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Oscillations and waves in a virally infected plankton system Part II: Transition from lysogeny to lysis

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ABSTRACT

A model of phytoplankton–zooplankton prey–predator dynamics is considered for the case of 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) as a Holling-type II predator is grazing on susceptibles and infected. The local and spatial analyses of the S–I–Z model with lysogenic infection have been presented in a previous paper (Malchow et al., 2004b. Oscillations and waves in a virally infected plankton system: Part I: The lysogenic stage. Ecol. Complexity 1 (3), 211–223). This lysogenic stage is rather sensitive to environmental variability. Therefore, the effect of a transition from lysogeny to lysis is investigated here. The replication rate of the infected species instantaneously falls to zero. A deterministic and a more realistic stochastic scenario are described. The spatiotemporal behaviour, modelled by deterministic and stochastic reaction-diffusion equations, is numerically determined. It is shown that the extinction risk of the infected is rather high in the deterministic system, whereas the environmental noise enhances their chance of spatial spread and survival.

1. Introduction

The discovery that viruses are extremely abundant in marine environments (by more than three orders of magnitude than previously thought) and that they infect a wide spectrum of hosts ranging from bacteria to eukaryotic primary producers (Bergh et al., 1989; Proctor and Fuhrman, 1990; Suttle et al., 1990) has stipulated a lot of research during the last one and a half decade. It is now known that marine viruses influence species diversity, phytoplankton mortality, gene transfer, global biogeochemical cycles and the termination of algal blooms. Although they are regarded as important members of the microbial food web and regulating factors in marine ecosystems, the understanding of the role of viral infection in the phytoplankton population is just at its beginning and continues to unfold; for recent reviews, see Fuhrman (1999), Suttle (2000b), Suttle (2005), Weinbauer (2004), Wilhelm and Suttle (1999) and Wommack and Colwell (2000).

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There are two main replication cycles of viruses that infect phytoplankton (Fuhrman and Suttle, 1993; Suttle, 2000a; Mann, 2003): (i) lytic (or virulent) infections with destruction and without reproduction of the host and (ii) lysogenic (or temperate) infections in which viruses integrate their genome into the host's genome and multiply along with the host until the lytic cycle is induced. The shift to the lytic mode can be triggered by a variety of environmental stresses such as radiation, pollution, temperature changes and nutrient depletion or may occur spontaneously. However, the exact mechanisms controlling the switch from lysogenic to lytic existence are currently unknown (Suttle, 2000a; Sullivan et al., 2003; Bidle and Falkowski, 2004; Hewson et al., 2004; McDaniel and Paul, 2005).

Lysogeny in freshwater filamentous cyanobacteria (which are major primary producers) is well-known since the early 1970s, cf. the review by Suttle (2000a) and the references therein. The relative importance of lysis and lysogeny in the marine environment is still unclear (Wilson and Mann, 1997; Jiang and Paul, 1998; Suttle, 2000a; Wommack and Colwell, 2000; Chen and Lu, 2002; Stopar et al., 2004), but there is increasing evidence for the occurrence of lysogeny in marine representatives. Sode et al. (1994) were the first to isolate a lysogen infecting a cultured *Synechococcus* species from the coastal waters off of Kyushu, Japan, and later described the induction by heavy metals (Sode et al., 1997). Lysogeny in the marine filamentous form *Phormidium* sp. has been reported by Ohki and Fujita (1996). There is also a report describing the induction in cultured and natural samples of the filamentous cyanobacterium *Trichodesmium* sp. (Ohki, 1999). Recently, it has been demonstrated that lysogeny occurs in natural populations of marine *Synechococcus*, namely in Tampa Bay, FL, USA (McDaniel et al., 2002), and during a natural bloom in a pristine fjord in British Columbia, Canada (Ortmann et al., 2002). Moreover, several studies provide evidence that viruses establish lysogenic associations and were induced to the lytic mode (Waterbury and Valois, 1993; Wilson et al., 1996, 1998; Marston and Sallee, 2002; Sullivan et al., 2003; Hewson et al., 2004).

