Multiscale analysis of compartment models with dispersal†

Yun Kang* and Carlos Castillo-Chavezb,c,d

aApplied Sciences and Mathematics, Arizona State University, Mesa, AZ 85212, USA; bMathematical, Computational and Modeling Sciences Center, Arizona State University, Tempe, AZ 85287-1904, USA; cSchool of Human Evolution and Social Changes and School of Sustainability, Santa Fe Institute, Santa Fe, NM, 87501, USA; dBiological Statistics and Computational Biology, Cornell University, Ithaca, NY 14853-2601, USA

(Received 7 March 2012; final version received 2 July 2012)

The characterization of the population dynamics of animal populations and dispersal provides the underlying setting of this article. Novel results emerge from our exploration of the role of disease in this context. We focus on the study of the impact of dispersal on the dynamics of populations that account for (a) induced Allee effects; (b) disease dynamics; and (c) spatial heterogeneity, using deterministic and stochastic models. Specifically, the models incorporate disease-driven effects on the individuals’ competitive ability to acquire resources as well as on their ability to move or reproduce. The results bring to the forefront the role of initial conditions and patch quality as well as ‘topological’ structure or connectivity landscape structure (the physical space where individuals move, reproduce, get sick, die, or compete for resources). The emphasis is placed on the dynamics of populations when disease is an important selective force. This article surveys the appropriate literature while including original research.

Keywords: Allee effects; competition; epidemics; infectious disease; reproductive fitness; metapopulation model; multiple interior equilibria; bifurcation; catastrophe; mathematical biology; conservation biology; sustainability

AMS Subject Classification: Primary: 37B25, 39A11, 54H20; Secondary: 92D25

1. Introduction

Differential patterns of dispersal are often responsible for generating and maintaining the variation required for evolution to operate over multiple organizational scales. Dispersal is a selective mechanism capable of re-shaping or maintaining community structure [34, 55, 77, 100]. The portfolio of survival mechanisms available to organisms includes a drive to colonize accessible or occupied habitats. Successful dispersers increase their sphere of influence or expand the size of their niche through the ‘sharing’ of vulnerable habitats: those occupied by ‘weak’ or ‘fragile’ populations [32, 108]. Colonization influences species diversity, plays an important role in the ability of a population to persist, and often determines a population’s size (abundance). In this
article, dedicated to our grand mentor, Simon Levin, the impact of dispersal in communities, mathematically defined or characterized via metapopulation models [55], is discussed under a mathematical framework that includes disease transmission. Exploring the role that disease, a selective force, plays in the life-history dynamics, including its impact on the competitive ability, of the populations involved [12,20,22] is one of the goals of this article.

Dispersal, often defined as the movement of organisms away from particular reference places, is ubiquitous across and over multiple spatial and temporal scales and yet the combined effects of disease and dispersal have not been systematically factored in. Disease can affect the rate of movement of individuals, or weaken resident populations, or diminish the ability of individuals to repel invaders, or minimize their effectiveness in competing for resources. Species mobility has generated changes in community structure as a result, for example, of the sudden arrival of competing species, the kind of events that may lead to the extinction of native subpopulations [119,121]. The dynamics of populations, connected by dispersal, are complex and adaptive, with dispersal often playing a defining role across multiple levels of organization, from bacteria to animals, including populations of organisms that spend most of their life cycle in sessile forms such as plants and fungi [97]. The overall role of dispersal and its impact on the world that we live in are articulated in compelling terms in Simon Levin’s *Fragile dominion: Complexity and the commons* (1999). Levin [80], on the issue of colonization, observes that

> History has restricted the composition of any ecological community, as well as the composition of its neighbors. When opportunity for colonization presents itself within an ecosystem, a limited set of colonists apply for admission; it is a case of being in the right place at the right time. And from those applicants, only those that can integrate themselves into the existing community will succeed. Pest species find such opportunities, usually with the help of humans, when they get to places where they have not been before...

> When broad-scale disturbance occurs, as, for example, following a volcanic lava flow, colonists are drawn from a larger geographic area. Still, there are strict rules governing the successional recolonization of the newly exposed surfaces... Within the constraints set by environment, history, and the composition of neighboring environments, ecological communities self-organize according to a roughly repeatable set of rules. The interplay of all of these factors provides us with insights into why certain communities exist; the challenge is to determine the relative importance of these various influences. (p. 48)

Dispersal shapes plant communities, determines invasion outcomes, alters the dynamics of infectious agents, and disentangles the dynamics of biological systems such as the marine open-ocean and intertidal systems. Dispersal is of interest to nearly every scientist working on questions where individuals’ mobility, across heterogeneous landscapes, is likely to give organisms an edge. The study of dispersal has naturally provided challenges and opportunities to researchers interested in the generation of useful and simple macroscopic mathematical descriptors of the dynamics of heterogeneous large ensembles of individuals.

Biologically inspired mathematical and modelling work involving dispersal can be traced back to the dazzling research of pioneers such as Fisher [42], Kolmogorov *et al.* [70], Skellam [104], and Kierstad and Slobodkin [69]. The classical work of Aronson and Weinberger [9,10], Hadeler [52,53], Hadeler and Rothe [54], Levin [77,79], Levin and Paine [82], Okubo [94], Weinberger [114,115], and Okubo and Levin [95] as well as the fascinating research developed by a large cadre of distinguished mathematicians and theoreticians interested in questions related to this aspect is central to the field of *population biology*. In today’s world, the mathematical and modelling literature on dispersal is routinely put to use, constantly revised, adapted, or modified in the search for solutions to questions intimately connected to the study of complex adaptive dynamical systems: efforts perhaps best exemplified by research aimed at understanding the dynamics of socio-biological and economic sustainable systems [27,41,80,81]. Levin [80] puts forward the value of the holistic vision inherent to the study of *Complex Adaptive Systems* when, in the context of successional processes in ecosystems, he observes that
The ecosystem is a complex adaptive system whose development over a period of years involves selection among its components over much shorter periods of time, while also reflecting other evolutionary processes that have occurred over much broader space and longer time scales. A forest is a complex of species, some adapted to the slow growth and eventual dominance of the canopy, others adapted to rapid exploitation of the temporary gaps that form. When a tree dies in the forest, or when a cluster of trees is felled by windthrow, a local successional process begins. The early colonists are adapted for rapid dispersal and growth; these include species such as grasses, forbs, and shrubs that require and thrive on lots of sunlight. As the forest develops, light availability in the understory diminishes, and these species are replaced in succession by those that make their living not by opportunism but by their ability to grow under low-light conditions and that hence will prosper in a highly competitive environment.

Is the forest landscape so different in this way from the economic landscape, in which opportunistic entrepreneurs leap in to exploit newly available possibilities, ultimately to be replaced by other companies better suited for the long haul?

Ecologically, specific traits adapt a species for exploiting a particular ecological niche; evolutionarily, the availability of new ecological opportunities creates pressure favoring their exploitation. Evolution hates a vacuum. That is not to say that every niche will be filled, or that a list of niches arrives in the same box with a newly minted ecosystem, with instructions on how to assemble... As a system develops, new opportunities are created as parasites or predators find new hosts and prey to exploit, or as pollinators and hosts develop their mutual interests. In this way, ongoing evolution changes the adaptive landscape for other species; this is a particular manifestation of the system’s nonlinearity, and it reinforces the importance of historical accidents that irrevocably influence the system’s later development.

Over the evolutionary time scale, a similar dynamic applies to ecosystem types; a hardwood forest in the northeastern United States, for example, is just one unit of a meta-forest, a collection of forests with similar but not identical compositions. Following a major disturbance, the reassembly of a forest is not a novel occurrence; it takes place within the context of that meta-forest, which provides the evolutionary background. The evolution of the northeastern forests as a group, in turn, represents the collective dynamics of a multiplicity of individual forests.

The interplay between ecological and evolutionary processes is central to understanding the emergence of biodiversity. Forest evolution involves processes identical in kind to those that determine the characteristics of the species that make a living on lava flows, or the intertidal invertebrates that exploit wave-induced gaps in mussel beds. (pp. 49–51)

The near absence of theoretical or mathematical work that explicitly incorporates the impact of disease on individuals and populations (but see \cite{12,20,22,99}) has restricted our understanding of the role that disease plays in the evolution of populations and communities. The overarching goal of this article is to identify the components of a programme that explores the joint role of disease and dispersal in shaping the dynamics of populations. Dispersal for the purposes of this article will be thought of as a dynamic descriptor that captures the macroscopic movement of spatially distributed individuals and it will be modelled in a rather simplified manner, a metapopulation framework. We follow the approach reported in Castillo-Chavez and Yakubu \cite{20}, Castillo-Chavez \textit{et al.} \cite{22}, and Berezovskaya \textit{et al.} \cite{12,14} in the rest of the article.

This article is organized as follows: Section 2 reviews briefly some of the literature on dispersal models and introduces a single-patch model that incorporates Allee effects as well as the impact of infectious, sometimes fatal, pathogens; Section 3 extends the single-patch framework described in Section 2 to one that allows for multi-patch deterministic network models; Section 4 explores a particular stochastic version of the models introduced in Section 3 using interacting particle system approaches; Section 5 presents some of our thoughts and conclusions on disease and dispersal.

2. Modelling dispersal: single-patch models

Mathematicians and theoretical and computational biologists have carried out studies on the effects of dispersal on the life-history dynamics of populations through the formulation and analyses of systems of nonlinear coupled differential equations (ODEs), or nonlinear reaction–diffusion or nonlinear integro-difference equations, or interacting particle systems \cite{5–7,18,23,24,31,32,35,36,42,70,71,77,78,83–85,88,92–94,100,108,119–121}.
The impact and usefulness of metapopulation models, models of the dynamics of populations of subpopulations occupying spatially separated patches [75,76], continue to rise. Metapopulation models are now routinely used to address questions and/or hypotheses that include (i) the belief that a population’s persistence may be achievable under scenarios where otherwise (single-patch models) extinction is the most likely outcome [116] and (ii) efforts to validate hypotheses using models or expanded paradigms that incorporate community elements ignored in ‘classical’ set-ups.

