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Risk Analysis For Invasive Species And Emerging Infectious Diseases: Concepts And Applications

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ABSTRACT.—Management and control of biological invasions and emerging infectious diseases are leading topics of research in theoretical and applied ecology. When the initial number of potentially invasive or infectious individuals is small, demographic stochasticity can lead to rapid extinction, suggesting that the transient dynamics of establishment for invasive species and epidemics should be modeled as a stochastic process. Quantitative risk assessment can exploit this conceptualization to calculate risk metrics such as the chance of invasion or epidemic and to study the potential effectiveness of rapid response interventions. Here I review some simple establishment models and demonstrate how understanding the underlying stochastic processes can lead to more effective policies for risk management. As examples, I study the dynamics of feral nutria (*Myocastor coypus*) in East Anglia, UK, and infection by bovine tuberculosis (*Mycobacterium bovis*) in various species. Nutria are fur-bearing aquatic mammals that have been introduced around the world intentionally and through escape from farms and cause severe damage to marsh vegetation. Bovine tuberculosis is a chronic wasting disease of diverse mammal species and results in long term emaciation and decline in animal fitness. I find that both nutria and bovine tuberculosis exhibit high intrinsic rates of increase as measured by their increase in abundance when the population or epidemic is small. While Allee effects may affect the chance of establishment for nutria, analysis of additional data will be required to reject the hypothesis that nutria dynamics follow the simple Ricker growth model. The distinction is important, however, as the estimated chance of establishment under the Allee effect model is considerably less than under the Ricker model for introduced populations less than about 850 individuals. The chance of bovine tuberculosis epidemic for introductions of small numbers of individuals infectious with bovine tuberculosis in populations of brushtail possums (*Trichosurus vulpecula*) or badgers (*Meles meles*) is high. Thus, if surveillance programs are not almost completely effective at preventing introductions of infectious individuals, periodic outbreaks are likely to occur. Epidemics in these species might be controlled by culling.

INTRODUCTION

Biological invasions and emerging infectious diseases of wildlife are key research frontiers in applied ecology (Kolar and Lodge, 2001; Williams *et al.*, 2002; Torchin and Mitchell, 2004). Invasive species are a leading cause of species extinctions (Wilcove *et al.*, 1998; Sala *et al.*, 2000) and a source of tremendous economic costs (Perrings *et al.*, 2000). Similarly, introduced pathogens in wildlife populations disrupt population dynamics (Grenfell and Dobson, 1995) and outbreaks of virulent diseases can decimate immunologically naïve populations (Dobson and May, 1986). Where wildlife populations are important to human societies as food or for recreation, the economic costs of a disease outbreak can be enormous (Burroughs *et al.*, 2002), but vary widely (Bernués *et al.*, 1997) and eradication is typically extremely costly (Nelson, 1999). Intentional and unintentional introductions of both non-indigenous species and pathogens represent a type of pollution. In contrast to

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chemical and radiological pollutants, however, environmentally discharged organisms and pathogens exhibit a distinctively biological property, the capacity to reproduce, leading initially to nearly exponential increase of the introduced agent. Consequently, management and control of introduced organisms might be more effective if focused on preventing introductions and rapid response after release occurs, rather than responding to introduced species that have become a nuisance. Determining when prevention is more effective than control is an important research objective in invasion biology (Smith *et al.*, 1999).

Risk assessment focuses on actions for preventing invasion or epidemic before naturalization of the agent in the environment and is an important potential contribution that applied ecology can make to human and animal welfare (National Research Council, 2002). One key component of risk assessment is risk analysis, which comprises estimating exposure to hazards and relating exposure to the chance and magnitude of undesirable consequences (USEPA, 1998). Other exercises that are crucial for deciding interventions include cost-benefit analysis and cost-effectiveness analysis (Sunstein, 2002). For non-indigenous organisms, zero tolerance to introductions is commonly not feasible, as organisms are continuously introduced during activities associated with global trade (Levine and D'Antonio, 2003). An important goal for ecological theory therefore is to relate the rate of introduction to the chance of establishment (Kolar and Lodge, 2001).

In contrast to this goal, most research to date has focused on estimating the effectiveness of efforts to control or eradicate invasive species and epidemics that are already established (Lefèvre, 1981; Shea *et al.*, 1998; Clancy, 1999). For example, Shea and Kelly (1998) used matrix models to estimate the potential impact of biological control agents on the population growth of nodding thistle (*Carduus nutans*) in New Zealand. El-Gohary (2001) developed a model for the optimal control of an epidemic of genital herpes. Another approach, in both animal (*e.g.,* Roberts, 1996) and human populations (*e.g.*, Katzman and Dietz, 1985), has been to estimate the fraction of the population that has to be vaccinated to halt the spread of an infectious disease (Anderson and May, 1991; Hethcote, 2000). Thus, while recent authors have suggested that a proactive approach to preventing the establishment of introduced organisms may be less costly than control or eradication after establishment has occurred (Mack *et al.*, 2000; Kolar and Lodge, 2002), most research to date has focused on invasions or epidemics already underway. As the goal of risk analysis is to formulate management strategies that are proactive rather than reactive, the following analyses focus on the relationship between naturalization of the species or disease and the initial number of individuals introduced.

