Individual-based modeling of the spread of pine wilt disease: vector beetle dispersal and the Allee effect

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Abstract Pine wilt disease is caused by the pinewood nematode *Bursaphelenchus xylophilus*, which is vectored by the Japanese pine sawyer beetle *Monochamus alternatus*. Due to their mutualistic relationship, according to which the nematode weakens and makes trees available for beetle reproduction and the beetle in turn carries and transmits the nematode to healthy pine trees, this disease has resulted in severe damage to pine trees in Japan in recent decades. Previous studies have worked on modeling of population dynamics of the vector beetle and the pine tree to explore spatial expansion of the disease using an integro-difference equation with a dispersal kernel that describes beetle mobility over space. In this paper, I revisit these previous models but retaining individuality: by considering mechanistic interactions at the individual level it is shown that the Allee effect, an increasing per-capita growth rate as population abundance increases, can arise in the beetle dynamics because of the necessity for beetles to contact pine trees at least twice to reproduce successfully. The incubation period after which a tree contacted by a first beetle becomes ready for beetle oviposition by later beetles is crucial for the emergence of this Allee effect. It is also shown, however, that the strength of this Allee effect depends strongly on biological mechanistic properties, especially on beetle mobility. Realistic individual-based modeling highlights the importance of how spatial scales are dealt with in mathematical models. The link between

mechanistic individual-based modeling and conventional analytical approaches is also discussed.

Introduction

Pine wilt disease in Japan

Pine wilt disease is a disease that infects and eventually kills pine trees. A typical, external symptom of the disease is red-brownish foliage, which is very conspicuous among a stand of healthy pine trees with evergreen foliage. In past decades, the disease has resulted in severe damage to pine trees in Japan. The disease was first recorded in 1905 at Nagasaki City in a southwest district of Japan. In the last century it has spread progressively eastwards along coasts, and by the 1990s the disease range covered almost all of Japan except the northernmost prefectures (Mamiya 1988; Kishi 1995).

The disease is caused by the pinewood nematode *Bursaphelenchus xylophilus* (Kiyohara and Tokushige 1971; Mamiya and Kiyohara 1972), which is vectored by the pine sawyer beetle *Monochamus alternatus* that disperses the nematode to healthy trees over space (Mamiya and Enda 1972; Morimoto and Iwasaki 1972). Since the discovery that the nematode is the causative agent, vectored by the sawyer beetle, extensive effort has been paid to control the vector beetle; aerial application of insecticides against flying beetle adults and insecticide spraying, fumigation or burning of nematode-killed trees in which the beetle immature stages reside have been conducted in

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the past decades. Despite extensive effort to control the disease, it has continuously expanded its range to include almost all of Japan.

The nematode is originally native to North America, where resistant pine species are distributed in warm-summer areas and susceptible pine species in cool-summer areas (Futai and Funuro 1979; Malek and Appleby 1984; Wingfield et al. 1984; Mamiya 1987; Rutherford and Webster 1987). It has been inferred that the nematode was accidentally brought to Japan through lumber trade at the beginning of the 20th century (Mamiya 1988; Beckenbach et al. 1992). The disease has spread quickly because two major pine species, Japanese black pine *Pinus thunbergii* and red pine *P. densiflora*, are extremely susceptible to the nematode infection. One of the causes of the severe damage to Japanese pine is the lack of resistance to the newly encountered nematode.

Lifecycle of the nematode and the vector beetle

Togashi and Shigesada (2006) reviewed the lifecycle of the nematode and the vector beetle in detail. In late June to early July, a large proportion of adult beetles emerge from pine trees that were infected and killed the previous year. These beetles carry nematodes within their tracheal system and fly to healthy pine trees to feed on twig bark for maturation. During this feeding, nematodes are transmitted to cortex and woody tissue through wounds on the twig made by beetles. Within 2–3 weeks infected trees lose the ability for oleoresin exudation. Soon afterwards, the trees emit ethanol and monoterpenes that attract mature beetles. Weakened trees eventually wilt and die and their foliage turns reddish brown, a typical symptom of pine wilt disease.

Beetle eggs laid on a weakened tree hatch after 1 week, and larvae feed on fresh inner bark of the tree to grow. By the end of autumn they construct pupal chambers in woody tissue, where they overwinter. The nematode begins to gather around the pupal chamber in winter and enter the tracheal system of newly eclosed adult beetle present in pupal chamber next late spring and early summer. Adult beetles then leave for healthy trees with lots of nematodes for new transmission the following June and July.

These lifecycles of the nematode and the beetle exhibit an obligatory mutualism in that the nematode supplies the beetle with newly weakened trees while the beetle transmits the nematode to a new host tree. As the two major Japanese pine trees have no resistance to the nematode infection, the disease spreads quickly nationwide under the mutualistic link between the beetle and the nematode.

Previous models of pine wilt disease

Yoshimura et al. (1999) modeled the population dynamics of the vector beetle and the pine tree based on detailed empirical data on the lifecycle of the nematode and the beetle in order to predict how the disease could be controlled by eradication of the beetle. They focused on the following nonspatial dynamics of the population density of the healthy pine trees, H_t, and the beetle, P_t, in year t given as difference equations

$$H_{t+1} = \exp[-\alpha P_t]H_t, \tag{1}$$

$$P_{t+1} = (1-\theta)F(P_t, \tilde{H}_t)\tilde{H}_t, \tag{2}$$

where α measures the rate of a healthy pine tree becoming infected by the nematode carried on beetles, $F(P_t, \tilde{H}_t)$ is the number of adult beetles emerging from an infected and dead pine tree the following year, $\tilde{H}_t$ is pine trees infected in year t ($\tilde{H}_t = H_t - H_{t+1}$) and θ is the eradication effort to reduce beetle density by spraying insecticide or fumigation of infected trees at the end of the year before adult beetles emerge ($0 \le \theta \le 1$).

