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Abstract: *Hepatozoan* spp. are apicomplexan parasites occurring in a wide range of mammals, birds, reptiles and amphibians. Between 1987 and 1991 within the Serengeti ecosystem, 24 cheetahs were examined for the presence of blood parasites. Microscopical examination revealed the presence of *Hepatozoan* in the leucocytes from thirteen cheetahs (54.2%). The highest parasitaemias occurred in October-November and might possibly be associated with increased tick activity during the short rainy season and with the use of habitat with long grass.



Notes and Records

Hepatozoonosis in cheetahs and wild dogs in the Serengeti ecosystem

Hepatozoon spp. are apicomplexan parasites occurring in a wide range of mammals, birds, reptiles and amphibians. There are about 85 named species (Levine, 1988). The most common mammalian species is probably Hepatozoon canis (James) occurring in domestic dogs in the tropics and sub-tropics where suitable tick vectors occur. Records of Hepatozoon from various African carnivores have frequently been described as being H. canis, and even some previously named species from specific hosts have been synonymized with the dog parasite (Levine, 1988). Hepatozoon has been recorded previously in Serengeti carnivores, most recently by Averbeck et al. (1990).

Between 1987 and 1991 within the Serengeti ecosystem, 24 cheetahs (Acinonyx jubatus (Schreber)) and sixteen wild dogs (Lycaon pictus (Temminck)) were examined for the presence of blood parasites. These studies were only part of larger investigations on the blood parameters of the hosts and, in the case of the wild dogs, the epidemiology of rabies (Gascoyne et al., 1993). Blood samples were collected in EDTA from animals immobilized for the fitting and removal of radio collars and thin smears prepared, air dried, fixed in methanol and

subsequently stained with Giemsa.

Microscopical examination revealed the presence of Hepatozoon in the leucocytes from thirteen cheetahs (54.2%) and thirteen wild dogs (81.5%). Of fifteen cheetahs which were re-sampled (twelve on one occasion and three on two occasions) after an interval of 3-14 months, nine had Hepatozoon in at least one sample (five had retained patency, two had lost it and two had gained). The highest parasitaemias occurred in October-November and might possibly be associated with increased tick activity during the short rainy season and with use of habitat with long grass. The wild dogs were sampled in all months except December and no seasonal prevalence with Hepatozoon was observed. These are the first records of Hepatozoon in African wild dogs. In addition to Hepatozoon three cheetahs also had low parasitaemias with intraerythrocytic piroplasms (either Theileria or Babesia). One wild dog also had microfilariae, and one had a single piroplasm, possibly Bahesia sp.

In an earlier study Averbeck et al. (1990) also found a high prevalence of Hepatozoon infection in carnivores within the Serengeti ecosystem although they concentrated mainly on lions (100% prevalence) and included only eight cheetahs (100% prevalence). Why the prevalence of Hepatozoon in cheetahs from the present study was lower is not clear, but may be due to sampling throughout the year rather than restricted to a 3-month span (July to September) by Averbeck

et al. (1990).

The pathogenicity of Hepatozoon in wild carnivores is unknown. Schizonts have been found in a wide range of tissues particularly in the lung, myocardium and skeletal muscle, but also in the spleen, liver and lymph nodes (McCully et al., 1975). In domestic dogs, severe infections with H. canis have usually been

accompanied by concomitant infection with other disease agents resulting in considerable necrosis and granuloma formation in the spleen, liver and lymph nodes (McCully et al., 1975). Fever, anaemia, weight loss and occasionally paralysis of the hind limbs have also been reported (Desser, 1993). Whether similar responses occur in wild carnivores is yet to be determined.

The morphology of Hepatozoon gametocytes in the present study indicates differences between those in cheetah and wild dogs. This suggests that synonymizing many African carnivore Hepatozoon spp. with H. canis (Levine, 1988) is clearly in error. In a recent review of Hepatozoon, Desser (1993) suggests that host specificity in mammals may be more prevalent than in reptiles. This view is further supported by studies in South Africa where unsuccessful attempts were made to transmit Hepatozoon from hyaena (Crocuta crocuta (Erxleben)) and jackal (Canis mesomelas Schreber) to domestic dogs (Basson et al., 1971; McCully et al., 1975). Further evidence of host specificity is presented by studies in the tick vectors. The most common vector of H. canis in domestic dogs is Rhipicephalus sanguineus (Latreille). However, specimens of R. sanguineus, R. simus Koch and Haemaphysalis leachii (Audouin) collected from hyaena revealed developmental stages of Hepatozoon only in R simus (McCully et al., 1975). In Tanzania, Yeoman & Walker (1967) recorded R. simus as the only species found on wild dogs (only three sampled) and no records of any ticks

Although ixodid ticks have traditionally been considered the principal vectors of large mammal Hepatozoon spp., recent studies on H. atticorae (de Beaurepaire Aragao) in the South African cliff swallow Hirundo spilodera Sundevall have indicated both an argasid tick and a flea as vectors (Bennett, Earle & Penzhorn, 1992). Other arthropods (mites, mosquitoes and tsetse) are known to be vectors of Hepatozoon spp. in small mammals and reptiles (Desser, 1993). Thus the potential vectors of Hepatozoon in African carnivores may not be restricted only to ixodid ticks.

This paper lends weight to the suggestion that host specificity, at least to familial level, may be as prevalent in mammals as recently described for avian species of Hepatozoon (Bennett, Earle & Peirce, 1992). To further elucidate this question material is currently being accumulated from a wider range of African carnivores as a prelude to a taxonomic review of Hepatozoon species. Research is also clearly required to determine the vectors responsible for Hepatozoon transmission, and pathogenicity in the host.

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