Plant community structure is shaped by interactions among plants and their pathogens and mutualists (collectively called symbionts). Some symbionts are highly specialized, others are generalists, adding to the complexity of interactions. The development of a conceptual framework that is based on idealized models is a necessary step in reaching a level of understanding that would allow us to predict outcomes of these interactions. In this paper, we introduce spatially explicit, stochastic models of multispecies host-symbionts interactions with a specific focus on how the degree of specialization affects coexistence of multiple hosts and their symbionts, and, in case of coexistence, what spatial patterns result. Our rigorous results, supplemented by simulations, give a complete description of the behavior of these host-symbionts models. It is proved analytically that generalist symbionts have only a limited effect on the structure of host communities which, in contrast, can be significantly altered by specialist mutualists, with the most beneficial mutualist together with its most preferred hosts outcompeting all the other species. Numerical simulations further suggest that, in the neutral case, specialist pathogens promote the local diversity whereas specialist mutualists lead to a coarse-grained habitat. This paper also contains conjectures for future research directions.

Keywords Contact process; Epidemic models; Interacting particle systems in dynamic environments; Mutualist; Pathogen; Voter model.

Mathematics Subject Classification Primary 60K35, 82C22.

1. INTRODUCTION

Darwin conjures up the image of an “entangled bank” at the closing of his book on the Origin of Species where species of “elaborately
constructed forms, so different from each other, and dependent on each other in so complex a manner, have all been produced by laws acting around us.” He then enumerates the laws: “Growth with Reproduction,” “Inheritance,” and “Variability.” Competition, or, to use Darwin’s words, “Struggle for Life,” leads to “Natural Selection” and consequently, to “Divergence of Character” and “Extinction of less-improved forms.” Darwin’s work on pollination added another dimension to understanding this “entangled bank,” namely the role of species interactions.

Species interact with each other and their environment in diverse ways. These interactions result in a diversity of spatial patterns, from monospecific stands of coniferous trees in boreal forests to high-diversity tropical forests. Continuing with plants to illustrate the types of interactions species encounter, we note that plants compete for soil nutrients, water, and light. They often depend on other organisms, such as mycorrhizal fungi, insect pollinators, nitrogen-fixing bacteria, and their existence might be threatened by pathogenic fungi, herbivorous insects, and viral or bacterial diseases. Organisms that have beneficial effects are called mutualists, while organisms that harm others are called pathogens. Together, they are referred to as symbionts. It soon became clear that not only were there numerous types of interactions but also the degree of specialization varied considerably. Some symbionts are highly specialized, whereas others are generalists. Specialization seems to be the more common lifestyle, in particular among pathogens where extreme specialization is often observed.

In Refs.\textsuperscript{21,22}, we introduced idealized mathematical models of multi-host and multi-symbiont interactions to aid in the understanding of this “entangled bank.” We considered both a static host environment\textsuperscript{22} and a dynamic host environment\textsuperscript{21}. The static host environment represents the permanent, spatial configuration of different host types, arranged in a checkerboard pattern. The size of host patches can vary and thus allows us to model both fine-grained and coarse-grained habitats. In this environment, we introduced symbionts (pathogens and mutualists) that differed in their degree of specialization. Our results showed that fine-grained habitats lead to generalist strategies, while coarse-grained habitats increased the competitiveness of specialists.

The dynamic host environment includes feedback between hosts and their symbionts. In Ref.\textsuperscript{21}, we compared a spatially explicit, stochastic model to its associated mean-field model (a non-spatial, deterministic version of the spatial model). The mean-field model pointed to a fundamental difference between mutualistic and pathogenic interactions. Whereas pathogenic interactions promoted diversity of multiple hosts, mutualistic interactions did not. In that paper, we also investigated the
spatial model and provided conditions for coexistence (or lack thereof) of multiple hosts as mediated by their associated symbionts.

In this paper, we introduce a stochastic model, which, similarly to the model in Ref.\cite{21}, describes the co-evolution of multiple hosts and symbionts in a dynamic host environment. The effects of the symbionts on their hosts was modeled in Ref.\cite{21} by a variation of the fertility of associated hosts whereas it is now modeled by a variation of their viability. Fertility and viability are two key life history traits that influence host competitiveness. The introduction of this model is also motivated by the fact that it is more tractable mathematically (duality techniques are available in the presence of generalist interactions) and leads to theoretical results stronger than in\cite{21}, while numerical simulations suggest that both models result in similar predictions. The main objective is to investigate how local interactions of multiple hosts and symbionts shape the spatial patterns of these communities. Host and symbiont dynamics are described by spatially explicit processes evolving simultaneously and interacting with each other. Parameters mimic both the effects of the symbionts on hosts (pathogen vs mutualist) and their strategy (generalist vs. specialist).

The number of types of hosts is denoted by $N_1$ while the number of types of symbionts is denoted by $N_2$. In the absence of symbionts, $N_2 = 0$, the host population evolves according to a voter model\cite{5,20}. In particular, the dynamics imply that each site remains occupied by a host at all times. Therefore, we assume that the number of host types $N_1 \geq 1$. When $N_2 \geq 1$, symbionts spread according to a (multitype) contact process\cite{18,26} with varying degrees of specificity and transmissibility. More precisely, our spatially explicit, stochastic model is a system of coupled interacting particle systems whose state at time $t$ is described by functions

$$h_t : \mathbb{Z}^d \rightarrow \{1, 2, \ldots, N_1\} \quad \text{and} \quad s_t : \mathbb{Z}^d \rightarrow \{0, 1, \ldots, N_2\}.$$  

The component $h_t$ describes the evolution of the host population, which represents the first trophic level, while the component $s_t$ describes the evolution of the symbiont population, which represents the second trophic level. Introducing the process $\eta_t = (h_t, s_t)$, the evolution of $\eta_t$ at site $x$ is described by

$$(i, j) \rightarrow (l, 0) \quad \text{at rate} \quad \delta_j \sum_{x \sim y} \mathbb{1}\{h(y) = l\} = \delta_s(x) \sum_{x \sim y} \mathbb{1}\{h(y) = l\}$$

$$(i, 0) \rightarrow (i, j) \quad \text{at rate} \quad \alpha_j \sum_{x \sim y} \mathbb{1}\{s(y) = j\} = \alpha_{h(x)} \sum_{x \sim y} \mathbb{1}\{s(y) = j\}$$

where $x \sim y$ indicates that $x$ and $y$ are nearest neighbors. Thinking of hosts and symbionts as evolving simultaneously on the same lattice, $\eta_t(x) =$
(i, j) indicates that site x is occupied by a host of type i associated with a symbiont of type j, with \( j = 0 \) denoting the absence of a symbiont, in which case the host is said to be unassociated. Note that, while \( \eta = (h, s) \) is a Markov process, its components \( h \) and \( s \) individually are not Markov since the transition rates of each component depend on both components. This is due to the fact that each trophic level evolves in a dynamic environment which is represented by the other trophic level: the symbiont component \( s \) shapes its host environment \( h \), which, in turns, shapes its symbiont environment, adding to the complexity of the interactions and the mathematical analysis of the model. The dynamics are divided into two parts corresponding to births of unassociated hosts and transmission of symbionts.

1. The transition \((i, j) \rightarrow (l, 0)\) is the birth of an unassociated host at \( x \) by either unassociated or associated neighboring hosts. To set the time scale, we assume that \( \delta_0 = 1 \) so that the death rate of unassociated hosts is equal to \( 2d \), while the one of hosts associated with a symbiont of type \( j = s(x) \) is equal to \( 2d\delta_j = 2d\delta_{s(x)} \). These parameters will allow us to mimic the effect of the symbionts on their hosts: the condition \( 0 \leq \delta_j < 1 \) indicates that symbiont \( j \) is a mutualist while the condition \( \delta_j > 1 \) indicates that it is a pathogen. Note that the host born at \( x \) is unassociated due to the fact that the symbiont is not passed to the offspring.

2. The transition \((i, 0) \rightarrow (i, j)\) is the transmission of a neighboring symbiont of type \( j \) to an unassociated host of type \( i \) at site \( x \). The parameters \( \alpha_{j \rightarrow i} = \alpha_{j \rightarrow h(x)} \) denote the rate at which symbiont \( j \) associates with host \( i = h(x) \). These parameters will allow us to mimic specialist and generalist symbionts.

We now investigate the spatially explicit, stochastic model in details. Recall first that the basic contact process is the Markov process whose state at time \( t \) is a function \( \zeta : \mathbb{Z}^d \rightarrow \{0, 1\} \) and whose evolution at site \( x \) is described by the transition rates

\[
0 \rightarrow 1 \text{ at rate } \beta \sum_{y \sim x} \zeta(y) \text{ and } 1 \rightarrow 0 \text{ at rate } 1.
\]

It is a well-known fact that this process exhibits a phase transition, namely there exists a critical value that we later denote by \( \beta_c \in (0, \infty) \) such that the process converges to the “all 0” configuration when \( \beta \leq \beta_c \), whereas strong survival occurs when \( \beta > \beta_c \).
1.1. Generalist Interactions: Evolution of the Symbionts

In the presence of generalist interactions, i.e., when the transmission rates $\alpha_{j \rightarrow i}$ do not depend on the host type $i$, the symbionts evolve regardless of the structure of the host population. The number $N_1$ of hosts is then unimportant to understand the evolution of the symbiont component $s_i$, which reduces to a multitype contact process in a homogeneous environment$^{[26]}$. Note however that the evolution of the host population depends on the configuration of the symbionts and does not reduce to a voter model.

