Short Communications

A survey of internal parasites in free-ranging African wild dogs (*Lycaon pictus*) from KwaZulu-Natal, South Africa

Gabriella Flacke[1]*, Penny Spiering[2,3], Dave Cooper[4], Micaela Szykman Gunther[2,5], Ian Robertson[6], Carlysle Palmer[1] & Kristin Warren[1]

[1]*Conservation Medicine Programme, School of Veterinary and Biomedical Sciences, Murdoch University, Perth, Western Australia, 6150 Australia*

[2]*Center for Species Survival, Smithsonian Conservation Biology Institute, National Zoological Park, Front Royal, VA 22630, U.S.A.*

[3]*Centre for Wildlife Management, University of Pretoria, Pretoria, 0002 South Africa*

[4]*Ezemvelo KZN Wildlife, Queen Elizabeth Park, Pietermaritzburg, 3202 South Africa*

[5]*Department of Wildlife, Humboldt State University, Arcata, CA 95521, U.S.A.*

[6]*Veterinary Epidemiology Programme, School of Veterinary and Biomedical Sciences, Murdoch University, Perth, Western Australia, 6150 Australia*

Received 28 April 2010. Accepted 29 July 2010

A study was undertaken between 1 January 2006 and 16 February 2007 to identify haemoparasites and gastrointestinal parasites infecting African wild dogs (*Lycaon pictus*) in KwaZulu-Natal (KZN) province, South Africa. Blood and faecal samples were opportunistically collected from wild dogs immobilized for collaring or translocation purposes (*n* = 24). Three common domestic canine gastrointestinal parasites, *Toxocara canis, Dipylidium caninum* and *Ancylostoma* spp., and two genera of canid protozoan GI parasites, *Sarcocystis* and *Isospora,* were identified in 12 fresh faecal samples. The seroprevalence of *Ehrlichia canis* from 24 individual serum samples analysed was 83%. However, only 21% of the 14 whole-blood smears evaluated for the presence of *E. canis* morulae within monocytes were positive. Twelve whole-blood smears were evaluated for the presence of *Babesia canis* trophozoites within erythrocytes and revealed 0% prevalence. Although there is currently no evidence of direct parasite-related mortality in the KZN population, the presence of internal parasites may be more detrimental to the overall health status of African wild dogs with immunosuppression as a result of other disease conditions, translocation stress, or inbreeding depression.

Key words: *Ancylostoma, Babesia canis, Dipylidium caninum, Ehrlichia canis,* internal parasites, *Isospora, Sarcocystis, Toxocara canis.*

The African wild dog (*Lycaon pictus*) is currently the most endangered carnivore in South Africa, where the total population of free-ranging animals has been estimated to be only 300–400 individuals (Woodroffe *et al.* 2004; Davies-Mostert *et al.* 2009). The largest and currently the only viable, self-sustaining population of African wild dogs is located within the Kruger National Park (KNP). As of 2007, the province of KwaZulu-Natal (KZN) had a population of 80–90 wild dogs, primarily located within Hluhluwe-iMfolozi Park (HiP), accounting for approximately a quarter of South Africa's total population (Davies-Mostert *et al.* 2009). The objective of this study was to identify the prevalence of various gastrointestinal and haematogenous internal parasites present in the KZN population of African wild dogs.

This project operated in collaboration with the KZN African Wild Dog Conservation and Reintroduction Programme, which utilizes radio collars for tracking breeding packs and individual dispersing animals. Faecal and blood samples were collected opportunistically for this study between 1 January 2006 and 16 February 2007 when wild dogs in the KZN population were immobilized for translocation or to place or remove radio collars. Data were gathered from a total of seven African wild dog packs in the KZN population; five packs were located in Hluhluwe-iMfolozi Park (28°4′59″S, 32°4′59″E), one pack in Mkhuze Game Reserve (27°37′59″S, 32°15′E), and one pack in Thanda Private Game Reserve (27°24′S, 32°9′E). As this project was a collaborative effort and samples were collected by different individuals at different times, there was not a serum sample, whole-blood smear, and fresh faecal sample available for each immobilized wild dog. Furthermore, since samples were only collected from individuals that were immobilized according to management needs, a majority (83%) of samples were from adult wild dogs (two years of age or older) instead of juvenile or sub-adult animals, slightly more females (58%) were sampled than males (42%), and the seven study packs were not equally sampled.

