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THE PARADOX OF ENRICHMENT, SPATIAL HETEROGENEITY, COMMUNITY EFFECTS AND THE PHENOMENON OF APPARENT DISAPPEARANCE IN THE MARINE BACTERIOPHAGE DYNAMICS

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Abstract

In aquatic microbial systems, high-magnitude variations in abundance, such as sudden blooms alternating with comparatively long periods of very low abundance ("apparent disappearance"), are relatively common. We suggest that in order for this to occur, such variations in abundance in microbial systems and, in particular, the apparent disappearance of species do not require seasonal or periodic forcing of any kind or external factors of any other nature. Instead, such variations can be caused by internal factors and, in particular, by bacteria–phage interaction. Specifically, we suggest that the variations in abundance and the apparent disappearance phenomenon can be a result of phage infection and the lysis of infected bacteria. To illustrate this idea, we consider a reasonably simple mathematical model of bacteria–phage interaction based on the model suggested by Beretta and Kuang, which assumes neither periodic forcing nor action of other external factors. The model admits a loss of stability via Andronov–Hopf bifurcation and exhibits dynamics which explains the phenomenon. These properties of the model are especially distinctive for spatially nonhomogeneous biosystems as well as biosystems with some sort of cooperation or community effects.

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Keywords and phrases: mathematical model, bacterioplankton, phage, host–parasite system, Hopf bifurcation, stability, limit cycle, self-sustained oscillations, kill the winner hypothesis, paradox of enrichment, spatial heterogeneity, community effects, cooperation.

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1. Introduction

In marine biosystems, high-magnitude variations in abundance and unstable dynamics, such as sudden planktonic and bacterial blooms alternating with extended periods of low abundances, are quite common. In particular, marine microbial communities frequently exhibit bacterial and bacteriophage blooms, which alternate with comparatively long periods of low abundance, when the concentrations of species are of several orders of magnitude lower than the peak bloom concentrations [11, 12, 39]. To describe such periods of very low abundance, when the species concentrations are extremely low and can drop below detection level, the microbiologist Prentice [31] suggested the term "apparent disappearance", which we adopt in this paper.

In many cases, the planktonic and microbial blooms are immediate consequences of seasonal variations in temperature, light and other conditions (the "spring blooms"). Increases in aquatic bacteria abundance can also be caused by external forcing of nonseasonal (and nonperiodic) nature, such as the ones that occur after dust pulses in the Western Mediterranean [27]. In these cases, an increase in bacterial abundance occurs due to a massive discharge of a certain nutrient benefiting a particular bacterial strain. In turn, this blooming bacterial strain would be infected by a specific bacteriophage, thus leading to a dramatic increase in abundance of a single phage genotype [39]. (This type of succession corresponds to the "kill the winner" hypothesis [34].) Nevertheless, while external forcing of seasonal and nonseasonal nature can be accountable for many cases of planktonic and microbial blooms, for many incidences links to external factors are dubious. In terrestrial ecosystems, Anderson and May [4] provided numerous examples of irregular explosions in abundance of insects, which are not linked to seasonal forcing and where the existence of external forcing of nonseasonal nature is unlikely or questionable.

In order to explain the high-magnitude outbreaks followed by periods of low abundance, a number of hypotheses were suggested. The hypothesis that in marine microbial communities the rapid decreases of abundance followed by comparatively long periods of low abundance (the "apparent disappearance") can be a result of phage infection and killing of a particular bacterial species (the "killing the winner population") is appealing and accepted. A certain drawback of this conjecture is that it does not provide the reasons behind the bacterial bloom of this specific bacterial species. As a result, this concept is often coupled with an assumption that the bacterial blooms are triggered by some favourable events, such as an influx of a particular limiting nutrient, which creates favourable conditions for the growth of a particular species, that is, a sort of external forcing, or an external factor is still implicitly assumed.

Hoffmann et al. [22] further developed this concept suggesting that the interaction of a pair of "target bacteria–phage" is sufficient for the rise of high-magnitude self-sustained oscillations in abundance of both species and that no external factor is needed. Unfortunately, to some extent this idea was handicapped by some shortcomings of a mathematical model used by Hoffmann et al. for illustrating this possibility. In particular, this model assumes the unlimited Malthusian growth of

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bacteria (in this aspect, it is similar to the first version of the Lotka–Volterra predator–prey model without carrying capacity of the environment) and, as a result, it is structurally unstable (and hence its dynamics should be destroyed by the slightest perturbation in the model). The phase orbits of this model form a one-parameter family of closed curves, that is, the model has a first integral (it is not clear, however, whether Hoffmann et al. recognized this fact). Consequently, the outcome of this model crucially depends on the initial conditions.

A few years ago, Sobolev proposed a purely theoretical hypothesis that in the predator–prey-type interactions with slow–fast dynamics, the phenomenon of apparent disappearance does not require seasonal forcing and can be explained with the existence of the so-called canard trajectories (see [21, 30, 33] and the references therein).

In order to explain irregular variations in abundance of high magnitude in a seemingly regular system and the phenomenon of apparent disappearance, in this paper we consider a simple model of bacteria–phage interaction, which is a straightforward development of a model proposed by Beretta and Kuang [6]. The model is autonomous and, hence, assumes neither seasonal or periodic forcing, nor external factors of any other nature. However, despite its simplicity, this model is capable of producing results that are able to explain the phenomenon.

