Discrete stochastic marine metapopulation disease model

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ABSTRACT

Some marine microparasitic pathogens can survive several months in the water column to make contact with or to be absorbed or filtered by hosts. Once inside, pathogens invade the host if they find suitable conditions for reproduction. This transmission from the environment occurs via pathogens released from infected and dead infected animals. Some recent modeling studies concentrated on the disease dynamic imposed by this complex interaction between population and water column at the host-pathogen level in single populations. However, only when a marine disease can be understood at the metapopulation scale effective approaches to management will become routinely achievable. The discrete-time disease model in this paper investigates both spatial and temporal dynamics of hosts and waterborne pathogens in a metapopulation system of three patches. This system with a patch providing infective particles and susceptible and infected individuals by dispersal tries to imitate the effect of current forces in the ocean on the passive dispersal of organisms. The model detects behaviours that are not present in single population continuous-time and deterministic models.

INTRODUCTION

The marine realm pathogens can be transmitted from host to host and from the environment to the host. The direct host to host transmission is commonly the case of diseases in fish such as salmons (e.g. Løvdal and Enger, 2002; Ogut et al., 2005) and mammals such as seals (Becher et al., 2002) where the disease is transmitted through contact (i.e. rubbing) with infected individuals since pathogens can reside on the skin of the infected animals. Marine invertebrates such as corals can also transmit pathogens through contact between sea fans when growing close together (Smith et al., 1996). Not only live infected animals but also dead infected animals can transmit the pathogen through contact. For example, polar bears, fish, shrimps, and amphipods can get infected by contacting or feeding on dead carcases (Lotz and Soto, 2002; Lotz et al., 2003; Rudolf and Antonovics, 2007).

The environment to host transmission occurs via pathogens released from infected animals that can survive in the marine environment for a certain amount of time from days to months (Casas et al., 2002) until they can invade host individuals finding suitable conditions for reproduction within the host. This is the case of the susceptible animal contacting with or filtering infective particles from the environment once are released by living or dead infected individuals; that is, the case of black-band disease (Richardson, 2004; Zvuloni et al., 2009) and Aspergillosis (Jolles et al., 2002) in corals, whithering syndrome (WS) in abalone (Moore et al., 2001, 2002) and transmission of trematode cercariae (De Montaudouin et al., 1998), shrimps with White-Spot disease (Rudolf and Antonovics, 2007) shedding particles during decay and scavenging processes, OsHV1virus in pacific oysters (Schikorski et al., 2011), MSX (Haskin et al., 1966) and Dermo (Mackin et al., 1950) diseases in oysters; Perkinsosis in clams (Paillard, 2004; Dang

et al., 2010). The proliferation of these marine infectious diseases are causing mass mortalities (Ward and Lafferty, 2004; Burge et al., 2014; Lafferty et al., 2015) threatening ecologically valuable habitats and resulting in substantial economic losses in fisheries and aquaculture (Walker and Winton, 2010; Lafferty et al., 2015).

Previous modeling studies on marine environmental pathogens have primarily focused on single population dynamics of uniformly distributed individuals on a single habitat bidegain. That is, continuous-time models, unstructured in spatial or age terms,and configured to simulate the dynamics of diverse dose (body burden)-dependent infectious disease transmission processes caused by susceptible individuals contacting or absorbing (filtering) infectious waterborne pathogens (Bidegain et al., 2016a, 2017). McCallumet al. (2005) similarly modeled the dynamics of withering syndrome in abalones by incorporating the free-living pathogen stage and disease transmission through contact between this stage and the host. Sokolow et al. (2009) and Yakob and Mumby(2011) formulated the dynamics of disease in corals describing the transmission of disease by contact between the host and free-living pathogens. Bidegain et al. (2016b) also formulated simple continuous-time compartmental models to yield the basic reproduction number *Ro* for a variety of marine host–pathogen systems to explore the relative importance of the host and pathogen traits that determine transmission.

This disease modeling approach has demonstrated that the water column provides a 'reservoir' for infective particles and the mechanisms by which particles are added to it or lost from it exert an important influence on the prevalence of disease and more importantly the difference between a disease exerting a local impact on a host population and pandemic disease affecting the host over large geographic regions. The local population modulates this effect through biological characteristics that affect the infective dose and through varying local availability by modulating particle incorporation and release rates. The dynamic imposed by this complex interaction between population and water column, potentially over metapopulation scales, is relatively unique to the marine world. Focusing on the details of this dynamic is critical to understanding the disease process in host populations and to improving management responses to marine disease challenges. However, only when a marine disease can be understood at the in vivo scale of the individual, the local scale of the population, and the metapopulation scale will effective approaches to management become routinely achievable.

