Absence of trypanosomes in polar bears (*Ursus maritimus*) from Svalbard

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MEMBERS of the protozoan genus *Trypanosoma* are obligate parasites, occurring in all classes of vertebrates. Most species of trypanosomes have a two-host life cycle alternating between a vertebrate and invertebrate host. In vertebrates, the majority of trypanosomes are found extracellularly in the blood or other tissue fluids, but some species, for example *Trypanosoma cruzi* of human beings and other mammals, are intracellular parasites. Most *Trypanosoma* species of mammals are transmitted by blood sucking insects, mainly tsetse flies, tabanid flies, and bugs (Hoare 1972). Thus, the geographical distribution of trypanosomes is dependent on the availability of suitable vectors (Hoare 1972, Levine 1985). A number of *Trypanosoma* species exist, having varying degrees of host specificity and pathogenicity (Hoare 1972). Some species are highly pathogenic in domesticated animals, but have limited pathogenicity in wild animals. Other species are non-pathogenic in all their hosts.

The occurrence of trypanosomes in mammals in the Arctic is poorly documented, but has been reported in reindeer (*Rangifer tarandus*) (Kingston and others 1982) and moose (*Alces alces*) (Kingston and others 1985). Svalbard has only two native species of mammals; the Svalbard reindeer (*Rangifer tarandus platyrhyncus*) and the arctic fox (*Alopex lagopus*). No blood parasites have been found in the Svalbard reindeer (Bye 1986), while arctic foxes have not been surveyed.

Trypanosomes have never been described from polar bears (*Ursus maritimus*) living in their natural habitat in the arctic region of the far north (DeMaster and Stirling 1981). As part of a larger study on the ecology of polar bears in the Barents Sea, an investigation of the presence of trypanosomes was undertaken. This was done in association with other investigations involving blood sampling. After this study was initiated, a report of a *T cruzi* infection in a polar bear in a zoo in Mexico was published (Jaime-Andrade and others 1997). However, this species is transmitted by reduviid bugs, a family of insects confined to the tropical areas of the world.

There are limited data on the parasite fauna of polar bears. Only nine parasites have been reported from this host (Rogers and Rogers 1976, Garner and others 1997, Jaime-Andrade and others 1997), and of these, only the nematode *Trichinella spiralis* (probably *Trichinella nativa*) has been reported in wild polar bears (Madsen 1961, Kjos-Hanssen 1984, Kumar and others 1990).

Blood samples were collected from polar bears at Svalbard and in the Barents Sea (76° to 79° N and 20° to 45° E) from late March until mid-May 1997 and from late April until mid-May 1998. Polar bears were temporarily captured by remote injection of a 1:1 combination of tiletamine and zolazepam (Zoletil; Virbac) administered by a projectile dart fired from a helicopter (Stirling and others 1989). Peripheral blood was collected from the femoral vein of the bears using evacuated blood collection tubes containing ethylene diamine tetraacetic acid or heparin as an anticoagulant. One or two blood smears were prepared from each blood sample on microscope slides, which were labelled with the identity number of the bear. The slides were air dried and stored in boxes until processed at the laboratory. In 1997, blood smears intended for trypanosome examination were prepared from 117 polar bears. In addition, a blood smear for haematology was prepared from each of 30 of these bears caught in the Barents Sea. In 1998, two blood smears intended for haematology were prepared from each of 38 polar bears caught in the central Barents Sea.

The 117 slides prepared for trypanosome examination in 1997 were fixed in methanol and stained by Giemsa stain according to standard procedures. The slides made for haematology in 1997 and 1998 were fixed in methanol and stained by a modified Wright-Giemsa stain using the fully automated Hema-tek 2000 Slide Stainer (Bayer Diagnostics) and a Hema-tek Stain Pak (Bayer Diagnostics). All blood smears were examined microscopically for the presence of trypanosomes.

Trypanosomes were not detected in any of the 223 blood smears from polar bears examined. Many of the 117 blood smears prepared for trypanosome examination only, were too thick and not well stained by the Giemsa stain used. However, all of these slides had areas where it would have been possible to detect trypanosomes, if present. All 155 haematology slides from 68 different polar bears were of good quality, but no trypanosomes could be found.

The most likely explanation for the absence of trypanosomes is that polar bears in the arctic are not natural hosts to such parasites. This is not unexpected, since there are few suitable blood sucking vectors in the Svalbard area (Sømme 1993). Another possibility is that a low level of infection might have gone undetected because few, if any, parasites were present in the peripheral blood at the time of sampling. If trypanosomes occur in the polar bear, they will probably be transmitted by blood sucking invertebrates only during the short summer period. Thus, the trypanosomes might be present in the peripheral blood only during the period when the vector is available, as is known to occur for some trypanosomes in birds (Levine 1985). Furthermore, the use of one or two blood smears from each animal is not a very sensitive method for detecting an infection. On the other hand, the total number of slides and animals examined was quite high in this investigation, thus increasing the chances of detecting any parasites.