When $\alpha_{j \rightarrow i} = \delta_j > 0$ for $i = 1, 2, \ldots, N_1$ and $j = 1, 2, \ldots, N_2$, the process $s_i$ reduces to a multitype contact process, and symbiont of type $j$ may survive in the absence of the other symbionts if and only if $\alpha_j > 2d_\delta \beta_c^{[1,25]}$, which we assume from now on for all $j$ to avoid trivialities. When the death rates of associated hosts are equal, $\delta_1 = \cdots = \delta_{N_2}$, the symbiont with the largest transmission rate, say symbiont $j_0$, outcompetes the other symbionts in the sense that, starting from a translation invariant measure with a positive density of each symbiont type,

$$\liminf_{t \to \infty} P(s_t(x) = j_0) > 0 \quad \text{and} \quad \lim_{t \to \infty} P(s_t(x) = j) = 0 \quad \text{for all } 0 < j \neq j_0.$$

When the death rates are not equal, it is conjectured that the symbiont with the largest $\alpha_j / \delta_j$ ratio outcompetes the other symbionts$^{[26]}$. It can also be proved that, all the parameters being fixed but the transmission rate $\alpha_1$, there exists a critical value $\alpha_{\alpha} \in (0, \infty)$ such that symbionts of type 1 outcompete the other symbionts provided $\alpha_1 > \alpha_{\alpha}$. The proof is a two-step process: first, the particle system viewed on suitable space and time scales is compared with an oriented percolation process on $\mathbb{Z}^d \times \mathbb{Z}^+$ in the limiting case $\alpha_1 = \infty$, and second, the result is extended to the region where $\alpha_1$ is large but finite through a perturbation argument. Even if (as far as we know) this result has not been proved elsewhere, we omit the details of the proof since the main mathematical objective of this article is the study of interacting particle systems in dynamic environments. This type of stochastic process appears in our model when the evolution of the trophic level under consideration is indeed affected by the spatial structure of the other trophic level, which is not the case of the symbiont population in the presence of generalist interactions.

When $\alpha_{j \rightarrow i} = \alpha_j > 0$ and $\delta_j = 0$ for all $i = 1, 2, \ldots, N_1$ and $j = 1, 2, \ldots, N_2$, the process $s_i$ reduces to the multitype Richardson model$^{[16]}$. Provided one starts with at least one associated host, fixation occurs for both the host and the symbiont components since the dynamics of associated hosts are frozen. The multitype Richardson model has been investigated in the case of two symbionts competing for space, $N_2 = 2$. We call mutual unbounded growth the event $A$ that the set of hosts associated
with a symbiont of type 1 and the set of hosts associated with a symbiont of type 2 both keep growing indefinitely. The combination of the results in Refs.\cite{10,15-17,19} shows that in any dimension $d \geq 2$ the process behaves as follows: if the transmission parameter $x_2 > 0$ is fixed, then starting from any initial configuration with a finite number of symbionts and in which none of the symbionts “surrounds” the other one (see Ref.\cite{10} for a precise definition of surrounding),

1. $P(A) > 0$ when $x_1 = x_2$ whereas
2. $P(A) = 0$ for all but at most countably many choices of $x_1$.

It is conjectured that $P(A) > 0$ if and only if $x_1 = x_2$, and that this conjecture can be extended to any finite value of the number of symbiont types, improving 1 and 2 above.

1.2. Generalist Interactions: Evolution of the Hosts

While in the case of generalist interactions the symbiont component $s_i$ reduces to well-known stochastic processes (multitype contact process and multitype Richardson model), the host component $h_i$ is an interacting particle system evolving in dynamic environment. It can be seen as a modification of the voter model in which the death rate at each site is a function of the spatial configuration of the symbionts which itself evolves stochastically. Our analysis reveals that generalist symbionts have only a limited effect on the structure of their habitat. More precisely, when all the symbionts are generalist, the long-term behavior of the host component $h_i$ is similar to that of the voter model in the sense that clustering occurs in $d \leq 2$ while hosts may coexist in $d \geq 3$, just as in the absence of symbionts. Note however that the evolution of the spatial structure of the host population differs from that of the voter model, as the mean cluster size strongly depends on the death rates $\delta_j$ of associated hosts. Indeed, in the presence of one generalist symbiont, at least when the transmission rate is large, the host component behaves nearly like a time-change of the voter model in which hosts update their type at rate $2d\delta_1$. This suggests that in $d \leq 2$ generalist pathogens speed up the clustering of the hosts whereas generalist mutualists slow down the clustering of the hosts, as indicated by the simulation pictures of Figure 1. The effect of generalist symbionts is to change the mean cluster size but not the typical geometry of the clusters. In particular, one still has diffusive clustering in $d = 2$, which strongly contrasts with the behavior of the process in the presence of specialist symbionts discussed below: specialist pathogens promote coexistence of the hosts in any dimension, whereas specialist mutualists lead to a coarse-grained habitat in which clusters have sharp boundaries.
Host-Pathogen and Host-Mutualist Interactions

FIGURE 1 Simulation pictures of the dynamic-host model on a 400×400 lattice with periodic boundary conditions at time 500. Host of type 1 is represented in black (associated) and dark grey (unassociated), and host of type 2 in pale grey (associated) and white (unassociated). The left picture shows the process with a generalist pathogen and the right picture the process with a generalist mutualist. The simulations indicate that generalists do not affect the structure of their habitat: the geometry of the clusters is similar to that of the voter model.

Theorem 1.1. Assume that \( \alpha_j \rightarrow i = \alpha_j \) and \( \delta_j > 0 \) for \( i = 1, 2, \ldots, N_1 \) and \( j = 1, 2, \ldots, N_2 \). Then

a. In \( d \leq 2 \) clustering of hosts occurs, i.e., for any initial configuration \( \eta_0 \), we have

\[
\lim_{t \to \infty} P(h_t(x) \neq h_t(y)) = 0 \quad \text{for all } x, y \in \mathbb{Z}^d.
\]

b. In \( d \geq 3 \) coexistence of hosts occurs, i.e., there is a translation invariant stationary distribution with a positive density of each host type.

The proof is based on duality techniques, though the coevolutionary process \( \eta_t \) does not have a dual process. The basic idea is to use the fact that, in the presence of generalist interactions, the evolution of the symbionts does not depend on the spatial structure of the host population. This allows us to first run the symbiont process \( s_t \) up to some time \( T \), and then, given a realization of the symbiont component, to define a dual process for the host component \( h_t \). This dual process consists of a collection of coalescing random walks in heterogeneous environment run at different rates, namely \( \delta_0, \delta_1, \ldots, \delta_{N_2} \), depending on the background configuration of the symbionts.
1.3. Specialist Interactions: Effect of a Single Symbiont

The next step is to look at the effects of a single specialist symbiont in terms of structure and diversity of the habitat. Note that in the presence of specialist interactions the coevolutionary process $\eta_t$ becomes more complicated because both its host component $h_t$ and its symbiont component $s_t$ consist of particle systems in dynamic environments. While the long-term behavior of the host population in the presence of generalist interactions is similar to that of the voter model, we now prove that the long-term behavior of the host component in the presence of a single specialist mutualist is similar to that of a biased voter model in which the preferred host has a selective advantage. Note again that the process $h_t$ is much more complicated than the biased voter model and that our result does not follow from the analogous result for the biased voter model. This is due to the fact that only a fraction of the preferred hosts are associated with their symbionts which, in the case of specialist interactions, evolve according to a process stochastically smaller than the contact process. The fact that the density of symbionts shrinks at the boundary of their habitat (the preferred host) is another difficulty.

**Theorem 1.2.** Assume that $N_2 = 1$, $\alpha_{1,i} = 0$ for all $i \neq 1$ and $\delta_1 < 1$.

a. In $d \geq 1$, there exists a critical value $\alpha_{c,1} \in (0, \infty)$ such that if $\alpha_{1,1} > \alpha_{c,1}$, then, starting from a Bernoulli product measure with a positive density of associated hosts of type 1,

$$\lim_{t \to \infty} P(h_t(x) = 1) = 1 \quad \text{and} \quad \lim \inf_{t \to \infty} P(s_t(x) = 1) > 0.$$ 

b. In $d = 1$, $\alpha_{c,1} = 2\delta_1 \beta_c$, and furthermore, symbionts go extinct if $\alpha_{1,1} \leq 2\delta_1 \beta_c$.

Part (a) will be generalized in Theorem 1.5 below. The idea is to observe that in the limiting case $\alpha_{1,1} = \infty$, the evolution of the host population is described by a biased voter model in which host of type 1 has a larger birth rate than the other hosts, and then apply a perturbation argument to prove that $\alpha_{1,1}$ large but finite does not change significantly the evolution of the process. In part (b), we give a necessary and sufficient condition for the survival of the mutualist and its preferred host in one dimension, which is the same condition as for the corresponding contact process. Note that this is not trivial since specialist symbionts only have a restricted habitat that evolves stochastically, and that the conclusion differs if the symbiont is not a mutualist (see our next result below). The idea of the proof is to let $f_0 = (-N, N]$, and start the process with hosts of type 1 associated with their mutualists in $f_0$ and other host types outside $f_0$. First, a comparison with the voter model implies that, regardless of the evolution
of the mutualists, the extinction time of hosts of type 1 is larger than a constant times $N^2$. Second, the number of times a mutualist associates with the rightmost or leftmost preferred host (we call this event a collision) in $N$ units of time is larger than a constant times $N$. Since the death rate $\delta_1 < 1$, this results in a linear expansion of the interval occupied by hosts of type 1 and their mutualists. The proof is made rigorous by comparing the process viewed on suitable space and time scales with a percolation process.

By using similar estimates on the mean number of collision events and assuming that the specialist is now a pathogen, we find that host of type 1 goes extinct when starting with a half-line occupied by associated hosts of type 1 and other host types outside this half-line.

