

# Climate-Driven Spatial Dynamics of Plague among Prairie Dog Colonies

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**ABSTRACT:** We present a Bayesian hierarchical model for the joint spatial dynamics of a host-parasite system. The model was fitted to long-term data on regional plague dynamics and metapopulation dynamics of the black-tailed prairie dog, a declining keystone species of North American prairies. The rate of plague transmission between colonies increases with increasing precipitation, while the rate of infection from unknown sources decreases in response to hot weather. The mean annual dispersal distance of plague is about 10 km, and topographic relief reduces the transmission rate. Larger colonies are more likely to become infected, but colony area does not affect the infectiousness of colonies. The results suggest that prairie dog movements do not drive the spread of plague through the landscape. Instead, prairie dogs are useful sentinels of plague epizootics. Simulations suggest that this model can be used for predicting long-term colony and plague dynamics as well as for identifying which colonies are most likely to become infected in a specific year.

**Keywords:** plague, host-pathogen dynamics, prairie dog, hierarchical, Bayesian.

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It has long been recognized that the dynamics of virulent pathogens and their hosts are closely related (Kermack and McKendrick 1927). Models of this interplay should therefore address the joint dynamics of hosts and pathogens, especially when infected hosts are short-lived and when the dispersal of the disease or the host is restricted (Fulford et al. 2002). Here, we present the first spatially explicit joint model of regional host and pathogen metapopulation dynamics fitted to empirical data.

Among the many emerging and reemerging infectious diseases affecting humans and wildlife (Dobson and Foutopoulos 2001; Hudson et al. 2001; Morens et al. 2004; Collinge and Ray 2006), plague is one of the most generalized, affecting >200 mammalian species worldwide (Poland and Barnes 1979). Epidemic plague is resurging in all major regions of the world (Gubler et al. 2001). It is caused by the bacterium *Yersinia pestis* and is lethal to humans without immediate access to effective antibiotics (Galimand et al. 1997). Because of its virulence and lethality, the effects of plague spread from susceptible species to ecosystems. On the North American prairie, the disease is one of the major threats (Cully and Williams 2001) to the black-tailed prairie dog (*Cynomys ludovicianus*), a keystone species with >100 associated species (Kotliar et al. 1999). The introduction of this exotic disease may thus threaten the functional role of prairie dogs in prairie ecosystems (Biggins and Kosoy 2001).

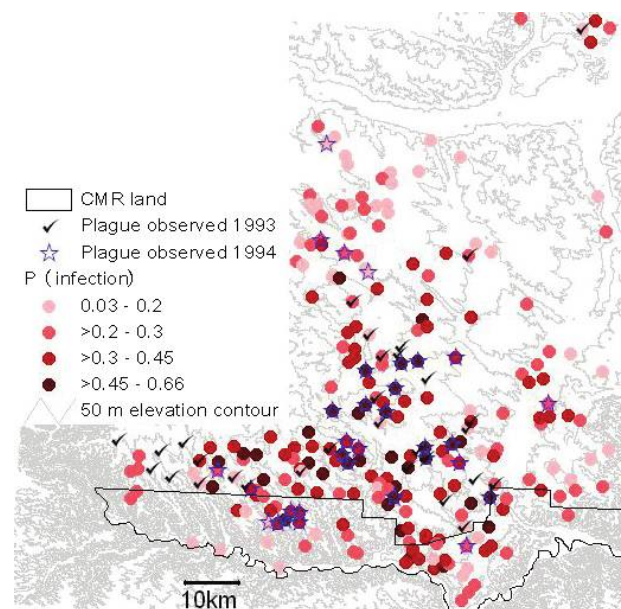
The black-tailed prairie dog is a colonial rodent occupying burrow systems that constitute discrete habitat patches in the landscape. Colonies are often extirpated by plague, but the unoccupied burrows that are left behind may be recolonized, leading to metapopulation dynamics (Lomolino and Smith 2001) driven largely by plague (Cully and Williams 2001; Stapp et al. 2004). Moreover, the occurrence of the disease is related to climatic conditions and to spatial colony structure (Girard et al. 2004; Collinge et al. 2005a). Although insights into the route of plague transmission between individuals within a colony have been presented recently (Webb et al. 2006), the spatial transmission of the disease between colonies is poorly understood. Moreover, despite the worldwide importance of

plague, no predictive models of host-pathogen metapopulation dynamics have yet been developed for this disease. Here, we model the joint metapopulation dynamics of both prairie dog colonies and plague, where colonies constitute patches for the disease (Hess et al. 2001). As one of the first models to address joint spatial and host-pathogen structure in a disease of wildlife, this study offers a template for future research (Murray et al. 1986; Smith et al. 2002).

The impact of landscape structure on disease dynamics is only beginning to be explored (Ostfeld et al. 2005). Some understanding of disease transmission has been gained through descriptive studies of disease occurrence and genetics (Bourhy et al. 1999; Cully and Williams 2001; Girard et al. 2004; Stapp et al. 2004; Collinge et al. 2005a). Spatial aggregation in disease outbreaks and parasite genetics may reflect spatially restricted dispersal of host species. With regard to plague, this hypothesis is strengthened by the finding that presumed barriers to the movements of prairie dogs explain both disease occurrences (Collinge et al. 2005a) and the spatial genetic structuring of *Y. pestis* (Girard et al. 2004). Alternatively, aggregated plague occurrence patterns may reflect aggregations of suitable reservoir hosts (Collinge et al. 2005a). Moreover, regional epizootics often skip local colonies. This pattern may be attributed either to imperfect transmission from a regional plague reservoir or to long-distance transmission among colonies facilitated by large mammals carrying infected fleas across the landscape (Barnes 1982).

