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Cooperation and Competition in Heterogeneous Environments: The Evolution of Resource Sharing in Clonal Plants

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Abstract

Plant species show great variation in the degree of physiological integration between developmental units (modules). When this degree is minimal, individual modules are self-supporting and compete with other modules. When the degree of integration is higher, modules remain physiologically connected and "cooperate" by sharing resources like water, nutrients, and photoassimilates taken up from their local environments. In such a manner, local differences in habitat quality can be diminished within a group of modules. Here we investigate how the evolutionarily optimal degree 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 these. 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 suffices 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.

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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 broad sense, is 'any distinguishable, repeated and multicellular structural unit within a genet' (Vuorisalo and Tuomi 1986: 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 degree of physiological autonomy. In the extreme, each module is fully self-supporting and able to develop all plant organs (root and shoot, including generative shoot) needed 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 the degree of physiological integration (Jónsdóttir and Watson 1997) and in the morphological pattern of connections (Watson 1986; Marshall and Price 1997). For example, in typical 'splitter' clones the degree of 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 (like *Aster lanceolatus* Willd.). Here the modules are interconnected, but can regain autonomy after the damage of rhizome connections (Schmid and Bazzaz 1987). Other species (like *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 itself, meaning that available resources are equally shared between members of a genet is an idealization. Even typical non-clonal plants, like small-sized annuals, show some degree of sectoriality, resulting in restrictions to transport (Watson 1986; Vuorisalo and Hutchings 1996). Physiological processes of resource integration, and their implications for the performance of genets, have been studied by radioactive labeling and through manipulating resource supply to different parts of the plant. For excellent reviews about the differential degree of integration in 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 the degree of integration. For example, Wijesinghe and Whigham (2001) compared the response of three *Uvularia* species to patchy distribution of nutrients, and demonstrated 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 degree of integration, between conspecific populations sampled from different habitats. These studies suggest that the degree of physiological integration is an evolutionarily flexible trait, and allows for adaptation to prevailing habitat conditions.

In this study we focus on the selective forces driving the evolution of integration strategies. In the course of the investigation we will 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 favor (re)integration?
- Are there circumstances that specifically select for intermediate degrees of integration?

A primary reason for splitting, supported by broad empirical evidence, is that physiological autonomy helps spreading 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. For illustration of this point, consider a simple case of two connected modules. One module grows on a favorable site, the other experiences unfavorable conditions. When is it then advantageous for the genet that these modules share a limiting resource, as opposed to being physiologically autonomous? Clearly, the degree 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 favored. If, by contrast, the function is concave, sharing pays and complete integration is selected for. If the function is linear, the degree of integration is expected to be neutral. In short, unless 1+1 is more than 2 in fitness terms, we should not expect to see 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 neighbors; 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 have differential 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 nonlinear 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 evolutionarily adjustment of their integration strategies.

The aim of this study is to delineate salient environmental conditions facilitating evolutionary transitions from integration to splitting and vice versa. For this purpose we analyze 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, while the diversity of habitat qualities alone suffices to explain evolutionary transitions from integrated growth to splitting, spatial population structure is critical for convincingly explaining 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 favorable (good) and unfavorable (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), i.e., 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. In other words, modules of a splitter genet attain physiological autonomy after their establishment, whereas those of 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 to the modules' generation time (as supported by earlier empirical literature; see, e.g., 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 the degree of integration is zero, modules on good sites have a net export, while those on bad sites benefit by experiencing a net resource import.

The degree 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

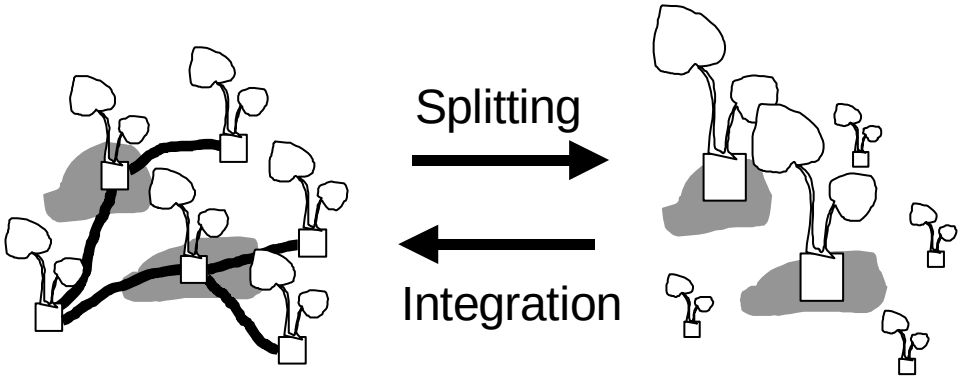

Figure 1 Implications of physiological integration and splitting for modules in resource-rich (grey) and poor (white) sites. In a complete integrator (left), modules equally share the available resource, resulting in equal chances for survival and reproduction. In a complete splitter (right), no resource 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 the optimal degree of resource sharing depends on the density and temporal constancy of rich sites.

basis, the invasion success of the mutant genet against the resident genet can be evaluated. This allows assessing the outcome of the evolutionary process resulting from successive successful invasions and to investigate 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 (Figure 2). The spatial version is implemented as a two-dimensional cellular automaton on a square lattice with von Neumann neighborhood (involving the next four neighbors of a site). Time is discrete and updating is synchronous. By contrast, in the non-spatial version, module growth is not restricted to next neighbors 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.

