Ecology, 84(5), 2003, pp. 1333–1339

HOW DEMOGRAPHIC STOCHASTICITY CAN SLOW BIOLOGICAL INVASIONS

ROBIN E. SNYDER[1]

Section of Evolution and Ecology, University of California, Davis, California 95616 USA

Abstract. Ecologists have traditionally neglected demographic stochasticity in describing the spread of an invading species. However, the region most critical in determining wave speed is often the leading edge, precisely the point where demographic stochasticity is most pronounced. In this paper, I analyze a common class of one-dimensional, single-species invasion models and find that, for very general conditions, demographic stochasticity slows biological invasions. Nonetheless, the slowing is not large enough to be noticeable in most ecological time series. I also briefly discuss the role of transient dynamics and rare, long-distance dispersal.

Key words: *biological invasions; demographic stochasticity; dispersal; integrodifference equations; invasion speed; population discreteness; spatial ecology.*

INTRODUCTION

Over the last several decades, researchers have expended great effort to predict the speeds of biological invasions. Traditionally, ecologists have predicted invasion speed by representing the mean population density as a function of position and time. They have ignored population discreteness and the resulting fluctuations in population levels (''demographic stochasticity''). However, one might well wonder about the effect of this omission. In the absence of an Allee effect, the speed of an advancing population wave is determined by what is happening in the leading edge, where population density is very low—precisely where demographic stochasticity is most evident. Indeed, discreteness-induced fluctuations have been shown to affect phenomena as diverse as the propagation of chemical waves (Brunet and Derrida 1997, 2001, Kessler et al. 1998, Kessler and Levine 1998, Pechenik and Levine 1999) diffusion-limited aggregation (Brener et al. 1991), the emergence of mutant strains (Kepler and Perelson 1995), and the evolution of viral colonies toward greater mean fitness (Tsimring et al. 1996).

Modeling invasions into virgin territory with no Allee effect, I have found that if the average per capita number of births is density-dependent, demographic stochasticity slows invasions. Transient dynamics are often slow to vanish for invasion models because the wave will not reach its asymptotic shape or speed until it has fully sampled the probability distribution that governs dispersal distances (the dispersal kernel). If population density can fluctuate to very high levels (such as in the absence of density-dependence), population fluctuations in the form of rare, long-distance dispersal can boost transient invasion speed somewhat, but this is a relatively minor effect.

I outline a qualitative mathematical explanation of how demographic stochasticity can slow invasion speed (see *Theory*) and discuss the effects of rare, long-distance dispersal and transient dynamics. I have also demonstrated these effects with some simple models of an invading population. I define my models and present my results in the sections *Models* and *Results*. To clarify the presentation and to use computing power most efficiently, both the mathematical explanation and the simulation models are presented in one dimension. However, the qualitative results should be unchanged in higher dimensions. I conclude in the *Discussion* by speculating about the magnitude of demographic stochasticity's effects in higher dimensions and by discussing other effects that may be present when the invader interacts with a resident species.

THEORY

I have chosen a discrete time model both because many populations have synchronized reproduction and to avoid some oddities associated with using dispersal kernels in continuous time, such as the possibility of moving a finite distance in infinitesimal time. The population at x in the next generation is given by the number of offspring produced at y times the prob-

Manuscript received 10 June 2002; accepted 12 August 2002; final version received 7 November 2002. Corresponding Editor: W. S. C. Gurney.

[1] E-mail: resnyder@ucdavis.edu

ability of dispersing from y to x, summed over all y. Thus, the equation for expected population density takes the form

$$n_{t+1}(x) = \int_{-\infty}^{\infty} (\text{expected number of offspring produced between positions } y \text{ and } y + dy \text{ at time } t) \times (\text{probability of dispersing from } y \text{ to } x \text{ between times } t \text{ and } t + 1)\, dy \quad (1)$$

