THE PARASITIC DISEASES OF MAN IN AFRICA *

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In opening the discussion on parasitic diseases of man in Africa I intend to adopt a broad definition of what are parasitic diseases by including not only those caused by protozoan and helminthic parasites but also other diseases in which the mechanism of transmission to man involves an intermediate host.

Broadly speaking the clinician practising in tropical Africa may divide parasitic diseases into 2 groups, the first composed of those which are more or less widely distributed and the second those with a focal distribution.

Diseases with a Widespread Distribution

Malaria, bilharziasis and hookworm fall into the first category for, although there may be regions in which they are absent, they must for practical purposes, be considered by every doctor engaged in clinical work in any part of tropical Africa. The frequency and distribution of malaria depends largely on the climate, acting through the mosquito vector; directly and particularly on rainfall and temperature and indirectly on altitude, since increase in altitude brings about a decrease in temperature and a more efficient natural drainage through swiftly flowing rivers. Other subsidiary influences also to be taken into consideration in certain areas are: (*a*) the distance from the sea or other large expanses of water, (*b*) ocean currents, (*c*) the direction of prevailing wind, and (*d*) the existence of forests.

In an equatorial climate the transmission period for malaria can be expected to last throughout the year, but this again will vary with the rainfall and altitude. As the altitude increases the transmission period becomes shorter. At 4,500 feet the incidence of endemic malaria is still heavy, but around 5,600 feet the epidemic type becomes the rule. It is even described up to 8,600 feet. Although malaria may be more frequent in low-lying areas it does not follow that it is more severe than the type encountered on the plateaux from 2,000 to 5,000 feet above sea level, where severe sporadic cases of virulent outbreaks are known to occur. The reason for this is that at the coast the population is exposed to the infection more or less throughout the year. This enables the individual to maintain a tolerance to a greater or lesser

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degree. But on the plateaux the transmission period is much shorter and after a long dry season much of the immunity built up in the previous rainy season has been lost by the time the following rains arrive.

There are few rivers in the centre of Africa free from bilharzial infection and the greater the rainfall the more the disease is encountered. In Mashonaland for example there is far more bilharziasis than in the drier and more arid parts of Matabeleland. Schwetz (1951) found *S. mansoni* at an altitude of 6,300 feet, but in Southern Rhodesia it is said not to occur above 6,000 feet. Inyanga, for instance, is believed to be free of the disease. Hookworm disease, too, is more frequent in lower areas than in higher regions where the water drains off more rapidly. But other factors come into play at the same time. A high temperature is more favourable to the parasite, which is also found more often in a clay soil than in the sand veld.

Parasites with a Focal Distribution

In contrast to those with a wide distribution, we meet parasites that are confined to focal areas, and the factors which determine this are largely unknown. Sleeping sickness, plague, kala-azar, yellow fever, amoebic disease and filariasis are examples of this. In some places a particular disease is common, yet in others it is rare. For instance the pathogenic amoeba is very common in and around Durban, where the peri-urban Africans seem to suffer more than the Europeans or Indians. It is possible that malnutrition may be a factor, or perhaps an increased virulence of the parasite, because not far away along the coast lies Lourenco Marques, where the disease is not encountered with the same frequency or intensity. Further, even in Southern Rhodesia I have noticed its incidence seems to vary almost from town to town. It is possible, of course, that the apparent difference may result from the varying skill of the technician. Judging by the frequency of liver abscess in my African patients I am inclined to believe that amoebic disease is commoner than many people believe.

Plague too is puzzling. It is seen fairly regularly in Barotseland and in some parts of Bechuanaland, but in Southern Rhodesia, which is adjacent to both these territories, no case has been notified since European settlement of the country, although the appropriate rodent reservoir exists there.

