A National Park Service Manager's Reference Notebook on Plague (*Yersinia pestis*)

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Background

New and emerging wildlife diseases will likely be one of the greatest challenges confronting the National Park Service (NPS) this century. As the world "gets smaller," and people and animals move more frequently and over greater distances, diseases will likely spread. Furthermore, disease severity can be exacerbated by environmental degradation, such as pollution, climate change, species invasions, and changes in land use. Increased prevalence and severity of disease may have ominous consequences for people, wildlife, and the ecosystems upon which they both depend. In fact, many of these incipient diseases can affect both humans and wildlife. The increased occurrence of such diseases may affect NPS operations more than most government bureaus, because the NPS both provides for human safety and well-being while also conserving wildlife and ecological integrity.

In 2003, the NPS Biological Resource Management Division (BRMD), Public Health Division (PHD) and the Risk Management Division (RMD) held a workshop to address this issue. The goal of the workshop was to develop a system of standard operating procedures for determining lead responsibilities and responses to categories of zoonotic and environmentally-transmitted diseases (ZEDs). In 2009,this so-called "ZED" group began transitioning into the NPS One Health Working Group. The One Health concept is based on the premise that the health of people, animals, and our environment are inextricably interconnected. Specifically, One Health advocates for "the establishment of closer professional interactions, collaborations, and educational opportunities across the health sciences professions, together with their related disciplines, to improve the health of people, animals, and the environment" (One Health Commission). This interdisciplinary approach is an exciting and potentially powerful model for health and disease management for veterinarians, physicians, public health officials, wildlife managers, and others. The NPS has been a leader in implementing the One Health approach by improving coordination, communication, and collaboration between the bureau's Wildlife Health Program and its Office of Public Health, as well as with numerous other NPS and external partners.

In May 2009, the One Health Working Group held a two-day workshop to bring together experts from the fields of wildlife health and conservation, public health, risk management, and integrated pest management, to have facilitated discussions on how NPS can use best management practices to approach plague management servicewide. This reference notebook is a result of those discussions and additional literature review and synthesis, and is meant to be a "living document" which will be updated regularly as new information on plague becomes available.

1. Introduction

Plague is an acute and often fatal zoonotic disease caused by the bacterium *Yersinia pestis*. *Y. pestis* mainly cycles between small mammals and their fleas; however, it has the potential to infect humans and frequently causes fatalities if left untreated. It is often considered a disease of the past; however, since the late 1800s, plague's geographic range has expanded greatly, posing new threats in previously unaffected regions of the world, including the western United States. Little is known about how plague, as an exotic invader with a potentially large host range, has influenced wildlife and biodiversity in North America.

The purpose of this notebook is to serve as an information reference summarizing the most pertinent plague literature, management options, and policies as they pertain to NPS units. It is not meant to be an all-inclusive review of the current literature or management options, but it gathers

as much of this information as possible into one resource to be used by NPS managers and employees interested in plague and how it can affect NPS units. Because plague is still considered an emerging disease, and knowledge about the disease continues to expand, this document will be updated regularly.

Acknowledgments

Many people helped put this notebook together. Jennifer Shoemaker and Amber Swallow were instrumental in helping with graphics. Megan K. Eberhardt-Frank provided help with an extensive literature search. Margaret Wild, Chuck Higgins, David Wong, Mike Quinn, and Bruce Badzik contributed to the development of this document. We thank Ken Gage and Pete Gober for their helpful peer review comments. This effort was funded by the NPS Natural Resource Protection Program.

2. Plague Background

2.1 Overview

Plague is a flea-borne zoonotic disease caused by a bacterium, *Yersinia pestis*. *Y. pestis* is essentially a bacterium of wild rodents and their fleas; however, it has the potential to infect humans and cause human fatalities if left untreated (Dennis et al. 1999). *Y. pestis* generally exists in an enzootic (maintenance) cycle where little host mortality occurs, and cycles between rodents and their fleas. When an epizootic event (outbreak of disease in a non-human animal population) occurs, humans and other mammals are at high risk of becoming infected. There have been three historical pandemics due to plague in human history, each of which have caused millions of deaths. The impacts of plague on wildlife species have been much less studied than the effects on humans; however, certain wildlife populations may be severely impacted, especially as plague invades into new geographic areas. Natural plague reservoirs, or rodent populations where plague endlessly circulates, can be found on every continent of the world except Australia and Antarctica.

Many think of plague as a disease of the past; however, it remains a current threat to public health in many parts of the world, particularly in Africa, where both the number of cases and the number of countries reporting plague have increased during recent decades (Stenseth 2008). Plague has been categorized as a re-emerging infectious disease (Schrag and Wiener 1995; Duplantier et al. 2005). It remains the world's most deadly bacterial disease. In North America, plague has not spread person to person since an outbreak in Los Angeles in 1924 when 31 people died of pneumonic plague (from rat-borne plague).

Plague is a new disease, relatively speaking. Between 1,500 and 20,000 years ago *Y. pestis* evolved from a closely related intestinal bacterium, *Y. pseudotuberculosis*. *Y. pseudotuberculosis* infection can result in a relatively mild gastro-intestinal disease that presents with sharp pains in the stomach, diarrhea, fever, vomiting, and bloody stools, but is usually handled effectively by the human immune system. Conversely, *Y. pestis* is highly virulent, often overwhelming the human immune system and causing a rapidly fatal septicemia with multi-organ failure.

Bubonic plague is the most common form of plague, while pneumonic is considered the most virulent but least common type of plague. Bubonic plague infection results from a bite from an infected flea or when plague-infected material enters through the skin. A second type of plague is septicemic plague, which occurs when plague bacteria infects the blood through flea bites or by direct contact with infective materials. Septicemic plague often occurs secondarily to bubonic plague. A third type of plague is pneumonic plague, which can result from inhalation of infectious respiratory droplets or other infectious materials (primary pneumonic plague) or as a result of a secondary blood-borne invasion of the lungs in a person with primary or secondary septicemic plague. Unlike bubonic and septicemic forms, pneumonic plague can potentially spread from person to person through coughing of infectious respiratory droplets (Dennis et al. 1999). Although plague is a serious disease with a case-fatality ratio between 30 to 60% if left untreated, quick diagnosis and treatment of plague with antibiotics with supportive therapy can reduce complications and fatality (Dennis et al. 1999).

2.2 History

World

There have been three recognized plague pandemics in world history attributed to *Y. pestis*. These pandemics resulted in millions of human deaths and have had a tremendous impact on the course of human history (Zinsser 1934; Gregg 1985; McEvedy 1988; Gage 1998). The first pandemic was Justinian's Plague. This outbreak was the first historically documented pandemic of plague and began sometime around 540 CE and persisted for 50-60 years. It is thought to have originated in East or Central Africa and spread from Ethiopia to Egypt and then to countries surrounding the Mediterranean basin (Achtman et al. 1999). It spread along the trade routes of the Byzantine emperor Justinian's realm and human deaths were mostly confined to ports. The black rat (*Rattus rattus*) was readily transported on ships and was retrospectively considered the culprit species, along with their fleas, that caused the spread of *Y. pestis*. No one knows how many people died in the Justinian Plague; however, it is commonly quoted that approximately 40 million people died during this pandemic (Campbell and Dennis 1998; Gage 1998).

The second pandemic, known commonly as the Black Death, occurred 750 years after the Justinian's pandemic. This outbreak lasted from approximately 1330 to 1352 and originated somewhere in inner Asia, then spread westward to the Middle East and Europe and eastward to China along international trade routes. The host species most commonly attributed with the origin of that pandemic has been Siberian marmots (*Marmota sibirica*), a presumably more ancient reservoir of *Y. pestis* than black rats (Drancourt and Raoult 2002; Kelly 2005). A frequently mentioned origin for the Black Death is the Mongolian Plateau. Another origin mentioned by historians is Lake Issyk Kul in Kirgizia near the northwest border of China (Kelly 2005). Unlike the Justinian Plague, which was due almost entirely to flea-borne bubonic plague, the Black Death also caused explosive outbreaks of primary pneumonic plague. This form of plague is virtually always fatal unless promptly treated with appropriate antibiotics (Gage 1998).

One of the sharpest differences between the Justinian pandemic and the Black Death was that the latter reached much deeper inland, while the earlier plague was largely confined to ports. The Black Death appeared to spread by "violent contagion" and was from its inception an extraordinarily virulent plague most likely due to a combination of factors (Kelly 2005). There is

some speculation that *Pulex irritans*, the so-called human flea was linked to the spread of plague during the Black Death pandemic, particularly in certain northern regions of Europe where black rats (*Rattus rattus*) and rat fleas (*Xenopsylla cheopis*) did not exist (Ell 1979; Gage and Kosoy 2005; Eisen et al. 2006). In Europe, the decades preceding the Black Death were exceptionally hazardous and unhealthy for both humans and domesticated animals (Kelly 2005). Almost everywhere in Europe there was war, overpopulation, economic stagnation and decline, filth, overcrowding, epidemic (nonplague) disease, widespread famine, and climatic and ecological instability. The Black Death affected every part of Eurasia. How many people perished is unknown: for Europe, the most widely accepted mortality figure is 33% of the population. Mortality varied across different parts of Europe; in some places plague claimed a third of the population; in others, half the population; and in a few regions, 60%. According to some reports from the time, plague not only affected humans, but also afflicted dogs, cats, birds, camels, and even lions with the tell-tale "boils" or buboes from the bubonic form of plague.

The most recent pandemic, which many authorities consider to be ongoing, is believed to have started in the 1880s in southwestern China, perhaps in Yunnan province. The so-called Third Pandemic, or Modern Pandemic, spread from southwestern China to ports of India, and then via shipping to Europe, Australia, Asia, and North and South America (Reisner 1996; Kupferschmidt 1997; Drancourt and Raoult 2002; Zietz 2004). Epidemics of plague have occurred in every continent except Antarctica. In some regions such as Australia and Hawaii, plague caused human epidemics and epizootics, but eventually disappeared, possibly due to the failure to become established in a suitable enzootic host (Perry and Fetherston 1997). The death toll exceeded 12 million in India and hundreds of thousands elsewhere (Poland et al. 1994). Although the Third Pandemic did not produce the tremendous mortality and social chaos associated with the previous two pandemics, it did result in a great expansion in the natural distribution of plague. Many regions of the world that were free of the disease before the last pandemic now have active foci, including North and South America, southern Africa, certain regions of Asia, and the islands of Java and Madagascar (Gregg 1985).

It was not until the beginnings of the Third Pandemic that the causative agent of plague was isolated and described. Alexandre Yersin, a Swiss scientist working in Hong Kong, described the plague pathogen in 1894. In 1898, fleas were incriminated as the vectors of plague by Simond and later proved to be the vectors by Liston in 1905. Studies conducted in India elucidated the roles played by commensal rats (*Rattus rattus* and *R. norvegicus*) and their fleas, particularly *Xenopsylla cheopis* (the oriental rat flea), in outbreaks of human bubonic plague (Gregg 1985; Gage 1998).

There are currently four recognized biotypes (biovars or strains) of *Y. pestis* based on different laboratory and phenotypic characteristics: Antiqua, Orientalis, and Mediaevalis. Recent molecular work has suggested that Antiqua caused the Justinian Pandemic, Mediaevalis caused the Black Death, and Orientalis initiated the Third Pandemic (Guiyoule et al. 1994; Achtman et al. 1999, 2004). Laboratory tests have yet to confirm a difference in virulence or pathology between these biotypes of *Y. pestis* (Perry and Fetherston 1997). More recently, a fourth biotype (Microtus) has been described from Asia. This biotype is unusual in being pathogenic for voles, but not for humans (Zhou et al. 2004).

United States

The history of plague in the United States (and in the western hemisphere in general) is a complicated one involving an introduced and invasive species, its eastward invasion into the continental U.S., and its cascading effects on wildlife and ecosystems. Plague most likely entered the continental U.S. through the port of San Francisco in the late 1890s and early 1900s (Kupferschmidt 1997; Adjemian et al. 2007; Eisen et al. 2007a). The first documented human plague case in the U.S. occurred in 1899 in a Japanese seaman (Link 1955). By the following year, human cases were occurring among U.S. residents, presumably from exposure to an urban rat-flea complex. From 1900 to 1907, 318 human plague cases reported resulted from "urban" plague exposure. In 1908, plague infections acquired from a wild rodent-flea complex (California ground squirrels) were reported from Contra Costa County, California (Link 1955). The disease moved steadily outwards from port cities into rodent populations inhabiting large expanses of western North America ("sylvatic" plague) (Adjemian et al. 2007). Plague has been documented in every state west of the Mississippi River except Arkansas, Iowa, Louisiana, Minnesota, and Missouri. By the mid-1940s, epizootics were documented in woodrat (*Neotoma* spp.), deer mice (*Peromyscus* spp.), ground squirrel (*Spermophilus* spp.), and prairie dog (*Cynomys* spp.) populations as far east as Oklahoma and Kansas (Cully et al. 2000).

The shift from urban-based plague to sylvatic rodent plague initially resulted in fewer reported human cases of plague (Mann 1984). For example, from 1927 to 1965, plague cases averaged only 2.3 per year. Beginning in the late 1960s, however, there was an increase in the number of human plague cases per year. This may be because of the further establishment of more sylvatic plague foci or from changing weather cycles leading to an overall increase in rodent populations. Larger populations of rodents would lead to more epizootics and therefore an increase in the number of human cases. From 1970 to 1983 there were 195 cases reported, averaging 13.9 cases per year. From 1983 to 2008 there were 261 cases reported, averaging 10.4 cases per year.

2.3 Distribution of Plague

World

Yersinia pestis can be found circulating in rodent populations on five continents (excepting Australia and Antarctica) where ground-dwelling rodents live in semi-arid climates (Antolin et al. 2010; Fig.1). Epidemics still occur frequently in developing countries where plague is endemic and people live in unsanitary, commensal rat-infested environments. Between 1987 and 2001, outbreaks involving hundreds of human plague cases occurred in at least 14 countries, usually as a result of exposures to infectious rat fleas (Gage and Kosoy, 2005). In the past 20 years, the World Health Organization (WHO) has reported 1,000 to 5,000 human cases of plague and 100 to 200 deaths per year in the world (Stenseth, 2008).

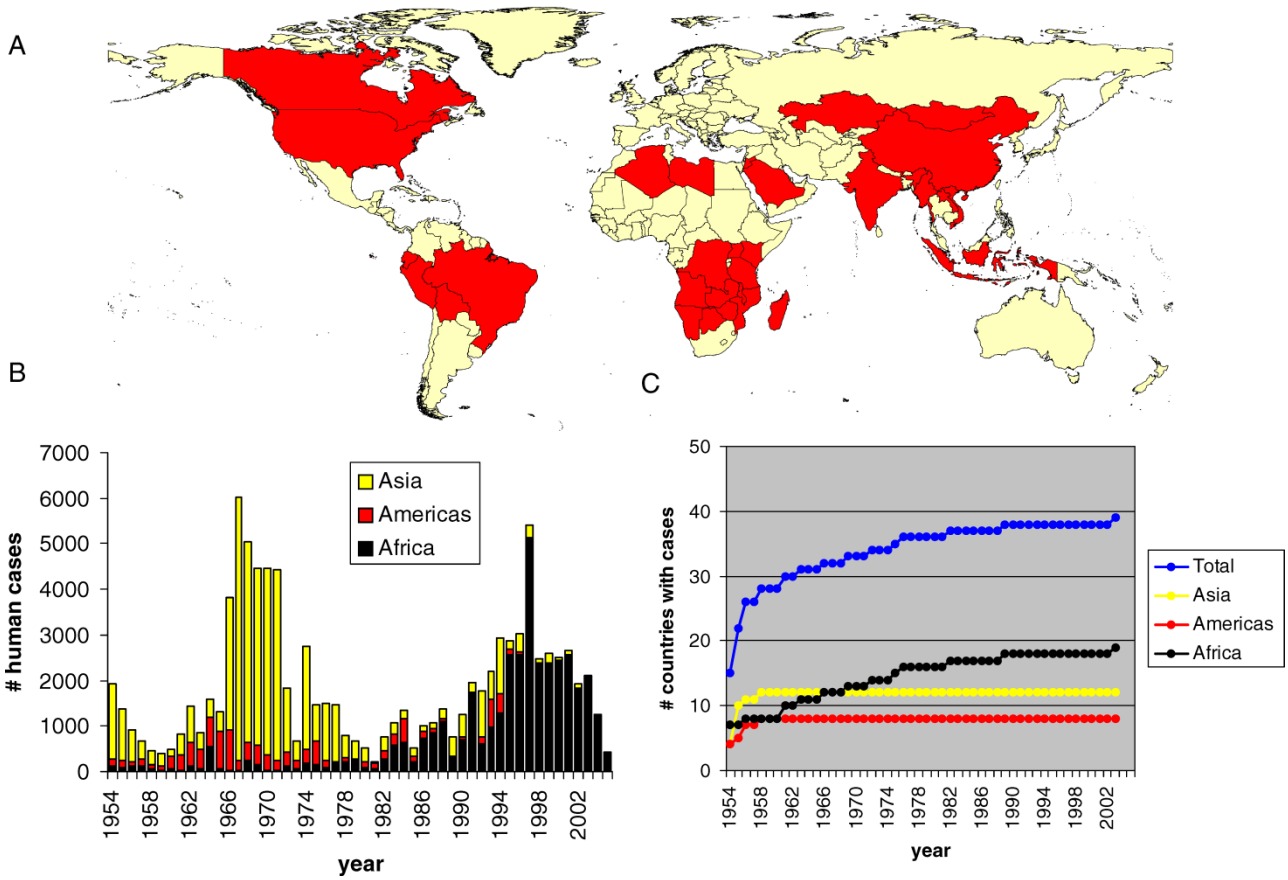

Figure 1. The global distribution of plague: (A) Map showing countries with known presence of plague in wild reservoir species (red). (B) Annual number of human plague cases over different continents, reported to the World Health Organization (WHO) in the period 1954-2005. (C) Cumulative number of countries that reported plague to WHO since 1954 (Stenseth et al. 2008).

United States

Most of the human cases in the U.S. are found in two regions: (1) northern New Mexico, northern Arizona, and southern Colorado; and (2) California, southern Oregon, and far western Nevada (Gage 1998; CDC 2009). The majority of human plague cases are reported from the southwest (Brown et al. 2010). Of the 456 cases reported to the Centers for Disease Control and Prevention (CDC) from 1950 through 2008, 82.5% of the cases occurred in either New Mexico (n=245), Arizona (n=63), Colorado (n=55), or Utah (n=13) (Brown et al. 2010). Human cases of plague are considered to be a by-product of the progression of plague in the rodent community (Keeling and Gilligan 2000).

Sylvatic plague occurs in every state west of the Mississippi River except Arkansas, Iowa, Louisiana, Minnesota, and Missouri (Fig. 2). Epizootics in sylvatic populations have been

documented in woodrat (*Neotoma* spp.), deer mice (*Peromyscus* spp.), ground squirrel (*Spermophilus* spp.), and prairie dog (*Cynomys* spp.) populations as far east as Oklahoma and Kansas (Cully et al. 2000). *Y. pestis* spread more than 2,250 km in a little more than 40 years, however its eastern spread has slowed. With the exception of parts of Texas, plague has rarely spread beyond the 103rd meridian, or the "plague line," in the western Great Plains (Antolin et al. 2002; Adjemian et al. 2007). Factors leading to the current distribution of enzootic plague and reasons why it has not spread eastward past the "plague line" are complicated and poorly understood.

Adjemian et al. (2007) retrospectively analyzed temporal and spatial dynamics of the expansion of plague over the U.S. landscape using historical human plague cases and wildlife plague reports. They found that there were between one and three introductions of plague along the Pacific coast after which it traveled from 45 to 87 km/year eastward. The coastal ranges of California were associated with slower spread, and the southern Rocky Mountains were associated with an increase in the rate of disease spread. The increase in velocity of spread in the southern Rocky Mountains was likely due to anthropogenic translocations of diseased wildlife. The southern Rockies include the ranges of black-tailed (*Cynomys ludovicianus*), Gunnison's (*C. gunnisoni*), and white-tailed (*C. leucurus*) prairie dogs. Prairie dogs are associated with a decreased natal dispersal rate relative to other rodents implicated in the spread and maintenance of plague throughout its range, such as ground squirrels (Garrett and Franklin 1988). This would imply a decreased velocity of the disease spread; however, in the early to mid-1940s, prairie dogs were considered a pest species to ranchers and deliberate translocations of diseased individuals to healthy populations likely resulted in the higher velocity of spread observed (Ecke and Johnson 1952; Adjemian et al 2007).

