



Serologic Survey for Canine Infectious Diseases among Sympatric Swift Foxes (*Vulpes velox*) and Coyotes (*Canis latrans*) in Southeastern Colorado

Authors: Gese, Eric M., Karki, Seija M., Klavetter, Mead L., Schauster, Edward R., and Kitchen, Ann M.

Source: Journal of Wildlife Diseases, 40(4) : 741-748

Published By: Wildlife Disease Association

URL: <https://doi.org/10.7589/0090-3558-40.4.741>

BioOne Complete (complete.BioOne.org) is a full-text database of 200 subscribed and open-access titles in the biological, ecological, and environmental sciences published by nonprofit societies, associations, museums, institutions, and presses.

Your use of this PDF, the BioOne Complete website, and all posted and associated content indicates your acceptance of BioOne's Terms of Use, available at www.bioone.org/terms-of-use.

Usage of BioOne Complete content is strictly limited to personal, educational, and non - commercial use. Commercial inquiries or rights and permissions requests should be directed to the individual publisher as copyright holder.

BioOne sees sustainable scholarly publishing as an inherently collaborative enterprise connecting authors, nonprofit publishers, academic institutions, research libraries, and research funders in the common goal of maximizing access to critical research.

Serologic Survey for Canine Infectious Diseases among Sympatric Swift Foxes (*Vulpes velox*) and Coyotes (*Canis latrans*) in Southeastern Colorado

Eric M. Gese,^{1,5} Seija M. Karki,² Mead L. Klavetter,^{2,3} Edward R. Schauster,^{2,4} and Ann M. Kitchen² ¹ US Department of Agriculture, Wildlife Services, National Wildlife Research Center, Department of Forest, Range, and Wildlife Sciences, Utah State University, Logan, Utah 84322, USA; ² Department of Forest, Range, and Wildlife Sciences, Utah State University, Logan, Utah 84322, USA; ³ Current address: US Fish and Wildlife Service, Colorado Fish and Wildlife Assistance Office, 36086 US Hwy. 350, Model, Colorado 81059, USA; ⁴ Current address: Science Applications International Corporation, PO Box 202, Driggs, Idaho 83422, USA; ⁵ Corresponding author (email: egese@cc.usu.edu)

ABSTRACT: Swift foxes (*Vulpes velox*) and coyotes (*Canis latrans*) are sympatric canids distributed throughout many regions of the Great Plains of North America. The prevalence of canid diseases among these two species where they occur sympatrically is presently unknown. From January 1997 to January 2001, we collected blood samples from 89 swift foxes and 122 coyotes on the US Army Piñon Canyon Maneuver Site, Las Animas County, SE Colorado (USA). Seroprevalence of antibodies against canine parvovirus (CPV) was 71% for adult (>9 mo old) and 38% for juvenile (≤9 mo old) swift foxes. Adult (≥1 yr old) and juvenile (<1 yr old) coyotes had a seroprevalence for CPV of 96% and 78%, respectively. Presence of antibodies against canine distemper virus (CDV) was 5% for adult foxes and 0% for juvenile foxes. Seroprevalence of CDV was 46% for adult coyotes and 18% for juvenile coyotes. No swift foxes had canine adenovirus (CAV) antibodies, whereas 81% and 63% of adult and juvenile coyotes, respectively, had antibodies for CAV. Seroprevalence of antibodies against *Yersinia pestis* was 68% among adult foxes and 34% among juvenile swift foxes. Seroprevalence of *Y. pestis* antibodies was 90% and 70% for adult and juvenile coyotes, respectively. No swift foxes had antibodies against *Francisella tularensis*, whereas seroprevalence was 4% among both adult and juvenile coyotes. Antibodies against CPV and plague were common in both species, whereas antibodies against CDV and CAV were more prevalent in coyotes compared to swift foxes.

Key words: Canine adenovirus, canine distemper virus, canine parvovirus, *Canis latrans*, coyote, *Francisella tularensis*, plague, swift fox, tularemia, *Vulpes velox*, *Yersinia pestis*.

Swift foxes (*Vulpes velox*) are small-sized canids that historically occupied much of the short- and mixed-grass prairie of North America (Scott-Brown et al., 1987). Changes in landscape practices

(conversion of prairie to agriculture), predator control, indiscriminate shooting, rodent control programs, and predation by domestic dogs brought about a general decline in swift fox abundance and distribution (Scott-Brown et al., 1987). Presently, swift foxes are distributed over a restricted part of their former range (Scott-Brown et al., 1987). Coyotes (*Canis latrans*) are medium-sized canids that occupy most habitats and regions of North America and are sympatric with swift foxes. Predation by coyotes is a leading cause of mortality in many swift fox populations (Covell, 1992; Sovada et al., 1998; Schauster et al., 2002). Resource partitioning between these two species has been documented to be intense, with coyotes possibly influencing the abundance and distribution of swift foxes across local landscapes (Kitchen et al., 1999; Kamler, 2002; Schauster et al., 2002; Karki, 2003). Whether disease plays a role in the relationship and interactions between these two sympatric canid species is unknown.