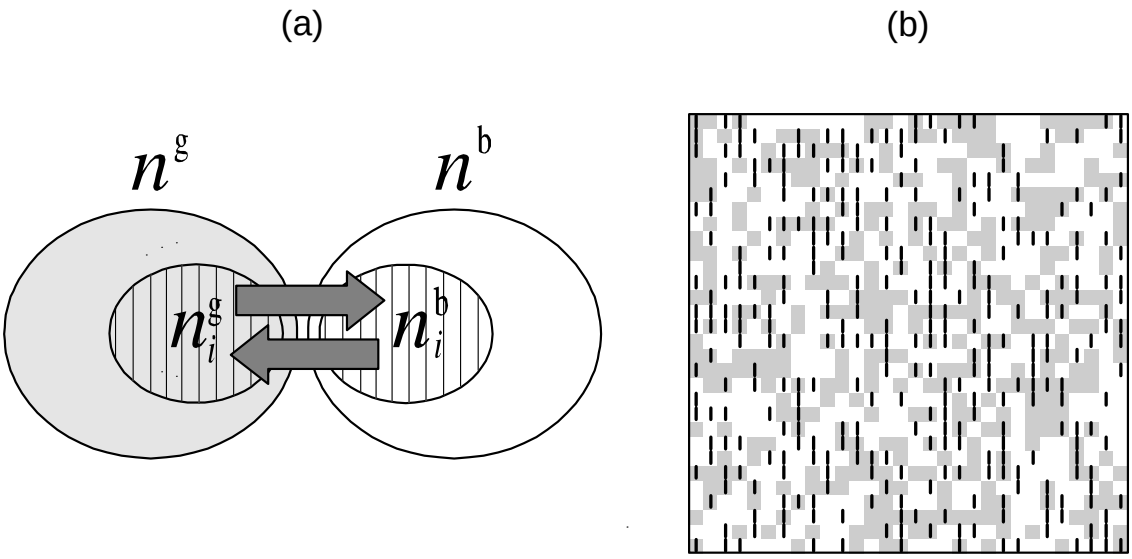

Figure 2 Schematic representation of states and state transitions in the (a) non-spatial and (b) spatial version of our plant population model. Sites of high habitat quality (good sites) are shown in gray, and sites occupied by a module are indicated by vertical lines. While the spatial model operates 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.

Environmental change

In each time step, a site of good quality changes to 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, while in the non-spatial version only the entire sets of good and bad sites need to be considered (Figure 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,

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

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

The case $c^{\mathrm{g}} = c^{\mathrm{b}} = 0$ corresponds to a constant environment, while $c^{\mathrm{g}} = c^{\mathrm{b}} = 1$ corresponds to one in which habitat qualities are alternating deterministically. Between these extremes, the ratio of good sites converges to the equilibrium value $p = c^{\mathrm{b}}/\left(c^{\mathrm{g}} + c^{\mathrm{b}}\right)$. We use p (characterizing habitat quality by the probability of a site to be of good quality) together with $c = c^{\mathrm{g}} + c^{\mathrm{b}}$ (characterizing habitat variability by the

speed of environmental change) as the primary parameters of our model and express the transition probabilities c^{g} and c^{b} accordingly,

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

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

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

For $0 \le c < 1$, 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 $1 < c \le 2$, 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 $0 \le x \le 1$ determines the fraction of the resource that a module shares with the other modules in its genet. Consider the *i*th 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 $x_i \cdot n_i^{\mathrm{g}} / (n_i^{\mathrm{g}} + n_i^{\mathrm{b}})$ and is equally shared 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 are given by