The effects of host or pathogen dispersal on spatial aggregation of epizootics can be summarized by using a metapopulation model to fit a dispersal function to the spread of disease (Hanski 1999; Xia et al. 2004; Snäll et al. 2005). A fitted metapopulation model serves two important purposes. First, the dispersal function can be compared with the dispersal kernels of potential disease carriers to help in the identification of the carrier(s) involved. Second, it may enable prediction of the spatial spread of the disease under different environmental conditions or intervention programs (Murray et al. 1986; Keeling et al. 2003). In fitting such a model, it should be recognized that both infection risk and subsequent infectiousness may be affected by local population size (Xia et al. 2004) or other conditions as well as by landscape heterogeneity (Ricketts 2001; Girard et al. 2004; Collinge et al. 2005a; Smith et al. 2005).

There has been a great interest in the effect of the size of prairie dog colonies on plague occurrence and local extinction risk. Descriptive analyses have suggested that larger colonies may be more likely to become infected if they are more likely to attract sources of infection, such as reservoir hosts or carnivores transporting infected fleas. Colonies that cover larger areas may also be more likely



**Figure 1:** Study area with plotted mean probability of colonies becoming infected by plague in 1994 as predicted by the spatial model for joint colony and plague dynamics based on the state observed in 1993 (see “Methods”). Predictions were not made for colonies that had become extinct due to plague in 1993. CMR = Charles M. Russell Natural Wildlife Refuge.

to overlap persistent or ephemeral sources of plague (Collinge et al. 2005a). Finally, disease risk in large colonies is of interest because large colonies may be necessary to support species that depend on the prairie dog (Kotliar et al. 1999). Nevertheless, studies have found no consistent effect of colony size (area covered) on disease occurrence and extinction risk (Cully and Williams 2001; Stapp et al. 2004; Collinge et al. 2005a; Wagner et al. 2006). Fitting a mechanistic metapopulation model allows the separation of effects of colony area on (1) the rate of colony-level infection and (2) the subsequent rate of extinction of the infected colony. Moreover, it allows a novel investigation of the effect of colony area on colony infectiousness.

Climate is known to affect the transmission of some pathogens (Koelle et al. 2005; Hudson et al. 2006), probably including plague (Parmenter et al. 1999; Ensore et al. 2002; Collinge et al. 2005b; Stenseth et al. 2006), but spatially explicit effects of climate on transmission have not been studied. Descriptive analyses of temporal patterns of plague occurrence in humans and prairie dogs have led to the “cascade hypothesis” (Parmenter et al. 1999) for plague prevalence: in arid ecosystems, increased precipitation increases primary productivity, which increases the densities of rodents, fleas, and plague. Formulated in a spatial context, the cascade hypothesis suggests that var-

iation in primary productivity among colonies may partially explain variation among colonies in infection risk. In addition, ambient temperatures affect both flea population growth and the efficient transmission of *Y. pestis* from fleas to hosts (Gage and Kosoy 2005).

The aim of this study was to develop a predictive model for the joint metapopulation dynamics of prairie dog colonies and plague. The colony submodel provides parameter estimates for colony growth and colonization-extinction turnover. To this submodel, plague transmission is added as a function of distance and potential barriers between infectious and susceptible colonies. We test for effects of colony area on infectiousness and on probabilities of colony infection and extinction, effects of intercolony distance and barriers in the landscape on plague transmission between colonies, effects of climate on the rate of plague transmission, and effects of primary productivity on the probability of colony infection. Finally, we performed a set of simulations to evaluate the ability of the final model to predict temporal and spatial plague dynamics.

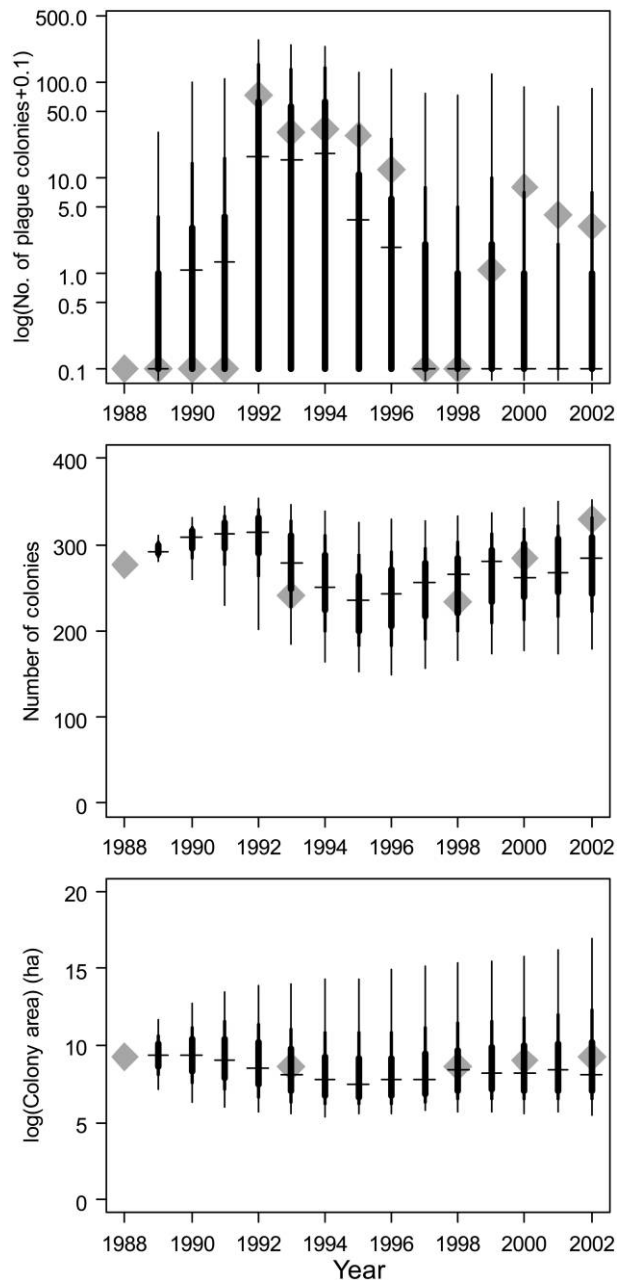
## Methods

### *Empirical Data*

The study landscape is located in Phillips County, Montana (fig. 1). It is composed of short- and mixed-grass prairie and sagebrush. The land is managed by the Charles M. Russell (CMR) National Wildlife Refuge, the Bureau of Land Management (BLM), and private owners. Cattle ranching and recreation are the dominant land uses.

