Spatiotemporal Patterns in an Excitable Plankton System with Lysogenic Viral Infection

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Abstract—An excitable 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, following a Holling Type-III functional response. 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 spatiotemporal behaviour, modelled by 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 for space. The external noise can enhance the survival and spread of susceptibles and infected, respectively, that would go extinct in a deterministic environment. In the parameter range of excitability, noise can induce local blooms of susceptibles and infected. © 2005 Elsevier Ltd. All rights reserved.

1. INTRODUCTION

Viruses are evidently the most abundant entities in the sea and the question may arise whether they control ocean life. However, there is much less known about marine viruses and their role in aquatic ecosystems and the species that they infect, than about plankton patchiness and blooming, for reviews, cf., [1]. A number of studies [2–6] shows the presence of pathogenic viruses in phytoplankton communities. Fuhrman [1] has reviewed the nature of marine viruses and their ecological as well as biogeological effects. Suttle *et al.* [5] have shown by using electron microscopy that the viral disease can infect bacteria and phytoplankton in coastal water. Parasites

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may modify the behaviour of the infected members of the prey population. Virus-like particles are described for many eukaryotic algae [7,8], cyanobacteria [9] and natural phytoplankton communities [10]. There is some evidence that viral infection might accelerate the termination of phytoplankton blooms [11,12]. Viruses are held responsible for the collapse of *Emiliania huxleyi* blooms in mesocosms [13] and in the North Sea [14] and are shown to induce lysis of *Chrysochromulina* [15]. Because most viruses are strain-specific, they can increase genetic diversity [16]. Nevertheless, despite the increasing number of reports, the role of viral infection in the phytoplankton population is still far from understood.

Viral infections of phytoplankton cells can be lysogenic or lytic. The understanding of the importance of lysogeny is just at the beginning [17–20]. 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 [21]. More recent work is of Chattopadhyay *et al.* [22,23]. The latter deal with lytic infections and mass action incidence functions. Malchow *et al.* [24] observed oscillations and waves in a phytoplankton-zooplankton system with Holling Type-II grazing under lysogenic viral infection and frequency-dependent transmission.

Numerous papers have been published about pattern formation and chaos in minimal prey-predator models of phytoplankton-zooplankton dynamics [25–32]. Different routes to local and spatiotemporal chaos [33–41], diffusion- and differential-flow-induced standing and travelling waves [26,42–46] as well as target patterns and spiral waves [47,48] have been found. Also, the impact of external noise on patchiness and transitions between alternative stable population states has been studied [41,49–51].

In this paper, we focus on modelling the influence of lysogenic infections and proportionate mixing incidence function (frequency-dependent transmission) [52–54] on the local and spatio-temporal dynamics of interacting phytoplankton and zooplankton with Holling Type-III grazing, i.e., with excitable dynamics. The latter has been introduced by Truscott and Brindley [28] to model recurrent phytoplankton blooms. Furthermore, the impact of multiplicative noise [55,56] is investigated.

2. THE MATHEMATICAL MODEL

The Truscott-Brindley model [28] for the prey-predator dynamics of phytoplankton P and zooplankton Z at time t and location $\vec{r} = \{x, y\}$ reads in dimensionless quantities,

$$\frac{\partial P}{\partial t} = rP(1 - P) - \frac{a^2 P^2}{1 + b^2 P^2} Z + d\Delta P, \tag{1}$$

$$\frac{\partial Z}{\partial t} = \frac{a^2 P^2}{1 + b^2 P^2} Z - m_3 Z + d\Delta Z. \tag{2}$$

There is logistic growth of the phytoplankton with intrinsic rate r and Holling-Type III grazing with maximum rate a^2/b^2 as well as natural mortality of zooplankton with rate m_3. 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 effects of nutrient supply and planktivorous fish are 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,

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

where

$$f_1 = r_1 X_1\,(1 - X_1 - X_2) - \frac{a^2 X_1\,(X_1 + X_2)}{1 + b^2\,(X_1 + X_2)^2}X_3 - \lambda\frac{X_1 X_2}{X_1 + X_2}, \qquad (3a)$$

$$f_2 = r_2 X_2\,(1 - X_1 - X_2) - \frac{a^2 X_2(X_1 + X_2)}{1 + b^2(X_1 + X_2)^2}X_3 + \lambda\frac{X_1 X_2}{X_1 + X_2} - m_2 X_2, \qquad (3b)$$

$$f_3 = \frac{a^2(X_1 + X_2)^2}{1 + b^2(X_1 + X_2)^2}X_3 - m_3\,X_3. \qquad (3c)$$

Proportionate mixing transmission with rate λ as well as an additional disease-induced mortality of infected (virulence) with rate m_2 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 equation (3b) would describe the losses due to natural mortality and competition. Here, lysogenic infections with $r_1 = r_2 = r$ will be considered. The lysogenic replication cycle of viruses is very sensitive to environmental variability and may switch to the lytic cycle. This situation is not considered here.

