7. THE ROLE OF TECHNOLOGICAL DEVELOPMENT IN PROMOTING DISEASE IN AFRICA

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For most countries of the world, this is an “age of development,” of planned endeavors to intervene into the status quo ante with the purpose of initiating change, both economic and social. But developmental activities often produce unexpected and sometimes vitiating consequences. The very nature of change itself (being an intervention in the physical and human environment) is predispositional to disease, although not necessarily causative of it, because it disrupts established ecologic patterns at many levels of interrelationship. This idea can be extensively corroborated in contemporary Africa.

Field evidence relating to malnutrition and three selected infectious diseases (viz. sleeping sickness, bilharziasis and malaria) is reviewed. In each case, the evidence is replete with examples of development interventions that inadvertently increase disease hazards. In regard to the infectious diseases, examples of such evidence are: (1) increased incidence of trypanosomiasis (sleeping sickness) along new road networks in Liberia and Nigeria; the cycling of trypanosomes by migrant labor between Ghana and the Upper Volta Republic; and an outbreak of trypanosomiasis near the man-made Lake Kariba; (2) increased incidence of bilharziasis (schistosomiasis), especially the more serious intestinal form, Schistosoma mansoni, around irrigation schemes, with examples ranging from the Gezira cotton scheme in Sudan to sugar schemes in

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1 This paper draws heavily from another discussion of this topic, including the psychosocial consequences of modernization. See References for Hughes and Hunter.
Nigeria and Tanzania, and tobacco farms in Rhodesia, with the dangers of further spread of the disease through presently planned agricultural schemes; and (3) an increasingly complex epidemiology of malaria in which, as a result of development activities and the emergence of insecticide-resistant vectors, sociocultural factors are playing a more involved role.

Ironically, nutritional deficiencies and diseases often accompany substantial regional agricultural development. The transition to a cash-crop economy (e.g., cocoa, rubber, cotton, groundnuts, coffee) in many cases has led to the neglect of food farming and the declining use of protein-rich sorghums and similar nutrients. Purchased foodstuffs such as protein-poor cassava, yam or plantain have become food staples, causing rural malnutrition.

Africa’s burgeoning towns are both a vehicle for, and a product of, development activities. And, as has been the case everywhere else in human history, the towns are also very effective as disease-fostering environments. Despite the provision of certain amenities, such as clinics, urban health standards are often lower than rural standards. For one thing, gross overcrowding and lack of sanitation cause deterioration in health status, and infectious diseases, such as tuberculosis and infant gastroenteritis, are rampant in towns. With regard to nutrition, the overwhelming impact of urbanization has been negative. Nutritional standards among urban migrants are unsatisfactory for a variety of reasons explained in this paper. Children are particularly prone to nutritional problems. It is well known, for example, that protein deficiency in early childhood (kwashiorkor) can cause irreversible intellectual impairment. Furthermore, for each case of kwashiorkor in African towns, diagnosed or not, there are countless other cases of subclinical malnutrition in which health deterioration is insidious and persistent.

The conclusion seems obvious, but inescapable. Because development activities have not been coordinated within a comprehensive ecologic framework, there has been a drastic deterioration in the social and economic conditions of life.

Our purpose in this paper is to focus on some of the disease consequences of technological development activities in Africa. This discussion will underscore the need to emphasize an ecologic frame of reference in research and planning.

Development programs, being an intervention into the affairs of nature, will have both intended and unintended consequences; that is to say, they enter the scene as elements in the ecologic dialectic in which all life is enmeshed, and they must be examined as such. In his *Man Adapting*, Rene Dubos has in a sense expressed the basic philosophy of this paper:

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All technological innovations, whether concerned with industrial, agricultural, or medical practices, are bound to upset the balance of nature. In fact, to master nature is synonymous with disturbing the natural order. While it is desirable in principle to maintain the “balance of nature,” it is not easy to define the operational meaning of this idea. Nature is never in a static equilibrium because the interrelationships between its physical and biological components are endlessly changing. Furthermore, man placed himself apart from the rest of nature when he began to farm the land and even more when he became urbanized. The survival, let alone growth, of his complex societies, implies that he will continue to exploit and therefore upset nature. The real problem, therefore, is not how to maintain the balance of nature, but rather how to change it in such a manner that the overall result is favorable for the human species. (1965, p. 416)

Dubos is, of course, echoing Hippocrates’
ancient observation that change itself, creating the necessity for readaptation to a complex of conditions, is predispositional to disease although not necessarily causative of it.

It is changes that are chiefly responsible for diseases, especially the greatest changes, the violent alterations both in the seasons and other things. But seasons which come on gradually are the safest, as are gradual changes of regimen and temperature, and gradual changes from one period of life to another. (Dubos, 1959, p. 116)

In this connection, let us note some of the associated alterations in life conditions affecting man-environmental relationships which, while they may not in all cases be part of the explicit goal of any given development scheme, are nevertheless involved in the course of the implementation of the scheme. Aside from major purposes abstractly stated (e.g., “to raise agricultural productivity”), what other kinds of changes which have obvious relevance to the health status of the population often come about under the aegis of “development”?

1. overall changes in man-habitat relationships (e.g., working in new farmland or under other new geographical and zoological conditions; relocation to different climatic and zoonotic areas)

2. increased population movement, mixing, and concentration (e.g., building roads, railways, and other transportation networks; migration to towns and sites of economic opportunity such as mines, factories, hydroelectric or irrigation projects; relocation of communities, etc.)

3. change of patterns of water flow and use (e.g., building irrigation schemes, dams and ponds; use of polluted water resources in undersanitized and overcrowded towns)

4. change of vegetation cover (e.g., cutting down forest or bush, altering ground cover; denudation of landscape)

5. changes in micro-environmental conditions (e.g., changes in housing, neighborhood, and settlement patterns; in house style and construction materials; in location with respect to modes of transportation, sources of water, kinsmen, etc.)

6. changes in value systems and social sanction systems (e.g., conjunction of alternative ways of life in urban environments or socially heterogeneous populations, as in large-scale economic schemes; the erosion of traditional systems in conditions of economic deprivation, as in urban slums, etc.)

This listing of what might be called the “hidden costs” of development is clearly incomplete at this stage and unquestionably could be expanded with additional comprehensive data on change projects. But let us review the extent to which a change in disease patterns has ensued from such changes in ecological situation as those suggested above. While the following selected illustrations make the point that frequently such consequent relationships do exist with some of the major pathological conditions in Africa, it should be clear that systematic epidemiological data are rare, and the data that do exist for regions or for the continent as a whole, are not comprehensive in their coverage. But they are compelling in their import—viz., that often the ecologically unenlightened introduction of a given scheme will result largely in a “robbing of Peter to pay Paul” as far as the overall improvement of health status is concerned.

THE ROLE OF DEVELOPMENT IN FOSTERING DISEASE

Trypanosomiasis (Sleeping Sickness). Sleeping sickness is a protozoan disease caused by the genus Trypanosoma, which can infect not only domesticated and wild animals, but also man.

The Kissi tribal region of northern Liberia, bordering upon Sierra Leone and Guinea, is a
prime focus of sleeping sickness in Liberia. Surveys in the early 1940’s showed that the infection rate had been pushed down by treatment from 26% to 2.5% (Veatch, 1946), but recent evidence shows that infection has increased, reaching 10% in many places. In some areas, 79% of the population are carrying *Trypanosoma gambiense* in their blood, although advanced cases are rare (Vauceil et al., 1963).

There are two sets of reasons for this endemic focus. The first deals with the traditional way of life. For one thing, the Kissi raise swamp rice in a tsetse habitat. Secondly, their migratory habits make adequate treatment difficult and many cases relapse. In addition, they live in small settlements whose surroundings are kept insufficiently cleared of vegetation, so that tsetse can breed in the immediate area. Finally, they keep pigs, which may be a reservoir of *T. gambiense* (Veatch, 1946).

Another set of reasons for the endemicity is based on “modernizing” trends and socio-political developments. The Kissi tribe lives in adjoining sections of three countries: Liberia, Guinea and Sierra Leone, and much movement takes place across the international borders for purposes of trade and of visiting relatives. Political changes in Guinea following independence caused the control practices originated by the French to be dropped, and now Liberia is open to the ingress of new cases from that country (Foster, 1963). Of late, such movement has been greatly facilitated by improved road linkages. Reintroduction of trypanosomiasis into areas which were once clear of the disease has resulted. Such diffusion of the disease is illustrated, for example, in the case of the new road through Kissi country in northern Liberia, which was constructed in 1960. Because *Glossina palpalis* is found at every river crossing, a particular hazard arises wherever a road is intersected by a stream or river; vehicles often stop at such places for water and to refresh passengers, who thereby become highly susceptible to the tsetse fly. To compound the jeopardy, tsetse flies are attracted to moving vehicles and may therefore be carried by them for some miles, a fact instrumental in spreading the disease into former tsetse-free areas (Abedi and Miller, 1964; Bequaert, 1946).

Among the Ashanti in Ghana, there is a clear illustration of the role of migrant labor, another aspect of population movement, in the dissemination of the disease and the reinfecting of controlled areas. From the 1920’s into the 1940’s many laborers from the savanna areas of northern Ghana and from other northern countries such as the (now) Upper Volta Republic, Mali, and Niger moved into southern Ghana for work. As they migrated, they passed through the tsetse belt immediately north of the forest, where they picked up the infection. Upon reaching their destination, Ashanti country in central Ghana, they became the source for spreading the disease. It spread to such an extent that Ashanti foci began to have higher rates than the north. At the same time, eradication campaigns in the northern areas were effective in reducing somewhat the incidence of infection there. With the periodic return of (infected) migrant laborers from the south, however, the process of reinfection began. Such migratory movements continue today. One of the consequences is the recycling of trypanosomes in the northern savanna (Scott, 1957).

The case of the Moshie of the Upper Volta Republic provided some supporting statistics for this phenomenon (Scott, 1957). A sleeping sickness survey in Ashanti from 1952 to 1954 showed an incidence of 1.05% among adult male Moshie. The rate for the general Ashanti population was 0.18%, that is, one-sixth of the Moshie infection rate. A concomitant survey at the Yeji ferry on the north-south migration route in 1952 to 1954 clearly shows the degree
of reinfection northward. The rate among Moshie males of ages 16 to 44, on their initial entry to Ashanti, was 0.14%; on leaving Ashanti, it was 1.44%. To a certain extent an increased risk of morbidity accompanied employment in Ashanti. While the difference in infection rates is not great in absolute terms, its demographic implication could be of much more significance, especially when coupled with the rest of the disease load under which most Africans labor. Davey (1948), for example, has estimated that in a population with a 6% trypanosomiasis infection rate, a stable population equilibrium is attained, while an infection rate as low as 3% allows only some natural increase. Other surveys, conducted along the road network connecting Ashanti with the north, show clearly that the highest incidence of sleeping sickness occurs along the major highways because such lines of communication facilitate man-fly contact (Scott, 1957).

