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The global properties of an age-dependent *SI* model involving pathogenic virus release and defence mechanisms for pests

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ABSTRACT

Insect pests are common but undesirable elements in ecosystems and represent thorny problems for most developing countries. To prevent pest outbreaks, growers often resort to insect–pathogenic viruses rather than to pesticides which affect human health and the environment. The purpose of this paper is to investigate a new age-structured pest management model which describes the interaction between susceptible insect pests, infected insect pests, pathogenic viruses and defence immunity mechanisms. A feature of this model is that it accounts for the dependence of the amount of pathogenic viruses released and of the efficiency of the defence mechanisms upon the so-called age of infection. First, the asymptotic behavior of the system is established via a monotonicity argument which makes use of several integral inequalities, being shown that the infection ultimately dies out, while under certain circumstances the susceptible pests also become extinct. By means of the Michailov criterion, one then analyzes the linearized stability of the trivial equilibrium and of the semi-trivial infected pest-free equilibrium. In this regard, it is observed that the defence mechanisms and maximal length of the infective period play important roles in the dynamics of the system. Several pest controls strategies are further investigated by means of numerical simulations, which show that when the dose of pathogenic viruses released initially is larger than a certain amount the profile of the response of defence mechanisms can be modified by changing this dose. Finally, the paper is concluded with a discussion on the biological significance of the mathematical results and framework.

1. Introduction

Insect pests are common but undesirable elements in ecosystems and represent thorny problems for most developing countries where pest outbreaks threaten food quality and food supply. Pest control strategies were described occasionally in writings of the ancient Chinese, Sumerian, and Egyptian scholars. Predatory ants, for example, were used in China as early as 1200 BC to protect citrus groves from caterpillars and wood boring beetles.

Nowadays, to prevent pest outbreaks growers often resort to pesticides, but their use may have unwanted consequences including insect resistance, resurgence, outbreak of secondary pests, and the accumulation of pesticide residues affecting human health and the environment. Generally, insects, like humans and other animals, can be infected by insect–pathogenic organisms such as bacteria, viruses and fungi. Under appropriate conditions, like high humidity or high pest abundance,

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these naturally occurring organisms may multiply to cause disease outbreaks or epizootics that can decimate an insect population. As a result, a wise alternative to chemical control is microbial control which is, generally, man's use of suitably chosen living entomopathogenic organisms, referred as the microbial agents, to control another living organisms. Control agents can be bacteria, fungi, viruses, nematodes and protozoa that either remove the harmful organisms or interfere with their biological processes [1–8]. For example, the control of rabbit pests in Australia [9] by the viral disease called "myxomatosis" provides a spectacular example of a virus controlling pest.

With the development of biotechnology and the beginning of ecologically advisable policies for pest management, microbial control policies have achieved immense importance in agriculture protection programs. A large number of baculoviruses offer potential as microbial control agents of insects. Amongst all the viruses, the greatest microbial potential is displayed by the Baculoviridae Nuclear Polyhedrosis Virus (NPV) and Granulo Virus (GV) [10]. The occurrence of many different baculoviruses, each with sometimes very restricted but different host stages, and their great potential for the pest control are well recorded. The use of NPVs [11] is growing gradually in agricultural management, because NPVs are often host specific, cause autoinfection and are usually fatal.

In India, *Spodoptera litura* has defoliated many economically important crops including cole, cotton, and soybean. Synthetic pesticides have been used against this pest, but many farmers have found that heavy doses of pesticides cause pest resistance, resurgence, and food contamination. Recently, some entomologists have tried to use the Spodoptera polyhedrosis virus (SlNPV) to control the fourth instar larvae which is the most damaging stage of the pest. For example, some 2006 field surveys were outlined by Prasad and Wadhwani [12] as follows. The viral preparation contained 2×10^9 polyhedra per ml. Considering this as stock solution, four different dilutions (viz. 0.1, 0.25, 0.5 and 0.75 ml) were prepared and fed to the target pest population—the fourth instar larvae of S. litura by leaf-dip method, Castor leaf of 6 cm diameter was dipped in prepared concentrations for 1 min and shade-dried for 30 min. Then leaf discs were placed in a slanting position in separate containers so that the larvae can feed on both the surfaces of the leaf. Ten fourth instar larvae were released in each container with three replicas and one control. Consequently, the histomicrograph clearly revealed that various midgut cells, fat bodies, connective tissues and integument either lost their identity or became highly disorganized. Furthermore, they found out that at the maximal period of treatment, i.e. 96 h, the mortality increased from 5.43% to 78.91%, from the lowest dose of 0.1 ml to the highest applied dose of 0.75 ml. Above all, the overall destruction of tissues led to liquefied contents inside the body cavity, giving the infected insect a turgid appearance. The infected larval bodies were laden with polyhedral occlusion bodies (POBs) which contain viral particles. Even a slight damage or disturbance of the integument released liquefied body fluid containing large number of POBs. This infected fluid further spread infection when healthy larvae came in contact with the fluid, causing autoinfection.

On the other hand, as part of a survival strategy, more than 645 species of insect species have perfected numerous effective resistance and defence mechanisms to most of the conventional chemical insecticides (see Fig. 1). Similarly, can an insect species that is susceptible to a pathogen become resistant to more microorganisms? In previous mathematical works mentioned above, many authors neglected insect defence during the use of pathogenic viruses since the precise resistance mechanism remained unknown. From a biological viewpoint, the host specificity which has been observed in many insect pathogens demonstrates that insect species are naturally resistant to these microorganisms. For example, Pieris brassicae, Heliothis zea, Plodia interpunctella and Bombyx mori have shown different degrees of resistance to insect viruses [13] (see Table 1). Compared to vertebrates, insects do not possess the ability to produce antibodies and, further, not produce alpha/beta interferons in response to viral infections. Nevertheless, they are capable of "immune"reactions which are characterized by non-specific defence mechanisms consisting of structural and passive barriers like cuticle, gut physicochemical properties and peritrophic membrane, as well as specific defence mechanisms including phagocytosis, nodulation and encapsulation [14]. The above survey briefly describes the insect-virus-defence mechanism from a biological viewpoint. In the following some of its mathematical aspects will be described.

2. The mathematical model: Previous work and motivation

At present, there is a vast amount of literature on the applications of entomopathogens to suppress pests [15,9, 16,17,7,18]. Recently, many scholars have attempted to formulate and analyze deterministic models of pest–pathogen dynamics [19–26,37].

