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THE EPIDEMIOLOGY OF SLEEPING SICKNESS IN THE HISTORICAL LUANGWA VALLEY

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Summary — This paper outlines the prehistorical and historical background of trypanosomiasis in Africa, with special reference to the Luangwa valley. It is thought that the haphazard contact of early man with flies of the *G. fusca* and *G. morsitans* groups led to his insusceptibility to *T. brucei* strains in a forest and woodland-savannah ecology, while the more frequent contact with flies of the *G. palpalis* group made him to retain his susceptibility to *T. brucei* strains of a river-lake ecosystem.

In the *G. morsitans* surroundings of the Luangwa valley, man-infective trypanosome strains could only manifest themselves after the arrival of (Ba-)ntu speaking people some 2,000 years ago had led to increased population densities. In more recent years the most outstanding epidemiological phenomenon has been a slow recovery of the Luangwa fly belt after the 1896 Rinderpest epidemic.

Three main reasons are given for the occurrence of epidemic sleeping sickness in the endemic Luangwa valley. One of these is *« the collision of an expanding fly belt with the human habitat »*. Another factor is that climatic stresses and a lack of game animals on the northern edge of the Luangwa fly belt force tsetse flies to feed more often on man. Finally, game movements make that tsetse flies thrive and greatly increase in number during the rainy season in the same areas, where they starve, accumulate near villages and heavily depend on man during the dry season.

The significance of subacute *T. rhodesiense* sleeping sickness in Zambia and other south-eastern *G. morsitans* belts is elaborated. Its origin is believed to lie in a selection by man-infective trypanosome strains of people who are able to develop a more efficient immune response against them. That is why, in general, Europeans and Nilotes (e.g. in south-west Ethiopia) follow an acute course while the Bantu (e.g. in Zambia, Rhodesia and south-east Tanzania) usually follow a more subacute course of the disease. (Ba-)ntu speaking people have a long history of almost uninterrupted contact with tsetse flies and, presumably, man-infective trypanosomes.

1. **Prehistorical and historical background**

Baker (1962, 1968) and Godfrey and Killick-Kendrick (1967) have shown that our closest hominoid relation, the chimpanzee is susceptible to *T. brucei* (*Trypanosoma* (*Trypanozoon*) *brucei brucei*). Since pongid evolution has been less rapid and involved less behavioural and metabolic changes than man's evolution, it is tempting to assume that also our hominoid ancestors (or the common man-chimpanzee ancestor) were susceptible to *T. brucei* or *T. brucei*-like trypanosomes.

In a masterly review of Africa's and, especially, East Africa's prehistory Clark (1970) has suggested that the homonid and pongid line separated some 4 to 10 million years ago. From a forest dweller, early man turned into a savannah dweller with special preference for lakes and

rivers. Subsequently, he lost contact with the *T. brucei* strains of the *forest-fusca system.*

In the *woodland-savannah-morsitans system*, the abundance of game animals made early man an, altogether, dispensable food host. His increasing ability to catch tsetse flies made him also a dangerous one and led to the selection of man-avoiding flies (Bursell, 1973). As a result, the man-*T. brucei* relationship became more and more haphazard and infrequent. Eventually, the *T. brucei* strains of the forest, woodland and savannah could no longer keep track of man and his metabolic changes and lost their man-infectivity *.

In the *lake-river-palpalis system*, the selection of man-avoiding tsetse flies did not take place because of a lack of permanent and reliable hosts. Here, tsetse flies are unable to develop a steady food preference and have to « *adapt themselves to whatever sources of blood are available* » (Weitz, 1970). In these circumstances the human host can not be neglected. It is the frequent man-fly contact around rivers and lakes which prevented a loss of man-infectivity in the prevailing *T. brucei* strains.

For millions of years man-infective *T. brucei* strains were almost completely restricted to the riverine and lacustrine ecology of the *G. palpalis* system. At first these strains were quite virulent, as any loss of virulence would have upset age-old adaptation mechanisms with the game host. For a long time, even in *G. palpalis* surroundings, the latter was much more important for the survival and transmission of man-infective *T. brucei* strains than the human host. Only in the neighbourhood of densely populated, agricultural societies could the transformation into mild *T. gambiense* (*Trypanosoma* (*Trypanozoon*) *brucei gambiense*) strains take place (Ford, 1965). According to Clark (1970), agriculture and the keeping of cattle were introduced in West Africa some 5,000 years ago.