Mathematical models of the growth and interactions of virally infected phytoplankton populations are correspondingly rare. The already classical publication is by Beltrami and Carroll (1994). They showed that a lytic viral infection can destabilize an otherwise stable phytoplankton–zooplankton food chain. In combination with seasonal forcing, their model exhibits irregular fluctuations which mimic the recurrent bloom patterns of the dinoflagellate *Noctiluca scintillans* in the German Bight (Uhlig and Sahling, 1992) as well as of diatoms at Scripps Pear at La Jolla (Tont, 1976). More recent work is of Chattopadhyay and Pal (2002) and Singh et al. (2004), but based on some debatable assumptions: recovery from disease, zooplankton feeds 10 times more on infected phytoplankton (Chattopadhyay and Pal, 2002) and infected phytoplankton is more vulnerable to predation by zooplankton (Singh et al., 2004). Moreover, both articles assume mass action transmission of the disease.

In this paper, we draw upon the spatially extended models by Malchow et al. (2004b, 2005) who observed oscillations and waves in a trophic food chain with more realistic Holling-types II and III grazing and frequency-dependent disease transmission. The latter is also called proportionate mixing or standard incidence (Nold, 1980; Hethcote, 2000; McCallum et al., 2001). These models consider lysogenic infection before the switch to the lytic cycle (thus actually resembling a chronic infection). Hilker and Malchow (2006) have provided a detailed mathematical and numerical analysis of the local model for lysogenic/chronic and lytic infections.

Here we focus on models incorporating the switch from the lysogenic to the lytic cycle and its consequences for the local and spatiotemporal dynamics of interacting phytoplankton and zooplankton. In the next section, the basic mathematical model is introduced. Then we consider a deterministic induction to lytic infection in the local dynamics. Section 4 deals with both a deterministic and stochastic switch from lysogeny to lysis in the spatially extended model. Furthermore, the impact of multiplicative noise (García-Ojalvo and Sancho, 1999; Allen, 2003; Anishenko et al., 2003) in the population dynamics is investigated.

2. The basic mathematical model

As in Part I of this paper (Malchow et al., 2004b), the prey–predator dynamics of phytoplankton and zooplankton is described by a standard model (Rosenzweig and MacArthur, 1963; Steele and Henderson, 1981; Scheffer, 1991; Malchow, 1993; Pascual, 1993). The phytoplankton population is split into a susceptible part X_1 and an infected portion X_2. Their growth rates $r_{1,2}$ are scaled as the ratio of the local rates $r_{1,2}^{\mathrm{loc}}$ and the spatial mean $\langle r \rangle$. Zooplankton X_3 is a Holling-type II grazer with maximum grazing rate a/b and natural mortality m_3. Then, the model system reads for symmetric inter- and intraspecific competition of susceptibles and infected in time t and two horizontal dimensions $\vec{r} = \{x, y\}$

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

where

$$f_1 = r_1 X_1(1 - X_1 - X_2) - \frac{aX_1}{1 + b(X_1 + X_2)} X_3 - \lambda \frac{X_1 X_2}{X_1 + X_2}, \tag{1a}$$

$$f_2 = r_2 X_2(1 - X_1 - X_2) - \frac{aX_2}{1 + b(X_1 + X_2)} X_3 + \lambda \frac{X_1 X_2}{X_1 + X_2} - m_2 X_2, \tag{1b}$$

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

All quantities are dimensionless. The diffusion coefficient d describes eddy diffusion. Therefore, it must be equal for both species. Proportionate mixing with transmission coefficient λ as well as an additional disease-induced mortality of infected (virulence) with rate m_2 is 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. (1b) would describe the losses due to natural mortality and competition. At first, lysogenic infections with $r_1 \geq r_2 > 0$ will be considered. The growth rate of susceptibles is often higher than that of infected

(Suttle et al., 1990). Secondly, the lysogenic viral replication cycle switches to the lytic one due to its high sensitivity to environmental fluctuations. In order to model the latter, multiplicative noise is introduced in Eq. (1), i.e.,

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

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

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

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

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

3. The local dynamics with deterministic switch

The local dynamics ($d = 0$) has been described by Malchow et al. (2004b) for the lysogenic case and $r_1 = r_2$. A general and more detailed analysis has been presented by Hilker and Malchow (2006). Their results will not be listed here. The interest is rather in the spatiotemporal dynamics, starting from a system with lysogenic and then switching to lytic infection of the prey. Only one local example for such a switch is drawn in Fig. 1. After the transition, there is no further replication of infected. For modelling purpose, we simply set $r_2 = 0$ when the switch occurs. A more technical assumption for the simulation is that the remaining natural mortality of the infected is added to the virulence, leading to a higher effective mortality of the infected, i.e., the parameter m_2 increases. Furthermore, the lytic cycle generates many more viruses, i.e., the transmission rate λ increases as well. And, finally, the intraspecific competition of the dying infected phytoplankton cells vanishes, whereas the interspecific competition of susceptibles and infected becomes non-symmetric, i.e., the dead and dying infected still influence the growth of the susceptibles and contribute to the carrying capacity but not vice versa.