Although ascariasis, the common parasite of the human intestine, is encountered in most parts of Africa, it is particularly prevalent in some regions. In a small survey in Ibadan Jelliffe (1951) found that over 80% of house servants harboured this parasite. He recorded cases of pulmonary eosinophilia, intestinal obstruction, perforation of the bowel, and obstruction of the common bile duct and trachea, due to this worm. In Mashonaland cases are not nearly so common and the complications are only rarely encountered, but in Manicaland the frequency is very much greater.

We know a little about the effects of an alteration in the environment of a parasite. Not all parasites are affected in the same way, but interference in the natural surroundings of some definitely influences their life cycle and thus their frequency in man. The organization of a civilized community in a malarial area tends to drive out the anopheline mosquito. Similarly, the clearance of vegetation in a trypanosomiasis area often disturbs the tsetse fly. This was evident in the Hartley district of Southern Rhodesia in the first decade of this century, when the cutting down of trees for the Cam and Motor Mine near Gatooma drove back the fly. The clearing of banks of rivers in Gambian sleeping-sickness regions removes the habitat of the tsetse fly. It is believed by some that game elimination leads also to the eradication of the fly.

But with bilharziasis interference with nature produces the opposite effect. The snails multiply with the digging of new dams, and the establishment of irrigation projects spreads the infection yet further afield.

Over the years Bancroftian filariasis seems to have existed continuously in certain defined pockets. When David Livingstone sailed down the Zambesi in 1856, he recorded the frequency with which the Natives suffered from elephantiasis of the limbs and scrotum. Today we see many cases from the same region complaining of the same complications, including orchitis, epididimo-orchitis, hydrocele, funiculitis, lymph scrotum, and even chyluria. The disease does not seem to have spread beyond these confines to Mashonaland where the same mosquito vectors are known to exist.

Sites of Election

Besides their focal distribution, many parasites, as Heisch of Kenya (1956) has stressed, select special local sites in which the disease may be contracted. For instance, relapsing fever results from the ticks that inhabit Native huts and plague is spread by the domestic rats which pillage the grain bins in the kraal after they themselves have been infected by the wild rodents which live in burrows in the fields. It is only when the wild rats are numerous that *P. pestis* reaches *Rattus rattus* and so man. In Kenya the sites infected with kala-azar are those round termite hills from which emerge the sand-flies responsible for this disease.

Heisch points out that the sites of infection may be relatively stable or unstable. Unstable areas he calls ecotones. Transition zones like forest edges and clearings and cultivated plains, are excellent examples of ecotones, which imply states of tension in nature, in which the ecological balance is liable to be upset. The balance is disturbed in a plague-infested area when the fields are ploughed, and the disease tends to disappear. Transition zones are important meeting-places for man, animals and insects, and result in an interchange of parasites. An excellent example of this is shown by workers in Uganda, where the mosquito *Aedes simpsoni*, the vector of yellow fever, lives in the forest edges. In the forest dwell the tree-top mosquitoes bearing the virus with which they infect the monkeys. The monkeys appear at the edges of the forest to raid the banana plantations and infect the *Aedes simpsoni*, which in their turn affect man, who comes into contact with them.

On the other hand some sites of infection known as 'habitat niches' are stable. Instances of these are Native huts, termite hills and even animal burrows that are away from interference by man. Even forest edges and other transition zones may be relatively stable.

Disappearance and Reappearance of Parasitic Diseases

Again, for reasons not yet clear, a parasitic disease may disappear from a region or become very rare, or it may appear in increased intensity in some other area far removed from its customary focus of infection. For instance, a very severe outbreak of yellow fever occurred in the Nuba mountains

in the Sudan, in which hundreds of Africans lost their lives. After the last war, too, a significant outbreak of kala-azar was reported in the Kitui district and another at Marigat, in the Rift Valley in Kenya, where hitherto this disease was rarely seen, in contrast to the Sudan, which is the accepted focus for kala-azar. After some investigation it was suggested by Heisch that sandflies emerging from termite hills were responsible.