In Nigeria transmission has increased sharply in localized pockets along the main Jos-Wamba road, a road used by migrant laborers going to the tin workings in the plateau (Thomson, 1967). Another, almost textbook, illustration of a man-made transmission site also comes from northern Nigeria, the area of Kanawa. In 1961, as a precautionary measure for laborers working on the Bornu railway extension, Kanawa was surveyed for prevalence of sleeping sickness. It was found to be free of the disease. Later, however, there was a sharp outbreak of sleeping sickness. Subsequent investigation indicated the source to be man-made; a small forest reserve was deliberately planted along the banks of a stream at the point where it was crossed by the main road—a place where people gathered to wash, drink and relax. In this case the trypanosome strain was one of high transmissibility but low virulence (Thomson, 1967).

Roads are thus linear-type transmission sites. Although systematic epidemiological data are not numerous, modern roads built for economic development seem to constitute a major health hazard in endemic regions, their very purpose being to encourage movement and mixture of peoples and goods but having, as implicated effects, the facilitation of man-vector contacts for several different kinds of insect-borne diseases. With the expanding road and physical communication networks in Africa, all of which facilitate population movement, the risks of rapid reinvasion of areas by tsetse (and consequent reinfection) are great. One obvious suggestion from the public health point of view would be the imposition of strict vigilance and control measures (Thomson, 1967; Scott, 1957).

Settlement relocation is often an etiological factor. In pre-colonial Africa, many traditional settlements were located in refuge areas, on mountaintops, on ridges, on high plateaus—inaccessible, easily defensible sites. These sites were elevated and therefore tended to be freer of fly-borne diseases than lowland sites. However, with the coming of the various European colonial regimes and the law and order they brought, settlements have tended to move down from the hills to better agricultural land on the plains. Agricultural developments of this sort have often led to a significant increase in sleeping sickness.

The Rukuba tribe of the Bauchi Plateau of northern Nigeria illustrates the point. This virile tribe came to the verge of extinction because of an epidemic of sleeping sickness. *Glossina palpalis* had inhabited the dense vegetation on the Rukuba escarpment long before the introduction of the trypanosomes themselves. After the Pax Britannica, homesteads were moved down onto the lowlands from the hilltop fortresses which had once repelled human invaders, thus bringing man and flies into ever increasing contact. During 1931 and 1932, farmers returning from Piti brought
trypanosomes first into Ikala and then Kakkek, thus providing the one biological necessity which had been lacking. Man, fly and parasite were thus brought together and a transmission cycle began, resulting in a devastating and cataclysmic epidemic. Ruins of once-prosperous compounds and farms became a feature of the Rukuba landscape (Duggan, 1962).

Another illustration of sleeping sickness as a result of opening up new farmland occurred in Nigeria. Movement onto the plains west of the Jos escarpment resulted in one thousand cases of diagnosed sleeping sickness from 1960 to 1965 (Thomson, 1967).

In 1956, the people from Muyama in the Kasulu District of Tanzania, a barren and heavily eroded area on the hillside above the Milangilizi Valley, obtained permission to go down into the valley to cultivate new fields because of pressure on available agricultural land. The valley was fertile and well watered, but much of the riverine thicket contained large concentrations of G. morsitans. For a year nothing happened, and then, in 1958 and in 1959, after man, fly, and parasite contact had been established, an epidemic outbreak of sleeping sickness occurred (Apted et al., 1963).

From the Dodos country in northern Uganda, another illustration shows how government action can trigger off an epidemic of sleeping sickness with repercussions in settlement, land use, economy and ecological balance. The government evacuated numbers of the Napore and Nyangeya tribes in 1924-25 in order to stop tribal warfare. As a result, a barrier to tsetse was removed because wild animals began to enter the territory formerly occupied (and extensively hunted in) by the Napore and Nyangeya. With the game the tsetse arrived. The advancing game and tsetse began to put pressure on the neighboring Dodos peoples. Tsetse advanced eighty miles and infested 1500 square miles of Dodos territory in the 1920’s. In the face of this tsetse hazard, cattle-keeping Dodos people were forced to migrate toward the south and west into a much more limited territory. This caused overpopulation, overgrazing, and soil deterioration, and seriously affected the health and demography of the Dodos people. However, in 1946, a tsetse clearance program was started and in three years two-thirds of the fly belt was cleared. The land which was reclaimed was in good condition after the long fallow (Deshler, 1960).

Another example of ecologically uninformed government action that resulted in an epidemic of sleeping sickness is from East Africa, in the endemic region around Lake Edward and Lake George, near the Semliki Valley in the (then) Belgian Congo. Native tradition holds that sleeping sickness had been present in the Semliki long before its occupation by the Belgians in 1896, and that it had decimated successive tribes who had tried to occupy this fertile plain. Soon after the European occupation, for greater ease of administration, the scattered agricultural population was moved into more compact settlements, which were placed on rivers to provide permanent water. In this part of the valley, the whole length of the Semliki and all its tributaries holding permanent water are infested with Glossina palpalis, the vector of Trypanosome gambiense. Consequently, the great amount of movement called for by the concentration of the inhabitants, and the position of the, settlements, brought about so much increased contact between the population and the tsetse that the existing endemic was rapidly turned into a fierce epidemic which literally wiped out many of these new villages. It was especially severe in the southern end of the Semliki Valley and around the shores of Lake Edward. Mortality was appalling. The epidemic reached its height between 1915 and 1923. In
1924 and 1925 the Belgian authorities took drastic action. The whole population of the valley was moved up onto the higher fly-free ground of the Mitumba and Ruwenzori Mountains and even deep into the Ituri forest along the new road being built from Beni to Irumu. In evacuating the Semliki Valley the Belgians eventually completely eliminated human trypanosomiasis (Morris, 1960).

Another example of population transfer forced by economic development occurred in Rhodesia, the Bumi River area, in 1958. The changes brought about by the Kariba dam produced an outbreak of sleeping sickness as well as a decline in nutritional standards (Apted et al., 1963) and overall health status (Scudder, 1966). A section of the Tonga tribe was displaced by the rising waters of Lake Kariba. They were moved to a dryer region in which their traditional crops would not ripen, but they were reluctant to use the new seed varieties given to them by the government. They therefore suffered from food shortage. When the group lived on the banks on the Zambesi River they had supplemented their diet with fish and rodents, which were not available in their new homeland. Severe malnutrition followed. A government grain store was established to help them, but because of its location, the store became a transmission site for sleeping sickness and a sharp outbreak occurred (Apted et al., 1963).

Bovine as well as human trypanosomiasis is a major disease obstacle to economic and social development in Africa. Huge areas on the African continent are infested by the tsetse fly, and the various trypanosomes which they carry are not only fatal and debilitating to man but also to domestic cattle. In the most highly infested fly belts, it is impossible to raise cattle. This produces a chronic shortage of animal protein; and protein deficiency, especially in early childhood, is one of the major health problems of Africa. As population increases, the problem will become more severe. Yet if the problem of tsetse infection could be solved in practical terms, the fly belts of Africa could be turned into productive cattle-ranching lands, thus in turn helping to solve the severe protein deficiency problem.

An experiment conducted in Nigeria illustrates the obstacles to development created by such bovine trypanosomiasis. In 1963, a herd of 28 Zebu cattle was trekked some 415 miles from a tsetse-free area in north Nigeria to Ilorin in the south. Although the sample size in this experiment was small, the implications are significant. Such a trek is typical of the pattern for the whole of West Africa, whereby cattle raised in the tsetse-free areas of the far north are trekked or conveyed by truck through the fly belt to the southern zone where there is a shortage of animal protein. The journey through the fly belt took twenty-eight days, and measurements were taken daily of the increasing load of trypanosome infection. On arrival at their destination, 39% of the herd were infected. Typical trade cattle surveyed in other studies had an infection rate of 42% (Jordan, 1965).

At their terminus, the herd of 28 Zebu were held over for a seventeen-day observation period. Within this seventeen-day period, 19 of the cattle died and of the 9 survivors only 1 was in good condition. The others were dying of sleeping sickness—illustrating the very high infectivity of sleeping sickness in the south and the obvious unfeasibility of raising cattle there (Godfrey et al., 1965).

In view of the vulnerability of the big Zebu cattle to trypanosomes and the great shortage of animal protein in the southern part of West Africa, it has been suggested that the N’dama dwarf cattle (which have some resistance to trypanosomes) should be developed for meat production in the fly belt. Veterinary opinion does not support this policy, however, because these cattle are slow-maturing, low-milk-
yielding and low-meat-yielding. One further aspect of this situation is that the tolerance of the dwarf cattle to infection is broken down when the animals are subjected (as they often are) to adverse conditions such as malnutrition, overwork, mineral and vitamin deficiencies, and intercurrent infections such as piroplasmosis, anaplasmosis, helminthiasis and rinderpest, or exposure to heterologous strains and species of the try-panosomes. At present in the fly belt and in lightly infested fly areas the only way to raise cattle is to use a supporting regimen of chemotherapy and chemophylaxis (Stephen, 1966).

**Bilharzia (Schistosomiasis).** Schistosomiasis is the disease caused by the worm *Schistosoma haematobium* or *S. mansoni*. The worm uses human beings as a primary host and snails as a secondary one. One of the chief ways of improving agricultural production in many parts of Africa is manipulation and control of water resources by dam construction and irrigation schemes. However, the reliability of water resources is not all that is ensured. In addition to creating dependable water resources, transmission networks are often produced for the diffusion of snails, which are the intermediate host of the schistosome. Indeed, the World Health Organization commented: “The incidence of bilharzia has increased but it is of man’s doing. As he constructs dams, irrigation ditches, etc., to alleviate the world’s hunger, he sets up the ideal conditions ... for the spread of the disease” (WHO, 1961, p. 431). Such schemes also tend to foster dense populations—both human and snail—and thereby increase rates of transmission. Because many new projects serve as foci for migrations of labor forces, they result in population concentration, and often the introduction of the schistosome itself (by workers from endemic areas). And, in passing, it may be noted that of the two types of schistosomiasis—the urinary or vesical (due to *Schistosoma haematobium*), and the more severe form, intestinal bilharzia (due to *Schistosoma mansoni*), it is the latter which is mainly increased by the irrigation schemes (Alves, 1958). Thus irrigation, as an example of a technological improvement oriented to increasing agricultural yield, poses many health problems. Closer attention to bilharzia implications of expanding irrigation schemes is necessary in Africa (Waddy, 1966).