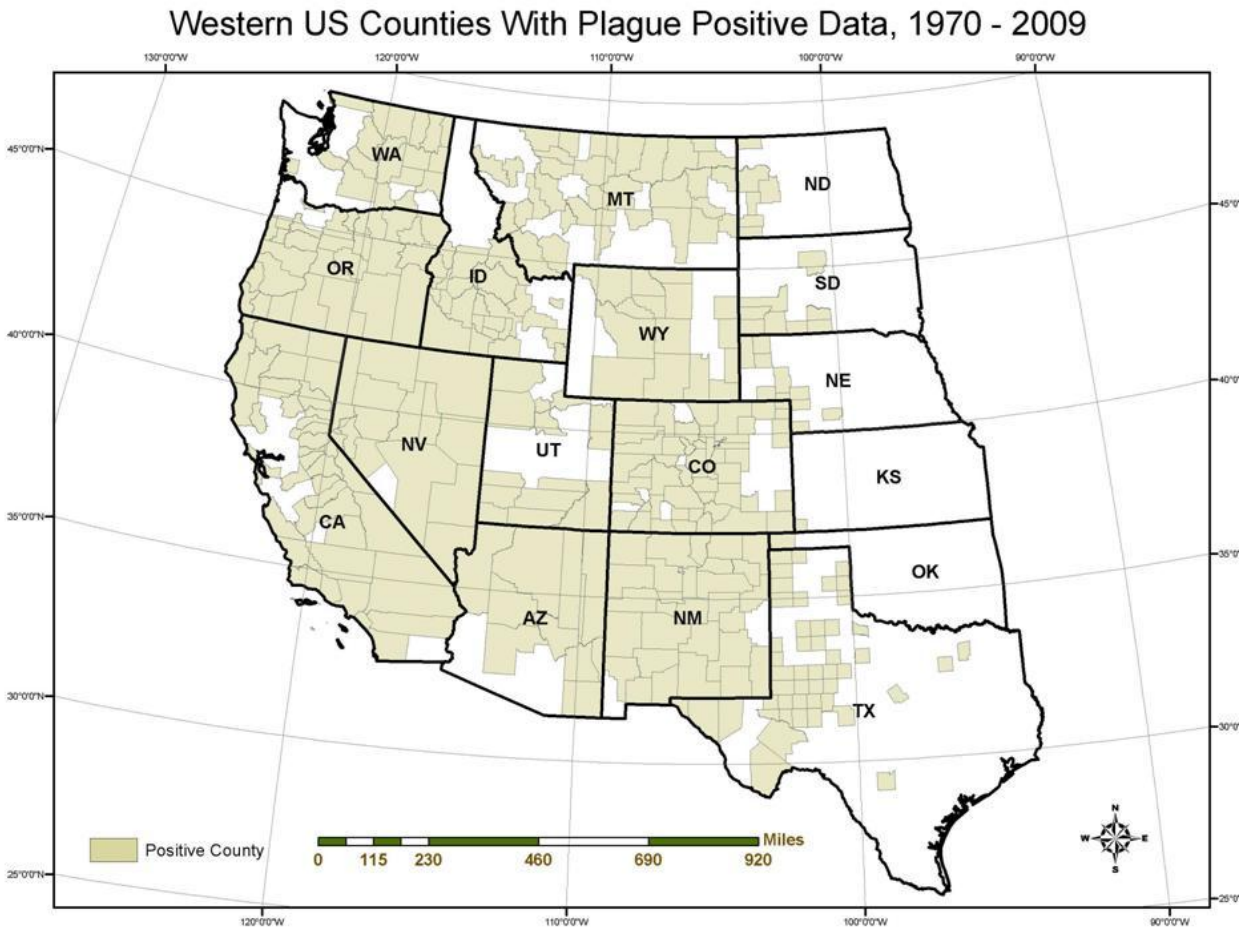

Figure 2. Geographic distribution of plague by county for the United States (Centers for Disease Control and Prevention, 2010).

A lack of suitable hosts and vectors, as well as habitats not typically associated with plague may be the reasons that plague did not become established in other parts of the continental U.S. where it was previously introduced (Adjemian et al. 2007). From 1914 to 1915, there was a large human plague outbreak in Louisiana resulting in 31 cases; and again in 1919-1920, Louisiana, parts of Florida, and Texas reported 61 cases. Also, incoming ships in New York in 1899 reported human plague cases (Caten and Kartman 1968; Kartman 1970). However, historical records indicate that plague did not become established in these or surrounding areas (Adjemian et al. 2007).

2.4 NPS history and distribution

At least 30 NPS units in the western U.S. have reported evidence of plague in wildlife, and many continue to report cycles of epizootic (= outbreaks within wildlife populations) activity (Figure 3). Plague has not necessarily disappeared from NPS units where it was found historically and not recently reported—it may still be present at enzootic levels. Additional parks are likely to

find evidence of plague, if the organism continues its current trend of expansion. For those reasons, NPS personnel within the current distribution of plague activity and those near the leading edge of the distribution must be conscious of the potential for plague to occur in wildlife and humans.

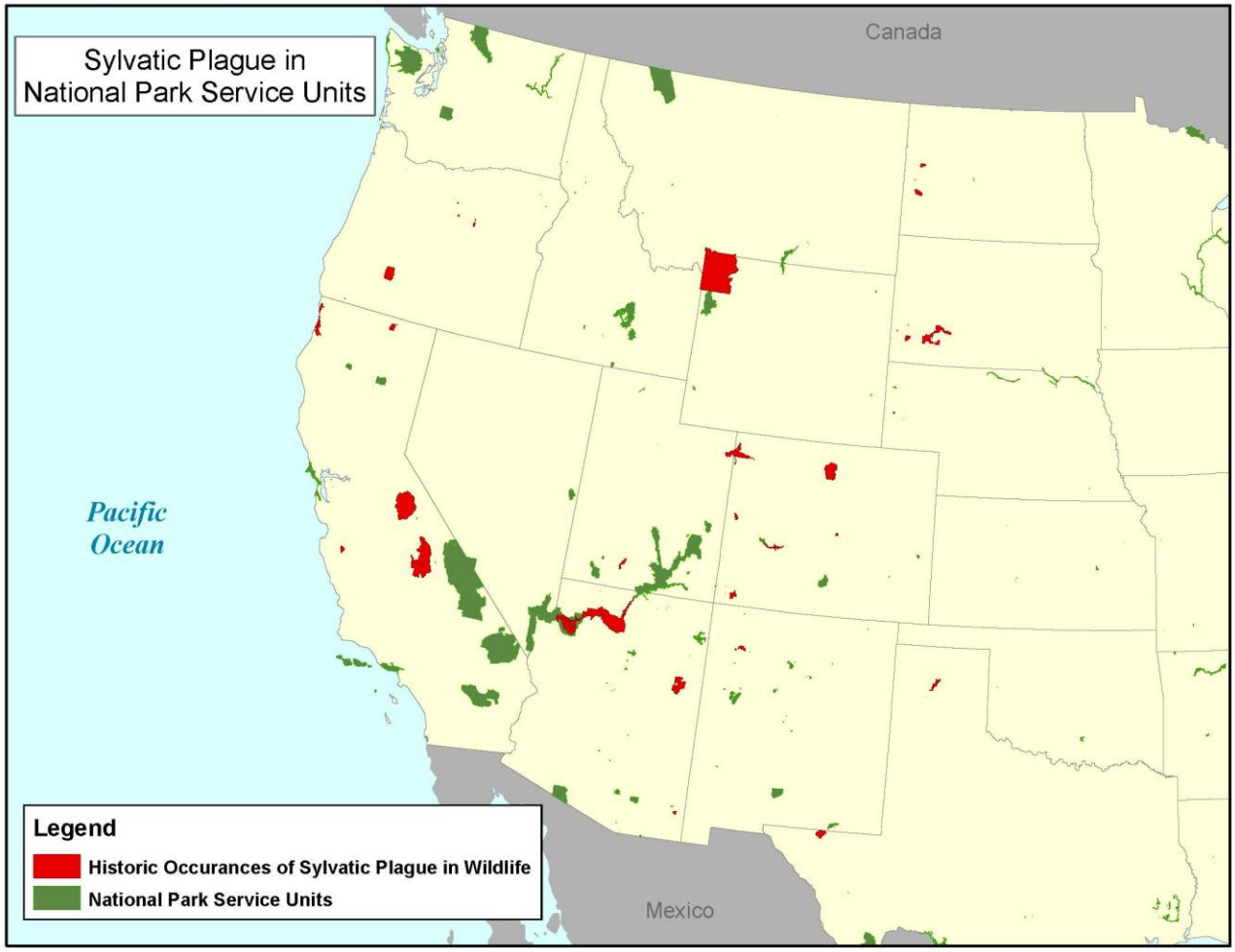

Figure 3. Geographic distribution of plague by National Park Service (NPS) units for the United States.

Management of plague may be necessary in NPS units to protect native wildlife species, ecosystem function, and human health. Because of the risks to human health, *Safe Work Practices (RM-50)* should always be followed when working in plague-endemic areas. This information can be found at the following link:
http://www1.nrintra.nps.gov/BRMD/Wildlife_Health_Management/Wildlife_Management/Handling_Wildlife.cfm

Case Studies of Plague at Parks

National parks have had extensive experience with plague. Plague has caused die-offs of prairie dogs and other ground squirrel species, and has affected populations of closely associated species, such as black-footed ferrets. Historically, NPS units have managed plague in a number of different ways. Many parks have used a combination of area closures and burrow dusting. Dusting has occurred proactively or as a response to an ongoing plague event. Three recent events regarding plague at parks will be highlighted to show how plague has affected parks and to highlight various responses. The plague event at Florissant Fossil Beds National Monument highlights an outbreak specific to wildlife; the plague event at South Dakota addresses plague prevention when it is found near park boundaries; and the plague event at Grand Canyon National Park is an example of direct human exposure to plague.

Florissant Fossil Beds National Monument

In July 2008, a retired NPS employee found a dead prairie dog on his property adjacent to the northeastern boundary of Florissant Fossil Beds National Monument (FLFO) in Colorado. Local public health officials were notified and the carcass was submitted to CDC in Fort Collins, CO for testing. A presumptive diagnosis of plague was quickly made by CDC and the finding was confirmed by additional testing shortly thereafter. Once the park was notified that the prairie dog was presumptive plague positive, FLFO closed public access to parking areas and trails nearest the location from which the prairie dog was collected, and posted health advisories at other high use areas. An NPS investigative team comprised of personnel from the Wildlife Health Program, Office of Public Health, and the Intermountain Regional Office was dispatched to FLFO. As part of the investigation, fleas were collected from burrows at three sites within the park: the inactive prairie dog colony in which the dead prairie dog was found, a colony near where a public event was scheduled to be held, and an active prairie dog colony 2 miles south of the infected colony.

Based on the close proximity of plague to the park, and based upon an upcoming annual public park event, the team recommended that the park consider the possibility of dusting prairie dog burrows with insecticide to kill fleas. The park hired a private contractor to apply Deltamethrin to burrows at the three sites. After dusting, unsuccessful attempts were made to collect fleas from the three sites. Because there was no evidence of an abundance of host-seeking fleas at the colonies, and because prairie dog and other ground squirrel activity was normal around the area where the public event would be held, the investigative team recommended to the superintendant that the public event could be held as planned. Health warnings to inform the public about plague in the area were placed around the area where the public event was held, as well as along trails and other high-use areas. After several weeks post-dusting, when no additional plague-killed rodents were observed, and after consultation with Public Health and Wildlife Health personnel, the warning signs were removed.

Unfortunately, the prairie dog colonies along the northern fence line of FLFO and near the Hornbek Homestead where the public event was held died out later that same year. Even though the contractor dusted all prairie dog burrows at north and south colonies, the smaller colony to the south disappeared the following year (2009).

Badlands and Wind Cave National Parks

Plague arrived in Conata Basin near Badlands National Park (BADL) in South Dakota in May of 2008. In summer 2009, plague was confirmed at a prairie dog colony within a wilderness area of BADL. Since its detection in Conata Basin/BADL, plague has reduced the acreage of active prairie dog colonies by about 50%; acreage fell from ~ 31,400 acres in 2007, to ~15,900 acres as of November 2009. In addition to the substantial decrease in a native rodent species, loss of prairie dogs threatens reintroduced black-footed ferrets (BFF) and other species that rely on prairie dogs as a keystone species. Until plague appeared, the Conata Basin/BADL BFF reintroduction area had been the most successful, self-sustaining recovery site for that species. Plague has directly (through BFF mortality) or indirectly (through loss of prey and burrows) led to an estimated 33% reduction in the BFF population. In response to the plague epizootic, an interagency collaboration to protect the ferrets and prairie dogs led to the application of insecticide powder (Deltamethrin) into prairie dog burrows. In 2008, 2009, and 2010 the interagency effort dusted approximately 11,000 acres each year, with the expectation that protecting the area with the highest concentration of ferrets would enhance survival of the population. In addition to the dusting effort, BFF were captured and vaccinated against plague. A prairie dog colony in BADL to the north of Conata Basin (the Robert's colony) was dusted as a preventive measure to protect prairie dog and human health. Plague has been detected within approximately 20 miles of Wind Cave National Park (WICA), and in 2010, *Y. pestis* DNA was detected in a small number of fleas collected from within the park. Biologists are closely monitoring the prairie dog populations within and around the park, and so far no evidence of epizootic plague has been detected at WICA. Wind Cave reintroduced BFF to one of its prairie dog colonies in 2007 and has reported successful reproduction in the wild. Because of its proximity to plague-infected sites, WICA elected to dust prairie dog burrows in 2008, 2009, and 2010, primarily to protect the prairie dogs and BFFs. While dusting does not convey 100% protection to colonies against plague, it has slowed the progression of plague in Conata Basin and has allowed prairie dogs and BFF to persist in areas that may have otherwise been decimated.

Grand Canyon National Park

On the morning of November 2, 2007, a 37-year old wildlife biologist was found deceased in his residence on the south rim of Grand Canyon National Park (GRCA) (Wong et al. 2009). Interviews with his colleagues revealed that the employee had developed a respiratory illness earlier in the week and had been examined at the GRCA medical clinic. Given the employee's occupation and clinical symptoms, zoonotic diseases, particularly pneumonic plague and hantavirus cardiopulmonary syndrome, were suspected as the possible cause of death. A team of investigators, including NPS Office of Public Health medical epidemiologist and public health consultant, a Wildlife Health Program veterinarian, GRCA management and law enforcement, and other collaborators including the CDC and the Arizona state and local health departments conducted an investigation into this event.

Upon further investigation, exposure history revealed multiple work and recreational contact with wildlife. Primary work duties included trapping and collaring mountain lions, and trapping nuisance rodents in historical and residential buildings. Colleagues reported that the employee had handled, skinned, and conducted a necropsy of a mountain lion carcass one week prior to his death. Aside from his work, the employee spent free time as a hunter and outdoorsman, and had recently returned from a deer hunting trip in California. Because pneumonic plague was suspected as a possible cause of death, a prophylactic course of antibiotics was offered to

individuals who were in close contact with the employee after he had developed symptoms. Multiple tissue samples from the employee's body was submitted for testing by the CDC and found to test positive for *Y. pestis.*

Once plague was identified as the probable cause of the employee's death, increased efforts were made to identify the source of infection. The mountain lion carcass that the employee had handled was suspected as a potential source of plague for multiple reasons: (1) mountain lions and other wild cats are highly susceptible to plague; (2) the carcass had been handled within the known incubation period; and (3) the retrieval of the carcass and subsequent necropsy would have allowed prolonged and intimate exposure to infected tissues. Parts of the mountain lion carcass were submitted for testing and samples from the lion's liver and lymph nodes tested positive for *Y. pestis*, suggesting that plague was the probable cause of the animal's death. Genetic signatures of the *Y. pestis* isolated from the employee and from the mountain lion were the same, providing strong evidence that the mountain lion carcass was the source of the employee's plague infection. Sampling of tissues for other possible diseases by CDC and other collaborators did not find evidence of any other source of the infection.

These plague case studies underscore the presence of plague at NPS and the need for all NPS employees in or near plague-endemic areas to be aware of the potential for exposure to the disease. In March 2010, representatives from a number of federal agencies, including NPS, met to begin formalizing a national plague management team that would investigate additional plague control and prevention methods, including an oral plague vaccine for prairie dogs.

3. Disease Ecology

3.1 Bacterial Characteristics

Yersinia pestis is an aerobic, nonmotile, gram-negative bacillus belonging to the family Enterobacteriaceae (Josko 2004). Gram-negative bacteria lose the crystal violet stain (and take the color of the red counterstain) in Gram's Method of staining (Shiel and Stoppler 2008). The genus *Yersinia* consists of 11 species, of which 3 are human pathogens (*Y. pestis*, *Y. pseudotuberulosis*, and *Y. enterocolitica*) (Perry and Fetherston 1997). *Y. pseudotuberculosis* and *Y. enterocolitica* are transmitted by fecal-oral routes and cause intestinal symptoms of moderate intensity rarely causing death (Gage 1998; Stenseth 2008). *Y. pestis* lives only within a mammal or a flea and is generally not found living free in the environment, whereas *Y. pseudotuberculosis* and *Y. enterocolitica* are viable for long periods of time in soil and water (Hinnebusch 1997). The genome of *Y. pestis* has been decoded, showing that the organism evolved from *Y. pseudotuberculosis* 1,500-20,000 years ago (Achtman et al. 1999). The transmission of *Y. pestis* by fleabite is a recent evolutionary adaptation that distinguishes it from *Y. pseudotuberculosis* (Hinnebusch 2005). The close genetic relationship between *Y. pestis* and *Y. pseudotuberculosis* indicates that just a few discrete changes were sufficient to give rise to flea-borne transmission and the *Y. pestis*-*Y. pseudotuberculosis* species complex provides an interesting case study in the evolution of arthropod-borne transmission (Hinnebusch 2005).

Since its first isolation by Yersin, *Y. pestis* has been extensively studied. The main characteristics of the species are its low number of positive biochemical reactions and its great homogeneity. There is only one serotype, one phage type, and four biotypes. The three major

biotypes, or biovars, are based on the conversion of nitrate to nitrite and the fermentation of glycerol: Antiqua is positive for both characteristics; Orientalis forms nitrite but does not ferment glycerol; and Mediaevalis ferments glycerol but does not form nitrite from nitrate. These three biotypes exhibit no difference in their virulence or pathology in animals or humans (Perry and Fetherston 1997), but each biotype has been implicated in the 3 different pandemics previously mentioned (Achtman et al. 1999, 2004; Guiyoule et al. 1994). The homogeneity of *Y. pestis* continually surprises scientists, since the bacterium has been able to adapt to various types of mammalian hosts and species of flea vectors across widely different climates and geographical locations (Guiyoule et al. 1994). A fourth biovar found in Asia has been proposed recently that lacks pathogenicity for humans, but remains pathogenic for voles.

3.2 Life Cycle and Transmission of Yersinia pestis

Plague is a zoonosis, or an infectious disease that can be transmitted between humans and other animals (see Fig. 4 for details on life cycle and transmission for the western United States). The natural reservoirs for the infectious agent, *Y. pestis*, are rodents and their fleas. Humans play no role in the long-term survival of *Y. pestis* (Butler 1983; Perry and Fetherston 1997). Transmission between rodents is usually achieved by their associated fleas. Infection can also occur by direct contact, ingestion, and inhalation, but these routes do not normally play a role in the maintenance of *Y. pestis* in rodent populations. Fleas acquire *Y. pestis* from an infected blood meal and the maintenance of plague in nature is entirely dependent on cyclic transmission between fleas and mammals.

Much of what we know about the plague life cycle is from the classic vector for plague, the oriental rat flea (*Xenopsylla cheopis*), considered the most efficient vector for plague for almost a century (Eisen et al. 2007d). *X. cheopis* will typically ingest from 0.03 to 0.5 ul of blood from its host (Perry and Fetherston 1998). *Y. pestis* is cleared from some fleas, but multiplies in the midgut (stomach) of others. The digestive tract of the flea is where the storage, digestion, and absorption of blood meals occur. The digestive tract is made up of the foregut, which includes the pharynx, esophagus, and proventriculus; the midgut, or stomach of the flea; and the hindgut. At the junction of the foregut and midgut lies a valve-like structure, the proventriculus (Vadyvaloo et al. 2007). The proventriculus is central to the mechanism of classic flea-borne transmission (Hinnebusch 2005). Two days after an infected blood meal, the stomach shows clusters of brown specks containing *Y. pestis*, which develop into dark brown masses. Between days 3 and 9 after ingesting the infected blood meal, the bacterial masses may completely block the proventriculus, extend into the esophagus, and prevent ingested blood from reaching the stomach. The flea will then repeatedly attempt to feed and blood sucked from the host distends the esophagus, mixes with the bacilli, and is regurgitated into the host when the feeding attempt is terminated (Perry and Fetherston 1997).

