

Rabies among African wild dogs (*Lycaon pictus*) in the Masai Mara, Kenya

P. W. Kat, K. A. Alexander, J. S. Smith,
J. D. Richardson, L. Munson

Abstract. A pack of African wild dogs (*Lycaon pictus*) ranging to the north of the Masai Mara National Reserve in southwestern Kenya was monitored from 1988 to 1989. During a 6-week period (August 1-September 13, 1989), 21 of 23 members of this pack died. Seven carcasses were retrieved, of which 4 were suitable for necropsy and histopathologic examination. Gross findings varied among individuals and included multiple bite wounds, synovitis, lymphadenopathy, submandibular, cervical, and vocal cord edema, blood in bronchi, bronchioles, stomach, and intestine, and anteroventral lung lobe consolidation. Histologic examination of 2 available brain samples revealed nonsuppurative encephalitis with eosinophilic intracytoplasmic inclusions (Negri bodies). An additional brain sample tested positive for rabies via a fluorescent antibody test. Other histologic features included severe suppurative bronchopneumonia, myocarditis, and lymphoid depletion of the lymph nodes, tonsils, and spleen. A 304-base pair (bp) nucleotide sequence from the N gene and a 310-bp sequence from the G gene from rabies isolates of 4 wild dogs indicated that infection was with a rabies variant common among domestic dogs in Kenya and Tanzania.

The African wild dog (*Lycaon pictus*) is one of the world's most endangered large carnivores; there may be as few as 2,000 adults remaining in sub-Saharan Africa.¹⁵ Surveys indicate that viable populations currently exist in only 6 of 34 African countries that historically comprised the species' geographic range.¹⁵ Factors such as human persecution, habitat loss, competition with other carnivores, and reduced prey availability have been proposed to account for the decline,^{10,15,17} but increasingly, disease is thought to play a major role in the numerical and distributional decline of the African wild dog.^{1,2} Disease-related mortality among wild dogs has been attributed to canine distemper,^{7,13,29,41} anthrax,³⁵ and ehrlichiosis.^{12,28} In addition, a variety of other pathogens have been recorded from free-ranging and captive wild dogs, including *Hepatozoon canis*,³⁶ *Toxoplasma gondii*,³⁶ *Encephalitozoon*,³⁷ *Eimeria* sp., *Isospora* sp., *Demodex* sp., *Sarcoptes* sp., *Ancylostoma* sp., and *Taenia* sp. (P. W. Kat and J. D. Richardson, unpublished data). In general, the effects of these pathogens on wild dogs are unknown. We have recorded serum antibodies to a va-

riety of pathogens, including canine parvovirus 2, canine adenovirus 1, canine coronavirus, and canine herpesvirus from wild dogs in eastern Africa (K. A. Alexander and P. W. Kat, unpublished data). Again, the consequences of such infections are unknown because all dogs were healthy when sampled and remained so for a considerable time afterwards.

We describe here the earliest outbreak of rabies documented among African wild dogs. The outbreak occurred in the Aitong pack, a group of wild dogs that ranged over an area of at least 650 km² to the north of the Masai Mara National Reserve in Kenya.¹⁷ The outbreak, which lasted August-September 1989, resulted in the deaths of 21 of 23 individuals in the pack. Rabies outbreaks in free-ranging canids have been reported for red and gray foxes (*Vulpes vulpes* and *Urocyon cinereoargenteus*),³⁹ bat-eared foxes (*Otocyon megalotis*),²² Blanford's foxes (*Vulpes cana*), arctic foxes (*Alopex lagopus*),³⁰ wolves (*Canis lupus*),⁹ jackals (*Canis mesomelas*),¹⁶ and Simien wolves (*Canis simiensis*).²³ Such canids often account for a significant percentage of reported rabies cases. For example, in Zimbabwe, jackals were responsible for 22% of reported cases,⁸ whereas in Europe, red foxes constituted 77% of 13,648 rabies reports in 1991.⁴⁰ Invariably, high mortality was associated with these outbreaks. The following report further documents the impact rabies has on free-ranging canid demography.

