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Pathogen exposure in endangered island fox (*Urocyon littoralis*) populations: Implications for conservation management

Deana L. Clifford[a,*], *Jonna A.K. Mazet*[a], *Edward J. Dubovi*[b], *David K. Garcelon*[c], *Timothy J. Coonan*[d], *Patricia A. Conrad*[a], *Linda Munson*[a]

[a]*Wildlife Health Center, School of Veterinary Medicine, University of California, One Shields Avenue, Davis, CA 95616, USA*
[b]*Diagnostic Laboratory, New York State College of Veterinary Medicine, Cornell University, PO Box 786, Ithaca, NY 14851, USA*
[c]*Institute for Wildlife Studies, PO Box 1104, Arcata, CA 95518, USA*
[d]*National Park Service, Channel Islands National Park, 1901 Spinnaker Drive, Ventura, CA 93001, USA*

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ABSTRACT

Island fox (*Urocyon littoralis*) populations on four California Channel Islands have declined severely since 1994. Canine distemper (CDV) was suspected to be responsible for the decline of the Santa Catalina Island fox, so knowledge of infectious disease exposure in the remaining island fox populations was urgently needed. This study reviewed previous pathogen exposure in island foxes and investigated the current threat by conducting a serologic survey of foxes on all islands and sympatric feral cats on three islands from 2001 to 2003 for antibodies against canid pathogens. Before the decline, foxes had evidence of exposure to CDV, canine adenovirus (CAV), canine parvovirus (CPV), and *Toxoplasma*, with exposure to these five pathogens differing greatly by island. Exposure to canine coronavirus (CCV), canine herpesvirus (CHV), and *Leptospira* was rare. In 2001–2003, wild-born foxes had evidence of exposure to CDV (5.2–32.8%) on 5 of 6 islands, CPV (28–100%) and CAV (4.7–100%) on five islands, and *Toxoplasma gondii* (2.3–15.4%) on four islands. Exposure to CCV, CHV and *Leptospira* was less common. Sharing of infectious agents between sympatric foxes and feral cats appeared minimal, but CDV exposure was detected in two cats on Santa Catalina Island. Domestic dogs have historically been present on the islands, but it is not known if canine diseases can be maintained in fox populations without the continual presence of dogs. Targeted vaccination programs against the most virulent pathogens and continued intensive disease surveillance may help protect the critically small remaining fox populations from disease outbreaks that could threaten the success of ongoing conservation efforts.

1. Introduction

Infectious disease has caused dramatic population declines and local extinctions in many canid species (Woodroffe et al., 2004). Their restricted distribution and small population size make the island fox (*Urocyon littoralis*) particularly vulnerable to catastrophic events such as disease epidemics. Island foxes are a diminutive relative of the mainland gray fox (*Urocyon cinereoargenteus*), found only on six of the eight Channel Islands located off the coast of Southern California, USA

* *Corresponding author:* Tel.: +1 530 752 4167; fax: +1 530 752 3318.
E-mail address: dlclifford@ucdavis.edu (D.L. Clifford).
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(Fig. 1). The islands range from 37 km^2 to 249 km^2 in area and are located between 30 and 98 km away from the mainland (Philbrick, 1967). The islands inhabited by foxes are San Miguel (SMI; 33°02′ N, 120°18′ W), Santa Rosa (SRI; 33°57′ N, 120°06′ W), Santa Cruz (SCZ; 34°0′ N, 119°45′ W), Santa Catalina (SCA; 33°24′ N, 118°24′ W), San Clemente (SCI; 32°55′ N, 118°30′ W) and San Nicolas (SNI; 33°14′ N, 119°30′ W).

Island foxes are the largest indigenous terrestrial mammal in these unique ecosystems and are distributed as six genetically and morphologically distinct island subspecies (Collins, 1982; Wayne et al., 1991). Island foxes co-exist and may compete for resources with a smaller endemic carnivore, the island spotted skunk (*Spilogale gracili amphiala*) on SCZ and SRI (Crooks and Van Vuren, 1995). Feral cats (*Felis catus*) are present on the three southern islands (SNI, SCI, and SCA). Domestic dogs (*Canis familiaris*) are commonly found only on SCA (which has a resident human population and seasonal tourists), but dogs have been present on most islands as pets or working dogs in recent history and their remains have been found at native American archeological sites on all six islands (Collins, 1982; Schoenherr et al., 1999).

1.1. *Population decline of the island fox*

Four of the six island fox subspecies have declined by as much as 95% since 1994 (Coonan, 2001; Coonan et al., 2005), resulting in the fox being listed as critically endangered by the International Union for Conservation of Nature (IUCN, 2001) and four subspecies listed as federally endangered (United States Fish and Wildlife Service, 2004). To safeguard remaining foxes and augment natural recruitment, the entire wild populations of SMI and SRI, and a portion of the populations of SCA and SCZ were placed into captive breeding programs with the intent of releasing foxes back into the wild after mortality factors were identified and mitigated.

Although golden eagle (*Aquila chrysaetos*) predation was identified as the principal cause of mortality on SCZ, SRI and SMI (Coonan et al., 2005; Roemer, 1999; Roemer et al., 2001), the threat of infectious diseases following the decline needed to be assessed. The vulnerability of island foxes to infectious disease was realized in 1999 when canine distemper virus (CDV) was suspected to cause a rapid population decline on SCA. Approximately 90% of the wild fox population on the eastern two-thirds of the island disappeared, leaving fewer than 150 known individuals (Timm et al., 2000). Distemper virus was confirmed in the only fox carcass available (Munson, L., unpublished). Concerns for future epidemics led to a re-assessment of pathogen exposure in the island fox.

1.2. *Historic infectious disease exposure in island foxes*

Prior to the population decline in the 1990s, there were few investigations of pathogen exposure in the island fox population. In 1973 antibodies to San Miguel sea lion virus (SMSLV) were detected in 6 of 85 (7%) SCZ foxes, thereby extending the host range for marine caliciviruses to canids (Prato et al., 1977). Foxes were presumably exposed to SMSLV through scavenging on beaches occupied by infected

