Research note

POSSIBLE CAUSES LEADING TO AN EPIDEMIC OUTBREAK OF SLEEPING SICKNESS: FACTS AND HYPOTHESES

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Abstract: Sleeping sickness epidemics have been noted to occur with some degree of periodicity and the question as to why this is so has been asked for quite a long time. These epidemics have been partially controlled in the past using the conventional methods of bush clearing, mass diagnostic surveys and treatment. Political, social and economic upheavals have been found to be very important factors in the recurrence of these epidemics. In addition, a number of facts and hypotheses have been advanced as possible causes of epidemic outbreaks of sleeping sickness.

This paper presents a brief account of factual epidemic outbreaks of sleeping sickness in south eastern Uganda (Busoga) and then proceeds to discuss, in general terms, a number of hypotheses that have been incriminated to date, as possible causes that might lead to an epidemic outbreak of the disease.

HISTORICAL PERSPECTIVE

The sleeping sickness epidemic which devastated the shores of Lake Victoria at the beginning of this century is a famous event in the annals of tropical medicine. It is famous because an estimated one quarter to one third of a million people lost their lives (Langlands, 1967). The same epidemic brought controversy as to whether Dr. Castellani or Colonel Bruce first identified the trypanosome as the cause of the epidemic (1962).

The cause of the epidemic was attributed to *Trypanosoma gambiense* introduced to this part of the country by a party accompanying the explorer Lugard from the Congo basin on relief of Emin Pasha expedition in 1894 (Christy, 1903). However, more properly the cause lay in the general increase in social, commercial and military mobility which developed throughout tropical Africa in the late nineteenth and early twentieth centuries.

Another outbreak involving about 2,500 persons occurred in the same area from Jinja eastwards to the border with Kenya between 1939 and 1945 (Machichan, 1944). The most striking feature of this epidemic was the virulence and rapid course of illness as contrasted with *T. gambiense* infection. An early and profuse appearance of posterior nucleate forms and a rapid and fatal course of the disease was observed on animal inoculation. Thus, it is believed, the epidemic was caused by *T. rhodesiense*. The first cases detected were among migrant workers employed on Kakira Sugar estates. Since these immigrants came from

areas of reasonable proximity to the infected areas of Tanganyika (now Tanzania), this epidemic was thought to have been introduced by them.

Since that outbreak, cases continued to be reported from within the infected area, though not in epidemic numbers. In 1971, infection spilled north of the usual focus and involved up to 169 persons. Through the combined efforts of EATRO (now UTRO) and the Ministry of Health, this outbreak was soon brought under control.

PRESENT SITUATION

Following the control of the small epidemic of 1971, surveillance programmes were not instituted because of the prevailing political and economic atmosphere in the country at that time. There was indiscriminate and haphazard movement of people and livestock across the traditional trypanosomiasis barrier zone. Besides smuggling of commodities including cattle between Kenya and Uganda across the zone, became a means of livelihood. It was therefore difficult for the Ministry of Health teams to enforce surveillance measures. The Tsetse Control Department could not carry out control programmes due to lack of insecticides, transport and manpower. Thus, there was total breakdown of control measures and hence, by 1976, the stage was set for another epidemic outbreak of the disease in the area.

In August, 1976 a report was received at the then East African Trypanosomiasis Research Organization (EATRO) now Uganda Trypanosomiasis Research Organization (UTRO) of an outbreak of sleeping sickness in Luuka County of Iganga District. A preliminary survey by EATRO (now UTRO) medical team revealed 12 positive cases out of 812 persons examined (prevalence rate of 1.5%) (EATRO unpublished observations, 1976). Unfortunately, in June 1977, the East African Community collapsed and EATRO lost valuable logistics to a partner state. These included vehicles and laboratory equipment. In addition, several members of staff were forced to go into exile because of the harsh political and economic atmosphere prevailing in the country at the time of Amin's rule. Thus, UTRO, probably the department in the best position to help contain this epidemic during its outset, was rendered helpless.

