

Threshold Conditions for the Persistence of Plague Transmission in Urban Rats

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In this article we derive a mathematical expression characterizing the tendency for *Yersinia pestis*, the etiologic agent of plague, to become established in an urban rat population upon introduction, and evaluate this risk for several cities. The expression gives a threshold condition for the persistence of *Y. pestis* transmission in terms of measurable attributes of a local urban rat population: the average flea density and the rat colony size. If the local rat and flea populations exceed this threshold, plague circulation is predicted to continue; if not, it will burn out of its own accord. This expression may be used to evaluate both the vulnerability of a specific neighborhood and the effect of pest control strategies upon that vulnerability.

KEY WORDS: Bubonic plague; equilibrium model; threshold condition

1. INTRODUCTION

In September 2005, three mice infected with *Yersinia pestis* (the bacterium that causes plague) were reported missing from a laboratory in Newark, New Jersey. The NJ Health Commissioner and the Center for Disease Control issued assurances that the risk to the community was miniscule, based on the rapidity with which *Y. pestis*-infected mice die and the failure to find evidence of plague in human and animal populations in the vicinity of the laboratory.⁽¹⁾ Although a plague outbreak did not ultimately occur in the surrounding community, the episode raises questions about the ability of urban small mammal populations to support and sustain *Y. pestis* transmission. The proliferation of research laboratories around the country working on Category A pathogens, in addition to the possibility of intentional

introduction through bioterrorism, makes this question especially relevant.

Of all the urban rodents, rats are the species that, if infected with *Y. pestis*, would be the most worrisome. This is on account of their close association with humans, their colonial nesting and gregariousness, their susceptibility to plague, and the high vector efficiency of their fleas.⁽²⁾ Rat-borne human plague was briefly a serious problem in the United States in the first quarter of the 20th century, when *Y. pestis*-infected rats and fleas in American port cities caused scores of human fatalities.⁽³⁾ This introduction is believed to be responsible for the plague that is still circulating in rodent populations in the western United States, primarily among prairie dogs and ground squirrels.^(4–6)

Reports of plague in domestic rats are now rare.⁽⁷⁾ The characteristics of urban rat populations have changed over the past century. The burrowing Norway rat (*Rattus norvegicus*) has replaced the climbing black rat (*Rattus rattus*) in much of the country, while flea infestations on rats have declined in major northeastern U.S. cities.^(8,9) It is not clear how these changes have affected the urban rat's

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potential as a *Y. pestis* reservoir. Nelson⁽¹⁰⁾ warned that the rat populations of sections of Los Angeles County could be vulnerable to the reestablishment of plague if the disease were to cross the rural-urban boundary. Poland and Barnes, citing several 20th-century observations of plague in rats, concluded that there was a persistent possibility of urban murine plague.⁽⁷⁾ Given the potential severity of a plague epizootic and the dependence of *Y. pestis* transmission on local geographic and environmental factors,⁽¹¹⁾ a more quantitative assessment is needed to predict the outcome of an introduction of the bacteria into specific wild urban rat populations.

As we will demonstrate, the ability of a population of rats to maintain *Y. pestis* transmission can be characterized in terms of the rat environmental carrying capacity (defined here as the steady-state colony size), the flea index (the average number of fleas per rat), and biological attributes of both populations. Above a critical flea index, introduced plague will become enzootic, with an established and persistent presence in the rat population.

The concept of a density-dependent threshold for plague enzooticity is not new in the plague literature. Davis *et al.*⁽¹²⁾ observed that *Y. pestis* circulation in Kazakhstan depends on the abundance of its main reservoir host, the great gerbil (*Rhombomys opimus*). Several authors have suggested threshold values for the flea index above which plague will persist in a rodent population. Vera⁽¹³⁾ found that reducing the flea index below 0.01 (an average of 1 flea per 100 rats) eradicated plague in Ecuadorean towns with large rat infestations. Eisen *et al.*⁽¹⁴⁾ found a flea index of 7.8 to be sufficient to maintain plague circulation in an experimental system consisting of the Norway rat and the Oriental rat flea. Lorange *et al.*⁽¹⁵⁾ derived a threshold index of 4.7, based on MacDonald's⁽¹⁶⁾ threshold equation for malaria persistence. Traub⁽¹⁷⁾ reported a flea index of 3–5 as “critical” in plague. More generally, Lopez *et al.*⁽¹⁸⁾ developed a framework for mathematically analyzing threshold and stability conditions of host-vector interactions.