The discrete-time disease model in this paper investigates both spatial and temporal metapopulation dynamics of hosts and waterborne pathogens in a three patch system. In order to detect system behaviors that are not present in deterministic models a stochastic version was implemented. In this model system, host and pathogen populations are growing and are subject to mortality. Each patch can both gain and loss hosts and pathogens by dispersion. One of the patches acts as a 'reservoir' and 'source' of pathogens and hosts nourishing of new individuals (animals) and pathogens the other two patches. This metapopulation with a patch providing infective particles and susceptible and infected individuals by dispersal tries to imitate the effect of current forces in the ocean and estuaries on the passive dispersal of organisms.

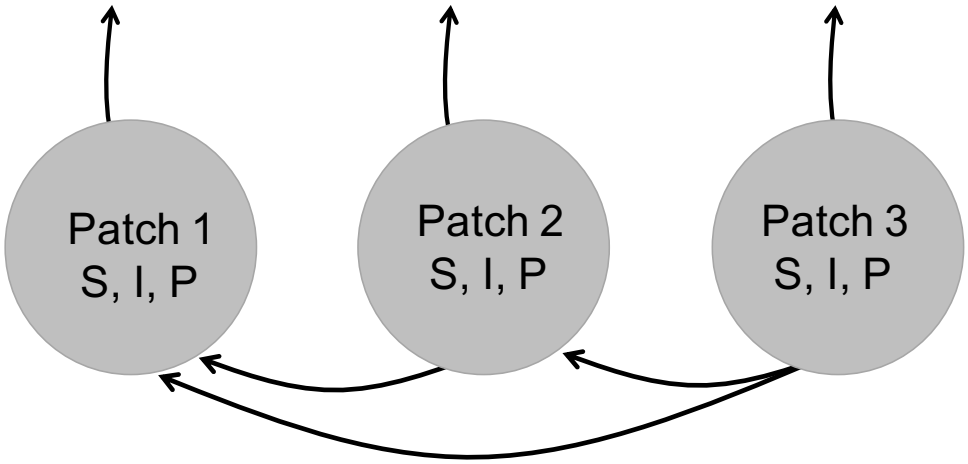

Figure 1. Model scheme. The population of hosts composed by susceptibles (S) and Infecteds (I) (depending of the infection state) and environmental pathogens (P) inhabit different patches (n patches, n=3 in this example). Dispersal of S, I and P occurs in all patches as represented by the black arrows. Individuals and pathogens are leaving each patch at a certain dispersal rate (specific for each subpopulation but the same for all patches) and function of the density of the patch. Each patch is also receiving individuals and pathogens from the patch nF (i.e. patch 3 in this example of three patches.

MODEL COMPONENTS, PROCESSES AND EQUATIONS

The main focus of this marine disease model is on a single species with multiple isolated subpopulations. The model is a general model for n patches, representing disease dynamics in discrete time. The model represents spatially distributed subpopulations, one next to but isolated from each other (a grid of 1 x n patches). In this study a three patch system is shown. In each patch, the host subpopulation is composed by two categories: susceptible individuals (S) and infected individuals (I) (depending of the infection state). In addition, environmental infectious pathogens (P) are also inhabiting the patches (Figure 1 and 2).

Susceptible individuals can be infected by both contact with infected individuals (i.e. pathogens can reside on the skin of the infected animals) and contact with free-living environmental pathogens. Infections can be produced (i) 'locally' within each patch and (ii) by infectious particles or infected animals immigrating from other patches (Figure 2). Individuals and pathogen population dynamics are forced by environmental stochasticity $\varepsilon_{n,t}$ which is coded to be as patch-specific but not species specific. However, for the simulation example in this document assumes a non patch-specific environmental stochasticity. Dispersal is assumed to be at a certain dispersal rate and function of the host population and pathogen population density in the source patches, respectively (Figure 2). More complicated connectivity can be imposed, but this should be a reasonable starting place.The purpose of these models is to analyze the spread of an infection due to migrating hosts and freely moving infectious pathogens. In this model, it is assumed that susceptible individuals become infected and it is not possible to be cured of the disease.

