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Authors: Creel, Scott, Creel, Nancy Marusha, Munson, Linda, Sanderlin, Dane, and Appel, Max J. G.

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SEROSURVEY FOR SELECTED VIRAL DISEASES AND DEMOGRAPHY OF AFRICAN WILD DOGS IN TANZANIA

Scott Creel,[1,5] Nancy Marusha Creel,[1] Linda Munson,[2] Dane Sanderlin,[3] and Max J. G. Appel[4]

[1] Department of Biology, Montana State University, Bozeman, Montana 59717-0346, USA
[2] Department of Pathology, School of Veterinary Medicine, University of Tennessee, Knoxville, Tennessee 37901, USA
[3] Viral and Rickettsial Zoonoses Branch, Centers for Disease Control, 1600 Clifton Road, N.E., Atlanta, Georgia 30333, USA
[4] James A. Baker Institute for Animal Health, College of Veterinary Medicine, Cornell University, Ithaca, New York 14853, USA
[5] Corresponding author

ABSTRACT: African wild dogs (*Lycaon pictus*) are endangered, with only 3,000–5,000 remaining in the wild. It is believed that wild dogs are unusually vulnerable to viral diseases, particularly rabies and canine distemper (CDV). However, canine distemper has been confirmed by laboratory diagnosis in only one free-living wild dog. The 43,000 km^2 Selous Game Reserve (SGR; Tanzania) holds approximately 900 adult wild dogs. In a study area of 2,600 km^2, the population maintained high density ($\geq$1 dog/20.5 km^2) from 1991 to 1996. The population was stable, varying 18% below and 9% above the mean density over the 6-yr period. Serum samples (n = 22) collected over 3 yr showed that most individuals were exposed to CDV (59%: 95% confidence interval = 43–76% seropositive) and canine parvovirus (68%: 95% CI = 54–81% seropositive), although none were seropositive for rabies (0%: 95% CI = 0–17%). CDV titers were positively related to age, with no seropositive dogs younger than 1.9 yr. At least five of 13 dogs positive for CDV seroconverted during the study. Dogs with high CDV titers did not survive better in the years after sampling (mean survival ± SE for those that died = 638 ± 92 days,). Variation in mean litter size was inversely related to CPV exposure in the SGR and elsewhere. Annual mortality rates were low in comparison to other populations for all age classes (pups: 31 ± 8%, n = 127, yearlings: 22 ± 10%, n = 93, adults: 20 ± 6%, n = 235). Annual mortality rates fluctuated little between 1992 and 1996. These data show that wild dog populations, like those of other canids, can remain stable and demographically healthy despite exposure to CDV and CPV.

Key words: African wild dog, canine distemper, conservation, demography, *Lycaon pictus*, parvovirus, rabies, serology.

INTRODUCTION

African wild dogs (*Lycaon pictus*) are endangered, with 3,000 to 5,000 individuals remaining in the wild (Ginsberg and Macdonald, 1990; Fanshawe et al., 1991). The species' precarious status is largely due to habitat loss combined with low population density under all conditions (Fanshawe et al. 1991; Creel and Creel 1996). Several factors may limit wild dog numbers, including human persecution (Childes, 1988), interspecific competition with larger carnivores (Estes and Goddard, 1967; Malcolm, 1979; Creel and Creel, 1996), and infectious diseases (van Heerden, 1986; Gascoyne et al., 1993; van Heerden et al., 1995; Alexander et al., 1996).

Until recently, most information on wild dogs came from a single population in the Serengeti National Park (SNP, Tanzania). The SNP population declined to extinction between 1970 and 1991, with recurrent outbreaks of disease. Schaller (1972) described a fatal illness among the members of one pack that caused anorexia and weight loss, mucopurulent discharge from the eyes, staggering and myoclonal twitching. Based on these clinical signs and postmortem examination of one carcass, mortality in three packs in 1967–1968 was ascribed to canine distemper. Malcolm (1979) attributed a decline in 1971–1973 to disease, though no serological or postmortem data were collected to identify the pathogen (*cf* Burrows et al., 1994). In the late 1980's and early 1990's, the last few

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wild dogs in SNP disappeared during a rabies epizootic. Rabies viral encephalitis was confirmed in four cases by brain histology and by isolation of the rabies virus, which was typed as a variant similar to that in domestic dogs neighboring the park (Gascoyne et al., 1993; Kat et al., 1995).

