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Extinction thresholds: insights from simple models

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There are two types of deterministic extinction thresholds: demographic thresholds such as the Allee effect, and parametric thresholds such as a critical effective colonization rate or a minimum amount of available habitat for metapopulation persistence. I introduce briefly both types of thresholds. First, I discuss the Allee effect in the context of eradication strategies of alien species. Then, I consider an example of parametric threshold: the critical amount of suitable habitat below which a metapopulation goes deterministically extinct. I review how this spatial threshold changes in relation to the level of spatial detail and the complexity of the food web. Since classical metapopulation models assume an infinite number of patches, I proceed by considering how the extinction threshold is affected by environmental variability acting on a small number of patches. Finally, I consider recent work suggesting that if the network of connectivity among patches is not random but highly heterogeneous, the extinction threshold may disappear.

"We don't record flowers," the geographer said.
"Why not? It's the prettiest thing!"
"Because flowers are ephemeral."
"What does ephemeral mean?"
"It means, 'which is threatened by imminent disappearance.'"
(A. de Saint-Exupéry 1943, "The Little Prince.")

Introduction

Extinction has played a major role in the organization of life on Earth. Almost all species which have existed at some point have already gone extinct. This unavoidable outcome, however, has been aggravated in the last few centuries due to human activity. Since 1600, the extinction of more than 485 animal and 585 plant species has been recorded (Lawton & May 1995), although the real number of species going extinct due to habitat fragmentation, biological invasions, climate change, pollution, and other types of environmental disturbance can only be guessed.

We face the big challenge of predicting under what circumstances extinction will occur. Currently, there are two main schools of thought when dealing with extinction. One deals with stochastic processes, assumes that extinction is unavoidable, and predicts the time to extinction. The other is deterministic and estimates the conditions beyond which a population goes extinct. It predicts extinction thresholds, that is, critical

values of some variable of interest beyond which a population can no longer persist.

In this paper I will review very simple deterministic models which predict extinction thresholds. In particular, I will focus on demographic thresholds, that is, critical population values below which the population goes extinct, and thresholds derived from the reduction in the fraction of available patches in a metapopulation context. These models are an oversimplification of reality, but they can provide *rules of thumb* to better understand extinction. I will explore how these thresholds change with the level of spatial resolution, the complexity of the food web, and the effect of environmental variability acting on small networks of patches. One goal is to explore whether these simple rules of thumb can be used regardless of the models' simplifying assumptions. Finally, I will consider how the shape of the connectivity distribution of nodes (e.g., patches) may affect the extinction threshold and even make it disappear.

Demographic thresholds: the Allee effect

The Allee effect is a mechanism first described by Allee *et al*. (1949) and describes any process in which any component of fitness is correlated with population size (Courcham *et al*. 1999, Stephens & Sutherland 1999, Stephens *et al*. 1999). In a population dynamics context, the Allee effect depicts a situation in which population growth rate decreases below some critical minimum density. In some circumstances this growth rate may even be negative, originating an extinction threshold. Different mechanisms responsible for this demographic threshold are failure to locate males, failure to satiate predators, and inbreeding depression.

The Allee effect can be incorporated into a logistic growth model in the following way (Dennis 1989, Amarasekare 1998, Keitt *et al*. 2001):

$$\frac{dn}{dt} = nF(n) = n\gamma\left(1-\frac{n}{k}\right)\left(\frac{n-c}{k}\right), \qquad (1)$$

where n is population size, γ is the intrinsic rate of natural increase, k is the carrying capacity, and c is the threshold population size below which $F(n) < 0$.

In the absence of the Allee effect, $F(n)$ is always positive, as shown in Fig. 1. For this situation there is a single solution, $n^* = k$, which is stable. On the other hand, Eq. 1 has two solutions, $n^* = k$ which is again stable, and $n^* = c$, which is unstable (population either increases or decreases towards extinction, Fig. 1).

A model like Eq. 1 may seem an oversimplification of reality, multiple factors are missing. However, it defines a threshold in very simple terms. One can plot the change in population size for different values of n in real data, and detect whether growth rate becomes negative at some critical density. One does not need further biological details. Also, although the previous model is deterministic, the Allee effect and demographic stochasticity may interact to drive a population extinct (Lande 1998, Dennis 2002). Furthermore, demographic stochasticity by itself can cause a population to decrease, which would constitute a type of stochastic Allee effect (Lande 1998).

The importance of the Allee dynamics in extinction has been widely explored in the context of conservation biology (Lande 1988, Groom 1998). Endangered species tend to have small densities, and under this situation, they may go extinct due to the Allee effect, demographic stochasticity, or a combination of both. This interaction between stochastic processes and the Allee effect is also important in determining the likelihood of successful establishment by alien species (Hacou & Iwasa 1996, Fagan *et al*. 2002, Petrovskii *et al*. 2002), and the extent of the range expansion during invasions (Lewis & Kareiva 1993, Keitt *et al*. 2001).

However, almost no study has incorporated the Allee effect in the context of biological control. Large efforts to eradicate alien animal and plant species have increased with the acceleration in the arrival of alien species due to global travel and commerce (Simberloff 2001). The introduction of alien species is now believed to be one of the major causes of biodiversity loss worldwide. It is usually accepted in studies of pest eradication, that eradication can only be achieved by the elimination of all the individuals. Liebhold and Bascompte (2003) have studied the

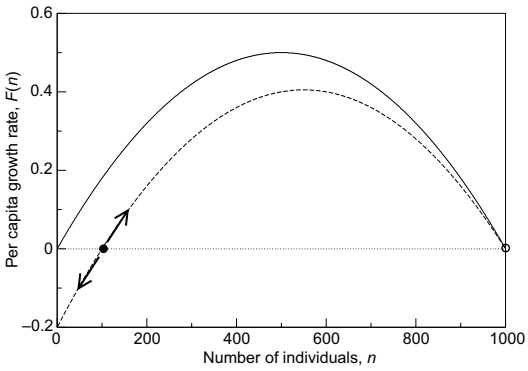

Fig. 1. Per capita growth rate as a function of population size according to Eq. 1. Continuous line depicts a situation with no Allee effect (c = 0), and broken line corresponds to a case with an Allee effect (c = 100, indicated by the solid dot). In the first case, there is a single solution n^* = k (empty dot), which is stable. In the last case, there is an additional, unstable solution indicated by the solid dot. Population sizes larger than c have a positive per capita growth rate and will grow until reaching their carrying capacity k. Population sizes below c have a negative per capita growth rate, and so, will go extinct (indicated by the arrows). Other parameters are: γ = 2, k = 1000.