Materials and methods

Pack history and observations. The Aitong pack was established in 1985 when a group of 9 adults (7 males, 2 fe-

From the Department of Veterinary Pathology, Microbiology and Immunology, School of Veterinary Medicine, University of California, Davis, CA 95616 (Kat, Alexander), the Rabies Section, Viral and Rickettsial Zoonoses Branch, M/S G-33, Centers for Disease Control, 1600 Clifton Rd, Atlanta, GA 30333 (Smith), Mpaka Road Veterinary Clinic, PO Box 14610, Nairobi, Kenya (Richardson), and the Department of Pathobiology, School of Veterinary Medicine, University of Tennessee, PO Box 1071, Knoxville, TN 37901 (Munson).

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males) appeared in the Masai Mara area.⁵ The pack was observed infrequently from 1985 to 1988, but 2 of 17 dogs born in 1986 were immobilized in April 1987 for blood sampling. In September 1988, permission was granted by the Kenya Wildlife Conservation and Management Department to radiocollar 2 of 18 dogs born in 1987. These radiocollared dogs made it possible to monitor the pack regularly,^{17,18} and considerable information was collected, including home-range size, habitat preference, prey preference and consumption rates, and contact rates with domestic dogs. Monitoring of the Aitong pack after September 1988 included monthly aerial telemetry flights followed by ground observations and a period of intensive study from June 17, 1989 to October 2, 1989.¹⁷ Ground observations consisted of documentation of wild dog activities, hunting success rates, and interactions with competitors, scavengers, domestic animals, and humans (Masai tribespeople), and behavioral interactions among pack members. Two dogs born in 1988 were radiocollared in June 1989, and 3 dogs in the Aitong Pack were vaccinated with a killed-virus rabies vaccine. One of these 3 dogs survived the rabies outbreak.

Necropsy and histopathology. Carcasses collected from the field were either given a complete necropsy on site or transported to a veterinary clinic in Nairobi. Necropsy procedure involved taking body weights and measurements and a complete external examination for wounds and/or signs of trauma and then a standardized gross examination of all internal organs. Tissues sampled for histopathology included brain, lung, spleen, lymph nodes, tonsils, salivary glands, liver, kidney, larynx, skeletal muscle, heart muscle, colon, stomach, small intestine, testis, prostate, diaphragm, adipose tissue, and skin (around puncture wounds). The types of tissues sampled and the quality of the necropsy were dependent upon the condition of the carcass; in several cases, carcasses were extensively decomposed and/or partially destroyed by scavengers such as vultures.

Rabies diagnosis and virus genetic analysis. Rabies diagnosis was conducted by immunofluorescent antibody (IFA) test performed at the National Veterinary Laboratory, Kabete, Kenya, and by histopathologic analysis performed at the Smithsonian Institution, National Zoological Park, Washington, DC.

Formalin-fixed, paraffin-embedded brain material of 2 Kenyan wild dogs and the salivary gland of a third wild dog was available for rabies viral genetic analysis. A virus isolate from a Tanzanian wild dog collected from the Serengeti National Park¹⁹ was available in the Centers for Disease Control and Prevention rabies sample bank. For the formalin-fixed brain material, tissues were disrupted with a lysis buffer containing proteinase K.²⁶ Rabies viral RNA was extracted from brain material by hypotonic lysis of cells, phenol/chloroform extraction, and alcohol precipitation.³¹ Primers 10g (5' CTA-CAATGGATGCCGAC, N gene) and 93g-5 (5' ATTTA-CACGATACCAGACAA, G gene) were used for reverse transcription,³¹ and the cDNA was amplified by polymerase chain reaction (PCR) with primers 10g and 304 (5' TTGAC-GAAGATCTTGCTCAT, N gene) and primers 93g-5 and 989-3 (5' CTGAGACGTCTGAAACTCAC, G gene) (Lin and J. S. Smith, unpublished data). The PCR product was directly sequenced.³¹ For the N gene, a 300-base pair (bp)

region between nucleotides 1,180 and 1,480 (base pair identification according to the Pasteur virus sequence³¹) was sequenced, which included 240 bp of the N gene and 60 bp of an intergenic nontranslated region. For the G gene, a 290-bp region between nucleotides 3,446 and 3,736 was sequenced. Wild dog rabies virus nucleotide sequences were aligned with sequences from domestic dog rabies virus isolates collected from a variety of African localities and with the sequence of a southern African yellow mongoose (*Cynictis penicillata*) rabies virus isolate (Tables 1, 2).^{4,32}