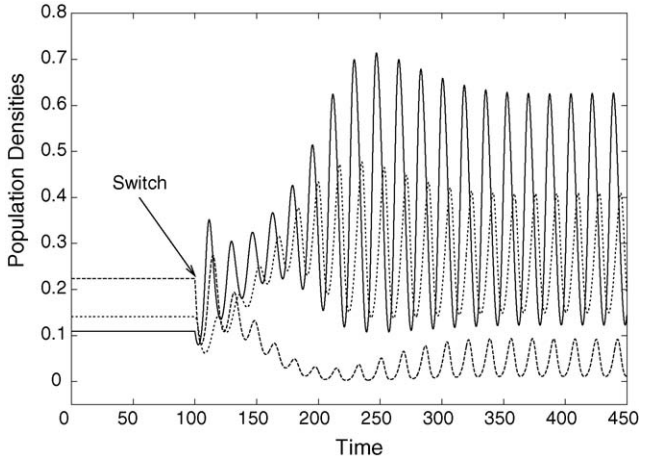

Fig. 1 – Non-oscillating endemic state with lysogenic infection switching at $t = 100$ to an oscillating endemic state with lytic infection. The growth rate of infected r_2 is set from $r_2^{\max} = 0.4$ to 0, the virulence m_2 from $m_2^{\min} = 0.2$ to $m_2^{\max} = 0.3$ and the transmission rate λ from $\lambda^{\min} = 0.6$ to $\lambda^{\max} = 0.9$, cf. text. Other parameter values: $r_1 = 1$, $a = b = 5$, $m_3 = 0.625$. Susceptibles are plotted with solid line, infected with dashed line and zooplankton with dots.

As to be expected, the switch from lysogeny to lysis results in a much lower mean abundance of infected though endemicity is still stable. However, the system responds rather sensitively to parameter changes, especially to variations of virulence and transmission rate and the infected can easily go extinct. As in the preceding paper, multiplicative noise supports the survival of the endangered species, i.e., there is always some probability to survive in a noisy environment while the deterministic setting inevitably leads to extinction.

4. The spatiotemporal dynamics

Now, we consider the spatiotemporal dynamics of the plankton model (2), i.e., zooplankton, grazing on susceptible and virally infected phytoplankton, under the influence of environmental noise and diffusing horizontally in two-dimensional space.

4.1. *Numerical methods, boundary and initial conditions*

The diffusion terms have been integrated using the semi-implicit Peaceman–Rachford alternating direction scheme (Peaceman and Rachford, 1955), cf. Thomas (1995). For the interactions and the Stratonovich integral of the noise terms, the explicit Euler–Maruyama scheme has been applied (Maruyama, 1955), cf. Kloeden and Platen (1999) and Higham (2001). The time step of the numerical scheme is $\Delta t = 0.01$. The spatial grid is a 99×99 point square with spacing $\Delta x = \Delta y = 1$.

Periodic boundary conditions have been chosen for all simulations (in order to avoid boundary effects).

The initial conditions are as follows: the space is filled with the non-oscillating endemic state $(X_1^S, X_2^S, X_3^S) = (0.109, 0.224, 0.141)$, cf. Fig. 1. Furthermore, there are two localized patches in space. They can be seen in Fig. 2. The

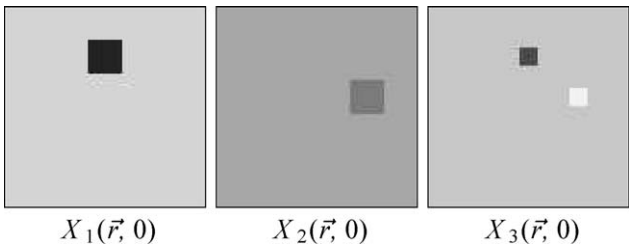

Fig. 2 – Initial conditions for all spatiotemporal simulations.