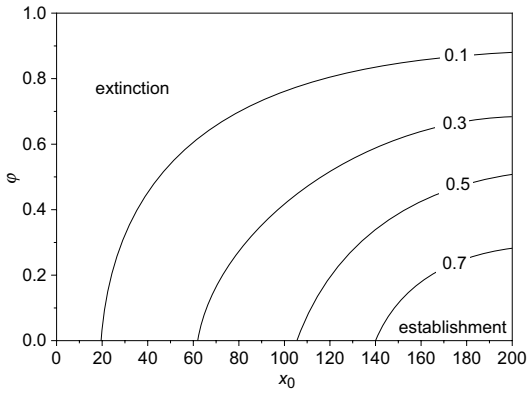

Fig. 2. The simulated probabilities of gypsy moth establishment are plotted as a function of the population level at the time of treatment (x_0), and eradication rate (fraction of individuals killed, φ). Based on Liebhold and Bascompte (2003).

combined role of stochasticity and Allee effect on the extinction of isolated populations following eradication treatments. As a case study, they used historical data on the dynamics of isolated populations of gypsy moth (*Lymantria dispar*) in North America. This insect species, a native to most of the temperate forest in Eurasia, was accidentally introduced in the east coast of the USA in 1869. Since then, it has been expanding through northeastern USA and southeastern Canada, where it has defoliated large forest areas (Liebhold *et al*. 1992).

Liebhold and Bascompte (2003) used temporal data on the total number of males of gypsy moth caught in pheromone traps to fit parameters of a discrete time version of Eq. 1, which can be written as follows:

$$\ln(r_t) = \gamma\left(1 - \frac{n_t}{k}\right)\left(\frac{n_t - c}{k}\right), \qquad (2)$$

where $r_t = n_{t+1}/n_t$ is the change in population density, and other parameters are as in Eq. 1. It can be noticed that when populations are very low, x_t is insignificant compared with k and thus $(x_t/k) \sim 0$. Equation 2 then becomes:

$$\ln(r_t) = \gamma\left(\frac{n_t - c}{k}\right) = \frac{-c\gamma}{k} + \frac{\gamma}{k} n_t. \qquad (3)$$

In this way, at the very low densities observed at the initial stages of a biological invasion, the Allee effect can be represented simply by $\ln(r_t)$ as a linear function of n_t, with intercept $-c\gamma/k$ and slope γ/k (Liebhold & Bascompte 2003). By fitting the data on gypsy moth populations to Eq. 3, Liebhold and Bascompte (2003) estimated the Allee extinction threshold as the negative intercept ($-c\gamma/k$) divided by the slope (γ/k), which yield $c \sim 107$ males trapped per colony. Thus, when the number of males is lower than this threshold, the population will go deterministically extinct. Note, however, that the Allee effect interacts with environmental stochasticity, which would transform the extinction threshold into a probability function (Lande 1998, Dennis 2002, Liebhold & Bascompte 2003). Thus, Liebhold and Bascompte (2003) modified Eq. 3 to add an additive environmental noise term, which is estimated from the data as the SD of the residuals (this makes the simplifying assumption that there is no sampling error and all the variation is due to environmental stochasticity).

With the parameterized model, Liebhold and Bascompte (2003) studied the probability of success of eradication treatments of varying strength. Figure 2 shows results of simulations plotting the probability of gypsy moth establishment as a function of the pre-treatment density

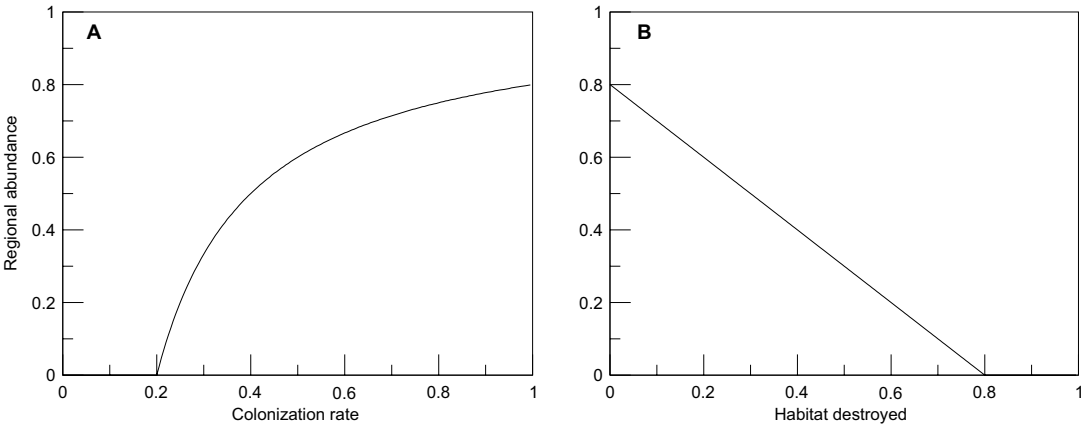

Fig. 3. Parametric extinction thresholds. Regional abundance is plotted as a function of (**A**) colonization rate *c*, and (**B**) fraction of habitat destroyed *D*, according to Eq. 5. Parameters are: $e = 0.2$, $D = 0$ (**A**); $c = 1$, $e = 0.2$ (**B**).

and eradication mortality rate. For example, they showed that as long as isolated populations were relatively low (< 100 males captured in pheromone traps), eradication treatments were likely to succeed if able to eradicate at least 80% of the individuals.

Parametric thresholds

Another type of threshold is the one given by a critical value in some meaningful parameter (e.g. the ratio extinction rate to colonization rate; a critical amount of habitat destroyed). We will briefly consider one example from metapopulation and epidemiological theory. We will focus on simple deterministic models. An extension of these results, including both more realistic and stochastic models, can be found in Ovaskainen and Hanski (2003).

