Cooperation and competition in heterogeneous environments: The evolution of resource sharing in clonal plants

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Cooperation and competition in heterogeneous environments: the evolution of resource sharing in clonal plants

Krisztián Mágori,[1,3] Beáta Oborny,[2]* Ulf Dieckmann[3] and Géza Meszéna[1,4]

[1]*Department of Biological Physics,* [2]*Department of Plant Taxonomy and Ecology, Eötvös University, Budapest, Hungary,* [3]*Adaptive Dynamics Network, International Institute for Applied Systems Analysis, Laxenburg, Austria and* [4]*Collegium Budapest, Institute for Advanced Studies, Budapest, Hungary*

ABSTRACT

Plant species show great variation in the degree of physiological integration between developmental units (modules). When this physiological integration is minimal, individual modules are self-supporting and compete with other modules. When there is greater physiological integration, modules remain physiologically connected and 'cooperate' by sharing resources like water, nutrients and photoassimilates taken up from their local environments. Thus, local differences in habitat quality can be diminished within a group of modules. Here we examine how the evolutionarily optimal amount of integration depends on habitat type – with habitats being characterized by the proportion of resource-rich and resource-poor sites and by the turnover rate between them. Two main questions are addressed: First, how does spatial heterogeneity influence natural selection for or against integration? Second, can adaptation, under reasonable ecological conditions, stabilize partial integration? A non-spatial version of the model, which assumes well-mixed populations, predicts the complete physiological independence of modules as the only evolutionarily stable outcome in any realistic habitat type. By contrast, a spatially explicit version of the model reveals the adaptive advantage of integration in typical high-risk habitats, where resource-rich sites are sparsely distributed in space and transient in time. We conclude that habitat diversity without spatial population structure is sufficient to explain the evolutionary loss of physiological integration. But only the additional consideration of spatial population structure can convincingly explain any backward transition and the stable existence of partial integration.

Keywords: adaptive dynamics, cellular automata, clonal growth, competition, cooperation, patchy habitats, physiological integration, plant development, spatially structured populations.

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* Address all correspondence to Beáta Oborny, Department of Plant Taxonomy and Ecology, Eötvös University, Pázmány Péter sétány 1C, H-1117 Budapest, Hungary. e-mail: beata@ludens.elte.hu

INTRODUCTION

All vascular plants are modular – that is, they grow by reiterating discrete developmental programs (Harper, 1985; Hallé, 1986; Vuorisalo and Tuomi, 1986; Schmid, 1990). A module, in the broadest sense, is 'any distinguishable, repeated and multicellular structural unit within a genet' (Vuorisalo and Tuomi, 1986, p. 383). In some species, modules are highly interdependent physiologically, and an intensive transport of resources (nutrients, water and photoassimilates) is observed between units. In others species, modules attain some physiological autonomy. In the extreme, each module is fully self-supporting and able to develop all plant organs (root and shoot, including generative shoot) required for their independent existence. With the fragmentation of a genetic individual (genet) into multiple physiological individuals (ramets) serving as a mode of asexual reproduction, plant species with largely self-supporting modules are called 'clonal'. Jackson *et al.* (1985) and de Kroon and van Groenendael (1997) provide surveys of clonal development in nature.

Plant species show great variation in their physiological integration (Jónsdóttir and Watson, 1997) and in their morphological pattern of connections (Watson, 1986; Marshall and Price, 1997). For example, in typical 'splitter' clones, integration is zero: each new module becomes self-supporting soon after its establishment and no longer exchanges any resource with the older parts of the genet. The offspring either physically detaches itself from the mother (as in *Sempervivum tectorum* L.) or the physical connections persist but carry no material transport (as in *Ranunculus repens* L.). Complete splitting, however, represents only one extreme; further along the continuum, we find species that are capable of partial autonomy (e.g. *Aster lanceolatus* Willd.). Here the modules are interconnected but can regain autonomy after the damage of rhizome connections (Schmid and Bazzaz, 1987). Other species (e.g. *Trifolium repens* L.) are closer to the other extreme – full integration – with the transport of material being intensive, rapid and far-ranging (Marshall and Price, 1997). Full integration – that is, available resources are shared equally between members of a genet – is an idealization. Even typical non-clonal plants, like small annuals, show some sectoriality, resulting in restrictions to transport (Watson, 1986; Vuorisalo and Hutchings, 1996). The physiological processes of resource integration, and their implications for the performance of genets, have been studied by radioactive labelling and through manipulating resource supply to different parts of the plant. For excellent reviews of the differential in integration between various species, see Pitelka and Ashmun (1985), Marshall (1990), Jónsdóttir and Watson (1997) and Marshall and Price (1997). In general, even closely related species can exhibit significant differences in integration. For example, Wijesinghe and Whigham (2001) compared the response of three *Uvularia* species to the patchy distribution of nutrients and found clear interspecific variation in the probability for new modules to enter into bad patches. Alpert (1999) and van Kleunen *et al.* (2000) even found intraspecific genetic variation in the amount of integration between conspecific populations sampled from different habitats. These studies suggest that physiological integration is an evolutionarily flexible trait that allows for adaptation to prevailing habitat conditions.

In this paper, we focus on the selective forces driving the evolution of integration strategies and suggest answers to the following questions:

• Under which environmental conditions is it selectively advantageous to split up a physiologically integrated organism into autonomous modules?

- By contrast, which conditions favour (re)integration?
- Are there circumstances in which intermediate amounts of integration are specifically selected for?

A primary reason for splitting, supported by broad empirical evidence, is that physiological autonomy helps to spread the risks of mortality and of reproductive failure between modules (as suggested by Eriksson and Jerling, 1990). Conversely, physiological integration enables risk-sharing between modules. It has therefore been proposed that spatial heterogeneity in the quality of habitat sites is an important factor selecting for or against physiological integration. As an illustration of this point, consider a simple case of two connected modules. One module grows on a favourable site and the other experiences unfavourable conditions. When is it advantageous for the genet that these two modules share a limiting resource, as opposed to being physiologically autonomous? Clearly, the amount of integration that is optimal under these conditions depends on how resource availability translates into reproductive success of the modules (Eriksson and Jerling, 1990). If the resource utilization function describing this relation is convex, the reproductive success of a module resulting from half the amount of resources is less than half the success expected without sharing, and the same applies to all other sharing ratios. Complete physiological autonomy is then favoured. If, by contrast, the function is concave, sharing pays and complete integration is selected for. If the function is linear, the amount of integration is expected to be neutral. In short, unless $1 + 1$ is more than 2 in fitness terms, we should not expect to find physiological integration.

It is evident that this simple analysis has to be extended to account for the evolution of integration strategies under more realistic conditions:

- First, resource transfer clearly extends beyond modules that are nearest neighbours; therefore, interactions between more than just two modules have to be considered.
- Second, we need to account for the fact that modules with different integration strategies differ in their probabilities of being situated on sites of high or low quality.
- Third, the particular spatial structure of a heterogeneous environment modifies the costs and benefits of physiological integration. In particular, barriers of low-quality habitat may effectively prevent the spreading of non-integrating modules (Oborny *et al.*, 2000, 2001; Oborny and Kun, 2002).
- Fourth, previous work has not offered an explanation for the wide range of intermediate integration strategies found in nature: selection resulting from non-linear resource utilization efficiency, as described above, is expected to lead to modules that are either maximally integrated or maximally autonomous.
- Fifth, and perhaps most important, earlier studies have not shown how the evolution of integration strategies is driven by environmental conditions. Establishing such a link could provide a compelling explanation for the supposedly recurrent evolutionary transitions between integration and splitting. Plants conquering new habitat featuring different environmental conditions would then be expected to undergo corresponding evolutionary adjustment of their integration strategies.

The aim of this study is to delineate salient environmental conditions that facilitate evolutionary transitions from integration to splitting and vice versa. For this purpose, we analyse the implications of spatial structure in habitats and of plant genets that can adapt

their developmental phenotype between completely integrated and completely split growth. After introducing a simple plant population model in a spatial and a corresponding non-spatial version, we investigate the adaptation of the integration rate to various types of environment. We show that evolutionary outcomes are expected to differ dramatically between spatially structured and unstructured populations. More specifically, we demonstrate that, although the diversity of habitat qualities alone is sufficient to explain evolutionary transitions from integrated growth to splitting, spatial population structure is critical for a convincing explanation of any backward transitions. Once spatial structure is accounted for, the entire range of integration strategies becomes evolutionarily feasible and, in particular, intermediate integration strategies can be evolutionarily stabilized. Actual evolutionary outcomes are shown to depend on the quality and temporal stability of habitats.

MODEL DESCRIPTION

We consider an environment that is a mosaic of favourable (good) and unfavourable (bad) sites, which offer different conditions for the survival and reproduction of the considered organism. Each site represents a microhabitat for a single plant module. The environment changes in discrete steps, with time steps corresponding to the generation time of the modules. Each site can change its quality independently (from good to bad or vice versa) – that is, the habitat is fine-grained in space. Transition probabilities are set so that the total proportion of good sites remains constant over time.

We study competition between genetic individuals with different integration strategies. Each genetic individual (genet) consists of multiple modules, occupying a corresponding number of sites. We focus on reproduction through clonal (vegetative) growth and thus disregard recruitment from seeds. Modules with full integration are referred to as 'integrators' and those with complete autonomy as 'splitters' (Fig. 1). In other words, modules of a splitter genet attain physiological autonomy after their establishment, whereas those of
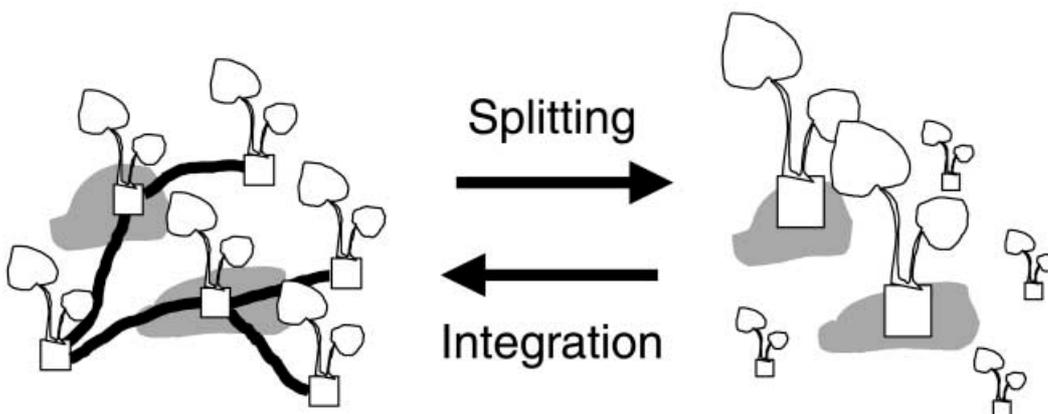

Fig. 1. Implications of physiological integration and splitting for modules in resource-rich (grey) and poor (white) sites. In a complete integrator (left), modules share the available resource equally, resulting in equal chances for survival and reproduction. In a complete splitter (right), no resource is transferred between modules. Each module survives and reproduces according to the local quality of its own site (depicted by the larger-sized modules in the resource-rich sites). We studied how optimal resource sharing depends on the density and temporal constancy of rich sites.

a (partial or full) integrator genet remain connected throughout their lives. For the sake of feasibility, the exact pattern of interconnections within genets is not tracked, and directional, age- or stage-dependent modes of resource transport between modules are not considered. Instead, all modules belonging to the same genet are assumed to be connected, and transport between modules is rapid compared with the modules' generation time (as supported by earlier empirical literature; see, for example, Marshall, 1990). Each module takes up a limiting resource from its local environment and, according to its integration strategy, shares a certain proportion of this uptake with the other modules of its genet. Unless integration is zero, modules on good sites have a net export of resources, whereas those on bad sites benefit by experiencing a net import of resources.

