

INVESTIGATION OF AND RESPONSE TO TWO PLAGUE CASES YOSEMITE NATIONAL PARK, CALIFORNIA, USA, 2015⁸

OVERVIEW

In August 2015, plague was diagnosed for two persons who had visited Yosemite National Park in California, USA. One case was septicemic and the other bubonic. Subsequent environmental investigation identified probable locations of exposure for each patient and evidence of epizootic plague in other areas of the park. Transmission of *Yersinia pestis* was detected by testing rodent serum, fleas, and rodent carcasses. The environmental investigation and whole-genome multilocus sequence typing of *Y. pestis* isolates from the patients and environmental samples indicated that the patients had been exposed in different locations and that at least two distinct strains of *Y. pestis* were circulating among vector—host populations in the area. Public education efforts and insecticide applications in select areas to control rodent fleas probably reduced the risk for plague transmission to park visitors and staff.

INTRODUCTION

Plague is a zoonotic disease caused by the gram-negative bacterium *Yersinia pestis*; the organism's reservoir is rodents, and the vectors are fleas [1,2]. Transmission to humans can occur through bites by infected fleas or through handling *Y. pestis*—infected rodents [1,2]. Epidemics of plague still occur on the continents of Africa, Asia, and North and South America [3]. Plague was introduced to California in 1900 [1,4–6] where over the next 25 years it caused occasional outbreaks in rats commensally residing with humans in urban areas [2,4,6]. Shortly after its introduction, *Y. pestis* moved into wild rodent populations, establishing a sylvatic transmission cycle [7,8]. In subsequent decades, plague spread across California and other western states [9] periodically affecting humans [4–6, 10–13].

The California ground squirrel plays a major role in human exposure in California because its predominant flea species *Oropsylla montana* is a competent *Y. pestis* vector [1,2] that is often abundant on this rodent and in its burrows [14] and will readily bite humans [1,11]. Since the 1980s, evidence of *Y. pestis* transmission in rodents in the Sierra Nevada mountains has been generally restricted to locations at elevations >1,200 meters (California Department of Public Health, unpub. data, 1983–2015). Despite ongoing sylvatic transmission, human plague remains rare in the western United States [15–17], including in California where no cases have been confirmed since 2006 [18,19].

During the summer of 2015, the Los Angeles County Department of Public Health (LAC DPH) and the Georgia Department of Public Health reported two cases of plague in persons who had recently travelled to Yosemite National Park (Yosemite). The California Department of Public Health (CDPH), in collaboration with the US Centers for Disease Control and Prevention (CDC) and the National Park Service (NPS), investigated the increased *Y. pestis* transmission in Yosemite. We summarize the epidemiologic, laboratory, environmental findings, and the public health response.

⁸ Full article published as: Danforth M, Novak M, Peterson J, et al. Investigation of and Response to 2 Plague Cases, Yosemite National Park, California, USA, 2015. Emerg Infect Dis. 2016 Dec; 22(12): 2045–2053.



RESULTS

Environmental Findings

Plague risk assessments were conducted for nine locations in Yosemite and the surrounding national forests visited by the patients. Within the park, eight more sites were also evaluated for *Y. pestis* transmission and potential risk areas for transmission to humans.

Flea Control

Sites with evidence of recent *Y. pestis* transmission and an increased risk for human exposure were temporarily closed, and rodent burrows were treated with insecticide to reduce flea populations and protect wildlife and human health. The following five areas in Yosemite were identified for insecticide treatments: Crane Flat Campground, Glacier Point, Tuolumne Meadows Campground, Tamarack Flat Campground, and the Crane Flat–NatureBridge campus. In total, 16.3 kg of 0.05% deltamethrin was used per label instructions to treat an estimated 3,700 rodent burrows. Although time and logistical constraints precluded pre- and post-treatment flea evaluations at all locations, evidence from limited sampling suggested that the insecticide applications reduced the local flea populations.

Public Outreach

To further reduce the plague risk for Yosemite visitors and staff, NPS and collaborating agencies initiated an aggressive public education campaign. The campaign included three news releases issued August 6–18, media interviews, and website alerts. The park newsletter, *The Yosemite Guide*, which was given to persons in every entering vehicle, included information about plague. Placards with plague information were posted at park entrances, locations with confirmed *Y. pestis* transmission, all campgrounds, and many day use locations and trailheads. Educational pamphlets were available to visitors at a variety of locations, including affected campgrounds.

DISCUSSION

In August 2015, these two cases of plague were linked to exposure in the internationally popular Yosemite National Park. The initial public health investigation and response with broad media coverage of the first case led to the rapid recognition and appropriate treatment of the second case-patient.

The investigation found little overlap in the travel itineraries of the two patients, and isolation of distinct strains of *Y. pestis* suggested that at least two *Y. pestis* strains were circulating among vector–host populations in the Yosemite area. In the only area visited by both patients, Yosemite Valley, no evidence of *Y. pestis* transmission in rodents was found, and *Y. pestis* has not been detected in the valley's rodent populations in recent decades (CDPH, unpub. data, 1984–2015). We were able to connect the exposure of patient 1 to epizootic transmission at the campground on the basis of the visual observations at Crane Flat Campground, the positive results for rodent serology and the pool of fleas collected there, and whole-genome MLST analysis of *Y. pestis* isolates from patient 1 and the flea pool. The most likely exposure site for patient 2 was Glacier Point, 20 km away, on the opposite side of Yosemite Valley. Although *Y. pestis*-



seropositive rodents were found at this location, we did not detect active infection in rodents or fleas and were therefore unable to directly link the patients' exposure to this site by whole-genome MLST.