Building upon recent mathematical models of bubonic plague transmission in rodents,^(19–21) we use a transmission model to derive an analytic expression describing plague persistence in urban rat populations. We derive a critical flea index, denoted ϕ_{crit} (Equation (5)), which expresses plague circulation persistence potential as a function of colony carrying capacity K_r . This expression is mathematically

equivalent to the basic reproduction number of epidemiology, R_0 , classically defined as the number of secondary infections produced by a single infected individual in a susceptible population.⁽²²⁾ R_0 has the property that, for $R_0 < 1$, an introduction of disease into a community will burn out due to insufficient transmission. For $R_0 > 1$, the disease will persist in the community. In our model, for colonies whose flea index ϕ exceeds ϕ_{crit} , introduced plague will become enzootic. Conversely, if $\phi < \phi_{\text{crit}}$, disease transmission will cease of its own accord. The ϕ_{crit} expression allows locations with different infestation levels to use the same expression to estimate their vulnerability.

2. METHODS

2.1. *Y. Pestis* Transmission Model

In urban environments, rat populations exist in semi-isolated colonies occupying an area generally smaller than a city block. Although rats can venture beyond this home range, they tend to restrict their movements to well-known haunts within which they can readily identify reliable food sources and harborage while avoiding pedestrian and vehicular traffic. The rate of new individuals invading neighboring colonies has been observed to occur only about once in 66 days.^(23–25) Therefore, we treat the rats within a city block as an isolated population, with fully homogeneous interactions among themselves, but no ingress or egress.

We model a single rat colony; rats and fleas are classified into compartments according to their plague status— S (susceptible to infection), E (exposed but not symptomatic nor infectious), I (symptomatic and infectious), and R (recovered and immune). Mathematically, the compartments are continuous functions, as approximations of discrete individuals.

The model is described by a system of coupled differential (Equation (1)), with the subscript “r” denoting the rat class, and a subscript “f” denoting the flea class. The sizes of the rat and flea populations are: $N_r \equiv S_r + E_r + I_r + R_r$ and $N_f \equiv S_f + E_f + I_f + R_f$, and are not assumed constant.

The set of equations describing the model take the form of mass-action, with input and output flows from each disease state described dynamically over time (parameters are defined in Table I).

Table I. Parameter Values Used in the Calculation of the Critical Flea Density, ϕ_{crit}

Parameter Symbol	Literature Value (day)	Parameter Values (day ⁻¹)(Source)	Description	Distribution (Beta[ϵ_1 , ϵ_2 , min, max])
μ_r	60 30	0.017 ⁽⁴³⁾ 0.039 ⁽⁵²⁾	Rat natural death rate	Beta[1.5, 1.5, 0.015, 0.035]
α_r ^a	2–3 2 2	0.33 ⁽⁵³⁾ 0.39 ⁽⁴⁷⁾ 0.39 ⁽¹⁵⁾	Rat plague death rate	Beta[3.8, 2, 0.27, 0.42]
r_r		0.0011 ⁽¹⁹⁾	Rat plague recovery rate	Beta[1.5, 1.5, 0.001, 0.0012]
σ_r ^a		0.21 ⁽²¹⁾ 0.25 ⁽⁵³⁾ 0.33 ⁽³⁴⁾	(Rat exposure period) ⁻¹	Beta[1.5, 2, 0.19, 0.36]
μ_f		0.061 ⁽⁵⁴⁾ 0.070 ⁽²¹⁾	Flea natural death rate	Beta[1.5, 1.5, 0.06, 0.071]
α_f	2.8 2–5 4.4 4	0.30 ⁽³⁸⁾ 0.25 ⁽¹⁵⁾ 0.20 ⁽³⁴⁾ 0.22 ⁽⁴⁸⁾	Flea plague death rate	Beta[1.6, 2, 0.18, 0.32]
r_f	Derived in text	0.026 ⁽³⁹⁾ 0.0084 ⁽³⁴⁾ 0.00093 ⁽³⁸⁾	Flea plague recovery rate	Beta[1.5, 2.0, 0.0001, 0.03]
σ_f	9–28 14 12.6 16 15–21	0.053 ⁽⁵³⁾ 0.069 ⁽¹⁵⁾ 0.076 ⁽³⁴⁾ 0.061 ⁽⁴⁸⁾ 0.054 ⁽³⁸⁾	(Flea exposure period) ⁻¹	Beta[1.5, 2.0, 0.05, 0.082]
β_r ^a		0.25 ⁽³⁸⁾ 0.38 ⁽¹⁵⁾	Infection rate: Rat to flea	Beta[1.2, 1.2, 0.24, 0.39]
β_f ^a		0.09 ⁽³⁸⁾ 0.15 ⁽³⁴⁾ 0.11 ⁽⁴⁸⁾	Infection rate: Flea to rat	Beta[1.5, 1.9, 0.084, 0.17]
a		0.046 to 0.14 [<i>this article</i>]	Flea searching efficiency	Beta[1, 1, 0.04, 0.15]