Equally interesting is the tremendous diminution in the incidence of tropical ulcer and tropical myositis, which have largely disappeared excepting in small isolated foci. In the days of Livingstone and for some years later these two diseases were amongst the most serious tropical conditions with which one was called upon to deal. Although they have been linked with a poor diet, the possibility of a parasitic origin has not been disproved.

CLINICAL ASPECT

Lack of Specific Pathological Changes

In countries where parasitic disease is not of major importance the clinician is aided in his diagnosis in a large number of day-to-day diseases by a confident pathological report. Many of the non-parasitic diseases produce specific histological changes. With the majority of the parasites, however, confirmation of the diagnosis of the cause of an illness and often even of a death may be impossible. It is difficult at autopsy to attribute the cause of death to relapsing fever or hookworm disease.

Most authorities do not agree with the high number of deaths attributed at autopsy to malaria in infants and young children in hyperendemic regions, since most infants at this time of life harbour the parasite and it may be coincidental with the presence of broncho-pneumonia, anaemia or other disease. Unless plugging of the capillaries of the brain with malarial parasites can be demonstrated, there is no definite proof that malaria is the cause of death in an indigenous population in many of whom the parasite is habitually harboured and in whose tissues the pigment is present. The same may be said of sleeping sickness. Filariasis, too, produces indefinite changes.

Bilharziasis may be regarded as an exception in this respect, since pathological changes with eventual fibrosis occur at the site of ovideposition. But even here the problem is by no means as simple as it might appear, because of the widespread deposition of ova in the different organs of the body. Many diseases occur coincidentally with bilharziasis in an organ, and as a result it is difficult to be certain whether or not the bilharziasis produced the lesion. Fibrosis in the ureter can be accepted as due to the ova because few other diseases are likely to affect this part of the body. But the finding of ova in a multilobular cirrhotic liver is a different matter, because a large section of the population has ova in the liver without cirrhosis. Many authorities, especially in Egypt and South America, are convinced that cirrhosis of the liver commonly follows bilharzial infiltration. They may be correct, but it is only fair to stress that cirrhosis of the liver occurs in parts of Africa where bilharziasis does not exist, and furthermore appears with the same intensity. The same may perhaps apply to cardio-pulmonary schistosomiasis, although here I am on less certain grounds. It is said that in Egypt some 2% of infected bilharzial subjects die of right heart failure due to arteriolar obstruction by ova deposited in them as emboli. The pressure in the pulmonary circulation is raised and varying degrees of heart strain commonly follow. Just as in the liver, over 50% of bilharzial subjects have ova in their lungs with apparently no ill-effects. We know that right heart failure from pulmonary hypertension of diverse causes occurs in non-bilharzial regions and there is little reason to suppose that these cases are not seen in bilharzial territories. It may be argued, therefore, that the right heart strain is incidental to the ova present in the lungs. A point favouring a bilharzial arteriolitis, however, is the angiomatoid formations which are said to be almost pathognomonic of bilharziasis.

Clinical Picture in the European and Bantu Patient

It is difficult to give an adequate description of a tropical disease which is generally applicable, because the disease usually manifests itself differently in the European and the Bantu. On the whole, the European is virgin soil for infection. When he becomes ill with a parasitic infection this generally occurs in a body which has never had the infection before and is free from other tropical diseases. He is a non-immune subject or one who has not been sensitized by previous infections. A truer picture of the acute uncomplicated effects of the disease can be obtained from him than from the Bantu, who has been exposed to parasitic infection from childhood and has acquired a hereditary racial or personal resistance to the disease.

It is not easy to draw a satisfying clinical picture of malaria, sleeping sickness, yellow fever or bilharziasis in the Bantu. The body responds differently according to its state of immunity to the particular parasite. No two individuals are alike. When an African (and perhaps his parents too) has been brought up in an area in which he has not been exposed to a particular infection, he is liable on contracting that infection to suffer from it in the same way as a European and thus for instance may as easily die of cerebral malaria and blackwater fever.