Collectively, data from SNP have shown that infectious diseases can cause substantial mortality, and can contribute to a local extinction. This has led to the widespread conviction that wild dogs are "particularly sensitive to disease" (Fanshawe et al., 1991, p. 140), and that disease has played "a main role in the numerical and distributional decline of African wild dogs" (Kat et al., 1995, p. 229). However, little is known about the regulatory role of diseases in other wild dog populations. The SNP population was unusual in several ways. Population density was very low (Frame et al., 1979; Malcolm, 1979), averaging <1 dog/200 km^2 over a 20 yr period (data from sources in Burrows et al., 1994). Competition from larger carnivores was intense (Fanshawe and Fitzgibbon, 1993), and might have limited wild dog numbers (Creel and Creel, 1996). Finally, many carnivores that could harbor and transmit viral infections were common in the SNP ecosystem. Common species with known exposure to canine distemper virus (CDV) and/or rabies virus included bat-eared foxes, *Otocyon megalotis* (Maas, 1993), domestic dogs (Alexander and Appel, 1994), jackals, *Canis aureus* and *C. mesomelas* (Alexander et al., 1994, Roelke-Parker et al., 1996), spotted hyenas, *Crocuta crocuta* (Alexander et al., 1995; Haas et al., 1996), and lions, *Panthera leo* (Roelke-Parker et al., 1996). Thus, it may be misleading to generalize conclusions from SNP about the role of disease in wild dog population dynamics.

Despite close monitoring, disease-related declines were not observed in Kruger National Park (KNP, South Africa) over a period of 22 yr, or in the Selous Game Reserve (SGR, Tanzania) over a period of 6 yr (Reich, 1981; Maddock and Mills, 1994). Combining serological and demographic data, van Heerden et al. (1995, p. 18) concluded that "disease could not be incriminated as an important cause of death" in KNP. It remains possible that these populations are vulnerable to viral diseases, but have not been exposed. In KNP, extensive serological screening showed no evidence of exposure to rabies virus, CDV or parvovirus (CPV). *Bacillus anthracis,* the bacterium causing anthrax, has caused several deaths in KNP, SGR, and in Luangwa, Zambia (Turnbull et al., 1991; Creel et al., 1995; van Heerden et al., 1995).

There is no direct evidence that CPV causes mortality in free-living wild dogs, although some populations are exposed (Fuller et al., 1992), and it is reasonable to hypothesize that CPV is a factor in some pup deaths. CPV can cause substantial mortality in young wolves (Peterson and Krumenaker, 1989; Johnston et al., 1994). For wolves, CPV has not been shown to cause population declines, but may hinder recovery after a decline (Mech and Goyal 1993). Because the carcasses of wild dog pups are virtually impossible to collect, it is plausible that some juvenile mortality is due to undetected CPV, among other factors. Pup mortality can be substantial for wild dogs, with as high as 70% annual mortality in KNP (van Heerden et al., 1995). No data exist to establish what proportion is caused by CPV.

No unvaccinated wild dog has been found to have antibodies to CDV (but see results), nor has canine distemper virus ever been isolated from this species. Nonetheless, population declines have been attributed to CDV on the basis of behavioral observations (Schaller, 1972; Malcolm 1979) or concurrent outbreaks in other species (Alexander and Appel, 1994; Roelke-Parker et al., 1996). Vaccine-induced canine distemper has caused deaths in captivity (Durchfield et al., 1990; McCormick, 1983; van Heerden et al., 1989). Recently, canine distemper was rigorously confirmed as the cause of death for a free-

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living wild dog for the first time. Ten wild dogs (from a pack of 12) died over 2 wk in Botswana's Chobe National Park. One carcass was recovered, and pathology and immunohistochemistry revealed pneumonia and meningoencephalitis due to CDV as the cause of death (Alexander et al., 1996).