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

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

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

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

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

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

3. THE LOCAL DYNAMICS

At first, the local dynamics is studied, i.e., it is searched for stationary and oscillatory solutions of system (3) for $d = 0$. To do that, system (3) is simplified through a convenient transformation, then describing the dynamics of the total phytoplankton population $P = X_1 + X_2$, the prevalence $i = X_2/P$ and zooplankton X_3. With $f(P) = a^2 P^2/(1 + b^2 P^2)$, the model equations read,

$$\frac{dP}{dt} = [\,r_1(1 - i) + r_2 i\,](1 - P)P - f(P)X_3 - m_2 iP,$$

$$\frac{di}{dt} = [(r_2 - r_1)(1 - P) + (\lambda - m_2)](1 - i)i, \qquad (5)$$

$$\frac{dX_3}{dt} = [f(P) - m_3\,]X_3.$$
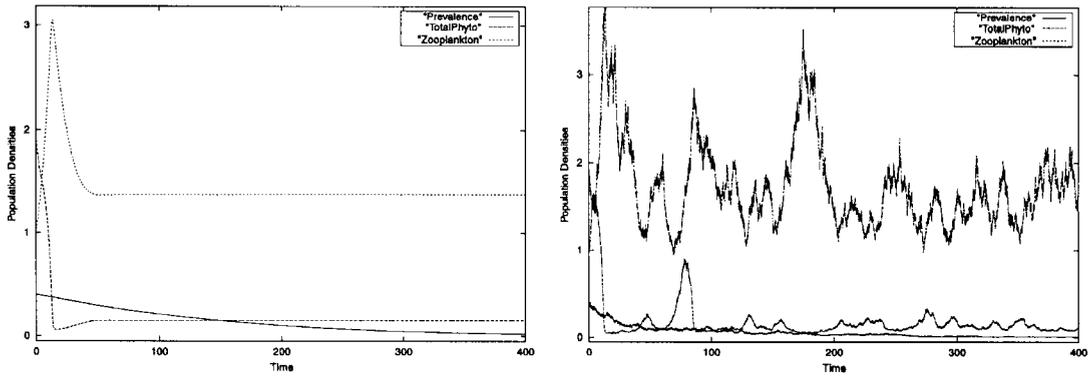
(a). $\lambda = 0.001 < m_2 = 0.01$.
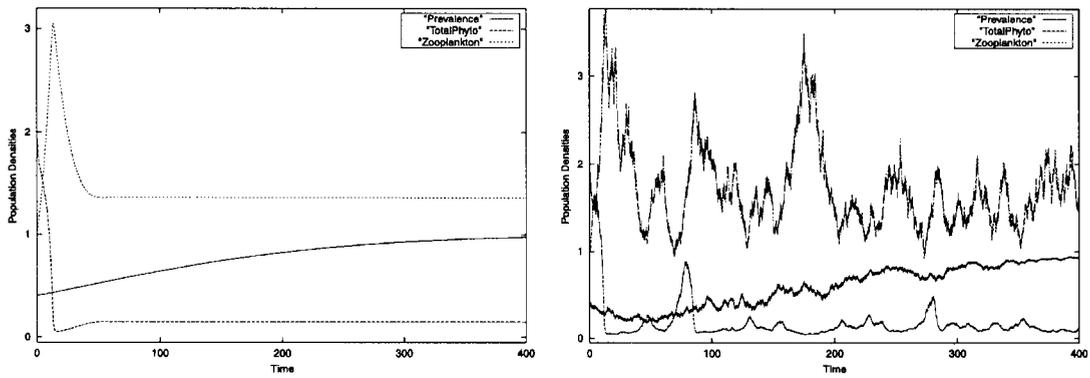
(b). $\lambda = 0.02 > m_2 = 0.01$.
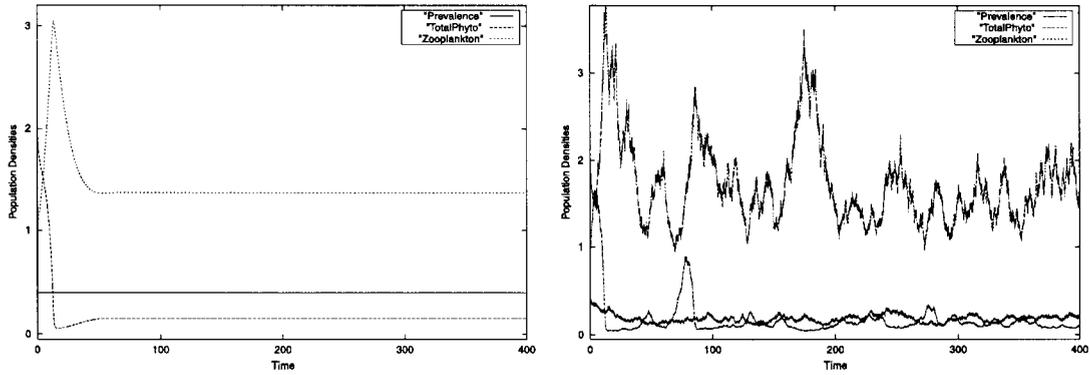
(c). $\lambda = m_2 = 0.01$.