Animal dispersal or movement is described macroscopically as the outcome of stages that include (i) the individual’s ability to disperse (patch departure); (ii) the individual’s invasion ability, that is, getting established, avoiding predation, surviving disease, or living with minimal resources (patch quality); and (iii) the population’s ability to expand and overtake new habitats. Determining the specific role of animal dispersal is rather complex since dynamic changes are most often generated in response to challenges faced by populations living in evolving plastic heterogeneous landscapes [2,3,26,32,39,40,50,63,108,112,117]. From a practical perspective, disentangling the dynamics that emerge from the interactions of disease and dispersal will help us understand, identify, test, develop, and improve management practices that are essential to the survival of natural populations, including rare and officially endangered species or communities [68].

Understanding the establishment, spread, or shrinking of populations subject to predation, mating limitations, and disease is handled in this article, with a class of deterministic patch models and some stochastic ‘counterparts’: models that simultaneously incorporate features often ignored, which include (a) induced Allee effects; (b) disease co-invasions; and (c) spatial heterogeneity. The nature of the dynamical interactions between some of the processes and factors noted above has not been studied fully. However, it is known that such interactions often lead to complex dynamical outcomes [12,14]. Stochasticity, a topic discussed later in this article, also plays an important role, particularly during the invasion process.

Theory brings tools that help us understand some of the aspects responsible for the complex dynamics that may emerge when specific details are incorporated into classical models. The study of the co-evolving dynamics of disease and dispersal is carried out using expanded deterministic and stochastic frameworks. The deterministic multi-patch models introduced, systems of nonlinear ordinary differential equations, account for the changes in the density of healthy and infected individuals within each patch, the result of local interactions (disease and competition) and global processes (dispersal). A stochastic version of our models, using the framework of interacting particle systems (individual-based models), is introduced later.

The ecological dynamic of a single species, in the absence of dispersal and disease, is described by \( N' = G(N) N - H(N) \), where \( N(t) \) denotes the population density at time \( t \) and \( G \) models the logistic component of population growth. That is, \( G(N) = r - bN \), where \( r \) denotes the per-capita intrinsic growth rate and \( b \) is the extra mortality caused by intra-specific competition. The function \( H \) models density-dependent demographic factors: a component Allee effect, the positive relationship between any measurable component of individual fitness and population density.

Ecologists have noted that species’ component Allee effects may include decreases in reproduction due to a shortage of mating encounters at low species densities or decreases in mortality due to weakening predation risks at high densities [29,65,102,105]. Taking \( H(N) = cN/(N + a) \) means that \( H \) corresponds to the assumption of a negative exponential distribution of mating encounters [28] or to the use of a predator’s Holling type II functional response [17,89]. These assumptions lead to the following single-species population model with component Allee effects:

\[
N' = N(r - bN) - \frac{cN}{N + a} = r \left( N - \frac{N}{K} \right) - \frac{cN}{N + a},
\]  

(1)
where $K = r/b$. If $r^2(K + a)^2 - 4rcK \geq 0$, Equation (1) can be rewritten as

$$N' = \frac{rN}{N + a} \left( N - \frac{r(K - a) - \sqrt{r^2(K + a)^2 - 4rcK}}{2r} \right) \times \left( \frac{r(K - a) + \sqrt{r^2(K + a)^2 - 4rcK}}{2r} - N \right).$$

Expression (1) is dynamically equivalent to the simplest single-species generic model with strong Allee effects, that is, to

$$N' = rN(N - \theta)(1 - N),$$

provided that

$$\min\{K, c\} > a \quad \text{and} \quad r^2(K + a)^2 - 4rcK \geq 0.$$  

Here, $\theta$ is an Allee threshold, which, after rescaling, must live between 0 and 1. The threshold ($\theta$) determines whether the population goes extinct or becomes established. Otherwise, Model (1) has weak Allee effects; that is, there is no critical threshold. We will discuss SI (susceptible–infected) models using either component Allee effects (1) or (heuristic) strong Allee effects (2). Here, we would like to point out that whether component Allee effects can lead to strong Allee effects or not depends on their strength. For example, Model (1) has component Allee effects that become strong Allee effects when inequalities (3) are satisfied. The analysis of the population dynamics of this single-patch SI model is carried out so as to generate baseline results, that is, a ‘classification’ of outcomes in the absence of dispersal and disease. These baseline results will be used to guide studies on the joint dynamics of disease and dispersal.

2.1. An SI model with strong Allee effects and disease-adjusted fitness

Macroparasitic and microparasitic infections are important regulators of natural populations [8]. Disease has a cost that often translates in reductions in the host’s fitness [49,73,74]. Differences in individuals’ fitness that arise from the cost of dispersal may lead to drastic changes in the overall dynamics of a population. It has been shown, for example, that dispersal can increase the number of population attractors, which brings up dependence on initial conditions [12,13,20]. Kang and Castillo-Chavez [64] introduced and analysed a minimal SI model with strong Allee effects, a phenomenological way of incorporating the fragility associated with small population sizes, in order to study the role of disease in a population’s fitness. The study is carried out under a model that a priori assigns higher fitness (increased reproduction and higher ability to acquire resources) to individuals in the S-class (in other words, infections are assumed to have a cost). Kang and Castillo-Chavez [64] have shown, for example, that fitness reduction factors lead to outcomes that cannot emerge from modelling protocols that ignore the impact of disease.

An outline of the typical results generated with a minimal SI model (4) that incorporates Allee effects, disease-dependent reproduction, and disease’s impact on the competitive ability of infected individuals can be found in [64]. Here, we use a system of nonlinear differential equations that describes the rates of change of the populations of susceptible and infected individuals at any time $t$ ($0 \leq t$) to describe some of the results found in [64]:

$$\frac{dS}{dt} = f(S, I) = \begin{cases} 0 & \text{if } S = 0 \text{ and } r \rho I(\alpha_1 I - \theta)(1 - \alpha_2 I) \leq 0 \\ r(S + \rho I)(S + \alpha_1 I - \theta) \times (1 - S - \alpha_2 I) - \beta SI & \text{otherwise} \end{cases}$$

$$\frac{dI}{dt} = \beta SI - dI.$$
where $S$ denotes the normalized susceptible and $I$ the infected populations relatively to $S$. ‘Relatively’ means that if the carrying capacity for susceptible individuals is $K$, then we use re-scaled variables with the same names, that is, $S/K \rightarrow S$, $I \rightarrow I/K$. Here, all the parameters are non-negative, with $\rho$ ($0 \leq \rho \leq 1$) describing the reduction in the reproductive ability of infected individuals; $\alpha_i$ ($0 \leq \alpha_i \leq 1$) $i=1,2$ capturing the competitive ability of infected individuals as a function of the total population size; $r$ representing the maximum per-capita birth rate of the species; $d$ modelling the per-capita death rate of infected individuals (a parameter that includes disease-induced deaths); $\theta$ ($0 < \theta < 1$) denoting the Allee threshold (for the normalized susceptible population); and $\beta$ representing the per-capita disease transmission rate.

The case of no infection ($I=0$) reduces Model (4) to $dS/dt = rS(S - \theta)(1 - S)$. We have shown that Equation (4) supports rich dynamics including the possibility of multi-stability (hysteresis, see Figure 1(a)), saddle node and Hopf bifurcations, and catastrophic events (disease-induced extinction, Figure 1(b)). From Figure 1(b), it can be seen that within a certain range of values for the basic reproduction number, $R_0$ [values ($R_0 = \beta/d \in (5.6, 6.2)$)], there is no, asymptotically speaking, disease dynamics, since the population experiences ultimate extinction. Thus, decreasing the value of $R_0$, starting with $R_0 = 6.2$, may result in the extinction of the species. We can see, perhaps unexpectedly, that high disease rates may, in some instances, guarantee the survival of a population. Our analyses suggest that current efforts to quantify species’ management efforts, which most often rely on approaches aimed at reducing the disease’s basic reproduction number, need to be re-assessed.

The work by Diekmann and Kretzshmar [33], researchers who studied the dynamics of two simple models for microparasitic and macroparasitic diseases but without Allee effects, is relevant since both models assume that (i) infectives experience reduced fertility due to the disease and (ii) the infection rate is an asymptotically homogeneous function. The microparasitic infectious disease model is given by the following system:

$$\frac{dS}{dt} = \beta \frac{(S + \xi I)^2}{S + I} - \mu S - \frac{\kappa SI}{c + S + I}$$
$$\frac{dI}{dt} = \frac{\kappa SI}{c + S + I} - \mu I - \alpha I.$$  (5)

If we let $H$ denote the host and $P$ the parasite populations, then the macroparasitic dynamic model is given by the following system:

$$\frac{dH}{dt} = -\alpha P - \mu N + \beta N \left( \frac{kN}{(1 - \xi)P + kN} \right)^k$$
$$\frac{dP}{dt} = P \left( \frac{\kappa N}{c + N} - (\mu + \alpha + \sigma) - \frac{(k + 1)P}{kN} \right),$$  (6)

where $\beta$ is the per-capita natural birth rate; $\mu$ is the per-capita natural death rate; $\alpha$ is the additional mortality rate caused by the disease; $\xi \in [0, 1]$ is a parameter describing the reduction of fertility of an infected individual due to the disease; $\kappa$ is the contact rate between infectives and susceptibles; $c$ is a constant; and $k$ is the clumping parameter. Diekmann and Kretzshmar [33] provided bifurcation diagrams involving several threshold values of the contact parameter $\kappa$ to show how $\kappa$ affect the dynamical patterns. The precise form of the bifurcation diagrams depends critically on the fertility of infectives. Their analysis also shows that for certain ranges of parameter values, a bistable behaviour occurs: either the population grows exponentially or it oscillates periodically with a large amplitude.
Figure 1. Bifurcation diagrams for System (4) highlighting the number of interior equilibria and their stability. The red colour indicates that the interior equilibrium is a source; the blue colour indicates that the interior equilibrium is a sink; and the green colour indicates that the interior equilibrium is a saddle. (a) $R_0^\alpha = \theta \beta / d < 1$: Bifurcation diagram of $\alpha_1$ when $r = 2.35, \alpha_2 = 0.01, \theta = 0.2, \rho = 0.8$ and $d = 0.85$ and (b) bifurcation diagram of $d$ when $r = 6, \beta = 1, \theta = 0.18, \rho = 0.1, \alpha_1 = \alpha_2 = \rho$. 

