A Naturalist on Lake Victoria, G D Hale Carpenter, London, 1920

(G.D. Hale Carpenter, 1882-1953, English entomologist and medical doctor worked in Uganda, 1910-16, for the Colonial Medical Service on tsetse flies and sleeping sickness and then in Tanganyika till 1918. This was his first book; he later specialised on mimicry in butterflies and co-authored *Mimicry* with EB Ford in 1933, one of the first texts on ecological genetics, He was Professor of Zoology at Oxford University, 1933–1948. All footnotes are the authors; the endnotes are links to modern biographies)

Sleeping Sickness

In this chapter a general account will be given of the history and symptoms of Sleeping Sickness as it was known during the epidemic in Uganda. It may be said at once that the diseases is entirely confined to tropical Africa, nor does there seem any reason, as will be explained later, to fear its spread beyond Africa[i].

In a few words it may be said that the cause of the disease is a minute unicellular creature, called a Trypanosome, belonging to the lowest order of animal life, which is as it were inoculated by the bite of a blood-sucking fly, the Tse-tse, or *Glossina*,

A common history given by patients who suffer from Sleeping Sickness is that they have been in a country where they were much bitten by Tse-tse flies, and that after a few days a painful swelling has appeared on the neck, accompanied by high fever. The swelling may appear to be on the point of becoming an abscess, but does not do so, and gradually subsides. It is probable that this represents the site where the fly which was the cause of the infection actually bit. The fever may subside in a few days, and recurs at irregular intervals lasting weeks or months; it often reached a high point attended with delirium.

There is a good deal of headache, debility and languor, and vague pains in legs. An interesting feature is the appearance of an erythematous rash, mainly on the chest and back. There is a great wasting and enlargement of the glands of the neck. Another interesting feature is puffy swelling of parts of the face and body: this oedematous swelling is a particular feature of the diseases of animals, which are, as will e seen, so very closely connected with Sleeping Sickness.

This condition may go on for years, and has been known to disappear altogether with an apparent cure; it is known as *Trypanosomiasis*. Next comes the stage to which the form Sleeping Sickness more properly applies. The drowsiness becomes accentuated, as that the subject takes no interest in his surroundings and does not trouble to eat, though he will eat food if it is brought to him, and he is fed. The fever continues irregularly, the eyes become more puffy, the lips and tongue tremulous, the wasting more and more pronounced, until death finally supervenes with the patient in a state of coma. At the last there may be mania and convulsions.

It has been noticed that natives suffering from Sleeping Sickness appear to feel the cold very acutely, and will often sleep so near a fire that they inflict severe burns on themselves. This also shows how the senses are dulled, so that one can conclude that they cannot suffer much.

Death may occur in a few months after the initial fever, but more usually after one to three years.

The earliest account of Sleeping Sickness which is known in print (London) dates from 1742 and is of considerable interest because it reflects the current medical opinion of the day.

Dr E D Warren in the *Malay Medical Journal* or April 1911, drew attention to a book by a naval surgeon named John Atkins, called *The Navy Surgeon: or a Practical System of Surgery with a Dissertation on Cold and Hot Mineral Springs and Physical Observations on the Coast of Guiney*. The following extract is given in *The Sleeping Sickness Bulletin*.

"The Sleepy Distemper (common among negroes) gives no other previous Notice, than a Want of Appetite two or three days before: Their Sleeps are sound, and Sense of Feeling very little; for pulling, drubbing, or whipping, will scarce stir up Sense and Power enough to move; and the moment you cease beating, the Smart is forgot, and down they fall again into a state of Insensibility, drivelling constantly from the Mouth, as if in a deep Salivation; breath slowly, but not unequally, nor snort.

"Young People are more subject to it than Old; and the Judgment generally pronounced is Death, the prognostick seldom failing. If now and then one of them recovers, he certainly loses the little Reason he had and turns Ideot.

"The immediate Cause of this deadly Sleepiness in the Slave, is evidently a Super-abundance of Phlegm or Serum extravated in the Brain, which obstructs the Irradiation of the Nerves; but what the procatartick Causes are, that exert to this Production, eclipsing the Light of the Senses, is not so easily assigned.

"We find sometimes in *Europe* that Enormities in the Non-Naturals, Surfeiting and Drunkenness do gradually, as Age and Custom advance, weaken the Tone of the Brain, to the Admission of serous and extrementitious Humours, including Sleepiness, etc. But here the case is different, they being young People that are generally afflicted, and who have been destitute of the Means of Surfeiting.

"I shall ascribe the Cause to *catching Cold, and their Immaturity; to Diet and Way of Living; and to the natural Weakness of the Brain*; some or all of these Causes co-operating to it.

"*First*. In *Immaturity*, or Childhood, it is a common and true Observation, that more of Phlegm and recrementitious Humour is bred, than in Manhood; because the Fibres, and consequently the Faculties resulting from their Constitution, have not attained their due Spring and Perfection; and it is only supposing the *Africans* continue longer Children than the *Europeans*.