**Theorem 1.3.** Assume that $d = 1$, $N_2 = 1$, $\alpha_{1-i,i} = 0$ for all $i \neq 1$ and $\delta_1 > 1$. If $\alpha_{1-1} > 2\delta_1\beta_c$, then, starting the one-dimensional process from any initial configuration with

$$h_0(x) = 1 \text{ if and only if } x \leq 0 \quad \text{and} \quad s_0(x) = 1 \text{ if and only if } x \leq 0,$$

we have, for any $x \in \mathbb{Z}^d$, $\lim_{t \to \infty} P(h_t(x) = 1) = \lim_{t \to \infty} P(s_t(x) = 1) = 0$.

More generally, Theorem 1.3 holds when starting with the same initial configuration of hosts and infinitely many hosts of type 1 associated with a pathogen. This, together with the fact that $d = 1$ and $\alpha_{1-1} > 2\delta_1\beta_c$, implies that, at any time, the set of sites occupied by hosts of type 1 is connex and contains infinitely many pathogens, which insures that, in any finite spatial box, host of type 1 goes extinct eventually. In higher dimensions, however, the dynamics produce an increasing number of finite connex components of hosts of type 1 that may not be associated with a pathogen. This results in a system that evolves eventually according to a voter model in which each host type is present. The same holds in one dimension when starting from an initial configuration with infinitely many finite intervals occupied by hosts of type 1. In conclusion, we conjecture that Theorems 1.2 and 1.3 can be improved by

**Conjecture 1.4.** Assume that $N_2 = 1$ and $\alpha_{1-i,i} = 0$ for all $i \neq 1$. In any dimension,

a. If $\delta_1 < 1$ and $\alpha_{1-1} > 2d\delta_1\beta_c$, then, starting from a Bernoulli product measure with a positive density of associated hosts of type 1,

$$\lim_{t \to \infty} P(h_t(x) = 1) = 1 \quad \text{and} \quad \liminf_{t \to \infty} P(s_t(x) = 1) > 0.$$
b. If $\delta_1 > 1$ then, for any transmission rate $\alpha_{1 \to 1}$ and starting from a Bernoulli product measure with a positive density of hosts of type 1,

$$\lim_{t \to \infty} \inf P(h_t(x) = 1) > 0 \quad \text{and} \quad \lim_{t \to \infty} P(s_t(x) = 1) = 0.$$ 

In part (a), the rate $\alpha_{1 \to 1}$ is chosen in such a way that the mutualists evolve according to a supercritical contact process on their hosts so the mean number of collision events (see the definition above) should be large enough to expand the set of sites occupied by hosts of type 1. The main problem in proving the result in $d \geq 2$ is that the boundary of $\{x : h_t(x) = 1\}$ is much more complicated than in the one-dimensional case. In part (b), the main difficulty comes from the lack of monotonicity of the process $n_t$ due to pathogens destroying their habitat. Numerical simulations suggest that the evolution of the process can be divided into three steps. First, clusters of hosts of type 1 start shrinking due to the presence of pathogens. Second, after destroying most of their habitat, pathogens are unable to survive due to the lack of space available to spread. Third, the process looks like a sea of hosts of type $\neq 1$ scattered with small islands of unassociated hosts of type 1, the host component evolving according to a voter model (see Figure 2).

**FIGURE 2** Snapshots of the dynamic-host model starting with hosts of type 2 inside the dashed circle and hosts of type 1 outside. The color code is the same as in Figure 1. In the left picture, all the hosts of type 1 are initially associated with a specialist pathogen and hosts of type 2 are unassociated, while in the right picture, hosts of type 1 are unassociated and hosts of type 2 are associated with a specialist mutualist. The simulations suggest that, for the process with a single specialist, the preferred host outcompetes the other host types in the mutualistic case, while, due to extinction of the symbiont, the process behaves eventually like a voter model in the pathogenic case.
1.4. Mixed Interactions: Effect of Multiple Symbionts

Our next result states that a specialist mutualist together with its preferred host outcompetes all the other species provided it has a more beneficial effect than the other symbionts and its transmission rate is sufficiently large. Note that this generalizes part (a) of Theorem 1.2. Theorem 1.5 reduces to Theorem 1.2(a) in the case $N_2 = 1$.

Theorem 1.5. Assume that $\alpha_1 \rightarrow i = 0$ for all $i \neq 1$ and $\delta_1 < \min(\delta_0, \delta_2, \ldots, \delta_{N_2})$. Then there is a critical value $\alpha_c \in (0, \infty)$ such that if $\alpha_1 > \alpha_c$ then, starting from a Bernoulli product measure with a positive density of hosts of type 1 associated with mutualists of type 1,

$$\lim_{t \to \infty} P(h_t(x) = 1) = 1, \quad \liminf_{t \to \infty} P(s_t(x) = 1) > 0 \quad \text{and} \quad \lim_{t \to \infty} P(s_t(x) \geq 2) = 0.$$  

To conclude, we formulate a conjecture describing the models with two specialists having both the same effect (pathogen or mutualist) on their respective host, and the same transmission rates.

Conjecture 1.6. Assume that $N_1 = N_2 = 2$, $\alpha_{11} = \alpha_{22} = \alpha$ and $\alpha_{12} = \alpha_{21} = 0$

a. If $\delta_1 = \delta_2 < 1$ and $\alpha > 2d\delta_1\beta_1$, then clustering of hosts occurs, i.e., we have

$$\lim_{t \to \infty} P(h_t(x) \neq h_t(y)) = 0 \quad \text{for all } x, y \in \mathbb{Z}^d.$$  

b. If $\delta_1 = \delta_2 > 1$ and $\alpha$ is sufficiently large, then coexistence occurs in $d \geq 2$, i.e.,

$$\liminf_{t \to \infty} P(h_t(x) = i) > 0 \quad \text{and} \quad \liminf_{t \to \infty} P(s_t(x) = i) > 0 \quad \text{for } i = 1, 2.$$  

This conjecture is reminiscent of recent results of Cox and Perkins\cite{Cox1, Cox2} about the Lotka–Volterra model introduced in Ref.\cite{Lotka} as it suggests that small perturbations of the voter model may radically change its set of invariant measures. This has also been observed through spatial simulations for another particle system based on the voter model introduced by the authors\cite{Voter}. For our host-symbiont model, the mechanisms that lead to clustering when both symbionts are mutualists, and to coexistence when they are pathogens, are the following. In both cases, the basic idea is that specialists of either type are unable to survive on a small cluster of the preferred host. In the mutualistic case, clusters of hosts with their preferred mutualists form and appear to continue to grow, while, due to the absence of mutualists, small clusters disappear quickly. This then results in a clustering effect more pronounced than in the voter model (compare the pictures of Figure 2 with the right-hand side of Figure 3). In the pathogenic case, cluster size, on the contrary, is limited
FIGURE 3 Simulation pictures of the dynamic-host model in the presence of two specialists on a 400 × 400 lattice with periodic boundary conditions at time 500. The color code is the same as in Figure 1. In the left picture, both specialists are pathogens, while in the right picture, both specialists are mutualists. The simulations show that pathogens alter the spatial structure of plant communities, promoting local biodiversity, while mutualists lead to coarse-grained habitats more pronounced than in the voter model.

by the presence of pathogens: In the absence of pathogens, clusters grow at the expense of neighboring clusters that contain symbionts. Upon invasion by the preferred symbionts, the clusters appear to shrink again (see left-hand side of Figure 3). Durrett and Lanchier\cite{13} recently proved a result similar to part (b) of Conjecture 1.6 for a system involving two hosts, with host of type 1 having a selective advantage (in the absence of symbionts, their model evolves according to a biased voter model rather than a voter model), and one pathogen specialized on host of type 1. Their result, however, was obtained under the assumption that the dispersal range of hosts and symbionts is large, in which case the process viewed on a finite space-time box can be approximated by its mean-field version. The process with short range interactions appears to be much more complicated.

1.5. Conclusion: Effects of Symbionts on the Structure of Their Habitat

The analytic study of the model shows that, in the presence of generalist symbionts, the host population evolves similarly to a voter model, just as in the absence of symbionts (see Theorem 1.1 and Figure 1), suggesting that generalists have only a limited effect on the structure of their habitat. Including host-specific symbionts with the same invasion rates as the generalists, specialists are unable to compete due to a lack of space
available to spread out\cite{22}. Increasing the competitiveness of the specialists, however, can significantly alter the spatial structure of the habitat. In the presence of mutualists, the specialist with the most beneficial effect as well as its most preferred hosts outcompete all the other species (see Theorems 1.2 and 1.5 and Figure 2), making coexistence impossible. In addition, numerical simulations indicate that, when all the specialists are pathogens with more or less harmful effects, they seem to be unable to survive, leaving the host population with the generalists: pathogens go extinct consecutively, starting from the pathogen with the most harmful effect and so on until the pathogen with the least harmful effect (see Figure 2). The most interesting behavior occurs in the symmetric case when all the symbionts are host-specific and have the same effect. In this case, simulations suggest that mutualist specialists modify the structure of their habitat so that it becomes coarse-grained, driving the host population to a clustering more pronounced than in the system without symbiont (see Figures 2 and 3). Pathogen specialists, on the contrary, modify their habitat so that it becomes finer-grained by promoting local diversity (see Figure 3). This difference in behavior is more pronounced the more host-specific the symbionts are.