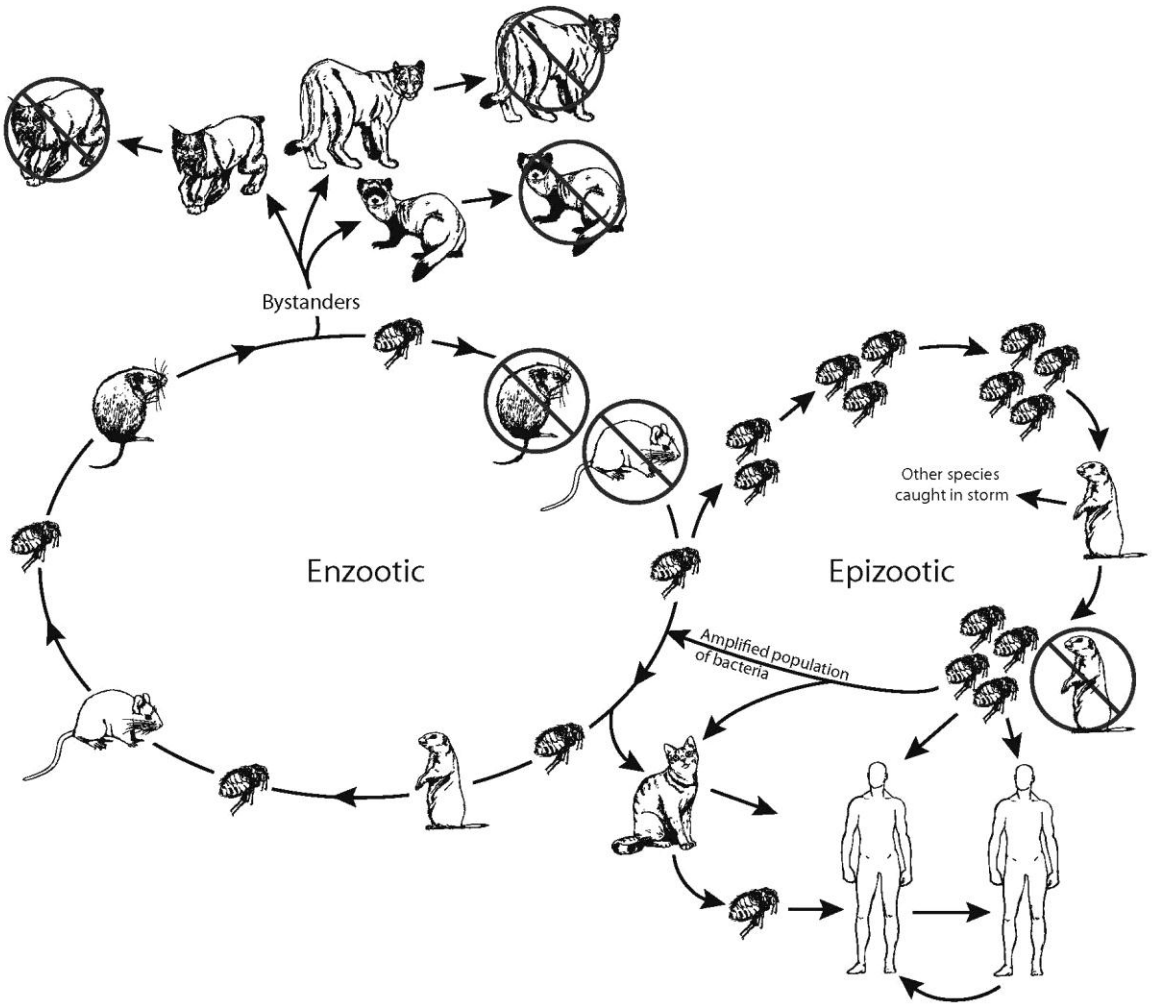

Figure 4. A schematic representation of hypothetical enzootic and epizootic plague cycles with comments on human and wildlife plague risks. Key characteristics are (1) increased flea densities that promote transition from the enzootic cycle into an epizootic (Lorange et al. 2005), (2) amplified populations of *Y. pestis* during an epizootic that feed back into the enzootic cycle, (3) possible involvement of key rodent species as both maintenance (i.e., enzootic) hosts and amplifying (epizootic) hosts (Biggins et al. 2010), (4) initiation and persistence of epizootics depending on threshold host densities (Davis et al. 2004), (5) highest human health risks occurring during epizootic cycles but human exposures occasionally occur during the enzootic phase, and (6) high risk to "bystander" wildlife species that are not involved in maintaining or amplifying plague, with substantial mortality even during the enzootic phase (Matchett et al. 2010). Wildlife conservation issues (indicated by the symbol Ø superimposed over an animal) arise during all phases.

Transmission by "blocked" fleas has traditionally been regarded as the most efficient means of transferring plague from the vector to its host (Prentice and Rahalison 2007) and dissemination of *Y. pestis* by blocked fleas has been the dominant paradigm for flea-borne transmission (Eisen et

al. 2007d). However, epidemiological models have suggested that because blocked fleas die shortly after becoming infectious, they are not infectious long enough to drive epizootics (outbreaks) unless the number of fleas per host is very high (Lorange et al. 2005; Webb et al. 2006). Additionally, it has been shown that some fleas can be long-lived successful vectors without blockage of the proventriculus. This includes the oriental rat flea (*X. cheopis*), which has been implicated as the primary vector of *Y. pestis* during plague pandemics, as well as more localized epidemics and epizootics in many tropical and semitropical regions (Eisen et al. 2006, 2007b, 2007d, 2009). A scenario of "early-phase" transmission by unblocked fleas has caused a shift in how scientists conceptualize *Y. pestis* transmission during rapidly spreading epizootics and epidemics, although the actual mechanism by which early phase transmission occurs remains unknown (Eisen et al. 2006). Vetter et al. (2010) demonstrated that early phase transmission did not depend on biofilm production of *Y. pestis* and it seems likely that the phenomenon results from regurgitation or a combination of regurgitation and mechanical transmission (contamination of flea mouthparts with viable *Y. pestis*).

Once transmitted by a flea bite (or less frequently by inhalation or ingestion), *Y. pestis* invades and multiplies within the host and is transported by phagocytes, or cells that can engulf bacteria, during the initial phases of the infection (Dennis and Gage 2004). It spreads from the site of the flea bite to the regional lymph nodes and grows to high numbers, causing the formation of a bubo, or swollen lymph node. Hosts that survive plague appear to arrest the progression of the disease at the lymph node level (bubo). The infection can then spread into the bloodstream, where bacilli are preferentially removed in the spleen and liver. Growth of the organisms continues in the blood, liver, and spleen and spreads to other organs and the progression is similar to that of other bacterial infections. Lungs and other internal organs may also become colonized by *Y. pestis*. Development of bacteremia, or the presence of live bacteria in the bloodstream, of sufficient degree and duration is essential for the effective transmission of *Y. pestis* to fleas in nature (Perry and Fetherston 1997). Infection of the flea via the blood of a bacteremic or septicemic rodent completes the cycle.

Two basic disease cycles have been associated with *Y. pestis*: "urban" and "sylvatic." Urban plague cycles primarily involve transmission of *Y. pestis* among human-associated rodents (e.g., rats), their fleas, and people. The commensal black or roof rat, *Rattus rattus*, the sewer rat, *R. norvegicus*, and their fleas (especially the oriental rat flea, *X. cheopis* described above) have been the principal sources of epidemics of plague and its pandemic spread (Dennis and Gage 2004). Domestic animals living in close association with humans, including cats and dogs, may play important roles in transmission to people in an urban cycle (Rust et al. 1971). Although canines are thought to be largely resistant to plague, some do develop illness (Orloski and Eidsen 1995). Cats that are allowed to roam outdoors may also introduce fleas into the human environment. Cats are also very sensitive to *Y. pestis* infection, and may transmit bacteria directly to people via respiratory secretions or, less likely, via direct contact with blood of bacteremic cats (Gage et al. 2000).

Sylvatic cycles of plague can be much more complex than urban cycles (Fig. 4). Generally, *Y. pestis* circulates within populations of wild rodents and their fleas in enzootic (quiescent or maintenance) cycles and periodic outbreaks or epizootic cycles. For reasons that are often unknown, *Y. pestis* will enter a population of host species and "amplify" or rapidly spread the

bacterium over wide areas causing an epizootic. In those instances, large numbers of susceptible animals may be killed, and humans and other atypical species (e.g., wild felids, mustelids) may be placed at greater risk. The next section will discuss the ecology of plague enzootic and epizootic cycles focusing on various examples of sylvatic plague in North America.

3.3 Ecology of Plague

How plague circulates in natural systems remains one of the great enigmas of the disease. However, in North America there appears to be two primary phases of plague ecology: a rapid expansion phase for growth and dispersal followed by a slower persistence phase (Girard et al. 2004). Most generally, plague persists in these so-called enzootic and epizootic cycles (Gage and Kosoy 2005). Enzootic (maintenance) cycles are often thought to involve resistant host populations that have many members that are likely to survive plague, but they also have some individuals that suffer more severe illness and experience bacterial loads sufficient to infect at least a proportion of the feeding fleas. Proposed enzootic hosts in North America include deer mice (*Peromyscus maniculatus*), California meadow voles (*Microtus californicus*), and northern grasshopper mice (*Onychomys leucogaster*) (Poland and Barnes 1979; Gage et al. 1995). An enzootic cycle is sometimes likened to the quiescent period of the disease dynamic. An epizootic (amplifying) cycle is one where susceptible hosts perish in high numbers and transmission rates are significantly higher than normal (i.e., during enzootic periods). Presumably epizootics occur when *Y. pestis* is introduced from enzootic cycles to highly susceptible epizootic host populations. Epizootic hosts include ground squirrels (*Spermophilus* spp.), prairie dogs (*Cynomys* spp.), woodrats (*Neotoma* spp.), and chipmunks (*Tamias* spp.) (Gage et al. 995). Epizootics of plague in prairie dogs have been likened to wildfires burning through prairie dog colonies and completely wiping out towns. To further the wildlife analogy, it appears that most of these epizootics die out only when "fuel for the fire" (susceptible hosts) becomes too limited to support continued transmission.

The mechanism(s) by which *Y. pestis* is maintained enzootically in nature is largely unknown (Eisen et al. 2008a). The occurrence of epizootic hosts is substantiated by obvious die-offs; however, little evidence exists to indicate that supposed enzootic hosts are truly essential for the maintenance of plague (Gage and Kosoy 2005). For example, the role of deer mice (*Peromyscus maniculatus*) as enzootic reservoirs for plague is controversial (Eisen et al. 2008a). Deer mice were implicated as enzootic hosts based on their high abundance in plague-endemic areas, their diverse flea fauna, presence of *Y. pestis* in fleas recovered from deer mice, and their observed resistance to infection (Barnes 1982; Gage et al. 1995; Gage and Kosoy 2005). Although the deer mouse flea, *Aetheca wagneri*, is capable of transmitting *Y. pestis* as early as three days post infection, transmission was found to be quite low (Eisen et al. 2008a). This study concluded that unnaturally high flea loads would be required to maintain enzootic transmission in an *A. wagneri*-deer mouse cycle of plague. Alternatively, it could be that plague is maintained between epizootics by low-level transmission among a number of potential host species and their fleas, causing epizootics only when environmental conditions (i.e., climate) are favorable and host populations are high. Patchy distributions of these multiple hosts and fleas within a variety of habitats could ensure that populations are never completely wiped out and that low levels of transmission can occur as plague slowly passes from patch to patch allowing host populations to recover from previous epizootics (Gage and Kosoy 2005).

Existing evidence suggests that epizootics occur most frequently when rodent and flea populations are high (Gage 1998; Davis et al. 2004). Many factors can affect rodent and flea abundances including climate. Seasonal shifts in precipitation, humidity, and temperature have been reported to affect the course of plague epidemics (India Plague Commission 1908; Cavanaugh 1971; Cavanaugh and Marshall 1972). Other studies have modeled the relationships between climate and the frequency of human plague cases in the southwestern U.S. (Parmenter et al. 1999; Enscore et al. 2002). Parmenter et al. (1999) found that human plague cases in New Mexico occurred more frequently following winter-spring periods (October to May) with above-average precipitation. Enscore et al. (2002) also found that increased precipitation during specific periods resulted in increased numbers of cases in both Arizona and northwestern New Mexico. Additionally, they found that when summer temperatures were relatively cool, numbers of human plague cases were relatively high. Similar results were found for animal-based surveillance data in New Mexico (Brown et al. 2010). The "trophic cascade hypothesis" was proposed to explain the observed patterns. This hypothesis suggests that suitable temperature and precipitation lead to an increased primary vegetative production; the increased food supply increases the carrying capacity of rodent populations, which also increase the number of hosts for fleas and this in turn increases the likelihood of epizootic spread (Parmenter et al. 1999; Enscore et al. 2002; Collinge et al. 2005; Eisen et al. 2006).

The rate of plague transmission in black-tailed prairie dog colonies in Montana was clearly explained by climate (Snall et al. 2008). Precipitation was found to affect the force of infection that regulates the spatial transmission, and temperature affects the rate of background infection. Hot days most likely reduced transmission throughout the landscape due to the negative effects of heat on flea survival and on flea-mediated transmission of *Y. pestis* (Cavanaugh and Marshall 1972, Enscore et al. 2002). Snall et al. (2009) predicted future plague levels in black-tailed prairie dogs in North America under different scenarios of climate change. Their simulations indicated there will potentially be less plague in the future, and even decreased plague with increased greenhouse gas emissions. Nakazawa et al. (2007) used ecological modeling to analyze the current and potential future distribution of human plague in the U.S. and concluded that, based on available models of climate change, the locations of future human plague cases might shift slightly northward.

Landscape features have been shown to influence the ecology of plague (Collinge et al. 2005a, 2005b; Eisen et al. 2007a; Collinge et al. 2008; Brown et al. 2010). Elevation, in particular has been shown to influence the distribution and occurrence of plague. In New Mexico, more aggregated distributions of human and pet plague cases and earlier onset dates were evident at lower elevation sites ($\leq$2,115 m) than at higher sites (>2,115m) (Brown et al. 2010). Lower elevation sites were characterized by drier and hotter conditions with transient presence of limited vegetation. Changes in landscape structure may also influence the transmission and spread of plague. In two sites in Colorado and Montana, Collinge et al. (2005a, 2005b) found that plague occurrence in black-tailed prairie dog colonies was negatively associated with the cover of lakes or reservoirs, streams and roads in the landscape surrounding colonies. These landscape features could act as barriers to the movement of prairie dogs, fleas, or other grassland rodents that serve as reservoir hosts for plague.

The effects of plague on rodent communities may cause major shifts in community composition and ecosystem function. For instance, it is likely that black-tailed prairie dogs

influence the abundance and distribution of several rodent species that act as alternative hosts for plague (Gage et al. 1995; Cully et al. 1997, 2000). Because prairie dogs suffer high mortality when infected with plague, this could amplify or dilute plague transmission in native rodent communities (Collinge et al. 2008). High mortality in prairie dogs could concentrate scavengers and amplify plague transmission or mortality could reduce host or host-vector contact rates, thereby diluting transmission. Prairie dogs also appear to affect the abundance and distribution of fleas that serve as the vectors for plague transmission probably due to the suitability of prairie dog burrows for fleas (Gage and Kosoy 2005). Flea abundance and flea species richness were significantly higher on mice captured within prairie dog colonies than at adjacent off-colony sites (Brinkerhoff et al. 2008, Collinge et al. 2008).

Prairie dogs play an important, possibly keystone, role in grassland ecosystems (Miller et al. 1994; Kotliar et al. 1999). They affect vegetation structure, productivity, nutrient cycling, and ecosystem processes. They are also important prey items for many terrestrial predators and raptors. Prairie dog communities have been shown to be important to nesting ferruginous hawks (Cook et al. 2003), burrowing owls (Desmond et al. 2000), red-tailed hawks, rough-legged hawks, bald eagles (Seery and Matiatos 2000), mountain plovers (Dinsmore et al. 2005; Augustine et al. 2008b), badgers (Goodrich and Buskirk 1998), arthropod diversity (Bangert and Slobodchikoff 2006), lesser earless lizards (Davis and Theimer 2003), black-footed ferrets (Biggins et al. 2006), and herbivorous rodents (Hartley et al. 2009). Therefore, epizootics of plague in prairie dog communities can indirectly affect all colony-associated species and grassland food webs. Even enzootic plague in prairie dog communities (presence of disease-causing *Y. pestis* without any noticeable prairie dog die-off), was found to reduce survival in the highly endangered black-footed ferret in Montana (Matchett et al. 2010).

3.4 Flea Vectors

More than 250 species of fleas, associated with about 200 wild rodent species, have been found to be infected with *Yersinia pestis* in nature, or to be susceptible to experimental infection (Pollitzer 1954, 1960; Gage and Kosoy 2005). At least 80 of these 250 flea species are thought to be involved in maintaining cycles of plague (Pollitzer 1954; Hinnebush 1997). About a dozen of these species have been implicated in transmission of urban plague around the world. These urban flea species are primarily ectoparasites of commensal and peridomestic rodents, but readily feed on humans (Gratz 1999a). Important flea species in urban plague cycles worldwide include *Xenopsylla cheopis* (oriental rat flea), *X. astia* (gerbils and rats), *X. brasiliensis* (rat flea), *Nosopsyllus fasciatus* (Northern rat flea), *Monopsyllus anisus* (common rat flea), *Pulex irritans* (human flea), and *Ctenocephalides felis* (cat flea). Many more species of fleas have been implicated in sylvatic plague worldwide (Gratz 1999a).

Different species of fleas naturally infected with plague vary substantially in their ability to transmit *Y. pestis* (Gage and Kosoy 2005; Hinnebusch 2005). Some species of fleas cannot or only rarely transmit *Y. pestis* (i.e., the common cat flea, *C. felis*). A model to estimate vector efficiency was developed in the 1940-50s and included the following variables: (1) infection potential (the percentage of fleas becoming infected after feeding on a septicemic animal); (2) vector potential (the percentage of infected fleas which become infective or blocked); (3) the transmission potential (the average number of successful transmissions per flea); (4) the life span of the infective (blocked) fleas; and, (5) the field prevalence index (the average number of fleas per species per

rodent or rodent nest) (Wheeler and Douglas 1945; Burroughs 1947; Kartman 1957). Using these measures, vector efficiency was calculated for selected species of fleas. The oriental rat flea (*Xenopsylla cheopis*) has most frequently been identified as the most efficient vector of plague worldwide (Hinnebusch 2005).

Determining flea vector efficiency is very important in understanding how plague is maintained in natural ecosystems. It is also useful information for identifying key flea vectors to be targeted for prevention and control activities during plague outbreaks (Eisen et al. 2008b). In North America, 28 species of fleas have been experimentally confirmed as vectors of *Y. pestis* (Eisen et al. 2009). *Oropsylla montana* is considered the primary vector of *Y. pestis* to humans in North America and naturally infests highly plague-susceptible California ground squirrels (*Spermophilus beechyii*) and rock squirrels (*S. variegatus*), both of which commonly succumb to plague during epizootics (Eisen et al. 2006). Although this flea only rarely becomes blocked, it does efficiently support early-phase transmission (i.e., transmission within 1-4 days post-infection of the flea vector). Along with *O. montana* (on ground squirrels), *O. hirsuta* and *O. tuberculata* are often associated with prairie dogs and other ground squirrels in North America. The primary fleas of black-tailed prairie dogs are *Oropsylla tuberculata cynomuris* and *Oropsylla hirsuta* (Wilder et al. 2008a). Although both of these species of fleas are competent vectors of *Y. pestis*, they rarely form proventricular blockages. However, Wilder et al. (2008b) investigated the efficiency of *O. hirsuta* to transmit *Y. pestis* to hosts and showed that it is a considerably more efficient vector during early-phase transmission periods (1-4 days post infection). They suggested that early-phase transmission can support plague at an enzootic level in a population where flea loads are within the average observed for black-tailed prairie dogs in nature. Epizootic spread of plague would be further enabled by shared burrows and sociality of prairie dogs leading to an accumulation of fleas when host populations are reduced as a result of the disease (Wilder et al. 2008b).

Wimsatt and Biggins (2009) reviewed the persistence of plague in western North America emphasizing the role of fleas. In the classic description of sylvatic plague, rodents are believed to act as primary hosts and serve as persistent disease carriers. Secondary hosts can be ancillary rodent species dependent on primary hosts. These secondary hosts and their fleas cannot maintain *Y. pestis* in the absence of the primary hosts but may assist in disseminating the disease (Gage and Kosoy 2005). A third group consists of incidental bystanders (e.g., humans, carnivores). They reviewed the major hypotheses explaining the continued existence of sylvatic plague and proposed that fleas are not only important vectors, but also play a potentially pivotal role as plague reservoirs during maintenance and outbreaks of plague in natural systems (Wimsatt and Biggins 2009).

3.5 Species Susceptibility to Plague

Rodent species (Order Rodentia) are the primary vertebrate hosts of *Y. pestis* (Gage 1998). However, other mammals can become infected and some develop severe illness. Over 200 species of mammals have been implicated in *Y. pestis* infections worldwide (Pollitzer 1954; Poland and Barnes 1979). Additional mammalian orders that have also shown evidence of infection: Artiodactyla (e.g., mule deer; Edmunds et al. 2008), Carnivora (e.g., canids, felids and mustelids), Hyracoidea (e.g., hyraxes), Lagomorpha (e.g., rabbits, hares, and pikas), Marsupialia (e.g., marsupial mice, opossums), and Primates (e.g., humans) (Gage 1998; Dennis et al. 1999). Most of these non-rodent species, with the possible exception of pikas (Lagomorpha) in central Asia, are

thought to be incidental hosts only, incapable of sustaining *Y. pestis* among their populations. Other vertebrates, including birds, reptiles, and amphibians appear to be completely resistant to plague infection. However, it is possible that scavenging birds may disperse infected fleas during an outbreak (Gage et al. 1994).

