# Plague outbreaks in prairie dog populations explained by percolation thresholds of alternate host abundance

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**Highly lethal pathogens (e.g., hantaviruses, hendra virus, anthrax, or plague) pose unique public-health problems, because they seem to periodically flare into outbreaks before disappearing into long quiescent phases. A key element to their possible control and eradication is being able to understand where they persist in the latent phase and how to identify the conditions that result in sporadic epidemics or epizootics. In American grasslands, plague, caused by *Yersinia pestis*, exemplifies this quiescent–outbreak pattern, because it sporadically erupts in epizootics that decimate prairie dog (*Cynomys ludovicianus*) colonies, yet the causes of outbreaks and mechanisms for interepizootic persistence of this disease are poorly understood. Using field data on prairie community ecology, flea behavior, and plague-transmission biology, we find that plague can persist in prairie-dog colonies for prolonged periods, because host movement is highly spatially constrained. The abundance of an alternate host for disease vectors, the grasshopper mouse (*Onychomys leucogaster*), drives plague outbreaks by increasing the connectivity of the prairie dog hosts and therefore, permitting percolation of the disease throughout the primary host population. These results offer an alternative perspective on plague's ecology (i.e., disease transmission exacerbated by alternative hosts) and may have ramifications for plague dynamics in Asia and Africa, where a single main host has traditionally been considered to drive *Yersinia* ecology. Furthermore, abundance thresholds of alternate hosts may be a key phenomenon determining outbreaks of disease in many multihost-disease systems.**

multihost disease transmission | pathogen percolation | *Yersinia pestis*

**Y**ersinia pestis, the bacterium that causes plague, has been responsible for three major human pandemics, including the Black Death, but is predominantly a disease of rodents (1). Plague is not a historical relic and remains a current public-health concern: there have been recent outbreaks in human populations (e.g., Madagascar, Uganda, and China), several human cases occur in the United States every year, and the pathogen is a potential threat as a bioterrorism agent (1–5). Introduced to the United States around 1900, plague currently poses a conservation threat to several animal species, including populations of the black-tailed prairie dog (*Cynomys ludovicianus*), a species of conservation concern in North American grasslands (6). Prairie dogs are social ground squirrels that occupy large towns or colonies on the prairie that can extend for hundreds of hectares and comprise several thousand individuals. Plague-induced prairie dog die-offs occur intermittently, but when they do, nearly 100% of prairie dogs in a colony die from the disease (7, 8). Based on observations of prairie dog die-offs, the general consensus suggests that plague outbreaks occur extremely rapidly, such that only a few survivors are observed after 6–8 wk (7, 9). However, the perceived short duration of an epizootic may be biased, because confirmation of plague often relies on enough luck to detect prairie dog carcasses above ground (most prairie dogs die in their burrows), and systematic surveillance for plague is infrequent.

Plague's remarkable pattern of sporadic explosive outbreaks (epizootic/epidemic phase) followed by extended periods of cryptic quiescence (enzootic/maintenance phase) is shared by many other highly deadly human diseases (e.g., hantaviruses, hendra virus, and anthrax). Frequently, key missing elements to the control and eradication efforts of such virulent diseases are an understanding of where pathogens persist during enzootic periods and an ability to identify the conditions that result in outbreaks or epizootics. There are two general hypotheses to explain enzootic–epizootic dynamics. The first hypothesis posits that pathogens spill over from one host species to a highly susceptible host species that suffers high mortality postinfection. The second hypothesis posits that pathogens simply circulate within one host species and that epizootics are the result of exacerbated transmission rates, perhaps as a result of changes in environmental conditions, host/vector abundance, and/or behavior (1, 10). Because of the unpredictable nature of sporadically occurring lethal diseases, there are few field-based investigations of free-living host populations that have managed to discern between these hypotheses (11).

The high lethality and rapid spread of plague in prairie dog populations have led to the consensus that the pathogen is most probably absent from healthy prairie dog colonies and instead, persists during interepizootics in an alternate rodent species (e.g., deer mice), although there is scant evidence to support this mechanism (1, 9, 12–14). Plague may also persist in the flea vectors after the local mammal host populations have been extirpated, although prairie dog fleas (*Oropsylla hirsuta*) experience high mortality in the absence of blood meals and flea abundance declines markedly postepizootic (15, 16). Nonetheless, because *Y. pestis* is a generalist pathogen that can infect multiple host and flea species in the prairie community, plague in prairie dog colonies provides an ideal system to examine the spillover and single-host persistence hypotheses for enzootic–epizootic pathogen ecology.

