SEROPREVALENCE, BLOOD CHEMISTRY, AND PATTERNS OF CANINE PARVOVIRUS, DISTEMPER VIRUS, PLAGUE, AND TULAREMIA IN FREE-RANGING COYOTES (CANIS LATRANS) IN NORTHERN NEW MEXICO, USA

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Seroprevalence, Blood Chemistry, and Patterns of Canine Parvovirus, Distemper Virus, Plague, and Tularemia in Free-Ranging Coyotes (*Canis latrans*) in Northern New Mexico, USA

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ABSTRACT: Wildlife diseases have implications for ecology, conservation, human health, and health of domestic animals. They may impact wildlife health and population dynamics. Exposure rates of coyotes (*Canis latrans*) to pathogens such as *Yersinia pestis*, the cause of plague, may reflect prevalence rates in both rodent prey and human populations. We captured coyotes in north-central New Mexico during 2005–2008 and collected blood samples for serologic surveys. We tested for antibodies against canine distemper virus (CDV, *Canine morbillivirus*), canine parvovirus (CPV, *Carnivore protoparvovirus*), plague, tularemia (*Francisella tularensis*), and for canine heartworm (*Dirofilaria immitis*) antigen. Serum biochemistry variables that fell outside reference ranges were probably related to capture stress. We detected antibodies to parvovirus in 32/32 samples (100%), and to *Y. pestis* in 26/31 (84%). More than half 19/32 (59%) had antibodies against CDV, and 5/31 (39%) had antibodies against *F. tularensis*. We did not detect any heartworm antigens ($n = 9$). Pathogen prevalence was similar between sexes and among the three coyote packs in the study area. Parvovirus exposure appeared to happen early in life, and prevalence of antibodies against CDV increased with increasing age class. Exposure to *Y. pestis* and *F. tularensis* occurred across all age classes. The high coyote seroprevalence rates observed for CPV, *Y. pestis*, and CDV may indicate high prevalence in sympatric vertebrate populations, with implications for regional wildlife conservation as well as risk to humans via zoonotic transmission.

Key words: Canidae, predator, serology, zoonotic disease.

INTRODUCTION

Infectious diseases are important factors in wildlife ecology and conservation. Pathogen prevalence and transmission may affect individual fitness, population dynamics, and community assemblages (Herrera and Nunn 2019; Wilson et al. 2019). Interspecific disease transmission among wildlife species is common (Allison et al. 2013; Beineke et al. 2015), and clinical effects may vary among species, from highly prevalent but asymptomatic infections to population losses. For example, plague (caused by *Yersinia pestis*) can be enzootic with little to no morbidity in some carnivores, but causes localized epizootic population crashes in prairie dog (*Cynomys* spp.) populations (Pauli et al. 2006). Life history traits such as sociality, mobility, prey species, and individual variation in behavior influence exposure risk and clinical vulnerability to pathogens, and may determine a species' potency as a reservoir (McGee et al. 2006; Kappeler et al. 2015; McDonald et al. 2018; Herrera and Nunn 2019). Pathogen prevalence and distribution may influence conservation of threatened and endangered species and complicate reintroduction and management plans (Barnes 1993; Murray et al. 1999). Conservation plans that consider wildlife

disease may be further complicated by global climate change and land-use conversion, as the spatial distribution of pathogens and hosts shift in response to changing environmental conditions (Holt et al. 2009; Brown et al. 2011; Gottdenker et al. 2014; Reddell et al. 2021).

Many diseases affecting wild mammals are of interest to wildlife conservation, domestic livestock, and human epidemiology. Coyotes (*Canis latrans*) can carry pathogens causing zoonotic diseases (e.g., sylvatic plague, tularemia [*Francisella tularensis* infection], rabies, toxoplasmosis, Lyme disease, anaplasmosis; Izac et al. 2020) and pathogens that affect domestic animals (e.g., canine distemper virus [CDV; Paramyxoviridae: *Canine morbillivirus*], canine parvovirus (CPD; Parvoviridae: *Carnivore protoparvovirus 1*), and canine adenovirus (Deem et al. 2000; Gier et al. 2001). Conflict with humans often results in lethal removal of coyotes in many regions of the US (Knowlton et al. 1999), and hypothesized risk of zoonotic disease transmission has occasionally been cited as a cause of conflict (Clark and Wilson 1995).