Observational data on plague occurrence (colony die-offs) on CMR and BLM lands have been collected since 1979 according to methods outlined in Collinge et al. (2005a). No colony die-offs were observed in the county before 1992. The area of all prairie dog colonies was mapped on both BLM and CMR lands in 1988, 1993, 1998, 2000, and 2002, and only on CMR land in 1995, 1997, 1999, and 2001. Each year, known colonies were checked for die-off and tested for plague where appropriate, but mapping was conducted only during survey years. Figure 2 shows the empirical data summarized by year.

The area of each colony was defined by the lines joining the outermost active burrows. Through time, colonies not only change in area but also split apart or fuse, making it difficult to follow and delimit individual colonies (Wagner et al. 2006). We therefore used the colony complex (Wagner et al. 2006) as the unit in our analysis, although we use the term “colony” in the text for convenience. The area of a complex was defined as the sum of the areas of the colonies comprising the complex. The location of each colony complex was fixed between years and defined as



**Figure 2:** Observed (gray diamonds) and predicted colony and plague dynamics based on a simulation using the spatial model. Horizontal lines show modes, and vertical lines show 95% (thick), 75% (medium), and 50% (thin) highest posterior density intervals for the predictions.

the coordinate of the centroid of the total areal extent of the colonies comprising the complex during 1988–2002. For further details, see Collinge et al. (2005a).

For each prairie dog colony, we calculated the primary productivity (kg/ha/year) for an area that was the sum of the area defined by the areal extent of the colony over all

years of mapping plus a surrounding buffer zone of 50 m. For details, see appendix A in the online edition of the *American Naturalist*.

We quantified topographic relief (ravines) between colonies by counting the number of crossings of 50-m elevation contours along a straight line between colony locations. Correspondingly, we counted the number of crossings of streams. For details, see below and appendix A.

We tested effects of climate on the rate of plague transmission using three variables previously shown to be associated with plague. The summed precipitation during April–July in the preceding year and the number of days  $>35^\circ\text{C}$  in the focal year were both identified as the best predictors of plague occurrence among prairie dog colonies in the data modeled here (Collinge et al. 2005b). The extensive set of models fitted in Collinge et al. (2005b) were based on hypotheses about relations between climate and plague dynamics on the North American prairie, and they showed a low sensitivity to the temperature limits chosen and to the period over which precipitation was summed. The mean temperature for March through May was identified as the best predictor of plague prevalence in other rodents (Stenseth et al. 2006).

#### *A Model for the Joint Metapopulation Dynamics of Prairie Dog Colonies and Plague*

The long-term data on both colony and plague dynamics allowed us to develop a model for both the spatial transmission of plague in the landscape and for the metapopulation dynamics of prairie dog colonies (fig. 3). The colony submodel (eqq. [1]–[3]) was used to predict the presence and size of each colony in years or in parts of the landscape (see “Empirical Data”) where no mapping had been conducted. These predictions were fed into the plague submodel (eqq. [4]–[7]). Since the submodels were fitted jointly, the uncertainty in colony dynamics affected the parameter estimates of the plague submodel, and vice versa.

We applied a Bayesian modeling approach because it is convenient for fitting complex hierarchical models with a formal mathematical treatment of natural variability and data uncertainty (Ellison 2004; Gelman 2004; Clark 2005; Snäll et al. 2007). For a description of parameters, symbols, and prior distributions, see table C1 in the online edition of the *American Naturalist*. The models were fitted using the software OpenBUGS 2.1 (Thomas et al. 2006).

*Colony Submodel.* In modeling the metapopulation dynamics of prairie dog colonies, we assumed that between years, colonies could either grow, shrink, become extinct, or arise (through colonization) in any location where a

colony had been observed during the study period (fig. 3). More specifically, we assumed that the logarithm of the area ( $A$ ) of a colony (ha) at location  $i = 1, \dots, M \leq 399$ , in year  $t = 2, \dots, 15$  (1989–2002) was

$$\log(A_{i,t}) \sim N(\mu_{\log(A_{i,t})}, \sigma_{A,S(i,t)}^2), \quad (1)$$

with mean  $\mu_{\log(A_{i,t})}$  and variance  $\sigma_{A,S(i,t)}^2$ , where  $S(i,t)$  is an indicator variable for whether colony  $i$  is infected (1) or not infected (0) by plague at time  $t$ . These higher-level mean and variances are called hyperparameters and are estimates of the mean and variance of the higher-level superpopulation (Gelman 2004). We modeled  $\mu_{\log(A_{i,t})}$  as

$$\mu_{\log(A_{i,t})} = \begin{cases} \theta_{S(i,t),t} + \log(A_{i,t-1}) & I_{i,t-1} = 1 \\ \Gamma_{i,t} \log(\Delta_{i,t}) & I_{i,t-1} = 0 \end{cases}, \quad (2)$$

where  $I_{i,t-1}$  is an indicator parameter specifying whether a colony was present (1) or absent (0) at location  $i$  in year  $t-1$ , and  $\theta_{S(i,t),t}$  is the colony area growth rate that is assumed to be normal distributed; hence,  $\theta_{S(i,t),t} \sim N(\mu_{\theta,S(i,t)}, \sigma_{\theta,S(i,t)}^2)$ . In other words, we present year-specific estimates of the growth rates ( $\theta$ ) that are different for infected and noninfected colonies. The parameter  $\sigma_{\theta,S(i,t)}^2$  describes the variation between years in these estimates, and  $\mu_{\theta,S(i,t)}$  provides general estimates of the growth rates for infected and noninfected colonies. The symbol  $\Delta$  is the area of a newly colonized colony; indexing  $\Delta$  by  $i, t$  shows that it is modeled as being year and location specific. Also, the events colonization (1) or not (0) were year and location specific and assumed to be Bernoulli distributed with probability  $\Gamma_{i,t}$ . The parameters  $\Gamma_t$  were drawn from a beta distribution with shape parameters  $\kappa_1$  and  $\kappa_2$ , that is,  $Be(\kappa_1, \kappa_2)$ . We define the posterior predicted colonization rate  $\bar{\Gamma}$  as  $\sim Be(\kappa_1, \kappa_2)$ .

