Fundamental Facts Concerning Malaria in the North-Eastern Transvaal

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SUMMARY

Facts are presented concerning the parasite, the invertebrate host, the vertebrate host, the distribution of the disease, control measures and recent epidemics in the north-eastern Transvaal. Comparisons are drawn between the position as it was in the past and as it is now. An attempt is made to read the message which the facts hold.

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Attention is focused upon some fundamental facts appertaining to malaria in the north-eastern Transvaal — facts upon which we, the medical practitioners in this area, must base our approach, not only for the prevention, diagnosis and treatment of malaria in this particular part of the globe, but also to the no less important duty of guiding public opinion on the subject.

THE PARASITE

The parasite in over 98% of malaria cases in the northern Transvaal is *P. falciparum*. *P. malariae, P. vivax* and *P. ovale* are found only very occasionally. In other words, the malaria with which we are dealing is, for all practical purposes, malignant tertian malaria. Seen from the patient's and from the clinician's points of view, this is the worst of the malarias, not only because it presents with such varied clinical pictures but also because, unrecognised and untreated, it is most liable to end fatally. From the public health official's point of view, on the other hand, it lends itself more easily to control measures than the other forms of malaria, because even untreated or insufficiently treated cases probably do not remain infective to the mosquito for more than a year, whereas in *P. vivax* infections (i.e. benign tertian malaria) gametocytes may persist for up to 3 years and in *P. malariae* infections (i.e. quartan malaria) gametocytes may persist indefinitely.

It is interesting to note that in 1953 Gelfand and Lane-Mitchell 'studied 100 consecutive cases of acute malaria admitted to the Salisbury African Hospital, from whom smears were taken every 24 hours while treatment continued unabated for 2 - 3 days until the fever subsided. After 24 hours' treatment 73% still had positive blood smears, after 48 hours 42% and after 72 hours 16%.' Although the antimalarial drugs used today may possibly clear the parasitaemia more rapidly, I think the importance of Gelfand and Lane-Mitchell's findings is that they stress that the vast majority of cases of malaria which occur, particularly in a known malarious area can, and should, be diagnosed and reported accurately and speedily. The public health significance of this lies in the fact that our epidemics in the north-eastern Transvaal spread from a few initial foci. Therefore, if these early cases are diagnosed and investigated, one has every hope of preventing an epidemic.

Modern control methods were introduced after World War II and the effect which they have had upon the parasite rate can be gauged from the records of the Annecke Institute. These statistics are, in my opinion, not really comparable; nevertheless, they are the only figures available.

Period	*Average No. of slides examined per year*	*% positive*
1939/46	6 746	48
1947/55	849	27
1956/58	9 153	3
1959/71	78 455	0,6
1971/72	78 940	2,3

THE INVERTEBRATE HOST OR VECTOR

In the north-eastern Transvaal (as indeed in the rest of South Africa) the only mosquitoes which have been proved to transmit malaria are 2 species of anophelines, viz. *A. funestus* and *A. gambiae*. Swellengrebel, in 1930, in his lucid report, tells us that both transmit malaria equally well, but their life-habits are entirely different and this has a marked influence on the epidemiological picture to which they give rise.

A. gambiae breed in small, exposed and often muddy puddles and are almost entirely dependent on local rains for the breeding places of their larvae. In a year when much rain falls or, more correctly, when the rain is evenly spread and when there are not long spells of drought between the rainy spells, the *A. gambiae* population explodes. It is then associated with epidemics.

A. funestus, which is a stream breeder, is for practical purposes really independent of rain. Naturally, a long-continued drought which dries up all water will destroy it, but an ordinary dry year when streams do not dry up does no harm to *A. funestus* at all. Therefore this mosquito is more likely to be associated with endemic malaria.