The amount of integration is a quantitative trait (metric character, continuous strategy) under frequency-dependent selection. New values of this trait can appear through mutations, which are considered to be rare on the time-scale of competitive exclusion between alternative integration strategies. A new mutant, therefore, typically encounters a population of resident modules that is at or close to its ecological equilibrium. On this basis, the invasion success of the mutant genet against the resident genet can be evaluated. This allows one to assess the outcome of the evolutionary process resulting from successive successful invasions and to determine how these outcomes depend on the environmental characteristics to which the population is exposed.

To highlight the effects of spatial structure on integration evolution, we consider a non-spatial and a spatial version of the model outlined above (Fig. 2). The spatial version is implemented as a two-dimensional cellular automaton on a square lattice with a von Neumann neighbourhood (involving the next four neighbours of a site). Time is discrete and updating is synchronous. By contrast, in the non-spatial version, module growth is not restricted to next neighbours and instead all modules compete for all empty sites. This implies that the spatial distributions of modules and genets are excluded from consideration. In both versions of the model, a time step consists of five subsequent processes: (1) environmental change, (2) resource redistribution within genets, (3) reproduction, (4) resource redistribution within genets and (5) survival.

Environmental change

In each time step, a site of good quality becomes bad with probability c^g and a bad site becomes good with probability c^b. In the spatial version, good and bad sites are distributed randomly over the lattice, whereas in the non-spatial version, only the entire sets of good and bad sites need to be considered (Fig. 2). If the total number of sites, n, is large enough, then the number of good and bad sites, n^g and n^b, change deterministically:

$$n^g \mapsto (1 - c^g) \cdot n^g + c^b \cdot n^b \tag{1a}$$

$$n^b \mapsto c^g \cdot n^g + (1 - c^b) \cdot n^b \tag{1b}$$

The case $c^g = c^b = 0$ corresponds to a constant environment, while $c^g = c^b = 1$ corresponds to one in which habitat qualities alternate deterministically. Between these extremes, the ratio of good sites converges to the equilibrium value $p = c^b/(c^g + c^b)$. We use p (characterizing habitat quality by the probability of a site to be of good quality) together with $c = c^g + c^b$ (characterizing habitat variability by the speed of environmental change) as
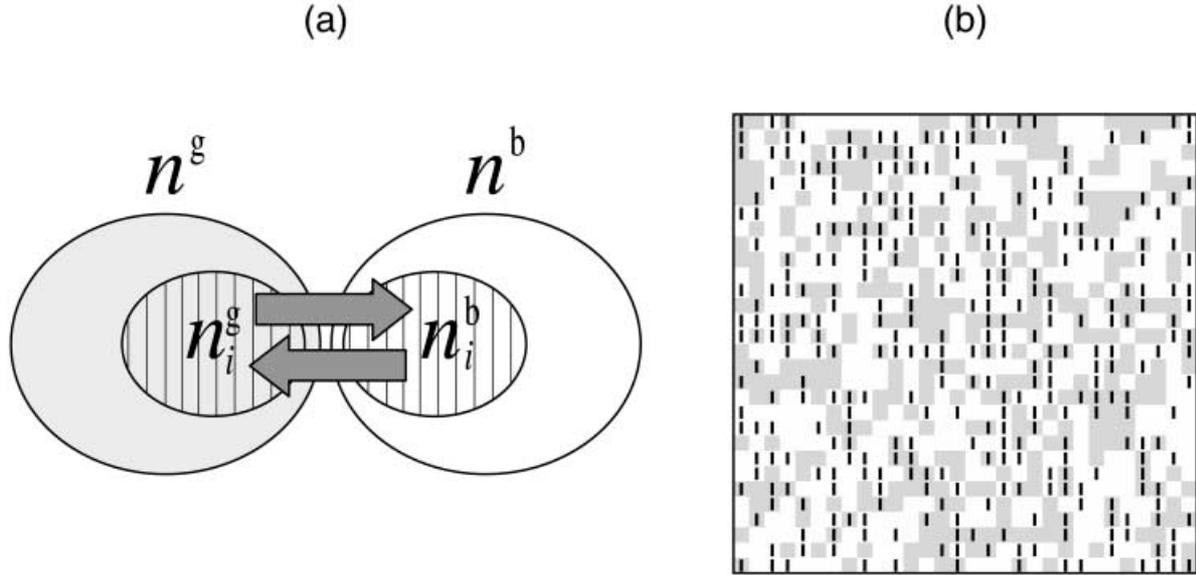
Fig. 2. Schematic representation of states and state transitions in the (a) non-spatial and (b) spatial version of our plant population model. Sites of high habitat quality (good sites) are shown in grey, with sites occupied by a module indicated by vertical lines. While the spatial model operates on a two-dimensional square lattice and colonization of empty sites is strictly local, the non-spatial model restricts attention to the global frequencies of good and bad sites that are occupied and empty, respectively, thus relying on the assumption of the system being well mixed with regard to these features. In (b), a reduced grid size of 30×30 has been chosen for the purpose of illustration.

the primary parameters of our model and express the transition probabilities c^g and c^b accordingly:

$$c^g = (1 - p) \cdot c \tag{2a}$$

$$c^b = p \cdot c \tag{2b}$$

Note that the consistency conditions $0 \leq c^g, c^b \leq 1$ imply that, for $0 \leq p \leq \frac{1}{2}$, c can be chosen from the range $[0, 1/(1 - p)]$, whereas for $\frac{1}{2} \leq p \leq 1$, the range $[0, 1/p]$ is feasible. The environmental process is initialized at equilibrium population sizes $n^g = p$ and $n^b = 1 - p$.

For $0 \leq c < 1$, equations (2a) and (2b) can be interpreted as indicating that a fraction c of all sites are reallocated between good and bad quality with probabilities p and $1 - p$, respectively. Environmental states are then positively correlated over time: good sites have a probability of more than p to retain their quality in one time step. The case $c = 1$ characterizes a random environment, in which qualities are uncorrelated between time steps. For $1 < c \leq 2$, environmental states are negatively correlated: in one time step, good sites then have a probability of less than p to keep their quality.

Resource redistribution

The amount of resource available on a single good site is set to 1, whereas bad sites provide no resource whatsoever. The integration strategy $0 \leq x \leq 1$ determines the fraction of the resource that a module shares with the other modules in its genet. Consider the ith genet of the population, with integration strategy x_i, occupying n_i^g good sites and n_i^b bad sites. The per capita amount of resource in the genet's resource pool then is $x_i \cdot n_i^g / (n_i^g + n_i^b)$ and is shared

equally between the modules of the genet. Modules on good sites have an additional amount of resource, $1 - x_i$. Consequently, the resource supply to a module in a bad and in a good site is given by

$$R_i^{\text{b}} = \frac{x_i \cdot n_i^{\text{g}}}{n_i^{\text{g}} + n_i^{\text{b}}} \tag{3a}$$

and

$$R_i^{\text{g}} = 1 - x_i + R_i^{\text{b}} \tag{3b}$$

respectively. The total amount of resource available to the whole genet is

$$R_i^{\text{tot}} = n_i^{\text{g}} \cdot R_i^{\text{g}} + n_i^{\text{b}} \cdot R_i^{\text{b}} = n_i^{\text{g}} \tag{3c}$$

and is not affected by redistribution of the resource. Resource availability has to be evaluated twice in each time step (before reproduction and before survival), since the new modules established during reproduction affect the amount of resource that is available to other modules in the genet.

Population dynamics: reproduction and survival

Modules reproduce by occupying empty sites in their neighbourhood. In the spatial version, neighbourhoods consist of the four nearest neighbours of a site, whereas in the non-spatial version, the neighbourhood extends to the set of all sites.

Modules differ in their chances of colonizing empty sites, owing to differential fertilities and competitive abilities. The fertility of a module of genet i situated on a site of quality q (good or bad) is assumed to be proportional to its resource supply R_i^q. (Here and below we focus on such linear relations because they provide the simplest plausible assumptions.) If two or more juvenile modules attempt to occupy the same empty site, they compete for establishment (local lottery competition; Chesson and Warner, 1981).

Since juveniles are not self-supporting before establishment and import their essential resource from their parent modules (if at all, connections are severed only after establishment), the competitive abilities of juvenile modules are assumed to be proportional to the resource supply R_i^q of their parents. The probability that a module succeeds in first producing and then establishing an offspring module on a given empty site in its neighbourhood is therefore proportional to $(R_i^q)^2$. To fully determine this probability, consider an empty site with a set N of occupied sites in its neighbourhood. The probability that the module on site $k \in N$ establishes its offspring on the empty site is

$$(R_{i(k)}^{q(k)})^2 \Big/ \sum_{k' \in N} (R_{i(k')}^{q(k')})^2 \tag{4}$$

where $q(k)$ is the quality of site k and $i(k)$ is the genet occupying site k.

Alternatives to the quadratic resource utilization function $(R_i^q)^2$ are highlighted in the Discussion, where we also explain why, in this function, an exponent larger than 1 appears plausible to us – even though we use the particular choice in equation (4) only for illustrative purposes. Since this choice intrinsically favours the strategy, $x = 0$, it renders conspicuous the effects of selection pressures favouring physiological integration and thus departures from $x = 0$.

After reproduction, the resource is redistributed between the old and newly established modules, and resource supplies are recalculated. A module of genet i situated on a site of quality q survives with a probability equalling its resource supply R_i^q.

Spatial and non-spatial versions of the model

The non-spatial and spatial versions of the model differ in their definition of neighbourhoods, although this only affects reproduction. However, because spatial structure is central to the latter version, implementation of these versions is entirely different. Relying on the convenient assumption of infinite (sufficiently large) population size, explicit recursion equations were derived and utilized for the non-spatial version. The corresponding results are presented in Appendix 1. By contrast, the numerical results had to be obtained for tracing through time the dynamics of the cellular automaton on which the spatial version is based. Implementation details for both model versions are described in Appendix 2. While the non-spatial version is based on deterministic dynamics, a finite lattice had to be used for the cellular automaton (Fig. 3), implying that demographic stochasticity was unavoidable in the spatial version.

