERENCES

1. Jeffreys MDW: Vernacular maize names and some African tribal migrations. Ann NY Acad Sc 118:557–673, 1965
2. Harlan JR, DeWet JM, Stemles ABL: The origins of African plant domestication. In World Anthropology. Edited by S Tax. The Hague, Mouton Publishers, 1967, p 296
3. Truswell AS: Diet and nutrition of hunter-gatherers. In Health and Disease in Tribal Societies. Ciba Foundation Symposium 49. Amsterdam, Elsevier, 1977, p 213
4. Truswell AS, Hansen JDL: Medical and nutritional studies of Kung bushmen in northwest Botswana: A preliminary report. S Afr Med J 42: 1338–1339, 1968
5. Murray MJ, Murray AB, Murray NJ, et al: Refeeding malaria and hyperferremia. Lancet 1:653–655, 1975
6. Milner RDG: Protein calorie malnutrition. In Present Knowledge in Nutrition. 4th Edition. New York, Nutrition Foundation, Inc, 1976, pp 428–436
7. Nutrition and the body's defense mechanism. Nutrition Reviews 31:115–116, 1973
8. Murray MJ, Murray AB: Starvation suppression and refeeding activation of infection—an ecological necessity? Lancet 1:123–125, 1977
9. Lawless J, Lawless MM, Garden AS: Admissions and mortality in a children's ward in an urban tropical hospital. Lancet 2:1175–1176, 1966
10. Braude-Heller A, Rotbalsam J, Elbinger R: in Maladie de Famine. Recherches, cliniques sur la famine executées dans le ghetto de Varsovie en 1942. Edited by A Apelbaum. Warsaw, American Joint Distribution Committee, 1946
11. Murray MJ, Murray AB, Murray NJ, et al: Diet and cerebral malaria: the effect of famine refeeding. Am J Clin Nutr 31:57–61, 1978
12. Ramakrishnan SP: Studies on Plasmodium Berghei. Vincke and Lips 1948. XVII: The effect of different quantities of the same diet on the course of blood induced infection in rats. Ind J Malarial 8:89–96, 1954
13. Edington GM: Pathology of malaria in West Africa. Brit Med J 1:715–718, 1967
14. Murray MJ, Murray AB, Murray MB, et al: Somali food shelters in the Ogaden famine and their impact on health. Lancet 1:1283–1285, 1976
15. Murray MJ, Murray AB: Suppression of infection by famine and its activation by refeeding—a paradox? Persp Biol Med 20: 471–483, 1977
16. Lapiccerella V, Lapiccerella R, Abboni F, et al: Enquête clinique biologique et cardiographique parmi les tribus nomades de la Somalie qui se nourrissent seulement de lait. Bull Wld Hlth Org 27:681–697, 1962
17. Murray MJ, Murray AB, Murray MB, et al: Adverse effects of iron repletion on the course of certain infections. Brit Med J 11:1113–1115, 1978
18. Trousseau A: Lectures on clinical medicine. London, New Sydenham Society, 1972, p 96
19. Murray MJ, Murray AB, Murray MB, et al: Parotid enlargement, forehead edema and suppression of malaria as nutritional consequences of ascariasis. Am J Clin Nutr 30:2117–2121, 1977
20. Murray MJ, Murray AB, Murray MG, et al: The biological suppression of malaria: an ecological and nutritional interrelationship of a host and two parasites. Am J Clin Nutr 31: 1363–1366, 1978
21. Homandberg GA, Penasky RJ: Characterization of proteins from Ascaris Lumbricoides which bind specifically to carboxypeptidase. J Biol Chem 251:2226–2231, 1976
22. Sohonie K, Bhandarakar AP: Trypsin inhibitors in Indian foodstuffs. Part I—Inhibitors in vegetables. J Sc Ind Res 13b:503–505, 1954
23. Jaffe WG: El valor biologico comparativo de algunas leguminosas de importancia en la alimentacion Venezolana. Arch Venez Nutr 1:107–125, 1950
24. Green GM, Lyman RL: Feedback regulation of pancreatic enzyme secretion as a mechanism for trypsin inhibitor-induced hyperscretion in rats. Proc Soc Exp Biol Med 140:6–11, 1972
25. Nacchiero M, Dreling D: The parotid and the pancreas I. The effects of secretin, CCK-PZ and prostaglandin E2 on canine parotid secretion. Mt Sinai J Med NY 45:187–195, 1978
26. Scrimshaw N, Tejada C, Arroyave G, et al: Changes in the liver and pancreas in Kwashiorkor with reference to the role of antecedent infections and infestations. Proc Wld Con Gastroenterol. Baltimore, Williams and Wilkins, 1958, p 677
27. Lawrence G, Walker PD: Pathogenesis of Enteritis necroticas in Papua, New Guinea. Lancet 1:125–126, 1976
28. Murrell TGC: Pig-bel, epidemic and sporadic necrotizing enteritis in the highlands of New Guinea. Australasian Ann Med 16:4–10, 1967
