A generalized definition of reactivity for ecological systems and the problem of transient species dynamics

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Abstract

1. Perturbations to an ecosystem's steady state can trigger transient responses of great ecological relevance. Asymptotic stability determines whether a generic perturbation will fade out in the long run, but falls short of characterizing the dynamics immediately after an equilibrium has been perturbed. Reactivity, traditionally defined as the maximum instantaneous growth rate of small perturbations to a stable steady state, is a simple yet powerful measure of the short-term instability of a system as a whole. In many ecological applications, however, it could be important to focus on the reactivity properties of just some specific, problem-dependent state variables, such as the abundance of a focal species engaged in interspecific competition, either predators or preys in a trophic community, or infectious individuals in disease transmission.
2. We propose a generalized definition of reactivity (g-reactivity) that allows to evaluate the differential contribution of the state space components to the transient behavior of an ecological system following a perturbation. Our definition is based on the dynamic analysis of a system output, corresponding to an ecologically-motivated linear transformation of the relevant state variables. We demonstrate that the g-reactivity properties of an equilibrium are determined by the dominant eigenvalue of an Hermitian matrix that can be easily obtained from the Jacobian associated with the equilibrium and the system output transformation.
3. As a testbed for our methodological framework, we analyze the g-reactivity properties of simple spatially-implicit metapopulation models of some prototypical ecological interactions, namely competition, predation and transmission of an infectious disease. We identify conditions for the temporary coexistence of an invader with a (possibly competitively superior) resident species, for transitory invasion of either prey or predator in otherwise predator- or prey-dominated ecosystems, and for transient epidemic outbreaks.
4. Through suitable examples, we show that characterizing the transient dynamics associated with an ecosystem's steady state can be, in some cases, as important as determining its asymptotic behavior, from both theoretical and management perspective. Because g-reactivity analysis can be performed for systems of any complexity in a relatively straightforward way, we conclude that it may represent a useful addition to the toolbox of quantitative ecologists.

1 Introduction

The study of ecosystem stability has been, and still is, one of the landmarks of theoretical ecology (Odum, 1953; MacArthur, 1955; Holling, 1973; May, 1973; Pimm, 1984; Tilman, 1996; McCann, 2000; Tilman et al., 2006). Several methods have been proposed to quantify the resilience of an ecological steady state to perturbations, most of which traditionally refer to the long-term dynamics of the system (see e.g. Grimm and Wissel, 1997; Arnoldi et al., 2016). While determining the asymptotic behavior of an equilibrium is of obvious importance from both theoretical and management perspective, characterizing its short-term transient dynamics immediately after a perturbation can be, in some

cases, at least as important. As an example, some invasive species may show a so-called boom-and-bust demographic pattern (Williamson, 1996; Simberloff and Gibbons, 2004), in which an abrupt increase in population abundance after introduction is followed by a sudden collapse. Similarly, in disease ecology, exposure to a pathogen can produce a transient epidemic wave in a naïve population (i.e. a population lacking pre-existing immunity to the disease); in this case, peak disease prevalence may be orders of magnitude larger than the one eventually observed after the pathogen has possibly become endemic (Anderson and May, 1992; Keeling and Rohani, 2007). These examples clearly illustrate a key ecological point: observation and theory must focus on process-relevant timescales, not necessarily (or not only) on asymptotic dynamics (Hastings, 2010).

A simple measure of a system's short-term instability to small perturbations was proposed by Neubert and Caswell (1997). Specifically, they introduced the notion of *reactivity*, defined as the maximum instantaneous rate at which perturbations to a stable steady state can be amplified. Revived by the seminal paper by Neubert and Caswell, the analysis of transient behavior in biological systems has been recognized as a key concept to long-term ecological understanding (Hastings, 2004). Reactivity has been studied in several ecological applications, such as food-web dynamics (Chen and Cohen, 2001), pattern-formation processes (Neubert et al., 2002), metapopulation dynamics (Marvier et al., 2004; Aiken and Navarrete, 2011), predator-prey interactions (Neubert et al., 2004), stage-structured populations (Caswell and Neubert, 2005; Stott et al., 2011), pathogen transmission (Hosack et al., 2008; Chitnis et al., 2013; Woodall et al., 2014) and community dynamics (Tang and Allesina, 2014; Barabás and Allesina, 2015; Suweis et al., 2015; Cortez, 2016).

In this work we present an extension of the basic definition of reactivity that seems to be especially suited for ecological applications. In fact, one possible setback of the original theory by Neubert and Caswell (1997) is that reactivity was defined as an isotropic quantity, i.e. all state variables are given equal weight in the evaluation of the system's short-term response to external perturbations. A measure of reactivity that allows unequal weighting of state variables was introduced by Verdy and Caswell (2008). However, their method does not include the situation in which the system is considered reactive if only some state variables display a positive growth rate in the aftermath of a perturbation to a stable steady state. In many ecological contexts, though, it could be important to focus on the short-term instability properties of just some specific, problem-dependent components of the state space – or of some suitable combination of state variables. To that end, a fully anisotropic definition of reactivity is needed. A few examples may help understand the ecological relevance of this issue, which we deem noteworthy. For instance, in a competition model describing resident-invader dynamics and characterized by a stable resident-only equilibrium, one might want to assess the short-term response of the system following the introduction of the invader in the ecosystem in terms of the abundance of the invader population; in a predator-prey system, one might be interested in the short-term fluctuations of just one of the interacting species (either the prey or the predator) while

neglecting (i.e. assigning a zero-weight to) the other; or, finally, in eco-epidemiological applications, one would perhaps limit reactivity analysis only to the variables pertaining to infection, thus assessing the likelihood of transient epidemic waves. None of these important ecological problems could be properly analyzed with the original definition of reactivity.

The paper is organized as follows. In the next section we briefly outline the isotropic reactivity theory elaborated by Neubert and Caswell (1997) and propose a simple test to detect short-term instabilities in a novel, fully anisotropic framework based on the dynamic analysis of a suitable system output. This corresponds to a linear transformation of the state variables – or, possibly, of a subset of the state variables – that is designed to be ecologically relevant. Then, as a testbed for our framework, we study the problem of transient species dynamics (*sensu* Hastings, 2004) in metacommunities. A discussion focused on the ecological implications of our generalized definition of short-term instability closes the paper.

