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A SUSPECTED CANINE DISTEMPER EPIDEMIC AS THE CAUSE OF A CATASTROPHIC DECLINE IN SANTA CATALINA ISLAND FOXES (*UROCYON LITTORALIS CATALINAE*)

Steven F. Timm,[1,8,9] Linda Munson,[2] Brian A. Summers,[3] Karen A. Terio,[4] Edward J. Dubovi,[5] Charles E. Rupprecht,[6] Sanjay Kapil,[7] and David K. Garcelon[1]

[1] Institute for Wildlife Studies, P.O. Box 1104, Arcata, California 95518, USA
[2] Department of Pathology, Microbiology, and Immunology, School of Veterinary Medicine, 1 Shields Ave., University of California, Davis, California 95616, USA
[3] Department of Pathology and Infectious Diseases, Royal Veterinary College, Hatfield, Herts AL97TA, UK
[4] University of Illinois Zoological Pathology Program, LUMC Building 101, 2160 S. First Ave., Maywood, Illinois 60153, USA
[5] Department of Population Medicine and Diagnostic Science, College of Veterinary Medicine, Cornell University, P.O. Box 786, Ithaca, New York 14853, USA
[6] Rabies Laboratory, Centers for Disease Control and Prevention, 1600 Clifton Road, Atlanta, Georgia 30333, USA
[7] Oklahoma Animal Disease Diagnostic Lab, Oklahoma State University, P.O. Box 7001, Stillwater, Oklahoma 740078, USA
[8] Current address: E9731 Co. Hwy. P, Westby, Wisconsin 54667, USA
[9] Corresponding author (email: stimm@mwt.net)

ABSTRACT: The island fox (*Urocyon littoralis catalinae*) population on Santa Catalina Island, California, USA declined precipitously in 1999 with an approximate 95% reduction on their eastern range, an area representing 87% of the island. During this investigation, between October 1999 and April 2000, evidence of live foxes dramatically decreased. The only carcass recovered during the decline succumbed to a co-infection of canine distemper virus (CDV) and toxoplasmosis. Sequence analysis of the viral P gene, derived by polymerase chain reaction, indicated that the virus was closely related to CDV from a mainland USA raccoon (*Procyon lotor*). Nine of 10 foxes trapped in 1999–2000, on the eastern portion of the island after the decline, had serologic evidence of exposure to CDV, whereas only four of 19 foxes trapped in this region in 1998 had antibodies reactive against CDV. The confirmation of CDV in one deceased fox, evidence of exposure to CDV in east-end foxes in 1999–2000 compared to 1998, and documentation of raccoon introductions to the island, implicates canine distemper as the cause of the population decline.

Key words: Canine distemper virus, demography, feral cats, island fox, raccoon, serology, toxoplasmosis, *Urocyon littoralis*.

INTRODUCTION

The island fox (*Urocyon littoralis*) is a relative of the mainland gray fox (*U. cinereoargenteus*), with a notably darker pelage and smaller size (Collins, 1982). Of the eight Channel Islands off the southern coast of California, foxes inhabit six of the largest islands, and genetic evidence supports separation of the species into six distinct subspecies (Gilbert et al., 1990). The subspecies *U. l. catalinae* is endemic to Santa Catalina Island (SCA), California, USA (33°18′N, 118°18′W), which is the third-largest Channel Island. The recent precipitous decline of island fox populations on the northern Channel Islands (Moore and Collins, 1995; Roemer, 1999), and the decline on SCA that is the subject of this report, prompted the U.S. Fish and Wildlife Service to list the island fox as an endangered species in 2004. The island fox has also been listed as a threatened species by the California Department of Fish and Game since 1987, principally due to their small population size and distribution, habitat destruction caused by feral goats (*Capra hircus*) and pigs (*Sus scrofa*) and by potential competition from feral cats (*Felis catus*).

Population estimates of SCA foxes have been obtained intermittently over the past 30 years by trapping on transects and grids. Studies by the Institute for Wildlife Studies (IWS) in 1989 and 1990 (Roemer et al., 1994) indicated that fox densities were high. Capture success on established trapping grids ranged from 10% in low-density areas (population estimate 2.4 foxes/km^2) to 31% in high-density fox

areas (population estimate 14.3 foxes/km^2; Roemer et al., 1994b). In July 1998, trap transects were operated on the eastern portion of the island to obtain fox blood samples for a serologic survey (Roemer et al., 2000). During 78 trap nights, a total of 20 foxes were captured, giving a capture success of 26%, similar to results from 1989 and 1990 trapping efforts (D. Garcelon, unpubl.).