*To whom correspondence should be addressed.
E-mail: gflacke@hotmail.com

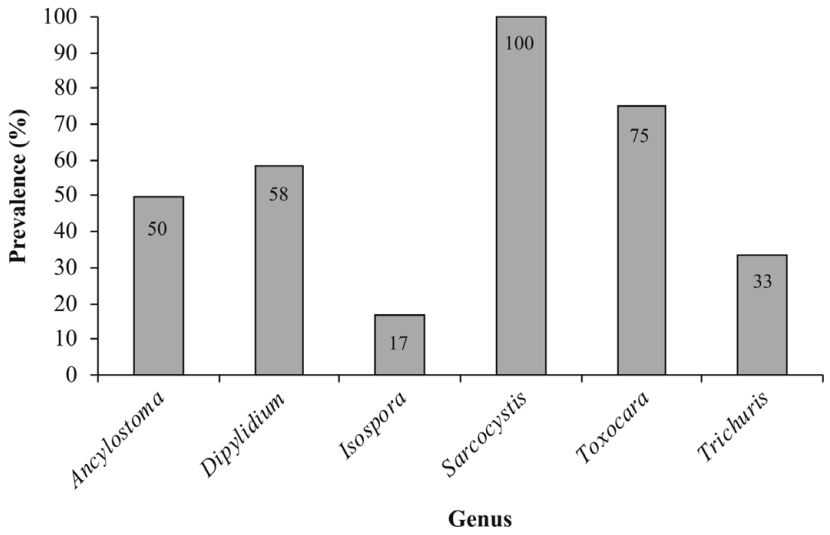

Fig. 1. Mean prevalence of the six genera of gastrointestinal parasites identified in 12 African wild dogs (*Lycaon pictus*) from the KwaZulu-Natal population between 1 January 2006 and 16 February 2007.

Over the study period, fresh faecal samples were collected from 12 African wild dogs for faecal parasite screening. Faecal samples were collected opportunistically (if produced by the animal during the immobilization procedure) and placed directly in Fecalyzer (EVSCO Pharmaceutical, Buena, New Jersey, U.S.A.) containers for storage until faecal float analysis could be conducted. Samples were analysed within 24 hours of collection for presence of parasitic eggs using a sodium nitrate egg flotation fluid (Kyron Laboratories Pty (Ltd), Benrose, South Africa.) using the faecal flotation method described by Zajac (1994).

Serum samples from 24 African wild dogs were collected in serum separator tubes and subsequently centrifuged within 24 hours of collection. The serum was frozen at –20°C until analysis was conducted. Serum antibodies reactive to *Ehrlichia canis* were detected by means of an indirect fluorescent antibody technique as previously described (van Vuuren 1990).

Whole-blood smears were created for 14 wild dogs at the time of sample collection to evaluate for the presence of *E. canis* within monocytes and *Babesia canis* trophozoites within erythrocytes. All whole-blood smears were of acceptable quality to evaluate white blood cell morphology and to determine the presence of potential parasites within these cells. However, on two of the 14 blood smears all of the red blood cells were lysed and only white cells remained intact (the reason for this occurrence is unknown). Thus, these particular slides could not be evaluated for the presence of intra-erythrocytic *B. canis* organisms and blood smears from only 12 animals remained available for this purpose. Evaluation of the slides was conducted by scanning 100 sequential high power microscopic fields (×100, oil emersion) per slide.

There were six genera of gastrointestinal parasites identified from the 12 wild dogs for which - faecal flotation examinations were performed (Fig. 1): three nematode genera (*Toxocara, Trichuris* and *Ancylostoma*), one genus of cestode (*Dipylidium*) and two protozoal genera (*Sarcocystis* and *Isospora*). The calculated seroprevelance of *E. canis* from the 24 serum samples analysed was 83% (95%; CIs 68, 98%). Of the 14 whole-blood smears examined for the presence of *E. canis* morulae within monocytes, three were positive (21%; 95% CIs 4.7, 51%). Of the 12 whole-blood smears examined for the presence of *B. canis,* none was positive for trophozoites within red blood cells (0% prevalence; 95% CIs 0.0, 27%).

Owing to the comparatively small number of fresh faecal samples available for examination (n = 12), the reported prevalence values for each of the six genera of gastrointestinal parasites identified in this study may substantially differ from the actual prevalence. Furthermore, when evaluating the results it must be remembered that most gastrointestinal parasites exhibit intermittent shedding of ova and that parasitic ova are not evenly distributed throughout the faeces (Little 2007). Therefore, any single faecal sample may not contain

parasite eggs even in the presence of a patent infection, making it possible that our data also underestimated the diversity of parasitic genera as well as the actual prevalence of these parasites in the KZN wild dog population.