The dynamical similarity between invasions and epidemics means that the chance of establishment is affected by the same mechanisms in each. In all biological populations the potential for growth is bounded by finite resources such as space and food. Epidemics are bounded by the number of susceptible individuals, which declines as the epidemic increases in severity. In paradigm cases for both free living populations (Gotelli, 2001) and epidemics (Allen, 2003), limitation increases as a linear function of population size, resulting in logistic growth, though more and less severe forms of regulation are possible. According to these models, the growth of the population or epidemic will be nearly exponential during the early stages and limitation only becomes severe when the population is large. In invasion biology, the logistic process describing local population growth is often characterized as proceeding in three phases: a lag phase, an expansion phase and a saturation phase (Fig. 1; Shigesada and Kawasaki, 1997). During the lag phase the expected population size grows nearly exponentially, but almost imperceptibly because the absolute number of organisms is small. Populations with Allee effects represent an exception to this rule, where social activities such as mate finding, foraging for food or colony defense are inhibited in extremely small or sparse populations (Dennis, 1989, 2002). The duration of the lag phase is

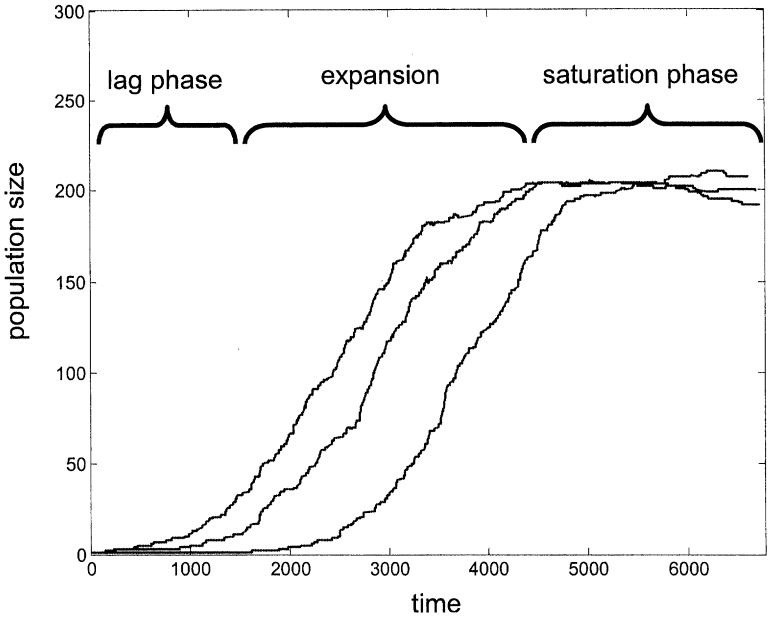


FIG. 1.—The dynamics of introduced populations and epidemics often exhibit three phases. Prevention of invasion or epidemic, in contrast to control, is typically only possible during the lag phase. Simulated trajectories for three iterations of a stochastic logistic growth process show that small fluctuations due to demographic stochasticity during the lag phase are amplified during expansion, but are erased during saturation. In come cases, these fluctuations can also lead to rapid extinction of the invasion or epidemic

crucial to determining the chance of establishment because in both invasions and epidemics the discreteness of individual propagules creates that possibility that demographic stochasticity might lead to rapid extinction. During the lag phase the chance that the introduction will fail on account of simple demographic fluctuations, independent of control efforts, is often non-negligible. Such fluctuations result from the fact that the number of organisms or infectious individuals is typically integer-valued, resulting in a situation where the chance demise of a small number of individuals can entirely eradicate the population or disease, a situation that is known in epidemiology as stochastic fade-out (Anderson and May, 1991). Because of this discreteness, deterministic models that are intended to model the average process may be misleading as the average population size can grow exponentially while the majority of populations go extinct (Bailey, 1960; Allen, 2003). Of course, if the invasion or epidemic manages to escape this phase then rapid extinction is extremely unlikely and the species or disease will have become naturalized. At this point the only possible options for intervention are control and eradication. In the remainder of this paper I will focus on conditions conducive to escape from initial transient dynamics. For the purposes of risk analysis, such escape can be considered equivalent to establishment.

The conditions under which invasion or epidemic can occur can be determined from population theory. Obviously, a necessary condition for invasion or epidemic is that the average rate at which new propagules are produced must exceed the average rate at which they are eliminated from the population through death, removal, recovery from infection or vaccination. This condition is not sufficient, however, as demographic stochasticity and environmental fluctuations create the possibility of invasion or epidemic failure even under

conditions where the expected population size increases (Bailey, 1960; Allen, 2003). In the simple case without environmental fluctuations, the chance of rapid extinction can be determined from a birth–death process. For a continuously reproducing population that initially grows nearly exponentially, the chance of extinction is approximately $(d/b)^N$ where d is the instantaneous death rate, b is the instantaneous birth rate and N is the initial number of individuals introduced (Allen, 2003). At a small cost to accuracy, this model and its extensions to density-dependent population dynamics and to population growth in fluctuating environments can be approximated by stochastic difference equations allowing greater flexibility in model structure (Morris and Doak, 2002; Lande *et al.*, 2003). These models can be studied to understand the dynamics of establishment, as I demonstrate with two examples.

First, using nutria (*Myocastor coypus* Molina) as a model species, I select and fit a stochastic population growth model to monthly censuses of a feral population in East Anglia, UK, for the period 1970 to 1979. Nutria are aquatic, fur-bearing social mammals native to South America. As a result of escapes and liberations from fur farms, feral populations now occur in Europe, Asia, Africa and North America (Woods *et al.*, 1992). In the United States, nutria have been introduced to nineteen states (USGS Nonindigenous Aquatic Species database; http://nas.er.usgs.gov) where they have been implicated in severe damage to marsh ecosystems and facilitation of invasion by nuisance plants. According to the Invasive Species Specialist Group of the World Conservation Union, nutria are among the 100 worst invasive species in the world (http://www.issg.org/database/species/search.asp?st=100ss&fr=1&sts=#SpeciesList). Nutria reproduce continuously and age of sexual maturity for females ranges from 5–10 mos in their native range and 3–8 mos in their introduced range (Guichón *et al.*, 2003a). Additionally, nutria exhibit a variety of cooperative behaviors including group foraging, nursing in groups, allogrooming and alarm calls (Guichón *et al.*, 2003b), suggesting possible regulation by Allee effects at low population densities. Selection and analysis of stochastic population growth models shows how the chance of establishment changes with the initial number of individuals introduced.