The prevalence of antibodies against various infections has been reported for many populations of coyotes. In US states containing both swift foxes and coyotes, antibodies against viral and bacterial infections have been reported for coyotes in Kansas (Gier and Ameel, 1959), Texas (Thomas et al., 1984), Colorado (Gese et al., 1991), and Wyoming (Williams et al., 1988; Gese et al., 1997). Unfortunately, there has been only one population of swift foxes sampled for canid infectious diseases and parasites (Miller et al., 2000).

There has been no survey of disease in coyotes and swift foxes in the same location during the same time period of sampling. We report results of a serologic survey for antibodies against canine parvovirus (CPV), canine distemper virus (CDV), canine adenovirus (CAV), *Yersinia pestis*, and *Francisella tularensis* among sympatric swift foxes and coyotes sampled during the same time period in SE Colorado.

Swift foxes and coyotes were sampled on the 1,040-km² US Army Piñon Canyon Maneuver Site (PCMS), Las Animas County, SE Colorado (37°20'N, 103°40'W). Elevation on the study area was 1,310–1,740 m above sea level. The climate is classed as midlatitude semiarid, with mean monthly temperatures of –1 C in January to 23 C in July (Andersen and Rosenlund, 1991). Annual precipitation averages about 32 cm (US Department of Army, 1980). The topography consists of broad, moderately sloping uplands, limestone hills, and sandstone canyons (Gese et al., 1988). Vegetation is dominated by short-grass prairie and pinyon pine (*Pinus edulis*) and one-seeded juniper (*Juniperus monosperma*) woodland communities (Costello, 1954; Shaw et al., 1989). Grasslands are composed of blue grama (*Bouteloua gracilis*), sideoats grama (*B. curtipendula*), western wheatgrass (*Agropyron smithii*), galleta (*Hilaria jamesii*), and needle-and-thread (*Stipa comata*).

Swift foxes were captured with box traps baited with chicken or an enclosure trap system (Covell, 1992; Schauster et al., 2002). A 3–4-ml blood sample was extracted from captured foxes via the jugular vein. Captured foxes were weighed, ear-tagged, aged by tooth wear (Rongstad et al., 1989), radio-collared (Advanced Telemetry Systems, Isanti, Minnesota, USA), and released at the capture site. Foxes were handled without anesthesia. Swift foxes were classified as juveniles (≤ 9 mo old) or adults (> 9 mo old).

Coyotes were sampled by capture with a hand-held net gun (Barrett et al., 1982; Gese et al., 1987) or during aerial gunning.

A 10–12-ml blood sample was extracted via the cephalic or saphenous vein from captured coyotes or by cardiac puncture from dead coyotes. All captured coyotes were weighed, sex determined, aged by tooth wear (Gier, 1968), ear-tagged, radio-collared (Advanced Telemetry Systems), and released. Captured coyotes were handled without chemical immobilization. For coyotes removed by aerial gunning, we determined their weight and sex and extracted a canine tooth for aging by cementum annuli analysis (Linhart and Knowlton, 1967). Coyotes were classed as juveniles (≤ 12 mo old) or adults (> 12 mo old).

Each blood sample was placed into a glass serum tube (Vacutainer, Becton Dickinson, Rutherford, New Jersey, USA) and centrifuged for 30 min; the serum was harvested and stored at –20 C. Serum samples were analyzed for antibodies against CDV, *Y. pestis*, and *F. tularensis* at the Wyoming State Veterinary Laboratory (University of Wyoming, Laramie, Wyoming, USA). Analyses for CPV and CAV antibodies were conducted at the Washington Animal Disease Diagnostic Laboratory (Washington State University, Pullman, Washington, USA). CDV antibody was detected by the serum virus neutralization test described by Appel and Robson (1973). An antibody titer $\geq 1:10$ was considered to be positive for antibodies against CDV. Antibodies against CPV were detected using an indirect fluorescent antibody test (Rose et al., 1992). A titer of $\geq 1:25$ was considered positive for CPV antibodies. Antibodies against CAV were detected by the virus neutralization test (Appel et al., 1975) which does not distinguish between CAV types 1 and 2. A titer level of $> 1:4$ was considered to be positive. To determine the prevalence of antibodies against *Y. pestis*, we used passive hemagglutination and inhibition tests and an enzyme-linked immunosorbent assay (Chu, 2000); a titer of $\geq 1:16$ was considered to be positive. We used the microscopic agglutination test as described by Gese et al. (1997) for detecting antibodies against *F.*

TABLE 1. Prevalence of antibodies against selected canine infection disease agent of sympatric swift foxes and coyotes, Piñon Canyon Maneuver Site, southeastern Colorado, 1997–2001.