A previous gastrointestinal parasite survey in African wild dogs conducted by van Heerden *et al.* (1995) in KNP identified three gastrointestinal parasites, nematodes *Ancylostoma* and *Toxascaris* and the cestode *Taenia.* Thus, the results of the present study show that the diversity of gastrointestinal parasites for wild dogs in KNP was lower than for the KZN population, and additionally there is apparently little overlap of parasite type between the two populations. One potential reason for the observed lower diversity of gastrointestinal parasites in KNP wild dogs compared with KZN is a lower rate of exposure to domestic dogs for the KNP population due to substantially larger reserve size and thus a smaller domestic animal/wildlife interface at reserve boundaries. Other potential reasons for the lack in overlap of gastrointestinal parasites between the KNP and KZN wild dog populations include climatic differences between the two reserves which can affect parasite diversity by affecting parasite life cycles, and the fact that a percentage of the KZN founder population of wild dogs was originally translocated from other reserves around South Africa, possibly bringing pre-existing parasitic infections with them.

The overall systemic health implications of gastrointestinal parasites in free-ranging African wild dog populations are not currently known. Young domestic dogs (*Canis familiaris*) can suffer from a number of gastrointestinal disturbances and weight loss associated with ascarid nematodes (e.g. *Toxocara canis*) and from significant anaemia secondary to *Ancylostoma* spp. (Bowman 1999). Severe infections of these parasites can result in death due to gastrointestinal intussusception or exsanguinations, respectively (Bowman 1999). Canine cestode infections with *Dipylidium caninum* are caused by ingesting an infected flea-intermediate host (primarily *Ctenoecephalides felis*). However, gastrointestinal cestodes are thought to be relatively non-pathogenic in otherwise healthy, adult domestic dogs, and are more of a concern due to their zoonotic potential (Little 2007). *Sarcocystis* spp., an apicomplexan protozoan parasite, also generally does not result in clinical illness in the carnivore definitive host, although reproduction via schizogony in the endothelial tissues of the herbivorous intermediate host can cause disease (Dubey & Odening 2001). The final gastrointestinal parasite identified, *Isospora* spp., a host-specific coccidian parasite, is generally only considered pathogenic in very young animals and establishes self-limiting, non-clinical infections with intermittent faecal shedding of oocysts during times of stress in adults (Duszynski & Upton 2001).

It is reasonable to assume that similar to the situation in domestic dogs, the effects of gastrointestinal parasitism in African wild dogs are most severe in very young and immunocompromized animals. Furthermore, as is the case with most free-ranging adult canids (Kennedy-Stoskopf 2003), it is likely that wild dogs evolved concurrently with a low level of chronic gastrointestinal parasitism and are therefore less susceptible to disease secondary to these parasites. For these reasons and because no immobilized animals exhibited clinical signs of severe intestinal parasitism (e.g. diarrhoea, weight loss, ascites, pale mucous membranes), it is unlikely that any of the gastrointestinal parasites identified in this survey are currently responsible for a high incidence of mortality in the KZN wild dog population. In most situations internal parasites are not considered to be a common cause of mortality among free-ranging canids; however, they can contribute to increased morbidity and indirectly affect survival by decreasing host immune system function, increasing host susceptibility to predation, and reducing the fitness of an infected host (Scott 1988).

The level of exposure to *E. canis* organisms, as evidenced by seropositivity, was found to be high at 83%. All immobilized animals had some degree of tick infestation, and although the various species of ticks harboured by immobilized wild dogs were not identified, *E. canis* is transmitted by the *Rhipicephalus sanguineus* tick (Neer 1998). Despite greater than 80% seroprevalence to *E. canis* and the fact that all immobilized wild dogs underwent complete physical examinations, there were no observed clinical signs of ehrlichiosis (e.g. fever, petechiation of or pale mucous membranes, lameness, peripheral lymphadenopathy). Even the three animals with *E. canis* morulae present in circulating blood monocytes, a finding which is usually consistent with active infection (Neer 1998), had no externally visible clinical manifestations of disease. This finding is either due to true absence of disease due to *E. canis* in these animals, or else clinical signs may have been too subtle to detect based on physical examination

alone. In a study by van Heerden (1979), captive wild dogs experimentally infected with *E. canis* did experience clinical signs of ehrlichiosis, although the symptoms were less severe than those observed in domestic dog controls. Under captive experimental conditions animals are generally more susceptible to disease due to an artificially high pathogen challenge and/or due to immunosuppression secondary to the stress of captivity (Funk *et al.* 2001). It has been hypothesized that sub-clinical infectious disease in African wild dogs may be more likely to manifest itself and cause illness in animals experiencing high levels of stress (Burrows *et al.* 1995).