2 Materials and methods

2.1 Background: reactivity in ecological systems

The asymptotic stability of a system's attractor defines the long-term response of the system to small perturbations, but does not provide information about transient behavior. To circumvent this limitation, in the case of equilibria, Neubert and Caswell (1997) proposed a way to characterize the short-term dynamics associated with small perturbations to the system. Given an asymptotically stable steady state of a linearized (or linear) system $d\mathbf{x}/dt = \mathbf{A}\mathbf{x}$ (with $\mathbf{A}$ being a square matrix of dimension n, and with $\mathbf{x}$ being the difference between the system state and the equilibrium) and generic perturbations $\mathbf{x_0} = \mathbf{x}(0)$, Neubert and Caswell defined

$$\text{reactivity} \equiv \max_{\mathbf{x_0} \neq \mathbf{0}} \left(\frac{1}{||\mathbf{x}||} \frac{d||\mathbf{x}||}{dt} \right) \Bigg|_{t=0} , \tag{1}$$

where $||\cdot||$ indicates the Euclidean norm of vectors. In other words, reactivity was originally defined as the maximum initial amplification rate of small perturbations to a stable equilibrium, evaluated over all possible local perturbations (or, for a linear system, over all possible perturbations with a given magnitude, say $||\mathbf{x_0}|| = 1$ as customary). Two cases are thus possible: either (i) all perturbations decay exponentially over time, or (ii) at least some perturbations can initially grow, before eventually decaying because of the asymptotic stability of the equilibrium. In the second case, reactivity as defined in (1) is positive, and the equilibrium is said to be reactive. Neubert and Caswell showed that reactivity is given by the dominant eigenvalue $\lambda_{\max}(H(\mathbf{A}))$ of the Hermitian part $H(\mathbf{A})$ of matrix $\mathbf{A}$, namely $H(\mathbf{A}) = (\mathbf{A} + \mathbf{A}^T)/2$, where T indicates matrix transposition. With this method, it is possible (and relatively straightforward) to evaluate a system's short-term response to small perturbations as

a function of the model parameters. However, Neubert and Caswell's (1997) measure relies on the Euclidean norm of the system state, so that all variables are given equal weight in the assessment of the reactivity properties of an equilibrium.

2.2 A generalized definition of reactivity

We build on the theory elaborated by Neubert and Caswell (1997) and propose an extended definition of reactivity, henceforth referred to as *generalized reactivity* (or, for brevity, g-reactivity), that is particularly suited for the analysis of ecological systems. To proceed with the evaluation of g-reactivity, we preliminarily introduce a suitable (i.e. ecologically motivated) linear output transformation $\mathbf{y} = \mathbf{C}\mathbf{x}$ for the linearized (or linear) system $d\mathbf{x}/dt = \mathbf{A}\mathbf{x}$, where $\mathbf{C}$ is a real, full-rank $m \times n$ ($m \leq n$) matrix defining a set of independent linear combinations of the system's state variables.

We define a stable equilibrium point as g-reactive if there exist some (small, yet non-zero) perturbations that are initially amplified in the system output (rather than in the state space), i.e. if

$$\left.\frac{d||\mathbf{y}||}{dt}\right|_{t=0} > 0 \tag{2}$$

for some $\mathbf{x_0} \neq \mathbf{0}$. Fig. 1 illustrates the differences existing among different definitions of reactivity. In the example (details in Appendix S1), $\mathbf{A}$ is a 2×2 upper triangular stable matrix. It may correspond, for instance, to the linearization of a predator-prey system (with a generalist predator) around the extinction equilibrium of the prey, with x_1 representing the difference between the predator's abundance and its carrying capacity, and x_2 representing the prey's abundance (panel A). Panel B shows different output transformations evaluated for a sample trajectory of the model. Specifically, the output can be represented by the two species' abundances (in this case g-reactivity corresponds to Neubert and Caswell's reactivity), possibly weighted (Verdy and Caswell's reactivity), by the abundance of only one of the two species (either the predator or the prey), or by a weighted sum of the two species' abundances (e.g. representing the total biomass of the system). The last three cases can only be addressed in our extended framework.

Figure 1 around here

2.3 A simple test to detect g-reactivity

Noting that $d\mathbf{y}/dt = \mathbf{C}\, d\mathbf{x}/dt = \mathbf{CAx}$, the initial amplification rate of perturbations to a stable steady state can be written as

$$\left.\frac{d||\mathbf{y}||}{dt}\right|_{t=0} = \left.\frac{d\sqrt{\mathbf{y}^T\mathbf{y}}}{dt}\right|_{t=0} = \left.\frac{\mathbf{y}^T\frac{d\mathbf{y}}{dt} + \left(\frac{d\mathbf{y}}{dt}\right)^T\mathbf{y}}{2\sqrt{\mathbf{y}^T\mathbf{y}}}\right|_{t=0} = \frac{\mathbf{x_0}^T\mathbf{C}^T\mathbf{CAx_0} + \mathbf{x_0}^T\mathbf{A}^T\mathbf{C}^T\mathbf{Cx_0}}{2\sqrt{\mathbf{x_0}^T\mathbf{C}^T\mathbf{Cx_0}}} = \\ = \frac{\mathbf{x_0}^T\left(\mathbf{C}^T\mathbf{CA} + \mathbf{A}^T\mathbf{C}^T\mathbf{C}\right)\mathbf{x_0}}{2\sqrt{\mathbf{x_0}^T\mathbf{C}^T\mathbf{Cx_0}}} = \frac{\mathbf{x_0}^T H\left(\mathbf{C}^T\mathbf{CA}\right)\mathbf{x_0}}{\sqrt{\mathbf{x_0}^T\mathbf{C}^T\mathbf{Cx_0}}}\,, \tag{3}$$

where $H(\mathbf{C}^T\mathbf{CA}) = (\mathbf{C}^T\mathbf{CA} + \mathbf{A}^T\mathbf{C}^T\mathbf{C})/2$ is the Hermitian part of $\mathbf{C}^T\mathbf{CA}$. The dimension (defined as the number of linearly independent rows) of the kernel of $\mathbf{C}$ (i.e. the subspace of $\mathbf{x}$ values such that $\mathbf{Cx} = \mathbf{0}$) is smaller than n, because $m \geq 1$ (actually, $\dim(\ker(\mathbf{C})) = n - m$). We thus have that $\mathbf{x_0}^T\mathbf{C}^T\mathbf{Cx_0}$ is positive for $\mathbf{x_0}$ not belonging to $\ker(\mathbf{C})$. Therefore, condition (2) is verified if