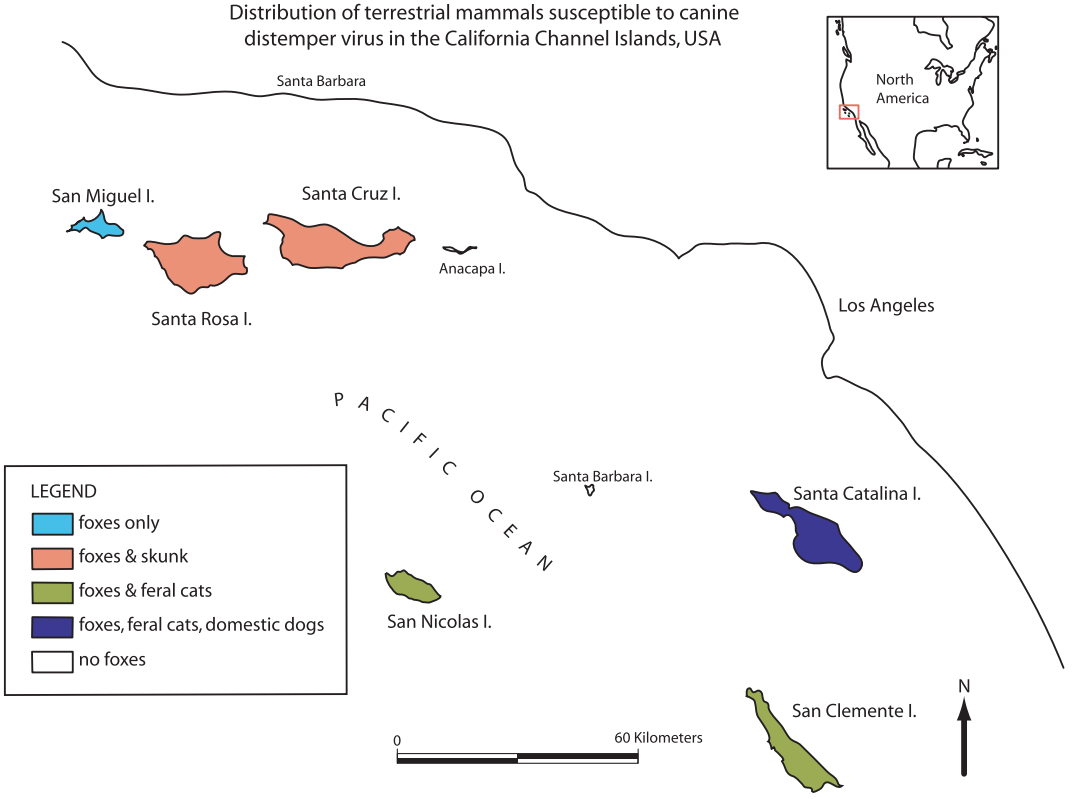

Fig. 1 – Map of the Channel Islands (California, USA) showing the distribution of terrestrial mammals on each island that are susceptible to canine distemper virus. Susceptible carnivores do not occur on Santa Barbara and Anacapa islands.

pinnipeds (Moore and Collins, 1995; Prato et al., 1977). A second survey of 100 SCZ foxes in 1973 did not detect antibodies against CDV, rabies, or leptospirosis, therefore Laughrin, (1977) cautioned that island foxes were at risk from health hazards presented by abandoned cats and dogs, and from bat species moving between the islands and the mainland.

All six fox populations were surveyed in 1988 for exposure to CDV, canine parvovirus (CPV), canine adenovirus (CAV), canine coronavirus (CCV), canine herpesvirus (CHV), *Toxoplasma gondii* and *Leptospira interrogans* (Garcelon et al., 1992). No antibodies against CDV were detected, but foxes on all six islands were exposed to CPV, with prevalence ranging from 5% to 59%. Exposure to CAV was common (72–97%), but absent in foxes on SCA and SCZ. Antibodies to CCV were found on only two islands, while CHV antibodies were found in a small number of foxes. Antibodies to the protozoal parasite, *Toxoplasma gondii* were present on all islands but SMI in 1988, and *Leptospira interrogans* serovar *icterohaemorrhagiae* antibodies were detected only on SCZ (Garcelon et al., 1992). A subsequent survey conducted during the northern island fox population decline (1994–1997) on SMI, SCZ and SCI detected fewer foxes exposed to CPV compared to 1988 and documented the new presence of CAV antibodies in 58% of SCZ foxes sampled (Roemer et al., 2001). The emergence of CAV on SCZ may have occurred via domestic dog contact or via spillover from the sympatric population of island spotted skunk. Immediately following the 1999 SCA fox decline, a survey of foxes detected CDV antibodies, including high titers suggestive of recent infection (Timm et al., 2000). Preliminary surveillance of sympatric feral cats detected CDV antibodies, suggesting spillover of CDV into this species (Timm et al., 2000).

Island foxes are likely susceptible to mortality from CDV because of the extreme susceptibility of the gray fox to both natural and vaccine-induced CDV infection (Halbrooks et al., 1981; Hoff et al., 1974; Nicholson and Hill, 1984), and the occurrence of fatal vaccine-induced CDV infection in captive island foxes (Munson, unpublished). Distemper has also caused significant mortalities in other wild carnivores, including black-footed ferrets (*Mustela nigripes*), African lions (*Panthera leo*), and wild dogs (*Lycaon pictus*) (Alexander et al., 1996; Roelke-Parker et al., 1996; Williams et al., 1988). The lack of evidence of exposure in foxes sampled in 1988 suggested the population was at substantial risk for a CDV epidemic in the future (Garcelon et al., 1992).

Canine parvovirus may also threaten island foxes, particularly juveniles, because it can cause debilitating enteritis and panleukopenia in juvenile and naïve adult domestic dogs (Pollack and Carmichael, 1990). Canine parvovirus may also have affected the recovery of some grey wolf (*Canis lupus*) and red wolf (*Canis rufus*) populations through increased pup mortality (Mech and Goyal, 1995) or poor juvenile survival (Munson, unpublished).

The high prevalence of CAV antibodies on some islands indicates exposure is common and suggests many foxes survive infection but introduction of CAV to the naïve SCA or SCZ fox populations could impact pup survival or cause disease in adults. Canine adenovirus type 1 causes infectious canine hepatitis and encephalitis in domestic dogs, wild canids, ursids and mustelids (Green, 1998; Woods, 2001). Mortality from CAV has occurred in silver foxes (*Vulpes vulpes*), with the disease primarily affecting younger animals (Woods, 2001). Neonatal dogs and captive coyote pups also can get a fatal generalized CHV infection (Appel, 1987; Evermann et al., 1984). Based on their natural history, clinical disease due to primary *T. gondii* or *Leptospira interrogans* would be rare in island foxes but other stressors (starvation, concurrent disease, placement in captivity) could make them more vulnerable to clinical disease.

1.3. *Current disease concerns for island foxes*

Based on studies showing that island foxes were naïve to many potentially dangerous pathogens, and evidence of CDV exposure in foxes and cats on SCA, a comprehensive serosurvey for infectious diseases was identified as a top research priority by the Channel Island Fox Working Group (Coonan, 2001). This survey was considered critical to ensure the health of foxes in captivity and to identify threats on islands where fox populations remained in the wild or would be released from captive breeding programs. Furthermore, the dramatic population declines and the subsequent placement of island foxes into captive breeding facilities may have altered the ecology of pathogens in island foxes through reduced acquired immunity or increased pathogen concentration at the sites of captive breeding. To address these concerns, exposure to selected infectious disease agents that could threaten fox persistence was examined by conducting a systematic serologic survey of foxes and sympatric feral cats for antibodies against pathogens known to infect canids. Feral cats were also surveyed for exposure to feline-specific diseases that cause immune suppression and thereby increase the susceptibility of cats to infection with canid pathogens.

2. Study area and methods

2.1. *Sampling*

We collected 312 serum samples from all six islands: SMI ($n = 16$), SRI ($n = 42$), SCZ ($n = 48$), SCA ($n = 58$), SCI ($n = 78$) and SNI ($n = 70$). Serum was collected in 2001 ($n = 69$), 2002 ($n = 145$) and 2003 ($n = 98$). Additionally, 117 archived samples collected in 1988 prior to the population declines were examined ($n = 19$ SMI, $n = 26$ SRI, $n = 14$ SCZ, $n = 16$ SCA, $n = 17$ SCI, $n = 25$ SNI).

Samples were collected opportunistically from wild foxes trapped as part of population monitoring efforts. Foxes were trapped using welded-wire box traps (Model #106, $23 \times 23 \times 66$ cm, Tomahawk Live Trap Co., Tomahawk, WI, USA) modified with plexiglass and bite bars to reduce tooth damage (Kohlmann et al., 2003; Roemer et al., 1994). Capture locations were recorded using handheld global positioning system units (Garmin 12XL, Garmin, Olathe, KS, USA; UTM, NAD 27 CONUS 11). On SMI and SRI, most foxes were held in captivity, thus samples were obtained during scheduled health examinations. Blood samples (up to 10 cc) were collected by femoral venipuncture using a 12- or 6-cc syringe and 22-gauge needle. Whole blood was placed into collection tubes with no anticoagulant, allowed to clot at room temper-

ature, then serum obtained by centrifugation. Serum was frozen at ⩽−40 °C until analysis.