2. GENERALIST INTERACTIONS: EVOLUTION OF THE HOSTS

This section is devoted to the proof of Theorem 1.1 which is based on duality techniques. Although the process $\eta_t$ does not have a dual process, conditioned on the environment of symbionts, a dual process for the host component can be defined. The first step is to run the symbiont component by going forward in time up to some time $T$ without taking into account the configuration of the host population. This can be done because, due to generalist interactions, the symbionts are “color-blind” in the sense that they associate with hosts of any type at the same rate. The configuration of the symbiont population being determined until time $T$, the second step is to keep track of the ancestors of the hosts by going backwards in time. This will result in a system of coalescing random walks run at varying rates depending on the dynamic environment of symbionts.

To avoid cumbersome notations, we prove the result when $N_2 = 1$ symbiont, but the proof easily extends to the general case. To construct our forward-backwards process, the first step is to define a graphical representation. For each pair of sites $(x, y)$ with $x \sim y$, we draw different types of arrows from site $x$ to site $y$ at the arrival times of independent Poisson processes (see Table 1). The first column indicates the intensity of the Poisson processes, and the last two columns, how to determine the evolution of hosts and symbionts given a realization of the graphical representation.
TABLE 1 Harris’ graphical representation

<table>
<thead>
<tr>
<th>Rate</th>
<th>Arrow</th>
<th>Effect on the host configuration</th>
<th>Effect on the symbiont configuration</th>
</tr>
</thead>
<tbody>
<tr>
<td>$x_{1 \rightarrow i}$</td>
<td>$\rightarrow$</td>
<td>None</td>
<td>If it exists, the symbiont at $x$ gives birth to an offspring sent to $y$</td>
</tr>
<tr>
<td>$\delta_0 = 1$</td>
<td>$\rightarrow \delta_0$</td>
<td>If the host at $\gamma$ is not associated, its type is replaced by the type of the host at $x$</td>
<td>None</td>
</tr>
<tr>
<td>$\delta_1 &gt; 0$</td>
<td>$\rightarrow \delta_1$</td>
<td>If the host at $\gamma$ is associated, its type is replaced by the type of the host at $x$</td>
<td>If it exists, the symbiont at site $\gamma$ is killed regardless of the state of $x$</td>
</tr>
</tbody>
</table>

As previously explained, the process $x_t$ can be constructed up to time $T$ by going forward in time regardless of the configuration of the host population: symbionts give birth through unlabeled arrows whose target site is not already occupied by a symbiont (the host at the tip of the arrow is not associated) and die when they cross a $\delta_1$. In particular, the evolution of the symbiont component depends only on the space-time location of the $\delta_1$’s and of the unlabeled arrows, but not on sites from which the $\delta_1$-arrows originate. The three-dimensional picture of Figure 4 shows a realization.

FIGURE 4 Realization of the graphical representation. The set of hosts associated with a symbiont is determined by going forward in time and is drawn in dashed thick lines on the second layer of the picture. Conditioned on this realization of the symbiont component, the dual process of the host component is determined by going backwards in time and is drawn in continuous thick lines on the first layer of the picture.
of the graphical representation using two layers, the first layer containing the $\delta_0$- and $\delta_1$-arrows, which may affect the hosts, and the second layer containing the unlabeled arrows and a reproduction of the $\delta_1$’s, which may affect the symbionts. The set of hosts associated with a symbiont is drawn in dashed thick lines on the second layer.

In order to determine the ancestry of the host component, we first ignore all the unlabeled arrows as well as all the $\delta_0$-arrows that point at an associated host and all the $\delta_1$-arrows that point at an unassociated host (all these arrows are in dashed lines in Figure 4). The reason why we now ignore those arrows is that they have no effect on the evolution of the hosts. Ignoring the dashed arrows, the dual process of the host component $ht$ is similar to the dual process of the voter model. That is, to keep track of the ancestor of the host at site $x$ at time $T$, we go backwards in time starting from the space-time location $(x, T)$ and, whenever we encounter a $\delta_j$, we cross the corresponding arrow in a direction opposite its orientation (see the first layer of the picture in Figure 4). As for the voter model, if we denote by $X_s(x, T)$ the location at time $T - s$ of the backward process we have just defined, the type of the host at point $(x, T)$ can be deduced from the configuration of the host population at earlier time through the duality relationship

$$h_T(x) = h_{T-s}(X_s(x, T)) \quad \text{for all } 0 \leq s \leq T.$$  

Unlike the dual process of the voter model, the process $X_s(x, T)$ jumps at rate $\delta_0$ or at rate $\delta_1$, depending on the configuration of the symbionts. However, since the process $s_s$ has been constructed regardless of the spatial location of the sites from which the $\delta_1$-arrows originate, at each jump of the process $X_s(x, T)$, the target site is chosen uniformly at random from the set of the $2d$ nearest neighbors. In particular, even if the process $X_s(y, T) - X_s(x, T)$ jumps at a rate that depends on the realization of the symbiont component, its discrete-time version is a symmetric random walk that is independent of the configuration of the symbionts. The rest of the proof of Theorem 1.1 follows the proof of the analogous result for the voter model. The basic idea is that, through the duality relationship between the host component and coalescing random walks, clustering of the process is equivalent to the recurrence property of symmetric random walks in $d \leq 2$, while the existence of nontrivial invariant measures of the hosts is equivalent to the transient property of symmetric random walks in $d \geq 3$ (see for instance Refs.\cite{12,20}, Section 3, for the details).
3. SPECIALIST INTERACTIONS: EFFECT OF A SINGLE MUTUALIST

This section is devoted to the proof of Theorem 1.2(b). Note first that, when \( \alpha_1 \leq 2 \delta_1 \beta_c \), extinction of the symbionts follows directly from the fact that the survival probability, as a function of the set of sites initially occupied by hosts of type 1, is nondecreasing, together with the fact that, when starting with all sites occupied by hosts of type 1, the symbiont component evolves according to a subcritical contact process. The fact that, when \( \alpha_1 \rightarrow 1 > 2 \delta_1 \beta_c \), survival occurs with positive probability is more difficult to establish, and relies on a block argument. The basic idea is to partition space into large intervals \( J_z \) and declare that \( J_z \) is good if it contains only hosts of type 1 and a significant number of mutualists. The process viewed on suitable length and time scales will be coupled with a supercritical oriented percolation process in such a way that the set of good intervals dominates the set of wet sites in the percolation process. More precisely, we set

\[
\mathcal{H}_1 = \{(z, n) \in \mathbb{Z}^2 : z + n \text{ is even and } n \geq 0\},
\]

and let \( N \) and \( T \) denote two (large) integers to be fixed later. For any \( z \in \mathbb{Z} \), we also let

\[
J_z = 2Nz + (−2N, 2N] \quad \text{and} \quad I_z = 2Nz + [−N, N]
\]

and declare that \( J_z \) is good if the following two conditions hold:

1. All sites in the interval \( J_z \) are occupied by hosts of type 1.
2. There is an interval \( I \subset I_z \) of length \( \sqrt{N} \) in which all sites are occupied by mutualists.

Declaring a site \( (z, n) \in \mathcal{H}_1 \) to be occupied if the interval \( J_z \) is good at time \( nT \), the aim is to prove that, for any small parameter \( \epsilon > 0 \), the length and time scales \( N \) and \( T \) can be chosen in such a way that the set of occupied sites dominates the set of wet sites in a 1-dependent oriented percolation process on \( \mathcal{H}_1 \) with parameter \( 1 - \epsilon \). By Theorem 4.3 in Ref.\[12\], this follows from

**Proposition 3.1.** Let \( \epsilon > 0 \). Assume that \( \delta_1 < 1 \) and \( \alpha_1 \rightarrow 1 > 2 \delta_1 \beta_c \). Then,

\[
P(J_{-1} \text{ and } J_1 \text{ are good at time } bN \mid J_0 \text{ is good at time } 0) \geq 1 - \epsilon
\]

for sufficiently large \( N \) and a suitable constant \( b > 0 \).
To avoid cumbersome notations, we prove the result in the presence of \( N_1 = 2 \) hosts, but the proof easily extends to the general case when a finite number of hosts are competing. Standard coupling arguments imply that, as previously mentioned, the survival probability of the symbionts is nondecreasing with respect to the set of sites initially occupied by hosts of type 1, where the collection of subsets of \( \mathbb{Z} \) is equipped with the inclusion relationship as a partial order. In particular, it suffices to establish Proposition 3.1 for the process starting with \( f_0 \) good at time 0 and only hosts of type 2 outside \( f_0 \). The first step is to prove that, for any constant \( b > 0 \), the probability that the interval \( I_0 \) remains occupied by mutualists until time \( T = bN \) goes to one as \( N \to \infty \). This is to build a source of mutualists likely to invade the leftmost and rightmost hosts of type 1. The second step is to estimate the fraction of time these hosts are associated. We call a collision the event that a mutualist associates with the leftmost/rightmost host of type 1. Our estimate on the number of collisions by time \( T \) and the fact that the death rate \( \delta_1 \) of associated hosts is strictly smaller than that of hosts of type 2, results in a linear expansion of the interval occupied by hosts 1.

Throughout this section, the process \( \eta_t \) is constructed from the Harris’ graphical representation described in Table 1. The effects of this graphical representation on the host and symbiont configurations are again the same as in Table 1, except that, due to specialist interactions, symbionts can only spread through an unlabeled arrow if its tip is occupied by an unassociated host of type 1, which makes the duality techniques developed in Section 2 no longer available.

### 3.1. Building a Source of Mutualists

First, we prove that the interval \( I_0 = [-N, N] \) remains available to mutualists, i.e., occupied by hosts of type 1, until time \( T \). Recall that the process starts with \( f_0 \) good at time 0 and only hosts of type 2 outside \( f_0 \). We define

\[
H^-_t = \inf\{x \in \mathbb{Z} : h_t(x) = 1\} \quad \text{and} \quad H^+_t = \sup\{x \in \mathbb{Z} : h_t(x) = 1\},
\]

the leftmost and rightmost host 1 processes, respectively. Also, we let

\[
\Theta^N_t = \{H^-_t \leq -N\} \cap \{H^+_t \geq N\}
\]

denote the event that the interval \( I_0 = [-N, N] \) is only occupied by hosts of type 1 at time \( t \).