$$\mathbf{x_0}^T H(\mathbf{C}^T\mathbf{CA})\mathbf{x_0} > 0 \tag{4}$$

for some $\mathbf{x_0} \neq \mathbf{0}$. Perturbations for which condition (4) is satisfied define the g-reactivity basin (a convex polytope in $\mathbb{R}^n$) of the equilibrium (see Hosack et al., 2008, for the corresponding definition in the isotropic case). Note that they cannot lie entirely in $\ker(\mathbf{C})$, otherwise $\mathbf{x_0}^T H(\mathbf{C}^T\mathbf{CA})\mathbf{x_0} = 0$. Only trajectories originating in a neighborhood of a stable (yet g-reactive) steady state within its g-reactivity basin will be initially amplified in the system output. Inequality (4) is verified if the quadratic form $\mathbf{x_0}^T H\left(\mathbf{C}^T\mathbf{CA}\right)\mathbf{x_0}$ is not negative semidefinite; we can thus conclude (Horn and Johnson, 1990) that condition (2) is equivalent to

$$\lambda_{\max}\left(H(\mathbf{C}^T\mathbf{CA})\right) > 0\,. \tag{5}$$

In other words, a stable equilibrium point is g-reactive if the dominant eigenvalue of the Hermitian part of matrix $\mathbf{C}^T\mathbf{CA}$ is positive (note that all the eigenvalues of $H(\mathbf{C}^T\mathbf{CA})$ are real because the matrix is real and symmetric), while $\lambda_{\max}(H(\mathbf{C}^T\mathbf{CA})) = 0$ marks the transition between a non-g-reactive equilibrium and a g-reactive one. Condition (5) thus represents a simple – yet generally applicable – test to discriminate between g-reactive and non-g-reactive equilibria. Obviously, it reduces to the original isotropic condition for reactivity proposed by Neubert and Caswell if $\mathbf{C}$ is the identity matrix ($\mathbf{I}$). In this respect, g-reactivity indeed represents an extension and a generalization of the original concept of reactivity. On the other hand, the above test for g-reactivity depends on matrix $\mathbf{C}$: as such, classifying an equilibrium point as g-reactive is contingent on an appropriate choice for the output transformation. We also remark that, in ecological applications, it may be important to check whether a g-reactive equilibrium is also endowed with a biologically meaningful g-reactivity basin.

Sometimes, it may be useful to assess g-reactivity in response not to any generic perturbation $\mathbf{x_0}$,

but rather to some particular class of perturbations. For instance, in an epidemiological system, we may be interested in analyzing the system's response (e.g. in terms of secondary infections) to the introduction of a few infected individuals in a disease-free population. Of great relevance is thus the case in which the perturbation involves only those state variables that are represented in the output transformation. Mathematically, this is equivalent to considering perturbations that belong to the orthogonal complement of the kernel of $\mathbf{C}$ (i.e. $\mathbf{x_0} \in \ker(\mathbf{C})^{\perp}$), defined as the set of vectors that are orthogonal to every vector in $\ker(\mathbf{C})$. It can be shown that so-structured perturbations can indeed be amplified in the system output if

$$\lambda_{\max}\left(H(\mathbf{CAC}^{+})\right) > 0\,, \tag{6}$$

where $\mathbf{C}^{+} = \mathbf{C}^{T}(\mathbf{CC}^{T})^{-1}$ is the right pseudo-inverse of matrix $\mathbf{C}$ (representing a generalization of a matrix inverse; in fact, matrix $\mathbf{C}$ may be not square, yet $\mathbf{CC}^{+} = \mathbf{I}$; note also that $\mathbf{CC}^{T}$ is invertible because $\mathbf{C}$ is full rank) and $H(\mathbf{CAC}^{+}) = (\mathbf{CAC}^{+} + (\mathbf{C}^{+})^{T}\mathbf{A}^{T}\mathbf{C}^{T})/2$. Details are given in Appendix S2. There, we also describe whether (and how, in case) it is possible to evaluate the maximum initial amplification rate and the overall maximum amplification of perturbations to a stable, g-reactive steady state, depending on the structure of the output matrix and/or the perturbations being considered.

3 Results

To show how our newly developed theory and methods can be applied in the actual ecological practice, we analyze the g-reactivity properties of some relatively simple models of typical ecological interactions, namely competition, predation and transmission of an infectious disease. Specifically, ecological dynamics are studied in a spatially-implicit metapopulation framework (Levins, 1969) that has widely been used in both theoretical and practical applications (Hanski, 1998, 1999). We focus on mean-field metacommunity models as prototypical cases of intermediate-complexity dynamical systems applied to ecological problems because they are simple enough as to allow a concise description, yet complex enough as to yield nontrivial results.

3.1 Interspecific competition

The first example concerns competition between two species (say A and B) inhabiting a fragmented landscape. Several studies (Cohen, 1970; Levins and Culver, 1971; Horn and Mac Arthur, 1972) have been devoted to this problem in the early years of multi-species metapopulation theory. Here we use the model proposed by Slatkin (1974) to describe interspecific competition in a patchy environment (later, the model has been also discussed by Hanski, 1983). If competition does not preclude coexistence of the two species, the landscape will comprise patches occupied by just one of the two species, patches where both species are present and empty patches.

Let p_A and p_B be the fractions of patches occupied only by species A or B, and let p_{AB} be the fraction of patches where A and B coexist (clearly, the fraction of empty patches is given by $1 - p_A - p_B - p_{AB}$). Following Slatkin (1974), patch occupancy dynamics can be described by the following set of ordinary differential equations (see Table 1 for a description of the state variables and the parameters of the model):

$$\begin{aligned}
\dot{p}_A &= c_A(p_A + p_{AB})(1 - p_A - p_B - p_{AB}) - e_A p_A - \epsilon_B c_B (p_B + p_{AB}) p_A + \kappa_B e_B p_{AB} \\
\dot{p}_B &= c_B(p_B + p_{AB})(1 - p_A - p_B - p_{AB}) - e_B p_B - \epsilon_A c_A (p_A + p_{AB}) p_B + \kappa_A e_A p_{AB} \\
\dot{p}_{AB} &= \epsilon_A c_A (p_A + p_{AB}) p_B + \epsilon_B c_B (p_B + p_{AB}) p_A - (\kappa_A e_A + \kappa_B e_B) p_{AB} \,.
\end{aligned} \tag{7}$$

Individuals of a given species, say A, dispersing from patches where they either live in isolation or coexist with species B, colonize empty patches or patches previously occupied only by B at rates c_A or $\epsilon_A c_A$, respectively. In the latter case, the factor $\epsilon_A \leq 1$ describes colonization competition, i.e. reduced colonization success because of the pre-establishment of a local competitor population in the patch (Levins and Culver, 1971; Slatkin, 1974). Colonization events are balanced by local extinctions: in patches occupied by species A alone, local extinction occurs at rate e_A; in patches where the two species coexist, extinction of species A occurs at rate $\kappa_A e_A$, with $\kappa_A \geq 1$ describing extinction competition, i.e. increased extinction risk in the presence of a competitor (see again Slatkin, 1974; Hanski, 1983). The same mechanisms apply, *mutatis mutandis*, to species B.

