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REVIEW

The spatial spread of invasions: new developments in theory and evidence

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Abstract

We review and synthesize recent developments in the study of the spread of invasive species, emphasizing both empirical and theoretical approaches. Recent theoretical work has shown that invasive species spread is a much more complex process than the classical models suggested, as long range dispersal events can have a large influence on the rate of range expansion through time. Empirical work goes even further, emphasizing the role of spatial heterogeneity, temporal variability, other species, and evolution. As in some of the classic work on spread, the study of range expansion of invasive species provides unique opportunities to use differences between theory and data to determine the important underlying processes that control spread rates.

Keywords

Diffusion, dispersal, integro-difference equations, invasions, reaction-diffusion, spatial spread.

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INTRODUCTION

The spatial spread of invasive species has been a subject of empirical and theoretical study for many decades (Fisher 1937; Skellam 1951). The earliest work seemed to point to a simple and robust prediction of a linear rate of spread, perhaps after some initial phase where spread was not linear (reviewed by Hengeveld 1989; Andow *et al.* 1990; Okubo & Levin 2002). The problem of spread has, however, turned out to be much richer and more interesting than was supposed 20 years ago. Some recent theoretical developments have called basic results into question, while others have clarified just when the earlier results should hold (Kot *et al.* 1996; Weinberger *et al.* 2002). Much more data are becoming available, and new statistical techniques are being developed to match data with theory. While the early models included only a single non-evolving species in a homogeneous habitat with random short range dispersal, newer theory is beginning to include the effects of changing all these assumptions.

The initial models of Fisher (1937) were phrased as partial differential equations that could be used to predict an asymptotic rate of spread. In the simplest form in one spatial dimension, with $p(x,t)$ a density function for the population level as a function of spatial location x at time t, $f[p(x,t)]$ the per capita rate of increase of the population at spatial location x and D a measure of the mean squared displacement of individuals per unit time, the model can be written as

$$\frac{\partial p(x,t)}{\partial t} = f\left[p(x,t)\right]p(x,t) + D\frac{\partial^2 p(x,t)}{\partial x^2} \qquad (1)$$

It is intriguing to note that rigorous results on the dynamics of eqn 1 are very difficult to obtain, and Bramson (1983) is one of the first papers where results describing the long-term dynamics of eqn 1 were fully justified.

Similar results for spread are obtained either with f as a constant, or with f decreasing monotonically as p increases (Okubo & Levin 2002), since at least at the initial stages of invasions population levels are small. The rate of spread in either case can be shown to be

$$2\sqrt{f(0)D} \tag{2}$$

with analogous results in more than one spatial dimension. This basic result has two implications. First the rate of spread is a linear function of time; second the rate of spread can be predicted quantitatively as a function of measurable life history parameters. However, these classical results have been shown to depend strongly on some of the explicit assumptions as well as the implicit assumptions inherent in the modelling framework of eqn 1.

Given the importance of invasive species in both basic and applied ecology, it is important to update and interpret these results in the light of recent empirical and theoretical developments. We begin with an overview of the empirical evidence that is available on spatial spread of invasions and how these data are collected. Then we review newer modelling and statistical approaches and indicate how these developments change the classic results. We then examine the potential to include, in both analytic and conceptual models, more biological information about long distance dispersal, temporal variability, spatial heterogeneity, interactions with other species, and evolution. The ultimate goal is a deeper understanding of spread, and improved ability to predict spread based on either determination of the underlying life history or on observations of initial rates of spread.

EMPIRICAL DATA ON THE SPATIAL SPREAD OF INVASIONS

The study of spread of species has been one of the areas of biology with the greatest interplay between models and data (Andow *et al.* 1993), so we begin with an exploration of the kinds of data available. The most accurate records of the spread of invasions come from field mapping of an invasion front over successive years (D'Antonio 1993; Holway 1998) or from time series of aerial photos (Lonsdale 1993). However, in most studies data are point locations of species, as recorded in such data bases as herbaria, county weed inventories, or species lists for parks and other protected sites. In the typical approach to analysing rates of spread, these point data are translated into presence/absence records for geographic areas such as counties (Forcella & Harvey 1982; Perrins *et al.* 1993; Suarez *et al.* 2001) or cells on a regular grid (Nash *et al.* 1995; Weber 1998). Sequential maps of these presence–absence data are taken to represent the expansion of the invasion.

Within a given geographic area, the cumulative numbers of records of a species over time is sometimes used as a measure of increasing abundance (Pysek & Prach 1993; Mihulka & Pysek 2001). If different areas are being compared, this measure may be standardized for the intensity of floristic research in each area.