During the summer of 1999, a substantial decrease in fox observations was noted by field biologists, and island residents reported numerous sick foxes and fox carcasses in the areas around the city of Avalon and various campgrounds. Because of these concerns, IWS conducted a small survey of 142 trap nights on the east end, and 14 trap nights on the west end from October–November 1999; we found an unusually low capture success on the east end. This apparent reduction in capture success prompted the Santa Catalina Island Conservancy (SCIC) to enlist IWS to conduct a demographic survey of the SCA fox population. The objective of this study was to further document the decline in the SCA fox population and its possible cause.

MATERIALS AND METHODS

Assessment of population status

Live trapping was conducted from October 1999 to April 2000 using conventional box traps (Tomahawk Live Trap Co., Tomahawk, Wisconsin, USA) baited with a combination of dog kibble, canned cat food, and a berry paste. Roadside transects were used, and traps were placed at 360-m intervals. In areas where live foxes were sighted, a slightly higher density of traps was set to increase the probability of capture. Up to 40 individual traps were set each night, and all traps were checked within a 24-hr period. Most transects were trapped for three consecutive nights unless inclement weather was predicted. Trapping was performed primarily by the same three experienced technicians. Clusters of fox observations in the same general area, or disturbed traps (digging or sprung traps) with fox sign, were interpreted as indicating a fox was present but not captured. Trapping results were analyzed separately for east- and west-end populations, which are delineated by an isthmus whereon the town of Two Harbors is a deterrent to fox dispersal (Kohlmann et al., 2005; Fig. 1). Capture success was calculated based on available traps to capture foxes, with traps containing nontarget species or with bait missing being considered unavailable.

Chi-square 2×2 contingency tests were used to analyze differences in capture success (number of captures/number of trap days) between the year prior to the apparent decline and the year after the decline, as well as to analyze spatial differences in capture success between the east and west ends of the island in 1999. To determine if capture success correlated with the estimated density of foxes, population estimates from 2001–2004, for both the east and west ends, were first calculated by adding the minimum number of foxes known to be alive (foxes either captured or known to be present due to signals from telemetry collars) to an estimated number of foxes in unsampled areas of the island. Density was then derived by dividing the fox population estimate for both the east and west ends by the area encompassed by each. The correlation between estimated density and capture success was then determined.

Assessment of fox health

All captured foxes ($n=59$) were given a thorough physical examination by a veterinarian (S.F.T.) to assess general health and to determine: 1) weight, 2) age class (0–4, as determined by dental wear patterns (Wood, 1958), 3) dental condition, 4) sex, and 5) reproductive condition. Foxes were examined for evidence of ocular or nasal discharge, ptyalism, diarrhea, coughing, poor hair coat, emaciation, injuries, aberrant behavior, or other evidence of disease. Foxes were aged according to tooth eruption and dentin exposure patterns relating to wear on the first upper molar (Wood, 1958; Collins, 1993) and assigned to one of five age classes: pups (age class 0: ca. <1 yr old), yearlings (age class 1: ca. 1–3 yr old), young adults (age class 2: ca. 3–5 yr old), mature adults (age class 3: ca. 4–6+ yr old), and old adults (age class 4: ≥7 yr old). Ear tags (Roto-Tag, Nasco West, Stockton, California, USA) were placed in the aural pinna to allow identification of individuals. Twelve foxes were fitted with mortality-sensing radiotelemetry collars (Advanced Telemetry Systems, Isanti, Minnesota, USA). Ten milliliters of blood were obtained from the femoral vein and allowed to clot for at least 1 hr. Blood samples were centrifuged, and

