REMARKS ON TRYPANOSOMIASIS IN RELATION TO MAN AND BEAST IN AFRICA.

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There is perhaps no disease that illustrates the association of zoology with pathology—both human and veterinary—more tragically than African trypanosomiasis or tsetse-fly disease, the natural history of which has been under continuous investigation since the classical discoveries of Bruce thirty-five years ago.

Research on the tsetse-fly problem continues and reliable data, slowly acquired by experiment and observation over vast regions of Africa, are accumulating to bridge the remaining gaps in our knowledge of the subject.

Now, in the light of such knowledge accumulated during the last seventeen years I can no longer hold the opinion to which I subscribed in the recommendations of the Royal Society's Commission on Sleeping Sickness in 1913, advising the extermination of wild animals in the "fly" country.

The alleged rôle of the great African Fauna in maintaining the tsetse-fly population and the incidence of human trypanosomiasis or sleeping sickness is still a debatable point and, as it is of special interest to zoologists, I propose to state the case for and against the game very

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The paper, illustrated by lantern slides, was read before the Zoological Society. It will not be published in their Proceedings, but has been accepted for publication in the Journal of the Society for the Preservation of the Fauna of the Empire.
briefly but I hope impartially; and incidentally to mention some of the more important observations that have caused me to modify my former views. There appears to be little doubt that the large ungulata on which the existence of the tsetse-fly was supposed to depend have been destroyed almost to the point of extermination in certain parts of the fly country, but the tsetse has remained in these districts as elsewhere; and its eradication, except in small areas that can be kept clear of bush, seems to be impracticable when one considers the vast areas over which some species of this noxious pest are distributed. Incidentally I may mention that on the two Portuguese Islands, St. Thomas and Princes Island, in the Gulf of Guinea, where there are no wild animals, tsetse-flies are numerous and outbreaks of sleeping sickness not uncommon, the causal trypanosome being introduced from time to time probably by native labourers from the West Coast. No man can foretell the result of depriving the tsetse of its accustomed diet of wild animal's blood; but so long as there are men and cattle about the "fly" will not starve, and evidence from various parts of Africa is accumulating in support of the view that game destruction, instead of being a remedy, increases the danger from the fly by causing dispersal of the insects which subsequently concentrate around villages in the bush where they feed mainly on man and inoculate into his blood certain animal-derived parasites which in the game are harmless, but become lethal when introduced into man and his domestic animals.

Instances in which annihilation of the large fauna has been followed by complete and permanent deliverance from the tsetse pest obtain only in certain small isolated spots of territory, south of the Zambesi from which the fly disappeared during the latter half of the last century, notably in parts of the Transvaal and Southern Rhodesia where European occupation and permanent deforestation has followed the destruction of game. Here again, reference to a map indicating the distribution of tsetse flies will show that it is impossible to apply to half a continent, measures that have been successful in the case of a few comparatively minute and isolated spots in the vastness of Africa. On the other hand instances where the extermination of game has failed to rid the country of the pest are numerous and increasing. The Director of Veterinary Research in Southern Rhodesia in a recent report (1928) [1] states:

"For several years large sums of money have been spent in the endeavour to eliminate the fly by eradication of the game upon which it is thought to depend, but these operations have not been successful."

An experience that is corroborated by many recent observers. The following may be cited:

An organized thorough and prolonged killing of game, resulting in the exile of large animals from a wide area in Tanganyika, south of Lake Victoria, was followed by an epidemic of sleeping sickness, from which over 600 natives in the experimental area died as a result of infection with *Trypanosoma brucei* vel *rhodesiense*. Swynnerton published an exhaustive
report on the entomological aspects of this outbreak [2]. He came to the conclusion that in the absence of game the tsetse-fly concentrates on man, and he states there is evidence to show that *T. rhodesiense* in man can originate from *T. brucei* in game in places where the fly has been forced to feed on man; and an infected man entering a village or locality where man is the centre of attraction for the tsetse is the primary factor in spreading the disease, which, once man has acquired, becomes solely man-carried and man-derived.

Lyndhurst-Duke, investigating the epidemic from the epidemiological aspect, came to the same conclusion. He found the natives had killed their cattle for food on account of famine, and in the absence of game, the fly also turned to man for sustenance, with the apparent result that he acquired a *T. brucei* vel *rhodesiense* infection, and the parasite, increasing in virulence in the human body, was rapidly transmitted from man to man by mechanical transmission on the needle-like proboscis of the fly.

Dye, investigating another outbreak of *T. rhodesiense* infection in another locality, states: “The observations recorded point to man being much more dangerous to man than any species of game, and that the infection is carried from man to man by the local village tsetse-fly.” Moreover, he found that after the human population had been evacuated from the infected area, the locality in due course became healthy again and safe for man, despite the presence of tsetse and the increase of game during his absence [3].

Lloyd and others working in Nigeria confirm the finding that in the absence of game the fly takes to feeding on human blood, and therefore they do not recommend game extermination, but wisely state “any increase of wild ungulata should be watched and should be checked if found to result in increase or spread of the fly.”

There is evidence, therefore, that a parasite normally and harmlessly living in the blood of game animals may, under certain conditions, become adapted to living in human blood in which it develops lethal properties.