^aThese parameters were not available for rats; experimental values were obtained from studies of prairie dogs, mice, or guinea pigs.

Notes: Beta distributions approximate the probability distributions around the parameter estimates. For parameters that were not directly available in the desired units, literature values given in (days) are listed, along with our conversion into (days)⁻¹.

$$\begin{aligned}
 \frac{dS_r}{dt} &= b_r S_r \left(1 - \frac{N_r}{K_r}\right) - \beta_f I_f \frac{S_r}{N_r} (1 - e^{-aN_r}) & \frac{dS_f}{dt} &= b_f N_f \left(1 - \frac{N_f}{\phi N_r}\right) - \beta_r S_f \frac{I_r}{N_r} (1 - e^{-aN_r}) \\
 \frac{dE_r}{dt} &= \beta_f I_f \frac{S_r}{N_r} (1 - e^{-aN_r}) - (\mu_r + \sigma_r) E_r & \frac{dE_f}{dt} &= \beta_r S_f \frac{I_r}{N_r} (1 - e^{-aN_r}) - (\mu_f + \sigma_f) E_f \\
 \frac{dI_r}{dt} &= \sigma_r E_r - (\mu_r + \alpha_r + r_r) I_r & \frac{dI_f}{dt} &= \sigma_f E_f - (\mu_f + \alpha_f + r_f) I_f \\
 \frac{dR_r}{dt} &= r_r I_r - \mu_r R_r & \frac{dR_f}{dt} &= r_f I_f - \mu_f R_f.
 \end{aligned} \tag{1}$$

Several assumptions have been made here. First and perhaps most importantly, rats reproduce under a logistic population limit $b_r S_r (1 - \frac{N_r}{K_r})$, where b_r is the birth rate. The limiting factor K_r , the environmental carrying capacity for rats, is affected by environmental factors such as food availability, shelter, and population density. This assumption, that rat populations approach an asymptotic upper bound when faced with resource constraints, is widely supported in the literature.^(19,26,27,28)

We make a similar logistic growth assumption for the flea population. The maximum number of fleas in the environment is given by the limiting factor ϕN_r ; that is, there is an average flea index ϕ , affected by environmental and biological characteristics, which is an upper asymptote for the actual number of fleas per host. The actual number of fleas in the environment, N_f , will fluctuate below the logistic limit ϕN_r . It is assumed that rats with large numbers of fleas will spend more time grooming, resulting in this asymptotic behavior of the flea count.⁽²⁹⁾

There are three potential transmission routes cited in the literature of plague in rodents: flea-bite, inhalation, and ingestion.⁽²¹⁾ In this article, a respiratory transmission term was not included, as direct contact transmission is an inefficient mechanism among rats.^(30–32) Some of the literature^(21,33) suggests that infection may be spread by cannibalism of plague-killed carcasses. Although this has been observed, the occurrence of cannibalism is sufficiently infrequent relative to flea exchange that the ingestion route can be neglected.^(32,34)

Transmission by flea bite is described in a hybrid frequency-dependent fashion, where the probability of infection is primarily dependent on the percentage of the rat population infected, and to a lesser degree on the absolute size of the population.

