

## Parvovirus enteritis in vaccinated juvenile bush dogs

Donald L. Janssen, DVM; Curtis R. Bartz, MBA, DVM, PhD; Mitchell Bush, DVM; Ruth H. Marchwicki, BS; Stephen J. Grate, DVM; Richard J. Montali, DVM

### SUMMARY

Parvovirus enteritis developed in 10 of 17 vaccinated juvenile bush dogs (*Speothos veneticus*) from 4 litters in a 5-month period. Nine dogs died.

The first outbreak involved 6 of 9 bush dogs from 2 litters. Each had been vaccinated with a killed feline-origin parvovirus vaccine at 11 and 14 weeks of age. The 6 affected dogs became ill at 29 weeks of age and died.

The second outbreak involved a litter of 6 bush dogs. Each had been vaccinated every 2 weeks starting at 5 weeks of age. Two were isolated from the colony at 16 weeks of age for treatment of foot sores. Three of the 4 nonisolated dogs developed parvovirus enteritis at 20 weeks of age; 2 died at 6 and 8 days, respectively, after onset of signs.

The 3rd outbreak involved a litter of 2 bush dogs. Both had been vaccinated every 2 to 3 weeks, starting at 6 weeks of age. One of these dogs became ill at 17 weeks and died 13 days later.

A litter of 6 maned wolves (*Chrysocyon brachyurus*) and a litter of 3 bush dogs were isolated from their parent colonies at 13 and 15 weeks of age, respectively. Each animal had been vaccinated weekly, beginning at 8 weeks of age, using an inactivated canine-origin parvovirus vaccine. None of the isolated animals developed the disease. Serologic testing during isolation did not reveal protective titers ( $\geq 1:80$ ) against canine parvovirus in the bush

dogs until they were 23 weeks old, whereas protective titers developed in the maned wolves when they were 14 to 18 weeks old. One hand-raised bush dog was vaccinated weekly, beginning at 8 weeks of age, and a protective titer developed by 21 weeks of age.

It was concluded that the juvenile bush dogs went through a period during which maternal antibodies interfered with immunization, yet did not protect against the disease. When the pups were isolated from the colony during this period, then vaccinated repeatedly until protective titers developed, the disease was prevented.

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CANINE PARVOVIRUS (CPV) infection has been reported worldwide in domestic dogs<sup>1</sup> and sporadically in a few species of nondomestic carnivores. Fletcher et al reported cases of CPV infection in maned wolves in 1979.<sup>2</sup> A parvovirus with different agglutination properties than those of CPV had been causing enteritis in a maned wolf colony in Krefeld Zoo, Germany.<sup>3</sup> Recently, CPV infection was reported in young raccoon dogs (*Nyctereutes procyonoides*) in fur farms in Finland.<sup>4</sup> An outbreak of CPV infection occurred in adult South American canids at the National Zoological Park in 1979.<sup>5</sup> No further outbreaks, until the ones presented in this report, had occurred here. The prevalence of CPV enteritis in adult domestic dogs and CPV myocarditis in young pups had begun to decline as more dogs had become resistant following vaccination or infection and as more breeding bitches had transferred passive protection to their pups.<sup>6</sup> The problem surfacing was that, despite vaccination, off-

From the Departments of Animal Health (Janssen, Bush) and Pathology (Montali), National Zoological Park, Smithsonian Institution, Washington, DC 20008; and the Department of Clinical Investigation and Research, Walter Reed Army Institute of Research, Washington DC 20012 (Bartz, Grate, Marchwicki).

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TABLE 1—Bush dog vaccination schedules and clinical history

Bush dog no.	Vaccination regimen* (week given)	Age disease began (week)	Duration of illness (days)	Survival (+ or -)	Comments
<b>Outbreak 1</b>					
Litter A					
BD 1	11, 14	29	6	—	No illness
BD 2	11, 14	—	—	+	
BD 3	11, 14	29	6	—	
BD 4	11, 14	—	—	+	
Litter B					
BD 5	11, 14	29	4	—	No illness
BD 6	11, 14	29	5	—	
BD 7	11, 14	29	6	—	
BD 8	11, 14	29	30	—	
BD 9	11, 14	—	—	+	
<b>Outbreak 2</b>					
Litter C					
BD 10	5, 7, 9, 11, 13, 15, 17, 19	20	4	+	Recovered
BD 11	5, 7, 9, 11, 13, 15, 17, 19	20	6	—	
BD 12	5, 7, 9, 11, 13, 15, 17, 19	—	—	+	Isolated at 16 weeks
BD 13	5, 7, 9, 11, 13, 15, 17, 19	—	—	+	
BD 14	5, 7, 9, 11, 13, 15, 17, 19	20	8	—	Isolated at 16 weeks
BD 15	5, 7, 9, 11, 13, 15, 17, 19	—	—	+	
<b>Outbreak 3</b>					
Litter D					
BD 16	6, 7, 10, 13, 15, 17†	—	—	+	No illness
BD 17	6, 7, 10, 13, 15, 17†	17	13	—	

\*Vaccine used was a feline-origin parvovirus vaccine (Parvocine) except for those marked †, which were vaccinated with an inactivated canine-origin parvovirus vaccine.

spring of bitches with high CPV titers often developed disease at 3 to 4 months of age.

