



Oscillations and waves in a virally infected plankton system Part I: The lysogenic stage[☆]

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Abstract

A model of phytoplankton–zooplankton dynamics is considered for the case of lysogenic viral infection of the phytoplankton population. The phytoplankton population is split into a susceptible (S) and an infected (I) part. Both parts grow logistically, limited by a common carrying capacity. Zooplankton (Z) is grazing on susceptibles and infected. The local analysis of the S – I – Z differential equations yields a number of stationary and/or oscillatory regimes and their combinations. Correspondingly interesting is the spatio-temporal behaviour, modelled by deterministic and stochastic reaction–diffusion equations. Spatial spread or suppression of infection will be presented just as well as competition of concentric and/or spiral population waves with non-oscillatory sub-populations for space. The external noise can enhance the survival and spread of susceptibles and infected, respectively, that would go extinct in a deterministic environment.

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1. Introduction

Numerous papers have been published about pattern formation and chaos in minimal prey–predator

models of phytoplankton–zooplankton dynamics (Scheffer, 1991a; Malchow, 1993; Pascual, 1993; Truscott and Brindley, 1994; Malchow, 1996, 2000b; Malchow et al., 2001, 2004b). Different routes to local and spatio-temporal chaos (Scheffer, 1991b; Kuznetsov et al., 1992; Rinaldi et al., 1993; Sherratt et al., 1995; Scheffer et al., 1997; Steffen et al., 1997; Petrovskii and Malchow, 1999, 2001; Malchow et al., 2002), diffusion- and differential-flow-induced standing and travelling waves (Malchow, 1993; Menzinger and Rovinsky, 1995; Malchow, 2000a; Satnoianu and

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Menzinger, 2000; Satnoianu et al., 2000; Malchow et al., 2003) as well as target patterns and spiral waves (Medvinsky et al., 2000, 2002) have been found. Also the impact of external noise on patchiness and transitions between alternative stable population states has been studied (Steele and Henderson, 1992; Malchow et al., 2002; Sarkar and Chattopadhyay, 2003; Malchow et al., 2004a).

Much less than on plankton patchiness and blooming is known about marine viruses and their role in aquatic ecosystems and the species that they infect, for reviews Fuhrman (1999); Suttle (2000) as well as Wommack and Colwell (2000). There is some evidence that viral infection might accelerate the termination of phytoplankton blooms (Jacquet et al., 2002).

Also the understanding of the importance of lysogeny is just at the beginning (Wilcox and Fuhrman, 1994; Jiang and Paul, 1998; McDaniel et al., 2002; Ortmann et al., 2002). Contrary to lytic infections with destruction and without reproduction of the host cell, lysogenic infections are a strategy whereby viruses integrate their genome into the host's genome. As the host reproduces and duplicates its genome, the viral genome reproduces, too.

Mathematical models of the dynamics of virally infected phytoplankton populations are rare as well. The already classical publication is by Beltrami and Carroll (1994), more recent work is of Chattopadhyay and Pal (2002) and Chattopadhyay et al. (2003). All these papers deal with lytic infections and mass action incidence functions (Nold, 1980; Dietz and Schenzle, 1985; McCallum et al., 2001).

In this paper, we focus on modelling the influence of lysogenic infections and proportionate mixing incidence function (frequency-dependent transmission) on the local and spatio-temporal dynamics of interacting phytoplankton and zooplankton. Furthermore, the impact of multiplicative noise (Allen, 2003; Anishenko et al., 2003) is investigated.

2. The mathematical model

The model by Scheffer (1991a) for the prey–predator dynamics of phytoplankton P and zooplankton Z is used as the starting point. It reads in time t and two spatial dimensions $\vec{r}=\{x, y\}$ with dimensionless

quantities, scaled following Pascual (1993)

$$\frac{\partial P}{\partial t} = rP(1 - P) - \frac{aP}{1 + bP}Z + d\Delta P, \quad (1)$$

$$\frac{\partial Z}{\partial t} = \frac{aP}{1 + bP}Z - m_3Z + d\Delta Z. \quad (2)$$

There is logistic growth of the phytoplankton with intrinsic rate r and Holling-type II grazing with maximum rate a as well as natural mortality with rate m_3 of the zooplankton. The growth rate r is scaled as the ratio of local rate r_{loc} and spatial mean $\langle r \rangle$. The diffusion coefficient d describes eddy diffusion. Therefore, it must be equal for both species. The dynamics of a, top predator, i.e., planktivorous fish is neglected because the focus of this paper is on the influence of virally infected phytoplankton. The phytoplankton population P is split into a susceptible part X_1 and an infected portion X_2 . Zooplankton is simply renamed to X_3 . Then, the model system reads for symmetric inter- and intraspecific competition of susceptibles and infected

$$\frac{\partial X_i(\vec{r}, t)}{\partial t} = f_i[X(\vec{r}, t)] + d\Delta X_i(\vec{r}, t), \quad i = 1, 2, 3; \quad (3)$$

where

$$f_1 = r_1 X_1 (1 - X_1 - X_2) - \frac{a X_1}{1 + b(X_1 + X_2)} X_3 - \lambda \frac{X_1 X_2}{X_1 + X_2}, \quad (3a)$$