For every susceptible flea, the probability that it will be able to find a new host following ejection from its current one is given by the exponential form, $(1 - e^{-aN_r})$,^(19,21) here a is a calibration constant corresponding to the likelihood of encountering a new host. This exponential term states that, for a small rat population of size N_r , fleas will have difficulty finding a new host; if N_r is large then this exponential form will be close to 1, indicating a high likelihood of fleas finding a new host.^(19,21) Upon finding a new rat host, there is an $\frac{I_r}{N_r}$ probability that the host is infectious. Then, there is a β_r probability for transmission to occur from the rat to the flea. Applying these probabilities to each of the S_f susceptible fleas yields a system-

wide flea transmission rate of $\beta_r S_f \frac{I_r}{N_r} (1 - e^{-aN_r})$.⁽¹⁹⁾ An analogous treatment is applied to determine the transmission rate from infectious fleas to susceptible rats.

2.2. Derivation of ϕ_{crit}

The threshold $R_0 < 1$ is mathematically equivalent to stability of the disease-free equilibrium (DFE) of Equation (1).⁽³⁵⁾ The DFE is defined as:

$$\begin{aligned} S_r &= K_r, S_f = \phi K_r \\ R_r &= E_r = I_r = R_f = E_f = I_f = 0 \\ \frac{dS_r}{dt} &= \frac{dS_f}{dt} = \frac{dE_r}{dt} = \frac{dE_f}{dt} = \frac{dI_r}{dt} = \frac{dI_f}{dt} = \frac{dR_r}{dt} = \frac{dR_f}{dt} = 0. \end{aligned} \quad (2)$$

The stability of this equilibrium can be determined by linearization (taking the Jacobian matrix) of Equation (1). The condition of negative real parts of the eigenvalues of this linearization, evaluated at the DFE, corresponds to $R_0 < 1$.⁽³⁵⁾ The characteristic polynomial of this linearization (the Jacobian determinant of Equation (1)) can be written in the form:

$$\sum_{i=0}^7 a_i \lambda^{7-i} = 0, \quad (3)$$

with $a_0 > 0$. Here, the a_i s are algebraic combinations of the parameter coefficients in Equation (1), while λ represents the eigenvalues. The Routh-Hurwitz theorem⁽³⁶⁾ provides conditions on the a_i s for which all roots of λ will have negative real parts:

$$\begin{aligned} |a_1| &> 0 \\ \begin{vmatrix} a_1 & a_3 \\ a_0 & a_2 \end{vmatrix} &> 0 \\ \begin{vmatrix} a_1 & a_3 & a_5 \\ a_0 & a_2 & a_4 \\ 0 & a_1 & a_3 \end{vmatrix} &> 0 \\ \begin{vmatrix} a_1 & a_3 & a_5 & \cdots & 0 \\ a_0 & a_2 & a_4 & \cdots & 0 \\ 0 & a_1 & a_3 & \cdots & 0 \\ \vdots & & \ddots & \ddots & \vdots \\ \cdots & \cdots & \cdots & \cdots & a_n \end{vmatrix} &> 0. \end{aligned} \quad (4)$$

Solving these determinant inequalities for Equation (1) is tedious but straightforward, and yields the

critical flea index ϕ_{crit} , such that $0 \leq \phi < \phi_{\text{crit}}$ satisfies Equation (4). ($\phi \geq 0$ is true by definition.)

$$\phi_{\text{crit}} = \frac{(\mu_r + \alpha_r + r_r)(\mu_f + \alpha_f + r_f)(\sigma_f + \mu_f)(\sigma_r + \mu_r)}{\beta_r \beta_f \sigma_r \sigma_f (1 - e^{-aK_r})^2} \quad (5)$$

If $\phi > \phi_{\text{crit}}$ (and hence, $R_0 > 1$), Equation (4) will not be satisfied, implying at least one positive root of Equation (3) and hence instability of the disease-free equilibrium. This implies that the system will move away from the DFE following a small perturbation; an introduction of plague will become enzootic.

For mathematical rigor, it is important to demonstrate the global asymptotic stability (GAS) of the DFE for $R_0 < 1$. Because the conditions of Equation (4) pertain to a first-order linearization about the DFE, it is not immediately obvious that this stability holds far from the DFE; e.g., for a nonnegligible number of infected rats or fleas in the environment. However, if the system has GAS about the DFE, the ϕ_{crit} condition will hold for any number of susceptible, exposed, infected, or recovered rats and fleas. GAS can be proven by applying a theorem developed by Castillo-Chavez *et al.*⁽³⁷⁾ It is straightforward to demonstrate that our model meets Castillo-Chavez's criteria, so further details are not included here.