Despite relatively high prevalence of several zoonotic diseases, presence of vulnerable species, and frequent human–wildlife interaction (USAF 2013), only plague has been investigated in coyotes in New Mexico, US (Bevins et al. 2021). Bevins et al. (2021) reported a 20.5% (95% confidence interval, 6.4–65.7) statewide prevalence of antibodies to plague across 17 mammalian species, including coyotes. Surveys for other pathogens in these coyotes have not been conducted.

We evaluated a coyote population in northern New Mexico for several pathogens and parasites in a region with potential for human–wildlife contact. Specifically, we examined prevalence in coyotes of antibodies against CPV, CDV, *Y. pestis*, *F. tularensis*, and of canine heartworm (Onchocercidae: *Dirofilaria immitis*) antigens. We tested for 1) differences among coyote pathogen seropositive rates; 2) variation in seroprevalence between sexes and among age classes; 3) differences in seroprevalences among coyote packs (i.e., family groups) and compared with transient coyotes; 4) patterns in accumulation of exposure through age classes of the number of different pathogens; and 5) relationships between antibody seroprevalence and coyote health condition as indicated by serum biochemistry panel variables.

MATERIALS AND METHODS

Study area

We conducted our study in the 360-km^2 Valles Caldera National Preserve (VCNP) in north-central New Mexico (Sandoval County; latitude 35°54′44.64″, longitude 106°30′48.24″). Elevations ranged between 2,450 and 3,400 m. The landscape included grassland meadow valleys (2,450–2,700 m) and ponderosa pine (*Pinus ponderosa*) and mixed-conifer forests (2,700–3,400 m). Mean annual precipitation was 594 mm ± 9.7 (SD), predominantly in the form of monsoon rains (June–September, 51%) and winter snow (November–March, 29%; Western Regional Climate Center 2022). Mean July temperature was 15.4 C, and mean January temperature was −4.8 C.

Coyote population

Life history, demographics, and population density of the coyote population in VCNP appeared similar to other coyote populations in the western US. Coyote packs consisted of an alpha male and alpha female with several subadult pups, comprising packs of 5–7 individuals (Gifford et al. 2017). Coyote territory size was mean 10.6 ± 2.2 (SD) km^2 (Gifford et al. 2017) and density was 0.6 coyotes/km^2 (S. Gifford, Utah State University, written communication, 2017). Data from another population in New Mexico (Sevilleta National Wildlife Refuge, Socorro County) also exhibited a mean annual density of 0.6/km^2 over 11 yr (1992–2002; density range of 0.2–2.0 coyotes/km^2; Parmenter 2013), and studies of coyote populations in Idaho produced a mean density of 0.7/km^2 (range 0.2–1.6 coyotes/km^2; Knowlton 1984) and 0.1–1.39/km^2 (Stoddart et al. 2001); hence, we assumed that the VCNP population was typical of regional coyote populations. The VCNP coyote population was protected from hunting, so we assumed that coyote social behaviors were not influenced by direct human activities. Coyote prey in VCNP included montane voles (*Microtus montanus*), neonate elk (*Cervus*

elaphus), cottontail rabbits (*Sylvilagus* spp.), beetles (Coleoptera), grasshoppers (Orthoptera), and prairie dogs (*Cynomys gunnisoni*) (Gifford et al. 2020).