The sex of all foxes was recorded, and individuals were uniquely identified with a colored ear tag (Rototag, Nasco-West, Stockton, CA, USA) or subcutaneous passive integrated transponder tag (Biomark Inc., Boise, ID, USA) (Kohlmann et al., 2003). Birthplace (wild or captivity) was noted for foxes being held in captivity. Age class was determined for wild-caught foxes using tooth eruption and wear pattern of the first upper molar (Wood, 1958). Actual or minimum estimated age in years was recorded for foxes sampled in captivity. No foxes younger than five months of age were sampled to eliminate the possibility of maternal antibody interference with test results. Fox age was classified as young (age class 0–1 or <2 years of age) or adult (age class 2–4 or >2 years of age) for statistical analyses, and both age classes were well represented, with 56% of the samples from young and 44% from adult foxes. The sex ratio of wild foxes sampled was approximately 1:1 (52% male: 48% female). Individual foxes were sampled only once throughout their lifetime. If multiple serum samples existed for certain individuals, a single sample was selected based upon the serum volume available. No previously vaccinated foxes were sampled. In order to determine possible disease risks from feral cats to island foxes, frozen archived sera from 92 feral cats collected between 2001 and 2003 on SNI ($n = 8$), SCI ($n = 21$) and SCA ($n = 63$) were tested for feline and canine pathogen exposure. Each cat was sampled once.

2.2. Serologic testing

Serologic assays for canine and feline viruses and *Leptospira* were conducted at the New York State Animal Health Diagnostic Laboratory, Cornell University (Ithaca, NY, USA). Serum samples from foxes on all six islands collected between 2001 and 2002 were assayed for antibodies against CDV, CAV, CCV and CHV, using serum neutralization (SN) tests (Appel and Robson, 1973); CPV using a hemagglutination inhibition (HAI) test (Carmichael et al., 1980) (positive titer ⩾ 1:10); and six *Leptospira interrogans* serovars (*icterohaemorrhagiae*, *pomona*, *canicola*, *hardjo*, *grippotyphosa*, and *bratislava*) using the microagglutination (MIA) test (Cole et al., 1973) (positive titer ⩾ 1:100). For CDV, the Onderstepoort viral strain was inoculated onto Vero cells. Positive antibody titers ⩾ 1:16 were considered indicative of previous CDV exposure, while positive antibody titers ⩾ 1:8 but <1:16 were classified as suspect. This test cutoff for island foxes was based on replicate testing of samples ($n = 26$ replicates) and performance of the Cornell CDV SN assay on serum from vaccinated foxes. For CAV, CCV and CHV, antibody titers ⩾ 1:8 were considered positive.

Fox samples were also tested for antibodies to the protozoal parasites *Toxoplasma gondii* and *Neospora caninum* at the University of California, Davis (California, USA) using an indirect fluorescent antibody test (IFAT) (Miller et al., 2001) with a 1:100 dilution of fluorescein isothiocyanate (FITC)-conjugated goat anti-dog or goat-anti cat (Jackson Immuno Research Laboratories, Inc., West Grove, PA, USA). Fluorescence at a titer ⩾ 1:640 was considered positive. The number of fox samples analyzed for each pathogen varied due to limited serum volume. Serum collected in 2003 from wild foxes on SCZ, SCA, SCI and SNI was tested for CDV, CAV, CPV, *T. gondii* and *N. caninum* antibodies, while 1988 fox samples were examined for CDV antibody only.

To examine the possibility of cross-reactivity in SN tests between closely related marine mammal morbilliviruses and canine distemper virus, a subset of 34 fox samples was tested for phocine distemper virus (PDV), phocine morbillivirus (PMV) and dolphin morbillivirus (DMV) using a differential SN assay at the Oklahoma Animal Disease Diagnostic Laboratory (Stillwater, OK, USA; positive titer ⩾ 1:8) (Duignan et al., 1995; Rossiter et al., 1985).

The possibility of pathogen sharing between cats and foxes was investigated by testing cats for antibodies to CDV (SN test, positive titer ⩾ 1:16); feline panleukopenia virus (FPLV) using HAI (positive titer ⩾ 1:10); and *T. gondii* (IFAT, positive titer ⩾ 1:640). The FPLV assay used in this study also detects antibodies to CPV (Carmichael et al., 1980), thus cats and foxes with positive titers may be exposed to either virus. All cats sampled, and a subset of 128 fox samples, were tested for feline calicivirus (FCV) antibodies by SN using virus ATCC 653 (positive titer ⩾ 1:8 for cats and foxes) (Harrison et al., 2004). The FCV SN may also detect related caliciviruses (Dubovi, unpublished).

Feral cat samples were tested for antibodies against FIV using a kinetic enzyme-linked immunosorbent assay (IDEXX PetChek® plate ELISA, IDEXX, Portland, ME, USA); and against feline enteric corona virus/feline infectious peritonitis (FCoV/FIP) using a kinetic ELISA with positive titers ⩾ 1:22 (Barlough et al., 1983). FIV results were reported as "positive", "equivocal", "high negative", or "negative" based on the kinetic ELISA slope and sample to positive ratio (Harrison et al., 2004). "Equivocal" samples were classified as "positive", and "high negative" samples were classified as "negative" for statistical analysis, as previous work has shown that the majority of cats testing "equivocal" are actually positive for FIV; and cats testing "high negative" are usually negative on confirmatory Western blot testing (Barr et al., 1991). The presence of FeLV p27 antigen was detected by microtiter plate ELISA (IDEXX PetChek®, IDEXX, Portland, ME, USA) and results classified as positive (including equivocal) or negative (Lutz et al., 1983). Although foxes were not tested for FIV, FeLV and FCoV/FIPV, as these disease agents are feline-specific, these pathogens may compromise immunity in infected cats (Hoover and Mullins, 1991; Knotek et al., 2000; Pedersen and Barlough, 1991), making cats more susceptible to opportunistic infections with other disease agents (such as CDV, FPLV, FCV or *T. gondii*) that could be transmitted to island foxes.

2.3. Analyses

The prevalence (number of exposed/number tested) of antibodies against each pathogen was determined for the wild (including wild-born) and captive-born fox population of each island as described (Thrusfield, 1995). Within each island population, prevalence was determined for age groups (young or adult), sex, and year (2001, 2002 or 2003). Differences in seroprevalence between islands were evaluated using χ^2 contingency tests or Fisher exact tests (EpiInfo StatCalc

ver. 3.2.2., Centers for Disease Control and Prevention, Atlanta, GA, USA). For each island, biologically plausible associations between pathogen seroprevalence and year, age group, sex, or exposure to other pathogens were evaluated using multiple logistic regression (STATA™ ver. 8.0, Stata Corporation, College Station, TX, USA). Regression model variables were selected based on univariate analysis and likelihood ratio (LR) tests while overall model fit was assessed using the Hosmer–Lemeshow test statistic (Hosmer and Lemeshow, 2000). The strength of associations were estimated using logistic odds ratios (OR) and 95% binomial confidence intervals (Long and Freese, 2001). For χ^2 and Fisher exact tests, odds ratios and 95% exact binomial confidence intervals (95%CI) were estimated using EpiInfo software (Mehta et al., 1985).

Locations where wild fox samples were collected on SCZ, SCA, SCI and SNI were examined for geographic clusters of higher or lower than expected CDV antibody prevalence using a Bernoulli probability model-based spatial scan statistic (Kulldorff, 1997) (SaTScan software ver. 2.1, Biometry Research Group, Division of Cancer Prevention, National Cancer Institute, Frederick, MD, USA). Repeatability of CDV assay results for replicate samples was assessed using the exact McNemar's change test for related samples with the strength of replicate agreement determined using the Kappa statistic (Thrusfield, 1995).

For feral cats, associations between antibody seroprevalence, island, and other pathogen exposure were examined using the statistical methods described for foxes. Age and sex data were not available for cats. The association between antibody seroprevalence in foxes and sympatric feral cats was evaluated using χ^2 contingency tables.