$$f_2 = r_2 X_2 (1 - X_1 - X_2) - \frac{a X_2}{1 + b(X_1 + X_2)} X_3 + \lambda \frac{X_1 X_2}{X_1 + X_2} - m_2 X_2, \quad (3b)$$

$$f_3 = \frac{a(X_1 + X_2)}{1 + b(X_1 + X_2)} X_3 - m_3 X_3. \quad (3c)$$

Proportionate mixing with transmission coefficient λ as well as an additional disease-induced mortality of infected (virulence) with rate m_3 are assumed. The vector of population densities is $\mathbf{X} = \{X_1, X_2, X_3\}$. In the case of lytic infection, the first term on the right-hand side of Eq. (3b) would describe the losses due to natural mortality and competition. Here, lysogenic infections with $r_1 = r_2 = r$ will be considered.

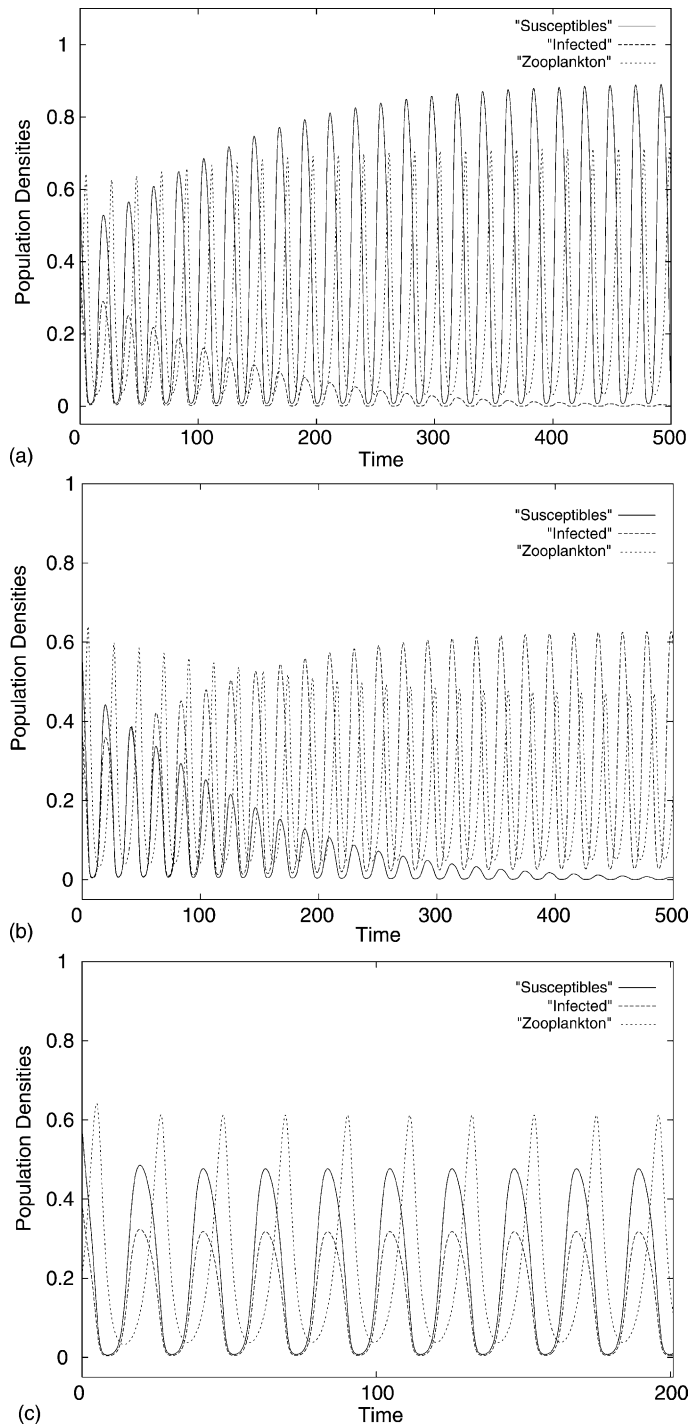


Fig. 1. Local dynamics with (a) extinction of infected for $m_2 > \lambda$, (b) extinction of susceptibles for $m_2 < \lambda$ and (c) coexistence of susceptibles X_1 , infected X_2 and zooplankton X_3 for $m_2 = \lambda$, $m_3 = 0.5$.

However, it is a highly simplified model because the growth rate of susceptibles is usually higher than that of infected (Suttle et al., 1990). Furthermore, the lysogenic replication cycle of viruses is very sensitive to environmental changes and very quickly switches to the lytic cycle. This is left for the second part of the paper.