Yoshimura et al. (1999) estimated the functional form of $F(P_t, \tilde{H}_t)$, the beetle's reproduction per tree, as a Holling type 2 response function of the beetle density P_t as

$$F(P_t, \tilde{H}_t) = \frac{bP_t}{S(c + \tilde{H}_t) + aP_t}, \tag{3}$$

where a, b, c, and S are parameters estimated by beetle breeding and average pine tree properties, and investigated how the eradication effort θ can control the beetle population to stop the disease epidemic.

In the Yoshimura et al. (1999) model, the beetle dynamics when the density P_t is low ($P_t \ll 1$) is reduced to

$$P_{t+1} \propto (1-\theta)H_t P_t^2, \tag{4}$$

and this dynamics shows a typical Allee effect in that the per-capita growth rate increases with the population density and that there exists a density threshold; once the beetle density is below this, it decreases monotonically to zero.

Takasu et al. (2000) extended the nonspatial dynamics to consider explicitly spatial expansion of the disease in a continuous one-dimensional space using an integro-difference equation. They analyzed the spatiotemporal dynamics of the beetle density, $P_t(x)$, and the healthy pine tree density, $H_t(x)$, at location x in year t. They introduced a dispersal kernel $w(x,y)$ which describes beetle mobility. The dispersal kernel can be interpreted as the probability that a beetle at position x moves to position y, which in many cases would be a function of the distance $d = |x - y|$ (Kot et al. 1996; Lewis 1997; Kot and Schaffer 1986). They assumed that newly emerged adults first disperse

according to the dispersal kernel and then interact with pine trees according to the local dynamics of Yoshimura et al. (1999). The beetle density at position x after dispersal, $P'_t(x)$, is given by

$$P'_t(x) = \int w(x,y)P_t(y)\mathrm{d}y. \tag{5}$$

After dispersal, beetles and pine trees at position x locally interact according to the Yoshimura et al. (1999) dynamics

$$H_{t+1}(x) = \exp\left[-\alpha P'_t(x)\right]H_t(x), \tag{6}$$

$$P_{t+1}(x) = (1-\theta)F(P'_t(x), \tilde{H}_t(x))\tilde{H}_t(x), \tag{7}$$

and the 1-year cycle completes.

Takasu et al. (2000) showed that the rate of disease spread depends critically on the functional form of the dispersal kernel $w(x,y) = w(d)$ and stressed the importance of precisely estimating the tail of the dispersal distribution for long-distance dispersal. Shigesada and Kawasaki (2002) reviewed the effects of long-distance dispersal in the context of species invasion.

These baseline models given as difference and integro-difference equations deterministically describe temporal and spatiotemporal change of population density as a nonnegative real value. These models, however, are descriptive in the sense that they do not explicitly consider mechanistic interactions of a beetle and a pine tree at the individual level.

Theoretical population biologists have recently noted the importance of "being individual and spatial" in population dynamics and started to pay attention to models that retain "individuality" in the framework (Durrett and Levin 1994). Needless to say, all biological populations consist of a certain number of individuals. All individuals experience discrete events of birth and death in their life as a discrete entity, i.e., the individual is the unit as it is not divisible.

Models retaining this individuality do not necessarily show qualitatively identical behaviors to those that ignore individuality and that focus on density as a continuous quantity, as described by conventional difference or differential equations, especially when Allee effects are involved in the dynamics (Dennis 2002; Snyder 2003; Kot et al. 2004).

In this paper I revisit the above baseline models of pine wilt disease with individuality retained explicitly, so that the individual is the unit of population dynamics, and mechanistic interactions at the individual level are directly modeled as an individual-based model. Construction and analysis of a realistic individual-based model helps us to understand better the mechanistic background of the Allee effect in pine wilt disease. The link with conventional analytical models by integro-difference equation and individual-based model is also discussed.

The model

Nonspatial individual-based population dynamics

I first revisit the nonspatial population dynamics of the beetle and the pine tree explored by Yoshimura et al. (1999) as an individual-based population dynamics. Let H_t denote the number of pine trees and P_t the number of beetles in year t. All beetles are assumed to carry enough nematodes to make a contacted tree infected. Both H_t and P_t are nonnegative integer-valued quantities.

I assume that P_t new adult beetles emerge at the beginning of year t and repeat a series of movements to a tree to feed for maturation T_M times, which I call the infection phase. As I here assume no spatial structure, movement of a beetle to a pine is random in the sense that each of P_t beetles moves to a randomly chosen pine tree among H_t trees.

The unit of time during the infection phase is taken as the time needed for a beetle to make one movement, or the time for which a beetle stays at a tree. This could be arbitrary unless detailed information is available on how often beetles fly to another tree. As a beetle stays on a healthy pine tree for 2–3 days on average (Togashi and Shigesada 2006), the time unit of infection phase is set as 3 days in this model for the sake of realism. Beetle females become reproductively matured 5–30 days after emergence (Togashi and Shigesada 2006) and the length of the infection phase is set as $T_M = 10$.

I assign each of H_t pine trees the set of three properties that crucially determine beetle reproduction: (a) tree status, (b) time of first infection, and (c) the number of beetles that have oviposited on it. Tree status is one of the following: (1) susceptible (S), when the tree is healthy and has potential to be infected by the nematode; healthy trees can exude oleoresin, which acts as a physical barrier to beetle oviposition, and beetles cannot oviposit on them due to the lack of emission of ethanol; (2) incubation of the nematode (I), when the tree has been infected by the nematode but still sustains the ability for oleoresin exudation; (3) ready for beetle oviposition (R), when oleoresin exudation ability has been lost and beetles can oviposit on it; and (4) dead (D), when the tree is dead. Transition through these status is irreversible: $S \rightarrow I \rightarrow R \rightarrow D$, or $S \rightarrow I = R \rightarrow D$ if oleoresin exudation ability stops immediately after first infection.