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In their reports Swellengrebel in 1930 and Annecke in 1950 make mention of both *A. gambiae* and *A. funestus* and leave no doubt that in the malarious areas, particularly during seasons favourable to mosquitoes, these insects were easily found in large numbers in all man-made shelters and habitations. However, the term *A. funestus* does not appear even once in the WHO report of 1959. One must assume that this species was then no longer found. Yet Hansford (1969 onwards), in his annual reports, states that both species still appear, although rarely, in human habitations; but, searched for correctly, they are found fairly easily out-of-doors. Over 90% of these 2 species collected, both indoors and out-of-doors, have fed on animal blood. How does one interpret these findings? It is clear that the use of residual insecticides (first BHC and more recently DDT) in the north-eastern Transvaal has decimated but not eradicated the 2 vector species. However, we are not certain whether the remaining population, of *A. gambiae* in particular, has changed its habits (from endophilic/anthropophilic to exophilic/zoophilic) or represents species which have always been exophilic/zoophilic in habit but which were masked previously by predominantly endophilic/anthropophilic strains which have now disappeared. We have a strong suspicion that transmission of the disease in the north-eastern Transvaal now occurs out-of-doors as well as indoors. Everything possible is being done to sort out this problem because the spraying of human habitations with residual insecticides, which forms the basis of our control measures, is directed at indoor transmission, and is not effective against outdoor transmission. However, I must stress that the solving of this entomological problem is an extremely difficult task because the vector mosquitoes now occur in such small numbers.

THE VERTEBRATE HOST

The vertebrate host is man. The effect which a malaria-infected mosquito-bite will have upon man depends on whether he has a degree of immunity, or is non-immune to the disease; in other words, whether he resides or has recently resided in an endemic area where he is or was regularly exposed to infection. An immune population suffers relatively little adverse effect from it, except in early childhood, while infection in a member of a non-immune population is almost certain to result in a clinical attack of malaria. In this connexion may I remind you that it is an acute attack which is associated with the highest gametocyte rate. In other words, it is the non-immune patient who infects the mosquito most readily. Our control measures have rendered virtually the entire population of the north-eastern Transvaal non-immune. Consequently, when an epidemic occurs a vicious cycle is started. Virtually every undiagnosed and untreated case of malaria develops a high gametocytaemia and most mosquitoes which feed on such persons become infected themselves. This leads to an explosive transmission rate. In this connexion may I remind you that although the sexual and asexual forms of the parasite appear roughly at the same time in *P. vivax* infections, the same does not hold true for *P. f lciparum* malaria. In *P. falciparum* infections gametocytes do not appear until 10 - 12 days after the first appearance of asexual parasites. This stresses, once again, how very important it is, from a malaria control point of view, that *P. falciparum* infections should be accurately diagnosed and treated in the 10 - 12-day period before they become infective to the mosquito.

DISTRIBUTION OF MALARIA PRIOR TO 1945

Full-scale control was introduced in 1945. Prior to this, as one would expect, the distribution and incidence of the disease reflected the distribution and the density of the 2 vector mosquito species. Moving from east to west through the north-eastern Transvaal, i.e. through the lowveld, up the escarpment of the Drakensberg and on to the middleveld plateau one could distinguish 3 malaria zones.

The first zone extended up to approximately 610 metres above sea level. Here one found perennial streams in which *A. funestus* bred and also a summer rainfall favourable to the breeding of *A. gambiae*. Consequently both species transmitted the disease, and in this area malaria was hyperendemic, i.e. both the spleen rate and the parasite rate in children 2 - 9 years of age were consistently over 50%.

The second zone consisted of the escarpment. Here too, perennial streams suitable for *A. funestus* breeding were to be found, but the efficient slope drainage and the high rainfall were usually inimical to the breeding of *A. gambiae*. Consequently in this belt malaria was transmitted mainly by *A. funestus* and was usually endemic.