Clearly, the kind and quality of data and the scale of mapping can influence estimates of spread rates. Weber (1998) compares spread rates for three congeners as calculated either by the number of localities or by the number of occupied grid squares. The species are ranked the same by either measure. However, measured by number of records, the fastest spreading species increases 18 times faster than the slowest, while measured by occupied grid squares it increases only seven times faster. Also, the way that range size is measured determines whether range size increases linearly or sigmoidally.

The appropriate measure of the rate of spread of occupied area also depends on assumptions about the dynamics of spread. Linear increase can be expressed in terms of a single measurement, with dimensions of $km^2\ year^{-1}$ if the area is increasing linearly, or $km\ year^{-1}$ if the radial expansion rate is constant. But if spread is more complex, the range expansion of an invasive species cannot simply be described in terms of a single rate. It is difficult to meaningfully compare spread rates between different species or locations if the underlying dynamics are not well specified (Grosholz 1996; Mack & Lonsdale 2001). In addition, few studies report the standard error for the observed velocity, or the root mean square of the regression model, which are needed in predictive models.

Independent estimates of dispersal are also required for mechanistic models of invasion. For plants, fitting methods have been developed to estimate the shapes of seed dispersal curves from seed trap data (e.g. Clark 1998). Animal tracking, mechanistic modelling of wind or water movement, and genetic approaches have also been employed in attempts to estimate or measure dispersal. For many invading species, the key to understanding dispersal is measuring human transport processes, such as the movement of Argentine ants (Suarez *et al.* 2001) and *Phytophthora lateralis* disease spores (Jules *et al.* 2002) by cars and trucks, or of zebra mussels by boats (Buchan & Padilla 1999).

It is commonly found, however, that empirically measured rates of dispersal combined with the model (eqn 1) and solution (eqn 2) do not accurately predict the rates of range expansion in invasions. This is usually explained by invoking rare long-distance dispersal events,

e.g. *Opuntia* cactus transport by animals (Allen *et al.* 1991), *Mimosa pigra* spread by water (Lonsdale 1993), and transmission of pathogens of the gypsy moth by its parasitoids (Dwyer *et al.* 1998). While the new theory described below provides a range of approaches for modelling dispersal, one of the major challenges to the field is the empirical estimation of low probability, long-distance dispersal events.

THE UNDERLYING GENERAL SPREAD MODEL

A general spread model would simply relate the position of reproducing individuals relative to the position of their parents (van den Bosch *et al.* 1992). This relationship enters into deterministic models as a dispersal kernel, $k(x,y,a)$ which is the probability that an individual born at location y produces an offspring when it is age a which then starts life at location x. In the most general circumstance, the kernel k could depend on a variety of factors, such as space and time, and the density of the species of interest or any other species. Essentially all deterministic spread models start from this basic formulation, and make various simplifying assumptions, as a general model of this form could not practically be solved. Thus the discussion of models in the remainder of the paper focuses on the kinds of assumptions made, and the resulting behaviour.

The simplest spread model (eqn 1) ignores any underlying variation in the environment and also ignores age and assumes that reproduction and movement occur randomly during the lifetime of the individual, and assumes that the movement is essentially the result of a very large number of steps of arbitrarily small size. The model (eqn 1) also assumes that the highest per capita growth rate occurs when the species is rare. Unfortunately, as we discuss below, the result (eqn 2) is not robust to changes in these assumptions. It is almost superfluous to say that the kinds of changes we can envision consist of different assumptions about movement, reproduction, the underlying environment, density dependence, and the effects of other species. Recent progress in understanding the dynamics of spread has essentially consisted of discovering other simplifying assumptions about the form of the kernel k that are biologically relevant and lead to tractable models.

NEW DEVELOPMENTS IN MODELS OF SPREAD

The main use of classical and neo-classical spread models has been to predict the velocity of the invasion front from species attributes related to reproduction and dispersal. We distinguish between two kinds of approaches, those that use independent data to estimate model parameters and those that use the velocity data to fit one or more of the model parameters. The basic approach with independent data is well illustrated by applications of the diffusion model, where just two parameters r and D are required to estimate velocity (eqn 1). For example, Andow *et al.* (1990) consider the spread of muskrat, cereal leaf beetles and cabbage white butterflies. They estimate $r = f(0)$ from life table data or from initial rates of increase for low-density populations, and D from mean displacement of recaptured individuals. The diffusion equation has been used to predict spread velocity from independent estimates of r and D for a variety of terrestrial and aquatic animals and some diseases, but curiously not for plants (van den Bosch *et al.* 1992; Holmes 1993; Buchan & Padilla 1999).