The clinical picture varies from year to year, season to season and district to district, and what makes it still more difficult to assess, in the African, is that he harbours more than one parasite at a time, sometimes even 3 or 4. Thus in endemic regions with a multiplicity of infections how can one adequately unravel the symptoms resulting from each? This is probably the reason why no clear cause has yet been established for the common tropical anaemia of Africa, despite the many interesting publications recently issued. Malaria, bilharziasis and hookworm disease all affect the haemopoietic system and, to complicate matters still further, the indigenous population exists on a diet which may be lacking in one or more factors essential in the manufacture of the red cell.

The Diagnosis

In England one might say that a clinician can practice satisfactorily with his stethoscope. In Africa he needs the microscope as well. He cannot move without it, else he misses the many parasites. Fortunately with the use of a microscope we can recognize the great majority of infections, such as malaria, bilharziasis, relapsing fever, sleeping sickness and filariasis, especially in the more acute stages of the illness. But the difficulty in the African is to know whether the presence of the parasite accounts for the symptoms. We know that malarial parasites are present in both febrile

and afebrile patients. The parasites may be extremely numerous in a child and yet he may be running round quite unconcerned. It is difficult to lay down a rule in the diagnosis of parasitic disease but, on the whole, it may be said with reasonable safety that, in an African, if a parasite is demonstrated and no other cause can be found for the symptoms the diagnosis of that parasitic disease may be made.

Bilharziasis is interesting in that it is one of the very few parasitic diseases in which stress is laid on the finding of viable eggs in order to establish an active infestation. When the ova are dead it is assumed that they are discharged in the same way as a foreign body and the individual is cured. Yet in other diseases no one insists on proving whether the organisms are alive or dead when found in the discharges. I personally can see very little reason for supporting such a point of view and maintaining that the bilharzia ova should be alive. There are two good reasons against this. Firstly, in urinary bilharziasis large numbers of terminal-spined ova are deposited in the rectal mucosa and yet it is exceptional for them to be discharged in the stools. Secondly, in many cases ova are deposited in the lungs in close vicinity to the alveoli, but are rarely found in the sputum. If these were foreign bodies, why are the terminal-spined ova not more commonly found in the stool or the ova of either variety in the sputum? It would seem to me that the ova of *S. haematobium* appear in the urine because it is part of the life cycle. The finding of dead ova does not imply in my opinion, that the worms are dead, but that the ova died soon after discharge.

TOLERANCE IN THE AFRICAN

Bilharziasis

It is convenient at this stage to discuss briefly the tolerance of the indigenous population to some of the main parasitic diseases. Our knowledge of this subject is by no means complete, but of special interest is the extraordinary battle put up by the host against most of the tropical parasites. The host does not appear ever quite to overcome the infection, except at certain periods of his life. The immunity is not as clear cut or as firm as in many non-tropical diseases, such as measles, poliomyelitis or typhoid fever. Many believe that the immunity, at least in malaria, is related to the nutritional status of the individual. At times, particularly with lack of protein, the amount of available globulin may be reduced and the infection may reappear in a severer form. Thus in hyper-endemic regions the lower protein intake is given as a reason for the increased frequency of malaria in the later months of pregnancy. With most of these tropical parasites we are not sure what happens with re-infection. Does a person with active bilharziasis become re-infected with more worms every time he exposes himself to infected waters or is there a limit to the amount of worms the body will accept? Does a state of immunity develop later and does the infant acquire some immunity from its mother? In my experience, it appears that in bilharziasis a person acquires a heavier infection with repeated exposures. How else can we explain the grosser lesions found in the Bantu as compared with the European, in whom the infection is usually light (Honey and Gelfand, 1957)? For example marked fibrotic thickening of the bladder and ureter, leading to hydronephrosis, is a very frequent occurrence in the Bantu. Compared with the African, the European exposes himself only occasionally. Experimentally, Vogel (1945) demonstrated that in monkeys (*Macacus rhesus*), after repeated exposure to the cercariae of *S. japonicum* over many months, there was a progressive decrease in ova. An immunity to superinfection was finally established so that a dose of cercariae which previously would have been fatal proved quite innocuous. Standen (1949) also observed a similar immunity in the *Macacus rhesus* monkey exposed to the cercariae of *S. mansoni*. Recent experiments by Lurie and De Meillon (1957) with mice infected with repeated small infections show that, once the mouse is infected, subsequent infestations do not aggravate the existing state. The initial dose of cercariae has an immunizing effect against further doses. Fairley (1951) states that there is no evidence that immunity of this type results in man from repeated exposure to schistosome cercariae over a number of years. He rightly draws attention to the importance of this subject, for if an immunity to super-infection exists in man, it might be preferable to employ a drug producing clinical rather than radical cure in heavily infected areas, particularly if it could be taken orally. In this way, I think, Miracil has a distinct future in *S. haematobium* infections. With this drug the load of worms would be reduced possibly to a sub-clinical level so that the body with a smaller number would be able to resist further infection.