2. Model

Bacteriophages (or, simply, phages) are small viruses which infect and kill bacteria, and these were discovered independently by Twort [35] and d'Herelle [16]. Phages are one of the most widespread and diverse entities in the biosphere [28] and it is in the ocean where the highest abundances are found. (There are 10^7 phages per millilitre of sea water; this figure grows up to 10 times at the surface in microbial mats reaching 10^8 virions per millilitre [38].) It was estimated that between 10% and more than 60% of bacterioplankton biomass is killed by bacteriophages [8, 18] and it is observed in different aquatic systems that bacterial mortality due to viral lysis is comparable with protist grazing [9, 10, 36].

While the bacteriophages that are able to infect two or more bacteria species are known [17, 23, 29], it is generally assumed that bacteriophages are usually specific for target bacteria, thus forming stable phage–bacteria pairs. There is no direct interaction between these pairs or, where there is some interaction, it is negligibly weak. However, there is indirect interaction between pairs, because bacteria from different pairs can compete for the same resource; for the sake of simplicity, in this paper we disregard this factor. Following Hoffmann et al. [22], we assume that the dynamics of the "bacteria–phage" pairs are identical in the sense that for all these pairs it is governed by the same principles and depends on the same factors, while the current state of each of these pairs is independent of the others.

A phage consists of a protein hull and the enclosed genetic material, which for the majority of the known phages consists of double-stranded DNA [1]. According

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to their mode of reproduction, phages can be roughly divided into two major types. The first type, the so-called lytic phages, is a highly virulent type. The phages of this type begin reproduction immediately after infecting a bacterium and after a short time lyse (destroy) the host bacterium, releasing new free phages. Phages of the second type, which are known as the non-lytic, or temperate, phages, are either integrating their genetic material into the chromosomal host's DNA or establishing themselves as plasmids. These endogenous phages are then copied with every cell division together with the DNA of the host. They do not kill the host cell until it starts to show signs of stress (meaning it might be about to die soon); at this stage the endogenous phages become active again and start their reproductive cycle, resulting in the lysis of the host cell.

In this paper, we consider the phages with the lytic reproduction cycle. To describe the interaction of a lytic phage–bacteria pair, a mathematical model of host–microparasite interaction with a free-living infective stage of a parasite can be employed. The basic concept for models of a host–microparasite interaction, where the microparasite has a free-living infective stage, was proposed by Anderson and May [4]. Such a model is typically composed of three populations, namely, the susceptible hosts (bacteria, in our case), the infected hosts and parasites in the free stage (phages, in our case), of sizes or concentrations $x(t)$, $y(t)$ and $v(t)$, respectively. The model postulates that the free-living parasites infect the susceptible hosts, that after an instance of infection the infected host moves into the infected class where it remains until death and that the infected hosts produce the free parasites. For a particular case of bacteria–lytic phage interaction, Beretta and Kuang [6] proposed a model where, instead of the unlimited Malthusian reproduction in the Anderson–May model [4], a limiting carrying capacity of the environment, K, was postulated and it was assumed that the susceptible hosts reproduce according to the logistic law. It is assumed that only uninfected bacteria are able to reproduce. This is equivalent to an assumption that the parasite's cytopathogenicity is high and the life span of the infected bacteria is comparatively short and, hence, their contribution to the proliferation of the population is negligible or that the parasite totally suppresses the reproductive ability of the infected bacteria. All the offspring are assumed to be susceptible.

The Anderson–May [4] and Beretta–Kuang [6] models assume homogeneous mixing of all populations and postulate that infective incidences occur according to the law of mass action and, hence, the incidence rate in both these models is bilinear and equal to αxv. However, in marine environment, neither bacteria nor phages are homogeneously distributed in sea water. Sea water is now viewed as a gel composed of microscopic particles ranging from colloids to marine snow, which are composed of organic matter and provide a source of concentrated nutrients for microorganisms [2, 5, 7, 13]. Microorganisms form colonies on the surface of these particles. Phage lysis of such a microbial colony would locally create high concentrations of both parasites and hosts. Moreover, the simultaneous massive release of phage from a dead bacterium in such a colony represents a cooperative effect. Furthermore, there is evidence of phage cooperation; it appears that a simultaneous attack of a bacterium by a few bacteriophages significantly increases chances of infection.

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In the framework of an ordinary differential equation model, a nonhomogeneity of an environment and community effects can be captured by a nonlinearity of functional responses [15]. Accordingly, we assume that the incidence rate is a nonlinear function with respect to both variables. These assumptions lead to the following system of differential equations:

$$\begin{aligned} \dot{x} &= ax\left(1 - \frac{x+y}{K}\right) - h(x)f(v), \\ \dot{y} &= h(x)f(v) - dy, \\ \dot{v} &= \sigma y - mv. \end{aligned} \tag{2.1}$$

Here, $x(t)$ and $y(t)$ are the concentrations of the susceptible and infected bacteria, and $v(t)$ is the concentration of free phages, a is the per capita bacteria reproduction rate, K is the carrying capacity and $1/d$ and $1/m$ are average life spans of the infected bacteria and the free phages, respectively. The parameter σ reflects the fact that the free phages are produced by the infected bacteria and are released at the moment of their death (that is, $\sigma = Nd$, where N is an average number of phages produced by a single infected cell). The functions $h(x)$ and $f(v)$ are positive and monotonically growing for $x, v > 0$ such that the conditions $h(0) = f(0) = 0$ hold.