In their 1927–1933 series of papers, Kermack and McKendrick [27–29] studied the following *SI* model with general infectivity

$$\begin{cases} S'(t) = S(t) \displaystyle\int_0^\infty A(\tau) S'(\tau) \mathrm{d}\tau \\ I(t) = -\displaystyle\int_0^\infty \frac{A(\tau)}{A(0)} S'(t-\tau) \mathrm{d}\tau, \end{cases} \tag{1}$$

where $S(t)$ and $I(t)$ denote the amount of susceptible and infective pests at time t and $A(\tau)$ represents the expected infectivity of an individual that became infected τ units of time ago. See also Diekmann, Heesterbeek and Metz [30] for an overview of the results presented in [27–29]. Kermack and McKendrick's model is often simplified in the form

$$\begin{cases} S' = -\rho SI, \\ I' = \rho SI - wI, \end{cases} \tag{2}$$

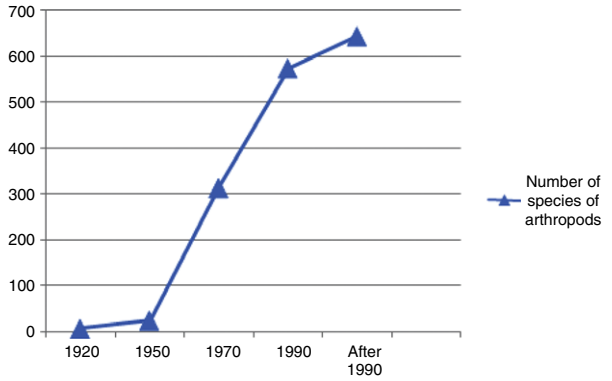

Fig. 1. Development of resistance by arthropods to insecticides.

Table 1
Insects showing resistance to insect–pathogenic viruses.

Species	Insect–pathogenic viruses
Pieris brassicae	GV
Heliothis zea	NPV
Plodia interpunctella	NPV
Bombyx mori	NPV

which is obtained from (1) for $A(\tau) = \rho e^{-w\tau}$, where w represents the total removal rate of infectives. Particularly, in 1980, Goh [25] studied (2) numerically, from the viewpoint of attempting to control a given pest population.

To adequately incorporate the effects of the pathogens on the pest population, in 1981, Anderson and May [20] used the system

$$\begin{cases} I' = \rho PS - (\theta + b + \mu)I, \\ H' = rH - \theta I, \\ P' = A - \omega I - (\gamma + \rho H)P, \end{cases} \tag{3}$$

where H represents the total host population $(S + I)$, P the pathogen population, ω the rate at which infective individuals produce pathogens, γ the mortality rate of the host population in the infective stage, A the release rate of the pathogen, b the death rate of the host if no pathogens are present, μ the host recovery rate and θ the pathogen-induced death rate. They showed that if the release rate A exceeded a critical level, the host (pest) population would decrease to zero.

Motivated by Anderson and May's modelling ideas, Brown [23] proposed and investigated a modified SIRS model developed as a framework for the study of epizootiological dynamics in an insect–pathogen system. From a linearized stability analysis, it has been shown that the system with one immune and one susceptible host class can exhibit stable, periodic or unstable behavior depending on model parameters. Furthermore, in 1997, Moerbeek and Van Den Bosch [26] developed stage-structured models of insect–pathogen interactions incorporating a constant maturation time which, from a mathematical point of view, introduces a delay in their systems. They pointed out that the interplay between insect-density dependence and stage-specific susceptibility has important consequences for the dynamics of insect–pathogen systems. Following this line of development, in 2008, Zhang et al. [31] dealt with an impulsively controlled epidemic model with delay, stage structure and a general form of the incidence rate. In [31], the pest population was subdivided into three subgroups: pest eggs, susceptible pests and infective pests, while being assumed that infective pests do not attack crops. The main results of [31] indicated that besides the release amount of infective pests, the incidence rate, time delay and impulsive period can have a great influence on the dynamics of the system. However, according to the comments made in Section 1, none of the models mentioned in Section 2 can completely characterize the interaction between insect pests, pathogenic viruses and the defence mechanism. More precisely, they cannot be employed to obtain precise quantitative assessments of the impact of the pathogenic virus density and infection age upon the spread of the infection and immunity in hosts for insect pest management.

Motivated by these comments and references, the aim of this paper is to construct a new model of pest management which involves the release of pathogenic viruses and defence immunity, and to investigate its dynamics. The model divides the pest population into a susceptible and an infective class, including also a class of pathogenic viruses as well as keeping track of the immune response, and uses an additional variable to describe the age of infection. Consequently, the model involves a transport type partial differential equation for infected hosts. The main results are obtained using by means of the linearized stability analysis of a functional differential equation which is equivalent to the original age-structured model. The use of Michailov criterion reveals the relationship between age structure, immune response and stability, specifically the

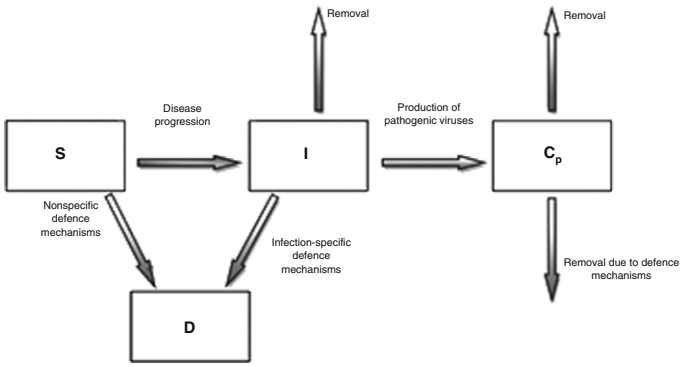

Fig. 2. Flow chart of infection spread via pathogenic viruses with defence immunity (S: susceptible insect pest population; I: infected insect pest population; C_p: pathogenic viruses; D: resistance mechanisms).

fact that the maximal length of the infective period and several coefficients characterizing defence immunity play important roles in the dynamics of the system.

The rest of the paper is organized as follows. The model to be studied is proposed in Section 3 together with its underlying biological assumptions, in a form which involves the use of a nonlinear partial differential equation of transport type. This model is then reformulated as a lower-dimensional functional differential equation. The influence of the viral particle self-regulation, age structure and immune response on the long term dynamics of the system is explored in Section 4. Several numerical simulations are performed in Section 5, a brief discussion of the biological significance of the mathematical results being given in Section 6.

3. Infection-age-structured model

As previously mentioned, we attempt to introduce a pest-virus-immune response model based on those of Goh [25], Anderson and May [20] and Anderson [21], which describes the dynamics of a host population that is infected by a disease possessing a free-living stage in the environment. Like those models, it includes time-dependent state variables that represent the densities of susceptible hosts $S(t)$, of infected hosts $I(t, a)$ and of the pathogenic viruses $C_p(t)$ in infected host organisms, the variable a describing the so-called age of infection. Also, the immune response denoted by $D(t)$ includes non-specific and specific defence mechanisms. The general scheme of the transitions between different pest states is presented in Fig. 2.

