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The influence of the multiplicity of infection upon the dynamics of a crop-pest–pathogen model with defence mechanisms

Hong Zhang [a], Paul Georgescu [b,*]

[a] Department of Financial Mathematics, Jiangsu University, ZhenJiang, JiangSu 212013, PR China
[b] Department of Mathematics, Technical University of Iaşi, Bd. Copou 11, 700506 Iaşi, Romania

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ABSTRACT

To prevent pest outbreaks, growers often resort to the release of insect-pathogenic viruses rather than to the use of pesticides, which threaten human health and adversely impact the environment. One of the unfortunate consequences of this approach, however, is the onset of resistance mechanisms in pests. In this paper, we propose a model which couples the analysis of the within-pest virus dynamics and the investigation of the evolutionary response of defence mechanisms. The ecosystem consisting in the soybean crop and its major pest, the fourth instar larvae of *Spodoptera litura*, which is infected by *Spodoptera polyhedrosis* viral particles, is used as our main example. Five types of resistance mechanisms are investigated, each expressing resistance through one or more model parameters. By means of linearization, phase field analysis and numerical simulations, we illustrate and analyze the stability of the steady states of our model. Moreover, we use a graph representing the values of the function of the lifetime reproductive success of the mutant in the resident pest population at equilibrium states to find which are the best and the worst pest control strategies. Our findings show the impact of evolutionary resistance on the selection for the multiplicity of infection of the within-pest virus, in the sense that to establish the viral infection there is a threshold of the mutant trait.

1. Introduction

Insect pests outbreaks, which threaten the supply of food and its quality, have always been major concerns for farming communities. Although chemical insecticides are easy to apply, their use may be detrimental in the long run for both humans and environment [1]. Biological controls, which represent an important alternative to chemical insecticides, can then be employed, leading to less adverse effects. Insects, like humans and other animals, can be infected by pathogenic organisms such as bacteria, viruses, fungi and protozoa [2] that either incapacitate them or interfere with their biological processes [3–9], being generally compatible with other natural enemies.

Baculoviruses are naturally occurring insect pathogens that can be used as biological control agents to control pest insects [10]. A field survey using the virus *Spodoptera polyhedrosis* against the fourth instar larvae of *Spodoptera litura* has been carried out by Prasad and Wadhwani [11] and is outlined as follows. The viral preparation contained 2×10^9 polyhedra per ml. Considering this as stock solution, four different dilutions were prepared and fed to the target pest population by leaf-dip method [12]. The polyhedra were taken up orally by the larvae together with the leaf. After ingestion, the polyhedra

* Corresponding author.
E-mail addresses: hongzhang@ujs.edu.cn (H. Zhang), v.p.georgescu@gmail.com (P. Georgescu).

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dissolved under the alkaline conditions in the midgut. The nucleocapsids were then released and consequently severely affected the midgut epithelial cells [13]. In the nucleus of these cells virus replication took place, budded viruses were produced in the late stages and then the occlusion bodies were produced in the final stage, the envelope being acquired from host cell nucleus and embedded in the matrix of occlusion body protein. These occlusion bodies were released when cells lysed to further spread baculovirus infection to next host. After infecting the larval midgut tissues, the budded virus particles entered the haemocoel, through which the virus invaded various tissues. Consequently, the histomicrograph clearly revealed that various midgut cells, fat bodies, connective tissues and integument either lost their identity or became highly disorganized [11].

The overall destruction of tissues led to the apparition of liquefied contents inside the body cavity, giving the infected insect a turgid appearance. The infected larval bodies were laden with polyhedral occlusion bodies (POBs) containing viral particles. Even a slight damage or disturbance of the integument releases liquefied body fluids containing large numbers of POBs. This infected fluids further spread infection when healthy larvae came in contact with it [14–16].

Given the rapid evolutionary changes in the pest population, it is not surprising that control agents are sometimes rendered ineffective, due to the natural evolutionary response to the environmental stress. This has been observed in [17–19] for Indian meal moth, *Plodia interpunctella*; American bollworm of cotton, *Heliothis virescens*; beet armyworm, *Spodoptera exigua*, and tobacco caterpillar, *Spodoptera litura*, which have all shown different degrees of resistance to *Bacillus thuringiensis* and insect viruses. Although significant efforts have been put into designing programmes of pesticide usage which avoid pesticide resistance, a more realistic goal is to delay the onset of resistance to pesticides, that is, to "mitigate resistance" [20].