Examination of blood smears revealed an apparent prevalence of 0% for intra-erythrocytic *B. canis* trophozoites. This finding is not surprising, as *B. canis* infection has infrequently been reported in the African wild dog, trophozoites are rarely found on blood smears of asymptomatic carriers, and even clinically ill domestic dogs often experience low levels of parasitaemia (Taboada & Lobetti 2006). Another potential explanation for the low prevalence is that *B. canis* is not endemic in the *Haemaphysalis elliptica* (=*Haemaphysalis leachii*) population in KZN and thus the exposure rate for African wild dogs is low. However, this scenario is unlikely as the prevalence of clinical babesiosis in domestic dogs in KZN is reported to be very high (D. Baxter, H. Kohrs & T. Viljoen, pers. comm., 2006).

Van Heerden (1980) demonstrated that although *B. canis* could be experimentally transmitted to captive African wild dogs and that these animals subsequently exhibited circulating trophozoites in their erythrocytes, clinical signs of babesiosis did not develop. A single case report by Colly & Nesbit (1992) describes a severe clinical case of babesiosis in a captive African wild dog which subsequently died secondary to infection. However, the authors hypothesized that the development of clinical disease was secondary to immunosuppression and that *B. canis* is likely not pathogenic in wild dogs under non-captive circumstances (Colly & Nesbit 1992). It is probable that African wild dogs evolved concurrently with *B. canis* and other internal parasites as symbiotic pathogens endemic in the environment, and thus, although they do experience active infection, clinical disease rarely develops. Alternatively, wild dogs may simply not be susceptible to disease caused by these organisms.

Although this study did not demonstrate direct evidence that ehrlichiosis, babesiosis, or the gastrointestinal parasites evaluated currently pose a substantial disease threat to the survival of the KZN African wild dog population, any of these agents may become a more serious problem in the future if wild dogs are increasingly immunocompromised by other infectious diseases, trauma, stress, or inbreeding depression (Spalding & Forrester 1993; Funk *et al.* 2001). Moreover, since smaller isolated populations are more vulnerable to disease-related mortality than are larger populations (Spalding & Forrester 1993) and the majority of wild dog subpopulations in South Africa are relatively small, infectious disease has the potential to play an important role in long-term persistence. Ongoing monitoring and disease surveillance is necessary for the KZN African wild dog population to detect any emerging parasite-related mortality and to more precisely determine the effects of internal parasitism on future population viability.

ACKNOWLEDGEMENTS

We thank the management of Hluhluwe-iMfolozi Park, the Isimangaliso (Greater St Lucia) Wetland Park Authority, Mkhuze Game Reserve and Thanda Private Game Reserve for permission to conduct this research and for logistical support. We also thank the Ezemvelo-KZN Wildlife Game Capture Unit for use of darting and laboratory equipment. Furthermore, we thank Moritz van Vuuren and the laboratory at the Department of Veterinary Tropical Diseases, Onderstepoort, for conducting the serology for this project, and Darryl Baxter, Heinz Kohrs and Trevor Viljoen, private practice veterinarians who contributed information on disease prevalence in domestic dogs in KZN. Finally, we wish to thank Sboniso Zwane and Thadaigh Baggallay for assistance with field monitoring. This project was supported by the National Geographic Society Conservation Trust (Grant Number C91-06) and the Conservation Medicine Small Research Grants Programme at Murdoch University.

REFERENCES

BOWMAN, D.D. 1999. Helminths. In: D.D. Bowman & R.C. Lynn (Eds), Georgis' parasitology for veterinarians, 7th edn (pp. 109–234). W.B. Saunders Company, Philadelphia.

BURROWS, R., HOFER, H. & EAST, M.L. 1995. Population dynamics, intervention and survival in African wild dogs (*Lycaon pictus*). *Proc. Roy. Soci. Lond. B* 262: 235–245.