Results

Behavioral observations

On June 17, 1989, the pack consisted of 30 dogs (4 adults, 7 dogs from the 1987 litter, and 19 dogs from the 1988 litter). One of 2 reproducing adult females previously with the pack was missing, and there had been a change of dominance among the males. The previous alpha male was limping, and the beta male had assumed dominant status. The pack was observed for 3 days before they moved into hills to the north of Aitong and were observed again until July 19. On that date, the beta male was missing, and the alpha male had a number of bite wounds on his head and muzzle. Over the next 10 days, the alpha male became progressively disoriented, aphagic, and uncoordinated and stopped feeding on July 29. On August 1, the alpha male was found alone about 3 km from the pack. He was emaciated, restless, disoriented, and ataxic with particular loss of control of the hind limbs. He was observed until dusk and could not be found the next day despite intensive searches in the area.

On August 3, all remaining dogs (6 males, 1 female) in the 1987 cohort left the pack. They were seen on September 13 near Keekorok Lodge (45 km to the southeast) and subsequently moved across the border into Tanzania.

The last surviving adult male was attacked on August 6 by dogs in the 1988 cohort and driven from the pack. He had been listless and disoriented for the previous 2 days but bit several of his attackers during the incident. He died during the night, and his carcass was flown to Nairobi for necropsy on August 8.

Beginning on August 14 and during the subsequent 30 days, the last adult female and 17 dogs of the 1988 cohort died. Two 1988 dogs survived, including 1 dog that had been vaccinated for rabies.

Clinical observations

The clinical signs observed among the wild dogs were similar in all animals, although there were variations in degree. All observed cases were paralytic and no furious symptoms were observed. The typical symptoms included loss of appetite and dull mentation, disorientation, stiff gait and ataxia, restlessness,

Table 1. A 300-bp nucleotide sequence of the rabies virus N gene in African wild dogs (AWD), a domestic dog from Nyeri in Kenya (DDKE), a domestic dog from Arusha in Tanzania (DDTZ), a domestic dog from Durban in South Africa (DDSA), and a yellow mongoose from Carnarvon in South Africa (YMSA).

	10	20	30	40	50	60	70	80
AWD	AACGGCTGAG	CTAACAAAAGA	CTGACGTGGC	ATTGGCAGAT	GATGGAAGT	TCAATTCTGA	CGATGAGGAC	TACTTCTCCG
DDKE	-----	-----	-----	-----	-----	-----	-----	-----
DDTZ	G-----	-----	-----	-----	-----	-----	-----	-----
DDSA	GG----A	--G----	-----	-----	-----	---C---	-----	-----
YMSA	GG-T-C-A	T-----	---ACAAT	-C-----	-----	---C-C--	--C----	-----G-
	90	100	110	120	130	140	150	160
AWD	GTGAAACCAG	AAGTCCAGAA	GCTGTCTATA	CCAGGATAAT	GATGAATGGA	GGTCGACTAA	AGAGATCGCA	TATACGGAGA
DDKE	-----	-----	-----	-----	-----	-----	-----	-----
DDTZ	-----	-----	-----	-----	-----	-----	-----	-----
DDSA	-----	-----	-----	-----	-----	-----	-----	-----
YMSA	-----	--C-T--	-----	-T-GA-C--	---C---	-----	---A--	C-A----
	170	180	190	200	210	220	230	240
AWD	TATGTCTCAG	TCAGTTCTAA	TCATCAAGCC	CGTCCAAACT	CATTGCTGA	ATTTTAAAC	AAGACATATT	CGAGTGACTC
DDKE	-----	-----	-----	-----	-----	-----	-----	-----
DDTZ	-----	-----	---N--	---C---	-----	-----	-----	-----
DDSA	-----	-----	-----	-----	-----	G-----	-----	-----
YMSA	---T-C-	---C--	---G--	---C---	-C-T-C--	G-C----	--G-C---	-T-A--T--
	250	260	270	280	290	300		
AWD	ATAAGGAGCT	GGACAACAAG	GTGCCGGGAA	TCTATTGATT	GTGTATATCC	ATCATGAAAA		
DDKE	-----	-----	-----	-----	-----	-----		
DDTZ	-----	-----	-----G	-----	-----	-----		
DDSA	-----	-A-T--T-	-----GG	-----	-----	-----		
YMSA	G-G-AA-TT-	AA-TGG---	AC--A-AC-	---C-C-	A--G---T-	CG-----		