Neo-classical models of spread use a similar approach, but more detailed data are used for the reproduction function and the dispersal kernel. By removing some of the assumptions underlying eqn 1 different results are obtained (Table 1). One of the implicit assumptions made in the classical model (eqn 1) is that movement and reproduction occur randomly during the lifetime of the individual. If instead, in a growing population, movement and reproduction occur at a specific age, then the rate of spread can be very different from the predictions of model (eqn 2) as shown by van den Bosch *et al.* (1992). They use a more detailed model that takes into account the age dependence of movement and reproduction and

Table 1 Steps in the theoretical understanding of the spread of invasive species, where the linear spread rate refers to a long-time spread that has the square root of area, or the linear extent, of the range increasing linearly with respect to time

Spread dynamics	Key reference
Linear rate of spread with density dependence	Fisher (1937)
Linear rate of spread in density independent deterministic model	Skellam (1951)
Equivalence of stochastic density independent spread rates and deterministic spread with density dependence or independence	Mollison (1991)
Timing of reproduction and dispersal can greatly influence spread	van den Bosch *et al.* (1992)
Allee dynamics can lead to increasing rates of spread	Lewis & Kareiva (1993)
Discrete time models with long distance dispersal can have very different rates of spread that can increase with time	Kot *et al.* (1996)
Competitive models can have nonlinear spread rates	Hosono (1998)
General conditions developed that guarantee linear rates of spread	Weinberger *et al.* (2002)

predict spread velocity. The data used were age-specific survivorship and fertility, and dispersal densities estimated from mark-recapture studies. The approach of van den Bosch *et al.* (1992) has been applied to birds (Lensink 1997) and earthworms in agricultural fields (Marinissen & van den Bosch 1992).

The result for the asymptotic rate of spread embodied in eqn 2 is quite general, but it assumes that there is no Allee effect in the per capita growth rate $f[p(x,t)]$. If the per capita growth rate is negative for small values of the population level (Lewis & Kareiva 1993), the initial rate of spread can be slower than predicted by eqn 2, and then increase with time. Thus, the linear rate of spread does not always hold. Interspecific competition can also lead to nonlinear rates of spread (Hosono 1998).

The implicit assumption of continuous time in the model (eqn 1) is perhaps even more subtle, but important. The role of changing this and other assumptions about the description of movement has been most extensively studied in the context of discrete-time integro-difference equations, as emphasized in the work of Kot *et al.* (1996). If the population is again studied in one spatial dimension, and for simplicity we ignore age structure, the probability of finding an individual at location x next year is found by summing up the probability over all locations y that an individual is produced at y and then disperses to location x

$$p_{t+1}(x) = \int p_t(y) f[p_t(y)] k(x,y) \mathrm{d}y \qquad (3)$$

Typically this model is further simplified by ignoring spatial heterogeneity and assuming that the dispersal kernel depends only on the distance between parent and offspring $k(x,y) = k(|x-y|)$. The dynamics of this model are similar to that of the model (eqn 1) only if the dispersal kernel is Gaussian, which is implicitly assumed in the derivation of the eqn 1 (resulting from the assumption of a large number of small steps). Different dispersal kernels can produce very different dynamic behaviour. For example, 'fat-tailed' dispersal kernels that are exponentially unbounded (the tail does not approach zero at least as fast as exponential) result in spread that accelerates and does not approach a constant velocity (Kot *et al.* 1996).

Integro-difference equation population models of the form (eqn 3) require parameter estimates for a reproduction function, which may be unstructured or stage structured, and a dispersal kernel. In some empirical studies the velocity of spread has been determined numerically or by simulation to accommodate unique combinations of the reproduction function and the dispersal kernel. A severe problem in fitting dispersal kernels is that the rate of spread is most sensitive to the behaviour of the long distance part of the dispersal kernel, for which the data are least available. Moreover, fitting the parameters in an assumed functional form for the dispersal kernel is difficult to justify. Obviously, infinite dispersal and unbounded acceleration of spread cannot apply to real systems or to real data, even if the best-fitting dispersal kernel confers these properties. However, the behaviour of accelerating spread still dominates over ecological time if the kernel is sufficiently 'fat-tailed' for large enough dispersal distances. Clark *et al.* (2001) show how to derive finite rates of spread by simulation from these badly behaved kernels. Integro-difference models have been used to estimate spread velocity for the house finch invasion of North America (Veit & Lewis 1996), the Holocene spread of trees (Clark 1998; Clark *et al.* 2001), and the spread of two herbaceous plant species (Neubert & Caswell 2000).