In endemic areas the highest incidence of bilharziasis is in children from the ages of 4 to 15, the number passing ova becoming less in adulthood. In Southern Rhodesia Dr. Morley Smith and I investigated its incidence in infants. Out of 27 infants tested, we found only 2 showed positive results, whereas in the 63 children we examined between the ages of 4 and 12, 37 passed ova. Workers in other territories have also found a low incidence in infants and a high one in children. At first we wondered if the infant was not exposed to infection in the same way as the child. We reasoned that the mother might be afraid that it might be drowned or injured by crocodiles. But on closer investigation, we found the mother bathed her infant in pools, especially when she was doing her laundry. It may be argued, therefore, that the infant has some immunity or protection against contracting the disease. We carried out a similar investigation at the other extreme of life and found that few elderly adults in the same region passed ova, despite their exposure to infection. It is probable that, by this time of life, they have acquired an immunity by which the body is able to reject invasion of the cercariae. Thus there appears to me to be some evidence that an immunity can be established in the body through infection and possibly a temporary one through the mother to her infant. An argument against this is that in other diseases, such as diphtheria, the immunity of a babe rarely lasts longer than 6 months or a year or two.

Hookworm Disease and Filariasis

I am not sure what the immune processes are in hookworm disease. As in bilharziasis, it is probable that there is a limit to the number of worms that can infest the body. As far as I have seen it at autopsy in Rhodesia, the infestation has been light, unlike Uganda, where hundreds of worms may be found in any one individual. In Gambia, MacGregor and Dean Smith (1952) have shown that the incidence of hookworm disease is very low in children up to the age of 5. They argue that the young child is not permitted to wander far from its hut and is left behind when the mother goes into the bush on her calls to nature. So far my experience con-

firms that of the workers in Gambia, but I would suggest that again the reason may be some protective power against the infection in the infant and young child.

Filariasis, especially that due to *Wuchereria bancrofti* and *Onchocerca volvulus*, is rare before the age of 4 in endemic regions. Dean Smith and MacGregor found that the first manifestations of bancroftian filariasis did not appear before the age of 7. They suggest that one of the reasons for this may be that, as malaria is usually present in the same region, the young child suffers from a continuous fever which protects it from acquiring filariasis. Is it possible to postulate a similar type of immunity for bilharziasis, filariasis and hookworm disease?

