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PATHOLOGY

Infectivity of *Trypanosoma rhodesiense* to Tsetse Flies fed through Animal Membranes

THE trypanosomes belonging to the *Trypanosoma brucei* group (agents of sleeping sickness and nagana) exist in the blood of the host in three main morphological forms—long thin, intermediate and short stumpy. When these are taken up by the tsetse fly they undergo developmental changes, namely trypanosome, crithridial and metacyclic forms, the last being infective to the vertebrate host. There is controversy about which forms are infective to the fly, the long thin, the short stumpy or both.

It is well established that the blood forms of the trypanosomes retain their viability as well as other biological characteristics after long periods of storage at −79° C (refs. 1 and 2). Feeding through membranes has been used for the regular maintenance of tsetse flies in the laboratory[3], and for the collection of metacyclic trypanosomes from infected flies[4]. An unsuccessful attempt to infect *Glossina pallidipes* with preserved *Trypanosoma brucei* group trypanosomes was made by Cunningham[5], who suggested that the failure was caused by the absence of short stumpy forms from the suspension used.

During a study on the infectivity of the two main morphological forms of these trypanosomes to tsetse flies, a batch of twenty-three *G. palpalis* were given an infective blood meal which contained only the long thin forms of *T. rhodesiense*, through a fresh mouse skin membrane. The morphology of the trypanosomes was determined by the method described by Ormerod *et al.*[6]. One of the flies later extruded trypanosomes in its salivary secretion, and transmitted the infection to a guinea-pig before it died (42 days after). This suggests that, under certain conditions, both morphological forms of these trypanosomes may become established in the fly[7–10], contrary to the results of several workers[11–13]. The blood of the guinea-pig, containing both long thin and short stumpy trypanosomes, was frozen at −79° C in ampoules.

After 14 months the contents of one ampoule were fed to nine newly emerged *G. austeni* through a fresh mouse skin membrane. Three flies died within 10 days and were not examined. All other flies were dissected as soon as possible after death. One infected fly was detected on the twenty-sixth day; however, when the flagellates obtained were subsequently inoculated into a rat, no infection occurred.

After 16 months' storage at −79° C, the contents of another ampoule were similarly fed to twenty-two *G. austeni*, of which eleven survived to the tenth day. Two flies which died on the fourteenth day contained active trypanosomes which produced an infection in a mouse. Five other flies dissected between the nineteenth and twenty-eighth days contained active trypanosomes, but these did not infect test animals.

It is therefore concluded that tsetse flies may be infected with preserved *T. brucei* group trypanosomes by feeding through membranes. The successful infection of a fly from a suspension which contained only long thin trypanosomes indicates that negative results, such as those of Cunningham[5], would result from some reason other than the absence of short stumpy forms in the blood meal. It is suggested that the technique of membrane feeding, as opposed to that of feeding flies on infected animals, offers more precise conditions for investigation of the factors which influence the infection rate of tsetse flies with these trypanosomes. These investigations should be based on the factors in the trypanosomes; the mammalian host; and the individual tsetse[14]. Other characteristics of the trypanosomes that may be investigated in this way include their infectivity, transmissibility by tsetse, antigenicity and drug resistance. The preservation of metacyclic trypanosomes derived from tsetse flies[4] adds considerably to the possibilities of such studies.

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Leaching of Constituents of Chrysotile Asbestos *in vivo*

IN recent years, Wagner[1] and Selikoff *et al.*[2] have shown that a rare tumour, the diffuse mesothelioma of the pleura and peritoneum, is associated with past exposure to asbestos. It appears that the amount of asbestos required to produce these tumours is small and that the latent period is very long. The connexion between exposure to asbestos and the production of mesotheliomas is being studied in a number of laboratories, and the possibility that trace metal constituents or contaminating oils may have a role has been suggested[3]. As yet, little is known about the fate of inhaled asbestos fibres and in particular about their movement out of the lung into other organs. The experiment described here was planned to assess the possibility of using radioactivity, induced in asbestos fibres by neutron irradiation, to trace their translocation in rats after administration by intrapleural injection.

A sample of Rhodesian chrysotile was irradiated in a high flux of thermal neutrons ($1{\cdot}5 \times 10^{14}$/cm^2/sec) for 10 h. After allowing some weeks for the short-lived activity to