

Human Trypanosomiasis in Rhodesia

A NEW HYPOTHESIS SUGGESTED FOR THE RARITY OF HUMAN TRYPANOSOMIASIS

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BACKGROUND

Rhodesia is a land-locked country in South Central Africa 150,000 square miles in extent. It lies between the Zambezi and Limpopo rivers and is a broad tableland of 3,000-5,000 feet above sea level, which extends from south-west to east in a broad arc. The eastern border of the country is a mountain range of 5,000 to 8,000 feet above sea level.

From the accounts of the nineteenth century travellers and missionaries, tsetse fly were not to be found on the top of the tableland and game was hunted on horseback. The Matabele, who had taken possession of the south-western part of the area about 1834, maintained vast herds of cattle; and the Shona-speaking people, who had lived in the northern and eastern portions for several centuries, also had herds of cattle. Hunters and missionaries, and no doubt the African inhabitants, recognised areas where it was unsafe to take cattle because of the risk of "ngana" (cattle trypanosomiasis).

In 1889 the British South Africa Company had secured a mineral concession from Lobengula, ruler of the Matabele nation, to enter and exploit the area occupied by the subject Shona-speaking tribes, now known as Mashonaland. To do this a Pioneer Column set out from Botswana and travelled northwards. The route followed had to avoid the Matabele homeland which was skirted to the east. They could not travel too far to the east or they ran the risk of entering country infested with tsetse fly, which would have wreaked havoc with the horses and draught oxen used by the Column for transport.

It is not known whether human trypanosomiasis occurred in Rhodesian tsetse fly belts during the nineteenth century. Any hunter or traveller in tsetse fly country who sickened and died of

an acute febrile illness in those days would have his death attributed to "fever." Whether the death was due to malaria or trypanosomiasis would not be known. This is quite different from the picture seen in West Africa, where the chronic disease "sleeping sickness" (due to *Trypanosoma gambiense*) was a well-recognised clinical entity despite the fact that the cause of the condition was not then known.

Soon after the occupation of Rhodesia the country was seriously affected by the pandemic of rinderpest which had swept down the continent of Africa in 1896. Rinderpest destroyed not only horses and cattle, but enormous herds of antelope, buffalo, etc., which had served as the food supply for female tsetse fly. As the game and cattle were destroyed, tsetse fly also disappeared from large tracts of Rhodesia and the insect was to be found only in a narrow belt in the Zambezi Valley proper and in a few isolated islands of territory where tsetse fly persisted (Jack's residual areas). The rinderpest pandemic had two important effects: the cattle and horses died, seriously impeding transport; and secondly, the destruction of the game population removed the food supply of tsetse fly over a large area. This enabled land settlement to be undertaken in many areas where previously "ngana" precluded stock farming.

THE WEST SEBUNGWE FOCUS, 1911-19

The first autochthonous case of Rhodesian trypanosomiasis to be discovered in a person who had not been north of the Zambezi river was diagnosed in London in August, 1911. Not only had the patient never been north of the Zambezi river, but his only contact with tsetse fly had been in the Sebungwe district bordering on the Zambezi river, downstream from the Victoria Falls. Fleming (1913) discusses the tsetse fly situation and the results of a clinical and blood film survey conducted in 1911 by Dr. F. O. Stohr and himself in the scanty African population in this area. Apart from the European case diagnosed in London, 11 Africans were found with trypanosomes in gland juice or blood. All age groups appear to have been affected, the youngest being 18 months old, the child of one of the other cases. It was surprising to see how many of the cases had been ill for as long as six months. The second case described by Fleming, Meleki, had been a personal servant at Kariangwe for some time; in March, 1911, he visited his father's village not far away and was sick when he returned and laid up for a week. In October, 1911, he was examined and appeared to be in good health. The patient claimed he was quite strong

and nothing the matter with him. Trypanosomes were found in gland juice. Another case, No. 10, an adult male named Sianyna, visited the Zambezi about March, 1911, was ill on his return, but was fit enough to be a carrier to help take the effects of the European official in late April to railhead on his journey on sick leave to London, where he was diagnosed as a case of human trypanosomiasis on 30th August, 1912. It is interesting to speculate that H.R.T., the first European known to have been infected in Rhodesia with trypanosomiasis, may well have been infected by a tsetse fly which had fed on one of his own carriers who may have been a "healthy" carrier case of trypanosomiasis.

While Dr. A. M. Fleming and Dr. Stohr were busy surveying the 600 African resident population in the Sebungwe tsetse fly area, Dr. A. J. Mackenzie was examining the people living in the Umniati tsetse fly area, 50 miles west of Gatooma, where no clinical indications of blood or gland infections were found.

In 1912 Dr. Stohr continued the survey of the population in the tsetse fly belts in the north of the Lomagundi district, but no further cases were discovered. The measures taken to control the disease were to depopulate the tsetse fly areas, resettling the people on fly-free land and throwing the affected areas open to shooting of game in an effort to reduce the tsetse fly population. In 1914 two European hunters who entered the Sebungwe open shooting area contracted the disease, one of whom died in Britain while undergoing treatment. The open shooting policy was abandoned as hunters concentrated their efforts on the more lucrative game animals, elephants, etc., ignoring the antelope class.

