



RESEARCH COMMUNICATION

Seroepidemiological survey of sympatric domestic and wild dogs (*Lycaon pictus*) in Tsumkwe District, north-eastern Namibia

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ABSTRACT

LAURENSEN, K., VAN HEERDEN, J., STANDER, P. & VAN VUUREN, M.J. 1997. Seroepidemiological survey of sympatric domestic and wild dogs (*Lycaon pictus*) in Tsumkwe District, north-eastern Namibia. *Onderstepoort Journal of Veterinary Research*, 64:313–316

Disease is a potential threat to many endangered populations and may originate from sympatric domestic species. This paper describes a cross-sectional serological survey of canine pathogens carried out in domestic ($n = 70$) and wild dogs (*Lycaon pictus*) ($n = 6$), in Tsumkwe District, north-eastern Namibia. Evidence of past exposure to canine distemper virus, canine adenovirus and parainfluenza virus was evident in both wild and domestic dogs with this, the first, documented exposure of free-living wild dogs to canine distemper. Domestic dogs were also exposed to rabies virus, canine parvovirus and coronavirus. There was no pathogen to which wild dogs, but not domestic dogs, were exposed. With wild dogs known to be susceptible to rabies and canine distemper, these may be the greatest threat to this population of wild dogs, although some wild dogs can clearly survive infection with canine distemper.

Keywords: Canine pathogens, domestic dogs, *Lycaon pictus*, seroepidemiological survey, sympatric, virus, wild dogs

INTRODUCTION

Periodic outbreaks of infectious disease are a critical ecological and selective pressure acting on free-ranging populations and are a stochastic factor which can drive endangered populations to extinction. Basic epidemiological information is, however, lacking for most pathogens, but it is unlikely that endangered populations, almost by definition, are ever above that

of the threshold population size that allow persistence of many pathogens (Anderson & May 1979). Nevertheless, if a pathogen has a generalized host range it may be sustained in a mixed-species population, which may include domestic species (McCallum & Dobson 1995). Therefore epidemiological studies of domestic populations may be required to understand the implication of infectious disease dynamics for endangered species.

The African wild dog (*Lycaon pictus*) is one of the most endangered carnivores in Africa, now numbering fewer than 5 000 individuals, and its numbers are declining throughout its range (Fanshawe, Frame & Ginsberg 1991). Potentially viable populations of more than 100 animals occur in only six countries (Fanshawe *et al.* 1991). Disease is thought to have played a central role in the decline of this species in some areas (Schaller 1972; Fanshawe *et al.* 1991), with rabies (Gascoyne, Laurenson, Lelo & Borne 1993; Alexander, Smith, Macharia & King 1993), canine distemper (Schaller 1972; Malcolm 1979; Alexander & Appel 1994) and anthrax (Turnbull, Bell,

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Saigawa, Munyenyembem, Mulenga & Makal 1991; Creel, Creel, Matovelo, Mtambo, Batamuzi & Cooper 1995) being identified as possible causes of mortality.

The north-eastern part of Namibia, comprising the communal lands of the Ju'/hoan San in Tsumkwe District, and Hereroland and the Kuadom Game Reserve, contains the last potentially viable population of wild dogs in Namibia, with an estimated 242–1 235 wild dogs (95% confidence interval, Stander, Ghau, Tsisaba & Txoma 1995). Domestic dogs live scattered throughout the area (0,027 km²) and could act as a reservoir for canine diseases. This study aimed to identify which canine pathogens occurred in this area—and were therefore a potential risk to wild dogs—by conducting a seroepidemiological survey of domestic dogs and of a limited number of sympatric wild dogs.

MATERIALS AND METHODS

Tsumkwe District, an area of 4 868 km², is situated in Otjozondjupa Region, north-eastern Namibia, adjacent to the Botswana border. The Kaudom Game Reserve to the north is part of the same continuous ecosystem of Kalahari sand and woodland interspersed by salt pans.