Next, I consider the chance of an epidemic that might result from unintentional introduction of wildlife infected with bovine tuberculosis (*Mycobacterium bovis*), a relative of *M. tuberculosis* (the agent causing tuberculosis in humans) that is most commonly found in livestock and wildlife. Bovine tuberculosis is endemic in brushtail possums (*Trichosurus vulpecula*) and ferrets (*Mustela furo* L.) in New Zealand (Caley and Hone, 2002, 2004), in badgers (*Meles meles*) in England and Ireland (Delahay *et al.*, 2000), and in white-tailed deer (*Odocoileus virginianus*) in the United States (Palmer *et al.*, 1999). In all three areas of endemism there is concern about spread to cattle herds and costs to agriculture (but *see* Bernués *et al.*, 1997). Additionally, an ongoing epidemic of bovine tuberculosis in African buffalo (*Syncerus caffer*; Rodwell *et al.*, 2001; Caron *et al.*, 2003) now threatens many wildlife populations in Kruger National Park through spillover effects, including baboon, lion, cheetah, kudu and leopard (De Vos *et al.*, 2001). Using data on bovine tuberculosis compiled from the published literature, I first study the relationship between the chance of epidemic and the number of introduced infectious individuals in brushtail possums and badgers. Then I explore how pre-emptive culling or rapid response to outbreak in brushtail possums might decrease the chance of epidemic.

METHODS

NUTRIA

Gosling *et al.* (1981) present a time series of nutria abundance at monthly intervals, obtained by retrospective census for a feral population of nutria in East Anglia, UK, for the

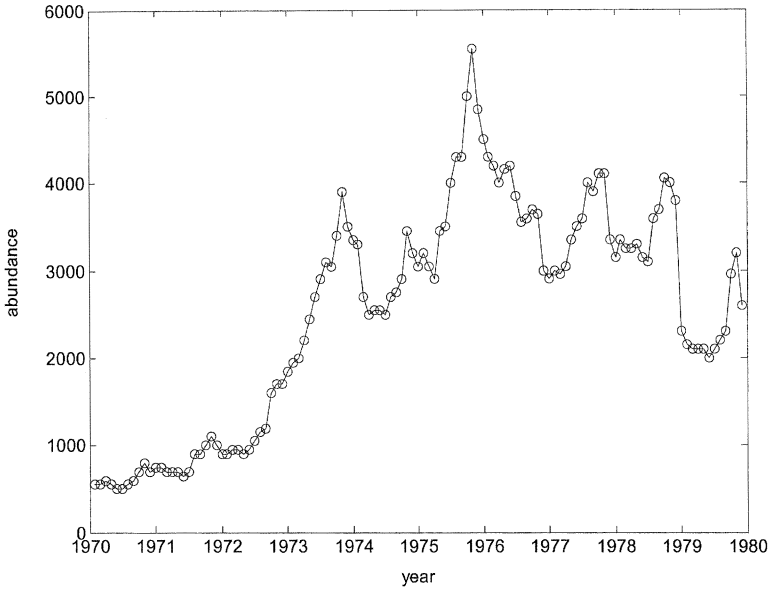


FIG. 2.—The abundance of female nutria in East Anglia, UK, from 1970 to 1979. Data were obtained from Gosling *et al.* (1981). High amplitude fluctuations are due to large inter-annual fluctuations in mortality (environmental stochasticity)

period 1970–1979. For the present analysis, only counts of adult females were used for model fitting, selection and forecasting (data available from the Global Population Dynamics Database at http://www.sw.ic.ac.uk/cpb/cpb/gpdd.html). Despite control efforts during this period (Gosling *et al.*, 1981), these data exemplify the stochastic logistic process predicted by theory (Fig. 2), with lag (1970–1972), expansion (1972–1974) and saturation phases (1974–1980). Distinguishing demographic stochasticity from environmental stochasticity in population time series is not possible without information on the fates of individual organisms (Lande *et al.*, 2003). In this analysis, I assumed that fluctuations were caused by environmental variation (the dominant cause of fluctuations in populations of hundreds to thousands of individuals), not demographic stochasticity, with the consequence that extinction risk is underestimated at small population sizes ($N \ll 100$). Four candidate models for population growth were fit to these data using the method of conditional least squares (Morris and Doak, 2002). Models included familiar density-independent and density-dependent population growth functions and a model for Allee effects (Table 1). Akaike's Information Criterion (AIC) and Bayesian Information Criterion (BIC) scores, which consider both the ability of the model to explain the data (model fit) and model complexity, were used for model selection. Low AIC and BIC scores indicate high model probability, relative to alternative models. For comparison, each model was also assigned a value Δ_i, representing the difference between the score for a particular model and the minimum score for all models. Burnham and Anderson (2002) argue that models with $\Delta_i < 2$ have essentially the same support as the best model, that there is considerably less evidence to support models with $2 < \Delta_i < 4$ and that models with $\Delta_i > 4$ can be rejected with considerable support.