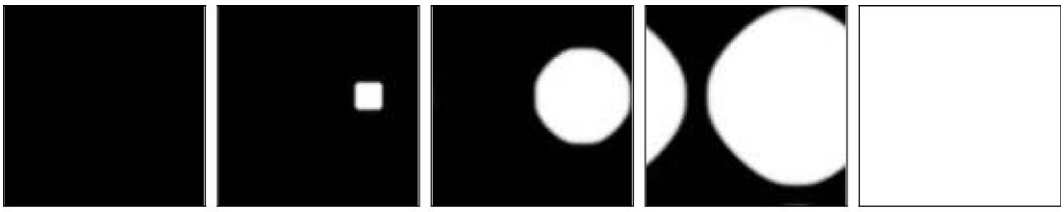

Fig. 3 – Spatial propagation of zero replication rate of the infected.

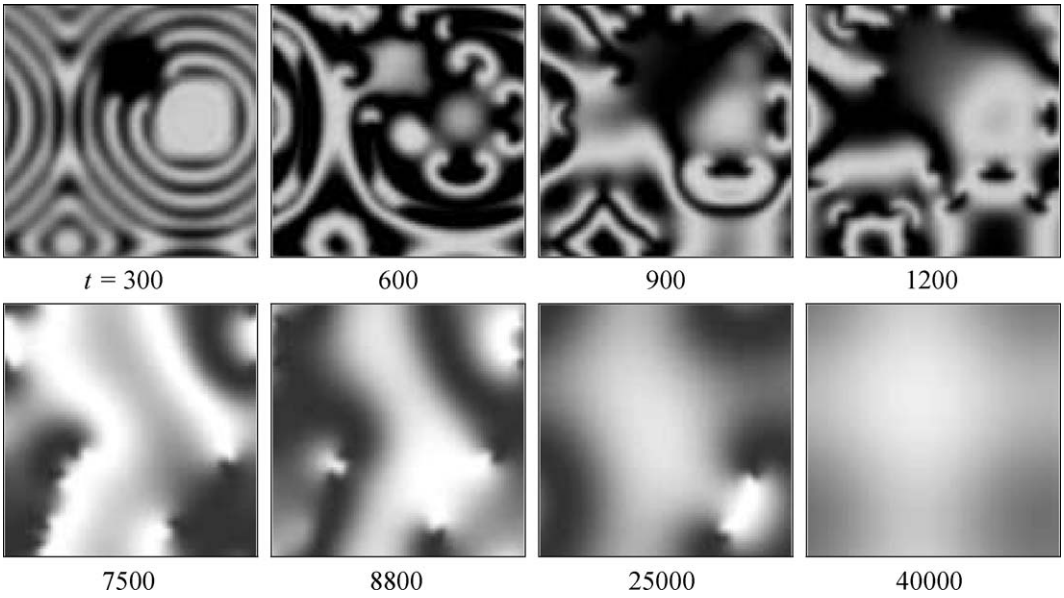

Fig. 4 – Dynamics of susceptibles. No noise.