Infected population

0 0.1 0.2 0.3 0.4 0.5 0.6 0.7 0.8 0.9 1

(a) $r=2.35; \theta=0.2; \rho=0.8; \alpha_2=0.01; \beta=1; d=0.85$

(b) $r=6; \rho=0.1; \theta=0.18; \alpha_1=\alpha_2=\rho; \beta=1$

d=\theta=0.18
2.2. General SI models with component Allee effects and disease selection pressures

The analyses of the SI model subject to Allee effects suggest that the incorporation of fitness reduction factors will naturally lead to outcomes that challenge the use of standard modelling protocols in the study of disease dynamics in non-domestic animal populations [64]. The following theoretical framework incorporates the following, often not included, factors:

(a) a disease that may affect the survival, reproduction, and competitive ability of infected individuals and
(b) density-dependent reproduction via a component Allee effect, which begins to remedy the limitations that arise from the inherent oversimplicity of the existing disease models.

Thus, the generalized SI model framework is given by the following nonlinear system:

\[
S' = \frac{B(S,I)P(S,I)}{N} - A(N) S, \\
I' = \Phi(S,I) \frac{S}{N} - \left(\frac{\alpha_2 A(N)}{\Phi_1(N)} + D(N) + \frac{\mu}{\Phi_1(N)}\right) I,
\]

where \(S(t)\) and \(I(t)\) denote, respectively, the susceptible and infected populations. Furthermore, \(N(t) = S(t) + I(t)\) denotes the total population at time \(t\), \(B(S,I)\) is the maximum reproduction rate when the resource is plenty (e.g. male population or food), and \(P(S,I)\) is the probability that a female individual mates during the reproductive period (or the ratio of available resource). In short, \(B(S,I)P(S,I)\) gives the birth term in the presence of disease; \(A(N)\) models the component Allee effect with \(\alpha_2\) capturing the disease effects; \(D(N)\) denotes the death rate, and \(\Phi(S,I)\) the force of infection. The condition \(B(0,0)P(0,0) \geq 0\) ensures that model (7) is positively invariant in \(\mathbb{R}_+^2\). We can see in System(7) that \(I = 0\) is an invariant manifold, that is, \(I(0) = 0\) implies that \(I(t) = 0\) for all \(t \geq 0\). However, \(S = 0\) may not be invariant due to the reproduction efforts of infected individuals. For example, we have \((dS/dt)|_{S=0,I>0} = B(0,I)P(0,I) > 0\) if infected individuals do reproduce. This model can be used to address questions such as

(i) How will component Allee effects affect disease dynamics?
(ii) How will different Allee threshold intensities (tipping points) affect population persistence and extinction when System (7) integrates an Allee threshold?
(iii) How will differences in disease transmission rates, \(\Phi(S,I)\), affect disease dynamics and the overall population dynamics?

The incorporation of Allee effects makes sense for many reasons. Some ecologists have used them to account (phenomenologically) for situations when a specie experiences mating limitation or is subject to predation by a generalist predator, with a saturating functional response [16,25,102]. The appropriate selection of \(B(S,I), P(S,I), D(S,I),\) and \(\Phi(S,I)\), in the context of System (7), accounts for the role of differential component Allee effects and alternate disease transmission rates in the system dynamics.

An SI model with a component Allee effect that sets \(B(S,I) = r(S + \rho I), P(S,I) = 1 - (S + \alpha_1 I)/K, A(N) = eC/(a + N), D(N) = d,\) and \(\Phi(S,I) = \beta IN\) is given by the following
nonlinear system:

\[
S' = \frac{r(S + \rho I)}{\text{reproduction effort}} \left(1 - \frac{S + \alpha_1 I}{K}\right) - \frac{d}{\text{the death rate}} S - eC \frac{S - \beta}{a + S + I} SI,
\]

where \(K\) denotes the carrying capacity; \(\rho \ (0 \leq \rho \leq 1)\) models the reduction in reproductive ability of infected individuals, with \(\rho = 0\) meaning no reproductive ability and \(\rho = 1\) meaning no impact on reproductive fitness; \(\alpha_1 \ (0 \leq \alpha_1 \leq 1)\) denotes the competitive ability of infected individuals relative to that of susceptible individuals; \(\alpha_2 \ (\alpha_2 \geq 0)\) represents the ability of infected individuals to avoid/escape predation, relative to that of susceptible individuals; \(r\) denotes the maximum birth rate; \(d\) denotes the death rate in the absence of disease; \(C\) denotes a constant predator population modelled via a Holling type II functional response; \(e\) scales the predator encounter rate of prey; \(a\) models predator saturation; and \(\beta\) is the disease transmission rate.

The formulation of Model (8) helps us to explore the effects of disease on fitness. This is done so by comparing the results of taking \(\rho = \alpha_1 = \alpha_2 = 1\) versus those generated when the \(\alpha\) values differ. The analysis of the dynamics can be used to contrast the dynamics of the disease-free system versus those when the disease prevails. These analyses can be carried out in the presence of weak Allee and strong Allee effects. The analysis and simulations of Model (4) [64] show that System (8) supports rich dynamics including but not limited to the existence of multiple interior attractors, saddle node, Hopf bifurcations, catastrophic events, and disease-driven extinction scenarios.

2.3. Single-patch model with Allee effects and disease: a brief review of the literature

We now briefly review the work carried out by researchers on models that incorporate the joint impact of Allee effects and disease. Throughout, we let \(S(t)\) be the susceptible population size, \(I(t)\) be the infected population size, and \(N(t) = S(t) + I(t)\) be the total population size at time \(t\).

Deredec and Courchamp [30] introduced an SI model, System (9), in order to compare the impact of the presence of Allee effects on disease dynamics under the following assumptions: (1) the host population occupies a constant area; (2) the reproductive capacity of an individual is not affected by its infective state; (3) disease transmission is horizontal (juveniles are born susceptible) and behavioural (through contacts between individuals); (4) the population dynamics face strong Allee effects already in the absence of disease; and (5) infected individuals die from infection at the rate \(\alpha\). Their model reads as follows:

\[
\frac{dS}{dt} = b f(N) N - mf(N) S - \beta \frac{SI}{N} \phi(N),
\]

\[
\frac{dI}{dt} = \beta \frac{SI}{N} \phi(N) - mf(N) I - \alpha I,
\]

where \(b = r - m\) with \(r, b, m\) denote the intrinsic growth, birth, and death rates, respectively. The case with no Allee effects takes \(f(N) = (1 - N/K)\); otherwise, we take \(f(N) = (1 - L/N)\).
\(1 - N/K\) with \(K\) denoting the carrying capacity and \(L\) the Allee threshold, below which the growth rate is negative. The term \(\beta(SI/N)\phi(N)\) stands for the number of transmission events, a function of the number of contacts between infected and susceptible individuals, which in turn depends on \(\beta\), the probability of transmission per encounter, \(\phi(N)\) denotes the number of conspecifics encountered by each infected, and \(S/N\) the proportion of susceptibles in such encounters. The disease would be transmitted horizontally and vertically if the infected hosts were capable of infecting newborns fast enough. In the last scenario, the division of the growth rate into birth and survival parts would be ‘justifiably’ avoided. System (9) is given by the following equations:

\[
\begin{align*}
\frac{dS}{dt} &= rf(N)S - \beta \frac{SI}{N} \phi(N), \\
\frac{dI}{dt} &= \beta \frac{SI}{N} \phi(N) + rf(N)I - \alpha I.
\end{align*}
\]

In addition, Deredec and Courchamp [30] developed an alternative model that includes (i) a growth rate that is divided into a constant birth rate and a density-dependent mortality rate and (ii) an Allee effect that acts only on mortality. Their alternative model reads as follows:

\[
\begin{align*}
\frac{dS}{dt} &= B(N)N - M(N)S - \beta \frac{SI}{N} \phi(N), \\
\frac{dI}{dt} &= \beta \frac{SI}{N} \phi(N) - M(N)I - \alpha I,
\end{align*}
\]

where \(B(N) = b\) and \(M(N) = b - rf(N)\).

Deredec and Courchamp [30] compared the dynamics of populations in the presence and absence of Allee effects within populations facing the possibility of microparasitic infections. These researchers found that the influence of Allee effects could be interpreted as the tradeoff between Allee effects and disease. Allee effects could protect native individuals by reducing the range of population sizes that facilitate parasitic spread, but, on the other hand, after the infection spreads, Allee effects weaken the host population, reducing its size and increasing the range of parasitic species that could lead to the population’s extinction.