Using Fig. 2 as a guide for different interactions we can formulate the governing equations as follows

$$\begin{cases} S'(t) = -\dfrac{\alpha_1 C_p(t) S(t)}{1 + k_1 S(t) + k_2 C_p(t)}, \quad t > 0 \\ \dfrac{\partial I(t,a)}{\partial t} + \dfrac{\partial I(t,a)}{\partial a} = -d_I(a) I(t,a), \quad t, a > 0 \\ C_p'(t) = -d_{C_p} C_p(t) + \displaystyle\int_0^h \beta(a) I(t,a)\mathrm{d}a - \dfrac{\alpha_2 C_p(t) S(t)}{1 + k_1 S(t) + k_2 C_p(t)} - \dfrac{\alpha_3 C_p(t) D(t)}{1 + k_3 D(t) + k_4 C_p(t)}, \quad t, a > 0 \\ D'(t) = \kappa S(t) - d_D D(t) + \displaystyle\int_0^h \gamma(a) I(t,a)\mathrm{d}a - \dfrac{\alpha_4 C_p(t) D(t)}{1 + k_3 D(t) + k_4 C_p(t)}, \quad t, a > 0 \end{cases} \tag{4}$$

with the following boundary condition

$$I(t, 0) = \frac{\alpha_1 C_p(t) S(t)}{1 + k_1 S(t) + k_2 C_p(t)}, \quad t > 0 \tag{5}$$

and initial conditions

$$S(0) = S_0, \qquad C_p(0) = C_{p_0}, \qquad D(0) = D_0 \tag{6}$$

$$I(0, a) = I_0(a), \quad a \geq 0. \tag{7}$$

Here, d_{C_p}, $d_I(a)$ and d_D represent the fractions removed per unit of time of the total amount of pathogenic viruses, infected hosts of age of infection a and resistance mechanisms, respectively. While the precise infection and defence mechanisms remain unknown, we are motivated to use the particular equations mentioned above by the specific forms of the functional responses of population dynamics. In this regard, we assume that α_1 is the fraction of susceptible pests converted to infected pests per viral cell and unit of time, α_2 and α_3 represent the fraction of pathogenic viruses consumed per susceptible pest and, respectively, per immune response at time t, k_1 and k_3 are nonnegative constants describing the effect of handling time

of the susceptible pest on the incidence rate of infection and, respectively, on the immune rate, k_2 and k_4 are also nonnegative constants representing the magnitude of the interference of pathogen cells in host organisms under infection and defence mechanisms, respectively.

Note that the infection rate

$$-\frac{\alpha_1 C_p(t)S(t)}{1+k_1S(t)+k_2C_p(t)},$$

degenerates into the bilinear incidence rate if $k_1 = k_2 = 0$, and also degenerates into saturated incidence rates if $k_1 = 0$ or $k_2 = 0$. Similar things happen to the infection rate

$$-\frac{\alpha_3 C_p(t)D(t)}{1+k_3D(t)+k_4C_p(t)}.$$

The following assumptions are also made to derive the mathematical model above:

- (A_1) Susceptible hosts, apart from their initial amounts, are not capable of reproduce once they are exposed to pathogenic organisms. No natural mortality of susceptible hosts is considered.
- (A_2) Infected hosts are parameterized by their age of infection, i.e., the time elapsed since the pest became infected, which is denoted by a. Pests of different age of infection may release pathogenic viruses at different rates.
- (A_3) Pathogenic viruses are degraded at a rate proportionate to their amount. Moreover, it is acceptable to consider independent rates for viral particles consumed for infection of pests and for viral particles degraded by defence mechanismsdepending on the infection age a.
- (A_4) The constants α_i $(i = 1, 2)$, d_{C_p} as well as d_D are all positive. The infected hosts mortality function d_I is dependent on infection age a, continuous, nonnegative and satisfies $\lim_{a\to h-}\int_0^a d_I(s)\mathrm{d}s = +\infty$ for some $h > 0$, which is understood as being the maximal age of infection. New pathogenic viruses are produced by infected host only in the bounded interval of ages of infection $[0, h)$, which leads to $supp\beta(\cdot) \subset [0, h)$, where $\beta(a)$ is the release rate of pathogenic viruses for an infected host of infection age a, and $I(t, a) = 0$ for $a \geq h$.
- (A_5) The function γ, which gives the production rate of defence immunity mechanisms by an infected host is continuous, nonnegative and $supp\gamma(\cdot) \subset [0, h)$. In other words, the effective defence mechanisms are observed in bodies of living infected hosts. On the other hand, excluding the effect of viral particles, the susceptible hosts are still capable of non-specific defence immunity. It is then safe to assume that the immune mechanisms also grow at a source rate κS.
- (A_6) Infective pests cannot attack crops.

Also, the initial data S_0, C_{p_0}, D_0, $I_0(\cdot)$ are assumed to be nonnegative.

Remark 3.1. In view of the assumption that $[0, h)$ is the maximal interval of infection age, one notes that the reasonable domain of function d_I is the same interval. This implies that infected hosts die before reaching the maximal infection age h.

Note also that the exponential $s(a) = \mathrm{e}^{-\int_0^a d_I(s)\mathrm{d}s}$ gives the fraction of a cohort infected at the same time which survives until their infection age is a, so it may be called a survival function. In Anderson and May's terms (see [32]), our mortality function is a modified type I mortality, in the sense that it is not assumed that d_I is 0 until the infection age h is reached.

Remark 3.2. The quantities S_0 and C_{p_0} represent the initial amount of target pests and, respectively, viral particles released by farmers. Also, D_0 describes the initial magnitude of the immune response, while I_0 represents the initial age distribution of the infected population.

To analyze the nonlinear PDE model introduced above, we first derive from (4), (5) and (7) that $I(t, a)$ is given by

$$I(t,a) = \begin{cases} \dfrac{\alpha_1 C_p(t-a)S(t-a)}{1+k_1S(t-a)+k_2C_p(t-a)}\mathrm{e}^{-\int_0^a d_I(\xi)\mathrm{d}\xi}, & \text{for } t > a \geq 0; \\ I_0(a-t)\mathrm{e}^{-\int_{a-t}^a d_I(\xi)\mathrm{d}\xi}, & \text{for } a \geq t > 0. \end{cases} \tag{8}$$

From the basic existence and uniqueness theory for functional differential equations (see [33,34]), it follows that the integrodifferential system consisting in the first, third and fourth equation of (4) in which $I(t, a)$ is substituted with its expression given in (8) has a unique global solution. In the following, we shall be concerned with the positivity and the asymptotic behavior of this solution.

4. The dynamical behavior of the pest–pathogen model

The key questions are, of course, whether or not the size of the susceptible pest class will be stabilized under a certain economically significant threshold level and whether or not the pathogenic viruses and resistance mechanisms will be active in the long term. In other words, we should determine the asymptotic behavior of the system for $t \to \infty$, which is what we attempt to do throughout this section.

4.1. Asymptotic behavior

Define the total size of the pest population at time t as

$$N(t) \doteq S(t) + N_I(t),$$

where the total size $N_I(t)$ of the infected pest population at time t is given by

$$N_I(t) = \int_0^h I(t, a)\mathrm{d}a.$$

The following theorem establishes the positivity of the solutions of (4), affirming also that the infection is removed in the long term.

Theorem 4.1. *Assume that all initial conditions are nonnegative. Then*

(i) *S, I, C_p, D, N_I and N are nonnegative.*
(ii) *N and S are nonincreasing.*
(iii) *N, S, N_I and $\int_0^t \left(\int_0^h d_I(a)I(s, a)\mathrm{d}a\right)\mathrm{d}s$ are bounded.*
(iv) *$\int_0^t N_I(s)\mathrm{d}s$ is bounded and N_I tends to 0 as $t \to \infty$.*

Proof. First, integrating the first equation of (4) on $[0, t]$, we get

$$S(t) = S_0 \mathrm{e}^{-\alpha_1 \int_0^t \frac{C_p(s)}{1+k_1S(s)+k_2C_p(s)}\mathrm{d}s}, \tag{9}$$

which implies that $S(t)$ is nonnegative since S_0 is nonnegative.