where $n_t(x)$ is the expected population density at x at time t. The probability of dispersing from y to x is given by the dispersal kernel, $k(x, y)$. I assume that this probability depends only on the distance between x and y, so that the dispersal kernel takes the form $k(x - y)$. Let $N(y)$ denote the population density at location y at time t for a given realization and let $\langle\cdot\rangle$ denote an expectation over an ensemble of realizations, so that $n_t(y) = \langle N_t(y)\rangle$. If R, the average number of offspring per individual (''fecundity''), is constant, then the expected number of offspring produced at y is $\langle RN(y)\rangle = R\langle N(y)\rangle = Rn(y)$, and Eq. 1 becomes

$$n_{t+1}(x) = \int_{-\infty}^{\infty} [Rn_t(y)]k(x - y)\, dy. \quad (2)$$

The other possibility is that fecundity is not density-independent but instead depends on the population density in some neighborhood about the potential parent. Let $\hat{N}_t(y)$ represent the local density about y in a given realization, and let $\hat{n}_t(y)$ represent the expectation of the local density over an ensemble of realizations. If fecundity is a function $R[\hat{N}(y)]$ of the local density, then the expected number of offspring produced at y is $\langle R[\hat{N}(y)]N(y)\rangle$, rather than $R\langle N(y)\rangle$. We can no longer slip the fecundity out of the average, so the expected number of offspring cannot be written simply as a function of the expected population density; i.e., Eq. 1 cannot be written in closed form. However, we can make an approximation that will let us see how the expected population density depends on variance and other moments of the population distribution. We find that in the leading edge of the wave, where the population is small,

$$n_{t+1}(x) \approx B(x) - V(x) - C(x) \quad (3)$$

where $B(x) = DI(x) - DD(x)$, and $V(x)$ and $C(x)$ are corrections for the effects of demographic stochasticity to $DI(x)$ and $DD(x)$, respectively. I discuss the form of each expression below; a derivation can be found in Appendix A.

The first expression is

$$B(x) = DI(x) - DD(x) = R\int_{-\infty}^{\infty} dy\, k(x - y)n_t(y) - \frac{\beta}{D}\int_{-\infty}^{\infty} dy\, k(x - y)n_t(y)\int_{y-D/2}^{y+D/2} dz\, n_t(z) \quad (4)$$

where

$$\beta = \left.\frac{-dn(\hat{n})}{d\hat{n}}\right|_{\hat{n}=0} = \frac{R(R - 1)}{K} \quad (5)$$

for Beverton-Holt reproduction (defined in the *Models* section), assuming R is small with respect to the carrying capacity, K. Here, $b(\hat{n})$ is the fecundity as a function of local density, $\hat{n}$, and I assume that the local density at y is determined by averaging the population in an interval of length D centered at y. This baseline expression summarizes the dynamics in the absence of fluctuations; if there were no demographic stochasticity, this is all we would have. The first term simply represents geometric growth. The number of offspring produced at location y is $Rn_t(y)$, and these disperse according to the dispersal kernel, $k(x - y)$. The second term approximates the effect of density-dependence. The population at location y interacts with individuals up to a distance $D/2$ away, and the integral $\int_{y-D/2}^{y+D/2} dz\, n_t(z)$ counts how many individuals there are in this interaction zone. The strength of the density-dependence, i.e., how much crowding reduces the fecundity, is given by β. Note that for ordinary density-dependence, in which births decrease with local density, β is positive.

The effects of the population variance are contained in the second expression,

$$V(x) = \frac{\beta}{D}\int_{-\infty}^{\infty} dy\, k(x - y)n_t(y). \quad (6)$$

(Appendix A shows that the population density variance equals the expected population density, $n_t(y)$.) This has the same structure as the first term of our baseline expression, $DI(x)$. Putting them together, we get $(R - \beta/D)\int_{-\infty}^{\infty} dy\, k(x - y)n_t(y)$, and we see that population variance, interacting with density-dependence, effectively reduces R, the fecundity in an uncrowded environment. This result seems counterintuitive but appears many times in ecology, perhaps most famously in Lewontin and Cohen's study of a geometrically growing population in a varying environment (Lewontin and Cohen 1969). They found that variance in the geometric growth rate subtracts from the long run growth rate (the average of the logarithm of the geometric growth rate).