The role of disease in regulating wild dog populations is still poorly understood. We use the term 'regulate' to mean 'affecting numbers without implying stable equilibrium,' as in May (1985). Susceptible-infected-recovered models show that a microparasite can regulate a host population if the pathogen is highly virulent, acquired immunity is weak, or the host's intrinsic rate of increase is low (Anderson and May, 1979). At least two of these criteria (high virulence, low host rate of increase) characterize interactions between wild dogs and rabies or canine distemper. Epidemiologic models of pathogens with multiple hosts (like rabies, CDV, and CPV) show that a small, low-density population of one host is vulnerable to spill-over transmission from a high-density host that supports an enzootic infection (Grenfell and Dobson, 1995). Because wild dogs invariably live at low density, often in populations with fewer than 100 individuals (Fanshawe et al., 1991), spill-over transmission is a concern.

Thus, there are theoretical models and empirical data suggesting that disease may regulate wild dogs in some ecosystems. Canine distemper and rabies have caused severe mortality and population declines in other carnivores (Williams et al. 1988; Osterhaus et al., 1995; Roelke-Parker et al., 1996), and some authors suggest that infectious diseases have been a major factor in the continent-wide decline of wild dogs (Kat et al., 1995; Alexander et al., 1996). In contrast, Pence (1995) argued that although CPV, CDV and rabies have caused substantial mortality among coyotes (*Canis latrans*), this mortality was compensatory, and did not affect population density in the long term (Thomas et al., 1984; Guo et al., 1986). It has been suggested (D. B. Pence, pers. commun.) that there is little direct evidence that disease regulates wild dog numbers. However, differences in the population biology of coyotes and wild dogs (population density, intrinsic rates of increase, presence of dominant competitors) are likely to modify their vulnerability to epizootics. In short, current data are compatible with widely divergent views of the role of disease in wild dog population dynamics. To help resolve this divergence, we present demographic and serological data from a large wild dog population in southern Tanzania.

MATERIALS AND METHODS

Data came from eleven packs in a 2,600 km^2 area of the 43,000 km^2 Selous Game Reserve (SGR, 7°15′ to 10°30′S, 36°00′ to 38°45′E). The population was under study from June 1991 to September 1996. All wild dogs on the study site were individually recognizable by variation in their black, white and tan coats, using a photofile. Age was known for most individuals in the study because they were identified at birth dens. Age was known for only half of the individuals sampled for serology, because many blood samples were taken when a newly-located pack (with unknown adults) was radiocollared. For dogs whose age was not known, estimates were based on body size, toothwear and pelage. We classified dogs as pups (<1 yr), yearlings (1–2 yr) and adults (≥2 yr). Age-specific annual mortality was calculated (n = 235 adult-yr, 127 pup-yr and 93 yearling-yr) as $m_x = (D_x - I_x)/T_x$, where D_x is the number of individuals of age x that died or disappeared, I_x is the number of unknown immigrants of age x, and T_x is the total number of individuals of age x. This method corrects for undetected emigration, assuming that the study area is neither a net source of emigrants or a net sink for immigrants (Waser et al., 1994). To measure reproduction, 27 litters were counted at dens over a 6 yr period. Most litters were counted approximately 1 mo after birth, when puppies begin spending time above ground. A few litters in very dense thickets were counted as late as 3 mo. Two litters were counted at 5 mo, after the denning period.

Between August of 1992 and October of 1995, we collected 22 blood samples from 21 wild dogs in 12 packs that were anaesthetized for radiocollaring (Creel et al., 1997). One serum sample came from an individual outside

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TABLE 1. The proportion of adult and yearling wild dogs in the Selous Game Reserve (Tanzania) that were positive for antibodies to canine distemper (CDV), canine parvovirus (CPV) and rabies between 1992 and 1995.

Year	CDV	CPV	Rabies
1992	5/8[a]	7/8	0/7[b]
	(0.63)	(0.88)	(0.00)
1993	2/5	3/5	0/5[b]
	(0.40)	(0.60)	(0.00)
1994	1/4	3/4	0/1[b]
	(0.25)	(0.75)	(0.00)
1995	5/5	2/5	0/0[b]
	(1.00)	(0.40)	(0.00)
Total	13/22	15/22	0/13[b]
	(0.59)	(0.68)	(0.00)

[a] Number seropositive/number sampled, with proportion in parentheses.