The environmental investigation found evidence of *Y. pestis* transmission in disparate locations of the park, including epizootic activity in the Tuolumne Meadows area, \approx 41 and 25 km from Crane Flat and Glacier Point, respectively. Evidence of *Y. pestis* transmission in rodents was found at 4 of the 5 areas trapped. Of the eight species of rodents live trapped in Yosemite, *Y. pestis* antibodies were detected in only 5 (15.2%) of 33 lodgepole chipmunks and 3 (7.3%) of 41 California ground squirrels. However, *Y. pestis* was also isolated from golden-mantled ground squirrel and Douglas squirrel carcasses and a deer mouse flea, indicating broader zoonotic involvement.

The 2015 findings for Yosemite share some striking similarities with those associated with the only human plague case previously associated with Yosemite [20]. In 1959, a teenage boy became ill after camping along Yosemite Creek trail, \approx 5 km from Crane Flat Campground. Subsequent investigation by CDPH and CDC found evidence of a recent epizootic plague event that had decimated the rodent populations near the campsite. During this investigation, *Y. pestis* transmission was also documented in Tuolumne Meadows and at Lake Tenaya.

The rapid interagency investigation and public health response to these cases probably reduced the risk for plague among Yosemite visitors and staff. Critical risk-reduction measures included expanding the investigation to recreational sites beyond those visited by the patients and localized insecticide treatments at sites with *Y. pestis* transmission. Increased educational efforts informing the public about how to reduce their exposure to the cause of this potentially fatal disease contributed to the early diagnosis for patient 2 and to increased reports of finding dead rodents in the park, which led to detection of *Y. pestis* transmission at additional locations.

REFERENCES

- 1. Pollitzer R. Plague. Geneva: World Health Organization. 1954.
- Perry RD, Fetherston JD. *Yersinia pestis*—etiologic agent of plague. Clin Microbiol Rev. 1997;10:35– 66.
- 3. Gage KL, Kosoy MY. Natural history of plague: perspectives from more than a century of research. Annu Rev Entomol. 2005;50:505–28.
- 4. Link VB. A history of plague in United States of America. Public Health Monogr. 1955;26:1–120.
- 5. Caten JL, Kartman L. Human plague in the United States, 1900-1966. JAMA. 1968;205:333–6.
- 6. Kugeler KJ, Staples JE, Hinckley AF, Gage KL, Mead PS. Epidemiology of human plague in the United States, 1900-2012. Emerg Infect Dis. 2015;21:16–22.
- 7. Barnes A. Surveillance and control of bubonic plague in the United States. Symposium of the Zoological Society of London. 1982;50:237–70.
- Inglesby TV, Dennis DT, Henderson DA, Bartlett JG, Ascher MS, Eitzen E, et al. Working Group on Civilian Biodefense. Plague as a biological weapon: medical and public health management. JAMA. 2000;283:2281–90.



- Antolin JF, Gober P, Luce B, Biggins DE, van Pelt WE, Seery DB, et al. The influence of sylvatic plague on North American wildlife at the landscape level, with special emphasis on black-footed ferret and prairie dog conservation. In: Transactions of the 67th North American Wildlife and Natural Resources Conference; 2002 Apr 3–7. Washington (DC): Wildlife Management Institute; 2002.
- 10. CDC. Human plague—four states, 2006. MMWR Morb Mortal Wkly Rep. 2006;55:940–3.
- 11. Craven RB, Maupin GO, Beard ML, Quan TJ, Barnes AM. Reported cases of human plague infections in the United States, 1970-1991. J Med Entomol. 1993;30:758–61.
- 12. Wong D, Wild MA, Walburger MA, Higgins CL, Callahan M, Czarnecki LA, et al. Primary pneumonic plague contracted from a mountain lion carcass. Clin Infect Dis. 2009;49:e33–8.
- 13. Lowell JL, Wagner DM, Atshabar B, Antolin MF, Vogler AJ, Keim P, et al. Identifying sources of human exposure to plague. J Clin Microbiol. 2005;43:650–6.
- 14. Lang JD. Factors affecting the seasonal abundance of ground squirrel and wood rat fleas (Siphonaptera) in San Diego County, California. J Med Entomol. 1996;33:790–804.
- 15. CDC. Imported plague—New York City, 2002. MMWR Morb Mortal Wkly Rep. 2003;52:725–8.
- Eisen RJ, Enscore RE, Biggerstaff BJ, Reynolds PJ, Ettestad P, Brown T, et al. Human plague in the southwestern United States, 1957-2004: spatial models of elevated risk of human exposure to *Yersinia pestis.* J Med Entomol. 2007;44:530–7 .10.1603/0022-2585(2007)44[530:HPITSU]2.0.CO;2
- 17. Kwit N, Nelson C, Kugeler K, Petersen J, Plante L, Yaglom H, et al. Human Plague United States, 2015. MMWR Morb Mortal Wkly Rep. 2015;64:918–9.
- 18. California Department of Public Health. Yearly summaries of selected general communicable diseases in California, 2001–2010. Sacramento (CA): The Department; 2015.
- 19. California Department of Public Health. Yearly summaries of selected general communicable diseases in California, 2011–2014. Sacramento (CA): The Department; 2015.
- 20. Murray KF, Kartman L. Plague in California during 1959. California Vector Views. 1959;6:66–7.