Compared to the Beretta–Kuang model [6], apart from the nonlinear incidence rate, the model (2.1) also disregards the loss of phages due to infection, since this is typically very small compared with the phages' mortality rate mv. Moreover, computations and analysis show that this term does not affect the qualitative dynamics.

As was mentioned above, the use of a nonlinear incidence rate $h(x)f(v)$ in this model is motivated by nonhomogeneity of the hosts' distribution and cooperative effects of the bacteriophages. For an ordinary differential equation model, a gathering of the hosts in dense colonies or clusters can be described by a concave function with respect to the variable $x(t)$ [15], whereas cooperation of predators or parasites can be captured by a convex function of $v(t)$. For simplicity, further in this paper, we assume that $h(x) = x/(1 + \nu x)$ and $f(v) = \alpha v(1 + \eta v^p)$ (where $\alpha > 0$ and $p > 0$ are parameters). Then $\eta = \nu = 0$ implies the bilinear response. The other functional responses in the model can be assumed nonlinear as well. However, simulations indicate that a nonlinearity of the incidence rate is most important for the model dynamics.

The nonnegative octant $R^3_{\geq 0} = \{(x, y, v) \in R^3 \mid x, y, v \geq 0\}$ is a positive invariant set of the model, that is, any solution with initial conditions in $R^3_{\geq 0}$ remains there indefinitely. Indeed, the natural constraints $h(0) = f(0) = 0$ and $h(x), f(v) > 0$ for $x, v > 0$ (which hold for the functions $h(x) = x(1 + \nu x)$ and $f(v) = \alpha v(1 + \eta v^p)$) ensure that $\dot{x} = 0$ at $x = 0$, and $\dot{y} \geq 0$ and $\dot{v} \geq 0$ hold at $y = 0$ and $v = 0$, respectively. These relationships imply that there is no phase flow through the plane $x = 0$, whereas phase flow through the planes $y = 0$ and $v = 0$ is only possible in the positive direction (that is, inside the positive octant). Furthermore, solutions of the system (2.1) are bounded. More precisely, there is a region Ω bounded by the coordinate planes and the planes $x + y = K$ and $v = \sigma K/m$, which is a positive invariant set of the system (2.1) (and hence any solution initiated in the region remains there, while solutions initiated

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outside of Ω eventually enter this region). Indeed, the total bacteria population $x + y$ does not exceed the carrying capacity K, and $\dot{x} + \dot{y} = ax - bx^2 - bxy - dy < 0$ (where $b = a/K$) holds for all $x + y \geq K$. Moreover, in the region $\{x, y, v \geq 0 \mid x + y \leq K\}$, $\dot{v} \leq 0$ holds for all $v \geq \sigma K/m$.

The origin, $E_0 = (0, 0, 0)$, is an equilibrium state of the model and it is always a saddle point. The yv-plane forms a stable manifold of this three-dimensional saddle, and the x-axis is an unstable manifold. The system also has a phage-free equilibrium state $E_K = (K, 0, 0)$. The stability and properties of this equilibrium state as well as the system general global properties depend on the basic reproduction number [24, 25]

$$R_0 = \frac{\sigma}{d}\frac{1}{m}\frac{\partial f(0)}{\partial v}h(K).$$

(This number is equal to an average number of offspring produced by a single phage introduced in a phage-free environment.) For the model with the bilinear incidence rate,

$$R_0 = \frac{\sigma}{d}\frac{\alpha}{m}K.$$

The phage-free equilibrium state E_K is asymptotically stable (at least locally) if $R_0 \leq 1$ and unstable (a saddle point) if $R_0 > 1$. Apart from these two equilibrium states, which always exist, the system can have positive equilibria, where phages and bacteria coexist. For the bilinear incidence rate αxv, the positive equilibrium state E^* is unique and has coordinates

$$x^* = \frac{md}{\alpha\sigma}, \quad y^* = \frac{am}{\alpha\sigma}\cdot\frac{R_0 - 1}{R_0 + a/d}, \quad v^* = \frac{\sigma}{m}y^*.$$

Note that the bilinear incidence rate $R_0 > 1$ is necessary and sufficient for the existence of the positive equilibrium state.

For the functions $h(x) = x/(1 + \nu x)$ and $f(v) = \alpha v(1 + \eta v^p)$, which we consider in this paper, the system has at most two positive equilibrium states. However, if $R_0 > 1$ holds, then for these functions the positive equilibrium state E^* is unique and appears in the positive octant of the phase space as a result of a saddle-node bifurcation of the point E_K at $R_0 = 1$ [24, 25]. For these functions $h(x)$ and $f(v)$, two positive equilibrium states are possible when $R_0 < 1$. In this case the point E_K is asymptotically stable, and two positive equilibrium states can appear as a result of a "blue sky" saddle-node bifurcation. This scenario corresponds to the Allee effect [3, 14], when a population should be over a certain threshold level to have the mean individual fitness (usually measured as current reproduction number) above level 1 and, thus, to persist [24, 25].