The qualitative prediction of the diffusion equation of a linear relationship between the square root of the area of an invasion and time seems to hold true for many invasions (Skellam 1951; Lubina & Levin 1988; Hengeveld 1989; Reeves & Usher 1989; van den Bosch *et al.* 1992; Nash *et al.* 1995), while spread rates of other invasions seem to increase exponentially with time (Perrins *et al.* 1993; Pysek & Prach 1993; Mihulka & Pysek 2001). In a review of 14 plants, seven demonstrated linear spread and seven exponential spread (Weber 1998). In some cases maps show that the invasion is proceeding from several locations or foci, which leads to much faster spread than from a single focus (Mack 1985). Shigesada *et al.* (1995) provide a mathematical model for this type of invasion, called stratified diffusion.

The robustness of the 'linear spread conjecture', i.e. the assumption that the asymptotic rate of spread is a linear function of time, is carefully examined by Weinberger *et al.* (2002), who focus on an abstract formulation of the problem that essentially includes all of the specific models as special cases. This work provides a clear description of the cases where the linear conjecture might fail, for example when a species is invading a region occupied by a competitor. However, verifying the conditions for which the linear spread conjecture holds can be difficult for some models.

Quantitative predictions from diffusion models parameterized using independent data have rarely predicted velocities that were much higher than observed. Velocities predicted by the linearized models provide an upper bound; thus, predicted rates that are too high could be because of the linear approximation (Mollison 1991). On the other hand, predicted rates of spread were often much lower than observed, suggesting departure from the model. For example, the rate of spread predicted for the cereal leaf beetle by the diffusion model, based on independent estimates of r and D, was about 20 times less than the observed rate (Andow *et al.* 1990). Because the model does not fit the independent estimates, Andow *et al.* (1990) hypothesize that the beetle is able to disperse much further

than estimated from mark-recapture studies, suggesting a long-distance dispersal mechanism such as human transportation. Similarly, in a number of other cases, the tendency to underestimate observed rates of spread has been attributed to the failure to accurately measure infrequent long-distance dispersal (Liebhold *et al.* 1992; Buchan & Padilla 1999; Neubert & Caswell 2000).

Mismatches between data and model predictions highlight one of the more important uses of the spread velocity models, which is in falsifying hypotheses about mechanisms of spread. Indeed, this was the original use put to the diffusion model by Skellam (1951) in his now classic analysis of the spread of oaks across Britain after the last glaciation. Testing hypotheses about dispersal mechanisms does not necessarily require independent estimates for both reproduction and dispersal. For example, if we can estimate the reproduction parameter, we can fit the dispersal parameters from the observed rates of spread and consider whether different dispersal mechanisms are consistent with the estimated dispersal kernel (Birks 1989). Where we do have independent estimates, a powerful use of the spread models is in confronting alternative dispersal models with the spread data (Allen *et al.* 1991; Clark 1998; Buchan & Padilla 1999; Clark *et al.* 2001).

Improvements to this approach include incorporating uncertainty and conducting sensitivity analyses. Mollison (1991) had emphasized, even under the assumptions about dispersal that lead to models of the form (eqn 1) how density dependent stochastic models could have different rates of spread from either the density dependent deterministic description or density independent models. Recent work emphasizing the stochastic aspects of invasions complements and extends the framework we have emphasized here (Lewis & Pacala 2001). This work could form the basis of careful statistical analyses because of its explicit inclusion of stochasticity, but as of now these models are quite complex and difficult to relate to the underlying data. Instead, we turn to simpler statistical approaches.

STATISTICAL FORECASTING APPROACHES

Thus far we have considered only approaches that use population dynamics models to predict spread. These are the most common approaches in the literature. However, there are powerful statistical tools, notably regression approaches, that could and perhaps should be used more commonly.

If the goal is to predict the future rate of spread, prediction from measured rates of spread may be more successful than parameterizing life-history models, especially if the linear spread conjecture holds. Measured rates of spread can sometimes be seen as implicit predictions of future rates, such as the classic muskrat example (reviewed in Okubo & Levin 2002), as long as the regression of square root of area on time is linear. However, no studies have properly estimated the uncertainty of these predictions. The use of standard statistical tools for forecasting would have the merit of providing confidence intervals for forecasted rates of spread. For example, with 95% confidence, what is the earliest date that we would expect to see the arrival of the invasion front in a particular region? Few studies supply standard errors for the regression used to estimate spread velocity, and even this is not enough for a prediction; a prediction interval is required.