Insofar, then, as the opening up of Africa means, in part, the tapping of considerable hydroelectric potential for the development of irrigation systems, it should be recognized that with every mile of water the bilharzia snail will very often appear.

Its debilitating effects will strike man down and make him an unproductive worker. And as the World Health Organization noted with respect to the planning, and therefore preventive, phase, “The successful attack on the disease will be accomplished by a team: the engineer, the malacologist, the parasitologist, the sanitarian, the physician, and the chemist, widely divergent in training, speaking in the beginning different technical languages, but with a common purpose in mind” (WHO, 1953, quoted by Odei, 1961 c). Such a plea for interdisciplinary coordination and planning is echoed by Lanoix (1958), who notes that irrigation systems have also been indicted by epidemiologists and other health authorities as the causal agents of several other diseases of man, such as enteric bacterial infections, diarrheas, cercarial dermatitis, guinea worm, polio and possibly histoplasmosis; such irrigation systems also provide suitable breeding places for dangerous insect vectors of malaria, dengue, encephalitis and filariasis. Thomas (1965), in dealing with the ecology of a small man-made lake in Ghana, has studied the great variety and number of potentially dangerous pathogenic parasites, of both man and domestic animals, for which an aquatic environment is
necessary. The numerous possibilities for the inadvertent spread of disease created by the spread of irrigation systems or the impounding of water can be seen in Appendix.

As far as general prevalence is concerned, it may be noted that bilharzia data on fourteen selected African countries collected by McMullen (1963) show that out of a population of 100 million persons, there were some 34 million infected. More than 50% of the population is infected in five of these countries: Egypt, 14 million out of 26 million; Ivory Coast, 1.75 million out of 3.1 million; Mozambique, 4.5 million out of 6.5 million; Rhodesia, 2.2 million out of 3.12 million; Tanzania, 5 million out of 9.5 million.

Other more specific rates and incidence patterns are those for French West Africa in 1952, which showed case mortality rates from vesical schistosomiasis of 0.3%, i.e., 3 per 1000, and that from intestinal schistosomiasis of 1.46%, i.e., 14.6 per 1000 (Odei, 1961 c).

Egypt, with its vast irrigation network, is the most heavily infected country in the continent. A study in 1949 claimed that “since the erection of the [first] Aswan dam, bilharzia has spread out and health and mentality of the individual deteriorated.” For example, in four selected areas in a three-year period bilharzia infection rates increased as follows: from 10% to 44%, from 7% to 50%, from 11% to 64%, from 2% to 75% (Lanoix, 1958).

Bilharzia is spreading gradually toward the west in Uganda. One study reports that *S. mansoni* is most prevalent in the Nile valley and along the shores of Lake Albert. In this disease, as in others, seasonal migration plays a role in the transmission (Nelson, 1958). In Kenya, the Lake Victoria area is one of the hyperendemic areas of Africa, with percentages for school children running up to 100%. Tanzania is likely to become a classic example of the dangers inherent in the economic opening up of Africa by irrigation and water con-
servation projects. It appears that infection is already fairly widespread throughout the country, but rates are not yet very high. However, irrigation schemes are certain to increase snail populations so that considerable epidemic bilharzia can be expected in the future (Alves, 1958).

In the Upper Volta Republic, about 50% of the population, more than 1.5 million people, are infected at some time during their life with *S. haematobium*. Yet here, as an example of a general point of this paper, an analysis in public health terms indicates that programs of bilharzia control are usually not of much practical value. One reason for this failure is that they are not combined with an integrated approach to general problems of environmental and urban sanitation. Ouagadougou, for example, has two public lavatories for a population of seventy thousand. Pollution by human waste along with refuse from houses and markets accumulate and offer harbor for flies and rats (McMullen and Francotte, 1962).

In the southwest Gambia coastal village of Kartung, the infection rates for the various age groups were as follows: 3 to 10 years, 59%; 11 to 12 years, 91%; 21 to 30 years, 52% (Odei, 1961 a). The decline in rates was probably due both to mortality and development of immunity. It may be predicted that the Volta River irrigation scheme in Ghana will result in the spread of the snail hosts and convergence of infected peoples. In north Nigeria, in the Wu-
lago region near Lake Chad, important irrigation works have been built, but in a location that reinforces an existing focus of bilharzia (Odei, 1961 a).

The Gezira area of the Republic of the Sudan provides a particularly good example of the dangers of lack of ecological foresight. The welfare and prosperity of this area is significant to the country as a whole because the Gezira provides nearly one-third of the total revenue of the country. Before 1925, the peo-
ple of the Gezira lived under primitive conditions, wholly dependent upon grain cultivation (mostly millet). Drinking water was scarce and drought was common. In 1918, work began on the dam at Sennar and it was completed after World War I. When the irrigation works were completed, cotton was planted and yielded abundantly. Prosperity came to the region.

Approximately three years after the establishment of irrigation, however, disease-transmitting snail vectors began to appear in the irrigation canals. The snail population increased from 1931 to 1953. The influx of migratory workers to the cotton fields, first from neighboring areas, then from western Sudan, and finally from West Africa, also increased. By 1954, half a million people worked regularly in the Gezira area. The total number of temporary migrants fluctuated between 150,000 and 200,000 per annum.

In 1942, attention was drawn to the ever increasing incidence of bilharzia, which, it is believed, was introduced into the area by the migrant workers from West Africa. In 1947, field investigations in northern Gezira showed a mean incidence of 21% among adults and 45% among children. Another survey showed that infection rates among males were twice as high as infection rates among females. Much of the bilharzia in Gezira is the more virulent intestinal variety, *S. mansoni* (El-Nagar, 1958).

In Nigeria, an irrigated sugar-cane scheme was recently instituted at Bacita on the river Niger east of Jebba. It attracted large numbers of people to an already endemic area. At the beginning of the season, the recruited labor force had a *S. haematobium* rate of 6.5%. At the end of the season, the rate was 20%. A preliminary morbidity survey showed the prevalence of not only *S. haematobium* but also *S. mansoni* in the area. It seems clear, therefore, that the Bacita scheme will increase the rate of transmission of intestinal as well as urinary bilharzia in this area by exposing a large number of people who are drawn there by economic opportunity (Thomson, 1967).

Another example is found in Yo, northern Nigeria in the Lake Chad Basin, where the River Yobe separates Nigeria from the Niger Republic to the north. The irrigation system functions for only four months of the year, but is sufficient to serve as a transmitter of both *S. haematobium* and *S. mansoni*. Here *S. mansoni* infected 15% of the irrigation staff and 10% of the local population. Liver enlargement was found in 18% of the adult villagers examined, and spleen enlargement in 17%. The possibility that schistosomiasis can produce serious liver damage may also be considered in this area. Here, as at Bacita, the main effect of irrigation appears to have been the attraction of more people to natural transmission sites as well as the creation of new modes of transmission through the network of irrigation (Thomson, 1967).

Irrigation schemes are being rapidly developed in Tanzania, and there is fear of increased bilharzial infection. Until recently, the only large-scale scheme was at Arusha Chini where sugar has been grown for several decades. But, between 1955 and 1961, nine new schemes were started, and, in addition, another fifteen are now proposed. Since both forms of bilharzia, *S. mansoni* and *S. haematobium*, are endemic in many areas of Tanzania, it seems inevitable that the disease will spread and become severe as irrigation networks are developed and concentrations of human populations around them increase. And in many cases, migrant workers carry infection into the new irrigation scheme and establish a focus of transmission. For example, in one particular scheme, when the labor force was examined in an initial data setting survey, it was found that 1425 (86%) out of a total sample of 1652 came from areas where *S. haematobium* is endemic, and 320 (19%) came from areas where *S. man-
soni is endemic (Sturrock, 1965).

In the Mbarali irrigation scheme, Rufiji Basin, Tanzania, development began in 1950 and settlement started in 1961. Surveys of bilharzial infection were made in 1962, 1963, and 1965. In the four-year period from 1962 through 1965, the S. haematobium rate remained stable, ranging from 9.5% in 1962 to 8.7% in 1965. The S. mansoni infection rate, however, increased from 14.5% in 1962 to 28.9% in 1965. In the age group 5 to 9 years, the infection increased from 16.3% to 39.5%; age group 20 to 39 years, 17.3% to 26.4%; age group 40 years and over, 24.0% to 35.0%. The S. mansoni rates were also differentiated according to occupation. Senior African Supervisors’ infection increased from 2.5% to 12.8%, while Tenant Laborers’ rose from 34.7% to 63.7%. Intestinal bilharzia is thus seriously increasing at both socioeconomic levels, and its prevalence is five times greater among tenant laborers, who work more in water on the irrigation scheme itself and whose villages are badly situated in relation to the irrigation scheme. The planned villages, in which the tenant laborers live, are all in the irrigated area and consist of mud huts which lack sanitation and depend for water on the secondary canal system of the irrigation scheme itself. This scheme provides numerous transmission sites. One of the obvious conclusions to be drawn is that all people moving into a new irrigation scheme should be examined for bilharzia, and infected persons treated (Sturrock, 1965).

In Tanzania, some five million people are infected. From northwest Tanzania, on the shores of Lake Victoria, figures from an unselected community sample indicate that S. mansoni infections are light but S. haematobium is rampant in the area, giving rise to hydronephrosis, ureteric lesions or non-functioning kidneys in more than 20% of the children and in over 10% of the adults (For-syth and Bradley, 1966).

In 1954, the Department of Health in Rhodesia warned that large-scale irrigation projects might well wreck the health of the country and bring the most grandiose of schemes to an end. For example, one of the first irrigation schemes established in that country after World War II has been a complete failure and is now largely abandoned because the effects of malaria and bilharzia were not calculated (Lanoix, 1958).