Furthermore, multiplicative noise is introduced in Eq. (3) in order to study environmental fluctuations, i.e.,

$$\frac{\partial X_i(\vec{r}, t)}{\partial t} = f_i[X(\vec{r}, t)] + d\Delta X_i(\vec{r}, t) + \omega_i[X(\vec{r}, t)]\xi_i(\vec{r}, t), \quad i = 1, 2, 3; \quad (4)$$

where $\xi_i(\vec{r}, t)$ is a spatio-temporal white Gaussian noise, i.e., a random Gaussian field with zero mean and delta correlation

$$\langle \xi_i(\vec{r}, t) \rangle = 0, \quad \langle \xi_i(\vec{r}_1, t_1)\xi_i(\vec{r}_2, t_2) \rangle = \delta(\vec{r}_1 - \vec{r}_2)\delta(t_1 - t_2), \quad i = 1, 2, 3. \quad (4a)$$

$\omega_i[X(\vec{r}, t)]$ is the density dependent noise intensity. The stochastic modelling of population dynamics requires this density dependence, i.e., multiplicative noise. Throughout this paper, it is chosen

$$\omega_i[X(\vec{r}, t)] = \omega X_i(\vec{r}, t), \quad i = 1, 2, 3; \quad \omega = \text{constant} \quad (4b)$$

3. The deterministic local dynamics

At first, the local dynamics is studied, i.e., we look for stationary and oscillatory solutions of system (3) for $d = 0$. Stationary solutions are marked by X_i^{Sno} , $i = 1, 2, 3$. Furthermore, we introduce the parameter

$$m_3^{\text{cr}} = \frac{a(X_1^{\text{Sno}} + X_2^{\text{Sno}})}{1 + b(X_1^{\text{Sno}} + X_2^{\text{Sno}})}.$$

Analytical and numerical investigations yield the following selected equilibria.

- (0) Trivial solution $X_1^{\text{S00}} = X_2^{\text{S00}} = X_3^{\text{S00}} = 0$, always unstable.
- (1) Endemic states with and without predation.

- (a) $X_1^{\text{S11}} > 0, X_2^{\text{S11}} > 0, X_3^{\text{S11}} > 0$ if $m_2 = \lambda$ and $m_3 < m_3^{\text{cr}}$, oscillatory or non-oscillatory stable, multiple stable equilibria possible.
- (b) $X_1^{\text{S12}} > 0, X_2^{\text{S12}} > 0, X_3^{\text{S12}} > 0$ if $m_2 = \lambda$ and $m_3 < m_3^{\text{cr}}$, non-oscillatory stable.
- (2) Extinction of infected with and without predation.
 - (a) $X_1^{\text{S21}} > 0, X_2^{\text{S21}} > 0, X_3^{\text{S21}} > 0$ if $m_2 > \lambda$ and $m_3 < m_3^{\text{cr}}$, oscillatory or non-oscillatory stable.
 - (b) $X_1^{\text{S22}} > 0, X_2^{\text{S22}} > 0, X_3^{\text{S22}} > 0$ if $m_2 > \lambda$ and $m_3 < m_3^{\text{cr}}$, non-oscillatory stable.
- (3) Extinction of susceptibles with and without predation.
 - (a) $X_1^{\text{S31}} > 0, X_2^{\text{S31}} > 0, X_3^{\text{S31}} > 0$ if $m_2 > \lambda$ and $m_3 < m_3^{\text{cr}}$, oscillatory or non-oscillatory stable.
 - (b) $X_1^{\text{S32}} > 0, X_2^{\text{S32}} > 0, X_3^{\text{S32}} > 0$ if $m_2 > \lambda$ and $m_3 < m_3^{\text{cr}}$, non-oscillatory stable.

For $m_2 > \lambda$, the infected go extinct (solutions 2a and b), for $m_2 > \lambda$, the susceptibles do (solutions 3a and b). In the case of $m_2 = \lambda$, susceptibles and infected coexist (endemic states 1a and b). Because of the symmetry of the growth terms of susceptibles and infected, the initial conditions determine their final dominance in the endemic state, i.e., if $X_1(t = 0) > X_2(t = 0)$ then $X_1(t) > X_2(t) \forall t$. A corresponding example is presented in Fig. 1 for $r = 1$ and $a = b = 5$. These three parameter values will be kept for all simulations.

The stable prey–predator oscillations are presentations of solutions (2a), (3a) and (1a), respectively.

The local dynamics of model (3) is well-known and simple for a single prey. There can be a Hopf bifurcation point, e.g., for decreasing mortality m_3 of the predator. For the parameters in Fig. 1, this point has been already passed. A slight increase of m_3 from 0.5 to 0.625 yields multiple equilibria and also demonstrates the dependence of the endemic states on the initial conditions. In Fig. 2a for $X_1(0) > X_2(0)$, one still finds the oscillatory solution (1a). However, the opposite choice $X_2(0) > X_1(0)$ in Fig. 2b, results in damping of the oscillations, i.e., the different initial conditions belong to different basins of attraction in phase space.

After this rough investigation of the deterministic local behaviour, we proceed now to the stochastic spatial dynamics.