To sum up the situation regarding indigenous human trypanosomiasis, there were from 1911 to 1919 four European cases and deaths and 11 African cases resulting in 10 deaths, all attributable to the Sebungwe area. One of the European cases was a woman who accompanied a hunting party moving in the southern part of the Sebungwe area. She had rarely left the base camp and it was hard to understand how she had contracted the infection. None of the other 62 members of the party, European and African, after a most careful examination, were found infected.

THE WEST HARTLEY FOCUS, 1923-34

No further cases were notified in Rhodesia until 1923, when a fatal case was discovered in an African woodcutter who had been working in

the West Hartley area on the Umniati river. Dr. Mackenzie surveyed the area in 1924 with negative results. From the same area two African cases, one fatal, were recorded in 1924; and in 1930 a further two African cases, one of whom died. In 1933 two European cases with one death, and six African cases resulting in two deaths, were reported, all these being associated with the West Hartley tsetse fly area. Early in 1934 a further African case infected from this focus was reported, and it was decided to undertake an extensive survey of the population in all the northern fly belts.

Dr. J. L. M. Jeffares surveyed the northern fly belts in Lomagundi and the people living on both banks of the Sanyati river. Dr. D. M. Blair surveyed the West Hartley focus and nearby parts of the Sebungwe district, covering the same territory as surveyed by Dr. Mackenzie in 1911 and 1924. The population was very sparse and it was possible to visit village by village and examine the whole population. Jeffares found no clinical or parasitological evidence of human trypanosomiasis. Three cases were found by Blair in West Hartley, one an acute case ending fatally, and two "healthy" carrier cases. In addition to these, there were in 1933-34 six other cases directly associated with the West Hartley tsetse fly area, and all nine had visited a small village of 66 inhabitants at Gowe, on the Umniati river. The headman of the village was Kahondera, a "healthy" carrier case.

In 1935 Blair spent five months in Sebungwe, surveying the scanty population who had drifted back into the area surveyed in 1912 by Fleming, and the dense population along the south bank of the Zambezi river between Walker's Drift and Kariba Gorge—an area now inundated by Lake Kariba. Other groups of Africans residing in north and central Sebungwe were also surveyed, but no evidence whatsoever of trypanosomiasis was discovered. Blair (1939) gives an account of this work and discusses the possible role of "healthy" carrier cases of the disease in providing *T. rhodesiense* for passage by tsetse fly to other people living in the area, but more especially to persons visiting the area. It was decided to move the human population from the tsetse fly areas along the Umniati river and to press on with the game shooting programme to eliminate the tsetse fly from the dangerous West Hartley focus.

No indigenous cases of human trypanosomiasis were reported from 1935 to 1944, although seven African cases, infected outside Rhodesia, were reported over this 10-year period. These

cases were found in persons who had come to Rhodesia seeking employment from countries to the north.

THE NORTH LOMAGUNDI FOCUS, 1945-54.

In 1945 cases of the disease were reported from the Rhodesian side of the Zambezi Valley, east from the Chirundu bridge to the border with Portuguese East Africa opposite the confluence of the Luangwa and Zambezi rivers. Further cases from this area were reported in 1946-48; at first all cases came from the eastern end of the area, but in 1949 two cases occurred north of Karoi. During 1950-52 further cases were reported from the area, but because of shortage of staff no survey could be undertaken.

However, in June and July, 1953, Dr. V. N. Barlow examined 1,589 people living in the Zambezi Valley east from the Chirundu road and a further 300 people living in tsetse fly areas on the south escarpment of the Zambezi Valley. An examination of blood films revealed two "healthy" carrier cases, a boy aged 11 years with a heavy blood infection and an adult. The distribution of the cases seen in 1953 were: four cases in villages opposite Feira, three from near Chirundu, one from the Rekometji river, 15 miles from the Zambezi, and one case from where the Sapi river enters the Zambezi. There was considerable domestic traffic across the Zambezi river in this locality and it would be difficult to say whether Rhodesian cases were infected south or north of the river and it is known that at this time there was some incidence of the disease on the north bank. No further cases were reported east of the Chirundu road until 1958, when three more cases were found in villages opposite Feira.