In Rhodesia, a study of parasitic infection among 80 laborers on a tobacco farm showed that 45 had S. haematobium, 18 had S. mansoni, and 11 had hookworm. The investigator, however, did not feel that these parasite loads impaired the performance of these laborers to any great degree (Young, 1955). But this observation was impressionistic rather than based on systematic data.

In the Republic of South Africa, bilharzia is well known in Natal and is now also being reported in the Transvaal, particularly in the eastern low veld and in the north. It is likely that the extension of irrigation schemes in these areas will make the Transvaal a much more dangerous area than Natal. The S. haematobium incidence was only slightly higher among Africans on European farms in the north and east of Transvaal than in the African reservations (Anneche, 1955). However, the S. mansoni infection rate was 68.5% on the European farms, as compared to 33.4% on the reserves. The difference presumably resulted from the irrigation on the European farms.

Again in the Transvaal, on protected farms in the irrigated low veld the infection rates for Africans were 29.3% and 49% for S. mansoni and S. haematobium, respectively. On farms without control, the S. mansoni rate was 68.5% and the S. haematobium rate was 66.7%. On the African reserves the S. haematobium rate was comparable to that of the unprotected farms, but the S. mansoni rate was
lower (Anneche, 1955).

It appears that bilharzia is spreading into Southwest Africa: *S. haematobium* and *S. mansoni* have been found in the east Caprivi Strip next to Zambia. The disease has also been found in the Okovango swamp at Maum, where previous surveys had been negative. The swamp provides an ideal snail habitat, and therefore the disease dangers are very considerable in this area. No bilharzia is found at present in Ovamboland, but a major water diversion scheme is planned whereby the Kunene River water in Angola will be conveyed through canals into Ovamboland for water supplies and possible irrigation in this densely populated area. When it is completed, bilharzia, which is present in the southern Angola rivers, will probably spread into Ovamboland (Geldenhuys *et al.*, 1967).

Other kinds of occupational activity or development schemes which involve manipulation of water resources may also assist the spread of bilharzia. In the southern part of Cameroon, for example, it is noted that rice cultivation is eminently favorable for the spread of infection, since dangerous transmission sites include slow-moving streams, ponds and marshes (Odei, 1961a). Liberia serves as another example. There, bilharzia is an occupational disease among women who spend a great deal of their time washing clothes along riverbanks. In the marshes of the coastal areas in Portuguese Guinea women grow the main crop, rice. They consequently develop a high rate of vesical bilharzia (Odei, 1961c). At another development project in a low-lying area of eastern Kenya, a rice scheme was developed which was a focus for bilharzia; in this case, however, attempts are being made to control the snail habitat. In rural areas where bilharazia infection is endemic, such scattered foci present a very difficult problem for public health planners because any water-connected development activity, such as a rice scheme, or the construction of fishponds, compounds the problem immeasurably (Fendall and Grounds, 1965b).

Numerous other deleterious effects of the disease can also be noted. On the basis of a study in Dakar, it is believed that schistosomiasis may give rise to cirrhosis (Charmot, 1954). Furthermore, it is possible that bilharzial infection can adversely affect nutrition. Disorders in the conversion of the amino acid tryptophan to nicotinic acid (the vitamin niacin) have been encountered in bilharzial cases. This would be significant in the upset of protein metabolism and in the increased likelihood of pellagra, another extensive public health problem in Africa (Mousa *et al.*, 1967). Regarding another complication, King (1965) reports that Bantu mineworkers in South Africa who have schistosomiasis develop irreversible urinary lesions with genito-urinary complications.

In this discussion, we cannot further develop the associated physiological concomitants of the disease. That is more properly done by specialists in the field. We would, however, with Farooq (1964), point to the widespread problem of decreased labor output in areas of endemicity.

During 1962 and 1963, an attempt was made to assess the economic effect of chronic schistosomiasis among a labor force of young male African adults employed on an irrigated estate at Arusha Chini in northern Tanzania. Some 75% of recruits in 1963 had current or previous experience with schistosomiasis. Workers lived in camps provided with borehole water, washing places, and latrine facilities. But where irrigation water ran close to habitation, it was used for domestic purposes. Because latrine facilities were available in the camp but not in the fields, contamination of canals and reservoirs by infected persons was widespread. Transmission continued. Date on daily absenteeism indicated that schistosomi-
asis was second only to injury among principal causes of lost working time (Foster, 1967a).

Estimates were made of the annual cost in lower productivity ascribable to schistosomiasis. Without control, the cost of schistosomiasis is £ st. 2.6 per worker per annum. The cost is made up of the following four items: direct treatment, additional absenteeism, additional inpatient treatment, and additional outpatient treatment. With control, the annual cost of schistosomiasis per worker is £ st. 5 per worker per annum. Control costs are made up as follows: molluscicide, additional staff, transport and application, treatment of new labor and dependents. With control, and the resulting increase in worker productivity, the saving is £ st. 1 per worker per annum (Foster, 1967b).

In Africa, the distribution of the snail hosts of bilharzia is more widespread than the distribution of schistosomes. Therefore, the disease can easily spread over a much wider area than it covers at present. Since man is the primary carrier of the disease, its spread is greatly fostered by migration, by concentration around water holes, and by development of new ways of farming which implicate surface water resources (Odei, 1961c). A technique for measuring the spread of bilharzia that might be of help to epidemiologists in the development of preventive measures is to ascertain the incidence of the disease by age groups. Normally, the measurable incidence, if not the actual incidence, is highest in young persons and somewhat lower in the adult population. In areas where it is believed that the disease has been recently introduced, measurable adult infection rates are significantly higher. Figures on incidence by age groups would help to portray the geographical spread of the disease.

Malaria. There are three types of malaria in Africa, caused by three different but closely related parasites: *Plasmodium falciparum*, *Plasmodium malariae*, and *Plasmodium vivax*. *Plasmodium falciparum* is the most common. The vector of the disease is the anopheline mosquito; two varieties are widespread and relevant to the dissemination of the parasite in Africa: *Anopheles gambiae* and *Anopheles funestus*.

When malaria does not kill, it debilitates. Victims develop a variety of symptoms: anemia, fever, high blood parasite level, and spleen enlargement. Populations in malarious regions are also characterized by high infant mortality and a greater susceptibility to many kinds of infection; in addition, pregnant women frequently suffer miscarriage as a result of the disease (Cannon, 1958). Thus, malaria contributes to the severity and exacerbation of a variety of morbid conditions in a population.

With malaria, as with bilharzia, the construction of irrigation schemes and expansion of agricultural programs has facilitated the spread of the disease; indeed, the spread of malaria in Africa was probably facilitated from the beginning by man’s own constructive activities. Livingstone, for example, notes that the clearing of tropical forest for agricultural purposes and the establishment of thatched-roofed permanent villages provided ideal habitats for *Anopheles gambiae* (1958). Similar processes, and similar effects, continue today. Wilson (1957) made a plea for coordinated environmental planning in this respect. The fears he expressed were realized in the Taveta area of Kenya and Tanzania when water from springs in the Kilimanjaro area was diverted for irrigation purposes and thereby provided ideal breeding sites for mosquitoes (Smith and Draper, 1959).

The fact that mosquitoes cannot transmit malaria directly to each other makes man’s role in its diffusion a primary one; and widespread population movements in Africa, both those linked with traditional patterns such as pastoral nomadism and those linked with
modern developments such as labor migration, are among the outstanding contributory features in this respect. As such, migrations both facilitate the spread of malaria and create serious difficulties in its eradication. Since most of these population movements are essentially uncontrolled and many are interterritorial and international, they often hamper effective eradication and control programs through the reinfection possible in uncontrolled areas. In fact, the eradication and control of malaria in Africa today increasingly has more to do with understanding and control of the human relations aspect of the problem than with the biological aspects. The techniques of the malariologists and entomologists are well established, as are methods of eradication (the main purpose of which is to interrupt the transmission cycle); but what remains is the articulation of these techniques within widely differing physical and sociocultural environments. Population instability and movement is only one consideration; the ways in which a population is distributed, its settlement patterns, house types, farming practices, communication patterns, and water-use patterns, are also important aspects of a malaria eradication campaign. It is in the area of the complex interrelationships among the parasite, the vector, and man that further data and studies are needed (Prothero, 1961; Fonoroff, 1963).

Malaria and attempts to control or eradicate the disease prove that the quest for health is continuous and that there is only temporary respite. Malarial programs are waging a continual fight to keep ahead of the proliferation of insecticide-resistant strains of the insect vectors which, through processes of natural selection, adaptive capabilities, and enormous reproductive capacity, are multiplying the disease threat. In a sense, the more control is attempted, the more the problem is exacerbated because of the need for new research to develop more effective and more specific insecticides. It might almost be said that unless the transmission cycle is broken at other points, insecticide spraying of the insect vector creates a new environment, an environment of “development,” and is an attack on a problem which itself has been created by earlier spraying—a “disease of development.”

This point has been documented in a number of studies. The study by Bruce-Chwatt (1956) with regard to DDT and dieldrin is a good example. After a four-year spraying campaign in the region of Thies, Senegal, the parasite rate among children under age fourteen was reduced from 22% to only 1%. However, the disease rebounded to 16% within only a year. In the opinion of the researcher, this rebound occurred because of the adaptation of the mosquito vector to the new environmental conditions (Escudie and Abonnenc, 1958). In the Para area of Tanzania, there was another outbreak of malaria transmission based on similar factors. The spraying campaign ceased in 1959, and thirteen months after the final round of dieldrin spraying, parasite rates had sharply increased, from a low figure of about 5% only six months after the cessation of the spraying campaign, to an average rate of 30% for children from 2 to 9 years. The factor causing the rebound was the development of a new strain of \textit{Anopheles gambiae}. Because the transmission cycle was not completely broken by the spray campaign, human susceptibility to the disease actually increased rather than diminished (Pringle, 1967).

Resistance of mosquito vectors to insecticides has three forms: (1) simple behavioristic, (2) excito-repellency, (3) physiological. Behavioristic resistance occurs when an insect population changes its habits to avoid contact with the insecticide. For example, mosquitoes may try to rest on clothing or furniture or leave the house immediately after taking a blood meal, instead of resting on the walls of a
Excitobompellency is a form of hypersensitivity to an insecticide. When a mosquito comes into contact with the poison, it may take off again before it has received a lethal dose. Physiological resistance arises when strains of mosquitoes emerge which are genetically tolerant to doses of toxicants which would be lethal for the majority of the mosquito population of the same species. It is this latter kind of resistance which poses such a severe problem and which has developed as a result of man’s own activities in spraying campaigns. In 1967, the WHO Expert Committee on malaria reported that twenty-four vector species of Anopheles show resistance to dieldrin, DDT, or both. In sixteen countries, eleven of these twenty-four vector species showed double resistance, which is much more difficult to counter than single resistance.