"*Secondly*. Promoted here *by their Diet and Way of Living*. At Home it is mostly on Roots, Fruits, and Herbage, greedily devouring such as are wild and uncultured; which together with the intolerable Heats of the Sun, weakening the concoctive Faculty, together with their Inactivity, render a very recrementitious Nutriment: Their Indolence is such (when shipped on Board for Slaves) as to be entirely dispassionate at parting with Wives, Children, Friends and Country, and are scarcely touched with any other Sense or Appetite, than that of Hunger; and even in this, for want of Custom or Instinct, they cannot distinguish proper Food, no know when to leave off, voraciously eating though Victuals be never so dirtily cook'd; and whether the Flesh be raw or dressed, whether of the Guts or a Sirloin; a Practise also that may sometime, by over-stretching the Fibres of the Stomach, occasion Crudity and Indigestion. By their Sloth and Idleness the Blood becomes more depauperated; and those recrementitious Humours bred from it, that Exercise would throw off through the proper secretory Organs, are her disposed toward the weakest Part, which in the Generality of Negroe Slaves I take to be the Brain.

"*Thirdly. The natural Weakness of the Brain*, I am apt to think the principal Cause of the Distemper. Doubtless that Part gains Strength by Exercise, i.e. by the Employment of our rational Faculties, as well as the Muscles and external Fibres of the Body by Labour; and since the *Africans* are hereditarily ignorant, destitute of all Art and Science, or any mechanical Knowledge to exercise the Brain, it consequently grows weaker in its inward Structure and Recesses; and falls together with the Judgement and Passions.

"The Cure is attempted by whatever rouzes the Spirits; bleeding in the Jugular, quick Purges, Sternutories, Vesicatories, *Acu-Puncture*, *Seton*, Fontanels, and sudden Plunges into the Sea, the latter is most effectual when the Distemper is new and the Patient not yet drivelling at Mouth and nose."

Noteworthy in this account are the number of causes assigned to this mysterious disease, and the naïve way in which the author assigns the conditions to "some or all of these causes"!

The essential characteristics of the disease in an advanced stage are all noted; the "Indolence", "State of Insensibility", "Hunger", "Sense of feeling very little", but the preliminary stages were not noted, for Atkins says it gives no other notice than a want of appetite two or three days before!

With the aid of a little imagination one can see a terrible picture of these wretched, somnolent natives being subject to "pulling, drubbimg or whipping" in the endeavour to give them sufficient "Exercise" to "throw off the recrementitious Humours" and afterwards "voraciously eating though Victuals be never so dirtily cook'd"!

The "Sudden Plunges into the Sea" are not used for curing Sleeping Sickness at the present day!

It was, I believe, well known to the African slave traders that natives with enlarged glands were of no use to them, as they always died and could not be made to work. So they were careful not to take any in this condition, but nevertheless took without knowing it, many cases who had the earliest stages of the disease, and the American slave-owners used to find their slaves dying of this peculiar drowsiness.

Yet it was not infectious, that is, it was not communicated from one to another; the reason for this will be entered into fully further on.

In 1890, a French doctor discovered in the blood of a patient from Africa who was suffering from a peculiar fever, a microscopic organism which he recognised as a *Trypanosome*, but he did not establish any relation between this and the fever, and the credit of first discovering this belongs to an Englishman. In 1901 Dr Forde, in the Gambia colony of the West Coast, had a English patient with a peculiar fever of a chronic and irregular type. He found in the blood a curious organism whose nature he could not recognise, and called in the late Dr Dutton[1], who at once recognised the creature as a *Trypanosome*; the first one to be discovered in man, though one had been described as the cause of a disease in horses in India some ten years before.

At first, and for some time, Sleeping Sickness was only known to occur in West Africa, but when equatorial Africa was gradually opened up the disease found its way into Uganda with disastrous results.

[1] *British Medical Journal*, 1902, Jan 4th and Nov 29th

This is believed to be due to Stanley's expedition for the relief of Emin Pasha, which in 1888 travelled from the Congo to the Lake Albert Nyanza. In 1891 the Sudanese soldiers of Emin's force were brought into South Toro with their followers, and eventually were brought into Uganda itself to be under control.

In July 1901, Dr Albert Cook of the Church Missionary Society at Mengo (Kampala) noted eight cases of a mysterious disease, and six months later reported that on Buvumu Island over two hundred natives had died and thousands appeared to be infected. The mortality became appalling, and the Government were at their wit's end, for it seemed as if the whole population was doomed.

In July 1902, the first Royal Society Commission arrived in Uganda, composed of Drs Low[ii], Christie[iii] and Castellani[iv], and Colonel Sir David Bruce[v] arrived in February 1903. ON April 28th, it was announced that the disease was caused by a Trypanosome[2] and conveyed by a Tse-tse fly *Glossina palpalis*.

It was suggested at once that as the haunts of this fly were strictly limited, it would be easy to check the disease by removing the population[3]; but the natives with their characteristic fatalism, refused to leave their villages along the shores of the lake. In the meantime the disease raged unchecked, and by the end of 1903 the number of deaths had reached over 90,000; whole villages were being depopulated, and great tracts of highly cultivated country relapsed into scrub and forest.

In March 1905 Lieutenant Tulloch, R.A.M.C., who had been sent out by the Royal Society to help in the investigations, became infected with the disease in its virulent form, and died a few months later.

By November 1904 the epidemic had appeared on the shores of Lake Albert in North West Uganda, and a survey of Uganda by six specially appointed medical officers in 1905 showed that the banks of the lakes and watercourses throughout Uganda were infected with the Tse-tse fly.

Statistics furnished in a dispatch by the Governor showed that "during the last five years the total mortality from this scourge in this Protectorate has considerably exceeded 200,000[4].