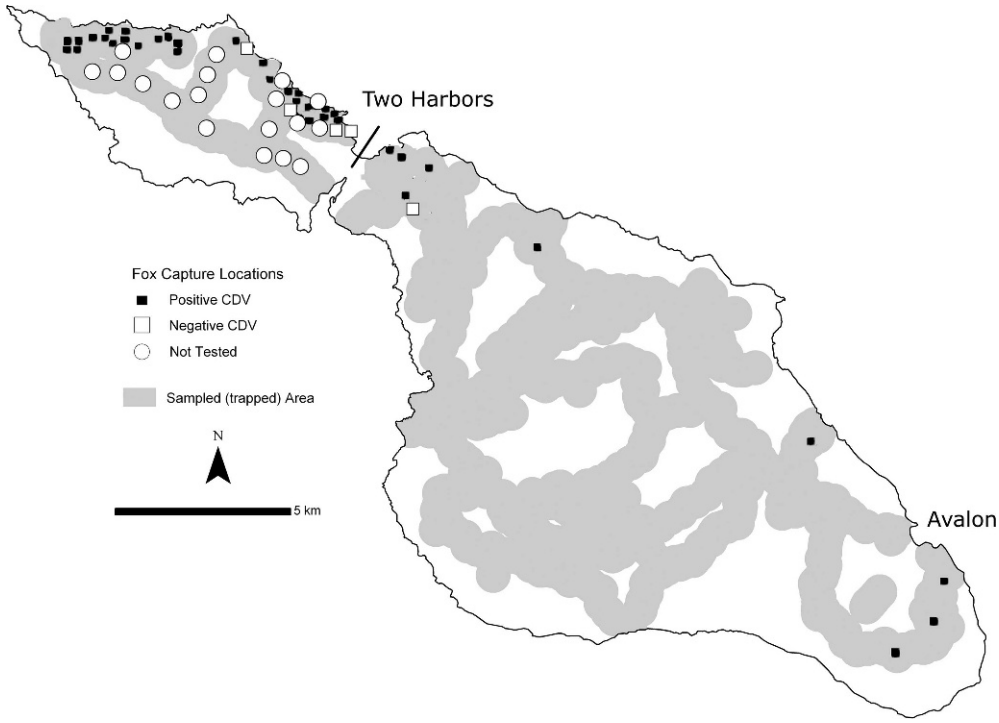

FIGURE 1. Island fox capture locations and distribution of foxes with antibodies reactive against canine distemper virus on Santa Catalina Island, California, USA (October 1999–April 2000).

serum was removed and frozen prior to shipment to a laboratory. The time, date, and GPS location were recorded for all captures. Other than those individuals brought into captivity for a captive breeding program and vaccine study ($n=6$), all foxes were released at the trap location.

Nontarget species captured were principally feral cats. Species, date, and location of each nontarget capture were recorded, and the animals were subsequently released. Some feral cats ($n=14$) were sedated with a combination of 11mg/kg ketamine hydrochloride (Fort Dodge, Fort Dodge, Iowa, USA) and 0.1mg/kg acepromazine maleate (Fort Dodge) injected intramuscularly. A blood sample was obtained, sex and age class (adult or juvenile) were recorded, and the animals were allowed to recover before release at the site of capture.

Laboratory tests

Serum from 41 foxes and 14 feral cats, collected from October 1999–April 2000, was frozen at −20 C and shipped to the New York State Veterinary Diagnostic Laboratory at Cornell University (Ithaca, New York, USA). Fox sera were tested for antibody titers reactive against canine distemper virus (CDV), canine adenovirus (CAV), and canine corona virus (CCV) by virus neutralization tests (VNT; Appel and Robson, 1973), canine parvovirus (CPV) by hemagglutination inhibition (HAI) test (Carmichael et al., 1980), *Leptospira interrogans* serovars *icterohaemorrhagiae, pomona, canicola, hardjo, grippotyphosa,* and *bratislava* using the microagglutination test (Cole et al., 1973), and *Toxoplasma gondii* by indirect hemagglutination test. For CDV, the Onderstepoort viral strain was inoculated onto vero cells. Positive antibody titers ≥8 were considered indicative of previous CDV exposure. This test cutoff for island foxes was based on replicate testing of samples ($n=26$ replicates) and performance of the Cornell CDV VNT on serum from vaccinated foxes (Clifford et al., 2006). Archived frozen plasma (serum not available) from 19 foxes trapped in 1998 on the east end of the island was also investigated for antibodies reactive against CDV by VNT. A subset of 10 serum samples (eight from foxes with antibodies reactive against CDV and two from foxes with no CDV-reactive antibodies) were tested for rabies-virus neutralizing antibodies via a rapid fluorescent focus inhibition test at the Centers for Disease Control and Prevention (Atlanta, Georgia, USA). A subset of 10 serum samples

from foxes with CDV-reactive antibodies were tested for phocine distemper virus (PDV), phocine morbillivirus (PMV), and dolphin morbillivirus (DMV) using a differential serum neutralization assay at the Oklahoma Animal Disease Diagnostic Laboratory (Stillwater, Oklahoma, USA; positive titer ≥8). Sera from feral cats were tested at the New York State Veterinary Diagnostic Laboratory for antibodies reactive against CDV by VNT and for antibodies reactive against feline immunodeficiency virus (FIV), feline infectious peritonitis (FIP), and toxoplasmosis by kinetic enzyme-linked immunosorbant assay. Positive FIV tests were confirmed by western blot. Feline leukemia virus (FeLV) antigens were tested by enzyme-linked immunosorbant assay.