Consider the following general metapopulation model which describes the dynamics in the fraction of patches occupied by the metapopulation (p) as a balance between extinction and colonization:

$$\frac{dp}{dt} = cp(1-p) - ep, \qquad (4)$$

where c and e represent the colonization and extinction rates, respectively. This very simple model was first described by Richard Levins (who coined the term metapopulation, Levins 1969a). The previous model describes an idealistic situation composed by an infinite number of available patches. We will return to some of these assumptions later on. Different versions of Eq. 4 have since then been used by many authors (e.g. Hanski & Gilpin 1997, Hanski 1998, 1999).

As easily seen, the long-term density of the metapopulation (the solution of Eq. 4) is $p^* = 1 - e/c$ if $e < c$, and $p^* = 0$ otherwise. This discontinuity in the solution is due to the existence of a threshold. The metapopulation will exist as far as the ratio $e/c > 1$. The extinction threshold lies in the equality (Fig. 3a).

Let us assume now that a fraction D of those patches is permanently destroyed. D can be incorporated into the Levins model as a reduction in the fraction of patches available to be colonized $(1 - p)$ in the following way:

$$\frac{dp}{dt} = cp(1-D-p) - ep. \qquad (5)$$

One can explore how the long-term regional abundance ($p^* = 1 - D - e/c$), decreases as D is increased. Interestingly enough, there is a critical value D_c beyond which the metapopulation goes extinct even when some habitat is still available (Fig. 3b). This extinction threshold is given by:

$$D_c = 1 - e/c. \qquad (6)$$

The concept of spatial extinction thresholds was introduced by Lande (1987) in a slightly

different version of Eq. 5 which included territoriality and life history. His model was applied to the case of the northern spotted owl (*Strix occidentalis caurina*).

Equation 5 is a very crude description of a real situation, and colonization and extinction rates may be difficult to estimate. But note that the critical amount of habitat destroyed at which the metapopulation goes extinct (Eq. 6), coincides with the fraction of habitat occupied when all habitat is available (solution of Eq. 4). This can be easily seen by looking at Fig. 3b: the value at which the curve crosses the two axis is the same. This important result, coined as the *Levins rule* (Hanski *et al*. 1996), is a useful rule of thumb. As emphasized by Nee (1994; *see* also Bascompte & Rodríguez-Trelles 1998), one does not need information of the relevant demographic processes to estimate the extinction threshold (we do not have to estimate parameters such as c and e).

The concept of extinction threshold illustrated by Eq. 6 is very general. Even when we have focused our discussion in a metapopulation context, models like Eq. 5 have been used in epidemiology, a comparison emphasized by Nee (1994), Lawton *et al*. (1994), and Bascompte and Rodríguez-Trelles (1998). In the epidemiological context, we would be describing the fraction of host infected by an infectious disease as a function of transmission rate (the rate at which susceptible individuals become infected after exposure to infected individuals), and clearance rate (the rate at which infected individuals recover). In epidemiology, D would correspond to the fraction of hosts vaccinated (unavailable to be colonized by the disease). The threshold in this context has been called *eradication threshold*. One would not need to vaccinate all individuals for the disease to disappear, a concept fully used in epidemiology (Anderson & May 1991).

The possibility that extinction thresholds may be predicted by simple models is supported by theory on phase transitions. A threshold can be considered as a critical point separating a phase transition (like the transition from liquid to solid). Below the threshold the state would be persistence, and beyond it would be absence. Near these critical points, several properties are described by a few variables such as critical exponents which do not depend upon the details of the situation but only on the dimension and the degrees of freedom of the system considered (Schroeder 1991, Solé *et al*. 1996). The behavior of the system is captured by very simple rules. Thus, regardless of simplifying assumptions, models such as Eq. 5 may be good metaphors of more complex scenarios near the extinction thresholds.

Up to here, we have reviewed a simple model, and we have learned the existence of an extinction threshold despite some habitat is still available. We have also learned that this extinction threshold can be predicted as the long-term regional abundance when all habitat is available. That is, we do not need any demographic information. However, one has to keep in mind that, as with each model, we have made a number of assumptions. In particular, the Levins model is a spatially implicit model which assumes that all patches are equidistant from each other. This means that when one individual leaves, it has the same probability to colonize every patch independently of its distance. This is a crude assumption for a great number of real situations (e.g., Hanski 1999). Also, we have focused on single species models. Since species are not isolated from each other but form food webs, it would be interesting to see how the extinction threshold depends on the complexity of such a web of interactions. Next, we will consider how the extinction threshold changes when (1) space, (2) the food web in which a species is embedded, and (3) environmental variability acting on a small network of patches, are explicitly considered.

Extinction thresholds in spatially explicit landscapes

The alternative to mean field-models such as the Levins model are spatially explicit models. In these models, space is considered like a lattice of points. Each point is determined by its specific coordinates. Dispersal can then be treated as a localized process. For example, an occupied patch has a probability to send propagules and colonize only one neighboring patch (for example one of its four nearest patches). Bascompte

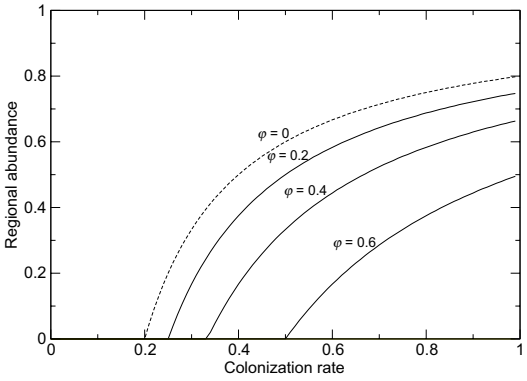

Fig. 4. Extinction thresholds as a function of the colonization rate and the spatial correlation φ according to Eq. 7. Broken line ($\varphi = 0$) corresponds to the homogeneous case (Eq. 5) plotted in Fig. 3a. Other parameters as in Fig. 3a.

and Solé (1996) considered such a scenario and explored how the threshold predicted by Lande (1987) is affected by explicit space. In particular, we are interested in checking whether the Levins rule is affected or not by such an additional level of reality.