pica, altered voice, progressive paralysis, dehydration and emaciation, swollen faces and necks (in part related to bite wounds), bloody nasal discharge, head shaking, and sneezing.

Clinically ill individuals were often the recipients of

aggression from packmates, and most clinically ill and dead dogs had bite wounds around the face and neck. Increased mouth licking was observed from packmates towards ill individuals. Such mouth licking and biting is a submissive gesture commonly observed during

Table 2. A 290-bp nucleotide sequence of the rabies virus G gene in African wild dogs (AWD), a domestic dog from Nyeri in Kenya (DDKE), a horse from Lagos, Nigeria (HNI), and a yellow mongoose from Carnarvon in South Africa (YMSA).

	10	20	30	40	50	60	70	80
AWD	CAAACAATTT	GGTGGTGGAG	GACGAAGGAT	GCACCAACCT	GTCAGGGTTC	TCCTACATGG	AACTTAAAGT	TGGATACATC
DDKE	-----	-----	-----	-----	-----	-----	-----	-----
HNI	-----	-----	-----	-T-----	-----	---T---	-----	-----
YMSA	-----	--A----	--T-----	--T----	A--G--A--	-----	-----	C-----
	90	100	110	120	130	140	150	160
AWD	TCAGCCATAA	AGGTGAACGG	GTTCACTTGC	ACAGGCGTTG	TGACAGAGGC	TGAAACCTAC	ACTAACTTTG	TTGGTTATGT
DDKE	-----	-----	-----	-----	-----	-----	-----	-----
HNI	-----	-----	-----	-----	--G-----	-----	-----	-----
YMSA	-----	-A-A-T--	--T-G--T	---T-G-	---G---	A-----T	--A-----	-----
	170	180	190	200	210	220	230	240
AWD	CACCACCACG	TTCAAAAAGAA	AGCATTTCCG	CCCAACACCA	GATGCATGTA	GAGCCGCCTA	CAACTGGAAG	ATGGCCGGTG
DDKE	-----	-----	-----	-----	-----	-----	-----	-----
HNI	-----	-----	-----	-----	-----	---G--	-----	-----
YMSA	-----	-----	---T--	-----	-----	---G--	-----	-----A
	250	260	270	280	290			
AWD	ACCCAGATA	TGAAGAGTCT	CTACACAATC	CGTACCCTGA	CTACCATTGG			
DDKE	-----	-----	-----	-----	-----			
HNI	-----	-----	---G--	-----	-----			
YMSA	-----	-----	-----	-----	-----			