All pine trees start from status S. When a pine tree i ($i = 1, 2, \ldots, H_t$) is visited by a beetle for the first time in the τ-th term of the infection phase ($\tau = 1, 2, \ldots, T_M$), the

tree becomes infected, the status changes from S to I, and τ is recorded as the time of first infection of tree i. The nematode loses oleoresin exudation ability after T_I time units, when the tree status changes from I to R ($0 \leq T_I \leq T_M$).

T_I is the incubation period of the nematode and measures how fast an infected tree is weakened and becomes ready for beetle oviposition.

In the last term of the infection phase $\tau = T_M$, all beetles are reproductively mature and oviposit, but oviposition is effective only on pine trees whose status is R, i.e., beetles cannot oviposit on trees I that have been infected for less than T_I. The number of beetles that have oviposited on tree i whose status is R is recorded as the number of ovipositions, n, which is translated into the number of newly adult beetles, $F(n)$, emerging from tree i in the following year $t + 1$. Here, n refers to how many beetles oviposit on a tree R with the assumption that the number of eggs per oviposition is constant. Due to density-dependent mortality of the beetle larva within a tree, I assume the following hyperbolic function, which saturates as the number of ovipositions n increases (cf. Eq. 3 in Yoshimura et al. 1999):

$$F(n) = \frac{bn}{1 + an},$$

where b is the net reproductive success when density-dependent mortality can be ignored and a controls the maximum number of beetles emerging from a tree. The actual number of beetle offspring is rounded to the nearest integer. Yoshimura et al. (1999) estimated that maximally 36 beetles emerge from an infected pine tree, considering intraspecific competition of beetle larvae. Here I assume $b = 20$ and $a = 1$ for the sake of the computational cost of the simulation to reduce the maximal number of beetles (maximally 20 beetles emerge from a tree in this model).

All beetles die after oviposition. All trees that have been infected in year t wilt and die by the next year $t + 1$, and their tree status becomes D. I ignore beetle mortality during the infection phase.

As I focus on a time scale too short for pine trees to regenerate, no reproduction of pine trees is considered in the model. Thus, the total number of pine trees remains constant. The number of pine trees of status S continuously declines as beetles make contact with healthy trees and transfer nematodes, and pine trees of status D continuously increase in number.

Spatial individual-based population dynamics

To implement spatial spread of the disease vectored by mobile beetles we need to assign each individual pine tree and beetle a position in two-dimensional space. I assume

that pine trees are configured on a two-dimensional regular lattice, i.e., the position of a tree is (x, y) where x and y are integers, with $-L \leq x, y \leq L$, and L controls the space size. I also assume that beetles move around according to a certain dispersal kernel and that movement direction is isotropic, i.e., the distance of one movement d follows a probability distribution $w(d)$ and the direction is uniformly random. After a movement, a beetle lands on a nearest-neighbor pine tree to feed for maturation to transmit nematodes or to oviposit. I also assume that the distance between two adjacent trees is the unit of length.

To exclude edge and corner effects due to the rectangular two-dimensional space, the space is assumed to be a torus; beetle movement beyond an edge results in reappearance at the opposite edge.

As the dispersal kernel, I assume a Gaussian with variance σ^2 which measures the mobility of beetles. A Gaussian kernel is best used for cases in which beetles repeat a large number of small-distance movements independently from each other. Actual beetle movements, however, would be better represented by other kernels such as a Laplacian with broader tail (Fujioka 1993; Takasu et al. 2000). Thus, the choice of Gaussian should be considered as arbitrary only for the sake of simplicity (see "Discussion"). As the unit of space is the distance between two adjacent pine trees, $\sigma = 1$ means that approximately 92% of beetles will land on 21 trees surrounding and including the tree from which they disperse (Fig. 1).

$$w(d) = \frac{1}{\sqrt{2\pi}\sigma} \exp\left[-\frac{d^2}{2\sigma^2}\right].$$

Figure 1 shows a schematic diagram of the individual-based population dynamics of the pine tree and the beetle.

It should be stressed that both the nonspatial and spatial individual-based population dynamics are stochastic because a beetle visit to a pine tree is a random event realized according to the algorithm of beetle movements described above.

It should also be noted that beetle movement is assumed to be unaffected by tree status, i.e., a beetle can land and stay on a dead tree in the present model. This is not realistic and underestimates the probability of a susceptible tree being contacted with beetles (see "Discussion").

Results

Nonspatial dynamics: how does the Allee effect emerge?

In this individual-based population dynamics, the number of adult beetles emerging from a tree, $F(n)$, is deterministically given as a function of the number of ovipositions on
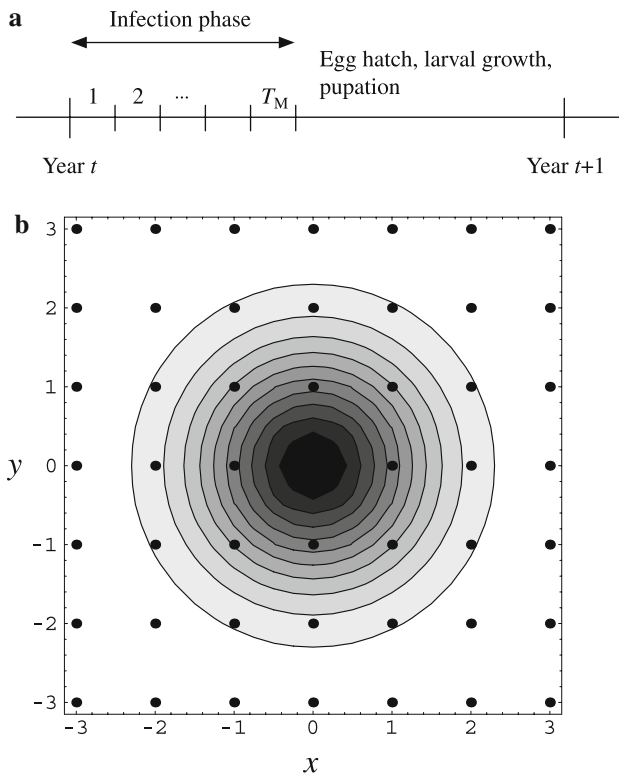
a

