



Are the speeds of species invasions regulated? The importance of null models

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Understanding the mechanisms and patterns that govern the invasion of species is essential for coping with global change of the biological world. A recent study highlights the possibility, based on data from a wide range of different taxa, that the invasion speed of species could be governed by a regulatory process. In principle, it is possible that mechanisms such as Allee effects could cause the invasion fronts to be regulated, such that the change in the rate of spread is negatively related to the current rate. This is very similar to how some populations are regulated around an equilibrium size, and finding the regulation structure if true, is of both pure and applied interest. However, here we will argue that the methods used so far are incomplete, thus even though there is a theoretical possibility that the speed of species invasions are regulated, more scrutiny is needed for its detection. Analysing changes of the ratio of current and past rate of spread against current ratios may give the impression of regulation in null models that are in fact unregulated. In addition we show that the apparent pattern is highly influenced by the spatial scale of investigation. Our results show that detecting regulatory patterns in species invasions is similarly non-trivial as is detecting density-dependence per se, but necessary, given the importance of this problem.

The impact of invasive species on natural ecosystems has been recognized for decades (Elton 1958), including the increasingly recognized possibility that invading species form a leading cause of animal and plant extinctions (Clavero and Garcia-Berthou 2005). It follows that ecologists need to understand the process of invasion, from first colonization to widespread presence (Sakai et al. 2001). The recent increase in the number of investigations of the dynamics of non-native species (Puth and Post 2005) has highlighted the need to disentangle the underlying processes that govern the particular rate of spread of an invading species (reviewed by Hastings et al. 2005). The standard prediction of spread rates are that they are constant (Fisher 1937, Skellam 1951), and recently an empirical investigation (Arim et al. 2006) suggested that the spread dynamics of invasive species might be a regulated process. Arim et al. (2006) contrast an unregulated process (exponential growth) with regulation, which they define as occurring if the change in invasion speed is negatively related to the current speed. They refer to change in per capita invasion rate in relation to the current rate as the R-function. The finding that the R-function has a negative slope appears to be consistent across biomes, taxa and temporal scales (Arim et al. 2006). The investigation was inspired by and utilized similar methods as in investigations of the regulation of population sizes (Berryman 1999, Berryman and Turchin

2001). However, the biological mechanisms leading to the regulation of invading fronts are still obscure, though speculations include competition for invadable areas (Arim et al. 2006), reproductive delays (Davis et al. 2004), Allee effects (Keitt et al. 2001, Tobin et al. 2007a), and other unknown potential mechanisms.

Here we will argue that while such mechanisms are not impossible (indeed, they may arise as emergent properties from spatial models of population growth, Ovaskainen and Cornell 2006), finding evidence for the regulation of invasion fronts requires more careful analysis than fitting an R-function to observed data on spread. Moving invasion fronts are the results of individuals reproducing and dispersing in space and the derivation of null expectations should therefore also be done in a spatial context. We construct two simple models of invasive species spread to show that a fitted negative slope of the R-function is not valid evidence that the invasion process is regulated, when regulation is defined as a negative causal relationship between current and future rate of spread. We will show that statistical fitting easily produces negative slopes, even if no regulation is inherent in the model. We also show that the strength of the apparent regulation can be an artefact of the spatial level of analysis. This highlights the importance of applying null-models in ecological research.

Two stochastic models of spatial spread

We construct two stochastic models of spatial spread to simulate an expanding population. We constructed two different models that make use of different assumptions, to increase the robustness of our results, and to highlight two different important points regarding spatial spread: the difficulty of separating apparent from real regulation, and the importance of the spatial scale used in the investigations. The first model utilizes a lattice structure to explicitly follow the dynamics of invadable sites, after which an individual-based model is built to follow individual reproduction and dispersal.