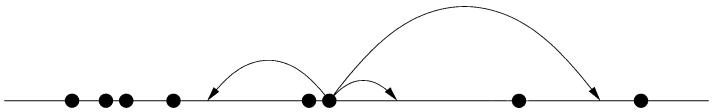

FIG. 1. Schematic of stochastic models. The black circles represent individuals at a time *t*. The arrows emanating from a circle represent the locations of that individual's offspring at time $t + 1$. Each individual produces a Poisson-distributed number of offspring, whose dispersal distances are drawn from a dispersal kernel. Space is continuous and one-dimensional.

Population fluctuations results not only in variance but also in covariance, which is taken into account by

$$C(x) = \frac{\beta}{D} \int_{-\infty}^{\infty} dy\, k(x - y) \int_{y-D/2}^{y+D/2} dz\, c_t(y, z) \quad (7)$$

where the population density covariance, $c_t(x, y)$, is defined as $\langle (N_t(x) - \langle N_t(x) \rangle)(N_t(y) - \langle N_t(y) \rangle) \rangle$. The second term in the baseline expression, $DD(x)$, assumes that the populations at location y and its environs do not fluctuate away from their expected values or that if they do, they fluctuate independently of each other. But this is false. Individuals who come from the same parent or whose parents shared an ancestor will be spatially correlated, since offspring tend to land near their parent. Thus, if the population at y fluctuates above or below its expected value, then nearby populations are likely to fluctuate in the same direction; that is, they will have a positive covariance. The expression $C(x)$ corrects for this covariance.

What is important in the context of biological invasions is that $V(x)$ and $C(x)$ are positive. (Recall that the strength of density-dependence, β, and the covariance, $c_t(y, z)$, are positive.) The fluctuation-dependent expressions $V(x)$ and $C(x)$ therefore slow population growth in the leading edge of the wave. (They subtract from $n_{t+1}(x)$.) The apparent forward motion of the wave is caused by small populations in the edge of the wave growing and becoming part of the bulk of the wave, and so slowed growth translates into a slowed invasion speed.

Fluctuations can also increase the transient invasion speed by increasing the likelihood of rare, long-distance dispersers. The wave will not reach its asymptotic shape and speed until it has fully sampled the tails of the dispersal kernel, which represent rare, long-distance dispersal. If the population density in some part of the wave briefly reaches a high level, there will be more offspring produced there, and more chances that one of those offspring will travel very far. The increased likelihood of sampling the tails of the dispersal kernel accelerates the rate at which the wave achieves its asymptotic form, increasing the transient speed. Note that sufficiently large fluctuations occur mostly in the bulk of the wave. (While relative fluctuations are greatest in the tip of the wave, the absolute magnitude of fluctuations is greatest in the bulk of the wave, where the greatest number of individuals are reproducing.) Large fluctuations require large populations, so this effect is most likely to be seen either with density-independent reproduction or in systems with a high carrying capacity.

MODELS

To demonstrate the effects just discussed, I compare speeds from a collection of related models. (I present definitions of invasion speed and explain how I measured invasion speeds in the stochastic simulations in Appendix B.) All use continuous, one-dimensional space and discrete time and represent a situation in which individuals reproduce at the beginning of a time step and then each of the offspring disperses according to a dispersal kernel (see Fig. 1). Parents die after reproducing, and the population is censused after dispersal. I have in mind here a semelparous population that reproduces in synchronized pulses, such as insects or, better still, annual plants.

The models are characterized by their dispersal kernel, which can be either a Laplacian or a ''top hat'' (defined below), and by whether reproduction is density-independent or density-dependent. Each model then has up to three variants: deterministic, deterministic births but stochastic dispersal (''stochastic dispersal'' runs), or stochastic births and dispersal (''fully stochastic'' runs). For the reasons I gave in the *Theory* section, deterministic analogues are not possible for models with density-dependence.