Stochastic simulations of the selected models were conducted to determine how the chance of invasion changes with the initial size of the population (Morris and Doak, 2002;

TABLE 1.—Statistical support for four models of nutria population growth in a fluctuating environment. Parameters are best fit values obtained by conditional least squares; ε in the model equations represents deviations from expected growth due to environmental variation and is normally distributed with mean 0 and variance σ^2; Δ_i is the difference between AIC/BIC scores for the selected model and the best fit model; σ^2 is the residual variance of the best fit model and an estimate of the variance of ε. The exponential and Ricker models are conventional models for density-independent and density-dependent growth, respectively (Morris and Doak, 2002). Lande *et al.* (2003) suggest that when modeling population dynamics with unknown functional form the theta-logistic model should be considered because it exhibits considerable flexibility while only requiring three structural parameters. The Allee effect model used here was first proposed by Morris and Doak (2002) to represent mate limitation. According to AIC, the Allee effect model is best supported by the data, but is only marginally superior to the Ricker model. BIC, which is sometimes preferred for forecasting (in contrast to inference), selects the Ricker model over the Allee effect model. The AIC results suggest that the exponential model can be rejected with considerable support, while the BIC results suggest that the theta-logistic model can be rejected with considerable support. Neither AIC or BIC provides much guidance in selecting between the Ricker and Allee effect models.

	Model	r	κ	θ	σ^2	*AIC*	$\Delta_{i,AIC}$	*BIC*	$\Delta_{i,BIC}$
Exponential	$\log_e(N_{t+1}/N_t) = r + \varepsilon$	0.0132			1.19	−207.2	4.4	−201.8	1.5
Ricker†	$\log_e(N_{t+1}/N_t) = r(1 - N_t/\kappa) + \varepsilon$	0.0582	3000.1		1.13	−211.4	0.3	−203.2	0
Theta-logistic	$\log_e(N_{t+1}/N_t) = r(1 - (N_t/\kappa)^\theta) + \varepsilon$	0.0416	3023.1	1.8068	1.12	−210.0	1.6	−199.2	4.0
Allee effect*	$\log_e(N_{t+1}/N_t) = \log_e(N_t) - \log_e(\theta + N_t) + r - \kappa N_t + \varepsilon$	0.1455	3.78e − 05	61.1432	1.11	−211.6	0	−200.9	2.4

† Best model overall (BIC)
* Best model overall (AIC)

Allen, 2003). This relationship is analogous to the concept of dose-response in chemical risk analysis (Sunstein, 2002). An unbiased estimate of environmental variation is $\sigma^2 = qV_r/(q-1)$, where q is the number of data points and V_r is the mean squared deviation between observation and model prediction (Morris and Doak, 2002). Simulations were conducted by iterating the models with best fit parameter estimates (Table 1) 100,000 times until the population went extinct or attained an invasion threshold. A threshold of $N = 1000$ was used as a benchmark for successful invasion. This is large enough that such a population would constitute an actual nuisance (Gosling *et al.*, 1981) and also ensures that subsequent extinction in ecological timeframes is extremely unlikely, even if the population exhibits Allee effects. In practice, the choice of invasion threshold is not extremely important as long as it is considerably greater than zero for models without Allee effects (exponential, Ricker and theta-logistic models) and the critical density for models with Allee effects (approximately 443 individuals). This is because the chance of becoming large and attaining quasi-stationary dynamics before going extinct quickly becomes great. Since the stochastic difference equations used to model nutria growth treat population size as a continuous variable, populations of size $N < 1$ were considered extinct. All population trajectories were followed until either invasion or extinction occurred.

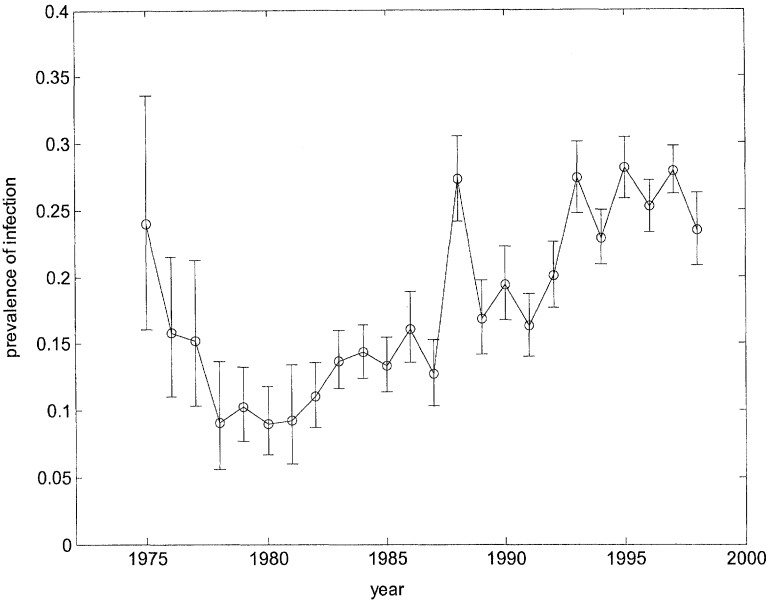


FIG. 3.—The prevalence of bovine tuberculosis infection in badgers in the UK, 1975–1998. Data collected by the Department for Environment, Food and Rural Affairs (DEFRA) are available from http://www.defra.gov.uk/animalh/tb/stats/stats4.htm. Error bars are 95% confidence intervals calculated from the binomial distribution

BOVINE TUBERCULOSIS

Bovine tuberculosis, once nearly eradicated from cattle in Britain (http://www.defra.gov.uk/animalh/tb/index.htm), is an example of a re-emerging infectious disease. First discovered in badgers in 1971, prevalence of infections from 1975–1998 first declined (1975–1978; Fig. 3), but then increased in a pattern consistent with the logistic process predicted by theory (Fig. 3). Changing strategies of controlling infections in badgers and spillover into cattle, including (beginning in 1973) a sequence of laws protecting badgers, have possibly contributed to the variability in infection prevalence over time.

