Department of Defense
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Spatial Ecology of the Island Fox

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Project Overview

Infectious diseases threaten the recovery of numerous threatened species managed by the U.S. Department of Defense (DoD), and pose a risk of reducing currently healthy wildlife populations to levels requiring intensive and expensive management or restricted activities to meet the natural resource management obligations under the Endangered Species Act (ESA) and other federal regulations. For example, withering foot disease has led to the severe reduction of black abalone (*Haliotis cracherodii*) populations in southern California, including the Naval Outlying Landing Field, San Nicolas Island, Ventura California, to the point where the species is recommended for protection under the ESA (National Oceanic and Atmospheric Administration 2008). Populations of amphibians and bats on DoD lands across the country are at risk from chytrid fungus (Petersen et al. 2011) and white nose syndrome (Frick et al. 2010) respectively.

The island fox (*Urocyon littoralis*) provides a particularly poignant example of the importance of understanding disease dynamics to managing wildlife on DoD lands. The DoD has designated the island fox as a species at risk candidate, and a focal point of conservation efforts. The
 Navy currently manages fox populations on San Clemente Island and San Nicolas Island,

Photo: Jessica Sanchez

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and owns San Miguel Island, which is managed by the National Park Service. Four of the six island fox subspecies, including the San Miguel Island fox, have been listed for protection under the Endangered Species Act due to rapid population declines. Notably, fox numbers on Santa Catalina Island dropped from 1300 animals to fewer than 200 following an outbreak of distemper virus (CDV), probably introduced by raccoons immigrating to the island (Timm et al. 2009). In response to the Catalina outbreak recovery for all four listed subspecies is contingent on having a plan in place to minimize the impact of future invasions by virulent pathogens (R. McMorran U.S. Fish and Wildlife Service, personal communication). The DoD has taken the lead in disease management supporting monitoring programs and preventative vaccination programs on San Clemente and San Nicolas islands and developing an epidemic response plan for San Clemente Island (Hudgens et al. 2011).

Currently, the most likely population level threat to island foxes on San Clemente Island is anticipated to be the introduction of a novel, virulent pathogen. Island foxes are susceptible to rabies and canine distemper, both of which are transmitted through close interactions between hosts. These diseases have caused epidemics in wildlife worldwide, and their spread has been well studied in mainland foxes (*Urocyon cinereargenteus*, *Vulpes macrotis macrotis*, *Vulpes vulpes*; Deem et al. 2000, Holmala and Kauhala 2006). Rabies is a pathogen that the island fox has not encountered in its recent evolutionary history and is deadly to nearly all mammals (World Health Organization 2005). The extreme population crash on Santa Catalina island due to canine distemper demonstrates what a serious threat introduced disease is to island foxes, a species that evolved in isolation from mainland pathogens for thousands of years (Clifford et al. 2006). In order to prevent future epidemics, foxes are currently vaccinated against rabies and CDV annually on all islands.

The ecology of disease in wildlife populations is complicated and often unpredictable; making the management of dangerous health risks an ongoing and adaptive process. One of the numerous variables that affect host-pathogen dynamics is how a host's behavior influences the rate and path of pathogen spread within a host population. For pathogens such as rabies and CDV that are passed directly between hosts the rate of transmission is often determined by the interactions of hosts with conspecifics. How often these potentially disease spreading interactions occur varies with the number of neighboring conspecifics and the amount of home range overlap with those neighbors (Woodroffe 1999). These factors depend on 3 aspects of the host's ecology: density of the population, home range sizes, and how individuals use and move within their home ranges (McCallum et al. 2001). Home range size and use can in turn vary with density, making it more difficult to predict the spread of disease in host populations with heterogeneous distributions.

We will use fox home range and contact data in conjunction with published data on disease transmission rates for canine rabies and distemper to develop a spatially explicit model for disease spread in San Clemente Island foxes. We will evaluate this model to 1) identify likely spatial patterns of disease spread, 2) based on these patterns, identify optimal vaccination or quarantine strategies to prevent introduced strains of these or other diseases from decimating fox populations, and 3) test alternative management strategies to slow or prevent disease spread across the island. This model will simulate the spread of disease initiated in animals at likely points of introduction (e.g. barge loading dock or town). The model will predict how quickly the disease spreads to different parts of the island providing guidance to inform emergency vaccination or quarantine strategies. We will also use the model to explore the efficacy of preventative measures, such as preemptive vaccination of a portion of the population. Although

the model was developed for San Clemente Island, our results are applicable to island fox populations on other islands.

Photo: Jessica Sanchez

Understanding host behavior is the first step in determining how to model disease spread. Island foxes are active throughout the day and night, with activity levels peaking in the late afternoon and early evening, likely due to the absence of predators and a food supply requiring a temporal division of activity (Crooks and VanVuren 1995, Hudgens and Garcelon 2011, Laughrin 1977). Island foxes form socially monogamous pairs that occupy the same home range year round, and remain together unless 1 member of the pair dies (Roemer et al. 2001). Adult pairing begins in January, with breeding occurring by mid-March (Laughrin 1977). Pairs are seen together at all times of the year, but most frequently during the breeding season from January through April (Laughrin 1977). Pups are born late-April to early-May, are first seen

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outside the den in early-June, and are independent and dispersing by September (Laughrin 1977).

Fox populations differ between and within islands in regards to size, density, and spatial distribution (Schmidt et al. 2005*a,b*; Coonan 2008; Garcelon et al. 2008; Garcelon and Hudgens 2008). Both fox home range size and the overlap between neighbors vary with density (Crooks and Van Vuren 1996; J. K. Young and D. K. Garcelon, Institute for Wildlife Studies, unpublished data). Because female foxes often do not disperse from natial territories until they are two years old, adult foxes that have substantial home range overlap with mated pairs are usually mature offspring (Roemer et al. 2001). Home range overlap (averaged over at least 1 year) is greater between mated pairs than between unmated pairs of any sex combination, and home range size does not differ between males and females within a study site (Crooks and Van Vuren 1996, Roemer et al. 2001). Crooks and Van Vuren (1996) tracked collared foxes from January to November 1992, and found that although there was some overlap between non-paired foxes on Santa Cruz Island, there was very little overlap between areas of core use. This implies that there may be some areas of exclusive use that are defended against, or avoided by, neighbors. This is supported by observations of aggressive behavior and subsequent injuries in wild foxes (Laughrin 1977). In contrast, mated pairs had significantly more overlap of core use areas than did non-paired individuals (Crooks and Van Vuren 1996). Annual estimates of home range size and core areas were not significantly different from wet (December-April) and dry (May-November) season estimates (Crooks and Van Vuren 1996). Institute for Wildlife Studies (IWS) collared and tracked 74 foxes at 4 sites of varying densities on San Clemente Island from January 2001 to April 2003 (J. K. Young and D. K. Garcelon, unpublished data). Home range size differed among sites for both sexes. Average home range size for all foxes on SCI was 0.94 km^2

(95% minimum convex polygon), and ranged from 0.50 km^2 at the highest density site to 2.33 km^2 at the lowest density site (10.5 and 1.4 foxes/km^2 respectively, using mark-recapture grid
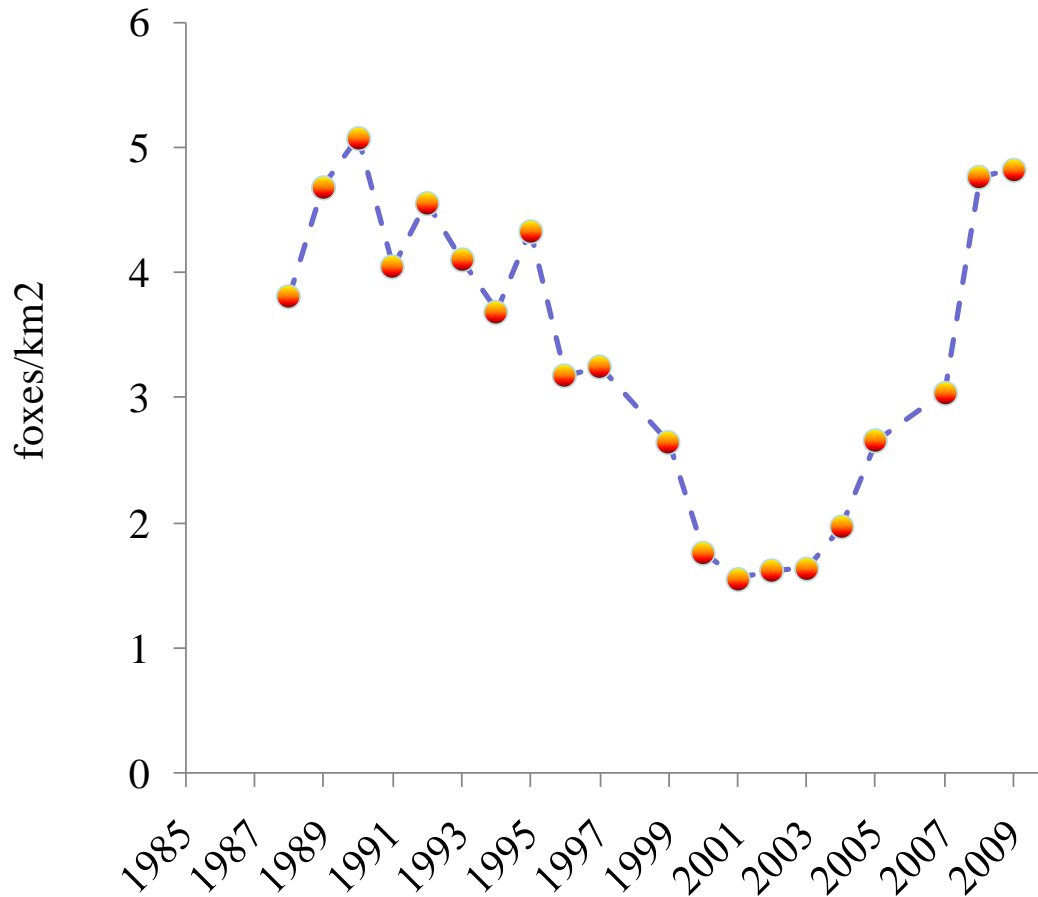

Fig. 1. Fox population estimates from 1988 to 2009 on San Clemente Island, CA.

trapping). Overlap between home ranges of any sex combination was greatest at the high-density site.