[b] If titers <11 are considered negative for hemolyzed samples, sample sizes from 1992 to 1995 were 8, 5, 4 and 4, totalling 21.

the study site but within the reserve. Only five sampled dogs were female, because we preferred to radiocollar (larger) males. Sex-biased sampling precluded analyzing serological data independently for each sex. We used an airgun (Vario 3V, Telinject, Saugas, California, U.S.A.) to fire a lightweight dart with 55 mg of Ketamine HCl (Ketaset, Fort Dodge, Port Washington, U.S.A.) and 60 mg of Xylazine (Rompun, Bayer, Suffolk, U.K.) into the hind leg, adjusting doses ±10% for variation in estimated body mass. Blood was drawn from the lateral tarsal vein. We reversed the effects of Xylazine with 560 μg of Yohimbine (experimental, provided by M. Bush, National Zoological Park, Washington, D.C., U.S.A.) injected into the lateral tarsal vein. Dogs were able to stand within 5 to 8 min of reversal, and typically rejoined packmates within 15 min.

Blood samples were separated and frozen in liquid nitrogen in the field, and transported from the field on dry ice. All samples thawed during transport for several hours on two occasions before reaching the lab. For the methods we used, thawing can reduce antibody titers and give false negative results, so the prevalences we report should be considered minimum estimates. Serum antibodies to canine distemper were measured by a microneutralization test with a positive reaction defined by a log titer >1.0 (Appel and Robson, 1973). Sera were evaluated for parvovirus antibody (CPV-2) by hemagglutination inhibition, with a threshold titer of ≥10 IU/ml considered positive (Carmichael et al., 1980). Sera were tested for rabies serum neutralizing antibody by rapid fluorescent focus inhibition (RFFIT) with a threshold of ≥5 IU/ml considered positive (Smith et al., 1996).

Data were analyzed with parametric tests (polynomial regression, ordinary least-squares regression) after testing that assumptions were met. We used Fisher's exact test (Sokal and Rohlf, 1995) to test for differences between proportions (e.g., seroprevalence, survival rates).

RESULTS AND DISCUSSION

The density of wild dogs in northern Selous was high and stable between 1991 and 1996. Total density (including pups) ranged from 1 dog/20.5 km^2 to 1 dog/15.8 km^2, with a mean of 1 dog/17.4 km^2. Excluding pups, density ranged from 1 adult/28.6 km^2 to 1 adult/21.8 km^2, with a mean of 1 adult/26.0 km^2. The density of other large carnivores was low (1 spotted hyena/3 km^2, Creel and Creel, 1996) to moderate (1 lion/11 km^2, Creel and Creel, in 1997). Smaller canids were rare: we saw black-backed and side-striped jackals 0–5 times a year, and never saw golden jackals (*Canis aureus*) or bat-eared foxes (*Otocyon megalotis*). Civets *Civettictis civetta*; dwarf and banded mongooses (*Helogale parvula* and *Mungos mungo*) were common. We saw no domestic dogs in the reserve. We kept records of domestic dogs along the two inhabited roads to the reserve, for the first 100 km from the reserve. We recorded no dogs to the east of the reserve, and interviews with villagers confirmed that dogs were not kept. We noted dogs in all villages to the north, and government veterinary staff estimated that 900 dogs lived in the seven villages nearest the reserve. In 1995, 14% of these dogs were vaccinated for rabies. In 1996, the veterinary staff killed five domestic dogs that showed behavioral signs of distemper (twitching, mucopurulent discharge) together with 23 domestic dogs suspected to be infected.

Of the wild dogs sampled, 59% were seropositive for CDV, with 95% confidence limits (CI) of 43 to 76% (Table 1). Seropositive individuals were found in ten of

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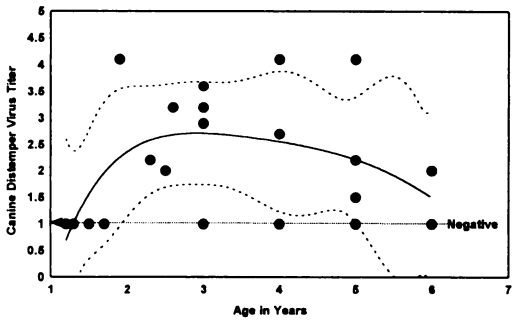

FIGURE 1. Among wild dogs in Selous Game Reserve, CDV titers increase sharply about 2-yr-old, and thereafter do not covary significantly with age. Solid line shows polynomial (third order) regression; dashed lines show 95% confidence belts for the regression.

