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MODELS ORDER REDUCTION AND EQUIVALENCE: PARADOX OF ENRICHMENT IS A 3-DIM BACTERIOPHAGES DYNAMICS MODEL AS CASE STUDY

VLADIMIR SOBOLEV, ELENA SHCHEPAKINA AND ANDREI KOROBEINIKOV

Abstract. The paradox of enrichment in a three-dimensional model for bacteriophage dynamics with a free infection stage of the phage and bilinear incident rate is considered. An application of the technique of singular perturbation theory allow us to demonstrate why the paradox arises in this three-dimensional model despite the fact that it has a bilinear incident rate (while in two-dimensional predator-prey models it usually associated with the concavity of the attack rate). Our analysis demonstrates that the commonly applied approach of the model order reduction using the so-called quasi-steady-state approximation can lead to a loss of important properties of an original system.

1. Introduction

The paradox of enrichment that is the loss of stability in a predator-prey model in response to an increase of the food availability to the prey was discovered by M. Rosenzweig in 1971 [11]. In a typical two-dimensional predator-prey system

$$\begin{aligned} \dot{x} &= ax\left(1-\frac{x}{K}\right)-\eta(x)y, \\ \dot{y} &= \kappa\eta(x)y-dy \end{aligned} \tag{1}$$

the concavity of the predation rate with respect to the prey population $\eta(x)$ is essential for the paradox to arise, and the general understanding of this phenomenon is that for an abundant food supply and for a predation rate concave with respect to the prey, the loss of prey due to predation increases slower than the growth of its population itself. Definitely, such a situation never occurs, and the paradox never arises for the standard bilinear attack rate or a rate convex with respect to the prey number. In model (1), $x(t)$ and $y(t)$ are the prey and predator populations (or concentrations), respectively; a is *per capita* prey reproduction rate; K is the carrying capacity of the environment; $\eta(x)y$ is the attack rate; κ is the consumption efficiency, and d is predator death rate in the absence of the prey.

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Mathematically, the phenomenon is a consequence of a supercritical Hopf bifurcation in the model, which occurs at a positive (coexisting) equilibrium state: as the carrying capacity K increases above a certain critical level K_{cr}, the coexisting equilibrium state loses its stability, and a stable limit cycle associated with self-sustained oscillations emerges.

While in a two-dim predator-prey system the concavity of the attack rate is necessary for the phenomenon to occur, in models which comprise more variables the supercritical Hopf bifurcation can occur even for bilinear incidence rate. Moreover, it can be expected that for three- or higher dimensional models the bifurcation may occur even for functional responses convex with respect to the prey. E. Beretta and Y. Kuang [3] suggested the following three-dimensional model of the marine bacteriophages dynamics:

$$\begin{aligned} \dot{x} &= ax - bx(x+y) - \alpha xv, \\ \dot{y} &= \alpha xv - dy, \\ \dot{v} &= \bar{\sigma} y - \bar{m} v - \alpha xv. \end{aligned} \tag{2}$$

This model is a development of the ideas and models of the dynamics of parasites with a free infectious stage suggested earlier by R.M. May and R.M. Anderson [1], and it is specifically tailored to describe the dynamics of bacteriophage in aquatic environment. The model comprises three variables, namely the host bacteria uninfected by phage of population size (or concentration) $x(t)$, the infected bacteria of population size (or concentration) $y(t)$, and the free phage of population (or concentration) $v(t)$, and postulate that (i) the host bacteria population grows according to the the logistic growth law; that (ii) the free phage are produced by the infected bacteria and are released at the moment of its death (and hence $\bar{\sigma} = Nd$, where N is an average number of phage produced by a single infected cell); that (iii) infection of the hosts occurs according to the law of mass action (and hence is represented by the bilinear incidence rate), and that (iv) average life expectations of the infected hosts and the free phages are $1/d$ and $1/\bar{m}$, respectively.