3. Results

3.1. *Island foxes*

Antibody prevalence in island foxes sampled from 2001 to 2003 is displayed in Table 1. Canine distemper virus antibody was detected in wild foxes and wild-born foxes in captivity on all islands but SMI. The prevalence of CDV-antibody positive wild or wild-born foxes on SNI was greater than SRI, SCZ, SCA and SCI, thus SNI foxes were 4.6 times more likely to be CDV-antibody positive than foxes on the other four islands during the study period. Wild (or wild-born) foxes with CDV-suspect antibody titers were present on all six islands, with the prevalence on SRI being significantly lower than SCZ, SCA, SCI and SNI. Although no CDV-antibody positive foxes were detected on SMI, 25% of wild-born foxes had suspect antibody titers. Canine distemper virus antibodies were absent in all captive-born foxes sampled. No antibodies were detected to marine mammal morbilliviruses (PDV, PMV, or DMV).

Table 1 – Seroprevalence (# exposed/# tested) of island foxes (Urocyon littoralis) sampled from 2001 to 2003 to canine distemper virus (CDV), canine parvovirus (CPV), canine adenovirus (CAV), canine coronavirus (CCV), canine herpesvirus (CHV), Toxoplasma gondii (TOXO) and Leptospira interrogans serovars pomona (LEPTO P.) and bratislava (LEPTO B.)

Island	Population sampled	Pathogens								
		CDV	CDV SUSP	CPV	CAV	CCV	CHV	TOXO	LEPTO P.	LEPTO B.
San Miguel (SMI)	Wild-born foxes (held in captivity)	0.0% (0/8)	25.0% (2/8)	0.0% (0/6)	100.0% (6/6)	0.0% (0/5)	0.0% (0/5)	0.0% (0/8)	0.0% (0/6)	0.0% (0/6)
	Captive-born foxes	0.0% (0/8)	0.0% (0/8)	28.6% (2/7)	71.4% (5/7)	0.0% (0/6)	0.0% (0/6)	0.0% (0/8)	0.0% (0/7)	0.0% (0/7)
Santa Rosa (SRI)	Wild-born foxes (held in captivity)	7.1% (1/14)	7.1%[b] (1/14)	92.9% (13/14)	100.0% (14/14)	0.0% (0/14)	0.0% (0/13)	14.3% (2/14)	14.3% (2/14)	21.4% (3/14)
	Captive-born foxes	0.0% (0/28)	0.0% (0/28)	89.3% (25/28)	17.9% (5/28)	0.0% (0/27)	0.0% (0/27)	0.0% (0/28)	0.0% (0/28)	3.6% (1/28)
Santa Cruz (SCZ)	Wild fox population	14.0% (6/43)	46.5% (20/43)	46.5%[c] (20/43)	4.7%[e] (2/43)	0.0% (0/28)	3.6% (1/28)	2.3% (1/43)	0.0% (0/28)	7.1% (2/28)
	Captive-born foxes	0.0% (0/5)	0.0% (0/5)	20.0% (1/5)	0.0% (0/5)	0.0% (0/5)	0.0% (0/5)	0.0% (0/5)	0.0% (0/5)	0.0% (0/5)
Santa Catalina (SCA)	Wild fox population	14.3% (8/56)	39.3% (22/56)	84.5%[d] (49/58)	0.0% (0/58)	9.4% (3/32)	0.0% (0/32)	5.2% (3/58)	0.0% (0/32)	9.4% (3/32)
San Clemente (SCI)	Wild fox population	5.2% (4/77)	39.0% (30/77)	100.0% (77/77)	74.4%[f] (58/78)	0.0% (0/46)	2.2% (1/46)	15.4%[g] (12/78)	0.0% (0/47)	21.3% (10/47)
San Nicolas (SNI)	Wild fox population	32.8%[a] (22/67)	41.8% (28/67)	98.6% (69/70)	40.0% (28/70)	0.0% (0/37)	0.0% (0/35)	0.0% (0/70)	0.0% (0/45)	0.0% (0/45)

CDV positive foxes had antibody titers $\geqslant$ 1:16, while CDV suspect foxes (CDV SUSP) had positive antibody titers $\geqslant$ 1:8 but less than 1:16.
a Greater than SRI, SCZ, SCA and SCI ($\chi^2 = 18.93$, $p < 0.001$).
b Lower than SNI, SCI, SCA and SCZ ($\chi^2 = 5.19$, $p = 0.023$).
c Lower than SNI ($\chi^2 = 43.16$, $p < 0.001$), SCA ($\chi^2 = 14.74$, $p < 0.001$) and SRI ($\chi^2 = 9.36$, $p = 0.002$).
d Lower than SNI (Fisher exact $p = 0.004$).
e Lower than SCI ($\chi^2 = 53.88$, $p < 0.001$) and SNI ($\chi^2 = 17.07$, $p < 0.001$).
f Greater than SNI ($\chi^2 = 17.89$, $p < 0.001$).
g Greater than SCZ ($\chi^2 = 4.93$, $p = 0.026$).

Distemper antibody prevalence varied by year on all islands, with SCI having the least variation (Fig. 2). On SRI, one of nine wild-born foxes tested had a positive CDV antibody titer in 2001, but none of five wild-born foxes tested in 2002 were positive. The prevalence of positive CDV antibody titers significantly decreased each year on SNI (LR $\chi^2 = 13.05$, $p < 0.001$; OR = 0.271, 95%CI for OR = 0.124–0.590). A similar trend was observed on SCZ, where CDV antibody prevalence decreased from 60% (3/5) in 2001 to 13% (3/23) in 2002 (Fisher exact $p = 0.050$), and to 0% in 2003.

When CDV-antibody suspect and positive groups were combined, the SCZ prevalence still decreased in a linear fashion over the three year period (LR $\chi^2 = 6.48$, $p = 0.011$; OR = 0.253, 95%CI for OR = 0.080–0.803). In contrast, the prevalence of CDV-antibody suspect and positive groups combined on SNI did not show a decreasing trend by year. The opposite trend was observed on SCA where the prevalence of CDV-antibody positive and suspect groups combined increased 4 times each year ($\chi^2 = 14.55$, $p < 0.001$; 95%CI for OR = 1.82–9.53).

Fig. 3 shows the proportion of young foxes that were CDV-antibody positive or suspect by sampling year. Positive distemper antibody titers were absent in young foxes on SCI ($n = 31$) and SRI ($n = 1$). On islands where CDV titers were present in young foxes (SCZ, SCA, and SNI), prevalence (18.6%) did not differ significantly from adults (29.2%). Similar to the wild population as a whole (Fig. 2), the prevalence of CDV positive young foxes decreased each year on SNI ($\chi^2 = 4.76$, $p = 0.029$; OR = 0.361, 95%CI for OR = 0.139–0.940), and on SCZ prevalence decreased markedly between 2001 and 2002 (Fisher exact $p = 0.016$), and was 0% in 2003. Although no CDV-antibody positive young foxes were detected during the study period on SCI, the proportion of CDV-antibody suspect young foxes increased significantly between 2002 and 2003 ($\chi^2 = 4.43$, $p = 0.035$).

There were no significant differences in CDV antibody prevalence between male and female island foxes. Spatial analysis did not reveal any statistically significant geographic clusters of greater or lower than expected CDV positive antibody prevalence on SCA, SCZ, SCI, and SNI.

Distemper antibodies also were detected by Cornell SN methods in samples collected from all six island populations in 1988; prevalences of positive tests ranged from 4.2% (1/24) on SNI to 17.4% (4/23) on SRI (Fig. 2). Foxes with suspect test results were also found on all islands, with the lowest prevalence on SNI (8.3%, 2/24) and the highest on SCA and SCZ (40%, both 6/15 foxes). Overall SRI had the highest proportion of antibody positive and suspect foxes combined (56.5%, 13/23 foxes), but prevalence did not differ significantly from SCZ, SCA or SCI. The proportion of antibody positive and suspect foxes combined was significantly lower on SNI than on SRI, SCZ, SCA and SCI ($\chi^2 = 10.25$, $p = 0.0014$), while the proportion of antibody positive and suspect foxes combined on SMI was not significantly different from the other islands. On islands where age data were available (SMI, SRI, SCZ and SCA), positive or suspect CDV-antibody titers were present in 10–50% of young foxes sampled in 1988.