Susceptible host population

Susceptible individuals (S) (or H in the Matlab code) are lost by three processes: infection, natural mortality and migration. The disease transmission rate is controlled by β_I for contacts between susceptibles and infecteds and β_P for contacts between susceptibles and environmental pathogens (Equation 1). The transmission of disease is density dependent here, that is proportional to infected animal or environmental pathogen density (Figure 1). The mortality rate for susceptibles or hosts is m_S. Susceptible and infected animals have specific intrinsic growth rates, r_S and r_I, respectively, and population increase due to growth is assumed to be free of the disease, so that we only have this growth term in the susceptible population equation. The per capita growth rate for hosts and infected populations is controled by the total population density $(S+I)$ and host population carrying capacity KH_n (Equation 1). $\varepsilon_{n,t}$ is the environmental stochasticity forcing susceptible population dynamics and also infected and pathogen dynamics.

The equation describing the susceptible host dynamics before dispersal of individuals is given as:

$$S^*_{n,t+1} = S_{n,t}\, exp\left[r_S\left(1-\left(\frac{S_{n,t}+I_{n,t}}{KH_n}\right)\right) exp\left(\varepsilon_{n,t}\right)\right] + I_{n,t}\, exp\left[r_I\left(1-\left(\frac{S_{n,t}+I_{n,t}}{KH_n}\right)\right) exp\left(\varepsilon_{n,t}\right)\right] - TF_t\, S_{n,t}\left(\beta_I\, I_{n,t} + TF_t\, \beta_P\, P_{n,t}\right) - m_S\, S_{n,t} \quad (1)$$

Where $S_{n,t}$ is the density of the susceptible host population in patch n (n=1,2,3,...,nF) at time t, where nF is the number of the last patch. TF_t is the time factor reproducing the dynamics of pathogen inactivation (see pathogen section below) which somehow also affect the transmission by contact with infected individuals since the model is assuming contact with pathogens on the skin of the infected individuals.

With the incorporation of the dispersal process the equation is given as:

$$S_{n,t+1} = S^*_{n,t+1} - dispersS\left(S^*_{n,t+1} + I^*_{n,t+1}\right) + dispersS\left(S^*_{nF,t+1} + I^*_{nF,t+1}\right) \quad (2)$$

Where individuals are (i) leaving each patch at a rate $dispersH$, specific to the susceptible host population, and depending on the population density in the patch, and also (ii) receiving individuals from the last patch nF.

Infected host population

Infected individuals are transfered from the susceptible subpopulation to the infected subpopulation by the infection process through contact with infected individuals or environmental pathogens. Infective individuals are lost due to disease mortality mortality at a rate m_I. The infected population intrinsic growth affects the susceptible population since new individuals are not infected. That is, this infected growth term is on the susceptible population equation (Equation 1) as commented above.

The equation describing the infected host dynamics is given as:

$$I^*_{n,t+1} = I_{n,t}\, exp\left(1-\left(\frac{S_{n,t}+I_{n,t}}{KH_n}\right)\right) exp\left(\varepsilon_{n,t}\right)\Big] + I_{n,t}\left(\beta_I\, I_{n,t} + -TF_t\, \beta_P\, P_{n,t}\right) - m_I\, I_{n,t} \quad (3)$$

Similarly, the incorporation of the dispersal process changes the equation to:

$$I_{n,t+1} = I^*_{n,t+1} - dispersI\left(S^*_{n,t+1} + I^*_{n,t+1}\right) + dispersI\left(S^*_{nF,t+1} + I^*_{nF,t+1}\right) \quad (4)$$

Individuals leave each patch at rate $dispersI$, specific to the infected host population, and function of population density in the patch. Similarly to susceptibles individuals are also coming to each patch from the last patch nF.