3. Apparent disappearance

A remarkable property of the model (2.1) is that even for the bilinear incidence rate αxv, it admits a supercritical Andronov–Hopf bifurcation. Specifically, a decrease of a, d or m or, alternatively, an increase of α, σ and, in particular, of the carrying capacity K, eventually leads to the loss of stability of the equilibrium state E^* and to

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the appearance of a stable limit cycle in the system phase space [6, 26]. In the model (2.1), the values of the parameters a, d, m and σ, as well as the forms of the functions $h(x)$ and $f(v)$, are specific for a particular phage–bacteria pair, and sizeable variations of these parameters are unlikely to occur. In contrast, in marine environment, the carrying capacity K can significantly vary (for example, seasonally). Moreover, it is commonly believed that in marine ecosystems there is no lack of nutrient for bacteria and that food is not a limiting factor for bacterial growth [2, 5, 13, 22, 38, 39]. These considerations make K the most important parameter for the phage–bacteria system dynamics, and indicate that principal changes of this dynamics are possible in response to variations of environmental conditions.

Such a loss of stability in a predator–prey system via the Andronov–Hopf bifurcation, which occurs in response to the growth of the carrying capacity of the environment, is referred to as "the paradox of enrichment". We have to stress, however, that a nonlinearity of the incidence rate (or the attack rate, in the case of a predator–prey system) with respect to the prey population $x(t)$ is essential for the Andronov–Hopf bifurcation in a two-dimensional predator–prey model [32]. Therefore, it is remarkable that in the model (2.1) the bifurcation occurs even for the bilinear incidence rate.

A stable limit cycle appears in the system phase space as a result of a supercritical Andronov–Hopf bifurcation, which occurs when a pair of isolated nonzero, simple complex-conjugate eigenvalues of a linearized system crosses the imaginary axis from left to right at a nonzero speed, while the rest of the spectrum (the third eigenvalue) remains in the open left half-plane. Let us assume for simplicity that the incidence rate is bilinear (that is, $h(x)f(v) = \alpha xv$) and that $R_0 = (\sigma/d)(\alpha/m)K > 1$ holds. Then the positive equilibrium state E^* exists. The Jacobian matrix at the point E^* is

$$J = \begin{pmatrix} -bx^* & -bx^* & -\alpha x^* \\ \alpha v^* & -d & \alpha x^* \\ 0 & \sigma & -m \end{pmatrix},$$

where $b = a/K$. The corresponding characteristic equation is

$$\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0, \tag{3.1}$$

where

$$\begin{aligned} a_1 &= -\operatorname{tr} J = bx^* + d + m > 0, \\ a_2 &= bx^*(d + m + \alpha v^*) > 0, \\ a_3 &= -\det J = \alpha x^* v^*(bm + \sigma\alpha) > 0. \end{aligned}$$

By the Routh–Hurwitz criterion [19, p. 197], conditions for the Andronov–Hopf bifurcation are satisfied if a_1, a_2, $a_3 > 0$ and $\Delta = a_1a_2 - a_3 = 0$. Here, the inequalities $a_1, a_2, a_3 > 0$ ensure that all roots of the characteristic equation are in the open left half-plane, while $\Delta = a_1a_2 - a_3 = 0$, together with $a_1, a_2, a_3 > 0$, is a sufficient condition

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for the existence of one negative real root and a pair of pure imaginary roots. For the characteristic equation (3.1),

$$\Delta = x^*[b(bx^* + d + m)(d + m) + \alpha v^* b(bx^* + d) - \sigma\alpha^2 v^*].$$

That is, $\Delta = 0$ holds when

$$m^3 \frac{d}{\sigma\alpha} b^3 + m[(d + m)^2 + d(m + a)]b^2 + \sigma\alpha[(d + m)^2 + d(m + a)]b - a(\sigma\alpha)^2 = 0.$$

Denoting $\kappa = K^{-1}$ and $M = [(d + m)^2 + d(m + a)]/\sigma\alpha$, and recalling that $b = a/K = a\kappa$,

$$P(\kappa) = \frac{a^2}{d^2}(x^*)^3\kappa^3 + \frac{am}{\sigma\alpha}M\kappa^2 + M\kappa - 1 = 0. \tag{3.2}$$

If the positive equilibrium state exists, then the product of the roots of the polynomial $P(\kappa)$ is positive (and equal to $d^2/a^2(x^*)^3$), whereas their sum is negative (and equal to $-Md^2/a^2(x^*)^3$). Hence, (3.2) always has one positive root $\kappa_{cr} > 0$ such that $\Delta(\kappa_{cr}) = 0$. (Two other roots are in the open left-half of the complex plane.) Furthermore, for $\kappa \to \infty$ (that is, for $K \to 0$), the polynomial (3.2) is positive and, hence, by the Routh–Hurwitz criterion, for $\kappa \to \infty$ all three roots of the characteristic equation (3.1) have negative real parts. When $\kappa \to 0$ (that is, for $K \to \infty$), the polynomial (3.2) is negative. This implies that if K is not sufficiently large, the equilibrium E^* is asymptotically stable. However, as K grows, $\Delta(K)$ decreases and eventually crosses zero level at $K = K_{cr} = \kappa_{cr}^{-1}$. At this value of K, a supercritical Andronov–Hopf bifurcation occurs, the equilibrium reverses its stability and a stable limit cycle appears. The size of this cycle grows as K further increases.