Capture, blood collection, and analysis

We captured 11 coyotes between August and November 2005 using padded-foothold traps (Livestock Protection Company, Alpine, Texas; Oneida Victor, Euclid, Ohio, USA). Coyotes were anesthetized for handling with 10 mg/kg tiletamine hydrochloride and zolazepam hydrochloride (Zoetis, Inc., Kalamazoo, Michigan, USA; Kreeger 1999). We captured 19 coyotes in May 2006 and 13 in March 2008 using a net-gun fired from a helicopter (Barrett et al. 1982; Gese et al. 1987); coyotes captured via helicopter were processed without chemical immobilization. Coyotes were examined for skin lesions and fur defects, external parasites, injuries, and body condition. We measured body size and mass; collected hair samples; determined sex; and estimated age based on either postmortem tooth analysis (for animals that died later or after the study) or by comparing tooth-wear photos to photos of known-age individuals in the study (Gier 1968). We fitted coyotes with GPS (Lotek Wireless, Inc., New Market, Ontario) and very high frequency (VHF) collars (Advanced Telemetry Systems, Isanti, Minnesota), or VHF collars alone (Lotek Wireless, Inc., New Market, Ontario). Capture and handling protocols were approved by the Institutional Animal Care and Use Committees at Utah State University (No. 1338) and the National Wildlife Research Center (QA-1492). We drew blood from the cephalic or lateral saphenous vein of each individual using a 20-gauge, 2.5-cm needle and a 10-mL syringe, and separated it in one 5-mL plain serum tube, one 5-mL serum-separator tube (2005 only), and one EDTA tube (all years; Becton Dickinson, Rutherford, New Jersey, USA). We stored samples on ice in the field, in a refrigerator in the VCNP laboratory, and submitted them to the analytical laboratory within 1–3 d of collection. From 2005 to 2008, we collected 43 samples from 36 coyotes. Seven coyotes were sampled twice; these individuals were intentionally recaptured to retrieve store-on-board GPS collars. For some samples, certain tests were not run because of insufficient blood quantity. As we did not have access to laboratory-raised pathogen-free coyotes, our study did not include negative control coyotes in the testing and analyses.

Serology tests were conducted by the New Mexico Department of Agriculture's Veterinary Diagnostic Services in the New Mexico Scientific Laboratories, Albuquerque, New Mexico, US. For pathogen surveillance, we tested for heartworm antigens in 2005, antibodies against CDV, CPV and *Y. pestis* in 2006 and 2008, and antibodies against *F. tularensis* in 2008. We detected antibodies against CPV using a hemagglutination inhibition test (HI, based on Carmichael et al. 1980), CDV with an indirect florescent antibody test (IFA, based on Rose et al. 1992), *Y. pestis* using a Centers for Disease Control (CDC) hemagglutination/hemagglutinin inhibition test (HA/HI, as described in Chu 2000), and *F. tularensis* using a CDC micro-agglutination test (MAT, based on Massey and Mangiafico 1974). Heartworm presence was assayed using a heartworm recombinant antigen ELISA test kit (IDEXX SNAP® Canine Heartworm Antigen Test Kit, IDEXX Laboratories, Inc., Westbrook, Maine) following manufacturer's instructions; this test has 97% specificity, and 81–90% accuracy with <1% false positive (1/175 dog serum samples tested; Atkins 2003). Antibody titer thresholds indicating an exposure to a disease were defined by the testing laboratory as follows: *Y. pestis* 1:32, CDVr 1:32, CPV 1:80, and *F. tularensis* 1:64.

We submitted blood samples to Tri Core Reference, Las Cruces, New Mexico, US, for standard domestic dog biochemistry panels in all years and compared our samples to reference ranges taken from pen-raised coyotes (Rich and Gates 1979), free-range coyotes (Smith and Rongstad 1980, Miller et al. 2009), and domestic dogs (*Canis familiaris*) for biochemistry variables without published coyote reference ranges.

We calculated Wald binomial proportion confidence intervals for each measure of seroprevalence (Wallis 2013). In analyzing demographic patterns, disease prevalence rates were examined for 1) occurrences among the three coyote packs and a transient class; 2) differences between sexes (Z-test for proportions); 3) differences among age classes (Pearson's chi-squared test with Yates' continuity correction for testing <1.5-yr-olds vs. adults, and linear regression for seroprevalence across all age classes); and 4) the cumulative number of exposures to disease per coyote with age class (regression analysis). In addition, we tested

for relationships of serum biochemistry panel variables between serologically positive vs. negative coyotes for *Y. pestis*, CDV and *F. tularensis* using analysis of variance (ANOVA) and Bonferroni-corrected *P* values for multiple comparisons; we performed a $Log_{(10)}$ data transformation on chemistry panel variables to achieve normality (Shapiro and Wilk 1965), and all variables met variance homogeneity requirements using tests from Levene (1960), O'Brien (1981), and Brown and Forsythe (1974). All statistical tests were carried out using the software Statistix 10 (Analytical Software, Tallahassee, Florida, USA).