It is seen from (8) that I is nonnegative, which implies that N_I and N are nonnegative. Also,

$$\frac{\mathrm{d}}{\mathrm{d}t}\left[C_p(t)\mathrm{e}^{\int_0^t \left(d_{C_p} + \frac{\alpha_2 S(s)}{1+k_1S(s)+k_2C_p(s)} + \frac{\alpha_3 D(s)}{1+k_3D(s)+k_4C_p(s)}\right)\mathrm{d}s}\right] \geq 0$$

and consequently $t \to C_p(t)\mathrm{e}^{\int_0^t \left(d_{C_p} + \frac{\alpha_2 S(s)}{1+k_1S(s)+k_2C_p(s)} + \frac{\alpha_3 D(s)}{1+k_3D(s)+k_4C_p(s)}\right)\mathrm{d}s}$ is increasing, which implies that C_p is nonnegative on its interval of existence. In addition, it follows from the nonnegativity property of S and C_P that S is nonincreasing. Clearly,

$$\frac{\mathrm{d}}{\mathrm{d}t}\left[D(t)\mathrm{e}^{\int_0^t \left(d_D + \frac{\alpha_4 C_p(s)}{1+k_3D(s)+k_4C_p(s)}\right)\mathrm{d}s}\right] \geq 0,$$

which implies that D is nonnegative. One notes that

$$\begin{aligned} N_I'(t) &= \frac{\mathrm{d}}{\mathrm{d}t}\left(\int_0^h I(t, a)\mathrm{d}a\right) \\ &= -\int_0^h d_I(a)I(t, a)\mathrm{d}a - \int_0^h \frac{\partial I(t, a)}{\partial a}\mathrm{d}a \\ &= -\int_0^h d_I(a)I(t, a)\mathrm{d}a + \frac{\alpha_1 C_p(t)S(t)}{1 + k_1S(t) + k_2C_p(t)} \\ &= -\int_0^h d_I(a)I(t, a)\mathrm{d}a - S'(t). \end{aligned} \tag{10}$$

From (A_4), the first equation of (4) and (10), we obtain that

$$N'(t) = -\int_0^h d_I(a)I(t, a)\mathrm{d}a \leq 0, \tag{11}$$

which implies that N is nonincreasing. Integrating both sides of (11) on $[0, t]$, we then derive

$$S(t) + N_I(t) + \int_0^t \left(\int_0^h d_I(a)I(s, a)\mathrm{d}a\right)\mathrm{d}s = S_0 + N_I(0), \tag{12}$$

from which the boundedness of S, N_I, N and of

$$\int_0^t \left(\int_0^h d_I(a)I(s, a)\mathrm{d}a\right)\mathrm{d}s$$

follows. To show that $\int_0^t N_I(s)\mathrm{d}s$ is bounded, note first that

$$\int_0^{h-a} d_I(\xi+a)\mathrm{e}^{-\int_a^{a+\xi} d_I(\tau)\mathrm{d}\tau}\mathrm{d}\xi = \int_0^{h-a} -\frac{\mathrm{d}}{\mathrm{d}\xi}\left(\mathrm{e}^{-\int_a^{a+\xi} d_I(\tau)\mathrm{d}\tau}\right)\mathrm{d}\xi = 1. \tag{13}$$

It then follows that

$$\begin{aligned}\int_0^t\int_0^h I(s,a)\mathrm{d}a\mathrm{d}s &= \int_0^t\int_0^h\left(\int_0^{h-a} d_I(\xi+a)\mathrm{e}^{-\int_a^{a+\xi} d_I(\tau)\mathrm{d}\tau} I(s,a)\mathrm{d}\xi\right)\mathrm{d}a\mathrm{d}s\\ &= \int_0^t\int_0^h\left(\int_0^{h-\xi} d_I(\xi+a)I(s+\xi,a+\xi)\mathrm{d}a\right)\mathrm{d}\xi\mathrm{d}s.\end{aligned}$$

With the change of variables given by $a' = a+\xi, s' = s+\xi$, one obtains that

$$\begin{aligned}\int_0^t\int_0^h I(s,a)\mathrm{d}a\mathrm{d}s &= \int_0^t\int_0^h\left(\int_\xi^h d_I(a')I(s+\xi,a')\mathrm{d}a'\right)\mathrm{d}\xi\mathrm{d}s\\ &= \int_0^h\left(\int_\xi^{t+\xi}\left(\int_\xi^h d_I(a')I(s',a')\mathrm{d}a'\right)\mathrm{d}s'\right)\mathrm{d}\xi\\ &\le h\left(\int_0^{t+h}\int_0^h d_I(a')I(s',a')\mathrm{d}a'\mathrm{d}s'\right).\end{aligned}$$

Consequently, $\int_0^t N_I(s)\mathrm{d}s$ is bounded. Also, since $N_I = N - S$ and N, S are nonincreasing, it follows that N_I has a limit as $t\to\infty$. Since $\int_0^t N_I(s)\mathrm{d}s$ is bounded, it is then seen that N_I tends to 0 as $t\to\infty$. This completes the proof. □

Naturally, as seen in the above result, the size of the susceptible pest population is expected to decrease, since there is no source term for susceptibles in the first equation of (4). Also, a disease with finite maximal age of infection cannot persist in a dwindling population.

Remark 4.2. The term $\int_0^t\left(\int_0^h d_I(a)I(s,a)\mathrm{d}a\right)\mathrm{d}s$ gives the total removed amount of infected hosts of all infection ages during the time interval $[0,t]$.

Next, to facilitate the further analysis of asymptotical behavior, we shall construct a connection between the release rate of new pathogenic viruses, the degradation rate of infected pests and the production rate of resistance mechanisms for an infected host of infection age a.

Lemma 4.3. *Suppose that there exists a nonnegative integrable function g defined on $[0,h)$ for the given h such that for every $a\in[0,h)$*

$$\beta(a) \le \int_0^{h-a} g(\xi)d_I(\xi+a)\mathrm{e}^{-\int_a^{a+\xi} d_I(\tau)\mathrm{d}\tau}\mathrm{d}\xi. \tag{14}$$

Then

$$\int_0^t\int_0^h \beta(a)I(s,a)\mathrm{d}a\mathrm{d}s \le \left(\int_0^h g(\xi)\mathrm{d}\xi\right)\left(\int_0^{t+h}\int_0^h d_I(a)I(s,a)\mathrm{d}a\mathrm{d}s\right). \tag{15}$$

The proof of this lemma is given in the Appendix.

Remark 4.4. Since $\int_0^{h-a} d_I(\xi+a)\mathrm{e}^{-\int_a^{a+\xi} d_I(\tau)\mathrm{d}\tau}\mathrm{d}\xi = 1$, it follows that (14) is satisfied with $g = C$ whenever β is bounded. Consequently, we may call (14) in what follows a quasi-boundedness condition. We also note that $a\to\int_0^{h-a} g(\xi)d_I(\xi+a)\mathrm{e}^{-\int_a^{a+\xi} d_I(\tau)\mathrm{d}\tau}\mathrm{d}\xi$ is a boundedness function which depends on the (weighted) total net loss rate of infected pests in $[a,h)$. Consequently, the meaning of (14) is that the release rate of pathogen viruses produced by an infected host of age of infection a is less than the weighted net loss rate of infected pests in $[a,h)$.

