defaecation, and dyspareunia. From being a bright girl she became peevish and despondent, she slept badly, her appetite failed, and she lost nearly 2 st. in weight. When she was sent to me I found the left side of the cervix split up to the vaginal roof, and a tense tender scar stretching from there across the left vaginal fornix. About this scar were fixed the uterus and the left appendages, and the whole mass was intensely tender. I operated on her, repairing the torn cervix, dividing the vaginal scar freely, and stitching the transverse incision across in the vertical direction, then opening the abdomen, freeing adhesions, removing the left appendages, and fixing the uterus to the anterior abdominal wall. She recovered well, and soon went to a convalescent home. Some weeks later her doctor again sent her to see me. She was still sleeping badly, and very easily got tired and worried. I found the pelvic organs in a very satisfactory condition, and was able to assure her that she ought soon to get rid of her neuroses. A course of nux vomica and bromide, continued for some weeks, helped towards this goal. Six months after her operation I again saw her, and hardly recognized her. She was fatter, brighter, and younger looking, was able to do her work with pleasure, and the pelvic symptoms had all gone.

Compare this with the case of a woman aged 44, on whom I did an operation for cystocele some months ago. Her one child was born seventeen years ago. The posterior vaginal wall and the perineum were torn, and a tense scar formed, which remained so tender that she was never able again to have intercourse with her husband. The falling birth-rate is not wholly due to the wilful practice of Malthusian methods. Efficient gynaecological treatment would enable not a few erstwhile sterile women to add their quota to the population.

These are a few of the many cases I have met with since I came to realize the frequency and importance of dyspareunia. I have to thank the physician whose clinical clerk I was some twenty years ago for impressing it upon me, with many an apt illustration, that in a considerable proportion of those bane of a practitioner's life—the "neurotic women"—careful search will discover a definite underlying pathological lesion. Dyspareunia is one among the multitude of these underlying conditions.

It does not require much imagination to understand how the prolonged mental and physical distress produced by this symptom, aggravated in many cases by the knowledge that the husband's affections are being thereby alienated, is likely to produce a morbid mentality. Says George Eliot in *Middlemarch*: "It is in the acts called trivialities that the seeds of joy are for ever wasted, until men and women look round with haggard faces . . . and say that earth bears no harvest of sweetness."

When the practitioner has realized that dyspareunia is no trivial condition, but one which may cast a deep cloud over a woman's whole married life, he will be much more likely to discover the cases in which it exists, and by adequate treatment to lift the cloud and earn the patient's undying gratitude.

THE IDENTITY OF *T. RHODESIENSE* WITH THE TRYPANOSOME OF THE SAME APPEARANCE FOUND IN GAME.

BY

WARRINGTON YORKE AND B. BLACKLOCK.

(From the Runcorn Research Laboratory of the Liverpool School of Tropical Medicine.)

At the beginning of 1912 Kinghorn and Yorke¹ published a paper announcing that a considerable proportion (16 per cent.) of the game in the Luangwa Valley of North-Eastern Rhodesia harboured the trypanosome which causes sleeping sickness in man. This discovery has since been confirmed by the Royal Society Commission,² who found that a similar percentage of the game in the proclaimed area of Nyasaland was infected with the human trypanosome. The German workers, notably Kleine and Eckard,³ Taute,⁴ and Fischer,⁵ although they agree that one of the trypanosomes found in game is very closely related to *T. rhodesiense*, both as regards its morphology and pathogenicity, hold that the parasites are not identical.

Owing to its practical importance the question has given rise to much discussion; those who are opposed to interference with the African fauna welcome the theory that the parasite found in the game is not the same as the human trypanosome. Much of the discussion is based on hypothesis and not on scientific observation, and is hence

of little value. The case of those who deny the identity of the two parasites has been stated recently by Kleine⁶ in a paper entitled "The Alleged Identity of *T. brucei* and *T. rhodesiense*." It is this paper that has called forth the present article, in which we propose first to review the evidence on which Kinghorn and Yorke concluded that the trypanosomes are identical; secondly, to examine the arguments advanced by Kleine and others who maintain that the parasites are different; and finally to consider certain additional facts which in our opinion support the view originally expressed by Kinghorn and Yorke.