Infection phase

Egg hatch, larval growth,
pupation

1 2 ... T_M

Year t Year $t+1$

b

Fig. 1 Schematic diagram of the model. **a** Beetles make a movement to a pine tree at each term of the infection phase T_M times. **b** Distribution of a beetle movement released from the origin, shown with a *grayscale* from high (*darker*) to low (*brighter*) probability, and contour lines. Distance of a movement follows a Gaussian with variance σ^2, which measures beetle mobility. Movement is isotropic and a beetle lands on a nearest-neighbor tree to transmit nematodes. *Black dots* are pine trees. About 92% of beetles land on 21 trees surrounding and including the departure point (within the outermost contour)

a Number of adult beetles (P_t)
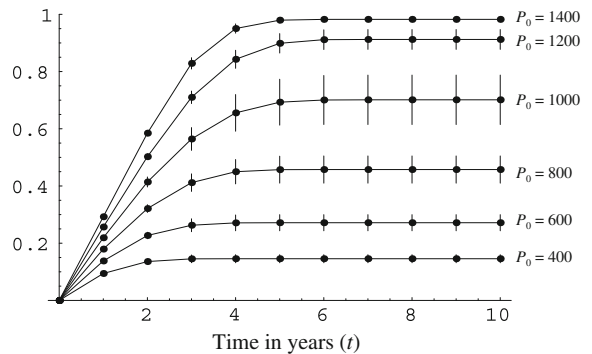

b Freq. H_t dead

Fig. 2 Population dynamics of the beetle and the pine tree. **a** Beetle population size P_t, **b** fraction of dead pines with status D. $T_M = 10$, $T_I = 5$, i.e., the infection phase is 30 days long and infected trees lose the ability for oleoresin exudation after 15 days. Initial number of beetles is $P_0 = 400, 600, 800, 1000, 1200, 1400$. Initial number of healthy pines of status S is $H_0 = 40401$ (no dead trees). Ensemble average and standard deviation for 20 trials starting from the same initial state are shown

the tree. Thus, stochasticity can arise only in the probability of a tree being visited by a beetle, which then affects the population dynamics of the beetle and the pine tree.

Typical population dynamics of the beetle and the pine tree are shown in Fig. 2 starting from a range of initial beetle numbers released when infected trees lose their ability for oleoresin exudation after $T_I = 5$ time units, (~ 15 days in the real system). The total number of pine trees is set to be large, $H_0 = 40401$ ($L = 100$), to exclude the effect of stochasticity.

Although the beetle is eventually doomed to become extinct as they continue to exploit healthy trees, they can increase in number in early dynamics when the initial number is larger than a threshold. When the initial number is less than the threshold, they decrease monotonically. This is typical of Allee effects, with disproportionate increase of the per-capita number of offspring as the population size increases.

Emergence of the Allee effect can be corroborated analytically as follows. For beetles to successfully oviposit

on and reproduce from a tree in year $t + 1$, the tree has to be contacted with beetles at least once during the first $1 \leq \tau \leq T_M - T_I$ terms in year t so that the tree gets infected and stops oleoresin exudation after T_I terms of incubation period. Such trees are ready for beetle oviposition by the last term of infection phase. With random movement of beetles, the number of beetle visits to a tree in one term of infection phase in year t follows a Poisson process with parameter $\lambda = P_t/H_t$. In the Yoshimura et al. (1999) model, $1/H_t$ corresponds to α. The probability that a pine tree can produce beetles the following year is the product of the probability that the tree was visited by beetles at least once in the first $T_M - T_I$ terms, $1 - e^{-(T_M - T_I)\lambda}$, and the probability that the pine tree was visited by beetles in the last term $1 - e^{-\lambda}$. Thus the average number of beetles in the following year is

$$P_{t+1} \propto (1 - e^{-(T_M - T_I)\lambda})(1 - e^{-\lambda})H_t \propto (T_M - T_I)P_t^2/H_t,$$

$$(8)$$

when $\lambda = P_t/H_t \ll 1$. Therefore, the Allee effect of the beetle reproduction arises due to the fact that beetles need to contact a healthy pine tree at least twice with a time lag of the incubation period.

The incubation period T_I needed for an infected tree to lose the ability for oleoresin exudation is likely to promote the Allee effect because availability of infected trees ready for beetle oviposition declines as the incubation period is increased. In other words, the incubation period can be critical, as it controls how fast an infected tree becomes ready for beetle oviposition. To see how the incubation period affects the strength of the Allee effect, the number of beetles at year $t = 1$, P_1, is plotted against the initial number P_0 for a range of incubation periods T_I in Fig. 3.

The longer the incubation period T_I, the stronger the Allee effect on beetle reproduction, and the higher the threshold for initial beetle number. No incubation period T_I means that a tree becomes ready for beetle oviposition immediately after first contact and beetles can oviposit on any tree in the last term $\tau = T_M$. In this case, no Allee effect emerges as beetles do not have to visit a tree at least twice.

Spatiotemporal dynamics: beetle dispersal and the Allee effect

First I focus on the trivial case in which infected trees immediately lose the ability for oleoresin exudation ($T_I = 0$) and beetles can oviposit on any tree. As we expect from Fig. 3, there should be no Allee effect and the beetle population will grow rapidly. Figure 4 shows snapshots of the spatiotemporal dynamics when the beetle mobility is $\sigma = 1.0$, i.e., 92% of movements are within 21 trees surrounding and including a focal tree, and ten beetles are released at the origin.

As we see in Fig. 4, after release beetles disperse and transmit nematodes to neighboring trees. The disease-damaged area of dead pines keeps expanding nearly concentrically due to the localized beetle dispersal. As the beetle population size can become large rapidly, the expansion is almost deterministic. The beetle population size increases linearly until the front of the damaged area reaches the edge of the space and all trees are dead. After having exploited healthy trees the beetle quickly declines in number and finally dies out. Distance to the front of the damaged area from the origin, roughly given as the square root of the number of dead trees, also increases linearly with time until most trees are dead (note that the spatial unit is arbitrary and chosen as the distance between two neighboring trees).