A basic statistic pertaining to the potential for a disease to propagate is the basic reproductive ratio, designated by R_0, which depends on social processes like the rate of contact between individuals, and biological parameters, like the duration of the latent period between infection and the onset of symptoms (Anderson and May, 1991). R_0 is a crucial statistic for the control of epidemics (Anderson and May, 1991; Keeling and Grenfell, 2000) as it represents a threshold between epidemics that have the possibility of propagating in the long-run ($R_0 > 1$) and those that will quickly fade out ($R_0 < 1$). R_0 is, therefore, analogous to the ratio (b/d) in the simple birth-death process. From the perspective of risk analysis, R_0 is a useful statistic for assessing the chance of epidemic for introductions of a small number of infectious individuals. Allen (2003, p. 120) observes that the model for the growth of an epidemic after a small number of infectious individuals are introduced into a large population is approximately a random walk on a semi-infinite domain. Thus, by analogy, that chance that the epidemic dies out quickly given k initially infected individuals is approximately $(1/R_0)^k$ (Allen, 2003). Here, the parameter k is envisaged as the number of

unintentionally introduced infectious individuals; it is equivalent to the concept of attack size in the literature on controlling epidemics from bioterrorist attacks (*e.g.,* Kaplan *et al.*, 2002). The approximate chance of epidemic then is just the complement,

$$p_{epi} \approx 1 - (1/R_0)^k. \quad (1)$$

There are two ways, therefore, in which epidemic risk can be decreased. First, by analogy to the case of biological invasion considered above, the number of initially infective individuals can be reduced through surveillance, removal and quarantine. Second, population-level interventions such as culling or vaccination might be implemented to interfere with the organism's population biology, reducing R_0.

R_0 has been estimated numerous times in the statistical literature on bovine tuberculosis in wildlife; *see* Smith (2001) for a review. For brushtail possums, the minimum estimate is given in Barlow (1991) as $R_0 = 1.6$ while the maximum estimate is given in Barlow (2000) as $R_0 = 2.13$. For badgers the range is $R_0 = 1.1$ to $R_0 = 1.2$, given by Delahay *et al.* (2000). Without knowing the sampling properties of these statistics, analysis of the entire interval is more appropriate (Ferson, 1996). First, I calculated the interval of the estimated chance of epidemic as a function of the number of infectious individuals introduced, according to equation (1). This is the epidemic analog to the idea of dose-response. Investigating possibilities for intervening in the population biology of the host requires considering factors that affect R_0. The transmission dynamics of bovine tuberculosis in wildlife are complicated and depend *inter alia* on population density, heterogeneous distribution of hosts, vertical transmission to offspring and infection from other species (Barlow, 2000; Caley and Hone, 2004). Roberts (1996) developed a reasonably realistic model of transmission of bovine tuberculosis among brushtail possums; this was updated by Barlow (2000) to account for host heterogeneity and additional data. According to the Roberts-Barlow model, the basic reproductive ratio is given by the formula

$$R_0 = (pb + \beta)/(\alpha + d), \quad (2)$$

where p is the rate of vertical transmission; β is a coefficient in the function for heterogeneous-mixing transmission; b is the maximum, density-dependent birth rate; α is disease mortality; and d is the minimum, density-dependent death rate. Barlow (2000) provides the following parameter estimates, which were used for this analysis: $p = 0.25$, $b = 0.3$, $\beta = 2.5$, $\alpha = 1$ and $d = 0.1$. By substituting equation (2) into equation (1) it is clear that to reduce the risk of epidemic requires increasing natural mortality (by culling) or disease mortality (by surveillance and removal). The appearance of these two terms together in the denominator of equation (2) suggests that the two interventions are equivalent and interchangeable, at least from a theoretical if not a practical standpoint. Below, I examine the effect of varying α from 1 (the natural mortality of the disease) to 2.4, where R_0 diminishes below the critical value and the chance of epidemic is eliminated. The parameter α can be interpreted as an induced mortality rate so that the sum of α and natural mortality is the reciprocal of individual life expectancy.

RESULTS

NUTRIA

Using AIC and BIC, the time series of nutria abundance in East Anglia (Fig. 2) is unable to adequately distinguish among the four candidate models (Table 1). While the exponential model might be rejected on the basis of the relative AIC scores and the theta-logistic model might be rejected on the basis of the relative BIC scores, neither AIC or BIC can