The evidence of identity adduced by these workers was derived from studies of the morphology, pathogenicity, and the development of the two parasites in the tsetse fly. *T. rhodesiense*, the cause of sleeping sickness in South Central Africa belongs to the polymorphic group of trypanosomes—that is, it possesses long free flagellar forms, short aflagellar forms, and intermediate varieties. In this respect it resembles *T. gambiense*. Its claim to be considered a good species depends on the fact that in a certain proportion of the short forms the nucleus is situated at the posterior extremity,⁷ and this character has up to the present sufficed to distinguish it from *T. gambiense*. Recently, however, it has been shown that certain other species, such as *T. pecaui*,⁸ *T. brucei* of Uganda (*T. ugandae*),⁹ and *T. equi*,¹⁰ hitherto considered to be parasites peculiar to animals, also exhibit this phenomenon. All these belong to the polymorphic group of trypanosomes, and are morphologically indistinguishable from each other and from *T. rhodesiense*. Almost all authorities now agree that *T. pecaui* and *T. ugandae* (*T. brucei* of Uganda) are synonymous. This trypanosome appears to be widely distributed throughout tropical Africa in game and domestic stock, and is most probably the parasite found in game by Kinghorn and Yorke in Rhodesia. We wish to emphasize here that the names applied to the trypanosome found in man, and to that found in game, are merely of secondary importance. The fact that the parasite infecting human beings happens to have been called *T. rhodesiense*, whilst a similar flagellate existing in animals has been designated *T. pecaui*, *T. brucei*, or *T. ugandae*, does not of itself exclude the possibility of their being identical. The real point at issue is whether or not the trypanosome infecting man is the same as that found in game and domestic stock.

As already mentioned, Kinghorn and Yorke, as the result of an exhaustive examination of the human and game trypanosomes, found that they were morphologically identical. The virulence of the two strains was examined in a large number of laboratory animals, and the trypanosomes proved to be alike. Finally the life-cycle of the parasites in the tsetse was examined. In laboratory-bred *G. morsitans*, whether fed on sleeping sickness patients or on animals infected with the game strain, the development of the trypanosomes was similar. The parasites multiplied in the intestine, and after a latent period, the length of which varied inversely with the temperature to which the flies were exposed, the salivary glands became invaded and the flies infective.¹¹ These observations have been confirmed by the Royal Society Commission and others, and are now generally accepted.

Kleine, Taute, Fischer, and others, while agreeing that the human and game trypanosomes are indistinguishable when judged by the methods of examination mentioned above, for various reasons deny that they are identical. In the first place they state that Kinghorn and Yorke failed to observe the presence of the widely spread *T. brucei* in a heavily infected *morsitans* area. Such a statement as this is, of course, of no value as criticism. It is in itself an assumption that *T. brucei* is a parasite capable of being distinguished from *T. rhodesiense*.

Again, on epidemiological grounds, Kleine⁶ asserts that the parasites are undoubtedly different, because in certain *morsitans* areas the animal trypanosome (*T. ugandae* vel *T. brucei* of Uganda vel *T. pecaui*) is found in game and stock and in tsetse fly without cases of human trypanosomiasis occurring. Moreover, numerous Europeans have hunted in such areas and have not contracted sleeping

* We must draw attention to the observation of Bouet and Roubaud¹² on the development of *T. pecaui* in *Glossina*. These authors found that *T. pecaui* instead of invading the salivary glands completes its life-cycle in the proboscis. If this observation is confirmed, then *T. pecaui* cannot be held to be identical with *T. rhodesiense* nor with *T. ugandae*.

sickness. In many districts of German East Africa cattle and dogs die at once from infection with this parasite whilst human trypanosomiasis is unknown. We cannot pass over such arguments as these without pointing out that it is by no means easy to make certain that any district is absolutely free from human trypanosomiasis. As a matter of fact sporadic cases of sleeping sickness have been found in widely separated areas throughout South Central Africa. Kleine considers that this state of affairs—the absence of sleeping sickness in such districts—cannot be due to immunity acquired as the result of a slight infection in youth. He examined some 1,500 children in German East Africa without finding any instances of infection. He goes on to argue that the assumption that the majority of human beings is resistant or only occasionally susceptible is not permissible. There is no analogy for this in other trypanosomal infections. If animals become infected with what is for them usually a non-infective trypanosome the infection invariably quickly dies out. Men die of *T. rhodesiense*.