2.3. Parameter Derivations and Sensitivity

As can be seen, Equation (5) is very sensitive to the parameter values. The transmission coefficients β_r and β_f are inversely proportional to the value of ϕ_{crit} . Conversely, it can be seen from Table I that α_r greatly exceeds μ_r and r_r in magnitude, as does α_f to μ_f and r_f . This leads to a nearly linear correlation between the magnitudes of the α s with ϕ_{crit} . To ameliorate this model stiffness and to represent uncertainty in the literature over the parameter values, we carefully defined probability distributions across all available parameter estimates (Table I). We chose to use beta distributions in all cases.

The birth rates b_r and b_f of Equation (1) do not appear in Equation (5), and are not included in Table I.

We made standard assumptions⁽¹⁶⁾ in the derivation of model parameter values from data (Table I). For example, Eskey and Haas⁽³⁸⁾ reported an average survival time of 2.8 days for infectious fleas. To

calculate the infectious flea death rate, α_f , we define p as the daily probability of an infectious flea surviving, such that $1 - p = \alpha_f$, the daily plague-induced mortality rate of infected fleas. We assume that the survival time follows an exponential distribution with parameter λ , so that $F(t) = 1 - e^{-\lambda*t}$ is the cumulative probability of the flea surviving up to t days after becoming infectious, with the expected value of survival time $E[t] = \frac{1}{\lambda}$. Thus, the daily death rate equals the cumulative probability of surviving 1 day or less, $\alpha_f = F(1)$. Therefore $p = 1 - F(1) = e^{-\lambda} = e^{-\frac{1}{E[t]}}$, so $\alpha_f = 1 - e^{-\frac{1}{E[t]}}$. Since the average survival time $E[t] = 2.8$ days, $\alpha_f = 0.30$.

The flea recovery rate r_f was estimated from observations in several studies. Engelthaler *et al.*,⁽³⁹⁾ studying *Y. pestis* infection in fleas, observed that by week 6, 40% (10/25) of the infected *X. cheopis* remained infected. We define $p_f = 1 - r_f$ as the daily probability that an infectious flea has not recovered. Assuming, as before, that p_f follows an exponential distribution ($p_f = e^{-\lambda*t}$), the expression $1 - F(t) = e^{-\lambda*t}$ gives the probability that an infected flea will not recover at least until time t . Thus, solving $e^{-\lambda*35} = 0.4$ (with the 35th day marking the start of week 6, and with the observation of 40% of fleas remaining infectious) gives a value of λ , which can be used to calculate the daily recovery rate $r_f = 1 - p = 1 - e^{-\lambda*1} = 0.026$. There were also two older studies of flea recovery. Burroughs⁽³⁴⁾ observed that, 39 days after infection, 28% of fleas had cleared their infection. Using the same method, we obtain a second r_f estimate of 0.0084. Eskey and Haas⁽³⁸⁾ reported 4% flea recovery over 44 days, yielding an r_f estimate of 0.00093.

We estimated values for the parameter a , the flea searching efficiency, based on rat behavior. Urban rat foraging is largely channelized in routes between sources of food and nests to such an extent that they create rat runways with evidence of heavy rat traffic.⁽²⁶⁾ The areas in which fleas would most likely be shed are the nest, the food source, and the runways, which are spatially quite small. Furthermore, it has been observed that poisoned rats retreat to the nest to die,⁽⁴⁰⁾ a behavior that, if also typical of *Y. pestis*-infected rats, would result in very efficient flea searching. Therefore, we estimate that the number of rats in a city block that would be required to give a questing flea a 75% probability of finding a host is between 10 and 30 rats. Solving $(1 - e^{-aN_r}) = 0.75$ for a across these host population sizes yields values of a from 0.046 to 0.14.