Figure 1. Local excitability with (a) decline of prevalence for $\lambda < m_2$, (b) saturation of prevalence for $\lambda > m_2$, and (c) constant prevalence for $\lambda = m_2$, $m_3 = 0.05$. No noise in left column. Localized outbreaks in a noisy environment with $\omega = 0.075$, right column.

For different ratios of λ and m_2 exist different stationary solutions. In order to characterize them, the following additional parameters are introduced,

$$i^S = \frac{X_2^S}{X_1^S + X_2^S}$$

and

$$m_3^{cr} = f(P^S) = \frac{a^2 (X_1^S + X_2^S)^2}{1 + b^2 (X_1^S + X_2^S)^2}.$$
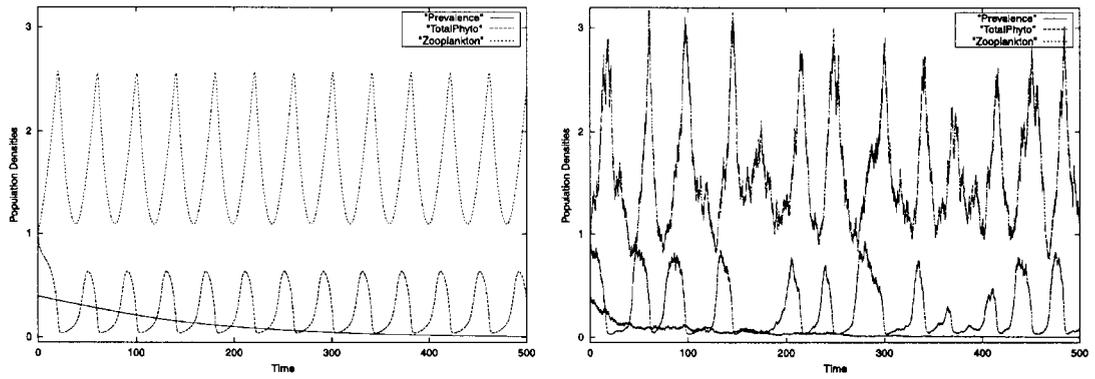
(a). $\lambda = 0.001 < m_2 = 0.01$.
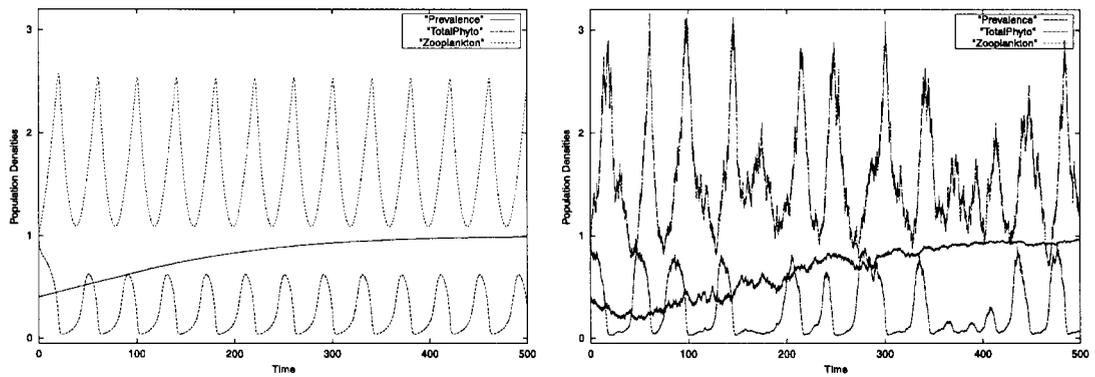
(b). $\lambda = 0.02 > m_2 = 0.01$.
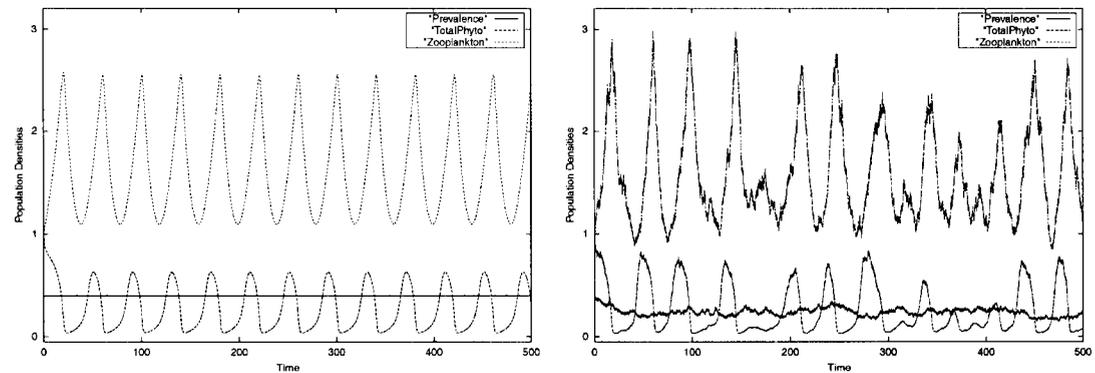
(c). $\lambda = m_2 = 0.01$.