As regards the former hypothesis, we agree with Kleine that there is no evidence to suggest that the native population enjoys an acquired immunity resulting from a mild attack of the disease during childhood. Analysis of the cases (about 300) of sleeping sickness recorded in Nyasaland and Rhodesia shows that children do not form an unduly large proportion, and, moreover, that they exhibit no tendency to recover. Again, such a hypothesis would not explain the fact that European hunters and others have travelled widely in *G. morsitans* regions with impunity. We are of opinion, however, that the second hypothesis—namely, that the majority of human beings is resistant and only occasionally susceptible—cannot be dismissed without very careful consideration. The statement that there is no analogy in the case of other trypanosome infections is not correct. It is a well-recognized fact that *T. vivax* does not, as a rule, infect small laboratory animals, such as dogs, rabbits, guinea-pigs, rats, and mice; these animals are immune. The immunity they enjoy is not acquired but natural; yet we have recently succeeded in showing that this immunity in the case of rabbits is by no means complete, and can in some cases readily be broken down.¹² A certain percentage of rabbits inoculated became infected with *T. vivax*, and of these about half died of an acute infection in from five to twenty days. Another striking instance of such natural immunity is that exhibited by certain species of animal to the small flagellar trypanosomes so commonly found in game and domestic stock in tropical Africa. In fact, so resistant were the small laboratory animals to some strains of this parasite that the name "*T. nanum*" was given to such strains in contradistinction to the term "*T. congolense*," which was applied to those strains found capable of infecting some or all of these animals. Recent work¹³ has, however, shown that such a distinction is not justifiable. The small flagellar trypanosome of domestic stock and game in Africa is at the time of its isolation from naturally infected animals of uncertain pathogenicity. Some strains are at once virulent, whilst others may only become so after many passages through laboratory animals. Kleine's statement that accumulated experimental evidence does not justify the view that the pathogenicity of a trypanosome strain can be permanently changed appears to be hardly in accordance with facts. In addition to the instance of *T. congolense* already cited, the permanent increase of virulence observed in certain strains of *T. gambiense* which have been maintained in European laboratories has been emphasized by numerous workers.

It has been held that the only way in which definite and conclusive proof of the identity of the two strains of trypanosome can be obtained is by inoculation of human beings with the game strain. Kleine, referring to experiments of this kind recorded by Todd and Taute, sees in their results evidence that the game and human strains are different. Kleine is, however, under a misapprehension when he writes that Todd inoculated himself with *T. brucei*. The observation recorded by Todd¹⁴ is to the effect that a European (Dr. Ascenso) and two natives were injected with blood from a cow suffering from trypanosomiasis, with negative result. It is important to note that the species of trypanosome with which the cow was infected is not mentioned: as *T. vivax* and *T. congolense*.

are more commonly found in cattle than the polymorphic trypanosome, the probability is that it was with one of these species that the experiment was performed. In Taute's experiment,⁴ however, the information given is quite definite. Taute fed upon himself, with negative result, laboratory-bred *Glossina morsitans*, which had been rendered infective with the *rhodesiense*-like game trypanosome. He also subsequently inoculated himself with 2 c.cm. of the blood of a dog naturally infected with the same trypanosome. Here again the result was negative. These experiments were carried out at Lubimbinu in Portuguese Nyasaland. Lubimbinu itself is fly free, but is surrounded by a large wooded fly belt in which *G. morsitans* is present in enormous numbers. The population is stated to be free from trypanosomiasis, but quite the reverse is the case with domestic stock. Cattle and sheep were not found, goats were rare, and dogs were very seldom seen and died rapidly. Very great importance has been attached to this experiment by those who hold the view that the game and human trypanosome are different. That such work is of the utmost value there is no doubt, but we cannot admit that the inference which has been drawn as a result of it is justifiable. All Taute proved is that a single healthy human individual was able to resist infection. The experiment simply confirms us in our belief in the relative immunity of man to infection with *T. rhodesiense*. It is exactly this natural resistance of man, which Kleine dismisses as a non-permissible assumption, that appears to us to afford the only satisfactory explanation of the comparative scarcity of cases of human infection with *T. rhodesiense*. The hypothesis of a relative immunity of the human race to *T. rhodesiense* is not improbable. Man exhibits a marked degree of resistance to infection with the genus *Trypanosoma* as a whole. No case of human infection with any of the common trypanosomes of domestic stock, such as *T. vivax* or *T. congolense*, has been recorded.