We modeled the probability of a colony persisting in year  $t$  as a function of previous colony area using a logistic regression approach (fig. 3); that is, we assumed that a colony persisted according to a Bernoulli distribution with probability  $\Omega_{i,t}$  and

$$\log it(\Omega_{i,t}) = \beta_{S(i,t)} + \omega_{S(i,t)} \log(A_{i,t-X}), \quad (3)$$

where  $\beta_{S(i,t)}$  and  $\omega_{S(i,t)}$  are parameters. During field surveys, plague occurrence was recorded at the same time as colony area. This means that at the time of the survey in year  $t-1$ , the area of infected colonies had probably begun to decrease due to plague. Using the measure of area recorded in  $t-1$  would lead to an incorrect estimate of the effect of area. For this reason, we modeled  $\Omega_{i,t}$  as a function of area in  $t-2$  ( $X=2$ ) for infected colonies and as a function of area in  $t-1$  ( $X=1$ ) for noninfected colonies.

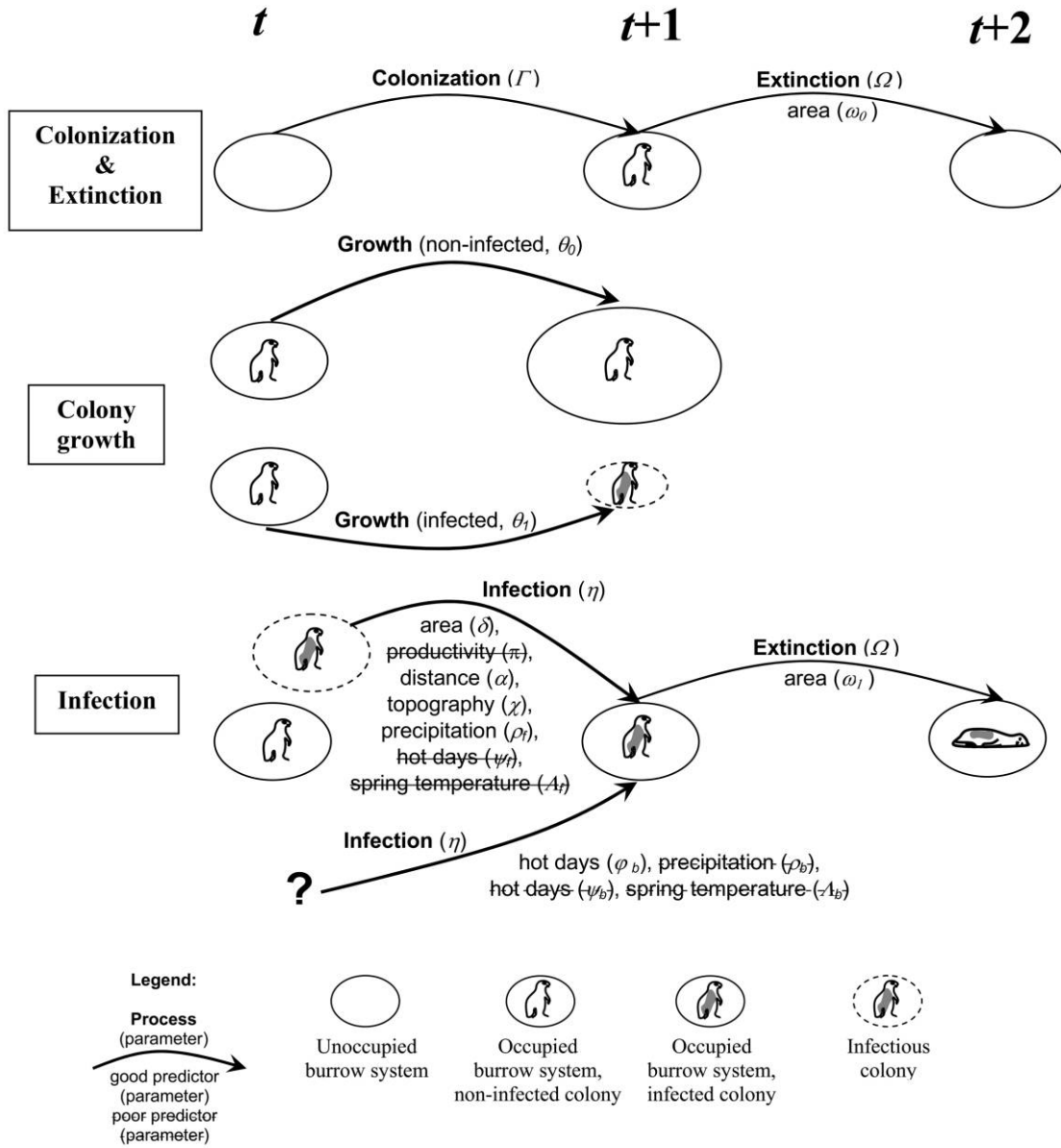


Figure 3: Schematic picture of the models fitted. Variables and associated parameters shown in strike-through text were not significant and were not included in the final spatial model.

Since colonies were not mapped at each location each year (see “Empirical Data”), we estimated colony presence ( $I_{i,t}$ ) and area ( $A_{i,t}$ ) at these locations ( $i$ ) in these years ( $t$ ). We estimated  $A_{i,t}$  using equations (1) and (2), and we estimated  $I_{i,t}$  using equation (2) ( $\Gamma_{i,t}$ ) if  $I_{i,t-1} = 0$  or equation (3) ( $\Omega_{i,t}$ ) if  $I_{i,t-1} = 1$ .