Lattice model

Consider a lattice of $n \times n$ cells, where each cell can be in three different states; empty (denoted 0), recently occupied (denoted 1) or occupied (2). There is no return from invaded to unoccupied (i.e. no local extinctions). We simulated a discrete-time invasion in this lattice, such that each time step all occupied cells send propagules to all cells within their neighbourhood. Each cell in the neighbourhood has a probability of being invaded given by p_1 or p_2 from cells in state 1 or 2 respectively, and every cell in the neighbourhood is evaluated independently (e.g. a cell with two recently occupied neighbours stays empty with probability $(1 - p_1)^2$, and becomes occupied otherwise). We varied the size of the diamond shaped neighbourhood by using the 4, 12, 24 or 40 closest cells. A cell that was 'recently occupied' (state 1) at the start of the time-step changes to state 2, and newly occupied cell is assigned state 1. Each time-step we recorded the number of cells that changed state from 0 to 1. Every simulation started with one cell in state 1, and the invasion never reached the end of the lattice.

A top-down imposed regulation of spread in this model is accomplished if a speedy invasion at one time step (which generates many recently invaded cells) leads to slowing down of new propagule formation. This is achieved by setting $p_1 < p_2$, which can represent a reproductive lag; a recently invaded area has a lower probability of invading other areas compared to an area where the invader has been present for some time. The degree of imposed regulation is denoted by $\kappa = p_2/p_1$, where 1 indicates no other regulation than potential competition for invadable sites (below) while $\kappa > 1$ indicates inherent regulation of spatial spread.

Individual-based model

The lattice model tracks the dynamics of habitat occupancy but it does not consider individual-level reproduction with subsequent dispersal of the offspring. Arguably, one can also consider it an inappropriate null model in the sense that vacant invadable sites can be in short supply if invasion of sites has been particularly successful in the recent past. Thus, although $\kappa > 1$ implies stronger regulation than $\kappa = 1$, the absence of all regulation at $\kappa = 1$ is debatable is therefore desirable to increase the robustness of our findings by considering a true null model where individuals truly do not interact with each other when sending out propagules.

To achieve this, we utilized an individual based approach under discrete time. In this model, an individual is

characterized by its position $[x, y]$ in the Euclidian space, and space is unbounded. Each time step, each individual produces one offspring with probability p . This offspring then disperses to a new location determined by the natal dispersal distance d and an angle θ . The distance, d , is drawn from a negative exponential probability distribution, $\exp(-d)$, and the angle, θ , is drawn from a uniform distribution $[0 \dots 2 \times \pi]$. The position of an offspring born of an individual at $[x, y]$ is thus $[x + d \times \cos(\theta), y + d \times \sin(\theta)]$. Time is updated after offspring production and dispersal of offspring has been evaluated for every individual. To gather data on invasion in a similar vein as done by Arim et al. (2006), we superimposed a grid structure onto the Euclidean space by dividing the area in which individuals occur into square grids (cells), each side measuring 1 units. The number of cells that are newly invaded was then recorded, for every time-step t . Notice that this grid structure does not influence the dynamics of the population which occurs in continuous space, and it exists only for the purpose of data collection. Also note that this model is a pure null model in the sense that we do not assume any population regulation. As before, we also ignore local extinction as established individuals do not die.

Data collection procedures for both models

We followed the definition of per capita rate of spread used

in Arim et al. (2006), $S(t) = \frac{N(t+1)}{\sum_{i=t-5}^t N(i)}$, where $N(t)$ is the

number of newly invaded cells at time t . Note that this is per capita rate and different from the usual use of rate as the number of newly invaded sites at a given time. We performed a $N+1$ - transformation (i.e. added 1 to each entry) for all time series that included at least one zero entry for $N(t)$. We then performed a linear regression of

$R = \log\left(\frac{S(t+1)}{S(t)}\right)$ against $\log(S(t))$, thus fitting the

function $R = \alpha + \beta \times \log(S(t))$ to observed values of R . The least-squares values of α and β were recorded for every simulation run.

Results

Null expectation in the models

Figure 1 shows examples of the regression of the change in the log rate of spread plotted against the current rate of spread (i.e. $R(t)$ against $\log(S(t))$, equivalent to Arim et al. 2006), developed from one simulation run without any regulation of spread ($\kappa = 1$), and one with regulation ($\kappa = 4$). Each simulation ran for 50 time steps. With a reproductive lag imposed there is a strong linear negative relationship between the current rate of spread and the change in that rate, but this also appears in the case without a reproductive lag.