Non-rodents

Susceptibility to plague infection varies among mammal species, and in some cases varies between populations of a given species (Gage 1998). Lagomorphs (rabbits and hares) are extremely susceptible to plague and occasionally suffer significant mortality during rodent epizootics (Pollitzer 1954; Von Reyn et al. 1976a). Carnivores also vary greatly in their susceptibility to plague. Of those tested in the lab, or for which evidence of exposure in the wild exists, dogs, coyotes, wolves, foxes, badgers, etc. seem resistant, while some mustelids (e.g., endangered black-footed ferrets, *Mustela nigripes*) and felids (cats) are highly susceptible. Mammalian predators can become infected with plague after ingesting rodent prey or being bitten by the prey's fleas. Blood sampled from carnivores in areas where plague is known to occur (i.e., serological surveys), frequently shows antibodies to *Y. pestis* (Barnes 1982; Salkeld and Stapp 2006). This, together with the laboratory challenges, implies that many carnivores are resistant to plague infection and could be important in the spread of *Y. pestis* across long distances (Gage et al. 1994; Antolin et al. 2002).

Members of the following families of carnivores have been reported to be seropositive to *Y. pestis* (showing a positive reaction to antibodies in blood serum): Canidae (dogs, coyotes, foxes), Felidae (cats, bobcats, mountain lions), Procyonidae (raccoons and ring-tailed cats), Mustelidae (weasels, ferrets, skunks, badgers, martens), Ursidae (bears), Viverridae (mongooses) (Gage 1998). With the notable exception of cats and some species of mustelids (e.g., the endangered black-footed ferret), however, it is rare to observe mortality of carnivores during rodent plague epizootics. Mortality in felids is high and there are numerous records of fatal plague infection in domestic cats, bobcats, lynx, and mountain lions (Tabor and Thomas 1986; Eidson et al. 1988; Gage et al. 1994, 1995; Wild et al. 2006; Wong et al. 2009).

There have been a number of serological surveys conducted on wild canids especially coyotes (*Canis latrans*). Arjo et al. (2003) found from 11-73% prevalence of antibodies for *Y. pestis* in 2 populations of coyotes in Utah. In a serological survey of coyotes in Yellowstone National Park from 1989-1993, a high prevalence of antibodies to *Y. pestis* was found, but seroprevalence (the rate or percentage of seropositivity in a population) declined over the 3-year period most likely due to the epizootic nature of plague in rodent populations (Gese et al. 1997). In southeastern Colorado, prevalence of *Y. pestis* antibodies among coyotes was 85% (Gese et al. 2004). (Swift foxes in this same study were 57% seropositive.) Seroprevalence in coyotes in California was much lower than in Colorado or Wyoming; only 11.7% of coyotes sampled were positive to *Y. pestis* (Hoar et al. 2003).

In a serological survey across western populations of Canada lynx (*Lynx canadensis*), 2 of 215 lynx tested positive for antibodies of *Y. pestis* (Biek et al. 2002). These two individuals were from Montana, the furthest south this particular study surveyed, and where plague is known to occur. Canada lynx (129 individuals) were reintroduced to southwestern Colorado from 1999 to 2003 as part of a species recovery program (Wild et al. 2006). The individuals originated in British Columbia, the Yukon, Manitoba, and Quebec, Canada, as well as Alaska. Of 52 lynx mortalities

documented by 2003, six had evidence of *Y. pestis* infection. Plague appeared to be the cause of death in five of these six animals. There was insufficient evidence to implicate plague in the 6th animal.

In a serological survey of 207 cougars (or mountain lions, *Puma concolor*) over 15 years in four populations of the Rocky Mountains, evidence was found of seroprevalence to *Y. pestis*, but at levels below 30% and these numbers varied geographically (Biek et al. 2006). In California from 1987 to 1990, 40% of mountain lions sampled were positive for antibodies of *Y. pestis* (Paul-Murphy et al. 1994). Primary pneumonic plague was contracted by a National Park Service employee at Grand Canyon National Park in late 2007 from a *Y. pestis*-infected carcass of a mountain lion (Wong et al. 2009; and see NPS Case Study Section 2.4).

Domestic cats have been shown to be susceptible to plague and have been implicated in human plague cases in the U.S. (Rust et al. 1971; Doll et al. 1994; Gage et al. 2000). To a lesser extent, dogs are also susceptible and have the potential to be a public health concern by transporting rodent fleas into homes especially in areas where plague epizootics are occurring (Rust et al. 1971; Chomel et al. 1994; Gould et al. 2008).

Salkeld and Stapp (2006) reviewed the literature for patterns of plague exposure in mammalian carnivores of North America, and explored the potential role that carnivores play in plague transmission cycles. As seen above, exposure to plague is common in carnivore species of the western U.S., which suggests that many are capable of surviving *Y. pestis* infection. Approximately 16% of all carnivores tested were seropositive, but prevalence varied taxonomically and geographically (Salkeld and Stapp 2006). Coyotes (*Canis latrans*), badgers (*Taxidea taxus*), and raccoons (*Procyon lotor*) were the most studied, and prevalence in badgers was much higher than that in raccoons and coyotes. This is most likely due to their propensity to prey on fossorial rodents. Salkeld and Stapp (2006) suggest that carnivores have the potential to act as plague reservoirs and their role in plague dynamics should not be overlooked, but this needs to be further evaluated. One likely problem with this hypothesis is that although these animals can become infected with *Y. pestis*, most are relatively resistant to infection and, therefore, would fail to develop the fulminant and ultimately fatal bacteremias that seem to be required for reliably infecting fleas.

Rodents

The susceptibilities of different wild rodent species to plague-related mortality vary greatly (Gage 1998). In western North America, virtually every rodent species has tested positive for plague at one time or another, but those showing high levels of resistance are few and include California voles (*Microtus californicus*), deer mice (*Peromyscus maniculatus*), and two species of kangaroo rats (*Dipodomys spectabilis* and *D. ordii*) (Antolin et al. 2002). Rodents particularly susceptible to plague include woodrats (*Neotoma* spp.) and sciurids (Antolin et al. 2002). Sciurid rodents include ground squirrels (*Spermophilus* spp., *Ammospermophilus* spp.), tree squirrels (*Sciurus*), chipmunks (*Tamias*, *Eutamias*), prairie dogs (*Cynomys* spp.), and marmots (*Marmota* spp.) (Poland and Barnes 1979; Barnes 1993; Biggins and Kosoy 2001a). In these species, epizootics can be explosive, with low resistance, high mortality, and rapid spread of plague across the landscape leading to amplification in the environment and spillover into other species including predators, peridomestic rodents, human pets, and humans themselves (Gage et al. 1994). Prairie

dogs (*Cynomys* spp.) in western North America are extremely susceptible to plague. The impacts of plague on all four species of prairie dogs are well known (Barnes 1982; Miller and Cully 2001; Gage and Kosoy 2005). Population declines at individual colonies of 85-100% have been recorded (Barnes 1982, 1993; Cully et al. 1997; Cully and Williams 2001; Antolin et al. 2002) and colony complexes have been reduced in size by 80-95% (Cully et al. 2010).

There is evidence that some populations of rodents exposed to enzootic cycles of plague are evolving a reduced susceptibility to plague (Cully 1993; Biggins and Kosoy 2001a). For example, individuals from a population of northern grasshopper mice (*Onychomys leucogaster*) from the Pawnee National Grassland where plague is known to occur, had higher resistance to laboratory plague challenges than did individuals from central Oklahoma, where plague has not been reported (Thomas et al. 1988). In California, the California ground squirrel began to have higher rates of survival during epizootics than early in the century when plague was newly introduced (Meyer et al. 1943; Nelson 1980). Rock squirrels (*Spermophilus variegates*) also developed resistance where contact with plague has been continuous, but not in areas where plague has not been identified for this species (Marchette et al. 1962; Quan et al. 1985).

3.6 Routes of Exposure

Human plague is generally the result of a bite from a rodent flea and this route of exposure occurs in the majority of human cases where the route can be identified (Gage et al. 1995). Flea bites are also the most likely route by which other animals are exposed; however, the route of exposure in wildlife and other non-human animals has rarely been closely examined. Approximately 20% of human cases are caused by exposure to tissues or body fluids of infected carcasses, or through scratches or bites from infected animals (Gage et al. 2000). *Y. pestis* has not been detected in saliva or salivary glands, so infections involving scratches or bites are due to other *Y. pestis*-infected fluids entering the wound. It is likely that wildlife is often exposed through ingestion of infected prey and/or scavenging carcasses.

Mode of transmission of *Y. pestis* to humans could be determined in 252 of 316 (80%) human cases reported from 1957 to 2004 in the Four Corners area of the U.S. (Eisen et al. 2007a). Flea-borne transmission was the most common (83.7%; n=211), followed by direct contact (15.1%; n=38) and air-borne exposure (1.2%; n=3) (Eisen et al. 2007a). Domestic cats served as the source of all air-borne exposures in this series of cases; infected cats can expel respiratory droplets infected with *Y. pestis* (Gage et al. 2000). The majority of cases associated with direct contact involved either rabbits or domestic cats, but also included prairie dogs, bobcats, gray foxes, and a coyote. The known vertebrate associations for flea-borne transmission (epizootic host-flea complexes) included mostly rock squirrels, followed by prairie dogs, ground squirrels, rabbits, domestic cats, woodrats, and a chipmunk (Barnes 1982). Recently, researchers have suggested that the rapidity with which plague epizootics move within large prairie dog colonies may be due to animal-animal spread via the pneumonic route.

3.7 Risk Factors

Humans at greatest risk for plague in the U.S. are those who live, work, and participate in outdoor recreational activities in areas of western states in which plague is endemic (Campbell and

Dennis 1998). The CDC has reported that most cases in the U.S. occur during the warmer seasons when epizootic activity is most common and humans are most likely to encounter host-seeking infectious fleas. However, it should be noted that plague exposure can happen any time during the year. Most often, winter cases in the U.S. are attributable to hunters, trappers, and cat owners coming in contact with infected animals, whereas warm season cases are most often caused by a bite from an infected flea (CDC 2005).

In general, risk factors are attributes of the landscape, environment, or host animals associated with a greater probability of plague occurring in a given region or animal population. By evaluating the risk factors, wildlife managers can attempt to predict the populations most likely to be affected by plague. Exposure and amplification risk factors include abiotic (e.g., climate, temperature, moisture, soil types) and biotic (e.g., flea and host densities, distributions, mobility, susceptibility) factors.

Areas of human risk for plague are typically defined by identifying the numbers of human cases by year, state, and county of exposure (Eisen et al. 2007b). The majority of human cases of plague are thought to occur during epizootics, when susceptible hosts (typically ground squirrels, prairie dogs, chipmunks or woodrats) perish in large numbers, and the infected fleas are forced to parasitize hosts upon which they would not ordinarily feed (Gage et al. 1995; Eisen et al. 2007a). However, most plague cases in New Mexico from 1976 to 2008 involved people residing in areas of endemic plague where the immediate source of infection was not readily apparent (Mann et al. 1979; Brown et al. 2010). These cases were strongly associated with the availability of harborage and food sources for wild rodents, which resulted from human activity in the immediate home environment. Among the factors found significant were the failure to store garbage in covered rodent-proof containers, gardens within 90 m of the home, unused outbuildings, feed storage in outbuildings, wrecked cars, lumber or wood piles, discarded tires, and miscellaneous trash piles (Mann et al. 1979). The association between the occurrence of human plague and the presence of harborage and food sources for rodents was also found in Bombay, India, where the Indian Plague Commission observed the plague attack rate was highest near the granaries, where rats abounded (Hirst 1953).

Because of the close association between pets and humans, there is increased risk to humans when a pet becomes infected with plague or where pets transport infected fleas into the home. The primary risk factor for feline plague appeared to be hunting and ingesting of rodents in plague-endemic areas in New Mexico from 1977 to 1985 (Eidson et al. 1988). Failure to control fleas on pets (dogs and cats) also appeared to be a risk factor in New Mexico (Mann et al. 1979). The ability to predict when and where infections are likely to occur is a useful tool to increase the local awareness of plague risk factors in the medical community and hence reduce the potential for plague-induced human mortality (Eisen et al. 2007a). It also improves the ability to target limited prevention resources. Climatological factors have been implicated in increased plague activity in several naturally occurring foci of plague, which could therefore increase human risk of exposure to plague. Epizootic activity appears to intensify when cool summer temperatures follow wet winters in the southwestern U.S. (Parmenter et al. 1999; Enscore et al. 2002). In a black-tailed prairie dog (*Cynomys ludovicianus*) in the north-central plains of Colorado, epizootic activity increased after El Nino events (Stapp et al. 2004). Although Collinge et al. (2005a, 2005b) failed to find an association between climatic variables and plague in black-tailed prairie dogs at their study

site in Boulder County, Colorado, they did observe positive associations between the occurrence of plague in prairie dogs in their Montana study area and the levels of April-July precipitation in the previous year and the number of "warm" (> 26.7^{o} C) days. A negative association was noted at this last site between plague in prairie dogs and the number of "hot" (> 29.4^{o} C) days.

Landscape and geography also can have an important role in the human risk for plague. Eisen et al. (2007a) developed spatial models for determining high risk habitats for human exposure in the Four Corners area of the U.S. (Arizona, Colorado, New Mexico, and Utah). The model they developed indicated that high-risk habitats can be classified accurately based on non-linear relationships with elevation and distance to key habitats. Their most conservative model predicted that 14.4% of the Four Corners region posed an elevated risk of peridomestic (occurring in and around human habitations) exposure. Of the four states, New Mexico had the highest coverage of peridomestic risk habitat (22.1%) and also reported the highest number of human cases (195 cases during 1957-2004). Utah, on the other hand, was classified as the state with the lowest coverage of peridomestic risk habitat (7.5%) and reported the fewest human cases (only four). Rodent and flea communities associated with key risk habitats included rock squirrels (*Spermophilus variegates*), woodrats (*Neotoma* spp.), chipmunks (*Tamias* spp.), and Gunnison's or white-tailed prairie dogs (*Cynomys gunnisoni* and *C. leucurus*), each of which was implicated in at least one human cases of plague. A geographic indicator of areas considered to be at high-risk for plague is habitats with high elevation. The risk of plague increases as elevation rises to approximately 2,300 m and then declines with further increases in elevation, where there is distance from high-risk areas (e.g., southern Rocky Mountain Pinon-juniper, Colorado plateau pinon-juniper woodland, Rocky Mountain ponderosa pine, and southern Rocky Mountain juniper woodland and savanna) (Eisen et al., 2007a).

Brown et al. (2010) also found a relationship between elevation and the number of plague cases using 29 years of pet-based surveillance data. They found that more aggregated distributions and earlier onset dates for plague cases were evident at lower elevations (1,238-2,115 m) as opposed to higher elevations (>2,115 m). They were also able to predict the number of cases reported per year at the lower elevations (1,238-2,115 m) based on warmer conditions 3-4 years prior and increased precipitation in the previous year. This is presumably based on the trophic cascade hypothesis, which suggests that suitable temperature and precipitation lead to an increased primary vegetative production; the increased food supply increases the carrying capacity of rodent populations, which also increases the number of hosts for fleas and this in turn increases the likelihood of epizootic spread (Parmenter et al. 1999; Enscore et al. 2002; Brown et al. 2010).

3.8 Clinical Forms of Plague

There are three predominant clinical manifestations of plague infection: bubonic, septicemic, and pneumonic. All three can be found in humans and non-human animals; however, they are usually most easily identified in humans. Common symptoms of plague include fever, headache, chills, myalgia (muscle pain), prostration (total exhaustion or weakness), malaise, gastrointestinal symptoms, and often acute lymphadenopathy (buboes) (Poland et al. 1994).

Bubonic plague is the most common form of plague in all animals, and occurs in approximately 80% of human exposures (Gage 1998). It is usually a result of a bite from an

infected flea, and may also occur when plague-infected material enters an animal through a break in the skin. This form of plague is characterized by "buboes," which are swollen lymph nodes that result from infection and inflammation due to *Y. pestis*. Buboes are usually found in the groin, armpit, or neck area, and can range in size from a few millimeters to as large as a hen's egg (Gage 1998). These buboes, which are often extremely tender and painful, usually develop in the lymph nodes, draining the initial site of infection. An inguinal bubo (in the groin area) will likely appear in those people who are bitten on the leg by an infectious flea, whereas those who contract plague as a result of handling an infected animal are likely to develop an axillary bubo (in the armpit area) (Gage 1998). The three most common locations for buboes in the U.S., in descending order, are inguinal, axillary, and cervical (neck area). Other lymph nodes can be affected, but this is most likely due to a secondary spread of *Y. pestis* via the bloodstream. In addition to swollen lymph nodes, a person infected with bubonic plague may experience mild to severe flu-like symptoms such as fever, headache, vomiting, nausea, chills, and exhaustion. Fatality rates for untreated bubonic plague cases are reported to be 50-60% (Poland et al. 1994; Levy and Gage 1999; Eisen et al. 2007a).

The second most common form of plague is septicemic plague. Septicemic plague occurs when *Y. pestis* enters into and spreads through the body via the bloodstream causing signs and symptoms of gram-negative sepsis. This is likely to occur in inadequately treated bubonic cases (secondary septicemic plague), but can also occur without prior evidence of lymphadenopathy (primary septicemic plague; Sebbane et al. 2006). The latter form can be dangerous because of the difficulties encountered in rapidly diagnosing human plague when there are no obvious buboes present. Septicemic plague can result in secondary plague pneumonia and occasionally meningitis; endophthalmitis (severe infections of the eye); and abscesses in the liver, spleen, kidneys, and lungs (Poland et al. 1994). Clinical signs of the septicemic form are similar to those associated with the bubonic form; however, they may become much more severe. In addition, the septicemic form may lead to respiratory distress syndrome, shock, and internal bleeding. In the U.S., about 10% of human plague cases do not develop buboes and about 50% of these non-bubo-forming cases die (Poland and Barnes 1979).

Pneumonic plague is the least common, but most dangerous, form of plague. It is characterized by severe, life-threatening respiratory and systemic illness. Primary pneumonic plague is acquired through inhalation of infectious respiratory droplets containing *Y. pestis*; secondary pneumonic plague occurs as a consequence of untreated bubonic or septicemic plague. Unlike bubonic and septicemic plague, pneumonic plague can potentially spread from person to person through infectious respiratory droplets (Gage 1998; Poland and Dennis 1999a; CDC 2005). Primary pneumonic exposure usually leads to clinical signs within 2-3 days. Symptoms of pneumonic plague can include high fever, chills, cough with bloody sputum, and severe breathing difficulty. Mortality rates for pneumonic plague are very high and probably reach 100% for untreated cases (Poland and Barnes 1979; Poland and Dennis 1999a). During 1925 to 2006, only 13 human cases of primary pneumonic plague were identified in the U.S.; among cases with known exposures, 6 of the 9 were associated with direct contact with infected pets (5 cats and one dog) and three cases were acquired in laboratory settings (Wong et al. 2009 summary of CDC unpublished data). The remainder had unknown exposures.

3.9 Diagnosis, Treatment, and Prevention of Plague (Human Considerations)

Plague has a high mortality rate if left untreated. Therefore, effective and timely antibiotic treatment is critical. When a diagnosis of human plague is suspected on clinical and epidemiological grounds, appropriate specimens for diagnosis should be obtained immediately and the patient should be started on specific antimicrobial therapy without waiting for a definitive answer from the laboratory (Poland and Dennis 1999b). However, even in places with known endemic plague and where human cases are the most prevalent (e.g., New Mexico), diagnosis is frequently delayed and treatment regimes often do not follow standard guidelines (Boulanger et al. 2004).

A person diagnosed with suspected plague should immediately undergo laboratory tests that include blood cultures for plague bacteria and microscopic examination of lymph gland, blood, and sputum samples. A definitive laboratory diagnosis of *Y. pestis* infection is based on the isolation and identification of the organism from clinical specimens or by demonstrating a four-fold change in antibody titer in paired serum specimens (Poland and Dennis 1999b). Diagnosis can be made from a variety of samples, including blood, aspirates from involved lymph nodes, and from sputum from patients with respiratory symptoms. In rare instances, skin scrapings, cerebrospinal fluid, and urine have also aided diagnosis (Rollins et al. 2003). A Gram stain will show polymorphonuclear leukocytes and gram-negative coccobacilli ranging from 1 to 2 um in length (Reisner 1996). The Wayson stain, a combination of basic fuchsia and methylene blue, should also be performed when available. In Wayson stain, the organism will appear as light blue bacilli with dark blue polar bodies against a pink background.

A course of antibiotics is given as part of the treatment process. The antibiotics most commonly used have included streptomycin, gentamicin, tetracycline (or doxycycline), and chloramphenicol (Perry and Fetherston 1997; Rollins et al. 2003). Since 1948, streptomycin has been considered to be the drug of choice for bubonic, septicemic, and pneumonic plague (Quan et al. 1948; Butler 1994; Boulanger et al. 2004). However, streptomycin is manufactured by one pharmaceutical company in North America and is available only in modest supplies and by request (Boulanger et al. 2004). Boulanger et al. (2004) found that gentamicin alone or in combination with a tetracycline was as efficacious as streptomycin for treating human plague. The efficacy of tetracycline alone could not be determined from their study, however.