Hilker et al. [59] studied a specific case of Model (11) with

\[
B(N) = a[-N^2 + (K + L + e)N + c], \quad M(N) = a(eN + LK + c) \quad \text{and} \quad \phi(N) = N.
\]

The selections of \(B(N), M(N),\) and \(\phi(N)\) specified above lead to the following model:

\[
\begin{align*}
\frac{dS}{dt} &= a[-N^2 + (K + L + e) + c]N - a(eN + LK + c)S - \beta SI, \\
\frac{dI}{dt} &= \beta SI - a(eN + LK + c)I - \alpha I,
\end{align*}
\]

where non-negative parameters \(e, c\) determine the effect of density dependence and independence on the demographic functions, respectively. Hilker et al. [59] showed that in the presence of a strong Allee effect in host demographics, System (12) can exhibit stable periodic orbits (by Hopf bifurcations), multiple stationary states, and catastrophic collapses of endemic equilibria. In addition, they noted that disease-induced extinction is possible for high transmissibility rates.

Friedman and Yakubu [44] used the same model, that is, Model (12), and used it to identify model parameter values that lead to host population persistence (with or without infected individuals)
and host extinction. These researchers showed that in the presence of an Allee effect in the host demographics, even at large population densities, a small perturbation from the disease-free equilibrium can lead to the host’s population extinction. In addition, they proved that the additional deaths that come with fatal disease infections could effectively alter, in fact increase, the Allee threshold of the host population.

Thieme et al. [110] analysed an SI model (13) under a strong Allee effect that incorporated distinct birth and death functions compared with those used in Model (12). Thieme’s model reads as follows:

\[
\frac{dS}{dt} = S(B(S) - d) - \beta SI,
\]

\[
\frac{dI}{dt} = \beta SI - (\alpha + d)I,
\]

where \(d\) is the natural death rate and \(B(S)\) is the birth rate. It is assumed in Model (13) that the infected population does not reproduce, which was not the case for Models (11) and (12). A specific example studied in Thieme et al. [110] uses the following specific form for \(B(S)\):

\[
B(S) = \frac{aS}{b + S^\gamma}, \quad \gamma > 1.
\]

Thieme et al. [110] established theorems highlighting the conditions under which a transition from population decline to collapse is mediated by a Hopf bifurcation and marked by the occurrence of a heteroclinic orbit.

The Hilker et al. [59] and Thieme et al. [110] models, Systems (12) and (13), are structurally similar to predator–prey models with Allee effects in the prey population; both involve linear functional responses (prey eaten per predator per unit time) (see [44]). Yet, Model (12) supports more complicated dynamics than Model (13). Specifically, Model (12) supports multiple stable interior equilibria, while Model (13) can only sustain an interior positive equilibrium. Most studies of the interplay between Allee effects and infectious diseases have focused on the role of Allee effects at small population densities, while Friedman and Yakubu [44] have shown that the presence of an Allee effect in host demographics also matters at large population densities. Model (4) developed by Kang and Castillo-Chavez [64] appears to be the first to incorporate Allee effects and reductions in fitness (competitive ability) from infection, the kind of modifications that lead to rich dynamical outcomes. In short, the lack of inclusion of selective forces in population biology models has led to the possibly misleading conclusion that selection-free models are robust. However, it is well known, from the epidemiological literature, that the inclusion of selective forces, in the context of fatal diseases such as HIV, leads to ‘undesirable’ outcomes (from the perspective of policy) such as the existence of multiple endemic states [21,22,37, 62]. In short, the exclusion of the role of selection raises concerns on the usefulness of the model results, particularly when it comes down to their role in the design of intervention or control policies. An important question would be the following: Have the limitations of classical models left the study of epidemic control and intervention efforts in the hands of non-generic models?

The fragility of biological systems to perturbations is central to conservation biology, disease management, or sustainability. Populations under Allee effects or under the selective pressure of fatal or debilitating diseases may experience abrupt and unexpected changes after even minor perturbations. In other words, the resilience of biological systems may be weakened by disease and the requirement of a minimal critical population size that guarantees survival. Deredec and Courchamp [30] and Hilker [58] have shown that the joint impact of parasitism and Allee effects increases the likelihood of extinction. Thieme et al. [110] and Hilker et al. [59] concluded that
the role of density-dependent transmission in host populations may lead to the host’s extinction. Our recent work [64] highlights the richness of the dynamics generated by the interplay between Allee effects, disease-adjusted fitness, and dispersal. The study of the joint impact of Allee effects, disease-dependent reproduction, and disease-adjusted individual competitive ability has generated classes of models that naturally support complex dynamic patterns. Specifically, the results of the analysis of toy models, such as the one given by System (4), show that the impact of disease, dispersal, and Allee effects on the survival and persistence of animal populations is dramatic. Hence, the development of intervention strategies that rely entirely on the basic reproduction number $R_0$ must be challenged. Disease can modify birth and death rates directly as well as the ability to disperse or compete for resources indirectly. Hence, not only the dynamics of populations facing local challenges (Allee effects), the influence of global effects (dispersal), and the selective pressures that come with parasitism or fatal or debilitating diseases need to be studied systematically, but perhaps past theoretical studies may also have to be re-evaluated. The following section studies the role of dispersal in multiple-patch models of populations under the selective pressures discussed above.

3. Multiple-patch models on small networks

Mathematical models of dispersal that focus on the study of invasion dynamics or on the impact of dispersal rates and dispersal kernels on evolutionary processes, including the so-called potential for evolutionary suicide, have often included Allee effects [5,6,38,47,51,96,106,109,111,113,122]. Species geographical distribution is constrained by a range of environmental variables that include anthropogenic barriers, such as roads, farms, farming lands, river dams, and more, which may be the most serious threat to dispersal given their impact on landscape fragmentation. Wiens [117] argued that the key to the management of populations living in such heterogeneous habitats lies in our understanding of the joint effects of landscape structure and patterns generated by the movement of individuals within and between habitat patches. Hence, it is not surprising to hear leading ecologists insist that mathematical models of ecological processes must in general account for the role of spatial heterogeneity [67]. The general models developed in the next two subsections explore the impact that dispersal, via some idealized dispersal corridors, has on the dynamics of populations living in multi-patch landscapes, under the influence of disease-induced mortality and Allee effects. The systematic use of dispersal corridors may be the best way of keeping otherwise isolated populations connected, perhaps the only way of reducing the negative impact that fragmentation has on biodiversity [12,20,64].

3.1. Single-species models with Allee effects and dispersal

Why should we focus on the inclusion of Allee effects? It is known that pest invasions are among the most significant threats to the stability of natural and agricultural ecosystems. Biologists have studied and documented the role of dispersal of species such as the gypsy moth [103], Argentine ant [107], Africanized honey bee (Apis mellifera scutellata) [118], hemlock woolly adelgid [90], emerald ash borer (Agrilus planipennis) [11,91], and horse chestnut leaf-miner (Cameraria ohridella) [46]. Furthermore, Allee effects have been detected in natural populations for a wide array of taxa [25,72]. In short, Allee effects may actually play a dramatic role in the consequences of biological invasion and related conservation biology issues [105].

Ackleh et al. [1] used Model (15) to investigate the establishment of invasive species within an $n$-group multi-patch system. These researchers considered a population living in $n$-interacting patches, with dynamics, linked by the matrix of dispersal rates $(l_{ij})$ with entries describing the
movement of individuals from patch $j$ to patch $i$, under the following constraint:

$$\sum_{i=1, i \neq j}^{n} l_{ij} = \sum_{i=1, i \neq j}^{n} l_{ji}. \quad (14)$$

Their model was formulated using the following system of nonlinear differential equations:

$$N_i' = r_i N_i (N_i - \theta_i)(1 - N_i) + \sum_{j=1, j \neq i}^{n} (l_{ij} N_j - l_{ji} N_i), \quad (15)$$

where $N_i(t), i = 1, \ldots, n$, denotes the population density in patch $i$. If we further assume that the dispersal rate between two patches is proportional to the difference in patch-specific population densities, then after dropping the assumption of Equation (14) in Equation (15) given in Ackleh et al. [1], we arrive at the following simpler nonlinear system (16):

$$N_i' = r_i N_i (N_i - \theta_i)(1 - N_i) + \sum_{j=1}^{n} l_{ij} (N_j - N_i), \quad (16)$$

with $l_{ij}$ representing the dispersal rate modelling the movement of individuals from patch $j$ to patch $i$. The weighted and directed adjacency matrix $L = (l_{ij})_{i,j=1}^{n}$ gives the connectivity between patches and dispersal strength; matrix $L$ defines the ‘topological’ structure of the landscape. System (16) can be used to explore the role of heterogeneity in the establishment and/or extinction of invasive species. Some questions that immediately emerge are as follows:

(i) What is the role of patch quality? Each patch is defined by different values of $r_i, \theta_i, l_{ij}$ representing the intrinsic growth rate, Allee threshold, and dispersal rate of species from patch $j$ to patch $i$, respectively.

(ii) What is the impact of initial conditions? Initial condition is expected to play a critical role in determining if a species spreads or becomes established, since each population is assumed to be operating under strong Allee effects.

(iii) How do different landscape structures (i.e. topological structures), modelled by the dispersal matrix $L = (l_{ij})_{i,j=1}^{n}$, affect population dynamics?

The pervasiveness of landscape structure in the time evolution of biological systems is unavoidable. Yet, theoreticians rarely incorporate the network structure. We revisit the four-patch version, shown in Figure 2, under a forest analogy described in [45] revisited below in order to begin to address the challenges that we face as we incorporate network structure. The Fulford et al. [45] configurations include

(a) A chain configuration (the first graph of the first row in Figure 2), motivated by the study of forest areas along a one-dimensional structure such as a river.

(b) A spider configuration (the second graph of the first row in Figure 2), motivated by the study of forest reserves with links to smaller forested areas on, for example, adjacent farmlands.