Environmental pathogen population

Environmental pathogens or free-living pathogens are living in the same environment as the host population. Pathogen density depends on the intrinsic growth r_P, which in turn is modulated by pathogen inactivation time factor TF_t (Equation 6), and on the mortality pathogens m_P (Equation 5).

$$P^*_{n,t+1} = P_{n,t}\, exp\left[r_P\left(1-\left(\frac{P_{n,t}+I_{n,t}}{KP_n}\right)\right) exp\left(\varepsilon_{n,t}\right)\right] TF_t - m_P\, P_{n,t} \quad (5)$$

The time factor TF_t (Figure 3, Equation 6) appears in several equations (equations 1 and 5). This is a mechanism to have the pathogen activation to be function of time. TF_t here reproduces the effect of

high temperatures (summers) on the reduction of disease virulence (i.e. pathogens on the skin of infected individuals, see Equation 1) and growth of pathogens (Equation 5) given as follows:

$$TF(t) = (1 - min)\, A\, (1 + \cos\left(\frac{2\pi t - lag}{365}\right)^{\delta} + min \tag{6}$$

(7)

Where *min* is the minimum value of the wave, A is the amplitude, *lag* is the time lag, and δ is the skewness of the waves.

Similarly, the incorporation of the dispersal process changes the pathogen population equation to:

$$P_{n,t+1} = P^*_{n,t+1} - dispersP\left(P^*_{n,t+1}\right) + dispersP\left(P^*_{nF,t+1}\right) \tag{8}$$

RESULTS AND DISCUSSION

Sumulationa are considered with values for parameters in Table 1. Initially, there is a random number of susceptibles, infecteds and pathogens in each patch. The simulation has a time step of 0.005 day and runs for 1000 days.

Parameter	Definition	Unit	Value
r_S	Growth rate of susceptibles	time $^{-1}$	1.5
r_I	Growth rate of infecteds	time^{-1}	1.0
r_P	Growth rate of pathogens	time^{-1}	2.0
KH_n	Host population carrying capacity	Number of individuals	[2.0:4.0:6.0]
KP_n	Pathogen population carrying capacity	Number of individuals	[2.0:4.0:6.0]
β_I	Disease transmission rate by contact with infected individuals	Individuals^{-1} time^{-1}	$5{\cdot}10^{-2}$
β_P	Disease transmission rate by contact with environmental pathogens	Pathogens^{-1} time^{-1}	$2.5{\cdot}10^{-2}$
m_S	Natural mortality rate	time^{-1}	$5{\cdot}10^{-3}$
m_I	Disease mortality rate	time^{-1}	$5{\cdot}10^{-2}$
m_P	Pathogen natural mortality	time^{-1}	$5{\cdot}10^{-2}$
dispersS	Dispersal rate of host population	time^{-1}	$1{\cdot}10^{-1}$
dispersI	Dispersal rate of infected population	time^{-1}	$5{\cdot}10^{-2}$
dispersP	Dispersal rate of pathogens	time^{-1}	$2.5{\cdot}10^{-1}$
min	The minimum value of the wave for the time factor (TF)	time $^{-1}$	0.2
A	The amplitude of the wave for TF	time $^{-1}$	0.5
lag	The time lag for TF	time $^{-1}$	0
δ	The skewness of the wave for TF	time $^{-1}$	1.5

Table 1. Parameters of the model. The model has an implicit surface area for the parameters. The units used in the simulations are assumed to be days for time and m^2 for area. Values in parenthesis represent the vector for patch-specific parameter values [Patch 1: Patch 2: Patch 3]

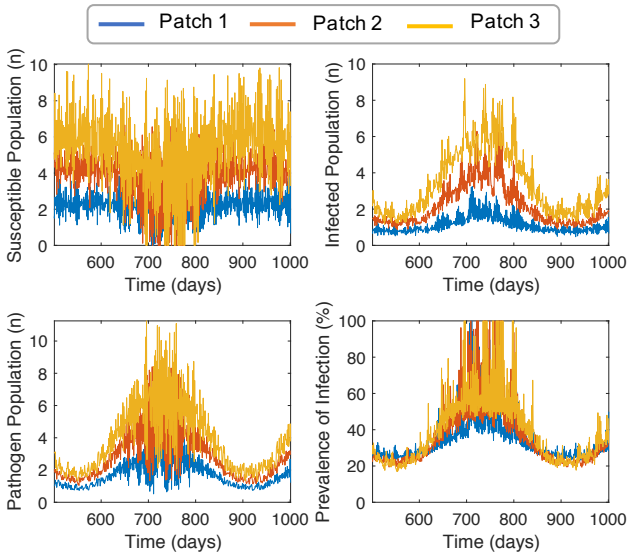

Figure 2. Susceptible (a), infected (b) and (c) pathogen population dynamics, and (d) prevalence of infection in each patch. The simulations are run for 1000 days and presented for the last 500 days. Day 1 represents January 1.