	CPV ^a	CDV ^b	CAV ^c	<i>Y. pestis</i>	<i>E. tularensis</i>
Swift foxes					
Adult females	85 (26) ^d	12 (26)	0 (26)	70 (27)	0 (27)
Adult males	61 (33)	0 (33)	0 (33)	67 (33)	0 (33)
Juvenile females	43 (14)	0 (14)	0 (14)	29 (14)	0 (14)
Juvenile males	33 (15)	0 (14)	0 (15)	40 (15)	0 (15)
Coyotes					
Adult females	92 (37)	35 (37)	87 (37)	96 (26)	0 (26)
Adult males	99 (58)	54 (56)	78 (58)	86 (44)	7 (42)
Juvenile females	78 (18)	17 (18)	56 (18)	64 (14)	7 (14)
Juvenile males	78 (9)	22 (9)	78 (9)	78 (9)	0 (9)

^a CPV = canine parvovirus type 2.^b CDV = canine distemper virus.^c CAV = canine adenovirus (types 1 and 2).^d % positive (sample size).

tularensis; a titer of $\geq 1:128$ was considered to be positive.

For all statistical tests, the sampling unit was the individual fox or coyote; all animals were represented by one sample. The χ^2 test was used to analyze the prevalence of antibodies between age classes, sexes, and species (Sokal and Rohlf, 1981). We used a Fisher's exact test when the contingency table contained an expected frequency of <1 in any cell (Zar, 1996). All statistical tests were performed using SPSS (SPSS Base 10; SPSS, Inc., Chicago, Illinois, USA).

Blood was collected from 89 swift foxes (48 males and 41 females) and 122 coyotes (67 males and 55 females) sampled from January 1997 to January 2001. Age classes of the swift foxes sampled were 60 adults (33 males and 27 females) and 29 juveniles (15 males and 14 females). Age classes of the coyotes sampled were 95 adults (58 males and 37 females) and 27 juveniles (nine males and 18 females). Thirty-one animals were sampled in 1997, 1 in 1998, 58 in 1999, 117 in 2000, and 4 in 2001.

Laboratory analysis for seroprevalence of CPV antibodies was completed on serum samples from 88 swift foxes and 122 coyotes. Among swift foxes, the overall seroprevalence of CPV antibodies was 60%; reciprocal antibody titers ranged from <25

to 1,600. Juvenile foxes (38%) had a lower prevalence of CPV antibodies, compared with adults (71%; $\chi^2=8.98$, 1 df, $P=0.0027$). Among juvenile foxes, there was no difference in seroprevalence between males and females (Table 1; $\chi^2=0.28$, 1 df, $P=0.597$). In contrast, adult female foxes had a higher seroprevalence than adult males (Table 1; $\chi^2=4.09$, 1 df, $P=0.043$). Among coyotes, the overall prevalence of CPV antibodies was 92%, with titers ranging of 10–5,120. Seroprevalence was lower among juvenile coyotes (78%) than among adult coyotes (96%; $\chi^2=9.06$, 1 df, $P=0.0026$). Among adult coyotes, there was no difference in the seroprevalence between the sexes (Table 1; adults: $\chi^2=2.28$, 1 df, $P=0.131$). Seroprevalence among juvenile coyotes was identical between the sexes (Table 1). When comparing the two canid species, the overall prevalence of CPV antibodies in swift foxes (60%) was lower than in coyotes (92%; $\chi^2=30.27$, 1 df, $P=0.0001$). Controlling for the influence of age, adult coyotes (96%) had a higher seroprevalence than adult swift foxes (71%; $\chi^2=18.71$, 1 df, $P=0.0001$). Juvenile coyotes (78%) also had a higher seroprevalence than juvenile foxes (38%; $\chi^2=9.06$, 1 df, $P=0.0026$).