Persons who have had close contact with the plague patient will be identified, especially if the patient is diagnosed with pneumonic plague (CDC 2005). Prophylactic (preventive) antibiotics may also be given to those who may have experienced likely exposures to plague, either through a potentially infected flea or when handling an animal that has been infected with plague. A person who will be in an area where plague outbreak is occurring can take a course of prophylactic antibiotics that will protect them from plague for 2 to 3 weeks, but such situations are rare in the U.S. and most frequently occur in plague-endemic developing countries that experience commensal rat-associated bubonic plague outbreaks or human to human spread of pneumonic plague.

Favorable clinical outcomes of plague in humans depend on prompt diagnosis and treatment (Dennis and Hughes 1997). According to some, treatment for pneumonic plague must be initiated within 12 hours of fever onset, or the infection is almost invariably fatal (Pollitzer 1954; Poland and Barnes 1979). Prompt and specific treatment reduces the case fatality rates from 60% or more

to less than 15% (Dennis and Hughes 1997). Individuals who are at risk (e.g., field biologists, NPS personnel in the field, outdoor enthusiasts, etc.) should be encouraged to learn about the risks of plague and be prepared to let health care professionals know they have been in close contact with high-risk animals. Many medical staff may be under the belief that plague is a thing of the past and may not treat accordingly or in a timely manner.

There is no currently available human vaccine for plague in the U.S., but plague vaccines are still being commercialized in other parts of the world (CDC 2005; Alvarez and Cardineau 2010). There are two "first generation vaccines" based on live attenuated and killed whole cells. The killed whole-cell vaccine protects against bubonic plague, but because of its multiple side effects and poor protection against pneumonic plague, it has not been available in the U.S. since 1999. The live attenuated vaccine provides protection against bubonic and pneumonic plague, but it is not commercially available in Europe and the U.S. because of its severe side effects that sometimes require hospitalization (Meyer et al. 1974; Alvarez and Cardineau 2010). Research is currently being conducted on so-called "second generation plague vaccines" (Alvarez and Cardineau 2010). The most promising candidates are recombinant subunit vaccines that express both F1 and V antigens of *Y. pestis* which are individually immunogenic and have additive protective effect in combination (Williamson 2001). The recombinant plague vaccine produced in *E. coli* is the most likely candidate to be licensed. Research is also being conducted on the efficacy of plant-derived vaccines for large-scale immunizations (Alvarez and Cardineau 2010).

4. Ecological and Conservation Implications of Plague

Infectious disease can represent a serious threat to threatened and endangered wildlife, occasionally causing sudden and unexpected local declines in abundance (Cleaveland et al. 2002). However, wildlife diseases are often overlooked as an important conservation issue until a major population decline occurs. Disease is likely to become increasingly important in wildlife conservation worldwide with expanding and growing human populations, increasing habitat loss and fragmentation, and changing climate.

Plague continues to cause large population reductions in rodents of several species within its native and introduced ranges (Biggins and Kosoy 2001b). For example, prairie dog populations have declined throughout western North America due to a combination of factors including habitat conversion, recreational shooting, and poisoning, but plague has also played an influential role (Miller and Cully 2001; Collinge et al. 2008; Cully et al. 2010). When plague enters a prairie dog colony, it can cause nearly 100% mortality in black-tailed (*Cynomys ludovicianus*) and Gunnison's (*C. gunnisoni*) prairie dogs (Cully 1997; Cully and Williams 2001), and high but more variable mortality in white-tailed (*C. leucurus*) (Anderson and Williams 1997) and Utah (*C. parvidens*) prairie dogs (Cully and Williams 2001). This has important conservation implications because prairie dogs have been shown to play key roles in grassland ecosystems. Black-tailed prairie dogs have been described as a keystone species in mixed- and shortgrass prairie ecosystems in western North America because they have been shown to influence the abundance and distribution of many native plant and animal species, including black-footed ferrets, burrowing owls, and mountain plovers (Kotliar et al. 1999; Kotliar 2000). Therefore, important current research topics include investigating the potential cascading effects on prairie ecosystems as a result of plague dynamics and prairie dogs.

More than half of the species of North American rodents of conservation concern occur within the range of plague in western North America (Biggins and Kosoy 2001a). Possible conditions that increase the vulnerability of rodents to plague include uniformly low resistance, high population densities (including coloniality and sociality), potentially abundant flea vectors that are efficient transmitters of plague, and a lack of evolved strategies to cope with demographic and environmental stochasticity (Biggins and Kosoy 2001a). Instability of rodent populations as a result of plague has the potential to affect their predators and even the structure of the ecosystems in general (Dobson and Hudson 1986).

Massive die offs of animals like prairie dogs can be caused by epizootic plague, but dramatic effects of the disease can occur via other routes also. The effect of plague can be amplified in a community of animals, whereby even relatively low incidence of the disease in a susceptible species becomes highly problematic for less common species. At least two mechanisms can account for this effect. First, highly susceptible "bystander" species that might play little or no role in maintaining *Y. pestis* in the animal community can become victims as a result of their susceptibility and proximity to the disease. Even a low rate of exposure might gradually cause their extirpation. The second route of amplified effects is exemplified by the black-footed ferret. Plague management (via reducing flea vectors) improved prairie dog survival by an estimated 31-45 % (Biggins et al. 2010), but flea control or an experimental plague vaccine improved survival of the associated ferrets by 82-91 % in Montana (Matchett et al. 2010). Ferrets and prairie dogs are probably similarly susceptible to the disease (90+ percent mortality compared to ~ 50% mortality in untreated human cases), but vulnerability of the ferrets might be increased due to their much larger home ranges and exposure via infected carrion. Regardless, ferret populations at the Montana study site have not been self-sustaining without plague management intervention, even when epizootics of plague were absent and prairie dogs persisted.

Although direct effects of plague on populations of susceptible species like prairie dogs, black-footed ferrets, and ground squirrels can be ominous, effects of plague can also be indirect, manifested through altered competition and predator-prey relationships. Plague can thus affect organisms that are resistant to the disease itself. For example, extreme oscillations of a prey species caused by density-dependent plague could have eliminated the peaks of prey populations that previously occurred during "boom years," preventing successful reproduction by carnivore or raptor species that relied upon those years to successfully rear young. The result could be gradual declines or extirpations of these predators even though the prey species persist. This phenomenon could be especially difficult to detect for long-lived predators with relatively low reproductive rates. Therefore, it is advantageous to keep plague in mind as a possible cause of decline in widely varying vertebrate taxa.

NPS employees should remain alert to short-term changes in abundance of rodents and carnivores, and report (or collect, following prescribed methods; Appendix 12.2) dead mammals encountered, especially if there is no evidence of accident or injury. Examples that have led to discovery of plague have included sudden disappearances of chipmunks and ground squirrels in campgrounds and other sites where they had been commonly seen, cessation of reported problems due to "nuisance" species such as deer mice and wood rats, and unexplained failures to find predators in preferred hunting areas where they had been commonly seen (e.g., harriers or coyotes hunting voles in meadows). Observant naturalists can detect changes that are sometimes subtle.

These phenomena have multiple potential causes, but their detection should trigger more careful monitoring.

Over the long term (decades or more), endemic plague has caused major declines or local extirpations of populations of some species. Plague invaded some of these areas many decades ago (1920-1960), and more recently-arrived resource managers and other residents may now consider vastly altered levels of abundance as normal. Our perceptions of normalcy might be another example of the shifting baseline syndrome associated with the general amnesia of passing human generations (Papworth et al. 2009). It is thus useful to query observers who have been familiar with an area for many years to accumulate evidence of past abundance of various animals and the potential for plague to have influenced the structure and function of various ecosystems. Because national park policy emphasizes maintenance of natural ecosystems to the extent possible, identifying these alterations caused by plague and attempting to restore the original structure of animal communities is important. Most importantly, NPS biologists and managers at parks in the western U.S. would be well-advised to critically examine their ecosystems and to keep plague in mind when considering potential sources of almost any demographic problem involving rodents and their predators. USGS biologists are attempting to investigate ecosystem-level disruptions caused by plague and will appreciate any evidence NPS employees can provide.

5. Plague Management Options

5.1 Prevention and Control

Surveillance, education, and environmental management are the cornerstones of plague prevention and control (Campbell and Dennis 1998). Generally, prevention measures should aim to educate the public regarding exposure sites, manifestations, diagnosis of disease, and what steps can be taken to protect individuals and their families (Poland and Barnes 1979). Residents living within highly suitable plague habitat are usually advised to eliminate harborage (e.g., piles of wood, brush, or debris) and food sources (e.g., pet food, garbage) for wild rodents. Handling sick or dead animals without adequate personal protective gear should be avoided in all locations. Owners of pets in plague endemic areas are advised to prevent their cats from hunting by keeping them indoors. Dusting cats with approved insecticide flea powders weekly during the summer was also suggested; flea collars were not considered to be as effective as regular dusting (Eidson et al. 1988). Dogs also should be prevented from roaming freely in areas where plague occurs in local rodent populations because of the risk they will transport infected fleas into homes (Gould et al. 2008). Dogs that are active in outdoor sites with rodent populations likely to be infected with plague should be treated for fleas using flea powders or more recently developed topically applied liquid or orally administered insecticidal formulations.

Control of transmission is usually directed at controlling the rodent reservoirs and flea vectors of plague (Gratz 1999b). The first step involves diagnosis and recognition of the disease, which is essential to establish firmly the existence of plague. However, it should be noted that laboratory confirmation of plague can take many days to a few weeks and in some situations, such as sudden die-offs in prairie dogs or other highly susceptible species, it might be advisable to begin plague control measures when other likely sources of mortality, including poisoning, can be eliminated. This problem illustrates why it is essential to have effective protocols for rapid sample

submission and plague testing. Plague control measures should start with the control of flea vectors rather than the reservoir rodent (Gratz 1999b). Although it might be feasible to control rodents in a plague focus, the death of large numbers of plague-infected rodents is likely to introduce large numbers of fleas into the environment, which could potentially spread the disease to a greater extent than would have been likely had the rodent hosts not been killed (Barnes 1982; Gage et al. 1995). Elimination of rodents on a site with abundant food and harborage also could lead in coming weeks to the immigration of plague-infected animals from surrounding regions, a situation that could prolong plague risks for humans. In such situations, it might be better to leave insecticide-treated rodents in place, a solution that also could provide conservation benefits for any local populations of threatened rodents. Also, use of rodenticides poses the risk of killing non-target species. The literature on control of fleas with the use of insecticides is extensive (Gratz and Brown 1983). Insecticidal dusts applied to rodent runways and burrows (for commensal rodents) or into rodent burrows (for wild rodents) are effective in controlling flea vectors (Seery et al. 2003). Rodents crossing the dust patches on runways or when exiting burrows pick up the insecticidal dust on their fur and spread it over themselves when grooming, killing the fleas. Bait boxes have also been used. Bait boxes include a food bait attractant in the interior of the box and insecticidal dusts at the entrance to the box. Rodents entering the box come across the dust, picking up the insecticide on their fur and carrying it back to their nests, killing fleas on their bodies and fleas in the nest (Kartman 1947; Barnes and Kartman 1960; Barnes et al. 1974).

In the U.S., insecticides commonly used to dust rodent burrows include permethrin, pyraperm, pyriproxyfen, deltamethrin, and carbaryl (Montman et al. 1986; Beard et al. 1992; Karhu and Anderson 2000; Seery et al. 2003; Hoogland et al. 2004; Mian et al. 2004; Matchett et al. 2010). Infusion of Utah prairie dog (*Cynomys parvidens*) burrows with Pyraperm killed fleas and immediately halted the spread of plague within colonies (Hoogland et al. 2004). A single application of deltamethrin significantly reduced populations of the plague vector *Oropsylla hirsuta*, and other flea species on black-tailed prairie dogs (*C. ludovicianus*) and in their burrows for at least 84 days at the Rocky Mountain Arsenal National Wildlife Refuge, Colorado (Seery et al. 2003). In California, lufenuron was used to control fleas on rodent communities (Davis 1999; Davis et al. 2008). Lufenuron is a chitin synthesis inhibitor and an insect development inhibitor that disrupts the hatching of flea eggs and the larval molting processes. The lufenuron was delivered via feed cubes to four different species of rodents and flea loads were significantly reduced on California ground squirrels (*Spermophilus beecheyi*), long-eared woodrats (*Neotoma macrotis*), and mice (*Peromyscus* spp.), but not on Merriam's chipmunks (*Tamias merriami*).

Insecticide resistance can be a problem in all insect groups that serve as vectors of emerging diseases (Brogdon and McAllister 1998). However, the full effect of resistance on control efforts is not known and specifically, flea resistance to insecticides can be a serious impediment to control of plague. Therefore, the susceptibility of target flea populations to locally-used insecticides should be determined on a periodic basis. A test to determine resistance in fleas is available from the World Health Organization (WHO) Regional Offices or from the Division of Control of Tropical Diseases (Gratz 1999b). Fortunately, most areas subject to plague epizootics, with the possible exception of a few sites in California, have had little previous exposure to insecticides and fleas in these areas are likely to be susceptible to most of the above insecticides. Continued use, however, could lead to resistance.

Promising research is currently being conducted on the prevention of plague in wild rodents by vaccination (Creekmore et al. 2002; Mencher et al. 2004; Rocke 2008a, 2010). Vaccination could reduce epizootics in rodent populations, but practical large-scale vaccination of free-ranging wildlife populations can only be achieved through voluntary consumption of an oral vaccine (Rocke et al. 2008a, 2010). Rocke et al. (2010) used two modified recombinant raccoon poxviruses (RCN-F1 and RCN-V307) to demonstrate that consumption of baits containing both vaccines by black-tailed prairie dogs (*Cynomys ludovicianus*) resulted in near-complete protection to subsequent plague challenge. The survival of prairie dogs administered oral baits with vaccine was better than prairie dogs injected subcutaneously with F1-V fusion protein, a vaccine demonstrated to induce immunity to plague in mice and other mammals (Powell et al. 2005), including the endangered black-footed ferret (Rocke et al. 2008).

The efforts currently used to halt the spread of plague in prairie dog colonies mostly relies on dusting individual burrows with pesticides that kill plague-infected fleas after a plague outbreak has begun (Seery et al. 2003; Hoogland et al. 2004). Pesticide application is labor intensive, costly, and difficult to sustain over time. Many times such efforts are initiated too late to substantially affect colony survival. The use of oral baits with plague vaccine could provide a practical, alternative approach, although more research is needed to determine the timing of bait/vaccine delivery, the duration of the immunity provided, and the effects on nontarget species (Rocke et al. 2010). Reduction of plague epizootics in prairie dog colonies through oral vaccination in targeted locations could minimize the risk of disease transfer to other animals, including humans. This approach could also enhance prairie dog conservation, and conservation of the endangered black-footed ferret, an animal that relies exclusively on prairie dogs for food and habitat.

5.2 Surveillance

Effective plague prevention and control programs require up-to-date information on the incidence and distribution of the disease (Gage 1999). The best means of gathering this information is through a surveillance program that collects, analyzes, and interprets clinical, epidemiological, and epizootiological data on plague. Systematic surveillance information over many years can provide information to predict areas where future human cases and rodent epizootics might occur, identify the most common zoonotic sources for human infection, identify the most important rodent and flea species maintaining a given focus of *Y. pestis*, indicate hosts and fleas that should be targets for control measures, assess the effectiveness of prevention and control programs, identify local ecological factors that may result in increased plague exposure risks for humans, and detect trends in epidemiology and epizootiology of plague (Gage 1999).

Human surveillance involves reporting confirmed cases of human plague to the CDC and World Health Organization (WHO). Local physicians and other health care workers should be familiar with the symptoms of plague and samples should be collected for diagnostic confirmation at a microbiological laboratory. Health care workers should know where to send samples for bacteriological or serological confirmation.

Rodents are the primary vertebrate reservoirs of plague so surveillance programs should monitor plague activity in susceptible rodent populations. The most common techniques used for monitoring plague in rodent populations include collecting and examining dead rodents, visually

monitoring activity among plague-susceptible rodents, trapping rodents for population data, serum, tissue samples, and ectoparasite collections, and conducting serological surveys of carnivore populations that consume rodents (Gage 1999).

Examples of methods for opportunistic and targeted wildlife surveillance are presented in Appendix 12.4. Unfortunately, the trapping and sampling of large numbers of rodents and the use of certain other forms of animal-based surveillance can be very labor intensive and costly. Surveillance systems also will be effective only if samples are analyzed in a timely manner and the results are quickly returned to local end users or others who need this information. For these reasons, care must be taken to carefully evaluate the goals of the program; choose the surveillance methodology(ies) most suitable for the local plague focus; and determine whether the likely results will justify the time and money spent collecting, shipping and testing the samples. Such cost-benefit analyses can help managers develop a plague surveillance program that will provide usable data and be sustainable over the long-term; rather than one that might initially provide more data, but must be abandoned in a few seasons because of its excessive expense and workload. For example, in those situations where many of the local plague hosts are diurnally active and relatively easy to observe (prairie dogs, ground squirrels, chipmunks), it might be advisable to institute a visual surveillance system where local park staff monitor rodent activity in campgrounds or other high use sites. If rodent activity suddenly diminishes, efforts can be made to collect rodent carcasses at the site or sample rodent burrows for fleas using the flagging (swabbing) method described elsewhere.

6. Intra-NPS Communication

Risk Communication and Plague

To much of the general public the word ‘plague’ conjures up images of the “Black Death” of the 14th century, one of the deadliest disease pandemics in human history. It is understandable, therefore, that many of today’s general population would be very concerned to hear that a case of plague may have recently occurred in a National Park near them. Proper communication to the public is critical during any investigation of a plague illness or death. Park Management and Public Affairs personnel stationed at locations in the western United States where plague is endemic should become familiar with plague and with the resources available to assist them in communication efforts related to plague.

Because most plague outbreaks in national parks involve wildlife resources, the NPS Wildlife Health Program will likely be the first to be contacted if plague is suspected as the cause of wildlife mortality or illness. The WHP Zoonotic Disease Veterinarian can provide initial information regarding ongoing surveillance for and the diagnosis of plague, and will communicate with NPS Office of Public Health personnel to help the park determine an appropriate course of action.

Each NPS unit has an assigned Public Health Consultant available to assist during human disease investigations. This Public Health Consultant should be contacted immediately should a

suspected human case of plague become known. The Public Health Consultant may then draw upon the resources of the NPS Office of Public Health, which includes a medical epidemiologist. Accurate information about plague and the risks of acquiring the disease should be provided to all members of the community as soon as possible before erroneous rumors and speculations begin.

Employee and Community Meetings should be held as soon as possible and at periodic times in any disease investigation. These meetings should include as many of the following personnel as possible, whether in person or via conference call: the park Superintendent or his/her designate, the park Public Affairs Specialist, the Public Health Consultant assigned to the park unit, the NPS Medical Epidemiologist, a Wildlife Health Program Veterinarian, the park Safety Officer, the local county Epidemiologist or other representatives from the local Health Department, and representatives from the State Division of Vector Borne and Zoonotic Diseases (or equivalent) or the state epidemiologist. High profile investigations may also include CDC personnel.

Collaboration between NPS and other federal agencies, local health departments, and state health agencies provides a pool of expertise that not only assists in the investigations process, but also increases credibility with the public during emergencies. However, for plague cases within a unit of the National Park System, the NPS takes the lead in risk communication efforts, with all other agencies providing a supportive role by invitation.

Press releases should be carefully crafted by Public Affairs in consultation with the NPS Wildlife Health Program and Office of Public Health in order to ensure they contain accurate information about the disease and timely diagnostic data. When informing the public about the progress of a plague investigation, great care should be taken to ensure that only confirmed information is disseminated. Positively confirming a case of plague may take a few weeks. While preliminary laboratory information may become available that indicate plague is likely, be careful to discuss the case in terms of "possible" or "potential" plague until the final confirmatory test result is available.

Risk communication is an essential part of a plague investigation. Taking steps such as these will help the public understand and respond correctly to the situation. See Appendix 12.3 for a sample communication flow chart.

7. Cooperation & Coordination with Other Agencies

Need for cooperation

Wildlife diseases in general—and plague specifically—do not respect political borders. This makes it difficult when trying to manage wildlife at risk to plague, as well as vectors of plague. Rarely if ever does a wildlife population reside completely within an NPS unit with no overlap onto other jurisdictions. Therefore, close partnerships with other agencies and private land owners or lessees are necessary to achieve management goals. Goals must be compatible with both agencies' missions and mandates. Thus, goals and management actions may not be mirrored among all agencies. While challenging, it may not be impossible to reach common ground with other agencies, considering there is a not always a universal goal of containing and controlling the spread of plague as well as increasing the body of knowledge pertaining to the disease.

International Health Regulations (mandated by the World Health Organization) require notification of the CDC in the event of a suspected plague outbreak in humans or other animals. The NPS Wildlife Health Program or Office of Public Health will communicate with CDC when such a situation exists (see Section 12.3, communications flow chart).

Potential cooperating agencies

Interested parties to keep in mind when considering and developing plague/wildlife management plans include: county and other local health departments, state wildlife agencies, state wildlife and public health veterinarians, private land owners, neighboring federal land management agencies (e.g., U.S. Forest Service, Bureau of Land Management, U.S. Fish and Wildlife Service), non-governmental agencies (e.g., Nature Conservancy), U.S. Geological Survey, Tribes, and the CDC.