The purpose of this report was to describe the epizootiology of 3 outbreaks of CPV infection in vaccinated juvenile bush dogs, and to demonstrate age-related decline in maternal antibodies followed by a rise in CPV titer after vaccination in 2 species of nondomestic canids.

### Materials and Methods

**Colony description**—The South American canid colony of the National Zoological Park consists of multiple indoor and outdoor enclosures situated in the central portion of the Department of Conservation at Front Royal, Va. At the time of these outbreaks, the adult colony included 14 bush dogs, 10 maned wolves, and 9 crab-eating foxes (*Cerdocyon thous*). Five or 6 bush dog litters and 1 or 2 maned wolf litters are produced each year.

**Vaccinations and blood sampling**—Prior to the current outbreaks, each adult canid in the colony had been vaccinated every 6 months with an inactivated feline-origin parvovirus vaccine.<sup>6</sup> Each juvenile canid was vaccinated with 1 of 2 vaccines according to the regimens given in Table 1. Originally the aforementioned feline-origin vaccine was used. Later, an inactivated canine-origin parvovirus vaccine was used.<sup>7</sup> Blood samples were taken by jugular venipuncture.

**Bacterial cultures**—Fecal samples from swabs were cultured for *Campylobacter* spp and other enteric pathogens, using previously reported techniques.<sup>8</sup>

**Viral studies**—Hemagglutination (HA) and hemagglutination-inhibition (HI) studies were done, using rhesus RBC in phosphate-buffered saline solution, pH 5.8, at 4C, as previously described.<sup>9</sup> Eight HA units of virus were used in

\*Parvocine, Dellen Laboratories Inc, Omaha, Neb.

TABLE 2—Fecal hemagglutination titers\* against CPV in bush dogs

Bush dog no.	Day of illness								Comments	
	1	2	3	4	5	6	7	8		
<b>Litter A</b>										
BD 1	40				0					Died (day 6) Died (day 8)
BD 3	>1,280†									
<b>Litter B</b>										
BD 5	>1,280†									Died (day 4) Died (day 5)
BD 6	>1,280									
BD 7	80			0	0					Died (day 6) Died (day 30)
BD 8	20				0	0	0	0		
BD 9	>1,280									No illness
<b>Litter C</b>										
BD 10	320	320	H	H	H	H	80	0		Recovered (day 4) Died (day 6)
BD 11	40,960†	40,960	40,960	H	H					
BD 14	40,960	40,960	640	H	H					Died (day 8) No illness
BD 15	320	320	H	H						
<b>Litter D</b>										
BD 16	H	0	20	20	0	0	0	20		No illness Died (day 13)
BD 17	1,280	80	80	20	20	H	H	160		

\*Values represent reciprocal of titers. †Inhibited by standardized feline parvovirus antiserum.

H = hemolysis; not able to evaluate.

the HI test, and a standardized-known-positive feline parvovirus serum was used as control.<sup>6</sup> Virus isolations were carried out on Walter Reed A-72 canine kidney cells, using 0.45- $\mu$ m filtrates of 10% fecal samples in veal infusion broth containing penicillin and streptomycin.

Electron microscopy was done, using negative staining with phosphotungstic acid, as previously described.<sup>10</sup>

**Isolation of juveniles**—After the 3 outbreaks of CPV infection, a litter of maned wolves (litter E) and a litter of bush dogs (litter F) were moved to a quarantine facility in an attempt to prevent further disease in juveniles. At the time of removal, the wolves were 13 weeks old and the bush dogs were 15 weeks old. Each animal had been vaccinated weekly, beginning at 8 weeks of age, with the canine-origin parvovirus vaccine. Blood samples for determination of HI titers against CPV were obtained every 2 weeks. Rectal swab specimens for determination of HA titers against CPV and for bacterial cultures were obtained weekly.