In the fully stochastic variants, the number of offspring for each individual is drawn from a Poisson distribution. Under density-independent reproduction, the mean number of offspring (fecundity) for all individuals is R. This model is the same as that used by Lewis and Pacala (2000). In the density-dependent case, each potential parent determines the local density, $\hat{n}$, in an interval of length D centered on itself. That parent's fecundity is then given by the Beverton-Holt model: fecundity $= R/(1 + (R - 1)\hat{n}/K)$. I have chosen $D = 1$ for all the data presented here.

Each of the offspring then disperses to the left or right, with the distance and direction drawn from a dispersal kernel, either Laplacian ($k(x) = (a/2) \exp(-a|x|)$) or top hat ($k(x) = 1/(2L)$ for $|x| \leq L$ and zero otherwise). Choosing a Laplacian kernel is equiv-

alent to assuming that dispersers have a constant probability of settling per unit distance as they travel. This might approximate light seeds settling out of an air stream, for example. What is more important is that the Laplacian kernel is leptokurtic; it has fewer intermediate-range dispersers and more long-range dispersers than a Gaussian kernel. Most dispersal distributions found in nature are believed to be leptokurtic (Neubert et al. 1995). A top hat kernel is obviously not realistic but it eliminates the possibility of rare, long-distance dispersal, which will be helpful for teasing apart different fluctuation-generated effects.

In the stochastic dispersal runs, the number of offspring is equal to the expected number of offspring, rounded to the nearest integer if need be. I chose R to be an integer ($R = 2$, specifically) so that for the all-important wave tip at least, where the dynamics are approximately linear, the mean number of offspring per individual would be the same for the stochastic dispersal runs and the fully stochastic runs. (The mean number of offspring is the same everywhere in the density-independent case.) Dispersal proceeds as in the fully stochastic runs.

The deterministic variants are equations for expected density. These are what is traditionally used to model invasions. Fluctuations are ignored and population size is not discrete.

For density-independent reproduction, the equation governing the expected population density is

$$n_{t+1}(x) = R \int_{-\infty}^{\infty} k(x - y)n_t(y)\, dy \qquad (8)$$

where $n_t(x)$ is the expected population density at point x at time t and $k(z)$ is the dispersal kernel. This equation has been used as the standard in a number of studies of invasion dynamics (Neubert et al. 1995, Kot et al. 1996, Clark 1998, Neubert et al. 2000), and it is easy to find the corresponding asymptotic invasion speed using the results in Kot et al. (1996). As discussed in the *Theory* section, there is no way to write such an equation for the density-dependent case in closed form without making simplifying approximations. However, Mollison states that the speeds of nonlinear stochastic models in continuous time will be no greater than the speeds of the equivalent linear models (Mollison 1991). This result should hold in discrete time as well, since in both cases the nonlinearities represent decreases in per capita growth rates at higher densities and these decreased growth rates can only reduce the invasion speed. Thus, the expected speed for the linear models should be an upper bound to the expected speed of the nonlinear models.

By comparing the speeds of different model variants, I am able to tease apart the effects of transient dynamics, demographic stochasticity, and rare, long-distance dispersal. The deterministic models give us asymptotic speeds for the density-independent models and upper bounds for the speeds of the density-dependent models. The slowing effect of demographic stochasticity is demonstrated in the density-dependent models. Both the stochastic dispersal runs and the fully stochastic runs experience population fluctuations due to demographic stochasticity, but the fluctuations are stronger in the fully stochastic runs (variance and covariance are larger), and the slowing should be greater there. In order to pick out the effect of rare, long-distance dispersal, I compare models with density-independent and density-dependent reproduction and Laplacian and top hat dispersal. The Laplacian kernel permits rare, long-distance dispersal while the top hat kernel does not. Density-independent reproduction permits the large fluctuations that make rare long-distance dispersal more likely while density-dependent reproduction (with a relatively low carrying capacity) discourages such fluctuations. However, speed differences will be complicated by transient effects and computer truncation effects. I discuss these issues in the *Results* section.