A remarkable feature of this model is that, despite the fact that the incidence rate is bilinear, it still admits the supercritical Hopf bifurcation, associated with the loss of stability of the positive equilibrium state and an emergence of a stable limit cycle, in response on an increment of the carrying capacity of the host bacteria K; that is, the model exhibits the enrichment paradox. A possible explanation why it may occur is that the free infectious stage of the phage is equivalent to the exponentially distributed delay in a two-dimensional delay model

$$\begin{aligned} \dot{x}(t) &= ax(t)\left(1 - \frac{x(t)}{K}\right) - \beta x(t) \int_0^\infty \exp(-\tau) y(t-\tau)\, d\tau, \\ \dot{y}(t) &= \kappa\beta x(t) \int_0^\infty \exp(-\tau) y(t-\tau)\, d\tau - dy(t). \end{aligned} \tag{3}$$

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Predator-prey model of such type with a finite or a distributed delay are known to exhibit self-sustained oscillations [2, 4, 6, 8, 9]. However, a delay is not the only possible explanation, and in fact this explanation is not the best possible, as it implies an establishing an equivalence of a reasonably simple three-dimensional system (2) with a considerably more complex infinitely dimensional delay system (3). Instead, we are to demonstrate that the three-dim model with bilinear incidence rate is equivalent, in a certain sense which will be explained below, to a two-dimensional predator-prey model, where the effect of the free infective stage of the phage is resulted in a non-linearity of the incidence rate, leading to the concavity of that, which, in its turn, leads to the paradox of enrichment.

2. Singularly perturbed system

The theory and applications of singularly perturbed systems of differential equations, traditionally connected with the problems of fluid dynamics and nonlinear mechanics, has been developed intensively and the methods are applied actively to the solution of a wide range of problems from other areas of natural science. This can be explained by the fact that such singularly perturbed systems appear naturally in the process of modelling various processes, that are characterized by slow and fast motions simultaneously present. In the most application, it is necessary to consider the behaviour of the system as a whole rather than separate trajectories, investigating the system dynamics by means of a qualitative analysis.

Typically, to investigate a singularly perturbed system a combination of asymptotic and geometrical techniques of analysis are applied [10, 13]. The essence of this approach consists in separating out the slow motions of the system under investigation. Then the order of the differential system decreases, but the reduced system, of a lesser order, inherits the essential elements of the qualitative behaviour of the original system in the corresponding domain and reflect the behaviour of the original models to a high order of accuracy when the slow integral manifold is attracting. A mathematical justification of this method can be given by means of the theory of integral manifolds for singularly perturbed systems [13].

Consider system of ordinary differential equations

$$\dot{X} = f(t, X, Y, \varepsilon), \tag{4}$$

$$\varepsilon\dot{Y} = g(t, X, Y, \varepsilon), \tag{5}$$

where $X \in R^m$, $Y \in R^n$, $t \in R$ and ε is a small positive parameter. Such systems are called *singularly perturbed systems*, since when $\varepsilon = 0$ the ability to specify an arbitrary initial condition for $Y(t)$ is lost. The usual approach to the qualitative study of (4) is to consider firstly the so-called *limiting system* or *quasi-steady-state*

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approximation ($\varepsilon = 0$):

$$\frac{dX}{dt} = f(t, X, Y, 0), \tag{6}$$

$$0 = g(t, X, Y, 0), \tag{7}$$

and then to draw conclusions about the qualitative behavior of the full system (4), (5) for a sufficiently small ε.

For many applied problems the use of the limiting system instead of the full system gives acceptable results. However, in some cases the approximation (6), (7) is too crude, lacks some important properties of the original system. A method that can be used to obtain a more precise approximation, which is needed for such models, relies on the theory of integral manifolds. The objective is to essentially replace the original system by another system on an integral manifold whose dimension is equal to that of the slow subsystem.