Table 1 around here

The metapopulation competition model has four possible steady-state solutions, namely

$$\mathbf{x_{ex}} = [0,\, 0,\, 0]^T \,, \quad \mathbf{x_A} = \left[\frac{c_A - e_A}{c_A},\, 0,\, 0\right]^T \,, \quad \mathbf{x_B} = \left[0,\, \frac{c_B - e_B}{c_B},\, 0\right]^T \,, \quad \mathbf{x_{AB}} = [\bar{p}_A,\, \bar{p}_B,\, \bar{p}_{AB}]^T \tag{8}$$

with $\bar{p}_A, \bar{p}_B, \bar{p}_{AB} > 0$. The four equilibria correspond, respectively, to extinction of both species, extinction of species B or A, and coexistence (a case in which computing the system state components at the equilibrium is analytically impractical but numerically easy). The asymptotic stability of these steady-state solutions can be assessed through linearization of model (7) around each equilibrium point. Linear stability analysis can be performed either analytically (for $\mathbf{x_{ex}}$, $\mathbf{x_A}$ and $\mathbf{x_B}$) or numerically (for $\mathbf{x_{AB}}$). As an example, details on the assessment of the stability of $\mathbf{x_{ex}}$ are reported in Appendix S3. The stability ranges in parameter space of the four equilibria of model (7) are shown in Fig. 2a. Note that the stability diagram is symmetric about $c_A = c_B$ because species A and B are assumed to be identical in the example shown in the figure, possibly except for their colonization rates. Asymptotic coexistence is possible (i.e. $\mathbf{x_{AB}}$ is stable) if the two species' colonization rates are not very different from each other (and obviously larger than the respective extinction rates), as observed by Slatkin (1974).

Figure 2 around here

To study the g-reactivity properties of the steady-state solutions of system (7), a suitable output matrix needs be specified. For instance, if we wanted to analyze the transient amplification of patch occupancy for species B, we could define

$$\mathbf{C_B} = \begin{bmatrix} 0 & u & 0 \\ 0 & 0 & v \end{bmatrix}, \tag{9}$$

with u and v being two positive parameters.

To detect g-reactive equilibria we apply condition (5) with $\mathbf{C} = \mathbf{C_B}$ and $\mathbf{A} = \mathbf{J_X}$ ($\mathbf{J_X}$ being the Jacobian matrix of the system evaluated at the four equilibria, $X \in \{ex, A, B, AB\}$). As an example, a detailed g-reactivity analysis for the extinction equilibrium $\mathbf{x_{ex}}$ is reported in Appendix S3. Fig. 2a shows that $\mathbf{x_{ex}}$ is g-reactive if the colonization rate of species B is sufficiently high; conversely, the g-reactivity of $\mathbf{x_{ex}}$ does not depend on the colonization rate of species A. In case of a g-reactive extinction equilibrium, the share of landscape patches occupied by species B can temporarily grow (before eventually vanishing, as implied by the stability of $\mathbf{x_{ex}}$) following a suitable perturbation. Specifically, such a perturbation must lie in the g-reactivity basin of the equilibrium (Fig. 2b), as determined by eqn. (4).

The g-reactivity of the single-species equilibrium $\mathbf{x_A}$ can be assessed through algebraic manipulations similar to those described in Appendix S3 for the extinction equilibrium. Interestingly, there are cases (i.e. the parameter combinations for which $\mathbf{x_A}$ is stable and g-reactive) in which species B can temporarily colonize the system before being outcompeted by species A, namely either for c_A and c_B relatively close to the two species' local extinction rates, or for high values of c_A. Under these conditions, an invader species (B) could temporarily coexist with the resident species (A). This result is also demonstrated in Fig. 2c, where the maximum initial amplification rate (r) and the maximum overall amplification ($\rho^\star$) of perturbations to the steady states of model (7), evaluated along a transect of Fig. 2a, is reported (see Appendix S2 for details on the evaluation of r and $\rho^\star$).

According to condition (5), the other steady states of the model (the single-species equilibrium $\mathbf{x_B}$ and the coexistence equilibrium $\mathbf{x_{AB}}$) are always g-reactive for generic perturbations (i.e. involving also patches occupied by species A alone). We could have come to the same conclusion by noting that $\ker(\mathbf{C_B}) \neq \ker(\mathbf{C_B J_X})$ ($X \in \{B, AB\}$, see again Appendix S2). However, because the focus of the output transformation is on species B, it might be interesting to assess whether perturbations not involving patches occupied by species A alone ($\mathbf{x_0} \in \ker(\mathbf{C})^\perp$) can temporarily be amplified as well. To do so, we evaluate the Hermitian matrices $H(\mathbf{C_B J_X C_B}^+)$ and $H(\mathbf{C_B J_{AB} C_B}^+)$ (note that the former can be worked out analytically, while evaluating the latter requires carrying out numerical simulations of model (7)) and apply condition (6). Fig. 2a shows that both $\mathbf{x_B}$ and $\mathbf{x_{AB}}$ are g-reactive

for perturbations $\mathbf{x_0} \in \ker(\mathbf{C})^{\perp}$ if the colonization rate of species B is either high or low.

Model (7) can also be used to analyze asymmetric interspecific competition (Tilman, 1994), i.e. cases in which one of the two species is competitively inferior to the other (e.g. in terms of higher extinction rate, smaller colonization competition factor and/or larger extinction competition factor). Fig. 2d reports the results of stability and g-reactivity analysis for the different equilibria of the model in a case in which species B is a lesser competitor than species A. Asymptotic species coexistence is still possible, provided that the inferior competitive abilities of species B be compensated for by a higher baseline colonization rate (i.e. as in the case of so-called fugitive species; see e.g. Horn and Mac Arthur, 1972; Hanski and Zhang, 1993). The parameter combinations for which species B can temporarily colonize an otherwise A-dominated ecosystem are remarkably reduced, yet conditions allowing a temporary increase of the fraction of patches occupied by the competitively inferior species still exist.