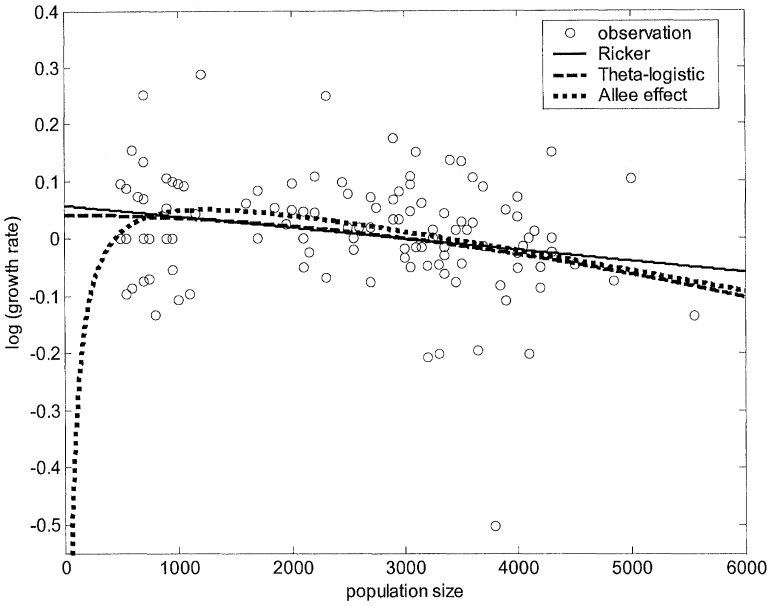


FIG. 4.—Observed instantaneous population growth rates of nutria in East Anglia, UK, from 1970 to 1979 are density-dependent. Ricker, theta-logistic, and Allee effect models cannot be distinguished with AIC or BIC (Table 1)

sufficiently distinguish between the Ricker and Allee effect models (Table 1). Further analysis of the data shows why this result was obtained. Recall that the instantaneous growth rate between time t and $t+1$ (the natural logarithm of the ratio of two censuses) was fit to a model by regressing on the population size at time t (Table 1). The plot of observed growth rate against population size N_t is apparently concave (Fig. 4), even excluding a possible outlier at ($x = 3800$, $y = -0.502$), but considerable scatter obscures any clear relationship. Proceeding from left to right, the initial increase in growth rate with population size is suggestive of Allee effects, and the best fit model according to AIC, the model with Allee effects, captures this shape. However, since there are few data in the range $500 < N < 1000$, and no observations for $N < 500$, model estimation in this range is extremely uncertain. Though the Allee effect model does give a better fit to the data (lower sum of squared residuals), the penalties in AIC and BIC for the additional parameter results in virtually identical support for the Allee effect model and the simpler Ricker model.

The consequences of these differences are important, however, as the establishment curve for the model with Allee effects (Fig. 5A) is considerably less than the establishment curves for the two models without Allee effects over the range from $N_0 = 0$ to $N_0 = 250$ (Figs. 5B–C). Indeed, for small introduced populations, the model with Allee effects predicts that the estimated chance of invasion is small, though still non-negligible (≈ 0.029), for populations less than approximately $N_0 = 50$, whereas both of the models without Allee effects estimate that for introduced populations of this size the chance of invasion is around 0.65. For these models, as the initial number of infectious individuals increases, the chance of invasion quickly approaches 0.8. Increasing the range of initial population sizes shows that for the model with Allee effects the establishment curve takes considerably longer to reach an asymptote, which it does at approximately 0.7 at population sizes around $N_0 \approx 850$ (not

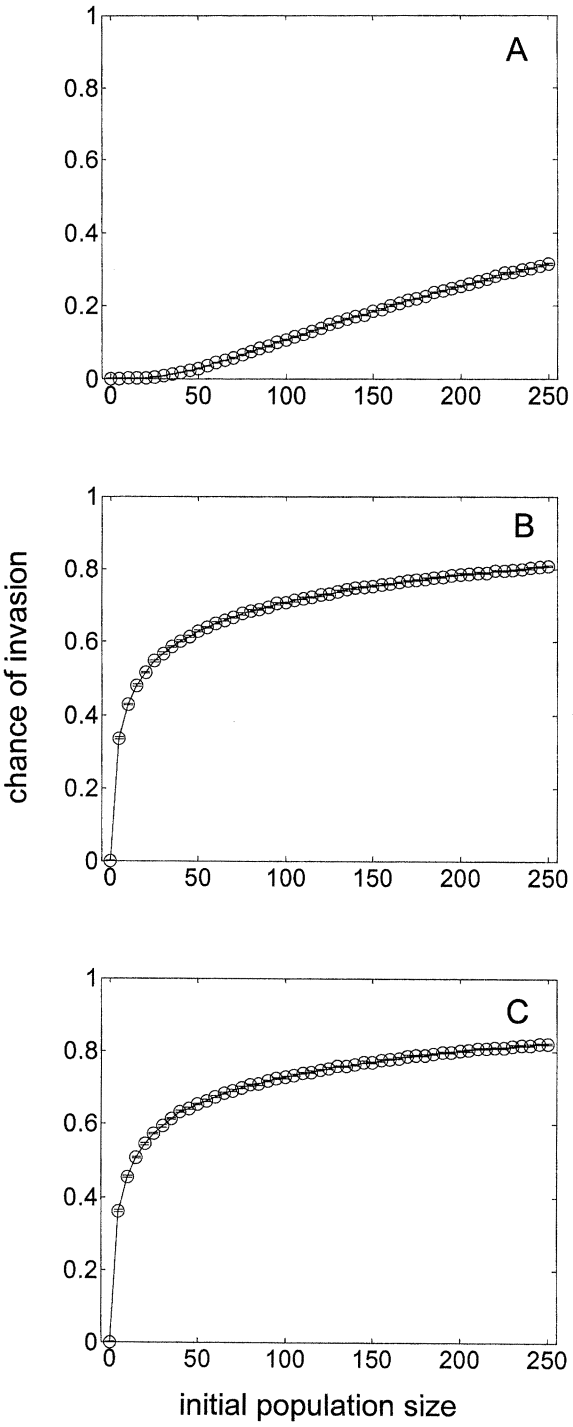
shown). Although model averaging is sometimes used for forecasts where multiple models are equally supported by the data (Burnham and Anderson, 2002), this is not appropriate here because the set of possible models is so small. The value of this analysis is to show that neither the Ricker model nor the Allee effect model can be rejected with the available data and that making this determination is important for estimating the chance of establishment.