The third zone incorporated virtually the entire middleveld plateau. *A. funestus* did not breed here nor did *A. gambiae* normally, although other species of anophelines were to be found. But every few years climatic conditions would favour *A. gambiae*. This mosquito would appear (whether it introduced itself by natural spread or through lines of communication was never established), breed prolifically and, because there was always a reservoir of parasites, malaria epidemics would result, such as occurred in 1939 and 1943.

In the most intensely malarious areas, namely the hyperendemic and endemic zones, it was only non-immune newcomers, that is neonates and visitors from non-malarious areas, who were really inconvenienced by the disease, whereas in the least malarious areas — the middleveld plateau — whenever an epidemic struck the susceptible population it left in its wake death, disaster and communities facing bankruptcy.

DISTRIBUTION OF MALARIA SINCE 1945

The introduction of modern control measures led to a rapid reduction in the incidence of malaria, and in 1959 the visiting WHO Assessment Team reported 'the presence of a number of small foci of endemicity separated by vast tracts of land in which there is no transmission at all'. This statement still holds true except that there have been 3 outbreaks of the disease, in 1967, 1971 and 1972.

In the non-epidemic years since 1965 we have had an annual average of approximately 300 indigenous and 200

imported cases of the disease, whereas in 1967 there were 1 197 local cases and 375 imported cases; in 1971 there were 607 local cases and 148 imported cases; and in the first 6 months of 1972 there were 4 083 local cases and 110 imported cases.

CONTROL MEASURES PRIOR TO 1932

Control measures were aimed at preventing contact between the mosquito and man, i.e. by larvicides, drainage schemes, siting of dwellings away from mosquito breeding sites, mosquito-nets, screening of housing, etc. These measures met with no real success.

In 1932 Park-Ross in Natal reasoned that, since the vector mosquitoes had been shown to spend the greater part of their lives indoors, there would be no need to prevent mosquitoes from breeding and from biting parasite carriers if one could destroy the adult insect within the 10-or-more-days period of the parasite cycle within the mosquito. He endeavoured to do this by spraying all habitations with a knock-down pyrethrum insecticide regularly each week. The immensity of the project foiled him; but not to such a degree that he and other malariologists failed to see a glimmer of hope.

CONTROL MEASURES FROM 1945 ONWARDS

The advent of residual insecticides which are effective for months (instead of a few minutes as is the case with pyrethrum sprays) made Park-Ross's dream come true. In Natal all other measures, including the use of larvicides, were abandoned, but in the north-eastern Transvaal Annecke superimposed the new method of attack upon the existing programme of larviciding in the vicinity of all built-up areas and of all major breeding sites of *A. gambiae*. These measures continue unabated to the present day, but to them has been added a programme of active and passive surveillance aimed at detecting and reducing to a minimum both persons suffering from clinical malaria and also asymptomatic carriers of the disease.

However, two matters cause us concern. Firstly, it is unfortunately our experience, in common with all other countries in which malaria has been brought under control, that we have lost much of the public co-operation which contributed to the initial success. This is perhaps understandable because in the north-eastern Transvaal we have now been in the attack phase for 25 years, compared with the 3 years which the experts envisaged when they still dreamt of a speedy eradication of malaria from the globe. Today, few Whites in these areas will consent to their homes being sprayed with residual insecticide and, in the case of the Blacks, we find many locked huts with the owners absent (or supposedly absent), and after the spray many of their womenfolk immediately set about replastering the interiors of the huts, thereby nullifying our efforts. Secondly, the strong suspicion that there is an increasing amount of outdoor transmission is a worrying one. Outdoor transmission can be interrupted only by eliminating the parasite or by preventing contact between the mosquito and man — that is, the very type of control which previous generations of malariologists failed to achieve.

RECENT EPIDEMICS

How does one account for the epidemics which occurred in 1967, 1971 and 1972?