3.2 Predator-prey dynamics

The second of our examples concerns predator-prey dynamics in a fragmented landscape. A simple metapopulation model for a prey and its specialist predator was proposed by Bascompte and Solé (1998). Their approach was later extended by Swihart et al. (2001) to account for generalist predators, i.e. predators that can survive in the absence of their preferred prey. Here we elaborate on the latter approach, so that the landscape can be partitioned into four categories of patches: empty, prey-only (predator is locally extinct), predator-only (prey is locally extinct) and predator-prey patches (where local populations of prey and predator coexist).

Let p_Y and p_D be the fractions of patches occupied only by the prey or the predator, respectively, and let p_{YD} be the fraction of patches where predator and prey coexist (the fraction of empty patches is given by $1 - p_Y - p_D - p_{YD}$). Following Swihart et al. (2001), patch occupancy dynamics can be described by the following set of ordinary differential equations (see again Table 1):

$$\begin{aligned}
\dot{p}_Y &= c_Y(p_Y + p_{YD})(1 - p_Y - p_D - p_{YD}) - e_Y p_Y + \epsilon_D e_D p_{YD} - c_D(p_D + p_{YD})p_Y \\
\dot{p}_D &= c_D(p_D + p_{YD})(1 - p_Y - p_D - p_{YD}) - e_D p_D + \kappa_Y e_Y p_{YD} - c_Y(p_Y + p_{YD})p_D \\
\dot{p}_{YD} &= c_Y(p_Y + p_{YD})p_D + c_D(p_D + p_{YD})p_Y - (\kappa_Y e_Y + \epsilon_D e_D)p_{YD}\,.
\end{aligned} \tag{10}$$

Prey and predator organisms colonize patches at species-specific rates c_Y and c_D. Note that possible mechanisms of preferential colonization (i.e. prey or predators showing a preference towards empty or prey-only patches, respectively) are not accounted for. Colonization events are balanced by local extinctions, occurring at rates e_Y in prey-only patches and e_D in predator-only patches. Prey or predator extinction in patches where the two species coexist occur at rates $\kappa_Y e_Y$ and $\epsilon_D e_D$, respectively. The coefficient $\kappa_Y \geq 1$ describes higher prey extinction risk in presence of the predator, while

$\epsilon_D \leq 1$ describes lower predator extinction risk in presence of the prey.

Model (10) has four possible steady-state solutions, namely

$$\mathbf{x_{ex}} = [0,\, 0,\, 0]^T\,, \quad \mathbf{x_Y} = \left[\frac{c_Y - e_Y}{c_Y},\, 0,\, 0\right]^T\,, \quad \mathbf{x_D} = \left[0,\, \frac{c_D - e_D}{c_D},\, 0\right]^T\,, \quad \mathbf{x_{YD}} = [\bar{p}_Y,\, \bar{p}_D,\, \bar{p}_{YD}]^T \tag{11}$$

with $\bar{p}_Y, \bar{p}_D, \bar{p}_{YD} > 0$. The four equilibria correspond to extinction of both predator and prey, predator extinction, prey extinction and predator-prey coexistence, respectively. The stability of these steady-state solutions can be evaluated through the analysis of the Jacobian matrix (and its associated eigenvalues) of model (10) evaluated at each equilibrium point (Fig. 3). The extinction equilibrium $\mathbf{x_{ex}}$ is stable if both the predator's and the prey's colonization coefficients are smaller than the respective extinction rates. Given the asymmetric nature of the predator-prey interaction, $c_Y > e_Y$ is a necessary (yet not sufficient) condition for the long-term persistence of the prey metapopulation, while $c_D > e_D$ is a sufficient (yet not necessary) condition for the persistence of the predator. In fact, the prey-only equilibrium $\mathbf{x_Y}$ is stable if $c_Y > e_Y$ and the colonization coefficient of the predator is low (at most, equal to its extinction rate e_D). Conversely, the predator-only equilibrium $\mathbf{x_D}$ is stable if $c_D > e_D$ and the colonization coefficient of the prey is not very high (yet including a sizable region characterized by $c_Y > e_Y$). Finally, predator-prey coexistence (stable $\mathbf{x_{YD}}$) requires $c_Y > e_Y$, but can be observed also if $c_D < e_D$.

Figure 3 around here

To study the g-reactivity properties of the steady-state solutions of system (10) we use two different output matrices, namely

$$\mathbf{C_Y} = \begin{bmatrix} u & 0 & 0 \\ 0 & 0 & v \end{bmatrix} \quad \text{or} \quad \mathbf{C_D} = \begin{bmatrix} 0 & u & 0 \\ 0 & 0 & v \end{bmatrix}\,, \tag{12}$$

which focus on the transient patch occupancy of either prey ($\mathbf{C_Y}$) or predator ($\mathbf{C_D}$). Clearly, with two output transformations, the procedure to evaluate g-reactivity has to be replicated twice. Results are shown in Fig. 3. Concerning output matrix $\mathbf{C_Y}$ (panel a), the most interesting result is that patch occupancy by the prey can temporarily increase in a predator-only situation (stable $\mathbf{x_D}$) for high values of the predator's colonization rate; also, the prey-only and coexistence equilibria are always g-reactive. Considering output matrix $\mathbf{C_D}$ (panel B), instead, it turns out that predators can temporarily increase in a global extinction (stable $\mathbf{x_{ex}}$) or prey-only situation (stable $\mathbf{x_Y}$) if the prey's colonization rate is either low or high; the predator-only (stable $\mathbf{x_D}$) and coexistence (stable $\mathbf{x_{YD}}$) equilibria are always g-reactive.

3.3 Pathogen transmission

The third example illustrates pathogen transmission in a metapopulation. Several mathematical models for host-pathogen interactions in spatially implicit metapopulations have been proposed: Hess (1996) formalized a first, simple model for susceptible-infectious (SI) dynamics; Gog et al. (2002) analyzed the effects of spillover from an alternative host on SI interactions; McCallum and Dobson (2002) studied susceptible-infectious-recovered (SIR) dynamics with a reservoir host; and Harding et al. (2012) analyzed Allee effect in the context of a SI model. All these studies have contributed to a lively debate on the long-term implications of conservation corridors for the spatial spread of infectious diseases and the ensuing conservation issues, as originally discussed by Hess (1994). Here we propose a model to describe susceptible-exposed-infectious-recovered-susceptible ($SEIRS$) dynamics in a metapopulation, namely to account for infections in which there is a non-negligible incubation period during which newly infected hosts are not yet infective (Anderson and May, 1992). For the sake of simplicity, and following the approach chosen in all the abovementioned host-pathogen metapopulation models, landscape patches are assumed not to host organisms with different infection status. Therefore, five categories of patches must be considered, corresponding to the four epidemiological compartments ($S/E/I/R$) and to empty patches.