It appears that a negative value of the slope β is a null expectation in the lattice model, and a reproductive lag only makes the slope steeper. To evaluate if this is true generally, we performed extensive simulations. For every neighbourhood we varied p_1 from 0.1 to 0.9 with 0.1 intervals. For

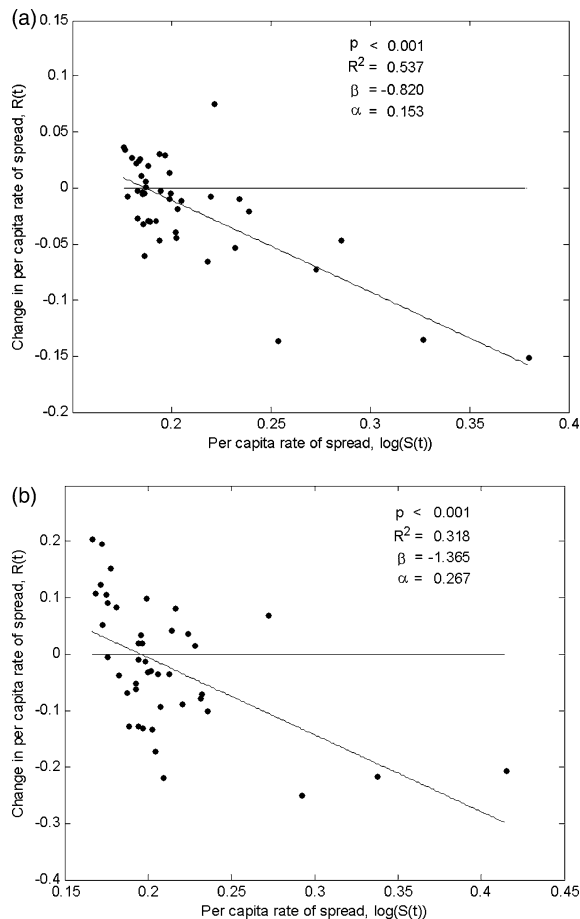


Fig. 1. Two examples of regressions of R against S in the lattice model. In (a) there is no top-down regulation of the invasion, whereas in (b) $\kappa = 4$. Even without regulation a negative relationship between current per capita rate of spread (ratio of newly invaded sites to invaded sites the previous five time-steps) and the change in that rate is expected.

each of these settings we also varied the degree of reproductive lag ($\kappa = 1, 4, 7, 10$). All these parameter settings yielded the same negative fit.

Figure 2 depicts the cumulative distribution of β with ($\kappa > 1$) and without ($\kappa = 1$) top-down regulation in the form of reproductive delay in the lattice model, for all four different neighbourhoods (each distribution based on 250 independent simulation runs of 50 time steps). In addition to larger neighbourhoods yielding less steep regression coefficients, the impact of a reproductive lag was weaker, the larger the neighbourhood. For the largest neighbourhood used, even fairly strong lag ($\kappa = 7$) was hard to distinguish from the scenario with no regulation inherent.

Figure 3 presents the result of varying the size of the grids in the individual-based model. We recorded the number of newly invaded sites using two different grid sizes, $l_1 = 1$ unit and $l_2 = 4$ units of length. The larger the size of the superimposed grid, the stronger the signal of apparent regulation. This indicates that if presence or absence is measured at larger spatial scales, null models with no regulation are more likely to yield a negative β , i.e. the wrong conclusion of regulation arises more easily.

Discussion

Intuitively, a negative relationship between the change of per capita rate of spread, $R(t)$, and the per capita rate of spread, $\log(S(t))$, observed immediately previously, appears to suggest that some regulatory process is at work. With an exception of very fine grids in the individual-based model, all of our simulations showed that this negative relationship is in fact the null expectation. Additionally, both models also showed that the spatial scale of investigation is of great importance. In the lattice model, the larger the neighbourhood, the harder it was to distinguish between the regulated and non-regulated stochastic expansion of populations, which also show the influence of competition for invadable sites. With small neighbourhoods there is higher competition for empty sites to invade, one of the proposed mechanisms creating the feedback structure. In fact the stronger the competition for empty sites, the easier it is to distinguish the invasions with a reproductive lag ($\kappa > 1$) from the ones without a lag ($\kappa = 1$). The individual-based model in turn shows that estimating spread rates from time series of newly invaded areas incorrectly suggests regulation most easily when the areas are large. This model constitutes a diffusion process, thus there are no biological mechanisms but only sampling effects creating this pattern.