Sir Hesketh Bell also reported "the natives have been almost completely wiped out everywhere along the lake shore, and in the islands the mortality has been even more appalling. Buvuma, for instance, which a few years ago was one of the most thickly populated and prosperous of all the islands, counted over 30,000 inhabitants. There are now barely 14,000. Some of the Sesse group have lost every soul; while in others a few moribund natives, crawling about in the last stages of the disease, are all that are left to represent a once teeming population".

In November 1906, it was again suggested that the only way to save the people was to remove them into fly-free areas, and segregate the infected natives into camps. The aid of the chiefs was sought and the matter fully explained to them, compensation was made to the heads of evicted families, they were given land away from the infected areas, and by degrees not only the mainland shores of the lake, but the islands also, were cleared of population, so that by 1909 all these were deserted and going back to the wild state[5].

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[2] *Reports of Sleeping Sickness Commission of the Royal Society*, 1903, nos. 1, IV
[3] *Bulletin of Sleeping Sickness Bureau*, vol. 4
[4] Dispatch no. 218 from Sir Hesketh Bell, November 1906
[5] *Bulletin of Sleeping Sickness Bureau*, vol. 4

Great difficulty was experienced in preventing the natives from returning to their homes, and some managed to obtain canoes and cross back to Buvuma and Damba Islands, but at length the evacuation was finally completed, and at the present day the whole of the fertile and valuable island territory is abandoned to the Tse-tse fly.

But the lake shore can only be kept in this condition by stringent regulations, and penalties, and a few natives are frequently discovered in the forbidden areas by the patrolling canoes.

By these means the inhabitants of Uganda were saved, and at the present day there are very few deaths a year; from 1905-1917 there were just over 30.000 deaths for the whole of the Uganda Protectorate.

At the end of 1909 considerable alarm was caused by the discovery in Nyasaland of Sleeping Sickness (or rather *Acute Trypanosomiasis*) in a native there, and since then a number of cases have been found, some of the Europeans, in Nyasaland, North-East Rhodesia, and Portuguese East Africa; in the case of some natives, they had certainly never left their homes. This was very interesting from a scientific point of view, because *Glossina palpalis*, the species of Tse-tse which carries Sleeping Sickness in Uganda and the West Coast, does not exist in those countries.

It was soon found that the carrier was another species, namely, the very one which had been so long known to travellers in Africa as the cause of Tse-tse fly disease or *Nagana* of horses, cattle and dogs.

This species is known as *Glossina morsitans*[6].

As this new form of human *Trypanosomiasis* appeared to be very much more acute than the form known as Sleeping Sickness, the discovery was disconcerting. It is, however, possible that our ideas of the severity of this form of *Trypanosomiasis* will need to be modified in the light of further knowledge, for during the campaign in East Africa a number of natives were found to have Trypanosomes in their blood while under treatment for other complaints and appeared to be little the worse for their presence; these natives had not been in the area of *Glossina palpalis*, so either the Trypanosome was *gambiense* carried by *morsitans*, or else it was *rhodesiense* in a non-lethal form. It is of interest to note that a case of true Rhodesian *Trypanosomiasis* has recently been recorded as the first to be cure by treatment; hitherto this form had always been regarded as fatal.

Figure 1 Close up of a Tse-tse fly taking blood meal https://www.cdc.gov/parasites/sleepingsickness/epi.html

[6] *Annals of Tropical Medicine and Parasitology,* Kinghorn and Yorke, vol. 6 pp. 1-23

Natural History of Sleeping Sickness

I propose now to deal at greater length first with Trypanosomes and next with the Tse-tse flies, then the inter-relation of the two will be considered and the relation of both to the wild animals of the countries in which they occur.

Trypanosomes are not 'microbes' in the accepted sense of the word as commonly used; that is, they are not micro-fungi, but belong to the *Protozoa*, the lowest members of the animal kingdom. The *Protozoa* can be divided into four main groups, in each of which are found species causing disease in man and other vertebrates. The *Sarcodina* is exemplified by the well-known *Amoea* and the species known as *Entomoeba*, which causes dysentery.

The next group, *Mastigophora*, is the one with which we are most concerned with at the moment, since to it belong the Trypanosomes and their allies. The name, which means 'Whip Bearers', refers to the possession of one or more lashes or *Flagella*, which by their rapid movements draw or push the animal through the fluid in which it lives.

The third group of *Protozoa* is the *Sporozoa*, which is only too familiar through the various species that cause malaria. To the last group *Infusoria* belong the myriad forms which are a delight to the amateur microscopist and may be seen so easily in stagnant water. One species, *Balantidium*, is a cause of dysentery.

Let us look more closely at the *Mastigophora*. The group is divisible into six sub-groups[7], but only that which includes the Trypanosomes need be mentioned here. This is named *Protomonadina*, and includes the following genera:

1 *Trypanosoma*: There is a single flagellum arising near the posterior nucleus and continued forwards as a marginal flagellum of an undulating membrane; usually continues into a free flagellum. The species are found mostly in blood and in the digestive tracts of invertebrates, but are also forms in the life cycle of species wholly parasitic in insects.

2 *Trypanoplasma*: There are two flagella, the posterior one united to the body by an undulating membrane along most of its length. According to their mode of life they fall into three groups.

Parasites in the blood of fresh-water fish and in the digestive tracts of leeches

Parasitic in the digestive tracts of marine fishes

Parasitic in invertebrates

3 *Crithidia*: A single flagellum arises about the middle of the body and runs forwards to form the marginal flagellum of a short or rudimentary undulating membrane, and is continued beyond as a free flagellum. It occurs as parasites in the gut of insects or as a stage in the life cycle of a Trypanosome.