Figure 2. Local oscillations with (a) decline of prevalence for $\lambda < m_2$, (b) saturation of prevalence for $\lambda > m_2$, and (c) constant prevalence for $\lambda = m_2$, $m_3 = 0.09$. No noise in left column. Noisy oscillations with $\omega = 0.075$, right column.

X_k^S, $k = 1, 2, 3$, are the stationary solutions of system (3). Both analytical and numerical investigations yield the following selected equilibria for $r_1 = r_2 = r$.

(0) Trivial solution $X_1^S = X_2^S = X_3^S = 0$, i.e., $P^S = i^S = X_3^S = 0$, always unstable.

(1) Extinction of infected with and without predation:

   (a) $P^S = X_1^S > 0$, $X_2^S = 0$, i.e., $i^S = 0$, $X_3^S = 0$, if $\lambda < m_2$ and $m_3 > m_3^{cr}$, nonoscillatory stable;

   (b) $P^S = X_1^S > 0$, $X_2^S = 0$, i.e., $i^S = 0$, $X_3^S > 0$ if $\lambda < m_2$, nonoscillatory or oscillatory stable depending on $m_3 >$ or $< m_3^{cr}$, respectively.

(2) Extinction of susceptibles with and without predation:

 (a) $X_1^S = 0$, $P^S = X_2^S > 0$, i.e., $i^S = 1$, $X_3^S = 0$ if $\lambda > m_2$ and $m_3 > m_3^{cr}$, nonoscillatory stable;

 (b) $X_1^S = 0$, $P^S = X_2^S > 0$, i.e., $i^S = 1$, $X_3^S > 0$ if $\lambda > m_2$, nonoscillatory or oscillatory stable depending on $m_3 >$ or $< m_3^{cr}$, respectively.

(3) Endemic states with and without predation:

 (a) $X_1^S > 0$, $X_2^S > 0$, i.e., $P^S > 0$, $0 < i^S = i(0) = $ const. < 1, $X_3^S = 0$ if $\lambda = m_2$ and $m_3 > m_3^{cr}$, nonoscillatory stable;

 (b) $X_1^S > 0$, $X_2^S > 0$, i.e., $P^S > 0$, $0 < i^S = i(0) = $ const. < 1, $X_3^S > 0$ if $\lambda = m_2$, nonoscillatory or oscillatory stable depending on $m_3 >$ or $< m_3^{cr}$, respectively.

For $\lambda < m_2$, the infected go extinct (Solutions 1a and 1b) and the prevalence i reaches zero. For $\lambda > m_2$, the susceptibles die out (Solutions 2a and 2b) and the prevalence approaches unity. In the case of $\lambda = m_2$, susceptibles and infected coexist (endemic states 3a and 3b) and the prevalence remains constant at its initial value. Moreover, if m_3 is greater or less than m_3^{cr}, the system becomes nonoscillatory or oscillatory stable, respectively. For low values of m_3, we observe excitation and the following relaxation to the nonoscillatory stable situation. A corresponding example is presented in the left column of Figure 1 for $r = r_1 = r_2 = 1$ and $a = 3.75$, $b = 10$. These three parameter values will be kept for all simulations. In the excitable parameter range with weak external noise, we also observe recurrent outbreaks related to planktonic blooming. This is shown in the right column. These stochastic sample runs have qualitatively the same outcome as the deterministic computations. However, one should have in mind that the latter must only hold for the average of a sufficient number of runs. In a noisy environment, there are only certain probabilities for the survival or extinction of the populations.

A slight increase of m_3 yields loss of excitability but oscillations in the system. These are drawn in Figures 2. The dynamics of the prevalence remains unchanged what is a very convenient property of system (5).

One can see that the transformation of the local part of model (3) to system (5) with $r_1 = r_2 = r$ reduces the considerations of deterministic stationarity and stability to a pseudo-two-dimensional problem because the prevalence can take only three values, i.e., zero for $\lambda < m_2$, unity for $\lambda > m_2$ or its initial value for $\lambda = m_2$. This simplifies the computations remarkably. However, for the investigation of the spatiotemporal system with external noise, we proceed with model (3).

4. THE SPATIAL DYNAMICS

Much has been published about the spatiotemporal selforganization in prey-predator communities, modelled by reaction-diffusion (-advection) equations, cf., 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 [57–59].

In this section, we consider the spatiotemporal 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, cf., [60]. For the interactions and the Stratonovich integral of the noise terms, the explicit Euler-Maruyama scheme has been applied [61,62].