BOVINE TUBERCULOSIS

There is a clear effect of species on the chance of bovine tuberculosis epidemic (Fig. 6). The curves in each panel of Figure 6 are the theoretical chance of epidemic (equation 1) for the maximum and minimum estimated values of R_0 and can be interpreted as bounds on the predicted chance of epidemic. Even though on an absolute scale the error in the estimated value of R_0 for possums (range: 1.6–2.13) is greater than for badgers (range: 1.1–1.2), the uncertainty in the chance of epidemic is less, reflected in the narrower range between the two establishment curves (Fig. 6). This is because the lower R_0 in badgers overall leads to a greater chance that an introduction will fail to produce an epidemic. In either case, the chance of epidemic is high for the introduction of even a few individuals. Indeed, the estimated range for the chance of epidemic for the introduction of just one infectious individual is 0.38–0.53 for possums and 0.09–0.17 for badgers. This implies that if epidemics are to be prevented not even a single infectious individual should be permitted to enter the population. Unfortunately, zero tolerance is generally not feasible. An alternative approach would be careful surveillance and removal of infectious individuals once they are detected or preemptive culling of the population. The effectiveness of culling on the chance of bovine tuberculosis epidemic increases dramatically as α, the parameter that governs induced mortality, approaches 2.4, *i.e.,* where R_0 approaches 1 and the chance of a major epidemic is eliminated (Fig. 7). One implication of this result is that endemic fadeout is extremely unlikely for bovine tuberculosis, a situation that would not necessarily obtain for less transmissible diseases.

DISCUSSION

Demographic analysis of biological invasions and epidemics can provide useful tools for risk analysis. Biological invasions and epidemics are special cases of population growth under limiting conditions. In simple cases, these dynamics lead to logistic growth processes in which the expected size of the population or epidemic initially increases approximately exponentially. In contrast, when Allee effects are present the population increases at a somewhat slower rate or may decline (Dennis, 1989, 2002). When reproductive or infectious individuals are discrete and their number is small, the introduction or epidemic may fail due to demographic stochasticity. Consideration of demographic stochasticity in invasions and epidemics leads to the idea of an establishment curve, which is analogous to dose-response in environmental risk assessment and might be used for managing risk within existing institutional structures and policies (*e.g.,* Sunstein, 2002). By relating the chance of invasion or epidemic to the initial number of individuals introduced, ecologists can provide useful information to assist managers in deciding actions to control risk. The concept of an establishment curve can be extended to populations that fluctuate because of environmental variation. As examples, I estimated establishment curves for a highly invasive aquatic mammal, nutria and a wildlife pathogen, bovine tuberculosis. The estimated establishment curve for nutria is based only on fluctuations from environmental stochasticity, which results in an underestimate of extinction risk and an overestimate of invasion risk. Further analysis of nutria population structure (Guichón *et al.*, 2003a) would further clarify invasion risk for extremely small introductions.



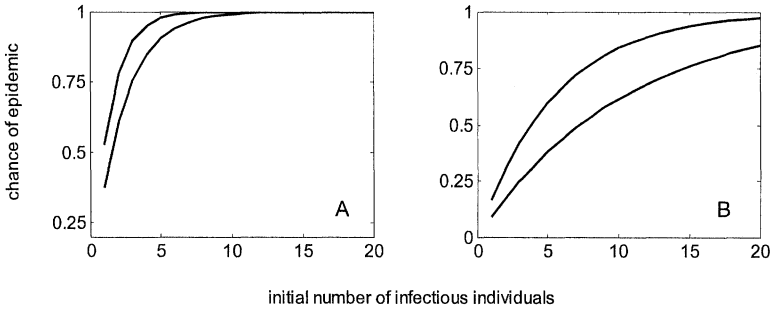


FIG. 6.—Interval estimates of the chance of bovine tuberculosis epidemic (equation 1) for an initial introduction of infectious individuals into populations of (A) brushtail possums, and (B) badgers. Uncertainty is reflected in the interval of estimated values for R_0 reported in the literature

For nutria, there were not sufficient data from populations at low density to reject the hypothesis that small populations are limited by Allee effects. The estimated chance of establishment differs considerably depending on whether or not nutria are regulated by Allee effects. Surprisingly, the risk of invasion for introductions of nutria at population sizes considerably less than the critical density can be non-negligible (Fig. 5A). This implies that even for species exhibiting severe Allee effects, introduction rates might need to be reduced to extremely low levels to prevent invasions. Quantifying Allee effects in nutria and other species is therefore an important goal for future research. Now that nutria is known to cause severe environmental damage, any future introductions are likely to be unintentional. The number of tolerable introductions and, therefore, the intensity of surveillance that will be required to prevent future invasions, depends largely on the severity of Allee effects. A better understanding of nutria population dynamics and regulation (Guichón *et al.*, 2003a) and social structure (Guichón *et al.*, 2003b) will be useful for establishing benchmarks for nutria eradication (Carter and Leonard, 2002).