An age class 1 male fox, found moribund on the southeastern side of SCA on 10 July 1999, died shortly after retrieval and was submitted for necropsy at a local diagnostic lab and then forwarded to the University of California, School of Veterinary Medicine (Davis, California, USA) for more comprehensive diagnostic tests. Formalin-fixed tissues were embedded in paraffin, sectioned at 5–7 μm, and stained with hematoxylin and eosin. Immunohistochemical procedures were applied to sections of formalin-fixed, paraffin-embedded lung and lymph nodes to demonstrate intralesional viral antigens using previously published methods (Roelke-Parker et al., 1996). Briefly, paraffin-embedded tissues were deparaffinized, treated to remove endogenous peroxidase, and then incubated with a mouse monoclonal antibody to a CDV-N protein (MAb N3.991; Orvell et al., 1985) or with rabbit primary polyclonal antibody raised with Rockborn strain of CDV. A commercial avidin–biotin kit was used to identify sites of MAb binding to tissues, and a commercial peroxidase–antiperoxidase kit was used for polyclonal antibodies; then tissue sections were counterstained with Gill's hematoxylin, dehydrated, and mounted with Permount (Fisher Scientific, Pittsburg, Pennsylvania USA). Negative controls were duplicate sections stained using an MAb for influenza virus replacing CDV MAbs, and positive controls were brain sections from a confirmed case of CDV in a domestic dog.

Similar immunohistochemical procedures were performed on the lungs and lymph nodes to detect *Toxoplasma*. After deparaffinization, tissue sections were predigested with 0.4% pepsin for 15 min, then a polyclonal rabbit anti-*Toxoplasma* antibody (Biogenex, San Ramon, California, USA) was applied at a 1:80 dilution. Antibody binding was visualized using a horseradish peroxidase system (Envision Rabbit, Dako Corporation, Carpenteria, California, USA).

A semi-nested reverse-transcription polymerase chain reaction (PCR) for a 149-base-pair fragment of the canine distemper virus phosphoprotein gene was conducted on formalin-fixed paraffin-embedded sections of lung, using previously described methods (Stanton et al., 2002). Each reaction step was conducted using appropriate negative and positive controls. Amplification products were separated on a 2% agarose gel by electrophoresis, visualized by ethidium bromide, and excised from the gels. DNA was extracted from the excised gel fragment using a commercially available kit (QIAquick Gel Extraction Kit, Qiagen, Valencia, California, USA). Nucleotide sequences were determined on an automated sequencer (Applied Biosystems 3730XL, Foster City, California, USA) at the University of Chicago Cancer Research Center DNA Sequencing and Genotyping Facility (Chicago, Illinois, USA). Nucleotide sequences were aligned with CLUSTAL W using MEGA 3.1 software, and comparison sequences were obtained from GenBank (Kumar et al., 2004). Phylogenetic analysis of the alignment was performed using distance matrix and maximum parsimony methods within the MEGA software package to produce a consensus tree, and data were subjected to bootstrap analysis based on 100 resamplings of the original data set.

RESULTS

Fox population demographics

From October 1999 through April 2000, 10 foxes were captured 16 times on the eastern portion of the island; captures occurred during 1,046 trap nights representing 449 unique trap locations along 134 km of roads and trails. This 2% capture success (based on 923 available traps) was a 16-time reduction from the previous year ($\chi^2=111.7$; $P<0.0001$; Table 1 and Fig. 1). The drop in capture success was not evident on the western portion of the island, where during the same time period, 140 trap nights of effort along 47 km of roads resulted in the capture of 49 individual foxes a total of 57 times, and in a capture success of 45% (based on 126 available traps). There was a significant difference in capture success

TABLE 1. Results of trapping efforts for island foxes (*Urocyon littoralis*) on Santa Catalina Island, California, USA (October 1999–April 2000).

Location	No. trap nights	No. individual foxes captured (No. males:No. females)	% capture success[a]	No. cats captured[b]	% capture success[a,b]
West end	140	49 (26:23)	45.2	6	8.1
East end	1,046	10 (4:6)	1.7	63	6.5
Total	1,186	59 (30:29)	6.9	69	6.6

[a] Capture success based on available traps.

[b] Feral cats (*Felis catus*).

between the east and west ends of the island during this time (χ^2=317.4; P<0.0001), suggesting that changes were spatially explicit and not associated with an island-wide phenomenon. Capture success in 2001–2004 was highly correlated with estimated fox density (r^2=0.985; Fig. 2), suggesting that the low capture success (2%) evident on the east end of the island in 1999 indicated an extremely low fox density.