Results

The results are summarized in Table 1. The speeds listed in the ''deterministic'' column are asymptotic speeds for the density-independent, stochastic model variants. As predicted, the fully stochastic density-dependent model variants were significantly slower than their stochastic dispersal counterparts, due to the increased levels of demographic stochasticity. On the other hand, the differences between the speeds of the fully stochastic and stochastic dispersal variants of the density-independent model were not statistically significant. Again, this is as predicted, because demographic stochasticity can only slow invasions through its interactions with density-dependence. (These speeds should also be statistically indistinguishable from their deterministic counterparts but are not. This issue is discussed below.)

Although the speeds are not all statistically distinguishable, it is striking that the mean speed of the fully stochastic variant was higher than that of the stochastic dispersal variant for a single case: the density-independent model with Laplacian dispersal. I could not run the density-independent models for very long due to computer memory constraints. If with more data the difference proved to be significant, I would suggest that the higher (transient) speed is due to rare, long distance dispersal. Large populations, such as those found with density-independent growth, provide an opportunity for large fluctuations when the number of offspring

TABLE 1. Comparison of dispersal speeds for invasion models with different degrees of stochasticity.

Kernel	Reproduction ($R = 2$)	Mean	Stochastic dispersal	Fully stochastic
Laplacian	dens.-ind.	3.80	3.37 ± 0.11	3.52 ± 0.13
	dens.-dep.	≤3.80	2.97 ± 0.03	2.56 ± 0.03
Top hat	dens.-ind.	1.261	1.198 ± 0.007	1.184 ± 0.009
	dens.-dep.	≤1.261	1.037 ± 0.003	0.872 ± 0.005

Notes: Speeds in the column labeled ''mean'' are for the mean population density. Speeds in the column labeled ''stochastic dispersal'' come from computer runs in which dispersal was stochastic but the number of offspring per individual was deterministic. Speeds in the column labeled ''fully stochastic'' come from computer runs in which both dispersal and the number of offspring per individual were stochastic. ''Dens.-ind.'' and ''dens.-dep.'' refer to density-independent and density-dependent (Beverton-Holt) reproduction, respectively. The Laplacian kernel has $a = 0.5$, and the top hat kernel has $L = 2$, so that in both cases, the mean absolute distance traveled ($\int dx\, k(x)|x|$) is 2. Confidence intervals represent one standard error. Population is not discrete in the equations for mean population density, and there is no demographic stochasticity. Population is discrete for both the stochastic dispersal runs and the fully stochastic runs, with greater levels of demographic stochasticity in the fully stochastic runs.

born is stochastic, and a Laplacian dispersal kernel permits rare long-distance dispersal.

The observant reader will have noted that speeds for the density-independent, stochastic model variants do not quite reach their predicted asymptotes. All of the stochastic speeds are a little slower than would be predicted because of long transients. Runs using a Laplacian kernel may also be slowed by computer truncation effects, since a computer cannot generate arbitrarily small numbers, and thus we do not explore the most extreme reaches of the kernel tails. However, this effect should be the same for all runs using a Laplacian kernel, and so comparisons within a model should still be valid.

While the effects of demographic stochasticity are clear in these models, no wave is slowed by >10%.

DISCUSSION

I have demonstrated that demographic stochasticity causes realized invasion speeds to be slower than would be predicted according to traditional methods. The primary effect of demographic stochasticity in the presence of density-dependent births is to slow the wave. The nonlinear fecundity couples the dynamics of the local mean population density to higher moments, which, in the absence of an Allee effect, slows the growth of the mean and hence slows the invasion. Transient dynamics are often slow to disappear for invasion models because the leading tip of the wave must be fully developed before the wave can reach its full speed. If the population is sufficiently large, demographic stochasticity can also enhance rare, long-distance dispersal, which increases speeds in the transient regime for both density-independent and density-dependent models. However, this is a relatively minor effect.

No wave in this study was slowed by > 10%, which is reassuring for those trying to match models to data. Given the noisiness and short duration of most ecological time series, it seems safe to ignore demographic stochasticity when formulating models.