The fox population on San Clemente Island experienced a gradual decline from a peak in 1990, reaching a low point in 2001 that was approximately 30% the number of animals that were

present 11 years earlier (Fig. 1). In the early 2000s their numbers started to increase again, and by 2008 it was estimated there were approximately 1,094 foxes on SCI, at densities ranging from 4.1 to 11.9 foxes/km^2 (Garcia and Associates 2009). Although this is similar to pre-decline estimates and is believed to indicate a healthy population, the reason for the previous decline and recovery is unknown. These population fluctuations in San Clemente foxes demonstrate that even large populations are at risk of substantial declines over relatively short time scales.

STUDY AREA

San Clemente Island (SCI) is owned by the U.S. Navy, and is located approximately 109 km west of San Diego, California. It has an area of 146 km^2, tapering from 6.4 km wide at its southern end to 2.4 km wide at its northern end (Schmidt et al. 2005*a*). Most of the island is a semi-flat plateau (the highest elevation is 559 m above sea level; Schmidt et al. 2005*a*) with steep cliffs on the eastern edge, and gradually declining marine terraces on the western side. Average annual precipitation is 13.2 cm (Olsen et al. 2000), and the dominant habitat types are sand dune, maritime desert scrub (MDS), and grassland (Spencer et al. 2006). MDS habitat is further separated into "gentle" if the terrain consisted of gently sloping marine terraces, and "rugged" if the area has steep, rocky canyons running through it.

METHODS

We first asked if differences in density affected home range behaviors by radio tracking foxes in 4 areas on San Clemente Island that have historically differed in fox densities. We recorded contact rates from a subset of these foxes at each site to determine how density-mediated changes in home-range behaviors affect contact rates. We used these data to inform a spatially explicit model of disease (rabies and CDV) spread to address questions about how fox density might affect spatial and temporal disease dynamics under different conditions of fox

vaccination and disease introduction. We also used this model to evaluate effective monitoring, vaccination, and response strategies to minimize the population level impact of these diseases.

Trapping

We trapped foxes from July to August 2010 to apply collars containing radio transmitters and assess current fox densities. Foxes were trapped at 4 sites of varying fox densities and habitat types (Fig 2). Traps were set 250 m apart in small 4×5 grids or larger 5×8 grids, and were trapped for a minimum of 4 nights so that density estimates can be compared to current and previous trapping efforts that use similar grid-trapping methods. Traps were run for up to 7 days if needed to collar the targeted set of animals. Density estimates for each site were calculated from mark-recapture data collected during trapping, using analyses of spatially explicit capture-recapture data developed by Efford et al. (2004) as implemented in Program DENSITY (University of Otago, Otago, New Zealand). Each trapping grid was almost completely contained within one habitat, so we used density estimates from the trapping grids to assign a fox density value to each habitat type.

Foxes were caught using box traps (23 23 66 cm, Tomahawk Live Trap Co., Tomahawk, WI) covered with burlap and vegetation to provide protection from the elements, and lined on the inside with grass as bedding material. Traps were baited using dry cat kibble and berry-scented lure (Knobb Mountain Fur Co., Berwick, PA). "Bite bars" (polypropylene tubing attached to the inside of the trap with flexible wire) were added to each trap for foxes to release stress without damaging their teeth. To compliment fox monitoring and data collection efforts by other agencies on SCI, for each animal captured we recorded: date, time, trap location (UTMs), passive integrated transponder (PIT) tag ID, sex, and age class. If an animal had not been previously tagged, a subcutaneous PIT tag was inserted between and just anterior to the scapulae. We estimated the age of foxes by assigning them to 1 of 5 age classes according to

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tooth eruption and dentin exposure patterns relating to wear on the first upper molar (Wood 1958, Collins 1993). Age classes approximately correspond to years as follows: age class 0: pups born within the previous year, age class 1: ca. 1 to 3 years old, age class 2: ca. 1 to 5 years old, age class 3: ca. 1 to 7 years old, and age class 4: ≥ 3 years old (Hudgens et al. 2007).

At each site, we fitted 8 foxes (4 males and 4 females) with "proximity collars" containing both UHF proximity loggers and VHF transmitters (Sirtrack, Havelock North, New Zealand). When two foxes wearing proximity collars came within approximately 5 m of one another both collars logged the collar ID of the other fox, the date and time, and number of seconds the contact lasted. An additional 10 collars containing VHF transmitters only (Communication Specialists, California, USA) were distributed at 2 sites in an attempt to collar a similar proportion of animals at each site. Neither collar type weighed more than 3% of an animal's body weight. We preferentially collared age class 2 and 3 foxes in good health, since they were the least likely to disperse from their natal range or die during the 6-month battery life of the radio transmitters (Hudgens et al. 2009). We also collared foxes caught in traps that were immediately next to one another or as close as possible to maximize the likelihood that foxes had "neighboring" (adjacent) home ranges. These methods were approved by Humboldt State University's Institutional Animal Care and Use Committee (09/10.W.43.A).

Telemetry

We tracked collared foxes via radio telemetry from July 2010 to January 2011. We located each fox 1 – 2 times per week over the 6 month study period. Locations were distributed as evenly as possible between dawn and dusk in order to capture variation in home range use. Due to safety restrictions, nocturnal locations were not collected. However, we believe our home ranges are representative of the true areas of use because island foxes are active throughout the 24 hour clock and crepuscular activity levels are similar to nocturnal activity

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(Laughrin 1977, Crooks and VanVuren 1995, Hudgens and Garcelon 2011). Locations consisted of at least three bearings, taken within 20 minutes of each other, each 30° to 150° apart, and were calculated in the program Location of a Signal ("LOAS;" Ecological Software Solutions, California, USA). We estimated telemetry errors from 9-12 test collars placed at each site and triangulated by a naïve tracker. Test collar locations were confirmed using a handheld gps with 5 m accuracy.

Photo: Andrew Bridges

Proximity collars logged a "contact" if 2 collared foxes came within approximately 5 m of one another, which is speculated to be a reasonable distance from which a rabid fox could detect another fox and attack, or for CDV to be transmitted via aerosol. A contact record ended once the foxes separate (are >5 m apart) for 120 seconds. We trapped foxes to remove collars in

January 2011, and downloaded contact data from the proximity loggers using software provided by Sirtrack. Contact data for each fox consisted of the total number of contacts, the start time and length of each contact, and the unique collar ID of each fox encountered.

Home Ranges and Contact Rates

We calculated home ranges using both fixed kernel (FK) and minimum convex polygon (MCP) estimators. FKs account for how frequently an animal uses different parts of its home range, but require more locations to estimate areas of core use (White and Garrott 1990). MCPs require fewer locations to estimate a home range, but do not account for differential area use within that home range (White and Garrott 1990). In order to control for locations that might represent estimation error or temporary movements outside the normal home range, 95%, 85% and 50% FKs were calculated by removing 5%, 15%, and 50% of the outliers, respectively (Okarma et al. 1998; Dickson and Beier 2002; J. K. Young and D. K. Garcelon, unpublished data). We entered fox locations into Program R ("R;" R Foundation for Statistical Computing, Vienna, Austria, ‹http://www.R-project.org›) and used the so-called "plug-in method" (Duong 2011) within the package "ks" to calculate the kernel bandwidths and utilization distributions for each fox. We used Hawth's Tools (Spatial Ecology LLC, ‹http://www.spatialecology.com/htools›) to generate FK (95, 85, and 50%) home range estimates from these utilization distributions by drawing contours around the area where 50, 85, or 95% of an animal's activity was concentrated. 100% MCPs were also generated in Hawth's Tools by drawing a convex polygon around the outermost locations for each fox. For each estimation method, we determined the area of overlap between the home ranges of each fox pair using Geospatial Modeling Environment ("GME;" Spatial Ecology LLC, ‹http://www.spatialecology.com/gme›). Home range overlap between each pair of foxes was calculated as a percentage using the Minta index (Minta 1992):

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$$\left(\frac{Overlap}{HRA} \times \frac{Overlap}{HRB} \right)^{0.5} \times 100,$$

with "HRA" and "HRB" being the home range sizes of fox A and B respectively, and "overlap" being the area of overlap between home ranges A and B. The resulting Minta values ranged from 0 to 1, with 0 indicating no overlap and 1 indicating that 2 home ranges occupy exactly the same area in space.