THE KARIBA FOCUS, 1955-67

Prior to 1955, cases of human trypanosomiasis had been recorded from Chirundu, where at the Alfred Beit bridge, the main road from Salisbury, Rhodesia, crosses the Zambezi to Lusaka, Zambia. It was assumed that these cases were all infected east of the main trunk road. Thereafter cases began to be reported west of the road in the area towards Kariba. As the preliminary work for the construction of the Kariba Dam had begun, it was feared that there might be a great increase in the number of cases of human trypanosomiasis and steps were taken to pass traffic along the access roads through "fly" chambers where an insecticide was sprayed on the vehicles in an enclosed space of the chamber. At the same time, insecticide was applied with a swing-fog machine to the vegetation around the

works and residential areas. The account of the anti-tsetse work carried out to protect the Kariba operation is described by Webster (1960). He also referred to what was possibly a "healthy" carrier case of trypanosomiasis. This was an African labourer who was admitted to hospital from the local prison for repair of an inguinal hernia. Trypanosomes were found in routine blood films taken on admission to hospital. He was quite asymptomatic and the source and duration of his infection was in doubt.

In 1959 five cases were reported as infected upstream from the Kariba Dam; a blood film survey revealed no "healthy" carriers. The next year two African cases occurred, one from the same village as three of the 1959 cases and the other the child of a "fly" gate attendant near Kariba on the south access road. During 1961-63 a total of three Europeans and six Africans were infected east of the dam on the shores of the lake where the Charara river enters. Tsetse fly in this area were particularly troublesome; one of the European cases had spent only a day or two in the area.

The African population in certain areas had to be moved from their homes, which were inundated as the lake filled. The lake reached its full level in 1963 and in the following year Africans living in their new villages away from the lake shore began to establish temporary fishing camps.

During 1964-66 11 European cases and 23 African cases were recorded—all from the same general area of the fishing camps on the river below the dam and in the bays in the lake east of the dam wall, where the Charara, Nyaodza and Sanyati rivers feed into the lake.

In 1967 there were eight cases of human trypanosomiasis, two European and six African, all but one of whom were infected in the area of the fishing villages east of Kariba. The African cases were all adult males who were temporary inhabitants of the fishing villages. By 1966 the inhabitants of the five fishing villages had ceased to be only African males who were temporarily engaged in fishing during the farming off-season. The fishing villages had assumed the role of being homes, albeit secondary homes, not only for fishermen, but their wives and families. In February, 1966, the total population of the five fishing camps was 251 men, 107 women and 205 children. The camps were at this time found free from tsetse fly except for Nyamunga's camp on the lake shore between the Sanyati and

Charara rivers. The bush between the camps was, however, infested with fly. It is interesting to note that six cases were directly associated with this camp in 1964 and 1966. Included in this number is one of the "healthy" carrier cases discovered by Dr. S. Graham in 1966. All the fishing camps were close to river or bush thickets in which tsetse fly were abundant and troublesome.

Since 1964 there have been increasing numbers of people patrolling areas in the Zambezi Valley, both up and down stream from the Kariba Dam. This has entailed fairly large numbers of Europeans and Africans who are not normally exposed to tsetse fly being bitten while on patrol operations in fly-infested areas. Despite this quite extensive exposure of fresh populations to tsetse flies in the areas east of the Chirundu road, west of the road and the area around the north-eastern end of the Kariba Lake, and the extensive area westwards along the southern shore of the lake to Binga, only three cases of human trypanosomiasis have been reported in patrolling personnel, all of whom were infected either near Kariba or in the fishing villages to the east of the dam.

DISCUSSION

Ormerod (1961) has given a masterly survey of the epidemic spread of human trypanosomiasis due to *T. rhodesiense*. He points out that it is not possible to distinguish *T. brucei* and *T. rhodesiense*; both produce heavy infections in experimental animals, and the only way these species of trypanosomes recovered from animals can be distinguished is that *T. rhodesiense* can infect man.

It is remarkable, however, that *T. rhodesiense* does not occur over the whole area where *T. brucei* is found. This is illustrated by the fact that not a single case of human trypanosomiasis has been discovered south of the Zambezi/Limpopo watershed despite the fact that there are tsetse fly areas in the south-east of Rhodesia which extend into Mozambique where all the conditions of dense *G. morsitans* and a plentiful game population exist. Ormerod (1961) supports the proposition that the original strains of *T. rhodesiense* arose in all probability in the Zambezi basin and spread northwards to be recognised as the epidemic strains seen in Malawi, Tanzania and eventually north to Uganda. It seems likely on epidemiological grounds that the disease as seen in Rhodesia since 1911 resembles the disease pattern seen in Botswana.

For 60 years cases of human trypanosomiasis have been reported in Rhodesia, most of the cases occurring in people who were infected in tsetse

fly areas within Rhodesia, although from time to time cases have been reported in African migrants from Zambia, Tanzania and Mozambique who had not visited or passed through tsetse fly areas in Rhodesia.

The indigenous cases can be grouped into three categories which are not clearly defined one from the other in the history of individual cases, but can be allotted to one of the three groups at a particular time in the history of a case. The categories are—

- (a) the "healthy" carrier case;
- (b) the sub-acute case with early neurological signs; and
- (c) the acute case with a rapid course if early treatment is not given.