As shown by Bascompte and Solé (1996), the regional occupancy of the metapopulation for the spatially explicit situation was lower than that predicted by the equivalent mean-field model. The extinction threshold took place sooner, that is, for lower values of habitat destruction. Thus, predicting the extinction threshold as the amount of empty habitat when all habitat is pristine, would lead to an overestimation. Further models incorporating non-random habitat loss have been developed by Dytham (1995), Hill and Caswell (1999), and With and King (1999). More recently, correlation in habitat destruction has been addressed analytically by means of pair-approximation techniques (Hiebeler 2000, Ovaskainen *et al*. 2002) and the concept of the metapopulation capacity of a fragmented landscape (Hanski & Ovaskainen 2000, Ovaskainen *et al*. 2002, Ovaskainen & Hanski 2003). I will not pursue this work any further here. Instead, I will further consider the case of the Levins rule in situations with localized dispersal.

If the extinction threshold takes place for lower values of habitat destroyed when we depart from the global mixing assumption of mean-field models, one can ask what component of explicit space is responsible for this and whether the change in the threshold can be corrected by some meaningful spatial variable. Spatially explicit models may be more accurate than their implicit counterparts, but this is easily explained because they contain many more variables (as many as sites). However, as noted by many authors, there is a trade-off between reality and simplicity. We need to understand what is essential and what is superfluous (Levin 1992, Levin & Pacala 1998). Bascompte (2001) tried to bridge between spatially implicit and explicit models by capturing spatial heterogeneities by means of a single macroscopic variable φ. This variable can be defined as $\varphi = p\,(1|1) - p(1)$, where $p(1)$ is the probability of a site being occupied, and $p(1|1)$ is the conditional probability of a site being occupied given that one of its four nearest neighbors is occupied. $p(1|1)$ has been called doublet density (Matsuda *et al*. 1992) or local density. Since mean-field models assume zero-correlation or spatial homogeneity (and thus $p(1|1) = p(1)$), φ describes the increase in local density above what we would observe in a homogeneous system. Spatial correlation can be incorporated into the Levins model (Bascompte 2001) in the following way:

$$\frac{dp}{dt} = cp(1 - D - p)(1 - \varphi) - ep. \qquad (7)$$

Bascompte (2001) showed that the aggregate measure of spatial correlation encapsulates well spatial heterogeneities: the behavior of Eq. 7 is very similar to the one exhibited by the equivalent spatially explicit simulation. Figure 4 shows how the long-term regional abundance (and the extinction threshold) for Eq. 7 depends on spatial correlation φ. The extinction threshold for Eq. 7 can be defined as follows:

$$D_c = (1 - e/c)(1 - \varphi^*), \qquad (8)$$

where φ^* is the spatial correlation at equilibrium near the threshold. If we compare the previous expression with Eq. 6, we can easily see that the extinction threshold is reduced by the term $(1 - \varphi^*)$. The Levins rule is still valid, but has to be "corrected" by the term measuring the degree of departure from the assumption of zero-correlation. As noted, both thresholds coincide

when spatial correlation is zero. Of course, this is an approximation. It is only appropriate if the aggregate model is a good description of the spatially explicit situation, but it is useful in relating the extinction threshold to some variable of spatial correlation.

Extinction thresholds and food web structure

Up to here we have considered single-species models. Tilman and colleagues (Tilman *et al.* 1994, 1997) extended previous work by Nee and May (1992) to consider the scenario of n competing species. Their model is a direct extension of the Levins model assuming a trade-off between competitive and dispersal abilities. They assumed that a superior competitor can instantly colonize a patch occupied by an inferior competitor, excluding it from such a site. If species are ranked by their competition ability, we can write the following equation for species i:

$$\frac{dp_i}{dt} = c_i p_i \left(1 - D - \sum_{j=1}^{i} p_j\right) - e_i p_i - \sum_{j=1}^{i-1} c_j p_i p_j \quad (9)$$

As described by the the first term in the previous equation, species i can colonize any non-destroyed site which is not occupied by superior competitors or individuals of its own species (species 1 to i). The last term indicates that species i can be displaced by any competitively superior species arriving at its patch. The long-term abundance of species i, that is, the solution of Eq. 9, can be written as (Tilman *et al.* 1994):

$$p_i^* = 1 - D - \frac{e_i}{c_i} - \sum_{j=1}^{i-1} p_j^* \left(1 - \frac{c_j}{c_i}\right). \quad (10)$$

It can be seen that the solution for the superior competitor is the same as the solution for the Levins model (Eq. 5), that is, $p_1^* = 1 - D - e_1/c_1$. In other words, as noted by Tilman *et al.* (1994), the Levins rule is again at work. The superior competitor goes extinct when the fraction of habitat destroyed equals the fraction of habitat occupied when all habitat was pristine.

Tilman *et al.* (1994) assumed that species abundance in a pristine habitat follows a geometric series, that is $p_i = q(1 - q)^{i-1}$ where q is the abundance of the best competitor. Assuming that extinction rates are the same for all species ($e_i = e_j = e$), we found that the required colonization rates compatible with the geometric series are given by $c_i = e/(1 - q)^{2i-1}$. Tilman *et al.* (1994) substituted this result into Eq. 10 to obtain the extinction thresholds, that is, the critical values of habitat destruction at which any given species becomes extinct. For the ith species, we have

$$D_{ci} = 1 - (1 - q)^{2i-1}. \quad (11)$$

The previous equation provides an interesting result already found by Nee and May (1992): species go extinct from the best competitor to the poorest competitor as more habitat is destroyed. Thus, the species more affected by habitat loss are the competitively superior, more dominant species. Furthermore, Tilman *et al.* (1994, 1997) found that the time lag between habitat destruction and the consequent extinction events may be of the order of hundreds of generations. They termed this delay the "extinction debt". The implication is that habitat loss may be more detrimental than previously thought, since some species, even when still present, will be doomed to extinction in the near future.

The next step is to introduce trophic structure, that is, to move from one trophic level to a web of trophic interactions. Several authors had already studied prey-predator metacommunity models (May 1994, Bascompte & Solé 1998, Swihart *et al.* 2001, Holt 1993, 1997). If predators are specialists, these studies showed that the higher trophic-level species become extinct sooner than the lower trophic-level species. Also, the effect of habitat loss may now be a complex interaction between different trends.