Serologic testing for CDV antibodies was completed on 87 swift foxes and 120

coyotes. Overall, we found that the prevalence of CDV antibodies was 3% among swift foxes, with reciprocal titers of <4–256. Seroprevalence was 5% among adult foxes and 0% among juvenile foxes ($P>0.20$, Fisher's exact test). Seroprevalence was 11% among adult female foxes and 0% among adult male foxes (Table 1; $\chi^2=3.59$, 1 df, $P=0.058$). Among coyotes, the prevalence of CDV antibodies was 40% for all animals combined, with titers of <4–512. Juvenile coyotes (18%) had a lower seroprevalence than adult coyotes (46%; $\chi^2=6.70$, 1 df, $P=0.001$). Among both adult and juvenile coyotes, seroprevalence was not different between the sexes (Table 1; adults, $\chi^2=3.05$, 1 df, $P=0.081$; juveniles, $\chi^2=0.12$, 1 df, $P=0.72$). When comparing the two canid species, overall seroprevalence among coyotes (40%) was higher than among swift foxes (3%; $\chi^2=36.29$, 1 df, $P=0.0001$). Adult coyotes (46%) had a higher seroprevalence than adult foxes (5%; $\chi^2=28.97$, 1 df, $P=0.0001$), and seroprevalence in juvenile coyotes (18%) was higher than that in juvenile foxes (0%; $\chi^2=5.70$, 1 df, $P=0.017$).

Prevalence of CAV antibodies was determined using samples from 88 swift foxes and 122 coyotes. No swift foxes were seropositive for CAV antibodies (Table 1). In contrast, the overall prevalence of CAV antibodies was 77% for all coyotes combined, with titers of 4 to >512. Seroprevalence was 63% among juvenile coyotes and 81% among adult coyotes ($\chi^2=3.89$, 1 df, $P=0.049$). Seroprevalence was similar between the sexes for both adult and juvenile coyotes (Table 1; adults, $\chi^2=1.165$, 1 df, $P=0.28$; juveniles, $\chi^2=1.27$, 1 df, $P=0.26$). The overall prevalence of CAV antibodies was different between swift foxes (0%) and coyotes (77%; $\chi^2=122.74$, 1 df, $P<0.0001$). Among the age classes, seroprevalence was different between adult coyotes (81%) and adult foxes (0%; $\chi^2=95.64$, 1 df, $P<0.0001$), as well as between juvenile coyotes (63%) and juvenile swift foxes (0%; $\chi^2=26.21$, 1 df, $P<0.0001$).

We analyzed serum samples from 89 swift foxes and 93 coyotes for antibodies against *Y. pestis*. The overall prevalence was 57% among all swift foxes. Seroprevalence varied between juvenile (34%) and adult swift foxes (68%; $\chi^2=9.16$, 1 df, $P=0.0025$). Seroprevalence was similar between the sexes for both adult and juvenile swift foxes (Table 1; adults, $\chi^2=0.09$, 1 df, $P=0.76$; juveniles, $\chi^2=0.42$, 1 df, $P=0.52$). The overall prevalence of *Y. pestis* antibodies among all coyotes was 85%. Seroprevalence was 70% among juveniles and 90% among adult coyotes ($\chi^2=5.65$, 1 df, $P=0.017$). Seroprevalence was similar between the sexes for both adult and juvenile coyotes (Table 1; adults, $\chi^2=1.74$, 1 df, $P=0.19$; juveniles, $\chi^2=0.47$, 1 df, $P=0.49$). The overall prevalence of *Y. pestis* antibodies was different between swift foxes (57%) and coyotes (85%; $\chi^2=17.03$, 1 df, $P=0.0001$). Controlling for the influence of age, seroprevalence was different between adult coyotes (90%) and adult foxes (68%; $\chi^2=9.48$, 1 df, $P=0.0021$) and between juvenile coyotes (70%) and juvenile swift foxes (34%; $\chi^2=6.31$, 1 df, $P=0.012$).

Serum samples from 89 swift foxes and 91 coyotes were analyzed for *F. tularensis* antibodies. No swift foxes had *F. tularensis* antibodies (Table 1). For coyotes, the overall seroprevalence was 4%. Seroprevalence was 4% among juvenile and 4% among adult coyotes ($\chi^2=0.0002$, 1 df, $P=0.99$). Seroprevalence was similar between the sexes for both adult and juvenile coyotes (Table 1; adults, $\chi^2=1.94$, 1 df, $P=0.16$; juveniles, $P>0.30$, Fisher's exact test). The overall prevalence of *F. tularensis* antibodies was different between swift foxes (0%) and coyotes (4%; $\chi^2=4.001$, 1 df, $P=0.045$). Among the age classes, seroprevalence was not different between adult coyotes (4%) and adult foxes (0%; $\chi^2=2.71$, 1 df, $P=0.10$) or between juvenile coyotes (4%) and juvenile swift foxes (0%; $P>0.20$, Fisher's exact test).