Statistical tools that are extensively used in epidemiology, such as logistic regression and survival analysis, can be very useful in this application. These tools are special cases of generalized linear models (GLM) in which nonlinear models are transformed to linearity by a link function; a wide range of distributional forms can be used to model random effects (McCullagh & Nelder 1989). Most standard statistical packages include convenient implementations of GLMs. These tools are especially useful for dealing with the heterogeneous nature of landscapes at within-region scales, especially in modelling the combined influence of abiotic conditions and invasion pathways. Survival analysis, which uses a GLM with reciprocal link and exponential distribution, is used to predict the probability of surviving for at least time t, given a set of explanatory variables. This is ideally suited to the question of how long a location will remain free of an invading species. Jules *et al.* (2002) used survival analysis to study the spread of *Phytophthora lateralis*, a root pathogen of the riparian tree Port-Orford-cedar in Oregon. They found that survival times were longer in locations away from roads, and were also affected by catchment area and host abundance.

Logistic regression, which is GLM with a logit link and binomial distribution, is concerned with the probability of a binary event given a set of explanatory variables. Of particular application to invasions is the probability that a location will become invaded, given the current or previous distribution of the invader in the landscape and a suite of abiotic variables. By including a dispersal or 'contact' kernel as well as local explanatory variables, logistic regression models can be constructed to mimic the structure of neo-classical and stochastic models discussed above. In logistic regression, probability is modelled by the logit link. Thus the probability p that location i becomes invaded is modelled as

$$p_i = \frac{\exp(\eta_i)}{1 + \exp(\eta_i)} \tag{4}$$

For invasion problems, a useful general form for the linear predictor η_i is

$$\eta_i = \beta_0 + \sum_{j=1}^{m} \beta_j x_j + \beta_s \sum_{k=1}^{n} d(i,k) w_k \quad (5)$$

where the β are the linear regression coefficients, $x_1, x_2, \ldots, x_m$ is a set of explanatory covariates that are location specific, and the last-term models the 'force of invasion' summing over the potential contributions from invaded locations at an earlier time. The force of invasion term can be seen to closely parallel in eqn 3, incorporating a contact kernel $d(i,k)$ weighted by w_k a measure of the potential output of invaders from each distant location k. For example w_k might be measured as the areal extent of the invasion at the distant site. The contact kernel need not represent Euclidean distances but might, for example, represent the transportation of propagules between locations. The β_j can be interpreted as a measure of site specific susceptibility and β_s can be interpreted as a measure of the transmissibility of the invasion.

Using this approach, Havel *et al.* (2002) modelled the spread of the exotic *Daphnia lumholtzi* between Missouri lakes. Using a 7-year time series of lake occupancies, they modelled the probability of invasion of each lake in a year as a function of the limnological characteristics of the lake and its spatial position relative to invaded lakes in the previous year. They used a negative exponential contact kernel and estimated the exponent by profile likelihood.

TEMPORAL VARIABILITY IN REPRODUCTION AND DISPERSAL

Episodic temporal variation may be a significant reason for our limited ability to predict the spread of invasions. For example, new invaders may only succeed if their arrival coincides with a disturbance, nutrient pulse, or rainfall event (Davis *et al.* 2000). Directional environmental change, such as global warming, increased atmospheric CO_2 concentrations, and increased nitrogen inputs may also affect invasions. However, evidence remains scarce on the effects of either episodic or directional variation on invasive spread.

Invasion of *Bromus mollis* into California serpentine grasslands increased in years following heavy rains, decreased dramatically after two consecutive years of drought, and was locally fostered by gopher disturbance (Hobbs & Mooney 1991). The native shrub *Artemisia rothrockii* 'invaded' montane meadows in the California Sierra Nevada during a dry period following the El Nino from 1982 to 1983, and remained established in subsequent years (Bauer *et al.* 2002). Recruitment of the invasive honey mesquite *Prosopsis glandulosa*, however, did not depend on rainfall events in a Texas savannah (Brown & Archer 1999).

Most of the relevant theory concerns temporal variance in reproduction. For populations with discrete reproductive events, population growth is determined by the variance in growth rates as well as the mean; as variance increases, the realized population growth decreases. However, this difference disappears in continuous time models. Neubert & Caswell (2000) showed in an Integro-difference model of the form (eqn 3) that periodic or stochastic variation in reproduction decreased the rate of spread. Also, Clark *et al.* (2001) found that temporal variance has an especially strong effect on spread in models with fat-tailed dispersal kernels.

Dispersal as well as recruitment may vary over time, although this has been relatively little studied either empirically or theoretically. Neubert & Caswell (2000) found the velocity of spread is increased when variable dispersal and recruitment rates are positively cross-correlated over time. Moreover, virtually all environmental variation has a non-stationary, positively autocorrelated structure (Cuddington & Yodzis 1999). We are aware of no theoretical studies addressing the effect of temporal autocorrelation on the dynamics of invasion, but analogous work in population genetics suggests that it will increase the rate of spread compared with random variance.