With a further increase of α, σ or, most importantly, of the carrying capacity K, or with a decrease of a, d or m, the size of this limit cycle grows as well. With the growth of K, the feasible region Ω also expands towards the inner part of the octant $R^3_{\geq 0}$. (Indeed, the planes $x + y = K$ and $v = \sigma K/m$ are parts of the boundary of the region.) However, only these parts of the boundary move with varying of K, whereas the rest of the boundary is formed by the coordinate planes and hence is fixed. Moreover, the coordinate x^* of the positive equilibrium state E^* does not depend on K either. This implies that as the size of the limit cycle grows, the cycle moves closer and closer to the coordinate planes. In particular, the cycle approaches the yv-plane, which is a part of the boundary and, at the same time, the stable manifold of the equilibrium state $E_0 = (0, 0, 0)$. Therefore, as the limit cycle size grows with the growth of K, the limit cycle is squeezed to the yv-plane and, as a result, it unavoidably comes into the vicinity of the origin, where all three populations, as well as their rates of change, are very low. The larger K becomes, the closer the limit cycle is squeezed to the yv-plane and, hence, it comes closer to the vicinity of the origin, where it remains longer.

This implies that to a large extent the system dynamics varies in response to variations (seasonal or random) of K. It also implies that this system can exhibit dynamics with long periods of low abundance, when one of the populations, or all populations, remain at very low and probably undetectable levels (the "apparent disappearance"), alternating with comparatively short periods of a high abundance.

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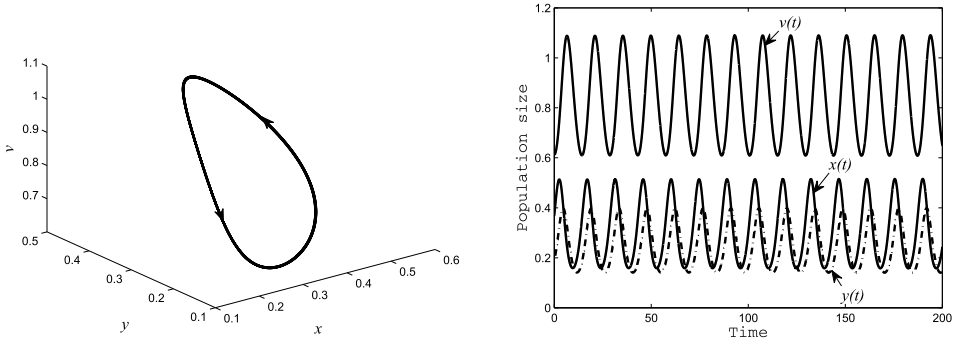

FIGURE 1. Appearance of the stable limit cycle; here, $K = 3.33$.

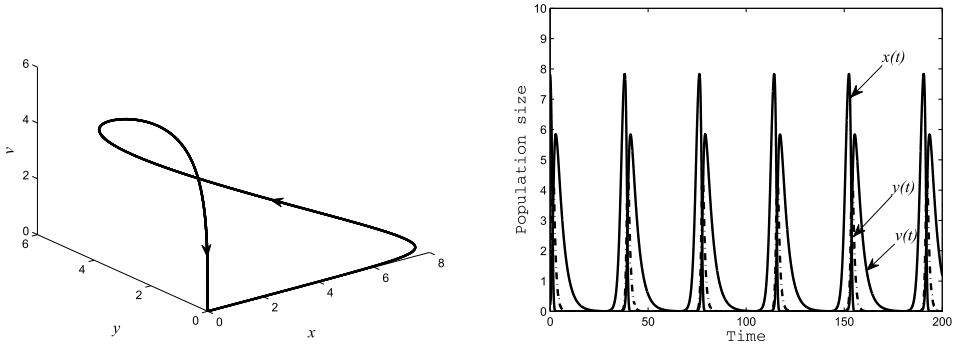

FIGURE 2. Further extension of the stable limit cycle: note the squeezing of the cycle to the yv-plane and x-axis; here, $K = 10$.

These speculative considerations are illustrated by Figures 1–3, where the appearance and development of the stable limit cycle, as well as an arising phenomenon of apparent disappearance in the model (2.1), is clearly seen. In these figures we used $\alpha = 0.1$. The other parameters are $a = 1.0, d = 1.0, \sigma = 1.0$ and $m = 0.3$. Furthermore, $K = 3.33$ in Figure 1, $K = 10$ in Figure 2 and $K = 20$ in Figure 3. Please note that a comparatively small increment of the carrying capacity (K is three times larger in Figure 2 compared with Figure 1) leads to the growth of the limit cycle size and to its squeezing to the yv-plane and to the origin, which is clearly seen in Figures 2 and 3. Note that the population size scales in these figures are different.