Let p_S, p_E, p_I and p_R be the fractions of susceptible, exposed, infectious and recovered patches (so that the share of empty patches is $1 - p_S - p_E - p_I - p_R$). Patch occupancy dynamics can be described by the following set of ordinary differential equations (see again Table 1):

$$\begin{aligned}
\dot{p}_S &= c_S p_S(1 - p_S - p_E - p_I - p_R) - e_S p_S - c_I p_I p_S + (1-\alpha)\eta p_E + \xi p_R \\
\dot{p}_E &= c_E p_E(1 - p_S - p_E - p_I - p_R) - e_E p_E + c_I p_I p_S - \eta p_E \\
\dot{p}_I &= c_I p_I(1 - p_S - p_E - p_I - p_R) - e_I p_I + \alpha\eta p_E - \gamma p_I \\
\dot{p}_R &= c_R p_R(1 - p_S - p_E - p_I - p_R) - e_R p_R + \gamma p_I - \xi p_R \,.
\end{aligned} \tag{13}$$

Local populations are endowed with different colonization (c_Σ) and extinction (e_Σ) rates according to their epidemiological status ($\Sigma \in \{S, E, I, R\}$). For the sake of parameter parsimony, we can assume that susceptible, exposed and recovered populations share the same baseline colonization ($c_S = c_E = c_R = c$) and extinction ($e_S = e_E = e_R = e$) rates, while infectious populations are characterized by lower colonization ($c_I = \epsilon_I c$, $\epsilon_I \leq 1$) and higher extinction ($e_I = \kappa_I e$, $\kappa_I \geq 1$) rates. Upon being colonized, empty patches assume the epidemiological status of the occupying organisms. Susceptible patches become exposed when colonized by organisms dispersing from infected patches, while the event that exposed organisms migrating to a susceptible patch would eventually become infectious (and thus make the whole patch exposed) is here neglected for simplicity. Patches that have been exposed to the pathogen progress to a different epidemiological status at a rate η, corresponding to the inverse of the average incubation time of the disease; specifically, if a local epidemic outbreak

unfolds (with probability α) they become infectious patches, otherwise they get back to a susceptible status (with probability $1-\alpha$). Infectious patches lose their status at rate γ, representing the inverse of the average duration of a local epidemic, and become recovered patches. Recovered patches become susceptible again at rate ξ, corresponding to the inverse of the average duration of the specific immunity to the disease.

Model (13) has three possible steady-state solutions, namely

$$\mathbf{x_{ex}} = [0,\, 0,\, 0,\, 0]^T\,, \quad \mathbf{x_{df}} = \left[\frac{c-e}{c},\, 0,\, 0,\, 0\right]^T\,, \quad \mathbf{x_{en}} = [\bar{p}_S,\, \bar{p}_E,\, \bar{p}_I,\, \bar{p}_R]^T \tag{14}$$

with $\bar{p}_S,\, \bar{p}_E,\, \bar{p}_I,\, \bar{p}_R > 0$. The three equilibria correspond, respectively, to metapopulation extinction, pathogen extinction (the so-called disease-free equilibrium, i.e. a state in which all occupied patches are susceptible to the infection) and pathogen establishment (endemic equilibrium, in which the pathogen can persist indefinitely in the metapopulation). The stability ranges of the three equilibria of model (13) can be obtained from the analysis of the dominant eigenvalues of the Jacobian matrices $\mathbf{J_0}$, $\mathbf{J_{df}}$ and $\mathbf{J_{en}}$ (for the extinction, disease-free and endemic equilibrium, respectively; Fig. 4a). The metapopulation is doomed to extinction for low values of the baseline colonization rate ($c < e$, Levins, 1969), persists in a disease-free state for intermediate values of c, or endures in an endemic-transmission setting for high values of c.

Figure 4 around here

Increasing values of c lead to higher overall patch occupancy, as expected, yet the fraction of susceptible patches peaks for intermediate values of the colonization rate, namely for c corresponding to the transition from a disease-free to an endemic state (Fig. 4b). Clearly, this transition is particularly important from an epidemiological perspective, because it marks a separation between conditions under which the disease is absent from the metapopulation and conditions under which sustained transmission is possible. Such a transition is triggered by a stability switch between the disease-free and the endemic equilibrium. With straightforward algebraic manipulations, it is possible to prove that the disease-free equilibrium becomes unstable if

$$\mathcal{R} = \frac{\alpha\epsilon(c-e)}{\gamma + (\kappa - \epsilon)e} > 1\,, \tag{15}$$

corresponding to the condition for the dominant eigenvalue of $\mathbf{J_{df}}$ to switch from negative to positive (or, equivalently, for the determinant of $\mathbf{J_{df}}$ to switch from positive to negative; note in fact that $\mathbf{J_{df}}$ is of even order). The threshold parameter $\mathcal{R}$ can be interpreted as the basic reproduction number of the disease (Anderson and May, 1992).