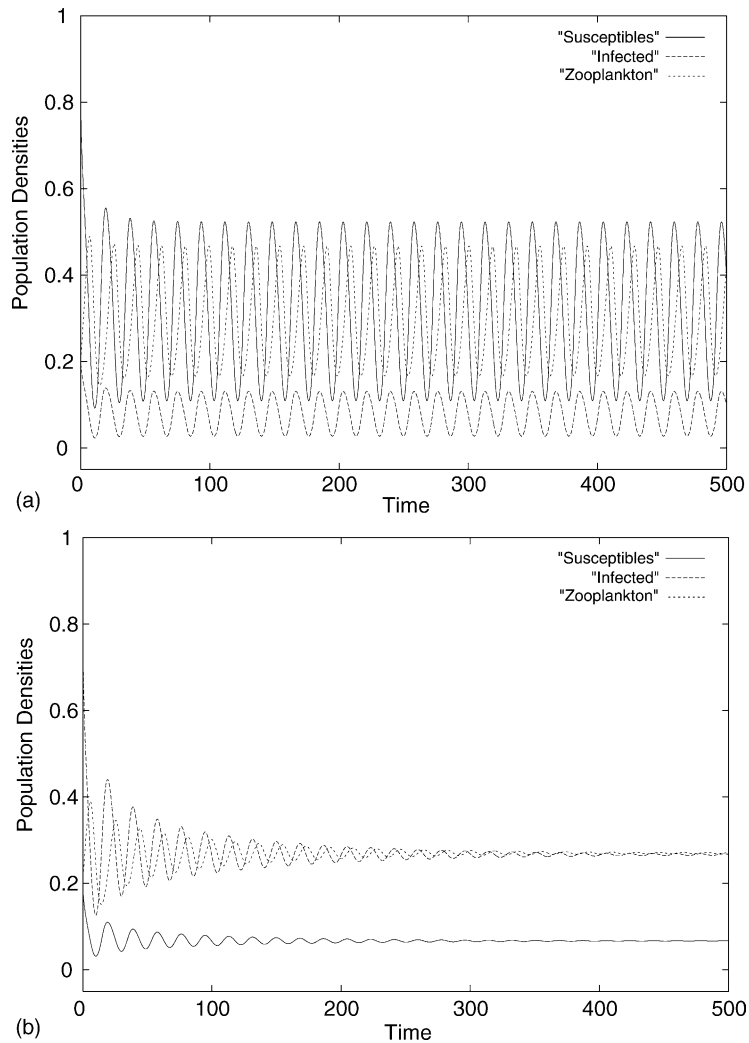


Fig. 2. Endemic state (1a) with initial conditions in different basins of attraction: (a) stable and (b) damped oscillation. $m_2 = \lambda = 0.2$, $m_3 = 0.625$.

4. The deterministic and stochastic spatial dynamics

Much has been published about the spatio-temporal selforganization in prey–predator communities, modelled by reaction–diffusion (–advection) equations, the references in the introduction. Much less is known about equation-based modelling of the spatial spread of epidemics, a small collection of papers includes Grenfell et al. (2001); Abramson et al. (2003); Lin et al. (2003) and Zhdanov (2003).

In this paper, we consider the spatio-temporal dynamics of the plankton model (4), i.e., zooplankton, grazing on susceptible and virally infected phytoplankton, under the influence of environmental noise and diffusing in horizontally two-dimensional space. The diffusion terms have been integrated using the semi-implicit Peaceman-Rachford alternating direction scheme, Thomas (1995). For the interactions and the Stratonovich integral of the noise terms, the explicit Euler-Maruyama scheme has been applied (Kloeden and Platen, 1992; Higham, 2001).