Increased human population densities later enabled man-infective *T. brucei* strains to manifest themselves in the woodland-savannah ecology of the *G. morsitans* system after many million years of absence (or near-absence and epidemiological insignificance). In the *G. morsitans* belts of Zambia's Luangwa valley, these manifestations probably started some 1,500 to 2,000 years ago. During the first centuries of the Christian era, Zambia and other parts of south-east Africa saw the introduction of iron and the arrival of (Ba-)ntu speaking people, who employed advanced farming methods and had a well-established political organization (Davidson, 1967; Fagan, 1970; Clark, 1970; Buyst, 1974). The resulting population increase favoured the transmission and spread of any existing, recently developed or newly introduced *T. rhodesiense (Trypanosoma* (*Trypanozoon*) *brucei rhodesiense*) strains.

Early workers in Zambia and Malawi, such as Kinghorn, Yorke, May and Bruce were all convinced that sleeping sickness was an old disease in these areas (Desart, 1914). There is no need to postulate that the

(*) Also the baboon's refractoriness to trypanosomes may be the end result of his ability to catch tsetse flies (according to Bursell (1973) « *the baboon probably catches more meals of tsetse flies than tsetse flies catch meals of it* ».

original spread of *T. rhodesiense* was a result of the colonial impact or a by-product of the « pax Britannica ». However, brusque changes which upset the man-fly equilibrium — obtained after many centuries of adjustment — led to epidemics in some areas (Ford, 1971).

The event which most brusquely upset the sleeping sickness situation in the Luangwa valley during the colonial and post-independence era, undoubtedly, was the Rinderpest epidemic, which almost annihilated this fly belt near the turn of the century (Ford, 1965; see also Hall, 1910; Neave, 1911). It was the subsequent recovery of the Luangwa fly belt which led to the detection of the disease in areas, where it had hitherto been unrecorded. The first documented case of sleeping sickness from the Luangwa valley was that of W. Armstrong. He travelled through the valley in 1909 and was later diagnosed in London. However, the most serious outbreaks in the Luangwa valley have only recently been recorded. On a survey in Chief Luembe's area (Petauke District, lower Luangwa valley), Rickman (1974) found human infection rates of up to 12.3 per cent. The author found annual infection rates of up to 16.5 per cent among people living in some villages in Chief Katyetye's area (Isoka District, northern edge of Luangwa fly belt) (Buyst, 1974).

The Isoka outbreak which had definite epidemic properties could be considered to be a late result of the 1896 Rinderpest epidemic. It is doubtful that with it the series of events which started before the turn of the century has come to an end.

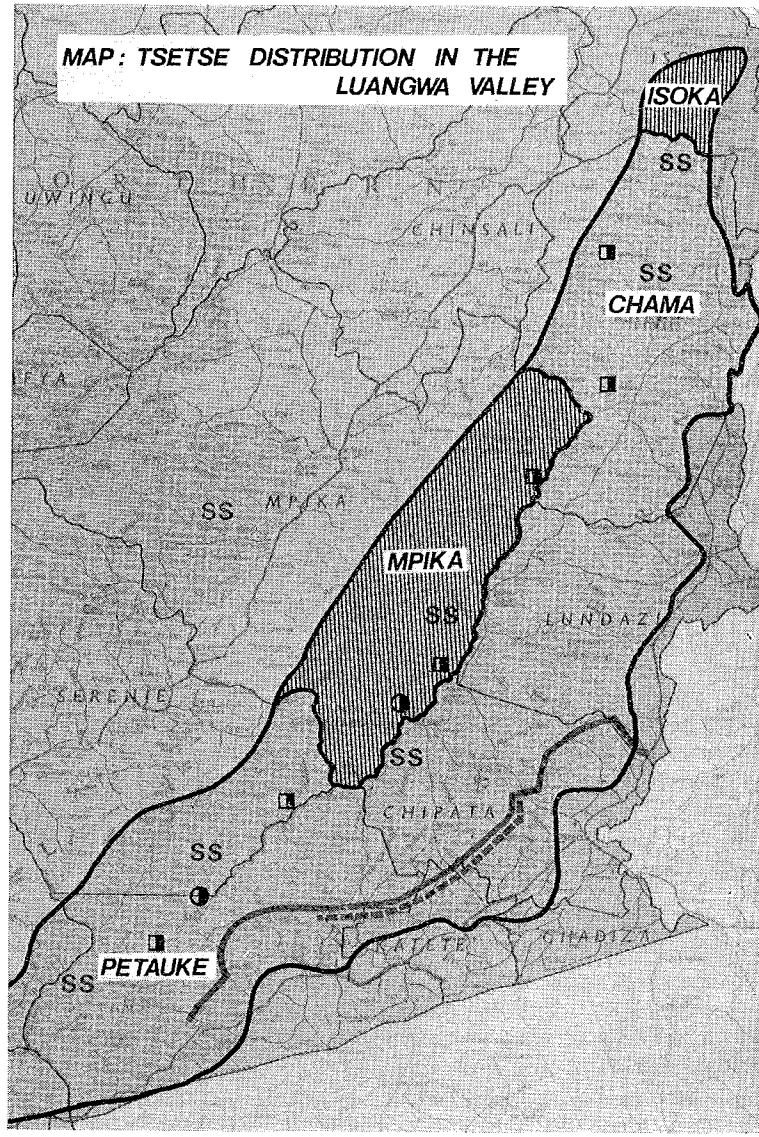
2. **Epidemic sleeping sickness in the endemic Luangwa valley**