Evolutionary invasibility analysis

To determine the evolutionary implications of the ecological setting described so far, we use the framework of adaptive dynamics (Metz *et al.*, 1992, 1996; Kisdi and Meszéna, 1993; Dieckmann, 1994, 1997; Dieckmann and Law, 1996; Geritz *et al.*, 1997, 1998). In line with the general definition of invasion fitness by Metz *et al.*, (1992), the invasion success of a mutant strategy x_m is judged by determining its growth rate $s_{x_r}(x_m)$ while rare in the environment set by a resident strategy x_r that has reached its ecological equilibrium (see also Turelli, 1978). Carrying out this investigation for many pairs of resident and mutant trait values provides information that, for one-dimensional quantitative traits, can be conveniently compiled into so-called pairwise invasibility plots (PIPs), which depict the sign of $s_{x_r}(x_m)$ as a function of x_r and x_m (Matsuda, 1985; van Tienderen and de Jong, 1986; Metz *et al.*, 1992; Kisdi and Meszéna, 1993; Geritz *et al.*, 1997; see also Taylor, 1989; examples of PIPs are shown in Figs 4a and 6a). For a detailed analysis of how to relate the long-term fitness of a mutant to its short-term net benefit, see Chesson and Peterson (2002).

By definition, a mutant population with a trait value equal to that of a resident strategy at equilibrium neither grows nor decreases, $s_{x_r}(x_r) > 0$. In each PIP, the main diagonal therefore separates regions of possible invasion success, $s_{x_r}(x_m) > 0$, from those of certain invasion failure, $s_{x_r}(x_m) < 0$. For a given resident strategy x_r, we can thus determine whether evolution favours a gradual increase or decrease of x_r by reading off from the PIP the sign of $s_{x_r}(x_m)$ right above and below the main diagonal. In this way, PIPs allow one to infer the direction of evolution by small mutation steps resulting from sequences of successive successful invasions.

In general, directional evolution converges either on an intermediate strategy or on one of the two extreme strategies represented in a PIP. So-called singular strategies are such internal strategies for which directional evolution comes to a halt. These strategies are recognizable in a PIP as intersection points between the main diagonal and the other curves on which the sign of $s_{x_r}(x_m)$ changes.
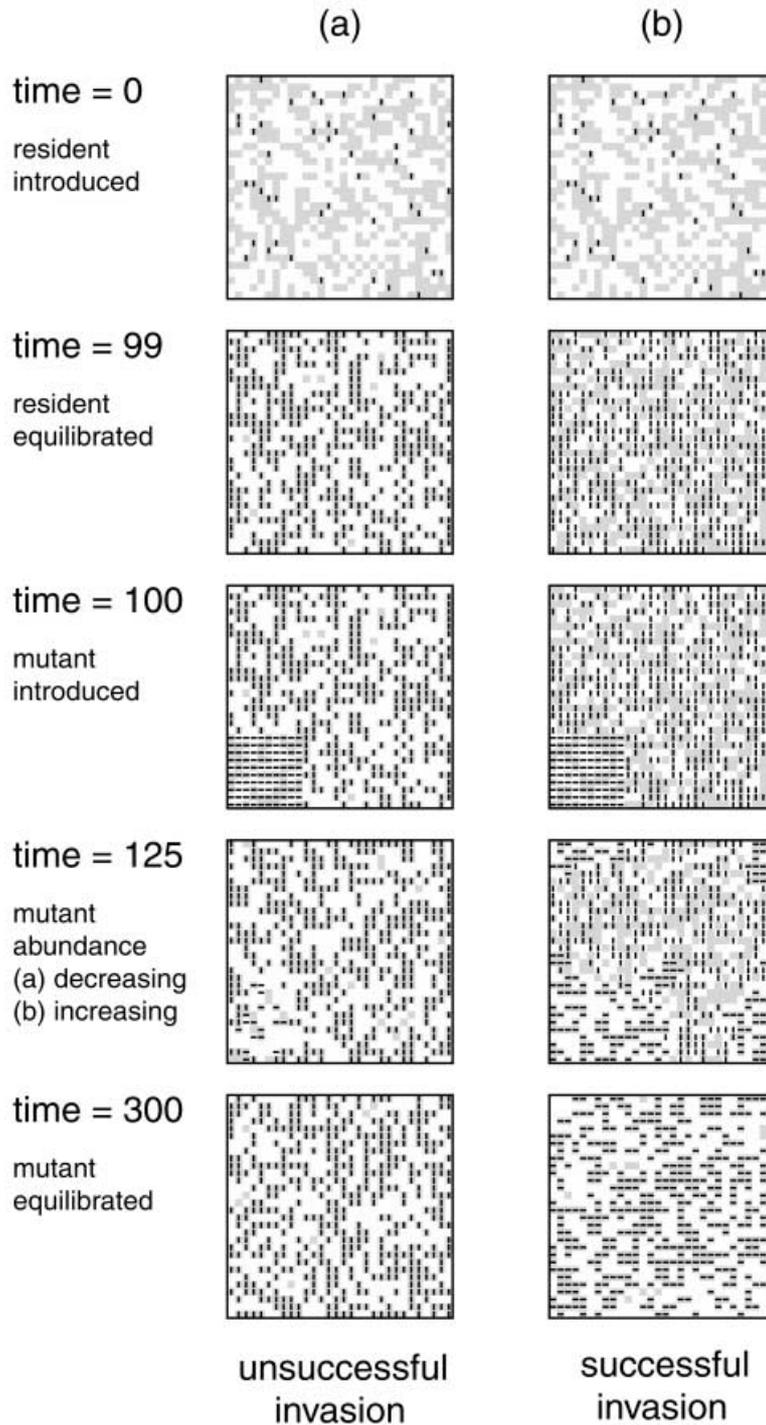
Fig. 3. Spatial invasion dynamics of mutant integration strategies. Good sites are shown in grey. Vertical and horizontal lines indicate sites occupied by modules with resident and mutant integration strategies, respectively. (a) Unsuccessful invasion. At time $t = 0$, resident modules are introduced to a 10% fraction of randomly chosen sites, upon which their abundance and spatial structure have time to equilibrate until $t = 99$. At time $t = 100$, mutant modules are introduced to a square-shaped subset of sites, giving the mutant an initial occupation of about 10%. Mutant modules decrease in abundance until $t = 125$ and vanish completely by $t = 300$. Parameters: $p = 0.5$, $c = 0.1$, $x_r = 0$, $x_m = 0.5$. (b) Successful invasion. After the mutant integration strategy has been introduced – again at $t = 100$ – it increases in abundance, as shown for $t = 125$, and has replaced all resident modules by $t = 300$. Parameters: $p = 0.5$, $c = 0.1$, $x_r = 1$, $x_m = 0$. A reduced grid size of 30×30 has been chosen for the purpose of illustration.

A singular strategy x^* is locally evolutionarily stable ([Maynard Smith, 1982](#)) if close-by mutants cannot invade. In the corresponding PIP, this means that $s_{x^*}(x_m)$ is negative for x_m above and below x^*. By contrast, a singular strategy x^* is convergence stable (acts as an evolutionary attractor; [Eshel and Motro, 1981](#); [Eshel, 1983](#); [Christiansen, 1991](#)) if close-by residents can be invaded by mutants that lie even closer to x^*. In the corresponding PIP, this means that to the left of x^*, $s_{x_r}(x_m)$ is positive above the main diagonal, and to the right of x^*, $s_{x_r}(x_m)$ is positive below the main diagonal.

RESULTS

When just a single integration strategy is present in the population, both the non-spatial and the spatial versions of the model exhibit the same simple behaviour: when alone, any strategy has an equilibrium population size of $p \cdot n$. This can be seen directly by considering that all empty sites are filled by individuals during the reproduction step, and that the average survival of individuals during one time step is p. The proportion p of good sites can therefore be interpreted as the carrying capacity of the environment and is identical for all integration strategies.

When two integration strategies are present simultaneously, it turns out that in our model competitive exclusion is inevitable. We carried out a full pairwise invasibility analysis (between mutant and resident integration strategies; see previous section) for all parameter combinations and for both model versions to confirm that one of the two strategies always outcompetes the other one. In other words, neither the non-spatial nor the spatial version of our model allows for the perpetual co-existence of two or more integration strategies. However, which of any two considered strategies will persist and oust the inferior one is a much more complex issue: the outcomes of this selection strongly depend on whether the non-spatial or spatial version of the model is considered and on the environmental conditions under which the competition process unfolds. Apart from the demographic stochasticity inevitable in the finite populations of the spatial model version, these outcomes turned out to be independent of initial condition (characterizing, for example, where and at what abundance the mutant was introduced). Figure 3 illustrates the process of competitive exclusion by showing, for the same environmental conditions, examples of successful and unsuccessful invasion resulting for two different pairs of resident and mutant integration strategies.