Of the 10 foxes trapped on the eastern portion of the island (the 87% of the island east of the Isthmus), four were within 2.3 km of the isthmus at Two Harbors, the division between the east and west ends of the island. Additional sightings and evidence of disturbed traps suggested approximately 18 other individuals resided on the eastern portion of the island in 2000. A population-size estimate of 28 individual foxes, based on trapping and sign, was considered the minimum number of foxes known to be alive on the eastern portion of the island at the time of this study.

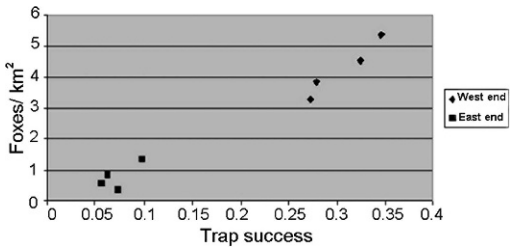

FIGURE 2. Correlation between estimated fox density and capture success on Santa Catalina Island, California, USA in 2001–2004 (r^2=0.985). This high correlation suggests that the low capture success (2%) on the east end of the island in 1999 reflected an extremely low fox density.

The number of males and females captured did not differ significantly from 1:1 (χ^2=0.01695; P=0.855). The majority of captured foxes (50 of 59; 85%) were in age classes 1 and 2, 15% were in age class 3, and no age class 0 (pups) or age class 4 (aged) individuals were captured. Nontarget species captured during trapping efforts included five Common Ravens (*Corvus corax*), eight ground squirrels (*Spermophilus beecheyi*), and 69 feral cats, with a capture rate of 7% for cats (Table 1).

Health of captured foxes

Only three captured-and-released foxes appeared in poor condition with rough, brittle hair coats and low body weights. These foxes had chronic injuries and were older individuals in age class 3. Of the 32 foxes examined otoscopically, 31 (97%) were found to have otitis and ear mites. Also found were mild to moderate tick infestations and occasional fleas.

Serologic results from 41 foxes are reported in Table 2. Nine of 10 east-end foxes and 27 of 31 west-end foxes had antibodies reactive against CDV, with titers ranging from 8 to 256 on the east end and titers from 8 to 32 on the west end. Foxes with detectable antibodies reactive against CDV were found in all age classes captured. Retrospective analyses detected antibodies reactive against CDV in four of 19 plasma samples taken in 1998. Two of those positive foxes were age class 1 and two were age class 2. Of 10 foxes with antibodies reactive against CDV that were captured in 1999 or 2000, nine

TABLE 2. Serum antibodies reactive against specific canine pathogens in 41 island foxes from Santa Catalina Island, California, USA (October 1999–April 2000). Antibody prevalence in foxes on the eastern portion of the island is compared to western island populations. Antibodies reactive against canine distemper virus (CDV), canine parvo virus (CPV), canine corona virus (CCV), *Leptospira* spp. (Lepto), and *Toxoplasma gondii* (Toxo) were tested.

Location	Age class[a]	CDV	CAV	CPV	CCV	Lepto[b]	Toxo
Eastern end	1	4/5	0/5	5/5	0/5	1/5	0/5
(n=10)	2	4/4	0/4	4/4	0/4	0/4	2/4
	3	1/1	0/1	1/1	0/1	0/1	0/1
Total		90%	0%	100%	0%	10%	20%
Western end	1	14/17	0/17	17/17	0/17	0/17	5/17
(n=31)	2	9/10	0/10	10/10	0/10	0/10	3/10
	3	4/4	0/4	4/4	0/4	0/4	0/4
Total		87%	0%	100%	0%	0%	26%

[a] Age classes are based on relative tooth wear after Wood (1958).

[b] All *Leptospira* serovars were negative with the exception of a single sample testing positive for *L. icterohaemorrhagia*.

foxes had antibodies reactive against PDV, with titers ranging from 8 to 32 (Table 3). None of the 10 foxes had antibodies that reacted against PMV or DMV. Of the 10 foxes tested for antibodies reactive against rabies virus, only one fox (the only animal included from a subset of foxes brought into captivity and vaccinated with a killed rabies vaccine) was positive.

All 41 foxes had antibodies reactive against CPV, and no foxes had antibodies reactive against CAV or CCV. Only one fox had antibodies reactive against *Leptospira,* and the proportion of animals with antibodies reactive against *Toxoplasma* was similar between east- and west-end populations. Serology results for the 14 feral cats indicated exposure to a variety of feline diseases, including exposure of four cats to CDV and of six cats to *Toxoplasma* (Table 4).