San Nicolas Island foxes sampled during 2001–2003 were 11 times more likely to have been CDV-antibody positive than SNI foxes sampled in 1988 ($\chi^2 = 6.25$, $p = 0.012$; 95%CI for OR = 1.58–483.87). One CDV-antibody positive fox was present on SMI in 1988 (1/19) but none were detected from 2001 to 2003. Overall CDV seroprevalence on SRI, SCZ, SCA and SCI did not significantly differ between 1988 and 2001–2003.

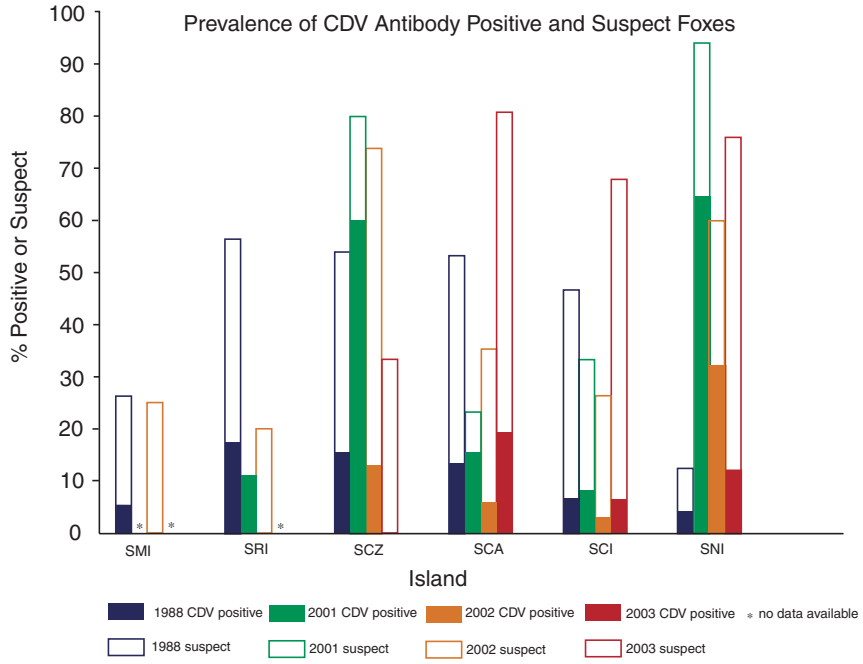

Fig. 2 – Seroprevalence of antibodies to canine distemper virus (CDV) in wild and wild-born island foxes sampled in 1988, 2001, 2002 and 2003. Results are reported as positive (titer ⩾ 1:16) or suspect (positive titer ⩾ 1:8 but <1:16). Years when no samples were available are indicated with a star (*). Island names are abbreviated as follows: San Miguel (SMI), Santa Rosa (SRI), Santa Cruz (SCZ), Santa Catalina (SCA), San Clemente (SCI) and San Nicolas (SNI).

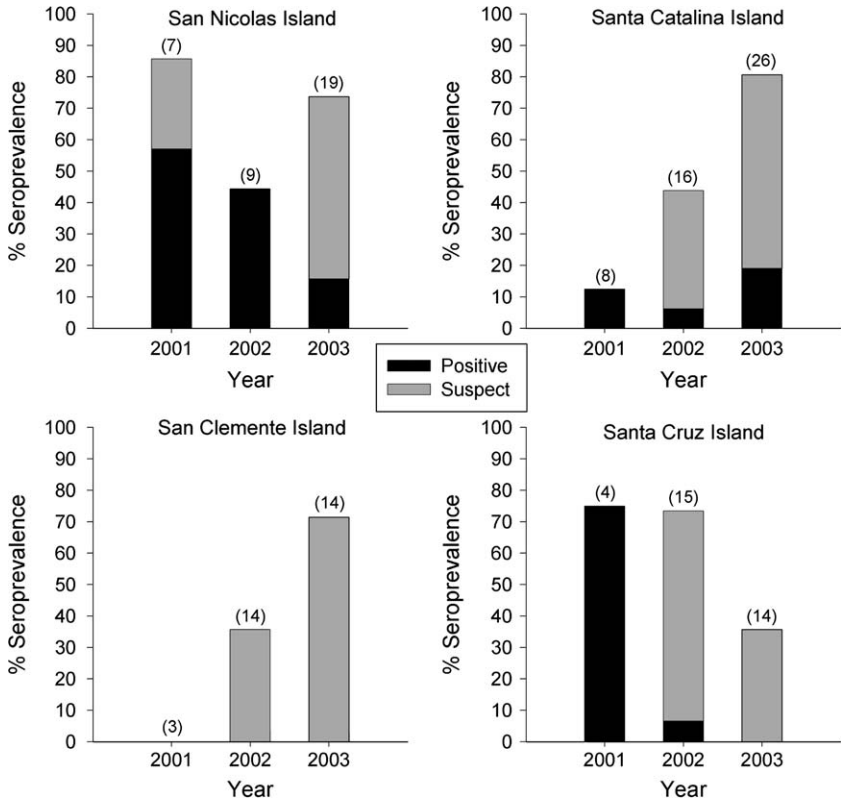

Fig. 3 – Seroprevalence of antibodies to canine distemper virus in young (Age class 0–1 or <2 years or age) wild island foxes sampled in 2001, 2002 and 2003. Results are reported as positive (titer ⩾ 1:16) or suspect (positive titer ⩾ 1:8 but <1:16). The number of individuals sampled is noted in parenthesis. San Miguel and Santa Rosa islands are not included in the figure, as those fox populations were extinct in the wild during this time period.

Cornell canine distemper virus SN assay results did not differ significantly for repeated test runs (exact McNemar's χ^2 $p = 0.219$). Replicate agreement was excellent for positive (antibody titer ⩾ 1:16) samples ($\kappa = 0.638$), but agreement decreased slightly when suspect samples were included ($\kappa = 0.572$).

Foxes sampled also had antibodies to CPV, CAV, CCV, CHV, *T. gondii* and two *Leptospira interrogans* serovars (*bratislava* and *pomona*; LEPTO B. and LEPTO P. respectively; Table 1). Canine parvovirus exposure was prevalent in wild and wild-born foxes in captivity on all islands but SMI. Prevalence on SCZ was significantly lower than SNI, SCA and SRI; and CPV prevalence on SCA was significantly lower than SNI. On SCZ, CPV prevalence was much greater in 2003 (93.3%) compared to 21.4% in 2001–2002 ($\chi^2 = 17.51$, $p < 0.001$). Both age (OR = 25.6, 95%CI = 2.32–280) and sex (OR = 12.68, 95%CI = 1.33–121) were significant predictors of CPV exposure on SCA, with young male foxes having the highest odds of being exposed to CPV. Even though CPV exposure was absent in wild-born foxes held in captivity on SMI, 28.6% of the captive-born foxes sampled were positive. Captive-born foxes were also exposed to CPV on SRI and SCZ.

Canine adenovirus antibodies were present in foxes on all but SCA (Table 1). Both SCI and SNI had greater CAV prevalence than SCZ, while SCI was also greater than SNI. Exposure to CAV did not differ significantly by sex or year sampled, but differed by age group on SCI and SNI. Mature foxes on SCI (87%) and SNI (61.8%) were 7 times more likely to be exposed to CAV than young foxes (SCI: 56.3%, $\chi^2 = 15.84$, $p < 0.001$, 95%CI for OR = 2.07–24.13; SNI: 19.4%, $\chi^2 = 13.52$, $p < 0.001$, 95%CI for OR = 2.28–19.65). High CAV antibody titers ⩾ 1:1024, suggesting recent infection were common on SMI, SRI, SCI, and SNI, but absent on SCZ (highest positive titer 1:12). Captive-born foxes on SMI and SRI were exposed to CAV, but antibodies were absent in captive-born foxes on SCZ.