Non-spatial version

Figure 4a shows four typical pairwise invasibility plots (PIPs) for the non-spatial version of our model. As explained in the previous section, the main diagonal $x_m = x_r$ is always a zero contour line of the mutant's invasion fitness, $s_{x_r}(x_m)$. In the most complex case (third column), the other, non-trivial zero contour line is elliptical and has two intersections with the main diagonal. Of the resultant two singular points, the one with the lower integration rate is convergence stable and thus represents an evolutionary attractor, whereas the other singular point is convergence unstable and thus acts as an evolutionary repellor. In the other three PIPs, either no non-trivial zero contour line exists (first and fourth columns) or it intersects the main diagonal only once (second column). All attractors prove to be locally evolutionarily stable and all repellors are evolutionarily unstable, which is a non-trivial property of this model.
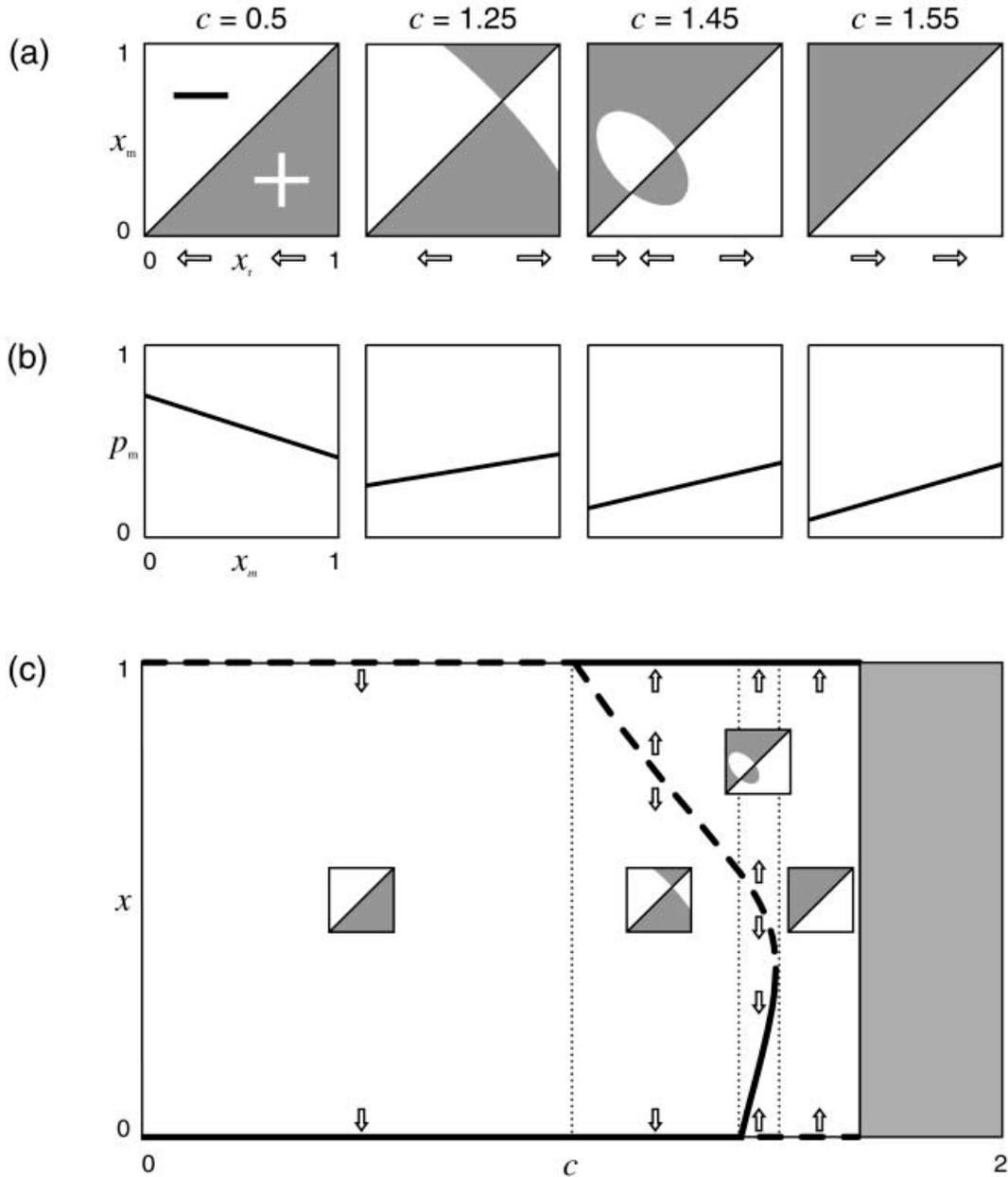
Fig. 4. Illustration of evolutionary regimes in the non-spatial model version for a particular proportion of good sites, $p = 0.4$. (a) Pairwise invasibility plots arising for four different rates of environmental change, c. In each of these plots, combinations of resident integration strategies x_r and mutant integration strategies x_m for which the mutant is successful in invading and replacing the resident are shown in grey. Hollow arrows indicate the resultant direction of evolution by small mutational steps. (b) Dependence of the proportion of mutant modules on good sites, p_m, on mutant integration strategy x_m. Panels characterize the four different evolutionary regimes for a resident integration strategy $x_r = 0.5$; other values of x_r give qualitatively similar results. (c) Bifurcation diagram for variation of c, showing the transitions between the four evolutionary regimes (dotted lines). Hollow arrows again show the direction of evolution. The location of convergence stable (unstable) integration strategies x is depicted by thick continuous (dashed) curves. The light grey area to the right corresponds to values of c that are infeasible at $p = 0.4$.

Figure 5 shows in detail how the type of PIP depends on average habitat quality (p) and habitat variability (c). If the temporal variability of the environment is not extremely high ($0 < c \leq 1$), mutants can invade whenever they have a lower integration rate than the resident ($x_m < x_r$), as can be seen from the PIP in Fig. 5d. Evolution, therefore, always proceeds towards splitting ($x = 0$). By increasing temporal variation such as to describe negatively
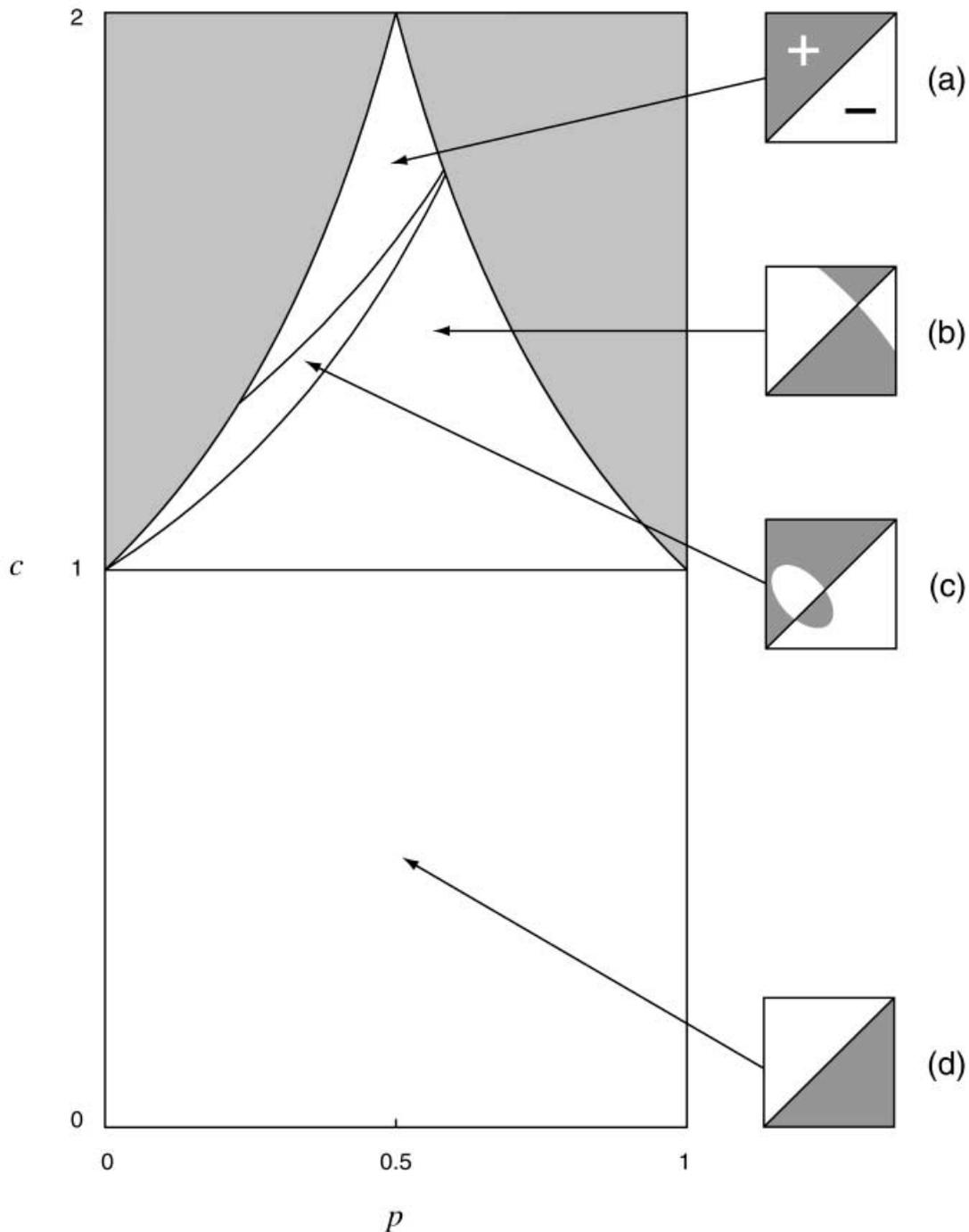

Fig. 5. Overview of evolutionary regimes in the non-spatial model version for dependence on habitat quality, p, and habitat variability, c. The two light grey areas at the top correspond to infeasible combinations of p and c. Altogether four evolutionary regimes are possible; however, for negatively autocorrelated environments, $c < 1$, evolution always favours complete splitting. Also note that the range of combinations of p and c that favour intermediate amounts of integration is narrow.

autocorrelated environments ($1 < c \leq 2$), an evolutionarily unstable internal repellor appears (Fig. 5b). This implies that, if environmental variation is larger than random, the extreme integration strategies $x = 0$ and $x = 1$ can both arise as the outcomes of the evolutionary process, depending on whether the process commences to the left or to the right of the repellor; this gives rise to evolutionary bistability. Increasing temporal variation further leads to the appearance of an interior evolutionary attractor and to a PIP with the elliptical zero contour line discussed above (Fig. 5c). An intermediate amount of integration is thus the expected evolutionary outcome if evolution starts to the left of the repellor, whereas starting to the right still results in complete integration. Finally, with extremely high temporal variation, both intermediate singular points collide and disappear, leaving complete integration as the only possible evolutionary outcome (Fig. 5a).

Figure 4c describes the transitions between these four fundamental evolutionary regimes in the form of a bifurcation diagram at $p = 0.4$. For $c < 1$, $x = 0$ is attracting and $x = 1$ is repelling. At $c = 1$, a bifurcation occurs: $x = 1$ becomes attracting with the emergence of an internal repellor with $x < 1$. At $c = 1.387$, the singular point $x = 0$ becomes repelling with the emergence of an internal attractor with $x > 0$. Finally, at $c = 1.469$, the internal attractor and repellor collide and thus disappear (a saddle-node bifurcation). As shown by Fig. 5, bifurcation sequences for other values of p are either similar or simpler.

Spatial version

Figures 6 and 7 summarize the results obtained for the spatial model version. Compared with the non-spatial version, a coarser resolution had to be chosen for the integration strategy to retain computational feasibility: Fig. 7 is based on computing PIPs for 147 combinations of habitat quality p and habitat variability c. These are the results of assessing the competitive outcomes of $11 \times 11 = 121$ combinations of resident and mutant strategy values, each of which in turn is based on 200 replicates of the individual-based, spatially explicit simulations illustrated in Fig. 3, involving 300 time steps. Figure 7 thus required 1.067 billion time steps to be carried out on a lattice of $100 \times 100 = 10{,}000$ sites.

In the spatial model version, populations are not viable in environments of low average quality, giving rise to the extinction region in Fig. 7 (dark grey area on the left). Not surprisingly, the sloped right boundary of this area indicates that environments with low temporal variability can sustain populations of slightly lower quality than highly variable environments.

The distribution of evolutionary regimes in the non-spatial and spatial model versions is fundamentally different (Fig. 5 and Fig. 7, respectively). In the spatial version, selection favours:

- full integration in almost all negatively autocorrelated environments (Fig. 7a);
- intermediate integration in low-quality and highly variable, yet positively autocorrelated environments (Fig. 7d); and
- complete splitting in high-quality and low-variability environments (Fig. 7e).