Attention has been drawn to an experience that appears to indicate the occurrence of the converse process in which a pathogenic trypanosome (*T. gambiense*) distinctly pertaining to man and carried by another species of tsetse (*Glossina palpalis*) has, after prolonged sojourn in the blood of the game during the absence of its human host, apparently reverted to its ancestral type, the distinctly animal trypanosome, *T. brucei*, and so has lost its pathogenicity to man.

The survivors of the great epidemic of sleeping sickness, caused by *T. gambiense* infection that decimated the population of the Sese Islands in Lake Victoria at the end of last century, were evacuated, and the islands remained absolutely uninhabited by man for twenty years.

The Sititunga antelope (*Tragelaphus spekii*) that is normally a host of trypanosomes of the brucei-gambiense type, increased enormously during the absence of man, and spread all over the islands, replacing man in
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providing mammalian blood for the tsetse-fly. Since 1919, several thousands of natives have been allowed to return to the islands where they have continued to live in close contact with the fly and game; yet, according to the most recent reports available, not one case of sleeping sickness has occurred among the island population.

If it can be proved that the trypanosome loses its virulence for man by passing into game, there will be a very good argument in favour of game protection.

It remains to be seen, however, whether this vindication of the game can be generally substantiated, but I think it gains some support from experiments by Taute and Huber, two German investigators operating in East Africa. Taute injected into himself antelope blood containing *T. brucei* vel *rhodesiense* which is believed to be the cause of human trypanosomiasis in Nyasaland, Rhodesia and Tanganyika. He also allowed himself to be bitten by tsetse flies infected with the parasite by feeding on an infected animal. Later he and Huber repeated the experiment on themselves and also inoculated 129 natives with animal blood containing *T. brucei* vel *rhodesiense* [4]. None of the persons under experiment became ill.

These experiments are of great interest, but all they show is that man is resistant to a certain animal parasite, viz., *T. brucei*, of which *T. rhodesiense* and possibly *T. gambiense* are variants or races that become habituated to living in human blood.

Further experiments are necessary to show whether a game trypanosome can become adapted to living in human blood, which in vitro appears to be an unfavourable medium, or that the trypanosomes *T. rhodesiense* and *T. gambiense* are human parasites only, like those of malaria. This seems to be an improbable supposition in view of the facility with which these two parasites can be conveyed from a sleeping sickness patient to experimental animals in whose blood they are indistinguishable from those trypanosomes commonly found in naturally infected game.

Now Warrington Yorke, Adams and Murgatroyd have demonstrated that animal-derived trypanosomes, namely, *T. brucei* vel *rhodesiense* and *T. congoense*, are rapidly destroyed in contact with human blood plasma or serum at normal blood-temperature 37° C., whereas *T. gambiense* is unharmed. This seems to afford grounds for assuming that man’s immunity to infection with the pathogenic trypanosomes of animals and his relative susceptibility to *T. gambiense* is bound up with this property of his blood-serum.

It must be noted, however, that after a period of several years’ passage through animals, a strain of *T. gambiense* was found to lose its resistance to the trypanocidal action of human serum and became readily destroyed by it.

In other words *T. gambiense* after prolonged sojourn in the blood of animals may revert to the *T. brucei* type as regards its serological reactions, a laboratory finding which, if confirmed, will form an interesting comparison with the Sese Island experience.
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After an elaborate and most important series of experimental investigations, Warrington Yorke, Adams and Murgatroyd enunciated the hypothesis that:

*T. gambiense*, like *T. rhodesiense*, is identical with *T. brucei*, the difference between them being due to numerous passages through the human host.

The source of both trypanosomes pathogenic to man—*T. gambiense* and *T. rhodesiense*—is *T. brucei*, of which the natural reservoir is the game.

The game trypanosome is not pathogenic to man because of the protective trypanocidal action of his blood.

Prolonged man-glossina-man passage has produced the modifications of the parasites which have resulted in the characters of *T. gambiense*, the most striking of which is its fixed resistance to the trypanocidal action of human serum [5].

In human trypanosomiasis, therefore, there is an unknown factor whereby certain animal trypanosomes can be adapted to live in human blood and destroy millions of men.

I hesitate to speculate as to what this factor may be, but I hope experiments may be carried out with a view to ascertaining whether the nourishing of the game-derived-trypanosomes, partly or wholly, on human blood during the prolonged cycle of their development in the body of the insect host, is an influence in adapting *T. brucei*, and possibly other animal trypanosomes, to life in the human body.

The solution of this problem seems to be possible only by carrying out elaborate fly-feeding experiments in which man would have to be the experimental animal.

The discovery of a safe and certain cure for human trypanosomiasis may eventually permit of such experiments being carried out. In the meantime, I submit, we have reason to believe that the large African fauna may act like a buffer state in absorbing the attacks of the tsetse-fly population and that by confining the transmission of their trypanosome parasites to their natural vertebrate hosts, the big game are a means of holding up the fly and preventing its dispersal through the haunts of man and the dissemination of trypanosomes among the human population.

I will conclude these remarks with a quotation from a most exhaustive treatise on the tsetse-fly problem, by two of the world’s greatest authorities on the subject, Major Austen and Emile Hegh [6].

"Though there be definite grounds for considering big game as constituting a reserve of food enabling tsetse-ﬂies to subsist in a given region, there is no scientific proof that the presence of these animals is indispensable to the continued existence of the fly."

In view of our limited knowledge the utmost caution seems to be necessary before advising the taking of measures (i.e., game destruction), which would have irreparable results.
REFERENCES.