To investigate enzootic persistence and epizootic outbreaks of plague, we modeled the dynamics of *Y. pestis* in prairie dog colonies using insights from field data, which suggest that the northern grasshopper mouse (*Onychomys leucogaster*) is intricately involved in plague ecology. Grasshopper mice are infected with plague during prairie dog epizootics but show heterogeneity to mortality. High grasshopper mouse abundance is associated with a higher

ECOLOGY

likelihood of plague epizootics on prairie dog colonies, and during epizootics, grasshopper mice harbor high numbers of *O. hirsuta*, the normally host-specific prairie dog flea (17).

## Results and Discussion

In the absence of grasshopper mice, plague tended to infect only a small proportion of the prairie dog population over the course of 5 y (Fig. 1). Although, in some cases, ∼20% of the prairie dog population was infected during the 5-y time span, daily attack rates were small (rarely exceeded 6/d). Prairie dog families defend their coterie territories from neighboring prairie dogs, but in the event that all coterie members are killed, prairie dog coteries are normally annexed by a single neighboring family (18). Therefore, plague tended to be transmitted successively between adjacent coteries, because transmission of infected fleas occurs when an infected prairie dog dies and the fleas jump off the carcass to another prairie dog. In other words, the predominant means of host-to-host flea-borne transmission from one coterie to another is highly spatially constrained, and because plague can only spread from one coterie to the next sequentially, epizootics cannot occur by this process alone. Even when factoring in prairie dog dispersal, plague is still likely to only move between adjacent coteries after it has traveled to a new family territory, and therefore, epizootics remain unlikely.

Of major interest is the observation that plague was able to persist in prairie dog populations at low frequencies for substantial periods of time (e.g., enzootic phases endured for more than 1 y in ∼25% of model runs). That is, our results suggest that plague can persist in populations of a highly susceptible host for long periods and that it is not necessary to invoke the existence of alternate host species to explain interepizootic plague dynamics.

When grasshopper mice were incorporated into the model, plague epizootics were more likely to occur. At low densities of grasshopper mice, plague activity frequently faded out (because grasshopper mice were so rare that their territories did not coincide with prairie dog coteries containing infectious flea populations), but low mouse densities occasionally caused large die-offs of the prairie dog population. However, at higher grasshopper mouse density, plague epizootics resulting in prairie dog colony extinction were virtually guaranteed, although the course

of the epizootic could last up to 4 y (Fig. 1*B*). The results show a clear threshold phenomenon between mouse abundance and plague outbreaks among prairie dogs: increasing the mouse density by a few percent results in a dramatic increase of the total number of infected prairie dogs. At low density, mice are not able to spread plague across an entire colony, but after the density of mice crosses a threshold value, large epizootics in prairie dogs are a frequent outcome.