Recall that a smooth surface S in $R \times R^m \times R^n$ is called an integral manifold of the system (4), (5) if any integral curve of the system that has at least one point in common with S lies entirely on S. Formally, if $\big(t_0, X(t_0), Y(t_0)\big) \in S$, then the integral curve $\big(t, X(t,\varepsilon), Y(t,\varepsilon)\big)$ lies entirely on S. The only slow integral manifolds of system (4), (5) discussed here are those of dimension m (the dimension of the slow variable X) that can be represented as graphs of vector-valued functions $Y = h(X, t, \varepsilon)$. Moreover, we consider here the attractive integral manifolds, only. It is important that any trajectory of (4), (5) can be represented as a trajectory on the attractive slow integral manifold plus an asymptotically negligible terms. The flow on the slow integral manifold is described by the equation

$$\dot{X} = f\big(t, X, h(t, X, \varepsilon), \varepsilon\big). \tag{8}$$

2.1. **Asymptotic expansions.** When the method of integral manifolds is being used to solve a specific problem, then a central question is the calculation of the function $h(X, t, \varepsilon)$ in terms of the manifold described. An exact calculation is generally impossible, and hence an approximation is necessary. One possibility is the asymptotic expansion of $h(X, t, \varepsilon)$ in integer powers of the small parameter ε:

$$h(t, X, \varepsilon) = \phi(t, X) + \varepsilon h_1(t, X) + \cdots + \varepsilon^k h_k(t, X) + \cdots . \tag{9}$$

By substituting this formal expansion into *invariance equation*

$$\varepsilon \frac{\partial h(t, X, \varepsilon)}{\partial t} + \varepsilon \frac{\partial h(t, X, \varepsilon)}{\partial X} f\big(t, X, h(t, X, \varepsilon), \varepsilon\big) = g\big(t, X, h(t, X, \varepsilon), \varepsilon\big) \tag{10}$$

and then equating powers of ε, we can obtain coefficients $h_1, h_2, ...$ and thus calculate an approximation to $h(X, t, \varepsilon)$ under taking into account that $\phi(X, t)$ is determined by $g\big(X, \phi(X, t), t, 0\big) = 0$.

Asymptotic expansions of slow integral manifolds were first used in [14]. A more detailed description of the method can be found in [10, 13].

To calculate the asymptotical approximation of slow invariant manifolds in this paper we will use another approach, which is referred to as "iterative method". The method was proposed by Fraser [5], and developed by Fraser and Roussel [12] for autonomous systems that are linear with respect to the fast variables in the case of scalar slow and fast variables. This method is described in Appendix.

2.2. **The idea of models asymptotic equivalence.** The concept of equivalence of models which, in general, have phase spaces of different size, is not trivial and its rigorous introduction and discussion are out of the scope of this paper. Here we introduce just a general idea of the concept that we refer to as "asymptotic equivalence".

We will call two models, with the phase spaces of dimensions n and m (where, generally, $n \neq m$), respectively, asymptotically topologically ω-equivalent, if phase spaces of both models contain invariant manifolds of equal dimensions, such that *(i)* each of the manifolds contains all ω-limit sets of the corresponding model, and *(ii)* the manifolds and the ω-limit sets of both models are topologically equivalent. It is easy to see that, since such a manifold contains all ω-limits, it is a global attractor of the system and there is no compact invariant sets off the manifolds. For convenience we can add a condition that the manifolds are globally stable, and hence all trajectories converges to the manifolds. The entire phase space of a smaller dimension can be such a manifold.

Analogically, two models are asymptotically topologically α—ω-equivalent, if, apart from the ω-limits, the manifolds contain all the α-limits (apart from the infinity) as well.

Since all trajectories of each system eventually converge to the corresponding manifold, we can state that the manifold contains all practically relevant motions of the system. The equivalent statement is that the pieces of trajectories which are outside of a ε-neighborhood of a manifold are irrelevant, because for any small ε and any initial condition a corresponding trajectory enters for a finite time the ε-neighborhood of the manifold and remains there.

The concept of asymptotic topological ω-equivalence (and α—ω-equivalence) is particularly obvious for the singularly perturbed (or slow-fast) systems, where the phase spaces are typically composed of a (globally stable) slow manifold and the fast motions converging to that. For such a system, unless one is interested in the fast motion and in short time intervals, all practically relevant trajectories belong to the slow manifold, and the system is equivalent (in the above defined sense) to the reduced system on the slow manifold.

