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THE POTENTIAL ROLE OF SWIFT FOXES (*VULPES VELOX*) AND THEIR FLEAS IN PLAGUE OUTBREAKS IN PRAIRIE DOGS

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ABSTRACT: Swift foxes (*Vulpes velox*) have been proposed as potential carriers of fleas infected with the bacterium *Yersinia pestis* between areas of epizootics in black-tailed prairie dogs (*Cynomys ludovicianus*). We examined antibody prevalence rates of a population of swift foxes in Colorado, USA, and used polymerase chain reaction (PCR) assays to examine their flea biota for evidence of *Y. pestis*. Fifteen of 61 (24%) captured foxes were seropositive, and antibody prevalence was spatially correlated with epizootic plague activity in prairie dog colonies in the year of, and previous to, the study. Foxes commonly harbored the flea *Pulex simulans*, though none of the fleas was positive for *Y. pestis*.

Key words: Carnivore disease, plague transmission, swift fox, *Vulpes velox*, *Yersinia pestis*.

INTRODUCTION

Though primarily a disease of rodents, plague presents a significant human health risk, and it also has important consequences for wildlife populations (Antolin et al., 2002; Gage and Kosoy, 2005). *Yersinia pestis*, the bacterium that causes plague, was introduced to the western United States in the 1900s and is now present throughout the Rocky Mountain States where plague outbreaks, or epizootics, occur sporadically following long periods of latent persistence (enzootic phase). Many North American mammal species show evidence of exposure or susceptibility to the disease, although the mechanism by which plague persists and is transmitted in wildlife populations remains poorly understood (Gage and Kosoy, 2005).

For example, in the USA, plague can be responsible for temporary extinctions of entire colonies of black-tailed prairie dogs (*Cynomys ludovicianus*), a species that plays an important ecological role in the grasslands of North America (Cully and Williams, 2001; Stapp et al., 2004). The mechanism of plague's movement and persistence between prairie dog colonies is not known, and although mammalian carnivores have been proposed as a carrier of plague-infective fleas (Gage et al., 1994; Salkeld and Stapp, 2006), this phenomenon has rarely been tested. Swift foxes (*Vulpes velox*) have been suggested as a potential carrier of plague-infective fleas (Harrison et al., 2003). Therefore, we investigated the possible role of swift foxes in the spread of plague by examining the rates of exposure to *Y. pestis* and patterns of flea infestation in a population in north-central Colorado, where plague is enzootic.

METHODS

The study took place as part of a swift fox reintroduction program that translocated foxes from Colorado to South Dakota, carried out by the National Park Service, Badlands National Park. In August 2005, swift foxes were captured on and around the Pawnee National Grassland (PNG), Weld County, Colorado (40°49′N, 104°47′W; elevation 1,650 m). The climate of the PNG is semiarid, and vegetation is shortgrass steppe, characterized by blue

grama (*Bouteloua gracilis*), buffalo grass (*Buchloe dactyloides*), and prickly pear cactus (*Opuntia polyacantha*).

Foxes were trapped in Tomahawk Single Door box traps (Tomahawk, Wisconsin, USA) and Tru-Catch Tuffy box traps (Belle Fourche, South Dakota, USA) baited with turkey poults and canned mackerel. Traps were opened in the afternoon and checked shortly after dawn the following day. Captured foxes were dusted with insecticide (Carbaryl, 12.5% active ingredient), and moved into portable, insecticide-dusted travel kennels. Exact locations of capture were recorded using a global positioning system (GPS; Garmin eTrex Legend, Garmin International, Inc., Olathe, Kansas, USA). Foxes were weighed, sex was determined, and up to 3 ml of blood was collected by femoral venipuncture. Adult foxes are heavier than juveniles, so weight was used as a proxy for age. Blood samples were centrifuged, and serum was tested for plague antibodies using an enzyme-linked immunosorbent assay (ELISA) at the Wyoming State Veterinary Laboratory, Laramie, Wyoming; seropositive or seronegative results were determined according to standards described by Chu (2000). Seropositive animals were released at their site of capture, and healthy seronegative animals were transferred for release in South Dakota. All trapping and animal-handling procedures were carried out in accordance with permits and licenses issued by the National Park Service, South Dakota Department of Game, Fish and Parks, South Dakota Animal Industry Board, and the Colorado Division of Wildlife.

Fleas were collected from the bottoms of insecticide-dusted travel kennels and refrigerated in 2% saline solution with a small amount of Tween-80 detergent (ICN Biochemicals, Ohio, USA) prior to identification. Fleas were identified to species level using classifications of Stark (1958) and Hubbard (1968), and identification was confirmed at the Centers for Disease Control and Prevention (CDC), Fort Collins, Colorado.