The autocorrelated structure of most environmental variation, together with the potential for positive correlation between dispersal and reproduction, should promote high temporal variation in rates of spread of invasive populations. In turn, high temporal variation provides one possible explanation for the time lag that is often noted between the arrival of an exotic species and its rapid spread. Other possible explanations for the time lag include Allee effects, adaptation, hybridization, or the delayed arrival of mutualists. In still other cases, the perceived lag may be an artefact of a high threshold of detection, as Carey (1991) argued was the case for the Mediterranean fruit fly in California.

SPATIAL HETEROGENEITY IN THE INVADED ENVIRONMENT

Environmental heterogeneity may influence all stages of the invasion process: dispersal, colonization, and population growth (see With 2002 for an excellent review). A handful of empirical studies have measured and compared rates of spatial spread in more than one type of habitat, and these generally find environmentally dependent spread rates. For example, Williamson & Harrison (2002) found that exotic species used for post-mining revegetation spread rapidly into oak woodland, slowly into serpentine grassland and seeps, and not at all into serpentine chaparral. Differences in the establishment phase appeared to be responsible for the variable rates of spread.

The spatial arrangement of habitat is another aspect of heterogeneity that has attracted interest. In a model system at a small spatial scale, Bergelson *et al.* (1993) found that three species of herbaceous plants in the genus *Senecio*

moved greater distances in disturbed sites that were uniformly rather than patchily distributed. In an agar plate system, Bailey *et al.* (2000) found that the spread of soil organisms was dependent on whether or not the patches of high quality habitat were separated by greater or less than a critical threshold distance.

At larger scales, the colonization of lakes by exotic *Daphnia* is affected by distances between lakes (Havel *et al.* 2002), and the spread of the tree pathogen *Phytophthora lateralis* depends on both connectivity by roads and distances between trees (Jules *et al.* 2002). Many studies have noted high frequencies of invasive plants by roadsides, and while this could simply reflect the disturbed nature of the roadside habitat, it has also been interpreted to mean that roads are corridors for the spatial spread of weeds. Gelbard & Harrison (2003) found that Californian grasslands 1000 m from the nearest road had a significantly lower percentage of alien individuals than sites that were 100 or 10 m from the nearest road: environmental differences could not explain this effect.

Environmentally dependent rates of spread pose a potential limitation on the generality of models, because even for a single species, a model parameterized in one type of habitat may not make accurate predictions elsewhere. For green crabs (*Cancer maenas*) spreading in different estuaries along the western US coast, Grosholz (1996) found that a diffusion model parameterized in one location was no better at predicting the spread of the green crab at a different location than it was at predicting the spread of a wide range of other species.

How to formally integrate spatial heterogeneity into models is a complex problem. We briefly discuss three approaches: reaction-diffusion, gravity, and individual-based models.

The difficult problem with including heterogeneity is to do so in a way that allows relatively general conclusions to be drawn. Shigesada and colleagues (Shigesada *et al.* 1986; Shigesada & Kawasaki 1997) introduced a reaction-diffusion approach, in which the spread of a single species is examined in an environment with periodic variation in diffusivity and/or growth rate. The most important outcome of the work of Shigesada and colleagues is that the rate of spread is determined by the geometric mean of the spread rates in different environments. Thus unfavourable habitats can have a dramatic effect in reducing spead rates. Subsequently, Marco & Paez (2000) studied the spread of the invasive plant *Gleditsia triacanthos* and the native *Lithraea ternifolia* in Argentine forests. They applied population growth rates from good and poor quality sites to a reaction-diffusion model, similar to the approach of Shigesada *et al.* (1986) except that diffusivity was held constant, and were able to demonstrate successful predictions.

Cruywagen *et al.* (1996) used the reaction-diffusion approach to model the spatial spread of a genetically engineered microbe in the presence of a naturally occurring competitor in a spatially varying environment. When diffusivity alone varied, a large enough unfavourable patch (low diffusivity) prevented spread, but when diffusivity and carrying capacity were varied together, unfavourable patch length alone did not determine the course of the invasion. One problem with modelling variation in diffusivity is that it is not clear how it should operate; species could move more or less quickly through poor habitat, depending on the organism's ability to identify and to traverse poor habitat.

Gravity models assume that movements are not random but are biased by the attractiveness of destinations. Bossenbroek *et al.* (2001) showed that such an approach was better at predicting zebra mussel spread than a simple diffusion model. These authors argued that such models may explain why some invasions do not occur as a moving front wave, but as satellite introductions. Gravity models may also be applicable to the spread of human-vectored organisms because site 'attractiveness' is based on human behaviour.