Villages in the district were visited between 10 December 1993 and 20 January 1994. The town of Tsumkwe was not included in the survey. Where possible, up to 20 ml of blood was taken from the cephalic or tarsal vein of all adult dogs (> 5 months of age) in the village. 2,5 ml of blood was immediately placed in anticoagulant (EDTA, Teklab UK) while the remaining blood was allowed to clot. After separation, serum was extracted, frozen and kept at -10°C until shipped on dry ice to the laboratory. Blood smears were prepared from the unclotted blood, air dried, fixed and stained before being examined under magnification.

Dog condition was scored on a scale of 1–5, with a score of 5 corresponding to a dog in good condition with no ribs showing, but not obese. The age of dogs was assessed by questioning the owners and examining the tooth wear. The age distribution was biased towards young dogs with 46% ($n = 94$) of dogs aged 1 year or less (Laurenson, Esterhuysen, Stander & Van Heerden 1997). In addition, one dog owner in each village was asked a series of questions about the dog. The dog population in the 28 villages visited totalled 102 dogs, a mean of 0,24 dogs per adult. Dogs were kept for herding (67%, $n = 27$ owners), guarding (37%), companionship (26%) and hunting (11%).

Six wild dogs were darted with 1 ml disposable darts (Pneu Dart Inc.) containing 70 mg of zoletil (Zoletil, Virbac) and 30 mg of xylazine (Rompun, Bayer). The

wild dogs were all adults and came from three packs. Two wild dogs were still alive 2,5 and 3 years after they had been darted, one wild dog survived for 17 months before it was found dead in a snare, and one dog's destiny is unknown as the collar failed. Two dogs were translocated elsewhere.

Blood samples were taken from the cephalic or tarsal vein and allowed to clot. Serum was drawn off and stored at -20°C until tested. A liquid-phase blocking ELISA was used to test serum for rabies antibodies (Esterhuysen, Thomson & Prehaud 1995). Titres were calculated as the dilution at which 50% of the maximal optical density was inhibited, and presented as log (10) reciprocal titres. Antibodies to parvovirus, rotavirus, parainfluenza virus, adenovirus and canine distemper virus were determined by the use of an indirect fluorescent antibody technique. Specimens were screened at a serum dilution of 1:20. The parvovirus strain used in the preparation of the antigen substrate slides was a field strain (P. Howell, Department of Veterinary Tropical Diseases, Faculty of Veterinary Science, University of Pretoria); the coronavirus strain WSU 79-1683 (3) (American Type Culture Collection {ATCC}, 12301 Parklawn Drive, Rockville, 20852, USA) and the rotavirus strain, a bovine field strain (V. Da Costa Mendes, Department of Infectious Diseases and Public Health, Faculty of Veterinary Science, Medical University of Southern Africa). The parainfluenza virus, strain 78-238, and canine adenovirus, strain Toronto A 26/61, were obtained from ATCC. Canine distemper antibodies were detected with a strain of measles virus (Department of Virology, Faculty of Medicine, Medical University of Southern Africa). Canine parvovirus and feline enteric coronavirus were propagated in Crandell feline kidney cells. Parainfluenza virus was grown in Vero cells and canine adenovirus in primary canine foetal kidney cells. Measles virus was grown in Hep-2 cells.

Standard errors of seroprevalence were calculated by the use of the angular transformation, which stabilizes the variance in proportion to the mean for binomial data. With this transformation, the standard error for zero seroprevalence = $(\sin [1,96 \sqrt{(821/N)}])^2 / 1,96$ and the upper 95% confidence limit = 1,96 (SE).

RESULTS

Well over half of the domestic dogs tested ($n = 70$) had been exposed to canine parainfluenza virus and canine adenovirus, whereas nearly half had been exposed to canine parvovirus and canine distemper virus (Table 1). Positive titres for rabies antibodies were found in 30% domestic dogs (see also Laurenson *et al.* 1997), but only a small proportion had been exposed to rotavirus or coronavirus. Wild dogs ($n = 6$) showed evidence of previous exposure to canine distemper virus, canine adenovirus and parainfluenza virus (Table 1). Although there was no evidence of

TABLE 1 Seroprevalence of selective canine pathogens in domestic and wild dogs, Tsumkwe District, Namibia