One approach recently developed by Melián and Bascompte (2002) is to represent different food web structures and to study how such structure modify the extinction threshold of the top predator. Melián and Bascompte (2002) considered four trophic web modules: simple food chain, omnivory, apparent competition, and intraguild predation. Each structure contained three trophic levels with three or four species. If we represent by r, c and p the regional abundance

of resource, consumer, and predator, respectively, we can write an extension of the Levins model for each trophic structure. For the simplest case, the simple food chain, we can write:

$$\frac{dr}{dt} = c_1 r(1-r-D) - e_1 r - \mu_1 rc, \qquad (12)$$

$$\frac{dc}{dt} = c_2 c(1-c-D) - e_2 c - \psi_1 c(1-r) - \mu_2 cp, \qquad (13)$$

$$\frac{dp}{dt} = c_3 p(1-p-D) - e_3 p - \psi_2 p(1-c), \qquad (14)$$

where the bulk of parameters are as above. The new parameters ψ_i and μ_i can be interpreted as follows. Colonization of a patch by the predator occurs independently of patch occupancy by its main prey. Therefore, in patches without prey, intermediate and top species pay an added cost ψ_i in terms of an increase in the rate of local extinction for mistakenly colonizing an inferior resource patch. In the case of a perfect generalist species, ψ_1 and ψ_2 would be equal to 0 (Swihart *et al*. 2001). μ_i represents the increase in mortality due to predation. This last parameter allows two scenarios to be considered: donor control ($\mu_i = 0$), and top-down control ($\mu_i > 0$).

From this simple food chain model we can build more complexity and introduce the other food webs structures (*see* Melián & Bascompte 2002 for details). For example, in omnivory, the predator consumes both consumer and resource; in apparent competition, there are two consumer species sharing the same predator; in intraguild predation, a trophic link is introduced between the two intermediate species (*see* inserts in Fig. 5). Figure 5 shows the reduction in the number of species as habitat is destroyed. As noted, food web structure alters the response of top species to habitat loss. This means that extinction thresholds are not only determined by life history traits, competitive-colonization abilities, and landscape properties, but also by the complexity of the food web. Omnivory confers the higher persistence to the top species, while interactions between the two intermediate species decrease its patch occupancy. Only the extinction threshold of the resource is constant and identical to that predicted by the Levins rule (since when all other species have gone extinct, we are dealing with a single species metapopulation model).

Critical number of patches for persistence

As noted above, metapopulation models assume an infinite number of patches. This assumption is unrealistic for many patchily distributed populations which are restricted to a small number of patches. Previous theory will be of little application in here because when the number of patches is so small, the effect of a fluctuating environment will be extremely important. Our goal in this section is to derive an extinction threshold for metapopulations living in a small network of patches and submitted to environmental stochasticity. Then we will be able to write this threshold as a function of the number of patches itself. Note that this is a slightly different approximation than the colonization-extinction stochasticity reviewed by Ovaskainen and Hanski in this issue.

Bascompte *et al*. (2002) used the concept of geometric mean fitness (GMF) to derive such an extinction threshold. The GMF is a widely used concept in ecology and evolutionary biology to understand persistence in fluctuating environments (Lewontin & Cohen 1969, Levins 1969b Gillespie 1974, Kuno 1981, Metz *et al*. 1983, Klinkhamer *et al*. 1983, Yoshimura & Jansen 1996, Jansen & Yoshimura 1998). The model, similar to early population genetic models assuming a mating pool and dispersal into separate demes (Levene 1953, Dempster 1955), and to models developed in relation to the evolution of dispersal (Kuno 1981, Metz *et al*. 1983), assumes spatial structure and geometric growth. The last assumption may be unrealistic for a large number of populations in which density-dependence operates. But the extinction threshold is independent of whether density-dependence operates or not. This, of course, would not be the case if we were trying to estimate long term population dynamics.

Bascompte *et al*.'s (2002) model assumes that juveniles (e.g. larvae) enter a common migrant pool and are redistributed as adults into *n* patches for reproduction. The growth rate at each patch is a random variable with some stationary probability-density function. This represents environ-