To further understand the role of grasshopper mouse abundance on disease dynamics in the main prairie dog host, we adapted the model to fully prevent prairie dog dispersal and annexation of abandoned coteries by neighboring prairie dogs. As a result, plague is spread exclusively through grasshopper mice, and epizootics in the main host are driven exclusively by the alternate host; consequently, we can investigate the disease dynamics from a network perspective. A percolation threshold occurs when vertices in a network are connected such that they form a giant component (i.e., a connected subgraph that contains the majority of vertices in the network) (19). In the context of infectious diseases, a vertex represents a host (or a group of hosts), and an edge represents a contact with the possibility of disease transmission between two groups of hosts. Below the percolation threshold, no giant component exists, and an initial infection in a random host cannot spread to the rest of the host population. In our plague model, the vertices represent the prairie dog coteries, and an edge exists between two vertices whenever a grasshopper mouse moves from its base coterie to another coterie. We calculate the number of components (single-vertex components are excluded, because they are irrelevant for disease spread) and the average size of the components. Starting from very low mice densities, increasing mouse density results in an increase of the number of components at first (because each mouse likely creates a new component) (Fig. 2). At a certain threshold density, the number of components starts to decrease as the mouse density increases, simply because additional mice are unlikely to create new components; instead, they will connect to already existing components and potentially even connect previously disconnected components. As more mice are added, the percolation threshold is crossed (i.e., most vertices are now connected and form a giant component), and the average number of components quickly approaches 1. In our plague model,
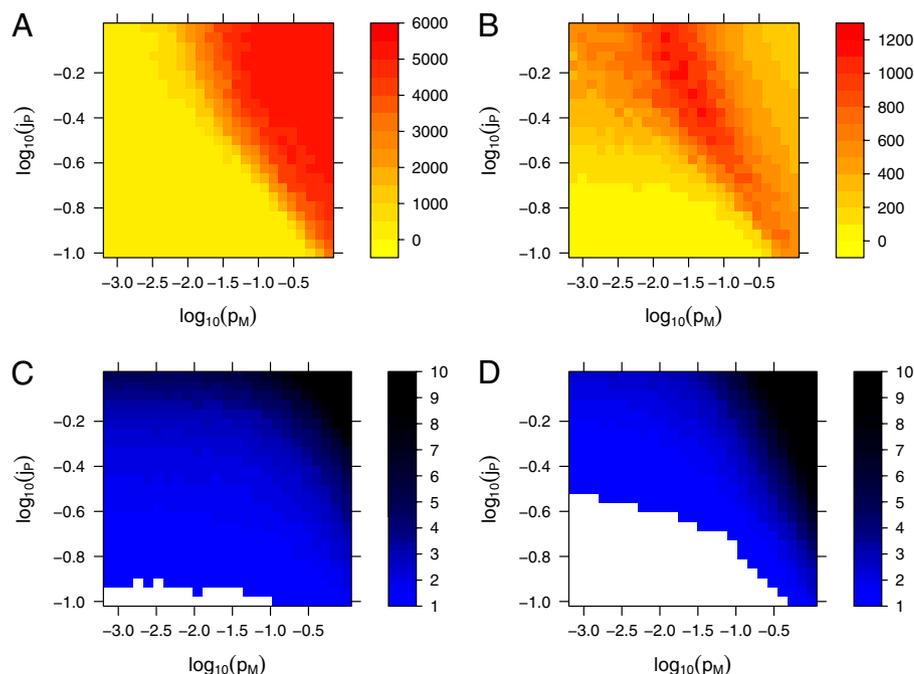


**Fig. 1.** The relationships between plague activity, grasshopper mouse density $p_M$, and flea jumping rate $j_P$ measured by (*A*) total number of infected prairie dogs during the simulation and (*B*) duration of plague activity in days. Each of the 625 pixels represents the means from 100 (*A* and *B*) or 1,000 (*C* and *D*) simulation runs, color-coded by value (see color strip to right of each graph). The axes are logarithmically scaled, although the left column of each panel represents 0 mice. The epizootic threshold is clearly visible in *A*. *B* shows that plague can persist in prairie dog populations over long periods of time, despite its high lethality. At low mouse densities, outbreaks fizzle out quickly and therefore, are of short duration. At intermediate densities, epizootics slowly burn through the colony, whereas at high densities, coteries are so well connected that plague moves rapidly through the colony. (*C*) Individual-based $R_0$. White denotes values <1. (*D*) Coterie-based $R_0$ ($R^*$) with the same color coding.
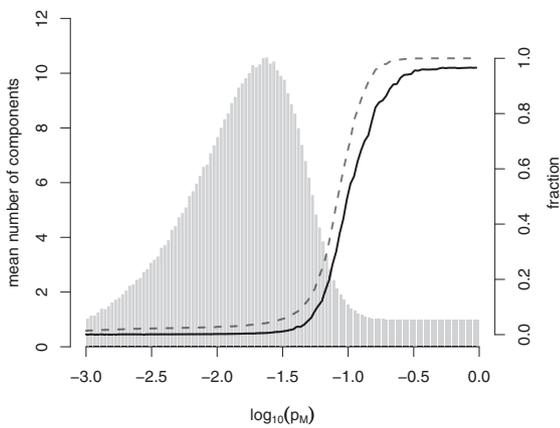
**Fig. 2.** Network components and epizootic thresholds. The gray bars show the number of components in the prairie dog colony, [i.e., in the network of coteries (nodes) connected by mouse dispersal (edges)] as a function of mouse density. The black solid line represents the average fraction of the prairie dog population infected. The dotted line represents the average graph component size (measured as the number of connected coteries) expressed as a fraction of the total number of coteries. The average epizootic size is dependent on the average graph component size, because any outbreak is limited by the size of the component in which the initial infection occurs. The outbreak curve is slightly shifted downward relative to the component curve because of stochastic disease transmission. (Parameters set to default values as specified in model description; $j_P = 0.6$).