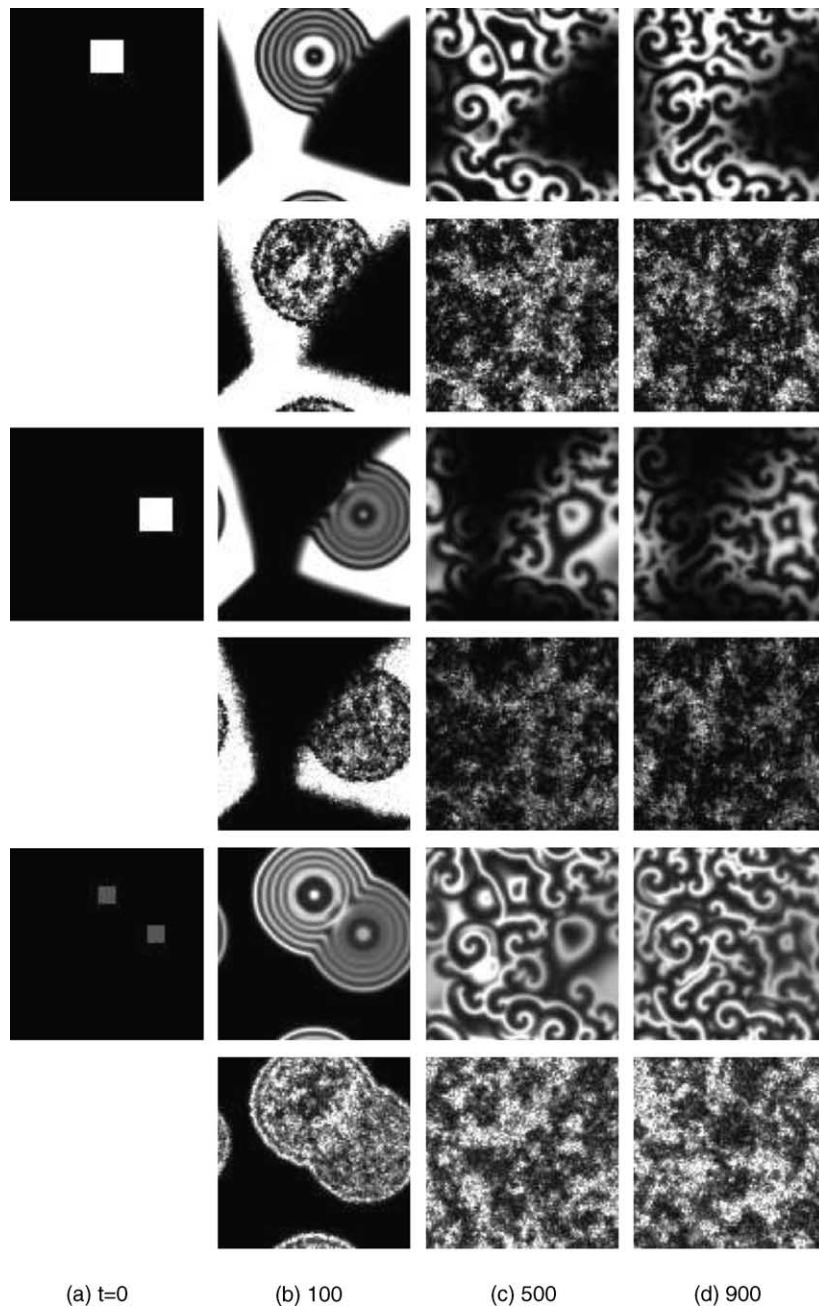


Fig. 3. Spatial coexistence of susceptibles (two upper rows), infected (two middle rows) and zooplankton (two lower rows) for $m_2 = \lambda = 0.2$, $m_3 = 0.5$, $d = 0.05$. No noise $\omega = 0$ and 0.25 noise intensity, respectively, with equal initial conditions (left column). Periodic boundary conditions.

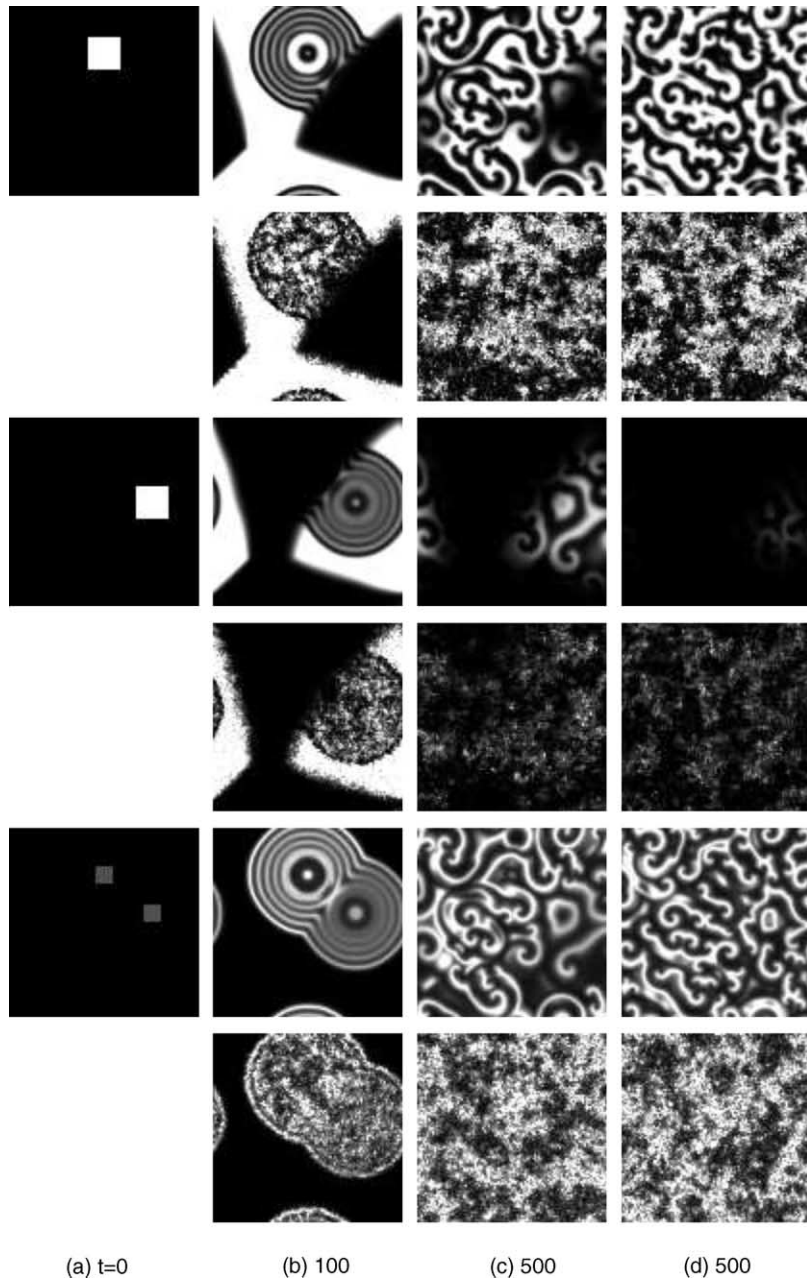


Fig. 4. Spatial coexistence of susceptibles (two upper rows) and zooplankton (two lower rows). Extinction of infected (third row) for $m_2 = 0.2 > \lambda = 0.19$, $m_3 = 0.5$, $d = 0.05$ and no noise. Survival of infected for $\omega = 0.25$ noise intensity (fourth row).