Pathogen	Domestic dogs <i>n</i> = 70	Wild dogs <i>n</i> = 6
Rabies virus	30,0 (5,8)	0 (7,7)
Canine distemper virus	44,3 (6,0)	66,7 (19,9)
Parvovirus	47,1 (6,0)	0 (7,7)
Adenovirus	64,3 (5,2)	83,3 (18,1)
Parainfluenza virus	78,6 (5,5)	83,3 (18,1)
Coronavirus	1,4 (3,2)	0 (7,7)
Rotavirus	4,3 (4,1)	0 (7,7)

exposure to rabies, parvovirus, rotavirus or coronavirus in this small sample, seroprevalence of up to 15,1% (95% confidence interval) could have occurred in the population without detection.

The mean condition score of domestic dogs was 2,9 (SE 0,15), therefore the dogs were generally underweight and had poor-quality coats. At least three dogs had clinical signs of disease. Most dogs appeared lethargic, even in the cool early morning and were generally easy to handle. Some dogs had suppurating wounds, and ticks (e.g. *Rhipicephalus* spp.) were observed on many. Dogs were fed mealie meal, milk and leftover offal from hunting, when available, as well as bushfood. Owners (*n* = 27) all reported 'dog sickness' as a cause of death, with ten specifically mentioning ocular and nasal discharge when asked to describe clinical signs, along with anorexia, shade-seeking and weakness. Four owners mentioned snake bites, and fighting and poisonous plants were mentioned as other mortality causes (once each).

DISCUSSION

This study provides evidence that canid diseases are a potential threat to the Tsumkwe District and Kaudom Game Reserve wild-dog population. Even with the limited number of wild dogs sampled, it was clear that wild dogs in this area had been exposed to a range of canine pathogens, that is canine distemper virus, canine adenovirus and canine parainfluenza virus. Wild dogs are known to be vulnerable to vaccine-induced distemper (McCormick 1983; Van Heerden, Bainbridge, Burroughs & Kriek 1989), but this study provides evidence that at least some dogs can survive infection.

The pathogens detected in the wild-dog population were also prevalent in the domestic-dog population, as were rabies, parvo and parainfluenza viruses. These pathogens were undoubtedly causing mortality, with clinical signs of canine distemper and parvovirus, described by the local people. The domestic-dog population of Tsumkwe District was in very poor condition and generally unhealthy with overt clinical

signs in some individuals. The mortality rate in this population was high, with a consequent low median age (1,5 years) and a high turnover rate of 46% each year (see Laurenson *et al.* 1997). Indeed, with 'dog sickness' being the most common reported cause of mortality, infectious diseases may be limiting or regulating the size of the population.

The density of wild dogs in the region is low at 0,019–0,031/km² (Stander, Tsusaba, Ghau, Txoma, Iui, Kagece, Nisa, Xuaq'an & Haden 1994) and it is unlikely that any of these pathogens could persist in this species alone. Indeed, there was no evidence of exposure to any pathogen in wild dogs but not domestic dogs. Domestic canids are a likely reservoir for at least some pathogens of wild dogs (Cleaveland & Dye 1995) and, although the domestic-dog density is relatively low in Tsumkwe district (0,27 dogs/km²) (Laurenson *et al.* 1997), surrounding districts have higher densities of domestic dogs from which infections might spread. Nevertheless, other wild carnivore species could be involved in the epidemiology of some of these pathogens, and further research is clearly required to elucidate how these pathogens persist, and the role of wildlife in their epidemiology.

In summary, this study has shown that a range of canine pathogens are prevalent in canids in Tsumkwe District. Wild dogs are probably most at threat from rabies and canine distemper virus as wild dogs are known to be susceptible to these pathogens. As human populations grow, contact between domestic and wild canids can only increase, thereby facilitating the transmission of pathogens between these groups.

ACKNOWLEDGEMENTS

Samples were collected with the help of Tsisaba Debe, //au, K/gau, Mark Jago and Simon Thirgood. We would like to thank Annatjie Bonthuys of the Namibian Nature Foundation for her logistical support. This project was funded by the British Ecological Society, with indirect help from Grand Canyon Spur, Windhoek, the Ministry of Environment and Tourism and WWF (US).

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