Wild and wild-born foxes on SCA, SCI, SCZ and SRI had antibodies to *T. gondii*. The prevalence of *T. gondii* antibodies in male SCI foxes (23.7%) was higher than females (7.5%), with males 4 times more likely to be exposed ($\chi^2 = 4.06$, $p = 0.045$, 95%CI for OR = 0.95–15.43). Antibodies to *T. gondii* were absent in all captive-born foxes sampled. Antibodies to *Neospora caninum* were not detected in any foxes sampled.

Wild and wild-born foxes in captivity on four islands (SRI, SCZ, SCA, and SCI) had exposure to LEPTO B. with SCI and SRI having the highest prevalence. Additionally, one captive-born fox sampled on SRI was positive for LEPTO B. exposure. Exposure to LEPTO P. was found in two wild-born foxes on SRI, although one of these foxes also had a positive antibody titer to LEPTO B. Exposure to LEPTO P. was not documented on the other five islands. No antibodies were detected against the other four *Leptospira interrogans* serovars tested. Exposure to CCV and CHV was rare in wild (or wild-born) foxes and absent in captive-born foxes. Canine coronavirus antibodies were only present in 3/32 SCA foxes (9.4%), and CHV antibodies were present in one fox on SCZ and SCI.

3.2. *Comparison of seroprevalences in island foxes and feral cats*

All three islands with feral cat populations had detectable antibodies to FCV and *T. gondii*, with SCI having a significantly lower prevalence of FCV than SCA and SNI, and lower *T. gondii* prevalence than SCA (Table 2). Antibodies to CDV, FIV, FPLV, and FCoV/FIP and FeLV antigen were only detected in the SCA cat population.

Even though 76% of foxes sampled coexist with cats, island foxes and feral cats had little similarity in seroprevalence to FCV, CDV, *T. gondii* and CPV/FPLV (Fig. 4). Although CPV is highly prevalent in foxes on SCA, SCI and SNI, only one cat on SCA had antibodies against CPV/FPLV. Prevalence differences between cats and foxes were statistically significant on SCA for FCV ($\chi^2 = 25.61$, $p < 0.001$), CDV (Fisher Exact $p = 0.046$), *T. gondii* ($\chi^2 = 19.94$, $p < 0.001$) and CPV/FPLV ($\chi^2 = 78.97$, $p < 0.001$). Exposure to CPV/FPLV and CDV was absent in cats but present in foxes on SNI and SCI. Foxes on islands with cats had higher CPV/FPLV prevalence (95.1%) compared to foxes on islands without cats (52.4%, $\chi^2 = 66.4$, $p < 0.001$). There was no significant difference in FCV or *T. gondii* prevalence between foxes on islands with cats (52.3% and 9.3%, respectively) and without cats (39.6% and 5.6%, respectively).

Table 2 – Seroprevalence of canine distemper virus (CDV), feline immunodeficiency virus (FIV), feline leukemia virus (FeLV), feline calicivirus (FCV), feline panleukopenia virus (FPLV), feline enteric coronavirus/feline infectious peritonitis (FCoV/FIP) and Toxoplasma gondii (TOXO) in feral cats sampled between 2002-2003 from Santa Catalina, San Clemente and San Nicolas islands California, USA

Island	*n*	CDV (%)	FIV (%)	FeLV (%)	FCV (%)	FPLV (%)	FCoV/FIP (%)	TOXO (%)
Santa Catalina	63	3.2	42.8	23.8	77.8	1.6	1.6	44.4
San Clemente	21	0	0	0	26.3%* $n = 19$	0	0	4.8[a]
San Nicolas	8	0	0	0	62.5	0	0	25.0

a Significantly lower than Santa Catalina Island ($\chi^2 = 9.29$, $p = 0.002$).
* Significantly lower than Santa Catalina and San Nicolas islands ($\chi^2 = 14.29$, $p < 0.001$).

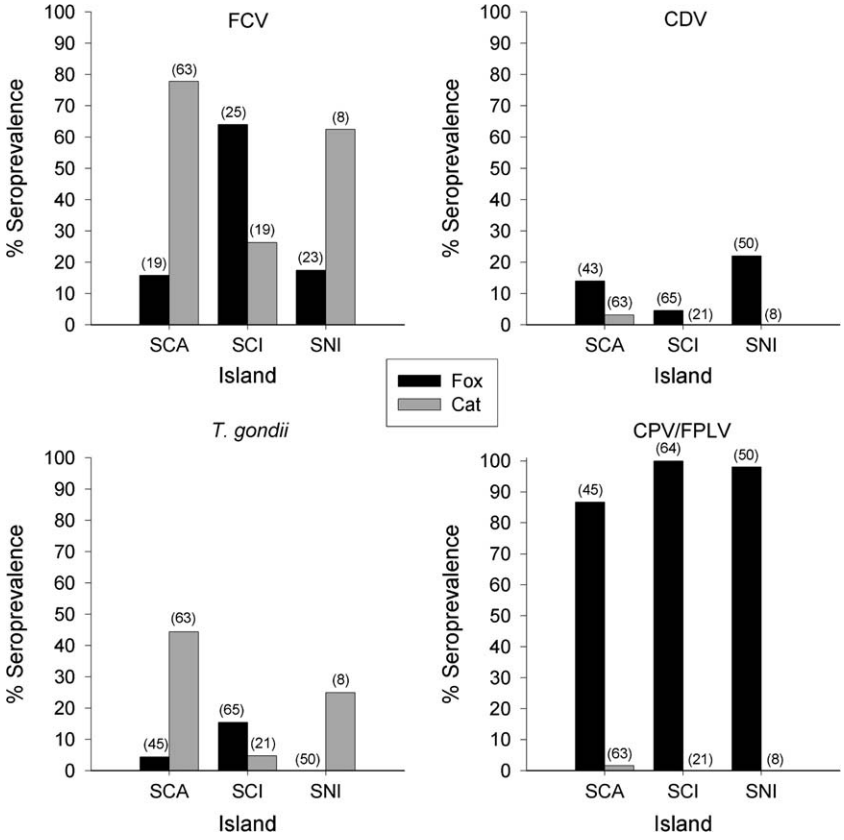

Fig. 4 – Seroprevalence of feline calicivirus (FCV), canine distemper virus (CDV), *Toxoplasma gondii* (*T. gondii*) and canine parvovirus/feline panleukopenia virus (CPV/FPLV) in wild (or wild-born) island foxes and feral cats sampled in 2002 and 2003 from Santa Catalina (SCA), San Clemente (SCI) and San Nicolas (SNI) islands. The number of individuals sampled is noted in parenthesis. Fox samples used for FCV testing were collected in 2001 and 2002.

4. Discussion

Although the distribution of the island fox is inherently limited to six of the California Channel Islands, human alterations of the ecosystems have put this unique species at risk of extinction from unnatural predation pressure (Roemer et al., 2001) and possibly through introduction of a more pathogenic strain of canine distemper virus (Timm et al., 2000). The placement of large numbers of remaining foxes into captivity, while critical for recovery efforts, further exacerbated the risk that disease could cause significant mortality or inhibit reproduction and eventual recovery. Our review of previous disease surveillance and recent survey results show that infectious disease exposure in island fox populations is dynamic over time and different on each island.