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

and

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

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

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

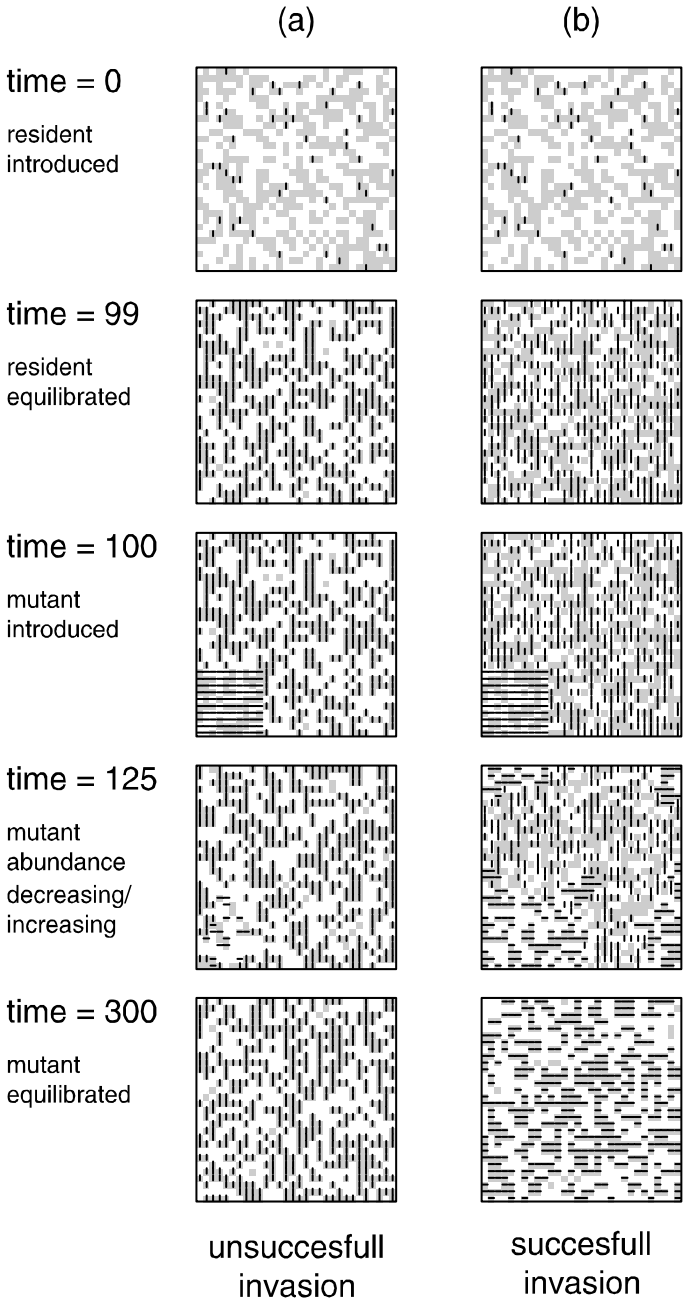

Figure 3 Spatial invasion dynamics of mutant integration strategies. Good sites are shown in gray. Vertical and horizontal lines indicate sites occupied by modules with resident and mutant integration strategies, respectively. (a) Unsuccessful invasion. At time $t = 0$, resident modules are introduced to a 10% fraction of randomly chosen sites, upon which their abundance and spatial structure has 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 have completely vanished until $t = 300$. Parameters: $p = 0.5$, $c = 0.1$, $x_{\mathrm{r}} = 0$, $x_{\mathrm{m}} = 0.5$. (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 until $t = 300$. Parameters: $p = 0.5$, $c = 0.1$, $x_{\mathrm{r}} = 1$, $x_{\mathrm{m}} = 0$. A reduced grid size of 30×30 has been chosen for the purpose of these illustrations.

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 neighborhood. In the spatial version, neighborhoods comprise of the four nearest neighbors of a site, whereas in the non-spatial version the neighborhood 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 neighborhood 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 neighborhood. The probability that the module on site $k \in N$ establishes its offspring on the empty site is then

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

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 seems plausible to us. Even though, we use the particular choice in Equation (4) only for illustrative purposes. Since this choice intrinsically favors the strategy $x = 0$, it renders conspicuous the effects of selection pressures favoring 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 equaling 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 the definition of neighborhoods, and 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. Corresponding results are presented in Appendix 1. By contrast, 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 (Figure 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 have employed 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 $s_{x_{\mathrm{r}}}(x_{\mathrm{m}})$ 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 gives information that, for one-dimensional quantitative traits, can be conveniently compiled into so-called pairwise invasibility plots (PIPs), which depict the sign of $s_{x_{\mathrm{r}}}(x_{\mathrm{m}})$ 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 Figures 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, $s_{x_{\mathrm{r}}}(x_{\mathrm{r}}) = 0$. In each PIP, the main diagonal therefore separates regions of possible invasion success, $s_{x_{\mathrm{r}}}(x_{\mathrm{m}}) > 0$, from those of certain invasion failure, $s_{x_{\mathrm{r}}}(x_{\mathrm{m}}) < 0$. For a given resident strategy x_{r}, we can thus determine whether evolution favors a gradual increase or decrease of x_{r} by reading off from the PIP the sign of $s_{x_{\mathrm{r}}}(x_{\mathrm{m}})$ right above and below the main diagonal. In this way, PIPs allow inferring 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.

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 behavior: 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 have 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 allow for the perpetual coexistence 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, e.g., 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.