Contacts between foxes were recorded by both collars in a pair, and often these records differed slightly between collars due to differences in transmitter strength, receiver sensitivity, and the location of each animal in the environment during a contact. To account for these differences, for each fox pair we merged contact records that overlapped so the resulting record reflected the maximum amount of time that each contact lasted. For each fox pair, we determined the total number of contacts and amount of time in contact over the life of both collars, and used these to calculate the number of contacts per day and seconds in contact per day.

In analyses of home range overlap and contact rates, we only included fox pairs that were determined not to be mates or family members, and had neighboring home ranges. We eliminated mates and family members because they are known to have much higher home range overlap than non-related pairs regardless of the local fox density (Crooks and VanVuren 1996, J. K. Young and D. K. Garcelon, Institute for Wildlife Studies, unpublished data). Male-female pairs were determined to be mates if they had large amount of home range overlap, were visually observed resting or foraging together, and spent more time together than other male-female pairs at the same site (according to the contacts logged by proximity collars). We defined "neighbors" as foxes with home ranges that overlapped or had borders ≤250 m of one another.

We determined which home range estimation method was most strongly correlated with fox contact rates by regressing the home range size of each fox, calculated with each FK and MCP method, with the per day rates of contact it had with neighbors. The home range method most strongly correlated with contact rates was used in further analyses. To determine if differences existed in contact rates between neighbors of various sex combinations, analyses of home ranges and contacts were done for all pairs together, and for female-female, male-male, and female-male pairs separately.

Fox densities appear to be influenced by habitat type, both of which vary over spatial scales smaller than our effective trap area in parts of the island. That is, although trapping grids were restricted to single habitats, some foxes captured and collared in a grid had home ranges that extended into other habitats. To account for this variation, we assigned each fox a weighted density based on the percentage of its home range area that occupied each habitat type. This density value was then used in regressions with home range size, the Minta value for home range overlap, and contact rates.

Disease Model

We used R to simulate the spread of a novel, virulent disease through an island fox population in a simplified island landscape. Foxes were spatially distributed at varying densities, reflecting the heterogeneity seen on San Clemente Island. Model treatments included changing the disease (rabies or CDV), the site on the island where the disease was introduced, and the number and location of vaccinated animals. These variations allowed us to explore the spatial and temporal spread of these diseases under different conditions, and assess the potential effectiveness of various management strategies that could be implemented on San Clemente Island.

Our model island approximated the dimensions of San Clemente Island and contained approximately 1,000 fox home ranges during each simulation (Fig. 3), approximating the current estimated fox population (Garcia and Associates 2009). Because mated pairs had such high overlap in their home ranges and were in close contact so much, we assumed that both members of a fox pair were infected at effectively the same time. Therefore, there was no distinction between mated pairs and only their shared home range was represented. The island was divided into 4 blocks, with each block reflecting the density of foxes observed at each of our 4 study sites. Briefly, from north to south the blocks were "high" density reflecting the dunes and developed areas on the north end of the island, "medium low" density representing the gentle sloping maritime desert scrub habitats dominating the northwestern portion of the island, "medium high" density representing the rugged maritime desert scrub dominating the southeastern and southern area of the island and "low" density representing the grasslands dominating the central plateau running down the island (Figs. 1, 3). High

Fig. 3. Model representation of San Clemente Island with circular fox home ranges at varying densities.

density habitat was assigned an area equivalent to 10% of the simulated landscape, while the remaining three habitats each covered 30% of the simulated landscape. The relative areas of the
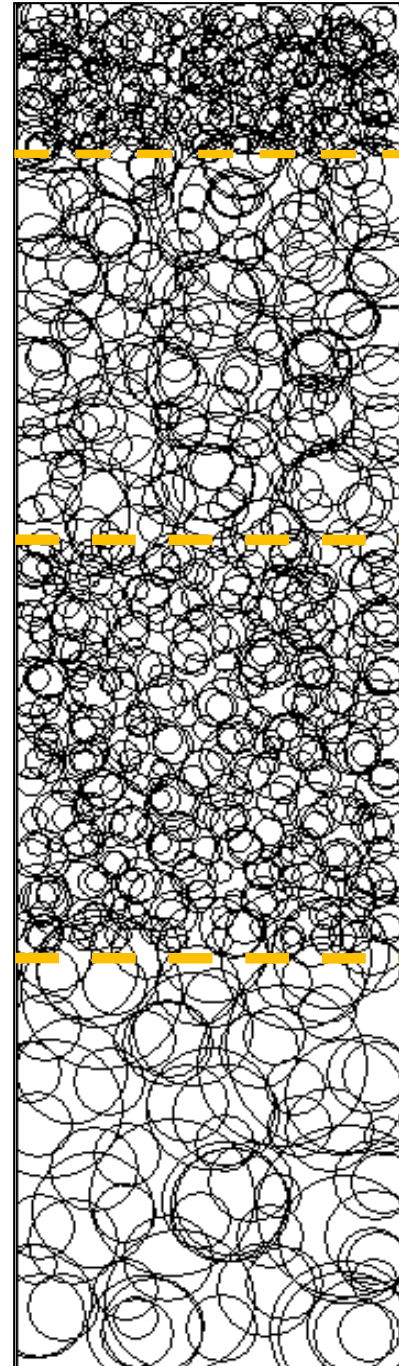
different habitat types were designated to roughly match the relative areas each habitat type comprises of SCI and to facilitate creating landscapes with a target population of 1000 home ranges.

In each block, the size of circular fox home ranges, was determined by the regression of fox densities and home range sizes of collared foxes. Simulated home ranges were restricted to overlap no more than the maximum degree observed between non-related pairs (Minta value ≤ 0.75).

At the start of each simulation, infection was forced into 1 home range located in a high or low-density area of the island (northern or southern end, respectively). The high-density northern end of San Clemente is where the town, airport, and harbor are located. This is considered a high risk area for pathogen introduction because people and objects are being transported to and from the mainland on a regular basis. The southern end of the island has a low to medium-density of foxes, and is a large, unoccupied area with many beaches not regularly patrolled by the U.S. Navy and with access by land restricted due to the nature of training activities in the area. Civilian boaters have been known to come ashore here with domestic dogs, which may expose foxes to canine pathogens.

Each model simulation consisted of 365 daily time-steps (t) representing 1 year, and 100 simulations were run for each scenario (different disease introduction sites and vaccination strategies). At each time-step, the risk of infection for susceptible animals was calculated, and the status of every fox updated based on its current disease class (susceptible [S], latent [E], infectious [I], and removed [R]). The infection probability (λ_{ij}) for each susceptible fox (i) was calculated based on the percent of home range overlap (m) and contact (c) with any adjacent infected fox (j) as well as the disease specific transmission rate (β). The parameters of the

regression line between overlap and contact rates were used as a model function to determine the amount of contact between simulated foxes based on the degree of overlap between their home ranges. Rabies is most commonly transmitted through saliva when an infected host bites a susceptible host (World Health Organization 2005), and because rabid animals are highly aggressive even brief encounters with rabid hosts are likely to result in bites. Since the risk of transmission depends on a susceptible host encountering an infectious host during a short period of infectivity before death (1–5 days; Appendix A), and this risk may increase with the frequency of contact, the parameters resulting from the regression of overlap with number of contacts per day were used when modeling rabies. Transmission of CDV generally occurs from inhalation of aerosol droplets from the respiratory tract, or contact with oral and ocular fluids of a sick animal (Deem et al. 2000, Williams 2001). Distemper can cause chronic disease, sometimes with no visible symptoms (Williams 2001). Because longer exposure to a sick animal may increase the chance of transmission via aerosol or contact with bodily fluids, the parameters resulting from the regression of overlap with the time in contact per day were used for modeling CDV. The probability of pathogen transmission during host-host contacts and the length of latent and infectious periods were obtained from the literature describing rabies and CDV in mainland species such as fox, mink, ferret, and domestic dogs (Appendix A and B).

Foxes may come into contact with non-neighboring animals due to juvenile dispersal, spatial shifts as infected foxes die and home ranges are left unoccupied, and behavioral changes associated with rabies and CDV. In order to account for these unknown variations in contact rates, all uninfected foxes had a low-level background risk of infection (μ) even if they were not adjacent to an infected home range. This background risk was based on the number of long distance forays we detected with radio telemetry and proximity collar data, and scaled with the number of infectious foxes in the population.

Once a fox was infected with a disease, it entered the latent class for a number of time-steps dependent on which disease is being modeled (Appendix A and B). On average this was 60 days for rabies and 7 days for CDV. Next it entered the infectious class, where it could infect other foxes through contact with neighbors or long distance transmission. This stage lasted for an average of 3 days for rabies and 18 days for CDV. In our model there was no recovery of infectious animals, and they eventually died and entered the removed class, where they remained for the rest of the simulation.

To evaluate the effectiveness of recent and proposed epidemic management strategies for SCI foxes, we simulated 1) when an epidemic would be detected using sentinel foxes recommended in the epidemic response plan (Hudgens et al., 2011) and 2) preventative vaccination strategies recently used on SCI (M. Booker, U.S. Navy personal communication).

"Sentinel" foxes are radio collared foxes have not been vaccinated and are monitored in the hope that managers will be alerted to an epidemic when several of the sentinel animals die in quick succession. To model this, we chose a random 10% of the susceptible animals in each simulation (approximately 100 foxes) to be disease sentinels, and recorded the days the first 5 of these animals died. Due to the logistical limitations and time delays in detecting mortality, retrieving it, sending it off island for necropsy, and receiving a cause of death, managers anticipate there may be a delay of a week or more between sentinel mortality and a potential disease diagnosis.