Our objective is to demonstrate that system (2) is equivalent, in the above-defined sense, to system (1) with the concave attack rate. For this we use the technique that is known as time scales separation.

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3. Bacteriophages interaction model

We rewrite model of host-microparasite interaction with a free-living infective stage of a parasite (2) as

$$\begin{aligned} \dot{x} &= ax - bx(x+y) - \alpha xv, \\ \dot{y} &= \alpha xv - dy, \\ \varepsilon\dot{v} &= \sigma y - mv - \varepsilon\alpha xv, \end{aligned} \tag{11}$$

where $\sigma = \varepsilon\bar{\sigma}$ and $m = \varepsilon\bar{m}$. The system can have a positive equilibrium state E^* with coordinates

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

Here $R_0 = a\alpha(\sigma - \varepsilon d)/bmd$ is the basic reproduction number; the condition $R_0 > 1$ is necessary and sufficient for the existence of this equilibrium state. We assume that $R_0 > 1$ is held, and hence there is a unique positive equilibrium state E^*.

For the bacteriophage model, small parameter ε has a transparent biological interpretation. As we mentioned above, the release of free bacteriophages occurs at the moment of death of the host bacteria, and hence for this model $\bar{\sigma} = Nd$ holds. Therefore, we can postulate that for this particular model $\varepsilon = 1/N$, and then $\sigma = d$ and $\sigma - \varepsilon d = d(1-\varepsilon)$ hold.

The corresponding limiting system is

$$\begin{aligned} \dot{x} &= ax - bx(x+y) - \alpha xv, \\ \dot{y} &= \alpha xv - dy, \\ 0 &= \sigma y - mv, \end{aligned} \tag{12}$$

or, by excluding v,

$$\begin{aligned} \dot{x} &= ax - bx(x+y) - \alpha x\sigma y/m, \\ \dot{y} &= \alpha x\sigma y/m - dy. \end{aligned} \tag{13}$$

This system is identical to the classic Lotka-Volterra predator-prey model. It has (for all $\bar{R}_0 = a\alpha\sigma/mbd > 1$) a positive equilibrium state (x^*, y^*) which is always globally asymptotically stable, and hence admits no Hopf bifurcation. That is, the limiting system is not equivalent to the original three-dim model, and a more precise approximation is needed.

For system (11), invariance equation (10) takes the form

$$\varepsilon h_x\big(ax - bx(x+y) - \alpha xh\big) + \varepsilon h_y\left(\alpha xh - dy\right) = \sigma y - mh - \varepsilon\alpha xh.$$

Taking into account that the limiting equation ($\varepsilon = 0$) gives $h(x, y, 0) = \sigma y/m$, we can rewrite the invariance equation in the form

$$\varepsilon\frac{\sigma}{m}\left(\alpha xh - dy\right) = \sigma y - (m + \varepsilon\alpha x)h.$$

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From this equality we immediately obtain the first order approximation for $h = h(x, y, \varepsilon)$:

$$h = \varphi^{(1)}(x, y) = \frac{\sigma(1 + \varepsilon d/m)y}{m + \varepsilon\alpha(1 + \sigma/m)x}.$$

For more detailed derivation of this expression see Appendix and [13].

Thus, the flow on the slow invariant manifold $v = \varphi^{(1)}(x, y)$ is given by the equations

$$\begin{aligned} \dot{x} &= ax - bx^2 - \left(\frac{\alpha\sigma(1 + \varepsilon d/m)x}{m + \varepsilon\alpha(1 + \sigma/m)x} + bx\right) y, \\ \dot{y} &= \frac{\alpha\sigma(1 + \varepsilon d/m)x}{m + \varepsilon\alpha(1 + \sigma/m)x} y - dy. \end{aligned} \tag{14}$$