4.1. *Canine distemper virus exposure in island foxes*

Five of the six island fox populations have been exposed to canine distemper virus and exposure appears to have occurred only in the wild. The existence of CDV-antibody positive and suspect young foxes on SCA, SNI and SCZ for three consecutive years indicates that some foxes were recently exposed to distemper in the absence of detectable mortality in radio-collared individuals and recovered carcasses. This apparent survival of exposed foxes contrasts with the high mortality suspected to be caused by CDV in 1999 on SCA (Timm et al., 2000). Mortality rates from CDV may differ among populations because of the genetic resistance of the host, virulence of CDV strains (Lednicky et al., 2004), or co-infection with other pathogens such as CPV or *T. gondii*. Concurrent CDV and toxoplasmosis has caused mortality in gray foxes (Davidson et al., 1992; Kelly and Sleeman, 2003), and other pathogens including rabies, *Yersinia pseudotuberculosis*, *Listeria monocytogenes* and *Cryptosporidium* sp. have also occurred concurrent with distemper in gray foxes (Black et al., 1996; Davidson et al., 1992; Hoff et al., 1974). Additionally, the abundance of domestic dogs on SCA increases the risk of a more virulent CDV strain being transmitted from dogs to foxes.

These patterns of CDV infection in island foxes mirror the situation in African wild dogs (*Lycaon pictus*), where fatal CDV-epidemics have occurred (Alexander et al., 1996), yet other populations have had up to 24% of wild dogs exposed to CDV without clinical disease (Creel et al., 1997). Epidemic cycles of CDV are common in raccoons and gray foxes when the number of susceptible (i.e. unexposed) individuals becomes high (Hoff et al., 1974; Roscoe, 1993). Accordingly, the absence of CDV-antibody positive young foxes all three years on SCI suggests recent exposure may not be occurring and that an increasing proportion of the population is susceptible.

In contrast to previous findings (Garcelon et al., 1992), our data indicate that a proportion of foxes on all six islands sampled prior to the decline in 1988 were exposed to CDV. The most likely reason for this discrepancy is higher test sensitivity of the Cornell SN assay, allowing the detection of low level antibody titers that were not detectable previously. False positive results could be due to non-specific antibody binding, but if this had occurred, all foxes tested would be expected to have some level of background activity in the assay. Although the occurrence of false positive results cannot be definitively ruled out, we carefully scrutinized positive test results, ruled out cross-reactions with closely related morbilliviruses, and evaluated assay performance on island foxes pre- and post-vaccination to ensure the test was discriminating between negative and positive results for the same individuals. Additionally, the long-term storage of the 1988 samples (likely reducing detectable antibody levels) and our conservative test cutoff make it even more unlikely that these are false positive results. Concerns that the high test sensitivity of the Cornell SN assay may over-estimate exposure to CDV were the reason for our designating a large proportion of population as CDV-antibody suspect. If suspect results represent true CDV exposure, there would be further evidence that CDV is endemic across most islands. Re-analyzing the 1988 fox samples with the more sensitive assay allowed us to compare our study results directly with samples from before the decline and document a previous exposure to canine distemper virus. It is possible that a low pathogenicity island-fox adapted CDV strain evoking low antibody titers evolved and now circulates among island foxes without the continued presence of domestic dogs.

Whether antibody levels detected in island foxes are protective against distemper infection cannot be determined. Most naturally infected animals are protected for life, regardless of antibody titer (Appel and Summers, 1995). However, in domestic dogs, previous vaccination does not guarantee protection from an overwhelming viral infection or challenge with a highly pathogenic CDV strain (Greene and Appel, 1998). All but one CDV-antibody positive island fox in our study had natural exposure titers of less than 1:100; similarly low level antibody titers (<1:100) occur in island foxes after CDV vaccination (Timm et al., 2000). Due to concerns about the lack of CDV exposure in captive-born foxes and that a strain of CDV more pathogenic to foxes might be introduced, all foxes in captivity (including those foxes subsequently released from captivity) are now vaccinated with a canary-pox vectored CDV vaccine shown to be safe for use in this species (Timm et al., 2000). Recently a sub-set of the wild populations on SCA, SCZ, and SCI also were opportunistically vaccinated against CDV due to the critically low population numbers left in the wild (Coonan, 2003). Although vaccination interferes with our ability to discern whether CDV is endemic or epidemic, the need to protect the small number of remaining foxes from extinction warrants this intervention for the most at-risk populations.

4.2. *Exposure to additional diseases of concern in island foxes*

While CDV may have caused a rapid population decline due to high mortality on SCA, distemper and other disease agents may also hinder recovery by reducing pup survival or reproductive success. Our results suggest that CPV is highly prevalent and remains endemic on all the islands, except possibly SMI. Although there was no evidence of CPV exposure in wild-born SMI foxes, the presence of CPV exposure in captive-born foxes suggests the virus is still present on the island. Canine parvovirus is long-lived in the environment (Pollack and Carmichael, 1990), creating a potential for viral accumulation in the captive breeding facilities and increased risk for parvoviral disease in pups born at these sites.

Canine adenovirus appears to be endemic on SNI, SCI, and SRI, and absent on SCA, but the pattern of infection on SCZ is unclear (Green, 1998). In 1988, no exposure to CAV was reported on SCZ (Garcelon et al., 1992), but then exposure was documented in SCZ foxes sampled from 1994 to 1997 (Roemer et al., 2001) and our study documented low positive CAV antibody titers in wild SCZ foxes after 2000. It is possible CAV was introduced sometime between 1988 and 1994, and exposure prevalence has subsequently decreased as a consequence of the severe population decline on SCZ, causing the virus to fade-out. Recent work indicates sympatric SCZ spotted skunks were not exposed to CAV, making skunks an unlikely source of exposure for foxes (Bakker et al., 1995).

The greater CAV seroprevalence in mature foxes on SCI and SNI is typical of an endemic disease where many individuals are exposed (or re-exposed) throughout their life. Despite the continuous presence of domestic dogs on the island and the highly contagious nature of CAV, foxes on SCA still remain naive to CAV. This is likely due to the fact that clinical disease is very rare in dogs because of vaccination (Green, 1998).

Canine adenovirus and parvovirus have not been documented as a cause of mortality in island foxes, but clinical disease from these pathogens may go undetected in wild populations, as young pups are most commonly affected and mortality and exposure rates may be low. Fox pup mortality on Santa Catalina Island can approach 50% in the wild (Clifford, unpublished). The contribution of disease to the observed mortality is not known due to the difficulty in recovering carcasses from dens.

Exposure to *T. gondii* is relatively common in foxes, and the occurrence of an immunosuppressive disease such as CDV or CPV may allow *T. gondii* tissue cysts to proliferate resulting in clinical disease. Our study results indicated that *T. gondii* exposure was present in SCA and SCI foxes where cats are present, but most likely absent in SNI foxes where cats also reside. Antibodies to *T. gondii* were also detected on two islands that do not have feral cats (SCZ and SRI), suggesting foxes were exposed through consumption of infected bird or marine mammal carcasses (Dailey, 2001; Dubey et al., 1999b). The lack of exposure to *T. gondii* in captive-born foxes supports the contention that transmission occurs via contact with feral cat feces or from infected prey found in the wild.

The absence of *Neospora caninum* antibodies in sampled foxes may indicate this protozoal parasite does not exist on the islands. A single report of *N. caninum* being found in the feces of two of nine SNI foxes (Roemer et al., 2001) contradicts our findings and warrants follow up to differentiate the oocysts of *N. caninum* by molecular methods from those of the morphologically identical fox parasite, *Hammondia heydorni* (Gondim et al., 2004a). Domestic dogs and coyotes are the only known species capable of shedding *N. caninum* oocysts (Gondim et al., 2004b; Lindsay et al., 1999) but antibodies have been found in multiple wildlife species including gray foxes (Lindsay et al., 2001), gray wolves (*Canis lupus*), coyotes, white-tailed deer (*Odocoileus virginianus*), and moose (*Alces alces*), suggesting a cervid-canid sylvatic cycle (Dubey et al., 1999a; Gondim et al., 2004a; Lindsay et al., 1996). Cervids capable of becoming infected with *N. caninum* co-exist with foxes on SCA and SRI, and foxes could ingest tissue cysts by scavenging on introduced ungulates. Because *Neospora* can cause debilitating neurological disease in canids (Dubey, 2003), continued surveillance of island foxes and sympatric ungulates is warranted.