To better understand the effects vaccination may have on an epidemic, we modeled 3 distributions of vaccinated animals. In all 3 treatments, vaccinated animals made up 10% of the fox population (approximately 100 foxes) and stayed in the vaccinated class for the entire simulation. Vaccinated animals were distributed at random across the island or clumped together in 1 of 2 core areas called a "firewall" (Fig. 4; Vickers and Clifford 2009). Firewalls were

bands of vaccinated animals spanning the width of the island. One firewall was placed at the border between high and medium-low density habitats, corresponding to a firewall placed to reduce the impact of an epidemic originating in high-risk entry points on the northern portion of the island (e.g., landing docks, town). The second was placed within the medium-high density block simulating vaccinations along the northern edge of the restricted access southern third of the island to restrict the impact of an epidemic originating in the relatively frequently (unauthorized) accessed and remote southern beaches.

We made several simplifying assumptions in the model that may influence model interpretation. These include:

1. No recovery. Almost all foxes that become infected with rabies or CDV eventually die, except for a small percentage that may survive as consistently infectious individuals. This reflects the reality that rabies is almost invariability fatal to terrestrial mammals, and only approximately 1% of the exposed foxes on Santa Catalina Island survived the 1999 CDV epidemic (Timm et al. 2009).

2. Vaccination is 100% effective. All vaccinated animals remain immune throughout the entire simulation. This is a reasonable assumption for rabies, however, only 50 – 83% of animals injected with CDV vaccine may develop immunity (Wimsatt et al. 2003). Therefore, in order to achieve the target number of immune animals, managers must vaccinate 20 – 50% more animals against CDV than they hope to ultimately have protected. In the model we assume that this has been done, and vaccinated animals represent the individuals who are completely protected.

3. Mates always infect one another. Due to the high Minta values and rates of contact between mated pairs, it is likely that if one individual in a pair is infected with either

disease it will quickly infect its mate. We did not model mates individually, but rather modeled home ranges which represent individual animals as well as mated pairs.
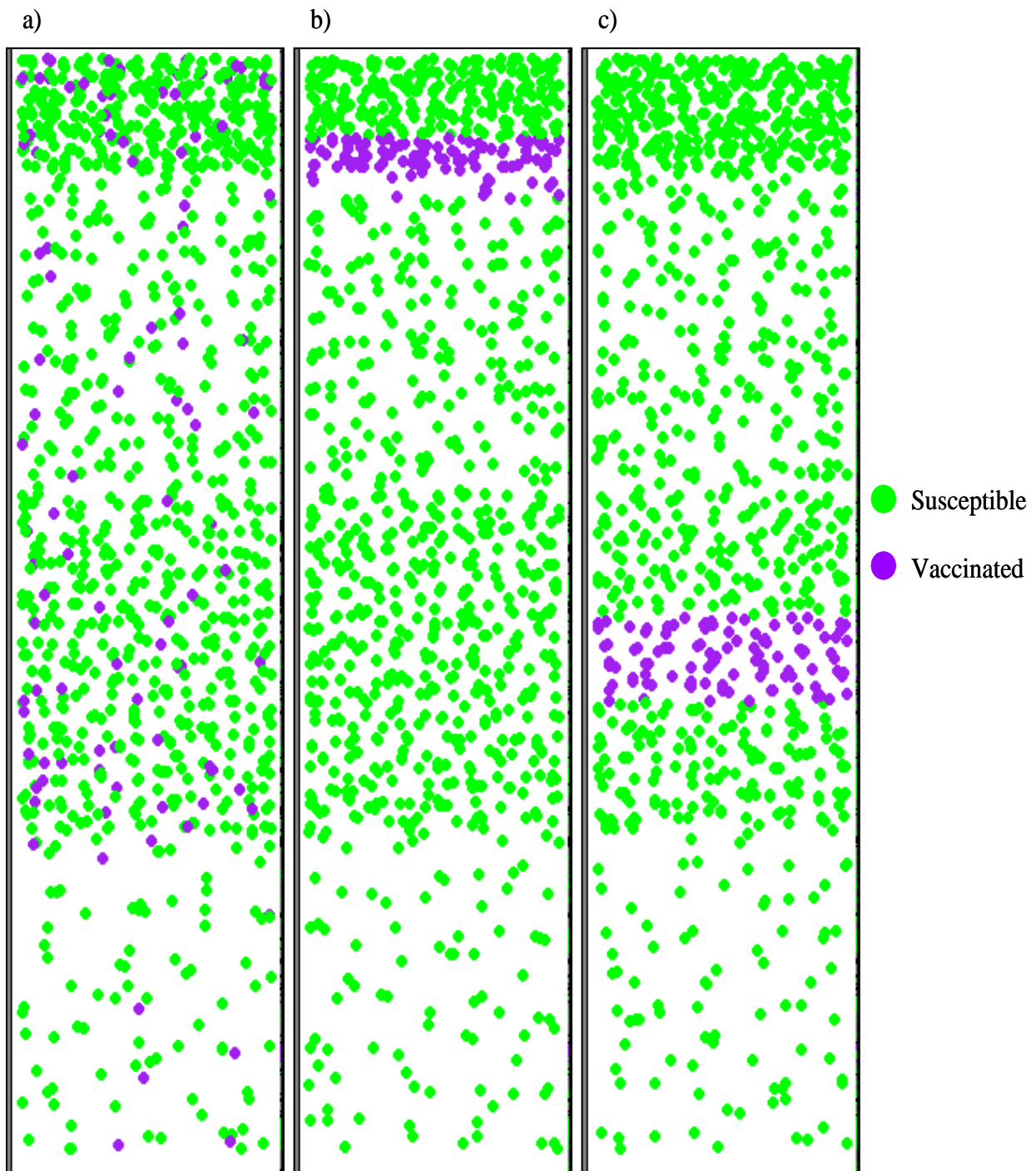

Fig. 4. Model representation of fox home range centers on San Clemente Island showing (a) random vaccination, (b) high-density firewall, and (c) medium-density firewall.

RESULTS

Trapping

We captured 12 – 45 animals on each grid. There was approximately a 15 fold difference in fox densities between grassland and dune habitats while the 2 MDS habitats supported intermediate densities of foxes (Table 2). Eight foxes (4 females and 4 males) at each site were fitted with proximity collars, and an additional 8 foxes (all females) at Dunes and 2 foxes (all males) at Horton were fitted with VHF only collars to ensure the majority of the animals at each site were collared.

Table 2. Results of grid trapping for foxes on San Clemente Island in July 2010. Local population size and density are estimates (±SE).

Study Site	Dominant Habitat	Individuals Caught	Number of Foxes Collared	Density (foxes/km^2)
Dunes	Sand Dune	45	16	42.85 (9.43)
Warren	MDS Rugged	21	6	11.33 (4.73)
West Shore	MDS Gentle	15	8	4.09 (1.82)
Horton	Grassland	12	10	2.87 (1.28)

Telemetry

Of the 42 foxes collared, 2 foxes at the Warren site were dropped from the analyses. One male was never relocated after being collared; we believe this was due to VHF transmitter failure. One female was located throughout the study period, but was found dead at the end of the season. We chose to remove her from home range analyses because her locations for the previous months were very close to one another, indicating that she may have been sick or

injured and not behaving normally for some time before her death. We located the remaining 40 foxes a total 11 – 30 times each.

Home Ranges and Contact Rates

Fox home ranges were not always located within just one habitat, and we noted difference in the size of home ranges within a study site when those home ranges were not in the same dominant habitat. We assigned each fox to the habitat that occupied the largest percentage of its home range area for further analyses. Average home range sizes varied between habitats with the highest density habitat (sand dune) having the smallest home ranges and the lowest density habitat (grassland) having the largest home ranges, regardless of home range estimation method (Table 2, Fig. 3).

Table 2. Mean (±SE) minimum convex polygon (100% MCP) and fixed kernel (50, 85, and 95% FK) estimates of home range size (km^2) of island foxes on San Clemente Island, California, from July 2010 to January 2011.

Habitat	n	Home Range Estimation Method			
		100% MCP	95% FK	80% FK	50% FK
Sand Dune	12	0.16 (0.04)	0.21 (0.05)	0.14 (0.03)	0.05 (0.01)
MDS Rugged	9	0.57 (0.15)	0.99 (0.18)	0.65 (0.10)	0.22 (0.03)
MDS Gentle	12	0.43 (0.09)	0.87 (0.14)	0.62 (0.10)	0.24 (0.04)
Grassland	7	0.66 (0.16)	1.39 (0.27)	0.95 (0.18)	0.36 (0.07)