The following series of figures summarizes the results of the spatiotemporal 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 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.
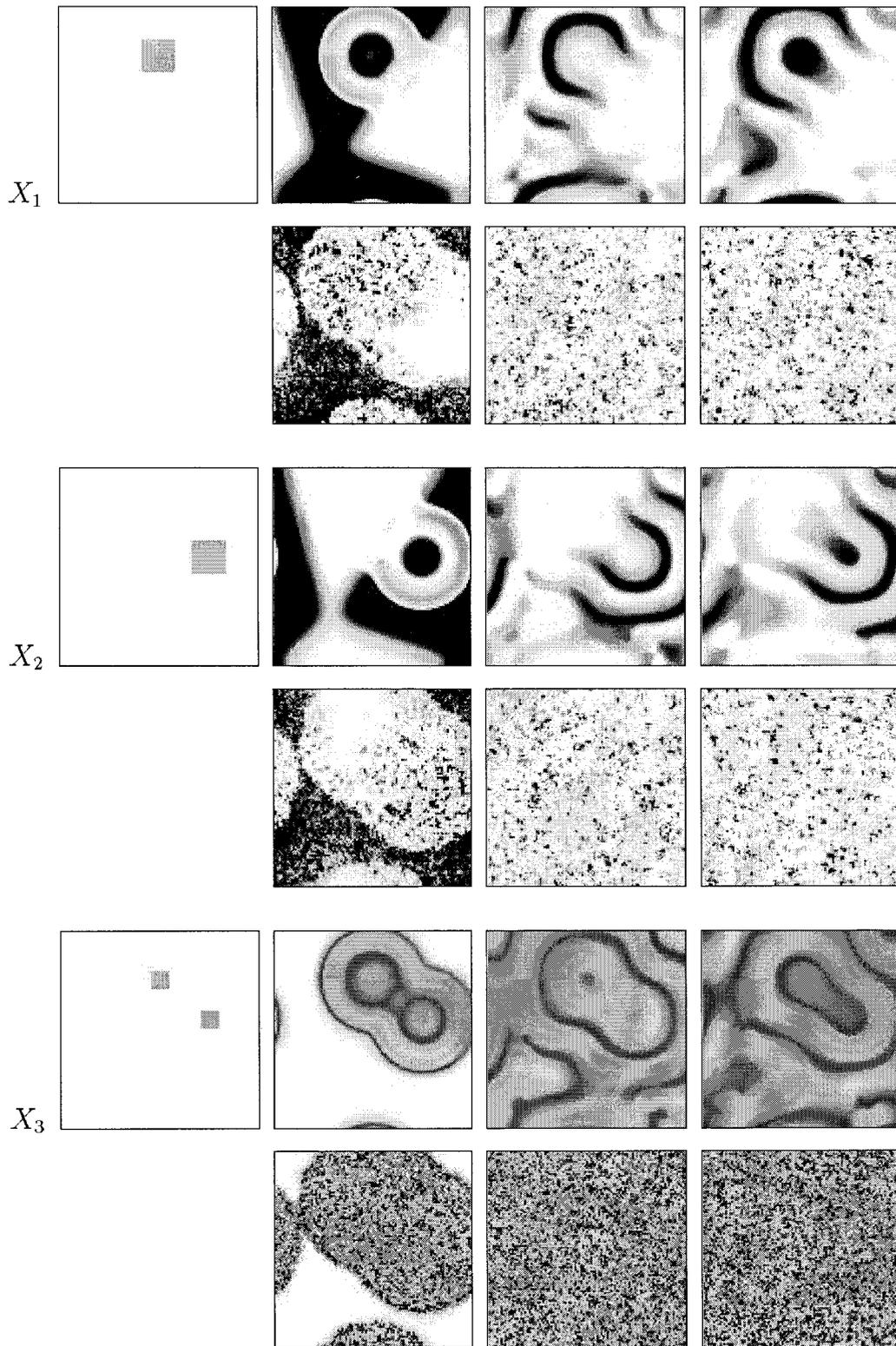
(a). $t = 0$.   (b). 300.   (c). 750.   (d). 1050.

Figure 3. Spatial coexistence of susceptibles (two upper rows), infected (two middle rows) and zooplankton (two lower rows) for $m_2 = \lambda = 0.01$, $m_3 = 0.09$. No noise $\omega = 0$ and 0.25 noise intensity, respectively, with equal initial conditions (left column). Periodic boundary conditions.