To determine the presence of *Y. pestis* in fleas, identified fleas were triturated with a MM301 mixer mill (Retsch, Newtown, Pennsylvania, USA) in 100-µl brain heart infusion (BHI; Difco, Kansas, USA) broth with three 3-mm sterile Pyrex glass beads at 20 beats/sec for 4 min. Triturated flea material was centrifuged at 15,600 × G for 10 sec and heated to 95 C for 10 min., and then centrifuged for an additional minute to pellet the flea debris. An aliquot of the supernatant containing the DNA (1.25 µl) was used for the polymerase chain reaction (PCR) template, and the remaining flea material was stored at −80 C.

Fleas were tested for *Y. pestis* using a PCR that targeted the *pla* gene as described previously with minor modifications (Hinnebusch and Schwan, 1993). Each 25-µl PCR reaction contained 1 X PCR buffer with 1.5 mM $MgCl_2$, 200 µM deoxynucleotide triphosphates (dNTPs), 0.5 U *Taq*-polymerase (Promega, Madison, Wisconsin, USA), 1.25 µl of flea triturate, and 10 uM each of primers Yp1 (5′-ATCTTACTTTCCGTGAGAAG-3′) and Yp2 (5′-CTTGGATGTTGAGCTTCCTA-3′) (Stevenson et al., 2003). Negative controls (master mix reagents only) and two positive controls (0.5 ng of *Y. pestis* DNA extracted from a wild type isolate [CO963188] from the CDC, and CO963188 culture diluted to 10-100 cfu/100 µl in HIB [heart infusion broth], added to uninfected flea triturate and heat lysed) were included in each 96-well plate of reactions. Reactions were amplified in a PTC-200 thermocycler (MJ Research, Watertown, Massachusetts, USA), with an initial denaturation step of 95 C for 5 min, followed by 35 cycles of 1 min at 95 C, 1 min at 55 C, 1 min at 72 C, and a final extension step of 10 min at 72 C. Amplification with the *pla* primers produced product lengths of 478 base pairs (bp). Products were visualized by separating 5 µl of the PCR reaction mixture on 2% agarose gels containing 1% ethidium bromide. Previous studies have shown the sensitivity of the *pla* PCR reaction to be 10-100 cfu per flea sample (Engelthaler et al., 1999; Stevenson et al., 2003), and this was confirmed by our diluted positive controls.

In 2004 and 2005, plague epizootics resulted in extinction of seven prairie dog colonies on the PNG (confirmed by the postmortem examinations of prairie dogs at the CDC). We sought to determine if seropositive foxes were captured in closer proximity to plague towns than seronegative foxes. The United States Forest Service has annually monitored the location, activity (i.e., extinct or active), and size of prairie dog colonies on the PNG since 1981, and from 1995, colonies have been mapped using global positioning system satellite technology (GPS) incorporated into a Geographic Information System (GIS) using Arc/INFO 9.0, ArcView 3.3, ArcMap 8.3, or Arc9 (Environmental Systems Research Institute, Redlands, California, USA). All data were differentially corrected and projected to UTM zone 13N WGS 84. Using the Spatial Analyst extension of Arc 9, we created a 30×30 m grid representing the Euclidean distance to the nearest prairie dog colony that tested positive for plague in 2004 or 2005;

TABLE 1. Flea infestation patterns of swift foxes (*Vulpes velox*; $n=37$) from north-central Colorado.

	Pulex simulans	*Thrassis fotus*	*Euhoplopsyllus glacialis*	All fleas
Proportion of foxes infested (%)	91.9	13.5	2.7	94.6
Number of fleas per animal	0–50	0–1	0–1	0–50
Number of fleas collected (% of total fleas collected)	656 (99.1)	5 (0.7)	1 (0.2)	662 (100)
Mean number of fleas per infested animal (SD)	19.3 (14.8)	1 (0)	1 (0)	18.9 (14.9)
Mean number of fleas per animal (SD)	18.2 (15.1)	0.13 (0.35)	0.02	17.9 (15.1)

distance values corresponding with fox capture locations were extracted using GridSpot. A Wilcoxon rank-sums test was used to compare median minimum distance to plague towns between seropositive and seronegative foxes.

Antibody prevalence was compared between sexes using chi-square tests. The relationship between seroprevalence and fox weight was analyzed using logistic regression.

RESULTS

Sixty-seven foxes were captured in 2005 during the translocation project. Of these, 61 were processed and examined for previous exposure to *Y. pestis*. Fifteen (24%) were seropositive. Males and females did not differ in antibody prevalence rates ($\chi^2=0.7$, $P=0.4$), but there was a significant positive relationship between fox weight and seroprevalence (logistic regression, $\chi^2=4.46$, $P=0.035$).