It is therefore clear that no single method will rid the world of malaria. Combination programs incorporating techniques such as spraying, draining marshland, clearing of the bush, and mass chemotherapy and chemoprophylaxis have the best chance of success although even with these measures it should be recognized that there may be a serious problem of drug resistance to the chemicals. Although such programs might be expensive to mount and complicated to administer, there is no question of the overall gain in terms of worker well-being and productivity. In the Transvaal and Natal, for example, malaria control programs decreased worker absenteeism by at least 30%; and in Rhodesia an antimalaria campaign reduced absenteeism during the harvest season in the Mazoe Valley from 25% to almost nothing (Winslow, 1951, pp. 24, 25; quoted in Taylor and Hall, 1967).

It is not appropriate here to enter into complicated matters of the economics of labor supply; but insofar as much of the worker absenteeism in developing countries is of an erratic and aperiodic nature (thereby making permanent replacement of trained workers from the unemployed pool difficult to accomplish), one further observation is relevant to the general issue. Calculations by Mushkin are reported to the effect that in a hypothetical developing country, with 80% of its population affected by malaria, for example (which would decrease productivity of agricultural workers by 30% during a three-month period), the output loss in the agricultural sector would be 6%. If agriculture accounted for one-third of the total output, the gross national product would be diminished by 1% for that period alone; and over a twelve-month period the loss would be 4% (Taylor and Hall, 1967). Of course, the specifics of such calculations might well vary; but what is quite clear is that sick workers simply do not work as well, nor as regularly, as those who have their health. Even an effective antimalarial campaign brings other problems. First, the complete eradication of malaria from holoendemic areas will increase susceptibility so that any attacks that do occur after the campaign will tend to be more crippling to adults. Thus, in the long run, partial control may prove to be a social detriment. Secondly, the demographic effects of an antimalarial campaign will include a rise in the rates of population increase. In fact, the rate of increase may exceed that of food resources. Other problems, such as malnutrition, may arise because antimalarial campaigns lead to a sharp increase in birth rates and sharp decrease in death rates (Learmonth, 1954; Cannon, 1958). In the short run, too, it is foreseeable that a sharp reduction of infant mortality rates (as, for example, from a successful antimalarial program) will lead to protein-calorie malnutrition because more surviving infants are competing for limited supplies of breast milk and other protein supplies, thus, even an effective antimalaria campaign may at best substitute complex new problems for old ones.

Rural Malnutrition. Many traditional Af-
African diets provide an excellent and well-rounded nutritional regime, especially where there is no population pressure and no cashcropping. For example, the Karamojong of Uganda possess many cattle, which are the mainstay of their economic and social life. Sorghum, milk and blood are the main foods and meat is eaten in times of famine. In this situation, protein-calorie malnutrition is rare (Jelliffe et al., 1964). The Mabaans occupy the bush country in the southeastern part of Sudan near the Ethiopian border, and they, too, have an excellent nutritional status; there are no nutritional deficiencies. Their major foods, all rich in protein, are guinea fowl, rodents, game, millet, sorghum, nuts, and dried fish (Rosen et al., 1962). In short, many indigenous diets are good diets which include a high proportion of protein-rich foods. Considerably more research, however, is needed in this area (Jelliffe, 1955).

On the other hand, it should also be noted that in numerous cases culturally based food taboos prevent consumption of animal protein which is available for either an entire group or for special classes of persons in the group. These food taboos play a part in denying children some of the vital nutritional elements required. In the Ankole area of Uganda, for example, many weaned children are deprived of goat milk because the goats are not used for this purpose; of eggs, because they are believed to be unsuitable for human consumption; and of meat and fish, or even an adequate quantity of cow’s milk, because of the expense. The value of vegetable protein, as in beans, peas, and ground nuts, is likewise not appreciated (Cook, 1966).

The introduction of cash crops in the colonial period often led to the neglect of traditional diets and proper food production. In many African countries, a high proportion of the men engaged in farming are still concerned primarily with cash-crop production, and the vigorous efforts to increase cash-crop yields have often been at the expense of subsistence farming. This is observable, for example, in the cocoa and coffee-growing districts of West Africa, where the best lands are used for cash-crop purposes. In many areas such a concentration on cash-crop production has reduced the quantity of locally produced food. Meanwhile, income derived from cash crops is subject to wide fluctuations and is not in every case wisely spent or wisely spread out. Lump sums derived from bulk sales tend to be quickly spent on items such as education fees or clothes and status symbols, leaving little for food purchases (Hendrickse, 1966).

In this connection, a study of the effects of cocoa production in West Africa are pertinent:

Economically, the most important finding for the country which came out of our survey concerns the cocoa villages. Cocoa is one of the best cash crops in the world, giving the highest cash yield for the smallest energy output. One might therefore expect the cocoa villagers to be well off, well fed, happy and gay. We found exactly the reverse. The people were dull, apathetic and unhappy. Their villages were run down, dirty and dilapidated and their children naked, pot-bellied and sickly. The reason for this is that it is not enough to introduce a highly paying cash crop to an illiterate peasantry and expect them to profit by it. What happens is that it tends to kill their traditional life, merely putting money in their pockets for a short period in the year, during which time they enjoy themselves. When the money gets scarce, months before the next harvest, they find themselves short of everything. In a pure cocoa village they have given up most of their land for cocoa and are no longer able to till the ground for food... Hence, with their money running out they can only buy the cheapest of food, e.g. cassava and yam.... At Igun [West Nigerian cocoa village] the villagers are apathetic, complaining it asked questions, and appear devitalized and sick. (Collis et al., 1962b, pp. 223-24)

Cash-crop production of mainly coffee and
cotton in the Kilimanjaro District of Tanzania, mostly by the Chagga people, leads to some neglect of local diets. The main crops are bananas, pulses, maize and yams. The diet is very high in starch and, not surprisingly, kwashiorkor is the main form of protein-calorie malnutrition (PCM) found in this area. It is observable in 2% of the children (Marealle and Kazungu, 1964). The real incidence of PCM will probably be much higher than the 2% figure for kwashiorkor, since subclinical PCM and post-PCM cases should be taken into consideration for such an assessment.

There are other examples of deleterious effects on diet and nutritional health following government programs. The Hadza (or Watindiga) are a small hunting and gathering tribe living in the tsetse-infested savanna adjacent to Lake Eyasi in northern Tanzania. It is believed that they may be related to the Bushmen of South Africa. Their diet consists of grain and gathered fruits, seeds and berries. Intercourse is forbidden during the prolonged lactations. At five months children are given bone marrow, seeds and pre-chewed meat. Overall, their nutritional status is excellent, and dental caries are almost absent. But tsetse clearance programs are changing the eco-logic and nutritional status of this group. Because of tsetse clearance, surrounding tribes are encroaching upon Hadza territory, and diet changes are inevitable. Corn meal, which can bring PCM and pellagra, is starting to make inroads on the diet (Jelliffe, Bennett et al., 1962).

The Lugbara, a large tribe living on both sides of the Congo-Uganda border, traditionally had a high-protein diet. Feeding and weaning of infants was satisfactory. The tribe had a high intake of beans, milk, peas, ground nuts, and sesame seeds; and their main food crops were millets and sorghum. Such a diet kept PCM incidence low. Following famines in the West Nile District, however, a law was introduced in 1950 requiring every householder to plant half an acre of cassava, a plant with very low protein content. The crop has now become a secondary staple, and if its cultivation further increases, as it may well do, the nutritional implications are obvious (Jelliffe, Bennett et al., 1962b). With mounting population pressure in rural areas, there is an inevitable tendency to change from protein-rich staples, such as millet and sorghum, to carbohydrate staples, such as cassava, yams and plantain. Areas of high rural population density are usually characterized by a dependence upon starchy staples, which yield more calories but less protein to the acre. For example, cassava produces four to five times the calories per unit area as does millet:

<table>
<thead>
<tr>
<th>Table 7-1</th>
<th>CALORIES PER HECTARE OF AFRICAN CROPS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cassava</td>
<td>7090</td>
</tr>
<tr>
<td>Maize</td>
<td>2845</td>
</tr>
<tr>
<td>Rice</td>
<td>2151</td>
</tr>
<tr>
<td>Sorghum</td>
<td>1854</td>
</tr>
<tr>
<td>Millet</td>
<td>1530</td>
</tr>
</tbody>
</table>

However, from the nutritional point of view, protein supplies are vital. Comparative protein content of various African foods are shown in Table 7-2.3

In such areas with considerable dependence upon starchy staples lacking supplementation, PCM is usually found. For example, in the Kayonza District of Kigezi, Uganda, a densely populated area in which the major food staple is plantain, a high incidence of PCM is found among the Bachiga children. This has occurred because of pressure on the land (Jelliffe et al., 1961).

When regional surveys of foodstuffs are taken into consideration with population density, they give a good indication of patterns of nutritional risks. In the Buganda region of Uganda, for ex-

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2 Table from J. F. Brock and M. Autert, 1952.
3 Ibid.
ample, there is a heavy dependence upon plantains (46%), at the expense of cereals (11.3%), pulses (9.7%), and animal products (4.2%). These figures express the percentage of total calories derived from the sources indicated. In Buganda therefore, protein intake is low, especially when compared to the northern region of Uganda, where total calories derived from cereals, pulses, and animal products were 39.3%, 24.1%, and 5.2% respectively. While the protein intake in the northern region is much better, cassava has recently spread into northern Uganda and at present accounts for 24.2% of total calories (Burgess, 1962).

The development of hybrids is often hailed as the agriculturists’ answer to the problem of population increase. And although hybrids do produce spectacular increases in yields, it is sometimes the case that protein content in hybrids is lower than in unimproved varieties. For example, Japanese hybrid rice has a 5% protein content (as compared to 10% for other rice), and U.S. hybrid maize has a 6% to 8% protein content (as compared to Nigerian or Brazilian maize with 10%). Allowances should be made for these differences in rural development planning; in particular, the amino acid composition of those food crops selected for promotion in rural areas should be carefully considered.

