

Appendix G Calculation of the basic reproductive ratio R_0

We partitioned the projection matrix \mathbf{A} , assuming no removals, into a transition matrix, \mathbf{T} , and a disease production matrix, \mathbf{D} . To simplify notation, we are dropping the subscript t from \mathbf{A} because we will subsequently linearize it, making it time invariant. The elements of the matrix \mathbf{T} give the probabilities that an animal in state i at time t is alive in state j at time $t + 1$ and is defined by the matrix \mathbf{A} without the top row. The fundamental matrix $(\mathbf{I} - \mathbf{T})^{-1}$ quantifies the expected amount of time that an individual spends in each stage. The elements of disease production matrix \mathbf{D} ($d_{(i,j)}$) give the number of new, infected individuals in stage i produced by infectious individuals in stage j during one time step. We excluded males, including juvenile males, from the elements of \mathbf{D} because they can never become infectious. The next generation matrix of the disease, \mathbf{G} , is calculated as $\mathbf{G} = \mathbf{D}(\mathbf{I} - \mathbf{T})^{-1}$. The elements of \mathbf{G} are the expected lifetime production of new infections (that can become infectious) by an individual in stage j over the duration of its infectious lifetime. (Allen and van den Driessche, 2008) and Oli et al. (2006) assert that the dominant eigenvalue of \mathbf{G} estimates R_0 .

This assertion is true for strictly linear models where transmission, and hence \mathbf{T} , can be treated as time-invariant. In this special case, the number of infections created by a single infected individual is itself a constant and does not depend on the infection status of the population. However, nonlinear dynamics are the hallmark of models of infectious disease, and we include these dynamics here (main text equation 4), which means that the probability of transmission depends on the state of the population, and hence, changes with time. Nonlinearity requires that we specify conditions for the matrix \mathbf{G} consistent with the definition of R_0 , the number of new infections created by a single infectious individual amidst a large population of susceptibles. Expressions for R_0 can be obtained symbolically (e.g, Oli et al., 2006) for simple models. However, to obtain a numerical estimate of R_0 for a high dimension matrix like \mathbf{A} , we must assume a population size of susceptible individuals. We choose 3000 because this choice is

consistent with management targets for abundance of bison in Yellowstone, but it turns out the results of the estimation of R_0 are quite insensitive to this assumption over a broad range of plausible values¹. To estimate the probability that a susceptible animal is exposed (ϕ^*) when there is a single infectious individual alive during the infectious period in a population totaling 3000, we used

$$\phi^* = 1 - \exp\left(\frac{-\beta}{3000\omega}\right), \quad (\text{G.1})$$

where ω is the proportion of the population that was juvenile or female, estimated from the stable age distribution of a healthy population experiencing exponential growth. The vector ω is needed because we exclude adult males from the calculation of frequency dependent transmission (equation 4). The stable age distribution for females was calculated as the normalized, dominant eigenvector of \mathbf{A} assuming recruitment rates for all adult females = $f_n/2$, $m = 0$, no management removals and $\phi_{(t)} = 0$. Thus, the quantity ϕ^* is the probability that a susceptible animal will become infected via horizontal transmission when there is a single infectious individual in an otherwise entirely susceptible population of 3000 bison.

The disease transmission matrix, \mathbf{D} , contains all zeros except for six entries

$(d_{(4,6)}, d_{(5,6)}, d_{(6,6)}, d_{(4,7)}, d_{(5,7)}, d_{(6,7)})$ specifying the number of new infections of type i created by a type j individual. To calculate the number of infected individuals created by a single individual the infected and infectious stage ($n_{(6)}$) we first calculated the probability that a susceptible animal would become infected from contact with a single infectious individual alive during the infectious period using:

$$\phi^* = 1 - \exp\left(\frac{-\beta}{\sum_{i=1}^3 n_i}\right) \quad (\text{G.2})$$

The denominator contains only female classes 1-3 because the entire population is susceptible.

The number of new infected juveniles produced by a single $n_{(6)}$ individual via horizontal and

¹Changing the assumed population size from 3000 to 5000 or from 3000 to 1000 changed the estimated value of R_0 by <.2%.

vertical transmission during one year was

$$d_{(4,6)} = (1 - m)(f_c v + \phi f_n p_{(2)} n_{(3)}^*) \quad (\text{G.3})$$

We include the term $(1 - m)$ because we exclude juvenile males that never become infectious. The number of new yearling infections produced annually via horizontal transmission from a single $n_{(6)}$ individual was

$$d_{(5,6)} = \phi^* p_{(1)} n_{(1)}^*, \quad (\text{G.4})$$

and the number of new infected and infectious individuals was

$$d_{(6,6)} = \phi^* p_{(2)} \left(n_{(2)}^* + n_{(3)}^* \right). \quad (\text{G.5})$$

We multiplied the right hand side of each of equations G.3 - G.5 by ψ and substituted f_p for f_c in equation G.3 estimate the contribution of a recrudesced individuals to new infections,

$$d_{(4,7)}, d_{(5,7)}, d_{(6,7)}.$$

To estimate R_0 , we substituted ϕ^* for $\phi_{(t)}$ in \mathbf{A} to obtain the linear projection matrix \mathbf{A}^* , partitioned \mathbf{A}^* into \mathbf{D} and \mathbf{T} to find \mathbf{G} , and numerically estimated the dominant eigenvalue of \mathbf{G} . We estimated the posterior distribution of R_0 by randomly sampling the output from the MCMC procedure (described in the section Parameter estimation), taking a single draw of the parameters of \mathbf{A}^* at each iteration to calculate a single value of R_0 . An accumulation of 30,000 of these values was used to describe the probability distribution of R_0 .

Literature Cited

- Allen, L. J. S. and P. van den Driessche. 2008. The basic reproduction number in some discrete-time epidemic models. *Journal of Difference Equations and Applications* **14**:1127–1147.
- Gall, D. and K. Nielsen. 2004. Serological diagnosis of bovine brucellosis: a review of test

performance and cost comparison. *Review of Science and Technology* **23**:989–1002.

Gall, D., K. Nielsen, L. Forbes, D. Davis, P. Elzer, S. Olsen, S. Balsevicius, L. Kelly, P. Smith, S. Tan, et al. 2000. Validation of the fluorescence polarization assay and comparison to other serological assays for the detection of serum antibodies to brucella abortus in bison. *Journal of Wildlife Diseases* **36**:469–476.

Nielsen, K. and D. Gall. 2001. Fluorescence polarization assay for the diagnosis of brucellosis: A review. *Journal of Immunoassay & Immunochemistry* **22**:183–201.

Oli, M. K., M. V., P. A. Kleinb, L. D. Wendlandc, and M. B. Brown. 2006. Population dynamics of infectious diseases: A discrete time model. *Ecological Modelling* **198**:183–194.

1.9