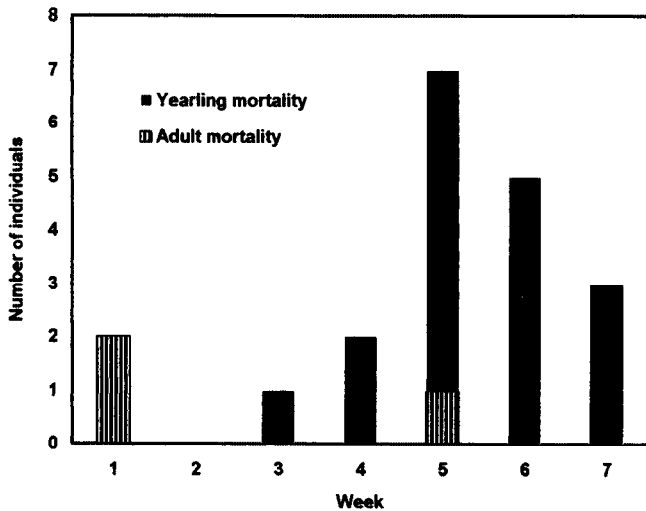


Figure 1. Temporal progression of the rabies outbreak through the Aitong pack of African wild dogs. The death of the first adult male on August 2, 1989, was taken as the point of initiation of the epidemic: the last juveniles died during the third week of September 1989.

daily greeting rituals when rank within the pack is reinforced.¹⁴ Terminally ill individuals would often seek out holes, and at least 2 individuals died underground. Throughout the outbreak, clinically ill animals appeared to make every effort to maintain normal behavior, including participation in hunts, appropriate participation in greeting rituals, and response to contact calls. Sick individuals would sometimes wander off by themselves or in small groups but would generally rejoin the pack after a few hours. Terminally ill wild dogs generally died a short distance away from the pack, as indicated by carcass locations.

The temporal progression of the outbreak through the pack is shown in Fig. 1.

Antemortem findings

Four dogs were examined antemortem. Two dogs were found comatose, and 2 other dogs were immobilized (Telazol, 2 mg/kg) for examination. Clinical findings included the symptoms listed above with additional findings of weight loss, subnormal rectal temperature, and bradycardia and/or tachycardia.

Postmortem findings

Seven carcasses were retrieved: 3 were badly decomposed, but 4 were suitable for necropsy and histopathology. Gross findings were remarkably similar among individuals and included multiple bite wounds, synovitis, lymphadenopathy, submandibular, cervical, and vocal cord edema, blood in bronchi, bronchioles, stomach, and intestine, and anteroventral lung lobe consolidation. Histopathologic observations were conducted on the following animals.

Dog 1: adult male, died August 7, 1989. This dog had been attacked by dogs in the 1988 cohort and had multiple bite wounds on the head, back, and rump. Histopathologic findings were consistent with a systemic bacterial infection and included suppurative vasculitis, valvular endocarditis, myocarditis, and lymphoid depletion of the lymph nodes, tonsils, and spleen. Brain tissue was not available for histopathology or IFA test, although a mild nonsuppurative myenteric ganglionitis was noted. Rabies was confirmed by extraction and reverse transcription of RNA from formalin-fixed salivary gland tissue and amplification of cDNA with primers specific for the rabies virus.

Dog 2: male, 1988 cohort, died September 9, 1989. This dog had been found moribund and hypothermic. The animal was emaciated and had multiple bite wounds on the head. Histopathologic findings included severe suppurative bronchopneumonia. Brain tissue was not available for histologic examination but was positive for rabies virus by IFA.

Dog 3: female, 1988 cohort, died September 10, 1989. The carcass was moderately decomposed when found. Brain stem fragments had mild perivascular aggregates of lymphocytes and neurons with pale eosinophilic intracytoplasmic inclusions (Negri bodies). The presumptive diagnosis was rabies viral encephalitis, which was confirmed by extraction and PCR amplification of rabies viral RNA from formalin-fixed brain tissue.

Dog 4: female, 1988 cohort, died September 11, 1989. This female was found comatose and hypothermic and subsequently died. The entire brain was submitted for histopathology. A moderate diffuse nonsuppurative encephalitis was noted. Many neurons, particularly in the hippocampus, contained eosinophilic intracytoplasmic inclusions (Negri bodies). The diagnosis was rabies viral encephalitis, confirmed by extraction and PCR amplification of rabies viral RNA from formalin-fixed brain tissue. Other notable histologic findings included lymphoid depletion and acute suppurative pneumonia.