The two ancillary regimes depicted in Fig. 7b and 7c do not play an important role; since fitness differences around $p = c = 1$ are minute, the corresponding small parameter regions in Fig. 7, despite massive numerical investment, cannot be demarcated with high accuracy. Compared with the non-spatial model version, the most striking feature of the
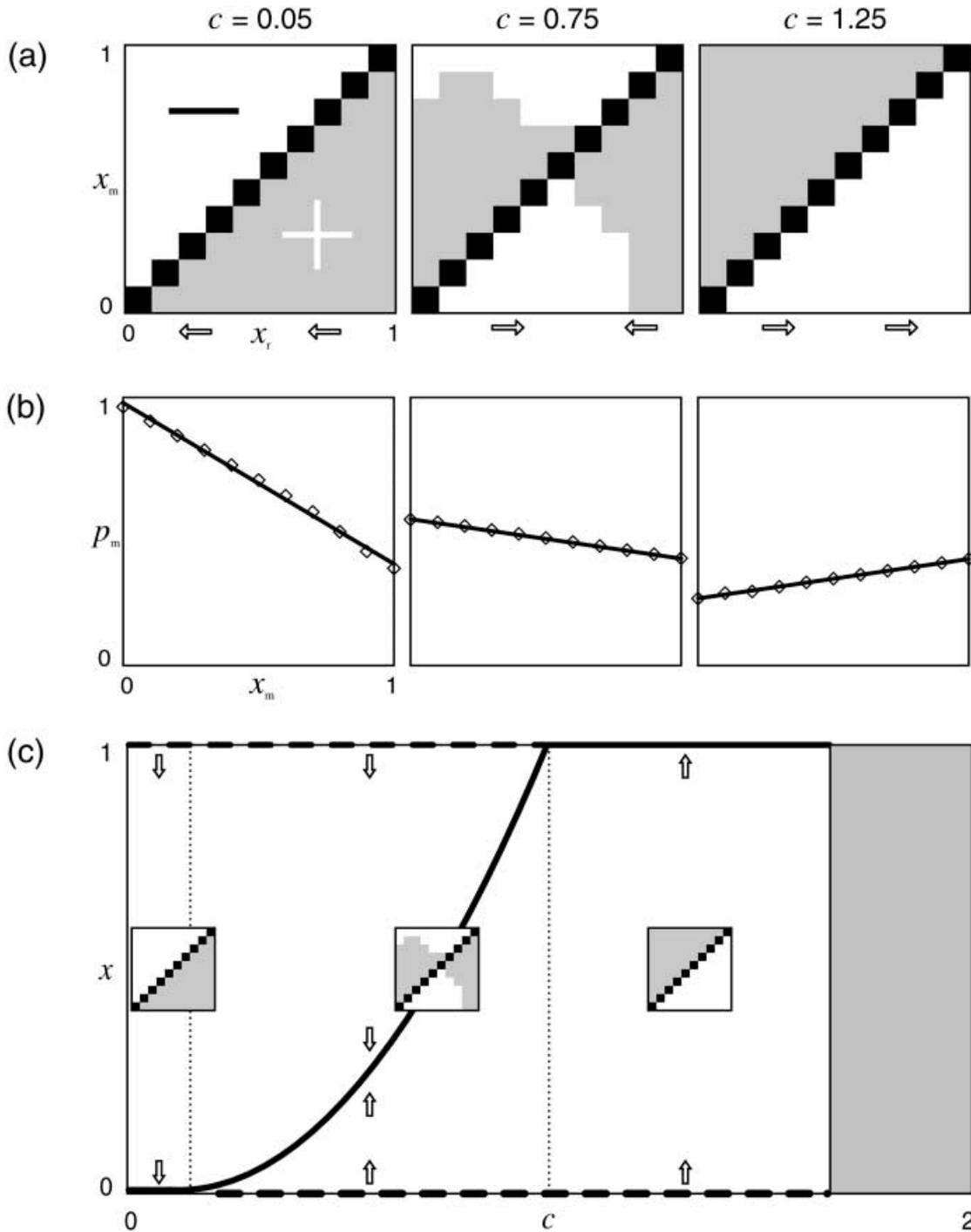
Fig. 6. Illustration of evolutionary regimes in the spatial model version for a particular proportion of good sites, $p = 0.4$. Graphical features are as in Fig. 4. (a) Pairwise invasibility plots arising for three different rates of environmental change, c. Due to demographic stochasticity, the results exhibit some noise. (b) Dependence of the proportion of mutant modules on good sites, p_m, on mutant integration strategy x_m. Panels characterize the three different evolutionary regimes for a resident integration strategy $x_r = 0.5$; other values of x_r give qualitatively similar results. Dependence of mutant habitat bias $p_m - p$ on resident and mutant integration strategies for the three evolutionary regimes. (c) Bifurcation diagram arising for variation of c, showing the transitions between the three evolutionary regimes. Note that, in contrast to Fig. 4, intermediate amounts of integration are favoured for $c < 1$.
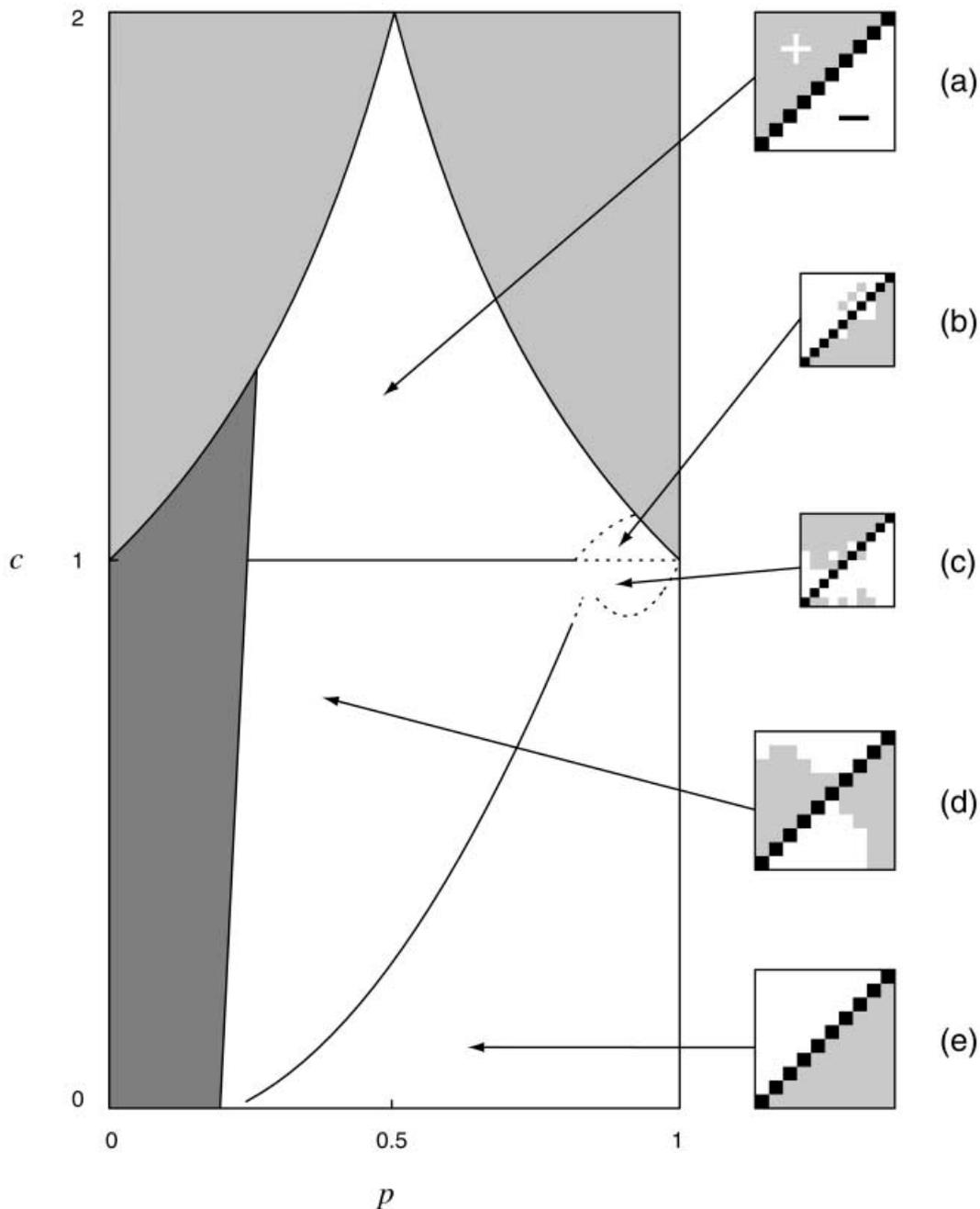
Fig. 7. Overview of evolutionary regimes in the spatial model version for dependence on habitat quality, p, and habitat variability, c. Graphical features are as in Fig. 5. The dark grey area to the left indicates combinations of p and c for which all resident integration strategies lead to extinction, an outcome that does not occur in the non-spatial version of the model. Four main evolutionary regimes are observed. Dashed curves in the vicinity of $p = c = 1$ enclose a small region for which, even with massive numerical investment, accurate localization of bifurcation curves turned out to be infeasible. A feature of primary interest in this plot is the existence of a wide range of combinations of p and c with $c < 1$ that favour intermediate amounts of integration.

spatial model version is the extended range of realistic environmental conditions that select for intermediate amounts of physiological integration (Fig. 7d). Note also that in positively autocorrelated environments, higher quality can compensate for higher variability: intermediate integration remains favoured in highly variable environments if these at the same time offer habitat of high average quality.

Figure 6c shows the bifurcation sequence of the spatial model version at $p = 0.4$. For very low temporal change c, a single evolutionary attractor is located at $x = 0$, indicating that, similar to the non-spatial model, full physiological autonomy is selectively favoured under such conditions. For environments with more variability, this attractor departs from the boundary $x = 0$ and leaves behind an evolutionary repellor. Increasing the temporal variability further, the attractor gradually moves from $x = 0$ towards $x = 1$ and arrives there for $c = 1$. For even larger variability, characteristic of negatively autocorrelated environments, only the boundary attractor at $x = 1$ remains and full integration is selected for.

Habitat bias

As a first step towards understanding the results described above, we consider p_m, the proportion of good sites among all the sites occupied by a rare mutant, when competing against a particular resident. We evaluate p_m for adult modules, before reproduction takes place. The departure of this proportion from p, the overall proportion of good sites, describes the mutant's habitat bias. For $p_m > p$, mutant modules in the resident's environment are favoured by a bias towards good sites, whereas for $p_m < p$, mutant modules are biased towards bad sites. The habitat bias $p_m - p$, therefore, serves as a convenient measure of module-environment correlation: only when $p_m - p = 0$ are a site's habitat quality and its occupation by the mutant uncorrelated.