*Plague Submodel.* To model plague transmission, we used a modified and extended version of a gravity model for epidemiological coupling (Xia et al. 2004) that also ac-

counts for environmental heterogeneity of susceptible colonies, landscape heterogeneity between colonies, and climatic conditions. We describe the full model below (fig. 3). The final model presented was the result of a selection procedure (see app. B in the online edition of the *American Naturalist*). The model assumed that the number of infectious units arriving at a colony follows a Poisson distribution, so that the probability of at least one successful infection is  $\eta_{i,t} = 1 - e^{-\lambda_{i,t}}$ , where  $\lambda_{i,t}$  is the rate of infection. In fitting the model, we acknowledged that plague

was not present in the landscape before the first observed colony die-off due to plague in 1992; hence, we restrict our calculations to  $t = 5, \dots, 15$  (1992–2002). The probability of a colony becoming infected was modeled as

$$\begin{aligned} \text{cloglog}(\eta_{i,t}) &= \log(\phi_t) + \log(K_{i,t}) \\ &+ \delta_t \log(A_{i,t-1}) \\ &+ \pi_t \log(P_i), \end{aligned} \quad (4)$$

where  $\text{cloglog}$  denotes the complementary log-log link function,  $\phi_t$  is the force of infection (which we modeled further as a function of climate; eq. [6]),  $K_{i,t}$  quantifies the epidemiological coupling (eq. [5]),  $A_{i,t-1}$  is the colony area (eq. [1]), and  $P_i$  is the primary productivity (kg/ha/year; see “Empirical Data”) of colony  $i$ . In order to investigate potential variation in the effect of area or productivity between years, we assumed that  $\delta_t \sim N(\mu_\delta, \sigma_\delta^2)$  and  $\pi_t \sim N(\mu_\pi, \sigma_\pi^2)$ .

Our measure of epidemiological coupling (fig. 3) not only accounts for the distance to the surrounding colonies that were infected by plague in the preceding year but also for the sizes of the infected colonies and for topographic relief and streams in the landscape between colonies. Moreover, this coupling accounts for infection originating from an unknown host at an unknown location (background infection). We investigate the potential effect of each of these factors by modeling the epidemiological coupling,  $K_{i,t}$ , of a colony at location  $i$  in year  $t$  as

$$K_{i,t} = e^{\bar{z}_t} + \sum_j e^{-\alpha D_{ij}} \alpha B_{j,t-1} A_j^{\gamma_t} \chi^{E_{ij}} \theta^{W_{ij}}, \quad (5)$$

where  $\bar{z}_t$  is the rate of background infection at time  $t$  (which we modeled further as a function of climate; eq. [7]),  $D_{ij}$  is the distance in kilometers between colonies at locations  $i$  and  $j$ , and  $\alpha$  is a parameter regulating the decay in the rate of plague transmission with distance. The second multiplication by  $\alpha$  improved model convergence (but does not change the likelihood). The mean dispersal distance of plague between colonies is  $1/\alpha$ . If colony  $j$  was infected by plague in year  $t-1$ ,  $B_{j,t-1}$  is 1; otherwise, it is 0. This means that only colonies that were infected in the preceding year are potential sources of infection. The motivation for using  $A_j$  in  $t-2$  is as in equation (3). The parameter  $\gamma_t$  scales the relation between colony area and infectiousness and has associated hyperparameters for investigating a potential variation between years in the effect of colony area (see table C1). The variable  $E_{ij}$ , a measure of topographic relief between colonies, is the number of times a straight line between colonies  $i$  and  $j$  crosses a 50-m elevation contour. The parameter  $\chi$  determines the proportional change in transmission rate for each elevation

contour between colonies  $i$  and  $j$ . Similarly,  $\theta$  determines the change in transmission rate per stream crossing, where the number of streams between colonies  $i$  and  $j$  is  $W_{ij}$ . We set  $E_{ij} = W_{ij} = 0$  for  $D_{ij} > 10$  km both for technical reasons and because the effect of the intervening topography and streams is relatively small across long distances. The second term in equation (5) is 0 for years following years when no plague occurrence was recorded (1992,  $t = 5$ ; 1998,  $t = 11$ ; 1999,  $t = 12$ ).

As stated above (eqq. [4], [5]), we modeled the force of infection ( $\log[\phi_t]$ ) and rate of background infection ( $e^{\bar{z}_t}$ ) further (fig. 3). We modeled them as  $\log(\phi_t) \sim N(\mu_\phi, \sigma_\phi^2)$  and  $e^{\bar{z}_t} \sim N(\mu_{\bar{z}_t}, \sigma_{\bar{z}_t}^2)$ , where their year-specific values ( $\mu_\phi$  and  $\mu_{\bar{z}_t}$ ) were potentially affected by climate (eqq. [6], [7]), and where the additional variation was accounted for by  $\sigma_\phi^2$  and  $\sigma_{\bar{z}_t}^2$ . More specifically,

$$\mu_{\phi_t} = \phi_0 + \rho_f(R_{t-1} - \bar{R}) + \psi_f(H_t - \bar{H}) + \Lambda_f(V_t - \bar{V}) \quad (6)$$

and

$$\mu_{\bar{z}_t} = \bar{z}_0 + \rho_b(R_{t-1} - \bar{R}) + \psi_b(H_t - \bar{H}) + \Lambda_b(V_t - \bar{V}), \quad (7)$$

