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Original Research Article

Spatial pattern as an adaptive phenotype

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ABSTRACT

Spatial patterns are common in nature across a broad range of scales, from body coloration patterns of animals to clustering of vegetation. The ultimate causes of these patterns are viewed very differently depending on whether they are traits of individuals or properties of aggregations. Traits of individuals are usually considered to be shaped directly by selection, while patterns of aggregation are typically viewed as incidental side effects of some other underlying processes or environmental heterogeneity. However, given the powerful influence that spatial structure can have on the susceptibility of a population to a dispersal-limited predator or pathogen, it may be useful to consider the possibility that spatial structure per se could serve as an anti-enemy adaptive phenotype. This group-level trait could evolve only if selection at the individual level does not overwhelm higher-level selection. To explore the plausibility of spatial structure as an adaptive phenotype, I consider the specific case of a spatially-explicit, evolutionary host–pathogen model. This model demonstrates the evolution of reproductive restraint, resulting in a low-density, poorly-connected landscape of host clusters that is resistant to the spread of the pathogen. Reimagining spatial structure as an adaptive phenotype may generate new insights of both theoretical and practical significance.

1. Introduction

Spatial patterns are ubiquitous in nature. Some, such as clumps of trees in a forest, patches of grass in a prairie, or clusters of individuals in a mussel bed, are formed by the aggregation of organisms. Others, like spots on a peacock butterfly or coat patterns of striped possums, are characteristics of individuals.

This distinction, between traits of individuals and properties of groups, profoundly shapes how we typically view the proximate and ultimate causes of these spatial patterns. The proximate causes of the spatial distributions of organisms are often thought to be some underlying environmental heterogeneity. For example, the change in vegetation patterns moving from timberline on a mountain to the valley below is usually considered to be a consequence of changes in temperature, soil type, moisture availability, etc. (Whittaker and Niering, 1975). This conventional view has been supplemented more recently by an appreciation for the role of self-organization, whereby local interactions give rise to emergent patterns at larger spatial scales (Bascompte and Solé, 1998; Klausmeier, 1999; Pascual et al., 2002; Rohani et al., 1997; Vandermeer et al., 2008). A now-classic example of this phenomenon is the formation of clumped patterns of vegetation in semi-arid ecosystems via the joint actions of local facilitation and long-distance competition for water resources (Rietkerk and van de Koppel, 2008). Self-organization is also proposed as the proximate cause of certain individual traits, e.g., the formation of pigmentation patterns on the bodies of animals through the diffusion of chemicals, as first developed theoretically by Turing (1952).

While there is some overlap in how we typically conceive the proximate causes of group-level and individual-level spatial patterns, the ultimate causes are almost always thought of very differently. Clustering of sessile animals is normally considered to be an incidental outcome of some other process, whether abiotic (in the case of environmental heterogeneity), or biotic (in the case of self-organization). Spatial pattern in these contexts is a side effect, not a driving motivation. In contrast, spatial patterns that are traits of individuals are usually seen as conferring some adaptive benefit, such as camouflage, aposematism, or distraction (Stevens, 2007). These spatial patterns are seen as having been shaped by the forces of natural selection; increased fitness is the ultimate cause.

The question I propose in the following is whether the spatial distribution of sessile organisms could be an adaptive phenotype. Could, for instance, the clumped distributions of vegetation that are observed so commonly in nature be actively selected for

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because they limit the ability of dispersal-limited predators and pathogens to spread through the populations, thereby increasing the fitness of the plants?

While it is clear how individuals arranged in a low-density landscape of isolated clumps might be protected from epidemics of pathogens or outbreaks of predators (Brown and Bolker, 2004; Ostfeld et al., 2005) – gaps between clumps would impede the spread of natural enemies much the same as firebreaks slow the progress of forest fires (Zinck and Grimm, 2009) – it is less clear that such a distribution could be favored by natural selection. For such a landscape of isolated patches to form, the expansion of clusters must be slow enough that the background mortality of individuals prevents the coalescence of clusters into larger clumps. This implies that individual reproduction is curtailed in the interest of the group-level distribution, which would ostensibly be opposed by natural selection on individual fitness.

This phenomenon, whereby individuals reduce their own rate of reproduction or spread such that the population as a whole benefits, has been termed "prudence" (Foitzik et al., 2001; Lion and Boots, 2010). The evolution of prudence has been demonstrated in spatially-explicit computer models of host–pathogen systems (Goodnight et al., 2008; Rand et al., 1995; Rauch et al., 2003; Sato et al., 1994). In these systems, pathogens evolve prudence in the sense that their transmissibility is less than a maximum value, preventing them from overexploiting the local supply of susceptibles. This result arises in spatially-explicit models, but prudence breaks down in the well-mixed, mean field model, as there is no local resource depletion experienced by pathogens that adopt a rapacious strategy of maximum transmissibility (Rand et al., 1995). More recently, prudence has been demonstrated in biological model systems (Boots and Mealor, 2007; Kerr et al., 2006; Szilágyi et al., 2009).