29. Gill CA: Seasonal periodicity of malaria. London, J and A Churchill, 1938
30. Edwards JT: In Discussion on Nutrition and its Effects on Infectious Disease. Proc Roy Soc Med 30:1046-1051, 1937
31. Chandra RK: Immunocompetence in undernutrition. J Pediat 81:1194-1200, 1972
32. Cooper WC, Good RA, Mariana T: Effects of protein insufficiency on immune responsiveness. Am J Clin Nutr 27:647-664, 1974
33. Rasmussen AF Jr, Wausman HA, Elvehjem CA, et al: Influence of the level of thiamine intake on the susceptibility of mice to poliomyelitis virus. J Inf Dis 74:41-47, 1944
34. Maegraith BG, Deegan T, Jones ES: Suppression of malaria (P. berghei) by milk. Brit Med J 2:1382-1384, 1952
35. Bray RS, Garnham PCC: Effect of milk diet on P. cynomolgi infection in monkeys. Brit Med J 1:1201-1201, 1953
36. Hawking F: Milk diet, p-aminobenzoic acid and malaria (P. berghei). Brit Med J 1:1201-1202, 1953
37. Kreschmar W: Die bedeutung der p-aminobenzoäsure für den krankheitsueralaufund die immunität bei der malaria im tier (plasmodium berghei) und in menschhen (plasmodium falciparum). Zeitschr Tropenmennis 17:301-320, 1966
38. Webb DH, Johnson AH, Alford JA: Fundamentals of Dairy Chemistry. Westport, CT, AVI Publication Co, Inc, 1974, p 386
39. Eaton JW, Eckman JR, Berger E, et al: Suppression of malaria infection by oxidant-sensitive host erythrocytes. Nature 264:758-759, 1976
40. Brown NC, Eliasson R, Reichard P, et al: Spectrum and iron content of protein B2 from ribonucleoside and diphosphate reductase. Eur J Biochem 9:512-518, 1969
41. Weinberg ED: Iron and susceptibility to infectious disease. Science 184:952-956, 1974
42. Houck JC: Circulating factors controlling cell proliferation. Prog Clin Biol Res 5:193-215, 1976
43. Lozio BB, Lozio CB, Bemberger EG, et al: Regulators of cell division: Endogenous mitotic inhibitors of mammalian cells. Int Rev Cytol 42:1-47, 1975
44. Prader A, Tanner JM, Von Harnack GA: Catchup growth following illness or starvation. J Pediat 62:646-659, 1963
45. Murray MJ, Murray AB, Murray CJ: An ecological interdependence of diet and disease? A study of infections in one tribe consuming two different diets. Am J Clin Nutr, in press
46. Diamond IS, Harlow DR, Phillips BP, et al: Entamoeba histolytica: iron and nutritional immunity. Arch Invest Med (Mex) 9 Suppl 2:329-338, 1978
47. Biss K, Kang-Jey H, Mikkelson B, et al: Some unique biologic characteristics of the Maasai of East Africa. New Eng J Med 284:694-699, 1971
48. Mann GB: A factor in yoghurt which lowers cholesterolemia in man. Atherosclerosis 26:335-340, 1977
49. Richardson T: The hypcholesterolemic effect of milk—a review. J Food Protect 41:226-235, 1978
50. Bernstein BA, Richardson T, Amundson CH: Inhibition of cholesterol synthesis and acetyl-coenzyme A synthetase by bovine milk and orotic acid. J Dairy Sci 60:1846-1853, 1978
51. Mann CH: Milk factor—a regulator of cholesterolemia. Clin Res 27:554A, 1979
52. Reiser R, Sidelman Z: Control of serum cholesterol homeostasis by cholesterol in milk of the suckling rat. J Nutr 102:1009-1016, 1972
53. Vwill GD: Medical treatment of gallstones. J Roy Coll Phys, London 13:47-52, 1979
54. Bang HO, Dryerberg J: Plasma lipids and lipoproteins in Greenland West Coast Eskimos. Acta Med Scand 192:85 94, 1972
55. Dryerberg J, Bang HO, Stofferson E, et al: Eicosapentaenoic acid and prevention of thrombosis and atherosclerosis. Lancet 2:117 119, 1978
56. Ross R, Glomset JA: The pathogenesis of atherosclerosis. New Eng J Med 295:420-425, 1976
57. Alpert ME, Hutt MSR, Davidson CS: Primary hepatoma in Uganda. Am J Med 46:794-802, 1969
58. Zuckerman AJ: The three types of human hepatitis. Bull Wld Hlth Org 56:1-20, 1978
59. Prince AM: In Viral Hepatitis. Edited by GN Vyas, SN Cohen, R Schmid. Philadelphia, 1978, p 460
60. Linsell CA, Peers FG: Aflatoxin and liver cancer. Trans Roy Soc Trop Med 71:471-473, 1977
61. Lutwick LI: Relation between aflatoxin, hepatitis-B virus and hepatocellular carcinoma. Lancet 1:755 757, 1979
62. Kagawa Y: Impact of westernization on the nutrition of Japanese: changes in physique, cancer, longevity and centenarians. Preventive Med 7:205-217, 1978