RESULTS

Pathogens

We tested 26 coyotes for CVD and CPV, plus six resamples (total of 32 tests). We found 19/32 samples had positive titers for CDV (59%; 95% confidence interval [CI], 42–76%) and 32/32 (100%) for CPV. Of the six coyotes resampled for CDV, three tested negative for anti-CDV antibodies in 2006 but were positive in 2008, and three were positive in both years. All six coyotes resampled for CPVs were positive in both 2006 and 2008. We found 26/31 tested for antibodies against *Y. pestis* and found 84%were positive (984%; 5% CI, 71–97%). Of the six coyotes resampled for plague, one had a positive titer result in 2006 and a negative result in 2008, while the other five had positive results in both 2006 and 2008. We found 5/13 coyotes antibody-positive against *F. tularensis* in 2008 (38%; 95% CI, 12–65). All nine heartworm screening tests were negative (n=8 coyotes, one resample).

Serum biochemistry

The serum biochemistry panel data comprised 33 samples from 30 coyotes (three coyotes were resampled on subsequent captures). Some blood samples were insufficient to run all 21 variables, as serology was prioritized. Results from our coyote samples and reference ranges for pen-raised and free-ranging coyotes and domestic dogs are listed in Table 1.

Of the 21 variables included in our serum biochemistry panels, 14 variables fell within the normal ranges of coyotes, and 14 of the variables fell within the normal range for dogs (Table 1). The most prominent abnormalities were elevations in lactate dehydrogenase (LDH) and total bilirubin compared to pen-raised coyotes and dogs, aspartate aminotransferase (AST) and alanine aminotransferase (ALT) compared to dogs, and direct bilirubin compared to coyotes.

In total, 28/33 (85%) of samples had low serum carbon dioxide levels (CO_2) compared to reference values for domestic dogs. Mild to moderate elevations in mean blood urea nitrogen (BUN) occurred without concurrent elevations in mean creatinine concentrations. All six electrolyte mean values were within normal limits for coyotes and dogs. Although mean total protein was normal, 3/32 (9%) results had high or elevated albumin levels. Overall glucose levels were comparable to both coyotes and dogs, but hyperglycemia occurred in 15/32 (6%) and hypoglycemia in 6/32 (19%) samples. Triglycerides were low in 5/32 (16%) of samples. The analyses of variance (ANOVAs) for chemistry panel results among seropositive and seronegative coyotes failed to produce significant relationships for any pathogen (Bonferroni-protected *P* values > 0.05; see Supplementary Materials Tables S1, S2, and S3).

Demographic patterns

A total of 19 coyotes were assigned to one of the three packs in the study area or could be classed as transient. Members of each of the three coyote packs (La Jara, Piñon, and East packs; Gifford 2013; Fig. 1) and transient coyotes produced positive titers against CPV, CDV, and *Y. pestis*. Antibodies against *F. tularensis* were detected in two members of the Piñon pack.

Although male coyotes tended to have higher seroprevalence values than females, these were not statistically distinguishable for any of the pathogens using the Z-test for proportions (M:F percentage seropositive: CDV 69:50, P=0.47; CPV 100:100, P=1.00; *Y. pestis* 93:75, P=0.33; *F. tularensis* 44:33,

TABLE 1. Results of serum biochemistry panels from coyotes (*Canis latrans*) in Valles Caldera National Preserve, New Mexico, USA, 2005–2008, compared to previously published reference values for coyotes and domestic dogs (*Canis familiaris*). Values = mean and (range).[e]