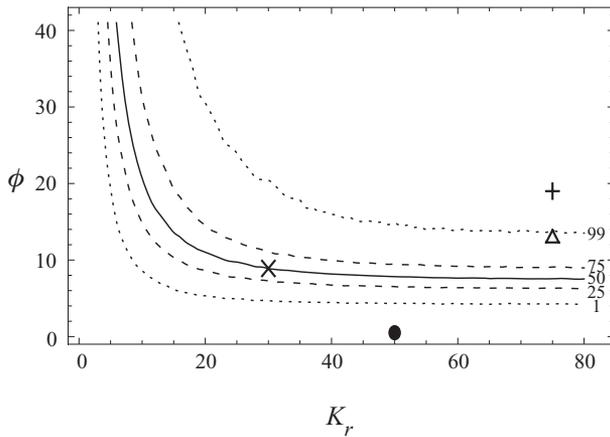


Fig. 1. Plot of the critical flea index ϕ_{crit} across a range of rat carrying capacities (K_r), with probability contours. The center curve shows the median value of Monte Carlo simulations; the dashed lines correspond to the 1st, 25th, 75th, and 99th percentiles. Four rat populations are located in this space, representing an industrial section of Los Angeles, CA (+),⁽¹⁰⁾ two mixed use retail/commercial areas in downtown Los Angeles (Δ , \times),⁽⁴⁰⁾ and an inner-city Baltimore, MD neighborhood (\bullet).⁽⁴³⁾

3. RESULTS

In Fig. 1, we plot ϕ_{crit} in flea-index/rat-carrying-capacity space. To locate an urban neighborhood on the threshold plot, field work is required, namely, measuring the rat carrying capacity and the flea index. Censuses of rat populations are typically conducted by lethal trapping, although hasty estimates may also be obtained through surrogate indicators (droppings, tracks, burrows). The flea index is determined by combing the pelts of the captured euthanized rats and counting the fleas.^(41,42)

Four rat-infested neighborhoods are plotted in the flea-index/carrying-capacity space of Fig. 1. It is estimated that the rat carrying capacity of one of the most rat-infested sections of Baltimore, MD is, at most, 50 rats per colony,⁽⁴³⁾ The associated flea index is on the order of 0.001,⁽⁴³⁾ so Baltimore has essentially zero risk of introduced *Y. pestis* becoming established in rats. Baltimore plots well below the critical flea index curve. New York City rats have comparable densities and flea burdens,⁽⁴³⁾ and comparably low potential for sustaining *Y. pestis* circulation.

The flea index of rats in a mixed commercial/industrial area of downtown Los Angeles varies dramatically with the seasons, and has been observed to be as high as 19 in late summer.⁽¹⁰⁾ *Y. pestis*, once introduced, would tend to be stably transmitted between the rats of this neighborhood, in accordance

with a more qualitative assessment by Nelson,⁽¹⁰⁾ at least while the flea index remained high. Since these flea populations plunge in winter, the enzootic phase should be short-lived.

Nelson's 1986 paper⁽¹⁰⁾ did not report rat colony size. We assume that it would have resembled current rat colonies in L.A., containing 75–100 individuals.⁽⁴⁰⁾ This does not have a strong effect on the plague stability predictions, on account of the asymptotic behavior of ϕ_{crit} as K_r increases. Two additional, recent observations⁽⁴⁰⁾ of downtown Los Angeles rat populations are also shown in Fig. 1. One neighborhood plots well above the threshold, with 75 rats and a flea index of 12.9; the other plots approximately at the 50% contour threshold, with 30 rats and a flea index of 8.9.

4. MODEL LIMITATIONS, ASSUMPTIONS, DISCUSSION

In this article, we derive a threshold model that predicts whether any given rat and flea system will become disease-free after the introduction of plague. The behavior of the system is predicted by two observations taken in the predisease state: rat carrying capacity K_r and flea index ϕ . As noted in Section 2, when the threshold ϕ_{crit} is exceeded, the disease-free state is unstable; as a result, an introduction of *Y. pestis* will cause the system to evolve towards the enzootic equilibrium. This behavior is dependent solely on the parameters in Equation (5) and on the specific values of K_r and ϕ . Fluctuations of rat and flea populations after the introduction of disease (as a result of the increased death rate) do not affect the prediction, as the disease-free equilibrium would still be unstable.

Rat-flea populations tend to peak and fall seasonally; as a result, the location of an environment on Fig. 1, along with the plague risk, will change over the year. It is important to bear in mind that the environments plotted in Fig. 1 represent a snapshot of plague risk at the time the data were collected, before the introduction of *Y. pestis*.

Urban rats often carry several species of fleas at once.^(44,45) Though *Xenopsylla cheopis*, the Oriental rat flea (used in our parameterization), is the most efficient plague vector, any flea capable of piercing skin can transmit *Y. pestis*, albeit less efficiently.^(34,46,47) This parameterization makes our model predictions conservative, representing a worst-case scenario.