Figures 4b and 6b show the dependence of p_m on the mutant integration strategy for the different evolutionary regimes that occur, respectively, in the non-spatial and spatial model versions. In positively autocorrelated environments ($c < 1$), habitat bias decreases when the mutant's integration rate increases. The reason is that diminished integration results in higher mortality differences between mutant modules located on good and bad sites, implying a higher relative occupancy of good sites after survival. This relation is reversed in negatively autocorrelated environments ($c > 1$): now high integration rates promote more favourable habitat biases for the mutant. The reason is that the higher relative occupancy of good sites after survival is turned on its head by the alternating nature of negatively autocorrelated environmental change. (As the reproduction step does not reverse this tendency, the behaviour of p_m is similar when calculated after the reproduction step.)

Understanding selection on physiological integration

The results we have obtained above can be understood with reference to three fundamental mechanisms that impose selection pressures on integration strategies:

1. Non-linear resource utilization efficiency selects for splitting in our model.
2. Habitat bias selects for splitting if $c < 1$ and for integration if $c > 1$.
3. The capacity for spatial spreading is enhanced by integration. Consequently, any habitat in which the ability to spread is important, but limited, selects for integration.

We now review these effects in sequence and use them to explain the outcomes of integration evolution noted above for various environmental conditions.

As we have already highlighted in the Introduction, the potential non-linearity of resource utilization alone can already select for full integration or complete splitting. If the efficiency of resource utilization decreases when more resource is available, the function that describes how the reproductive output of a module depends on its resource availability is

concave. Under such circumstances, passing on a certain amount of resource to an adjacent resource-deprived module makes the amount more valuable, as the poor recipient's utilization efficiency exceeds that of the rich donor. Sharing a resource between such modules of a genet thus increases the genet's reproductive output and full integration is selected for (Eriksson and Jerling, 1990). By contrast, if the resource utilization function is convex, the richest modules are maximally efficient. Under such conditions, the sharing of a resource is wasteful and complete splitting is selected for. This primary selection pressure operates independently of any module–environment or module–module correlations. In this study, we have focused on a convex resource utilization function. The quadratic function in equation (4) is a natural choice when assuming that the fertility of a module, as well as the establishment success of its offspring, increases linearly with the amount of resource available to the parent. Resource redistribution from rich to poor modules then handicaps reproduction of the rich modules more than its helps reproduction of the poor ones. Consequently, as shown in Appendix 1, the mutant population's average reproductive success is a decreasing function of x_m, its degree of integration. If this selection pressure were acting alone, we would see evolution towards complete splitting under all environmental conditions, both for the non-spatial and spatial versions of our model.

The selection pressure arising from habitat bias leads to a first correction of this expectation. Integration also affects the average amount of resource available to modules of the mutant genet, which equals the proportion p_m of mutant modules located on good sites. As shown above, this proportion is a decreasing function of x_m for $c > 1$ and an increasing function for $c > 1$; for random environments, $c = 1$, there is no habitat bias. Consequently, for $c < 1$, habitat bias favours decreasing integration rates: the resultant genet is better concentrated on good sites and thus enjoys a higher average amount of resource available to its modules. Analogously, for $c > 1$, habitat bias favours increasing integration rates. The selection pressure resulting from habitat bias only comes into play when modules are not fully randomly distributed over sites; in other words, it originates from module–environment correlations. Such correlations are ubiquitous in nature (Caldwell and Pearcy, 1994): biases of modules towards relatively resource-rich sites have been explicitly measured in studies of plant foraging (Sutherland, 1990; Hutchings and de Kroon, 1994; Oborny *et al.*, 2001).

The following relations help to assess the interplay of Effects 1 and 2 as described above:

A. Effect 1 gradually weakens towards, and ceases at, full integration, $x = 1$, as the difference between rich and poor modules diminishes.
B. Effect 1 weakens when p_m approaches 0 or 1, since the qualities of occupied sites then become more and more homogeneous.
C. Effect 2 disappears at $c = 1$, because random environments do not allow for biased occupation of good and bad sites. Habitat bias becomes stronger when c departs from 1 in either direction.

Calculations corroborating the first two relations are presented in Appendix 1. Based on Effects 1 and 2 and with the help of Relations A–C, we can now explain the evolution of integration strategies in the non-spatial model (Figs 4 and 5).

Habitat bias selects against integration in positively autocorrelated environments. This means that for $c < 1$, Effects 1 and 2 act synergistically, implying evolution towards complete splitting.

For negatively autocorrelated environments, $c > 1$, Effects 1 and 2 act antagonistically, which entails that the outcome of evolution depends on the relative strength of these selection pressures: where the effect of habitat bias prevails, selection favours increased integration. According to Relation C, this is the case for large values of c. By contrast, for lower values of c, the impact of habitat bias decreases and the relative strength of the two effects depends on the amounts of integration. In particular, at low values of x, Effect 1 dominates and selects for decreasing integration (Relation A); for higher x, Effect 2 prevails and selects for increasing integration. This is the reason for the emergence of an evolutionary repellor at intermediate values of x (such that any perturbation drives evolution away from the singular point). Decreasing c towards 1 reduces the range where Effect 2 dominates (Relation C), so that the position of the repellor converges to $x = 1$ (Fig. 4c).

For a narrow range of c in Fig. 4c, an internal evolutionary attractor can also appear. Within this range, Effect 2 dominates Effect 1 not only for high but also for low integration, while for intermediate integration Effect 1 remains stronger. Note that this range is located at $c > 1$: the proportion of good sites changing into bad sites within one time step is thus high. Since weakly integrated genets are more dependent on good sites, they experience more severe environmental change than do strongly integrated genets, such that p_{m} tends to be small for low integration. According to Relation B, Effect 1 then becomes weaker, enabling a balance with Effect 2. This gives rise to an internal evolutionary attractor. Convergence to this attractor applies only locally, with the extent of its basin of attraction delimited by the evolutionary repellor described above. This means that initial integration strategies above the repellor do not converge towards the internal evolutionary attractor but instead to full integration. The range of environmental parameters that allow for such an internal attractor is rather narrow; since Effect 2 rapidly weakens towards, $c = 1$, the attractor approaches the boundary value $x = 0$ (Fig. 4c). In the non-spatial model version, evolutionary convergence towards intermediate integration strategies is thus of very limited relevance and requires positively autocorrelated environmental change, fine-tuned combinations of average habitat quality and habitat stability, as well as restrictive initial conditions for the integration strategy.

The stability or instability of complete integration deserves special attention (see the line $x = 1$ in Fig. 4c). At $x = 1$, Effect 1 vanishes completely (Relation A) and the direction of evolution is determined solely by Effect 2. Habitat bias favours splitting at $c < 1$, and supports integration at $c > 1$, with this qualitative change in selection pressure being applicable to all values of p. In Fig. 5, $c = 1$ therefore separates the region $c > 1$ in which evolution converges locally towards full integration and the region $c < 1$ in which $x = 1$ is repelling.

The slopes of the boundary lines between the regions characterized by Fig. 5a, 5b and 5c are explained by a weakening of Effect 1 for low values of p. In random environments, $c = 1$, this weakening is a direct consequence of Relation B (the habitat bias $p_{\mathrm{m}} - p$ vanishes here); the same tendency must prevail for values of c near to 1.

To further verify the validity of these explanations, which are all consistent with the results shown in Fig. 5, we investigated two variations on the non-spatial model version. First, by using the linear function R_i^q instead of the convex function $(R_i^q)^2$ for determining the probability of offspring production in equation (4), Effect 1 disappears because the values of a shared resource between a donor and a recipient module are identical. Only Effect 2 remains, which implies that complete splitting is favoured for $c < 1$, while full integration evolves for $c > 1$. Second, when using the concave function $\sqrt{R_i^q}$ in equation (4), the

selection pressures resulting from Effect 1 are reverted. Effects 1 and 2 are then antagonistic for $c < 1$ and synergistically favour physiological autonomy for $c > 1$. Since Effect 2 becomes stronger at lower integration rates, intermediate integration strategies are then evolutionarily stabilized in a region below $c = 1$.

The additional Effect 3 is present only in the spatial model version. With the non-spatial version being the mean-field approximation of the spatial one (Law *et al.*, 2001), differences in evolutionary outcomes between the two are, by definition, a consequence of spatial population structure and therefore of module–module correlations. The most compelling differences are, first, a radical expansion of the range over which full integration is selected, resulting in this regime's spanning the entire feasible parameter range for $c > 1$, and, second, selection for intermediate integration rates over a large range of environmental conditions for $c < 1$ (Figs 5 and 7). Since Effects 1 and 2 are independent of module–module correlations, these striking differences can only be explained by a markedly increased advantage of integration in spatially structured module populations. We posit that the additional benefit to integration originates from the capacity of genets with integrated modules to traverse barriers of unsuitable habitat (Oborny *et al.*, 2000, 2001; Oborny and Kun, 2002).

Such a capacity for spatial spreading is essential, since module clusters of finite size go extinct with certainty. Integration allows genets to spread through regions of bad sites, which, at any given moment, separate clusters of good sites. Such an improved spreading capacity confers advantages in competing for newly emerging clusters of good habitat (for studies of this selection pressure on dispersal rates in metapopulation models, see Levin *et al.*, 1984; Metz and Gyllenberg, 2001; Kisdi, in press). In general, therefore, spatial population structure introduces a potent selection pressure towards integration.

The following intuitively evident relations help to assess the interaction of Effect 3 with Effects 1 and 2:

D. Effect 3 gradually weakens towards full integration, $x = 1$, as the difficulty of spreading through unsuitable habitat vanishes when the difference in resource supply to modules located on good and bad sites fades.

E. Effect 3 diminishes in environments of high quality, since a high proportion of good sites intrinsically facilitates spatial spread, without depending on integration.

F. Effect 3 diminishes in environments of low variability, in which the extinction risk of module clusters is low.

The qualitative expectations resulting from these relations are fully consistent with the results depicted in Figs 6 and 7. For $c > 1$, Effect 3 acts synergistically with Effect 2, so that the two effects together can overcome Effect 1, except in the region corresponding to Fig. 7b. For $c < 1$, Effect 3 opposes Effects 1 and 2. Because of Relation D, only low values of integration allow Effect 3 to dominate and to select for increasing integration. In other cases, Effects 1 and 2 drive evolution towards decreasing integration. These antagonistic effects give rise to an internal attractor for a rather broad range of parameter combinations (Fig. 7d). However, in typical low-risk environments (with high quality and low variability), Effect 3 prevails according to Relations E and F, and integration evolution converges towards full splitting (Fig. 7e). Like for the non-spatial version $c = 1$, delineates two different regimes, since the direction of evolution at full integration is solely determined by Effect 2.