		Coyote reference values			Dog reference values[d]	Coyote data, this study
Analyte	SI unit	Pen-raised[a] $n = 48$	Wisconsin[b] $n = 40$	South Carolina[c] $n = 22$	US	New Mexico $n = 33$
Calcium	mmol/L	2.4 (2.1–2.8)	2.3	2.5 (2.2–2.7)	2.7 (2.4–3.0)	2.3 (1.8–2.6)
Chloride	mmol/L			115 (106–122)	114 (110–118)	118 (111–129)
Magnesium	mmol/L				0.84 (0.70–0.99)	0.96 (0.74–1.64)
Phosphorus	mmol/L	1.2 (0.6–2.0)	1.6	1.8 (0.9–3.2)	1.6 (0.7–2.6)	1.8 (0.8–3.1)
Potassium	mmol/L	4.9 (4.1–6.2)		4.2 (3.0–5.7)	4.2 (3.5–5.0)	4.9 (3.9–6.4)
Sodium	mmol/L	145 (135–154)		153 (147–161)	143 (138–148)	145 (141–150)
CO_2	mmol/L				21.0 (16.0–26.0)	10.5 (2.5–18.0)
Glucose	mmol/L	7.0 (4.4–10.6)	9.1	5.6 (0.8–10.5	5.9 (3.7–8.2)	6.5 (3.0–8.7)
Triglyceride	mmol/L			1.05 (0.47–1.53)	0.77 (0.24–1.31)	0.77 (0.23–3.02)
Total bilirubin	µmol/L	4.3 (1.7–6.8)	2.1	2.9 (1.7–8.6)	6.8 (1.7–12.0)	12.0 (1.7–63.3)
Direct bilirubin	µmol/L	0.7 (0.2–1.9)				10.3 (1.7–32.5)
Creatinine	µmol/L	115 (71–186)		67 (35–106)	88 (44–133)	72 (35–115)
Total protein	g/L	65 (55–72)	64	75 (51–96)	57 (48–66)	62 (54–87)
BUN	g/L	0.27 (0.12–0.40)	0.21	0.40 (0.10–1.03)	0.18 (0.04–0.30)	0.34 (0.12–0.72)
Albumin	g/L	27 (19–34)	30	32 (25–40)	31 (23–39)	33 (28–51)
Globulin	g/L	37 (29–43)	34	37 (29–43)		29 (24–36)
LDH	U/L	143 (54–443)			894 (105–1,683)	2,764 (340–9,591)
AST (SGOT)	U/L				19 (1–37)	151 (51–616)
ALT (SGPT)	U/L	155 (28–346)			26 (3–50)	90 (39–179)
GGT	U/L				15 (5–25)	12.9 (6.0–38.0)
Alkaline phosphatase	U/L	35 (20–119)		80 (0–275)	88 (20–155)	49 (21–159)

[a] From Rich and Gates (1979); published values converted to SI units.
[b] From Smith and Rongstad (1980); published values converted to SI units.
[c] From Miller et al. (2009); published values converted to SI units.
[d] From TriCore-Laboratories reports.
[e] BUN = blood urea nitrogen; LDH = lactate dehydrogenase; AST = aspartate aminotransferase; SGOT = serum glutamic-oxaloacetic transaminase; ALT = alanine aminotransferase; SGPT = serum glutamic-pyruvic transaminase; GGT = gamma-glutamyl transferase.

P=1.00; *D. immitis* tests were all negative). We found no differences in prevalence of any of the five pathogens between juveniles (<1.5 yr) and adults (≥1.5 yr; P>0.23). Antibodies against CPV were observed in all coyotes across all age classes (Fig. 2A). We found antibodies against CDV in increasing frequency with age (linear regression $CDV+ = 0.275Age - 0.083$, r^2=0.995, P<0.00001, Fig. 2B). Antibodies showing exposure to *Y. pestis* occurred commonly in all age classes, and by age 4+ yr, 100% of coyotes had been exposed sometime during their lives (Fig. 2C). Our small sample size for antibodies against *F. tularensis* (5/13 positive, 8/13 negative) prevented adequate testing for age-exposure patterns. Finally, coyotes showed no pattern of increasing cumulative exposure to pathogens with age (r^2=0.0003).

DISCUSSION

The results of our serum biochemistry analyses and serological survey indicate that coyotes in VCNP exhibit generally normal values for most analytes but relatively high seropositivity rates for several pathogens. Despite serological evidence of exposure to the study pathogens, coyotes appeared healthy (i.e., active and alert) at the time of sample

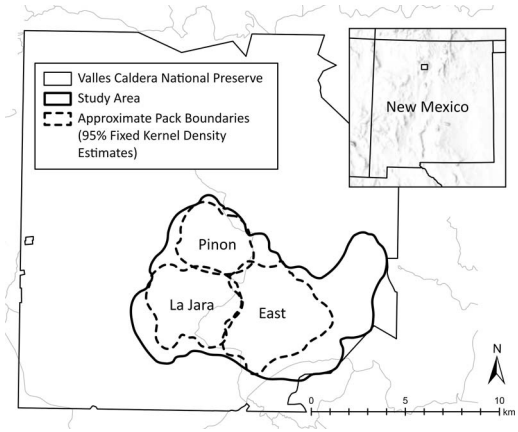

FIGURE 1. Delineation of the study area where blood samples were collected from coyotes (*Canis latrans*) from 2005 to 2008, and boundaries of resident coyote pack home ranges during 2006 in Valles Caldera National Preserve, New Mexico, USA. Adapted from Gifford et al. (2017).

collection, although some serum analytes indicated increased stress levels from being captured. Many of the diseases we evaluated may be enzootic, contributing little to mortality within a population during favorable environmental conditions (Trainer and Knowlton 1968). Although the VCNP coyote population exhibits high exposure to viral and bacterial diseases, it is unknown whether coyotes function as reservoirs for further transmission to other vertebrate wildlife species.