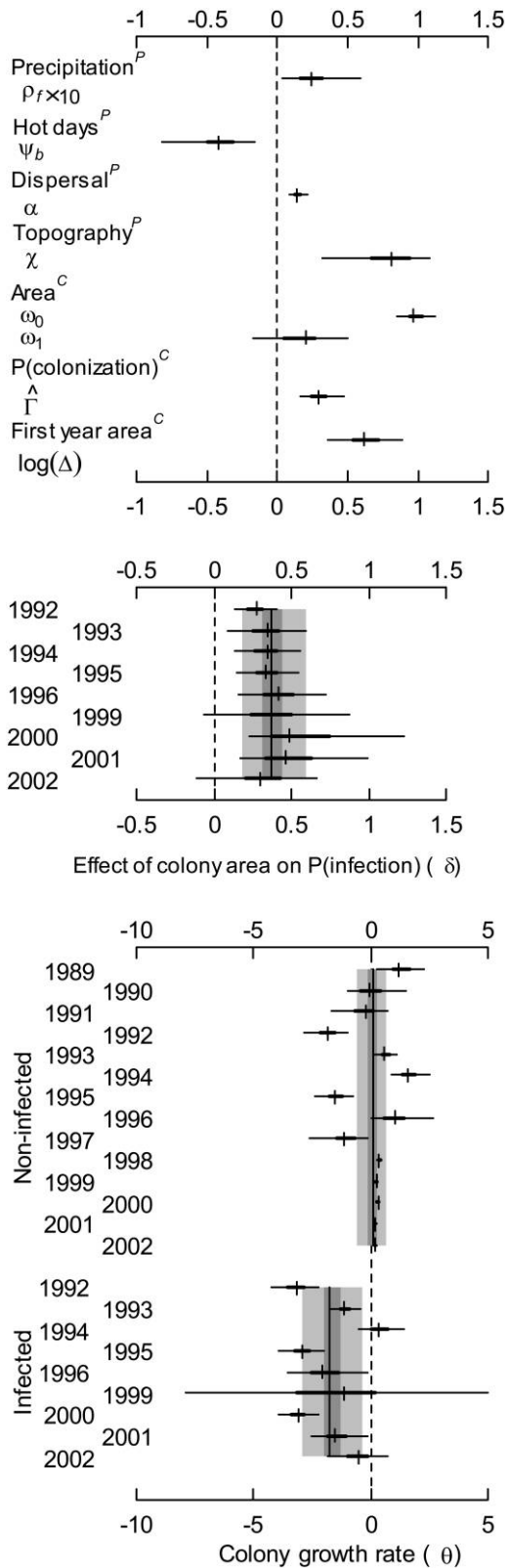
where  $R_{t-1}$  denotes the summed precipitation (in mm) April–July in year  $t-1$ . The variable  $H_t$  denotes the number of days  $>35^\circ\text{C}$  in year  $t$ , and  $V_t$  denotes the mean temperature March–May. We subtracted each observation of a climate variable from its mean ( $\bar{R} = 217$  mm;  $\bar{H} = 12.1$  days;  $\bar{V} = 5.9^\circ\text{C}$ ) to reduce parameter correlations. Parameters to be estimated are  $\bar{z}_0$ ,  $\phi_0$ ,  $\rho_{f,b}$ ,  $\psi_{f,b}$ , and  $\Lambda_{f,b}$ .

### Model Evaluation through Simulation

Three models were evaluated through simulation: the final spatial model described above, a nonspatial model assuming global dispersal, and a no-climate model ignoring climatic variation. In fitting the nonspatial model,  $\alpha$  was fixed at 0 and  $\chi$  at 1 (eq. [5]). In fitting the no-climate model, single  $\mu_\phi$  and  $\mu_{\bar{z}_t}$  were fitted and used in drawing values of  $\log(\phi_t)$  and  $e^{\bar{z}_t}$ .

In the simulations, we drew a new set of parameter values from the distributions defined by the hyperparameters for each year. Hence, year-specific estimates were not used. The simulations reflected parameter uncertainty: a new joint set of parameters was drawn from the posteriors for each of the simulation replicates. We wrote our own code for performing the simulations using R 2.1.1 (R Development Core Team 2005), with the add-on library *geOR* (Ribeiro and Diggle 2001).

Three simulations (each replicated 1,440 times) were



used to test the ability of the three models to predict and reconstruct the observed temporal dynamics over the period 1988–2002. Simulations started at the state observed in 1988, that is, with no colony infected. Note that plague was not observed in the landscape until 1992 and that plague data from 1992–2002 were used to fit the plague submodel. Hence, the models perform well if they predict no plague during 1988–1991, if they predict triggering of plague in 1992, and if they reconstruct data observed during 1992–2002. The performance of these three models was also compared by calculating, for each year, the proportion of samples from the posterior distribution (simulation replicates) in which the spatial model predicted the observed value better than the nonspatial and no-climate models. These comparisons were evaluated for three observed values: (1) number of infected colonies, (2) total number of colonies, and (3) total colony area.

In the fourth and fifth simulations, we tested the abilities of the spatial model and the nonspatial model to identify which colonies will become infected in a year of epizootics, based on the observed location, the area, and the infection status of colonies in the preceding year. We investigated model performance using the statistics sensitivity and specificity, both of which measure agreement between predicted and observed. In this case, sensitivity is the probability that a colony observed to become infected was also predicted to become infected, while specificity is the probability that a colony that did not become infected was not predicted to become infected. For this test, we simulated 1993–1994 because 1993 was the only survey year during severe epizootics.

### Results

Here we describe the final model and discuss several variables. The selection procedure is presented in appendix B, and the estimates of all 94 parameters of the spatial model and the nonspatial model (both including the colony submodel) are presented in table C2 in the online edition of the *American Naturalist*.

The rate of plague transmission in the landscape was clearly explained by climate. The force of infection, which

**Figure 4:** Parameter estimates for the spatial model of the joint dynamics of prairie dog colonies and plague. The 50% (thick horizontal lines) and 95% (thin horizontal lines) highest posterior density intervals (HPDIs) and modes (short vertical lines) are shown. The posterior distributions of hierarchical means are shown as shaded areas; 50% (dark gray), 95% (light gray) HPDI, and modes (long vertical line). Superscripts refer to the effect of the variable on probability of plague infection (P) or on colony dynamics (C); subscripts refer to noninfected (0) and infected (1) colonies;  $\rho$  has been multiplied by 10 for ease of illustration.

regulates the rate of transmission between infectious and susceptible colonies, increased with increasing summed precipitation during April–July in the preceding year; the 95% highest posterior density interval (HPDI) of  $\rho_f$  was greater than 0 (fig. 4). Temperature also affected transmission: the rate of infection from an unspecified background source,  $\Xi$ , decreased with the number of hot days in the current year (fig. 4). Spring temperature also explained the force of infection, but not when hot days and precipitation were included in the model.