this means that almost all coteries are now connected by mouse movement, and thus, plague can spread and infect the majority of the prairie dog population. Indeed, the fraction of the prairie dog population that is infected increases dramatically as the number of components decreases, and the average number of the components is a reliable predictor of the size of an epizootic (clearly, no epizootic can be larger than the number of hosts connected by mouse dispersal, and thus, the dotted line in Fig. 2 indicates the maximum epizootic size possible). That is, we show that the mouse abundance threshold behavior is a percolation phenomenon: at low densities, mice can spread plague locally only, but at higher density, the territories of mice overlap sufficiently that plague can be spread across the entire colony (Fig. 2).

The ability to predict disease thresholds has traditionally been based on the concept of $R_o$, the number of secondary cases caused by the first infectious individual in a completely susceptible population. However, the standard invasion threshold $R_o = 1$ (above which a disease can invade) has been shown to be a poor predictor of disease outbreaks in populations that are subdivided into groups with limited movement among groups. In such populations, $R^*$, a group-level equivalent of $R_o$ (20), is a better predictor of disease invasion (21–23). More recently, a percolation threshold has been shown to occur in great gerbil populations in central Asia, where disease outbreaks seem to occur only above a certain host-abundance threshold (24). Our model suggests that a percolation threshold of the secondary host predicts plague epizootics in prairie dogs (Fig. 2). To show that this percolation threshold is distinct from both the $R_o$ and $R^*$ thresholds, we measured the individual-based and coterie-based $R_o$ (i.e., $R^*$). Individual-based $R_o$ is the average number of secondary prairie dog infections caused by the first infected prairie dog in the initially susceptible population. Coterie-based $R_o$ ($R^*$) is the average number of coteries with at least one prairie dog infection that was caused by a prairie dog in the initially infected coterie.

$R_o$ failed to accurately predict plague outbreaks, because although plague is easily transmitted between prairie dogs within a coterie, disease spread between coteries depends largely on grasshopper mice and thus, is independent of $R_o$ (Fig. 1C). $R^*$ is a better predictor of plague outbreaks, because it accounts for

disease spread between coteries. Nonetheless, this is only true if mouse densities are high enough (Fig. 1D), because only then are most coteries connected by mouse movement, forming a giant component (Fig. 2). At low mouse densities, groups of coteries form many isolated small components, and although $R^*$ correctly predicts transmission within these components, large-scale outbreaks cannot occur.

Recent evidence from field research supports our model's description of plague's ecology in prairie dog populations. High abundance of grasshopper mice has been associated with increased likelihood of plague-induced prairie dog die-offs during the following year (17). Also, increasing numbers of grasshopper mice are infested by prairie dog fleas several months before large-scale prairie dog die-offs are actually observed, which we interpret as evidence of prairie dog family groups being killed by plague, although declines in the prairie dog population remain hard to detect (17). *Y. pestis*-infected fleas have been isolated from prairie dog colonies several months in advance of epizootics (25). Furthermore, population genetic structure of *Y. pestis* in Arizonan prairie dog populations suggests periods of very rapid plague transmission followed by periods of slower localized differentiation (26).