(a). $t = 0$.   (b). 300.   (c). 750.   (d). 1050.
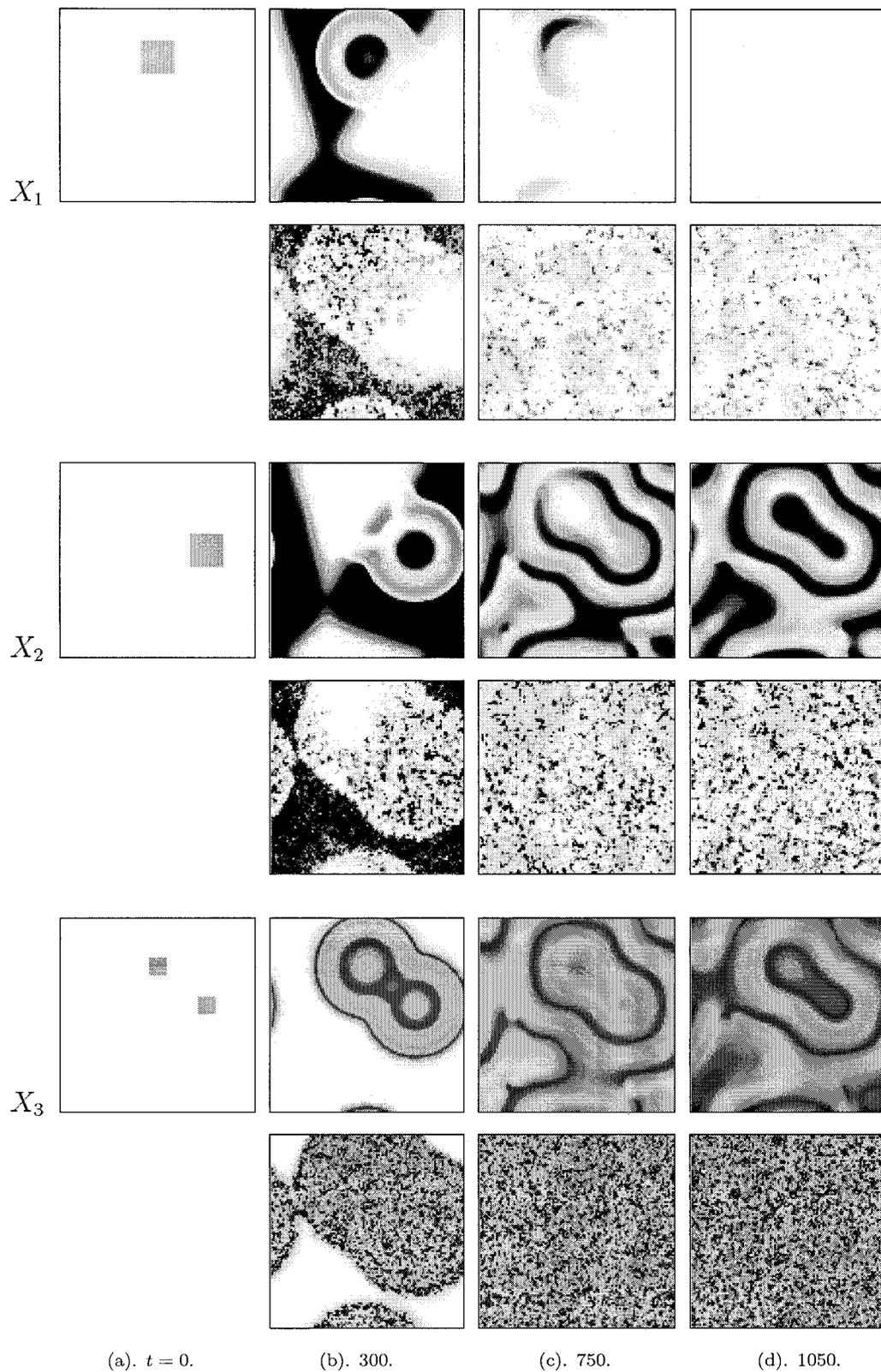
Figure 4. Spatial coexistence of infected (two middle rows) and zooplankton (two lower rows). Extinction of susceptibles (first row) for $m_2 = 0.01 < \lambda = 0.03$, $m_3 = 0.09$, and no noise. Very low survival of susceptibles for $\omega = 0.25$ noise intensity (second row).