A concavity of the incidence rate with respect to x, which corresponds to the spatial heterogeneity of the environment or the target bacteria spatial distribution, makes the apparent disappearance phenomenon more distinctive. Figure 4 shows the variation of the bacterial (the dashed black curves) and viral (the solid blue curves) abundance in time for a nonlinear incidence rate of the form $\alpha xv/(1 + \nu x)$ for $\nu = 0.1, 1.0, 2.0, 2.5, 2.9$. Figure 4(f) exhibits the limit cycle in the system phase space for $\nu = 2.9$. In Figure 4, $K = 20$ and the other parameters are the same as in Figures 1–3. Please note that the time interval in Figure 4 is 800 (days), whereas in

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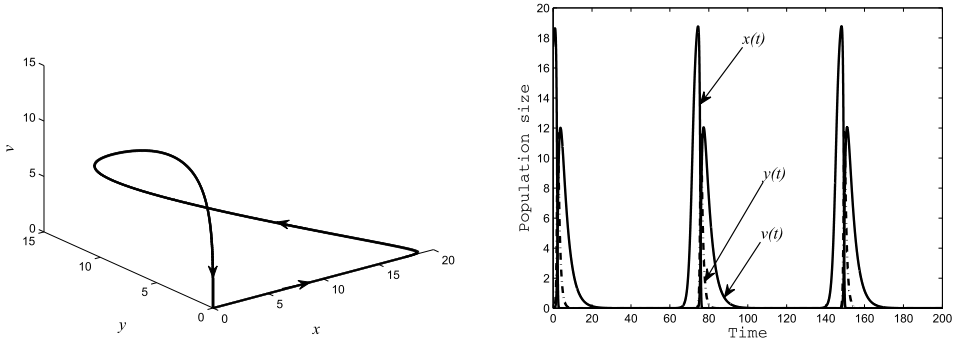

FIGURE 3. Further expansion of the limit cycle and arising of apparent disappearance. Here, $K = 20$; the other parameters are the same as in Figures 1 and 2.

Figures 1–3 it is 200 (days). Note that for small concavity ($\nu = 0.1$), the period of oscillations does not differ from that in Figure 3. However, as the concavity increases (ν grows), the period increases as well. Thus, for $\nu = 2.0$ the period is approximately two times longer than that for $\nu = 1.0$.

Another distinctive feature of the system with the concave incidence rate is that in this case the bacterial abundance exhibits alternating phases of very low (the apparent disappearance) and very high (close to K) abundance. Moreover, for larger ν, the phases of high bacterial abundance are longer than those of low abundance. This implies that for the concave incidence rate, the stable limit cycle in the phase space is tightly squeezed to the phage-free equilibrium state E_K, and the system remains in the vicinity of this saddle point for a comparatively longer time.

The convexity of the incidence rate with respect to v makes the properties exhibited in Figure 4 even more striking, since for such an incidence rate the null-surface of the variable $y(t)$ is not a vertical plane (as it is for the linear function $f(v)$), but it is bent in such a way that its point of intersection with the x-axis moves closer to the equilibrium state E_K (see Figure 5). Hence, the canard point also moves in the right-hand direction closer to the equilibrium state E_K and, accordingly, the system remains in the vicinity of this equilibrium state longer.

4. Discussion and conclusion

In marine ecosystems, significant variations of microbial abundances of nonseasonal nature are common. In many cases, such variations are caused by seasonal variations. Nevertheless, unexpected outbreaks and blooms that cannot be explained by seasonality are not unusual either. During such outbreaks, microorganisms undergo the phenomenon of “apparent disappearance”. Moreover, in such cases, the transitions from high to low abundance and then to the succeeding blooms are usually very fast.

With an aim to demonstrate why and how such irregular variations of abundance and the apparent disappearance can arise in a seemingly regular marine environment, in this paper we considered a simple model of bacteria–phage interaction. As a