We are now ready to complement Theorem 4.1 with a result which describes the asymptotic behavior of S and C_p provided that the release rate of pathogenic viruses β is quasi-bounded. The quasi-boundedness assumption tames the force of infection β and it is natural to expect that the susceptibles will survive in the long term.

Theorem 4.5. *Assume that condition (14) holds. Then*

(i) *C_p and $\int_0^t C_p(s)\mathrm{d}s$ are bounded.*
(ii) *S tends to a positive limit S^* as $t\to\infty$ provided that $S_0 > 0$.*
(iii) *D is positive.*

Proof. By integrating the third equation of (4) from 0 to t, one obtains that

$$C_p(t) + d_{C_p}\int_0^t C_p(s)\mathrm{d}s + \alpha_2\int_0^t \frac{C_p(s)S(s)}{1+k_1S(s)+k_2C_p(s)}\mathrm{d}s + \alpha_3\int_0^t \frac{C_p(s)D(s)}{1+k_3D(s)+k_4C_p(s)}\mathrm{d}s$$
$$= C_{p_0} + \int_0^t\int_0^h \beta(a)I(s,a)\mathrm{d}a\mathrm{d}s, \tag{16}$$

which implies that

$$\int_0^t C_p(s)\mathrm{d}s \le \frac{C_{p_0}}{d_{C_p}} + \frac{1}{d_{C_p}}\int_0^t\int_0^h \beta(a)I(s,a)\mathrm{d}a\mathrm{d}s. \tag{17}$$

From Lemma 4.3 and the boundedness of $\int_0^t\int_0^h d_I(a)I(s,a)\mathrm{d}a\mathrm{d}s$, it is then concluded that $\int_0^t C_p(s)\mathrm{d}s$ is bounded. Furthermore, since S is decreasing and $\int_0^t \frac{C_p(s)}{1+k_1S(s)+k_2C_p(s)}\mathrm{d}s$ is also bounded, it follows from (9) that $S(t)$ tends to a positive constant S^* as $t\to\infty$. It is then observed that

$$D'(t) \ge \kappa S^* + \int_0^h \gamma(a)I(t,a)\mathrm{d}a - \left(d_D + \frac{\alpha_4}{k_4}\right)D(t)$$
$$> \int_0^h \gamma(a)I(t,a)\mathrm{d}a - \left(d_D + \frac{\alpha_4}{k_4}\right)D(t). \tag{18}$$

It is easily seen that

$$D(t) > D_0\mathrm{e}^{-(d_D+\frac{\alpha_4}{k_4})t} + \mathrm{e}^{-(d_D+\frac{\alpha_4}{k_4})\theta t}\int_0^t\int_0^h \gamma(a)I(t,a)\mathrm{d}a$$

for some $\theta\in(0,1)$ and all $t\in\mathbb{R}_+$, from which we can obtain the positivity of D. □

Remark 4.6. In particular, it follows from Remark 4.4 and Theorem 4.5 that if β is bounded, then C_p and $\int_0^t C_p(s)\mathrm{d}s$ are bounded, while S tends to a positive limit S^* as $t\to\infty$ provided that $S_0 > 0$.

Remark 4.7. From Theorem 4.5, it is seen that if the release rate of pathogenic viruses is exceeded by the weighted net loss of infected pests, then the susceptible pest population tends to a positive equilibrium S^*. As seen from (9), the success of our control strategy is then conditioned by the initial size of the susceptible pest class S_0 and by the value of α_1. In this regard, it is observed that the immune response is uniformly persistent if the susceptibles also persist uniformly.

Let

$$\Gamma_0(0) \doteq \nu(0) + \frac{\alpha_2}{\alpha_1}N_I(0)$$

and

$$\nu(t) \doteq C_p(t) - \frac{\alpha_3}{\alpha_4}D(t).$$

Next we shall consider the case of $d_{C_p} = \kappa \equiv 0$. The following result provides an estimation of the auxiliary function ν as $t\to\infty$.

Lemma 4.8. *Assume that $d_{C_p} = \kappa \equiv 0$. Further, assume that*

$$\beta(a) \ge \frac{\alpha_2}{\alpha_1}d_I(a) + \frac{\alpha_3}{\alpha_4}\gamma(a) \tag{19}$$

Then $\liminf_{t\to\infty}\nu(t) \ge \Gamma_0(0)$.

Proof. Indeed, by the third and fourth equations of (4) and (10), one obtains that

$$\nu(t) - \nu(0) = \int_0^t\int_0^h \left(\beta(a) - \frac{\alpha_2}{\alpha_1}d_I(a) - \frac{\alpha_3}{\alpha_4}\gamma(a)\right)I(s,a)\mathrm{d}a\mathrm{d}s + \frac{\alpha_2}{\alpha_1}(N_I(0) - N_I(t)) + \frac{\alpha_3}{\alpha_4}\int_0^t d_D D(s)\mathrm{d}s. \tag{20}$$

Then

$$\nu(t) = \Gamma_0(0) - \frac{\alpha_2}{\alpha_1}N_I(t) + \frac{\alpha_3}{\alpha_4}\int_0^t d_D D(s)\mathrm{d}s + \int_0^t\int_0^h \left(\beta(a) - \frac{\alpha_2}{\alpha_1}d_I(a) - \frac{\alpha_3}{\alpha_4}\gamma(a)\right)I(s,a)\mathrm{d}a\mathrm{d}s. \tag{21}$$

It follows from Theorem 4.1 and (21) that $\liminf_{t\to\infty}\nu(t) \ge \Gamma_0(0)$. This completes the proof. □

Theorem 4.9. *Assume that $d_{C_p} = \kappa \equiv 0$ and (19) holds, together with the following condition*

$$\Gamma_0(0) > 0. \tag{22}$$

Then $S(t)$ tends to 0 as $t \to \infty$.

Proof. It follows from Lemma 4.8, (19) and the nonnegativity of $D(t)$ that $S(t)$ tends to zero as $t \to \infty$. This completes the proof. □

Remark 4.10. The product terms $\frac{\alpha_2}{\alpha_1} d_I(a)$ and $\frac{\alpha_3}{\alpha_4}\gamma(a)$ approximate the practical removal efficiency and practical immune efficiency, respectively, when the infection age of infected pests is a. In this regard, Theorem 4.9 can be interpreted as if there is no non-specific defence immunity, the pathogenic viruses are not naturally degraded and are produced fast enough, compensating the loss due to the immune response and to the removal of infected pests, and the initial density of viral particles is not less than a certain value depending on S_0, D_0 and $N_I(0)$, then the susceptible pest population S will become extinct. Note that condition (19) is an "unboundedness" condition for β, as seen, for instance, from the fact that if (19) is satisfied, then $\int_0^h \beta(a)\mathrm{d}a = +\infty$.