4 *Leptomonas* or *Herpetomonas*: There is a single anterior flagellum but no undulating membrane. These are parasitic on invertebrates, chiefly insects, but also as a form of the next genus in invertebrate hosts or

[7] This classification is taken from Minchin

in artificial cultures. It is also of great interest that they are found in the milky juice of certain plants (*Euphorbiaceae*)

5 *Leishmania*: There is no flagellum, but there are two nuclei as in the other genera. These animals have become specialised to live in the tissue cells of vertebrates and have no need of organs of locomotion. It is of much interest that in artificial cultures *Leishmania* develops into forms like the two preceding genera or like Trypanosomes. The type species cause a fatal disease in Asia known as 'Kala-azar'.

Now let us consider a little more fully the Trypanosomes themselves. They are elongated bodies with a pointed posterior extremity where is a small nucleus, and at the anterior extremity the whip-like flagellum which by vigorous movements drags the Trypanosome along. The undulating membrane pursues a wavy course along the body and is responsible for the name, which is derived from *τρμπανου*, meaning a carpenter's tool: the allusion is probably to the spiral thread on an auger. In the middle of the body is a large nucleus. *T, gambiense* in the blood is several times as long as the diameter of a red corpuscle, but much narrower. The different species that may be found in the blood vary greatly in size and activity; *gambiense* is not very active and merely wriggles, but a species such as *vivax* in goats can hardly be kept in the field of a microscope. The first one to be seen was found in the blood of a frog, but the first reliable description was not given until 1841, when a specimen was described from the blood of a trout[8]. Not until 1879 was one found in a mammal, and this was *Trypanosoma lewisi*, seen in the blood of a rat. It is now known that in nearly every place rats are infected with this species, often to the extent of 50 per cent, but it is not harmful to them unless present in great numbers. The first disease-producing Trypanosome described was *T. evansi*, which kills numbers of horses, camels, elephants and dogs in Asia and North Africa by causing a disease known as 'Surra'; it was discovered in 1880. Not until 1895 was the next pathogenic species discovered, and this was an extremely important one, the knowledge of which was of utmost help in the subsequent investigations in to Sleeping Sickness.

This species was names *T. brucie*, after Sir David Bruce, who proved it to be the cause of 'Tse-tse fly disease' or '*Nagana*' in South Africa.[9]

In 1901 another species was described as *T. equiperdum*, the cause of 'Dourine' in stallions, brood mares and donkeys in America and North Africa. Also in 1901 was discovered *T. gambiense*, the first Trypanosome to be known foer a cause of disease in man.[10]

In 1902 *T. equinum* was shown to be the cause of '*Mal de Caderas*', a fatal disease of horses in Brazil and the Argentine Republic. Another human Trypanosome was discovered in 1909 by Chagas[11]; it causes in children and adults who have not become immune in childhood, chronic fever, enlargement of the thyroid gland, and puffiness of face, and may rarely result in death. This species was found to differ sufficiently in its life history to be put into a new genus, and has ben names *Schizotrypanum cruzi*. In 1910 it was shown that6 the cause of the acute Trypanosomiasis of Rhodesia was differentiated, not by its

[8] By Valentin. Recent work by Mlle M Gauthier, however, place this species in the genus *Trypanoplasma*
[9] Preliminary Report on the Tse-tse fly disease or Nagana in Zululand, 1895
[10] *British Medical Journal*, 1902, January 4th p. 42
[11] Chagas, *Brazil Medico*, 1909, April 22nd. See Bull. Inst. Pasteur, 1909, May 30th pp. 453-4. Also Bulletin of the Sleeping Sickness Bureau, vol. 1 p. 252; vol. 2 p. 117

morphology, but by is effects on animals, from its close allies *T. gambiense* and *T. brucei*, and is now known as *T. rhodesiense*[12].

In 1913 another species was found in man, causing in natives of Nigeria a chronic swelling of the glands in the neck very like the early stages of Sleeping Sickness, but very rarely going beyond that; it has been named *T. nigeriense*[13].

It must not be thought that every Trypanosome is a cause of disease. This is very far from being the case; the production of disease is a mere accident from the Trypanosome's point of view, an occurrence of fatal import for itself as well as the host should the latter die!

Although only a few of the pathogenic species have been mentioned, all of them together are a very small fraction of the total number of Trypanosomes and their allies that are known. They occur in the blood of fishes, reptiles, amphibian, birds and mammals; in the vital fluids of molluscs, in the alimentary canal of insects and other invertebrates.

By cultural methods it has been found that normal English cattle have Trypanosomes in their blood, and Bruce first found that even in species that cause disease in domestic animals lives harmlessly in the indigenous wild animals. Dr Duke has shown that the 'Situtunga' antelope (*Tragelaphus spekei*) carries in its blood the deadly *T. gambiense* without any harm to itself. It must be remembered that Trypanosomes are not like the bacteria such as that of anthrax, which form highly resistant spores, and by the death of their host and its disintegration are disseminated more widely in a condition in which life may be maintained indefinitely.

The position seems to be this. Many insects, for instance the house fly, contain in the alimentary canal Flagellates of a type closely allied to Trypanosomes, which live a natural parasitic life in the fly only.

When the ancestors of the present blood-suckers became addicted to this habit, probably a development of the habit of feeding on the fluid exuding from sores, it may well have happened that some of these internal parasites were inoculated into the blood of the animal. Those that survived may well have caused disease as a result of their vital activities; if so, by the process of natural selection, those which soonest became adapted to the new conditions and no longer brought about a condition unfavourable to the host, on whose life they would now depend, would have most chance of surviving.