The prevalence of CPV antibodies was high in coyotes (92%) and was relatively lower among swift foxes (60%). Among

both species, adults had higher prevalence of CPV antibodies than younger animals, which indicates that the adults likely had been exposed to CPV, or a closely related parvovirus, and survived. Compared with previous samples from the same study area collected in different time periods, the prevalence of CPV antibodies increased in both swift foxes (from 39%; Miller et al., 2000) and coyotes (from 71%; Gese et al., 1991). The prevalence of CPV antibodies among San Joaquin kit foxes (*V. macrotis nutica*), a close relative of the swift fox (Mercure et al., 1993), ranged 67–100% in California (McCue and O'Farrell, 1988). The prevalence of CPV antibodies in coyotes from other western states (Arizona, Idaho, Utah, and Wyoming) was generally >70% (Thomas et al., 1984; Gese et al., 1997; Grindler and Krausman, 2001; Arjo et al., 2003). A high prevalence of antibodies is associated with a highly contagious, but nonfatal, infection, because prevalence is measured among survivors (Thomas et al., 1984). The impact of CPV infection on canid populations is largely unknown. However, evidence of CPV infection has been implicated as a mortality agent among young coyotes (Gese et al., 1997) and wolves (*C. lupus*) (Mech and Goyal, 1993; Johnson et al., 1994).

The overall prevalence of CDV antibodies among swift foxes and coyotes was 3% and 40%, respectively. In contrast to CPV, the prevalence of CDV antibodies declined from previous sampling. Miller et al. (2000) documented 18% prevalence of CDV antibodies among swift foxes, whereas Gese et al. (1991) found a seroprevalence of 57% among coyotes. McCue and O'Farrell (1988) reported CDV antibodies in 0–14% of the kit foxes sampled in California. We found that levels of CDV antibodies increased with age among both species, similar to results among coyotes in Texas (Guo et al., 1986), Wyoming (Gese et al., 1997), Arizona (Grindler and Krausman, 2001), and Utah (Arjo et al., 2003). The higher prevalence of CDV antibodies in adults may be a result of adults being

more likely to survive exposure or adults having a longer exposure period to the virus and developing a persisting titer (Gorham, 1966; Green et al., 1984). Among coyote populations sampled in western states, the prevalence of CDV antibodies was 23–76% (Trainer and Knowlton, 1968; Guo et al., 1986; Williams et al., 1988; Gese et al., 1997; Grindler and Krausman, 2001; Arjo et al., 2003).

The prevalence of CAV antibodies was high among coyotes (77%), and antibodies were not detected in swift foxes. The prevalence of CAV antibodies has not been previously examined among swift foxes. McCue and O'Farrell (1988) reported a prevalence of CAV antibodies among San Joaquin kit foxes of 6–21% in California. The prevalence of CAV antibodies among coyotes in Arizona, Texas, Utah, and Wyoming has been reported to be 31–100% (Trainer and Knowlton, 1968; Gese et al., 1997; Grindler and Krausman, 2001; Arjo et al., 2003). The degree to which CAV virus affects canid population demographics is unknown.

The high prevalence of *Y. pestis* antibodies in swift foxes (57%) and coyotes (85%) indicates relatively equal exposure. Canids may become infected with *Y. pestis* by being bitten by fleas or by ingesting infected rodents (Thomas et al., 1989). Small mammals make up a large component of the diets of both canid species in our study area (Kitchen et al., 1999). When canids are infected, they generally do not develop clinical signs, but they do develop antibodies (Barnes, 1982), making them an indicator species for plague. Changes in the prevalence of plague antibodies in canids may be related to changes in the prevalence of plague in prey. The prevalence of *Y. pestis* antibodies has not been reported for swift foxes. Among kit foxes, no evidence of *Y. pestis* antibodies was reported in California (McCue and O'Farrell, 1988). Among coyotes, the seroprevalence of *Y. pestis* antibodies was reported to be low in California (<6%; Thomas and Hughes, 1992). In contrast, coyote popu-

lations in Wyoming (Gese et al., 1997) and Utah (Arjo et al., 2003) had prevalence similar to those in this study. The impact of plague on canid populations is unknown.