The fact that in Northern Rhodesia—and this applies also to other countries in South Central Africa—the disease is scattered sporadically in widely separated areas suggests that there must be some endemic reservoir of the disease apart from human beings. It is hardly conceivable that widely separated cases of sleeping sickness should have been infected one from another in the absence of considerable foci of human infection. If men were readily susceptible to infection only a single case would be required in a heavily infested *morsitans* district to cause an epidemic. Up to the present, however, an epidemic of sleeping sickness due to *T. rhodesiense* is unknown. Instead of considerable foci, which one would expect if infected man were the reservoir from which *G. morsitans* derived its infection, we have isolated cases scattered apparently haphazard over enormous tracts of country. It appears to us that the most probable explanation of these facts is that the virus exists endemically in game and fly all over the country—allowance, of course, being made for altitude and other circumstances that may influence the infectibility of *G. morsitans*—and that here and there scattered throughout the country an individual, the natural immunity of whom is for some unknown reason insufficient, becomes infected.

Moreover, it is quite clear that a very large proportion of game must be infected with *T. rhodesiense*. In the Rovuma district of German East Africa, Week¹⁵ inoculated antelope with blood from a human being infected with *T. rhodesiense*. The antelope became infected, and its blood was infective for monkeys and dogs five weeks later. The buck itself exhibited no signs of disease. This experiment shows that antelope can harbour the human trypanosome in their blood for some time—how long is not yet known—without exhibiting symptoms. As at the end of five weeks the blood was infective for monkeys and dogs, there is no reason to doubt that it would also infect *G. morsitans*. Nor is there any ground for doubting that if antelope can be infected directly from the human being they could also be infected from the tsetse fly. This being the case it follows that antelope must be infected with *T. rhodesiense* one from the other by *G. morsitans*, precisely in the same way as they are with the common trypanosomes of domestic stock, *T. vivax* and *T. congolense*.

In view of the fact that antelope can harbour

T. rhodesiense in their blood, it is incredible that they do not constitute in nature a reservoir of the virus. The fact that a large proportion of the antelope do actually harbour a trypanosome indistinguishable from *T. rhodesiense* is very suggestive. To assume that the game is infected with two different trypanosomes, alike in every respect with the single exception that one variety is fatal to man whilst the other is innocuous, would be, in our opinion, excessive theorizing.

Summary.

In favour, then, of game being the reservoir of human trypanosomiasis in South Central Africa we have the following facts:

1. Human beings and game are known to be infected with trypanosomes identical as regards morphology, pathogenicity in laboratory animals, and their development in *G. morsitans*.

2. The human trypanosome can be successfully inoculated into game.

3. The peculiar sporadic occurrence of the disease in human beings suggests that they were infected from a widely spread reservoir of the infection (the game) rather than from one another.

In conclusion, we submit that the hypothesis that man enjoys marked natural immunity and is in consequence to a great extent resistant to infection with this parasite affords a satisfactory explanation of the distribution of the disease, of its comparative rarity, and of the fact that Taute's attempt to infect himself failed.

REFERENCES.

- ¹ *Annals Trop. Med. and Parasit.*, 1912, vol. vi, p. 1. ² *Proc. Roy. Soc.*, 1913, vol. B., lxxxvi, p. 263. ³ *Zeitschr. f. Hygiene und Infektionskr.*, 1913, vol. lxxv, p. 118. ⁴ *Arbeit. a. d. Kais. Gesundheitsamt.*, 1913, vol. xlv, p. 102. ⁵ *Archiv f. Schiffs- und Trop. Hygiene*, 1913, vol. xvii, p. 621. ⁶ *Zeitschr. f. Hygiene und Infektionskr.*, 1914, vol. lxxvii, p. 184. ⁷ *Proc. Roy. Soc.*, 1910, vol. B., lxxxiii, p. 28. ⁸ *Journ. Trop. Med. and Hygiene*, 1912, vol. xv, p. 195. ⁹ *Proc. Roy. Soc.*, 1913, vol. B., lxxxvi, p. 187. ¹⁰ *Proc. Roy. Soc.*, 1913, vol. B., lxxxvii, p. 89. ¹¹ *Bull. Soc. Path. Exot.*, 1911, vol. iv, p. 539. ¹² *Annals Trop. Med. and Parasit.*, 1913, vol. vii, p. 563. ¹³ *Annals Trop. Med. and Parasit.*, 1913, vol. vii, p. 603. ¹⁴ *Sleeping Sickness Bulletin*, 1911, vol. iii, p. 174. ¹⁵ *Archiv f. Schiffs- und Tropen-Hygiene*, 1914, vol. xviii, p. 113.

ELECTRARGOL IN SMALL-POX AND PLAGUE.

BY

ROBERT DENMAN,

DIRECTOR, MEDICAL AND HEALTH DEPARTMENT, MAURITIUS.