In the present work, I employ a spatially-explicit, evolutionary host–pathogen model to explore the hypothesis that the spatial distribution of hosts could be subject to evolutionary pressure, and that this pressure could lead to the evolution of prudent hosts that cooperate to form a low-density, poorly-connected landscape of clusters that is resistant to the spread of the pathogen. This question – whether clustering can drive process as opposed to being a byproduct of process – is a general one that may apply to many victim-exploiter systems comprised of sessile victims and dispersal-limited exploiters.

2. The model

The model is a discrete time, probabilistic cellular automata on a square lattice with periodic boundary conditions. Each cell in the lattice can be in one of three states: empty, occupied by a susceptible host, or occupied by an infected host. Time advances by synchronously updating the states of all cells in the lattice based on their own states and the states of their neighboring cells in the previous time step.

The life cycle of the pathogen is assumed to operate at a much faster time scale than that of the host, such that the host demographics are assumed to be static throughout the complete progression of an epidemic, from initiation to extinguishment. This is implemented by pausing all host activity while an epidemic is in progress. In each iteration of the model, either the pathogens will execute their actions, if there are pathogens present, or the hosts will execute their actions. This results in the pathogen life cycle being effectively instantaneous compared to the host life cycle; the hosts reproduce and die of natural causes as long as there are no pathogens present, but once a pathogen infects a single host, host activity is frozen while the pathogens sweep through the host population. Host activity resumes only after the epidemic runs its course and the last pathogen dies.

Host activity includes reproduction, death by natural causes, and pathogen-induced mortality. Reproduction of a susceptible (healthy) host, i, into an empty cell in its von Neumann neighborhood (its four nearest neighbors) occurs with probability g_i; infected hosts are unable to reproduce. Reproduction is strictly local, with no long-distance, i.e., global, dispersal. Each reproduction attempt is an independent event, meaning that a host surrounded by four empty cells can produce up to four offspring in a single time step. If multiple hosts attempt to reproduce into a single cell, the winner is chosen randomly. Death by natural causes occurs with a fixed probability, m. Pathogen-induced mortality is determined by the pathogen virulence, ν. In the current study, virulence is fixed at a probability of 1 unless otherwise noted, meaning that hosts only live for one time step after becoming infected. When ν is less than 1.0, the pathogen is cleared after one iteration if the host survives the infection.

Pathogen activity begins with an initial infection event that occurs with probability l. The initial infection targets a randomly-chosen host. The pathogen subsequently spreads via transmission to susceptible hosts in the von Neumann neighborhood of the infected host with probability τ. All transmission is local, with no long-distance dispersal. Unless otherwise noted, τ is fixed at 1 for all pathogens. Collisions, in which multiple pathogens attempt to infect a single host, are resolved by choosing a winner at random. As with host reproduction, transmission is determined independently for all of an infected host's susceptible neighbors, so an infected host with n susceptible neighbors can infect between 0 and n individuals.

Evolution occurs during host reproduction. When host i reproduces, its offspring normally inherit its reproduction probability, g_i. However, mutations of $\pm\in$ occur with probability μ. Therefore, the reproduction probability of offspring j of host i is defined as follows:

$$P(g_j = g_i) = 1 - \mu \tag{1}$$

$$P(g_j = g_i + \in) = \frac{\mu}{2} \tag{2}$$

$$P(g_j = g_i - \in) = \frac{\mu}{2} \tag{3}$$

The default parameter values used for all simulations, unless otherwise noted, are shown in Table 1.

Under this framework, the host spatial distribution emerges due to the interaction between the hosts' reproduction probabilities, g_i, the background mortality rate, m, and the intermittent removal of hosts by epidemics. Epidemics occur at random and then spread through the host population. If the hosts are distributed in a well-connected landscape, the pathogen will sweep through a large portion of the host population. If the hosts

Table 1
Default parameter values.

Parameter	Description	Default
g_i	Reproduction probability of host i	Variable
m	Baseline (natural) mortality rate	0.2
l	Probability of spontaneous infection	0.0016
ν	Virulence: mortality probability of infected host	1
τ	Probability of transmission to susceptible neighbor	1
μ	Probability of mutation of g_i	0.15
$\in$	Magnitude of mutation of g_i	0.01
X	Width of lattice (cells)	100
Y	Height of lattice (cells)	100
N_0	Initial host population size	500
P_0	Initial number of pathogens	50