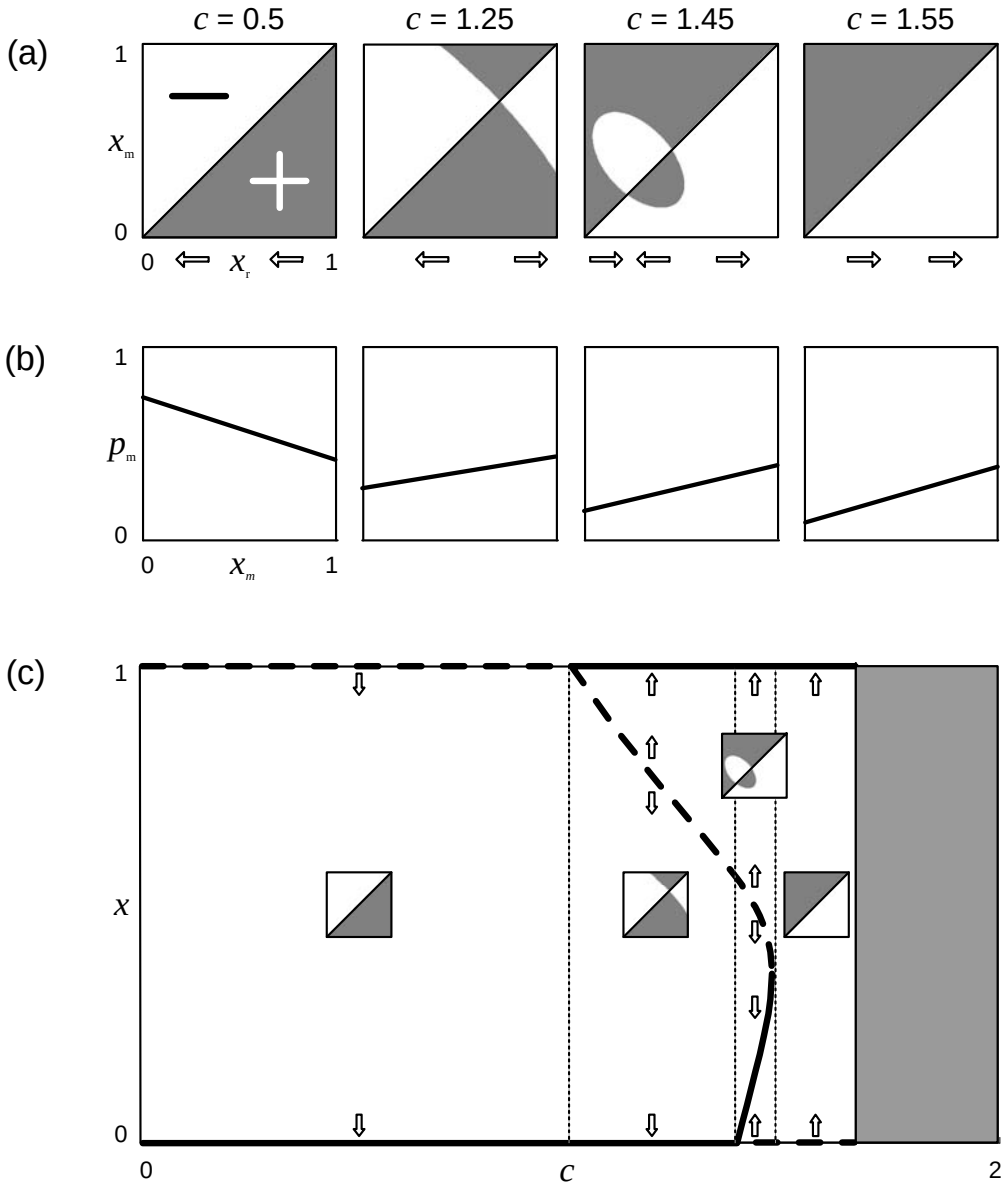

Figure 4 Illustration of evolutionary regimes in the non-spatial model version for a particular proportion of good sites, $p = 0.4$. (a) Pairwise invasibility plots arising for four different rates of environmental change, c. In each of these plots, combinations of resident integration strategies x_r and mutant integration strategies x_m for which the mutant is successful in invading and replacing the resident are shown in gray. 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 gray area to the right corresponds to values of c that are infeasible at $p = 0.4$.

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 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.

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 Figure 5d. Evolution therefore always proceeds toward splitting ($x = 0$). By increasing temporal variation such as to describe negatively autocorrelated environments ($1 < c \leq 2$), an evolutionary unstable internal repellor appears (Figure 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 (Figure 5c). An intermediate degree of integration is thus the expected evolutionary outcome if evolution starts to the left of the repellor, while starting to the right still results in complete integration. Finally, at extremely high temporal variation, both intermediate singular points collide and disappear, leaving complete integration as the only possible evolutionary outcome (Figure 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 Figure 5**,** bifurcation sequences for other values of p are either similar or simpler.