The following series of figures summarizes the results of the spatio-temporal simulations for growth and interaction parameters from Section 3, but now including diffusion and noise.

Periodic boundary conditions have been chosen for all simulations.

The initial conditions are localized patches in empty space, and they are the same for deterministic and

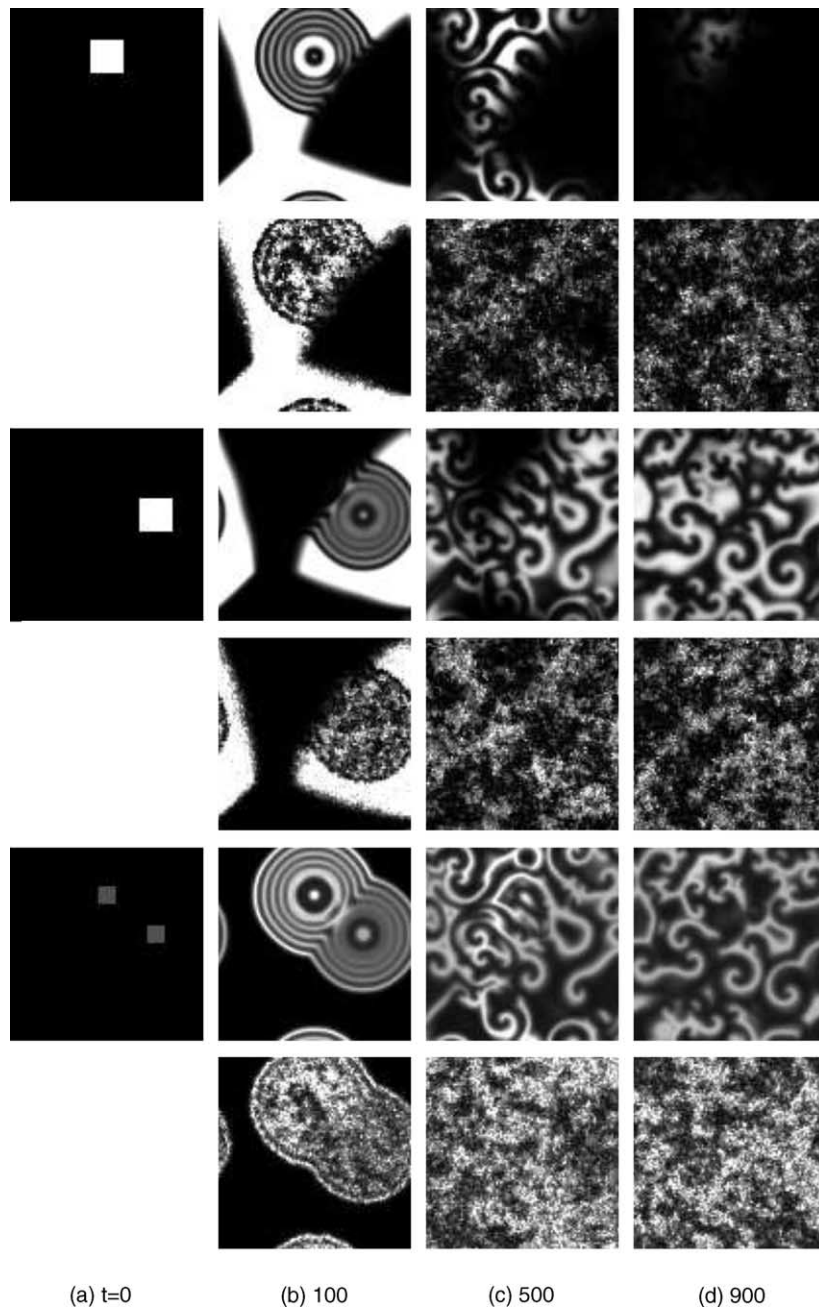


Fig. 5. Spatial coexistence of infected (two middle rows) and zooplankton (two lower rows). Extinction of susceptibles (first row) for $m_2 = 0.2 < \lambda = 0.21$, $m_3 = 0.5$, $d = 0.05$ and no noise. Survival of susceptibles for $\omega = 0.25$ noise intensity (second row).

stochastic simulations. They can be seen in the left column of all following figures. The first two rows show the dynamics of the susceptibles for deterministic and stochastic conditions, the two middle rows show the

infected and the two lower rows the zooplankton. For Figs. 3–5, there are two patches, one with zooplankton surrounded by susceptible phytoplankton (upper part of the model area) and one with zooplankton sur-