(c) A loop configuration (the third graph of the first row in Figure 2), motivated by the study of forest areas surrounding small villages or lakes (where the species cannot live).

In the absence of dispersals, System (16) becomes the $n$ uncoupled system of equations given by Model (2). In the presence of dispersal, we can deduce that, for System (16), the existence of a patch where the species persists implies species persistence in all patches. Similarly, the existence of a patch where the species goes extinct implies species extinction in all patches. A study of the
dynamics of System (16) when $n = 2$ [65] has shown that (1) there is no limit cycle; (2) the two-patch model can support up to four attractors, two interior generated by source–sink dynamics; and (3) the extinction and establishment state basins of attraction determined by $r_i, \theta_i$ and $l_{ij}$.

It is still an open question whether or not these three dynamical properties will carry over to System (16) when $n \geq 3$. However, if it is assumed that all patches are initially unoccupied, then we have shown that a species that arrives in patch $i$, with initial condition $N_i(0)$ will expand if $r_i(N_i(0) - \theta_i)(1 - N_i(0)) \geq \sum_{j=1}^{n} l_{ij}$ or shrink if this condition is not satisfied. The challenging task at the moment (at least for us) is to identify the minimal biologically adequate condition that guarantees either the establishment or the extinction of an invasive species, as a function of the topological structures. Kang and Lanchier’s higher dimensional approach, using a stochastic version of Model (16), seems quite promising and therefore it will be discussed briefly in Section 4.1.

3.2. SI models with component Allee effects, disease-modified fitness, and dispersal

Mammals, birds, fish, and insects undertake regular long-distance movements to track resources and, in the process, they often shift habitats. Migrations, a particular form of long-distance dispersal, not only facilitate the global spread of disease but may also provide opportunities for hosts to escape from habitats where disease prevalence is high [4]. Studies of disease dynamics in migratory species and their response to global changes are important for a multitude of reasons; some of the most pressing reasons put forward come from the desire to limit the spread of avian influenza [98], a disease that impacts not only wild and domestic avian populations but also the whole humans and a disease that seems to be a key reservoir for the genetic variability responsible for the generation of new strains of influenza A that are responsible for influenza pandemics [19].

Recent studies [1,12,15,20,57,86,87,98,99,101,122] have shown that the movement of susceptible or infected individuals can enhance or suppress the spread of diseases when the outcome is a function of spatial heterogeneity and connectivity. We introduce a multiple-patch model that extends the general SI system to investigate the impact of landscape network structure on the
We start from System (7) and make use of the fact that when the dispersal between any two patches is proportional to the difference in population sizes of the patches involved. Under this assumption, we arrive at the following multi-patch SI system:

\begin{align}
S_i' &= B_i(S_i, I_i)P_i(S_i, I_i) - A_i(N_i)S_i - \Phi_i(S_i, I_i) \frac{S_i}{N_i} - D_i(N_i)S_i + \sum_{j=1}^{n} l_{ij}(S_j - S_i) \\
I_i' &= \Phi_i(S_i, I_i) \frac{S_i}{N_i} - (\alpha_{2i}A_i(N_i) + D_i(N_i) + \mu_i)I_i + \sum_{j=1}^{n} m_{ij}(I_j - I_i),
\end{align}

where $S_i(t), I_i(t), N_i(t), i = 1, \ldots, n$, denote the susceptible, infected, and total populations in patch $i$, respectively; $l_{ij}$ denotes the dispersal rate of susceptible individuals from patch $j$ to patch $i$; and $m_{ij}$ denotes the dispersal rate of infective individuals from patch $j$ to patch $i$. The dispersal matrices of the susceptible population $L = (l_{ij})_{j=1}^{n}$ and the infective population $M = (m_{ij})_{j=1}^{n}$ are constrained by pre-agreed topological structures imposed on the network.

Model (17) is powerful enough to allow for the study of questions that include:

(i) **The effects of patch quality on disease persistence and extinction:** Fixing the landscape structure $L, M$ and incorporating component Allee effects in System (17) allow for the study of situations where it is, for example, assumed that each patch is under either weak Allee effects or strong Allee effects. Identifying conditions for disease persistence and extinction as well as disease-driven extinction of the host could be tackled with Model (17).

(ii) **Disease-induced network structure effects:** The study of the impact of pre-selecting the $M$ and $L$ dispersal structures may start under the assumption that all patches are identical (regarding strong Allee effects) in the absence of dispersal and then proceed by studying the network structure effects when $M = N$ or when $M \neq L$.

(iii) **The effect of initial conditions:** How the spreading of disease, a function of the initial conditions within the infected patch as well as a function of the connecting degree structure associated with the initial patch, can be explored with Model (17).

We are painfully aware that it is quite challenging to obtain analytical results for System (17) because of the nonlinearity introduced by component Allee effects, the complexity of the disease transmission term (incidence), and the increased dimensionality. Nevertheless, we believe that the analyses of special cases and the use of extensive and systematic simulations will eventually lead to the identification of characteristic patterns, as a function of the network structure overimposed by Model (17).

The initial analytical work has therefore focused on the study of two-dimensional (2D) systems. We start from System (7) and make use of the fact that when $L = M = 0$ the 2n-D. System (17) is equivalent to $n$ independent 2D systems, with all being equivalent to Model (7). Thus, System (17) is capable of supporting multiple stable limit cycles (under certain parameter ranges). What would the basins of attraction of the interior attractors supported by System (17) be, as a function of the dispersal matrices $L$ and $M$, under the presence of Allee thresholds and weak dispersal?

The derivation of the multi-patch setting encoded in Model (17) has been influenced by the research of Hastings [56] on dispersal under passive animal dispersal (density-independent dispersal, see [43]). Insights into the differential role played by active (density-dependent) dispersal as opposed to passive dispersal in the dynamics of populations can be gained from the simulation of our multi-patch models after we replace $\sum_{j=1}^{n} l_{ij}(S_j - S_i)$ with $\sum_{j=1}^{n} (l_{ij}(N_j)S_j - l_{ij}^S(N_j)S_i)$. Note that $l_{ij}^S, l_{ij}^P$ are functions of $N_i, N_j$, respectively. For example, we can assume that $l_{ij}^P(N_j) = l_s(N_j^P/(h_j^P + N_j^P))$, where $l_s$, a constant, denotes the maximal per-capita dispersal rate...
for susceptible individuals, a parameter that captures the ability of a species to disperse (or a parameter that is used to calibrate or account for the distance between patches). The half satu-ration parameter \( h_S \) is a constant that is given by the density of susceptibles that corresponds to the case when the per-capita dispersal rate \( l_S^i(N_j) \) is half of its maximal value \( l_S \). The parameter \( \gamma \) determines the shape of the density dependence on dispersal. Hence, when \( \gamma = 0 \), dispersal is independent of density, occurring at a constant per-capita rate \( l_S/2 \), while when \( \gamma > 0 \), the dispersal rates increase with density. On the other hand, \( \gamma < 0 \) corresponds to decreases with density. We can add terms that account for the (possibly distinct) dispersal rates of infectives in the population.

3.3. Other models with Allee effects and dispersal

There are few patch (metapopulation) models that include Allee effects and disease in the literature. Currently, Kang and Castillo-Chavez (ongoing work) are focusing on the study of System (4), a two-patch extension model (that follows after the structure of Model (18) given below), under the assumption that between-patch dispersal can be captured by a term involving the difference between the population densities of the two patches involved. The system of equations in this case becomes

\[
\begin{align*}
\frac{dS_1}{dt} &= f_1(S_1, I_1, S_2, I_2) \\
\frac{dI_1}{dt} &= f_2(S_1, I_1, S_2, I_2) = \beta_1 S_1 I_1 + l_{12}(I_2 - I_1) - d_1 I_1 \\
\frac{dS_2}{dt} &= g_1(S_1, I_1, S_2, I_2) \\
\frac{dI_2}{dt} &= g_2(S_1, I_1, S_2, I_2) = \beta_2 S_2 I_2 + l_{22}(I_1 - I_2) - d_2 I_2,
\end{align*}
\]  

(18)

where

\[
f_1(S_1, I_1, S_2, I_2) = \begin{cases} 
\quad r_1(S_1 + \rho_1 I_1)(S_1 + \alpha_{11} I_1 - \theta_1) \\
\times (1 - S_1 - \alpha_{12} I_1) \\
\quad -\beta_1 S_1 I_1 + l_{11}(S_2 - S_1) \\
0, & \text{if } S_1 = 0 \text{ and } r_1 \rho_1 I_1 (\alpha_{11} I_1 - \theta_1) \\
\times (1 - \alpha_{12} I_1) + l_{11} S_2 < 0
\end{cases}
\]

and

\[
g_1(S_1, I_1, S_2, I_2) = \begin{cases} 
\quad r_2(S_2 + \rho_2 I_2)(S_2 + \alpha_{21} I_2 - \theta_2) \\
\times (1 - S_2 - \alpha_{22} I_2) \\
\quad -\beta_2 S_2 I_2 + l_{21}(S_1 - S_2) \\
0, & \text{if } S_2 = 0 \text{ and } r_2 \rho_2 I_2 (\alpha_{21} I_2 - \theta_2) \\
\times (1 - \alpha_{22} I_2) + l_{21} S_1 < 0
\end{cases}
\]

In System (18), \( S_1 \) denotes the normalized susceptible population and \( I_1 \) denotes the infected population, relative to \( S_1 \) at location A; similarly, \( S_2 \) is the normalized susceptible population and \( I_2 \) is its relative infected population at location B; all parameters are non-negative; \( 0 \leq \rho_i \leq 1, i = 1, 2 \), describe the reduced contribution of the infected population of newborns; \( l_{ij}, i, j = 1, 2, \) denote the dispersal parameters from patch \( j \) to patch \( i \); \( 0 \leq \alpha_{ij} \leq 1, i, j = 1, 2, \) represent the relative
competitive ability of the infected individuals with respect to the non-infected; $r_i, i = 1, 2,$ are the patch-specific maximum birth rates of species; $d_i$ are the death rates of the infected population, including additional death caused by disease; $0 < \theta_i < 1, i = 1, 2,$ denote the Allee thresholds; and $\beta_i, i = 1, 2,$ denote the disease transmission rates.