The Ministry of Health, Uganda Government, posted microscopists to the area and later opened up treatment centres in the area. Since then, however, the incidence of the disease has continued to increase from 52 cases in 1976 to over 8,000 cases in 1980 (see Table 1).

HYPOTHESES OF EPIDEMIOLOGICAL IMPORTANCE

To date, a number of general hypotheses have been advanced to explain the occurrence of sleeping sickness epidemic outbreaks. Some of these hypotheses are discussed as follows.
Increased man-fly contact:

This phenomenon occurs most commonly during the hot, dry season with the result that transmission is enhanced. Indeed a period of drought almost invariably means an increase in the number of infections because few sources of water are shared by man, fly and game animals, in the case of *T. rhodesiense*, in close association; there is also more hunting and more searching for wild forest products at times when crops are bad. This phenomenon has been described by Willet (1965) and many other workers.

Changes in climate, vegetation and tsetse fly distribution:

Climate appears to be of more than ordinary importance. At higher temperatures, there is increased salivary gland infection rate in the tsetse flies, and in addition to this direct effect, there are many ways in which the climate influences a closer association between man and fly.

In Kenya a succession of heavy rains provided *G. f. fuscipes* with suitable conditions well outside its usual riverine habitat so that it was able to live and breed in the vegetation surrounding the homesteads-conditions which gave rise to the Alego outbreak of 1965. This same factor has had a role in the current epidemic in Uganda where the abundant growth of *Lantana camara* thickets near homesteads, has attracted *G. f. fuscipes* into peridomestic contact with man.

There is too the influence of climate in determining where people choose to live, and the population density of both flies and human beings, as discussed by Ford (1965) and this is relevant to the proper use and planned full development of land which is the ideal at which to aim in the eradication of the tsetse fly and trypanosomiasis.

Infection rates in the tsetse flies and their infectivity:

Wijers (1958) observed that infection rates were highest in flies taking an infective blood meal on the day on which they emerged, somewhat lower on the second day after emergence, and did not occur thereafter. Thus the fact that flies emerging during the hot season are likely to feed early in their adult life means that infection rates in the fly are maximal during the hot, dry season. However, the number of trypanosomes inoculated by an infected tsetse fly varies greatly even among flies infected from the same host and in the same fly at different times.

It has been estimated that the minimum infective dose of *T. rhodesiense* for man is of the order of 350 parasites and it is probable that a similar number would be required to establish infection with *T. gambiense* (Fairbairn and Burtt, 1946)

Population density of the tsetse flies and their feeding behaviour:

A tsetse fly feeding on a number of animals and possibly also on man, may become infected with many different strains of trypanosome. Most of these strains will be non-pathogenic for man and even if a man-infective strain is acquired by the fly, the tendency will be for it to be so diluted by the non-pathogenic strains that it will not be passed on in a number sufficient to cause an infection in man.

Tsetse longevity:

Flies emerging at the end of the hot, dry season are particularly receptive to trypanosome infection since they will feed early in adult life. With the onset of the rainy season, the

Table 1 Annual incidence of sleeping sickness in south eastern Uganda 1976-1987

Year	Number of cases
1976	52
1977	586
1978	2,076
1979	4,991
1980	8,465
1981	1,938
1982	1,309
1983	1,199
1984	1,956
1985	3,517
1986	4,446
1987 (up to end of July)	5,053
Total	35,588

expectation of life of the tsetse flies is maximal so that a combination of these factors produces a situation in which infected flies are liable to survive for protracted periods which enhances the potential for these flies to transmit the disease, of course depending on their infection rates.

Presence of domestic and wild animal reservoir hosts:

The pig in the case of *T. gambiense* and cattle in the case of *T. rhodensiense* have been incriminated as domestic animal reservoir hosts, while the kob and hartebeest in the case of *T. gambiense* and the bushbuck in the case of *T. rhodesiense* have been incriminated as wild game reservoir hosts. The bushbuck is particularly important because it tends to live in thickets near human habitation which puts it in close contact with man.

Appearance of different forms of the parasite:

The appearance of such parasites may be due to either the parasites being introduced from outside the area or genetic changes in the parasite.