The "Healthy" Carrier Case

This expression is not perhaps a good clinical description, but is a useful term in the study of the epidemiology of human trypanosomiasis in Rhodesia. As far as can be ascertained, these cases have all been Africans and have been discovered and diagnosed in similar circumstances. They give a history of having lived in tsetse fly-infested areas for a considerable period and generally have been inhabitants of small villages of about 50 persons or less. They are rarely women or small children and most commonly are people who are hunters, fishermen, honey gatherers, woodcutters, etc., who spend considerable time away from their village homes. At the same time it must be realised that women and children collecting wood and wild food also frequent the bush. The cases are found in areas where tsetse fly and game are plentiful. They are not found in large villages of 100 inhabitants or more and villages which are surrounded by stretches of cultivated land. These larger villages and the area of cultivated land discourage game, and the noise of village activities seems to keep tsetse fly away.

The "healthy" carrier case gives a consistent history, commencing with a febrile illness which is generally of little consequence and from which a full recovery is made. The patient returns to his normal occupations and probably remains in this state for months, and in fact does so until accidentally discovered. These cases are generally discovered by the finding of trypanosomes in a blood smear in a routine survey of the human population of an area, and the patients deny feeling ill and are generally apyrexial. Naturally, when such persons are discovered to have a try-

panosome infection of the blood, they are speedily removed to a medical centre and treated. Blair (1939) recorded the case of Kahondera, the headman of a village at Gowe, on the Umniati river in the West Hartley focus, who was found on 21st May, 1934, to have a heavy *T. rhodesiense* infection in his blood. He was re-examined early in July when he had walked 22 miles to pay his respects. His blood infection of *T. rhodesiense* was still present, but he was well apart from a degree of chronic bronchitis. On 11th July, 1934, he was visited at Gowe; his blood picture was unchanged. The second "healthy" carrier case from the same village, Zariro by name, was diagnosed on 11th July, 1934; trypanosomes were seen again in a blood film taken on 12th September after he had just walked 40 miles in two days, and again on 18th September, 1934, when his village was visited. It was difficult to persuade him to go to hospital for treatment, but eventually he walked 70 miles to Gatooma hospital escorted by a Native Department messenger, and his blood infection was confirmed at the hospital.

The significance of these two cases is that they were able, despite heavy blood infections of trypanosomes, to work hard and walk considerable distances, and both strenuously denied that they were ill. Kahondera had his blood infection for at least two months and Zariro for over three months with no deterioration in their condition and certainly no signs of any early involvement of the central nervous system.

In 1934-35, 12,839 Africans were examined in Sebungwe, West Hartley and North Lomagundi tsetse fly areas. At the beginning of the survey blood films were taken only if the patient appeared ill. When trypanosomes had been discovered in Kahondera and Zariro, persons who were not ill, it was decided to take blood films from everyone living in tsetse fly areas and 1,157 smears were examined, mostly in West Hartley and South Sebungwe.

In retrospect, it would seem that Fleming (1913) described two cases who would probably now be considered "healthy" carrier cases:—

Meleki, resident on the Lubu river, had been a personal servant at Kariangwe, the district headquarters nearby, for some time. In March, 1911, he visited his father's village on the Busi river about 20 miles to the east. He was ill when he returned to his work and was laid up for a week. Dr. Stohr found trypanosomes in gland juice and occasionally in the blood in October, 1911. He

asserted he was quite strong and well and that there was nothing the matter with him.

Sianyyna had his home on the Lubu river near Kariangwe, where he was employed as a carrier. About May, 1911, he went to the Zambezi river and returned by way of villages on the Busi river. He was not ill and was one of the carriers who accompanied the European case H.R.T. to the railway and had been living at his home village since. He was examined on 11th November, 1911, and trypanosomes were found in lymph gland juice. No trypanosomes were seen in his blood smear.

In 1946 it was observed that cases were being discovered in Africans who had travelled south from the north Lomagundi area north of Sipolilo. Of eight adult cases seen in this year, five had been working for five to seven months on farms or mines and the others for 19, 20 and 28 months, all employed in areas far from tsetse fly; yet when the disease first manifested itself the patients deteriorated rapidly, cerebral symptoms soon developed, and death occurred speedily in some cases.

Dr. Barlow in 1953 examined the population in the North Lomagundi fly area and 1,850 blood films were taken and examined. Two "healthy" carrier cases were detected in people who were not ill—a child aged 11 years with a heavy blood infection and an adult also in good health.

Webster (1960) described a case which seems to have been a "healthy" carrier case.

Dr. Graham in February, 1966, carried out a survey of the population of the fishing villages on the lake shore east of Kariba Dam, in which area a number of acute cases of trypanosomiasis had been attributed over the past four years. Blood films were taken from every person examined and two (or perhaps three) "healthy" carrier cases were diagnosed in 465 films.

(1) *Enos*, a young male aged 19 years, arrived at Nematombo's fishing camp on 1st February, 1966, from his home in the eastern part of the Urungwe Reserve, where tsetse fly are virtually unknown. The blood taken on 9th February showed trypanosomes; he was apyrexial and not feeling ill. It seems more likely that the infection was contracted on an earlier visit to the fishing camp rather than after 1st February.