4.2. Linearized stability

Let us first observe that the solutions of (4) satisfy the following integrodifferential system for $t > h$, as in this situation t is larger than all possible infection ages and consequently $I(t, a)$ is always given by the first half of (8)

$$\begin{cases} S'(t) = -\dfrac{\alpha_1 C_p(t) S(t)}{1 + k_1 S(t) + k_2 C_p(t)}, \\ C_p'(t) = -d_{C_p} C_p(t) + \displaystyle\int_0^h \frac{A(a) C_p(t-a) S(t-a)}{1 + k_1 S(t-a) + k_2 C_p(t-a)}\mathrm{d}a - \frac{\alpha_2 C_p(t) S(t)}{1 + k_1 S(t) + k_2 C_p(t)} - \frac{\alpha_3 C_p(t) D(t)}{1 + k_3 D(t) + k_4 C_p(t)}, \\ D'(t) = \kappa S(t) - d_D D(t) + \displaystyle\int_0^h \frac{B(a) C_p(t-a) S(t-a)}{1 + k_1 S(t-a) + k_2 C_p(t-a)}\mathrm{d}a - \frac{\alpha_4 C_p(t) D(t)}{1 + k_3 D(t) + k_4 C_p(t)}. \end{cases} \tag{23}$$

Let us also denote

$$A(a) = \alpha_1 \beta(a) \mathrm{e}^{-\int_0^a d_I(\xi)\mathrm{d}\xi}$$

and

$$B(a) = \alpha_1 \gamma(a) \mathrm{e}^{-\int_0^a d_I(\xi)\mathrm{d}\xi}.$$

We consider two critical states: the pest-free state and the initial state, in which all pests are susceptible. Mathematically, we obtain two equilibria, i.e., a trivial equilibrium $E_0 = (0, 0, 0)$ and a semi-trivial infected pest-free equilibrium $E_1 = (\overline{S}, 0, \frac{\kappa \overline{S}}{d_D})$, $\overline{S} = S_0$, although it should be noted that the semi-trivial equilibrium is not unique, as $(\overline{S}, 0, \frac{\kappa \overline{S}}{d_D})$ is an equilibrium for all $\overline{S} > 0$. Obviously, this precludes any kind of asymptotic stability for both E_0 and E_1.

Remark 4.11. $\frac{\kappa \overline{S}}{d_D}$ describes the initial density of the non-specific defence immunity, which includes the inherent resistance mechanisms of cuticle, peritrophic membrane, etc.

The characteristic equations at E_0 and E_1 have the following forms

$$\det \begin{pmatrix} \lambda & 0 & 0 \\ 0 & \lambda + d_{C_p} & 0 \\ -\kappa & 0 & \lambda + d_D \end{pmatrix} = 0$$

and, respectively,

$$\det \begin{pmatrix} \lambda & \dfrac{\alpha_1 \overline{S}}{1 + k_1 \overline{S}} & 0 \\ 0 & \lambda + d_{C_p} + \dfrac{\alpha_2 \overline{S}}{1 + k_1 \overline{S}} + \dfrac{\alpha_3 \kappa \overline{S}}{d_D + k_3 \kappa \overline{S}} - \dfrac{\overline{S}}{1 + k_1 \overline{S}} \displaystyle\int_0^h A(a) \mathrm{e}^{-\lambda a}\mathrm{d}a & 0 \\ -\kappa & \dfrac{\alpha_4 \kappa \overline{S}}{d_D + k_3 \kappa \overline{S}} - \dfrac{\overline{S}}{1 + k_1 \overline{S}} \displaystyle\int_0^h B(a) \mathrm{e}^{-\lambda a}\mathrm{d}a & \lambda + d_D \end{pmatrix} = 0.$$

For the trivial equilibrium E_0, one obtains the non-positive eigenvalues 0, $-d_{C_p}$ and $-d_D$. For the infected pest-free equilibrium E_1, one obtains the eigenvalues 0 and $-d_D$, the remaining eigenvalues being determined by the roots of the following equation

$$\lambda + d_{C_p} + \frac{\alpha_2 \overline{S}}{1 + k_1 \overline{S}} + \frac{\alpha_3 \kappa \overline{S}}{d_D + k_3 \kappa \overline{S}} - \frac{\overline{S}}{1 + k_1 \overline{S}} \int_0^h A(a) \mathrm{e}^{-\lambda a}\mathrm{d}a = 0. \tag{24}$$

It is then seen that E_0 has a two-dimensional stable manifold and a one-dimensional center manifold. Also, E_1 has an at least one-dimensional stable manifold and an at least one-dimensional center manifold, while in certain situations it also has a one-dimensional unstable manifold. Further details regarding the stability of the equilibria are given below; unfortunately, we have not been able to compute the center manifold neither for E_0 nor for E_1, due to the complexity of (23). See Khalil [35] or Coddington and Levinson [36] for further details regarding center manifold theory for continuous-time systems.

The following preliminary result is taken into account by the argument principle known as Michailov criterion.

Lemma 4.12. *Let*

$$\Phi(\lambda) = \lambda + d_{C_p} + \frac{\alpha_2\overline{S}}{1+k_1\overline{S}} + \frac{\alpha_3\kappa\overline{S}}{d_D+k_3\kappa\overline{S}} - \frac{\overline{S}}{1+k_1\overline{S}}\int_0^h A(a)\mathrm{e}^{-\lambda a}\mathrm{d}a$$

and let $\Delta_C \operatorname{Arg}\Phi(\lambda)$ denote the change of the argument of a curve Φ in C considering windings and orientation. Also, let Δ be the variation of the argument for $\gamma(\mu)$(noted by $\operatorname{Arg}\gamma(\mu)$) as μ varies from 0 to ∞ for a curve γ in C, i.e.,

$$\Delta = \Delta_{\mu\in[0,\infty)}\operatorname{Arg}\gamma(\mu). \tag{25}$$

Suppose that

(a) (24) *has no pure imaginary roots;*
(b) *there exists a positive constant k^* such that $|\lambda| < k^*$ for all complex roots of (24) with* $\mathrm{Re}\lambda > 0$.

Then the number of roots in the right half-plane equals $\frac{1}{2} - \frac{\Delta}{\pi}$.

The proof of this lemma is given in the Appendix. To apply Lemma 4.12, we shall prove that $\Phi(\lambda)$ satisfies conditions (a) and (b) in Lemma 4.12.

Lemma 4.13. *Suppose that either of the following conditions hold*

(a) $d_{C_p} + \frac{\alpha_2\overline{S}}{1+k_1\overline{S}} + \frac{\alpha_3\kappa\overline{S}}{d_D+k_3\kappa\overline{S}} > \frac{\overline{S}}{1+k_1\overline{S}}\int_0^h A(a)\mathrm{d}a$;
(b) $d_{C_p} + \frac{\alpha_2\overline{S}}{1+k_1\overline{S}} + \frac{\alpha_3\kappa\overline{S}}{d_D+k_3\kappa\overline{S}} \neq \frac{\overline{S}}{1+k_1\overline{S}}\int_0^h A(a)\mathrm{d}a$ *and* $\frac{\overline{S}}{1+k_1\overline{S}}\int_0^h A(a)\mathrm{d}a < \pi$.

Then Φ has no zeros on the imaginary axis.

