

Article 162

THE PATHOGENICITY OF LOCAL STRAINS OF TRYPANOSOMA

BRUCEI FOR BOVINES IN RHODESIA

BY P.K.I. MACKENZIE AND W.P. BOYT

DEPARTMENT OF VETERINARY SERVICES, TSETSE AND TRYPANOSOMIASIS CONTROL
BRANCH,

P.O. BOX 8283, CAUSEWAY, SALISBURY, S.RHODESIA

INTRODUCTION

H.E. Hornby stated in his report on African Trypanosomiasis in East Africa (1949) that as a rule Trypanosoma brucei provokes a reaction in the bovine so mild as to pass unnoticed, however he goes on to say that occasionally cases may be seen showing the typical wasting and anaemia normally associated with the disease due to T.congolense.

Henning (1956) also remarks on the tolerance of cattle, sheep and goats to T.brucei.

Killick-Kendrick (1971) draws attention to certain errors made in the past regarding the pathogenicity of T.brucei in the bovine but also records that although the organism is commonly apathogenic in this species, producing a sub patent infection, there are recorded cases where deaths have occurred. He quotes Hornby (1930) who in an experiment records that 83% of the infected animals recovered (and so presumably 17% died) and Gray (Leach 1964, 28) who infected two cattle with T.brucei - one recovered and the other died a year later due to trypanosomiasis brucei.

In Rhodesia it is seldom that T.brucei is diagnosed in cattle - during 1966-67, 20 such cases were diagnosed in 8 134 smears examined; in 1967-68 seven cases from 6 314 smears and only one case in 1968-69 from the 5 336 smears examined.

MATERIAL AND METHODS

In June 1969, during the routine collection of trypanosome isolates from donkeys in the Gokwe district, four strains of T.congolense were stored in liquid nitrogen, to be tested later for resistance to isometamidium*. When these isolates were subsequently inoculated into

*Samorin - May and Baker Ltd. Dagenham, England

non-immune cattle outside the tsetse area, the parasitaemias which developed proved to be mixed T.congolense and T.brucei.

One of the animals infected, No. A285, was treated with isometamidium at the dosage rate of 0.25 mg/kg body weight on the 25th day after infecting and although this animal subsequently relapsed with T.congolense, no further evidence of T.brucei was noted. (See Fig. 1)

Haemoglobin estimations, packed cell volumes and erythrocyte counts were carried out on days 49, 65, 80, 93 and 107 to indicate the pathogenic effects of the isolates. As may be seen from the data collected (See Table 1), a low grade anaemia developed in all the cattle over a period of three months.

An interesting feature of this particular experiment was the disappearance of T.congolense and the persistence of T.brucei. T.congolense disappeared after the 29th day in animal A296 and earlier in the other untreated animals. (See Fig. 1)

The development of this low grade anaemia with the persistence of T.brucei, led us to believe that this trypanosome species might be more pathogenic than had been previously thought.

In November 1970 three bovines were infected with a polymorphic trypanosome which had been recovered from a Puku (Anenota vardonii vardonii) in the Luangwa Valley in 1962. This isolate had been passaged in rats three times and then stored in liquid nitrogen, being designated Strain P3.

Blood films from the three bovines were examined twice weekly using the thick haemolysed smear stained by Giemsa's method, 300 fields being scanned on each one taken. Haematological examinations carried out once a week for 30 weeks, comprised haemoglobin estimations, red and white cell counts, haematocrits, erythrocyte sedimentation rates and serum protein estimations. Three uninfected bovines were treated in a similar manner to detect environmental effects.

RESULTS:

Initially the infection followed the usual pattern of a fairly constant patent parasitaemia. Within seven weeks however, parasites could only be detected in the peripheral circulation at irregular intervals, that is to say by blood film examination. These occult phases became more frequent and longer in duration as time elapsed: Sub-inoculation into mice was commenced on day 164 after infection, 1cc of blood being injected intraperitoneally into each of four mice from every animal weekly for eight weeks - with the exception of one animal (C359 on the 4th week) all mouse sub-inoculations were fruitful, prepatent periods varying from seven to 20 days. Further sub-inoculations from these three bovines on day 269 again showed two of the 3 animals to be harbouring a subpatent parasitaemia. (See Fig. 2)

As a result of these observations, one of the animals used in the initial investigation with the mixed T. brucei/T. congolense strain, vide supra, was subjected to further examination. Sub-inoculations of blood into mice on days 693 and 707 were fruitful, the mice developing a parasitaemia within 12 and 14 days respectively.