Density was negatively correlated with home range size ($R^2 = 0.414$, $F_{1,38} = 26.83$, $P < 0.001$), uncorrelated with overlap ($R^2 = 0.029$, $F_{1,274} = 8.041$ $P = 0.005$), and was weakly, positively correlated with number of overlapping home ranges ($R^2 = 0.123$, $F_{1,38} = 5.314$, $P = 0.027$). Home
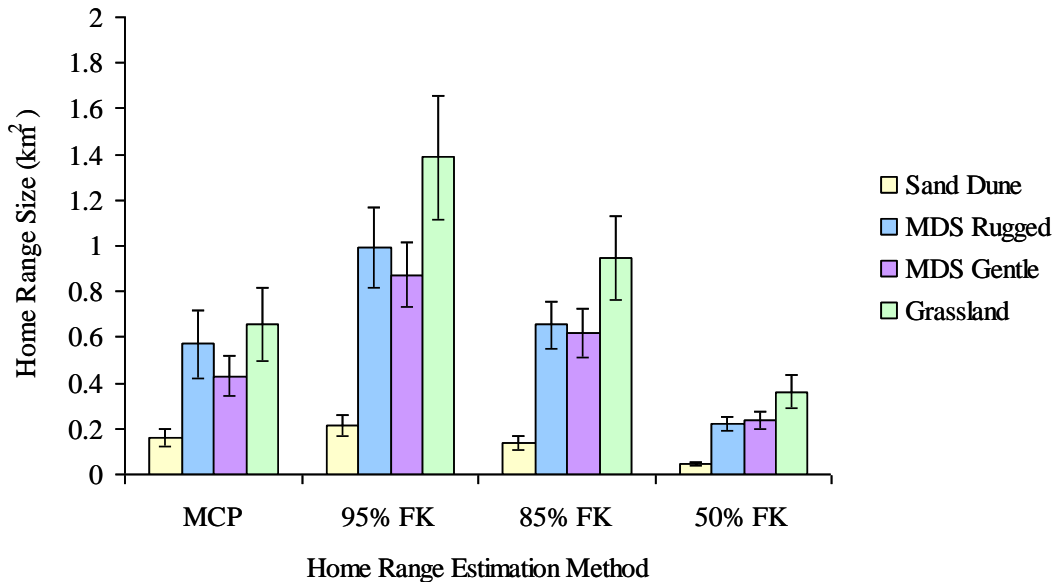
Fig. 5. Mean (±SE) home range sizes in each habitat type for each home range estimation method.

range overlap was positively correlated with the number of contacts per day ($R^2 = 0.210$, $F_{1,71} = 18.86$, $P < 0.001$) and seconds in contact per day ($R^2 = 0.088$, $F_{1,71} = 6.865$, $P = 0.011$) between fox pairs. Density was neither correlated with number of contacts per day ($R^2 = 0.003$, $F_{1,71} = 0.183$, $P = 0.670$) nor with seconds in contact per day ($R^2 < 0.001$, $F_{1,71} = 0.003$, $P = 0.957$). There was no difference in any of these relationships between sexes or among different sex pairs.

Disease Model

Rabies moved slowly through the fox population, and the number of infected (latent or infectious) or dead animals increased slowly over the course of one year, with the peak number of infected animals occurring at the end of the year (Fig. 6). With no vaccination and high density introduction, the first 5 sentinel animals died between days 131 and 220, when on

average 89.9 – 95.5% of the original population was still susceptible and 2.3 – 3.5% of the original population was latent (Fig. 6). Pathogen introduction at low densities resulted in sentinels dying later, between days 211 and 283, when on average 98.7 – 99.0% of the original population was still susceptible and 0.2% of the original population was latent (Fig. 6).
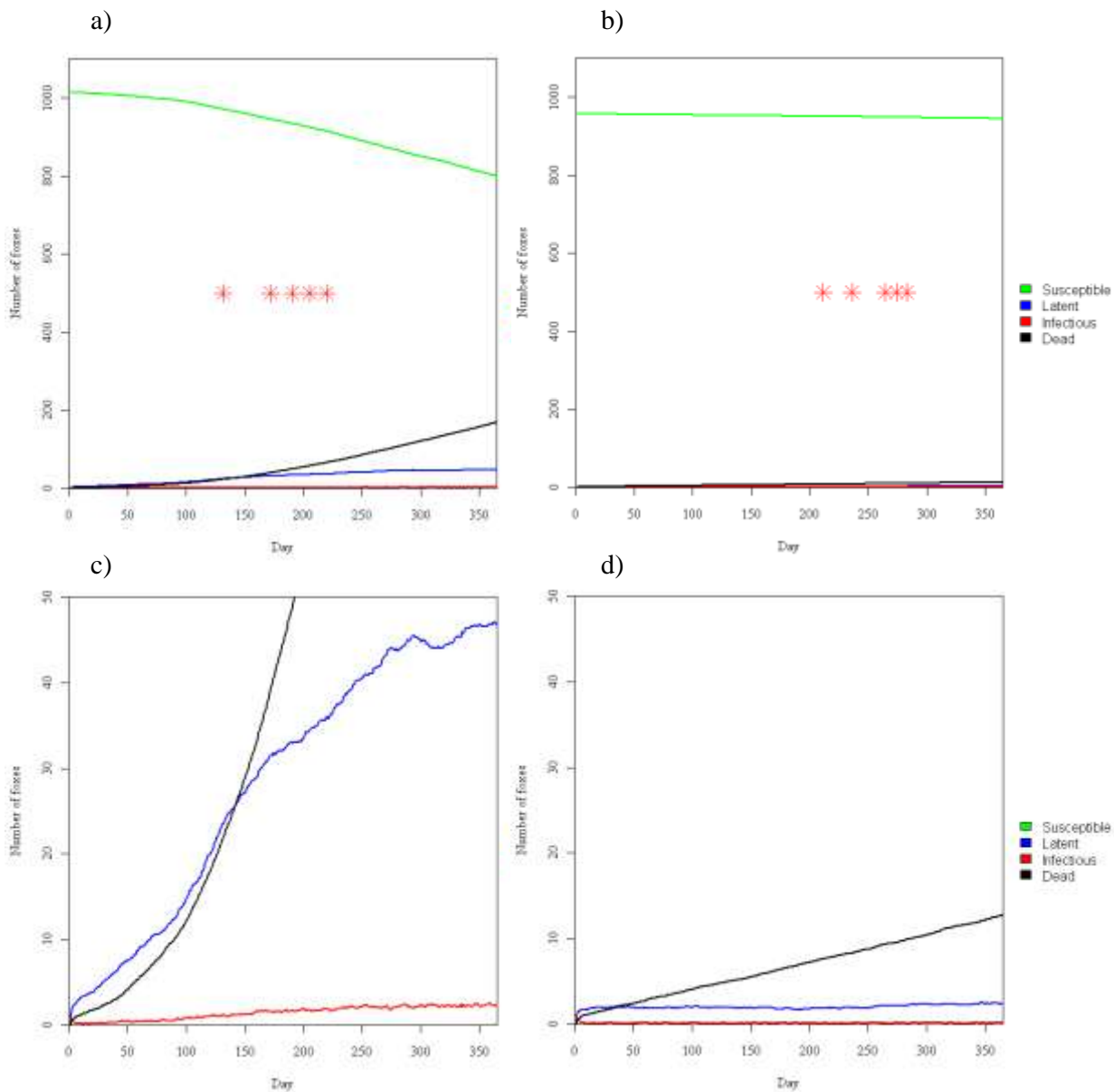

Fig. 6. Mean number of animals in each disease stage class averaged over 100 simulations of rabies introduction at (a) high-density and (b) low-density sites with no vaccination. Asterisks represent the average day at which the first 5 sentinels died. Plots c and d are the same as plots a and b but zoomed in to better show the latent and infectious classes.

When rabies was introduced in high-density areas with no vaccination, a higher percentage of the original susceptible population were infected or dead at the end of one year (mean = 21.3%) compared to the introduction of disease at low densities (mean = 1.6%; Fig. 7). When foxes were vaccinated at random, the average percentage of foxes infected or dead at the end of one year decreased by one quarter for high density pathogen introduction (16.0%), but remained almost the same for low density introduction (1.9%; Fig. 7). When foxes were vaccinated in a high-density firewall and disease was introduced into the high-density area, the percentage of infected or dead animals leveled off and started to decline near the end of one year (15.0%). When foxes were vaccinated in a medium-density firewall and disease was introduced into the low-density area, the percentage foxes infected or dead at the end of one year remained low (3.4%; Fig. 7).
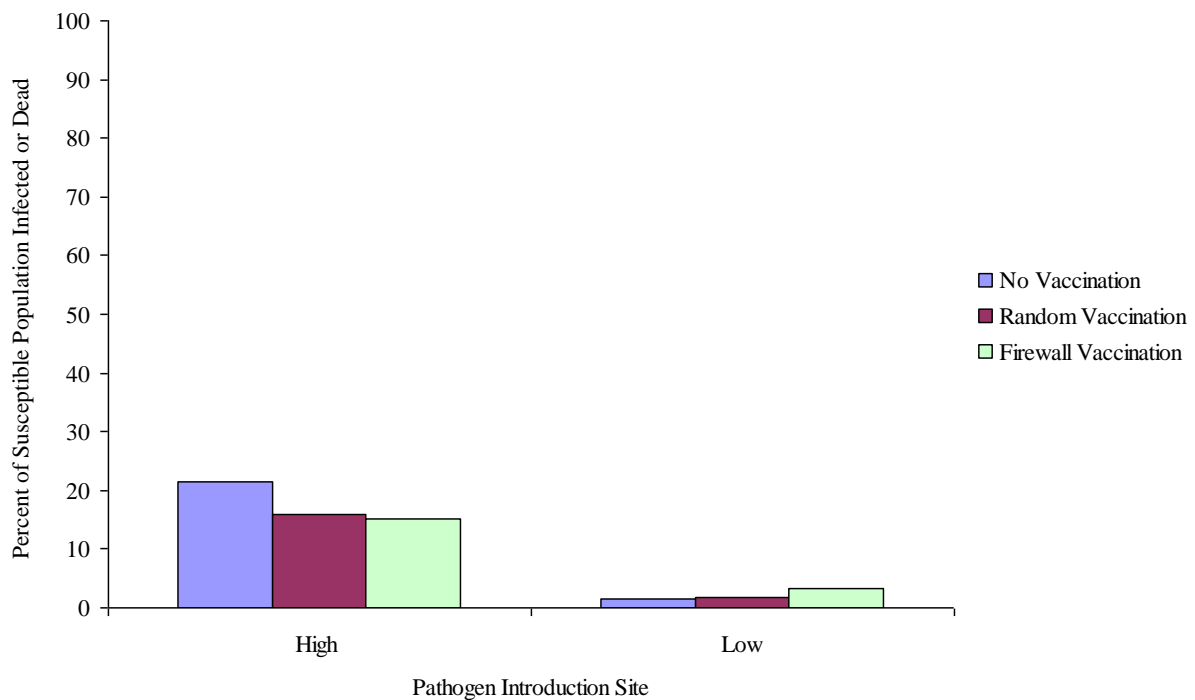

Fig. 7. Mean percentage of original susceptible population infected or dead at the end of one year after the introduction of rabies using different vaccination strategies.