In Mpika, the Luangwa fly belt covers a vast area and contains a very dense game and *G. morsitans* population. The Mpika fly belt is situated near the middle of the Luangwa valley, where high trypanosome infection rates have been recorded in both game animals and tsetse flies (Kinghorn and Yorke, 1912; Keymer, 1969; Clarke, 1969). From this enormous fly belt about 15 sleeping sickness cases were diagnosed in a 3-year period ending, May, 1970 (see also Buyst, 1970, 1976).

In Isoka, the Luangwa fly belt is much smaller (see Map) and has a much sparser game and *G. morsitans* population, while the number of people living in the infested areas is only slightly higher than in Mpika. From this small fly belt 241 sleeping sickness cases were diagnosed in just under 3 years (31 August 1971 - 7 July 1974).

Mpika and Isoka are hardly 125 km apart. In both districts cattle disappeared from the infested areas many years ago. Recent encroachment by tsetse flies of 2 cattle areas (Nachisitu, Muyombe) on the eastern border of the Isoka fly belt did not immediately give rise to a high number of sleeping sickness cases. *G. pallidipes* and *G. brevipalpis* have occasionally been recorded in Mpika, but never in Isoka. Three factors may explain why a serious outbreak occurred and endemo-epidemic conditions prevailed in the latter, while a situation of low endemicity and stable sleeping sickness existed in the former.

1. After the Rinderpest epidemic had swept through the Luangwa valley and almost completely wiped out its fly belt around 1896, tsetse



flies quickly reinvaded part of what is now Mpika District (Hall, 1910; Neave, 1911). Soon Kinghorn and Yorke (1912) were able to establish a field station near Munyamazi river in Chief Nabwalya's area (Mpika District). The reinvasion of the Isoka fly belt, on the other hand, started around 1935 and had not yet completely ceased by 1972 (Buyst, 1974). Similarly to what happened in the epidemic outbreaks of western Tanzania, *a collision of an expanding fly belt with the human habitat* (Ford, 1965) was partly responsible for the Isoka epidemic.

2. Since Isoka is situated at *the northern edge of the Luangwa fly belt*, *stress conditions* force tsetse flies to feed more often on man. There is a relative scarcity of game animals in the Isoka fly belt, which is higher, drier and colder, and undergoes more abrupt temperature changes than the rest of the valley. In areas with extreme climatic conditions flies emerge with a low fat reserve (Bursell, 1960, 1963). They quickly starve and more readily take their first blood meal from man. Where subacute cases of *T. rhodesiense* sleeping sickness prevail, starving teneral flies may easily become infected and, in suitable circumstances, they will transmit the disease to man.

3. In Isoka's climatic stress situation a slight *change in the availability of game animals* must be a matter of life or death for a number of starving tsetse flies. Migratory tendencies do occur as parts of the lower Luangwa valley get flooded during the heavy rains, when some game animals find rescue in Isoka.

According to local evidence, buffalo and roan antelope are each year seen in Isoka for only a limited number of months. (Jarman (1972) has recorded important seasonal movements of buffalo, warthog and other herbivores in unflooded parts of the Zambesi valley and has given supportive evidence for similar migrations in other parts of Africa. He found that game animals move away from the flood plains during the rainy season and return there in the dry season.)

Because of the abundance and even distribution of game animals in the Isoka belt during the rainy season, tsetse flies thrive, greatly increase in number and stay far away from villages. Consequently, men (hunters) are much more commonly infected than women and children.