Proof. Indeed, $\Phi(\mathrm{i}\omega) = 0\ (\omega \neq 0)$ if and only if $\mathrm{Re}\,\Phi(\mathrm{i}\omega) = 0$ and $\mathrm{Im}\,\Phi(\mathrm{i}\omega) = 0$, that is,

$$\begin{cases} d_{C_p} + \dfrac{\alpha_2\overline{S}}{1+k_1\overline{S}} + \dfrac{\alpha_3\kappa\overline{S}}{d_D+k_3\kappa\overline{S}} - \dfrac{\overline{S}}{1+k_1\overline{S}}\displaystyle\int_0^h A(a)\cos\omega a\mathrm{d}a = 0, \\ \omega + \dfrac{\overline{S}}{1+k_1\overline{S}}\displaystyle\int_0^h A(a)\sin\omega a\mathrm{d}a = 0. \end{cases}$$

Condition (a) guarantees that $\mathrm{Re}\,\Phi(\mathrm{i}\omega) > 0$, so clearly $\Phi(\mathrm{i}\omega) \neq 0$. The second condition of condition (b) ensures that $\Phi(0) \neq 0$. It is clear that $\mathrm{Im}\,\Phi(\mathrm{i}\omega) > 0$ for $\omega \in (0, \pi]$. In addition, for $\omega > \pi$, the second condition also guarantees that $\mathrm{Im}\,\Phi(\mathrm{i}\omega) > 0$. Hence, we have proved that $\Phi(\mathrm{i}\omega) \neq 0$ for all $\omega > 0$. As a result, we conclude that $\Phi(\lambda)$ has no zeros on the imaginary axis since zeros of $\Phi(\lambda)$ come in complex conjugate pairs. This completes the proof. □

Lemma 4.14. *There exists a positive constant k^* such that $|\lambda| < k^*$ for all complex roots of (24) with* $\mathrm{Re}\lambda > 0$.

Proof. Letting $\lambda = x + yi (x > 0)$ in (24), we have

$$\begin{cases} x + d_{C_p} + \alpha_2\dfrac{\overline{S}}{1+k_1\overline{S}} + \dfrac{\alpha_3\kappa\overline{S}}{d_D+k_3\kappa\overline{S}} - \dfrac{\overline{S}}{1+k_1\overline{S}}\displaystyle\int_0^h \big(A(a)\mathrm{e}^{-xa}\cos ay\big)\mathrm{d}a = 0, \\ y + \dfrac{\overline{S}}{1+k_1\overline{S}}\displaystyle\int_0^h \big(A(a)\mathrm{e}^{-xa}\sin ay\big)\mathrm{d}a = 0. \end{cases}$$

We may choose k^* as

$$k^* = 2\left(d_{C_p} + \alpha_2\overline{S} + \frac{\alpha_3}{k_3} + \overline{S}\int_0^h A(a)\mathrm{d}a\right),$$

which completes the proof. □

According to Lemmas 4.12–4.14, we may then deduce the following result.

Table 2
The stable and unstable manifolds for the linearized system at the equilibrium ($\overline{S} > 0, 0, \overline{D} > 0$).

h	Two-dimensional stable manifold	One-dimensional unstable manifold
All ($\theta^* \geq \theta_2$)	Yes	
All ($\theta_1 \geq \theta_2$)	Yes	
$h < \frac{1}{d_I} \ln\left(\frac{\theta_2}{\theta_2 - \theta_1}\right) + \epsilon$ ($\theta_2 > \theta_1$)	Yes	
$\frac{1}{d_I} \ln\left(\frac{\theta_2}{\theta_2 - \theta_1}\right) + \epsilon < h < \frac{1}{d_I} \ln(\frac{\theta_2}{\theta_2 - \pi}) + \epsilon$ ($\theta_2 > \max\{\theta_1, \pi\}$)		Yes

Theorem 4.15. *If*

$$d_{C_p} + \frac{\alpha_2 \overline{S}}{1 + k_1 \overline{S}} + \frac{\alpha_3 \kappa \overline{S}}{d_D + k_3 \kappa \overline{S}} > \frac{\alpha_1 \overline{S}}{1 + k_1 \overline{S}} \int_0^h \beta(a) \mathrm{e}^{-\int_0^a d_I(\xi) \mathrm{d}\xi} \mathrm{d}a, \tag{26}$$

then all possible roots of (24) lie in the left half-plane.

Remark 4.16. The sufficient condition (26) guarantees that all real parts of eigenvalues are negative. That is, (26) has a stabilizing effect.

Theorem 4.17. *If*

$$d_{C_p} + \frac{\alpha_2 \overline{S}}{1 + k_1 \overline{S}} + \frac{\alpha_3 \kappa \overline{S}}{d_D + k_3 \kappa \overline{S}} < \frac{\alpha_1 \overline{S}}{1 + k_1 \overline{S}} \int_0^h \beta(a) \mathrm{e}^{-\int_0^a d_I(\xi) \mathrm{d}\xi} \mathrm{d}a < \pi, \tag{27}$$

then a single root of (24) lies in the right half-plane.

Proof. In fact, it follows from $Arg \Phi(0) = \pi$ that $\Delta = -\frac{\pi}{2}$, and then $N^\infty = 1$. This completes the proof. □

Remark 4.18. The sufficient condition (27) guarantees that the infected pest-free equilibrium is unstable. That is, (27) has a destabilizing effect.

The following result proves that, under some circumstances, the supremum infection age h and initial density of the inherent non-specific defence immunity $\overline{D} \doteq \frac{\kappa \overline{S}}{d_D}$ play important roles in dynamics of structure.

Corollary 4.19. *Suppose that the following parameters take the form*

$$\beta(a) = \beta, \quad d_I(a) = \begin{cases} d_I \in (0, 1], & 0 < a \leq h - \epsilon; \\ +\infty, & a > h - \epsilon \end{cases}$$

for some $\epsilon > 0$. Then the trivial equilibrium (0, 0, 0) is unstable. For the linearized system at the equilibrium ($\overline{S} > 0, 0, \overline{D} > 0$), the h-dependence of the stable and unstable manifolds is summarized in Table 2.

Here

$$\theta_1 \doteq \theta^* + \frac{\alpha_3 \kappa \overline{S}}{d_D + k_3 \kappa \overline{S}},$$

$$\theta^* \doteq d_{C_p} + \frac{\alpha_2 \overline{S}}{1 + k_1 \overline{S}}$$

and

$$\theta_2 \doteq \frac{\alpha_1 \overline{S} \beta}{(1 + k_1 \overline{S}) d_I}.$$

Corollary 4.20. *Assume that*

$$\beta(a) = \beta, \ d_I(a) = \begin{cases} d_I \in (0, 1], & 0 < a \leq h - \epsilon; \\ +\infty, & a > h - \epsilon. \end{cases}$$

Then the trivial equilibrium (0, 0, 0) is unstable. For the linearized system at the equilibrium ($\overline{S} > 0, 0, \overline{D} > 0$), the $\overline{D}$-dependence of the stable and unstable manifold is summarized in Table 3. Here, ϵ, θ_1, θ_2 and θ^* are as defined in the corollary above.

Table 3
The stable and unstable manifolds with respect to the linearizes system at the equilibrium ($\overline{S} > 0, 0, \overline{D} > 0$).