Colony location and landscape heterogeneity also affected plague transmission. The mean annual plague dispersal distance,  $1/\alpha$ , was 6.9 km (HDPI 4.5–12.6 km; fig. 4). Transmission was retarded by topography, with a 20% reduction in transmission per 50-m elevation contour (mode of  $1 - \chi$ , fig. 4). Although the HPDI for  $\chi$  (0.29–1.09; fig. 4) slightly overlapped 1, the value defining no effect, we retained this effect because of the large variation in topography in the landscape (fig. 1). Crossings of streams, often located in ravines, did not affect the rate of plague transmission: the HPDI of  $\theta$  clearly overlapped 1.

Certain colony characteristics affected plague transmission. The probability of a colony becoming infected by plague increased with increasing colony area: the HPDI of  $\mu_s$  summarizing the yearly area parameters ( $\delta$ ) was  $>0$  (fig. 4). However, colony area did not affect subsequent infectiousness. Primary productivity also did not affect the probability of a colony becoming infected.

The joint model also allowed inference regarding prairie dog metapopulation dynamics. The persistence of non-infected colonies increased with increasing colony area up to a colony size of about  $e^2 = 7$  ha (fig. 5). Larger colonies persist with a high probability. The probability of persistence of infected colonies was lower (0.5–0.8; fig. 5, *light gray*), and any relation to area was much weaker (fig. 4). The widening of the confidence interval reflects sparse data—most colonies had an area of 7 to 150 ( $e^2$ – $e^5$ ) ha. The estimated time for recolonization of an extinct burrow system was 2.1–6.0 years ( $1/\hat{\Gamma}$  in fig. 4). Persisting, non-infected colonies generally increased in area between years; the HDPIs were positive for that period and the portion of the study with complete data (see  $\theta_{1998-2002}$  in fig. 4, CMR lands in fig. 1, and “Empirical Data”). However, colonies were also predicted to decrease in area in some years (negative  $\theta$ ). Although biologically reasonable, this predicted decrease may reflect the large uncertainty in colony growth during the first 10 years of the study ( $\theta_{1989-1997}$ ), when data were scarce (see “Empirical Data”). This annual variation leads to a posterior distribution of mean growth ( $\mu_\theta$ ) for noninfected colonies clearly overlapping 0 (fig. 4). In contrast, infected colonies that survived infection shrank on average by 85% (posterior mode

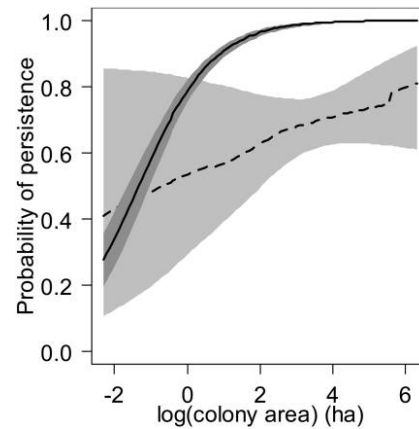


Figure 5: Probability of persistence of infected (*light gray*) and non-infected (*dark gray*) colonies as a function of colony area. Lines and colored areas show modes and 95% highest posterior density intervals, respectively, of predictions given by equation (3) using parameter estimates given in figure 4.

of  $1 - \exp[\mu_\theta]$  for infected colonies; fig. 4). Such declines significantly decreased the chance of colony persistence in the next year—they are likely to decrease in size to  $<7$  ( $e^2$ ) ha, below which persistence decreases (fig. 5, *dark gray*).

The simulations starting at year 1988 using the spatial model showed good fit to the data (fig. 2): the triggering of epizootics in 1992 was predicted, and the amplitude and fade-out of subsequent epizootics were reconstructed. More specifically, the number of colonies with plague and the total number and area of colonies observed were generally within the 50% HPDI of the predictions. During 2000–2002, when observed values were within the 75% HDPI, the observed effect of plague was negligible. There was no difference in the ability of the nonspatial model to predict the triggering of epizootics and to reconstruct the number of infected colonies, total number of colonies, and total colony area (see figs. C1, C2 in the online edition of the *American Naturalist*). Note, however, that the nonspatial model does not predict a decrease in total number of colonies during the epizootics of 1992–1995. The no-climate model showed inferior ability in predicting all three quantities (see figs. C2, C3 in the online edition of the *American Naturalist*).

The spatial model reconstructed well which colonies did not become infected in 1994, based on a simulation starting at the state observed in 1993 (fig. 1). Moreover, infections occurred among colonies with high predicted probabilities of infection. The median sensitivity was 0.38. There was a large uncertainty in this estimate (fig. C4 in the online edition of the *American Naturalist*), and this is explained by the between-year variation ( $\sigma^2$ ) accounted for in the force of infection ( $\log[\phi]$ ), background infection

( $\Xi$ ), and colony area ( $\delta$ ). A shortcoming of the model was that the predicted infection probabilities were high for many more colonies than those observed to get plague. This, in combination with few infected colonies observed in 1995, explains the relatively high estimate of median specificity, 0.88; predicting that a colony does not become infected is a good guess, since few colonies in fact did become infected.

The nonspatial model performed poorly in predicting which colonies became infected (median sensitivity = 0.09; fig. C4). The median specificity was high (0.96), with the same explanation as above. A comparison of the map showing the predictions by the spatial model (fig. 1) with a corresponding map for the nonspatial model (fig. C5 in the online edition of the *American Naturalist*) shows that in the latter, many colonies that were isolated from colonies that were infected in the preceding year had high predicted infection probabilities. These high predictions were due to the large sizes of these colonies (fig. C6 in the online edition of the *American Naturalist*).