This theory is supported by some very interesting work done by Fantham and Porter, who showed that *Herpetomonas jaculum*, a normal inhabitant of the gut of the 'Water scorpion' (*Nepa cinerea*), when introduced into the peritoneal cavity of mice or even taken by them in food eventually finds its way into the blood and cause symptoms like those of 'Kala-azar' in man, from which the mouse dies.[14] But if the mouse had not died so quickly there might have developed such immunity to the new substances circulating in its blood as a result of the vital activities of the *Herpetomonas* that friendly relations between the two would result and mammal and parasite would become mutually immune to each other, as appears to be the case with wild game and *T. brucei*.

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[12] Stephen and Fantham, *Proc. Royal Soc*, Series R 1910, no 561, pp. 28-32
[13] J W Scott Macfie, *Annals of Tropical Medicine and Parasitology*, 1913, August, vol. 7, no. 3A, pp. 339-56
[14] Fantham and Porter, *Proc. Camb. Phil. Soc*., 1915, vol. 18, pp. 39-50 & 137-48

When man with his non-immune domestic animals come into relation with this equilibrised system the equilibrium is disturbed; the Trypanosome finds itself introduced into the circulation of hosts to which it is a novelty and 'disease' results.

But even man himself shows a gradual acquirement of immunity. In West Africa Sleeping Sickness is not generally so virulent as in the Uganda epidemic, and the disease has been known in that are for a very much longer time. The species of Trypanosome known as *T. nigeriense* causes only a mild form of disease which is very rarely fatal, so it would appear to be the species which has been in longest contact with man. On the other hand *T. rhodesiense* is very much more virulent that nay other human Trypanosome known to us, and it appears to have arisen as a sudden 'mutation' from the animal-inhabiting species *T. brucei*. It is only ten years since this undesirable addition to the list of 'human' Trypanosomes made its presence known to us by causing a disease previously unknown in that part, or at least unrecognised as distinct from malaria.

Trypanosomes are transferred from one host to another by the agency of various invertebrates, which are as necessary for its existence as are the animals in whose blood they also live.

In the blood they only multiply by fission, asexually or vegetatively; but in the invertebrate host they go through a sexual form of reproduction. Development generally commences in the alimentary canal, whence the Trypanosomes find their way into the 'salivary glands', so that they are inoculated into the new vertebrate host when the blood-sucker injects the irritating fluid secreted by those glands in order to produce a free flow of blood. The effect of this fluid is familiar to all who have been 'bitten' by mosquitos. Such an alternation of sexual with asexual methods of reproduction is called a 'cycle'; the complicated life history of the malaria parasite is a more familiar example.

I think the fact that the sexual process takes place in the invertebrate host points to that being the original host for the Trypanosomes; the life in the blood is as it were an accident, and many close allies of Trypanosomes can do perfectly well without it.

Trypanosomes of fishes are carried by leeches[15]; of birds, by mosquitoes; those of mammals by various blood-sucking insects. Thus the rat Trypanosome is transmitted by the rat flea, the species causing 'Surra' in animal by the large flies often called 'Cleggs' (*Tabanida*), and those causing 'Nagana' in animals and Sleeping Sickness in man are carried by Tse-tse flies (*Glossina*).

The Trypanosome of 'Dourine' is particularly interesting, because it seems to have found that it can do without an intermediate insect host, and is transmitted directly from male to female animal, thus having severed relations with the ancestral home!

It may be pointed out that there is a close analogy shown by Spirochaetal diseases in man, some of which are carried by an intermediate host while others are not; the pathology of the two classes also shows much in common, which is not surprising, considering how closely allied are Trypanosomes and Spirochaetes.

Lastly, the very interesting species which causes inflammation of the thyroid gland with fever in Brazil, is carried by an insect (*Conorhinus*) of the order *Hemiptera* or 'bugs'. an order which numbers extremely few blood-suckers among its ranks, although all are adapted for obtaining food by suction. Darwin made

[15] See Miss M Robertson's paper in *Phil. Trans. Royal Soc.*, (?) Series B, vol. 202, pp. 29-50

special mention of this bug in his journal, and remarks that "one feast kept it fat during four whole months"[16].

The Tse-tse flies will now be dealt with more fully but in this chapter I will only give a general account reserving a full account of their natural history of *Glossina palpalis* for another chapter.

Some sixteen species exist, which are entirely Ethiopian in their distribution; for though one species (*tachinoides*) is found in the south-west corner of Arabia, that area is fantastically part of the Ethiopian region. Tse-tse do not occur in Africa north of the Sahara nor south of St Lucia Bay in Zululand. It is remarkably interesting, therefore, that fossil flies have been found in Colorado referable to this group[17], and it has been suggested by Osborn that they were responsible at least in part for the extinction of many of the large mammals which abounded in Cainozoic times and by their migrations came into contact with blood-sucking flies to which, and their associated flagellates, they had not yet become habituated.[18]

The first Tse-tse which I wish to mention particularly is *Glossina morsitans*, known to travellers in Africa as 'The Fly', or, collectively, as 'Fly'. According to Austen[19] the exact origin of the name is uncertain; it is believed to be a corruption of 'Nsi-nsi', said to be the name given to blood-sucking flies y natives of some parts of Africa. A passage in the Old Testament[20] possibly refers to *Glossina*, although it may also apply to other flies with the same habits: "And it shall come to pass in that day, that the Lord shall hiss for the fly that is in the uttermost part of the rivers of Egypt, and they shall come, and shall rest all of them in the desolate valleys, and in the holes of the rocks, and upon all thorns, and upon all bushes".