(2) *Water*, an adult African, had lived at Nyamanga's fishing camp for a year. Clinically he was not ill and was apyrexial.

(3) *Thomas*, an adult African who lived in Kariba township, but his duties entailed that he visited the fishing camps collecting loads of fish for transport to his employers at Kariba. He was not ill, nor did he give any history of illness, and could see no reason why he should have to go to hospital.

It will be noted that no "healthy" carrier cases have been discovered in Europeans. All cases described have been in Africans and in Africans who have spent long periods of time in close contact with tsetse fly. It might be argued that some of these so-called "healthy" carriers were not carriers at all, but merely cases whose disease was diagnosed before it had proceeded to the stage of cerebral symptoms. This argument would not explain the two cases at Gowe described by Blair (1939).

The epidemiological importance of the "healthy" carrier case is that the person is not ill, continues his normal vocation and continues to have the trypanosome blood infection tapped by biting tsetse fly. If a "healthy" case continues to live and work in a tsetse fly area it can be readily understood why a number of other cases of human trypanosomiasis are found in the other inhabitants of the area, and particularly in people who visit the focus where the "healthy" carrier lives and works.

The Sub-Acute Case with Early Neurological Signs

People who continue living in tsetse fly areas where they are constantly being bitten by tsetse fly appear to develop a rather sub-acute form of the disease. Most of the cases described by Fleming (1913) are in this category. They give a history of several months of illness, loss of weight and an inability to carry on their work. Most of them were too ill to walk far and were confined to their villages or even to their houses. Because of their illness, such cases, even with a trypanosome infection in the blood, were not as exposed to tsetse fly as frequently as the active "healthy" carrier cases and so are of less significance in the maintenance of *T. rhodesiense* in tsetse flies.

The Acute Case with Rapid Deterioration

This type of case is seen not only in persons who live in the fly areas, but also in those persons who have only paid short visits to tsetse fly areas and who become ill with an acute pyrexial illness within some days after the introduction of the



infection; they progress rapidly to a fatal issue if a diagnosis is not promptly made and specific therapy given.

Acute cases of human trypanosomiasis began to occur in Central Africa about 1909; in Europeans who had entered tsetse fly areas on hunting expeditions and had soon afterwards developed the disease rapidly, with early death resulting. It is worth recording these early cases because they emphasise and illustrate how often the acute case has had only a brief contact with tsetse fly. Gelfand (1961) describes four such cases. Mr. O. Philips from Rhodesia contracted the disease in 1909 after a short visit to the Luangwa river valley on a hunting expedition. In the same year Messrs. Beith and Armstrong arrived back in Rhodesia with 10 Africans from a shooting expedition in the same area. They became ill soon after their return and trypanosomes were found in their blood. Both died of their disease. It was from trypanosomes recovered from Mr. Armstrong that Stephens and Fantham (1910) made their description of *Trypanosoma rhodesiense*. In 1910 a Dr. J. B. Greathead died near Serenje, Zambia, of acute trypanosomiasis while on a shooting expedition. Gelfand (1964) described the case of a Rev. Paul H. Roux who left Mvera, Malawi, on a shooting trip on 13th August and trypanosomes were discovered in his blood six days later. Two such European cases were described by Blair (1939). They paid a brief visit to the West Hartley area on hunting trips in June, 1933, and teamed up for three days at Gowe; they became ill six to ten weeks later. One was diagnosed as a case almost immediately, but died suddenly when undergoing treatment. The second case was at first considered to be one of relapsing fever. At the time the first case had been under treatment at another hospital, the second case returned to his mine and struggled on with his work for 3½ months before he was admitted to hospital and his condition diagnosed on 30th January, 1934. Blair described a third case, an African servant who visited tsetse fly country for four days in August and three days in September, 1933, when he accompanied his employer on a fishing visit to Gowe. He became ill on 10th January, 1934; trypanosomes were found in his blood and he died, despite treatment, in March, 1934.

Gelfand (1968) describes the case of an adult African who went in 1966 on a mineral prospecting expedition to the south shores of Lake Kariba. He contracted a fever soon after arriving in the area; the condition was thought to be

malaria, but failed to respond to treatment, and he returned to Salisbury near where he had lived all his life, never having had previous contact with tsetse fly. He was admitted to hospital a few weeks later, very ill; found to have trypanosomes in blood and cerebro-spinal fluid. He died a year later despite treatment. Gelfand had nine European cases in his care within the last eight years. All, except one, lived far from tsetse fly areas and had visited the Zambezi Valley on hunting and fishing trips of a few days' duration, though some of the cases had paid similar brief visits in previous years. All these cases developed acute trypanosomiasis within a matter of weeks of their visit to tsetse fly areas; only one case showed cerebral symptoms. All responded well to treatment.

The ninth case, the exception, was a European employed as a tsetse fly ranger who had lived on the edge of the fly area for five years, paying occasional visits of inspection into tsetse fly areas.