The dynamics of System (18) are richer than those generated by System (4) as a result of dispersal coupling. It is observed, in the simulations described below, that weak dispersal can support source–sink dynamics. In fact, setting $l_{ij} = 0$ in the uncoupled system generated from Model (18) leads to simplified sub-models capable of supporting stable limit cycles in a single patch (for some parameters). Adding patch coupling via dispersal will, in general, have a significant impact on the dynamics. The coupled version of System (18) under weak dispersal is capable of supporting four stable limit cycles (Figure 3).

Thus, weak dispersal rates cannot, in general, stop the disease from spreading, since the established infected population via System (4) serves as a source, while the connected patch serves as a sink (source–sink dynamics). In general, the source–sink dynamics of System (4) can be generated by the following two mechanisms:

(i) Allee effects and spatial heterogeneity are tied into differences in initial conditions. In the absence of dispersal, population size in a patch is above its Allee threshold, and thus the infected population persists, while the population in the connected patch is below its Allee threshold, and thus the infected population goes extinct. In the presence of dispersal, the patch with population size above the Allee threshold serves as the source and the one with population size below the threshold serves as the sink. This analysis holds even in the case when the two patches are identical.

(ii) Differences in patch quality. The high-quality patch supports an endemic state that serves as the source, while the lower quality patch living under disease-free dynamics serves as the sink.

The effects of dispersal on the dynamics of System (18) become quite complicated when dispersal between patches changes from weak to strong; that is, the coupling terms $(l_{ij}, i, j = 1, 2)$ are not small any more. Numerical simulations lead to the following conclusions as dispersal moves from weak to strong:

(i) Small dispersal intensities can generate source–sink dynamics and thus weak dispersal cannot stop the spread of disease.

(ii) When uncoupled system (18) supports a stable limit cycle, the coupling, via intermediate dispersal intensities, can stabilize the coupled system, driving the coupled system into one supporting a stable focus. In the last case, dispersal simply modifies the dynamical pattern; that is, it has no effect on coexistence. As dispersal continues to increase, we cross a threshold after which the reappearance of a stable limit cycle is possible. The two-patch dynamics become synchronized as dispersal tends to infinity (Figure 4).

(iii) It is seen that intermediate dispersal intensities can drive the coupled system into one that supports stable limit cycles when a patch in uncoupled system (18) supports a stable limit cycle, while the second patch undergoes a heteroclinic bifurcation. Further, as dispersal rates increase, a threshold is reached and crossed. Crossing the threshold forces each stable limit cycle to merge with the adjacent saddle, a process that leads to the annihilation of both. This annihilation is recognized as a catastrophic event in dynamical systems theory [48,61], a catastrophe that wipes out the whole two-patch population. Hence, the combined interactions of Allee effects and large dispersal rates can result in the elimination of an invasive species that suffers from a disease (Figure 5).
Figure 3. Coupled system (18) that has four stable limit cycles when $r_1 = 0.2; \theta_1 = 0.1; \rho_1 = 0.15; d_1 = 0.5; \beta_1 = 1$; 
$r_2 = 1; \theta_2 = 1; \rho_2 = 0.15; d_2 = 0.5; \beta_2 = 1$; $T = 1000$ 
Initial conditions generate blue: $S_1(0) = 1; I_1(0) = 1; S_2(0) = 0.45; I_2(0) = 0.3$; 
Initial conditions generate red: $S_1(0) = 0.5; I_1(0) = 0.2; S_2(0) = 0.1; I_2(0) = 1$; 
(a) Source patches: red is patch one, blue is patch two 
(b) Sink patches: red is patch one, blue is patch two
The goal of the model was to explore the spatiotemporal spread of an infectious disease in animal populations. In the case that $a = 0$, both patches have a stable limit cycle with different amplitudes and periods. In the case that $a = 0.0001$, the small dispersal generates the source–sink dynamics. While large dispersals stabilize the coupled system by driving the couple system into one exhibiting a stable focus, e.g. $a = 0.1$, the extremely large dispersals, e.g. $a = 100.4$, drive the dynamics in two patches synchronized.

Hikler et al. [60] introduced a reaction–diffusion SI model using SI model (12) as a platform. The model of the goal was to explore the spatiotemporal spread of an infectious disease in animal populations:

$$\frac{\partial S}{\partial t} = D_S \Delta S + a(-N^2 + (K + L + e) + c)N - a(eN + LK + c)S - \frac{\beta SI}{N}$$
$$\frac{\partial I}{\partial t} = D_I \Delta I + \frac{\beta SI}{N} - a(eN + LK + c)I - aI,$$

(19)

where $\Delta$ is the Laplacian; $D_S \geq 0, D_I \geq 0$ (km$^2$ per year) are the diffusion coefficients for the susceptible and infected individuals, respectively.

Hikler et al. [60] concluded that the spatiotemporal dynamics of (19) are complex and that the disease turned out to have a considerable impact on the spread of a host population in the following sense: (1) There are waves of extinction arising when the disease is introduced in the invading host population wave. Further, these waves of extinction destabilize locally stable endemic coexistence states. (2) Spatially restricted epidemics are possible as well as travelling infection pulses corresponding to either fatal epidemics resulting in the host population extinction or to epidemics with host recovery.
Figure 5. The phase plane of coupled system (18) when \( r_1 = 1; \theta_1 = 0.1; \rho_1 = 0.15; d_1 = 0.2; \beta_1 = 1; l_11 = l_12 = l_{21} = l_{22} = a; r_2 = 1; \theta_2 = 0.1; \rho_2 = 0.15; d_2 = 0.4; \beta_2 = 1; S_1(0) = 0.5; I_1(0) = 0.15; S_2(0) = 0.6; I_2(0) = 0.1; \) and \( a = 0, 0.01, 0.1, \) respectively. In the case that \( a = 0, \) the red patch \((S_1, I_1)\) has a stable limit cycle, while the blue patch \((S_2, I_2)\) is undergoing heteroclinic bifurcation that leads to the extinction of infected populations. In the case that \( a = 0.01, \) the small dispersal drives the coupled system that has stable limit cycles, while dispersal becomes large, e.g. \( a = 0.1, \) the infected populations in both patches go extinct. (a) The phase plane of uncoupled system (18), (b) the phase plane of coupled system (18) when dispersals are equal to 0.01, and (c) the phase plane of coupled system (18) when dispersals are equal to 0.1.

The predator–prey two-patch model given by System (20) \([12,14]\) incorporates Allee effects in the population. Nevertheless, in some respects, it is quite similar to the SI formalisms involving Allee effects. Model (20) has been used to explore the population dynamics of predator–prey systems when their interactions are mediated by a prey population access to a refuge \([12]\) or when patch–prey abundance drives prey dispersal between patches \([14]\). The model equations are as follows:

\[
\begin{align*}
  u_1' &= \beta_1 f(u_1) - u_1v_1 + \alpha_1(u_2 - u_1) = F_1(u_1, v_1, u_2, v_2) \\
  v_1' &= \gamma_1 v_1 (u_1 - m) = G_1(u_1, v_1, u_2, v_2) \\
  u_2' &= \beta_2 f(u_2) - u_2v_2 + \alpha_2(u_1 - u_2) = F_2(u_1, v_1, u_2, v_2) \\
  v_2' &= \gamma_2 v_2 (u_2 - m) = G_2(u_1, v_1, u_2, v_2),
\end{align*}
\]

\( (20) \)
where \( f(u_i) = u_i(u_i - l_i)(1 - u_i) \); \( \beta_i > 0 \) characterize the rates of prey growth; \( 0 \leq l_i \leq 1 \) denote the critical densities of the prey population; \( \gamma_i > 0 \) denote the coefficients of conversion of prey into predator biomass; \( m_i \geq 0 \) is a measure of the predators’ adaptation to the prey, and \( \alpha_i \geq 0 \) characterize migrations of preys in \( i \)th patch.

In situations when differences in prey abundance between the refuge and non-refuge are the drivers of prey dispersal, Berezovskaya et al. [12] have shown that as dispersal between the prey refuge and the predator–prey habitats increases, the system experiences transitions from predator extinction (for all initial conditions) to predator–prey oscillatory coexistence, to predator–prey non-oscillatory coexistence (outcomes depend on initial conditions). They also discussed the possibility of bi-stability, tri-stability, and related outcomes.

In situations when the prey population has access to both habitats, Berezovskaya et al. [14] were able to perform quite a bit of the mathematical analysis with the help of lower dimensional sub-models, with outcomes quite sensitive to the structure of the system, the range of parameter values, and initial conditions. They showed that the system can support multi-stability and a diverse set of predator–prey life-history dynamics including rather complex dynamical system outcomes. Their work supports the view that if the ‘goal’ is to generate variability in life-history outcomes, the kind of variability needed for selection to operate, then evolution must favour heterogeneous settings that include Allee effects, prey refuges, and patch-specific predators all connected by dispersal.