Serum biochemistry

Most of the deviations in hepatic, renal, and electrolyte serum biochemistry levels from normal ranges were probably capture-related. Elevated LDH and AST in dogs commonly result from overexertion or muscle trauma (Bedrak 1965; Burr et al. 1997). Although ALT is primarily liver-specific, transient elevations related to capture cannot be ruled out; sample hemolysis, toxins, some anesthetic agents, infections, neoplasia, and direct liver injury from trauma, shock, or dehydration in dogs can all cause elevated ALT (Center 2007). Normal alkaline phosphatase and gamma-glutamyl transferase levels support that these coyotes did not have

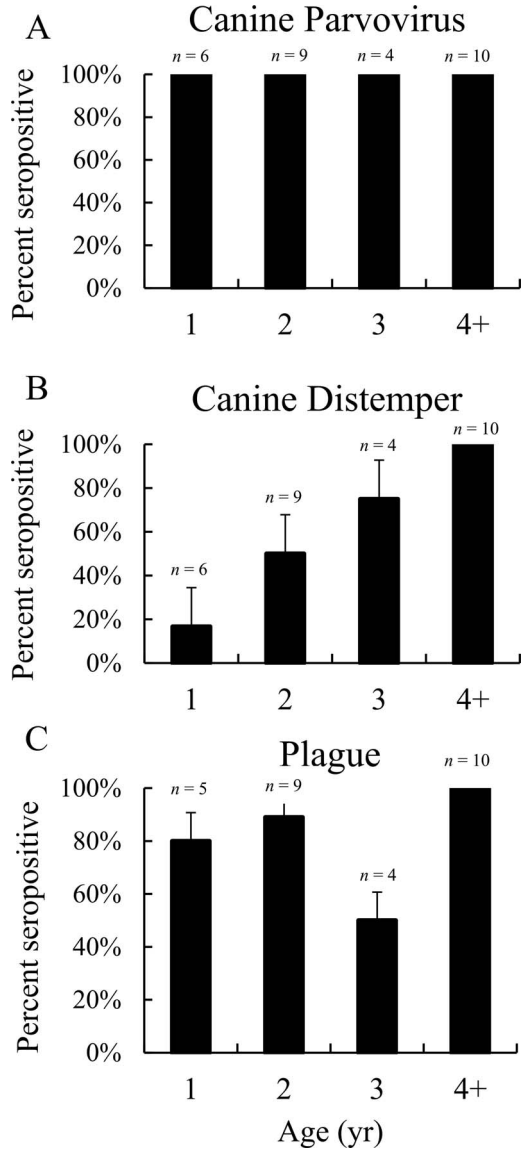

FIGURE 2. Influence of age on antibody seroprevalence of (A) canine parvovirus, (B) canine distemper virus, and (C) plague in coyotes (*Canis latrans*) in Valles Caldera National Preserve, New Mexico, USA, 2005–2008. Includes three resampled individuals; error bars are standard error.

severe, underlying hepatic disease; renal function also appeared to be normal compared to dogs (Fernandez and Kidney 2007). In the absence of azotemia, BUN elevations were probably due to dehydration or high-protein diet (Finco and Duncan 1976). In all coyotes, low CO_2 levels appeared to be the result of

exertion or overheating during capture (Casaburi et al. 1979). Hyperglycemia in some of the coyote samples is consistent with physiologic stress (Miles et al. 1991). Low triglyceride levels, as seen in some of our coyotes, are associated with inadequate food intake in dogs (Usui et al. 2015) and may indicate potential malnutrition in some VCNP coyotes.