Introducing an incubation period $T_I > 0$ should suppress beetle reproduction as it limits the availability of weakened trees ready for oviposition. Figure 5 shows the
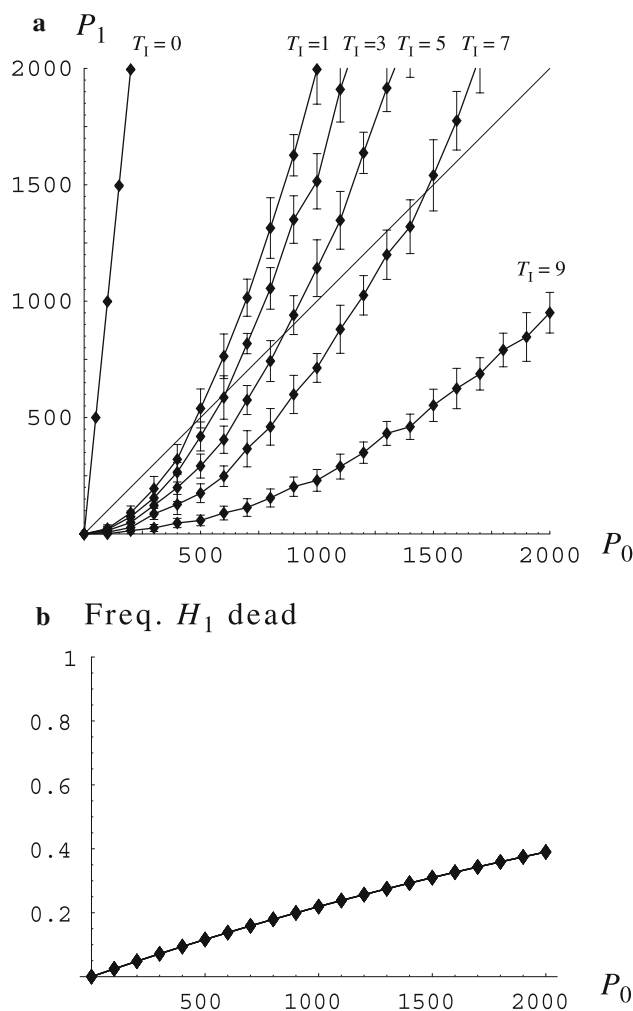

Fig. 3 a Return map of P_1 plotted against P_0 (average ± standard deviation for 20 trials). **b** Frequency of pines dead at year 1. $L = 100$, $H_0 = 40401$. $T_M = 10$, $T_I = 0, 1, 3, 5, 7, 9$. When $T_I = 0$, beetles landing on a healthy pine tree in the last term $\tau = T_M$ can infect it with nematodes, cause it to lose the ability for oleoresin exudation immediately, and oviposit, thus the per-capita reproductive rate is nearly $b/(1 + a) = 10$ because most pine trees have up to one beetle ($n = 1$). The incubation period T_I affects beetle reproduction but has nothing to do with dead pine trees H_1 because trees visited by beetles at least once eventually die by the end of the year. Thus, the frequency of dead pine trees as a function of P_0 is the same for all values of T_I

spatiotemporal dynamics when ten beetles are released with the same mobility $\sigma = 1.0$ with an incubation period of $T_I = 5$.