**Lemma 3.2.** Assume that \( f_0 \) is good at time 0 and \( \delta_1 \leq 1 \). Then, for any \( c > 0 \),

\[
P(\Theta^N_t \text{ for all } t \leq cN) \geq 1 - C_1 \exp(-\gamma_1 N)
\]

for suitable \( C_1 < \infty \) and \( \gamma_1 > 0 \).
Proof. A standard coupling argument implies that $H_t^+$ (respectively, $H_t^-$) is nonincreasing (respectively, nondecreasing) with respect to $\delta_1$. In particular, by letting $P_{\delta_1}$ be the law of the process in which the death rate of associated hosts is equal to $\delta_1$, we obtain

$$P_{\delta_1}(\Theta^N_t \text{ for all } t \leq cN) \geq P_1(\Theta^N_t \text{ for all } t \leq cN)$$

for all $\delta_1 \leq 1$. But when $\delta_1 = 1$ the host component $h_t$ reduces to a voter model so that the processes $H_t^+$ and $H_t^-$ perform symmetric random walks. Since $H_0^- = -2N + 1$ and $H_0^+ = 2N$, it follows from well-known random walk estimates that

$$P_1(\Theta^N_t \text{ for all } t \leq cN) \geq 1 - C_1 \exp(-\gamma_1 N)$$

for suitable $C_1 < \infty$ and $\gamma_1 > 0$. The lemma follows. \qed

Given a realization of the Harris’ graphical representation introduced in Table 1, we say that there is an invasion path from $(x_0, 0)$ to $(y, t)$ if there is a sequence of times $s_0 = 0 < s_1 < \ldots < s_{n+1} = t$ and spatial locations $x_0 = x, x_1, \ldots, x_n = y$ such that

1. For $i = 1, 2, \ldots, n$, there is an unlabeled arrow from $x_{i-1}$ to $x_i$ at time $s_i$ and
2. For $i = 0, 1, \ldots, n$, the vertical segments $\{x_i\} \times (s_i, s_{i+1})$ do not contain any tip of $\delta_1$-arrows.

Finally, we let $\Omega^N_t$ be the event that there exists an invasion path $\{\pi_i : 0 \leq s \leq t\}$ included in $I_0$, that is, there exists an invasion path with $x_0, x_1, \ldots, x_n \in I_0$.

Lemma 3.3. Assume that $J_0$ is good at time 0. Then, for any $c > 0$ and any $\epsilon > 0$,

$$P(\Omega^N_t \text{ for all } t \leq cN \mid \Theta^N_t \text{ for all } t \leq cN) \geq 1 - \epsilon$$

for all $N$ sufficiently large.

Proof. Since $J_0$ is good at time 0, there exists an interval $I \subset I_0$ of length $\sqrt{N}$ that initially contains only hosts of type 1 associated with a mutualist. Moreover, this interval remains void of hosts of type 2 until time $T = cN$ on the event $\{\Theta^N_t \text{ for all } t \leq cN\}$, which implies that, at least until time $T$, the symbiont component is stochastically larger than the contact process with birth parameter $\alpha_1 \rightarrow 1$ and death parameter $2\delta_1$ restricted to the interval $I$. Denoting by $\sigma_N$ the time to extinction of this contact.
process starting with all sites in $I$ infected, Theorem 2 in Ref.14 states that there exists a constant $\gamma_2 > 0$ such that $(\log \sigma_N)/\sqrt{N} \to \gamma_2$ in probability as $N \to \infty$. Using this result, together with the stochastic domination above, implies that

$$P(\Omega_i^N \text{ for all } t \leq cN | \Theta_i^N \text{ for all } t \leq cN) \geq P(\sigma_N \geq cN) \geq P(\sigma_N \geq \exp(\gamma_2\sqrt{N}/2)) \geq P(\log \sigma_N \geq \gamma_2\sqrt{N}/2) \geq 1 - \epsilon$$

for all $N$ sufficiently large.

With Lemma 3.3 in hand, we are now ready to build a source of mutualists. We introduce the process $\bar{\eta}_t = (\bar{h}_t, \bar{s}_t)$ with the same evolution rules and constructed from the same graphical representation as the process $\eta_t = (h_t, s_t)$ but starting from the initial configuration

$$\bar{h}_0(x) = \begin{cases} 1 & \text{when } x < -N \\ h_0(x) & \text{when } x \geq -N \end{cases} \quad \bar{s}_0(x) = \begin{cases} 1 & \text{when } x < -N \\ s_0(x) & \text{when } x \geq -N \end{cases}$$

Lemma 3.4. $P(\eta_t(x) = \bar{\eta}_t(x) \text{ for all } x \geq N \text{ and } t \leq T | \Theta_i^N \cap \Omega_i^N \text{ for all } t \leq T) = 1.$

Proof. Since the equality has to be checked for every site in the set $[N, H_t^+ + 1]$ which is finite, we can prove the result by induction. We think of the processes as being generated by the graphical representation introduced in Table 1, and assume that the property to be proved holds until the arrival time $t < T$ of a Poisson process in $[N, H_t^+ + 1]$. When time $t$ is the arrival time of a $\delta_0$-arrow or a $\delta_1$-arrow, the configuration of the processes can undergo the following two transitions.

1. The host of type 1 at site $H_t^+$ gives birth to an offspring sent to site $H_t^+ + 1$.
2. The host of type 2 at site $H_t^+ + 1$ gives birth to an offspring sent to site $H_t^+$ and, if it exists, the mutualist at the target site is killed.

In both cases, it is easy to check that such a transition occurs for both processes. We now assume that a mutualist is born at a site $y \in [N, H_t^+]$ at time $t$ in the process $\bar{\eta}_t$. This implies the existence of an invasion path $\bar{\pi}_t$ starting at some site $x$ with $\bar{s}_0(x) = 1$, ending at site $\bar{\pi}_t = y$, and contained in the space-time region occupied by hosts of type 1. Then, we have the following alternative.
1. $\bar{\pi}_s \geq -N$ for all $s \leq t$. Since $h_s(x) = \tilde{h}_s(x)$ for all $x \geq N$ and $s < t$ and the event $\Theta^N_s$ occurs for all $s \leq T$, this invasion path can be used by mutualists in $\eta$, so $s_t(y) = 1$.

2. $\pi_s < -N$ for some $s \leq t$. In this case, we observe that, since $\Omega^N_s$ occurs for all $s \leq T$, there is an invasion path $\pi^*_s$ with $\pi^*_s \in I_0$ for all $s \leq T$. Moreover, site $\pi^*_0$ is occupied by a mutualist at time 0. Due to nearest neighbor interactions, both paths $\pi_s$ and $\pi^*_s$ intersect, so there exists an invasion path $\pi$, with $\pi_s \geq -N$, starting at site $\pi^*_0$ and ending at $(y, t)$. It suffices to follow $\pi^*_s$ until the last time we encounter $\bar{\pi}_s$ then follow the path $\bar{\pi}_s$ until time $t$ (see Figure 5).

In both cases $s_t(y) = 1$ so that $s_t(y) \geq \bar{s}_t(y)$ for all $y \geq N$ and $t \leq T$. The reverse inequality is a straightforward consequence of attractivity and the choice of the initial configuration $\bar{\eta}_0$. □

3.2. Number of Collisions

We now investigate the gap process $H_t^+ - S_t^+$ where

$$S_t^+ = \sup\{x \in \mathbb{Z} : s_t(x) = 1\}$$

denotes the rightmost mutualist process. The following results are proved for the rightmost mutualist only but the analogous results for the leftmost mutualist hold as well and can be deduced by symmetry. Each time the rightmost mutualist is sent to the rightmost host of type 1, we call this event a collision, and let $D_t$ denote the number of collisions by time $t$. Our objective is to prove that, with high probability, the random variable $D_t$ is
greater than a constant times $t$. Motivated by the results of Subsection 3.1, we now focus on the process with initial configuration

$$h_0(x) = \begin{cases} 
1 & \text{when } x \leq 2N \\
2 & \text{when } x > 2N
\end{cases}$$

$$s_0(x) = \begin{cases} 
1 & \text{when } x < -2N \\
0 & \text{when } x \geq -2N
\end{cases}$$

We denote by $\tau_i$ the $i$th time a collision occurs. More precisely, we introduce two sequences of stopping times $\{\tau_i : i \geq 0\}$ and $\{\sigma_i : i \geq 0\}$ by setting

$$\tau_i = \inf\{t \geq \sigma_{i-1} : S_i^+ = H_t^+\}$$

$$\sigma_i = \inf\{t \geq \tau_i : S_i^+ < H_t^+ \text{ or } s_i(S_i^+ - 1) = 1 \text{ and } H_t^+ \text{ jumps to the left}\},$$

with the convention $\tau_0 = \sigma_0 = 0$. In particular, if at time $\sigma_i$ the rightmost mutualist has another mutualist on its left-hand neighboring site and that the rightmost host of type 1 jumps to the left, then $\tau_{i+1} = \sigma_i$ and we also call this event a collision.