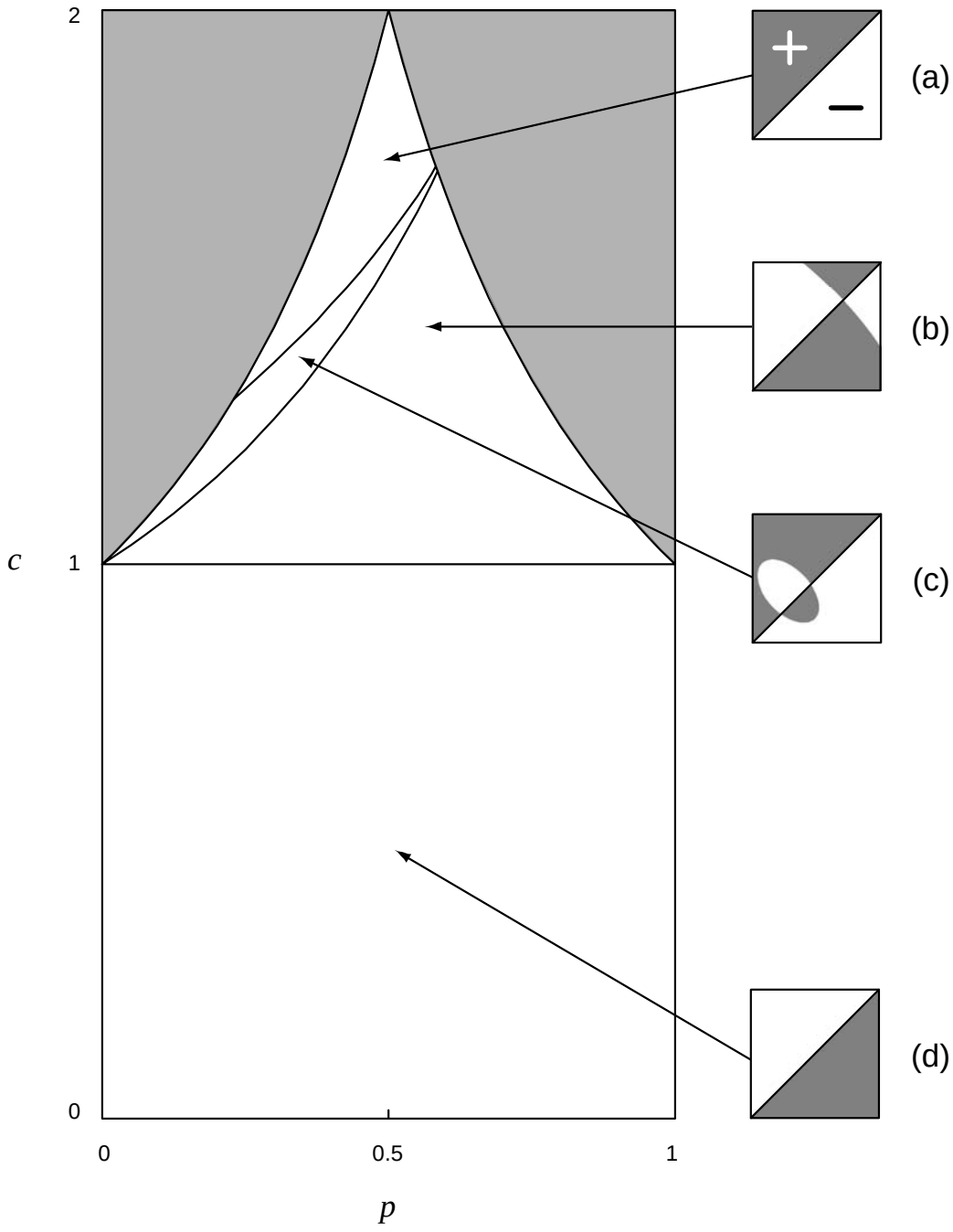

Figure 5 Overview of evolutionary regimes in the non-spatial model version, in dependence on habitat quality, p, and habitat variability, c. The two light gray areas on the top correspond to infeasible combinations of p and c. Altogether four evolutionary regimes are possible; however, for negatively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

Spatial version

Figures 6 and 7 summarize the results obtained for the spatial model version. Compared to the non-spatial version, a coarser resolution had to be chosen for the integration strategy in order to retain computational feasibility: Figure 7 is based on computing PIPs for 147 combinations of habitat quality p and habitat variability c. Each of these results from assessing the competitive outcomes of $11\times11=121$ 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 Figure 3, involving 300 time steps. Figure 7 thus required 1.067 billion time steps to be carried out on a lattice of $100\times100=10{,}000$ sites.

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

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

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

The two ancillary regimes depicted in Figures 7b and 7c do not play an important role; since fitness differences around $p=c=1$ are minute, the corresponding small parameter regions in Figure 7, despite massive numerical investment, cannot be demarcated with high accuracy. Compared with the non-spatial model version, the most striking feature of the spatial model version is the extended range of realistic environmental conditions that select for intermediate degrees of physiological integration (Figure 7d). Notice also that in positively autocorrelated environments higher quality can compensate for higher variability: intermediate levels of integration remain favored in highly variable environments if these at the same time offer habitat of high average quality.