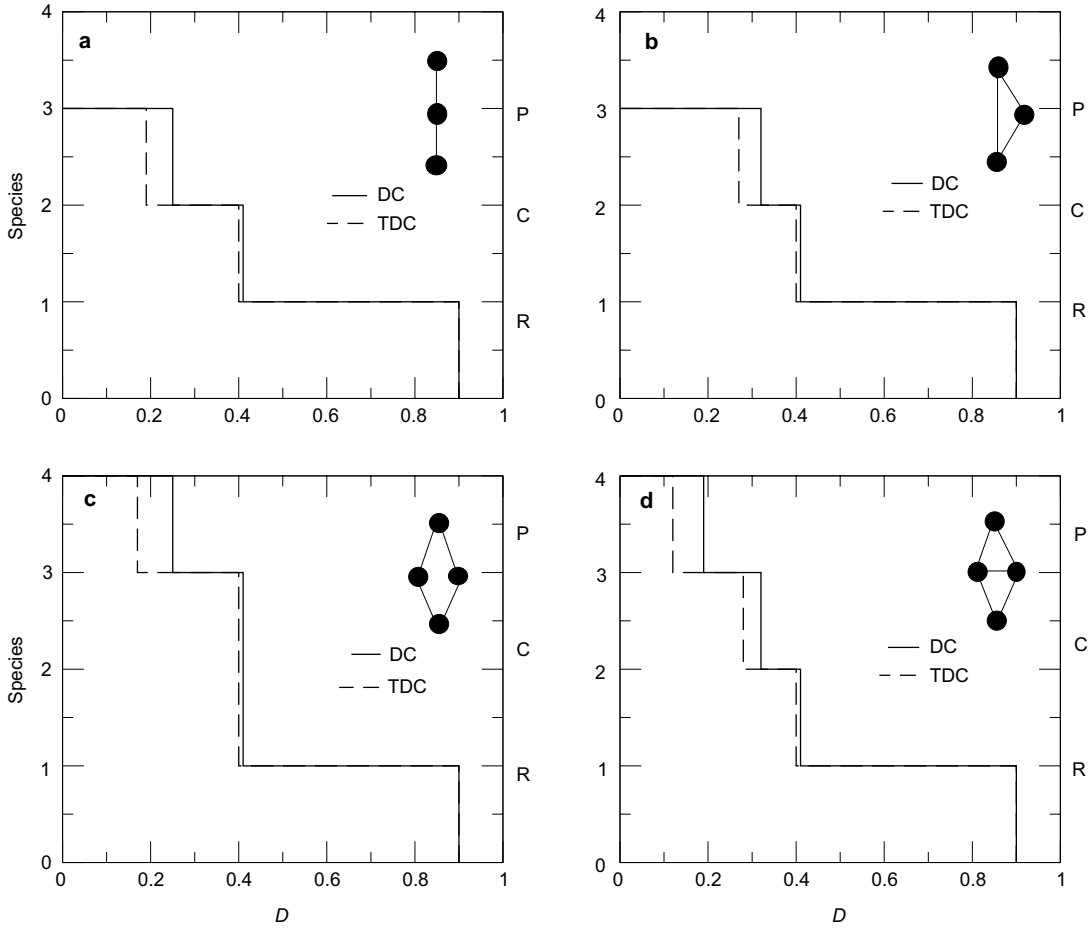

Fig. 5. Reduction in the number of species in four models of food webs as the amount of habitat destroyed is increased. The inset in each figure depicts the type of trophic web: (**a**) simple food chain; (**b**) omnivory; (**c**) apparent competition; (**d**) intraguild predation. P, C, and R stand for predator, consumer and resource, respectively, and represent the species which will go extinct next. Modified from Melián and Bascompte (2002). Continuous line represents donor control, and discontinuous line represents top-down control. Note the position of the extinction thresholds in relation to the type of food web.

mental stochasticity. Note that, as opposed to the patch occupancy (extinction-colonization) models reviewed in previous sections, we are now considering a metapopulation model with explicit local dynamics.

If N_0 is the initial metapopulation size, and $R_{i,0}$ is the growth rate of patch i at year 0, the metapopulation size in the next generation is given by:

$$N_1 = \frac{1}{n} N_0 \sum_{i=1}^{n} R_{i0} = N_0 \overline{R}_0, \qquad (15)$$

where $\overline{R}_0$ is the arithmetic mean of growth rates among the n patches at year 0. Since the metapopulation size one generation later is $N_2 = N_1 \overline{R}_1 = N_0 \overline{R}_0 \overline{R}_1$, an expression for the metapopulation size after t generations can be generalized in the following way:

$$N_t = N_0 \prod_{j=0}^{t-1} \overline{R}_j. \qquad (16)$$

If we denote the geometric mean (GM) of $\overline{R}$ by $G(\overline{R}) = \left(\prod_{j=0}^{t-1} \overline{R}_j \right)^{1/t}$, Eq. 16 can be written as:

$$N_t = N_0 G(\overline{R})^t. \qquad (17)$$

Then, the metapopulation will persist in the long-term ($N_t \geq N_0$) if (Lewontin & Cohen 1969,

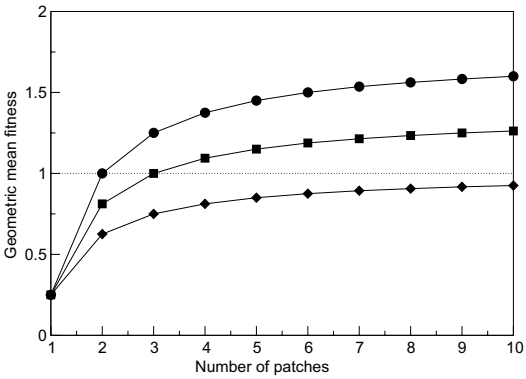

Fig. 6. Geometric mean fitness GMF according to Eq. 19 as a function of the number of patches. Dotted line indicates the extinction threshold. Metapopulation goes extinct when GMF is lower than one. $\overline{R}$ = 1.75, σ^2 = 3 and spatial correlation is 0 (circles), 0.25 (squares), and 0.5 (diamonds).

Kuno 1981, Yoshimura & Jansen 1996, Bascompte *et al*. 2002):

$$G\left(\overline{R}\right) \geq 1. \qquad (18)$$

The GM in Eq. 18 can be approximated by an expression known as *variance discount approximation* which is derived by means of Taylor expansions (Gillespie 1974, Yoshimura & Jansen 1996). For the case of a metapopulation inhabiting n patches, this can be written as (Bascompte *et al*. 2002):

$$G\left(\overline{R}\right) \approx \overline{R} - \frac{\sigma^2 + (n-1)\mathrm{cov}}{2n}, \qquad (19)$$

where $\overline{R}$ and σ^2 are the arithmetic mean and variance, respectively, of R within patches, and cov is the spatial covariance between any pair of growth rates. As noted, both the variance and the spatial covariance in growth rates tend to reduce the GM, while GM increases with the number of patches (Fig. 6). This relationship can be understood if we consider that: (1) it is the geometric mean and not the arithmetic mean which is relevant for population growth (it is a multiplicative process, growth rates at different years do not add but multiply each other); (2) the GM is equal to the arithmetic mean when variance is zero and decreases as σ^2 increases (while the arithmetic mean is not affected); (3) the variance in this context is analogous to sampling error associated to the sampling of the spatial arithmetic mean of growth rates (sampling error, and so variance, decreases with n).

Equation 19 provides an analytical expression which can be used to define the extinction threshold as a function of n. The critical number of patches at which the metapopulation goes extinct is:

$$n_c = \frac{\sigma^2 - \mathrm{cov}}{2\overline{R} - \mathrm{cov} - 2}. \qquad (20)$$

As n is reduced below this critical value n_c, the metapopulation becomes extinct despite that there are still available patches. All the information we need in order to measure this threshold are estimates of growth rates at different times and locations. Note that any further complexity in demographic parameter such as the density-dependent term does not affect n_c. This having been said, however, we have to keep in mind that the previous model is based on a series of assumptions such as global mixing in a pool and equidistant redistribution. The threshold will, of course, be modified if we depart from this set of assumptions. Specifically, both partial mixing (a fraction of larvae stays in its patch), and an uneven redistribution among patches (patches which are bigger or are located closer to the pool may attract more individuals) tend to reduce the geometric mean fitness and so locate the metapopulation closer to its extinction threshold. However, as noted by Bascompte *et al*. (2002), the threshold is very robust for moderate deviations of the assumptions.