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Population and pathogen dynamics in each patch are presented for last 500 days of the simulation (Figure 4) and last 50 days of the simulation for a more detailed picture (Figure 5). Figure 6 shows the net migration (i.e. the net result after gaining of individuals or pathogens coming from the last patch nF and loss due to some proportion of the populations leaving the patch).

The results show that with this parameterization host and pathogen populations are locally and globally persistent. Note that initial population values for S, I and pathogens are random, so that it could be necessary to run the model a few times to have a solution with a persistent population.

Increasing carrying capacity of hosts and pathogens between patches (from Patch 1 to 3) results in higher populations and relatively higher prevalence of infection (Figures 4 and 5). The 'winter activation' of pathogens (c.a. day 750) increases the number of pathogen population to a maximum (Figure 4, bottom left) which results in similar increase of the infected population (Figure 4, top right). The fluctuations of the populations at this maximum are also more intense.

The ' summer inactivation' has the opposite effect with a reduction of the number of infections in all patches. The pathogen inactivation effect on the susceptible population is less intense and more fluctuating due to the compensation by population growth.

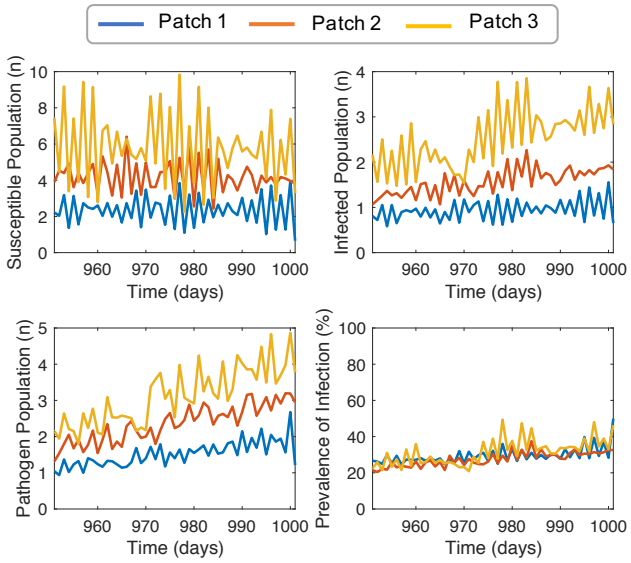

Figure 3. Susceptible (a), infected (b) and (c) pathogen population dynamics, and (d) prevalence of infection in each patch. The simulations are run for 1000 days and presented for the last 50 days. Day 1 represents January 1.

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The dispersal of the host population, in terms of net migration, is positive for patch 1 and 2, due to the strong 'source' effect of the patch 3 with higher population density. The net migration is higher for susceptibles (Figure 6, top left) than for infecteds (Figure 6, top right, patch 1 and 2) because of a higher dispersal ability of susceptible animals; the model assumes some limitation of movement when the animal is sick. The pathogen dispersal rate is assumed to be highest than that for the host, however this is only mirrored in a higher net migration when the pathogen inactivation is relatively low (Figure 6, bottom left, see increasing net migration as the pathogens are activated by colder temperatures (see time factor in Figure 3).

The net migration for the patch 3 is null. The model assumes, in this last and higher carrying capacity patch, that the host population individuals and pathogens are leaving this patch at the same specific rate as immigrants are arriving. This patch acts within the metapopulation as the source for the rest of the patches. Note that as some initial values, such as host and pathogen populations, are random other simulations could lead to different results in net migration.

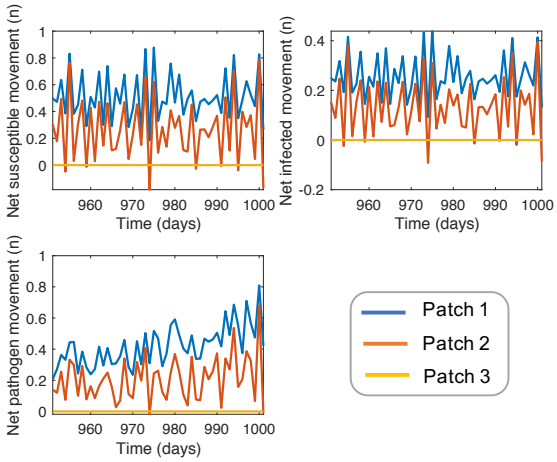

Figure 4. Net migration dynamics for susceptible (a), infected (b) and (c) pathogen population in each patch. The simulations are run for 1000 days and presented for the last 50 days. Day 1 represents January 1.