In the beginning of the dry season the migratory animals return to (the flood plains in) the south, while the remaining animals are restricted to the immediate vicinity of permanent water sites in Isoka's relatively arid environment. As a result, tsetse flies become more dependant on man as a food source and some start accumulating near villages where they infect men and women in almost equal numbers. This intimate man-fly contact may lead to the appearance of a man-fly-man cycle of *T. rhodesiense* and an explosive, epidemic situation may evolve.

Quite characteristically, the age and sex distribution of Isoka's sleeping sickness patients underwent dramatical seasonal changes (see table, which takes into account the time lapse between infection and diagnosis). While *male-female-child percentages* were *66-24-10* for patients infected during the 1972 heavy rains, these changed to *34-41-25* for patients infected during the 1972 dry season epidemic.

However, once « early diagnosis and adequate treatment » had reduced the human trypanosome reservoir, almost cut off the man-fly-man cycle and restored endemic conditions, *T. rhodesiense* transmission near villages became relatively insignificant. Seasonal fluctuations in the age and sex distribution of sleeping sickness patients tended to disappear, the percentage of adult men remaining high throughout the year (*66-20-14*). Obviously, although they were still bitten by tsetse flies near their homes during the dry season, few women and children became infected.

In Mpika, on the other hand, where the Luangwa valley contains two game reserves and a game management area, animals abound throughout the year, irrespective of seasonal migrations. Here, tsetse flies never accumulate near villages and, male-female-child percentages of sleeping sickness patients do not undergo seasonal changes, but reflect endemic conditions (Buyst, 1970, 1977).

It is important to note that Isoka is situated 125 km north and Luembe — the other endemo-epidemic area in the Luangwa valley — 100 km south of a game reserve. Isoka is near and Luembe still in a game management area. An epidemic flare-up in an endemic *G. morsitans* belt of south-east Tanzania was described by Dye (1926, 1927) and occurred in an area with relatively few game animals but close to a game reserve. *Seasonal game movements* may be of greater epidemiological importance than has hitherto been suspected and should be studied from a new angle.

Other factors, which played an important role in the Isoka — and also in the Luembe — outbreak, were the existence of small scattered villages and the occurrence of subacute *T. rhodesiense* cases. These factors have been discussed elsewhere (Buyst, 1974; Rickman, 1974). However, the significance and origin of subacute *T. rhodesiense* sleeping sickness may need further elaboration.

TABLE

Showing seasonal fluctuations in male-female child percentages of sleeping sickness patients, infected and diagnosed in Isoka

PATIENTS MAINLY INFECTED DURING	MA-FA-C NUMBERS	MA-FA-C PERCENTAGES	COMMENTS
1971 DRY SEASON AND EARLY RAINS	27-19-9	49-35-16	MEN LEAD WAY TO HOSPITAL
1972 HEAVY RAINS	25*-9-4	66-24-10	LOW MAN-FLY CONTACT NEAR VILLAGE
1972 DRY SEASON AND EARLY RAINS	31-37-23	34-41-25	HEAVY MAN-FLY CONTACT NEAR VILLAGE
1973 HEAVY RAINS	9-2-2	70-15-15	LOW MAN-FLY CONTACT NEAR VILLAGE
1973 DRY SEASON 73-74 WET SEASON	29-9-6	66-20-14	CONTROL BY DIAGNOSIS-TREATMENT HAS MADE TRYPANOSOME TRANSMISSION NEAR VILLAGE ALMOST NEGLIGIBLE.

*this number includes one patient, originally diagnosed in Mbala.

3. **The significance of subacute *T. rhodesiense* sleeping sickness**

Wijers (1974, 1974a, b, c) does not believe that the human *T. rhodesiense* reservoir is of great epidemiological significance in Kenya and Uganda. He also minimizes the importance of the man-fly-man cycle of *T. rhodesiense* there, nonwithstanding the presence of *G. fuscipes*. This is not very surprising, because *T. rhodesiense* sleeping sickness in Uganda and Kenya has in general been described as quite an acute disease and, patient-fly contact must be minimal.

A different picture emerges in Zambia's (and other south eastern) *G. morsitans* fly belts. Foulkes (1970), Rickman (1974) and Buyst (1974) all agree that most sleeping sickness patients in Zambia do not feel very ill during an early stage of the disease and, that they are usually ambulant for quite a number of months. Examples have been given of how patients remained relatively well without treatment for periods up to 10 months after their initial diagnosis (Buyst, 1974). In the same paper it was reported that one patient « *diagnosed on a wet film during a population survey, at first asked permission to finish harvesting before coming to hospital, but on admission trypanosomes were demonstrated in the CSF, which also contained 44 leucocytes per c.mm.* ».