COLLY, L.P. & NESBIT, J.W. 1992. Fatal acute babesiosis

in a juvenile wild dog (*Lycaon pictus*). *J. S. Afr. Vet. Med. Assoc.* 63: 36–38.

DAVIES-MOSTERT, H.T., MILLS, M.G.L. & MACDONALD, D.W. 2009. A critical assessment of South Africa's managed metapopulation recovery strategy for African wild dogs. In: M.W. Hayward & M.J. Somers (Eds), Reintroduction of top-order predators. Wiley-Blackwell Publishers, London.

DUBEY, J.P. & ODENING, K. 2001. Toxoplasmosis and related infections. In: W.M. Samuel, M.J. Pybus & A.A. Kocan (Eds), Parasitic diseases of wild mammals, 2nd edn (pp. 478–519). Iowa State Press, Ames.

DUSZYNSKI, D.W. & UPTON, S.J. 2001. *Cyclospora, Eimeria, Isospora* and *Cryptosporidium* spp. In: W.M. Samuel, M.J. Pybus & A.A. Kocan (Eds), Parasitic diseases of wild mammals, 2nd edn (pp. 416–459). Iowa State Press, Ames.

FUNK, S.M., FIORELLO, C.V., CLEAVELAND, S. & GOMPPER, M.E. 2001. The role of disease in carnivore ecology and conservation. In: J.L. Gittleman, S.M. Funk, D.W. MacDonald & R.K. Wayne (Eds), Carnivore conservation (pp. 443–466). Cambridge University Press, Cambridge.

KENNEDY-STOSKOPF, S. 2003. Canidae. In: M.E. Fowler & R.E. Miller (Eds), Zoo and wild animal medicine, 5th edn (pp. 482–491). W.B. Saunders Company, Saint Louis.

LITTLE, S.E. 2007. An overview of cestode infections in dogs and cats. *Supp. Vet. Med.* 3: 15–17.

NEER, T.M. 1998. Ehrlichiosis. In: C.E. Green (Ed.), Infectious diseases of the dog and cat, 2nd edn (pp. 139–147), W.B. Saunders Company, Philadelphia.

SCOTT, M.E. 1988. The impacts of infection and disease on animal populations: implications for conservation biology. *Conserv. Biol.* 2: 40–56.

SPALDING, M.G. & FORRESTER, D.J. 1993. Disease monitoring of free-ranging and released wildlife. *J. Zoo Wildl. Med.* 24: 271–280.

TABOADA, J. & LOBETTI, R. 2006. Babesiosis. In: C.E. Greene (Ed.), Infectious diseases of the dog and cat, 3rd edn (pp. 722–736). Elsevier Publishing Inc., Saint Louis.

VAN HEERDEN, J. 1979. The transmission of canine ehrlichiosis to the wild dog *Lycaon pictus* (Temminck) and black-backed jackal *Canis mesomelas* (Schreber). *J. S. Afr. Vet. Med. Assoc.* 50: 245–248.

VAN HEERDEN, J. 1980. The transmission of *Babesia canis* to the wild dog *Lycaon pictus* (Temminck) and black-backed jackal *Canis mesomelas* (Schreber). *J. S. Afr. Vet. Med. Assoc.* 51: 119–120.

VAN HEERDEN, J., MILLS, M.J.L., VAN VUUREN, M.J., KELLY, P.J. & DREYER, M.J. 1995. An investigation into the health status and diseases of wild dogs (*Lycaon pictus*) in the Kruger National Park. *J. S. Afr. Vet. Med. Assoc.* 66: 18–27.

VAN VUUREN, M. 1990. Serological studies of bovine respiratory syncytial virus in feedlot cattle in South Africa. *J. S. Afr. Vet. Med. Assoc.* 4: 168–169.

WOODROFFE, R., McNUTT, J.W. & MILLS, M.G.L. 2004. African wild dog. In: C. Sillero-Zubiri & D.W. MacDonald (Eds), Foxes, wolves, jackals and dogs: status survey and conservation action plan, 2nd edn (pp. 174–183). IUCN, Gland.

ZAJAC, A.M. 1994. Fecal examination in the diagnosis of parasitism. In: M.W. Sloss, R.L. Kemp & A.M. Zajac (Eds), Veterinary clinical parasitology, 6th edn (pp. 3–93). Iowa State Press, Ames.

Corresponding Editor: M.J. Somers