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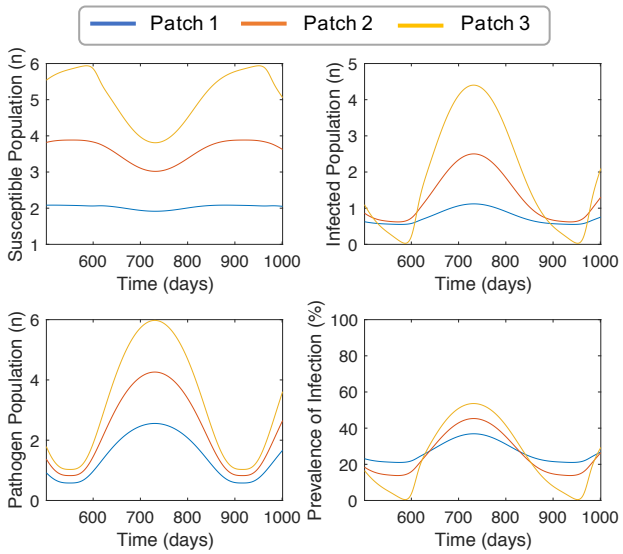

Figure 5. Deterministic model. Susceptible (a), infected (b) and (c) pathogen population dynamics, and (d) prevalence of infection in each patch. The simulations are run for 1000 days and presented for the last 500 days. Day 1 represents January 1.

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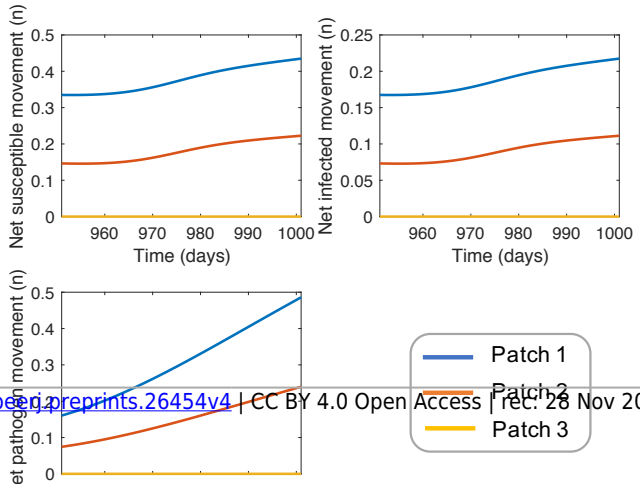

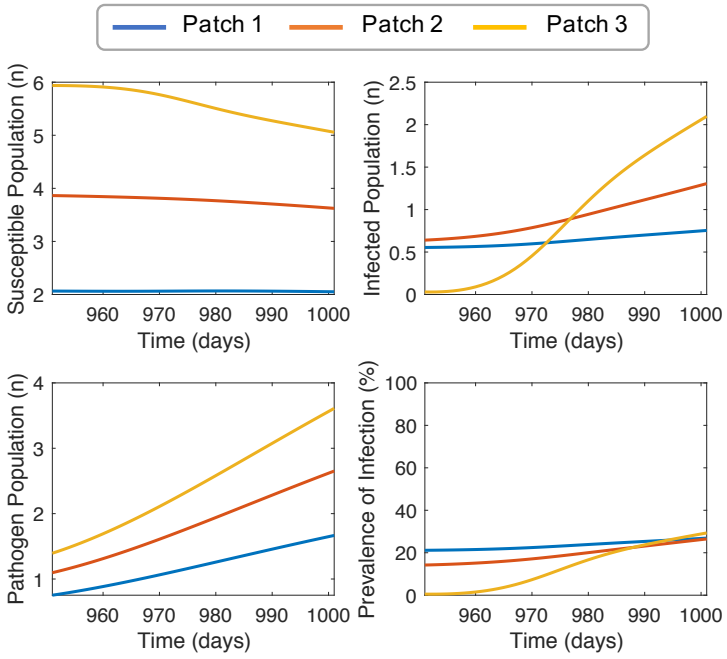

Figure 6. Deterministic model. Susceptible (a), infected (b) and (c) pathogen population dynamics, and (d) prevalence of infection in each patch. The simulations are run for 1000 days and presented for the last 50 days. Day 1 represents January 1.

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