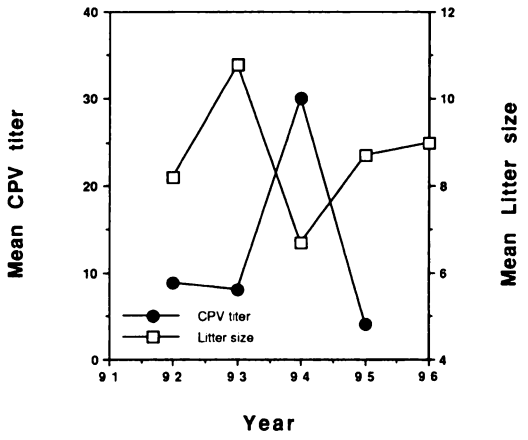

FIGURE 2. Variation in mean CPV titer and mean litter size over four years for Selous wild dogs.

12 packs, showing that exposure is widespread. Of 13 seropositive individuals, five (38%) were born during the study, and thus were known to have seroconverted while under observation. The proportion of individuals with antibodies to CDV did not vary significantly across 4 yr (Fisher's exact test: 92 versus 93, $z = 0.15$, $P = 0.88$; 93 versus 94, $z = 0.01$, $P = 0.99$; 94 versus 95, $z = 0.95$, $P = 0.34$). CDV titers were positively related to age (Fig. 1: polynomial regression, $r^2 = 0.56$, $P < 0.02$), with no antibodies in dogs <1.9-yr-old. This suggests two possible explanations, which are not mutually exclusive. First, individuals that contract canine distemper before 2-yr-old might be more likely to die. Second, exposure to CDV may be limited prior to 2-yr-old. CDV has poor survival outside of a host; it is transmitted by inhalation, which requires fairly direct contact (Appel 1987). If spotted hyenas are a source of infection (Alexander et al., 1996; Haas et al., 1996), yearlings may be exposed less frequently than adults, because they rarely fight with spotted hyenas at carcasses (S. Creel and N. Creel, unpubl. obs.). Regardless, the age-distribution of seropositivity for CDV suggests that vaccination programs, if considered, should focus on young individuals.

For 10 individuals that died during the study, post-sampling survival averaged 638 ± 92 (SE) days, and did not correlate with CDV titers (least squares regression, $P = 0.37$). Although the population was under near-daily observation, no wild dogs or other species were observed with clinical signs of canine distemper. Because five of 13 seropositive dogs were born during the study, we can exclude the possibility that seropositive dogs were simply the survivors of a CDV epidemic prior to the study. Among all sampled individuals (seropositive or negative), at least 24% were exposed to CDV during the study.

Of 22 dogs evaluated for antibodies to CPV, 68% were seropositive (Table 1: 95% CI = 54–81%). Seropositives came from 10 of 11 packs sampled, showing that exposure to CPV is widespread. CPV titers were not significantly related to age (least squares regression, $F_{1,20} = 0.25$, $P = 0.62$), and did not vary significantly among years (Table 1: Fisher exact test, all interyear pairs not significant). Pups occasionally had diarrhea, and three lethargic pups disappeared. The effect of parvovirus (if any) is most likely to be detected through high juvenile mortality or small litters at first count. Across years, mean litter size dropped by 38% when mean CPV titer peaked in 1994 (Fig. 2; regression of mean litter size on mean CPV titer, $F_{1,3} = 2.06$, $P = 0.29$, $R^2 = 0.51$).