Individual-based models are a popular framework for incorporating detailed information about individual fecundity, dispersal, and landscape structure, but lack the generality of approaches based on the reaction-diffusion framework. Variation in habitat suitability can easily be represented on a digital map, and may lead to unpredictable changes in patterns of individual dispersal. Higgins *et al.* (1996) used such an approach to make landscape-level predictions of the spread of *Pinus radiata* spread in South Africa.

Such simulation approaches are also useful for incorporating corridors and barriers to dispersal. Using a data-based stochastic model, Smith *et al.* (2002) studied spread of rabies on heterogeneous landscapes and found that rivers reduce local rates of spread sevenfold; they argue that the irregular pattern of rabies spread can be explained by the spatial distribution of major rivers combined with long-distance dispersal. Percolation theory is another approach that examines how spatial heterogeneity affects not only the rate of spread, but its final outcome. So-called 'percolation thresholds,' or critical levels of connectivity, determine whether the organism eventually reaches all suitable sites or is limited to a subset of suitable sites by distance or barriers. Percolation theory may be helpful in understanding the relationship between disturbance and the spread of invaders (With 2002).

COMPETITORS, MUTUALISTS, AND NATURAL ENEMIES

The concept of biotic resistance has played an important role in invasion biology, and there is a longstanding

expectation that diverse communities should be less invasible. However, relatively few studies have examined the rate of spatial spread of invasions as a function of diversity or interactions in the invaded community.

The red-whiskered bulbul, a cage bird originally from India, spread more slowly in continental areas where local bird diversity was high (Florida, USA and New South Wales, Australia) than on the Mascarene islands where diversity was less than half that of Florida and New South Wales (Clergeau & Mandon-Dalger 2001). However, competitive resistance from the native ant community did not affect rates of spread of Argentine ants in California (Holway 1998). In three related oak gall wasps in Britain, interspecific competition did not appear to affect rates of invasion (Walker *et al.* 2002).

Mutualisms have also been suggested to affect the spread of invasions; many examples are cited by Richardson *et al.* (2000) of species that became invasive only after the arrival of their pollinators or other mutualists. None of these studies, however, explicitly considered rates of spread. Likewise, while many studies have examined the impacts of natural enemies on invasions, very few of these have examined spatial spread. However, Lonsdale (1993) found that seed predators combined with folivores can considerably slow the rate of spread of the woody weed *Mimosa pigra* in wetlands in northern Australia.

Both classical reaction diffusion models (Okubo *et al.* 1989) and integro-difference equations (Hart & Gardner 1997) have shown that established competitors can slow an invading species. A superior resident competitor will prevent invasions, but an inferior resident will slow the invasion. In both cases, the invasion speed is a decreasing function of the competition strength, with zero invasion speed when the competitors are equal. Okubo *et al.* (1989) accurately predicted the rate of spread of an invasive gray squirrel in the presence of a native red squirrel in Britain using a diffusion-competition model.

The results of Okubo *et al.* (1989) depended on the approximation of the nonlinear model by a linear one, and may not be valid for all choices of parameters. Using the results proved by Lewis *et al.* (2002); Weinberger *et al.* (2002) derived a set of sufficient conditions for linear spread rate, in the Lotka–Volterra competition model with diffusion: 'sufficiently large dispersal of the invader relative to dispersal of the out-competed resident and sufficiently weak interactions between the resident and invader'.

Predators and parasitoids may have the ability to stop, slow or reverse an invasion of the victim species. Owen & Lewis (2001) considered the spread of a prey species followed by the spread of a more mobile predator. For one-dimensional spread on a continuous semi-infinite domain, the invasion speed of the prey is unaffected by the predator when the prey population does not have an Allee effect. When the prey has a strong Allee effect, i.e. per capita growth is negative for small populations, the prey invasion may be slowed or reversed by the predator. When space is modelled as a finite domain with no-flux boundary conditions, the predator may stop the prey invasion, but this occurs for a narrower range of parameters when there is no Allee effect than when there is one. Stationary, slowed, or reversed prey spread can also occur when space is semi-infinite and patchy.

Most invasion models do not consider the effects of species interactions on dispersal behaviour. However, in a host-parasitoid metapopulation model, French & Travis (2001) showed that dispersal in response to other species may influence the rate of spread. They found that the presence of a competitor can reduce the rate of spread of invasion for both fixed and density-dependent dispersal, but the spread rate is slowest when the invader has density-dependent dispersal and encounters an established competitor. Empirical evidence suggests that parasitoids disperse more as the parasitoid to host larvae ratio increases, suggesting an important role for competition.