I AM now able to carry out the promise in my letter of August 22nd last, published in the JOURNAL for October 11th, p. 906, to send more definite figures concerning the results obtained by the treatment of small-pox with electrargol.

We had altogether 1,914 cases and 196 deaths, giving a mortality of 10.24 per cent. for the whole epidemic. Of these, 1,309 were treated in one central small-pox hospital and 124 of them died, giving a hospital mortality-rate of 9.46 per cent.

The majority of cases were of a mild type and only required segregation and recovered practically without treatment, but we had 259 cases of the confluent and haemorrhagic types, and of them we lost 99. I have divided these types from one another, and in the following table will be seen the difference between the death-rate among those treated with electrargol and those who were not. At the beginning and before we were certain of the value of the treatment patients were allowed to choose whether they would accept the treatment or not, and I think I am right in saying that it was owing to this unfortunate fact that our death-rate was so high. Later on, when we had established the value of the treatment, it was made compulsory, and every patient who was suffering

TABLE I.

Nature of Case.	Treatment.	Number.	Cured.	Died.	Mortality.
Confluent	Simple	94	29	65	69.1%
	With electrargol	136	115	21	11.4%
Haemorrhagic	Simple	9	1	8	88.8%
	With electrargol	20	15	5	25.0%

from the severer forms of the disease had to submit to injection. As I said in my letter of August 22nd, the injections were all intravenous, and we afterwards increased the dose—in one case going up to 50 c.cm. every twenty-four hours for three days—the patient, an apparently hopeless case, delirious, with a confluent rash and passing bloody stools and urine, recovered.

The figures show that patients treated with electrargol had a much better chance of recovery than those who were not. Cases were not picked out for treatment, but they were taken as they came, and I can confidently recommend this mode of treatment to any one who may find himself in charge of a small-pox lazaret during a severe epidemic with many confluent and haemorrhagic cases to look after.

The drug is absolutely stable and reliable and may be kept in stock for an unlimited time. The mode of administration is simple and can be carried out by a well-trained hospital attendant. It consists of the injection into a vein of the required amount of the drug with the usual precautions, and if its use in 259 cases of small-pox and 48 of plague without accident means anything, it is perfectly safe.

With regard to its use in plague, I have had only a very limited number of cases to treat this year, the whole plague season giving 313 cases and 261 deaths, with a mortality of 83.4 per cent. nearly. Only 48 of these cases came under treatment at the lazaret, and we managed to save 19 of them, reducing the mortality to 60.4 per cent.

The following table shows very well that early treatment is the main thing, as out of 20 cases treated within forty-eight hours of the commencement of the disease only 5 died, whereas of the 21 treated for the first time on the third and fourth days 18 died.

TABLE II.

Day of Disease.	No. of Cases.	Cured.	Died.	Death-rate.
1st	10	8	2	25.00%
2nd	10	7	3	42.85%
3rd	10	1	9	90.00%
4th	11	2	9	81.00%
5th	5	0	5	—
6th	2	1	1	—

So far, I am sorry to say that we have found treatment of any kind hopeless in cases of abubonic or pneumonic plague, all these patients having died, no matter what treatment they received.

These types were very common during this epidemic, and we had nine abubonic and one pneumonic case in our lazaret. If we deduct these cases from the total we have 38 cases and 19 deaths left, reducing our mortality to 50 per cent. for the simple bubonic type. This result suggests the necessity for continuing the experiment when an opportunity occurs, and if we are unfortunate enough to have an epidemic in the next plague season I mean to continue it here. If any one in charge of a plague lazaret wishes to do the same let me advise him to begin his treatment as soon after his diagnosis is made as possible, to give his injections intravenously every twenty-four hours, not to be afraid of large doses, and always to carry on his treatment to the end of the fifth day, no matter what the temperature and the pulse are doing. In plague we have gone as far as 60 c.cm. without any ill results in cases of multiple bubo.

Further information will be found in my reports on plague and small-pox, and in my Annual Medical Report for 1913.

THE foundation stone of the new hospital and nurses' home for the Chelsea Hospital for Women will be laid by Earl and Countess Cadogan on Tuesday next at 3.30 p.m.

GENERAL LYAUTEY has forbidden the sale of absinthe in Morocco, where the use of that beverage has caused an immense amount of evil. On the other hand, one of the last acts of the late Chamber of Deputies was to reject a proposal to increase the tax on absinthe. The French colonies are thus better protected from this particular poison than France itself.