Antibodies against tularemia were not found in swift foxes and were low in the coyote population (4%). Evidence of tularemia has not been reported for swift foxes. In kit foxes, *F. tularensis* antibodies were reported to be 8–31% in California (McCue and O'Farrell, 1988). Among coyotes, evidence of tularemia antibodies was found in Wyoming (Gese et al., 1997) and Utah (Arjo et al., 2003) but at low levels. In Texas, Trainer and Knowlton (1968) found no serologic evidence of tularemia. In contrast, 88% of coyotes sampled in Idaho were seropositive (Gier et al., 1978). The impact of tularemia on canids is unknown. They may contract the disease, but they appear to be relatively resistant and probably recover (Gier and Ameel, 1959; Zarnke and Ballard, 1987).

Reasons for differences in seroprevalence for some canine infections (mainly CPV, CDV, and CAV) between swift foxes and coyotes are unknown. It is possible that coyotes are more resistant to some of these infections than are swift foxes. We documented three radio-collared swift foxes dying from CDV infections in the study area (Karki, 2003). Similarly, Olson and Lindzey (2002) reported two swift foxes dying from CDV in Wyoming. Although we did not document direct mortality among the swift foxes that was caused by CPV or CAV infection, foxes in a weakened condition from infection could be more vulnerable to predation. Coyote predation is the leading cause of swift fox mortality on the PCMS (Kitchen et al., 1999; Schauster et al., 2002; Karki, 2003). Predation could potentially mask an underlying infectious disease that increased the vulnerability of the foxes. Alternatively, different rates of exposure may explain the disparity of the antibody prevalence between the two species. With both species sharing the same landscape and diet

(Kitchen et al., 1999), it seems unlikely that exposure to infectious agents differs between the two species, but this remains a possible explanation.

The high prevalence of CPV, CDV, and CAV antibodies in the coyote population over time is evidence that these infections persist in these population and that coyotes are a potential source of viral infection to the foxes. However, the close encounters needed for transmission between coyotes and foxes seems to be unlikely, because coyote predation is a major source of mortality for swift foxes. Thus, varying resistance to CDV may be the most plausible explanation for the different levels of CDV antibody prevalence between the two canids; the three fox deaths from CDV support this supposition. For CPV and CAV, exposure and transmission may occur from within and between canid species because of the resistance of CPV and CAV in the environment (Thomas et al., 1984). Exposure from prey is the most likely source of exposure to plague for foxes and coyotes. Conservation efforts for swift foxes, a species of special concern in many states, should consider infectious diseases, particularly in areas that have a high prevalence of CDV. Canine distemper was the only disease causing mortality in our foxes, although other infections could make swift foxes more vulnerable to predation.

We thank E. Williams, J. Edwards, and H. Edwards of the Wyoming State Veterinary Laboratory for their expertise and laboratory assistance; numerous technicians for field assistance; Hawkins and Powers Aviation and Miller Aero Service for helicopter flying; W. Aaroe for demonstrating jugular draws on swift foxes; and E. Williams for review of the manuscript. We also thank T. Warren, G. Belew, R. Bunn, and B. Rosenlund for providing funding, support, and project coordination for many years. Funding and logistical support provided by the US Army, Directorate of Environmental Compliance and Management, Fort Carson, Colorado, through the US Fish and Wildlife Service,

Colorado Fish and Wildlife Assistance Office, Lakewood, Colorado, and the Utah Cooperative Fish and Wildlife Research Unit at Utah State University. Additional support provided by the US Department of Agriculture, Wildlife Services, National Wildlife Research Center, Logan Field Station at Utah State University. Research protocols were approved by Institutional Animal Care and Use Committees at Utah State University and the National Wildlife Research Center.