The most important feature of this system, compared with the limiting system (13), is that the incident rate in system (18) is nonlinear and concave. In fact, the "consumption rate" is of the Holling type 2 functional response, whereas the "attack rate" is of the same type modified by a bilinear term bxy. The Holling type 2 functional response (which is also known as Michaelis-Menten kinetics in biochemistry, or the Monod equation in microbiology) usually implies the assumption that predation is limited by the predator ability to handle the killed prey and process food. Accordingly, in the two-dim reduced model the nonlinearity of incidence rate can be interpreted as the necessity to spend time producing the free stage infective agents. The mentioned bilinear term bxy appears only in the first equation and can be interpreted as infection-induced mortality of the uninfected bacteria (a sort of apoptosis). In a predator-prey model such a term can be interpreted as "killing for sport", or "killing without consumption" [7]. Since this term is bilinear it does not affect the global dynamics of the model. As a result of the nonlinearity of the functional responses, the system admits the supercritical Hopf bifurcation, and hence exhibits the paradox of enrichment. Moreover, the sizes of the stable limit cycles which emerge in systems (11) and (18) as the result of the bifurcation are very close (see Fig. 1), and the three-dim limit cycle located on the two-dim slow manifold practically coincides with the two-dim limit cycle in system (18).

4. Conclusion

The objective of this paper was demonstrate why the paradox of enrichment, which in two-dimensional predator-prey models is usually associated with the concavity of the attack rate, arises in three-dimensional bacteria-phage systems with a free infective stage of the phage and bilinear incidence rate. In order to do this, we reduced the three-dim model to a two-dim system on the slow manifold; the resulting two-dim system is equivalent to a standard predator-prey model with the Holling type 2 attack rate. This type of functional response, which is

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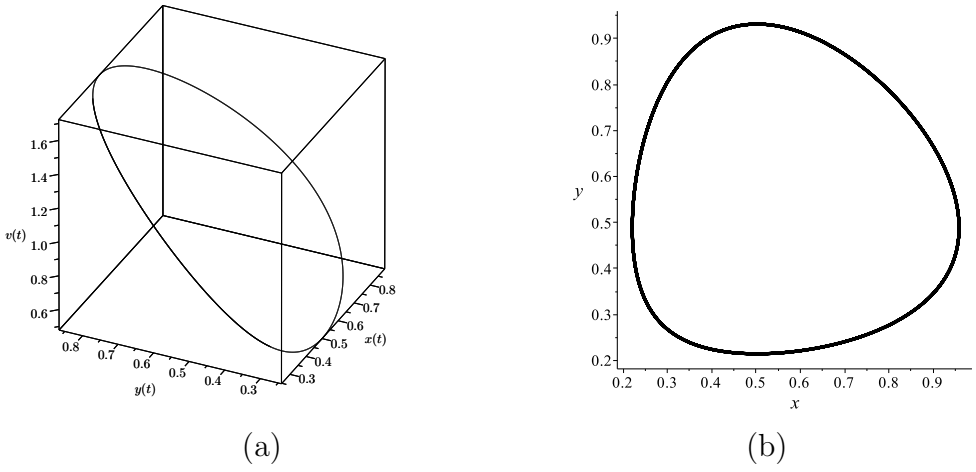

(a) (b)

FIGURE 1. The limit cycles in systems (11), plot (a), and (18), plot (b). Please note that both limit cycles are of the same size. Both figures are for $a = 1.0$, $\alpha = 1.0$, $d = 1.0$, $\sigma = 2.0$, $m = 1$, $\varepsilon = 0.01$, and $K = 34.96$.

identical to Michaelis-Menten kinetics in biochemistry, or the Monod equation in microbiology, corresponds to the assumption that predators are limited by their ability to handle the killed prey and process food. Thus, in the two-dim reduced model the nonlinearity of incidence rate can be interpreted as the necessity to spend time producing the free stage infective agents. It could be noteworthy that in some cases, a concave functional response of this type can arise as a consequence of a negative feedback [15].