It must be borne in mind that although malaria control may have decimated the mosquito population and reduced the parasite rate to a very low figure, it has nevertheless not eradicated it entirely. Still less has it altered the climate and environment. Consequently the malarious areas of the north-eastern Transvaal retain (and it is important that we understand this fact) their full potential as far as malaria is concerned.

Briefly, what the malaria control programme has achieved is: the eradication of indigenous malaria from the previous epidemic (middle-veld) zone; and the reduction of malaria in the endemic and hyperendemic zones to a few residual pockets of low endemicity. It is from these pockets and, in our experience, even more from imported cases, that malaria spreads in years when swollen rivers and impassable roads hamper control measures and *A. gambiae* breeds to such a degree that it swamps our defences.

We border on 3 neighbouring territories, only one of which, according to our information, has attained the same degree of control over malaria as we have. If international boundaries presented impassable barriers to human beings I have no doubt that we could soon eliminate the few residual foci of malaria endemicity but, since they do not, the parasite is being imported continuously. Not only do visiting South Africans contract infections outside our borders, but the Black ethnic groups in the north-eastern Transvaal and neighbouring territories are not confined to our man-made boundaries; nor, since they often travel by foot, do they always pass through immigration controls. Consequently, there is a constant flow to and fro of Vendas, Shangaans and Swazis.

The control of malaria has led to economic development, particularly in the previous hyperendemic areas. This in turn has brought an influx of persons who not only are non-immune to the disease but who also, unlike the old-timers in the area, are not malaria-minded. Consequently this large group assists neither the Department of Health nor themselves in the prevention of the disease.

The community, and this applies particularly to the Black community, is not scattered as it was in the 1930s and early 1940s, but is now grouped into communities. This grouping facilitates the transmission of all communicable diseases, including malaria. Transport facilities have increased and have been speeded up considerably. This factor must also play a role in the propagation of malaria epidemics.

In short, the disease presents in epidemic form because we have a reservoir of parasites, a non-immune population and an explosion of *A. gambiae*, plus economic development, concentration of population and improved transport facilities — all factors which favour epidemic spread of the disease.

DISCUSSION

What messages do these facts hold for us? The environment of the north-eastern Transvaal lowveld is such that it was, is, and always will be, a potentially hyperendemic malaria area. While the vector mosquitoes have been decimated, the control measures available at present offer no possibiilty of eradicating them entirely. Similarly, there is no possibility of eradicating the parasite reservoir from the north-eastern Transvaal while it is exposed to continuous reimportation of parasites.

Malaria epidemics in the north-eastern Transvaal are caused by *A. gambiae*. Therefore the risk is greatest when the normal high summer temperatures are interspersed with rainy spells occurring regularly and not more than a week apart. In such years the local population should be on their guard more than ever. In these high-risk years prophylactic drugs should be taken and the populace should be encouraged to remain indoors after dark.

Dry years with relatively little malaria should not lull us into such a false sense of security that we fail to take full advantage of the malaria control services offered by the health authorities. It is at such times that the parasite rate, and consequently the risk of subsequent malaria outbreaks, can be reduced to low levels.

The farming community and the Black community should be encouraged to insist that recently-arrived visitors, relatives and workseekers from across the border should submit themselves to blood examination and presumptive treatment by the Department of Health's malaria surveillance teams which cover the area every 6 weeks. This will eliminate a great deal of the parasite reservoir which exists among asymptomatic carriers of the disease.

Medical practitioners in the north-eastern Transvaal have a particularly important role to play, not only in the cure but in the prevention of malaria.

Malaria epidemics in the north-eastern Transvaal appear to stem from small foci of either local or imported infections. Therefore, it is of vital importance that each suspected pyrexia case should have a blood examination which, if positive, should in turn trigger off immediate focal investigation of contacts, the introduction of chemotherapy and intensive mosquito control measures by the health authorities. They should be distributors of accurate information concerning drug prophylaxis. In their capacity as leaders of community thought they should encourage and explain to the public the need for continuing cooperation with the health authorities.