Flea pools were examined from 37 foxes. Flea species diversity was low, and only three identifiable species of flea were present: *Pulex simulans*, *Thrassis fotus*, and *Euhoplopsyllus glacialis*. In total, 95% (35/37) of foxes were found to harbor fleas; the two uninfested animals were both female (one subadult and one juvenile). All but one flea-infested fox hosted *P. simulans* fleas, *T. fotus* was much less common, and *E. glacialis* was found on only one fox (Table 1). For all flea-infested hosts, there was no relationship between host weight and total number of fleas ($r^2=0.24$, $P=0.82$, $n=35$). There was no difference in *Pulex* flea load between males and females (Wilcoxon rank sum test: $Z=-1.01$, $P=0.31$), and *Pulex* flea load was unrelated to fox weight (linear regression: $r^2=0.01$, $F_{1,30}$, $P=0.58$). PCR analysis of fleas found no evidence of *Y. pestis* infection, although all positive controls amplified the *pla* fragment, indicating that if the fleas had been infected, we would have detected it. When compared with seronegative foxes, seropositive foxes were captured in closer proximity to prairie dog colonies that experienced plague outbreaks (median values 19.35 and 1.33 km, respectively; $\chi^2=19.43$, df=1, $P<0.0001$; Fig. 1).

DISCUSSION

On the PNG, 24% of captured foxes were seropositive for plague antibodies. This level of plague exposure is similar to antibody prevalence levels previously reported for Colorado, which range 0–57% (Miller et al., 2000; Gese et al., 2004). The likelihood of being seropositive increased with weight, which implies increasing age, and presumably increasing opportunity for plague exposure. Previous studies have demonstrated seroprevalence variation in relation to age in coyotes (*Canis latrans*) (Gese et al., 1997).

Exposure to plague was strongly associated with prairie dog colonies that had experienced recent plague epizootics. Transmission of *Y. pestis* by the consumption of infected hosts has been demonstrated in domestic cats and dogs (Rust et al., 1971), as well as northern grasshopper mice (*Onychomys leucogaster*) and other rodents (Rust et al., 1972; Thomas et al.,

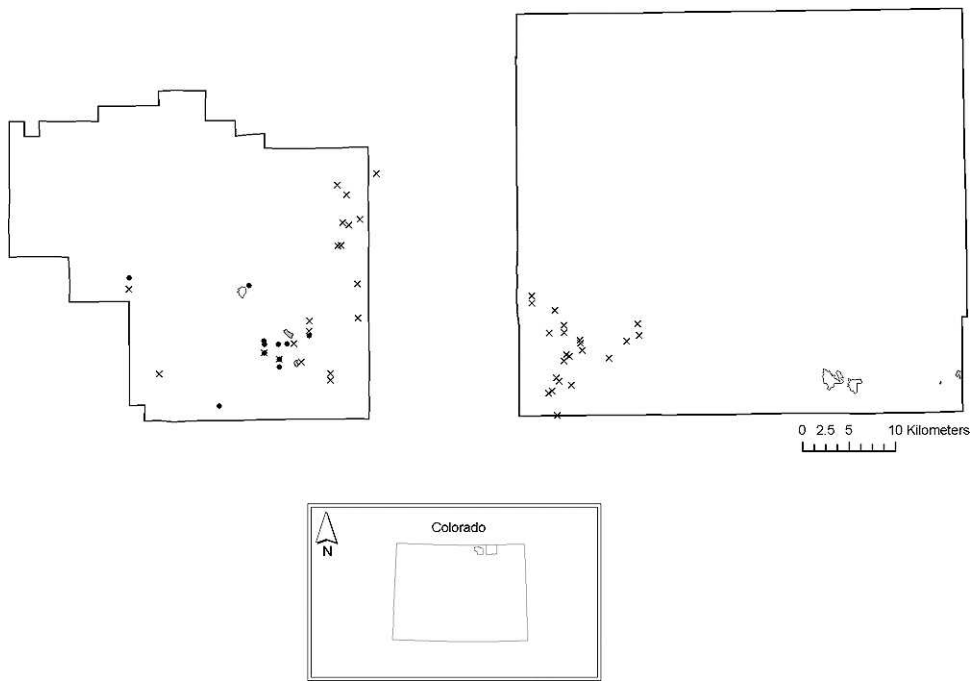

FIGURE 1. Locations of sampled swift foxes on the eastern and western section of the Pawnee National Grasslands (PNG), Colorado. Prairie dog colonies that experienced outbreaks of plague in 2004 are shown in gray, and outbreaks in 2005 are shown as white. Foxes that were seropositive (•) were significantly nearer to prairie dog colonies with plague than seronegative foxes (x). The location of the PNG within Colorado is shown in the inset.

1989) and is probably an important source of infection for wild carnivores. Prairie dogs dead from plague can be found above ground and are probably consumed readily by scavenging swift foxes. Furthermore, plague antibodies can persist for several months in carnivores (Barnes, 1982). Thus, foxes that were seropositive near colonies that had experienced plague a year earlier (2004) were quite probably exposed to plague-infected animals during that year's epizootics.