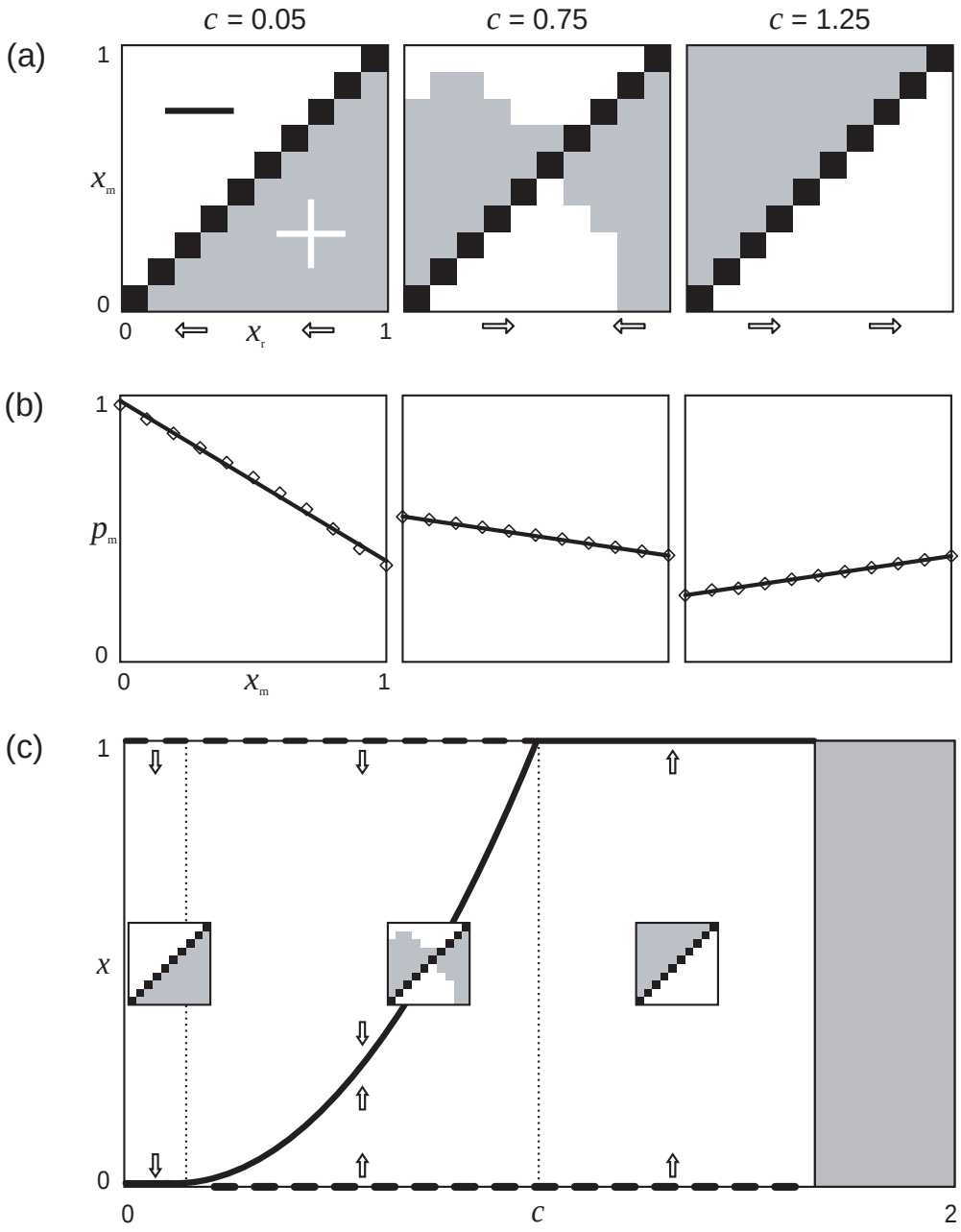

Figure 6 Illustration of evolutionary regimes in the spatial model version for a particular proportion of good sites, $p = 0.4$. Graphical features are as in Figure 4. (a) Pairwise invasibility plots arising for three different rates of environmental change, c. Due to demographic stochasticity, results 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. Notice that, in contrast to Figure 4, intermediate degrees of integration are favored for $c < 1$.