In our investigations with the polymorphic strain P3 it can clearly be seen that liveweight increases were approximately the same as in the non-infected control animals. (See Fig. 3)

The values for haemoglobin, haematocrit and erythrocyte counts showed no significant deviation from the corresponding figures recorded for the control animals, with the possible exception of a slightly lower haematocrit in the initial period of four weeks. (See Figs. 4,5 and 6)

The serum protein values did however show certain differences between infected and non infected groups. It can be seen that the infected animals showed a higher total protein, this being almost solely due to an increase in the globulin fraction. (Figs. 7,8, and 9) Although no immunological investigation was carried out, it is presumed that the increase in globulin was due to the production of antibody to the infecting organism. We therefore assume that the strain used was antigenically potent. This

host response however does not immediately eliminate the parasite.

DISCUSSION:

Trypanosomes of the brucei group appear to persist in the bovine for long periods as a sub clinical infection. This poses problems in the control of trypanosomiasis amongst a heterogeneous population of domestic stock where the trypanosome risk is light enough for the animals to be maintained by the use of the curative drug Berenil. (^{diminazine} iminbazine diacetate). The makers recommended that this drug should be used at twice the normal dosage rate of 3.5 mg/kg to cure an animal infected with T. brucei. Thus, where this chemotherapeutic agent is used at the normal dosage rate one might expect bovines to continue to harbour trypanosomes of the brucei group thus providing a reservoir of infection for tsetse which could in time infect other species such as members of the equine and canine families.

Cunningham and van Hove (1964) in a survey carried out in Zebu cattle from the Alego location in Kenya recovered 43 isolates; all of these were confined to the brucei group and some later proved to be T. rhodesiense.

This demonstrates that not only does the reservoir hazard apply to other livestock but might provide the basis for a zoonosis of human sleeping sickness.

In Rhodesia it is indeed fortunate that human trypanosomiasis does not occur to the extent noted elsewhere and moreover is at present confined to areas where there is little or no domestic stock. Onyango et al (1966) considered the epidemic of human sleeping sickness in the Alego location of Kenya to be mainly due to the reservoir status of the domestic bovines in the area.

To summarise, the evidence we have to date suggests that trypanosomes of the brucei group are usually apathogenic in the domestic bovine, and as such are not a serious veterinary problem, but the host parasite relationship appears to be such as to allow the survival of the organism for a long period within that host.

ACKNOWLEDGEMENTS

We wish to acknowledge the technical assistance of Miss M. Moorcroft, Mr. V. Luesely and Mr. S. Masimbe. The Director of Veterinary Services is thanked for permission to publish this paper.

REFERENCES

- Cunningham, M.P., van Hove, K. (1964) Diagnosis of trypanosomiasis in cattle. I.S.C.T.R. 1964 p51
- Henning, M.W. (1956) Animal diseases in South Africa. Central News Agency - South Africa
- Hornby, H.E. (1949) Animal trypanosomiasis in Eastern Africa. London. Her Majesty's Stationary Office 1952
- Hornby, H.E. (1930) Control of Animal trypanosomiasis - In Proc. 11th Int.Vet.Congr., London. Bale, Sans and Danielson
- Killick-Kendrick, R. (1971) The low pathogenicity of *Trypanosoma brucei* to Cattle. Trans.Roy.Soc.Trop.Med.Hyg. 65 104
- Leach, T.M. (1964) Ann.Rep.Nig.Inst.Tryp.Res. p.28
- Onyango, R.J., van Hove, K., De Raadt, P. 1966 The epidemiology of *Trypanosoma rhodesiense* sleeping sickness in Alego Location, Central Nyanza, Kenya. Trans.Roy.Soc.Trop.Med.Hyg. 60 175

SUMMARY

The main reports and investigations relating to the pathogenicity of T. brucei for the bovine are briefly reviewed.

The paper describes an experience during the course of an experiment connected with drug resistant trypanosomiasis in cattle in which there were indications that T. brucei might be more pathogenic than was hitherto believed.

A further experiment was undertaken using three head of cattle infected with an isolate of the brucei sub-group from an antelope. The main blood parameters, bodyweight and parasitaemia were monitored and the results confirmed the general opinion that trypanosomiasis of the brucei sub-group are virtually apathogenic for the bovine.

Xeno-diagnosis using mice indicated that the parasite persisted for long periods at a sub-patent level. The danger of cattle as potential reservoirs of T. rhodesiense is underlined.