Supporting Information

George et al. 10.1073/pnas.1010875108

SI Methods

Estimation of Transmission Rates from Serology Data. We modified our methodology for estimating nest survival (1) to estimate the rate of seroconversion in a bat rabies system. From this we can determine parameter values for transmission. Serology data were obtained from a 5-y study on bat rabies virus in big brown bats in Fort Collins, CO, and the methodology for their collection is described elsewhere (2–4). The utility of this method centers on dealing with data that come from repeated sampling events. The time between sampling events is considered in the estimation of the parameter as follows:

$$P(Y = y|p) = (p^t)^y(1-p^t)^{1-y},$$

where y is the serological state of the sample ($y = 1$ for positive or $y = 0$ for negative), p is the daily survival probability, and t is the time interval between sampling events. So the likelihood function becomes

$$\prod_{t\in T}(p^t)^y(1-p^t)^{1-y}. \qquad [S1]$$

Ultimately, using standard maximum-likelihood methods, one can estimate p.

Additionally, p was further interpreted for the bat rabies virus (BRV) system within the context of a simple death process where $p = \exp(-\theta t)$. If we think of seroconversion as a simple death process that describes how individuals move from seronegative to seropositive, then we can provide more structure to p. Following Renshaw (5), we can characterize a simple death process (transition from seropositive to seronegative in this case) as follows:

$$q(t+h) = q(t)(1-\theta t),$$

where

$q(t + h)$ = Pr (it is alive at time t and does not die in the subsequent small time interval h).

On letting $h \to 0$:

$$\frac{dq(t)}{dt} = -\theta q(t),$$

which solves as

$$q(t) = exp(-\theta t)$$

and

$$\begin{aligned} p(t) &= Pr(organism\ is\ dead\ by\ time\ t) \\ &= 1-q(t) \\ &= 1-exp(-\theta t) \end{aligned}$$

Thus, in onr situation,

$$p = exp(-\theta t),$$

and this gives us the following likelihood function:

$$\prod_{t\in T}[\exp(-\theta t)]^y[1-\exp(-\theta t)]^{1-y} \qquad [S2]$$

We were able to estimate θ in a Bayesian framework in WinBUGS using likelihood Eq. S2, giving us the estimate θ = 0.00465, which corresponds to a rate of something happening approximately every 214 d.

We gave further definition to θ by considering which classes in the model can be considered seronegative and seropositive. The infectious and recovered individuals are considered seropositive, and all others will be seronegative. We ean reasonably exclude considering the pathway to infectious individuals when estimating the rate of seroconversion (Fig. 4*A*) beeause these individuals are short-lived and most likely will not be captured and represented in the data. Thus, for a seronegative bat to become a seropositive bat, it must follow the pathway from susceptible to recovered individual that is eomprised of the following rates:

Thus, $\theta = (1 - p)\beta + \sigma_R = 1/214$.

Because we have independent estimates of the per capita per day immunity rate for BRV, $\sigma_R = 1/24$, we can further bound our θ estimate by subtracting the immunity rate, which gives $(1-\rho)\beta = 1/119$.

Alternative Model Structures for the Early Transmission Season. We considered different model structures for the early transmission season. We considered that the early transmission season was no different from hibernation in terms viral activity. Thus, none of the disease classes changed during the early transmission season.

$$\begin{aligned} \frac{dS_j}{dt} &= -\mu_{pj}S_j \\ \frac{dL_{Rj}}{dt} &= -\mu_{pj}L_{Rj} \\ \frac{dL_{Ij}}{dt} &= -\mu_{pj}L_{Ij} \\ \frac{dR_j}{dt} &= -\mu_{pj}R_j \\ \frac{dI_j}{dt} &= -\upsilon I_j \end{aligned}$$

An alternative form of early transmission season was also considered to test the importance of this season to overall pathogen dynamics. It assumes that rabies transmission does not occur during the early transmission season, but other viral activity does at a slower rate because of greater use of daily torpor and cooler temperatures in the spring. This version is characterized as follows:

$$\begin{aligned} \frac{dS_j}{dt} &= \mu_{pI}S_j \\ \frac{dL_{Rj}}{dt} &= -\sigma_{pR}L_{Rj} - \mu_{pj}L_{Rj} \\ \frac{dL_{Ij}}{dt} &= -\sigma_{pI}L_{Ij} - \mu_{pj}L_{Ij} \\ \frac{dR_j}{dt} &= \sigma_{pR}L_{Rj} - \mu_{pj}R_j \\ \frac{dI_j}{dt} &= \sigma_{pI}L_{Ij} - \upsilon I_j \end{aligned}$$

Last, we considered models without density dependence in the transmission season. All of these models behaved qualitatively similar to those presented in the main text. The major difference was that in some regions of parameter spaee there was exponential population growth. However, this does not change the major findings of the paper.