The early African travellers of the middle of the nineteenth century were, naturally, much impressed by the fly which was well known to the natives for inflicting a 'poisonous bite' upon their cattle.

Thus Gordon Cumming in 1850 wrote as follows[21]: "When under the mountains on the south bank of the Limpopo river I met with this famous fly whose bite is certain death to oxen and horses. This fly is similar to the fly in Scotland called 'Kleg', but a little smaller. They are very quick and active, and storm a horse like a swarm of bees, alighting on him in hundreds and drinking his blood. The animal thus bitten pines away and dies at periods varying from a week to three months."

And again: "The next day one of my steeds died of Tse-tse. He had been bitten under the mountain range lying to the south of this fountain. The head and body of the poor animal swelled up in a most distressing manner before he died. His eyes were so swollen that he could not see, and in darkness he neighed for his comrades who stood feeding beside him."

Again, Livingstone in 1857 wrote as follows[22]: "The peculiar buzz when one heard can never be forgotten by the traveller, for it is well known that the bite of this poisonous insect is certain death to ox, horse or dog. In this journey we lost forty-three oxen by its bite. We watched the animals carefully, and believe that not a score of flies were ever upon them.

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[16] *Voyage round the World*, edition 1890, p. 316
[17] Cockerell, *Proc. Of the United States National Museum*, vol. liv. 1918, p.308
[18] Letter from Professor Cockerell in *Nature*, 1919, June 5, vol. ciii, p. 265
[19] *Monograph on the Tse-tse Flies*, 1908, p. 32
[20] Isaiah, chapter vii verses 18,19
[21] *Five Years of a Hunter's Life in the Far Interior of South Africa*, vol. ii, p. 219, etc.
[22] *Missionary Travels and Researches in South Africa*, p. 79, etc.

"A most remarkable feature is the perfect harmlessness of the bite to man and *wild animals*." The italics are mine, to emphasize that the scientific mind of the greatest explorer had noticed this pregnant fact, although the full significance was not made plain until Bruce's work was published,

"We never experienced the slightest injury from them ourselves, although we lived two months in their habitat, which was in this case as sharply defined as in many others; for the south bank of the river was infested by them, and the north bank, where our cattle were placed, only fifty yards distant, contained not a single specimen. This was more remarkable as we often saw natives carrying over raw meat to the opposite bank, with many Tse-tses settled upon it."

This account makes clear one of the most remarkable points in the natural history of this species of *Glossina*, namely, the very sharply marked areas which it inhabits; this must depend upon the presence or absence of certain factors in its environment, but has not yet been thoroughly explained.

After noting the symptoms of 'fly disease' Livingstone continues: "These symptoms seem to indicate a poison in the blood, the *germ of which enters when the proboscis is inserted to draw blood. The poison germ, contained in a bulb at the root of the proboscis, seems capable, although very minute in quantity, of reproducing itself*," The words which I have put in italics were written bt Livingstone about fifty years before the discovery of Trypanosome by Bruce, *and some years before the first discovery of micro-organisms of disease by Louis Pasteur!*[23]

The genus *Glossina* was founded in 1839, when Wiedemann described a new species of fly from Sierra Leone, and in the same years another species was described from the Congo by Robineau-Desvoidy; thus is the one now known as *Glossina palpalis*. It is interesting that the first scientific description of a Tse-tse was not that of the one so long known to natives of South Africa; this was not named until 1850, when Gordon Cumming's travels made the fly well known in England.

Glossina palpalis frequents forested and humid country, and is not found so far south as its more widely distributed relative *morsitans*. The great river courses and lake shores in the tropics furnish the shade and humidity that it requires, hence the Congo basin, the upper Nile, and the shores of Lake Victoria abound with it.

Tse-tse flies are on the whole diurnal, though one or two species are known to bite at night; but the traveller is safe from n=both *morsitans* and *palpalis* after dark.

The life history of the Tse-tse flies is very remarkable and almost unique among insects. No eggs are laid, but a single egg is hatched within the abdomen of the mother fly and the larva, a white grub, is fed by the secretions of special glands, When the larva is full grown the mother fly seeks a suitable spot and the larva is extruded. It is a very active little creature, and crawls about seeking for a spot where it can burrow into the soil. It is helped to do this by two curious bosses at the posterior end of the body which are of hard chitin and give the larva a firm purchase when it begins to burrow. As soon as it has buried itself the skin hardens and it becomes a dark brown oval pupa with the two bosses at the posterior end which were seen in the larva, and differ in shape according to the species of fly.

[23] In 1857 Pasteur first showed that fermentation was due to micro-organisms; in 1865 he showed that silkworm disease was due to micro-organisms. In 1876 the first 'bacterium' (of anthrax) was isolated by Koch

After a period of varied duration the perfect fly emerges by cracking the pupa-case or 'puparium' at one end and by means of an extrusible bladder on the front of the head works its way up through the soil covering it and creeps up a stem in a great hurry to let the wings hang down and expand before they have hardened. I have seen many *palpalis* thus emerge from the ground, and they always give the impression that it is a matter of the utmost importance that they should lose no time!

Let us now glance at the development of our knowledge of Tse-tse flies and disease. It was quite clear to the earliest travellers in South Africa that 'The Fly', as *Glossina morsitans* was called, was a cause of the fatal disease of cattle and horses to which allusions have been made.