In summary, SI models with Allee effects and dispersal can produce extremely complicated dynamics. Examining the effects of the interactions that emerge from Allee effects, disease, and dispersal requires minimally the integration of studies of the local (single-patch) dynamics, the nodes of a network landscape structure. Further, the edges of this network are dynamic, modelled under passive or active dispersal assumptions. Interacting particle systems deal with the overall properties of large ensembles of nodes. Hence, the study of high-dimensional versions of our systems within a stochastic framework (interacting particle systems) may bring the kind of flexibility that is needed to study networks, that is, large ensembles of patches linked by dispersal (see the next section).

4. Stochastic models

To understand the effects of stochasticity and spatial arrangements on species establishment and species/disease spread, we introduce stochastic versions of the deterministic models described in the previous section using interacting particle systems, that is, individual-based models or continuous-time Markov chain models that include two components: a connected graph to be thought of as a network of interactions and a set of heuristic rules that model the transition rates at each vertex of the graph. It is expected that the study of these models will generate insights on the macroscopic behaviour and spatial patterns that emerge from microscopic interactions.

4.1. Multiple-patch models with Allee effects

Our previous work [65] on the establishment of species with Allee effects in patchy environments increased our understanding of the role of stochasticity in the timing of two key transitions: species establishment and species spread. In this section, the work of Kang and Lanchier [65] is extended to landscapes defined by small networks in order to study the role of network structure (‘topology’) in the expansion/extinction of invasive species under heuristic strong Allee (2) and stochastic effects.
We start by re-stating the symmetric version of our deterministic when \( n = 2 \), that is, the following two-patch model:

\[
N'_i = rN_i(N_i - \theta_i)(1 - N_i) + \mu(N_j - N_i) \quad \text{for } i = 1, 2 \text{ and } j = i + 1 \mod 2,
\]

(21)

where \( N_i(t) \), \( i = 1, 2 \), denotes the population density in patch \( i \) and \( \mu \) the dispersal rate of species moving from one patch to another. The stochastic version of Equation (21) can be seen as an interacting particle system on a graph with only two vertices and one edge.

\( N_i(t) \), \( i = 1, 2 \), denotes the population density at patch \( i \) and local expansions/extinctions are modelled by the transitions

\[
N_i(t) \rightarrow 1 \text{ at rate } r \text{ when } N_i(t) > \theta_i \quad \text{and} \quad N_i(t) \rightarrow 0 \text{ at rate } r \text{ when } N_i(t) < \theta_i,
\]

while dispersal is modelled by assuming that the edge connecting both the patches becomes active at rate one, which results in the transitions

\[
N_i(t) \rightarrow \mu N_j(t) + (1 - \mu)N_i(t) \quad \text{for } i = 1, 2 \text{ and } j = i + 1 \mod 2.
\]

While both models share some similarities, they also exhibit interesting differences, particularly in the presence of weak dispersal. The deterministic model has four attractors, possibly three in the asymmetric case \( \theta_1 \neq \theta_2 \), suggesting that at high and low densities, the coexistence of populations at equilibrium in nearby patches is possible. In contrast, the stochastic model supports only equilibria that correspond to global expansion and global extinction with the ‘missing’ equilibria becoming metastable states. Moreover, we have shown that starting with one empty and one fully occupied patch then, as the dispersal parameter tends to zero, in the symmetric case, the probability of global expansion tends to one when the Allee threshold is smaller than one-half, and the probability of global expansion tends to zero when the Allee threshold is larger than one-half.

These sufficient conditions for expansion/extinction depend on local population size, migration rate, and connectivity even though we are aware that stochastic variability in births, deaths, and migration can significantly affect species establishment and spreading. Thus, the main focus of this section is to discuss ways in which a species expands its population to other patches once it has become established in a patch, using the framework of interacting particle systems.

In order to understand how the geometry of the network affects the macroscopic behaviour of populations subject to an Allee effect, an interacting particle system model is presented. The network used to support dispersal is represented by an arbitrary connected graph where each vertex is identified as a patch, say \( x \), and characterized by its density \( N_x(t) \). Each edge of the network, say \( (x, y) \), becomes active at rate one, which results in an exchange of a fraction \( \mu \) of the population or in other words

\[
N_x(t) \rightarrow \mu N_y(t) + (1 - \mu)N_x(t) \quad \text{and} \quad N_y(t) \rightarrow \mu N_x(t) + (1 - \mu)N_y(t).
\]

In addition, each patch experiences local expansion or extinction, at rate one, depending on whether its density is above, respectively, below, the Allee threshold \( \theta \). Local extinction means that the patch becomes empty and local expansion means that the density jumps to 1. This model is a generalization to arbitrary graphs of the stochastic two-patch model introduced by Kang and Lanchier [65]. Standard results of the theory of Markov chains imply that this process converges almost surely to one of its two absorbing states: the configuration in which all patches are fully occupied or the configuration in which all patches are empty. We call the events that the process converges to absorbing states or, ‘biologically’ speaking, global expansion and global extinction.
Preliminary simulations on all possible networks with four vertices starting with a single fully occupied patch, which we call the source, suggest that $\mu$ only has a limited effect on the asymptotic behaviour of the system, though it has a strong effect on the time to fixation, with small $\mu$ leading to the existence of metastable states. In contrast, the value of $\theta$ as well as the geometry of the network and the degree of the patch initially occupied has a strong effect on the probability of global expansion/extinction. The smaller the $\theta$, the larger the probability of expansion, a result that can be proved analytically using standard coupling techniques for particle systems.

Starting with a single source, numerical simulations indicate that the probability of global expansion decreases with the number of edges as well as with the degree of the source, as shown in Figure 2, a graph that suggests that dispersal promotes extinction. However, the application of standard coupling techniques implies that the probability of expansion of the process obtained by exchanging initially occupied and empty patches while replacing $\theta$ with $1 - \theta$ is equal to the probability of the extinction of the original process; therefore, dispersal promotes survival when the starting state has a majority of occupied patches.

The mathematical analysis of the model on large networks (e.g. regular graphs) is possible. We focus on two extreme cases in order to prove that dispersal promotes extinction in situations when we have a single source: the complete graph case, in which all the vertices are connected to each other, and the ring case, in which all the vertices have degree two. For complete graphs, the idea is to map the dynamics of mixing events into a dynamic graph in which the edges are created by a pair each time individuals disperse. Large deviation estimates for the first time a cycle appears in this dynamic graph and the number of leaves just before this stopping time imply that, with a high probability when the network is large, all patches are below $\theta$ by the time the first local expansion occurs, which leads to extinction. In contrast, for the process on the ring, random-walk estimates imply that if a patch is fully occupied, then its two neighbours become fully occupied with a high probability when $\theta$ is smaller than some positive threshold, which, together with a comparison with oriented site percolation, leads to expansion (see Figure 6 for simulation results).

From the work of Kang and Lanchier [65,66], we expect results that highlight a similar disagreement between deterministic models and their stochastic counterparts as the number of patches increases. The results have been generated from preliminary simulations, starting with a single fully occupied patch, of all possible networks with four vertices (Figure 2) in the symmetric cases. Analytical proofs are being tried by Nicolas Lanchier.

Figure 6. Fraction of patches above the Allee threshold after 200,000 updates on the ring with 200 vertices and after 2000 updates on the complete graph with 200 vertices, as a function of the Allee threshold and the initial density of the occupied patches. The colour of each point is computed from the average of 100 independent realizations. Black means that all patches are below the Allee threshold, while white means that they are all above the threshold.
4.2. Multiple patches with Allee effects and disease

To understand the combined effect of dispersal and infection in a stochastic framework, we extend Model (17). We let each vertex, say $x$, be characterized by two random variables:

$$S_x(t) = \text{density of healthy individuals} \quad \text{and} \quad I_x(t) = \text{density of infected individuals}.$$ 

Each edge of the network, say $(x, y)$, becomes active at rate one, which results in a fraction $\mu$ of the population in each patch dispersing to the other patch, that is,

$$S_x(t) \rightarrow \mu S_y(t) + (1 - \mu) S_x(t) \quad \text{and} \quad I_x(t) \rightarrow \mu I_y(t) + (1 - \mu) I_x(t),$$

and the analogous transition obtained by exchanging $x$ and $y$. In addition, each patch experiences a local expansion, respectively, extinction, at rate one depending on whether

$$S_x(t) + \rho I_x(t) > \theta \quad \text{or} \quad S_x(t) + \rho I_x(t) < \theta,$$

(22) where $0 \leq \rho \leq 1$ models the variation in fecundity of individuals which are infected. Local expansion now means that $S_x(t) \rightarrow 1 - I_x(t)$. Finally, the spread of the infection is modelled by

$$I_x(t) \rightarrow S_x(t) + I_x(t) \quad \text{and} \quad S_x(t) \rightarrow 0 \text{ at rate } \beta I_x(t),$$

where $\beta$ is the infection parameter. Although this new model has three absorbing states, its dynamics imply that there is always a residual of infected individuals as long as the global density is positive; therefore, it has only two possible asymptotic behaviours on finite graphs: global extinction or global expansion with all individuals infected.

To understand the role of dispersal in the presence of disease, we assume that $\theta < \frac{1}{2}$ and that the initial density is fixed so that a healthy population can expand. When all the individuals are infected, the left-hand side of Equation (22) is equivalent to $I_x(t) > \theta / \rho$. This, together with our assumption and coupling techniques for interacting particle systems, implies that, regardless of the infection rate, there is global expansion whenever $\rho > 2\theta$. In the region $\rho < 2\theta < 1$, numerical simulations of the processes on the ring and a finite regular graph with a large degree give the schematic phase diagrams shown in Figure 7. For small values of the infection rate, there is global expansion with all individuals infected if $\rho > \theta$ but coexistence between mildly infected patches and empty patches when $\rho < \theta$. This follows from the fact that the left-hand side of Equation (22) is always satisfied for fully occupied patches when $\rho > \theta$ but not for fully infected patches when

Figure 7. Schematic phase diagrams of the patch model on regular graphs with degree two on the left and a large degree on the right. In the second picture, the continuous lines represent the transition curves when starting with a lower density and the dashed lines represent the transition curves when starting with a larger density.
\( \rho < \theta \). Note that coexistence here means that there is a quasi-stationary distribution, as opposed to a stationary distribution, with a positive density of empty and occupied patches. In contrast, when \( \beta \) is large, global extinction occurs, which follows from the fact that a fully infected population behaves like a healthy population with Allee threshold \( \theta / \rho \). The larger the dispersal, the larger the critical infection rate when starting with a large density of occupied patches – see the dashed transition curves on the right-hand side of Figure 7 – but the smaller the critical infection rate when starting with a small density – see the continuous transition curves of the same figure: as in the absence of disease, dispersal may promote either survival or extinction depending on the initial density.