Canine distemper virus and canine parvovirus virus

The prevalence of CDV antibodies in the VCNP coyote population (59%) was within ranges found in other US states. Reported rates in previous studies ranged from 10 to 23% in Utah (Arjo et al. 2003), 15% in South Carolina (Miller et al. 2009), 17% in North Carolina (Chitwood et al. 2015), 25% in Pennsylvania (Kimpston et al. 2022), 27% in Arizona (Grinder and Krausman 2001), 27% in South Dakota (Schuler et al. 2021), 37% in California (Cypher et al. 1998), 44% in urban environments in Denver, Colorado (Malmlov et al. 2014), 48% in Georgia (Gates et al. 2014), 56 to 61% in Texas, Nebraska, and Colorado (Guo et al. 1986; Gese et al. 1991; Bischof and Rogers 2005), and 76% in Wyoming, all US (Gese et al. 1997). Previous serologic surveys reported lower prevalence in juveniles than adults (Guo et al. 1986; Grinder and Krausman 2001; Arjo et al. 2003), similar to our observed pattern across age classes (Fig. 2B).

Canine parvovirus infection is a recently emerged disease, appearing in the mid-20th century (Allison et al. 2013). Since then, the disease has become enzootic in many populations of wild carnivores in the US. Where it is enzootic, it probably only causes high mortality during stressful environmental conditions such as drought and ensuing limited prey abundance (Trainer and Knowlton 1968). Canine parvovirus spreads by ingestion of an infected animal's feces or vomit, and stays viable in the environment for several months. Cross-species transmission is common (Allison et al. 2013). In coyotes, CPV infection has caused mortality by hemorrhagic enteritis (Evermann et al. 1980; Holzman et al. 1992). All 32 coyotes tested in the VCNP were seropositive for CPV. Reported rates from the US ranged from 46% in Pennsylvania (Kimpston et al. 2022), to 66 to 77% in California and Colorado (Cypher et al. 1998; Gese 2004), 91% in Oregon (Dunbar and Giordano 2003), 96% in North Carolina (Chitwood et al. 2015) and 100% in Utah, Wyoming, Arizona, and Georgia, US (Gese et al. 1997; Grinder and Krausman 2001; Arjo et al. 2003; Gates et al. 2014), and 84% within the Denver, Colorado, metropolitan area (Malmlov et al. 2014).

Tularemia

Tularemia mainly affects lagomorphs and rodents but can also infect humans and wild and domestic carnivores (CDC 2013). Antibody prevalence to *F. tularensis* observed in the VCNP coyote population (38.5%) was relatively high. Reported rates in coyotes in US states range from 0% in South Dakota (Schuler et al. 2021) and 0–4% in Texas, Utah, and Quebec (Trainer and Knowlton 1968; Arjo et al. 2003; Gabriele-Rivet et al. 2016), to 21% in Wyoming (Gese et al. 1997), and 32% in Nebraska (Bischof and Rogers 2005). Coyotes, especially adults, can be healthy carriers of the pathogen (Lundgren et al. 1957), but it can be fatal to pups (Gier et al. 2001). Three cases of *F. tularensis* transmission from coyotes to humans have been documented—two from coyote bites (Parker and Francis 1926; Chomel et al. 2016) and one from disposal of a coyote carcass in New Mexico, US (Kunkel 1930).

Canine heartworm

Canine heartworm affects wild and domestic canids, and coyotes can be a reservoir for transmission of *D. immitis* to domestic dogs (Weinmann and Garcia 1980). None of the nine coyote samples in 2005 had evidence of the presence of . *D. immitis*. This finding is consistent with previous continental-scale analyses of heartworm infection frequency (Brown et al. 2012; Self et al. 2019), indicating that northern New Mexico has a very low

infection occurrence. We note, however, that these screening tests detect antigen from adult female worms. False-negative results may occur because of the type of test being used, the presence of immature heartworm infections (prepatent period 6–7 mo), male-only heartworm infections, or single-worm infections (Atkins 2003; Sobotyk et al. 2022). Even incorporating potential testing bias, if current regional trends in heartworm distributions continue, the frequency of heartworm exposure in VCNP coyotes will probably increase (Self et al. 2019).

