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## **Serum from the cotton rat (*Sigmodon hispidus*) lacks lytic activity against some *Trypanosoma vivax* stocks**

### **Short communication**

P. R. GARDINER, R. THATTHI, R. C. KING

The investigation of naturally occurring reagents which specifically agglutinate or lyse trypanosome populations may reveal exploitable target molecules of the parasite, other than the variable surface glycoproteins, for biochemical or immunological therapy of trypanosomiasis. In the case of *Trypanosoma vivax* two types of reagents have been identified, the bacterial toxin aerolysin (Gardiner et al., 1987) and normal serum from the cotton rat, *Sigmodon hispidus* (Terry, 1957; Hudson and Terry, 1970). However, Cover (1984) has reported that the agglutinating and trypanolytic properties of normal cotton rat serum for some stocks of *T. vivax* (Terry, 1957; Hudson and Terry, 1970) were limited to one or two trypanosome stocks. The susceptible stocks were the rodent-infective (Desowitz, Wellcome) stock and a stock isolated from a tsetse-infected goat in Nigeria. The anti-*vivax* factor was thought to be IgA (Hudson and Terry, 1970).

We have sought to extend these observations to comparable but different stocks and clones of *T. vivax* using freshly collected cotton rat serum from the ILRAD colony, or reconstituted cotton rat serum (kindly supplied as a lyophilisate by Professor Charles Tanner of the Institute of Parasitology, McGill University, Canada). The trypanosomes used were IL 1392, a clone of the naturally rodent infective stock, originally isolated as Y486 (Leefflang et al., 1976) in Nigeria (also used by Cover, 1984); clones IL 2931 and IL 2932 derived respectively from stocks of *T. vivax* from Lugala and Teso in Uganda which have been recently adapted to rodents (Gathuo et al., 1987) and IL 2946 a derivative of EATRO 1721 isolated in Nigeria, which is not infective for rodents and in these experiments was taken from an infected goat. Only IL 2931 was homogeneous for variable antigen type (VAT).

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Agglutination and lysis tests employing trypanosomes in infected, heparinised blood or separated from blood elements by DEAE-cellulose chromatography (Lanham and Godfrey, 1970) were performed. In neither test did whole cotton rat serum have any effect on any of the trypanosome stocks. In contrast, specific mouse antisera to the three rodent-infective populations lysed 70–100% of the homologous populations (in the presence of guinea pig complement). Total globulin fractions (precipitated with saturated ammonium sulphate) of both preparations of cotton rat sera, or cotton rat IgA (eluted from jacalin columns; Pierce Chemical Company, Rockford, IL, USA), similarly failed to lyse any of the trypanosome stocks with or without the addition of complement. These serum fractions and whole cotton rat serum did not neutralize the infectivity of the rodent-infective stocks for Balb/c mice, following a 30 min incubation of  $10^3$  trypanosomes in the various serum preparations prior to i.v. inoculation of the mice.

Direct inoculation of 2-month-old cotton rats with  $10^6$  trypanosomes of the rodent-infective stocks led to the sporadic appearance of parasites for up to 4 days in some animals, but trypanosomes were not detected thereafter. Such observations tend to confirm the insusceptibility of cotton rats to infection with *T. vivax*, although an immediate lysis of the inoculated trypanosomes by the hosts' serum did not apparently occur.

It is not known whether the 'natural' antibody of cotton rats recognised variant or invariant antigens of the Desowitz Wellcome stock and the Nigerian stock of *T. vivax*. Exploitation of this phenomenon for the immunological therapy of *vivax* trypanosomiasis will be difficult, or impossible, if the target antigens are expressed by so few *T. vivax* stocks, or the natural antibody is confined to some colonies of cotton rat.

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