**Lemma 3.5.** There exists $c_1 > 0$ such that $P(\tau_1 > c_1 N) \leq C_3 \exp(-\gamma_3 N)$.

**Proof.** Since $\alpha_1 \to 1 > 2\delta_1$, the Shape Theorem in Ref. [25], p. 128, implies that

$$P(S_i^+ - S_0^+ \leq c_2 t \mid \tau_1 \geq t) \leq C_4 \exp(-\gamma_4 t)$$

for some constant $c_2 > 0$, and suitable $C_4 < \infty$ and $\gamma_4 > 0$. This together with the fact that the process $H_t^+$ performs a symmetric random walk until time $\tau_1$ implies the result. $\square$

The next step is to prove that, after the mutualists hit the interface, their distribution on the set of sites occupied by hosts of type 1 dominates the upper invariant measure $\tilde{\mu}$ of the basic contact process with parameter $\alpha_1/2\delta_1$. The aim is to control the gap $S_i^+ - S_i^-$ at times $\sigma_i$.

**Lemma 3.6.** For all $i \geq 1$, the distribution of $s_i$ dominates $\tilde{\mu}$ on $(-\infty, H_t^+ - 1]$ at time $\sigma_i$.

**Proof.** Let $K_t = (-\infty, H_t^+]$ and denote by $\{\xi_t : t \geq 0\}$ the 1-dimensional contact process with birth rate $\alpha_{1}\delta_{1}$ and death rate $2\delta_{1}$ starting with all sites infected, constructed from the same graphical representation as $s_t$ using the unlabeled arrows as birth marks and the $\delta_{1}$’s as death marks. The distribution of $\xi_t$, that we denote by $\mu_t$, is stochastically larger than the measure $\tilde{\mu}$ so it suffices to prove that the distribution of $s_t$ in $K_t - 1$ is
equal to \( \mu_t \) at times \( \sigma_i \). Assume that \( s_t \) is distributed according to \( \mu_t \) in \( K_t \) at time \( \tau_i \) for some integer \( i \geq 1 \). Note that the argument in the proof of Lemma 3.4 implies that this holds at time \( \tau_1 \).

1. Between time \( \tau_i \) and time \( \sigma_i \), site \( H^+_t \) is occupied by a mutualist so the argument described in the proof of Lemma 3.4 implies that

\[ \xi_t(x) = s_t(x) \quad \text{for all } x \in K_t. \]

In particular, \( s_t \) is distributed according to \( \mu_t \) in \( K_t \) until time \( \sigma_i \).

2. At time \( \sigma_i \), the rightmost host 1 process \( H^+_t \) keeps still or jumps to one of its nearest neighbors so in the worst case our property holds in the interval \( K_t - 1 \).

3. Finally, between time \( \sigma_i \) and time \( \tau_{i+1} \), the process \( s_t \) evolves according to a contact process so, by using one more time the argument in the proof of Lemma 3.4, we obtain that \( s_t \) is distributed according to the measure \( \mu_t \) in \( K_t \) at time \( \tau_{i+1} \).

The lemma follows by induction. \( \square \)

**Lemma 3.7.** There exist \( C_5 < \infty \) and \( \gamma_5 > 0 \) such that \( P(\tau_{i+1} - \tau_i > t) \leq C_5 \exp(-\gamma_5 t) \).

**Proof.** By Lemma 3.6, the distribution of \( s_t \) is stochastically larger than the upper invariant measure \( \tilde{\mu} \) in the interval \( K_t - 1 \) at all times \( \sigma_i \). In particular, Theorem 4.20, p. 41, in Ref.\(^\text{[1]}\) implies that there exist two constants \( C_6 < \infty \) and \( \gamma_6 > 0 \) depending on \( \alpha_{i-1} \) and \( \delta_1 \) such that

\[ P(H^+_{\sigma_i} - S^+_{\sigma_i} > s) \leq C_6 \exp(-\gamma_6 s). \]

The same argument as in Lemma 3.5 then implies the existence of \( \epsilon_3 > 0 \) such that

\[ P(\tau_{i+1} - \sigma_i > \epsilon_3 s \mid H^+_{\sigma_i} - S^+_{\sigma_i} \leq s) \leq C_7 \exp(-\gamma_7 s) \]

for suitable \( C_7 < \infty \) and \( \gamma_7 > 0 \). In particular, by setting \( t = \epsilon_3 s \), we obtain

\[ P(\tau_{i+1} - \sigma_i > t) \leq P(H^+_{\sigma_i} - S^+_{\sigma_i} > t/\epsilon_3) + P(\tau_{i+1} - \sigma_i > t \mid H^+_{\sigma_i} - S^+_{\sigma_i} \leq t/\epsilon_3) \leq C_6 \exp(-\gamma_6 t/\epsilon_3) + C_7 \exp(-\gamma_7 t/\epsilon_3). \]

Since \( \sigma_i - \tau_i \) is exponentially distributed with parameter \( 2\delta + 1 \), we can conclude that

\[ P(\tau_{i+1} - \tau_i > t) \leq P(\tau_{i+1} - \sigma_i > t/2) + P(\sigma_i - \tau_i > t/2) \]
\[ \leq C_6 \exp(-\gamma_6 t/2\epsilon_3) + C_7 \exp(-\gamma_7 t/2\epsilon_3) + \exp(-(2\delta_1 + 1)t/2). \]

This completes the proof of the lemma. \(\Box\)

**Lemma 3.8.** Let \( T \geq 2\epsilon_1 N \) and denote by \( D_t = \max\{i \geq 0 : \tau_i < t\} \) the number of collisions that occur by time \( t \). Then, there exists a constant \( a > 0 \) such that

\[ P(D_T \leq aT) \leq C_8 \exp(-\gamma_8 N) \]

for \( N \) sufficiently large, and suitable \( C_8 < \infty \) and \( \gamma_8 > 0 \).

**Proof.** We first observe that, by Lemma 3.5, we have

\[ P(D_{T/2} = 0) = P(\tau_1 > T/2) \leq P(\tau_1 > \epsilon_1 N) \leq C_3 \exp(-\gamma_3 N). \]

In other respects, Lemma 3.7 together with large deviation estimates implies the existence of a constant \( a > 0 \) such that, for suitable \( C_9 < \infty \) and \( \gamma_9 > 0 \),

\[ P(D_T \leq aT | D_{T/2} \neq 0) \leq C_9 \exp(-\gamma_9 N) \]

from which it follows that

\[ P(D_T \leq aT) \leq P(D_{T/2} = 0) + P(D_T \leq aT | D_{T/2} \neq 0) \leq C_3 \exp(-\gamma_3 N) + C_9 \exp(-\gamma_9 N). \]

This completes the proof. \(\Box\)

### 3.3. Invading \( J_{-1} \) and \( J_1 \)

To conclude the proof of Theorem 1.2(b), we now return to the stochastic process \( \eta_t \), starting from any initial configuration with the interval \( J_0 \) good and only unassociated hosts of type 2 outside \( J_0 \). We will prove the existence of a constant \( b > 0 \) such that, with probability close to 1 when \( N \) is large, \( J_1 \) is good at time \( T = bN \). By symmetry of the evolution rules in space, the same holds for the interval \( J_{-1} \).

First of all, the combination of Lemmas 3.4 and 3.8 implies that for the process starting with the interval \( J_0 \) good and only hosts of type 2 outside \( J_0 \) we have

\[ P(D_T \leq aT | \Theta_t^N \cap \Omega_t^N \text{ for all } t \leq T) \leq C_8 \exp(-\gamma_8 N) \]
for all $T \geq 2c_1 N$. This, combined with Lemmas 3.2 and 3.3, implies that, for any $\epsilon > 0$,

$$P(D_T \leq aT) \leq P(\Theta^N_T \text{ does not occur for some } t \leq T)$$

$$+ P(\Omega^N_T \text{ does not occur for some } t \leq T | \Theta^N_T \text{ for all } t \leq T)$$

$$+ P(D_T \leq aT | \Theta^N_T \cap \Omega^N_T \text{ for all } t \leq T) \leq \epsilon$$

for $N$ large. Furthermore, from time $\sigma_{i-1}$ to time $\tau_i$ when a collision occurs, the process $H_i^+$ evolves according to a symmetric random walk, between time $\tau_i$ and time $\sigma_i$ it is frozen, while denoting by $x$ the position of the process $H_i^+$ at time $\sigma_i$ we have the following alternative at time $\sigma_i$.

1. There is a $\delta_1$-arrow from $x + 1$ to $x$ which causes $H_i^+$ to jump to $x - 1$.
2. There is a $\delta_1$-arrow from $x - 1$ to $x$ which has no effect on the process $H_i^+$.
3. There is a $\delta_0$-arrow from $x$ to $x + 1$ which causes $H_i^+$ to jump to $x + 1$.

Since $\delta_i$-arrows between adjacent sites occur at rate $\delta_i$ and $\delta_0 > \delta_1$ (mutualist), the evolution rules above cause the process $H_i^+$ to drift to the right. More precisely, we have

$$\mathbb{E}(H^+_T - H^+_0 | D_T) = \frac{\delta_0 - \delta_1}{2\delta_1 + \delta_0} D_T$$

so that large deviation estimates imply that

$$P\left(\frac{H^+_T - H^+_0}{2\delta_1 + \delta_0} \leq \frac{\delta_0 - \delta_1}{2\delta_1 + \delta_0} aT \bigg| D_T > aT\right) \leq C_{10} \exp(-\gamma_{10} N)$$

for suitable constants $C_{10} < \infty$ and $\gamma_{10} > 0$. Setting $T = bN$ where

$$b = \max\left(2c_1, \frac{4\delta_1 + \delta_0}{\delta_0 - \delta_1}\right),$$

Lemma 3.8 holds with the same constant $a$ since $b \geq 2c_1$ and none of the constants $a$ and $b$ depends on the parameter $N$. The choice of $b$ also implies that

$$\frac{\delta_0 - \delta_1}{2\delta_1 + \delta_0} aT \geq 2N$$

from which it follows that, for any $\epsilon > 0$,

$$P(H^+_T - H^+_0 \leq 2N) \leq P(H^+_T - H^+_0 \leq 2N | D_T > aT) + P(D_T \leq aT) \leq \epsilon$$
for all $N$ sufficiently large. This proves that, with probability arbitrarily close to 1 when $N$ is large, all sites in the interval $J$ are occupied by hosts of type 1 at time $T = bN$. Finally, we observe that, by Lemma 3.6, the distribution of $s_i$ is stochastically larger than the upper invariant measure $\bar{\mu}$ in the intervals $L_1$ and $I_1$ at time $T$. Since $\bar{\mu}$ is ergodic by Proposition 2.16 in Ref. [24], p. 143, there exist, with probability close to 1 when $N$ is large, two sub-intervals, each of length $\sqrt{N}$, in which all the hosts are associated with a mutualist, and included in $L_1$ and $I_1$, respectively. This completes the proof of Proposition 3.1 and Theorem 1.2(b).