Pathology

Necropsy findings in the age class 1 male fox, recovered in July 1999, included pneumonia and lymphadenopathy. Histopathologic findings included severe necrotizing bronchopneumonia with bronchial epithelial syncytia that had eosinophilic intracytoplasmic and intranuclear viral inclusions. There were also viral inclusions in lymphocytes and histiocytes, epithelial syncytia with inclusions in the epididymis, footpad, and urethra, and pheochromocyte syncytia with inclusions in the adrenal medulla. The fox had marked lymphoid hyperplasia in lymph nodes, lymphocytolysis in the spleen, and lymphoid depletion

TABLE 3. Antibody test results for rabies virus, phocine distemper virus (PDV), porpoise morbillivirus (PMV), and dolphin morbillivirus (DMV) in selected island foxes from Santa Catalina Island, California, USA for which serum was available from October 1999–April 2000.

Location	Rabies	PDV	PMV	DMV
Eastern end	0/5[a]	5/5	0/2	0/2
Western end	1/5[b]	4/5	0/5	0/5

[a] Fractions indicate number positive over number tested.

[b] The one positive fox had previously been vaccinated against rabies virus.

TABLE 4. Positive test results to select feline pathogens in feral cats from Santa Catalina Island, California, USA (December 1999–March 2000). Antibodies reactive against canine distemper virus (CDV), feline immunodeficiency virus (FIV), feline leukemia virus (FeLV), feline infectious peritonitis virus (FIPV), and *Toxoplasma gondii* (Toxo) were measured.

Age group	Number tested	CDV	FIV	FeLV	FIPV	Toxo
Juvenile	2	1	1	0	0	1
Adult	12	3	4	1	0	5
Total	14	29%	36%	7%	0%	43%

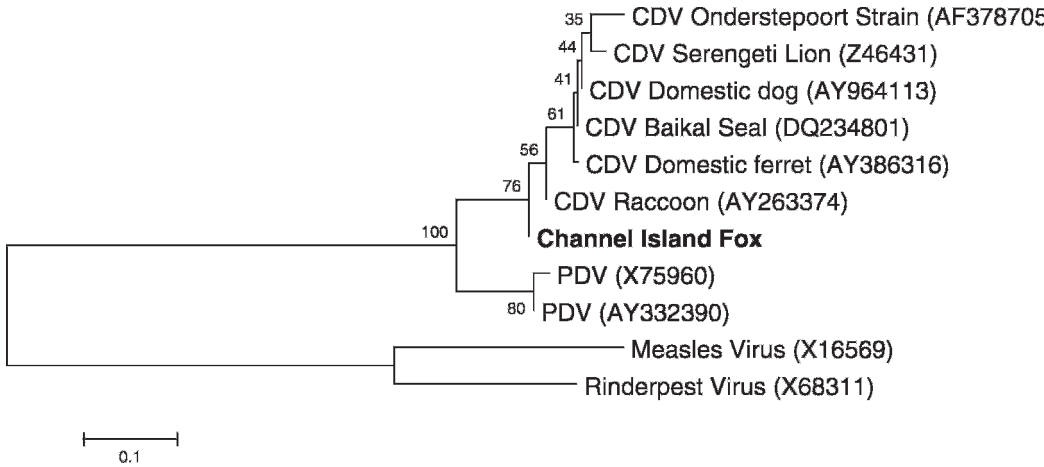

FIGURE 3. Phylogenetic tree for partial P gene sequences of representative morbilliviruses and a sequence from an island fox from Santa Catalina Island, California, USA. Phylogenetic and evolutionary analyses were conducted using MEGA version 3.1 (Kumar et al., 2004). Numbers at roots are the number of bootstrap iterations (out of 100) that support the nodes. Numbers in parentheses are the GenBank accession numbers for the reference sequences chosen for comparison.

of the tonsils. The brain was not available for examination. Canine distemper virus immunohistochemical stains strongly reacted with antigens in bronchial epithelial cells, lymphocytes, and all epithelial syncytia. There were widespread *Toxoplasma* zoites throughout alveolar septa and in macrophages around blood vessels and bronchi. The cause of death was diagnosed as bronchopneumonia due to canine distemper and toxoplasmosis.

The PCR amplified an amplicon of the P gene of appropriate size from formalin-fixed, paraffin-embedded sections of lung. A 109-base-pair segment of readable sequence, with primer sequences removed, was determined. This sequence was most similar (98%) to a strain of canine distemper virus isolated from a mainland USA raccoon (GenBank accession #AY263374; Fig. 3). The most similar marine morbillivirus was phocine distemper virus (GenBank accession #ZS75960) that had only 92% similarity to the sequence from the fox.