Plague

Plague is a significant concern for human health, and many wildlife species serve as reservoirs (e.g., canids, felids, and mustelids; Salkeld and Stapp 2006). The prevalence of antibodies indicating past or present *Y. pestis* infections in the VCNP coyote population is high (86% over all age classes and 100% in coyotes 4 yr or older). During our study, concurrent surveys of *Y. pestis* in VCNP flea (*Oropsylla* spp.) and rodent communities found two plague epizootics in prairie dog colonies in 2004 (Friggens et al. 2010). In 2005–2006, they found *Y. pestis*–positive fleas and prairie dogs with *Y. pestis* antibodies (Friggens et al. 2010). Across the US, reported seroprevalence in coyotes ranges from 0 to 73% in California, Utah, and Wyoming (Arjo et al. 2003), 12% in California (Hoar et al. 2003), 13% in South Dakota (Schuler et al. 2021) and 28% in urban environments in Denver, Colorado (Malmlov et al. 2014). Bevins et al. (2021) reported 8.5% plague seroprevalence in New Mexico's coyotes, and 20.5% exposure rates to plague among all mammals monitored in New Mexico.

Wet winters and springs may be accompanied by elevated frequency of human plague cases, presumably resulting from a trophic cascade leading to increases in small mammal abundance, coupled with enhanced flea survival (Parmenter et al. 1999). The years preceding our study (2004–2006) had normal mean winter–spring (October–May) precipitation, but 2007–2008 winter-spring precipitation (368 mm) was 26% higher than the long-term average (293 mm). Winter-spring precipitation and resulting rodent abundance and flea survival may influence coyote exposure to plague similarly to humans, but further longitudinal monitoring is necessary.

Coyotes infected with *Y. pestis* show few, if any, clinical signs (Vernati et al. 2011; Baeten et al. 2013). They become infected by consuming infected rodents or lagomorphs, or by being bitten by infected fleas (CDC, 2022). The pathogen can be directly transmitted from infected coyotes to humans by exposure (e.g., while skinning coyote carcasses; von Reyn et al. 1976). Antibody titers in coyotes will indicate recent exposure to plague (up to 6 mo postexposure), and they have been used as sentinel species to monitor plague (Willeberg et al. 1979; Buller et al. 2018). Seroprevalence in coyotes can accurately reflect infection rates in humans (Willeberg et al. 1979) and in coyotes' prey species (Gese et al. 1997). Because most new human cases of plague originate in New Mexico (CDC 2022; New Mexico Department of Health 2020), continued monitoring of coyote infection prevalence could serve as an index to plague dynamics in the environment.

Plague may threaten conservation of several species of concern in the southwestern US. Gunnison and black-tailed prairie dogs (*Cynomys gunnisoni, Cynomys ludovicianus*) are both considered species of greatest conservation need by the New Mexico Department of Game and Fish and act as amplifying hosts of *Y. pestis*, with rapid spread and colony die-off (Conover and Vail 2015). Federally endangered black-footed ferrets (*Mustela nigripes*) are vulnerable to plague-driven population crashes in prairie dogs (Barnes 1993; Shoemaker et al. 2014) plus direct mortality from plague (Matchett et al. 2010). Adult pumas (*Puma concolor*) in VCNP from 2014–2017 had a 75% prevalence ($n = 6/8$) of antibodies against *Y. pestis* (R. Parmenter and M. Peyton, National Park Service, pers. comm.). Plague can be a significant source of mortality in puma populations (Elbroch et al. 2020), which may have

outsized impacts on local ecosystems (Ripple et al. 2014). Reintroduced populations of federally threatened Canada lynx (*Lynx canadensis*) also are vulnerable to plague-induced mortality (Wild et al. 2006; Devineau et al. 2010). As a result of landscape changes, fragmented populations, and increased domestic animal interactions, disease is likely to play a larger role in future conservation considerations of felids (Munson et al. 2010). Close monitoring and additional research are needed to understand the potential impacts of plague on wildlife conservation better.

Baseline seroprevalence and pathogen distribution surveys will assist in monitoring shifts in pathogens related to global change (Buller et al. 2018). The present work provides a valuable baseline for monitoring infectious wildlife diseases in northern New Mexico. However, serologic tests indicate previous exposure to a disease, not current infection. Given the limited spatial and temporal scale of this study, further monitoring is needed to understand spatial and temporal dynamics of these diseases for regional conservation efforts. Additional research could focus efforts on identifying the full range of diseases present on the landscape and attempt to further understanding of the implications for wildlife conservation efforts.

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SUPPLEMENTARY MATERIAL

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