3.4. Proof of Theorem 1.3

To prove Theorem 1.3, we now assume that $\delta_0 < \delta_1$ modeling the fact that the symbiont is a pathogen, and start from the initial configuration

$$h_0(x) = \begin{cases} 1 & \text{when } x \leq 0 \\ 2 & \text{when } x > 0 \end{cases} \quad s_0(x) = \begin{cases} 0 & \text{when } x \leq 0 \\ 1 & \text{when } x > 0 \end{cases}$$

Defining the rightmost host 1 process $H^+_t$, the rightmost pathogen process $S^+_t$, and the number of collision events $D_t$ occurring by time $t$ as previously, the proof of Lemma 3.8 implies that

$$P(D_T \leq aT) \leq C_8 \exp(-\gamma_8 N)$$

for all $T \geq 2_1 N$ and $N$ sufficiently large, in the pathogenic case as well. This follows from the fact that the process $s_i$ evolves according to a basic contact process with parameter $\alpha_{1-1/2} \delta_1 > \beta_{c}$ on the dynamic set $(-\infty, H^+_T]$. The transition rates of the process $H^+_t$ when the rightmost host 1 is associated with a pathogen also indicate that

$$\mathbb{E}(H^+_t | D_T) = \mathbb{E}(H^+_t - H^+_0 | D_T) = \frac{\delta_0 - \delta_1}{2 \delta_1 + \delta_0} D_T.$$ 

In particular, setting

$$\epsilon = \frac{a \delta_0 - \delta_1}{2 \delta_1 + \delta_0}$$

and using large deviation estimates, we obtain

$$P(H^+_T \geq cT) \leq P(H^+_T \geq cT | D_T > aT) + P(D_T \leq aT) \leq C_{11} \exp(-\gamma_{11} N)$$
for suitable $C_1 < \infty$ and $\gamma_{11} > 0$. Theorem 1.3 follows by observing that $c < 0$ (pathogen) and by taking the limit as $N \to \infty$ in the previous inequality.

4. MIXED INTERACTIONS: EFFECT OF MULTIPLE SYMBIONTS

This section is devoted to the proof of Theorem 1.5. To avoid cumbersome notations, we prove the result when $N_1 = 2$ hosts and $N_2 = 2$ symbionts, but as previously the proof easily extends to the system with a finite number of species. Recall that $\delta_1 < \min(\delta_0, \delta_2)$, indicating that symbiont of type 1 has the most beneficial effect on its preferred host. We will show first that, in the limiting case $\alpha_{1 \to 1} = \infty$, the set of sites occupied by hosts of type 1 dominates the set of sites occupied by particles of type 1 in a two-color biased voter model in which particles of type 1 have a selective advantage over particles of type 2. Since the latter converges to the “all 1” configuration this will produce the result when $\alpha_{1 \to 1} = \infty$. To conclude, we will apply a perturbation argument to extend the result to the region where the transmission rate $\alpha_{1 \to 1}$ is large but finite.

Without loss of generality, we may assume that all the hosts of type 1 are initially associated with a mutualist of type 1. To define the process when $\alpha_{1 \to 1} = \infty$, we first observe that whenever a host of type 1 associated with a mutualist of type 1 gives birth to a host of its own kind, the latter becomes instantaneously associated with a mutualist of type 1. Therefore, at any time, all the hosts of type 1 are associated with a type 1 mutualist. The state space of the process thus reduces to all the functions that map $\mathbb{Z}^d$ into $\{(1, 1), (2, 0), (2, 2)\}$, and the evolution rules at site $x$ are described by the transition rates

\[
(i, j) \to (1, 1) \quad \text{at rate} \quad \delta_j \sum_{x \sim y} \mathbb{1}\{h(y) = 1\}
\]

\[
(i, j) \to (2, 0) \quad \text{at rate} \quad \delta_j \sum_{x \sim y} \mathbb{1}\{h(y) = 2\}
\]

\[
(2, 0) \to (2, 2) \quad \text{at rate} \quad \alpha_{2 \to 2} \sum_{x \sim y} \mathbb{1}\{s(y) = 2\}.
\]

The transition rates indicate that hosts of type 1 die at rate $\delta_1 < \min(\delta_0, \delta_2)$ while hosts of type 2 die at rate $\delta_0$ or $\delta_2$ according to whether they are unassociated or associated with a symbiont of type 2, respectively. At death, hosts of either type are replaced by a new host whose type is randomly chosen from the nearest neighbors. Symbionts of type 2 associate with unassociated neighboring hosts of type 2 at rate $\alpha_{2 \to 2}$ whereas symbionts of type 1 associate instantaneously with unassociated neighboring hosts of type 1. The death rates of the hosts suggest that hosts of type 1 outcompete
hosts of type 2, which is proved by coupling the process $\eta_t$ with a biased voter model with appropriate parameters. We introduce the continuous-time Markov process whose state at time $t$ is a function $\xi_t: \mathbb{Z}^d \rightarrow \{1, 2\}$ and whose evolution at site $x$ is described by

$$1 \rightarrow 2 \text{ at rate } \delta_1 \sum_{x \sim y} \mathbb{I}\{\xi(y) = 2\}$$

$$2 \rightarrow 1 \text{ at rate } \min(\delta_0, \delta_2) \sum_{x \sim y} \mathbb{I}\{\xi(y) = 1\}.$$  

Particles of type 1 die at rate $\delta_1$ while particles of type 2 die at rate $\min(\delta_0, \delta_2)$. At death, particles of either type are replaced by a new particle whose type is randomly chosen from the nearest neighbors. The death rate of one type can also be interpreted as (a constant times) the birth rate of the other type, in which case the process can be seen as the biased voter model in which particles of type 1 give birth at rate $2d \min(\delta_0, \delta_2)$ and particles of type 2 at rate $2\delta_1$. Results in Refs.\cite{3,4} imply that, starting from any initial configuration with infinitely many 1's, the process converges almost surely to the “all 1” configuration provided $\delta_1 < \min(\delta_0, \delta_2)$. The following lemma implies that hosts of type 1 outcompete hosts of type 2 as well in the process $\eta_t$ when the transmission rate of symbionts of type 1 is infinite.

**Lemma 4.1.** Assume that $\delta_1 < \min(\delta_0, \delta_2)$ and that $\alpha_{1-t} = \infty$. Then, $\eta_t = (h_t, s_t)$ and the biased voter model $\xi_t$ can be constructed on the same probability space in such a way that

$$\{x \in \mathbb{Z}^d : \xi_t(x) = 1\} \subset \{x \in \mathbb{Z}^d : h_t(x) = 1\} \text{ at any time } t \geq 0$$

provided the inclusion holds at time 0.

**Proof.** The proof relies on a coupling argument where both processes are constructed from the same Harris’ graphical representation. We first assume that $\delta_1 < \delta_2 < \delta_0$ indicating that both symbions are mutualists. For each ordered pair $(x, y)$ of adjacent sites, we draw different types of arrows from site $x$ to site $y$ at the arrival times of independent Poisson processes whose rates are indicated in the first four rows of Table 2. The processes $\eta_t$ and $\xi_t$ evolve simultaneously along this graphical representation according to the rules described in the third and fourth columns, respectively. To understand the third column, recall that, since all the hosts of type 1 are initially associated with symbionts of type 1 with their transmission rate to hosts of type 1 being equal to infinity, offspring of hosts of type 1 become instantaneously associated with a symbiont of type 1. In particular,
potential transmission events of a symbiont of type 2 to a host of type 1 can be ignored since all the hosts of type 1 are permanently associated with a symbiont of type 1. One can check that these rules induce our coevolutionary process in the limiting case when the transmission rate $\alpha_{1\to 1} = \infty$ and the biased voter model introduced above. Moreover, the processes are coupled in such a way that the inclusion to be proved holds at any time provided it holds at time 0. The result when $\delta_1 < \delta_0 < \delta_2$ can be proved in a similar way but replacing the rates and effects of $\delta_0$- and $\delta_2$-arrows as indicated in the last two rows of Table 2. □

Lemma 4.1 together with results in Refs.\cite{3,4} implies Theorem 1.5 in the limiting case $\alpha_{1\to 1} = \infty$. To extend the result to the region $\alpha_{1\to 1}$ large but finite, the idea is to compare the process $\eta_t$ viewed on suitable length and time scales with an oriented site percolation process on the lattice 

$$\mathcal{H}_d = \{(z, n) \in \mathbb{Z}^d \times \mathbb{Z}_+ : z_1 + \cdots + z_d + n \text{ is even}\}$$

### TABLE 2 Construction of the processes $\eta_t$ and $\xi_t$ when $\delta_2 < \delta_0$ and $\delta_0 < \delta_2$, respectively