### Results

Three outbreaks of CPV enteritis occurred from September 1981 to February 1982 in 4 litters of juvenile bush dogs, as summarized in Table 1. Ten of 17 pups were affected. In each outbreak, all affected pups became ill within a 1- to 3-day period. Clinical signs were anorexia, vomiting, bloody diarrhea, and depression. Severe leukopenia developed early in the course of the disease. The disease was confirmed by specific inhibition of hemagglutination of rhesus erythrocytes with a known-positive goat anti-feline panleukopenia virus antiserum (Table 2). A 27-nm particle morphologically similar to parvovirus was found by electron microscopy of fecal samples from 2 affected pups. The agent caused cytopathic effect, which was specifically inhibited by the anti-feline panleukopenia virus antiserum. Three clinically normal pups (BD 9, BD 15, and BD 16) had fecal HA titers during the period of the outbreaks in their littermates.

*Campylobacter fetus* subsp *jejuni* was isolated from 4 of 5 pups in litter B, and 3 of those infected

<sup>10</sup>Immune feline panleukopenia serum, Philips-Roxane, Goat, National Cancer Institute Cat. No. 9910-641, Lot No. 66-6900.

TABLE 3—Predisease HI titers\* against CPV in bush dogs

Bush dog no.	Age of pups (weeks)							Comments
	3	5	12	15	16	20	26	
Litter C								
BD 10			<10	<10		80		Ill at 20 weeks
BD 11		40	10	<10				Ill at 20 weeks
BD 12	80	40	10	10	40			Isolated at 16 weeks
BD 13			10	10				Isolated at 16 weeks
BD 14			<10					Ill at 20 weeks
BD 15	160	40	<10	10				No illness
Litter D								
BD 16		160			20			No illness
BD 17		40		40	20			Ill at 17 weeks

\*Values represent reciprocal of titers.

later died. The 1 that was not infected later died of CPV enteritis. Pups in litters A,C,D, and E were culture-negative for *Campylobacter* spp, and pups in all litters were culture-negative for other enteric pathogens.

Treatment included lactated Ringer's solution, B-complex and C vitamins, intestinal protectants, and antibiotics. Neomycin or erythromycin was given orally. Ampicillin with gentamicin or kanamycin was given parenterally. Each sick pup received a transfusion of 50 ml of whole blood from bush dogs with high CPV titers. Nine of the treated pups died.

Pathologic findings in the 9 fatally afflicted dogs included extensive necrosis of crypts of the small intestine. Neutrophils were rare or absent in the lesions. Commonly found were lymphoid depletion of the spleen and mesenteric lymph nodes, and pulmonary hemorrhages. In 2 dogs there was evidence of septicemia; 1 dog (BD 1) had necrotic hepatitis, and the other (BD 14), embolic pneumonia. *Pseudomonas aeruginosa* was isolated from the lesions in each of those 2 dogs.

Predisease HI titers against CPV were determined in litters C and D (Table 3). Two bush dogs isolated from the colony for treatment of foot sores at 16 weeks of age (BD 12 and BD 13) continued to receive vaccinations and never became ill. In these 2 bush dogs, CPV titers considered protective in domestic dogs ( $\geq 1:80$ ) developed by 29 weeks of age.<sup>6</sup>

## Discussion

The pathologic findings were similar to those previously described in bush dogs and other non-domestic canids with CPV infection.<sup>5</sup> *Campylobacter fetus* subsp *jejuni*, which has been found in association with parvoviral enteritis,<sup>11</sup> was a secondary pathogen only in litter B.

These outbreaks suggest either a problem with the vaccine or with the immune response of bush dogs. Adequate response to the vaccine was seen in the isolated maned wolves. Predisease CPV titers of the juveniles demonstrated waning passive immunity. Studies in laboratory dogs have shown that

maternal serum antibody against CPV infection interferes with active immunization but is not sufficient to protect against the disease. Cases of CPV infection continue to occur under these circumstances, despite repeated vaccination with a killed or attenuated vaccine.<sup>6</sup> A similar situation appeared to exist in our canid colony. Exposure of the bush dog pups to inapparent shedding by adults or from organisms remaining in the environment might have occurred during the period when their maternal antibody was low, with resultant infection of the pups. However, it is unclear why CPV infection did not begin until 8 weeks after HI titers dropped to nonprotective levels (<1:80) and why some animals with low titers escaped illness (Table 3).

The isolation of bush dogs and maned wolves provided an opportunity to study their response to vaccination. The dams of both litters had high HI titers against CPV a few weeks after the birth of the pups. The development of HI titers in the maned wolves was similar to that reported in domestic dogs whose dams had high HI titers.<sup>6,c</sup> Maternal antibodies declined to a low point at approximately 14 weeks of age. Protective titers from active immunization developed between 14 and 18 weeks of age. Maternal antibodies in the bush dogs declined to a low point at about 15 weeks of age; however, active immunization was not effective until the pups were 23 weeks old. This suggests a difference in immune response to parvovirus vaccine in these 2 species of canids.

<sup>c</sup>Folkers C, Wilson JHG, Roerink JHG, et al: Vaccination against canine parvovirus disease (letter). *Vet Rec* 110:235, 1982.

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