We have performed carefully designed numerical simulations to obtain the schematic phase diagrams shown in Figure 7 for two network structures: the ring and a finite regular graph with a large degree. We are working, as we speak, on simulations involving alternative topological network structures. Detailed analytical results are still missing albeit Nicolas Lanchier’s research has shown promising avenues of success (pers. communication). If obtaining analytical results is not possible for the asymmetrical stochastic multi-patch models that include differential dispersal strategies in heterogeneous large networks, we will proceed to carry out extensive and systematic numerical simulations that we hope will allow us to collect valuable insights into the long-term behaviour of the system. In addition, we plan to continue to compare and contrast the results generated here with those that arise from the use of deterministic models.

5. Discussion

Simon A. Levin’s work on the role of dispersal in shaping communities, maintaining biodiversity, and building a science of sustainability represents one of his major contributions to the fields of ecology and evolutionary biology. His achievements include over 400 publications, mentoring more than 50 PhD students, countless numbers of postdoctoral students, and a cadre of young and old researchers who have benefited from his vision, friendship, and generosity. This article, dedicated to him on the occasion of his 70th birthday, expands on our recent efforts to build a framework where the joint dynamics of ecological and epidemiological processes can be explored and analysed.

The focus is on the study of the role that disease has in the life-history dynamics of subpopulations living in patchy landscapes connected by dispersal. The framework makes an honest effort to include the impact of disease as a selective force through the incorporation of disease-specific reductions in fertility or increases in mortality, disease-driven effects on the competitive ability of interacting subpopulations, and disease effects on host mobility. Our framework develops and makes use of deterministic and stochastic models that incorporate features rarely included simultaneously in the literature, such as (a) component Allee effects; (b) the impact of disease dynamics on the host’s fitness; and (c) spatial heterogeneity (modelled via a metapopulation). This article highlights the initial results of a group effort aimed at disentangling the impact of disease on the dynamics of mobile populations. Special attention is paid to the role of initial conditions, patch quality, and ‘topological’ or connectivity landscape structure (defined by the flow of individuals via dispersal corridors). In order to explore the role of stochasticity and the spatial arrangement of individuals/patches, we constructed stochastic analogues of the deterministic models, using the framework of interacting particle systems. We have done the following:

(1) Introduced generalized SI models with component Allee effects and disease-adjusted fitness.

This setting has allowed us to (1) compare disease dynamics subject to weak Allee effects with disease dynamics subject to strong Allee effects and (2) investigate the impact of distinct Allee thresholds on the persistence/extinction of the population.
Incorporated spatial heterogeneity, habitat connectivity, and movement rates between patches into the general SI model with disease and Allee effects, in order to address questions such as how do the number of infected patches and the connectivity of patches affect the spread of disease? How do initial conditions affect species’ persistence and disease dynamics? Which landscape structures are more likely to drive disease-aided species extinction? Comparing the dynamical results of deterministic models with those of their stochastic counterparts will help us understand the role of stochasticity. The mathematical results on the dynamics of these models provide useful insights into the identification, development, management, and/or control of endangered species in biology conservation and invasion programme.

Extended a two-patch model with Allee effects and dispersal following Kang and Lanchier [65] into a multi-patch framework involving different topological network structures, using interacting particle systems, a modelling approach that allows us to explore the role of topological structures in the expansion/extinction of species subject to Allee effects.

Mathematical challenges resulting from studying the details of the deterministic models (e.g. Equations (4) and (17) discussed in the article include, but are not limited to, the following:

(a) Multi-parameter bifurcation analyses that explore the role of reductions in reproduction or competition ability as well as in changing Allee thresholds.
(b) The estimation of attractor-specific positively invariant sets that include interior (endemic) and boundary attractors (disease extinction).
(c) The identification of stable limit cycles and heteroclinic orbits that have emerged from our high-dimensional ODE systems.

We have introduced a modelling framework that has been used to study the joint dynamics of mobile populations in systems where disease is a selective force. This is not a new perspective and yet the number of studies that have not ignored the role of selection via infections in the dynamics of heterogeneous populations has been rather miniscule. The modelling framework introduced here will be used in the development of management or control strategies aimed at reducing disease morbidity and mortality under more realistic settings. The control or management studies that will emerge from the systems presented in this article will have to deal with the fact that the underlying dynamics are complex and that the likelihood of the existence of stable multiple attractors is most likely generic. Hence, the tradition of using the basic reproductive number or ratio as the basis of such analyses will have to be re-formulated. We believe that models that include selective forces and network structure will eventually lead to the revision of some of the theories that have been built under highly simplified sets of assumptions.

Acknowledgements

The research of CCC is partially supported by the grant number 1R01GM100471-01 from the National Institute of General Medical Sciences (NIGMS) at the National Institutes of Health and partially supported by the grant number U54GM088558 from NIGMS to Marc Lipsitch and the Center for Communicable Disease Dynamics. The research of YK is partially supported by Simons Collaboration Grants for Mathematicians (208902). We thank Nicolas Lanchier for his great help in generating figures and his insights on the stochastic models. We also thank the referee for comments that have improved this article greatly.

References

[1] A.S. Ackleh, L.J.S. Allen, and J. Carter, Establishing a beachhead: A stochastic population model with an Allee effect applied to species invasion, Theor. Popul. Biol. 71 (2007), pp. 290–300.
[2] F.R. Adler, Migration alone can produce persistence of host-parasitoid models, Am. Nat. 141 (1993), pp. 642–650.
[3] L.J.S. Allen, B.M. Bolker, Y. Lou, and A.L. Nevai, Asymptotic profiles of the steady states for an SIS epidemic patch model, SIAM J. Appl. Math. 67 (2007), pp. 1283–1309.
[107] A.V. Suarez, D.A. Holway, and T.J. Case, *Patterns of spread in biological invasions dominated by long-distance jump dispersal: insights from Argentine ants*, Proc. Nat. Acad. Sci. USA 98 (2001), pp. 1095–1010.

[108] R.K. Swihart, Z. Feng, N.A. Sladea, D.M. Mason, and T.M. Gehring, *Effects of habitat destruction and resource supplementation in a predator–prey metapopulation model*, J. Theoret. Biol. 210 (2001), pp. 278–303.

[109] C.M. Taylor and A. Hastings, *Allee effects in biological invasions*, Ecol. Lett. 8 (2005), pp. 895–908.

[110] H.R. Thieme, T. Dhirasakdanon, Z. Han, and R. Trevino, *Species decline and extinction: Synergy of infectious disease and Allee effect?*, J. Biol. Dyn. 3 (2009), pp. 305–323.

[111] J.M.J. Travis and C. Dytham, *Dispersal evolution during invasions*, Evol. Ecol. Res. 4 (2002), pp. 1119–1129.

[112] M.G. Turner, R.H. Gardner, and R.V. O’Neill, *Landscape Ecology in Theory and Practice: Pattern and Process*, Springer-Verlag, New York, 2001.

[113] M.-H. Wang and M. Kot, *Speeds of invasion in a model with strong or weak Allee effects*, Math. Biosci. 171 (2001), pp. 83–97.

[114] H.F. Weinberger, *Asymptotic behavior of a model in population genetics*, in *Nonlinear Partial Differential Equations and Applications*, J.M. Chadam, ed., Lecture Notes in Mathematics, Vol. 648, Springer-Verlag, Berlin, 1978, pp. 47–96.

[115] H.F. Weinberger, *Long-time behavior of a class of biological models*, SIAM J. Math. Anal. 13 (1982), pp. 353–396.

[116] J.A. Wiens, *Wildlife in patchy environments: Metapopulations, mosaics, and management*, in *Metapopulations and Wildlife Conservation*, D.R. McCullough, ed., Island Press, Washington, DC, 1996, pp. 53–84.

[117] J.A. Wiens, *Metapopulation dynamics and landscape ecology*, in *Metapopulation Biology: Ecology, Genetics, and Evolution*, I.A. Hanski and M.E. Gilpin, eds., Academic Press, San Diego, CA, 1997, pp. 43–62.

[118] M. L. Winston, *The biology and management of Africanized honey bees*, Ann. Rev. Entomol. 37 (1992), pp. 173–193.

[119] D. Xu and Z. Feng, *A metapopulation model with local competitions*, Discrete Contin. Dyn. Syst. Ser. B 12 (2009), pp. 495–511.

[120] D. Xu, Z. Feng, L.J.S. Allen, and R.K. Swihart, *A spatially structured metapopulation model with patch dynamics*, J. Theoret. Biol. 239 (2006), pp. 469–481.

[121] Z. Feng, R. Liu, Z. Qiu, J. Rivera, and A.-A. Yakubu, *Coexistence of competitors in deterministic and stochastic patchy environments*, J. Biol. Dyn. 5 (2011), pp. 454–473.

[122] S.-R. Zhou and G. Wang, *Allee-like effects in metapopulation dynamics*, Math. Biosci. 189 (2004), pp. 103–113.