Interestingly, the seropositive smaller foxes, equivalent to young born in 2005, were also found near the prairie dog colonies that had experienced epizootics in 2004; the plague epizootic had occurred before they were born. Several explanations can account for young seropositive foxes, which were approximately 4-mo old at the time of sampling (unpubl. data). Maternal antibodies can persist in weaned rats (*Rattus norvegicus*) for up to 3 mo (Williams et al., 1977), so it is possible that the young foxes were seropositive as a result of their mothers' exposure to plague the previous year. Young foxes may have developed plague antibodies after being bitten by infectious prairie dog fleas, but flea abundance in colony burrows drops to very low numbers the year after plague epizootics (P. Stapp and D. Salkeld, unpubl.). On the other hand, foxes may have been exposed to plague in 2005 by consuming other small mammal plague hosts. We also cannot rule out the possibility that seropositive foxes may have traveled from the one colony in the western PNG that was actively experiencing an epizootic in 2005 (Fig. 1), since young males disperse during mid-late August of their first year, and foxes are capable of moving the relatively short distances between most PNG colonies (me-

dian distance = 3.6 km; Stapp et al., 2004) over short periods of time (Roell, 1999).

Although evidence for the role of swift foxes as plague reservoirs is lacking, it is possible that foxes may act as a source of infection to rodent communities. *Yersinia pestis* has been detected in other canid species, and gray foxes (*Urocyon cinereoargenteus*) have been responsible for zoonotic (animal to human) plague transmission (Rust et al., 1971; Gage et al., 1994; Salkeld and Stapp, 2006). Since there was no pre-outbreak monitoring, we cannot determine whether our observation that seropositive foxes were associated with infected prairie dog colonies resulted from exposure of foxes to infected prairie dogs or through prairie dog colonies becoming infected through exposure to infected swift foxes or their fleas. However, this study demonstrates the use of carnivores as sentinel species for detecting prior plague activity in rodents, as antibody prevalence rates demonstrated a relationship with plague activity in smaller mammals up to a year previous to trapping.

A high proportion (95%) of the foxes harbored fleas, most commonly *Pulex simulans*. Previous studies of swift foxes have found a similarly high prevalence of infestation by *Pulex* fleas (Williams et al., 1993; Miller et al., 1998; Harrison et al., 2003; Pence et al., 2004). *Pulex simulans* has also been found in prairie dog burrows and on prairie dogs on the PNG (unpubl.), and plague-positive *Pulex* sp. specimens have been found in prairie dog burrows in Kansas (Cully et al., 2000). In 2006 on the PNG, we found a prairie dog carcass, confirmed dead from plague by the CDC, which still harbored 16 *Oropsylla hirsuta* fleas and 33 *Pulex* sp. fleas. Of these fleas, 62.5% of the *O. hirsuta* and 45.5% of the *Pulex* fleas were found positive for plague, using the same *pla* primers described earlier. Although it is unknown how much flea transfer takes place between carnivores and prairie dogs, the possibility of such host sharing has great implications for the spread and persistence of plague.

Five animals hosted *Thrassis fotus*, which, on the PNG, is commonly found on thirteen-lined ground squirrels (*Spermophilus tridecemlineatus*), and secondarily on northern grasshopper mice (*Onychomys leucogaster*; Stapp and Salkeld, unpubl.), and has been found occasionally on black-tailed prairie dogs (Antolin and Tripp, unpubl.). One fox harbored a single *Euhoplopsyllus glacialis*, more commonly found on cottontail rabbits (Hubbard, 1968). Therefore, we assume that these infestations were the results of fleas jumping from their typical rodent hosts or burrows during predation or foraging activity, rather than utilizing swift foxes as regular hosts. Even if such an association is fleeting, plague could be transmitted between foxes and rodents if infectious rodent fleas bite foxes, or if rodent fleas bite infectious foxes and then subsequently feed on other rodent species. Alternatively, carnivores may simply act as carriers of infectious rodent fleas between locations (Gage et al., 1994).

Some rodent flea species collected from foxes have been identified as potential vectors of plague (Gage et al., 1994; Harrison et al., 2003). However, in our study and in an investigation in northwestern Texas (McGee et al., 2006), none of the fleas found on swift foxes were positive for plague using PCR techniques, a finding that raises questions about the oft-stated hypothesis that carnivores act as carriers of plague-infective fleas. However, capture of foxes on the PNG was designed for translocation purposes, and although some foxes were caught in the vicinity of plague epizootics, few foxes were captured directly on colonies with plague. More concentrated sampling, over a more extended period, may yield plague-positive fleas on foxes and help resolve the role of foxes in the spread of plague, particularly if temporal changes in plague antibody titers can show when plague exposure in

foxes occurs in relation to plague activity in rodents.

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