Molecular genetics and serology

Wild dog rabies virus isolates from Kenya (dogs 1, 3, 4) and an isolate collected from a wild dog in Tanzania^{19,20} were fundamentally identical in nucleotide sequence to virus isolates from rabid domestic dogs in Kenya and Tanzania⁴ (Tables 1, 2). Over the 300-bp region of the nucleocapsid (N) gene, the wild dog rabies isolates were identical in sequence to domestic dog isolates collected in the Masai Mara, Nairobi, and Machakos in Kenya. Base-pair substitutions occurred at 3 sites when wild dog rabies virus isolates were compared with a domestic dog isolate from Arusha, Tanzania, and occurred at 11 sites when wild dog isolates were compared with isolates from the Republic

Table 3. Number of nucleotide substitutions in the rabies virus N gene sequence and G gene sequence among isolates from wild dogs in Kenya (AWD) and other African isolates from domestic dogs (DDKE, DDTZ, DDSA), a mongoose (YMSA), and a horse (HNI).

N gene				
	DDKE	DDTZ	DDSA	YMSA
AWD	0	3	11	64
DDKE	...	2	11	64
DDTZ	10	63
DDSA	58
G gene				
	DDKE	HNI	YMSA	
AWD	...	5	22	
DDKE	...	5	22	
HNI	23	

of South Africa (Table 3). The wild dog rabies isolate differed substantially in genetic sequence (64 substitutions, i.e., > 20% sequence difference) from an African endemic rabies virus occurring among mongooses (*Cynictis penicillata*) in southern Africa (Table 3). Over the 290-bp region of the glycoprotein (G) gene, wild dogs virus isolates were again identical in nucleotide sequence to domestic dog isolates collected in Kenya. Slight differences in nucleotide sequence (5 bp, i.e., about 2% sequence difference) occurred between the wild dog isolates and a rabies virus isolate from Nigeria (Table 3). As with the N gene sequence, the G gene sequence from wild dog isolates differed substantially from that of the endemic mongoose rabies virus (22 bp, i.e., about 8% sequence difference) (Table 3). The limited nucleotide sequence analysis of both the N and G genes of rabies viruses isolated from wild dogs revealed a variant common in areas of Kenya where rabies occurs in domestic dogs.

Discussion

Field observations and laboratory findings presented here indicate that over a period of 44 days, 21 of 23 African wild dogs from the Aitong pack died of rabies or rabies-associated diseases. Diagnosis was based on 1) the presence of encephalitis with Negri bodies in hippocampus neurons, 2) positive immunofluorescence of neurologic tissue with anti-rabies antibodies, and 3) amplification and sequencing of rabies viral RNA from infected tissues. Although the diagnosis was only confirmed in 4 dogs, the similarity of neurologic signs in other pack members suggests an analogous etiology, especially because each pack member was observed with disease symptoms before they disappeared. Other pathologic findings in this outbreak likely were secondary to rabies. The severe bite wounds from intrapack aggression could explain the findings of secondary bacterial septicemia and bacterial endo-

carditis. The anteroventral bacterial pneumonia noted in 2 dogs may have resulted from aspiration of oral or gastric contents due to pharyngeal paralysis, a common neurologic disorder in rabies.⁶

The temporal and age-class pattern of mortality in the epidemic curve suggests a mixture of both point and propagated epidemics (Fig. 1). Initial deaths occurred among the adult males, and these deaths were followed by an apparent second wave of mortalities among the yearlings and the pregnant female. This pattern can be partially interpreted within the context of age-class dependent behavior among African wild dogs. Aggression towards a pack generally elicits a defensive response from adult pack members rather than yearlings. The severe bite wounds on the head of the dominant male observed on 19 July likely constituted the introduction of rabies virus into the pack. Secondary spread to the rest of the pack could have occurred during normal social interactions between pack members and virus-shedding adults. African wild dogs are the most social of canids, exhibiting a wide diversity of social interactions.¹⁴ Mouth-licking behavior occurs frequently in association with pack greeting rituals. This type of interaction would facilitate rapid dissemination of a saliva-borne pathogen such as rabies. All observed rabies cases were paralytic in nature, and clinically ill wild dogs were often the recipient of aggression from packmates. During such aggressive encounters, the clinically ill wild dogs would reciprocate and bite packmates, potentiating the spread of the rabies virus. African wild dogs live within a complex system of social cues and appropriate responses. We postulate that the inability of clinically ill animals to respond appropriately to social cues precipitated aggression from packmates.