Leptospira interrogans exposure was evident in a small number of foxes on four islands. Leptospirosis is an endemic disease in California sea lions (*Zalophus californianus*) that breed on the islands and has caused reproductive failure and periodic epidemics of severe renal disease in these populations (Gulland et al., 1996; Smith et al., 1974). Island foxes may serve as an important sentinel for the presence of different *Leptospira* serovars in the California Channel Islands ecosystem as they inhabit the near-shore environment and interact with both terrestrial and marine mammals that may be harboring *Leptospira*.

Antibodies to CHV and CCV remain restricted to a few islands, with an apparent prevalence decrease since 1988 (Garcelon et al., 1992). We found no exposure to CHV in our SRI sample, although this subspecies had evidence of exposure in 1988 (12%). Additionally, Santa Cruz foxes no longer have evidence of exposure to CCV, although past prevalence was 7% (Garcelon et al., 1992). The pathogenicity of CHV and CCV in wild canids is unclear, thus the effects of an introduction of either pathogen to naive island foxes is unknown (Green et al., 1984; Pollack and Carmichael, 1990).

Given our study results indicating that island foxes are exposed to multiple canine-origin diseases, and the historic and recent presence of domestic dogs on the islands (Collins, 1982), it is possible that foxes acquired infections from domestic dogs. Most extinctions and near-extinctions of wildlife populations from disease are caused by generalist pathogens with a wide host range that "spill over" from domestic to wildlife species (Woodroffe, 1999). Rabies outbreaks in African wild dogs (Kat et al., 1995) and Ethiopian wolves (*Canis simensis*) (Randall et al., 2006; Sillero-Zubiri et al., 1996) and the CDV epidemic in lions (*Panthera leo*) from the Serengeti (Roelke-Parker et al., 1996) were believed to have originated from nearby domestic dog populations. On the California Channel Islands there is also risk of generalist pathogens like CDV spilling over from foxes or dogs into sympatric marine mammal populations, as was observed in Lake Baikal (*Phoca sibirica*) and Caspian seals (*Phoca caspica*) (Grachev et al., 1989; Kennedy et al., 2000). Domestic dogs are rarely permitted on the islands except for Santa Catalina where residents and tourists can bring their dogs. The unregulated travel of people and their pet dogs between Catalina and mainland California poses a constant threat to foxes.

4.3. *Pathogen sharing between island foxes and feral cats*

The disease risk feral cats pose to island foxes is unclear. Our data indicate that fox–cat pathogen sharing is minimal, but not absent. The presence of CDV antibodies in two feral cats on SCA likely occurred via spillover from foxes or dogs. The two CDV-antibody positive cats were also exposed to FIV and FeLV, possibly making these individuals more susceptible to CDV infection. Exposure to CDV in feral and domestic cats has been previously reported (Appel et al., 1974; Ikeda et al., 2001), but it is not known whether cats that are immunocompromised due to FeLV or FIV infection are capable of shedding CDV into the environment. However, immunocompromised

cats are vulnerable to other opportunistic diseases, including toxoplasmosis (Hoover and Mullins, 1991; Lin et al., 1992; Pedersen and Barlough, 1991). Foxes living on islands with feral cats have the opportunity to ingest *T. gondii* oocysts in cat feces, in addition to the tissue cysts in prey items (Tenter et al., 2000). The documentation of greater mortalities with concurrent distemper and *T. gondii* infection in gray foxes and domestic dogs provides support for controlling cats to reduce the numbers of infectious oocysts that are shed into the environment.

Although foxes on islands with cats have a higher prevalence of CPV/FPLV than foxes on islands without cats, the lack of CPV/FPLV antibodies in sympatric cats provides strong evidence that cats are not a primary source of exposure for foxes. While caliciviruses have been shown to infect a variety of hosts and could possibly be passed between cats and foxes (Smith et al., 1998), the inverse relationship between fox and cat calicivirus exposure in our study and the presence of calicivirus antibodies in foxes on islands without cats suggests this interaction is not necessary for fox infection. Given our results, it is likely that the biggest threat to island foxes from feral cats is competition.

4.4. *Conservation implications*

Due to their inherently small population sizes and limited geographic distribution, island foxes will always be vulnerable to stochastic events, including disease outbreaks. Previous studies and anecdotal reports from island residents suggest that fox population sizes fluctuated widely in the past (Laughrin, 1980). Island foxes can occur at much higher densities than gray foxes (3–14/km^2 vs. 0.4/km^2) which may increase the rate of pathogen transmission between susceptible animals (Garcelon and Schmidt, 2005; Grinnell et al., 1937; Laughrin, 1980). During the 1970s, fox numbers and density remained very low (0.5 foxes/km^2) on SCA. Additionally, a dramatic decrease in foxes trapped (from 24 to 2) and fox density (11 foxes/km^2 to 2 foxes/km^2) occurred on SNI between 1971 and 1974, and further decreased (3 foxes, 0.5 foxes/km^2) in 1977 (Laughrin, 1980). Laughrin's data correlates well with recent work examining major histocompatibility complex (MHC) gene diversity in island foxes, which suggests that the current degree of genetic monomorphism at neutral loci and high MHC gene variation in SNI foxes resulted from an extreme population bottleneck occurring between 20 and 40 years ago and resulting in less than 10 individuals (Aguilar et al., 2004). Although speculative, an infectious disease outbreak on SNI similar to what was observed on SCA in 1999 could explain the dramatic decrease in fox numbers observed by Laughrin, and resulted in the population bottleneck described by Aguilar and colleagues. Strong selection for resistance to CDV, as a result of previous epidemics, could explain the relatively high CDV exposure prevalence in the absence of clinical disease in some island fox populations today.

Even though our study suggests that CDV is endemic in some island fox populations, the recent suspected epidemic on SCA and the cyclical nature of CDV epidemics in other naturally exposed wildlife populations support concerns that island foxes are at risk from fatal epidemics of canine distemper. The continued presence of domestic dogs (whether constant or sporadic) on the California Channel Islands poses a risk to island foxes. In addition to dogs, the presence of introduced feral cats on three islands and the endemic island spotted skunk on two islands may pose some disease risk to foxes, as pathogens such as distemper and rabies could circulate among sympatric carnivores on the island.

The recent placement of significant portions of three of six island fox populations into captivity may decrease the opportunity for foxes to be exposed to low virulent pathogens present in their native environment. Accordingly, foxes in captivity should be vaccinated for the most potentially dangerous pathogens (CDV and rabies) to avoid release of a totally naïve captive-born population. Vaccination of a portion of the wild foxes against CDV and rabies should also be considered where the remaining wild populations are at critically low numbers. If possible, efforts should be made to allow natural cycling of less pathogenic viruses already in the ecosystem to help protect against future epidemics, as long-term vaccination of island foxes will likely become impractical once populations have recovered. The suspected susceptibility to CDV and absence of documented morbidity or mortality in foxes exposed to other pathogens strongly argue for continued intensive disease surveillance. This surveillance will provide the basis for management decisions on all the islands, especially during active recovery efforts.

Generalist pathogens like CDV, which can be maintained by both domestic and wild carnivores in an ecosystem, threaten the persistence of small populations of canids worldwide (Woodroffe et al., 2004). During a suspected disease epidemic, data from sick or dead animals are often incomplete as these individuals are difficult to detect and recover. Pathogen exposure can usually be determined in live animals, but must be interpreted with caution as exposure may not cause clinical illness or mortality. The development of sensitive diagnostic methods increases our ability to detect pathogen exposure in wild populations, a critical first step towards understanding disease risks to threatened species. By examining the relationship between pathogen exposure and spatial, temporal, and population demographic factors, along with multi-species host transmission dynamics, we can better understand the epidemiology of diseases and provide objective data on which to base conservation management decisions.

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