Much of the acute rural poverty on the South African reserves, where there is heavy soil erosion and serious malnutrition, is a consequence of the phenomenon of urbanization, for the rural hinterlands of towns serve as labor reservoirs. Government restrictions on movement mean that wives and children are left on the reserves, in many instances to fend for themselves, while the men work in the towns. Food production, consequently, has fallen rapidly behind food needs on the Bantu reserves (Fox, 1954).

A study of an African reserve in northeast Transvaal (Sekhukuniland) clearly shows some of the severe social and nutritional problems. Soil is poor and rainfall is highly seasonal and sparse—averaging only 23 inches per year. Women and children form the bulk of the population because the able-bodied men have migrated to the cities. Child wastage because of malnutrition is an important problem. This study, among numerous others, illustrates the nutritional stress of the transition from tribal life to wage-economy in South Africa (Waldmann, 1960).

In one health survey of a rural Zulu community in southwest Natal, in the Polela District, it was shown that about three-fourths of the males between 25 and 40 years are absent from the community most of the time. Although from 1945 to 1955, health, education and welfare services aimed at improving the nutritional status of infants and at developing preventive measures pushed down the infant annual mortality rate from 202 to 86, the present high mortality levels caused by poverty and malnutrition cannot be reduced without economic assistance (Bennett, 1960).

### Table 7-2

<table>
<thead>
<tr>
<th>Grams of Protein Per 100 Calories</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dried fish</td>
</tr>
<tr>
<td>Soya (whole dry seed)</td>
</tr>
<tr>
<td>Beef</td>
</tr>
<tr>
<td>Beans or Peas</td>
</tr>
<tr>
<td>Ground nuts</td>
</tr>
<tr>
<td>Millet (Pennisetum)</td>
</tr>
<tr>
<td>Sorghum vulgare</td>
</tr>
<tr>
<td>Maize (whole meal)</td>
</tr>
<tr>
<td>Yams</td>
</tr>
<tr>
<td>Plantain</td>
</tr>
<tr>
<td>Cassava (fresh)</td>
</tr>
<tr>
<td>Cassava (flour)</td>
</tr>
</tbody>
</table>

**THE ROLE OF URBANIZATION**

The relationship between “development” and urbanization is obviously not a simple one.
But that there is a relationship of evolving complexity in today’s context of accelerating urbanization all over the continent is quite clear (Steel, 1961). Many of the tangible instances of “development” schemes (schools, amenities, multifarious goods, occasional wage-jobs, health clinics, etc.) are concentrated in towns and cities, and whether accurately or not, the city or town is taken as the exemplar of the process of betterment. It thus serves as the magnet drawing population from a hinterland, and creates that mix of peoples and ideas which, in the absence of ameliorating influences, is commonly predispositional to diverse pathology (Brockington, 1958, Chap. 10). The growth of urban areas in many parts of Africa today may therefore be taken as at least a corollary, if not a simple result, of developmental ideologies and programs. But it is a corollary which includes many ecologically based conditions conducive to widespread pathology. Such pathology is both physically rooted (as in crowded and unsanitary living conditions) and psychosocially rooted (as in widespread conditions of anxiety and stress deriving from economic deprivation, unemployment, physical threats, insecurity and social alienation).

Recalling the pathogenic conditions accompanying the industrial development of the Western world, a fairly recent summation (1959) by the World Health Organization of African urban conditions is pertinent, and essentially unchanged in its gross details today:

The situation created by the rapid urbanization of Africa was examined by the WHO Regional Committee for Africa at its ninth session in Nairobi in September 1959. The influx of immigrants into the towns leads to overcrowding, which in turn produces water shortages, overloading of existing sewage disposal systems, the creation of fresh sanitary problems in towns without such systems, and the risk of spread of infectious diseases. The rural immigrant ... is the least adequately protected of all town dwellers from the standpoint of environmental sanitation because of his poverty and his ignorance of the ways of the town. He is poorly housed and badly fed; he is without access to a wholesome water supply; his personal hygiene is of the lowest standard. Sanitary measures seem to be designed less to protect his health than to protect the town community from any infection he might carry. His attempts to earn a living by hawking food and drink, for example, are frowned upon. Householders are prevented by anticrowding measures from giving him cheap shelter. His very presence in the town is discouraged, not only by the health authorities but also by the police. He accordingly settles outside the town, entering it daily to look for employment; and the overcrowded, unsanitary hovels in which he and his fellow-immigrants live form the peri-urban slums, the “shanty towns,” of modern Africa. From the sanitary aspect Engels’ description of the Manchester slums in 1844 is applicable to these shanty towns. Sanitation is non-existent, and open drains run down what passes for streets. The shanties are built of mud and wattle, old packing-cases, or kerosene tins, with tattered blankets as doors. Children crawl among the uncollected rubbish or in the drains. Water has to be fetched from a pump, well or tap, and may be contaminated. The atmosphere is unlikely to be polluted, as it was so often in Industrial Revolution towns, and the warmer climate leads to life being spent out of doors to an extent not possible in more northern latitudes; but the climate also encourages vast numbers of flies and mosquitos and, in places, the snails which transmit bilharziasis. Occasional floods convert the ground into a quagmire. Malaria, the diarrhoeal diseases, tuberculosis, bilharziasis, and the helminthiases all abound. Malnutrition is common, with its train of deficiency diseases and kwashiorkor. The destruction of tribal traditions and the general social disorganization lead to alcoholism, prostitution, and venereal disease; and mental disorders are frequent. Morbidity and mortality are both high. (WHO, 1960, p. 176)

Urbanization is perhaps the most salient social and economic feature in the life of Africa today. While populations of African countries are doubling in a period of from twenty-five to forty years, African urban populations are dou-
bling in less than fifteen years. The towns in Senegal have increased by more than 100% in a decade. Enugu, in Nigeria, was an empty site in 1914 and now has a population of more than eighty thousand; Ibadan, also in Nigeria, has trebled its population in twenty years. And much of this population concentration is packed into the peri-urban fringes, slums and shanty-towns. A recent housing survey in Jinja, Uganda, for example, showed that one-third of the workers live in mud-and-thatch- or metal-roofed huts on the fringe of town, and such peri-urban slums are a constant feature of the large cities and towns (Fendall, 1963). Migrants to these slums often lack a good understanding of how to operate in a monetary economy, and face the stresses of unemployment and social heterogeneity often without the support of traditional relationships (Fendall, 1963; Little, 1966).

Such social heterogeneity—particularly in the “new” towns and cities of Nairobi, Johannesburg, Abidjan, Dakar, Freetown, Accra, Lagos, and Kumasi—is manifested not only in economic and wealth differentials, but also in considerable tribal and linguistic diversity (H. Kuper, 1965). In Accra, for example, more than sixty ethnic groups are represented. Many people are strangers to each other; they huddle together in areas where there are kinsmen who speak their languages; they feel a sense of estrangement from much of their environment. An additional socially disruptive feature is that many towns (especially in industrial or mining areas) have an excess of males, particularly single males, or married men without their families.

The stresses which affect life are therefore not simply those relating to the traditional concerns of public health; they also derive from the psychosocial dimension of a human environment. For man lives not only in terms of physical parameters; he is also enmeshed in a framework of social relationships which constitute varying patterns of threat and support. He functions within a symbolically transmitted framework of values and orientations which define the nature of legitimate goals, means, and aspirations. One salient feature of the widespread phenomenon of urbanization in Africa today is the disruption of these frameworks of social relationships and value systems. This social aspect of the urban context is described by us in another work. (Hughes and Hunter, 1968). Here we shall review some of the primarily organic pathogenic conditions, focusing on communicable diseases and malnutrition.

Nonpsychological Disorders and Pathogenic Conditions: Tuberculosis and Other Communicable Diseases. The association between health status and overcrowded, unsanitary conditions is well known in public health literature. Africa’s towns are no exception to this maxim; they serve as the breeding ground and reservoir for many diseases of mass contact: whooping cough, smallpox, polio, malaria, typhoid, gastroenteritis. Many examples of overcrowding can be cited. There is the case of Kericho, a small town in Kenya, where two-fifths of all families live in single rooms (Fendall, 1959).

Tuberculosis is commonly associated with other diseases such as malnutrition, anemia, helminth diseases, and malaria. Not unexpectedly, then, tuberculosis in Africa is primarily an urban slum disease. In Kenya it is of fairly recent origin. Reports of the disease from the coast early in this century describe it as “galloping consumption,” of acute onset and rapid fatality. Immediately after World War II the disease assumed primarily an urban locale, and in the last twenty years the most prevalent type of tuberculosis has changed from tuberculosis septicemia to chronic pulmonary tuberculosis. A 1948 survey estimated a rate of proved and suspected cases at 11.1 per 1000 for the country as a whole.

A WHO survey of 1958 and 1959 in Kenya confirmed that the disease was more prevalent
in central and coastal regions than elsewhere. Except for the Lake Victoria region, these regions have higher humidity and greater population densities than anywhere else in the country (Fendall and Grounds, 1965a). Indeed, the disease is most closely tied to the density of population rather than to any other single feature of the urban milieu. A study in Kenya corroborates the finding of the highest rural rates in areas of highest population concentration: the Myeri district, an area in which the population has almost doubled in the last twenty years, is associated with a concomitant precipitous increase in tuberculosis (Turner, 1962). Another study similarly reports the highest prevalence in the Zambian countryside in those areas of highest population density (Grave and Ililonga, 1962).

A TB study of forty-four thousand African children in Rhodesia showed the highest rates in urban areas (Shennan, 1960). Another study of children in the Kano area of Northern Nigeria showed an infection rate in the congested central area almost twice as high as the infection rates in the better-quality housing of the suburbs and villages (McArdle, 1961). And a general WHO summary of TB surveys in African countries shows that the highest rates are found among men rather than women, and in urban rather than rural areas. These data support the inference of a close relationship between the migration of labor to congested urban areas and the prevalence of the disease (Roelsgaard et al., 1964).

Through migration, especially labor migration, TB, originating in urban slum areas, is being transmitted to the rural areas in Africa. These areas then become additional foci (e.g., Fendall and Grounds, 1965a).

Migrants, for example, come to town for work and contract TB there, and then often return to their rural homeland because they are too sick to work. One study traces the diffusion of the disease from Abidjan to rural areas in the Ivory Coast (Delormas, 1960). Another example of TB spread by such migrant labor occurs in Gao. Many farmers of Gao apparently contracted the disease while they were working or searching for work in Ghana. One practical implication would be the imposition of control and inspection of migrants returning from Ghana to Gao (Plessis et al., 1959).