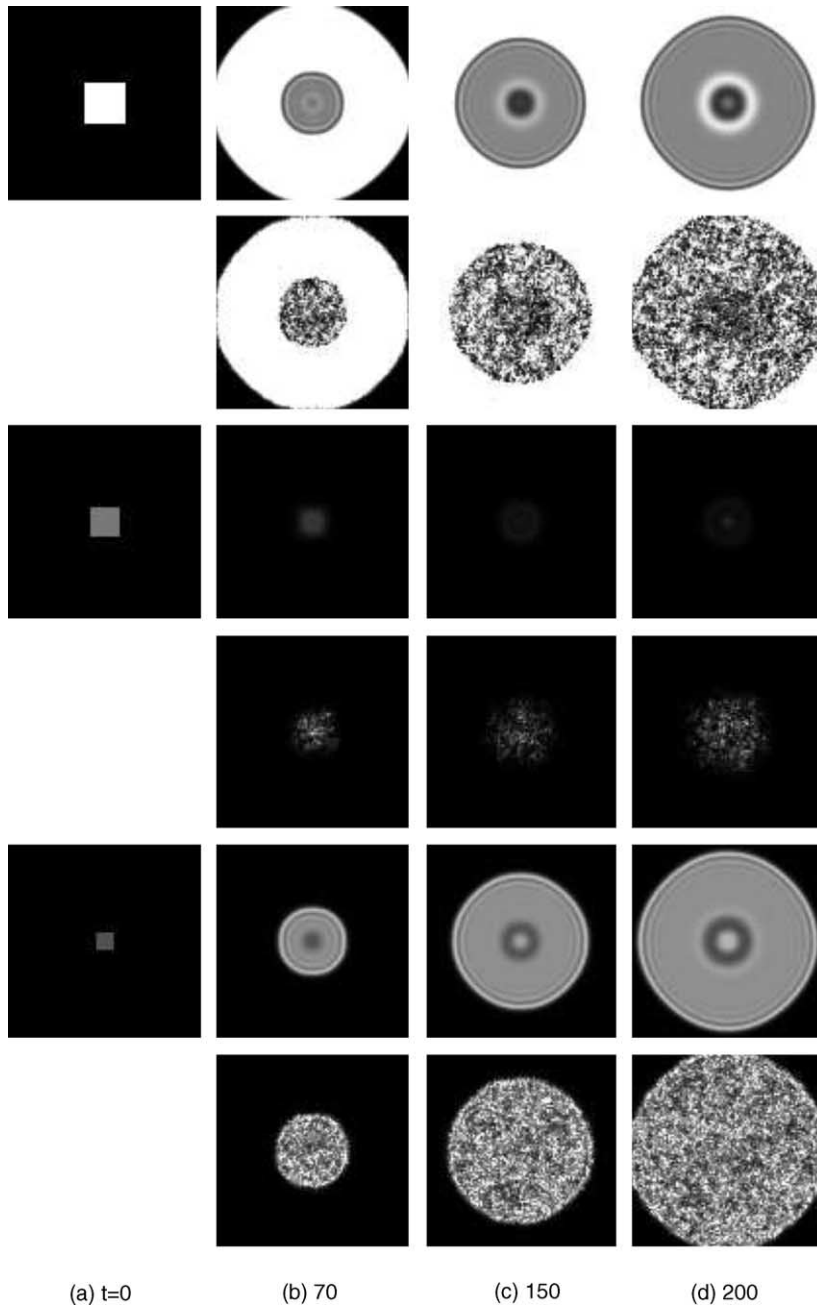


Fig. 6. Spatial coexistence of susceptibles (two upper rows), infected (two middle rows) and zooplankton (two lower rows) for $m_2 = \lambda = 0.2$, $m_3 = 0.625$, $d = 0.05$. Without noise trapping and almost extinction of infected in the center (third row). With $\omega = 0.25$ noise intensity noise-enhanced survival and escape of infected (fourth row). Phenomenon of dynamic stabilization of a locally unstable equilibrium (first and fifth row).