The only European case to occur in 1967 had an interesting history. He was a young soldier who had only two contacts with tsetse flies. In July he was patrolling in the Zambezi Valley near Feira for about 10 days. Few tsetse fly were seen. He then returned to barracks, where he remained for August and was fit and well. On 2nd September he went on patrol to the area between the Chirundu bridge and the gorge below the Kariba Dam. Tsetse fly were numerous and very troublesome, especially at the fishing camps on the river. He returned to his barracks on 2nd October, when he had a large well-developed sore. He became ill on 6th October and it was thought he might have malaria, but on 10th October he was found to have a heavy blood infection with *T. rhodesiense*. Despite treatment, he developed a cerebro-spinal fluid infection, but now seems to be fully recovered.

All the cases described shared one characteristic in that they had visited tsetse fly areas either only once for a few days or infrequently, and when they developed trypanosomiasis it was in an acute form. In other words, these visitors to a fly belt were unfortunate that they were bitten by a tsetse fly carrying the rather uncommon human trypanosome and so becoming ill with the acute form of the disease. Far more likely, however, is it for such an individual to be inoculated by the much more prevalent non-human forms of trypanosome which, although short-lived in man, set up a brief immunity in him and thus protect him if he is bitten again by a tsetse carrying *T. rhodesiense*.

The Progress of Disease from Infection to Diagnosis or Death

Every case of human trypanosomiasis starts as a blood infection. Presumably in the invasive stage the trypanosomes stimulate a reaction in the host with pyrexia and illness. In European persons, and in Africans who have only made fleeting visits to tsetse fly areas, the disease progresses rapidly to the stage of cerebral involvement, and the whole illness runs an acute course with an early fatal issue if specific treatment is not given. In Africans who live in tsetse fly areas and are constantly exposed to biting by flies, the disease can proceed as described above, but on occasions may run a more chronic course. It seems that only in this group will "healthy" carrier cases be discovered. Even when cerebral involvement occurs, the disease may run a much more chronic course. This is evident from the published work on the disease in Rhodesia, but is also clearly evident from unpublished records and hospital reports. An indication of the possible importance of constant fly exposure is shown by the experience of 1946, when a number of Africans left the area of the north Lomagundi focus or the neighbouring human trypanosomiasis areas of Zambia and Mozambique and sought employment on farms and mines far removed from any possible contact with tsetse fly. It must be assumed that these cases left the focus already charged with a trypanosome infection of their blood. Of the Rhodesian cases numbering five, four were juveniles leaving their homes for the first time to seek employment. Of eight cases whose movements could be checked, five developed symptoms of trypanosomiasis seven to 12 months after leaving tsetse fly areas and three others 19, 20 and 28 months later. There was an impression that the younger the person, the sooner the disease became manifest. Once the disease was clinically manifest it pursued an acute course. This is particularly so in a number of

the cases described by Fleming (1913). It has not been possible to carry out a test as suggested by Blair (1939) that a "healthy" carrier case once found should be observed at intervals, say, for a period of a year. If he continued to show trypanosomes in the blood and no cerebral symptoms for this time, it would be difficult to suggest that he was still only in the early stages of infection. Even in the limited time of two to three months Blair (1939) observed two cases who constantly showed a heavy blood infection with no physical deterioration or development of symptoms. What are the factors that would seem to be operating in such cases which prevent an invasion by trypanosomes from the blood into the cerebro-spinal system? Does a "healthy" carrier case, as distinct from an early case of the disease, maintain a trypanosome infection of his blood for months or even years? If this is so, the danger and importance of these cases are obvious.

Foci of Infections with Human Trypanosomiasis in Rhodesia

Table I and Figure 1 show the way in which at any one time in the history of human trypanosomiasis in Rhodesia the disease flares up in one area, only to die down to extinction, to re-appear some time later in another focus. The tsetse fly belts south of the central tableland in the Sabi-Lundi rivers system and the tsetse fly areas along the north-east perimeter of the country bordering on Mozambique, to the east of the north Lomagundi focus, seem never to have contributed any cases of human trypanosomiasis. In retrospect, and studying the incidence of cases over the past 55 years, it can be seen that a new focus of human trypanosomiasis becomes manifest to the health authorities by the discovery of acute or sub-acute cases in people who have paid short visits to a particular tsetse fly area. When such cases are found their previous movements should be carefully chronicled and an effort made to establish

Table I
DISTRIBUTION OF INDIGENOUS CASES OF HUMAN TRYPANOSOMIASIS IN RHODESIA

Period	Area	CASES	
		European	African
1911-19	South and West Sebungwe	5	12
1924-34	West Hartley area	2	20
1945-53	Zambezi Valley from Chirundu road east to Portuguese border	5	59
1954-67	Kariba and Urungwe west of Chirundu road	20	59
1958	Zambezi Valley (opp. Feira)	—	3

a likely site of infection. At the earliest opportunity, and especially if a number of cases are found who seem to have a common focus of infection, then an exhaustive blood film survey of the whole population of the area should be made. It is of little avail to try and discover persons who may be ill, thin, showing cerebral symptoms or who are pyrexial and limiting blood film taking to this group. If the whole population in an area suspected to be a focus of infection are seen and blood films taken, it may be that one or two "healthy" carriers may be found per 1,000 persons examined. In such areas the human population may be sparse and the small villages scattered over a large territory, but it is easier to take blood films from everyone and to spend the time saved by not carrying out detailed clinical examinations; in following up and blood filming all people who happen to be away when the village is visited—the hunters, the fishermen, the salt gatherers, the honey gatherers, the very groups of the African population living in tsetse fly areas in which infected persons, including "healthy" carriers, will be found.