From the present study it seems unlikely that polar bears in the Svalbard area are hosts for trypanosomes. Investigation of polar bears in Hudson Bay, where they are harassed by several species of biting insects (Derocher and Stirling 1990, Clark and others 1997), may, however, provide evidence of trypanosomes. As reported by Jamie-Andrade and others (1997), polar bears forced to live in other regions of the world might become infected by *Trypanosoma* species present in those areas.

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Pathological changes in free-ranging African ungulates during a rinderpest epizootic in Kenya, 1993 to 1997

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RINDERPEST, caused by a morbillivirus of the family Paramyxoviridae, produces disease in wild and domestic ruminants and pigs. Historically, it has devastated cattle populations in Africa, Asia and Europe, promoting national and international control strategies for eradication. As the virus is highly susceptible to environmental inactivation, maintenance in populations requires continual transmission by direct contact with secretions from infected animals to susceptible hosts. Vaccination is effective in preventing the disease, but outbreaks occur periodically, presumably when disease surveillance and/or vaccine regimens are relaxed. Epizootics involving wildlife have occurred relatively recently in East Africa, a 1982 outbreak in Tanzania (Nyange and others 1983) and a 1960 to 1962 outbreak in Kenya (Stewart 1964). An epizootic among wild ruminants occurred in Kenya between 1993 and 1997, which resulted in an estimated overall decline in certain wild ruminant populations of between 50 to 80 per cent but had minimal effect on cattle (Kock and others 1999).

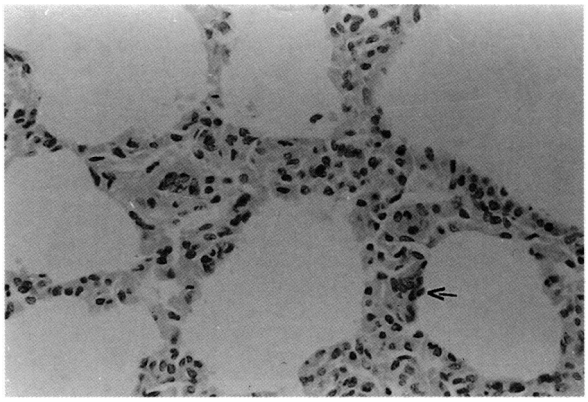

FIG 1: Lung from a lesser kudu (*Tragelaphus imberbis*) with rinderpest. Syncytial cells can be seen lining the airways (arrow). Haematoxylin and eosin × 400

Fifty-seven wild ruminants and a warthog suspected of having rinderpest were necropsied during the epizootic. Tissues from 21 buffalo (*Synceros caffer*), six lesser kudu (*Tragelaphus imberbis*), four eland (*Taurotragus oryx*), two impala (*Aepyceros melampus*), one bushbuck (*Tragelaphus scriptus*) one giraffe (*Giraffa camelopardalis*) and one warthog (*Phacochoerus aethiopicus*) were examined microscopically. Formalin-preserved samples of heart, lung, kidneys, liver, spleen and intestine were examined in most cases, and lymph node, salivery gland, abomasum, conjunctiva and eye, pancreas and oesophagus were examined in some cases. All samples were submitted to the University of Zimbabwe (Faculty of Veterinary Science, Harare, Zimbabwe). The tissues were trimmed, embedded in paraffin, cut at 6 to 10 µm, stained with haematoxylin and eosin and examined by light microscopy.

Clinically, the buffalo were often emaciated, with peripheral lymphadenopathy, and had ulcers of the lips, mouth, stomach, nares and cornea. Gross findings in eland and kudu were similar, although less pronounced. Vision loss, corneal opacity, ulcerative keratoconjunctivitis and tear staining of the face were consistently seen in kudu.

The most striking histopathological lesions occurred in the lesser kudu, where individual epithelial cell necrosis was found in renal tubules, abomasum, small and large intestine, lung, conjunctiva, and salivary, pancreatic and bile ducts, often with the formation of syncytial cells (Fig 1). The most chronic lesions were present in the conjunctiva, possibly indicating the point of viral entry. Single cell necrosis was also found in renal tubules, salivary ducts, abomasum, small and large intestine and conjuctiva in the buffalo, but typically without syncytia (Fig 2). Epithelial cell necrosis was present in the oesophagus, abomasum and small and large intestines in the eland, with syncytia only found in the small intestine. Lymph nodes and spleen in all three species often had haemosiderin deposits, suggesting prior haemorrhage, either in the lymph nodes themselves, or in the tissues of drainage. None had lymphoid necrosis typical of rinderpest in cattle (Barker and others 1993) or, as has been reported previously, in wildlife species (Scott 1981). Although general malaise and poor body condition were also seen in the impala, giraffe, bushbuck and warthog during the epizootic, histopathological lesions did not support a diagnosis of rinderpest.

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