LITERATURE CITED

- ANDERSEN, D. E., AND B. D. ROSENBLUND. 1991. Fish and wildlife management recommendations: Pinon Canyon Maneuver Site, Las Animas County, Colorado. US Fish and Wildlife Service, Golden, Colorado, 68 pp.
- APPEL, M., AND D. S. ROBSON. 1973. A microneutralization test for canine distemper virus. *American Journal of Veterinary Research* 34: 1459–1463.
- , L. E. CARMICHAEL, AND D. S. ROBSON. 1975. Canine adenovirus type-2-induced immunity to two canine adenoviruses in pups with maternal antibody. *American Journal of Veterinary Research* 36: 1199–1202.
- ARJO, W. M., E. M. GESE, C. BROMLEY, A. KOZLOWSKI, AND E. S. WILLIAMS. 2003. Serologic survey for diseases in free-ranging coyotes (*Canis latrans*) from two ecologically distinct areas of Utah. *Journal of Wildlife Diseases* 39: 449–455.
- BARNES, A. M. 1982. Surveillance and control of bubonic plague in the United States. *Symposia of the Zoological Society of London* 50: 237–270.
- BARRETT, M. W., J. W. NOLAN, AND L. D. ROY. 1982. Evaluation of a hand-held net-gun to capture large mammals. *Wildlife Society Bulletin* 10: 108–114.
- CHU, M. C. 2000. Laboratory manual of plague diagnostic tests. Centers for Disease Control and Prevention and World Health Organization, Atlanta, Georgia, 129 pp.
- COSTELLO, D. F. 1954. Vegetation zones in Colorado. In *Manual of plants of Colorado*, H. D. Harrington (ed.). Sage Books, Denver, Colorado, pp. iii–x.
- COVELL, D. F. 1992. Ecology of swift fox (*Vulpes velox*) in southeastern Colorado. MS Thesis, University of Wisconsin, Madison, Wisconsin, 111 pp.
- GESE, E. M., O. J. RONGSTAD, AND W. R. MYTTON. 1987. Manual and net-gun capture of coyotes from helicopters. *Wildlife Society Bulletin* 15: 444–445.
- , ———, AND ———. 1988. Home-range and habitat use of coyotes in southeastern Colorado. *Journal of Wildlife Management* 52: 640–646.
- , R. D. SCHULTZ, O. J. RONGSTAD, AND D. E. ANDERSEN. 1991. Prevalence of antibodies against canine parvovirus and canine distemper virus in wild coyotes in southeastern Colorado. *Journal of Wildlife Diseases* 27: 320–323.
- , ———, M. R. JOHNSON, E. S. WILLIAMS, R. L. CRABTREE, AND R. L. RUFF. 1997. Serological survey for diseases in free-ranging coyotes (*Canis latrans*) in Yellowstone National Park, Wyoming. *Journal of Wildlife Diseases* 33: 47–56.
- GIER, H. T. 1968. Coyotes in Kansas (revised). Agricultural Experiment Station Bulletin 393, Kansas State University, Manhattan, Kansas, 118 pp.
- , AND D. J. AMEEL. 1959. Parasites and diseases of Kansas coyotes. Technical Bulletin 91, Agricultural Experiment Station, Kansas State University, Manhattan, Kansas, 34 pp.
- , S. M. KRUCKENBURG, AND R. J. MARLER. 1978. Parasites and diseases of coyotes. In *Coyotes: Biology, behavior, and management*, M. Bekoff (ed.). Academic Press, New York, New York, pp. 37–71.
- GORHAM, J. R. 1966. The epizootiology of distemper. *Journal of the American Veterinary Medical Association* 149: 610–618.
- GREEN, J. S., M. L. BRUSS, J. F. EVERMANN, AND P. K. BERGSTROM. 1984. Serologic response of captive coyotes (*Canis latrans* Say) to canine parvovirus and accompanying profiles of canine coronavirus titers. *Journal of Wildlife Diseases* 20: 6–11.
- GRINDER, M., AND P. R. KRAUSMAN. 2001. Morbidity-mortality factors and survival of an urban coyote population in Arizona. *Journal of Wildlife Diseases* 37: 312–317.
- GUO, W., J. F. EVERMANN, W. J. FOREYT, F. F. KNOWLTON, AND L. A. WINDBERG. 1986. Canine distemper virus in coyotes: A serologic survey. *Journal of the American Veterinary Medical Association* 189: 1099–1100.
- JOHNSON, M. R., D. K. BOYD, AND D. H. PLETSCHE. 1994. Serologic investigations of canine parvovirus and canine distemper in relation to wolf (*Canis lupus*) pup mortalities. *Journal of Wildlife Diseases* 30: 270–273.
- KAMLER, J. F. 2002. Relationships of swift foxes and coyotes in northwest Texas. PhD Dissertation, Texas Tech University, Lubbock, Texas, 116 pp.
- KARKI, S. M. 2003. Effects of coyote removal on swift fox (*Vulpes velox*) population ecology in southeastern Colorado. MS Thesis, Utah State University, Logan, Utah, 82 pp.
- KITCHEN, A. M., E. M. GESE, AND E. R. SCHAUSTER. 1999. Resource partitioning between coyotes and swift foxes: Space, time, and diet. *Canadian Journal of Zoology* 77: 1645–1656.
- LINHART, S. B., AND F. F. KNOWLTON. 1967. Deter-