During this outbreak, the pack maintained cohesion. Clinically ill dogs stayed in close contact with the pack even when advanced clinical signs were evident. Small groups of dogs (2-4 individuals) would sometimes split off from the main pack. These groups did not travel far from the pack and often rejoined after a period of hours. Such small groups were sometimes led by clinically ill and therefore disoriented individuals, which were followed by apparently normal littermates. At other times, groups would split off while the pack was moving, perhaps as a result of incomplete establishment of dominance among the surviving yearlings. The pack remained entirely within its previously established home range during the outbreak, and the average daily distance travelled decreased from 10.5 km¹⁷ to 3.5 km. All individuals participated in hunting, although clinically ill dogs did not feed from the kills.

The epizootiology of rabies in Kenya²¹ and Tanzania²⁴ has been described. In Tanzania, rabies spread northwards from a reservoir near the Zambia/Malawi bor-

der, reaching the Tanzanian Mara Region (which contains the Serengeti National Park) in 1978-1979. Twenty-seven human deaths were associated with the advent of rabies in that area alone. In Kenya, rabies had been kept under control until the late 1970s; e.g., in the Narok District, which encompasses the Masai Mara area, no rabies cases were reported from 1961 to 1978. At the end of 1979, however, this situation changed drastically with the introduction and spread of rabies from Tanzania and southwestern Kenya across to the western and central parts of the country.²¹ A similarly rapid spread of rabies was reported from Zimbabwe,¹⁶ where rabies was carried 200 km by jackals through a previously uninfected area in 2 years. In both Tanzania and Kenya, however, where rural rabies vaccination is practically nonexistent, domestic dogs were the principal vectors responsible for the spread of rabies.²¹ Consistent with the reported epizootiology of rabies in Tanzania, the rabies virus isolated from a wild dog that died in the Serengeti was identified as serotype 1 (domestic dog rabies), using a panel of antinucleocapsid monoclonal antibodies.¹⁹ This classification has been corroborated and expanded by our nucleotide sequence analysis; the wild dogs that died of rabies in the Masai Mara and the Serengeti were infected with a rabies variant common among domestic dogs in Kenya and Tanzania. In agreement with the observed spread of rabies from southern Tanzania into Kenya, this variant is most closely related to southern African isolates collected in Zimbabwe and South Africa.⁴

This outbreak illustrates the susceptibility of African wild dogs to disease-mediated population fluctuations. Rapid dissemination of virus through the pack, as noted in this enzootic (Fig. 1), could also occur with other lethal viruses, such as canine distemper.¹ The large home ranges of African wild dogs¹⁷ often exceed the areas available within reserves. In the Masai Mara, the Aitong pack ranged almost entirely outside the reserve, as did 4 other packs in the area (P. W. Kat, unpublished data). Once outside protected areas, wild carnivores increasingly come into contact with domestic dogs and the diseases they transmit; in Kenya and Tanzania, 65% and 89%, respectively, of the total reported rabies cases occur in domestic dogs.²¹ Domestic dogs are primarily responsible for transmission rabies in other areas of Africa as well,^{33,38} but the virus also occurs among a wide variety of wildlife species, such as jackals, genets (*Genetta* spp.), civets, bat-eared foxes, and honey badgers (*Mellivora capensis*).^{11,33} In some cases, rabies viruses associated with wildlife transmission cycles are genetically distinct (Table 3).³² However, the rabies virus variant that caused mortality among the Masai Mara and Serengeti African wild dogs was identical in nucleotide sequence to rabies viral isolates common

among domestic dogs in Tanzania and Kenya. The large unvaccinated domestic dog population that surrounds the reserves likely maintains and transmits a number of canid pathogens.¹⁻³ Continued survival of endangered canid species sympatric with domestic dogs would benefit from a carefully conducted and maintained program of domestic dog vaccination.

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