8. Data Management

8.1 Within the NPS

It is essential to maintain complete records of animals tested for plague or other infectious diseases within an NPS unit. Basic animal identification information should include: date, species, sex, estimated or exact age, location animal was found (preferably with UTM coordinates), clinical signs of disease or a visual description of the carcass, status of any other animals seen in the vicinity, samples taken, submitted and banked, results of any diagnostic test(s), and results of the complete necropsy if performed (see Appendix 12.6 for a link to a sample submission form). One efficient way to accomplish this is with a computer spreadsheet with a column dedicated to each category. Additionally, plague test results can be compiled in a separate hard copy notebook. Finally, in order to have a centralized database within the NPS, it is requested that all plague testing results be forwarded to the Biological Resource Management Division (BRMD) zoonotic disease coordinator. Much of the time plague testing will be coordinated through the BRMD office, however, if alternative laboratories are utilized, please forward these results to the coordinator (see Appendix 12.3 for contact information).

8.2 Outside of the NPS

A national database consolidating plague information for wildlife is not available. The CDC, the National Wildlife Health Center, and many states have records of samples they have received; however, there is little or no regular communication of these results except via periodic written reports.

9. Education & Outreach

As previously stated, the word “plague” can invoke a deep-seated fear in employees and visitors alike. A carefully crafted, proactive message is necessary to convey the appropriate level of risk, which is typically quite low, and to avoid needless over- or under-reaction by employees and

user groups. Resource managers, information officers, and interpretive staff in parks with historic and/or ongoing wildlife plague should make information available to park employees and visitors via all available means, including periodic staff briefings, informational kiosks, interpretive programs, and web-based media. Additional information can be found on the NPS One Health website, and websites for the Office of Public Health and the Wildlife Health Program. Examples of signs that can be used to inform the public of ongoing plague investigations can be found in Appendix 12.5.

10. NPS Policies and Implications for Plague Management

Any proposed plague management plans or activities must be developed in accordance with applicable Federal and NPS laws and policies, including the Organic Act of 1916, NPS Management Policies 2006, and NPS Director's Orders. Plague management is not explicitly directed by law or by NPS management policies; however, NPS units are required to manage plague under sections of the Organic Act and Management Policies 2006, in order to protect native species and ecosystems, to remove exotic species, and to protect human health. For example, the Organic Act of 1916 states:

> "The National Park Service will strive to understand, maintain, restore, and protect the inherent integrity of the natural resources, processes, systems, and values of the parks…"
>
> Plague epizootic and enzootic activities clearly threaten the integrity of numerous animal species and associated ecosystems, so management is warranted. NPS Management Policies 2006 pertinent to plague management include:
>
> 4.1 General Management Concepts:
> "The Service will not intervene in natural biological or physical processes, except
> • to restore natural ecosystem functioning that has been disrupted by past or ongoing human activities; or
> • when a park plan has identified the intervention as necessary to protect other park resources, human health and safety, or facilities."
>
> 4.4.1.3 Definition of Native and Exotic Species
> "... <u>Exotic species</u> are those species that occupy or could occupy park lands directly or indirectly as the result of deliberate or accidental human activities."
>
> 4.4.2 Management of Native Plants and Animals
> "The Service may intervene to manage individuals or populations of native species only when such intervention will not cause unacceptable impacts to populations of the species (and) management is necessary … to protect human health as advised by the U.S. Public Health Service (which includes the Centers for Disease Control and the NPS public health program)."
>
> 4.4.4.2 Removal of Exotic species Already Present
> "All exotic plant and animal species that are not maintained to meet an identified park purpose will be managed—up to and including eradication—if (1) control is prudent and feasible, and (2) the exotic species

- interferes with natural processes and the perpetuation of natural features, native species or natural habitats, or
- disrupts the genetic integrity of native species, or
- disrupts the accurate presentation of a cultural landscape, or
- damages cultural resources, or
- significantly hampers the management of park or adjacent lands, or
- poses a public health hazard as advised by the U. S. Public Health Service (which includes the CDC and the NPS public health program), or
  creates a hazard to public safety."

4.4.5.3 Pesticide Use
"All prospective users of pesticides in parks must submit pesticide use requests ... The decision to incorporate a … pesticide … will be based on a determination by a designated IPM specialist."

8.2.5.5 Public Health Program
"The Service will work to identify public health issues and disease transmission potential in the parks and to conduct park operations in ways that reduce or eliminate these hazards. Park managers will pursue these goals with technical assistance provided under the auspices of a Service-wide public health program. The public health program will use the consultation services of commissioned officers of the U.S. Public Health Service."

Director's Order #83

- "It is the policy of the NPS to protect the health and well-being of NPS employees and park visitors through the elimination or control of disease agents and the various modes of their transmission to man and to ensure compliance with applicable Federal, State and local public health laws, regulations and ordinances. Implementation of this policy will be tempered by the Organic Act's requirement that the NPS conserve the scenery and natural and historic objects and the wildlife therein.
- Managers will reduce the risk of transmission of vector-borne and zoonotic diseases to park visitors and employees through education, surveillance, and control efforts … while minimizing adverse impact on natural and cultural resources.
- Guidance and information will be provided on specific diseases by the Zoonotic, Vector-borne and Environmentally Transmitted Disease (ZED) Steering Committee, a collaborative effort of the Biological Resource Management Division and the Public Health and Risk Management Programs."

NEPA Considerations

Plague management plans and activities must incorporate requirements of the National Environmental Protection Act (NEPA) at the appropriate (earliest possible) time in the planning process. Appendix 12.1 provides additional information and forms pertaining to NEPA compliance. At this time, dusting or the use of insecticide-containing bait boxes or systemic insecticide-containing baits are the only real options for plague control [e.g., Davis et al. (2008), Dolan et al. (2004), Gage et al. (1997)]. Therefore, most plague management has focused on using Categorical Exclusion 3.4 E(3):

> "Removal of individual members of non-threatened/endangered species or populations of pests and exotic plants that pose imminent danger to visitors or an immediate threat to park resources."

However, the impacts of broad scale application of an insecticide on non-target invertebrate and vertebrate species and on other environmental parameters such as water quality have not been assessed, and warrant additional NEPA consideration and consultation with the NPS Environmental Quality Division (EQD). To avoid harming non-target species, using host-targeted bait for flea control or bait tubes (boxes) is warranted because these methods greatly reduce the amount of pesticide applied at a given site compared to area-wide or even burrow-level applications. In general, a Categorical Exclusion may not be sufficient if plague management activities are ongoing; in that case it may be more appropriate to consider producing an Environmental Assessment to address NEPA requirements. If additional control methods (such as oral plague vaccines) become available, EQD input and additional NEPA consideration will be necessary prior to implementation.

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12. Appendices

12.1 NEPA Compliance & Information

Excerpt from Director's Order 12: Determining the Appropriate NEPA Pathway

A. Documentation

A NEPA review includes documentation of the analysis, which then becomes available to the public. The degree of public input and the detail included in the documentation varies depending on the severity, in context, of the environmental impacts of the proposal and alternatives. Managers involved with writing and implementing a plague management plan should consult with their park and regional environmental compliance personnel.

B. Five options

Five options to document a NEPA analysis are available. In addition, depending on where prairie dogs or other potential plague hosts occur, it may be necessary to prepare an AEF (Assessment of Effects Form) if the colony is located in a historic district, or if concerns about historic structures, features, etc. occur. This would be completed in addition to the Environmental Assessment, Environmental Impact Statement, and/or Categorical Exclusion forms.

1. Memo to file—Prepare a memo to file when the proposal has already been analyzed in site-specific detail in a previous NEPA document, no different impacts or changes to the project are expected, and

environmental conditions have not changed. A notation to this effect should be prepared and placed in the project files.
2. CEs for which no formal documentation is necessary—This option is applicable when the action is described using one of the categories in section 3.3 and no exceptions (section 3.5) exist. If appropriate, and if a project file exists, a memo to file may be placed in the project file.
3. CEs for which a record is needed—Prepare these records when the action is described using one of the categories in section 3.4 and no exceptions (section 3.5) exist. Complete the ESF (appendix 1) and the CEF (appendix 2) and include them with the project file.
4. EIS—Prepare an EIS when the potential for significant impact to the human environment exists (see section 4.2), as indicated by an EA, an ESF, or other scoping, or because the proposed action or alternative is described in section 4.4.
5. EA—Prepare when:
(a) the significance of impacts is unknown (e.g., to determine whether an EIS is required).
(b) the proposed action is not described on either of the CE lists (sections 3.3 and 3.4) or the list of actions that normally require an EIS (section 4.4).
(c) the proposed action would take several CE categories to describe fully, would involve one or more of the exceptions described in section 3.5, or would involve unresolved conflicts concerning the use of resources.
Each of these options has specific content and public involvement requirements.
For options 3, 4, and 5, you should complete the clear definition of objectives, an initial range of alternatives and actions including connected and cumulative actions, internal interdisciplinary scoping (see section 2.6), and an ESF (see section 2.6 (B) (1)) before you determine the appropriate NEPA pathway.
For options 1 or 2, no ESF is required, although you should consult the list of exceptions to categorical exclusions (see section 3.5) to see whether any apply.

C. The choice of pathways

1. If you believe option 1 above applies, the IDT should re-read the NEPA document that it believes already describes and analyzes the impacts of the action. If it does so in site-specific detail, and the analysis is up-to-date (see section 2.6 (C)), no further documentation is required, although for the administrative record, you must write a memo to file as described above. (Also note that this memo should be approved by the Superintendent or his/her designee after consultation with the regional environmental coordinator.)
2. If option 1 does not apply, but you believe option 2 does, check the list of actions in section 3.3. If it is described on this list (and no exceptions in section 3.5 apply), you may take action without further paperwork.
3. If the action is not on the list in section 3.3, or if it is described and analyzed in a previous NEPA document, you should complete internal scoping, complete the ESF form, and check the list of actions in section 3.4.
4. If the action is described in section 3.4, and no exceptional circumstances (section 3.5) exist, the CE is likely the appropriate pathway. Section 3.2 of this handbook describes the categorical exclusion process in more detail.
5. If the action is not described on the CE list, check the list of actions that normally require the preparation of an EIS (section 4.4). If it is on the list, or the potential for significant impacts exists as indicated by the ESF, you must write an EIS. Chapter 4.0 of this handbook details the process to follow in preparing an EIS.
6. You should prepare an EA if the action is not described on any list, or if one of the following applies:

• one or more of the categories on the ESF apply or are checked “data needed” or “yes,” but you do not know if any will result in significant impacts;
• several categories in section 3.4 are required to completely describe the project;
• the action is described in section 3.3 or 3.4, but one or more of the exceptions in section 3.5 apply;
• unresolved conflicts concerning the use of resources exist; or
• the significance of impacts is unknown.

If the EA indicates there may be significant impacts, you must prepare an EIS, unless the unique and limited circumstances described in section 5.4 (F)(3) apply and a proposal can be modified with mitigation measures to lessen the severity of impact, sometimes referred to as a “mitigated EA.” The use of the terms “significant” and “significance” over the years has become quite contentious in NEPA documents. It is highly recommended that these terms be avoided in EAs and EISs, since these terms apply primarily to the determination of the most appropriate NEPA pathway.

ENVIRONMENTAL SCREENING FORM

This form, along with all other NEPA-related forms, should be filled out in PEPC and park staff should have access to this program as should regional office staff. PEPC is the NPS standard for NEPA compliance and ensures that the documentation is reviewed by appropriate staff, includes check boxes for when review begins and ends and when documents are signed by the decision making individual. There is also a pathway to post documents for public review and allows the public to post comments online.

ENVIRONMENTAL SCREENING FORM

This form must be attached to all documents sent to the regional director's office for signature. Sections A and B should be filled out by the project initiator (may be coupled with other park project initiation forms). Sections C-I are to be completed by the interdisciplinary team members. Although you may modify this form to fit your needs, you must ensure that the form includes information detailed below and must have your modifications reviewed and approved by the regional environmental coordinator.

A. PROJECT INFORMATION

Park Name_____ Project Number _____

Project Type (*Check*): ☐ Cyclic ☐ Cultural Cyclic ☐ Repair/Rehab ☐ ONPS
☐ NRPP ☐ CRPP ☐ FLHP
☐ Line Item ☐ Fee Demo ☐ Concession Reimbursable
☐ Other (specify) _____

Project Location _____ Project Originator/Coordinator _____

Project Title _____

Contract # _____ Contractor Name _____

Administrative Record Location _____

Administrative Record Contact _____

B. PROJECT DESCRIPTION/LOCATION *[To begin the statutory compliance file, attach to this form, maps, site visit notes, agency consultation, data, reports, categorical exclusion form (if relevant), or other relevant materials.]*

Preliminary drawings attached? ☐Yes ☐No Background info attached? ☐Yes ☐No

Date form initiated _____ Anticipated compliance completion date _____

Projected advertisement/Day labor start _____ Construction start _____

Is project a hot topic*? ___ Yes ___ No SHPO Log No ***(NM only)*** _____

(*controversial or sensitive issues that should be brought to the Regional Director's attention)

_____

_____

_____

_____

_____

_____

C. RESOURCE EFFECTS TO CONSIDER *(Tailor the following to meet individual park/unit project needs.)*

Please see section F (Instructions for Determining Appropriate NEPA Pathway) prior to completing this section. Also, use the process described in DO-12, §2.9 and §2.10; §3.5; §4.5(G) to (G)(5) and §5.4(F) to help determine the context, duration and intensity of effects on resources.

Are any impacts possible on the following physical, natural or cultural resources?	Yes	Level of Effect	No	Data Needed to Determine
1. Geological resources – soils, bedrock, streambeds, etc.				
2. From geohazards				
3. Air quality				
4. Soundscapes				
5. Water quality or quantity				
6. Streamflow characteristics				
7. Marine or estuarine resources				
8. Floodplains or wetlands				
9. Land use, including occupancy, income, values, ownership, type of use				
10. Rare or unusual vegetation – old growth timber, riparian, alpine				
11. Species of special concern (plant or animal; state or federal listed or proposed for listing) or their habitat				
12. Unique ecosystems, biosphere reserves, World Heritage Sites				
13. Unique or important wildlife or wildlife habitat				
14. Unique or important fish or fish habitat				
15. Introduce or promote non-native species (plant or animal)				
16. Recreation resources, including supply, demand, visitation, activities, etc.				
17. Visitor experience, aesthetic resources				
18. Cultural resources including cultural landscapes, ethnographic resources, sacred sites				
19. Socioeconomics, including employment, occupation, income changes, tax base, infrastructure, concessions				
20. Minority and low income populations, ethnography, size, migration patterns, etc.				
21. Energy resources				
22. Other agency or tribal land use plans or policies				
23. Resource, including energy, conservation potential				
24. Urban quality, gateway communities, etc.				
25. Long-term management of resources or land/resource productivity				
26. Pollution prevention (greening the parks)				
27. Wilderness – suitability, recommended, potential, designated				
28. Park operations				
29. Other important environmental resources (e.g. geothermal, paleontological resources, night skies)?				

a) D. Mandatory Criteria

Mandatory Criteria: If implemented, would the proposal:	Yes	No	Comment	Data Needed to Determine
A. Have material adverse effects on public health or safety?				
B. Have adverse effects on such unique characteristics as historic or cultural resources; park, recreation, or refuge lands; wilderness areas; wild or scenic rivers; national natural landmarks; sole or principal drinking water aquifers; prime farmlands; wetlands; floodplains; or ecologically significant or critical areas, including those listed on the National Register of Natural Landmarks?				
C. Have highly controversial environmental effects?				
D. Have highly uncertain and potentially significant environmental effects or involve unique or unknown environmental risks?				
E. Establish a precedent for future action or represent a decision in principle about future actions with potentially significant environmental effects?				
F. Be directly related to other actions with individually insignificant, but cumulatively significant, environmental effects?				
G. Have adverse effects on properties listed or eligible for listing on the National Register of Historic Places?				
H. Have adverse effects on species listed or proposed to be listed on the List of Endangered or Threatened Species or have adverse effects on designated Critical Habitat for these species?				
I. Require compliance with Executive Order 11988 (Floodplain Management), Executive Order 11990 (Protection of Wetlands), or the Fish and Wildlife				

Mandatory Criteria: If implemented, would the proposal:	Yes	No	Comment	Data Needed to Determine
Coordination Act?				
J. Threaten to violate a federal, state, local, or tribal law or requirement imposed for the protection of the environment?				
K. Involve unresolved conflicts concerning alternative uses of available resources (NEPA sec. 102(2)(E)?				
L. Have a disproportionate, significant adverse effect on low-income or minority populations (EO 12898)?				
M. Restrict access to and ceremonial use of Indian sacred sites by Indian religious practitioners or adversely affect the physical integrity of such sacred sites (EO 130007)?				
N. Contribute to the introduction, continued existence, or spread of federally listed noxious weeds (Federal Noxious Weed Control Act)?				
O. Contribute to the introduction, continued existence, or spread of non-native invasive species or actions that may promote the introduction, growth or expansion of the range of non-native invasive species (EO 13112)?				
P. Require a permit from a federal, state, or local agency to proceed, unless the agency from which the permit is required agrees that a CE is appropriate?				
Q. Have the potential for significant impact as indicated by a federal, state, or local agency or Indian tribe?				
R. Have the potential to be controversial because of disagreement over possible environmental effects?				
S. Have the potential to violate the NPS Organic Act by impairing park resources or values?				

E. OTHER INFORMATION *(Please answer the following questions/provide requested information.)*

Are personnel preparing this form familiar with the site? ☐ Yes ☐ No

Did personnel conduct a site visit? ☐ Yes ☐ No *(If yes, attach meeting notes noting when site visit took place, who attended, etc.)*

Is the project in an approved plan such as a General Management Plan or an Implementation Plan with an accompanying environmental document? ☐ Yes ☐ No

If so, plan name _____

Is the project still consistent with the approved plan? ☐Yes ☐ No *(If no, prepare plan/EA orEIS.)*

Is the environmental document accurate and up-to-date? ☐ Yes ☐ No *(If no, prepare plan/EA orEIS)*

FONSI ☐ ROD ☐ *(Check one)* Date approved _____

Are there any interested or affected agencies or parties? ☐ Yes ☐ No
Did you make a diligent effort to contact them? ☐ Yes ☐ No

Has consultation with all affected agencies or tribes been completed? ☐ Yes ☐ No
(If so, attach additional pages detailing the consultation, including the name, the dates, and a summary of comments from other agencies or tribal contacts.)

Are there any connected, cumulative, or similar actions as part of the proposed action? ☐ Yes ☐ No
(If so, attach additional pages detailing the other actions.)

F. INSTRUCTIONS FOR DETERMINING APPROPRIATE NEPA PATHWAY

First, always check DO-12, § 3.2, "Process to Follow" in determining whether the action is categorically excluded from additional NEPA analyses. Other sections within DO-12, including §2.9 and 2.10; §3.5; §4.5(G)(4) and (G)(5), and §5.4(F), should also be consulted in determining the appropriate NEPA pathway. Complete the following tasks:

conduct a site visit or ensure that staff is familiar with the site's specifics; consult with affected agencies, and/or tribes; and interested public and complete this environmental screening form.

If your action is described in DO-12 section 3.3, "CE's for Which No Formal Documentation is Necessary," follow the instructions indicated in that section. If your action is not described in DO-12, section 3.3, and IS described in section 3.4, AND you checked yes or identified "data needed to determine" impacts in any block in section D (Mandatory Criteria), this is an indication that there is potential for significant impacts to the human environment, therefore, you must prepare an EA or EIS or supply missing information to determine context, duration and intensity of impacts.

If your action is described in section 3.4 and NO is checked for all boxes in section D (Mandatory Criteria), BUT you have initially checked "yes" in section C (Resource Effects to Consider) during internal scoping, this means that the team should do additional analyses to determine the context, duration and intensity of effects. If the magnitude of effects is then determined to be at the negligible or minor level, then usually there is no potential for significant impacts, then an EA or EIS is not required. If, however, during internal scoping and further investigation, resource effects still remain unknown, or are at the minor to moderate level of intensity, and the potential for significant impacts may be likely, an EA or EIS is required.

In all cases, data collected to determine the appropriate NEPA pathway must be included in the administrative record.

G. INTERDISCIPLINARY TEAM SIGNATORY *(All interdisciplinary team members must sign.)*

By signing this form, you affirm the following: you have either completed a site visit or are familiar with the specifics of the site; you have consulted with affected agencies and tribes; and you, to the best of your knowledge, have answered the questions posed in the checklist correctly.