- mining age of coyotes by tooth cementum layers. *Journal of Wildlife Management* 31: 362–365.
- MCCUE, P. M., AND T. P. O'FARRELL. 1988. Serological survey for selected diseases in the endangered San Joaquin kit fox (*Vulpes macrotis mutica*). *Journal of Wildlife Diseases* 24: 274–281.
- MECH, L. D., AND S. M. GOYAL. 1993. Canine parvovirus effect on wolf population change and pup survival. *Journal of Wildlife Diseases* 29: 330–333.
- MERCURE, A., K. RALLS, K. P. KOEPFLI, AND R. K. WAYNE. 1993. Genetic subdivisions among small canids: Mitochondrial DNA differentiation of swift, kit, and arctic foxes. *Evolution* 47: 1313–1328.
- MILLER, D. S., D. F. COVELL, R. G. MCLEAN, W. J. ADRIAN, M. NIEZGODA, J. M. GUSTAFSON, O. J. RONGSTAD, R. D. SCHULTZ, L. J. KIRK, AND T. J. QUAN. 2000. Serologic survey for selected infectious disease agents in swift and kit foxes from the western United States. *Journal of Wildlife Diseases* 36: 798–805.
- OLSON, T. L., AND F. G. LINDZEY. 2002. Swift fox survival and production in southeastern Wyoming. *Journal of Mammalogy* 83: 199–206.
- RONGSTAD, O. J., T. R. LAURION, AND D. E. ANDERSEN. 1989. Ecology of swift fox on the Pinon Canyon Maneuver Site, Colorado. Final report, US Army, Directorate of Engineering and Housing, Fort Carson, Colorado, 53 pp.
- ROSE, N. R., E. DEMARIO, J. L. FAHEY, H. FREEDMAN, AND G. M. PENN. 1992. Manual of clinical laboratory immunology. American Society for Microbiology, Washington, D.C., 987 pp.
- SCHAUSTER, E. R., E. M. GESE, AND A. M. KITCHEN. 2002. Population ecology of swift foxes (*Vulpes velox*) in southeastern Colorado. *Canadian Journal of Zoology* 80: 307–319.
- SCOTT-BROWN, J. M., S. HERRERO, AND J. REYNOLDS. 1987. Swift fox. In *Wild furbearer management and conservation in North America*, M. Novak, J. A. Baker, M. E. Obbard, and B. Malloch (eds.). Ontario Trappers Association, Toronto, Ontario, Canada, pp. 433–441.
- SHAW, R. B., S. L. ANDERSON, K. A. SCHULZ, AND V. E. DIERSING. 1989. Plant communities, ecological checklist, and species list for the US Army Pinon Canyon Maneuver Site, Colorado. Colorado State University Range Science Department, Range Science Serial 37, Fort Collins, Colorado, 71 pp.
- SOKAL, R. R., AND F. J. ROHLF. 1981. *Biometry*, 2nd ed. Freeman, San Francisco, California, 859 pp.
- SOVADA, M. A., C. C. ROY, J. B. BRIGHT, AND J. R. GILLIS. 1998. Causes and rates of mortality of swift foxes in western Kansas. *Journal of Wildlife Management* 62: 1300–1306.
- THOMAS, C. U., AND P. E. HUGHES. 1992. Plague surveillance by serological testing of coyotes (*Canis latrans*) in Los Angeles County, California. *Journal of Wildlife Diseases* 28: 610–613.
- THOMAS, N. J., W. J. FOREYT, J. F. EVERMANN, L. A. WINDBERG, AND F. F. KNOWLTON. 1984. Seroprevalence of canine parvovirus in wild coyotes from Texas, Utah, and Idaho (1972 to 1983). *Journal of the American Veterinary Medical Association* 185: 1283–1287.
- THOMAS, R. E., M. L. BEARD, T. J. QUAN, L. G. CARTER, A. M. BARNES, AND C. E. HOPLA. 1989. Experimentally induced plague infection in the northern grasshopper mouse (*Onychomys leucogaster*) acquired by consumption of infected prey. *Journal of Wildlife Diseases* 25: 477–480.
- TRAINER, D. O., AND F. F. KNOWLTON. 1968. Serologic evidence of diseases in Texas coyotes. *Journal of Wildlife Management* 32: 981–983.
- US Department of the Army. 1980. Draft environmental impact statement for training land acquisition. Fort Carson, Colorado, 284 pp.
- WILLIAMS, E. S., E. T. THORNE, M. J. G. APPEL, AND D. W. BELITSKY. 1988. Canine distemper in black-footed ferrets (*Mustela nigripes*) from Wyoming. *Journal of Wildlife Diseases* 24: 385–398.
- ZAR, J. H. 1996. *Biostatistical analysis*, 3rd edition. Prentice-Hall, Inc., Upper Saddle River, New Jersey, 662 pp.
- ZARNKE, R. L., AND W. B. BALLARD. 1987. Serologic survey for selected microbial pathogens of wolves in Alaska, 1975–1982. *Journal of Wildlife Diseases* 23: 77–85.

Received for publication 3 November 2003.