Malaria

Malaria is different in that it is the infant who seems to bear the brunt of the infection in a hyper-endemic region. Afterwards the child is able to survive the attacks and by the time he reaches 15 years is probably so resistant that, unless the infestation is extremely heavy or virulent or there is some other good reason, he is not likely to suffer much sickness or risk of death from the disease. The infant up to the age of 3 months rarely shows parasites in the blood, but by the age of 1 or 2 years most infants have had the disease. The immunity conferred on the infant by its mother stands it in good stead, not only for the first 3 months, but even later, for the attacks tend to be milder in all respects than those suffered by European children without that immunity. There is much controversy about the number of infants who die from acute malaria. The work of Garnham in East Africa in recent years shows that, in actual fact, fewer die from the disease than was formerly believed. Yet Bruce-Chwatt considers that 5% of children under 5 die directly from fever each year. Another school of thought believes that, whilst there may not be many direct deaths from malaria, the disease is an aggravating factor in many of the deaths in infants and young children from gastro-enteritis, broncho-pneumonia, tuberculosis, anaemia and even kwashiorkor, by lowering of resistance. In my experience, malaria rarely kills the African infant, and I am doubtful whether it is an important factor in the morbidity rate. The immunity of the baby in its first few months of life must be very high in hyper-endemic regions, since congenital malaria is rare. But other factors may be responsible. There is experimental evidence that breast milk renders an attack of malaria less severe and it is possible that in certain regions the possession of the sickle-cell trait may protect the infant against the disease.

It has been suggested that a raised body temperature in a hyper-endemic zone is an indication of malarial infection, but on closer investigation, Colbourne (1955) showed that this elevated temperature in children up to the age of 14 years is not due to malaria, but is a normal occurrence. He is careful to point out that we do not know what is the normal temperature in the Bantu. He treated febrile school children of the age of 7 with antimalarial drugs, but they did not appear to gain any tangible benefit from the treatment except perhaps a slight increase in weight and less absenteeism.

The effect of malaria on the foetus has also been investigated by workers in Africa. At one time it was believed that abortion was likely to result when a pregnant woman contracted malaria. I think this is the case in a non-immune mother and possibly in an immune one when the infestation is heavy. But workers have found little or no increase in the abortion rate in hyper-endemic areas during the malaria season. The same findings apply to the stillbirth rate. But in a hyper-endemic zone the incidence of malaria in pregnant women towards the end of parturition is approximately double that of the general population. It may be that her resistance has been reduced by the strain of the pregnancy and she is then more liable to the infection than the ordinary adult.

An excellent example of acquired immunity to malaria by the African is shown by the frequency of *P. malariae* and *P. vivax* in infancy and early childhood, after which these two strains disappear and are rarely found in the adult (Schwetz, 1949). The African appears therefore to develop an immunity to these two parasites, but is not quite able to protect himself from the malignant tertian form of the disease.

In a hyper-endemic zone children, on the whole, suffer little disability from malaria. It is believed that people moving from an endemic area to a non-endemic one lose their immunity and on their return suffer severely when re-infected. But Colbourne (1955) investigated this subject by studying Gold Coast students who had spent some years in England and found they did not suffer unduly on their return. They contracted no severe attacks and managed to control the fever quite readily. We are also taught that Africans who move from one endemic region to another are liable to suffer acute attacks of malaria owing to their coming into contact with a different strain of parasite (Schwetz, 1949). While this may be so, I have not noticed it in Africans coming to Mashonaland from Northern Rhodesia, Nyasaland and Portuguese East Africa.

Garnham (1949) classifies immunity in malaria into 3 types :

1. *The racial type*, in which the individual has lived in the area for generations and built up an immunity. This is long lasting in contrast to Africans brought up in non-endemic areas, who suffer severely on moving to an endemic one.

2. *The transmitted or passive immunity* which is passed on from the mother to her child and lasts only about 3 months.

3. *An acquired immunity* (*premunition*) for which, for the purposes of this paper, I prefer to use the word tolerance.

Relapsing Fever and Sleeping Sickness

Relapsing fever bears some similarity to malaria in that the infant is frequently infected. Bell (1953) from East Africa reports that the incidence of this disease varies inversely with the age of the patient. In infants and children, too, the prognosis is more serious. Differing in this respect from malaria, congenital relapsing fever has been described a number of times in endemic regions.