To reduce the model order and to demonstrate the equivalence, we apply the technique of the time scales separation. The problem considered in this paper demonstrates that this technique should be applied with a certain degree of care. The reason for this is that, when this technique is applied, often only the very first approximation, referred to as the quasi-steady-state approximation or the limiting system and which is in fact the zero-order approximation, is obtained, while the higher order approximations are disregarded. A usual motivation for this is the simplicity and the transparency of such an approach. However, the quasi-steady-state approximation, which is in fact the zero-order approximation, can lead to a loss of many features of the original system. Thus, for model considered in this paper the zero-order approximation results in a two-dim system with the bilinear incidence rate, which is identical to the original Lotka-Volterra predator-prey model, is always globally asymptotically stable and hence does not admit the Hopf bifurcation. This example shows the importance of a more precise analysis. As a general rule, the more complex model behaviour is, the higher order of approximation the model requires.

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References

[1] R.M. Anderson and R.M. May, The population dynamics of microparasites and their invertebrate hosts. Phil. Proc. Roy. Soc. Lond. B **291** (1981), 451–524.

[2] E. Beretta and Y. Kuang, Convergence results in a well-known delayed predator-prey system. J. Math. Anal. Appl. **204** (1996), 840–853.

[3] E. Beretta and Y. Kuang, Modeling and analysis of a marine bacteriophage infection. Mathematical Biosciences **149** (1998), 57–76.

[4] T. Faria, Stability and bifurcation for a delay predatorprey model and the effect of diffusion. J. Math. Anal. Appl. **254** (2001), 433–463.

[5] S.J. Fraser, The steady state and equilibrium approximations: A geometric picture. J. Chem. Phys. **88** (1988), 4732–4738.

[6] X.-zh. He, Stability and delays in a predator-prey system. J. Math. Anal. Appl. **198** (1996), 355–370.

[7] A. Korobeinikov, Global properties of a general predator-prey model with non-symmetric attack and consumption rate. Discrete and Continuous Dynamical System B **14**(3) (2010), 1095–1103.

[8] Y. Kuang, Delay Differential Equations with Applications in Population Dynamics., Academic Press, Boston (1993).

[9] R.M. May, Time delay versus stability in population models with two and three trophic levels., Ecology **4** (1973), 315–325.

[10] M.P. Mortell, R.E. O'Malley, A. Pokrovskii and V.A. Sobolev (eds): Singular Perturbation and Hysteresis, SIAM, Philadelphia (2005).

[11] M. Rosenzweig, The paradox of enrichment. Science **171** (1971), 385–387.

[12] M.R. Roussel and S.J. Fraser, Geometry of of the steady–state approximation: Perturbation and accelerated convergence method. J. Chem. Phys. **93** (1990), 1072–1081.

[13] E. Shchepakina, V. Sobolev and M.P. Mortell, Singular Perturbations: Introduction to System Order Reduction Methods with Applications. Springer Lecture Notes in Mathematics, Vol. **2114**, Springer International Publishing, Switzerland (2014).

[14] V.V. Strygin and V.A. Sobolev, Effect of geometric and kinetic parameters and energy dissipation on orientation stability of satellites with double spin. Cosmic Research **14**(3) (1976), 331–335.

[15] C. Vargas-De-León and A. Korobeinikov, Global stability of a population dynamics model with inhibition and negative feedback. Math. Med. Biol. **30**(1) (2013), 65–72.

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Appendix: Iterative Method

Formally, the essence of the iterative method for the autonomous system with scalar variables

$$\begin{aligned} \dot{X} &= \zeta(X,\varepsilon) + F(X,\varepsilon)Y, \\ \varepsilon\dot{Y} &= \xi(X,\varepsilon) + G(X,\varepsilon)Y, \end{aligned} \tag{15}$$

is as follows. For the autonomous system (15) we have

$$\varepsilon\frac{\partial h}{\partial X}(\zeta + Fh) = \xi + Gh$$

and then

$$h = \frac{-\xi + \varepsilon\zeta h_X}{G - \varepsilon F h_X}.$$

This representation is used to organize the iterative process by the algorithm

$$\varphi^{(0)} = \phi(X) = -\frac{\xi}{G}, \quad \varphi^{(k)} = \frac{-\xi + \varepsilon\zeta\varphi_X^{(k-1)}}{G - \varepsilon F\varphi_X^{(k-1)}}, \quad k = 1, 2, 3, \dots ,$$