In Fig. 5, we see that the damaged area expands nearly concentrically only in early dynamics. Beetle population growth is much slowed compared with in Fig. 4 with no incubation period $T_I = 0$, and due to the stochasticity of tree visited by the smaller number of beetles, the front of the damaged area becomes irregular as time passes. In some trials beetles become extinct by chance before exploiting all healthy trees. The distance to the disease
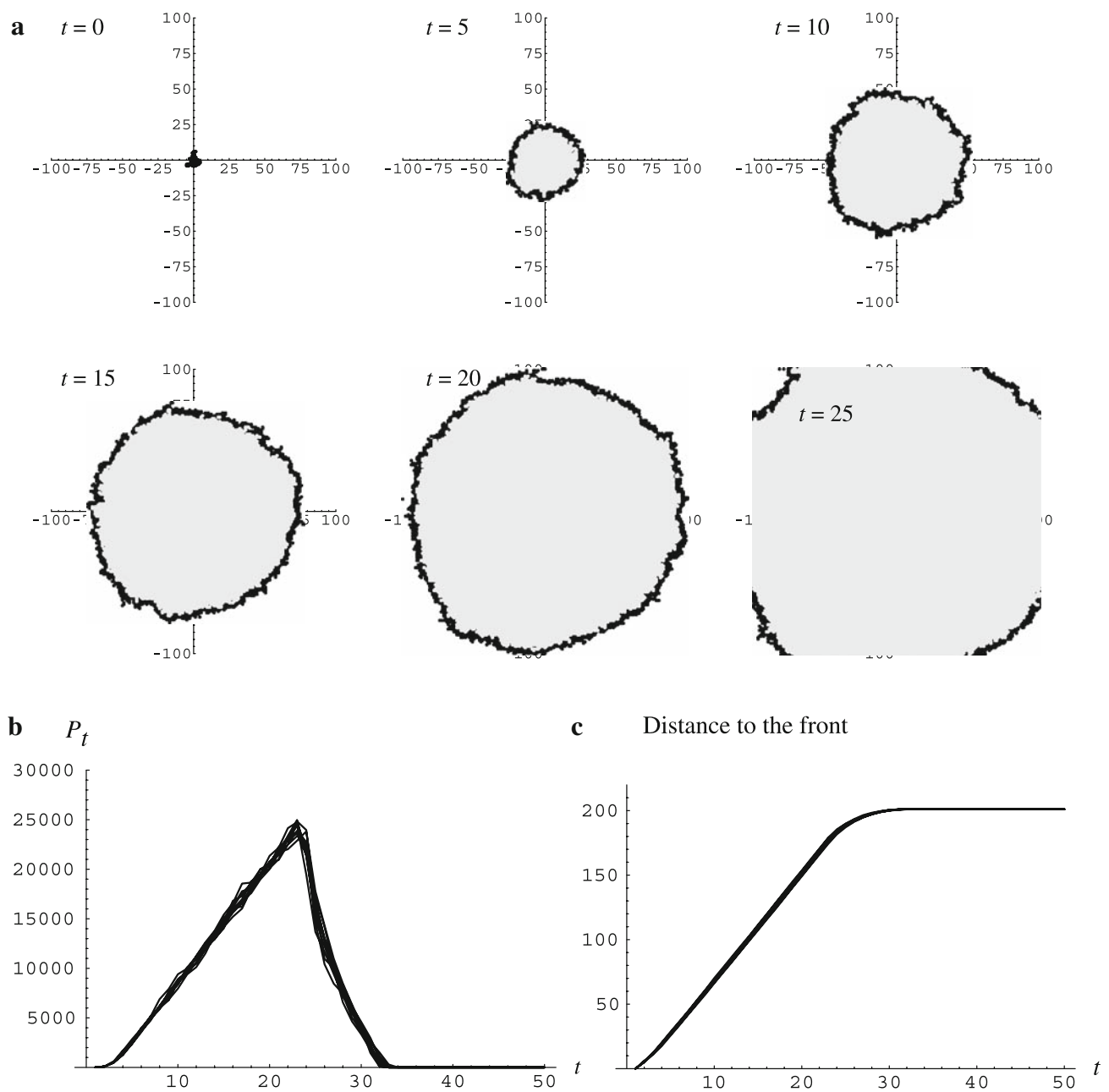
Fig. 4 Spatiotemporal dynamics of the pine trees when ten beetles are released at the origin in a pine stand of $(2L + 1) \times (2L + 1)$ trees ($L = 100$, 40401 in total). **a** Snapshots of the distribution of pine trees at the end of the infection phase are shown for $t = 0, 5, 10, 15, 20, 25$. *Black dot* is a tree of status R, gray is D. Healthy trees S are not shown. **b** Temporal change of the number of beetles P_t, and **c** square root of the number of pine trees dead as a measure of the distance to the front of the damaged area. Ten realizations are shown. Beetle dispersal distance follows Gaussian with $\sigma = 1.0$, $T_M = 10$, $T_I = 0$

front eventually increases linearly as time passes unless beetles become extinct, but the speed is much slower than the case with no incubation period.

We next focus on how beetle dispersal affects the Allee effect seen in the nonspatial model. Figure 6 shows a return map of the number of beetles P_1 plotted against initial P_0 for a range of the beetle mobilities σ.

As beetle mobility becomes smaller, beetle distribution tends to become more clumped over a smaller number of pine trees and the Allee effect becomes less obvious. Localized dispersal reduces the effective number of pine trees potentially visited by the beetle and this contributes to increase the coefficient of P_t^2, $(T_M - T_I)/H_t$, in Eq. 8. When $\sigma = 1.0$, the curve of P_1 plotted against P_0 is above the line
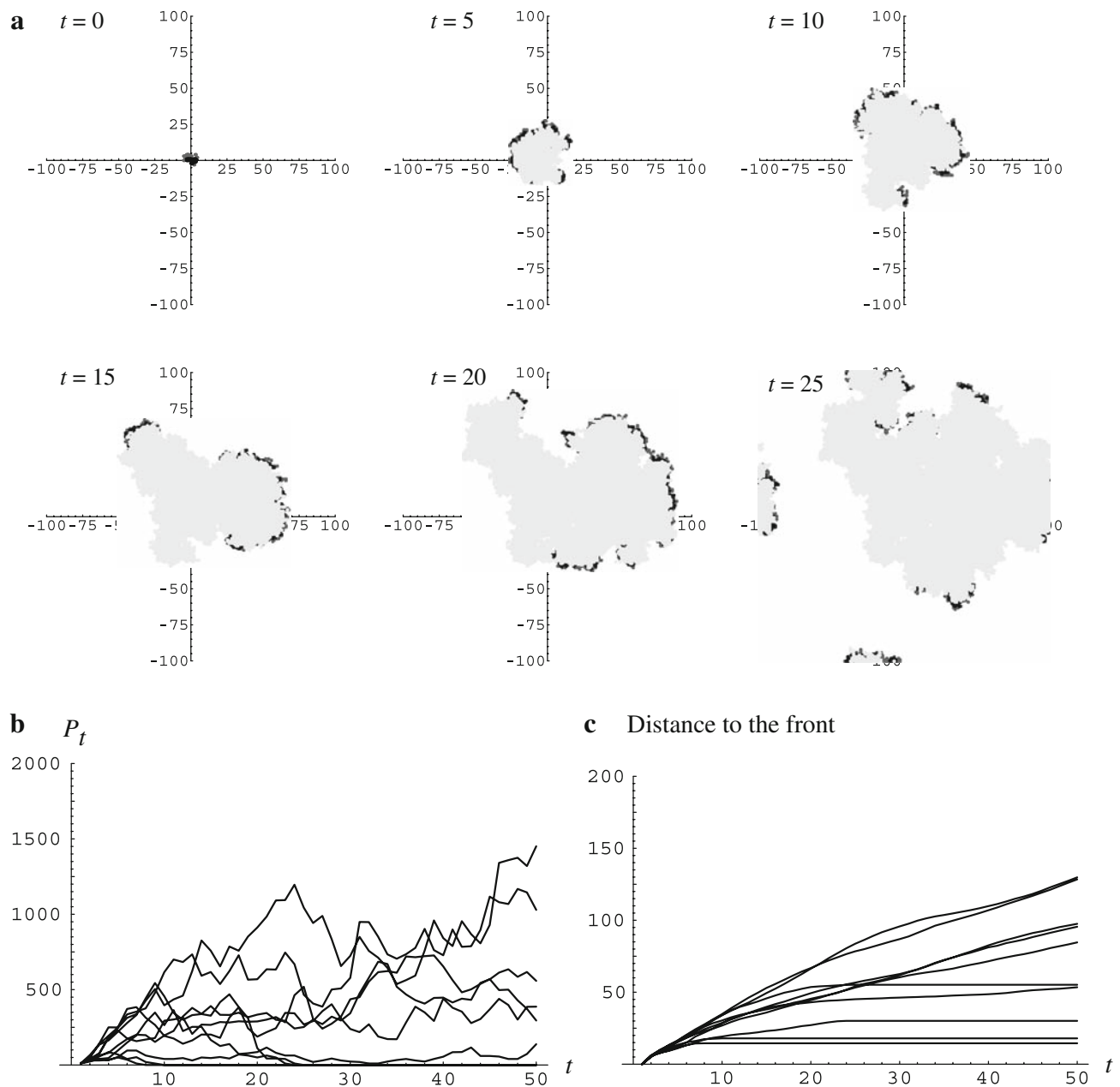
Fig. 5 Spatiotemporal dynamics of pine trees when ten beetles are released at the origin in a postulated pine stand. **a** Snapshots of the distribution of pine trees are shown for $t = 0$, 10, 20, 30, 40, 50. **b** Temporal change of the number of beetles P_t, and **c** square root of the number of dead pine trees as a measure of the distance to the front of the damaged area. Ten realizations are shown. Beetle dispersal distance follows Gaussian with $\sigma = 1.0$, $T_M = 10$, $T_I = 5$