Even in both the Rhodesian and Gambian forms of sleeping sickness, there seems to be an immunity to this disease, which is normally regarded by many as fatal. Mild cases are frequent in sleeping-sickness belts and carriers of the rhodesiense type of the disease have also been recognized. It is known that a mild case may sooner or later be complicated by involvement of the nervous system, a development which may occur at any time—even months or years after infection.

Summary

Thus to summarize, the clinical picture of the main parasitic disease of Africa depend on complicated immune

processes. The Bantu has acquired a racial form of tolerance to these diseases, producing a clinical picture quite different from that of the European living in these parts in whom the clinical features are more distinct and more uniform.

MALNUTRITION, ANAEMIA AND PARASITIC DISEASES

An important subject which should be discussed is the effect of these parasitic diseases on the nutritional state of the body. Many workers, including myself, have said that bilharziasis, malaria and hookworm disease reduce the appetite and induce debility and, in this way, tend to hasten the onset of nutritional disease in a population whose diet is already on the borderline or distinctly lacking in proteins and vitamins. Furthermore, these diseases often cause either a loss of blood or blood destruction and so are considered responsible for the anaemia so common in these people. Recent evidence, based on some interesting surveys, has given rise to doubt whether nutritional disease or anaemia is in fact hastened by these diseases. A useful research was that by MacNamara (1948), who carefully investigated a number of school-children of the same social grade in Central Nigeria, where bilharziasis, hookworm and malnutrition are frequent. One group of children was given a good diet, another was treated for parasitic diseases, and the third, the control group, was left to carry on in the normal way. After an interval of 100 days MacNamara found that the children on the good expensive diet had gained the most weight—an average of 1 lb. 10½ oz. Those treated for helminths had gained an average of 9½ oz. and the control group also very little. All signs of malnutrition had disappeared from the well-fed group, but there was no change in the nutritional state of those who were deparasitized. He found that the children on the good diet showed the best results in endurance tests, but their teachers noticed no difference in their mental ability. In this connection, in South Africa, Kieser (1934) maintained that the child with bilharziasis was lethargic and had poor powers of concentration, so that his school work suffered. In Southern Rhodesia work on similar lines was recently carried out by Blair *et al.* (1949), who found that the children with bilharziasis showed better school results than those free of the disease.

Beet (1949) investigated the haemoglobin levels of children in the Lala district near Broken Hill in Northern Rhodesia, where bilharzia, hookworm and malaria are endemic. He first compared the mean haemoglobin levels in children with hookworm and those without and found no difference (12·3 g.% in both groups). He therefore concluded that hookworm disease was not an important cause of anaemia in these people. He then compared a large group of children with *S. haematobium* with a similar group without the disease and found the average haemoglobin (11·6) of the children with bilharziasis was lower than that of those without (12·5). He considered, therefore, that bilharziasis tended to lead to slight anaemia. But Gerritsen *et al.* (1953) in Johannesburg did not find much anaemia in the 8 Africans with obvious haematuria in whom they studied the degree of blood loss and reached the conclusion that bilharziasis was not an important factor in the production of anaemia.

Colbourne, Edington and Hughes (1950) in West Africa approached the problem from a different angle. They compared the incidence of hookworm disease, bilharziasis, malaria and ascariasis in children with deficiency disorders and those without signs of malnutrition and found no difference. Thus from these surveys, it would appear that the importance of parasitic diseases in the causation of malnutrition has been over-stressed in the past.

So far we have tried to assess the harm that the parasite can cause man. We are agreed that each one can lead to disease, but we are not sure whether it influences the nutritional state of the body. Do the parasites in any way protect the body or confer any benefit to the individual? It could be argued that malaria, bilharziasis, sleeping sickness and kala-azar produce an excess of globulins in the blood, which may confer an immunity to bacterial infection. But this does not seem to be the case. Does the malarial fever so frequent in children in an endemic area protect them against nephritis or does a malarial infection in an adult African render him less liable to general paralysis of the insane? In my experience both these diseases are sufficiently common to lead me to believe that the fever does not confer this protection. Nor am I convinced that parasitic diseases protect the body against other infections such as pneumonia and tuberculosis.