We have described a mechanism for plague activity on a single prairie dog colony, which involves exacerbated disease transmission among a prairie dog population when combined with high densities of grasshopper mice. Patterns of prairie dog colony extinctions suggest that colony area and the fate of adjacent colonies are important influences on the likelihood a colony suffering die-offs (8). This pattern suggests that the movement of plague between prairie dog colonies is caused by the movement of infectious animals or animals carrying infectious fleas and that metapopulation dynamics may be important in explaining plague's ecology at the landscape scale (1, 8, 26, 27). Nonetheless, our model and field observations suggest that, if plague arrives at a prairie dog colony, the local grasshopper mouse abundance will be a strong influence on the likelihood of *Y. pestis* transmission erupting into an outbreak as opposed to smoldering cryptically through the prairie dog population. Grasshopper mouse abundance fluctuates over time and also varies across the landscape (17, 28), suggesting that prairie dog die-offs are the consequence of coincidental mouse population dynamics and *Y. pestis* movement (i.e., simultaneous interactions of disease movement and host ecology). Interestingly, our results based on model interpretations of empirical observations echo previous theoretical work examining disease persistence and outbreaks.

## Conclusion

In summary, using field data to parameterize a spatially explicit multihost model of plague dynamics provided surprising insights into the disease's ecology that suggest that (*i*) plague is able to persist in prairie dog colonies for prolonged periods at low enzootic levels simply by moving sequentially between neighboring family groups (coteries) and (*ii*) grasshopper mice, even at low frequencies, exacerbate plague spread throughout a prairie dog colony, causing large epizootics. These results provide an insight into plague dynamics, i.e., that complex multihost interactions drive disease outbreaks but that the process of spillover from enzootic hosts is not necessarily crucial to interepizootic pathogen persistence. Indeed, the propensity of plague for social burrowing animals that maintain territories (e.g., prairie dogs, ground squirrels, great gerbils, etc.) (1) may explain its ability to persist in the host population as a highly lethal pathogen.

The observation that additional species can strongly impact disease dynamics is particularly interesting, because it exemplifies a classical percolation-threshold phenomenon. Although the importance of percolation thresholds in disease systems has been established before (24), percolation thresholds in which alternate hosts are the key element determining outbreaks of disease in a primary host have not previously been reported. Many disease

ECOLOGY

systems involve multiple hosts infected by generalist pathogens (for example, canine distemper virus in Serengeti carnivores and Lyme disease in small mammals) (29, 30) and furthermore, involve species that occupy more or less discrete home ranges that induce spatial structure pertinent to disease transmission. Consequently, insights provided by percolation dynamics involving alternate hosts may be of crucial importance in understanding the complex disease dynamics of wildlife and zoonotic diseases.

## Materials and Methods

We developed a simple computational model to simulate plague dynamics in prairie dog colonies, predominantly using data from our study site in the Pawnee National Grasslands (PNG), northern Colorado (17). On the PNG, prairie dog colonies range in size up to ~250 ha, and prairie dog density has been conservatively calculated at 22 ha$^{-1}$ (31), suggesting that large colonies are populated by over 5,500 prairie dogs. Coterie (family group) territories are usually contiguous (18) and in northwest Colorado, normally occupy ~0.2 ha (range = 0.12–0.61 ha) (32). Prairie dog families defend their coterie territories from neighboring prairie dogs, but in the event that all coterie members are killed, prairie dog coteries are normally annexed by a single neighboring family (18). Prairie dog dispersal is limited to a 10-wk window each year (33).

Grasshopper mice are abundant on prairie dog colonies (28), with densities of 0–10 ha$^{-1}$ or the equivalent of up to 2 mice per prairie dog coterie. Radio-tracked grasshopper mice on prairie dog colonies had mean home ranges of 3.84 ha (SD = 1.64) when followed over a 7-d period, which suggests that that mice traverse and interact with at least three coteries a night.

Prairie dogs are predominantly infested by the normally host-specific flea *O. hirsuta* (25). After infected with plague, prairie dogs die in approximately 6 d (12, 34), and the fleas will subsequently search for new hosts. Dying prairie dogs infest a high proportion of their fleas with the plague bacterium: 18/22 (or 82%) *O. hirsuta* fleas found on dead prairie dogs were plague-positive, and fleas are likely to be infected only in the terminal stages of the host's *Y. pestis* infection (35, 36). Grasshopper mice are normally infrequent hosts of *O. hirsuta* but during plague epizootics 81% of grasshopper mice, can be infested with this flea species (17).