$P_1 = P_0$ and the beetle can successfully establish itself starting from as low an initial number as ten (see also Fig. 5). The quadratic relationship of P_1 and P_0 always holds for any beetle mobility $\sigma > 0$ and gives a threshold for the beetle density below which the beetles become extinct. In the individual-based simulation, however, the threshold density makes no biological sense when it is less than 1, the unit of individual.

As the mobility is increased, on the other hand, the Allee effect starts to become conspicuous and the beetle becomes extinct unless it starts from an initial number greater than a threshold. Given a fixed number of initial beetles, larger mobility results in "dilution" of beetles per pine tree as they disperse to a wider area covering more trees, which discourages beetle reproduction because healthy pine trees are less likely to be visited at least twice by beetles. In the
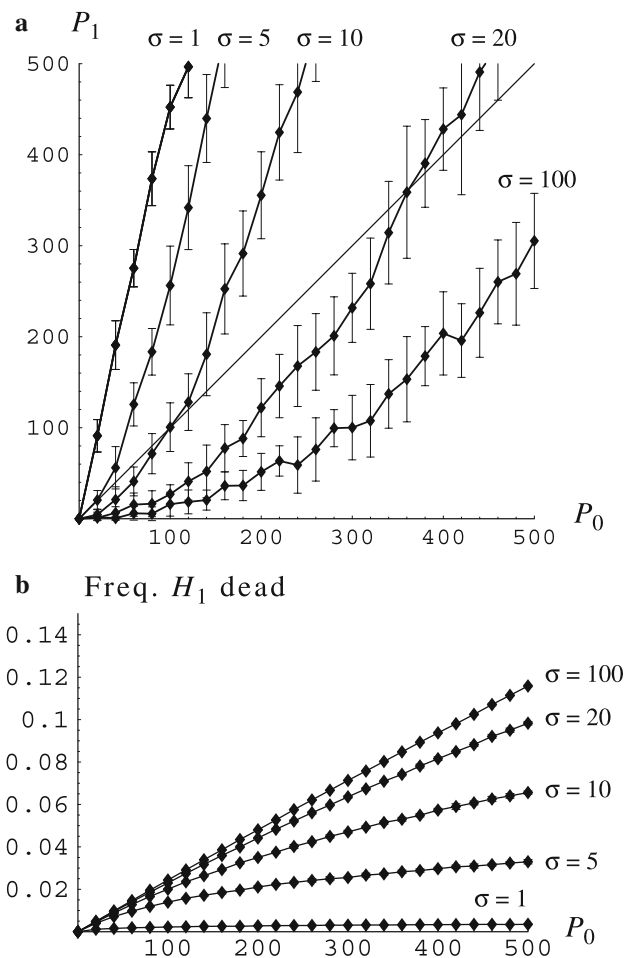
Fig. 6 **a** Return map of P_1 plotted against P_0 (average $\pm$ standard deviation for 100 trials). **b** Fraction of dead pine trees at the end of year $t = 0$ plotted against P_0 for various beetle mobilities $\sigma = 1, 5, 10, 20, 100$. $T_M = 10$, $T_I = 5$

limit $\sigma \to \infty$, all trees suffer from an equal number of beetles as a Poisson process and the return map $P_1(P_0)$ becomes the same as that of $T_I = 5$ in Fig. 3. Actually, the nonspatial population dynamics analyzed above was simulated by assuming that the landing position (x, y) of a beetle is uniformly random distributed within the range $-L \leq x, y \leq L$, which is equivalent to $\sigma \to \infty$ in the spatial population dynamics in toroidal space.

The fraction of dead pine trees monotonically increases as the initial beetle number P_0 is increased and it also positively depends on the beetle mobility; large beetle mobility results in a larger fraction of dead pine trees. Longer incubation period also results in fewer pine trees ready for beetle reproduction, which makes the beetle population small (not shown).

The irregular front and large variation of the successful beetle invasion shown in Fig. 5 results from the stochasticity of the dispersal of the finite number of beetles, which we may call stochastic dispersal.

Discussion

I revisited the nonspatial population dynamics of the beetle and the pine tree by Yosimura et al. (1999) and the spatial population dynamics by Takasu et al. (2000) with individuality retained explicitly through an individual-based model.

