

# Comparative study of the susceptibility to infection with "Trypanosoma simiae" of "Glossina morsitans" and "G. tachinoides"

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## **Comparative study of the susceptibility to infection with *Trypanosoma simiae* of *Glossina morsitans* and *G. tachinoides***

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### **Summary**

The susceptibility to infection with *Trypanosoma simiae* of *Glossina morsitans* and *G. tachinoides* was compared. A total of 592 *G. tachinoides* and 348 *G. morsitans* were used in trying to transmit *T. simiae* to pigs. *G. morsitans* were very good at transmitting *T. simiae* infection to pigs while *G. tachinoides* were very poor. The epidemiological importance of the results is discussed.

**Key words:** *Trypanosoma simiae*; *Glossina morsitans*; *G. tachinoides*; pigs; trypanosomes; parasitaemia; transmitting; epidemiology.

### **Introduction**

The distribution of different *Glossina* species in Nigeria is well known (Davis, 1977). However, evidence of differences in susceptibility to trypanosome infections between the species is scanty in Nigeria. Harley and Wilson (1968) and Harley (1971) have shown marked differences in infection rates between species of *Glossina* infected with the same species of trypanosome. There is indication that genetic and/or environmental factors are involved in controlling fly susceptibility (Maudlin, 1982).

The objective of this study was to compare the susceptibility to infection with *Trypanosoma simiae* of *Glossina morsitans* and *G. tachinoides*.

### **Materials and Methods**

*Animals.* 6 large-white pigs were used for the study. They were housed in concrete pens and fed on concentrates and vegetables. Salt licks were also provided.

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*Tsetse flies.* 592 *G. tachinoides* were used. 141 were wild flies caught at Yankari, Bauchi State, Nigeria; 451 were hatched in the laboratory from pupae collected also at Yankari. 348 *G. morsitans* were used, consisting of 320 wild flies caught at Yankari and 28 flies hatched from pupae collected at the same place.

#### *Experimental procedure*

The experiment was divided into two parts. 141 wild *G. tachinoides* were fed on pigs Nos. 10 and 14 for 30 days. Blood films were examined daily to determine if the pigs had become infected. 320 wild *G. morsitans* were fed on pig No. 4 and similar daily examinations made.

One pig was inoculated intramuscularly with *Trypanosoma simiae* which had been stored in liquid nitrogen. When the pig showed a heavy infection of *T. simiae* (40 trypanosomes per microscope field in a wet preparation,  $\times 10$  eye-piece and  $\times 40$  objective), 451 newly emerged (one day old) *G. tachinoides* and 28 newly emerged (one day old) *G. morsitans* were fed on the pig twice daily (morning and evening) for 3 days. The *G. tachinoides* were then fed on pig No. 16 for 27 days while the *G. morsitans* were fed on pig No. YR for 17 days. Roberts (1971) has shown that cyclical development of *T. simiae* in tsetse flies takes between 16 and 25 days. Blood films were examined daily to determine if the pigs had become infected.

## **Results**

No trypanosome was seen in the peripheral blood of pigs Nos. 10 and 14, fed on by wild *G. tachinoides*, even after over 50 days of examining their blood each day.

The results of the *G. morsitans* experiment were different. On the 6th day of feeding wild *G. morsitans* on pig No. 4, *T. simiae* identified morphologically and by rodent inoculation (Hoare, 1936) appeared in the peripheral blood of the pig. The pig died 13 days later after showing heavy parasitaemia.

In the second part of the experiment, no trypanosome was seen in pig No. 16 fed on by *G. tachinoides* while *T. simiae* parasites were demonstrated in the peripheral blood of pig No. YR, fed on by *G. morsitans*. The parasitaemia commenced 21 days after the first feeding by the flies. The pig died on the 4th day after parasitaemia became patent.

## **Discussion**

This work agrees with that of others (Desowitz and Watson, 1953; Stephen and Gray, 1960; Isoun, 1968) in showing that *G. morsitans* is an efficient transmitter of *T. simiae* in pigs. The literature is scanty on experimental transmission of *T. simiae* in pigs by *G. tachinoides*. The present work shows that *G. tachinoides* is not a suitable vector of *T. simiae* in pigs. Baldry (1964), in a survey of tsetse flies at Nsukka, Nigeria noted that *G. tachinoides* was very prevalent and showed a marked preference for the local pig as a host. His dissection studies on the flies showed 6.6% infection rate with *T. vivax*, *T. congolense* and *T. brucei*. Killick-Kendrick and Godfrey (1963) examined pigs in close contact with *G. tachinoides* at Nsukka, Nigeria and found that 86% of the 35 pigs examined were infected with *T. congolense* and/or *T. brucei*. The authors did not identify

*T. simiae* in the pigs, however, commented that some of the *T. congolense* infections identified in the pig survey could be due to *T. simiae* of low virulence. Janssen and Wijers (1974) reported that the virulence of *T. simiae* in pigs depended on the species of tsetse transmitting the trypanosomes; *G. brevipalpis* transmitted virulent infections while *G. pallidipes* transmitted chronic infections. Work is continuing to find out if this is so with *G. morsitans* and *G. tachinoides*. Roberts (1971), however, showed that *G. tachinoides* could transmit *T. simiae* to pigs though the infection rate in *G. morsitans* was higher than in *G. tachinoides*. He found by dissection that infection rates of up to 6% and 8.6% may be obtained in *G. tachinoides* and *G. morsitans*, respectively.

No explanation can so far be given for the poor vectorial capacity of *G. tachinoides* for *T. simiae* in pigs demonstrated in the present work. Harley and Wilson (1968) showed that *G. fuscipes* was a poor vector for *T. congolense* when compared with *G. pallidipes*. They thought that the reason for the difference was the frequent feeding of *G. fuscipes* on hosts such as man, reptiles and birds, which were not susceptible to *T. congolense* infection. Genetic factors have been found to control the susceptibility of vectors to infections (Wakelin, 1978; Maudlin, 1982).

My results are significant in the epidemiology of *T. simiae* in pigs, particularly in areas of poor traditional pig husbandry where the pigs roam and scavenge in the compound. Obviously, in areas where *G. morsitans* is prevalent, there would be very limited success in establishing piggeries, except in fly-proof buildings. In areas where *G. tachinoides* are found, there would be a good chance of a successful out-door pig farm.

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