1. Stanley TR (2000) Modeling and estimation of stage-specific daily survival probabilities of nests. *Ecology* 81:2048–2053.
2. Wimsatt J, O'Shea TJ, Ellison LE, Pearce RD, Price VR (2005) Anesthesia and blood sampling of wild big brown bats (*Eptesicus fuscus*) with an assessment of impacts on survival. *J Wildl Dis* 41:87–95.
3. Pearce RD, O'Shea TJ, Shankar V, Rupprecht CE (2007) Lack of association between ectoparasite intensities and rabies virus neutralizing antibody seroprevalence in wild big brown bats (*Eptesicus fuscus*), Fort Collins, Colorado. *Vector Borne Zoonotic Dis* 7 489–495.
4. Shankar V, Bowen RA, Davis AD, Rupprecht CE, O'shea TJ (2004) Rabies in a captive colony of big brown bats (*Eptesicus fuscus*). *J Wildl Dis* 40:403–413.
5. Renshaw E (1991) *Modelling Biological Populations in Space and Time* (Cambridge Univ Press, New York).

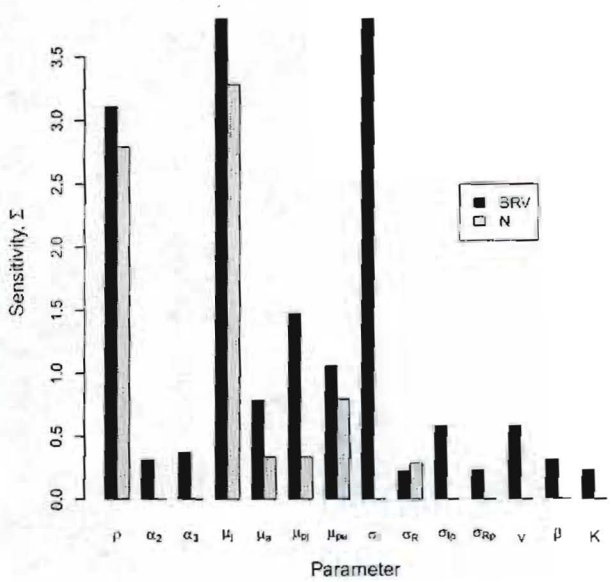

Fig. S1. Analysis determining how sensitive model output, probability of pathogen extinction (BRV), and probability of bat population extinction (N) were to changes in all model parameters. The *y* axis represents absolute values of sensitivity.