In contrast with the situation in Uganda and Kenya, the human trypanosome reservoir has great epidemiological significance in Zambia and, where tsetse flies regularly bite the human host, there is an imminent danger of a man-fly-man cycle of trypanosomes. If not proof, at least circumstantial evidence, of the epidemiological importance of infected patients has been given above, where it was reported that « early diagnosis and adequate treatment » drastically reduced the number of infections which were being acquired near the village during the dry season.

An interesting phenomenon, recorded in Isoka during 1972, was the effect the first heavy rains had on the number, age and sex of infected patients. The rising number of people infected with sleeping sickness around January, 1972, was a continuation of the dry season epidemic trend, but their age and sex distribution changed significantly and became typical for infections acquired during the rainy season (Buyst, unpublished data). Obviously, tsetse flies which had been infected in great number near the village by subacute sleeping sickness patients at the end of the dry season, diverted their attention after the first heavy rains to an increasingly abundant game host, infecting both the animals and their hunters.

4. **The origin of subacute** *T. rhodesiense* **sleeping sickness**

When trying to explain the existence of subacute sleeping sickness in Zambia and other south eastern *G. morsitans* fly belts, a number of facts have to be taken into account.

1. The same trypanosome strains which give rise to subacute sleeping sickness in some people living in endemic *G. morsitans* belts give an acute disease in non-immune visitors and laboratory rodents (Bevan, 1935; Lamborn and Howat, 1936; Blair, 1939; Deckelbaum and Lowenthal, 1970; Foulkes, 1970).

2. Treatment results in subacute patients are typical for *T. rhodesiense* — as opposed to *T. gambiense* — sleeping sickness [high mortality rate after Tryparsamide; high percentage of fatal complications during, low relapse rate after, Mel B (Buyst, 1975)].

3. A very acute type of *T. rhodesiense* sleeping sickness has been described in Ethiopia (Hutchinson, 1971; Baker, 1974), where exceptionally high prevalence rates of positive I. F. A. tests point to « *constant challenge with trypanosomes which are not infective to man* » (McConnell *et al.*, 1970).

4. Mixed infections of *T. brucei* or *T. rhodesiense* and *T. vivax* or *T. congolense* do occur in game animals and cattle (Kinghorn and Yorke, 1912; Keyrner, 1969; Geigy *et al.*, 1971; Geigy and Kauffman, 1973; Mwambu and Mayende, 1971; Willett, 1972).

Therefore, it is unlikely that mild *T. rhodesiense* strains are of epidemiological importance in the south eastern *G. morsitans* belts and, if acquired immunity plays any role at all in the pathogenesis of subacute sleeping sickness, it should be due to abortive infections with *T. brucei* or sub-infective doses of *T. rhodesiense* and not with *T. vivax* or *T. congolense.* The « *wide range of antigenically distinct strains* » in trypanosomes belonging to the *T. brucei* subgroup (Gray, 1969) makes it less likely that acquired immunity is an important factor in the field. However, Powell's finding (1976, 1976a) of functional cross-immunity between *T. brucei* and *T. rhodesiense* may have opened a new chapter.

Natural selection of a human host with an innate ability to develop a more efficient immune response to *T. rhodesiense* may also play a role. According to this hypothesis, the minimum infective dose of *T. rhodesiense* (and *T. gambiense*) is higher in people whose ancestors have had frequent contact with infected tsetse flies and have been selected by man-infective trypanosomes. When these people are inoculated with low doses of *T. rhodesiense* they completely resist, and when they are inoculated with somewhat higher doses they eventually throw off, the infection (see Buyst, 1974, where 3 cases of « *spontaneous cure* » are reported). However, when the same people are infected with massive doses of *T. rhodesiense*, they succumb but follow a subacute course of the disease.

Incidentally, this hypothesis is not inconsistent with extensive work on man-infectivity by Fairnbairn and Burtt (1946), who mention « *the high resistance to infection possessed by some men* ». It could hold the answer to some of the conflicting results which are characteristic of man-inoculation experiments. The latter's unreliability has still recently been emphasized (Rickman and Robson, 1974). When man-inoculation experiments are described, the origin and subsequent passages of the trypanosome strains are usually very accurately recorded but, unfortunately, little attention is paid to the volunteer, whose personal and ethnic background (with regard to tsetse contact) are seldom mentioned in detail.