To study the g-reactivity properties of the steady states of model (13) from an epidemiologically-

relevant perspective, we use the following output transformation matrix

$$\mathbf{C_{EI}} = \begin{bmatrix} 0 & u & 0 & 0 \\ 0 & 0 & v & 0 \end{bmatrix}, \tag{16}$$

where only the infection-related state variables (exposed and infectious) are accounted for. A transient disease epidemic can thus be defined as the extinction or the disease-free equilibria being g-reactive. Using condition (5), we find that the extinction equilibrium is g-reactive if

$$\mathcal{E}_{ex} = \frac{v^2}{4u^2} \frac{\alpha^2\eta^2}{(c-e-\eta)(\epsilon c - \kappa e - \gamma)} > 1\,, \tag{17}$$

while the disease-free equilibrium is g-reactive if

$$\mathcal{E}_{df} = \frac{\left[u^2\epsilon(c-e) + v^2\alpha\eta\right]^2}{4u^2v^2\eta[\gamma + (\kappa-\epsilon)e]} = \mathcal{R} + \frac{\left[u^2\epsilon(c-e) - v^2\alpha\eta\right]^2}{4u^2v^2\eta[\gamma + (\kappa-\epsilon)e]} > 1\,. \tag{18}$$

Note that these two conditions hold for both generic perturbations and perturbations involving only exposed and infected patches ($\mathbf{x_0} \in \ker(\mathbf{C_{EI}})^\perp$), because $\ker(\mathbf{C_{EI}}) = \ker(\mathbf{C_{EI}J_X})$ ($X \in \{ex, df\}$, Appendix S2). Inequality (18), in particular, implies that the parameter region for which the disease-free equilibrium is g-reactive lies close to the boundary $\mathcal{R} = 1$ separating the stability region of the disease-free equilibrium from that of the endemic equilibrium. Interestingly, Snyder (2010) found that, in general, weakly stable systems have a greater capacity to be reactive. $\mathcal{E}_{ex}$ and $\mathcal{E}_{df}$ represent thresholds for transient epidemicity (see Hosack et al., 2008): if one of the two above conditions is verified, a temporary epidemic outbreak may be possible even in the absence of endemic transmission. As for the endemic equilibrium, condition (5) holds true independently of parameter values, hence a stable $\mathbf{x_{en}}$ is always g-reactive for generic perturbations (in fact, $\ker(\mathbf{C_{EI}}) \neq \ker(\mathbf{C_{EI}J_{en}})$, see again Appendix S2). To assess whether it is also reactive to perturbations $\mathbf{x_0} \in \ker(\mathbf{C_{EI}})^\perp$, we simulate model (13) and evaluate numerically the dominant eigenvalue of $H(\mathbf{C_{EI}J_{en}C_{EI}^+})$. Fig. 4a shows that the endemic equilibrium subject to perturbations $\mathbf{x_0} \in \ker(\mathbf{C_{EI}})^\perp$ is g-reactive for parameter sets close to $\mathcal{R} = 1$. Therefore, transient epidemic waves are expected to occur especially in the disease-free/endemic transition region embedding $\mathcal{R} = 1$, where the fraction of susceptible patches is maximum, leading to either temporary pathogen invasion (in a previously disease-free metapopulation, $\mathcal{R} < 1$) or a transient increase in the fraction of infected/infectious patches (in a metapopulation in which pathogen transmission is endemic, $\mathcal{R} > 1$).

4 Discussion

In this work we have proposed a novel, generally applicable test to evaluate the reactivity of dynamical systems. Our definition of reactivity (g-reactivity) is especially suited to analyze ecological problems

in which it may be necessary to track the transient dynamics of just some of the state variables of the system, or a suitable linear transformation thereof. In this respect, g-reactivity overcomes a possible limitation of previous approaches, which basically neglected (Neubert and Caswell, 1997) or only partially addressed (Verdy and Caswell, 2008) the problem of the state variables' differential contribution to the transient behavior of a system following a perturbation to a stable steady state. Our framework includes Neubert and Caswell's and Verdy and Caswell's definitions of reactivity as particular cases, thus representing a useful tool for the study of transient dynamics in ecological systems.

Although the level of technicality involved in the derivation of our new method might seem bewildering at first sight, the use of g-reactivity is actually quite straightforward in practice. Given a linear or linearized system described by the state matrix $\mathbf{A}$, a necessary, preliminary step is the definition of a suitable output transformation matrix, $\mathbf{C}$. Then, if only the existence of generic perturbations that will initially be amplified in the system output needs be assessed, one can simply use condition (5) and determine the sign of $\lambda_{\max}(H(\mathbf{C}^T\mathbf{C}\mathbf{A}))$, possibly in conjunction with the evaluation of the g-reactivity basin (eqn. 4). The analysis of perturbations endowed with a specific structure and the evaluation of the initial/overall maximum amplification of perturbations require some additional steps, but the simple procedure just outlined should be sufficient to conduct g-reactivity analysis for a variety of applications.

To show how our methodological framework can be applied to ecological problems, we have performed g-reactivity analysis for three simple, yet paradigmatic metapopulation models describing, respectively, competition, predation and disease transmission in a fragmented landscape (Hanski, 1998, 1999). Case-specific ecological lessons can be learned from each of these applications:

- the analysis of the model for interspecific competition has shown that two species can temporarily coexist in a fragmented landscape even if the study of long-term dynamics would predict extinction of either (or both) metapopulations. This result could be particularly important if one of such species were an invader for which control measures had to be sought: in this case, linear stability theory could help design strategies for the eradication of the pest in the long-run, while g-reactivity analysis could suggest ways to limit short-term outbreaks. Our analysis also sheds new light on the transient dynamics of fugitive species, i.e. of species that can compensate for inferior competitive traits with better dispersal abilities. Specifically, we have shown that the likelihood of temporary colonization by the competitively inferior species is remarkably reduced in the case of asymmetric competition;

- the model for predator-prey dynamics has clearly shown that a distinction between g-reactive and non-g-reactive steady states cannot be made in absolute terms, because different choices for the output transformation (e.g. focusing on either the predator's or the prey's transient

dynamics in the problem at hand) can lead to different outcomes. Therefore, deciding whether or not an equilibrium point is g-reactive requires a suitable (possibly ecologically motivated and certainly problem-dependent) definition of the output transformation matrix. This message is general in scope, evidently extending beyond this predator-prey example;

- finally, the model for pathogen transmission in a fragmented landscape has shown that, while increasing colonization rates (possibly as a result of increased habitat connectivity) can favor the spread of an infectious disease in a metapopulation (as measured by the prevalence of infected/infectious patches at the equilibrium), the likelihood of transient epidemic waves is maximum for intermediate colonization rates, namely around values of the colonization rate close to the disease-free/endemic transition, for which the share of susceptible patches peaks.

Although relatively simple, the examined models were not completely amenable to analytical treatment. Conditions for g-reactivity have been evaluated numerically whenever a formal derivation was impossible or impractical. This in turn demonstrates how the methods described here can provide ecologists with quantitative tools that are ready to be used in applications of any degree of complexity. In particular, g-reactivity analysis could be usefully applied to spatially explicit ecological models, such as structured metapopulations (Hanski and Ovaskainen, 2000; Ovaskainen and Hanski, 2001; see also Casagrandi and Gatto, 1999, 2006; Mari et al., 2014; Bertuzzo et al., 2015; Grilli et al., 2015). By doing so, it would be possible to complement stability analysis for such models (which is often framed in terms of the so-called metapopulation capacity; see again Hanski and Ovaskainen, 2000) with a study of the transient dynamics that may follow spatially heterogeneous perturbations, i.e. perturbations acting only on some specific habitat patches. Also, coupling g-reactivity analysis with elements of generalized stability theory (see Farrell and Ioannau, 1996, for a review) would allow the detection of the fastest-growing perturbation geometries, with possibly profound implications for the conservation and management of populations inhabiting fragmented ecosystems.