The approach presented in Arim et al. (2006) is analogous to detecting evidence of density-dependence in time series of population data within a locality (Berryman 1999, Berryman et al. 2002). Detecting density dependent population regulation is no easy task (Murdoch 1994, Fowler et al. 2006, Freckleton et al. 2006), and only the newest developments allow unambiguous detection of density dependence in a large number of species (Brook and Bradshaw 2006). One would not expect detection of regulation to be any simpler in the context of spatial spread. It is important to note that our results do not mean that the regulatory process suggested by Arim et al. (2006) does not exist; instead, we suggest that the matter deserves much more sophisticated analysis, such as precise documentation of how Allee effects operate during an ongoing invasion (Johnson et al. 2006, Tobin et al. 2007b).

In the debate on the regulation structure of population densities, regulation has by many been defined as done in Arim et al. (2006). This approach assumes that a biological variable can be treated as a dynamical one and that there are negative feedbacks leading to the inverse proportionality of the change of such a variable and its current or past states (Berryman et al. 2002). In population dynamics the potential negative feedbacks from current to future population size are fairly intuitive. Because populations can only persist for appreciable time when regulated, the regulation structure of populations are often viewed as a fundamental framework for research, rather than a hypothesis to be tested (Turchin 1999). It is less clear if (and why) an invading front should be regulated, and therefore it appears useful to uncover the expected patterns when there is no regulation.

However, in this case it may be hard to agree on how to construct and choose the appropriate null model. This is most notably because the potential mechanisms leading to regulation are unknown, and therefore they might be difficult to completely leave out in a model generating the null expectations. For instance, Arim et al. (2006) suggested