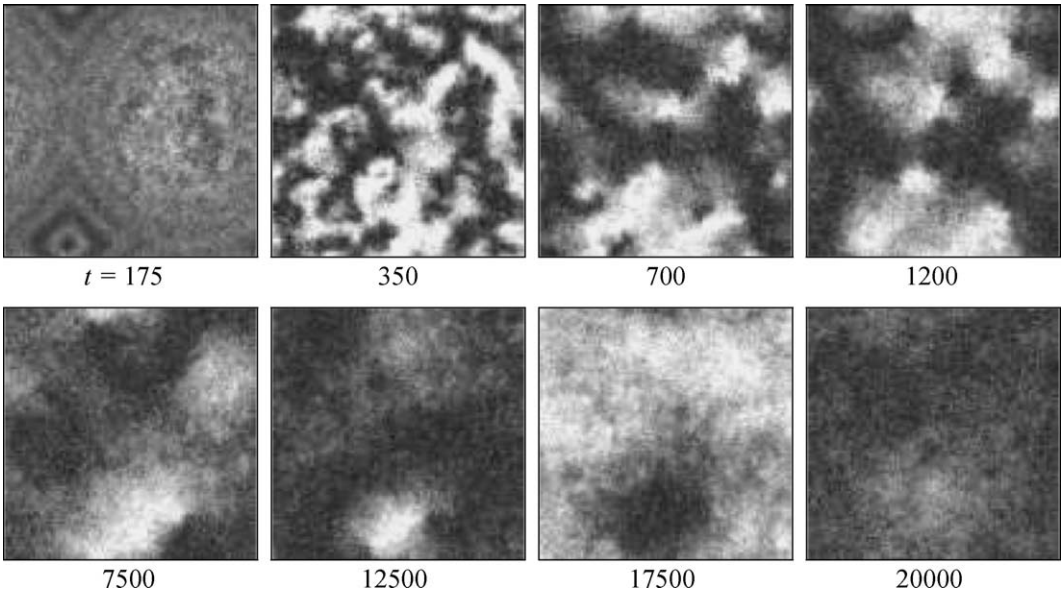

Fig. 5 – Dynamics of susceptibles. Noise intensities $\omega_1 = \omega_2 = \omega_3 = 0.05$.

grey scale changes from high population densities in black colour to vanishing densities in white.

One patch is located in the upper middle of the model area with susceptibles $X_1 = 0.550$, four grid points further away from zooplankton $X_3 = 0.450$ in each direction. The infected are at X_2^S. In the other patch at the right, the infected $X_2 = 0.333$ are further away from zooplankton $X_3 = 0.036$, whereas the susceptibles are at X_1^S. These initial conditions are the same for deterministic and stochastic simulations.

The chosen system parameters generate oscillations in the center of the patches. The latter act as leading centers for target pattern waves that collide and break up to form spirals. Increasing noise blurs this (naturally unrealistic) patterning, cf. also Malchow et al. (2004a,b).

4.2. *Deterministic switching from lysogeny to lysis*

At first, switching begins in the area with the highest initial density of infected, i.e., in the right-hand patch. The growth rate of infected r_2 vanishes, whereas virulence and natural mortality of infected add up to a higher effective virulence m_2 and also the transmission rate λ increases as described in Section 3. It is assumed that these parameter changes propagate through space like a Fisher wave (1937). If an auxiliary quantity r_3 with Fisher dynamics is introduced,

$$\frac{\partial r_3(\vec{r},t)}{\partial t} = r_3(1-r_3) + d\,\Delta r_3, \tag{3}$$

then

$$r_2(\vec{r},t) = r_2^{\max}(1-r_3), \tag{3a}$$

$$m_2(\vec{r},t) = m_2^{\min} + (m_2^{\max} - m_2^{\min})r_3, \tag{3b}$$

$$\lambda(\vec{r},t) = \lambda^{\min} + (\lambda^{\max} - \lambda^{\min})r_3. \tag{3c}$$

The initial conditions are $r_3 = 1$ in the right patch and 0 elsewhere. For simplicity, the diffusivity d is assumed to be the same as for all the populations. Its value of $d = 0.05$ has been chosen from Okubo's diffusion diagrams (1971) in order to model processes on a kilometer scale. The resulting spatial propagation of $r_2 = 0$ is drawn in white colour in Fig. 3.

The corresponding dynamics of susceptibles is plotted in Fig. 4. The presentation of susceptibles has been chosen because of richer contrast. The patterns of infected are similar.

The break-up of concentric waves to a rather complex structure with spirals is nicely seen. In the long run, pinning-like behaviour of pairs of spirals is found. This effect is well-known from excitation waves in cardiac muscles, cf. the classical publications by Davidenko et al. (1992) and Pertsov et al. (1993). Here, the biological meaning remains unclear. The almost fixed pair-forming and rapidly rotating spirals approach each other extremely slowly, collide and burst. A weak multiplicative noise accelerates this process, a fact shown in Fig. 5. Stronger noise, i.e., higher environmental variability, suppresses the generation of pins. Finally, the homogeneously oscillating endemic state remains.