In the nonspatial individual-based population dynamics, I obtained qualitatively the same results as Yoshimura et al. (1999) that there is a threshold for the beetle population size, a typical symptom of the Allee effect, which is mathematically expressed as an increasing per-capita growth rate as the population size increases when it is low. I have shown, by considering mechanistic contact of a beetle and a pine tree, that the Allee effect emerges from the fact that beetles have to make a second contact with pine trees to reproduce successfully. I have also shown that the incubation period after which an infected tree loses the ability for oleoresin exudation is critical in determining the strength of the Allee effect. Increasing the incubation period does not help to rescue pine trees already infected but it may be quite effective in controlling the beetle population as it induces a strong Allee effect in the beetle dynamics.

I then extended the nonspatial population dynamics to explore spatiotemporal dynamics by explicitly considering beetle mobility in a two-dimensional space. I showed that the Allee effect can emerge as we increase the beetle mobility and the length of the incubation period in the spatially explicit model, but that localized beetle dispersal hinders the emergence of the Allee effect; the beetle can successfully establish starting from a small initial number when beetle mobility is low. This suggests the importance of the spatial scale over which beetles disperse.

Although I made several simplifications to make the model as easy to simulate as possible with limited computation power, any mechanistic process can be readily embedded in the model and simulated on an individual basis.

In the spatial model I assumed that pine trees are configured on a regular lattice grid. However, relaxing this constrain is straightforward to allow pine trees to be distributed in a continuous space as realistic as in the real system based on a vegetation map of a focal regional area.

Beetle movement was described by a Gaussian dispersal kernel, which means that almost all movements are locally clustered, roughly within a distance proportional to the standard deviation σ from a departure tree. However, any functional form based on an empirical estimate could be implemented. If the dispersal kernel is given as Laplacian that one movement distance follows an exponential distribution with broader tail than Gaussian, we expect it to be more difficult for the beetle to establish successfully

because of amplified dilution of beetles per tree in the distribution tail, and this effect would be enhanced if the initially introduced population were smaller in size. Assuming realistic mechanistic interactions and using parameter values which can be empirically estimated will help to predict how pine wilt disease will expand in the future and shed light on practical control measures to stop pine wilt disease expansion. Further empirical study together with development of a detailed tailor-made individual-based model warrants future research.

It was also assumed that tree status does not affect beetle mobility and that beetles can land and stay on dead trees. This simple algorithm is likely to underestimate the chance of a tree being contacted by beetles. Actually, beetles are attracted to ethanol and monoterpenes, which weakened trees emit (Ikeda and Oda 1980). Implementation of this beetle movement is needed to understand fully the spread of the disease in relation to beetle dispersal distance.

It has long been suggested that difficulty in finding a mate at low population density in sexually reproducing species is the most prevalent mechanism that gives rise to Allee effects (Dennis 1989; Boukal and Berec 2002). Detailed processes have been analyzed using realistic individual-based model (Berec and Boukal 2004). The beetle, of course, reproduces sexually but the present study suggests another important mechanism responsible for Allee effects: that a tree has to be contacted by beetles at least twice within a time span. Stephens and Sutherland (1999) suggested "modification of the environment" as a potential mechanism of Allee effects. In the context of the mutually beneficial lifecycles of the beetle and the nematode, the beetle modifies the pine tree as a reproductive resource with the help of the nematode to enhance reproduction.

Apart from the empirical consequences mentioned above, analysis of the spatially explicit individual-based model points to future modeling research. Conventional models of population dynamics in spatial ecology focus on temporal change of population density as a nonnegative real-valued quantity. Such models are analytic as they are described as a dynamical system and various mathematical techniques are available to investigate their behaviors. These models, however, completely ignore the individuality of discrete entities.

In conventional modeling of spatiotemporal population dynamics in continuous space and discrete time, we first derive a local population dynamics given in the form

$$N_{t+1} = F(N_t)N_t, \tag{9}$$

where $F(N)$ is per-capita growth rate as a function of population density N. Then we introduce a dispersal kernel with assumptions that (1) the local dynamics runs at every spatial position at x, and (2) individuals disperse according

to the kernel $w(x, y)$ as the probability that an individual at x moves to y.

Assumption (1) is translated mathematically as

$$N_{t+1}(x) = F(N_t(x))N_t(x) \tag{10}$$

and assumption (2) is given as

$$N_t'(x) = \int w(x, y)N_t(y)\mathrm{d}y, \tag{11}$$

where the prime denotes population density after dispersal. We then combine the local dynamics and the dispersal as an integro-difference equation

$$N_{t+1}(x) = \int w(x, y)F(N_t(y))N_t(y)\mathrm{d}y \tag{12}$$

if dispersal takes place after local dynamics within year t.

Once we have combined the local dynamics with a certain mode of dispersal as the integro-difference equation, mathematical tools are available to predict the speed of the range expansion (Neubert et al. 1995, 2000; Kot et al. 1996; Clark 1998; Wang et al. 2002). The functional form of the dispersal kernel, especially the form of the distribution tail, is crucial to determining the expansion speed (Kot et al. 1996). Shigesada and Kawasaki (1997) gave a comprehensive review on modeling of biological invasion. Hastings et al. (2005) reviewed recent developments in theory and practice.

Individual-based models, on the other hand, can implement mechanistic processes at the individual level in a very realistic way. Birth–death processes of individuals in space are readily described as a set of algorithms, and these are inherently stochastic. Although such algorithmic models are easy to simulate, it is not easy to extract meaningful information from the noise of stochasticity and it is usually difficult to understand the model's behavior as a whole, such as parameter dependency, etc. Bridging mathematically tractable analytic models and mechanistic individual-based model is a challenging task in theoretical population biology, i.e., how algorithmic descriptions of birth and death of individuals can be translated into analytic models (Dieckmann et al. 2000; Law et al. 2003; Kot et al. 2004). Further theoretical study that retains individuality is needed.

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