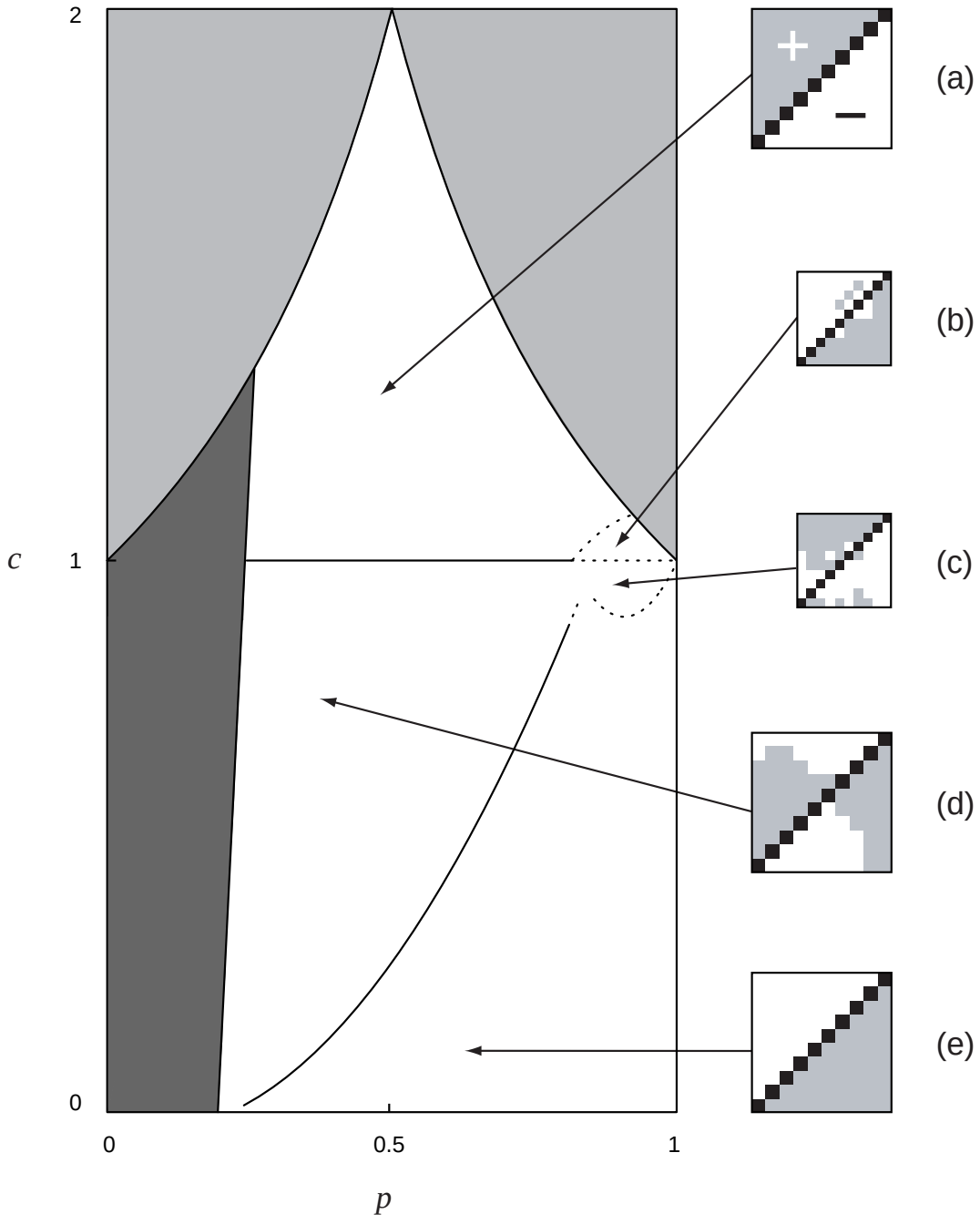

Figure 7 Overview of evolutionary regimes in the spatial model version in dependence on habitat quality, p, and habitat variability, c. Graphical features are as in Figure 5. The dark gray area to the left indicates combinations of p and c for which all resident integration strategies lead to extinction, 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 favor intermediate degrees of integration.

Figure 6c shows the bifurcation sequence of the spatial model version at $p = 0.4$. For very low levels of 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 favored under such conditions. For environments with more variability, this attractor departs from the boundary $x = 0$ and leaves behind an evolutionary repellor. Further increasing the temporal variability, the attractor gradually moves from $x = 0$ toward $x = 1$ 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 toward understanding the results described above, we study 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 favored by a bias toward good sites, whereas for $p_m < p$ mutant modules are biased toward bad sites. The habitat bias $p_m - p$ therefore serves as a convenient measure of module-environment correlation: only for $p_m - p = 0$, a site's habitat quality and its occupation by the mutant are uncorrelated.

Figures 4b and 6b show the dependence of p_m on the mutant integration strategy for the different evolutionary regimes occurring, 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 versed in negatively autocorrelated environments ($c > 1$): now high integration rates promote more favorable 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 behavior 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 by reference to three fundamental mechanisms that impose selection pressures on integration strategies:

1. Nonlinear 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 utilize them for explaining the outcomes of integration evolution found above for various environmental conditions.

As we have already highlighted in the Introduction, the potential nonlinearity 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 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 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 toward 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 favors 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 favors 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 toward relatively

resource-rich sites have been explicitly measured in studies on plant foraging (Sutherland, 1990; Hutchings and de Kroon, 1994; Oborny et al., 2001).
The following relations help assessing the interplay of Effects 1 and 2 as described above:

A. Effect 1 gradually weakens toward, 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 (Figures 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 toward 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 favors 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 degree 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 toward 1 reduces the range where Effect 2 dominates (Relation C), so that the position of the repellor converges to $x = 1$ (Figure 4c).
For a narrow range of c in Figure 4c, also an internal evolutionary attractor can 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. Notice that this range is located at $c > 1$: the proportion of good sites change into bad sites within one time step thus is 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 degrees of 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 toward 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 toward $c = 1$, the attractor approaches the boundary value $x = 0$ (Figure 4c). In the non-spatial model version, evolutionary convergence toward intermediate integration strategies thus is 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 Figure 4c). At $x = 1$ Effect 1 vanishes completely (Relation A) and the direction of evolution is determined solely by Effect 2. Habitat bias favors 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 Figure 5 $c = 1$ therefore separates the region $c > 1$ in which evolution locally converges toward full integration and the region $c < 1$ in which $x = 1$ is repelling.

The slopes of the boundary lines between the regions characterized by Figures 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 $p_m - p$ 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 Figure 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 for a donor and a recipient module are identical. Only Effect 2 remains, which implies that complete splitting is favored 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 favor 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 of 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 $c < 1$ (Figures 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 that, 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 toward integration.

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

D. Effect 3 gradually weakens toward full integration, $x = 1$, as the difficulty of spreading through unsuitable habitat vanishes when the differences 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 Figures 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 Figure 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 toward decreasing integration. These antagonistic effects give rise to an internal attractor for a rather broad range of parameter combinations (Figure 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 toward full splitting (Figure 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 degree of physiological integration. To our knowledge, this is the first study that allows for a continuum of integration strategies (rather than considering only two extreme types) and that systematically evaluates how environmental conditions affect gradual evolutionary change in these strategies. Salient environmental factors have been analyzed, 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: Nonlinear 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 nonlinear 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 degree 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 neighboring 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, nonlinear 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), does not suffice 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 suffice 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 to 3 allows us to understand the evolutionary outcomes observed in the spatial model version examined in this paper.

We must 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 Figures 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 degrees 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

In order to better appreciate the finding just summarized it is interesting 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 timescale 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 to 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 employed a convex utilization function of quadratic shape. Assuming 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 seemed to us like a plausible minimal assumption. Yet, many other function shapes can reasonably be

considered. In particular, situations in which 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 seem unlikely. Notice 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, at the present stage of empirical knowledge we essentially have to remain agnostic about their particular shape. While 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 favorable 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 favored in typical high-risk environments.