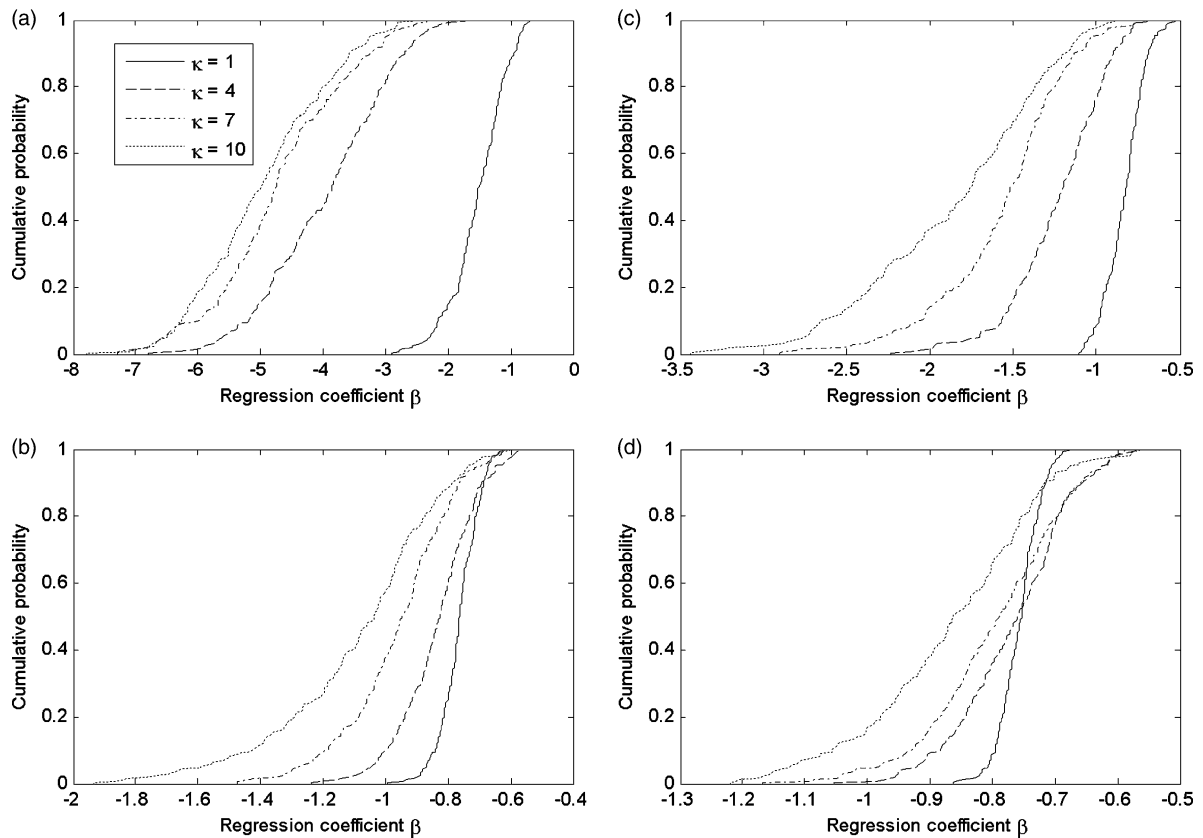


Fig. 2. Cumulative distributions of regression coefficients for 250 simulation runs in the lattice model, for the case of no regulation ($\kappa = 1$, solid line), and three different strengths of top-down regulation ($\kappa = 4$, dashed line, $\kappa = 7$, dot-dash, and $\kappa = 10$, dotted line). Each simulation ran for 50 time steps. The size of the neighbourhood was varied; (a) 4, (b) 12, (c) 24 and (d) 40 closest cells. Note the different scales on the x-axis. Probability of invasion from an occupied cell (p_2) was 0.5. Regressions of R against S were significant ($p < 0.05$) in all simulations.

that competition for empty sites to invade (on its own, or together with a reproductive lag) can be a mechanism leading to regulation. In our first model we let the competition for invadable sites vary through different choices for the sizes of the invasion neighbourhoods, and we also varied the reproductive lag. This model cannot completely remove the first mechanism because competition for invadable sites always applies in a lattice model. In the second model, however, we modelled a diffusion process which allowed us to completely exclude competition for invadable sites as well as any reproductive lags. With all so far proposed regulatory mechanisms excluded, this model (and its relatives, Hastings et al. 2005) constitutes an appropriate null model. This model, too, produces negative slopes of the R-function.

It is also important to realize that regarding regulation of population densities, the prerequisite of treating densities as a dynamical variable is fairly straightforward; births and deaths map current densities to future densities. The premise that we can build a similar mapping function from current to future per capita rates of spread is less easily legitimized. Most models of invasive species predicts linear rates of spread (Hastings et al. 2005), and if there is some stochastic noise in these rates over time they will appear bounded and high invasion rates will most likely be

followed by lower, due only to chance (Freckleton et al. 2006). The biological reality of treating these rates as a dynamical variable should be a subject for future research.

The upside of the similarity of the problems inherent in detecting local density dependence and regulated spread is that many of the lessons learnt in the context of local density-dependence can be applied in this new context. An important insight from the literature on population regulation relates to the spatial scale over which data are gathered (Levin 1992, Ray and Hastings 1996, Freckleton et al. 2006). The choice of a proper spatial scale is probably even more important in studies of invasive species since this is by definition a spatial process and very different patterns can emerge depending on which scale is used (Pysek and Hulme 2005). The appropriate level of spatial resolution will vary between species and should be based on a biological understanding of the population under investigation. In our individual-based model, decreasing the size of the data sampling units decreased the strength and significance of the negative relationship between rates of spread and change in rates of spread, thus larger spatial scales wrongly suggest stronger regulation.