Other researchers have investigated the velocity of stochastic invasion processes. Mollison's papers (1977, 1991) provide the most comprehensive review of early results. To cite some specific studies, Mollison considers a different epidemic model on a one-dimensional lattice and proves that, given some easily met conditions, the expected front velocity is constant if and only if the infection kernel (equivalent to the dispersal kernel here) has finite variance (Mollison 1970). In simulations described in the same paper, Mollison notes that when there is a limit to the number of infected individuals at a site, equivalent to an extreme form of density-dependence, the velocity of the stochastic model is less than that predicted by a naive deterministic equivalent (i.e., without $V(x)$ and $C(x)$). However, he does not suggest any explanation for why this should be so, and provides no mathematical analysis.

Bartlett (1960) was one of the first to consider how population density covariance affects the spread of an invasion. His work was corrected and extended by Daniels (1977). More recently, Ellner and others (1998) have modeled the spread of the contact process on a two-dimensional lattice. There, the relevant covariances are nearest-neighbor correlations, which they estimate using a pair approximation.

Lewis's paper (2000) exploring the spread rate for a particular nonlinear stochastic invasion model is closest in spirit to the present work. He starts with an equation for the expected population density in terms of expected density and covariance, as done here, and also includes an equation for the covariance in terms of the

same quantities. Lewis, however, directs his energies toward finding a moment closure scheme (a way of approximately writing the dynamics for the expected density in closed form) for a specific model instead of considering density-dependent dynamics generally, as this paper does.

One question not fully addressed in this paper is how demographic stochasticity affects invasion speed in higher dimensions. Mollison (1991) has observed that nonlinear stochastic invasions progress at different speeds in different dimensions. The procedure described in Appendix A can easily be generalized to higher dimensions, and doing so results in a multidimensional equivalent of the approximation obtained in one dimension (Eqs. 3, 4, 6, 7), with integrals over dy and dz becoming integrals over $d^d y$ and $d^d z$, for example, in d dimensions. We can make the same arguments about the variance expression $V(x)$ and the covariance expression $C(x)$ subtracting from $n_{t+1}(x)$, and so demographic stochasticity should also slow invasions in higher dimensions. What is less certain is whether the slowing will be more than or less than the slowing in one dimension. In general, spatial correlations become weaker in higher dimensions because individuals interact with a larger number of neighbors or, in this case, because the number of locations for offspring to land that are ''far'' from their parents grows faster than the number of ''near'' locations. (Recall that only correlations up to lag $D/2$ contribute to $C(x)$.) I therefore suspect that the effects of demographic stochasticity on invasion speed will be even less noticeable in higher dimensions.

Demographic stochasticity can cause additional effects when the invading species interacts with a resident species. One traditionally models such systems by assuming that individuals interact only in pairs, not in clusters, and that population levels are always equal to the local means (a ''mean field'' assumption). However, if the wave front width narrows to the point that it is comparable to the distance between individuals, then population discreteness becomes unignorable and one can no longer equate local densities with mean densities. Likewise, if the wave front width becomes comparable with the distance within which individuals interact, one can no longer assume interactions are restricted to pairs. Mai et al. (1998) see both of these effects in their simulations of chemical waves, and find that for their system, wave speed increases as a result.

Population discreteness and demographic stochasticity can have an additional effect if the invading species must interact with the resident species to spread, e.g., predators moving into a region of prey, parasitoids sweeping across a field of hosts, or a disease spreading among new susceptibles. If there is no Allee effect, one ordinarily finds the invasion speed by linearizing the equations, since it is the wave tip that determines speed and population density is low there. However, if interaction rates are too high, then the area where linearized equations are valid may not contain both types of individual, and nonlinear interactions become important. In a separate study of chemical reactions, Mai et al. (2000) note this effect and find that it slows wave speed.

This study has found that while demographic stochasticity does slow wave fronts involving a single species, the reduction in speed is small. It is unclear how the speed would change in the presence of interactions with other species, such as predation or competition, and further investigations in this direction would be useful.

ACKNOWLEDGMENTS

I thank Maury Bramson, Peter Chesson, Bernard Derrida, Parviez Hosseini, Bruce Kendall, Aaron King, and especially Mark Lewis and Roger Nisbet for helpful conversations. Subject editor Bill Gurney and three anonymous reviewers provided helpful comments. This work was partially supported by NSF grants BIR94-13141 and GER93-54870.