Sir David Bruce in 1894 was able to report that the actual cause of the disease 'Nagana' was a Trypanosome which was named *brucei* after its discoverer, and was taken up by a Tse-tse when it fed on the blood of an animal suffering from Nagana. It was believed that the fly inoculated the Trypanosome into a fresh animal when it fed again in the same manner that a vaccinator introduces the lymph on the point of his lancet, that is to say, by direct inoculation. Thus in the case of Nagana we have started with the carrier of the disease and found the germ which it carried.

When the problem of Sleeping Sickness became so acute in Uganda, the Royal Society sent out a Commission to investigate the disease, and the members arrived in Uganda in July 1902. But the first step towards the elucidation had already been made in West Africa. In 1901 an Englishman in charge of a steamer on the Gambia river was admitted to hospital at Bathurst for 'fever', and Dr Forde found in his blood peculiar organisms whose nature was unknown to him. The patient was sent to Liverpool, and Dr Dutton recognised the new organism to be a Trypanosome: the patient eventually died at the commencement of 1903.

In 1902 Drs Dutton and Todd found Trypanosomes in the blood of several West African negroes suffering from the early stages of what we now know as Sleeping Sickness, but in those days the condition had not been recognised as connected with that well known disease. This early stage of fever now came to be known as 'Trypanosome fever' or '*Trypanosomiasis*', and in March 1903 Dr Baker found the Trypanosome in a case in Uganda[24], though he did not recognise the full importance of this fact. The next development was the finding by Dr Castellani of Trypanosomes in the cerebro-spinal fluid of a case of Sleeping Sickness in April 1903[25], and afterwards by Bruce and Nabarro in every case of Sleeping Sickness examined. Thus was made clear the fact that 'Trypanosome fever' is the early stage of the fatal disease, and the next step was to find out how the disease was transmitted.

At this step a comparison of what was known about Nagana with what had been found out about Trypanosomiasis aids in the understanding of further developments. In the former case investigation commenced with strong presumptive evidence that a Tse-tse fly was the agent through which the disease was acquired; in the latter case the germ was discovered and it became necessary to ascertain how it was transmitted. Since Nagana had been proved to be due to a Trypanosome carried by the blood-sucking fly *Glossina morsitans*, and Sleeping Sickness had now been sown to be due to another species of Trypanosome, evidence pointed to a blood-sucking fly as the carrier of this new species *Trypanosoma gambiense*, and requests were made to officials in the Sleeping Sickness areas to send specimens of all

[24] *British Medical Journal*, 1903, May 30th p. 1254
[25] *Proc. Roy. Soc.*, 1903, vol. lxxi, pp. 501-8

biting flies in the neighbourhood to the laboratory in Entebbe. It very soon became clear that one fly was found throughout the areas being ravaged by the disease, that is the shaded margins of great lakes and rivers; this fly was *Glossina palpalis*. Accordingly experiments were made to test whether the fly can be the carrier, and specimens captured on the shores of the lake were fed upon monkeys whose blood was examined daily, the procedure being the same as in Bruce's classical work on Nagana. Conclusive proof was obtained when the Trypanosome was found in the blood of monkeys, and the discovery was announced by the Commission in 1903[26]. Further proof was obtained by feeding bred flies on monkeys which had been already infected by wild flies. And then making them feed upon another monkey; the second monkey in a proportion of cases also showed the Trypanosome in its blood. Control experiments showed that a freshly hatched fly contains no Trypanosomes, so that those in the above experiments must have acquired them from the first monkeys fed upon.

The two diseases are thus entirely parallel…

It has been said that the transmission of the Trypanosome of Nagana was believed to be entirely mechanical, and for some years was also thought to be the case with *T gambiense*. With further knowledge, however, it became clear that there was a period after the fly had fed during which the Trypanosome could not be transmitted to a fresh animal. It had previously been supposed that this was because Trypanosome was no longer alive in the fly, but Kleine, working in German East Africa, showed in 1908 that the non-infectivity of the fly after a few days did not mean the death of the Trypanosome, but that it was going through a cycle of development in the alimentary canal of the fly, and was not in an infective condition. For when the development was complete Kleine found that the fly could convey the disease fifty days after it had acquired the Trypanosome. These most important results were fully confirmed in Uganda in 1909[27] and it was found that the time required for the cycle of development in the fly varied from eighteen to forty-five days, after which time a fly will remain infective and be able to introduce the Trypanosome into every animal it bites for as long as seventy-five days!

The complete life cycle of the Trypanosome in the fly was worked out fully in Uganda by Miss Robertson[vi] in 1913[28], The Trypanosome multiplies in the gut of the fly, but the forms of multiplication are not those which will live in the blood stream of the vertebrate host, and, a somewhat different form is developed which finds its way into the 'salivary' glands of the fly and is injected with the irritant secretion of the gland which presumably is intended to produce a free flow of blood in the site of the puncture. We have now arrived at the most important fact that when once it has acquired the Trypanosome the Tse-tse fly can infect for the rest of its life.

We must now consider the relations between the Tse-tse, Trypanosome, and the 'alternative hosts' of the latter from which the fly acquires it.