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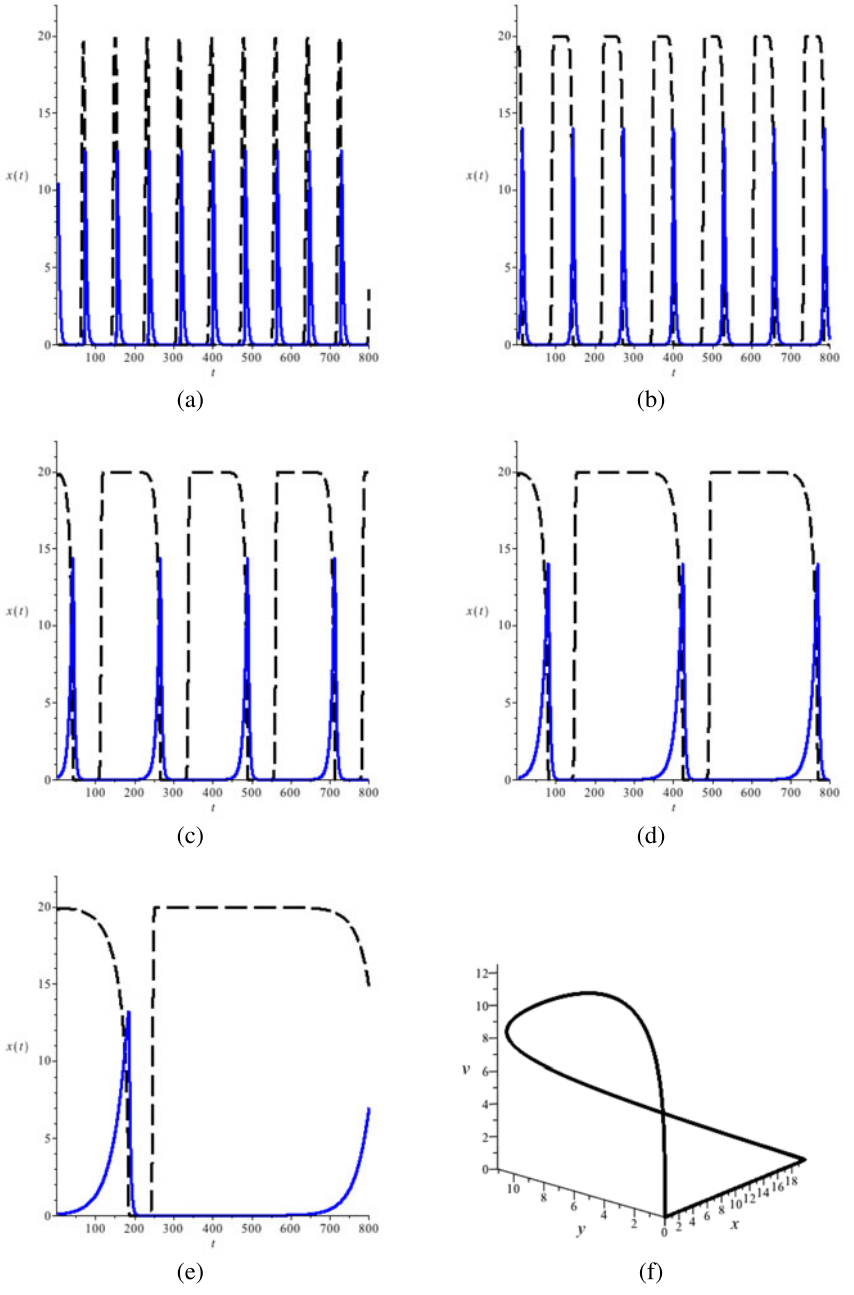

FIGURE 4. Subfigures (a)–(e): variation of the bacterial (the dashed black curves) and viral (the solid blue curves) abundance in time for the model (2.1) with incidence rate $\alpha xv/(1+\nu x)$ for $\nu = 0.1, 1.0, 2.0, 2.5, 2.9$, respectively. Subfigure (f): the limit cycle in the phase space for $\nu = 2.9$. In all figures, $K = 20$ and the other parameters are the same as in Figures 1–3.

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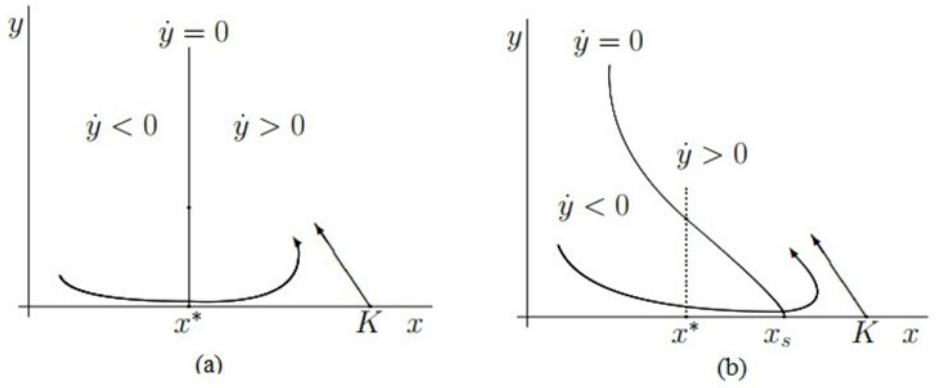

FIGURE 5. Schematic representation of the mutual location of the null-surface of the variable $y(t)$, the canard point and the phage-free equilibrium state E_K for the linear (in (a)) and convex (in (b)) with respect to y incidence rates.

basis for this study, we took the Beretta–Kuang host–parasite model [6] with a free-living infective stage of the phage, where we postulated the logistic proliferation of the bacteria and bilinear infection rate. Despite its simplicity, this model exhibits complex dynamics and demonstrates that the behaviour of the system may undergo a radical transformation in response to a variation of the parametrization. In particular, in response to an increment of the environment carrying capacity (simply speaking, an increase of nutrient concentration in the sea water), this model admits a supercritical Andronov–Hopf bifurcation leading to a loss of stability and a rise of self-sustained oscillations. This resembles the enrichment phenomenon in a two-dimensional predator–prey model. A principal difference is that the enrichment phenomenon crucially depends on the nonlinearity of the incidence rate with respect to the prey population, whereas in the Beretta–Kuang model the loss of stability and the rise of the self-sustained oscillations occur even for the standard bilinear incidence rate. Furthermore, due to a significant difference in the life cycle time scales of the host bacteria and the phage, and to the delay between the instance of infection and the death of infected hosts, which is captured to some extent by this model, the loss of stability and the appearance of the stable limit cycle can eventually lead to the phenomenon of apparent disappearance.