Comparisons with two other popula-

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tions can test for effects of CPV on juvenile survival. In KNP, 0% of 43 wild dogs were seropositive for CPV, litters were large at 3 mo (11.9 pups), and juvenile survival was 30% (Maddock and Mills, 1994; van Heerden et al., 1995). In SNP, 67% of six individuals were seropositive for CPV, litters averaged 10.0 pups at first count, and juvenile survival was 39% (Fuller et al., 1992; Burrows et al., 1994). In SGR, 68% of 22 wild dogs were seropositive for CPV, litter size was 8.6 ± 0.7 (n = 27), and juvenile survival was 69% (n = 127). These data suggest that exposure to CPV might reduce litters before their emergence from the den (Fig. 3a) but that it does not cause significant mortality after the denning period (Fig. 3b). Nonetheless, high CPV seroprevalence was not associated with low recruitment of yearlings (Fig. 3c). Because factors other than CPV are likely to cause mortality during the denning period, these data are not conclusive, but suggest that CPV and pup survival should be examined further. Direct data on the cause of pup deaths are needed, but will be difficult to obtain.

There was no evidence that wild dogs in SGR have been exposed to rabies, as all were seronegative (Table 1). No wild dogs or other species in SGR were observed with clinical signs of rabies, despite 6 yr of close observation. Because rabies is normally acute and fatal, it might go undetected by serological survey, though high rabies seroprevalence has been reported from wild dogs in SNP (Gascoyne et al., 1993). Rabies is known to occur in domestic dogs in and around the city of Morogoro, <70 km from the study area (Magembe, 1985).

Despite their exposure to CDV and CPV—two of the viruses that could affect wild dog numbers—mortality in SGR is low (Table 2). For adults, annual mortality in SGR (20%) is lower than in KNP (31%: van Heerden et al., 1995) and Okavango (ONP, Botswana, 42%; McNutt, 1995), where large and stable populations are found. Among yearlings, mortality in SGR (22%) is similar to that in ONP (23%; McNutt, 1995) and lower than that in KNP (36%; van Heerden et al., 1995). The mortality of pups in SGR (31%) is particularly low in comparison to other populations (KNP 70%; ONP 64%; SNP 61%). Some of the variation in pup mortality is probably related to the age at which pups can be counted. In SGR, dens are generally in dense thickets, so the first accurate count is occasionally as late as 3 to 5 mo. Consequently, some mortality is incorporated in the initial litter size. This may partially explain why initial litter sizes in SGR (8.6 pups) are lower than in some other populations (KNP, 11.9 pups; SNP, 10.0 pups; Okavango, 8.4 pups). However, variation in pup survival is clearly meaningful, because there are large differences among populations in the mean number of pups raised to 1 yr (Fig. 3c), in the opposite direction to differences in initial litter size.

Survival rates for wild dogs in SGR did not vary significantly between 1992 and 1995 (Fig. 4: Fisher exact test: 92 versus 93, z = 1.07, P = 0.28; 93 versus 94, z = 0.007, P = 0.99; 94 versus 95, z = 1.21, P = 0.22). Annual survival rates did not covary with seroprevalence of antibodies to CDV, CPV, or rabies (Table 1). Population density also varied little, with a maximum of 1 dog/15.8 km^2 in 1993 and a minimum of 1 dog/20.5 km^2 in 1992. These values equate to deviations of 18% below and 9% above the mean density.

Our data show that a wild dog population can remain demographically healthy and maintain a high population density, despite exposure to CDV and CPV. This scenario differs from that reported for wild dogs in SNP, and tends to contradict the hypothesis that wild dogs are unusually susceptible to diseases (Fanshawe et al., 1991; Alexander et al., 1996). Only 4% of 45 deaths of known cause in SGR were due to disease. Similarly, 5% of deaths in KNP were attributed to disease (van Heerden et al., 1995). In the SNP ecosystem, population declines attributed to viral diseases have occurred among lions, jackals