Although the previous model deals with environmental stochasticity, it has a deterministic structure, and it provides a deterministic criterion for persistence. This is different in truly stochastic metapopulation models in which a time to extinction is predicted. Despite this difference, an analogous dependence on n in stochastic colonization-extinction models can be found for the metapopulation time to extinction or the occupancy state (*see* Ovaskainen & Hanski 2003). Stochastic models explore the effect of a small network of patches. Stochasticity works even in a constant environment. In here, we have focused on the effect of environmental stochasticity on a small network of patches.

Until here we have revolved around the very concept of extinction thresholds which seems to

be a very general property. However, we will turn in the next section to a specific situation in which such extinction thresholds disappear. We will come back to epidemiology, but in this case the viruses are not biological, but computer viruses.

Absence of eradication thresholds in scale-free networks

In the previous sections we have considered that dispersal is either a global mixing or it is restricted to the nearest neighbors. In both cases, each patch is connected to the same number of other patches. In epidemiology, this means that each infected individual has the same average probability of infecting a susceptible individual. But this may not be necessarily the case as we will see in this section.

Pastor-Satorras and Vespigniani (2001a) studied the persistence time of several computer viruses. This was found to be much larger than expected. It did not seem that there was an extinction threshold: viruses remained even when a large number of computers had the appropriate anti-virus. Note that despite the different nature of a computer, the system is analogous to an epidemiological system. Computers interact among them (they are connected through the Internet). If a computer is "susceptible" it can become infected if it interacts with an infected computer. A number of computers are then "vaccinated" against such a virus. Thus, standard theory on epidemiology has been applied to computer viruses. Theory predicts that after a critical fraction of computers have been vaccinated, the virus disappears. But this was not what Pastor-Satorras and Vespigniani (2001a) found. What was the reason for the discrepancy?

As with the above models, it was assumed that computers are randomly connected. But Albert *et al*. (2000) had described that this is far from true. These authors had proven that the probability $p(k)$ that a computer is connected to k other computers follows a power law of the form:

$$p(k) \propto k^{-\gamma}. \qquad (21)$$

The previous distribution is called scale-free, because the tail of the distribution is so large that there is no characteristic mean (Schroeder 1991). It is not defined for a particular scale. For example, if k is rescaled (multiplied by some constant α), $p(\alpha k)$ is still proportional to $(\alpha k)^{-\gamma}$. This is not the case for the Gaussian distribution characteristic of a random process. In the latter, there is a well-defined average, the scale of the system. Rescaling the system would not maintain the proportionality, because in such a case, the proportionality is defined only at the appropriate scale.

The scale-free distribution in computer connectivities means that, as opposed to a random network, the network of computer connections is highly heterogeneous: the bulk of computers are connected to a small number of other computers, but a few computers interact with a huge number of other computers. These *hubs* would be impossible to observe in a random network (for a general introduction to scale-free networks *see* Barabasi 2002 and Buchanan 2002).

The question then was to see whether this scale-free distribution was the cause of the lack of eradication thresholds in computer viruses. Pastor-Satorras and Vespignani (2001a, 2001b) used a similar model to Eq. 4 but evolving in several types of interacting networks.

If we assume that the network is random, then we will have that the number of connections per node follows a Gaussian distribution. There is an average number of connections and the probability of having a node much more connected or much less connected than the average drops very fast. We can say that connectivity has only small fluctuations, that is, $\sigma_k^2 \sim \bar{k}$, where $\bar{k}$ and σ_k^2 are the mean and variance, respectively, of the number of connections per node. Thus, we can assume that each node has the same number of links, $k \sim \bar{k}$, and this is equivalent to the homogeneity assumption of the mean-field approximation, or of the spatially explicit simulation. In particular, a mean-field epidemiological model (or the Levins model) evolving on a random network of patches could be written (in an equivalent way to Eq. 4):

$$\frac{dp}{dt} = c\bar{k}p(1-p) - ep. \qquad (22)$$

The only difference in relation to Eq. 4 is that an individual infected (or a patch occupied)

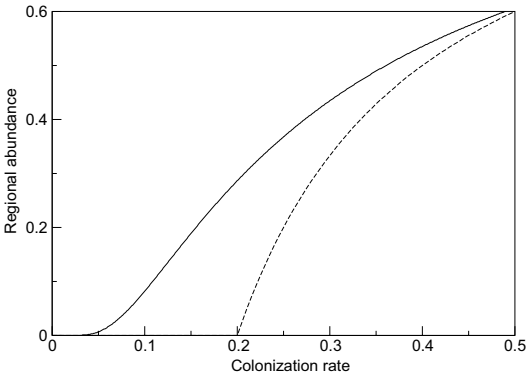

Fig. 7. Absence of eradication threshold in scale-free networks. Regional abundance is plotted as a function of colonization rate. Solid line corresponds to a metapopulation on a scale-free network according to Eq. 25. As a comparison, broken line corresponds to the Levins model depicted in Fig. 3a, for which an extinction threshold occurs. Parameters are $e = 0.2$ and $m = 0.8$. Based on Pastor-Satorras and Vespignani (2001b).

can not infect all other individuals, but only an average of other individuals with which the infected individual interacts. Thus, we recover the same results as before, that is, the existence of an extinction threshold (Pastor-Satorras & Vespigniani 2001a, 2001b): if the spreading rate falls below a critical threshold ($c_c = e/\bar{k}$) then the epidemics disappear. This is equivalent to the threshold for Eq. 4.