A BALANCED APPROACH

There are a number of rival schools of thought, each stressing the responsibility of a particular parasite or other disease for much of the ill-health or morbidity in the people. One group claims that if malaria is eradicated, the capacity of the African will be vastly increased. Another group, especially the Egyptians, maintains that if only bilharziasis can be eliminated a healthy nation will be produced. Yet another considers that if the diet is balanced and adequate in quantity and quality the parasites can be disregarded and the problem of ill-health will be solved. Not one alone is correct. Although, in my opinion, the improvement of the nutrition is perhaps the most important single factor that helps to preserve a healthy population, one should not forget that the diet of the African has some advantage. Its low fat-content tends possibly to militate against degenerative heart disease. To obtain the best results, however, I believe we should approach the problem of disease from a wider aspect so that it is attacked from all angles at once.

THE CYCLE

The outlook for mankind exposed to these parasites is ultimately good. We can be hopeful because the parasites infect man only through his close contact with nature. The closer this association, the more likely he is to be infected with one or more of them. The parasitic diseases may thus be regarded as diseases of nature or environment, including the soil, the water, the vegetation, animals and insects. If in any of the diseases one can succeed in breaking a link in the chain of events, there is a good chance of avoiding the infection. Thus even if he lives in an endemic region, man can avert bilharziasis by avoiding contact with the waters. He need not contract hookworm as long as he wears shoes. If he lives in a mosquito-gauzed house, the cycle of the malaria parasite is affected and he stands a good chance of surviving the malarial season. Good drainage and spraying are also effective measures of breaking the cycle.

While there are many ways of preventing the disease I

think that perhaps the factor which most helped the White man in early settlements in Africa was his continuing to follow a European way of life and particularly his living in a European type of house. As long as his nights were spent in that house his chances of escaping the fever were good. In the small townships of Rhodesia and Nyasaland, at the turn of the century, few people died, but when they moved out to the country in the rainy season, even for a few days, they ran a grave risk of being cut off. The European settler who died of fever was not the townsman but the traveller, prospector, tramp, and transport rider. A very good example of the importance of this factor in the prevention of malaria is shown by the experience of the U.M.C.A. mission on Likoma island of Lake Nyasa in its early days. These brave missionaries believed that in order to convert the Natives they should live like them in the same type of huts and eating the same food. This experiment was a catastrophe. Their mortality was so terrifying that the London board had to intervene. This was largely through the efforts of Dr. Robert Laws, a medical man in charge of the Scottish Mission on the Lake shore nearby, whose men suffered far more lightly from malaria because they lived in brick houses. On the Scottish mission the mortality from the fever was negligible. When the U.M.C.A. missionaries changed their housing they too withstood the malaria.

Before 1898, when Ronald Ross proved the part played by the anophelene mosquito in the transmission of malaria, the outlook for European settlement in Africa seemed hopeless. The various governments, backed by the opinion of the medical profession, were loath to permit large-scale settlement on account of the fever. In fact tropical Africa was known as 'the White man's grave'. Except for the missionaries, even the few people who came out did not intend to settle and it was believed that the future of the White man in Africa could be 'counted in years rather than generations' (Felkin, 1895). Today the White man can live in any part of tropical Africa with little risk to himself or his family; provided he avoids close contact with the elements of nature the hazards of parasitic diseases can be overcome.

Finally we should remember that the tropical diseases of Africa are not static. Each forms a complex unit in which the parasite, the animal, man and his environment build up a chain or a whole. It is dynamic, evolutionary and ever changing and, as pointed out by Heisch in Kenya (1956) reacts with other wholes. At any time a disease may disappear or diminish, with man receding into the background and the disease confining itself to animals. Heisch has in fact evoked the theory of holism as propounded by that great South African statesman and philosopher, Jan Smuts, in order to explain the behaviour of some of these parasites.

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