LITERATURE CITED

Bartlett, M. 1960. Stochastic population models in ecology and epidemiology. Methuen's Monographs on Applied Probability and Statistics, Methuen, London, UK.

Brener, E., H. Levine, and Y. Tu. 1991. Mean-field theory for diffusion-limited aggregation in low dimensions. Physical Review Letters **66**:1978–1981.

Brunet, E., and B. Derrida. 1997. Shift in the velocity of a front due to a cutoff. Physical Review E **56**:2597–2604.

Brunet, E., and B. Derrida. 2001. Effect of microscopic noise on front propagation. Journal of Statistical Physics **103**: 269–282.

Clark, J. S. 1998. Why trees migrate so fast: confronting theory with dispersal biology and the paleorecord. American Naturalist **152**:204–224.

Daniels, H. 1977. The advancing wave in a spatial birth process. Journal of Applied Probability **14**:689–701.

Ellner, S. P., A. Sasaki, Y. Haraguchi, and H. Matsuda. 1998. Speed of invasion in lattice population models: pair-edge approximation. Journal of Mathematical Biology **36**:469–484.

Kepler, T., and A. Perelson. 1995. Modeling and optimization of populations subject to time-dependent mutation. Proceedings of the National Academy of Sciences (USA) **92**: 8219–8223.

Kessler, D., and H. Levine. 1998. Fluctuation-induced diffusive instabilities. Nature **394**:556–558.

Kessler, D., Z. Ner, and L. Sander. 1998. Front propagation: precursors, cutoffs, and structural stability. Physical Review E **58**:107–114.

Kot, M., M. A. Lewis, and P. van den Driessche. 1996. Dispersal data and the spread of invading organisms. Ecology **77**:2027–2042.

Lewis, M. 2000. Spread rate for a nonlinear stochastic invasion. Journal of Mathematical Biology **41**:430–454.

Lewis, M. A., and S. Pacala. 2000. Modeling and analysis of stochastic invasion processes. Journal of Mathematical Biology **41**:387–429.

Lewontin, R., and D. Cohen. 1969. On population growth in a randomly varying environment. Zoology **62**:1056–1060.

Mai, J., I. Sokolov, and A. Blumen. 1998. Discreteness effects on the front propagation in the a+b to 2a reaction in 3 dimensions. Europhysics Letters **44**:7–12.

Mai, J., I. Sokolov, and A. Blumen. 2000. Front propagation in one-dimensional autocatalytic reactions: the breakdown of the classical picture at small particle concentrations. Physical Review E **62**:141–145.

Mollison, D. 1970. The rate of spatial propagation of simple epidemics. Pages 579–614 *in* Proceedings of the Sixth Berkeley Symposium on Mathematical Statistics and Probability. Volume 3. University of California Press, Berkeley, California, USA.

Mollison, D. 1977. Spatial contact models for ecological and epidemic spread. Journal of the Royal Statistical Society, Series B **39**:283–326.

Mollison, D. 1991. Dependence of epidemic and population velocities on basic parameters. Mathematical Biosciences **107**:255–288.

Neubert, M., M. Kot, and M. Lewis. 1995. Dispersal and pattern formation in a discrete-time predator–prey model. Theoretical Population Biology **48**:7–43.

Neubert, M. G., M. Kot, and M. A. Lewis. 2000. Invasion speeds in fluctuating environments. Proceedings of the Royal Society Biological Sciences Series B **267**:1603–1610.

Pechenik, L., and H. Levine. 1999. Interfacial velocity corrections due to multiplicative noise. Physical Review E **59**: 3893–3900.

Tsimring, L., H. Levine, and D. Kessler. 1996. RNA virus evolution via a fitness-space model. Physical Review Letters **76**:4440–4443.

APPENDIX A

A description of the second-order expansion for mean density is available in ESA's Electronic Data Archive: *Ecological Archives* E084-029-A1

APPENDIX B

A description of the definitions and methods for invasion speeds is available in ESA's Electronic Data Archive: *Ecological Archives* E084-029-A2