Distemper virus had a much greater impact on the fox population in one year than did

rabies, and with no vaccination it infected almost all the animals on island by the end of one year

(Fig. 8). With no vaccination and high density introduction, the first 5 sentinel animals died

between days 25 and 41, when on average 69.6 – 86.8% of the original population was still

susceptible and 5.3 – 7.5% of the original population was latent (Fig. 8). Pathogen introduction

at low densities resulted in sentinels dying later, between days 51 and 84, when a greater

proportion (75.4 – 91.4%) of the original population was still susceptible and fewer animals (1.9
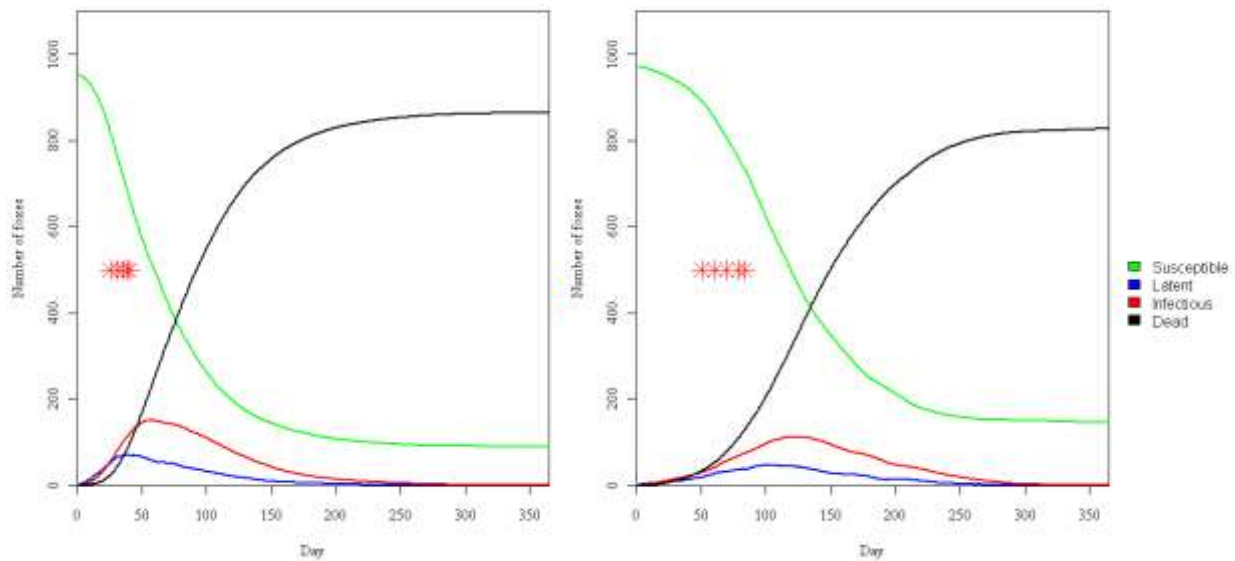

Fig. 8. Mean number of animals in each disease stage class averaged over 100 simulations of CDV
introduction at (a) high-density and (b) low-density sites with no vaccination. Asterisks represent
the average day at which the first 5 sentinels died.

– 3.8%) were latent (Fig. 8).

In contrast to rabies, at the end of most CDV simulations the number of animals latent or

infectious had started to decline, and the majority of the original population was susceptible or

dead. When CDV was introduced in high-density areas with no vaccination, a similar

percentage of animals were infected or dead at the end of one year compared to the introduction of disease at low densities (90.0% and 84.7%, respectively; Fig. 9). However, at high densities the disease moves more quickly, with the number of infected animals peaking on average at day 53, compared to a peak at day 120 when the disease is introduced at low densities (Fig. 8). When foxes were vaccinated at random and disease was introduced at high densities, the percentage of foxes removed at the end of one year decreased slightly (85.0%), however, random vaccination decreased the percentage of infected or dead animals by approximately half when disease was introduced at low densities (44.0%; Fig. 9). There was only a slight decrease in the percentage of animals infected between high-density and low-density firewalls (77.4% and 72.4%, respectively; Fig. 9).
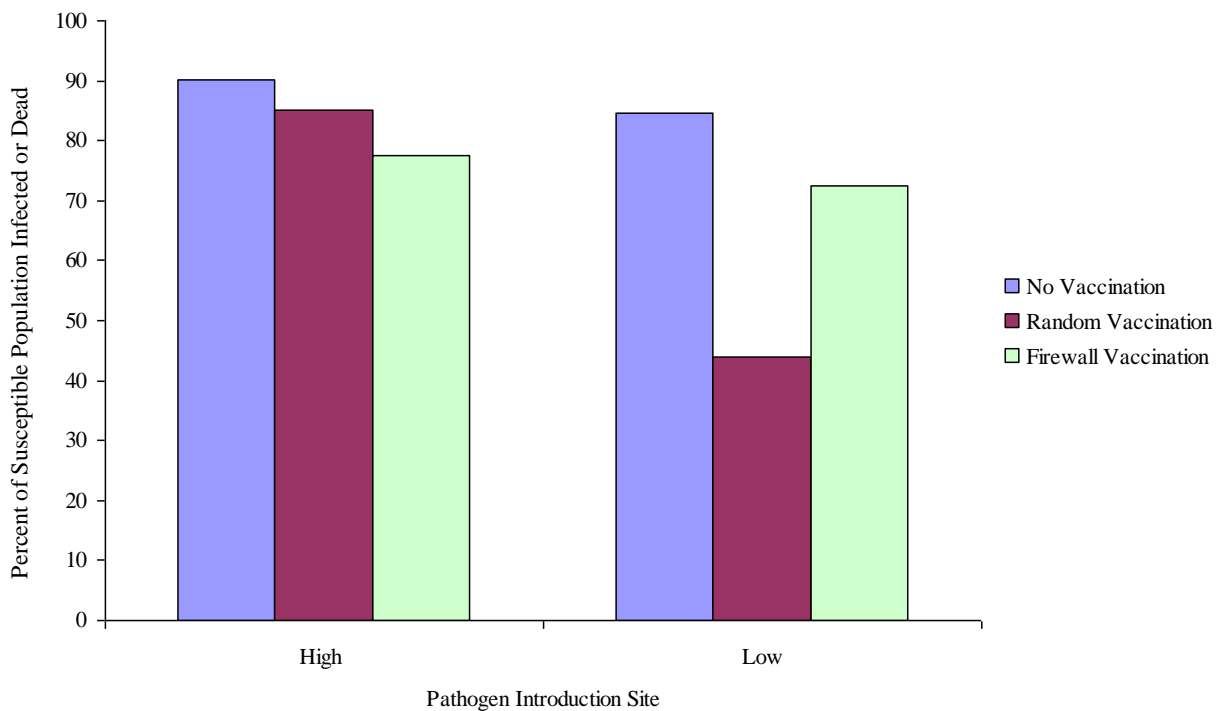
Fig. 9. Mean percentage of original susceptible population infected or dead at the end of one year after the introduction of CDV using different vaccination strategies.

When either disease was introduced in a part of the island with a low density of foxes, the majority of simulations resulted in epidemic fadeout (i.e., no latent or infected foxes remain so that the pathogen is extirpated from the island).. The percentage of simulations resulting in fadeout was higher for CDV than for rabies regardless of where the disease was introduced (fadeout occurred in 94% and 95% of CDV simulations compared to 17% and 62% of rabies simulations introduced at high and low density regions, respectively,). At high-density disease introduction the average percentage of the fox population left susceptible and infected at the end of one year was lower and higher, respectively, compared to low-density introduction.

DISCUSSION

Foxes at high densities experience greater overall amounts of contact with neighbors than foxes at lower densities. This is primarily due to an increase in the number of neighbors with overlapping home ranges, and not an increase in the amount of pair-wise overlap between neighbors. Although a fox in a high-density area may not overlap with each neighbor more or contact those neighbors more often, it has a greater number of neighbors that it interacts with, thereby increasing its total amount of contact.   Our estimate of the number of neighbors at high density is likely conservative, because we collared a lower proportion of the local fox population than at the lower density sites. The number of neighbors and overall contact experienced by foxes at high densities is likely higher than we were able to measure. The contact between two neighboring foxes is best predicted by the degree of overlap between their two home ranges, and not the local density of foxes.