Now, what happens if interactions take place on the scale-free network observed for real computers? In this case, as noted before, there is not a well-defined scale, so fluctuations in connectivity are unbounded ($\sigma_k^2 = \infty$). This means that some nodes are much more connected than for a random case, and so we can no longer accept the homogeneity assumption. Following Pastor-Satorras and Vespignani (2001a, 2001b), the model for the spreading of a virus in a scale-free network reads:

$$\frac{dp_k}{dt} = ck\Theta(p)(1 - p_k) - ep_k, \qquad (23)$$

where now we describe the fraction of nodes of connectivity k infected (p_k). The extinction term is the same as before, but the positive term defines the probability that a node with k links is healthy ($1 - p_k$) and gets the infection from one of the nodes with which it interacts. The last event has a probability equal to the product of the infection rate c, the number of connections k, and the probability of a link pointing to an infected node $\Theta(p)$. The solution of the model (Eq. 23) is given by:

$$p_k^* = \frac{kc\Theta^*}{kc\Theta^* + e}. \qquad (24)$$

Since it was assumed that $\Theta(p)$ is a function of the total density of infected nodes, and this density in the steady state is a function of c/e, then Θ^* is also a function of c/e.

The previous equation indicates that the higher the node connectivity, the higher its probability of being infected. Pastor-Satorras and Vespignani (2001a, 2001b) introduced this relationship into the calculation of Θ^*, that is, they assumed that any node is more likely to be connected to an infected node highly connected. By introducing this assumption and doing some algebra, Pastor-Satorras and Vespignani (2001a, 2001b) ended up with the following expression for the stationary fraction of nodes infected:

$$p^* \sim \exp(-e/mc), \qquad (25)$$

where m is a parameter from the connectivity distribution and can be understood as the minimum number of connections at each node. As noted, the extinction threshold has vanished. The expression for p^* is continuous for all the range of values of c. Figure 7 represents the relationship between p^* and c for the random and the scale-free network. The elimination of the eradication threshold can be understood in the following way. It is well-known that in regular or random matrices, the higher the node's connectivity, the lower the eradication threshold. Since scale-free networks can be considered to have an infinite connectivity, the threshold just disappears.

Although we have developed the previous result in relation to the threshold in the effective colonization rate c/e, we would obtain the same result, that is, the absence of the eradication threshold, if we would be considering an increasing fraction of nodes vaccinated. For real viruses, this has deep implications. It is well-known that the network of sexual interactions can be reasonably well approximated by a scale-

free network over a substantial part of the scaling range (Liljeros *et al*. 2001). This suggests that AIDS also lacks an extinction threshold. This is bad news. The good news is that research on stability of complex networks provides guidelines for how to best attack these systems: although scale-free networks are very robust to failure (i.e., the treatment of a random node), they are very sensitive to attack (the treatment of the most connected node) (Albert *et al*. 2000). This means that epidemiological strategies should focus on the hubs, that is, the patients with the largest number of sexual partners (Albert 2002).

Now, let us come back to metapopulations. From the issues considered in this section, it becomes clear that a key question before predicting the existence of an extinction threshold is to determine the shape of the connectivity distribution function among patches. If this curve fits a Gaussian distribution, with a variance in the number of connections per patch similar to the average number of connections, then the predictions about the existence of an extinction threshold developed in the first part of this paper should be observed regardless of specific details not incorporated in the model. But if connectivity distribution has longer tails (that is, the variance in the number of connections per patch is much larger than the mean), then we may be in a situation closer to the second part of this paper in which the extinction threshold disappears or at least is reduced.

If a network of habitat patches is highly heterogeneous, and colonization is a local event, one can envision a situation in which a patch in the periphery may be connected through colonization processes to only another patch, while one patch in the center of the network may be connected to many other patches. Besides traditional estimates such as number of patches, distances, isolation, etc., one could estimate a measure of heterogeneity in the connectivity distribution and see how this could shift the extinction threshold or even make it disappear. It is still not clear, however, to what extent this result is relevant for metapopulations. One can easily see that a connectivity distribution following a scale-free distribution is more likely in epidemiology than in metapopulations. Several authors have looked at spatially explicit realistic metapopulation models (*see* Ovaskainen & Hanski 2003), in which real networks of patches were modeled, and still found the existence of an extinction threshold (although of course, this may have been shifted in relation to the predictions of an equivalent mean field model). Future work should identify situations (if any), in which the results by Pastor-Satorras and Vespignani (2001a, 2001b) are relevant for metapopulation dynamics.

A situation of high heterogeneity in the connectivity among patches is observed in plant species dispersed by frugivorous birds. Depending on the type of patch (e.g. its microhabitat), it will be visited by many more bird species, which may disperse the seeds to a much larger variety (e.g., type, distance) of patches. For example, in a study about the seed dispersal process in a Mediterranean scrubland, Jordano and Schupp (2000) found that microhabitats differed strongly in the proportion of seeds delivered by the main frugivorous, and also differed in the number and identity of contributing disperser species. Overall, the seed rain was found to be strongly nonrandom (Jordano & Schupp 2000). This type of scenario is likely to be more relevant from the point of view of the theory reviewed in this section.

Similarly, in network design one could modify the distribution of patches' connectivities to reduce the extinction threshold to some tolerable value. This could be achieved by increasing the number of corridors in patches already well connected. Although these ideas are still preliminary, this is an interesting area that deserves further study.

Concluding remarks

In this paper I have reviewed the two types of deterministic extinction thresholds. First, I have introduced the Allee effect, which is a demographic threshold, i.e., a critical population density below which growth rate may be negative. The Allee effect may interact with environmental stochasticity to drive a population extinct. I have illustrated this idea in the context of strategies for eradication of alien species.

Second, I have considered parametric extinction thresholds, that is, thresholds for a critical

value of a parameter such as an effective colonization rate or a minimum amount of available habitat. I have illustrated these ideas using very simple metapopulation models which can be used to generate "rules of thumb." Localized dispersal tends to shift the extinction threshold towards lower values of habitat destroyed. Also, the extinction threshold is modified by the complexity of the food web in which a species is immersed.

Since the bulk of mean-field metapopulation models assume an infinite number of patches, I have also considered the effect that the number of patches has on the persistence of a small metapopulation in a variable environment. As both the magnitude and spatial correlation of environmental fluctuations increase, the extinction threshold takes place for a larger number of patches.

All the metapopulation and epidemiological models showing extinction thresholds assume random connectivities of patches. When the network of interactions is not random but scale-free, the extinction thresholds vanish. Thus, it is important to take into account the topology of the network of connectivities.

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