How Best to Eradicate a Focus of Infection?

The Sebungwe and West Hartley foci were brought under control by a combination of two policies—the removal of all the inhabitants to new villages in tsetse fly-free areas and a policy of game destruction. These measures seem to have been satisfactory in eliminating both areas of human trypanosomiasis. Tsetse fly are still present in much of the first area, but have been completely eliminated from the second.

Except in rather special circumstances, these measures are unlikely now to be effective because of less room and facilities for mass movement of population. It is quite impracticable, for instance, to try and deny the Batonka people, who were dispossessed of their homes by the construction of the Kariba Dam, access to the lake shore to continue with fishing—a vocation they have practised for many years.

What can be done is to make their fishing villages more permanent, clear the bush from their neighbourhood and discourage the establishment of further small villages. The aim should be to have villages with, say, 100-200 inhabitants with the village surroundings well cleared of bush; with this arrangement man-fly contact for much of the population is greatly reduced, and if acute cases of human trypanosomiasis occur in visitors to such areas, extensive blood film surveys should soon uncover any early blood infection or "healthy" carrier case who can be removed and

treated. Past experience has shown that if these measures are taken early, further cases of human trypanosomiasis are not likely to occur.

Where Does the Human Infection Originate?

Ormerod (1961) discusses this problem at great length. Are people infected by *T. brucei* from a game host which might be considered as an accidental inoculation into man, or are human beings only infected by a strain of *T. brucei* (which we call *T. rhodesiense*) which has the power of being transmitted from animal to animal, but not losing its power to infect man when the opportunity occurs? Workers in East Africa, he shows, have maintained a strain of *T. rhodesiense* from a human being, passaged for many generations over years through animals which, when experimentally inoculated into human volunteers, produces human trypanosomiasis. It may be that if *T. brucei* is inoculated into a human being in a certain dose, in the right person it produces a blood parasitaemia only in the infected person, which may perhaps persist for months or years as a blood infection causing no serious illness and no cerebro-spinal involvement.

If human trypanosomiasis was caused by *T. brucei*, transferred by tsetse fly from an animal host, then one would expect cases to occur widely in the tsetse fly belts where this parasite is established and there are ample game reservoirs. This is not so. Human trypanosomiasis in Rhodesia is limited to certain small foci in the vast areas over which tsetse fly and game range, and is not found outside these circumscribed areas.

Recently large numbers of Europeans and Africans whose homes are in fly-free areas have been spending periods of time in the Zambezi Valley. Despite the widespread exposure of these people to tsetse fly in areas where the local African people live in villages throughout the area, only three cases have occurred in the visiting groups. The three acute cases were in persons who had visited and stayed near fishing villages on the Zambezi river below the Kariba Dam and on the lake shore east of Kariba.

Why Do the Local Population in an Endemic Focus Not Suffer from an Epidemic of Trypanosomiasis?

The Rhodesian experience seems to show a consistent pattern of events. An endemic focus of human trypanosomiasis first becomes evident when visitors to the locality contract acute infections soon after leaving the area. By back-

checking of the movements of such cases it can generally be found that they have one factor in common: a visit to one particular locality.

When the presence of a focus is suspected a clinical and blood film survey of the local inhabitants of the areas reveals few, if any, frank cases of trypanosomiasis and no history of an abnormal mortality consistent with trypanosomiasis. However, one or two persons may be found who have trypanosomes in blood or gland juice who give no history of illness nor can be said to be suffering from trypanosomiasis in its sub-acute or chronic stage with cerebral symptoms. Why, therefore, do persons paying brief visits into tsetse fly areas sometimes contract the disease in an acute form while the resident inhabitants contract the disease sporadically, although they are constantly exposed to attack by tsetse flies?

It is possible that persons constantly exposed to tsetse fly may receive inoculations of other trypanosomes such as *T. vivax* or *T. congolense*, which may be able to build up a non-specific resistance to infection by *T. rhodesiense* when this parasite is inoculated. This may be the reason why over a period of more than 50 years so few African hunters in the employ of the Game Department have contracted trypanosomiasis. The great likelihood is that anyone in these regions will be bitten by a tsetse fly harbouring *T. vivax*, *T. congolense* or *T. brucei* rather than by one carrying *T. rhodesiense*, since the latter is very uncommonly encountered in these parts. The infection with the non-human trypanosome dies out, but the individual is left with an acquired resistance, which is constantly being maintained by repeated inoculations of non-pathogenic trypanosomes.