Following the natural selection hypothesis, a very poor immune response may be expected in people whose ancestors have had little or no contact with tsetse flies during the last ten thousand year or more. Hence, the very acute disease, almost invariably reported, in Europeans and the relative frequency with which they are infected.

Also Nilotes, who are adjusted to a « hot and dry environment », as found in the extreme north of sub-saharan Africa (Hiernaux, 1974), must have had little contact with tsetse flies and man-infective trypanosomes until relatively recently. Hence, the very acute type of sleeping sickness reported in Nuer, Anuak and Masengo speaking people (all nilotic tribes) of south-west Ethiopia (Hutchinson, 1971). The virulent character of sleeping sickness in the north eastern corner of Lake Victoria and the high incidence of the disease among fishermen coming from these areas (Christy, 1903; Robertson, 1963), which stand out for their « *extraordinary mixture of*

ethnic and tribal groups » (Barnley, 1968) may be due to a predominance of nilotic (« nilo-hamitic ») genes.

(Ba-)ntu speaking people, on the other hand, are well adapted to an ecology of light woodlands with medium humidity (Hiernaux, 1974). They most likely originated in central Cameroon or in the central Benue valley of eastern Nigeria, migrated along the Zaire river system and continued their explosive expansion from a nucleus in the Luba area along the upper Lualaba (Oliver, 1970). They have a history, extending over several thousand years, of almost uninterrupted contact with tsetse flies and, presumably (see above) man-infective trypanosomes. This may be the reason behind the subacute type of sleeping sickness which has been reported for Kaonde and Senga speaking people in Zambia (Foulkes, 1970; Buyst, 1974), the Tonga speakers in Rhodesia (Ford, 1971) and the Batawana and Makoba in Botswana (Apted *et al.*, 1963).

Dye (1926, 1927) has described an epidemic « flare-up » of sleeping sickness in an area of south-east Tanzania, where he thought the disease had been endemic for a considerable length of time. Most patients followed a fairly subacute course, showing few symptoms and signs during the first 2 or 3 months of infection.

In western Tanzania, on the other hand, *T. rhodesiense* sleeping sickness was first recorded in 1920, when epidemic foci emerged in the aftermath of the Rinderpest epidemic, and a more acute clinical pattern was noted (Ford, 1971; Buchanan, 1929; Apted *et al.*, 1963). If sleeping sickness existed in western Tanzania prior to the Rinderpest epidemic, it must have been in a form of extremely low endemicity because of the low susceptibility to infection recorded in local tsetse flies and the low human population densities which prevail in the area (Ford and Leggate, 1961; Ford, 1965).

Also in Zambia, some (Ba-)ntu speaking people, whose ancestors have had little or no contact with man-infective trypanosomes for several centuries [e.g. Isoka's Benamwanga (Buyst, 1974)] have lost some of their « *immune ability* » and follow a more acute form of the disease. Perhaps, this may explain the acute type of sleeping sickness recorded in a district secretary and some other Zambian and Zimbabwean visitors to endemic fly belts in their country of origin (Foulkes, 1970; Blair, 1939). After staying on a tsetse free plateau for several centuries, some Bantu tribes may have partly lost or diluted those genes which gave them the ability to develop a more efficient immune response against man-infective trypanosomes and which they had acquired through a process of natural selection in their original West African home and during their long eastward march.

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P. de Raadt : The question of spontaneous cure : we have seen several cases in Kenya also and our conclusion has always been that this may have been a translent infection with *T. brucei.* Have you been able to observe the duration of their patent infection and can you give any details ?

H. Buyst : We found altogether some 14 positive cases on various surveys. It took some time before some came to the hospital and by the time we saw them again, 4 were negative.

We examined them every three months for about two years and when Dr Ormerod came after two years, he inoculated their blood in rats and they were negative. So it's very difficult to say whether these were abortive *T. brucei* or selfcuring *T. rhodesiense* infections.

P. G. Janssens : I am not asking a question, but want to say to Dr Buyst that even if his hypothesis is completely erroneous it remains a fascinating story. To my opinion it is important that someone at least does try to explain along some epidemiological lines the difficult problem of the differences in reaction of the individual human host against the same *T. rhodesiense.* What you are stating about visitors, whether Europeans or Africans is only too obvious : they run a very acute disease and to the opposite those living in the endemic area for generations they run a more subacute disease. One important conclusion is that the statement found in so many textbooks asserting that *T. rhodesiense* is always and only an acute disease is totally wrong.