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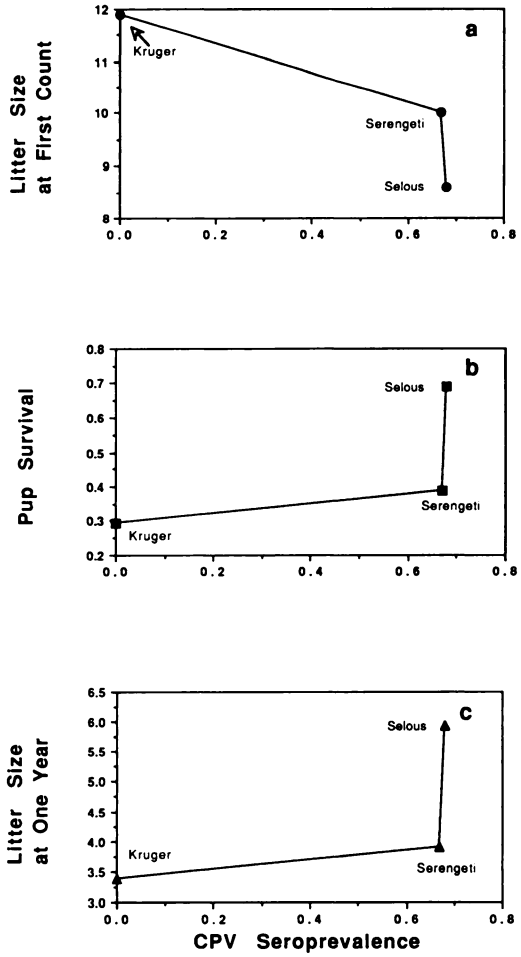

FIGURE 3. Litter size at first count and juvenile survival in relation to seroprevalence of canine parvovirus in three wild dog populations: (a) litter size, (b) juvenile survival, (c) litter size at one year.

and bat-eared foxes (Moehlman, 1983; Maas, 1993; Roelke-Parker 1996), and individual hyenas have died of canine distemper (Haas et al., 1996). Collectively, it appears that the effect of viral diseases on wild dogs is similar to their impact on sympatric carnivores. Wild dogs are not apparently more vulnerable than other carnivores in a physiological sense. Their vulnerability to extinction by spill-over transmission is due to low population density, which also affects their vulnerability to extinction by factors unrelated to disease, such as competition and predation (Ginsberg et al., 1995; Creel and Creel, 1996).

TABLE 2. Annual rates of survival for wild dogs in the Selous Game Reserve (Tanzania) between 1991 and 1996.

	Number of individuals			Proportion surviving		
Age class	Female	Male	Both sexes	Female	Male	Both sexes
Juveniles	62	65	127	0.69	0.68	0.69
Yearlings	43	50	93	0.84	0.74	0.78
Adults	106	129	235	0.79	0.81	0.80

CDV is thought to be a particularly important threat (Alexander and Appel, 1994; Alexander et al., 1996), but has previously been detected only once in a free-living population (Alexander et al., 1996). Despite heavy exposure to CDV, survival rates in SGR are higher than in other populations. However, the variant of CDV in SGR has not been identified, and a more pathogenic variant could produce markedly different results. The apparently low pathogenicity of CDV in SGR suggests that this variant might be promising for the development of vaccines for wild dogs that are less likely to induce the disease (Durchfield et al., 1980; McCormick, 1983; van Heerden et al., 1989). Given severe population declines due to CDV and closely related morbilliviruses in other carnivores (Appel and Summers, 1995; Osterhaus 1995; Roelke-Parker et al., 1996),

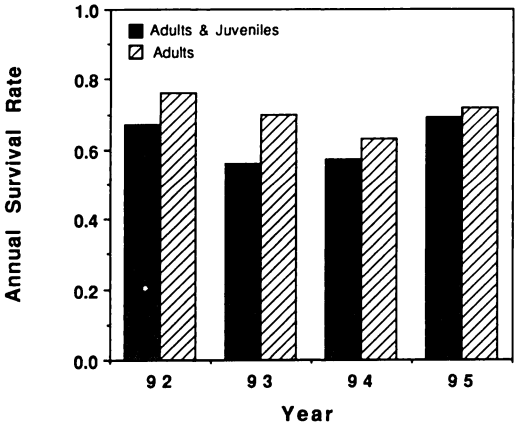

FIGURE 4. Variation in survival rates of wild dogs in Selous Game Reserve between 1992 and 1995.

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it would be unwise to conclude that wild dogs are not endangered by viral diseases. Theoretical models suggest that species living at low density (like wild dogs) are likely to be endpoints of infection transmitted by other species, and this increases their risk of local extinction (Grenfell and Dobson, 1995). Wild dogs' low density may increase the risk that an epizootic causes local extinction, but current empirical data do not show that viral diseases have a greater impact on wild dogs than on sympatric carnivores.

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