Interdisciplinary Team Leader Name	Field of Expertise	Date Signed
Technical Specialists' Names		

H. *This section may be filled out either as the project progresses or when environmental documentation is complete.*

National Environmental Policy Act Data entered by: _____
(*Choose one and fill in blanks*)

☐ CE *Complete sections A-F before checking this box.* _____ CE Citation (from 3-4 of DO-12)

☐ EA Public scoping date _____ IMR Review date _____
EA release to public _____ FONSI date _____

☐ EIS NOI in FR _____ NOA for DEIS _____
NOA for FEIS _____ ROD date _____

Will the EA/EIS be used as the §106 compliance document? ☐ Yes ☐ No If yes, you must notify *in advance* the SHPO/THPO and ACHP of your intent to do so. Date notified _____

National Historic Preservation Act Data entered by: _____

Has the area been surveyed and NRHP resources identified? ☐ Yes ☐ No

Archeological resources affected? ☐ Yes ☐ No
Historic structures affected? ☐ Yes ☐ No
Cultural landscapes affected? ☐ Yes ☐ No
Ethnographic resources affected? ☐ Yes ☐ No (If yes, interested parties contacted? ☐ Yes ☐ No)

Choose one for determination of effect on NRHP properties:

☐ *No Historic Properties Affected* Date to SHPO/THPO _____

☐ *No Adverse Effect* ☐ Programmatic Exclusion (Exclusion # _____)

If using combined EA/AEF/ESF, date letter to SHPO/THPO declaring intention to use combined document. _____

Date AEF/ESF or combined EA/AEF to SHPO/THPO _____

Date response from SHPO/THPO _____

Date mitigation completed _____

☐ *Adverse Effect* If using combined EA/AEF, date letter to SHPO/THPO declaring intention to use combined document. _____

Date AEF/ESF or combined EA/AEF to SHPO/THPO _____

Date to ACHP, if required _____

MOA Date _____

Date mitigation completed _____

Endangered Species Act Data entered by: _____

Any threatened/endangered species in area? ☐ Yes ☐ No

If species in area ☐ No effect ☐ Not Likely to Adversely Affect ☐ Likely to Adversely Affect *(If checked, consider EIS)*

Date to FWS _____ Date FWS response _____

Floodplains/Wetlands/§404 Permits Data entered by: _____

Is project in 100- or 500-year floodplain, flash flood hazard area, or wetlands? ☐ Yes ☐ No *If yes, statement of findings approval date* _____

404 permit needed? ☐ Yes ☐ No Date _____

State 401 certification? ☐ Yes ☐ No Date _____
Note: If 404 permit needed, so is 401 permit

State Water Quality permit? ☐ Yes ☐ No Date _____

Tribal Water Quality permit? ☐ Yes ☐ No Date _____

CZM Consistency determination needed? ☐ Yes ☐ No Date _____

Other Permits/Laws

Data entered by: _____

Consistent with Wilderness Act ☐ Yes ☐ No ☐ N/A Date _____

Wilderness minimum requirement (tool) decision needed? ☐ Yes ☐ No Date _____

Wild and scenic river concerns? ☐ Yes ☐ No Date _____

National Trails concerns? ☐ Yes ☐ No Date _____

Air Quality consult w/State? ☐ Yes ☐ No ☐ N/A Date _____

Consistent w/Architectural Barriers, Rehabilitation, and Americans with Disabilities Acts? ☐ Yes ☐ No ☐ N/A Date _____

Other _____ ☐ Yes ☐ No Date _____

I. MITIGATING MEASURES TO BE INCLUDED IN PROJECT:
(Specify here or attach appropriate pages from EA, EIS, FONSI, MOA, or ROD)

J. SUPERVISORY SIGNATORY
Based on the environmental impact information contained in the statutory compliance file and in this environmental screening form, environmental documentation for the subject project is complete. If the project involves hot topics or sensitive issues, I have briefed the deputy or regional director.

Recommended:

Compliance Specialist	Telephone Number	Date

Approved:

Superintendent	Telephone Number	Date

SHPO Concurrence (***NM only***)**:**

Name/Title	Telephone Number	Date

12.2 Template for Plague Outbreak and Treatment in Prairie Dog Colonies Standard Operating Procedure (SOP)

TEMPLATE FOR PARK S.O.P.

The following is an example from Bryce Canyon National Park. Modify as needed for your use.

PLAGUE OUTBREAK AND TREATMENT IN PRAIRIE DOG COLONIES STANDARD OPERATING PROCEDURE (UPDATED XX-XX-XX)

This document identifies standard operating procedures for treatment of prairie dog colonies with pesticide when outbreaks of sylvatic plague occur. Also outlined in this document are procedures to follow for the collection of biological samples, notification of cooperating agencies, and protocols for posting warning and closure signs.

I. Background
II. Purpose and Need
III. Monitoring Prairie Dog [or other species] Population and Occurrence of Fleas that Inhabit Prairie Dog Burrows
- A. Prairie Dog Monitoring
- B. Flea Monitoring

IV. If an Outbreak of Plague is Suspected: Collecting, Handling, and Shipping Prairie Dogs
- A. Prairie Dog Sample Collection
- B. Flea Sample Collection
- C. Burrow Dusting Protocol
- D. Communication Protocol

V. Detection Determination
- A. If Plague IS NOT Detected
- B. If Plague IS Detected

I. Background

II. Purpose and Need

The most recent plague detections in [Park Name] prairie dog population occurred [ ]. This plague outbreak action plan will outline steps that should be followed and contacts that should be made both within the park and with other agencies should plague be detected in [Park Name]. Prompt and proper action will be necessary to prevent the spread of this disease to protect not only human health, but also the population of prairie dogs within the park.

III. Monitoring the Prairie Dog Population and Occurrence of Fleas That Inhabit Prairie Dog Burrows

A. Prairie Dog Monitoring

Currently, [Park Name] has a plan in place for the weekly monitoring of the prairie dog populations throughout the park. ***Any die-offs of prairie dogs or observations of prairie dogs acting abnormally are to be reported immediately to park Resource Management staff or a protection ranger (see communication flow chart).*** That Resource

Management or Law Enforcement employee should then notify the Superintendent of the possibility of a plague outbreak. Park Resource Management staff will then proceed to the area of the suspected outbreak, collect any available dead prairie dogs **(see below for details on how to safely collect suspected plague-killed prairie dogs),** and send them to a laboratory for testing to see whether plague was the cause of death.

B. Flea Monitoring

Flea sampling will not occur on a regular basis as does prairie dog monitoring. When a suspected plague outbreak occurs, any dead prairie dogs will be collected as well as fleas in the area of the burrow. These samples will then be shipped to a laboratory for testing to see if they are carrying the plague or not **(see below for details on how to safely collect and store collected fleas).**

IV. If an Outbreak is Suspected: Collecting, Handling, and Shipping Prairie Dogs and Fleas

A. Prairie Dog Sample Collection

If plague is suspected in the deaths of large numbers of prairie dogs, it is imperative to collect as many viable specimens (whole animals) as possible and ship them to a laboratory for analysis and clarification as to what actually killed the animals. Shipments should be kept to 12-15 dead prairie dogs at one time. If more than 12-15 dead prairie dogs are collected at one time, keep the remaining dead animals in a freezer to be shipped at a later time.

Materials you will need:

Data sheets	Gloves
Large ziplock freezer bags	Long sleeve shirt
Labels	Long Pants
Crush-proof shipping box	Insect repellent (with DEET)

As per Dr. Ken Gage of the CDC, this is the absolute minimum personal protective equipment (PPE) that you will need to wear. However, if you feel safer wearing more PPE than suggested here (i.e., face mask, goggles, Tyvek suit, etc.), that is acceptable and encouraged.

When going to collect dead prairie dogs:

1. Apply an insect repellent that contains DEET to your exposed skin and clothes. This will prevent fleas that may jump off of the dead animal from remaining on you and potentially transmitting the plague virus to you when they make their next blood meal (i.e., YOU!). You can also use a pyrethrin-containing powder (Deltamethrin) or permethrin-containing spray, if available to dust or spray your clothes; this will also keep fleas off of your body **(see attached MSDS sheet under Tab 5 for Deltamethrin information).** If you are collecting fleas, be careful to keep the powder (Deltamethrin) off of the collection rag because synthetic pyrethroids have repellent as well as insecticidal activity.

2. Next, take one of the large ziplock freezer bags, turn it inside out, and insert your gloved hand into the bag. Grab the dead prairie dog and pull your arm out of the bag while still holding on to the dog, thus pulling it into the freezer bag without having to touch it with your gloved hand. Zip the bag closed and place inside a second ziplock bag.

3. Label the bag with a carcass number, location of collection, date of collection, estimated date of death, if exact date is unknown (if date is known then put that date here), county, state, and name of collector. Also include this information on a **prairie dog mortality data sheet**. This will assist in matching up the results in the unlikely event of a mix-up at the testing facility or in the packaging of the animals.

4. As soon as possible after collection and bagging, freeze the animal in the Resource Management Freezer located [ ].

To prepare prairie dogs for shipping:

1. Select a biological sample shipping box that is constructed of heavy duty, crush-proof cardboard and has a solid Styrofoam container inside. These are stored [Location]. Be sure that all frozen prairie dog bags still have the labels attached, and place them in the shipping container with dry ice to keep them frozen; unfrozen animals should be shipped with ice packs (never "wet ice").

2. Next, make copies of the data sheets. Put the originals in an envelope inside a ziplock bag, and place in the shipping box on top of the bagged animals.

3. Follow additional packing instructions found at:

4. Before shipping the samples, call the laboratory or the BRMD to make sure they are aware of the shipment.

5. Ship the package overnight to the cooperating lab or to BRMD:

NPS-BRMD Wildlife Health Program
1201 Oakridge Dr.
Suite 200
Ft. Collins, CO 80525

B. Flea Sample Collection

If plague is suspected in the deaths of large numbers of prairie dogs, it is imperative to collect as many viable flea specimens as possible and ship them to a cooperating laboratory for plague testing.

Materials you will need:

Insect repellent (with DEET)	Collection vials
Plumber snake	Saline
White cloth	Collection forms
Large ziplock freezer bags	Long sleeve shirt
Tweezers	Long Pants
Gloves	Crush-proof shipping box

Tyvek suit is also acceptable PPE for keeping fleas off of your body

When going to collect fleas:

1. Apply an insect repellent that contains DEET to your exposed skin and clothes. This will prevent fleas that may jump off of a dead prairie dog from remaining on you and potentially transmitting the plague virus to you when they make their next blood meal (i.e., YOU!). You can also use a pyrethrin-containing powder (Deltamethrin), or permethrin-containing spray, if available to dust or spray your clothes; this will also keep fleas off of your body. HOWEVER, be careful to keep the powder (Deltamethrin) off of the collection rag so as not to repel or kill the fleas you are collecting in this manner. Synthetic pyrethroids have repellent as well as insecticidal activity

2. Next, make sure to sample from burrows that appear active, or where dead prairie dogs have been collected/observed. Attach the cloth to the alligator clip on the end of the plumber's snake. Try to get the cloth into

the burrow as far as possible and allow it to remain in the burrow for about one minute. Have a large ziplock freezer bag ready. Pull the cloth out, immediately place it in the bag, and secure the top with your gloved hand.

3. Holding the bag closed, squeeze the alligator clip to release the cloth. Pull the cable from the bag and zip the closure on the bag to close it. Repeat this procedure with different cloths and bags at as many burrows as seems prudent based on the extent of die-off observed. It is imperative to get as representative a sample as possible, instead of sampling from only a few burrows in the affected colony. Be sure to label the bags with the location of collection, date of collection, name of collector, county and state in which they were collected.

4. When done collecting the fleas, place the bags in a freezer [Location] to kill the fleas.

To prepare the fleas for shipping:

After 24–48 hours in the freezer, the fleas should be dead, and they can be sorted. You will need: a metal tray or some other light-colored background to work on, tweezers, collection vials, and saline. Fill out the **Flea Collection Form** as you go along. This is the same data sheet you use to fill out when collecting dead prairie dogs. The data sheet has spaces for date collected, date picked, and columns for each location where they were collected.

1. Starting with a bag collected at one location, empty the bag onto your tray or light-colored background, ensuring that every bit of dirt, etc., comes out of the bag.

2. Pick up the cloth and carefully examine it for fleas on both sides. Fleas are usually 1/16–1/8" long, and brownish-red in color, with long back legs. They often look shiny. Run your fingers down the cloth to brush off any debris clinging to it, and put it back in its bag.

3. Now, examine the tray to see what fell off the cloth or out of the bag. Often, most of the fleas are found here in the tray. If fleas are found, pick them up with the tweezers and place them in a vial. Label the vial with the same location as was on the bag.

4. Empty the tray between each sample rag.

5. For each sample, write on the data sheet the number of fleas found, even if it is zero.

6. Once all the bags have been gone through, fill each vial with saline to preserve the fleas. Cap them tightly and place duct tape or electrical tape around the lids to ensure a tight seal.

7. Wrap the taped vials in bubble wrap and place in a crush-proof box with blue ice or dry ice and absorbent material in case the vial breaks or leaks.

8. Select a biological sample shipping box that is constructed of heavy duty, crush-proof cardboard and has a solid Styrofoam container inside. These are stored [Location]. Be sure that all frozen prairie dog bags still have the labels attached, and place them in the shipping container with dry ice to keep them frozen; unfrozen animals should be shipped with ice packs (never “wet ice”).

9. Next, make copies of the data sheets. Put the originals in an envelope inside a ziplock bag, and place in the shipping box on top of the bagged animals.

10. Follow additional packing instructions found at:

11. Before shipping the samples, call the laboratory or the BRMD to make sure they are aware of the shipment.

12. Ship the package overnight to the cooperating lab or to BRMD:

NPS-BRMD Wildlife Health Program
1201 Oakridge Dr.
Suite 200
Ft. Collins, CO 80525

C. Burrow Dusting Protocol

The use of pesticides within NPS units is governed by the Pesticide Use Proposal System, so potential users must contact their park or regional IPM coordinator for approval.

If plague is suspected in the deaths of the prairie dogs, then Resource Management staff will immediately start treatment of affected burrows before any test results come back, but after sample collection is completed. As per DO12 handbook Chapter 3 Section 4 E (3), which states, *Removal of individual members of a non-threatened /endangered species or population of pests and exotic plants that pose an imminent danger to visitors or an immediate threat to park resources*, we have the authority to act in such a manner. The following protocol should be followed.

Notify the NPS Wildlife and Public Health Program contacts.

Until we hear back from the NPS Wildlife and Public Health Program contacts, Resource Management staff should dust the affected colony (where dead prairie dogs were found) with insecticidal dust (Deltamethrin).

A store of bubonic plague warning or closure signs and Deltamethrin for controlling fleas will be kept available [Location]. Personal protective equipment, including respirators, goggles, disposable coveralls, and gloves, is located in the resource management garage in the historic cabin area. Disposable face shields should be used for each dusting event. It is also important to wear appropriate PPE (pants, long sleeve shirts, chemical resistant gloves, eye protection) when applying the Deltamethrin. Care should be taken when applying the insecticide on windy days to prevent the dust from blowing toward the person applying it.

A job hazard analysis for burrow dusting is located [Location].

D. Communication Protocol

All research and other human activity in close proximity to the affected burrows should be immediately suspended until the extent of the outbreak is defined and treatment efforts have been completed.

Until we hear back from the NPS Wildlife and Public Health offices, Law Enforcement staff should mark the affected area with warning/closure signs, which are stored [Location].

A **draft press release** is located below [ ] for disseminating information to the public and NPS and concession employees. Simply fill in the dates, which colony is affected, and what closures, if any, have been enacted. This information should be communicated to the Chief of Interpretation for release to the media. A briefing statement to provide information on the location of the outbreak, actions we are implementing, and the phone number of the Public Health Department should be included in the release. The park's Public Information Officer (PIO) should develop this briefing with input from Resource Management staff. The PIO will handle dissemination of information to the media. It is important to get the information out to the public early, before rumors start to develop.

A **copy of the warning/closure signs** should be distributed to interpretive staff at the visitor center (VC), along with information pertaining to the event so it can be passed on to visitors who may only stop at the VC. If the outbreak takes place near public use areas (e.g. housing areas, trails, or campgrounds), contact the on-duty law enforcement ranger and arrange for a meeting with campground hosts and residents. They should be briefed in order to provide the public and campers with accurate information, including recommendations to stay out of the colonies and to keep their pets inside or on leashes away from prairie dogs. Information regarding other animals that can also harbor the plague organism

should also be disseminated to the public so visitors do not contract plague while attempting to feed ground squirrels, chipmunks, or other rodents. **The intent of this meeting should be to inform, NOT to alarm people.** If park visitors follow the recommended precautions, their plague risk should be extremely low.

Law Enforcement personnel should be notified to strictly enforce the leash law so as to prevent spreading the disease to domestic pets and then to humans.

Signs should be posted at campground entrances, at all loop entrances, and at the entrances of both men's and women's restrooms.

V. Detection Determination

A. If Plague is NOT Detected

If plague is not detected, your efforts have not been in vain. If the samples were sent to the CDC and state health laboratories, the only other likely possibility would be tularemia because plague and tularemia tests would be the only tests conducted. If further testing is needed, the samples might need to be sent to a wildlife disease laboratory. If die-off is still occurring, it would be advisable to continue to collect prairie dog and fleas samples and ship them to the lab or BRMD in case the plague organism was simply not detected in the previous samples, or in order to determine what is actually killing the prairie dogs.

b. If Plague IS Detected

If the lab report comes back with positive results for plague, then immediately notify the Superintendent, all Division Chiefs, and call NPS Wildlife and Public Health contacts, who may conduct a site visit, and make recommendations as to potential closures in conjunction with the Superintendent and Resource Management staff, depending on where the colony is located relative to human activity. They will also make recommendations regarding whether or not it is necessary to dust the burrows with Deltamethrin. It may be necessary to issue a press release to describe the situation and explain why closures are going into effect.

The continued monitoring of the colonies in and around the park for further prairie dog die-offs will be crucial in determining the spread, or lack thereof, of the plague outbreak. If animals in other colonies begin to die off then sampling should be conducted to verify if plague is the causative agent or not.

The outbreak will be considered contained when no more prairie dog die-off is observed and when the park receives direction to lift restrictions from the Wildlife and Public Health offices. At that time, all closed or restricted access areas will be reopened, closure signs will be removed and replaced with warning signs, and Resource Management personnel will continue to monitor the affected colony for prairie dog die-off. Warning signs should only be removed after consultation among park staff and NPS Wildlife Health and Public Health personnel, and re-initiation of any suspended activities in the area of the affected colony will only occur after such consultation.

12.3 Example of Contact Information and a Flow Chart for Wind Cave (WICA)

Contact Information for below Flow Chart:

WHP: Dr. Kevin Castle; 970-219-0104

OPH: Dr. David Wong; 202-538-9969

Mr. Adam Kramer; 202-641-0013

Plague Contact Flow Chart Example: WICA

NPS employee observes or receives report of prairie dog die-off or multiple prairie dogs observed dead or acting abnormally.

Person who observed or received report notifies Resource Management staff and/or Law Enforcement ranger of this event.

Law Enforcement

Notify Superintendent and Resource Management staff of potential plague outbreak.

Resource Management

Notify Superintendent and Law Enforcement staff of potential plague outbreak. Notify BRMD Wildlife Health Program (WHP) that we have a potential wildlife plague outbreak and prepare to collect samples if so advised. WHP will contact NPS Office of Public Health (OPH).

Respond to site and collect any visible dead Prairie dogs and fleas from affected burrows.

Submit samples/carcasses as recommended by NPS WHP.

If confirmed or highly suspicious, notify SD Dept. of Health and Custer State Park, of potential plague outbreak. Notify Regional Office. The WHP or OPH will maintain contact with other federal agencies. Request NPS IPM approval for Delta dust application if not already approved.

Conduct all-employee briefing so that all employees are informed and have good information to pass along to visitors.

Post closure or warning signs around the affected colony. Suspend all research and other human activities in close proximity to the outbreak until the extent of the outbreak is defined and treatment efforts are completed.

Until we hear back from WHP or OPH, proceed with the dusting of affected burrows with DeltaDust, or other recommended management actions

If Plague is **NOT** detected, keep warning signs posted around colony and keep collecting dead animals and fleas until a cause of death can be determined. Removal of any closures will be with the mutual consent of the OPH and WHP.

Continue monitoring.

If plague **IS** detected, immediately:
(1) Notify Superintendent, and all Division Chiefs Recommendations will be made as to whether continued dusting is necessary, which areas to close, and which areas to re-open, if necessary.
(2) Finalize draft press release and distribute to appropriate agencies/contacts.
(3) Notify MWRO, SD Dept. of Health, Custer State Park.

Discussions with OPH and WHP may trigger deployment of the Disease Outbreak Investigation Team.

Continue monitoring colonies throughout the park and treating those affected with DeltaDust as directed.

Conduct After Action Review to assess actions and identify areas to improve.

Interpretation

Produce handout for the visitor center staff to provide to visitors (if test results come back positive for plague outbreak). Also, notify interpretation staff if an outbreak occurs so they can place information on the Travel Information Station

Access Draft Press Release. Fill in appropriate information and prepare to send out, pending plague test results or advisement from OPH and WHP.

12.4 Plague and Flea Monitoring Methods

PLAGUE AND FLEA MONITORING METHODS

(Information in this appendix is from: Western Association of Fish and Wildlife Agencies. 2006. Draft Gunnison's prairie dog conservation plan: addendum to the white-tailed and Gunnison's prairie dog conservation strategy. Western Association of Fish and Wildlife Agencies. Laramie, Wyoming. Unpublished Report. 41 pp.)

Plague Monitoring Protocol

Plague epizootics may originate from focal areas, with possible maintenance in non-focal areas between epizootics. During epizootics, plague can spread over great distances and in the process affect humans, most often during and shortly following epizootics (Cully 1993). Detection and monitoring of plague at NPS units may be accomplished using a variety of methods, as summarized below.