(a). $t = 0$.   (b). 150.   (c). 300.   (d). 450.
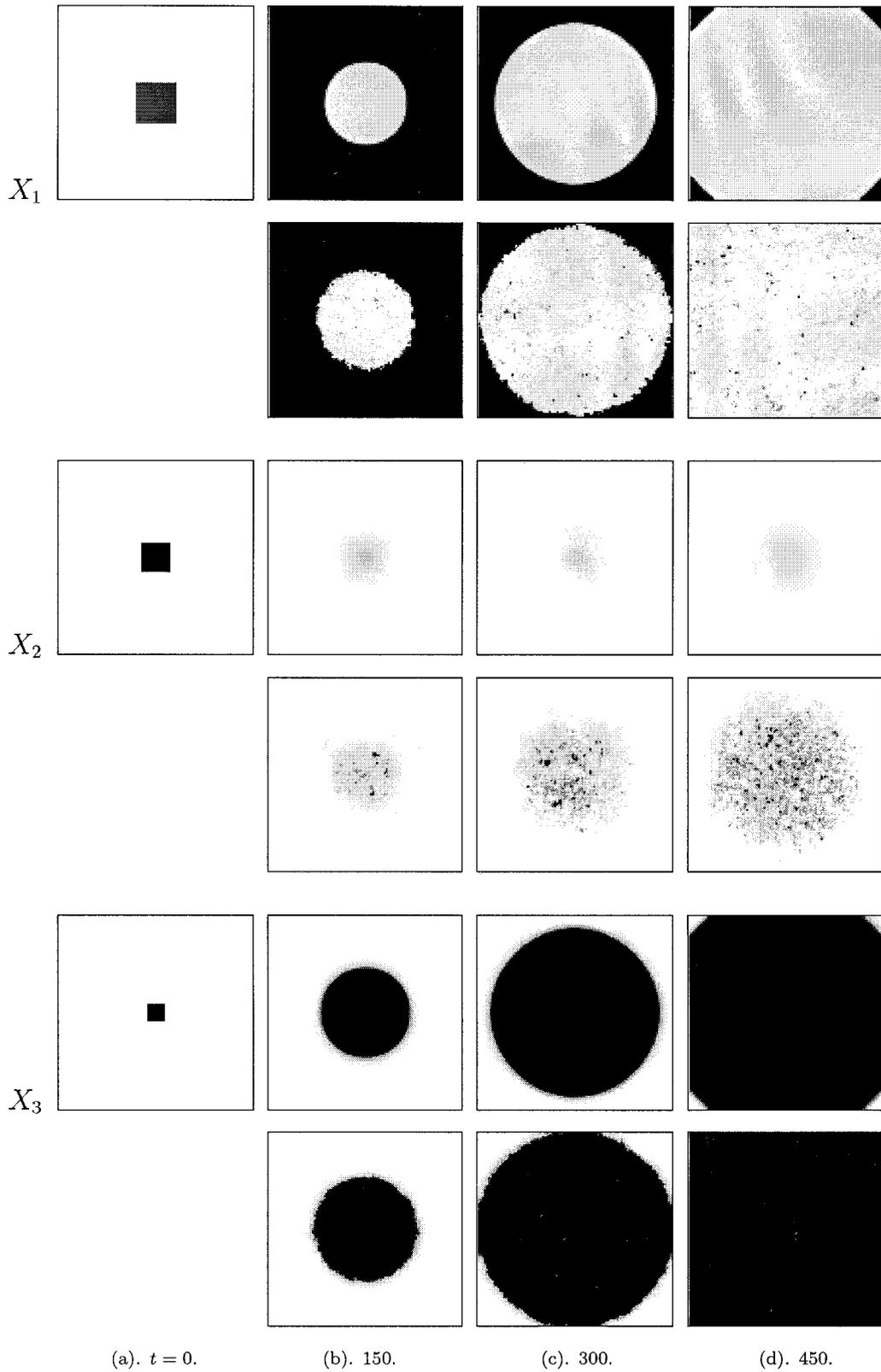
Figure 5. Parameter range of deterministic excitable spatial coexistence of suscep-
tibles, infected and zooplankton for $m_2 = \lambda = 0.01$, $m_3 = 0.05$. Without noise
trapping and almost extinction of infected in the center (third row). With $\omega = 0.25$
noise intensity noise-enhanced survival and spread of infected (fourth row).

In Figures 3 and 4, there are initially two patches, one with zooplankton surrounded by susceptible phytoplankton (upper part of the model area) and one with zooplankton surrounded by infected (on the right of the model area). For Figures 5, there are central and concentric patches of all three species.

In Figures 3, one can see the final spatial coexistence of all three species for $\lambda = m_2$. 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 black colour to vanishing densities in white.

(a). X_1.

(b). X_2.