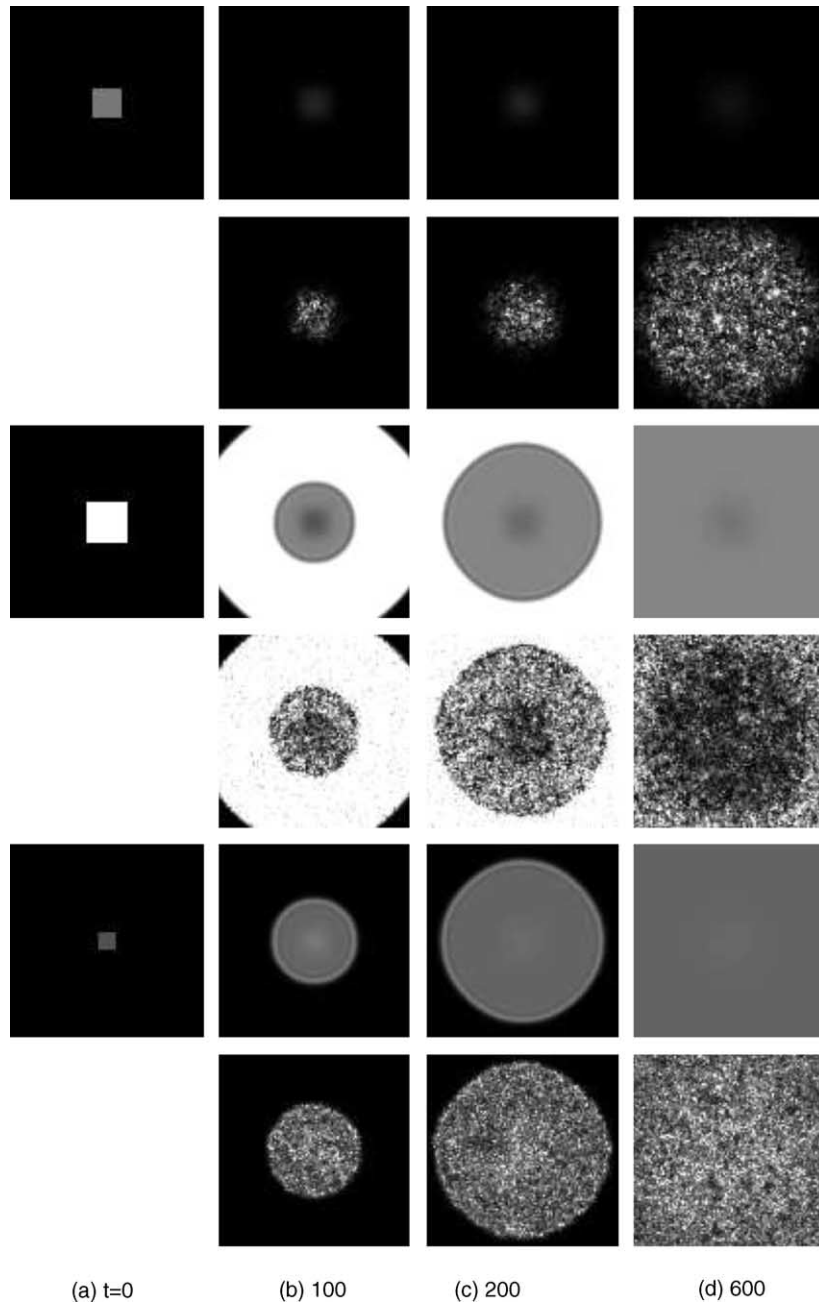


Fig. 7. Spatial coexistence of susceptibles (two upper rows), infected (two middle rows) and zooplankton (two lower rows) for $m_2 = \lambda = 0.2$, $m_3 = 0.625$, $d = 0.05$. Without noise trapping and almost extinction of susceptibles in the center (first row). With $\omega = 0.25$ noise intensity noise-enhanced survival and escape of susceptibles (second row).

rounded by infected (on the right of the model area). For Figs. 6 and 7, there are central patches of all three species. In Fig. 6, susceptibles are ahead of infected that are ahead of zooplankton. In Fig. 7, infected are ahead of susceptibles that are ahead of zooplankton. In all figures, this special initial configuration leads at first to the propagation of concentric waves for the deterministic case in rows 1, 3 and 5. For the stochastic case in rows 2, 4 and 6, these (naturally unrealistic) waves are immediately blurred and only a leading diffusive front remains.

In Fig. 3, one can see the final spatial coexistence of all three species for $m_2 = \lambda$. The localized initial patches generate concentric waves that break up after collision and form spiral waves in a deterministic environment. The noise only blurs these unrealistic patterns. The grey scale changes from high population densities in white colour to vanishing densities in black.

This changes for $m_2 > \lambda$ and $m_2 < \lambda$ in Figs. 4 and 5, respectively. Whereas in the deterministic case infected or susceptibles go extinct, the noise enhances their survival and spread under unfavourable conditions.

In Fig. 6, the deterministic simulations yield the dynamic stabilization of the locally unstable focus in space and a long plateau is formed with a leading diffusive front ahead, cf. Petrovskii and Malchow (2000); Malchow and Petrovskii (2002). Furthermore, the infected are somehow trapped in the center and go almost extinct. The noise enhances the “escape”, spread and survival of the infected.

In Fig. 7, the dynamic stabilization is not so clearly seen. However, the noise enhances the “escape”, spread and survival of the susceptibles here.

5. Conclusions

A conceptual biomass-based model of phytoplankton–zooplankton prey–predator dynamics has been investigated for temporal, spatial and spatio-temporal dissipative pattern formation in a, deterministic and noisy environment, respectively. It has been assumed that the phytoplankton is partly virally infected and the virus has a lysogenic replication cycle, i.e., also the infected phytoplankton is still able to reproduce.

The equal growth rates of susceptibles and infected have led to the situation that, in a non-fluctuating environment, the ratio of the mortality of the infected and the transmission rate of the infection controls coexistence, survival or extinction of susceptibles and infected. A fluctuating environment enhances the survival and the spatial spread of the “endangered” species. However, noise has not only supported the spatio-temporal coexistence of susceptibles and infected but it has been necessary to blur distinct artificial population structures like target patterns or spirals and to generate more realistic fuzzy patterns.

Forthcoming work has to consider differing growth rates of susceptible and infected species as well as the critical noise-induced switch from lysogenic to lytic viral replications and the resulting spatio-temporal dynamics of the plankton populations.

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