Of the 12 foxes radio-collared in 1999–2000, two were killed by automobiles 3 mo and 12 mo after being radio-collared, three died in 2003 from traumatic injuries, and one was brought into captivity because of injuries in 2005. Five others were recaptured at least once from 2001–2005 during annual-census trapping, and one was lost to follow-up in 2001 due to transmitter failure.

DISCUSSION

Based on the very low capture rates on the eastern 87% of the island after October 1999, and on previous census information, it is evident that the island fox population on SCA had undergone a significant decline between the fall of 1998 and mid-1999. Based on a population estimate of 1,200 foxes in 1991 (Roemer et al., 1994), approximately 85% of the entire SCA fox population was lost. As is often the case in wildlife not under close observation, the decline occurred before an investigation could be organized. Therefore, optimal diagnostic samples during the peak of the epidemic were not available, and the cause of the dramatic decline in this endangered species cannot be stated with certainty. However, an epidemic of CDV is suspected because CDV was the cause of death of

the only fox necropsied during the decline, and some foxes tested after the decline had high titers of antibodies reactive against CDV.

The capture success rate of 2% in 1999–2000 was extremely low when compared to historic rates on SCA and other islands. During 1989 and 1990, island fox capture rates on SCA were between 10% and 31% on three trapping grids (Garcelon et al., 2003). Furthermore, during a brief trapping effort in July 1998, capture success was 26% in a representative area on the eastern end of SCA (D. Garcelon, unpubl. data). Extrapolating the capture success of 2% on the east end of the island, with an average previous east-end capture success of approximately 20%, suggests that only 5% of the fox population survived the epidemic on the east end of the island. Our capture success of 45% on the west end of SCA in 1999 was typical of a healthy fox population. Over a 10-year period on San Clemente Island, capture success for island fox captures was 28% (n=13,560 trap nights) on three established trapping grids (Garcelon, 1999). Between 1993 and 1995, capture success for island foxes on Santa Cruz Island (SCZ) averaged 35% (n=2,520 trap nights; Roemer, 1999); and between 1973 and 1977 capture success on SCZ ranged from 49% to 78% (n=819 trap nights; Laughrin, 1980). Trapping efforts on San Nicolas Island in 2002 resulted in high capture success (49%; n=888 trap nights), and fox sign was very evident on the island (Schmidt and Garcelon, 2003). Taken together, these data indicate that the density of the fox population on the east end of SCA in 1999 was dramatically reduced.

A canine distemper epidemic was suspected because of the magnitude of the decline, the susceptibility of the 1998 population due to the low prevalence of antibodies reactive against CDV (21%), and the confirmation of CDV in the single carcass retrieved during the decline. In addition to these findings, island residents reported disoriented and dying foxes on the east end of the island during winter and spring of 1999, signs compatible with a neurologic disease such as canine distemper. Before 1999, sightings of dead or dying foxes reported to IWS or SCIC were rare. The absence of juveniles (age class 0) among trapped foxes in 1999–2000 is typical of canine distemper, where young animals are most susceptible (Williams, 2001). The increased prevalence of CDV-reactive antibodies in foxes (and with its detection in feral cats) in 1999–2000 provides strong evidence that CDV (or a closely related morbillivirus) was circulating at the time of the decline. The lack of mortality in west-end foxes was likely because of a lack of exposure to the virulent strain due to the geographic deterrent of the isthmus. The high prevalence of antibodies reactive against CDV after the decline suggests recent exposure to CDV across the population (Clifford et al., 2006). Although fox antibodies reactive against CDV also reacted against PDV, PDV is not endemic in this region, and no epidemic was noted in the carefully monitored marine mammals on SCA rookeries during this time (F. Gulland, pers. comm.). Cross-reactivity of CDV and PDV in virus neutralization tests is common, and PDV titers were lower than CDV titers in most foxes, again typical of a cross-reaction. Negative rabies tests do not completely rule out a rabies epidemic because few canids survive infection to seroconvert. However, the absence of clinical signs or mortalities in other mammals on the island makes rabies virus an unlikely cause of the decline in island foxes.