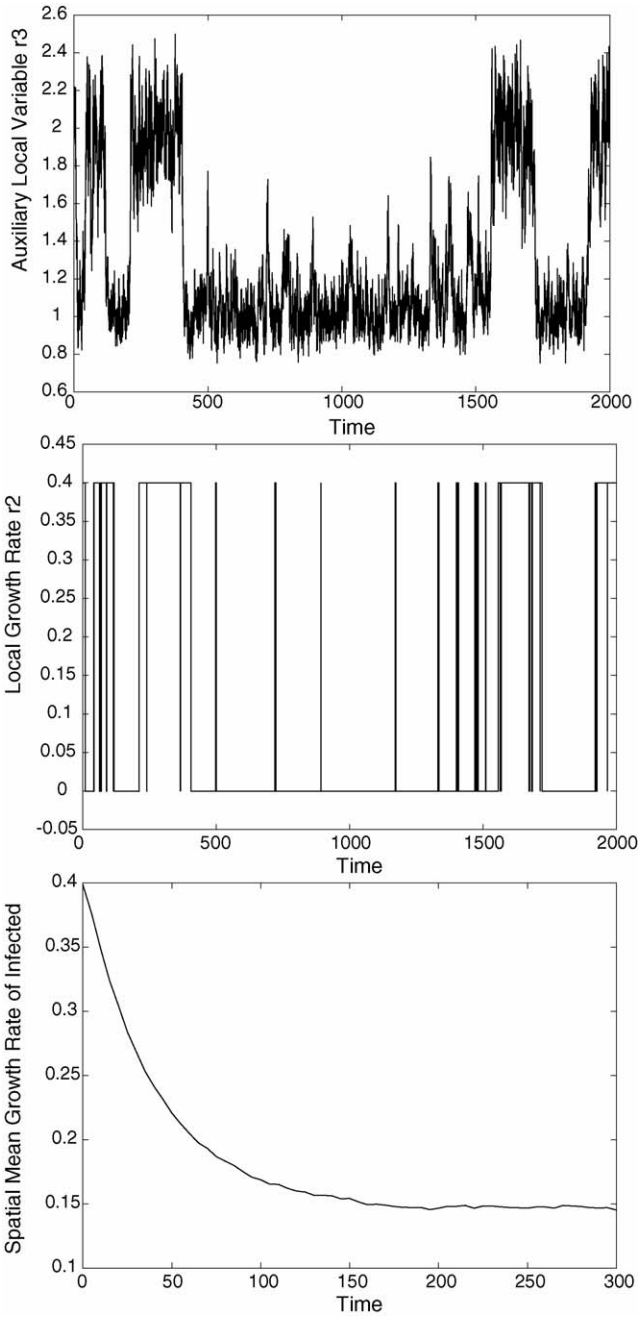

Fig. 6 – Noisy bistable dynamics of r_3 and resulting local switches of r_2 for $r_3^{\max} = 2$, $r_3^{\text{crit}} = 1.5$, $r_3^{\min} = 1$ and $\omega_4 = 0.1$. The spatial mean of r_2 decreases from the maximum as homogeneous initial condition to a value of approximately 0.15. The growth rate of infected r_2 switches from $r_2^{\max} = 0.4$ to 0, the virulence m_2 from 0.2 to 0.3 and the transmission rate λ from 0.6 to 0.9, cf. text. Other parameter values: $r_1 = 1$, $a = b = 5$, $m_3 = 0.625$.

The system parameters have been chosen to guarantee the survival of all three populations under deterministic conditions. The sample run for Fig. 5 with 5% noise also yields this final endemic coexistence. However, after the switch to cell lysis, it should be noted that there is only a certain survival probability for all three populations, the lowest of course being the one for the infected.

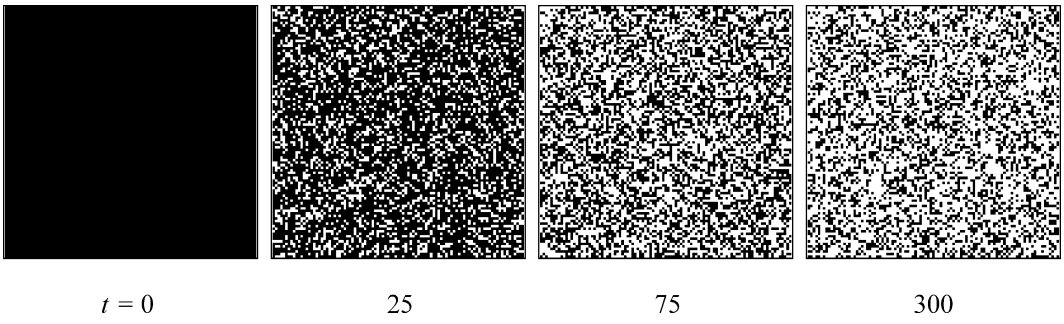

Fig. 7 – Spatiotemporal pattern of r_2.