It was known from Bruce's Research on Nagana[29] that *Trypanosoma brucei* is a natural and harmless inhabitant of the blood of various species of big game in the 'fly areas', and as soon as Sleeping Sickness was proved to be due to another Trypanosome, efforts were made to discover its natural host or reservoir. The Commission in Uganda made a series of experiments with the blood of such animals and birds that,

[26] Reports of the Sleeping Sickness Commission, No IV, pp. 56-65
[27] Reports of the Sleeping Sickness Commission, vol. x, p 46, etc., vol. xi, p. 12 etc.
[28] Reports of the Sleeping Sickness Commission, no. XIII, p. 119 etc
[29] Appendix to *Further Report on Tse-tse Fly disease or Nagana in Zululand*, 1903, p 8 London, Harrison & Sons

inhabiting the lake shore, might be justly suspected of harbouring the Trypanosome, but with negative results. One important species of antelope, however, was not at this time (1908-10) examined. The next step was to *infect* captive antelope and native cattle by feeding upon them flies caught in the Sleeping Sickness area, and it was found that this could readily be done; waterbuck, bushbuck and reedbuck in captivity could all be made reservoirs of the Trypanosome without any harm to themselves, and could continue to infect bred flies fed upon them for more than twelve months after they had been artificially infected[30]. Finally, in March 1912, Dr Duke announced that the antelope known to the natives as '*Enjobe*' was a natural reservoir of *Trypanosoma gambiense,* and the chain was thus completed[31].

It had seemed for some time probable that there must be a natural host, although it had not been found. Four years and a half after the natives had been removed from the islands the fly there was still infective, and it was impossible to suppose that the same flies were still alive that had been the cause of the epidemic. With one exception the animals and birds and reptiles within reach of the fly had been sufficiently examined to make it almost certain that they were not incriminated, and the one large antelope living on the islands was yet to e excluded. Accordingly, in the latter part of 1911 Dr Duke came over to my camp on Damba Island to shoot and investigate the very abundant *Enjobe* there. This antelope, the Situtunga (*Tragelaphus spekei*), lives in the most intimate association with the fly among the shaded forests at the water side.

A number were shot and their blood was injected into monkeys, one of which, injected on November 5th and 6th, showed Trypanosomes in its blood on the 18th. It was taken to the laboratory on the mainland and the nature of the Trypanosome investigated in every possible way, and Dr Duke considered that there was no doubt that he had found at last the source from which the flies on the island acquired the cause of Sleeping Sickness.

This discovery made the question of the return of the natives to the lake shore a far more difficult one than was anticipated when they were removed during the height of the epidemic. It was then thought that as the fly was merely a mechanical carrier of Trypanosome, an interval during which all known sources of infection (the natives) were kept away from the fly would allow the disease to die out, and when the life of the infected flies had come to an end, the natives who were free from Trypanosomes would return without danger. But now that it is known that there is a 'vicious circle', the fly acquiring the Trypanosome from the antelope and in turn inoculating it into fresh animals, the islands and mainland shore of the lake are still dangerous. It is true that some consider that it is possible that the Trypanosome has been so long away from man's blood that it may no longer be pathogenic to him, but against this is the fact that as a result of living on the islands with me in 1911-12 three of my native employees were found to be infected when I examined them before returning to the islands in 1914, and one at least is reported to have died,

So far as we know at present, the fly would be harmless without the antelope and the antelope without the fly, and to eliminate the disease from the most fertile and beautiful part of Uganda these two must be kept apart; that is to say, one of them must be exterminated.

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[30] Reports of the Sleeping Sickness Commission, no XI p 71 etc.
[31] *Ibid* no XII p 117 et seq. See also article by Duke in *British Medical Journal*, 1914 February 7th

In 1914 I obtained most interesting conformation of Dr Duke's results. The Island of Nsadzi, lying opposite to Entebbe and south of it, was well populated in the old days, and there was very little refuge for *enjobe* there; I was told by my canoe-men that the antelope was not to be found on the island in those days. In 1911 the flies on Nsadzi were tested and were found to be free from infection, for as many a 5,763 failed to cause infection in a monkey. In 1914, however, I frequently saw footprints of *enjobe* on the island, which had presumably come across the narrow channel between Nsadzi and the large isle of Kome to the west where *enjobe* abound. The flies were again tested and found to be infected, for after 2,071 had fed upon a monkey it showed the Trypanosome in its blood[32]. Less than half the number of flies that did not produce an infection in 1911 produced an infection in 1914, and this is associated with the arrival of the (?) in the continued absence of the population,

It seems hardly possible to entertain the idea of destroying the *enjobe*, since when hard pressed it takes refuge in dense papyrus swamps, and even if it could once be eliminated from the islands would soon find its way back by swimming from the mainland, and would resume its former relations with the native. It is, then, possible to eliminate the fly?

On this question I shall have something to say in the chapter devoted to the natural history of *Glossina palpalis*

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[i] For a modern analysis, see https://www.who.int/trypanosomiasis_african/country/history/en/index6.html (omits Muriel Robertson) and https://www.who.int/trypanosomiasis_african/diagnosis/en/ and https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1832067/

[ii] See https://en.wikipedia.org/wiki/George_Carmichael_Low

[iii] See https://en.wikipedia.org/wiki/Cuthbert_Christy

[iv] See https://en.wikipedia.org/wiki/Aldo_Castellani

[v] See https://en.wikipedia.org/wiki/David_Bruce_(microbiologist)

[vi] Muriel Robertson, see https://www.cambridge.org/core/journals/parasitology/article/remarkable-dr-robertson/6EDB4A8A2A860D9C7B86DACCFFE52D6E/core-reader

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[32] Reports of the Sleeping Sickness Commission, no xvii, 1919 pp. 71-74