where $\varphi^{(k)}$ is considered as an approximation to h. It can be shown that

$$h(X,\varepsilon) - \varphi^{(k)} = O(\varepsilon^{k+1}).$$

The Fraser and Roussel [12] approach was extended to systems (15) with vector variables in [13]. We solve the equation

$$\varepsilon\frac{\partial h}{\partial X}(\zeta + Fh) = \xi + Gh$$

for the vector function $h(X,\varepsilon)$ and obtain

$$h = (G - \varepsilon F h_X)^{-1}(-\xi + \varepsilon\zeta h_X).$$

As in the scalar case, this formula is the basis for the iterative procedure

$$\varphi^{(0)} = -G^{-1}\xi, \quad \varphi^{(k)} = \left(G - \varepsilon F\varphi_X^{(k-1)}\right)^{-1}\left(-\xi + \varepsilon\zeta\varphi_X^{(k-1)}\right), \tag{16}$$

for the vector function $\varphi^{(k)}$, $k = 1, 2, 3\dots$ and the asymptotic relationship

$$\|h(X,\varepsilon) - \varphi^{(k)}\| = O(\varepsilon^{k+1})$$

holds.

Thus, for example,

$$\varphi^{(1)} = \left(G - \varepsilon F\varphi_X^{(0)}\right)^{-1}\left(-\xi + \varepsilon\zeta\varphi_X^{(0)}\right). \tag{17}$$

We will use the iterative method to construct the first approximation to the slow invariant manifold. For system (11) the functions in (15) have the form

$$X = \begin{pmatrix} x \\ y \end{pmatrix}, \quad \zeta(x,y,\varepsilon) = \begin{pmatrix} ax - bx(x+y) \\ -dy \end{pmatrix}, \quad F(x,y,\varepsilon) = \begin{pmatrix} -\alpha x \\ \alpha x \end{pmatrix},$$

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$$Y = v,\ \xi(x,y,\varepsilon) = \sigma y,\ G(x,y,\varepsilon) = -(m + \varepsilon\alpha x).$$

Then the zero–order approximation to the slow invariant manifold $v = \varphi^{(0)}(x,y)$ takes the form

$$v = \varphi^{(0)}(x,y) = -G^{-1}(x,y,0)g(x,y,0) = \frac{\sigma}{m}y,$$

and

$$\varphi_X^{(0)} = \begin{pmatrix} \dfrac{\partial\varphi^{(0)}}{\partial x} & \dfrac{\partial\varphi^{(0)}}{\partial y} \end{pmatrix} = \begin{pmatrix} 0 & \dfrac{\sigma}{m} \end{pmatrix}.$$

According to Eq. (17) for the first-order approximation we have

$$\begin{aligned}
\varphi^{(1)}(x,y) &= \left(G - \varepsilon F\phi_x^{(0)}\right)^{-1}(-\xi + \varepsilon\zeta\phi_x) \\
&= \left[-(m+\varepsilon\alpha x) - \varepsilon\begin{pmatrix} 0 & \dfrac{\sigma}{m} \end{pmatrix}\begin{pmatrix} -\alpha x \\ \alpha x \end{pmatrix}\right]^{-1} \\
&\quad \times \left[-\sigma y + \varepsilon\begin{pmatrix} 0 & \dfrac{\sigma}{m} \end{pmatrix}\begin{pmatrix} ax - bx(x+y) \\ -dy \end{pmatrix}\right] \\
&= \frac{\sigma(1+\varepsilon d/m)y}{m + \varepsilon\alpha(1+\sigma/m)x}.
\end{aligned}$$

This apparent contradiction is easily removed if instead of limiting system to consider the first-order approximation of the system on the integral manifold $v = \varphi^{(1)}(x,y)$:

$$\begin{aligned}
\dot{x} &= ax - bx(x+y) - \alpha x\varphi^{(1)}(x,y), \\
\dot{y} &= \alpha x\varphi^{(1)}(x,y) - dy.
\end{aligned} \tag{18}$$

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