We simulated plague dynamics on one large prairie dog colony, organized as a square grid of 33 × 33 sites (total = 1,089), where each site is initially occupied by one prairie dog coterie that is comprised of five prairie dog individuals and provides an initial population size of 5,445 prairie dogs. Grasshopper mice were assigned to each site as a Bernoulli trial with probability $p_M$ (i.e., for $p_M = 0$, plague dynamics are simulated without mice present) and allowed to disperse to three random coteries in the vicinity of their base coterie.

We model a single large colony as a spatial regular network where each vertex represents a coterie initially inhabited by one family of five prairie dogs. A simulation runs for 1,825 time steps, where each time step represents a day (to incorporate daily differences in plague-transmission likelihoods), and the simulation run approximates plague dynamics for 5 y. On a random day during the first 365 d of the simulation, a randomly assigned prairie dog individual is infected with plague. At each time step, the following processes occur: (*i*) exposure of prairie dogs to infectious fleas jumping off carcasses of plague-killed prairie dogs, (*ii*) transmission of *Y. pestis* from fleas to prairie dogs, (*iii*) exposure of prairie dogs to fleas jumping off mice, and (*iv*) exposure of mice to infectious fleas and *Y. pestis* infection. Further details can be found in *SI Materials and Methods*.

To understand patterns of plague dynamics, we explored the parameter space by running 100 simulations per parameter combination. (For a few test parameter combinations, we ran 1,000 simulations, and the observed patterns remained robust). We used the following parameters: $j_P$, the probability that hosts are jumped on by fleas abandoning plague-killed prairie dogs, varied from 0.1 to 1.0, mouse frequency varied from 0 to 1.0, and prairie dog dispersal to random coterie territories varied from 0% to 20%. We measured three outputs: the total number of infected prairie dogs, the maximum daily attack rate (the number of prairie dogs newly infected during 1 d), and the duration of plague activity (the length of time between first infection of a prairie dog and the last infection of a prairie dog).