Compared to vertebrates, insects do not possess the ability to produce antibodies against foreign antigens, and hence cannot release alpha/beta interferons in response to viral infections. Nevertheless, the defence mechanisms of insects can be classified into two broad groups. The first group consists of structural and passive barriers such as cuticle, gut physicochemical properties and peritrophic membrane, that is, of non-specific mechanisms. The second one consists of specific defence mechanisms, including cellular and humoral immunity, and inhibitors of apoptosis. Specifically, cellular responses involve phagocytosis, nodulation and encapsulation, humoral reactions include activation of prophenoloxidase (ProPO) cascade and induction of immune proteins like lysozymes, lectins and antibacterial and antifungal proteins [21,22], and inhibitors of apoptosis (IAP) are a family of functionally and structurally related proteins, which serve as endogenous inhibitors of programmed cell death.

In recent decades, besides investigations on the experimental and theoretical applications of microbial pathogens to suppress pests [4,23–28], many studies have attempted to formulate and analyze deterministic models of pest-pathogen dynamics [30–39], making use of analytic results to set up field experiments and to analyze their conclusions. To adequately incorporate the effects of the pathogens on the pest population, Anderson and May [32] investigated the pathogen–host model and showed that if the release rate exceeded a critical level, the host (pest) population would decrease to zero.

Resistance modeling usually takes one of the following two forms: (1) modeling the processes which take place within a single host, or (2) modeling the spread of resistance through a population of hosts [40]. However, both types are rarely combined, although exceptions do exist [41,42]. Modeling within-host bacterial dynamics or within-ecosystem dynamics in insect populations is usually accomplished using ordinary differential equations. Examples of models of this type include those of [43–46]. The models describing the spread of resistance factors through a population of hosts usually follow the SIS models; see, for instance, studies on the epidemiology of resistance spread [47,48]. Further, [42] explored both of the above situations in a model that incorporates within-host dynamics and tracks the average infection rates across hosts, pointing out that indiscriminate use of antibiotics greatly increases the rate of defence development. [41] provided another excellent modeling framework in which the direct interaction of hosts is considered, rather than interaction through an environmental pool, and found that resistance can be managed by cycling different antibiotics or using combinations of antibiotics.

On the other hand, of all physical parameters tested for in vitro baculovirus infection, the multiplicity of infection was found as being the most important factor to influence the total percentage of infected cells [49]. However, in the above-mentioned models, the authors ignored the effect of defence mechanisms upon the variation of the viral multiplicity of infection of cells within infected insects.

The aim of this paper is to introduce a model of a pest management strategy which accounts for defence responses depending upon a variable multiplicity of infection (MOI). The latter is understood as the number of viruses that are added per cell during infection [50], in response to pest control measures. In this regard, the MOI is highly relevant for understanding the virus dynamics and its evolution. Obviously, mixed genotype infections can occur only if the MOI is > 1, while recombination or complementation between different genotypes can occur only in mixed-genotype infected cells. Also, higher MOI raises the ploidy, partially masking deleterious variants. This reduces the deterministic forces by which these deleterious variants are removed by natural selection, maintaining a greater genetic diversity and thereby increasing the pool for recombination or complementation, possibly for longer periods of time [51,52]. In turn, rapid evolution during periods of high MOI may generate new viral strains. Lower ($\leqslant 1$) MOI levels relax selection between virus genotypes at the within-cell level, since virions rarely have to share a cell, increasing the competition at higher levels (within-tissue, within-host, between-host). That is, MOI is relevant not only for phenomena which occur at the cellular level, but also for the viral evolution at higher levels.

An extensive spatio-temporal monitoring of the cellular MOI of a eukaryotic virus, the *Cauliflower mosaic virus* (CaMV), from the onset of the systemic invasion until the senescence of its host plant, has been performed in [53], wide variations