Canine parvovirus can also cause high mortality rates in juvenile animals, and new strains can cause considerable mortality in adults. However, all SCA foxes tested had antibodies reactive against CPV, and CPV appears to be endemic on all the Channel Islands (except possibly San Miguel Island), as indicated by the consistently high prevalence of antibodies

(Clifford et al., 2006). In the face of such high antibody prevalence, and the high disappearance rate across all age classes during the decline (an epidemiologic feature that is not typical of CPV infections in an endemic region [Barker and Parrish, 2001]), a parvovirus epidemic would be unlikely. Furthermore, no clinical signs of CPV, such as diarrhea or vomiting, have been observed in any examined fox, nor have lesions of CPV been detected in any deceased fox. Other potential causes, such as toxins or a stochastic event, are unlikely to have caused the decline because other mammalian species in the area were not known to be affected.

The concurrent occurrence of toxoplasmosis with CDV may have increased the mortality during the epidemic. Toxoplasmosis has not been reported to cause catastrophic losses in canids, but could be a copathogen contributing to mortalities. *Toxoplasma* zoites incite widespread damage when disseminated throughout the body, and can result in higher morbidity and mortality in immunosuppressed animals such as those infected with CDV or CPV (Greene and Appel, 1998; Williams, 2001). Finding both CDV and *Toxoplasma* spp. in the one confirmed mortality supports this hypothesis. Feral cats are presumed to be the source of *Toxoplasma* spp. As cats have only recently been introduced to the Channel Islands, island foxes may be more susceptible to feline pathogens that are new to their evolutionary history. Domestic cats can become infected with, and shed, CDV (Appel, 1974; Munson, unpubl.); however, their role in CDV transmission is unknown. Other carnivores such as skunks or raccoons that commonly maintain CDV in other ecosystems do not permanently reside on SCA, although accidental introductions of raccoons are known to have occurred.

The decline of the fox population is typical of CDV epidemics in susceptible wildlife populations (Williams, 2001). Canine distemper has been associated with declines of an African wild dog (*Lycaon pictus*) population (Alexander et al., 1996), widespread losses of black-footed ferrets (*Mustela nigripes*; Williams et al., 1988), and the disappearance of one third of the Serengeti lions (*Panthera leo*) in 1994 (Roelke-Parker et al., 1996). Foxes in the genus *Urocyon* have historically been particularly sensitive to both natural CDV infections (Halbrooks et al., 1981) and vaccine-induced disease caused by modified-live viruses (Munson, unpubl. data).

The island fox population previously was thought to be naïve to CDV because no antibodies reactive against CDV were detected in a 1988 survey that included SCA foxes (Garcelon et al., 1992). However, subsequent studies using a more sensitive test disclosed low levels of antibodies reactive against CDV in 1988 (Clifford et al., 2006). Antibodies reactive against CDV have been detected in many SCA foxes sampled recently, as well as in other island fox populations, suggesting that CDV is circulating through these populations without detected CDV-associated mortalities in these now closely monitored populations. Introduction of more-virulent biotypes from translocated mainland wildlife or domestic pets may explain differences in mortality rates among CDV exposures (Clifford et al., 2006). In support of that hypothesis, several "stowaway" raccoons on boats anchoring at Avalon have recently been accidentally introduced to SCA from the mainland (Garcelon and Munson, unpubl.). It is interesting to note that the virus isolated during the epidemic was most-closely related to mainland USA raccoon virus. Domestic dogs that reside on the island could have served as amplifying hosts if raccoon CDV was introduced. Introduction of a virus in Avalon is suggested by the fact that the two highest CDV titers were in foxes near Avalon, the confirmed CDV-caused fox death was near Avalon, and the foxes that

survived the epidemic were on the west end of the island furthest from Avalon.

In summary, a catastrophic decline in the SCA fox population occurred in 1999, and CDV is suspected to have played a role. The rapid decline in the east-end island fox population illustrates the vulnerability of insular carnivore populations to infectious agents, as well as the risk of mingling domestic pets or introduced wildlife with endangered-species populations. What remains unresolved is why 27 of 31 foxes from the relatively isolated population on the west end of the island also had antibodies reactive against CDV in 1999–2000, while the population remained stable during the decline of the east-end fox population. Because foxes are monogamous, have low reproductive rates, and have relatively short life spans, recovery of this endangered population would likely be protracted without human intervention. Recovery efforts have included: 1) vaccination of the remaining SCA foxes with a recombinant canary pox–CDV vaccine; 2) translocation of juvenile foxes from the west to the east end of the island; 3) development of a captive breeding and release program as a safeguard against extinction and to enhance population recovery; 4) long-term population monitoring using radio-collared animals to identify mortality factors; 5) encouraging vaccination of all domestic dogs on Santa Catalina Island and; 6) establishment of a feral cat and introduced-wildlife control program.

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