**Urban Malnutrition.** Protein-calorie malnutrition is emerging as a significant force in the rapidly growing towns of Africa as well as in rural areas. Urban diets, in fact, are often deficient in vitamins, minerals, and proteins. These deficiencies occur, in part, when important nutritional elements are lost in the processing of grains and cereals (Jelliffe, 1962; Mead, 1955). Traditional methods of preparing these plants for consumption in the countryside do not result in comparable vitiation of nutritional elements. One study of polyneuropathy (a condition strongly related to pellagra) in Dar es Salaam, Tanzania, indicates that the condition is probably of nutritional origin and could be based on the use of highly refined (and therefore niacin- and thiamine-deficient) maize flour (Ebrahin and Haddock, 1964). Kwashiorkor and marasmus were found in some 2% of the children in a sample in Dar es Salaam. These diseases occurred because of high starch diets and, in some cases, early weaning followed by poorly administered artificial feeding (Marealle et al., 1964).

The Acholi peoples inhabit the north central part of Uganda. In a study comparing rural Acholi with urban Acholi the latter were found to have a higher rate of PCM, presumably because of the substitution of maize flour and other foods for traditional vegetable proteins, including millet, sesame seed and ground nuts (Jelliffe, Bennett et al., 1963). In Durban, where 1565 cases of kwashiorkor in children admitted to clinics were studied, the peak age of prevalence was 1 to 3 years. Fifty-three per cent of the children died, and in some instances
their condition was complicated by pneumonia and gastroenteritis. Excessive use of starch products was judged to be the main reason for the poor diets of these urban Africans (Scragg and Rubidge, 1960).

Several factors are implicated in urban malnutrition. One of the chief culprits is money and the high cost of protein-rich foods. The urban environment, to a much greater extent than the rural, revolves, of course, around a money economy. This factor is the core of many aspects of the malnutrition often found as a concomitant of the shift from rural to urban areas (Mead, 1955). A study done in West Africa, for example, indicates that malnutrition is more common in urban and peri-urban areas than in rural areas. It states that the value of money income in the towns is diminished by (1) obligatory expenditures such as rents, transportation expenses, and clothing; (2) by higher food prices; and (3) by having limited and even no access to home-produced foodstuffs (Hendrickse, 1966). But there are other features in a monetary economy which are detrimental to nutritional standards. One study, for example (Fendall, 1963), shows that twice as much is spent on food in the first ten days immediately after payday than in the last ten days of the month. Similarly, three times as much is spent on alcohol in the first ten days than in the last ten days.

When men work in towns and live without their families, they frequently feed themselves much less adequately than when they live in the rural areas. They lack women’s expertise in food preparation and purchase. They often eat in canteens which provide monotonous and poor-quality diets.

Living in towns also frequently leads to extramarital liaisons which involve a man in a new set of familial obligations under which either his village family or his city family will suffer financially (Hendrickse, 1966).

Because of less effective child care in the towns, urbanization frequently results in infant malnutrition. In the towns considerable pressure is placed on young mothers to go to work. In their mother’s absence, babies from two to three months old are often cared for by children from seven to ten years old. The diet for such babies is often poorly prepared canned foods. This poor diet can produce gastroenteritis and eventual malnutrition. Kahn (1962) also reports kwashiorkor in children of working mothers.

Not only are there fewer relatives to help care for the children in towns and cities, but there is also less sunlight and therefore more rickets. In addition, there tends to be earlier weaning in the town than the country, with consequent intake of less protein. Subsistence depends mainly on expensive and poorly carried out artificial diets (Jelliffe, 1962).

Ignorant and uninformed adoption of European-type baby bottle-feeding also contributes to infant malnutrition in the towns. The practice of feeding powdered milk to babies is rapidly growing in urban areas, but this practice is being used by women with no knowledge of hygiene, no ability to read the instructions on the can, and no money with which to buy sufficient powdered milk. Thus, diluted powdered milk from dirty bottles and dirty teats is substituted for breast milk. This leads to malnutrition and dietary disorders such as marasmus, diarrhea, or vomiting (Cook, 1966). Welbourn (1958) also comments on the uninformed use of bottle-feeding and its deleterious effects. In Uganda, bottle-fed children were two pounds lighter in average weight than breast-fed children.

It is worth looking at some specific studies of relationships between overall physical and mental development and early protein deficiency. For eleven years, Stoch and Smythe studied twenty Cape colored infants who were grossly undernourished during infancy. The undernourished group lived under unhealthful slum conditions in one-room shanties built on
sand without any sanitary facilities. Those who lived in houses were crowded into unventilated dark back rooms. Thirteen were illegitimate and, in another three cases, the fathers had deserted. Mothers appeared too apathetic to care for their children. Social workers and district nurses repeatedly requested the mothers to take their children to municipal soup kitchens or to clinics for meals but there was little cooperation. Compared to matched controls, the head circumference, height, weight, intellectual and psychological assessment and encephalograms of these children show that undernutrition during the period of active brain growth has resulted in a significant reduction in brain size and impairment of intellectual development (Stoch and Smythe, 1963, 1967).

In brief the findings are as follows (1967):

| Table 7-3 |
|---------------------|-----------------|-----------------|
| CONTROL GROUP | GROUP | MAGNITUDE OF DIFFERENCE |
| Average head circumference | 52.04 cm | 49.58 cm | 2.46 cm smaller |
| Average height | 133.68 cm | 125.73 cm | 7.95 cm shorter |
| Average weight | 29.45 kg | 24.38 kg | 5.07 kg lighter |

In addition, electroencephalogram data indicate that twelve of the undernourished children had poorly formed low-voltage alpha waves with poor response to eye opening, whereas seventeen of the controls had well-formed high-voltage alpha waves, especially posteriorly, with an excellent response to eye opening. In terms of the New South African Intelligence Scale (not standardized for Cape colored children), the mean for the control group was 76.70, and that for the undernourished group, 61.15—some 15.5 points lower.

Another study points to the same result—i.e., long-term crippling effects from nutritional deficiencies in early infancy, the period of maximum brain growth. From 1953 to 1964, data were collected on 1094 autopsies performed at the Mulago Hospital at Kampala, Uganda, on children from the age of birth to fifteen years old. The children were divided into two groups: those who had suffered from malnourishment and those who had not. The malnourished children had been afflicted with kwashiorkor or marasmus. In each autopsy the child’s brain was weighed. The findings for the age group 4 to 5 years indicated that the non-malnourished brain weight was 1127 grams, and the malnourished brain weight, 985 grams. The malnourished brain was 13% lighter than the non-malnourished brain (Brown, 1965).

At birth the human brain is approximately 40% of its adult weight; it increases to 70% after one year and to 80% by the age of two years. The brain increases in weight in incremental stages, but because of the metabolic stability of many of its constituents, once these constituents are laid down they may be inaccessible to the general metabolic pools of the body in times of shortage. Therefore, even good nutrition in later life cannot repair the irreversible damage done to the brain in infancy, damage which may well result in poor learning capacity in adult life (Brown, 1965; Scrimshaw, 1968). Such an assertion is supported not only by clinical experience with human beings, but also by experimental data on animals such as the pig and the rat (Barnes, 1967). A recent article covering studies of this kind notes that they

...indicate that poverty and the poor nutrition that almost invariably accompanies it may bring into the world children who are less able to learn and to earn than their genetic potential would otherwise allow.

The studies give the expression “food for thought” a new dimension. They have shown that children who receive inadequate nourishment both before and shortly after birth suffer a diminished capacity for intellectual achievement.
In fact, studies of infants and laboratory animals have indicated that children inadequately nourished while still in the womb may start life with a sub-normal number of brain cells—a deficit which can never be made up. (Brody, 1968)

Thus, the concept of a close, reciprocal inter-relation between aspects of organic growth and psychological capacity, influenced by both a social and an ecologic context, is a critical area for further research. Indeed, the spiraling effect, once set in motion, is difficult to break. Cravioto, after commenting on the pervasive and permanent psychobiological effects of protein malnutrition in Central America, another “underdeveloped” area, says of the “PCM spiral”:

A low level of adaptive capacity, ignorance, social custom, infection, or environmental paucity of foodstuffs appears to result in malnutrition, which may produce a large pool of individuals who come to function in sub-optimal ways. Such persons are themselves more ready to be victims of ignorance and less effective than would be the case in their social adaptations. (1966, p. 320)

A concept like the PCM spiral would seem to be particularly useful in understanding many aspects of the social history of West and Equatorial Africa. Vansina (1966) discusses the rise and fall of traditional political-military empires in the Congo in terms of the spread of cassava, a very-low-protein staple. Dependence on cassava is predispositional to the development of kwashiorkor and other deficiency diseases. Such a “cassava belt” stretches along the coast of West Africa where kwashiorkor was first clinically described. And the tragic events of today in eastern Nigeria and Biafra are only too compelling in their underscoring of the close relationship between a protein-adequate diet and effective bodily and psychological functioning. On the latter note—that of the inter-penetration of “social” or “political” events and nutritional health—one may recall the pessimistic appraisal by the African pediatrician Hendrickse, who said:

The high incidence of nutritional disorders in Africa today reflects the seriousness of the social and economic problems of the people of Africa. Current trends in African affairs offer little hope of improvement. On the contrary, the direction of social change and the long-term effects of economic policies currently in operation will inevitably result in deterioration of the present position. Meanwhile, the unstable political situation in the continent threatens constantly to disrupt the existing economic structure and to precipitate disaster. (Hendrickse, 1966, p. 346)

Beyond the obvious clinical cases of protein deficiency in African children in the towns, there are numerous cases of sub-clinical protein-calorie malnutrition, sometimes known as mild-moderate malnutrition. It is difficult to diagnose. Children suffering from mild-moderate malnutrition are not suffering from kwashiorkor nor are they visibly emaciated, yet they are completely outside of the normal range of body weight for age, and they have an increased susceptibility to disease. How common this nutritional growth failure is can be judged by field surveys of preschool children and records of young-child clinics that often show that between 15% and 30% of children weigh below 75% of that which is normal for their age, and therefore fall into this mild-moderate malnutrition group (Cook, 1966).

Thus, it would appear that the major benefit of urbanization for child nutrition is the provision of clinics and other health facilities in which mothers can eventually be educated and severe cases of malnutrition can be treated. But thus far the overwhelming impact of the urban environment on child and adult nutrition has been negative.