Our model allows a simple interpretation for the irregularities in the abundance and apparent disappearance of microorganisms. This interpretation is based on a combination of three factors, namely: (i) instability of a positive equilibrium state, (ii) the existence of a stable limit cycle in a bounded feasible region, (iii) the existence of a saddle-type equilibrium state at the origin, with a stable manifold forming a part of the boundary of the feasible region. Under these conditions, as the size of the limit cycle grows, it is squeezed to the boundary of the feasible region and closely approaches the equilibrium state along its stable manifold. In the vicinity of an equilibrium state, the rates of changes for the phase variables are low and, given that this equilibrium

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is at the origin, their values are small. Consequently, as the trajectory is squeezed to the equilibrium state, the system exhibits lengthy periods of very low abundance. The difference in the bacterial and viral life cycle time scales changes the abundance from high to low very fast.

Moreover, in the vicinity of the origin, the rates of change of the phase variables are proportional to the distance between a current state and the equilibrium. Hence, durations of the low-abundance periods essentially depend on how closely the cycle is pressed to the boundary and the origin. This implies that these durations are very sensitive to variations of the system parameters and, in particular, to variations of the carrying capacity K. In marine ecosystems, the carrying capacity can vary significantly (for instance, seasonally) and, as a result, irregularities of the dynamics can be rather typical for marine microbial systems, especially for marine bacteria–phage interactions. These also indicate that the results of isolated tests, and even a sequence of tests taken with too wide intervals, can be misleading.

For viruses, the periods of low abundance can be even longer if the x-axis is a heteroclinic orbit connecting two saddle points (as in our model and, in fact, as it should be in any realistic model of bacteria–virus interaction). In such a case, as the size of the stable limit cycle grows, the cycle comes into the vicinity of the second saddle-type equilibrium state, where $x \approx K$, $v \approx 0$ and $\dot{v}$ is small. As a result, the system exhibits an oscillatory regime of alternating prolonged periods of high and low bacterial abundance with fast switchings. Such regimes arise and become more apparent as the canard point on the x-axis moves to the right-hand side.

In this paper, we initially considered the model with the standard bilinear incidence rate and postulated that the other functional responses are linear. However, there are a number of reasons to believe that in marine ecosystems the functional responses are nonlinear [20, 22]. In particular, the bilinear incidence rate is based on the assumption of a homogeneous distribution of the bacteria and bacteriophage populations in sea water, whereas, in reality, in the marine environment, bacteria often form dense colonies on the surface of small particles providing a source of concentrated nutrients for them [2, 5, 7, 13]. The distribution of phages is also nonhomogeneous, because they are released as a cohort at the moment of death of their bacterium host; community effects and some form of cooperative effects are likely to occur for such massive simultaneous releases of the virus [18]. In the framework of an ordinary differential equation model, a nonhomogeneity of the host distribution and cooperative effects of the phage can be captured, to some extent, by nonlinearities of the functional responses. Specifically, the nonhomogeneous spatial distribution of the bacteria can be mimicked by an incidence rate which is concave with respect to the host population, whereas community effects in the viral population correspond to an incidence rate that is convex with response to the phage population [15]. The nonhomogeneity of the environment can also be captured by nonlinear death/removal rates. Note that for such an incidence rate, the phenomenon of apparent disappearance is more intense. By the analogy with the phenomenon of enrichment, for such an incidence rate, the Andronov–Hopf bifurcation (which leads to the loss of stability and the appearance of

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the stable limit cycle) occurs at a lower level of K. Moreover, for such incidence rates, the canard point is located on the x-axis further to the right-hand side and, hence, the break up from the canard is delayed, compared with the bilinear incidence rate. As a result, the cycle is pressed closer to the virus-free equilibrium point $(K, 0, 0)$. This yields solutions with prolonged periods of high bacterial abundance, longer periods of viral apparent disappearance and a sharper and shorter viral outbreak.

In this model, irregularities in the dynamics can arise due to slow variations of the carrying capacity K, which hardly remains constant in the real aquatic systems. However, apart from that, the model indicates another possibility that should be taken into account as well. As we mentioned above, the bacteria–phage pairs can indirectly interact, because the bacterial species in different pairs compete for the same resource. As a result, fast changes of this resource concentration (and, consequently, changes in the carrying capacity) caused by blooms of the competing bacterial species are also possible. In a system where several bacterial species compete for the same resource and are parasitized by viruses, a likely outcome would be an extremely irregular (and probably chaotic) dynamics.

In conclusion, in the dynamics with the alternations of abundance, when fast explosions of a population are followed by equally fast declines, the phenomenon of apparent disappearance is not uncommon in the population dynamics in general, including terrestrial ecosystems (in particular, in invertebrates and microorganisms) and host–parasite systems [4, 37]. It is likely that in many other cases, where the phenomenon of apparent disappearance cannot be influenced by seasonal impact, the mechanism behind this irregular dynamics is the same as, or similar to, one that we described in this paper. An essential element of this dynamics is the existence of a stable trajectory (a stable limit cycle) that passes near one of the several saddle-type equilibrium states. It is not necessary for one of these saddle points to be located at the origin, as it is in the model (2.1). However, their stable manifolds should be part of the boundary of the feasible region, to which the trajectory is squeezed, and, hence, the saddle points should be located on the boundary as well.

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