DISCUSSION

Three fundamental selection pressures on physiological integration

In this study, we have investigated the interplay between three fundamental selection pressures that are expected to jointly determine the extent of physiological integration. To our knowledge, this is the first study to allow for a continuum of integration strategies (rather than considering only two extreme types) and which systematically evaluates how environmental conditions affect gradual evolutionary change in these strategies. Salient environmental factors have been analysed, including, most importantly, the quality and stability of spatially structured habitats. To explain their evolutionary implications, a hierarchical pattern of three mechanisms has been established and examined:

- *Effect 1: Non-linear resource utilization efficiency.* As Eriksson and Jerling (1990) have demonstrated, the advantage of resource sharing depends on how the available resource is converted into reproductive output of modules. When resource utilization functions are linear, physiological integration is predicted to be selectively neutral, while convex (concave) functions select against (for) physiological integration. Effect 1 already applies to a pair of interconnected modules; it is particularly strong for highly non-linear resource utilization functions.
- *Effect 2: Habitat bias.* Depending on their integration strategy, the distribution of modules over good and bad sites can systematically deviate from randomness. The resulting habitat bias selects for physiological autonomy in relatively stable (positively autocorrelated) environments, while in very unstable (negatively autocorrelated) environments habitat bias selects for integration. Oborny *et al.* (2000, 2001) have demonstrated that habitat bias readily occurs in realistic models of spatially extended populations. Effect 2 results from correlations between the quality and occupancy of sites; it is particularly strong when local habitat quality is strongly (positively or negatively) correlated over time.
- *Effect 3: Capacity for spatial spread.* The amount of physiological integration also affects the pace at which modules can spread over a heterogeneous habitat and (re)colonize distant high-quality patches (Oborny and Kun, 2002). Spatial barriers of low-quality habitat can only be traversed by physiological integration, and this confers an important selective advantage to integration. Effect 3 results from correlations between the occupancy of neighbouring sites; it is particularly strong when integration is low, habitat quality is low or habitat variability is high.

Understanding the evolution of integration strategies in realistic ecological settings requires the joint consideration of all three driving forces. Effect 1, non-linear resource utilization efficiency, is sufficient for explaining integration evolution in spatially unstructured populations, supporting predictions by Eriksson and Jerling (1990). Such reasoning, however, is limited to a pair of modules and, as shown by Oborny *et al.* (2001), is not sufficient to predict evolution in spatially structured populations or environments. Effect 2, habitat bias, is superimposed on this primary effect if the quality and occupancy of sites are correlated. Effects 1 and 2 together are sufficient to explain the evolutionary outcomes observed in the non-spatial model version examined in this paper. Finally, Effect

3, capacity for spatial spread, is superimposed on Effects 1 and 2 if occupied sites are spatially correlated. The combination of Effects 1–3 allows us to understand the evolutionary outcomes observed in the spatial model version examined in this paper.

We thus conclude that, in positively autocorrelated environments, in which utilization of a limiting resource is described by a convex function, Effects 1 and 2 select against physiological integration. Results derived in this paper (see Figs 6 and 7) demonstrate that Effect 3 not only counteracts the combined selection pressure from Effects 1 and 2, but that it can actually be strong enough to provide a net evolutionary benefit to intermediate amounts of integration. Under such circumstances, Effect 3 is thus critical for explaining the evolutionary emergence and maintenance of physiological integration.

Temporal autocorrelation and resource utilization functions

To better appreciate the findings just summarized, it is helpful to reflect on the likelihood of encountering negatively autocorrelated environments or concave resource utilization functions in nature.

It has to be emphasized that negative temporal autocorrelation of habitat qualities is very rare in nature, especially on the fine time-scale considered here. In our model, a time unit corresponds to the developmental time of a module; this can range from days to years, depending on the species, but is most likely to be short compared with the average time it takes for habitat qualities to become reversed. In nature, positively autocorrelated environments must hence be considered as being far more widespread than negatively autocorrelated environments.

By contrast, no agreement exists in the literature about the likely shape of resource utilization functions. To illustrate the analysis in this paper, we used a convex utilization function of quadratic shape. Assuming the probabilities of development of a new module and of maintenance of that module until self-support to be both linearly dependent on the amount of resource available to the mother appeared to us a plausible minimal assumption. Yet, many other function shapes can reasonably be considered. In particular, when there is such an oversupply of resource that modules get saturated could lead to a diminishing return of resource retention and thus to concave utilization functions. Even mixed cases, in which a resource utilization function is convex at low resource availability and becomes concave at high availability, could then arise. However, since the resource considered in this study is limiting, such situations are unlikely. Note also that the separate dependences of module development and initial maintenance on resource availability both have to be sufficiently concave for their product still not to be convex.

Even though there are thus reasons to expect convex rather than concave resource utilization functions, with the empirical knowledge we currently have we essentially have to remain agnostic about their particular shape. Although this may be deplorable, it leaves the main insights from our study unaffected: these are based on disentangling the selection pressures acting on physiological integration according to the trinity of effects presented above and on understanding how the strengths of these selection pressures vary with environmental conditions.

It is reassuring to realize that, contrary to Effect 1, Effects 2 and 3 do not sensitively depend on the shape of resource utilization functions: habitat bias and a capacity for spatial spread are expected to robustly select for splitting and integration, respectively, under realistic assumptions about environmental conditions.

High-risk environments, dispersal limitation and frequency dependence

The balance between the three fundamental selection pressures described above can only be appreciated in spatially structured evolutionary models. This balance offers an explanation for the existence of intermediate integration strategies in nature, and for the occurrence of evolutionary transitions from splitting to integration and back. The reason for the significance of spatial effects is that physiological integration enables modules to disperse across gaps of low habitat quality. This facilitates the escape from shrinking patches of favourable habitat and the colonization of newly emerging high-quality patches. We have demonstrated that the resulting selection pressure is strong when temporal fluctuations are relatively large and average habitat quality is low. Put differently, integration is favoured in typical high-risk environments.

This is consistent with the hypothesis, frequently suggested in the empirical literature, that integration helps to buffer local fluctuations in site qualities (Hartnett and Bazzaz, 1985; Pitelka and Ashmun, 1985; Alpert and Mooney, 1986; Hutchings and Bradbury, 1986; Eriksson and Jerling, 1990; Pennings and Callaway, 2000). The results presented here shed some new light on this hypothesis by clarifying that buffering cannot be expected to select for integration in the absence of dispersal limitation. In the non-spatial version of the model, dispersal is unlimited, and then even large fluctuations of habitat conditions in space and time (up to random change) proved to be insufficient for promoting integration. Only when considering the dispersal limitation inherent in the spatial version of our model, a high risk of resource shortage combined with dispersal barriers imposed by clusters of bad sites can exert a sufficiently strong selection pressure for integration to become advantageous. As shown in Fig. 7, selection for full integration still ceases for particular combinations of average habitat quality and stability. This underlines the fact that the extent to which an advantage of buffering environmental fluctuations selects for integration can only be properly appreciated in quantitative models, which assess the balance between the various selection pressures that simultaneously affect the evolution of integration strategies.

The intermediate integration strategies found in our analysis are stabilized by frequency-dependent selection. This implies that in the evolutionary processes we have considered, the selective advantage of a particular integration strategy depends on the prevalent strategy against which it competes. We believe that this basic feature is an indispensable property of realistic models of competition between different strategies of physiological integration; models in which this feedback on fitness is not incorporated fail to capture a critical aspect of integration evolution. Analysing the outcomes of pairwise contests allowed us to assess the expected course of evolution. Such evolutionary invasibility analyses, based on quantitative characters and realistic ecological dynamics involving both density- and frequency-dependent selection, lie at the heart of adaptive dynamics theory (Brown and Vincent, 1987; Hofbauer and Sigmund, 1990; Metz *et al.*, 1992, 1996; Kisdi and Meszéna, 1993; Dieckmann, 1994, 1997; Dieckmann and Law, 1996; Geritz *et al.*, 1997, 1998). The evolutionary implications of many interesting ecological settings have already been analysed in such a manner (e.g. Brown and Pavlovic, 1992; Meszéna *et al.*, 1997; Kisdi and Geritz, 1999; Doebeli and Dieckmann, 2000; Mathias *et al.*, 2001; Mizera and Meszéna, 2003). The present study is the first to extend this approach to a cellular automaton model.

Limitations

The analysis presented here has focused on the resource budget of potentially autonomous modules, and inevitably failed to capture some other interesting effects. For example, we assumed that: (a) the lifespan of connections between integrated modules was unconstrained; (b) the direction and magnitude of transport did not depend on the age or developmental stage of modules; (c) modular growth was the only method for dispersal; and (d) differences in resource supply did not cause any morphological change in the direction or distance of module placement (i.e. foraging responses were excluded). In addition, we assumed that (e) within a genet ramets shared resources through a common pool.

Assumption (e) appears to be a reasonable simplification, since resource transport is typically very fast compared with clonal growth. The time scale at which a newly established module develops can range from several days to years, depending on the species. By contrast, the transport of resources through the vascular system is estimated to take hours or days. For example, D'Hertefeldt and Jónsdóttir (1999) studied the translocation of a tracer, acid fuchsin dye, in *Carex arenaria*. They treated the root system of a single ramet by the dye and observed the distance of translocation within a whole, interconnected system of ramets. They found that the dye reached 90% of the distance to the rhizome apex within 72 h. On average, the tracer diffused through 28 ramet generations (with a maximum of 48 generations) and travelled more than 2 m (with a maximum of 4 m). Considering the rate of clonal growth of the species (D'Hertefeldt and Jónsdóttir, 1999), we can estimate that the development of this rhizome length requires at least 3–4 years. Therefore, the product of more than 3 years of clonal growth was traversed by diffusion within 3 days. A common resource pool hence describes such situations adequately, provided that the connected parts of a genet are large against the scale of spatial heterogeneity.

The other simplifications are more critical. Several studies have suggested, directly or indirectly, that relaxing assumptions (a) to (d) can influence the pattern of spatial spreading (a: Jónsdóttir and Watson, 1997; b: Marshall, 1990; c: Eriksson, 1997; Winkler and Fischer, 2002; d: Hutchings and de Kroon, 1994; Wijesinghe and Whigham, 2001; Herben and Suzuki, 2002) and could thus interfere with the results presented here. The potentially intricate interactions between these separate effects are not yet understood in any general way. Clearly, such investigations must remain a challenge for future research (Cain *et al.*, 1996; Oborny *et al.*, 2001). As a proximal aim, tactical models for specific plants could take into consideration the whole developmental process of the plant as a basis for studying the selective value of integration (as exemplified by studies on *Podophyllum peltatum* and *Carex bigelowii* by Jónsdóttir and Watson, 1997). In this context, it is especially important to consider the morphological and physiological constraints on integration that are characteristic for a particular species (Stuefer, 1996).