These relationships between density, home range dynamics, and contact rates are important for understanding potential pathogen spread. Our disease model showed that fox contact rates and spatial distribution may determine the course of an epidemic. For rabies and CDV, an introduction to areas of high fox density resulted in more animals becoming infected

more quickly than if the disease was introduced at lower densities. This is because each infectious fox has more potentially susceptible neighbors it is in contact with, and can therefore cause a greater number of secondary infections. In contrast, at low densities each infected fox has fewer neighbors it can infect, which results in the disease spreading more slowly.

In the case of rabies, at lower densities where fewer home ranges overlap one another, there is a greater chance of epidemic fadeout due to all neighbors surrounding an infectious fox already being infected or removed, or because an infectious fox has fewer susceptible neighbors it can infect. Rabies also has a very short infectious period (approximately 1 – 5 days), so infectious animals may die before they are able to infect their neighbors. In contrast, CDV has a long infectious period (approximately 14 – 21 days), which increases the probability of contact and disease transmission before death. The result is that CDV fades out much less often than rabies, even at low densities, and when fadeout does occur it is due to the disease infecting the entire fox population and running out of susceptibles rather than simply not being transmitted often enough. Even at high densities, over the course of a year rabies may spread more slowly and infect fewer animals than CDV due to this difference in latent and infectious periods.

Traditional non-spatial models can overestimate the minimum level of immunization that is required to control a disease (Eisinger and Thulke 2008). Our spatially explicit model more accurately determines the best method of vaccination by simulating the presence of vaccinated hosts in the population and predicting how the location of those animals may affect pathogen spread. Our simulations show that vaccinating foxes randomly or in a firewall may prevent or slow disease spread. Compared to no vaccination, random vaccination reduced the overall number of removed animals for both diseases whether the pathogen was introduced at high or low density. This reduction was larger when the disease was introduced at low densities. Foxes at lower densities have fewer neighbors, therefore vaccination of even one

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neighbor greatly reduces the chance that an infected fox will transmit a pathogen. In contrast, at high densities an infectious animal has so many neighbors that one being vaccinated will not help slow disease spread because many neighbors are still capable of being infected.

Firewalls stopped the epidemic in simulations of rabies at both pathogen introduction sites, but not for CDV. This is likely due to the background infection rate used to account for the potential of long-distance dispersal events by infected animals. On average rabies simulations only had a few animals in the infectious class at any time-step, so the background infection rate was low and it was rare that an infectious animal would infect a susceptible fox on the other side of the firewall within the short time before it died. However, foxes infected with CDV have a shorter latency period and are infectious much longer. Consequently, there were more animals in the infectious class at the same time, which increased the background infection rate to a level at which it was not uncommon for a susceptible fox on the other side of the firewall to contract the disease. A solution to this may be to vaccinate more animals near likely disease introduction sites so fewer animals can get infected, thereby lowering the number of infectious foxes that may cross the firewall.

This model can also be used to determine manager response in the face of an epidemic. If managers can approximate how fast disease will spread across the island based on where infected animals have been observed, they can decide if the best response would be to trap at the front of the disease wave to stop immediate infections, if trapping should occur on the far end of the island in an attempt to vaccinate the foxes least likely to have been exposed, or if (in the case of rabies) the human health risk is too great for hand vaccination and oral baits containing vaccine should be distributed instead. In our model, the first sentinels died earlier in simulations of CDV, which is consistent with CDV infecting foxes more quickly than rabies. However, even though managers may detect a CDV epidemic sooner, it is probable that a higher

proportion of the starting population will already infected at that point. Infected animals cannot be cured and are effectively removed from the population of foxes that can be saved once an epidemic is underway. Regardless of vaccination strategy, managers may have less time to respond to a CDV epidemic with additional vaccinations or quarantine of susceptible animals. Although managers may detect a rabies epidemic later, it is likely that fewer animals will be already infected, regardless of introduction site. This suggests that mangers may have more time to respond to a rabies epidemic, and may be more successful in saving a large proportion of the original fox population.

Our model assumes no recovery after infection, but in reality a small percentage of foxes may recover from CDV to become immune. This means our model predictions of the number of

Photo: Jessica Sanchez

foxes remaining alive may be overly pessimistic. Incorporating recovery may result in there being a larger proportion of the original susceptible population alive at the end of 1 year. Although recovery would likely be too slow to change the rate of CDV spread across the island, it would give managers a larger population of survivors after an epidemic to recover the population through natural or captive breeding. Reduced mortality from CDV, however, is unlikely to prevent the necessity for intensive management to recover fox populations in the absence of preventative measures or a quick and effective response. It is also possible that some sentinel animals may become infected but recover, and therefore never be detected as disease mortalities, increasing the amount of time before managers detect an epidemic.

We also assumed that mates become infected at the same time, which could mean our results of the speed at which rabies and CDV spread are worst case scenarios. Mates have large amounts of home range overlap, so disease does not spread very far spatially when it infects the second fox in a mated pair. If there is a significant delay between when one mate gets infected and when it infects its mate, disease transmission across the island could be slower than our model expects.

MANAGEMENT RECOMMENDATIONS

CDV may be a greater threat to island fox populations than rabies because it will move more quickly through the susceptible population. Therefore we recommend vaccinating to maximize the number of animals that will survive a CDV epidemic. When CDV was introduced at high densities, the number of susceptible foxes alive at the end of one year was only slightly higher using high-density firewall versus random vaccination. When CDV was introduced at low densities, the number of susceptible foxes alive at the end of one year was substantially higher when foxes were vaccinated randomly instead of in a medium-density firewall. It appears that random vaccination is the best strategy for ensuring the most foxes across the island survive

a CDV epidemic no matter where it is introduced. All vaccination strategies were successful in reducing the number of animals infected with rabies, although firewalls eventually stopped the epidemic completely while random vaccination only stopped the epidemic by chance. However, because there will likely be more time for managers to respond to a rabies epidemic, the increased risk of a continuing rabies epidemic might be a worthwhile tradeoff if random vaccination is more successful than a firewall in preserving animals during a CDV epidemic.

Future work addressing disease introduction on San Clemente Island should focus on exploring how variations in the parameters modeled here might affect disease dynamics. Vaccinating animals closer to high-risk disease introduction sites or increasing the proportion of the total population vaccinated may help stop disease spread earlier in an epidemic. Incorporating recovery into simulations of CDV may increase the number of foxes we expect to be alive at the end of 1 year, or alter our detection of an epidemic due to sentinel animals recovering. Changing the site of disease introduction may also be a realistic scenario to explore. These are variations that will give managers more information on how they can best prepare for the introduction of a novel, virulent disease in a way that maximizes fox survival, and minimizes the necessity of further human intervention.

ACKNOWLEDGEMENTS

LITERATURE CITED

Appel, M. J. G. 1987. Canine distemper virus. Pages 133–149 *in* M. J. G. Appel, editor. Virus Infections of Carnivores. Elsevier Science Ltd, Amsterdam, Netherlands.

Blancou, J. 1988. Ecology and Epidemiology of Fox Rabies. Reviews of Infectious Diseases 10:S606–S609.

Blancou, J., M. F. A. Aubert., and M. Artois. 1991. Fox Rabies. Pages 257–290 *in* G. M. Baer, editor. The Natural History of Rabies. Second edition. CRC Press, Boca Raton, Florida, USA.

Clifford, D. L., J. A. K. Mazet, E. J. Dubovi, D. K. Garcelon, T. J. Coonan, P. A. Conrad, and L. Munson. 2006. Pathogen exposure in endangered island fox (*Urocyon littoralis*) populations: Implications for conservation management. Biological Conservation 131:230–243.

Collins, P. W. 1993. Taxonomic and Biogeographic Relationships of the Island Fox (*Urocyon littoralis*) and Gray Fox (*U. cinereoargenteus*) from Western North America. Pages 351–390 *in* F. G. Hochberg, editor. Third California Islands Symposium: Recent Advances in Research on the California Islands. Santa Barbara Museum of Natural History, Santa Barbara, California, USA.

Coonan, T. J. 2008. Island Fox Recovery Program: San Miguel and Santa Rosa Islands 2008 Annual Report. The National Park Service Technical Report 08–01, Channel Islands National Park, California, USA.

Crooks, K. R., and D. Van Vuren. 1995. Resource utilization by two insular endemic mammalian carnivores, the island fox and island spotted skunk. Oecologia. 104:301-307

Crooks, K. R., and D. Van Vuren. 1996. Spatial organization of the island fox (*Urocyon littoralis*) on Santa Cruz Island, California. Journal of Mammalogy 77:801–806.

Deem, S. L., L. H. Spelman., R. A. Yates, and R. J. Montali. 2000. Canine Distemper in Terrestrial Carnivores: A Review. Journal of Zoo and Wildlife Medicine 21:441–451.

Dickson, B. G. and P. Beier. 2002. Home-Range and Habitat Selection by Adult Cougars in Southern California. Journal of Wildlife Management. 66:1235–1245

Duong, T. 2011. ks: Kernel smoothing. R package version 1.8.2. ‹ http://CRAN.R-project.org/package=ks›

Eisinger, D., and H. H. Thulke. 2008. Spatial pattern formation facilitates eradication of infectious diseases. Journal of Applied Ecology 45:415–423.

Efford, M. G., D. K. Dawson, and C. S. Robbins. 2004. DENSITY: software for analyzing capture-recapture data from passive detector arrays. Animal Biodiversity and Conservation 27:217–228.