If *T. rhodesiense* is just *T. brucei* which has an ability to infect human beings, it might be asked why cases of human trypanosomiasis have not been reported from many different localities where *T. brucei* abounds. If, however, as Ormerod (1961) suggests, *T. rhodesiense* is a human parasite which can exist in the antelope population as an infection quite indistinguishable from *T. brucei*, but maintaining its virulence for men when the opportunity of infection arises, one would expect to find very many more casual infections of human beings direct from antelope, and these would occur randomly throughout tsetse fly areas. This does not occur. Endemic foci in Rhodesia are generally of a very restricted size; the West Hartley focus of 1924-34 was probably restricted to the area of a single small village, and the same picture seems to hold now

for the fishing villages east of the Kariba Dam. In each endemic focus that has been recognised, careful blood film surveys of the whole resident population have revealed one or two "healthy" carrier cases. It is believed that when such cases are discovered, removed and treated, the endemic focus disappears. Can it be that the "healthy" carrier represents a person who by constant exposure to tsetse fly and to frequent inoculations of game trypanosomes develops an ability to allow a *T. rhodesiense* infection to establish itself in the blood stream, but the infection has not the ability to pass the blood-brain barrier and produce the signs of cerebral trypanosomiasis which so quickly becomes evident in persons who are visitors to an endemic focus and contract the disease? Presumably game and antelope can harbour blood infections of *T. brucei*, but do not succumb to cerebral symptoms.

SUMMARY

(1) Human trypanosomiasis (*T. rhodesiense*) has been known to occur in Rhodesia since 1911. Despite the fact that this disease spread in an epidemic form northwards in Africa about this time, no epidemic occurrence has been reported south of the Zambezi river, although the scattered and sparse population of human beings living in small villages in close association with tsetse fly and game animals seemed to provide conditions suitable for epidemic spread.

(2) The cases reported in any one year or period of years of indigenous origin have been small in number and at any one period have been traceable to infection contracted at an endemic focus of limited size; even a single village, in one instance, constituted the focus. Four such endemic foci have been in operation—one after the other, over nearly 60 years.

(3) The endemic foci have all been located on the Zambezi river side of the central watershed. No cases of human trypanosomiasis have ever been reported in Rhodesia in the Sabi-Lundi tsetse fly area.

(4) It has been found that each endemic focus appears to be based on one or more "healthy" carrier cases of the disease, persons with a trypanosome infection of their blood stream who are not ill and continue in their normal pursuits and provide a ready source of infection for local tsetse fly.

(5) Visits to an endemic focus by persons who live in fly-free areas, whether they be Africans or Europeans, result in a number of cases of

acute trypanosomiasis, and it is the occurrence of a series of such cases that generally draws attention to the existence of such a focus. It appears that the indigenous African population living in the focus do not succumb to the infection readily, and even when they do contract the disease it seems to run a more chronic course.

(6) The problems of the identity or otherwise of *T. brucei* and *T. rhodesiense* are discussed. If *T. rhodesiense* is merely *T. brucei* inoculated into a human being, one would expect cases to occur widely in tsetse fly areas where *T. brucei* abounds. This is not so.

(7) If *T. rhodesiense* is a human parasite able to maintain itself over the years by passage through game animals, the surprising localisation of cases is difficult to explain. It may, however, be a very rare parasite even in the Zambezi basin, where it is considered to have had its origin. If, however, it is inoculated into a person who, although developing a heavy blood infection, does not develop cerebral symptoms and a progressive deterioration of health, a "healthy" carrier is created and may be the source of the build-up of a more frequent *T. rhodesiense* infection of the tsetse fly in a particular locality.

(8) It is difficult to explain the tolerance of the "healthy" carrier to his infection and the general resistance of the indigenous inhabitants of an endemic focus to infection. It may be that the constant assault by tsetse fly infected with *T. vivax* and *T. congolense* may create some degree of non-specific resistance to infection with *T. rhodesiense* when this infection is introduced by a tsetse fly. It is also possible that when an individual, not previously exposed to tsetse fly, enters a focus in which cattle and human strains

exist, the great chances are that he will be inoculated with one of the non-human trypanosomes. These trypanosomes do not survive in him, but induce a temporary acquired immunity which depends on repeated inoculation of non-human trypanosomes. Should such a person then be inoculated with the much rarer human strain, he will not develop trypanosomiasis unless his state of immunity is not fully protective, in which event he will contract a mild form of the disease or become a "healthy" carrier.

(9) In investigating an endemic focus it is important to examine all the people in the area, including those temporarily absent. Blood films must be taken from all the inhabitants and not only those who are ill, have temperatures or have enlarged cervical glands. If a "healthy" carrier is present he will be active and well and strenuously deny that he is in any way ill.

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