EVOLUTION OF INVADING ORGANISMS

There is increasing evidence that evolution can influence the dynamics of invading populations (Hänfling & Kollmann 2002). Repeated introductions, hybridization with native species, novel selective regimes, and the stochastic effects of rapid expansion can change the genetic makeup of introduced populations. In turn, rates and patterns of spread can be shaped by local adaptation, the development of phenotypic plasticity, and/or changes in basic life history characteristics. However, we are far from understanding the role of evolution in invasive spread.

During the last century, North American populations of common reed (*Phragmites australis*) experienced a dramatic increase in range and abundance. It is now clear that this explosion was partly the result of the introduction of a novel European strain that replaced native genotypes and spread into new habitats (Saltonstall 2002). Such multiple introductions of distinct genotypes from disparate parts of a native range and their cryptic spread through introduced populations appears to be common (Geller 1996; Novak & Mack 2001).

Hybridization with native lineages also frequently creates new genetic combinations in introduced populations. In San Francisco Bay, introgression between the introduced smooth cordgrass (*Spartina alterniflora*) and the native cordgrass (*S. foliosa*) has created a hybrid swarm of novel genotypes of which many are invasive (Ayres *et al.* 1999). Ellstrand & Schierenbeck (2000) present 28 examples of plant species that became invasive only after a hybridization event.

Sexton *et al.* (2002) proposed a conceptual model in which phenotypic plasticity allows species to initially naturalize over a wide range of environments, and selection then favours local adaptation and an increase in local invasiveness. They argue this model is supported by the combination of broad plasticity and local adaptation in North American populations of saltcedar (*Tamarix ramosissima*). North American populations of cheatgrass (*Bromus tectorum*) display a similar pattern (Rice & Mack 1991). However, Californian populations of *Verbascum thapsus* have expanded across a wide elevational gradient almost exclusively by phenotypic plasticity with little local adaptation (Parker *et al.* 2003). Recent models have shown that migration between spatially variable populations favours the evolution of plasticity rather than local adaptation (Sultan & Spencer 2002).

García-Ramos & Rodríguez (2002) modelled the influence of local adaptation on invasion in a spatially heterogeneous environment, and found that the rate of adaptation to local conditions can be the key limiting factor to spread, especially when there are large differences between habitat patches. Paradoxically, in such cases, spread may be slowed by excessively high dispersal because of its homogenizing effect on genetic structure.

The emergence of new pathogens and the increasing spread of resistant strains has sparked interest in modelling how evolution and population biology interact to influence the dynamics of disease, but modelling efforts for other kinds of invasions are largely limited by the lack of relevant data. Simply identifying which genotypes within an invading population contribute most to spread is a significant empirical problem.

An important area for application of evolutionary models of invasives is the management of resistance to chemical and bio-control agents. Emerging strategies include the use of rotation systems for pesticides and herbicides, and the establishment of control-free areas to serve as refuges for non-resistant genotypes (Stockwell *et al.* 2003). Evolutionary models are also being used to forecast the spread of new pathogens and resistant genotypes (Mundt 1995).

CONCLUSIONS

The study of spread rates of invasions has been and continues to be one of the most exciting areas of interplay between theoretical and observational work in ecology. Recent work has emphasized how to move beyond the simplest results of linear rates of spread, yielding very different potential results for the rates of spread in some cases. This work provides new opportunities to match the theory and models and determine the processes that control spread rate. Yet, despite recent advances, there is both a need and an opportunity for new advances in both empirical and theoretical work. The unique opportunities presented by spread of invasive species in providing a dynamic window into ecological and evolutionary processes also provide challenges for future work.

Careful study of the dispersal process is required to fit the newest models, and, although there has been recent progress in statistical analyses of this problem there is substantial room for further improvement. Inclusion of other factors in the analysis such as spatial heterogeneity and other species provides an important challenge. In particular, we have shown that the empirical data do exist to support further development of the theory to include, especially, the role of heterogeneity and temporal variability. Just as studies of the purely ecological aspects of invasive species spread have both greatly improved our understanding of the process of spread and the underlying process of dispersal, future work focusing on evolutionary aspects is likely to have a large impact. This work could move beyond the important models of Shigesada *et al.* (1995) to include other factors.

Applications of the kinds of work presented here also are a strong justification for future efforts, as rate of spread can be predicted in at least some cases. In fact, recent efforts have begun to incorporate the study of control of invasions, even incorporating economic aspects. Given the great economic and biological impact of invasive species, the benefits of deeper understanding of the process of spread of invasive species will be great. One initial effort by Sharov & Liebhold (1998) illustrates how future inclusion of heterogeneity may be the key to a unified bioeconomic approach to understanding, and perhaps controlling, spread of invasive species. Additionally, further understanding of invasion dynamics is important in other applied areas, such as implementation of biological control (Fagan *et al.* 2002).

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