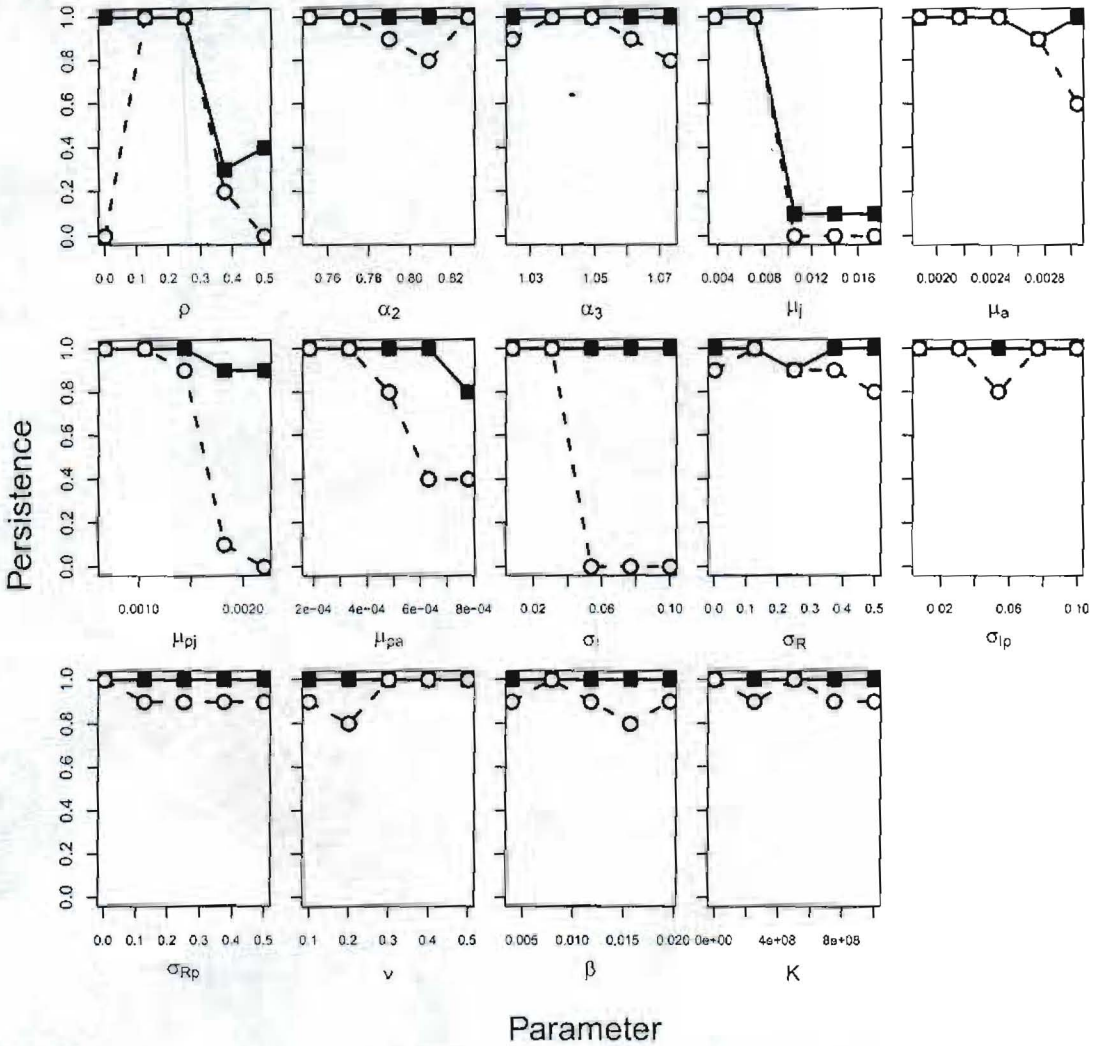

Fig. S2. Model sensitivity (percent of simulations that have bat population or rabies virus maintenance) for all model parameters: proportion exposed that become infectious (ρ), reproductive rate of first year adults (α_2), reproductive rate of adults >1 y (α_3), juvenile natural mortality rate in the main and early transmission season (μ_j), adult natural mortality rate in the main and early transmission season (μ_a), juvenile natural mortality rate during hibernation (μ_{pj}), adult natural mortality rate during hibernation (μ_{pa}), incubation rate in the main transmission season (σ_I^{-1}), immunity rate in the main transmission season (σ_R^{-1}), incubation rate in the early transmission season and hibernation (σ_{Ip}^{-1}), immunity rate in the main transmission season and hibernation (σ_{Rp}^{-1}), disease-induced morality rate (ν^{-1}), transmission rate (β), and carrying capacity (K). Solid squares (■) represent the proportion of simulations with persistent bat populations. Open circles (○) represent the proportion of simulations with rabies maintained in the bat population.

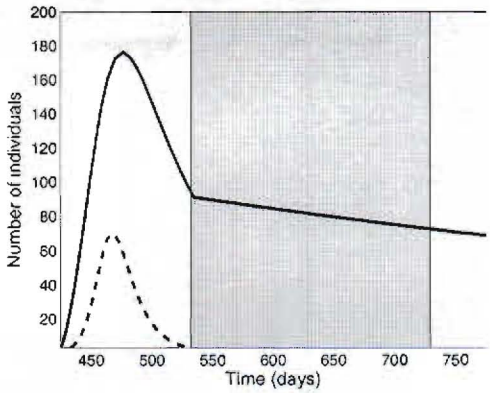

Fig. S3. Deterministic dynamics of exposed individuals that will become infectious evaluated for short (dashed line, 1/0.055) and long (solid line, 1/0.007) incubation periods, σ_I. The plot covers all three seasons of the model: main transmission (white), hibernation (dark gray), and early transmission (light gray). The shorter the incubation period the greater the chance of epizootic fadeout, because fewer exposed individuals enter hibernation and carry the pathogen to the next birth pulse.