The different datasets presented by Arim et al. (2006) varied in terms of the spatial scale, but most of them were at the level of the county, which can be considered a rather

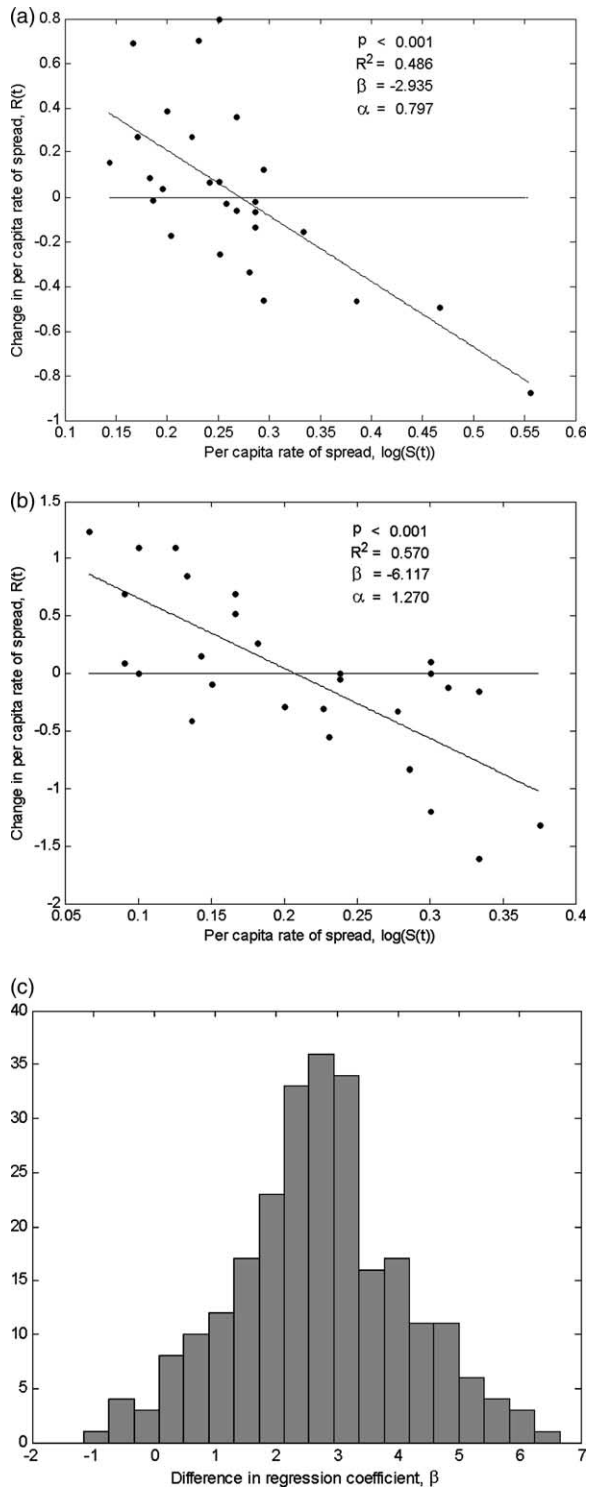


Fig. 3. Results from the individual-based model for two different spatial scales of data collection, (a) $l_1 = 1$ units, (b) $l_2 = 4$. (c), the distribution of the difference in estimated regression coefficients β (estimate using l_2 – estimate using l_1), for 250 simulation runs of 35 generations each, with $p = 0.5$. Larger grids produce stronger apparent regulation.

large spatial scale. Artefactual patterns suggestive of regulation might arise easily in such data. If a county or region of substantial size was invaded recently, the invader will most

likely spread within this area before invading the surrounding areas, yielding an apparent feedback between current and future spread that, in fact, only reflects a sampling effect. Another important aspect of the detection of regulation of invasion fronts that has not been investigated relates to the non-uniform distribution of detection errors in species invasions (Costello and Solow 2003). This could affect findings heavily, but again the problem of census-errors has a cousin in the population regulation debate (Freckleton et al. 2006) which might yield analogous insights.

Here we have shown that the analysis of the potential regulation of expanding populations is far from a trivial issue, but has several complications, much akin to the analogous debate in population regulation. Such regulation of spatially expanding populations are, however, theoretically plausible, and more thorough analysis is obviously needed, as links between individual movements and the invasion process are still fairly badly understood (Facon et al. 2006, Kokko and López-Sepulcre 2006, Sax et al. 2007). We also need a theoretical investigation of the impact of Allee effects and reproductive lags and their potential for giving rise to regulation of fronts. One approach to follow would be to develop other ways of measuring the rate of spread, for instance to also take the respective densities into account, or try experimental methods for detecting regulation.

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