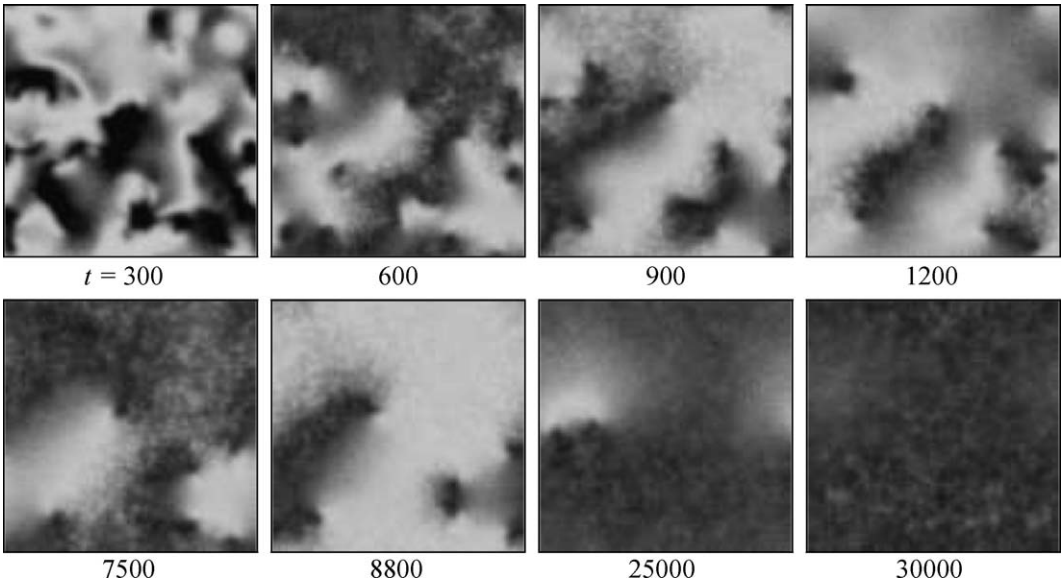

Fig. 8 – Deterministic dynamics of susceptibles with noisy switch. $\omega_1 = \omega_2 = \omega_3 = 0$, $\omega_4 = 0.1$. The almost stationary spiral pairs do still exist.