There is at least a suspicion, based on field observations, that zymodemes of trypanosome introduced into fresh localities may exhibit an enhanced ability to spread through the community. Scott (1961) reported two instances in which the introduction of infected persons from an established epidemic area resulted in sharp outbreaks of the disease in endemic localities far removed from the original focus of infection. There are other similar observations suggesting that severe local outbreaks which quickly follow the introduction of infected persons to fresh localities are in some way connected with enhanced ability of the zymodeme to spread. Indeed, the possible existence of epidemic trypanosome zymodemes has been advanced by some workers.

Changes in population movements and population growth:

It is generally supposed that population movements are liable to precipitate epidemics. Refugees displaced as the result of war, famine, earthquakes and other similar occurrences are notoriously prone to disease in epidemic form as are also immigrant labourforces recruited for large scale construction work (tropical aggregation of labour) and pilgrims attending major religious festivals.

A new population in an area may spark off an epidemic outbreak of sleeping sickness in the area as a result of imported cases among them, which may be sufficiently large to augment the reservoir of infection available to the insect vector and so in a quantitative manner promote transmission. An imported strain may also show quantitative differences such as enhanced virulence or ability to spread or may be one to which the indigenous population has not been previously exposed and to which no resistance has been acquired. This phenomenon can also operate vice versa. Further, as with some other diseases, the periodicity of epidemics of sleeping sickness may be associated with growing up of a new generation of people with no previous experience of the disease.

Occurrence of subacute cases of the disease:

Another factor which might conceivably influence the spread of sleeping sickness is the presence of an undetected and perhaps unsuspected reservoir of infection in the form of human 'healthy carriers' of the disease which has been reported by several workers (Buyst, 1977; Rickman, 1974; Woodruff, 1982).

Under conditions in which man-biting tsetse are common where people congregate, the ambulant human carrier assumes a powerful potential for the onward transmission and spread of sleeping sickness. The exact extent of asymptomatic carriers is certainly low.

However, sleeping sickness cases with non-specific symptoms (fever, headache) who remain ambulant for several weeks are common, and they too may be important reservoirs of infection where man-fly contact is intense (Wurapa, 1984). This threat is also present among many early cases of the Gambian disease in which the initial stages are generally relatively mild and the victim may continue at work for many months or even for years, before he is eventually driven by increasing illness to seek treatment or to retire to his home. During this time, he is a constant source of infection to tsetse so that the very nature of the illness provides great opportunities for its spread. In the Ugandan situation, however, the question of delayed diagnosis and treatment is a big factor.

Human behaviour and activities in the fly habitat:

Man becomes infected during travel, hunting, fishing, collection of honey, or when working in the fly bush. Fishing and 'honey-hunting' are particularly hazardous occupations. While fishing in the riverine pools surrounded by thickets, man may be in close contact with tsetse fly for many days at a time, a situation in which the association between man, bushbuck and the tsetse fly is likely to be significant and therefore conducive to transmission and spread of the disease. Wyatt *et al.* (1985) working in north east Zambia found fishing to be more common among cases of sleeping sickness than controls and that it represented a hazard either while walking to the stream or while engaged in the activity of fishing itself.

Large regional and national development projects:

Projects like the on-going Onchocerciasis Control Programme in the Volta River Basin, construction of dams for power generation or irrigation, and agricultural or agro-industrial projects bring about changes in the local ecology in general. Moreover, such projects attract migrants and, whether spontaneous or planned, the resulting settlements are often inadequately supervised from the health and sanitation point of view. Also, the risk of sleeping sickness is rarely specifically considered when such projects are planned.

Political and economic upheavals:

These cause extensive and often uncontrolled movements of people into areas that were previously abandoned because of epidemics thereby promoting circulation of the parasite in the population and the risk of contact between people and the tsetse flies. These upheavals lead, in the final analysis, to a breakdown in vital social services including systematic medical and vector surveillance programmes. This is obviously the most important factor in the case of the current epidemic in Uganda as is the case in other countries like Angola, Mozambique and the Sudan which are also bedevilled with civil strife.

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