(c). X_3.
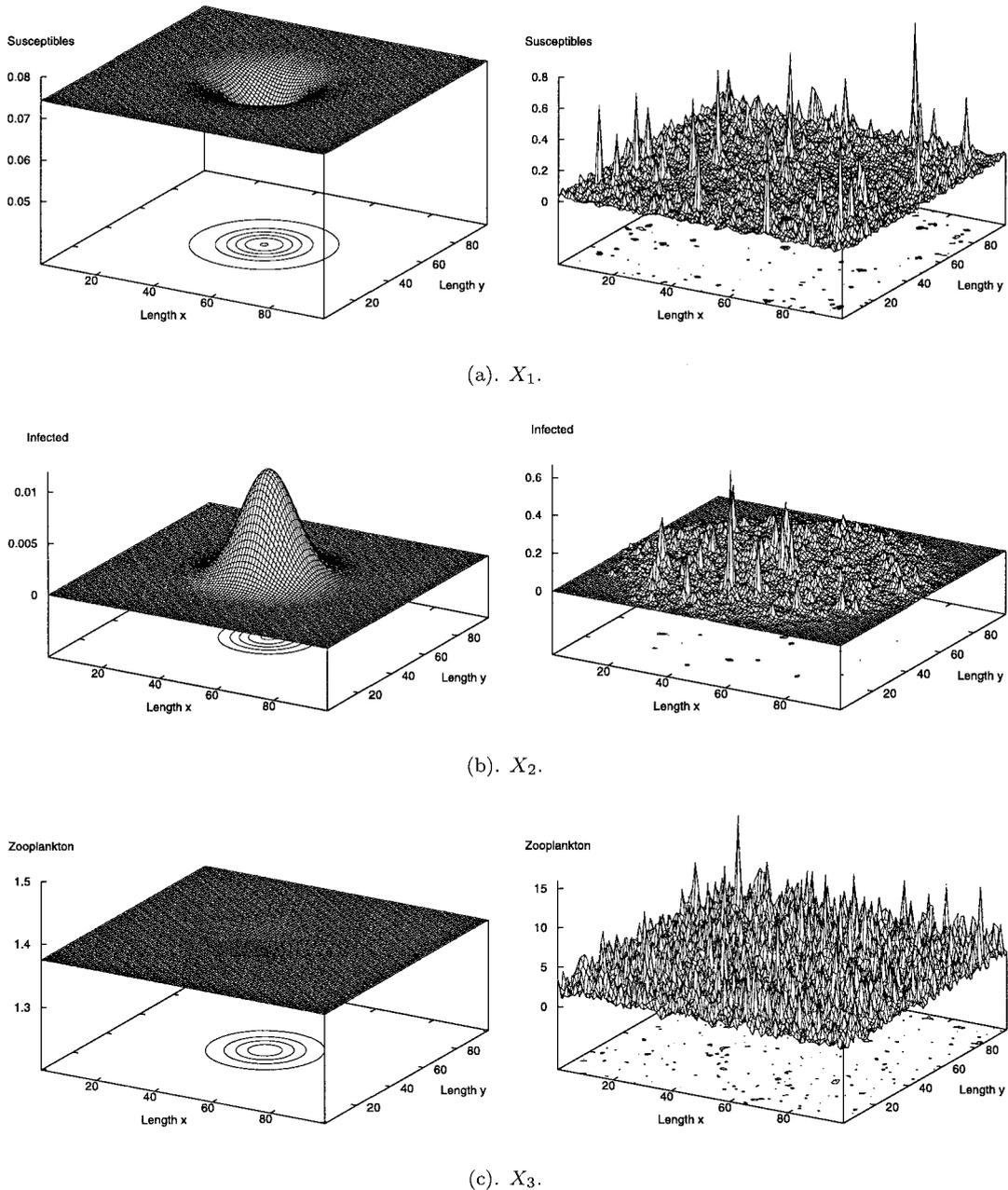
Figure 6. Density plot at $t = 550$ for the simulation shown in Figure 5. No noise in left column, noise intensity $\omega = 0.25$ in right column. $m_2 = \lambda = 0.01$, $m_3 = 0.05$. The trapping of infected without noise and their noise-enhanced spread are readily seen. Noise-induced local outbreaks due to the excitability of the system can also be observed.

This changes for $\lambda \neq m_2$. Whereas in the deterministic case infected or susceptibles go extinct, respectively, the noise enhances their survival and spread under unfavourable conditions. An example is given in Figures 4 for $\lambda > m_2$, i.e., the deterministic extinction and noise-induced survival and spread of susceptibles. An example for the opposite case is omitted here.

In Figures 5, the deterministic conditions allow for the excitable, nonoscillatory coexistence of susceptibles and infected. Susceptibles are initially ahead of infected that are ahead of zooplankton. This special initial configuration leads to the propagation of diffusive fronts in rows 1, 3, and 5. However, the infected are somehow trapped in the centre and go almost extinct. For the stochastic case in rows 2, 4, and 6, the noise enhances the "escape", spread and survival of the infected. The opposite case is also possible if the infected are initially ahead of susceptibles.

In Figures 6, spatial snapshots of all the three populations at $t = 550$ are provided, without noise in the left column and with noise in the right one. The noise-induced enhancement of the spread of infected is just as readily seen as the localized noise-induced outbreaks.

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. Holling Type-III zooplankton grazing has been considered in order to study the interplay of excitability, infection and noise.

The equal growth rates of susceptibles and infected have led to the situation that, in a non-fluctuating environment, the ratio of virulence and transmission rate of the infection controls coexistence, survival or extinction of susceptibles and infected, respectively. A fluctuating environment enhances the survival and the spatial spread of the "endangered" species. Furthermore, the noise has induced localized outbreaks or bloom phenomena in the parameter range of excitability. However, noise has not only supported the spatiotemporal coexistence and spread of susceptibles and infected but it has blurred distinct artificial population structures like target patterns or spirals and generated more realistic fuzzy patterns.

Forthcoming work has to include modelling of the transition from lysogenic to lytic viral replications, induced by noise with an intensity above a critical threshold, and its impact on recurrent phytoplankton outbreaks. Furthermore, different incidence functions and noise characteristics and the resulting local and spatiotemporal dynamics of the plankton populations have to be considered.

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