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Authors' contributions

LM and MG designed methodology and led the writing of the manuscript. All authors analyzed the results, contributed critically to the drafts and gave final approval for publication.

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Tables

Table 1: List of symbols used in the metapopulation models. State variables/model parameters are listed in the top/bottom part of each subtable.

	Interspecific competition
p_A	fraction of patches occupied only by species A
p_B	fraction of patches occupied only by species B
p_{AB}	fraction of patches occupied by both species
c_Σ	baseline colonization rate of species Σ ($\Sigma \in \{A, B\}$)
e_Σ	baseline extinction rate of species Σ
ϵ_Σ	colonization competition factor for species Σ (≤ 1)
κ_Σ	extinction competition factor for species Σ (≥ 1)
	Predator-prey dynamics
p_Y	fraction of patches occupied only by preys
p_D	fraction of patches occupied only by predators
p_{YD}	fraction of patches occupied by both preys and predators
c_Σ	colonization rate of species Σ (prey: $\Sigma = Y$; predator: $\Sigma = D$)
e_Σ	extinction rate of species Σ
ϵ_D	extinction rate reduction factor for the predator
κ_Y	extinction rate increase factor for the prey
	Pathogen transmission
p_S	fraction of susceptible patches
p_E	fraction of exposed patches
p_I	fraction of infectious patches
p_R	fraction of recovered patches
c_Σ	colonization rate of compartment Σ ($\Sigma \in \{S, E, I, R\}$)
e_Σ	extinction rate of compartment Σ
η	incubation rate
α	probability of developing infection
γ	recovery rate
ξ	immunity loss rate

Figures

Figure 1: A theoretical example to show the differences among different definitions of reactivity. a) Simulations of the linear dynamical system described by matrix $\mathbf{A}$: all trajectories converge to the stable steady state $x_1 = x_2 = 0$. b) Effect of different output transformations for a sample trajectory of the model (thick line in panel a). The steady state of the model described by matrix $\mathbf{A}$ is non-g-reactive for matrices $\mathbf{C_1}$ (original reactivity *sensu* Neubert and Caswell, 1997) and $\mathbf{C_4}$, while it is g-reactive for matrices $\mathbf{C_2}$ (weighted reactivity *sensu* Verdy and Caswell, 2008), $\mathbf{C_3}$ and $\mathbf{C_5}$. The cases pertaining matrices $\mathbf{C_3}$, $\mathbf{C_4}$ and $\mathbf{C_5}$ can only be studied in our newly developd framework. See Appendix S1 for a detailed g-reactivity analysis of this example.

Figure 2: Stability and g-reactivity analysis of model (7), describing interspecific competition in a fragmented landscape. The focus is on the transient dynamics of species B. a) Stability and g-reactivity ranges of the steady-state attractors of the system: black curves represent separations between the stability regions of different equilibria (labels), while gray shading indicates parameter regions where the relevant steady state is g-reactive (dark gray indicates that the equilibrium is g-reactive also for perturbations involving only the variables included in the output transformation). b) Basin of g-reactivity for a stable, g-reactive extinction equilibrium ($c_A = c_B = 0.9$), evaluated either analytically via linearization (light shading) or through the numerical computation of $d||\mathbf{y}||/dt$ at $t = 0$ (dark shading). Black curves show some sample trajectories of the system in a neighborhood of the global extinction equilibrium ($p_A(0) = 0$ in all simulations). c) Maximum initial amplification rate (black line, left axis) and maximum overall amplification (gray line, right axis) of perturbations to the steady-state attractors (labels on top) of the system for different values of the colonization rate of species A ($c_B = 0.9$). Other parameters (panels a–c): $e_A = e_B = 1$, $\epsilon_A = \epsilon_B = 1/3$, $\kappa_A = \kappa_B = 3$. d) As in panel a, for a case of asymmetric competition in which species B is assumed to be competitively inferior to species A. Parameter values: $e_B = 2e_A$, $\epsilon_B = \epsilon_A/2$, $\kappa_B = 2\kappa_A$; other parameters as in panels a–c. In all panels, g-reactivity has been evaluated with respect to output matrix $\mathbf{C_B}$, with $u = v = 1$.

Figure 3: Stability and g-reactivity ranges of the steady-state attractors of model (10), describing predator-prey interactions in a fragmented landscape. Black curves indicate separations between the stability regions of different equilibria (labels), while gray shading illustrates parameter regions with g-reactive steady states (dark gray indicates that the equilibrium is g-reactive also for perturbations involving only the variables included in the output transformation), respectively for the prey-focused output matrix $\mathbf{C_Y}$ (panel a) or the predator-focused output matrix $\mathbf{C_D}$ (panel b). Parameter values: $e_Y = 1$, $e_D = 10$, $\epsilon_D = 1/10$, $\kappa_Y = 10$, $u = v = 1$.

Figure 4: Stability and g-reactivity analysis of model (13), describing pathogen transmission in a metapopulation ($SEIRS$ dynamics). a) Stability and g-reactivity ranges of the steady-state attractors of the system: black curves separate the stability regions of different equilibria (labels), while gray shading indicates parameter regions where the relevant steady state is g-reactive according to the epidemiologically-motivated output matrix $\mathbf{C_{EI}}$ (dark gray indicates that the equilibrium is g-reactive also for perturbations involving only the variables included in the output transformation). b) Asymptotic fractions of $S/E/I/R$ patches and total patch occupancy (legend) for different values of the baseline colonization rate c ($e = 0.1$). Labels on top show the stability ranges for the three equilibria of the system. Other parameters: $c_S = c_E = c_R = c$, $c_I = \epsilon_I c$, $\epsilon_I = 1/3$, $e_S = e_E = e_R = e$, $e_I = \kappa_I e$, $\kappa_I = 3$, $\alpha = 0.75$, $\eta = 1$, $\gamma = 0.1$, $\xi = 0.05$, $u = v = 1$.