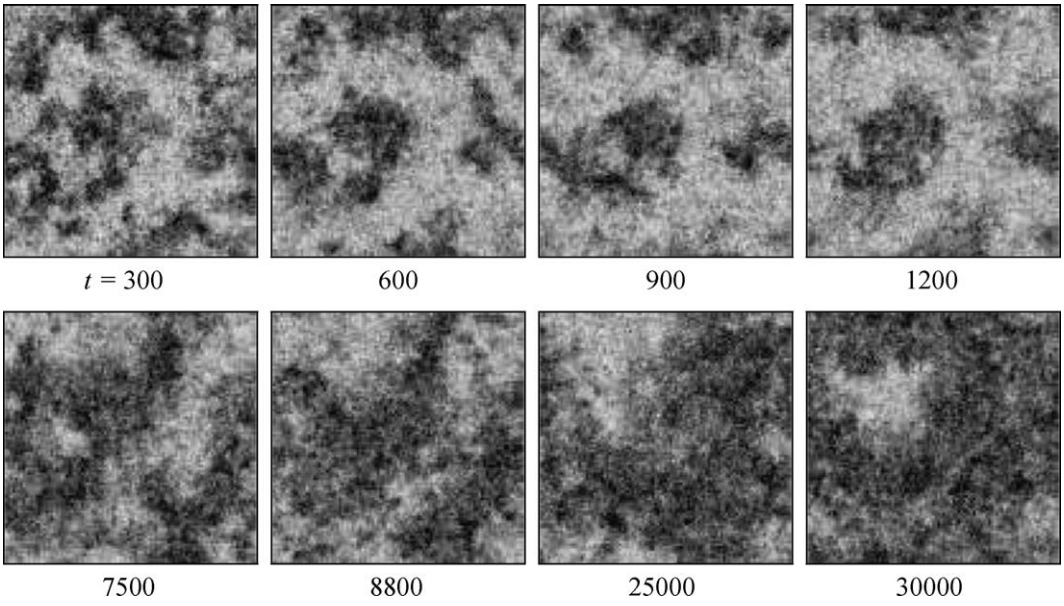

Fig. 9 – Noisy dynamics of susceptibles and noisy switch. $\omega_i = 0.1$, i = 1–4. The "pins" cannot be found anymore.

4.3. *Stochastic switching*

The deterministic once-for-ever switching mechanism is highly unrealistic. Moreover, there is some evidence that lytic infections can become lysogenic (Herskowitz and Hagen, 1980; Moebus, 1996; Wilson et al., 1996; Oppenheim et al., 2005). One has to consider that only a certain fraction of viruses begins with the lysogenic replication cycle locally and then switches. To model this, we redefine the auxiliary quantity r_3 to obey bistable kinetics and multiplicative noise, i.e.,

$$\frac{\partial r_3(\vec{r},t)}{\partial t} = (r_3 - r_3^{\min})(r_3 - r_3^{\text{crit}})(r_3^{\max} - r_3) + \omega_4 r_3 \cdot \xi(\vec{r},t). \tag{4}$$

The noise forces system (4) to switch between its stable stationary states $r_3^{\min}$ and $r_3^{\max}$ (Nitzan et al., 1974; Ebeling and Schimansky-Geier, 1980; Malchow and Schimansky-Geier, 1985). It is assumed that the replication rate of the infected switches accordingly, i.e.,

$$\text{if } r_3 > r_3^{\text{crit}} \quad \text{then } m_2 = m_2^{\min},\ \lambda = \lambda^{\min},\ r_2 = r_2^{\max}\ (\text{lysogeny}), \tag{4a}$$

$$\text{if } r_3 \le r_3^{\text{crit}} \quad \text{then } m_2 = m_2^{\max},\ \lambda = \lambda^{\max},\ r_2 = 0\ (\text{lysis}). \tag{4b}$$

This phenomenon as well as the temporal development of the spatial mean and the spatiotemporal pattern of r_2 is drawn in Figs. 6 and 7, respectively. Initially, the whole system is in the lysogenic state (4a).

At first, the simulation is run with noisy switches of r_2, λ and m_2 but deterministic population dynamics. In this unrealistic setting, the pins can still be seen, cf. Fig. 8.

If also the population dynamics is subject to noise, the result becomes more realistic. The pins are suppressed and the plankton forms a rather complex noise-induced patchy structure, cf. Fig. 9.

5. Conclusions

A conceptual biomass-based stochastic reaction-diffusion model of phytoplankton–zooplankton prey–predator dynamics has been investigated for temporal, spatial and spatiotemporal dissipative pattern formation in a deterministic and noisy environment, respectively. It has been assumed that the phytoplankton is partly virally infected and the virus switches from a lysogenic to a lytic replication cycle.

The logistic growth rate of lysogenically infected has been about 40% of the growth rate of susceptible phytoplankton. The local dynamics has been used to tune the system parameters in a way that the infected survive after switching from lysogeny to lysis.

In the spatial model, the switch has first been assumed to be deterministic, beginning at a certain position and then propagating like a Fisher wave. The populations have shown a complex wavy structure with formation of almost pinned pairs of spirals. The biological meaning of this pattern is unclear. The pair formation has disappeared for increasing noise.

Deterministic switching is an unrealistic once-for-ever mechanism. Therefore, a stochastic method has been developed and applied. Noisy switches and population dynamics generate a complex patchy spatiotemporal structure that is typical for natural plankton populations. The presented sample runs have led to a final endemic state with coexistence of susceptibles, infected and zooplankton like in the deterministic case. However, one should be aware that the survival probability of the system with all non-zero populations is smaller than 1. Thus, there are good chances that the three-component system switches to one of its subsystems. Noise has not only an impact on the spatiotemporal coexistence of populations but its presence is necessary to blur distinct artificial population structures like target patterns or spirals and to generate more realistic fuzzy patterns.

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