Farrell, R. K., J. R. Gorham, and L. O'Neill. 1955. The response of young mink to varying lengths of exposure to nebulized egg-adapted distemper virus (DV). Veterinary Medicine 50:412–414.

Fekadu, M. 1988. Pathogenesis of Rabies Virus Infection in Dogs. Reviews of Infectious Diseases 10:S678–S683.

Frick W.F., Pollock J.F., Hicks A.C., Langwig K.E., Reynolds D.S., Turner G.R., Butchkoski C.M., and T. H. Kunz. 2010. An emerging disease causes regional population collapse of a common North American bat species. Science. 329:679-682

Garcelon, D. K., and B. R. Hudgens. 2008. Island fox monitoring and demography on San Nicolas Island – 2007. Unpublished report prepared by the Institute for Wildlife Studies, Arcata, California, USA.

Garcelon, D. K., J. A. Fox, and J. N. Sanchez. 2008. Island fox recovery efforts on Santa Cruz Island, California, February 2007 – March 2008. Unpublished report by the Institute for Wildlife Studies, Arcata, California for The Nature Conservancy, Santa Cruz Island Project, Ventura, California, USA.

Garcia and Associates. 2009. Island Fox (*Urocyon littoralis clementae*) Monitoring and Research on Naval Auxiliary Landing Field, San Clemente Island, California. Draft Large Report Option Period 2; 2009 Field Season.

Gillespie, J. H. 1962. The virus of canine distemper. Annals of the New York Academy of Sciences 101:540–547

Gorham, J. R. 1999. Some experiments and filed observations of distemper in mink and ferrets. Advances in Veterinary Medicine 41:557–570

Gorham, J. R., and C. A. Brandly. 1953. The transmission of distemper among ferrets and mink. Pages 129-141 *in* Proceedings Book. American Veterinary Medical Association, 90th Annual Meeting, 20–23 July 1953, Toronto, Canada.

Hampson, K., J. Dushoff, S. Cleaveland, D. T. Haydon, M. Kaare, C. Packer, and A. Dobson. 2009. Transmission dynamics and prospects for the elimination of canine rabies. Public Library of Science Biology 7:462–471.

Headley, S. A. and D. L. Graca. 2000. Canine distemper: epidemiological findings of 250 cases. Brazilian Journal of Veterinary Research and Animal Science [online] 37.

Holmala, K., and K. Kauhala. 2006. Ecology of wildlife rabies in Europe. Mammal Review 36:17–36.

Hudgens B., Ferrara F., and D. Garcelon. 2007. Remote monitoring of island foxes. Final Report to DoD Legacy Resource Management Program.

Hudgens, B. R. and D. K. Garcelon. 2011. Induced changes in island fox (*Urocyon littoralis*) activity do not mitigate the extinction threat posed by a novel predator. Oecologia 165:699–705.

Laughrin, L. 1977. The Island Fox: A Field Study of Its Behavior and Ecology. Dissertation, University of California at Santa Barbara, Santa Barbara, USA.

Kitala, P., J. McDermott, and C. Dye. 1997. Transmission dynamics of dog rabies in Machakos District, Kenya. Épidémiologie et Santé Animale 31-32:04.04.1–04.04.3.

McCallum, H., N. Barlow, and J. Hone. 2001. How should pathogen transmission be modeled? TRENDS in Ecology and Evolution 16:295–300.

Minta, S. C. 1992. Tests of Spatial and Temporal Interaction Among Animals. Ecological Applications 2:178–188.

National Oceanic and Atmospheric Administration. 2008. Proposed Rules. Federal Register 73:1986.

Okarma, H., W. Jedrzejewski, K. Schmidt, S. Śnieżko, A. N. Bunevich, and B. Jedrzejewska. 1998. Home Ranges of Wolves in Białowieża Primeval Forest, Poland, Compared with Other Eurasian Populations. Journal of Mammalogy 79:842–852.

Olsen, T. L., P. J. Gulman, and E. McKenna. 2000. Wind Power Plant Evaluation, Naval Auxiliary Landing Field, San Clemente Island, California. The National Renewable Energy Laboratory, Golden, Colorado, USA.

Petersen, C. Lovich R., Lanoo M., and P. Nanjappa 2011. Do frogs still get their kicks on Route 66? A transcontinental transect for amphibian chytrid fungus (*Batrachochytrium dendrobatidis*) infection on U.S. Department of Defense installations. Final Report to DoD Legacy Resource Management Program.

Rhodes, C. J., R. P. D. Atkinson, R. M. Anderson, and D. W. Macdonald. 1998. Rabies in Zimbabwe: reservoir dogs and the implications for disease control. Philosophical Transactions of the Royal Society B 353:999–1010.

Roemer, G. W., D. A. Smith, D. K. Garcelon, and R. K. Wayne. 2001. The behavioral ecology of the island fox (*Urocyon littoralis*). Journal of Zoology 255:1–14.

Spencer, W., E. Rubin, J. Stallcup, V. Bakker, B. Cohen, S. Morrison, and R. Shaw. 2006. Framework Monitoring Plan for the San Clemente Island Fox. United States Navy Region Southwest, California, USA.

Schmidt, G. A., B. R. Hudgens, and D. K. Garcelon. 2005*a*. Island fox monitoring and research on Naval Auxiliary Landing Field, San Clemente Island, California, Final Report May 2004 – June 2005. Unpublished report prepared by the Institute for Wildlife Studies, Arcata, California, USA.

Schmidt, G. A., T. W. Vickers, and D. K. Garcelon. 2005*b*. Island fox recover efforts on Santa Catalina Island, California, 1 April 2004 – 30 April 2005. Unpublished report prepared by the Institute for Wildlife Studies, Arcata, California for Santa Catalina Island Conservancy, Avalon, California, USA.

Smith, G. C. and D. Wilkinson. 2002. Modelling disease spread in a novel host: rabies in the European badger *Meles meles*. Journal of Applied Ecology 39:865–874.

Timm, S. F., L. Munson, B. A. Summers, K. A. Terio, E. J. Dubovi, C. E. Rupprecht, S. Kapil, and D. K. Garcelon. 2009. A suspected canine distemper epidemic as the cause of a catastrophic decline in Santa Catalina Island foxes (*Urocyon littoralis catalinae*). Journal of Wildlife Diseases 45:333–343.

Vickers, W. and D. Clifford. 2009. Fox Health Working Group *in* T. J. Coonan, editor. Eleventh Annual Meeting Island Fox Working Group: Summary Report. National Park Service, Ventura, California, USA.

White, G. C., and R. A. Garrott. 1990. Analysis of Wildlife Radio-Tracking Data. Academic Press, Inc., San Diego, California, USA.

Williams, E. S. 2001. Canine Distemper. Pages 50–59 *in* E. S. Williams and I. K. Barker, editors. Infectious Diseases of Wild Mammals. Third edition. Blackwell Publishing, Ames, Idaho, USA.

Wimsatt, J., D. Biggins, K. Innes, B. Taylor, and D. Garell. 2003. Evaluation of Oral and Subcutaneous Delivery of an Experimental Canarypox Recombinant Canine Distemper Vaccine in the Siberian Polecat (*Mustela eversmanni*). Journal of Zoo and Wildlife Medicine 34:25-35

Wood, J. E. 1958. Age Structure and productivity of a gray fox population. Journal of Mammalogy 39:74–86.

Woodroffe, R. 1999. Managing disease threats to wild mammals. Animal Conservation 2:185–193.

World Health Organization. 2005. WHO Expert Consultation on Rabies: First Report. WHO Press, Geneva, Switzerland.

Zinsstag, J., S. Durr, M. A. Penny, R. Mindekem, F. Roth, S. Menendez Gonzalez, S. Naissengar, and J. Hattendorf. 2009. Transmission dynamics and economics of rabies control in dogs and humans in an African city. Proceedings of the National Academy of Sciences 106:14,996–15,001.

Photo: Brian Hudgens

APPENDIX A: Parameter values used to model the spread of rabies in island foxes on San Clemente Island, CA.

Parameter	Symbol	Rabies	Source
Home range size	r	Determined by local density	Field data
Home range overlap (Minta value)	m	≤ 0.75	Maximum overlap between non-related pairs observed field data
Contact rate	c	Number of contacts per day	Field data
Transmission probability given a contact	p	0.49	Hampson et al. 2009
Background infection rate	μ	$1-(1-0.000001)^I$	Long distance forays observed in field data
Transition rate from E to I	σ	1/60	Blancou 1988, Fekadu 1988, Blancou et al. 1991, Kitala et al. 1997, Rhodes et al. 1998, Smith and Wilkinson 2002, Hampson et al. 2009, Zinsstag et al. 2009
Transition rate from I to R	α	1/3	

APPENDIX B: Parameter values used to model the spread of canine distemper virus in island foxes on San Clemente Island, CA.

Parameter	Symbol	Canine Distemper	Source
Home range size	r	Determined by local density	Field data
Home range overlap (Minta value)	m	≤ 0.75	Maximum overlap between non-related pairs observed field data
Contact rate	c	Seconds in contact per day	Field data
Transmission probability given a contact	p	Distribution based on c	Farrell et al. 1955
Background infection rate	μ	$1-(1-0.000001)^I$	Long distance forays observed in field data
Transition rate from E to I	σ	1/7	Gorham and Brandly 1953, Gillespie 1962, Appel 1987, Gorham 1999, Deem et al. 2000, Headley and Graca 2000
Transition rate from I to R	α	1/18	