This is consistent with the hypothesis, frequently suggested in the empirical literature, that integration helps buffering 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 Figure 7, selection for full integration still ceases for particular combinations of average habitat quality and stability. This underlines 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. Analyzing 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 analyzed 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 hours. On average, the tracer diffused through 28 ramet generations (with a maximum of 48 generations), and traveled more than 2 meters (with a maximum of 4 meters). 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 the 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: Wijesinghe and Whigham 2001; Herben and Suzuki 2002; Hutchings and de Kroon 1994), and could thus interfere with the results presented here. The potentially intricate interactions between these separate effects are not yet understood in any generality. Clearly, such investigations must remain a challenging target 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 nicely 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 favoring resource sharing can occur when modules critically depend on more than one resource (Chesson and Peterson 2002; see also Stuefer and Hutchings 1994; Stuefer et al. 1994; Stuefer 1996; Alpert and Stuefer 1997; and Hutching et al. 2000 about reciprocal translocation of limiting resources). Whereas such considerations clearly are 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, like storage, as 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 (Mogie and Hutchings 1990; de Kroon and van Groenendael 1990; Klimeš et al. 1997; Sachs 2002). Instead, clonal growth seems to be an evolutionarily flexible trait, which has appeared, disappeared, and probably sometimes re-appeared, on several branches on 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 for enabling the lateral colonization of neighborhoods, we can expect evolutionary transitions from aclonal to clonal growth.

Appendices

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 ($q = \mathrm{g}$ for good sites and $q = \mathrm{b}$ 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

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

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). Notice 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

$$n_{\mathrm{m}}^q \mapsto n_{\mathrm{m}}^q + \left(n^q - n_{\mathrm{r}}^q \right) \cdot \frac{n_{\mathrm{m}}^{\mathrm{g}} \cdot (R_{\mathrm{m}}^{\mathrm{g}})^2 + n_{\mathrm{m}}^{\mathrm{b}} \cdot (R_{\mathrm{m}}^{\mathrm{b}})^2}{n_{\mathrm{r}}^{\mathrm{g}} \cdot (R_{\mathrm{r}}^{\mathrm{g}})^2 + n_{\mathrm{r}}^{\mathrm{b}} \cdot (R_{\mathrm{r}}^{\mathrm{b}})^2} \ . \tag{A1b}$$

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

$$n_i^q \mapsto R_i^q \cdot n_i^q \ . \tag{A2}$$

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 $n_{\mathrm{m}} = n_{\mathrm{m}}^{\mathrm{g}} + n_{\mathrm{m}}^{\mathrm{b}}$ during a time step is

$$n_{\mathrm{m}} \mapsto S_{\mathrm{m}} \cdot (1 + F_{\mathrm{m}}) \cdot n_{\mathrm{m}} \ , \tag{A3a}$$

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 $p_{\mathrm{m}}^{\mathrm{b.s.}}$ of mutant modules that are situated on good sites before the survival step,

$$S_{\mathrm{m}} = p_{\mathrm{m}}^{\mathrm{b.s.}} \cdot R_{\mathrm{m}}^{\mathrm{g}} + (1 - p_{\mathrm{m}}^{\mathrm{b.s.}}) \cdot R_{\mathrm{m}}^{\mathrm{b}} = p_{\mathrm{m}}^{\mathrm{b.s.}} \cdot (1 - x_{\mathrm{m}} + p_{\mathrm{m}}^{\mathrm{b.s.}} x_{\mathrm{m}}) + (1 - p_{\mathrm{m}}^{\mathrm{b.s.}}) \cdot (p_{\mathrm{m}}^{\mathrm{b.s.}} x_{\mathrm{m}}) = p_{\mathrm{m}}^{\mathrm{b.s.}} \quad . \qquad \text{(A3b)}$$

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,

$$F_{\mathrm{m}} \propto p_{\mathrm{m}} \cdot (R_{\mathrm{m}}^{\mathrm{g}})^2 + (1 - p_{\mathrm{m}}) \cdot (R_{\mathrm{m}}^{\mathrm{b}})^2 = p_{\mathrm{m}} \cdot (1 - x_{\mathrm{m}} + p_{\mathrm{m}} x_{\mathrm{m}})^2 + (1 - p_{\mathrm{m}}) \cdot (p_{\mathrm{m}} x_{\mathrm{m}})^2 \ , \qquad \text{(A3c)}$$

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

$$\frac{\mathrm{d}\, F_{\mathrm{m}}}{\mathrm{d}\, x_{\mathrm{m}}} \propto -2 p_{\mathrm{m}} (1 - p_{\mathrm{m}})(1 - x_{\mathrm{m}}) \ , \qquad \text{(A4)}$$

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, $x_{\mathrm{m}} = 1$, as well as near $p_{\mathrm{m}} = 0$ and $p_{\mathrm{m}} = 1$.

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 Figures 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 Figure 7. The lattice size for the cellular automaton was set to 100×100 sites, and 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.

$$n_{\mathrm{r}}^{g}(0) = p/10\,, \tag{A5a}$$

$$n_{r}^{b}(100) = (1-p)/10\,. \tag{A5b}$$

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,

$$\sigma_{x_{\mathrm{m}}|x_{\mathrm{r}}} = \frac{n_{\mathrm{m}}(300)}{n_{\mathrm{r}}(300)} - \frac{n_{\mathrm{m}}(100)}{n_{\mathrm{r}}(100)}\,. \tag{A7a}$$

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

$$s_{x_r}(x_m) = \sigma_{x_m|x_r} - \sigma_{x_m|x_m} \quad . \tag{A7b}$$

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

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