### Discussion

We have successfully developed a spatially explicit model for the joint metapopulation dynamics of a host-pathogen system. Our results strongly suggest that the spatial transmission of plague is climate driven: precipitation affects the force of infection that regulates the spatial transmission, and temperature affects the rate of background infection. Two processes probably explain these dynamics. First, increased precipitation increases primary productivity, which is followed by increased rodent and flea density (Parmenter et al. 1999; Ensore et al. 2002; Yates et al. 2002; Collinge et al. 2005b). These affect the rate of spatially restricted plague dispersal over 10-km distances. Second, hot days reduce transmission throughout the landscape due to the negative effects of heat on fleas (Cavanaugh and Marshall 1972) and on flea-mediated transmission of *Yersinia pestis* (Ensore et al. 2002). This negative effect of hot days is stronger than the positive effect of warm spring days that has been shown to explain plague prevalence in central Asian great gerbils (Stenseth et al. 2006).

We have estimated both the effect of intercolony distance and the effect of landscape heterogeneity on plague transmission. The plague dispersal function predicts a mean dispersal distance of 4–12 km between years. This estimate is consistent with patterns of plague occurrence (fig. 5 in Cully and Williams 2001; fig. 3 in Stapp et al. 2004) and spatial genetic structuring of *Yersinia pestis* (fig. 2 in Girard et al. 2004) in other regions. Similarly, previous studies of plague occurrence patterns have shown effects of landscape heterogeneity (Girard et al. 2004; Collinge et

al. 2005a). Our spatially explicit model allowed us to quantify how topography reduced the transmission rate. It is crucial to account for these spatial landscape properties for predicting plague dynamics, as shown by the poor predictive ability of the nonspatial model. Moreover, knowing how topography reduces the transmission rate also suggests certain modes of intercolony transmission over others (see below).

The infection probability increased with colony size (area), but a colony's size did not affect its subsequent infectiousness. The positive effect of area on infection probability is consistent with some previous studies of plague occurrence (Cully and Williams 2001; Lomolino and Smith 2001; Collinge et al. 2005a). The most likely mechanisms are that the population size of putative reservoir hosts (small rodents) increases with increasing colony area, leading to increased contacts with infective hosts, and that large colonies attract more rodent-consuming predators that may carry infected fleas between colonies (Cully and Williams 2001). Stapp et al. (2004) found that intermediate-sized colonies were most likely to become extinct due to plague. By modeling infection and extinction separately, we can explain this observation as a combined effect: infection risk rises with colony size, while risk of subsequent extinction falls with colony size.

Plague strongly affects the long-term metapopulation dynamics of prairie dogs. Commonly, infected colonies are either extirpated or reduced in size. Both effects lead to colony loss, because smaller colonies are at greater risk from other factors. The resulting decrease in colony numbers (fig. 2), followed by recolonizations, allowed us to estimate the area of new colonies (table C2). Our estimate of time until recolonization is consistent with Webb et al. (2006), but our persistence estimate for infected colonies is much higher than the 2% they report. However, Webb et al. (2006) assume a maximum colony size of 200 individuals. The colonies we model are often larger and may behave differently. For example, some individuals in a large colony have been observed to escape epizootics due to landscape heterogeneity (Cully et al. 1997).

The estimated mean dispersal distance for plague (6.9 km) exceeds the observed maximum dispersal distance for prairie dogs (Garrett and Franklin 1988) by over 25%. Moreover, the rate of disease spread from a colony was not affected by its area. If prairie dogs were the main carrier, we would expect an increasing number of individuals infecting surrounding colonies with increasing area of the infected colony (Xia et al. 2004). Finally, the transmission rate decreased with increasing topographic relief (ravines between colonies). Ravines generally contain taller, more dense vegetation, which has been observed to facilitate rather than retard prairie dog dispersal (Garrett and Franklin 1988). Our results therefore suggest that prairie

rie dogs are not the main carrier of plague between colonies. It should, however, be noted that the longest dispersal event observed (5.5 km) was accomplished in <6 hours (Garrett and Franklin 1988). Hence, if the movements of individuals are unaffected by infection, prairie dogs may carry plague short distances. But what carries the disease across the landscape? Although our findings (effects of precipitation and colony area) suggest that small rodents play a role in plague transmission, it is unlikely that individual small rodents disperse 10 km/year, as we estimate for plague. Hence, if plague dispersal is solely by small rodents, then it must occur through a stepwise contact process between many individuals. Other likely plague carriers are large carnivores that can carry diseases tens of kilometers in a year (Smith et al. 2005). The foraging behavior of predators may explain the positive relationship between colony area and risk of infection (Cully and Williams 2001; Collinge et al. 2005a). Moreover, large carnivores often carry plague antibodies and have been observed to carry infected fleas in the vicinity of infected colonies. Long-distance transmission by carnivores does not necessarily reduce the importance of small rodents in overall transmission: increased numbers of infected rodents should increase contact rates and transmission events of plague to large carnivores. Clearly, we need to understand more about the roles of the different flea hosts in the population dynamics of *Y. pestis*.

Our simulations suggest that the spatial model for joint host-parasite dynamics can predict both long-term temporal and short-term spatial colony and plague dynamics. During epizootics, this model can be used to quantify the infection risk of specific colonies. Plague is not likely to occur among colonies predicted to have low infection risk. Among colonies predicted to have high infection risk, only a portion will become infected. However, by identifying colonies not at risk, management actions can be targeted more efficiently. It can be seen as a forecasting problem that data on the number of hot days cannot be obtained before the summer of plague transmission. However, hot days decrease the risk of infection equally for all colonies, so relative infection risks remain unchanged. Hence, several forecasts should be conducted before the summer, each assuming a different number of hot days. Precautions or intervention programs can then be updated throughout the period as hot days occur.

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