There are several historical surveys of flea indices during plague outbreaks that can be compared

with our model predictions. Although rat carrying capacity estimates are not available for any of these sources, it is reasonable to believe that rat carrying capacity would be sufficiently high (≥ 60 in a typical environment) that the data would lie in the asymptotic region of the threshold plot. Olson⁽⁴⁹⁾ reported a flea index of 8.3 in the Vietnamese city of Nha-Trang during a 1966 plague outbreak. Macchiavello,⁽⁵⁰⁾ surveying the 1945 bubonic plague outbreak in Tumbes, Peru, reported a flea index of 11.3 for rats “in the epizootic foci,” and 2.1 for rats in the rest of the town. Carrion⁽⁵¹⁾ reported a flea index of 7.1 for the rats of San Juan, Puerto Rico. Although plague was not ongoing at the time of Carrion’s survey (1926–1929), there were outbreaks in 1912 and 1921. These data are plotted on the transmission plot in Fig. 2, demonstrating that our threshold model is consistent with these historical observations.

One limitation of our model is the literature estimates of flea transmission probability. Burroughs⁽³⁴⁾ observed that: “*In nature fleas have an opportunity, except in deserted burrows, to feed at will, and may frequently remain on the host for long intervals . . . Had these blocked fleas [in the experiment] been on a host for longer periods of time than permitted at laboratory feedings, they might have transmitted more frequently.*”

Burroughs explicitly names his own study, along with those of Eskey and Haas⁽³⁸⁾ and Wheeler and

Douglas,⁽⁴⁸⁾ as being vulnerable to this phenomenon. As these studies are our primary references in parameterizing β_f and β_r , and we are not aware of any alternative studies that compensate for this error, the underestimation of transmission probability that Burroughs cites results in our expression of ϕ_{crit} potentially overestimating the critical flea index. A more accurate estimate of transmission probability may lower our ϕ_{crit} contour plot. Furthermore, these laboratory experiments were conducted on mice^(34,48) and guinea pigs⁽³⁸⁾ rather than rats.

To account for uncertainty, we estimated probability distributions for each of the model parameters (Table I). The probability contours around ϕ_{crit} in Fig. 1 are the result of 10,000 Monte Carlo realizations for each K_r value, sampling from these parameter distributions. This represents our confidence in the parameter estimates. When evaluating a city’s rat population, evaluating its ϕ relative to a probability contour below the median ϕ_{crit} introduces a safety factor to compensate for parameter uncertainty.

If the probability of a plague introduction to a community is considered to be high, we imagine that city sanitation officials might use Fig. 1 to identify neighborhoods with elevated transmission risk to help them allocate pest-control resources. Furthermore, our analysis provides quantitative benchmarks for determining the adequacy of their efforts to reduce the risks.

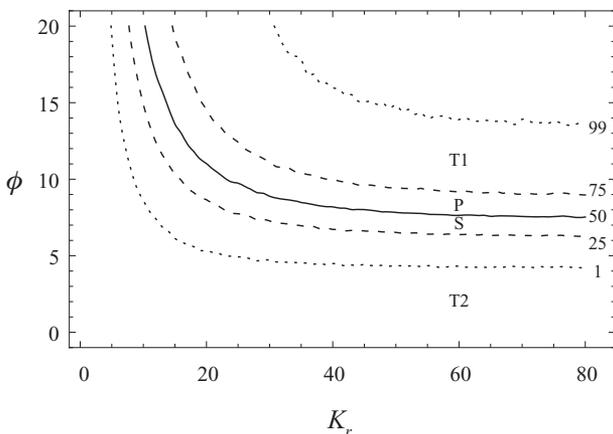


Fig. 2. Enzootic threshold plot with flea indices from several past plague outbreaks. Since rat carrying capacities were not recorded, we locate all points at “ K_r ” = 60 rats. “P” is the flea index measured in a 1966 plague outbreak in the Pleiku area of Vietnam.⁽⁴⁹⁾ “S” indicates the flea index during the late 1920s in plague-prone San Juan, Puerto Rico.⁽⁵¹⁾ “T1” and “T2” are flea indices from two neighborhoods of Tumbes, Peru in 1945, where “T1” experienced a plague outbreak, and “T2” did not.⁽⁵⁰⁾

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