Directions for future research

There are two exciting, more general directions for extending this study. First is the consideration of additional factors that can influence the selective advantage of physiological integration. It has been convincingly argued that additional selection pressures favouring resource sharing can occur when modules critically depend on more than one resource

(Chesson and Peterson, 2002; for reciprocal translocation of limiting resources, see Stuefer and Hutchings, 1994; Stuefer *et al.*, 1994; Stuefer, 1996; Alpert and Stuefer, 1997; Hutchings *et al.*, 2000). Although such considerations are clearly beyond the scope of the present paper, it would be very worthwhile to extend the model presented here to accommodate multiple resources, multivariate resource utilization functions, and multi-component integration strategies regulating resource exchange in such a much more complex system. Suggesting another direction of extension, connections between modules may serve as pathways for the spreading of pests (Wennström, 1999), thus detracting from the benefits of integration. In addition, interconnecting tissues may have specific functions, such as storage, which can be observed in many rhizomatous and stoloniferous plants (Suzuki and Hutchings, 1997; Stuefer and Huber, 1999; Suzuki and Stuefer, 1999).

Second, the current study has focused on the evolutionary implications of temporally and spatially heterogeneous environments, the latter being characterized by the emerging module–environment and module–module correlations. To cover an even wider range of environmental settings, it would be interesting to consider the potential evolutionary implications of spatial autocorrelations in habitat qualities (environment–environment correlations; Oborny *et al.*, 2000; Law *et al.*, 2001). In many natural systems, a high-quality site is more likely to be surrounded by other sites of comparable quality than by those of low quality. The resultant average spatial distance over which habitat quality is correlated can be small or large and may well fine-tune the evolution of integration strategies as described here. In addition, in a possible multi-resource extension of our model, spatial cross-correlations between different resources (e.g. light and water) would certainly influence the evolving integration strategies.

We have shown that frequency-dependent selection pressures emerging in spatially structured populations are required to understand the evolution of integration. We have also described how the resultant evolutionary outcomes depend on the quality and stability of spatially structured habitats. The present results have clear implications for understanding the evolution of clonal growth. An important element in clonality is that individual modules attain physiological autonomy, allowing a genetic individual (genet) to split up into multiple physiological individuals (ramets). This transition was not a unique event in plant phylogenesis (de Kroon and van Groenendael, 1990; Mogie and Hutchings, 1990; Klimeš *et al.*, 1997; Sachs, 2002). Instead, clonal growth appears to be an evolutionarily flexible trait, which has appeared, disappeared and probably sometimes re-appeared on several branches of the phylogenetic tree. This observation makes it important to understand the selection pressures that can lead towards or away from clonality. Our results suggest a need for adaptation to environmental heterogeneity to play a key role for this evolution. But the direction of selection (for or against clonality) depends on the actual pattern of environmental heterogeneity. Whenever spatial spreading is limited by the scarcity or ephemeral nature of resource-rich sites, clonal growth is unlikely to emerge. By contrast, when the density and persistence of resource-rich sites are high enough to enable the lateral colonization of neighbourhoods, we can expect evolutionary transitions from aclonal to clonal growth.

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APPENDIX 1: RECURSION EQUATIONS FOR THE NON-SPATIAL MODEL VERSION

Provided that populations are large enough to be described deterministically, recursion equations for the non-spatial model version can be derived. The number of modules of genet i on sites of quality q ($q = $ g for good sites and $q = $ b for bad sites) is denoted by n_i^q.

1. *Environmental change*. Population sizes n_i^q change according to equations (1).
2. *Resource redistribution*. Resource supplies R_i^q are calculated according to equations (3).
3. *Reproduction*. Population sizes n_i^q change according to

$$n_i^q \mapsto n_i^q + \left(n^q - \sum_j n_j^q\right) \cdot \frac{n_i^g \cdot (R_i^g)^2 + n_i^b \cdot (R_i^b)^2}{\sum_j [n_j^g \cdot (R_j^g)^2 + n_j^b \cdot (R_j^b)^2]} \tag{A1a}$$

where the summation extends over all genets. The expression in parentheses is the number of empty sites with quality q, and the subsequent fraction follows directly from equation (4). Note that in this

step all empty sites become occupied. Equation (A1a) simplifies for pairwise invisibility analyses, when a rare mutant genet competes against a resident genet. Given the equilibrium population sizes n_r^q of the resident, the population sizes n_m^q of the rare mutant change according to

$$n_m^q \mapsto n_m^q + (n^q - n_r^q) \cdot \frac{n_m^g \cdot (R_m^g)^2 + n_m^b \cdot (R_m^b)^2}{n_r^g \cdot (R_r^g)^2 + n_r^b \cdot (R_r^b)^2} \qquad \text{(A1b)}$$

4. *Resource redistribution.* Resource supplies R_i^q are again calculated according to equations (3).
5. *Survival.* Population sizes n_i^q change according to

$$n_i^q \mapsto R_i^q \cdot n_i^q \qquad \text{(A2)}$$

The recursion equations for the non-spatial model version are thus fully established.

To study the effects of habitat bias, it is instructive to reformulate the recursion equations for a mutant genet in terms of the mutant's population-level averages of fecundity and survival. The change of the total mutant population size $n_m = n_m^g + n_m^b$ during a time step is

$$n_m \mapsto S_m \cdot (1 + F_m) \cdot n_m \qquad \text{(A3a)}$$

where F_m is the mutant's average effective fecundity (involving both offspring production and establishment) and S_m is the mutant's average survival probability. The latter can be calculated from the proportion $p_m^{b.s.}$ of mutant modules that are situated on good sites before the survival step:

$$S_m = p_m^{b.s.} \cdot R_m^g + (1 - p_m^{b.s.}) \cdot R_m^b = p_m^{b.s.} \cdot (1 - x_m + p_m^{b.s.} x_m) + (1 - p_m^{b.s.}) \cdot (p_m^{b.s.} x_m) = p_m^{b.s.} \qquad \text{(A3b)}$$

Therefore, the average survival probability of (adult) modules does not depend directly on the mutant's integration strategy, but only on the proportion of mutant modules on good sites. (A similar argument leads to the conclusion that, for the resident population, effectively being alone, the average survival probability is p, and thus equals the proportion of resident modules on good sites.) In other words, redistribution of the resource does not affect the average survival probability of modules. However, it does affect the average effective fecundity:

$$F_m \propto p_m \cdot (R_m^g)^2 + (1 - p_m) \cdot (R_m^b)^2 = p_m \cdot (1 - x_m + p_m x_m)^2 + (1 - p_m) \cdot (p_m x_m)^2 \qquad \text{(A3c)}$$

where p_m denotes, as in the main text, the proportion of mutant modules on good sites before reproduction. In equation (A3c), we only consider the numerator of equation (A1b), since the denominator does not depend on the mutant's integration strategy. From this we obtain

$$\frac{dF_m}{dx_m} \propto -2p_m (1 - p_m)(1 - x_m) \qquad \text{(A4)}$$

which shows that resource redistribution via integration has an adverse effect on the population-level average of effective mutant fecundity. This effect vanishes near full integration, $x_m = 1$, as well as near $p_m = 0$ and $p_m = 1$.

APPENDIX 2: IMPLEMENTATION DETAILS

The process of competition between different mutant–resident pairs (varying x_m and x_r) was studied in different environments (varying p and c). For each individual pairwise invasibility plot (PIP), x_r and x_m were independently increased from 0 to 1 (in steps of 0.01 in the non-spatial and 0.1 in the spatial version). For Figs 5 and 7, p changed from 0 to 1 and c from 0 to 2 (in steps of 0.01 in the non-spatial and 0.1 in the spatial version). For the spatial version, PIPs for many additional combinations of p and c were established to accurately identify the bifurcation curves shown in Fig. 7. The lattice size for the cellular automaton was set to 100×100 sites and the boundary condition was periodic.

To obtain an individual PIP at fixed values of p and c, the occupation of good and bad sites by mutant and resident modules was tracked over time for all combinations of x_r and x_m. Each simulation was initialized with a 10% occupation by the resident genet, placing the initial modules only into good sites:

$$n_r^g(0) = p/10 \tag{A5a}$$

$$n_r^b(0) = (1-p)/10 \tag{A5b}$$

In the spatial version, the sites thus occupied were chosen randomly. For a duration of 100 time steps, the resident population was then allowed to equilibrate. After that, a mutant genet was introduced, again with an initial occupation of 10%:

$$n_m^g(100) = p/10 \tag{A6a}$$

$$n_m^b(100) = (1-p)/10 \tag{A6b}$$

Sites for mutant occupation were chosen independently of their previous occupation (empty or occupied by a resident module). In the spatial version, sites occupied by the mutant were chosen within a square (the initial number of mutant modules was thus truncated to a square number). Simulations were stopped at time 300. The 100 time steps allowed for the resident dynamics and the 200 time steps for the mutant-resident dynamics were chosen to ensure essentially complete equilibration under all conditions. For the deterministically behaving non-spatial version, a single simulation at each parameter combination was sufficient, whereas for the spatial version, 200 replications were carried out and averaged for each parameter combination to account for the effects of demographic stochasticity.

In the non-spatial version, changes of the population sizes of mutant and resident genets were strictly monotonous after the establishment of an equilibrium distribution of mutant modules between good and bad sites. This monotony allowed for a direct estimation of invasion fitness. However, for the spatial version, characterizing the invasion success of a mutant in a resident population is not trivial because of the confounding effects of demographic stochasticity: simply calculating the difference between mutant and resident population sizes or growth rates did not give satisfactory results. We therefore compared the success of the mutant genet when competing against a resident genet with the success the mutant genet had when competing against a resident with exactly the same strategy. For this purpose, we first evaluated the change in the mutant-to-resident ratio between times 100 and 300:

$$\sigma_{x_m}|_{x_r} = \frac{n_m(300)}{n_r(300)} - \frac{n_m(100)}{n_r(100)} \tag{A7a}$$

A negative (positive) value of $\sigma_{x_m}|_{x_r}$ indicates a loss (gain) of mutants between the two measurements. In the absence of demographic stochasticity, we would have $\sigma_{x_m}|_{x_m} = 0$ (i.e. a rare mutant genet that competes against a resident genet with exactly the same integration strategy is neutral, and its popula-

tion size neither grows nor shrinks). However, in the presence of demographic stochasticity, the rare mutant genet is at an intrinsic disadvantage and is much more likely than the abundant resident genet to go extinct by chance effects. Therefore, $\sigma_{x_m}|_{x_m}$ does not vanish on a finite lattice (it tends to be negative) and we need to recalibrate the mutant's success against the neutral case,

$$s_{x_r}(x_m) = \sigma_{x_m}|_{x_r} - \sigma_{x_m}|_{x_m} \qquad\qquad (\text{A7b})$$

Based on this measure of invasion fitness, $s_{x_r}(x_m)$, we can conclude, both for the non-spatial and the spatial model versions, that the mutant can successfully invade the resident if $s_{x_r}(x_m)$ is positive.