I found that the chance of bovine tuberculosis epidemic for introductions of a small number of infectious individuals is great for both badgers and brushtail possums, although greater for possums. This is not surprising given that transmissibility is known to be high (Barlow, 2000; Smith, 2001; Caley and Hone, 2002). One way to reduce the chance of epidemic is by careful surveillance and removal of infected individuals before introduction. However, because the estimated value of R_0 is high, controlling epidemic risk by reducing the initial number of infective individuals will be difficult because it must be almost 100% efficient. Thus, pre-emptive culling in populations at risk may be a more effective means of control. Of course, actions such as pre-emptive culling are likely only to be taken in areas where the chance of introducing an infectious individual is relatively high, for instance in regions adjacent to areas of disease endemism. The accuracy of risk predictions that depend on epidemiological parameters will depend on the preciseness with which R_0 can be estimated and the degree to which extrapolation to other populations is warranted. Keeling

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FIG. 5.—The estimated chance of invasion for introductions of nutria according to three models with environmental stochasticity. The chance of invasion is much lower (A) if the population is limited by Allee effects, compared with (B) if the population grows according to the theta-logistic model or (C) if the population grows according to the Ricker model. Data do not strongly support the selection of any one of these models over the other two

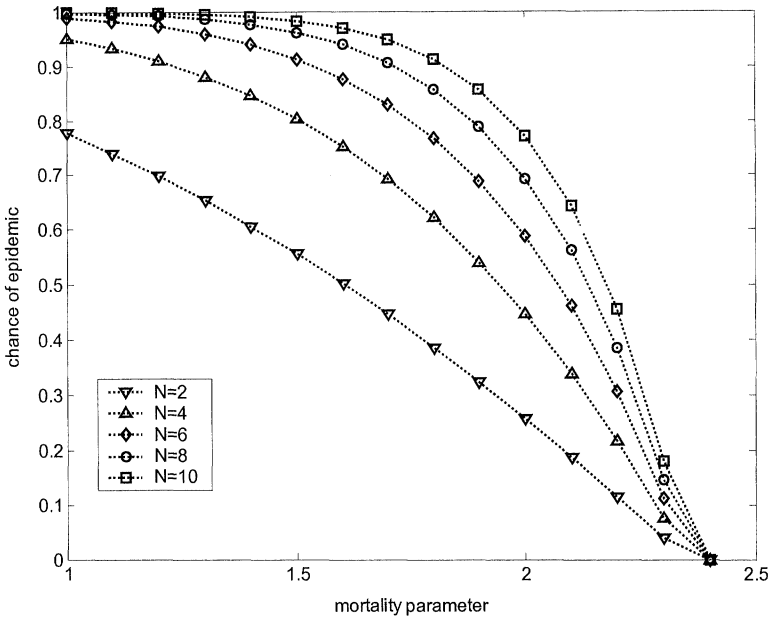

FIG. 7.—The chance of bovine tuberculosis epidemic for introductions of 2, 4, 6, 8 or 10 individuals can be reduced by culling the population. The chance of epidemic declines to zero when induced mortality (x-axis) occurs at a rate of 2.4 y^{-1}. For an intrinsic death rate of $d = 0.1$ this corresponds to an individual life expectancy of $(2.4 + 0.1)^{-1} \approx 5$ mo

and Grenfell (2000) have shown that the assumption of homogenous mixing in model construction may result in R_0 being severely overestimated, particularly for diseases spread by social contacts rather than aerial transmission. The robustness of model forecasts to errors of this kind (errors that result from assuming an inappropriate model structure) is an important feature of risk assessment that must be undertaken before recommendations are judged to be reliable.

In contrast to most existing optimal control studies, which focus only on control and eradication of endemic disease (Lefèvre, 1981; Clancy, 1999), future research should address prevention and rapid response, too (*e.g.*, Kaplan *et al.*, 2002). One approach is to formulate an optimal control model for population processes like the birth-death-immigration models studied by Matis and Kiffe (2000), but with multiple control parameters. This would allow models of epidemic and endemic disease in closed populations to be extended to study the growth of populations or epidemics with recurrent immigration or infections from outside the population and thus to determine the relative cost-effectiveness of preventive measures (which are reductions in the immigration rate) compared to control measures (which are increases in death and removal rates).

An important question concerning all risk assessments, but particularly risk assessments for introductions of living organisms, is the extent to which models derived from data collected under one set of circumstances can be generalized to predict hazards in other contexts. The nutria data used here were collected in the UK during the period from 1970–1979. It remains to be shown that these data are a reliable indication of the potential for an introduced

population to establish elsewhere, for instance in the Midwestern and Southwestern United States. Previous studies have shown that a history of invasiveness is a consistent predictor of future invasions (Kolar and Lodge, 2001), lending credibility to extrapolations in the absence of countervailing evidence. Nonetheless, where feasible and ethically permissible (*i.e.,* in laboratories), experimental introductions or epidemics should be undertaken to validate theoretical predictions. Further analysis of historical invasions and epidemics might also help to determine the conditions under which extrapolation is acceptable.

The methods used here are quite general and could be applied to a variety of invasive species and human and wildlife diseases. In each case, the quantitative assessment of invasion or epidemic risk will depend on model parameters such as transmission and birth and death rates. While gross generalizations about the risk of invasions and epidemics will probably continue to elude analysis (Kolar and Lodge, 2001), statistical correlates of model parameters might result in useful approximations. For instance, vital rates such as birth and death rates and parameters governing mate search are all related to body size (Blueweiss *et al.*, 1978; Gerritsen, 1980) while R_0 depends on ecological characteristics like population density, the maintenance of the disease in other host species and broad scale biogeographical patterns and climate. Presently, risk analysis for biological invasions and epidemics remains relatively undeveloped and much could be gained by synthesizing the statistical and mechanistic understandings of population growth and disease propagation.

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