In conclusion, we firmly assert that programs of economic or agricultural development, popu-
lation relocation, industrial construction, or any program which either deliberately or inadvertently changes pre-existing relationships between man and any aspect of his environment must be viewed from the outset in an ecologic framework. We must realize the serious hidden costs of a new “ecologic contract” between man and his surroundings. Perhaps it would be useful for public health specialists to start talking about a new category of diseases analogous to the “iatrogenic” diseases known in medicine. Such diseases could be called the “diseases of development” and would consist of those pathological conditions which are based on the usually unanticipated consequences of the implementation of developmental schemes.
### Appendix

**SOME PARASITES OF MAN AND DOMESTIC ANIMALS IN AFRICA FOR WHICH AN AQUATIC ENVIRONMENT IS NECESSARY**

*(Thomas, 1965)*

<table>
<thead>
<tr>
<th>PARASITES OF MAN</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PARASITES</strong></td>
</tr>
<tr>
<td>Viruses:</td>
</tr>
<tr>
<td>About 32 mosquito-borne viruses are associated with human infections</td>
</tr>
<tr>
<td>Protozoa:</td>
</tr>
<tr>
<td>Trypanosoma gambiense Dutton</td>
</tr>
<tr>
<td>Plasmodium spp.</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Nematoda:</td>
</tr>
<tr>
<td>Wuchereira bancrofti (Cobbold)</td>
</tr>
<tr>
<td>Setaria equina (Abildgaard)</td>
</tr>
<tr>
<td>Dipetalonema perstans (Manson)</td>
</tr>
<tr>
<td>Dipetalonema streptocerca (Macic and Carson)</td>
</tr>
<tr>
<td>Mansonella ozzarii (Manson)</td>
</tr>
<tr>
<td>Dirifilaria repicii (Railliet and Henry)</td>
</tr>
<tr>
<td>Loa loa (Guyot)</td>
</tr>
<tr>
<td>Onchocerca volvulus (Leuckart)</td>
</tr>
<tr>
<td>Dracunculus medinensis (L.)</td>
</tr>
<tr>
<td>Trematoda:</td>
</tr>
<tr>
<td>Fasciola gigantica Cobbold</td>
</tr>
</tbody>
</table>
### Appendix (continued)

#### PARASITES OF DOMESTIC ANIMALS

<table>
<thead>
<tr>
<th>PARASITES</th>
<th>INTERMEDIATE HOST</th>
<th>METHOD OF INFECTION</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Trematoda:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gastrodiscus aegyptiacus</td>
<td>Snail</td>
<td>Eating infested vegetation</td>
</tr>
<tr>
<td>(Cobbold)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Schistosoma bovis</td>
<td>Snail</td>
<td>Infective stages penetrate skin</td>
</tr>
<tr>
<td>(Sonsino)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Schistosoma mattheel</td>
<td>Snail</td>
<td>Infective stages penetrate skin</td>
</tr>
<tr>
<td>Veglia and la Roux)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Schistosoma leiperi</td>
<td>Snail</td>
<td>Infective stages penetrate skin</td>
</tr>
<tr>
<td>(la Roux)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Schistosoma margrebowiel</td>
<td>Snail</td>
<td>Infective stages penetrate skin</td>
</tr>
<tr>
<td>(la Roux)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Schistosoma curassoni</td>
<td>Snail</td>
<td>Infective stages penetrate skin</td>
</tr>
<tr>
<td>Brumpt</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Nematoda</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Solaria equiina</td>
<td>Simulium, mosquitoes</td>
<td>Fly biting horse</td>
</tr>
<tr>
<td>(Abildgaard)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Setaria digitata</td>
<td>Mosquitoes</td>
<td>Fly biting sheep, horse</td>
</tr>
<tr>
<td>(Lin.stow)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Setaria lahiato</td>
<td>Mosquito</td>
<td>Fly biting cattle, sheep</td>
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<tr>
<td>papiHosa (Alesc)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dipetaloncina ruandae</td>
<td>Mosquitoes, ticks</td>
<td>Fly biting dog, cat, etc.</td>
</tr>
<tr>
<td>VM\ anil Hercin</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Brugia patei</td>
<td>Mosquito</td>
<td>Fly biting dog</td>
</tr>
<tr>
<td>(Buckley et al.)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dirofilaria immitis</td>
<td>Mosquito</td>
<td>Fly biting dog</td>
</tr>
<tr>
<td>(Leiily)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dirofilaria rcpens</td>
<td>Mosquito</td>
<td>Fly biting dog, cat, etc.</td>
</tr>
<tr>
<td>(Raillet anil Henry)</td>
<td></td>
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<tr>
<td>Onclwcerca armillata</td>
<td>?</td>
<td>Fly biting cattle</td>
</tr>
<tr>
<td>(Raillet and Henry)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Onchocerca fasciata</td>
<td>?</td>
<td>Fly biting camel</td>
</tr>
<tr>
<td>Railliet and Henry</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Onchocerca gibsoni</td>
<td>Culicoides</td>
<td>Fly biting cattle</td>
</tr>
<tr>
<td>(Cleland and Johnston)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Onchocerca gutturosa</td>
<td>Simulium</td>
<td>Fly biting cattle</td>
</tr>
<tr>
<td>(Neumann)</td>
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<td></td>
</tr>
<tr>
<td>Eln’ opiiora poeti</td>
<td>?</td>
<td>Fly biting cattle</td>
</tr>
<tr>
<td>(Vryhurg)</td>
<td></td>
<td></td>
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<tr>
<td>Dracunculins incJtiensis</td>
<td>Copepods</td>
<td>Fly biting cattle, ox</td>
</tr>
<tr>
<td>(L.)</td>
<td></td>
<td>Cattle, dog, etc., drinking water containing infested copepods</td>
</tr>
</tbody>
</table>
### PARASITES OF MAN

<table>
<thead>
<tr>
<th>PARASITES</th>
<th>INTERMEDIATE HOST</th>
<th>METHOD OF INFECTION</th>
<th>MOST COMMON DISEASES TRANSMITTED</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Trematoda</strong> (continued)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fasciolopsis fuelleborni Rodenwaldt</td>
<td>Aquatic snail</td>
<td>Infective stage on water plants</td>
<td>Intestinal fluke</td>
</tr>
<tr>
<td>Heterophyes heterophyes (Siebold)</td>
<td>Aquatic snail, fish</td>
<td>Infective stage in fish</td>
<td>Intestinal fluke</td>
</tr>
<tr>
<td>Stictidora tridactyla Martin and Kintz</td>
<td>Aquatic snail, fish</td>
<td>Infective stage in fish</td>
<td>Intestinal fluke</td>
</tr>
<tr>
<td><em>Echinostoma revolutum</em> (Froelich)</td>
<td>Aquatic snail</td>
<td>Infective stage in snail or second intermediate host</td>
<td>Intestinal fluke</td>
</tr>
<tr>
<td>Echinoparyphium recurvatum (Linstow)</td>
<td>Aquatic snails, frogs</td>
<td>Infective stage in snails or frogs</td>
<td></td>
</tr>
<tr>
<td>Paragonimus westermani (Kerbert)</td>
<td>Aquatic snail, crustacean</td>
<td>Infective larvae enter through Paragonimiasis (lung fluke)</td>
<td></td>
</tr>
<tr>
<td>Schistosoma mansoni Sambon</td>
<td>Aquatic snail</td>
<td>Infective larvae enter through skin of man</td>
<td>Intestinal schistosomiasis</td>
</tr>
<tr>
<td>Schistosoma haematobium (Billharz)</td>
<td>Aquatic snail</td>
<td>Infective larvae enter through skin of man</td>
<td>Genitourinary schistosomiasis</td>
</tr>
<tr>
<td>Watsonius watsonsi (Coryngam)</td>
<td>Aquatic snail</td>
<td>Infective stage on water plants</td>
<td>Intestinal fluke</td>
</tr>
</tbody>
</table>

**Cestoda:**
- Diphyllobothrium sp. larva: Copepod, man
- *Spirometra pretonensis* (Baer): Copepod, reptiles, amphibia, mammals (including man)
- *Echinostoma revolutum* (Froelich): Aquatic snail
- *Echinoparyphium recurvatum* (Linstow): Aquatic snails, frogs
- *Paragonimus westermani* (Kerbert): Aquatic snail, crustacean
- *Schistosoma mansoni* Sambon: Aquatic snail
- *Schistosoma haematobium* (Billharz): Aquatic snail
- *Watsonius watsonsi* (Coryngam): Aquatic snail

**Protozoa:**
- Trypanosoma brucei: Glossina palpalis and other Glossina sp. Mechanically by tabanids or mosquitoes
- Trypanosoma vivax: Glossina palpalis, etc. Also mechanically by tabanids
- *Trypanosoma cruzi* (Kobold): Snail
- Paramphistomum cervi: Snail
- Paramphistomum microbothrium Frischoeder: Snail
- Cotylophorone cotylophorum (Frischoeder): Snail

*Recorded in Africa but in mammalian hosts other man. However, man is a potential host.
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Turner, P. P. Home Treatment of Tuberculosis in the Nyeri District of Kenya.” Tubercle, 43 (1962), 76-82.
(1958).
Technological advancement always upsets the man/environment relationship, but, as Rene Dubos points out, “the real problem is not how to maintain the balance of nature, but rather how to change it in such a manner that the overall result is favorable for the human species.” Developing a country may mean building new roads, providing more water, increasing urbanization, and constructing factories. Consideration, however, should also be given to what increased migration, lack of water purification facilities, overcrowded towns, and the breakdown of a homogeneous community may mean to the people in the country.

Hunter and Hughes in their paper THE ROLE OF TECHNOLOGICAL DEVELOPMENT IN PROMOTING DISEASE IN AFRICA (Page 69), discuss these problems in terms of malnutrition and disease.

Photo 7-1 shows an urban slum in Freetown, Sierra Leone. The African shantytown shown in Photo 7-2 is the result of an influx of migrants from the rural areas.

Clean water pipes can significantly lower morbidity. Photo 7-3 shows villagers in Suhum, African Ghana. In contrast, many streams are still used for drinking water, washing, personal hygiene, laundry, and for playing by children, thereby becoming potential sites for bilharzia.

In Suhum (Photo 7-4), outside food preparation and vending risks gastroenteritis infection carried by flies. Unhygienic market conditions are also seen in Photo 7-5. The dry fish being sold here is a valuable source of animal protein in West Africa’s forest zone. It is imported from the coast and from the Niger River Delta in Mali.