1. Gage KL, Kosoy MY (2005) Natural history of plague: Perspectives from more than a century of research. *Annu Rev Entomol* 50:505–528.
2. Boisier P, et al. (2002) Epidemiologic features of four successive annual outbreaks of bubonic plague in Mahajanga, Madagascar. *Emerg Infect Dis* 8:311–316.
3. Ogen-Odoi A, et al. (2009) Bubonic and pneumonic plague—Uganda, 2006. *MMWR Morb Mortal Wkly Rep* 58:778–781.
4. World Health Organization (2009) Global alert and response. Plague in China. Available at http://www.who.int/csr/don/2009_08_11/en/index.html. Accessed June 30, 2010.
5. Stenseth NC, et al. (2008) Plague: Past, present, and future. *PLoS Med* 5:e3.
6. United States Fish and Wildlife Service (2004) Endangered and threatened wildlife and plants; finding for the resubmitted petition to list the blacktailed prairie dog as threatened. *Fed Regist* 69:51217–51226.
7. Pauli JN, Buskirk SW, Williams ES, Edwards WHA (2006) A plague epizootic in the black-tailed prairie dog (*Cynomys ludovicianus*). *J Wildl Dis* 42:74–80.
8. Stapp P, Antolin MF, Ball M (2004) Patterns of extinction in prairie dog metapopulations: Plague outbreaks follow El Niño events. *Front Ecol Environ* 2:235–240.
9. Webb CT, Brooks CP, Gage KL, Antolin MF (2006) Classic flea-borne transmission does not drive plague epizootics in prairie dogs. *Proc Natl Acad Sci USA* 103:6236–6241.
10. Davis S, et al. (2004) Predictive thresholds for plague in Kazakhstan. *Science* 304:736–738.
11. Lloyd-Smith JO, et al. (2005) Should we expect population thresholds for wildlife disease? *Trends Ecol Evol* 20:511–519.
12. Cully JF, Williams ES (2001) Interspecific comparisons of sylvatic plague in prairie dogs. *J Mammal* 82:894–905.
13. Pollitzer R, Meyer KF (1961) *Studies of Disease Ecology*, ed May JF (Hafner, New York), pp 433–501.
14. Salkeld DJ, Stapp P (2008) No evidence of deer mouse involvement in plague (*Yersinia pestis*) epizootics in prairie dogs. *Vector Borne Zoonotic Dis* 8:331–337.
15. Wilder AP, et al. (2008) *Oropsylla hirsuta* (Siphonaptera: Ceratophyllidae) can support plague epizootics in black-tailed prairie dogs (*Cynomys ludovicianus*) by early-phase transmission of *Yersinia pestis*. *Vector Borne Zoonotic Dis* 8:359–367.
16. Salkeld DJ, Stapp P (2008) Prevalence and abundance of fleas in black-tailed prairie dog burrows: Implications for the transmission of plague (*Yersinia pestis*). *J Parasitol* 94:616–621.
17. Stapp P, et al. (2009) Evidence for the involvement of an alternate rodent host in the dynamics of introduced plague in prairie dogs. *J Anim Ecol* 78:807–817.
18. Hoogland JL (1995) *The Black-Tailed Prairie Dog* (University of Chicago Press, Chicago).
19. Grimmett G (1999) *Percolation* (Springer, Berlin).
20. Ball F, Mollison D, Scalia-Tomba G (1997) Epidemics with two levels of mixing. *Ann Appl Probab* 7:46–89.
21. Hagenaars TJ, Donnelly CA, Ferguson NM (2004) Spatial heterogeneity and the persistence of infectious diseases. *J Theor Biol* 229:349–359.
22. Cross PC, Lloyd-Smith JO, Johnson PLF, Getz WM (2005) Duelling timescales of host movement and disease recovery determine invasion of disease in structured populations. *Ecol Lett* 8:587–595.
23. Cross PC, Johnson PLF, Lloyd-Smith JO, Getz WM (2007) Utility of R0 as a predictor of disease invasion in structured populations. *J R Soc Interface* 4:315–324.
24. Davis S, Trapman P, Leirs H, Begon M, Heesterbeek JAP (2008) The abundance threshold for plague as a critical percolation phenomenon. *Nature* 454:634–637.
25. Tripp DW, Gage KL, Montenieri JA, Antolin MF (2009) Flea abundance on black-tailed prairie dogs (*Cynomys ludovicianus*) increases during plague epizootics. *Vector Borne Zoonotic Dis* 9:313–321.
26. Girard JM, et al. (2004) Differential plague-transmission dynamics determine *Yersinia pestis* population genetic structure on local, regional, and global scales. *Proc Natl Acad Sci USA* 101:8408–8413.
27. Salkeld DJ, et al. (2007) The potential role of swift foxes (*Vulpes velox*) and their fleas in plague outbreaks in prairie dogs. *J Wildl Dis* 43:425–431.
28. Stapp P (2007) Rodent communities in active and inactive colonies of black-tailed prairie dogs in shortgrass steppe. *J Mammal* 88:241–249.
29. Craft ME, Hawthorne PL, Packer C, Dobson AP (2008) Dynamics of a multihost pathogen in a carnivore community. *J Anim Ecol* 77:1257–1264.
30. Brisson D, Dykhuizen DE, Ostfeld RS (2008) Conspicuous impacts of inconspicuous hosts on the Lyme disease epidemic. *Proc Biol Sci* 275:227–235.
31. Magle SB, et al. (2007) Mark-resight methodology for estimating population densities for prairie dogs. *J Wildl Manage* 71:2067–2073.
32. Tileston JV, Lechleitner RR (1966) Some comparisons of the black-tailed and white-tailed prairie dogs in north-central Colorado. *Am Midl Nat* 75:292–316.
33. Slobodchikoff CN, Perla BS, Verdolin JL (2009) *Prairie Dogs: Communication and Community in an Animal Society* (Harvard University Press, Cambridge, MA).
34. Rocke TE, Smith SR, Stinchcomb DT, Osorio JE (2008) Immunization of black-tailed prairie dog against plague through consumption of vaccine-laden baits. *J Wildl Dis* 44:930–937.
35. Engelthaler DM, Gage KL (2000) Quantities of *Yersinia pestis* in fleas (Siphonaptera: Pulicidae, Ceratophyllidae, and Hystrichopsyllidae) collected from areas of known or suspected plague activity. *J Med Entomol* 37:422–426.
36. Hinnebusch BJ (2005) The evolution of flea-borne transmission in *Yersinia pestis*. *Curr Issues Mol Biol* 7:197–212.