<table>
<thead>
<tr>
<th>Rate</th>
<th>Arrow</th>
<th>Effect on the process $\eta_t$</th>
<th>Effect on the process $\xi_t$, respectively</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\alpha_{1\to 2}$</td>
<td>$\rightarrow 22$</td>
<td>If the host at site $x$ is associated with a symbiont of type 2 and the host at site $y$ is an unassociated host of type 2, the latter becomes associated with a symbiont of type 2</td>
<td>None</td>
</tr>
<tr>
<td>$\delta_1$</td>
<td>$\rightarrow \delta_1$</td>
<td>The host at $y$ is replaced by a host of the same type as the host at $x$; if the new host at $y$ is of type 1 it is associated with a symbiont of type 1, while if it is of type 2 it is unassociated</td>
<td>The particle at $y$ is replaced by a particle of the same type as the particle at $x$</td>
</tr>
<tr>
<td>$\delta_0 - \delta_1$</td>
<td>$\rightarrow \delta_2$</td>
<td>If the host at $y$ is unassociated or associated with a symbiont of type 2 then the effect is the same as for $\delta_1$-arrows</td>
<td>If the particle at $y$ is of type 2 it is replaced by a particle of the same type as the particle at $x$</td>
</tr>
<tr>
<td>$\delta_0 - \delta_2$</td>
<td>$\rightarrow \delta_0$</td>
<td>If the host at $y$ is unassociated then the effect is the same as for $\delta_1$-arrows</td>
<td>None</td>
</tr>
<tr>
<td>$\delta_0 - \delta_1$</td>
<td>$\rightarrow \delta_0$</td>
<td>If the host at $y$ is unassociated or associated with a symbiont of type 2 then the effect is the same as for $\delta_1$-arrows</td>
<td>If the particle at $y$ is of type 2 it is replaced by a particle of the same type as the particle at $x$</td>
</tr>
<tr>
<td>$\delta_2 - \delta_0$</td>
<td>$\rightarrow \delta_2$</td>
<td>If the host at $y$ is associated with a symbiont of type 2 then the effect is the same as for $\delta_1$-arrows</td>
<td>None</td>
</tr>
</tbody>
</table>
Host-Pathogen and Host-Mutualist Interactions

The reason why we invoke a rescaling argument is to later compare the processes with \( z_{1 \to 1} \) infinite and \( z_{1 \to 1} \) finite within each space-time box of the block construction, and apply a perturbation argument to extend our results from one process to the other. Given two integers \( \Gamma \) and \( L \) to be fixed later, a site \((z, n) \in \mathcal{H}_d\) is said to be good if for all \( x \in Lz + (-L, L)^d\)

1. The host at site \( x \) is of type 1 at time \( n \frac{\Gamma}{L} \) and
2. There is a site \( y \in \mathbb{Z}_d \) with \( x \sim y \) which is occupied by a mutualist of type 1 at time \( n \frac{\Gamma}{L} \).

Assume that \( \delta_1 < \min(\delta_0, \delta_2) \) and start the biased voter model with a single particle of type 1 at site 0. Then, conditioned on nonextinction of the 1’s, there is an in-all-directions expanding region which is void of type 2. This together with Lemma 4.1 implies the following lemma, which is proved in Section 3 in Ref.[21].

**Lemma 4.2.** Assume that \( \delta_1 < \min(\delta_0, \delta_2) \) and that \( z_{1 \to 1} = \infty \). Then, there exists \( \Gamma \) such that, for any \( \epsilon > 0 \), the set of good sites dominates the set of wet sites in a 3-dependent oriented site percolation process with parameter \( 1 - \epsilon \) for all \( L \) sufficiently large.

We now consider the process with \( z_{1 \to 1} \) finite, which is constructed from a modification of the graphical representation of Table 2 for which

1. We change the interpretation of \( \delta_1 \)-arrows (and \( \delta_0 \)- and \( \delta_2 \)-arrows accordingly) by assuming that offspring of hosts of either type are unassociated.
2. For each \((x, y)\) with \( x \sim y \), we draw a 11-arrow from \( x \) to \( y \) at the arrival times of a Poisson process with parameter \( z_{1 \to 1} \) to indicate that if the host at \( x \) is associated with a type 1 symbiont and site \( y \) is occupied by an unassociated host of type 1 then the latter becomes associated with a type 1 symbiont.
3. For each \((x, y)\) with \( x \sim y \), we draw a 21-arrow from \( x \) to \( y \) at the arrival times of a Poisson process with parameter \( z_{2 \to 1} \) to indicate that if the host at \( x \) is associated with a type 2 symbiont and site \( y \) is occupied by an unassociated host of type 1 then the latter becomes associated with a type 1 symbiont.

The next step is to prove the analog of Lemma 4.2 for the process with a finite but sufficiently large transmission rate \( z_{1 \to 1} \) and oriented site percolation with parameter \( 1 - 3\epsilon \). To achieve this objective, it suffices to prove the existence of a large enough \( z_{1 \to 1} \) such that, with probability greater than \( 1 - \epsilon \), the following event \( E \) occurs:
$E$ = each time a host of type 1 associated with a mutualist of type 1
at some site $x \in [-3L, 3L]^d$ gives birth by time $\Gamma L$ to a host of type 1
which is then sent to site $y$, the next occurrence of a Poisson process
at either $x$ or $y$ causes the host at site $y$ to become associated with a
mutualist of type 1.

We focus only on the spatial box $[-3L, 3L]^d$ because of the 3-dependency
of the percolation process introduced in Lemma 4.2. To estimate the
probability of the event $E$, we first pick a pair of adjacent sites $(x, y)$ and
assume that site $x$ is occupied by a host of type 1 associated with a mutualist
of type 1 that gives birth at time $T$ to an unassociated host which is sent
to $y$. Excluding 11-arrows, the rate at which arrows point at either site
$x$ or site $y$ is given by

$$4d(x_{2-1} + x_{2-2} + \max(\delta_0, \delta_2)).$$

In other respects, since the host at site $x$ is associated with a mutualist
of type 1, the host at site $y$ becomes associated with a mutualist of type 1 at
rate at least $\alpha_{1 \to 1}$. In particular, the probability that the next occurrence
after time $T$ of a Poisson process at either site $x$ or site $y$ results in the
association of a type 1 mutualist at site $y$ is greater than

$$p = \frac{x_{1-1}}{x_{1-1} + 4d(x_{2-1} + x_{2-2} + \max(\delta_0, \delta_2))}.$$

Now, let $N$ denote the number of births of a host of type 1 in $B =
[-3L, 3L]^d \times [0, \Gamma L]$. Since the death rate of host of either type is at most
$2d \max(\delta_0, \delta_2)$, we have

$$\mathbb{E}(N) \leq 2d \max(\delta_0, \delta_2) \times |B| = 2d \max(\delta_0, \delta_2)(6L + 1)^d \Gamma L.$$

Large deviation estimates for the Poisson distribution then imply that

$$P(N > 4d \max(\delta_0, \delta_2)(6L + 1)^d \Gamma L) \leq C_{12} \exp(-\gamma_{12} \Gamma L)$$

for suitable $C_{12} < \infty$ and $\gamma_{12} > 0$. It follows that

$$1 - P(E) \leq C_{12} \exp(-\gamma_{12} \Gamma L) + 4d \max(\delta_0, \delta_2)(6L + 1)^d \Gamma L (1 - p).$$

By taking first $L$ sufficiently large so that both $C_{12} \exp(-\gamma_{12} \Gamma L) \leq \epsilon$ and
Lemma 4.2 holds, and then the transmission rate $x_{1-1}$ large so that

$$4d \max(\delta_0, \delta_2)(6L + 1)^d \Gamma L \leq \epsilon$$

\frac{4d(x_{2-1} + x_{2-2} + \max(\delta_0, \delta_2))}{x_{1-1} + 4d(x_{2-1} + x_{2-2} + \max(\delta_0, \delta_2))} \leq \epsilon
we have $1 - P(E) \leq 2\epsilon$, which together with Lemma 4.2 implies that

$$P(\text{site } (z, 1) \text{ is not good for some } z \sim 0 \mid \text{site } (0, 0) \text{ is good}) \leq 3\epsilon.$$  

In conclusion, we obtain the following

**Lemma 4.3.** Assume that $\delta_1 < \min(\delta_0, \delta_2)$. Then, there exists $\Gamma$ such that, for any $\epsilon > 0$, the parameters $L$ and $\alpha_{1 \rightarrow 1}$ can be chosen in such a way that the set of good sites dominates the set of wet sites in a 3-dependent oriented site percolation process with parameter $1 - \epsilon$.

This implies the existence of a critical value $\alpha_c < \infty$ such that, under the assumptions of Theorem 1.5, type 1 hosts and type 1 mutualists survive whenever $\alpha_{1 \rightarrow 1} > \alpha_c$. Our last problem is that oriented site percolation has a positive density of unoccupied sites, which may result in survival of type 2 hosts and type 2 symbionts. To prove that hosts and symbionts of type 1 outcompete the other species, namely that there is an in-all-directions expanding region which is void of hosts and symbionts of type 2, we apply a result from Ref.\textsuperscript{[11]} that shows that sites that are not occupied do not percolate when $\epsilon$ is close enough to 0. Since species of either type cannot appear spontaneously, once a region is void of one type, this type can only reappear in the region through invasion from the outside. This concludes the proof of Theorem 1.5.

**ACKNOWLEDGMENTS**

The authors thank two anonymous referees for their interesting comments and suggestions.

Partially supported by NSF Grants DMS-00-72262 and DMS-00-83468 to C. Neuhauser.

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