Technique	**Description**
"Windshield surveys"	General observations of prairie dog towns to detect die-offs, with follow-up evaluations needed to confirm cause and status.
Collection and analysis of dead prairie dogs	Prairie dogs often die in burrows, but a small percentage of those exposed to plague die above-ground and can be picked up if colonies are regularly surveyed for dead and dying prairie dogs
Collection and analysis of fleas from prairie dog burrows (= burrow "swabbing")	This technique has had widespread use as a surveillance technique for human health concerns.
Collection of blood samples from members of Order Carnivora (= serosurveys)	Although such species as badgers and coyotes can become infected with plague, their primary role in the disease cycle is the transport of plague-infected fleas (Poland and Barnes 1979 cited in Gage et al. 1994). Nobuto blood-sampling papers have been used extensively, since the technique does not require access to refrigerators and requires only 0.2 ml of blood (Wolff and Hudson 1974, Gage et al. 1994). This technique has recently been used in association with black-footed ferret reintroduction, either via collection of blood samples from live animals, dead animals collected for this purpose, or animals killed during animal damage control activities (Anderson et al. Undated, Williams et al. 1998, Matchett 2001). In addition, black-footed ferrets captured for removal of radio collars, for implantation of transponder chips, or for canine distemper vaccination can be bled for disease analysis samples.
Collection of blood samples from domestic dogs	Barnes (1982) reported using domestic dogs as sentinels for exhibiting antibodies to plague. This technique has been effective on Native American reservations in the Southwest to detect seroconversion before plague was observed in rodents or humans.
Collection of blood from	Certain rodent species appear to be resistant to plague and may

Technique	Description
potentially resistant small mammals	serve as maintenance or enzootic hosts that maintain plague between epizootics (Cully 1993, Gage et al. 1994). The Wyoming Game and Fish Department has monitored small mammals for plague seroconversion in Shirley Basin, Wyoming (Luce et al. 1994, 1996, 1997). Trapping efforts focused on deer mice and grasshopper mice, with the assumption that active plague would be detectable by antibodies produced during the short life span of these rodents. These investigations detected a relationship between seroprevalence of plague in deer and grasshopper mice and status of white-tailed prairie dog populations in Shirley Basin.

ACTIONS:

1. Staff initiates a public information program to inform adjacent landowners, visitors, and other members of the public concerning the need to notify the agency of die-offs of prairie dogs or ground squirrels.

2. Staff informs state wildlife management agency personnel, adjacent land management agencies, local veterinarians, and other government personnel that deal with animal control, or have regular contact with landowners and the public, of the need for reporting die-offs.

3. Staff may provide, as needed, information for state wildlife management agency personnel, adjacent land management agencies, local veterinarians, and other government personnel that deal with animal control, on protocols for collection of dead prairie dogs and ground squirrels, packaging and record keeping.

4. Staff will develop windshield survey routes throughout the prairie dog habitat to be conducted on a routine basis (e.g. biweekly, monthly, annually) by staff where prairie dogs occur, particularly during March and April. Windshield surveys will follow the CDC protocol (attached). Significant decline in any colony or complex should be immediately reported to the park resource manager.

   In the event of a suspected die-off (if a windshield survey route reports a significant loss of prairie dogs or ground squirrels), the staff will implement the plague contingency plan immediately (attached).

   A. Make inquiries to determine whether or not the colony was poisoned, or whether mortalities were due to shooting.

   B. If neither shooting nor poisoning occurred, the colony or complex should be searched for prairie dog and ground squirrel carcasses as soon as possible after discovery of the population decline. Carcasses should be handled in the field according to protocol (attached).

C. In the event that carcasses cannot be found, and the disappearance of prairie dogs is verified as recent, burrow swabbing should be conducted to collect fleas according to CDC protocol (attached).

6. If plague is verified, the resource manager, in cooperation with NPS Wildlife Health Program and Office of Public Health (One Health) officials and should immediately notify, and make plague contingency recommendations to, the following: landowners and wildlife agency personnel in the affected area, state Department of Agriculture, USDA-Wildlife Services, NRCS, veterinarians, and local government personnel that deal with animal control, and the general public through local media sources. NPS public health and wildlife health personnel should be consulted on the need for insecticide treatment at this time to control fleas in burrows, and therein reduce the potential for further plague infections.
7. Post-plague monitoring of prairie dog colonies should be conducted throughout the year to document the rate of re-colonization and verify occupied acreage. Initial monitoring, which will take place from one to several years, should consist of windshield surveys. When visual surveys indicate prairie dog colonies are recovering, a quantitative survey method should be initiated. The recommended method, due to widespread use, particularly on black-footed ferret reintroduction sites, is the Biggins transect method (Biggins et. al. 1993) that equates active and inactive burrow densities to population density.

8. The resource manager and staff should evaluate the extent of the impact of the epizootic as it affects the acreage and distribution objectives in existing prairie dog and, if applicable, black-footed ferret management plans. The group should determine whether or not there is a need to modify prairie dog or ferret management in the plague area, and potentially elsewhere in the park, if occupied acreage is below the objectives in the management plan.

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Procedure for Visual Evaluation of Prairie Dog Colonies for Plague

Source: Enscore, R. CDC, NCID, Division of Vector Borne Infectious Diseases, Plague Section, Fort Collins, Colorado.

<u>HEALTHY COLONY</u>

OBSERVATION: The vast majority of burrows show signs of recent use, unless it has rained within the past 24 hours – in which case the colony should be reexamined following a period of at least 24 hours without precipitation. Active prairie dogs are observed during periods of acceptable weather conditions. Only a relatively few (<10%) burrow openings appear inactive (lack of disturbed soil, presence of cobwebs or wind-blown vegetation over the entrance). An occasional carcass or dried bones may be present as a result of non-plague death or predation.

EVALUATION: Unless recently (days) introduced, plague is not likely to be present. Fleas are not likely to test positive.

SAMPLE RECOMMENDATIONS: No samples recommended.

<u>DEAD COLONY</u>

OBSERVATION: The colony appears completely inactive. Burrows show no signs of recent use (re-examine if it has rained within 24 hours). An occasional desiccated carcass and bones may be present, and have likely been scavenged.
EVALUATION: 1) Make inquiries to determine if the colony was poisoned. This is especially likely if it appears that dirt was shoveled into the burrows. If there is no evidence of poisoning and the food supply appears ample: 2) it is likely that plague or some other zoonotic disease killed the colony. An experienced observer can usually make an estimate (recently, 1 season, or 2 seasons) on how long the colony has been inactive by considering the soil type and degree of burrow degeneration.
SAMPLE RECOMMENDATIONS: Sample only if there is no evidence of poisoning. A recent (same season) die-off might produce many fleas through burrow swabbing. Older die-offs will likely produce few or no fleas. Typically, many burrows (dozens or even hundreds) may be swabbed with only a few producing flees. If burrowing owls are using the inactive burrows, small black stick-tight fleas may be present in large numbers (in contrast to the larger, reddish-brown prairie dog fleas). Fresh or desiccated prairie dog carcasses may also be collected for analysis.
SCATTER PATTERN:
OBSERVATION: Inactive burrows constitute an unusually high (typically 20-90%) percentage of the total burrows. Active burrows however are clearly evident and active prairie dogs are observed during periods of acceptable weather. Active and inactive burrows are scattered amongst each other in no particular pattern (see below), keeping in mind that family units may have multiple burrow openings and hence an inactive unit may produce a small cluster of 2-5 inactive burrow openings. An occasional carcass (fresh or desiccated) and bones may be present.

EVALUATION: Several scenarios could account for these observations – and more than one scenario may be in play at the same place and time. Presented in order of likelihood: 1) make inquiries to determine if the colony was poisoned. This is especially likely if it appears that dirt was shoveled into the burrows. This scatter pattern could be produced if the application of poison was scattered and not comprehensive, 2) if there is no evidence of poisoning, assess the available food supply. Such a pattern of death could also be attributable to a population crash as a result of lost carrying capacity of the site or over-population, 3) if there is no evidence of poisoning or population crash, hunting by humans or excessive predation by carnivores or birds of prey are highly likely. Human hunting usually produces physical evidence such as footprints, tire tracks and spent ammunition shells. Depending upon the local culture, human hunters may collect their prey (many Native American groups regard prairie dogs as a delicacy) or leave it for scavengers. Experienced observers can often spot carnivore tracks and recognize hunting and attack patterns in these tracks near burrow entrances, 4) finally, a zoonotic disease could be responsible, but given this mortality pattern, a disease with a lower mortality rate than plague is more likely.
SAMPLE RECOMMENDATIONS: If there is no evidence of poisoning, population crash, or excessive human hunting: collect fleas by swabbing burrows – especially inactive burrows – and collect fresh or desiccated prairie dog carcasses if available.
DEAD ZONE
OBSERVATION: Within an otherwise healthy appearing colony, there is a zone of inactive burrows. This zone may encompass a relatively small or large proportion of the colony, and may be located anywhere in the colony. Eventually it spreads to encompass a section of the colony and appears to be spreading, along a discernable line of demarcation, over the remaining section of the colony. Experienced observers can often clearly distinguish and mark (flagging tape) this

demarcation line between active and inactive regions. Marking allows for periodic re-examination to assess the rate of spread and facilitates sampling. Fresh or desiccated carcasses may be present. Near the demarcation line, recently inactive burrows may reveal the odor of decaying carcasses and flies may be common at burrow entrances.

EVALUATION: 1) There is a high probability that plague is active in such a colony. Although other zoonotic diseases are possible, plague is most likely. 2) Depending upon the location of the dead zone with respect to other human activity (homes, barns, etc.) poisoning is also a possibility and should be investigated.

SAMPLE RECOMMENDATIONS: Collect fleas by swabbing burrows immediately along both sides of the demarcation line, concentrating a majority of your efforts immediately along (within 10 meters) the inactive (dead) side of the line. Fleas are likely to be numerous. You may wish to apply extra insect repellent but be extremely cautious not to directly or indirectly get repellent on your burrow swab. (If this happens: discard it, wash your hands, and start with a new one). If others in a group are getting fleas and you are not, and you are swabbing essentially the same area, you likely have repellent on your swab. Collect any available rodent carcasses (fresh or desiccated, prairie dog or other rodent) for testing.

Additional Notes: Include GPS coordinates for all samples. One set of coordinates per colony is acceptable. Specify the type of inactivity pattern noted for each sampled colony: dead colony, scatter pattern, dead zone. Analysis of samples from "dead zone colonies" will receive laboratory priority.

The above activity patterns are typical for the warm months. Visual examination during winter months is more difficult due to decreased daily activity among even healthy animals.

Field Procedures for Collecting and Handling Carcasses as Diagnostic Specimens

1. Search prairie dog colonies systematically using walking or 4-wheeler transects spaced at about 50 meters.
2. When a carcass is discovered, ascertain, if possible, whether or not the animal was shot. If mortality by shooting is confirmed, there is no need to collect the specimen.
3. Before you collect a carcass, prepare a tag with the following information: species, date, location (both legal description and UTM is recommended), name of collector, agency or affiliation of collector, telephone number and address of collector, and a brief description of circumstances for collection.
4. When collecting a carcass, the collector should wear leather or latex gloves, and a long sleeved shirt or jacket that is tight at the wrist, to ward off fleas.
5. Invert a one-gallon plastic ziplock freezer bag over your hand, grasp the carcass in your hand, quickly fold the bag over the carcass, roll the bag on the ground, away from your body, to expel the air, and seal the ziplock.
6. Immediately place in a second ziplock bag, put in the tag, roll and seal the second bag.
7. As soon as possible after collection, freeze the specimen.
8. Sample Size:
   A) If specimens are from a single sample area (one prairie dog colony or area), collect as many specimens as is practical up to 15, but initially ship only the freshest five specimens to the diagnostic lab.
   B) Freeze the additional specimens that were collected, up to ten, and save for further testing needs, depending upon the results from the testing of the first five specimens. Keep the samples until notified by the lab that results were obtained from the first five samples and that the additional specimens will not be needed.

9. Shipping Carcasses
   A) Carcasses must be individually labeled and bagged in watertight bags (minimum triple bag in ziplock bags).
   B) Placement of absorbent packing material around the carcass (crumpled newspaper, etc.).
   C) Use of approved laboratory shippers or hard-sided containers, adequately taped closed.
   E) Carcasses should be frozen or packed with frozen ice packs (no wet ice).

Procedure for Flagging (Swabbing) Rodent Burrows

Source: Gage, K. Centers for Disease Control, Ft. Collins, CO.

Some important flea vectors of plague infest rodent species that live in burrows. Although these fleas usually can be found in abundance on live hosts, they also can be collected by a procedure known as burrow flagging or burrow swabbing.

This procedure requires:

1) Burrow swabbing device consisting of a flexible cable, wire, or strong rubber hose with spring-loaded clip attached to the end. We prefer a steel plumber's "snake" that has an alligator clip attached to (screwed onto) the end as a means of attaching the flag. A simple burrow swab can be made by attaching a flag to the end of a piece of wire (about the thickness of a coat hanger), but this primitive swab allows only the top 2 or 3 feet of a burrow to be swabbed and will miss

some fleas. Despite the shortcomings of the latter technique, it can be useful when die-offs are encountered unexpectedly and more sophisticated means of swabbing fleas are not available.

2) Flags consisting of white flannel cloth squares (approx. 25 cm^2 or 10 in^2). We prefer white flannel because it is easier to see the fleas on white cloth than on cloth of other colors. Flannel is better than most other cloth because of its deep nap, which increases the likelihood that fleas will continue to cling to the cloth flag after it is removed from the burrow.
3) Plastic bags (approx. 20-40 cm^2 or 8-15 inches) (ziplock type are best)
4) Insect repellent (DEET) to spray on clothes and exposed skin on arms, legs, etc. Although this is recommended for safety reasons, care must be taken not to apply repellents to hands because the repellent is likely to transfer to the flagging material, thus preventing fleas from jumping onto the flag. Note: clothing also can be treated with permethrin-containing sprays but these sprays should not be applied directly to the skin.

Procedure:

1. Attach a flag to the clip on the end of the burrow swab.
2. Slowly insert the flag as far as possible down the burrow. The fleas confuse the flag with their normal host and cling to it as it passes through the burrow.
3. Slowly withdraw the flag from the burrow after approximately 30 seconds.
4. Quickly place the flag in a plastic bag.
5. Seal the bag to prevent the fleas from escaping.
6. Keep track of the number of burrows swabbed so that a burrow index can be calculated.

Burrow index = no. fleas collected/no. burrows sampled - This value often increases dramatically during die-offs among prairie dogs, rock squirrels, California ground squirrels, or other ground squirrel species.

7. Place another flag on the swab and repeat steps 1-6 for each burrow.
8. Transport flags back to laboratory in the plastic bags. Keep the bags in a reasonably cool place to prevent desiccation of the flea samples (*Yersinia pestis* is very susceptible to death by desiccation) or death of the plague bacilli due to excessive heat (remember pick-up hoods can get very hot in direct sunlight; fried samples will come back negative for plague every time).
9. Place bags in a freezer overnight to kill the fleas.

****If the park does not have resources to separate fleas as described below, contact the Wildlife Health Program; in some instances the entire frozen bag, including flags and fleas, can be shipped frozen to Ft. Collins for separation. This should be determined before flagging burrows!**

10. Place the flags and loose contents of the plastic bags in a white enamel pan. Fleas may be picked from the flags and bottom of the pan with forceps.
11. Place fleas in vials containing 2% saline and a very small amount of Tween-80 detergent (<0.0001% of solution). Remember that the detergent is added to reduce surface tension and allow the fleas to sink to the bottom of the vial. Too much detergent will kill the plague bacteria and prevent successful isolation. Fleas can be submitted in 2% saline without Tween-80, but an effort should be made to submerge the fleas. If the fleas have been killed by freezing, this should not be a problem. Although not recommended for routine collecting, some investigators occasionally remove live fleas directly from the flags and place them in vials of saline. Live fleas placed in saline containing the Tween-80 detergent will be unable to float on the surface of the liquid, thus ensuring that they will drown soon after being placed in the saline. Without the detergent, surface tension can become a problem because the numerous bristles and setae found on fleas enable them to remain afloat on the surface of saline. This can be a potential safety problem because floating

fleas often survive shipment and arrive at the laboratory ready to jump onto lab personnel. Rapid freezing of the fleas obviously eliminates this problem, but adding Tween-80 to the saline also helps reduce the growth of fungi on flea samples. Dead fleas trapped in the surface tension at the air-saline interface rapidly become overgrown with fungi making identifications more difficult.

12. Vials containing 2% saline and fleas can be shipped for taxonomic identification and analysis of the fleas for *Yersinia pestis* infection. The fleas can be shipped at ambient temperature in the vials of 2% saline. For best results, ship the specimens as soon as possible because the fleas will start to decay soon after collection. Be sure and double wrap the vials in a leak-proof material and then place them in a crush-proof box or metal mailing tube for shipment.

FLEA COLLECTION FORM EXAMPLE

Prairie Dog Flea Flagging, and Picking Data Form

Date Collected: _____, Collected by:_____

Date Picked: _____, Picked by:_____

Date Samples Shipped to CDC:_____
Fed Ex Tracking Number:_____

Colony A		Colony B		Colony C	
Sample #	#of Fleas	Sample #	#of Fleas	Sample #	#of Fleas
1		1		1	
2		2		2	
3		3		3	
4		4		4	
5		5		5	
6		6		6	
7		7		7	
8		8		8	

12.5 Examples of Closure Signs

CAUTION

SYLVATIC PLAGUE HAS BEEN FOUND IN PARK PRAIRIE DOG POPULATIONS
PLEASE EXERCISE THE FOLLOWING PRECAUTIONS DURING YOUR VISIT:

- Stay out of all areas that have been CLOSED by order of the Superintendent.
- DO NOT go near or poke into any rodent holes or prairie dog burrows.
- DO NOT handle any wild animals. Avoid dead or sick animals.
- Pets are NOT allowed on the trails or in the backcountry.
- If you observe dead animals, contact a ranger or notify the visitor center as soon as possible.

What is Sylvatic Plague?

Plague is an acute infectious disease which primarily affects rodents, including prairie dogs. Plague can be passed to humans by wild rodents and by their fleas. The incubation period is usually 2-5 days but can be as short as 1 day or as long as 12 days.

What are the symptoms?

- Feeling sick all over.
- Sudden onset of fever.
- Headache, nausea, vomiting, diarrhea.
- Painful and swollen glands in the groin, armpits, and neck.

Plague is curable when treated in time.

Prompt diagnosis and treatment with antibiotics can stop the disease.

For more information, contact the park:
National Park Service
YOUR Park

CAMPGROUND/FACILITIES NOTICE

Prairie Dogs, chipmunks, ground squirrels and other wild rodents in this area may be infected with plague. Plague can be transmitted by animal contact or by fleas.

- Avoid all contact with prairie dogs, chipmunks, ground squirrels and other wild rodents.
- Do not feed or play with wild animals.
- Avoid fleas by protecting pets with flea collars and keeping pets on a leash and out of prairie dog colonies.
- See a physician if you become ill within one week of your visit to this area. Plague is a treatable disease.
- Do not touch sick or dead animals
- Apply insect repellents to reduce the risk of a flea bite

For further information contact:
National Park Service
YOUR Park

WARNING
AREA CLOSED
(Name of Area)

Sylvatic plague has been found in prairie dog populations in the (name of area) area. Please exercise the following precautions during your visit to the park:

- Stay out of areas that have been CLOSED by order of the superintendent
- DO NOT go near or poke anything into rodent holes or prairie dog burrows
- DO NOT handle any wild animals, living or dead

What is Sylvatic Plague?

Plague is an acute infectious disease which primarily affects rodents, including prairie dogs. Plague can be passed to humans by wild rodents and their fleas. The incubation period for the disease in humans is usually 2-5 days, but can range from 1 day to as long as 12 days.

What are the symptoms?

General flu-like symptoms, including headache, fever, and swollen glands in the groin, armpits, or neck.

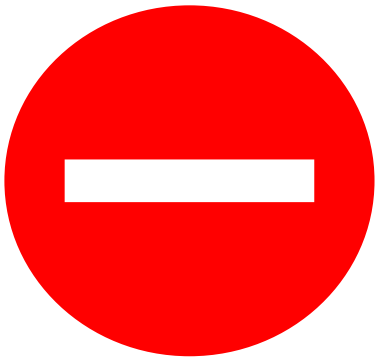
PLAGUE IS CURABLE WHEN TREATED IN TIME!

If you develop these symptoms within 7 days of possible exposure, notify your doctor.

(Map of closed area)

The area marked above is CLOSED until further notice.

IF YOU HAVE QUESTIONS, CONTACT A PARK RANGER OR CALL THE PARK AT:



12.6 Useful Links to Important Documents and Information Resources

NPS Wildlife Health Branch:
http://www1.nrintra.nps.gov/BRMD/Wildlife_Health_Management/Wildlife_Health/Wildlife_Health.cfm

NPS Office of Public Health
http://inside.nps.gov/waso/waso.cfm?prg=44&lv=2

NPS Office of Risk Management:
http://inside.nps.gov/waso/waso.cfm?prg=46&lv=2