$\overline{D}$	Two-dimensional stable manifold	One-dimensional unstable manifold
All ($\theta^* \geq \theta_2$)	Yes	
All ($\theta^* \geq \theta_2(1 - \mathrm{e}^{-d_I(h-\epsilon)})$)	Yes	
$\overline{D} > \frac{\theta_2(1-\mathrm{e}^{-d_I(h-\epsilon)})-\theta^*}{k_3\left[\frac{\alpha_3}{k_3}-(\theta_2(1-\mathrm{e}^{-d_I(h-\epsilon)})-\theta^*)\right]}$ $\left(\frac{\alpha_3}{k_3} > \theta_2(1 - \mathrm{e}^{-d_I(h-\epsilon)}) - \theta^*\right)$	Yes	
All $\left(\frac{\alpha_3}{k_3} < \theta_2(1 - \mathrm{e}^{-d_I(h-\epsilon)}) - \theta^* < \pi - \theta^*\right)$		Yes
$\overline{D} < \frac{\theta_2(1-\mathrm{e}^{-d_I(h-\epsilon)})-\theta^*}{k_3\left[\frac{\alpha_3}{k_3}-(\theta_2(1-\mathrm{e}^{-d_I(h-\epsilon)})-\theta^*)\right]}$ $\left(\frac{\alpha_3}{k_3} > \pi - \theta^* > \theta_2(1 - \mathrm{e}^{-d_I(h-\epsilon)}) - \theta^*\right)$		Yes

Table 4
Threshold values for the stable and unstable manifolds corresponding to the linearizes system at the equilibrium ($\overline{S} > 0, 0, \overline{D} > 0$).

$\alpha_1 = \alpha_3 = 0.3, \alpha_2 = \alpha_4 = 0.5,$ $k_1 = k_3 = 0.2, k_2 = k_4 = 0.3,$ $d_I(a) \equiv 1, a \in [0, h-\epsilon](\equiv \infty, a > h-\epsilon),$ $\beta(a) \equiv 10/3, d_{C_p} = 0.1, d_D = 1, \gamma = 0.01$	Two-dimensional stable manifold	One-dimensional unstable manifold
$\overline{S} = 50, \overline{D} = 0.5$	$h < 0.803 + \epsilon$	$0.803 + \epsilon < h < 1.174 + \epsilon$
$\overline{S} = 100, \overline{D} = 1$	$h < 0.852 + \epsilon$	$0.852 + \epsilon < h < 1.077 + \epsilon$
$\overline{S} = 200, \overline{D} = 2$	$h < 0.937 + \epsilon$	$0.937 + \epsilon < h < 1.032 + \epsilon$
$\overline{S} = 300, \overline{D} = 3$	$h < 1.007 + \epsilon$	$1.007 + \epsilon < h < 1.017 + \epsilon$

5. Pest control strategies

To confirm our mathematical findings and facilitate their interpretation, we proceed to investigate further by using numerical simulations (see Table 4).

Let $S(t)$ denote the number of pests in the larvae and adult stages. In each generation there is a burst of new larvae and adults. This resets the initial amount S_0 for each generation in an insect population with nonoverlapping generations. Hence, the time variable is on a fast time scale. It is suitable for describing processes within a generation [25].

Suppose that pathogenic viruses are grown in laboratories and then released during the time interval $(-h, 0]$. This means that we can control the initial amount of viral particles C_{p_0} and thus C_{p_0} is a control parameter. Given a pest population S_0, we can then use Fig. 3 to compute the least value C_{p_0} so that the limit of $S(t)$ as $t \to \infty$ will be below a desirable level. More precisely, setting the economic threshold (ET) $= 20.51$ and the control period ($t = 9$), from Fig. 7 (the magnified view of Fig. 4(a)), we may choose the highest applied dose of 20 to control the amount of the susceptible pest population below the given ET under the assumption that the period of treatment approximates 6.4. To make the management more economically viable, it is recommended to use the dose of 15 to control the amount of the pest population below the given ET under the assumption that the period of treatment approximates 7.5. On the other hand, Fig. 4(b) reveals that defence mechanisms can prolong the life time of infected hosts (it is fated that infected hosts should die out).

From Figs. 5 and 6, one notes that the immune response is of no effect since the low dose effects induced by viral particles. However, at a feasible dose of pathogenic viruses released initially, there are a "moderate" accumulation in pathogenic viruses and an "abrupt" increase in defence mechanisms accumulation for a period of time, and subsequently a "moderate" dissipation in pathogenic viruses and an "abrupt" increase in the density of the immune response. On the other hand, graphically, we find that the defence immunity is sensible to the change in the amount of pathogenic viruses in advance. In this regard, Figs. 8–11 describe the dynamics of the linearized stability of the equilibrium $E_1 = (\overline{S}, 0, \overline{D})$. From a viewpoint of pest management, we should avoid such a situation by setting a series of feasible and economical control policies.

6. Concluding remarks

The present paper attempts to formulate and study an integrated pest management model which relies on the use of pathogenic viruses as biocontrol agents. The pest population is divided into susceptibles and infectives, pathogenic viruses and defence mechanisms being also employed. Our model keeps track of the so-called "age of infection", that is, the time elapsed since the pest became infected. Consequently, a transport equation is used to model the dynamics of the infective class. Through the use of some reasonable hypotheses, our mathematical model can be reduced to a three-dimensional integrodifferential system. The asymptotic behavior of the system is then studied using monotonicity methods and integral estimations. It is seen that the size of the susceptible pest population stabilizes in the long term at a certain positive level and the total size of the infective class tends to 0, while if the decay rates of the pathogenic viruses and defence mechanisms are null, the pathogenic viruses are released fast enough and the initial amounts of susceptible hosts, viral particles and defence mechanisms satisfy some conditions, then the susceptible pests become extinct as well.

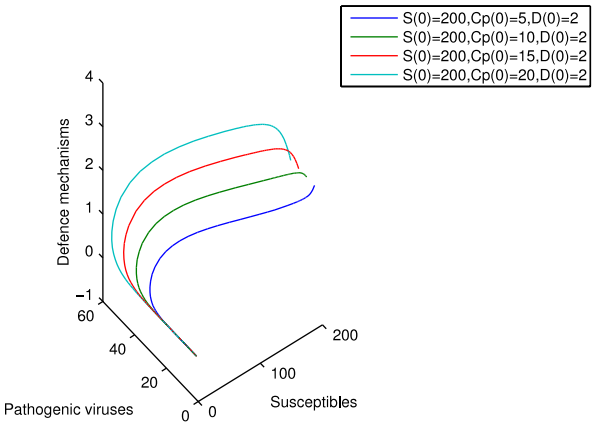

Fig. 3. $\alpha_1 = \alpha_3 = 0.3, \alpha_2 = \alpha_4 = 0.5, k_1 = k_3 = 0.2, k_2 = k_4 = 0.3, d_I(a) \equiv 1, a \in (0, h-\epsilon]$ $(d_I(a) \equiv +\infty, a > h-\epsilon)$, $\beta(a) \equiv 10/3, \gamma(a) \equiv \frac{1}{3}$, $d_{C_p} = 0.1, d_D = 1, \kappa = 0.01, h = 1.0001$ and $\epsilon = 0.0001$. The trajectories with the same initial values $S(0)$ and $D(0)$, and different initial values $C_p(0)$.

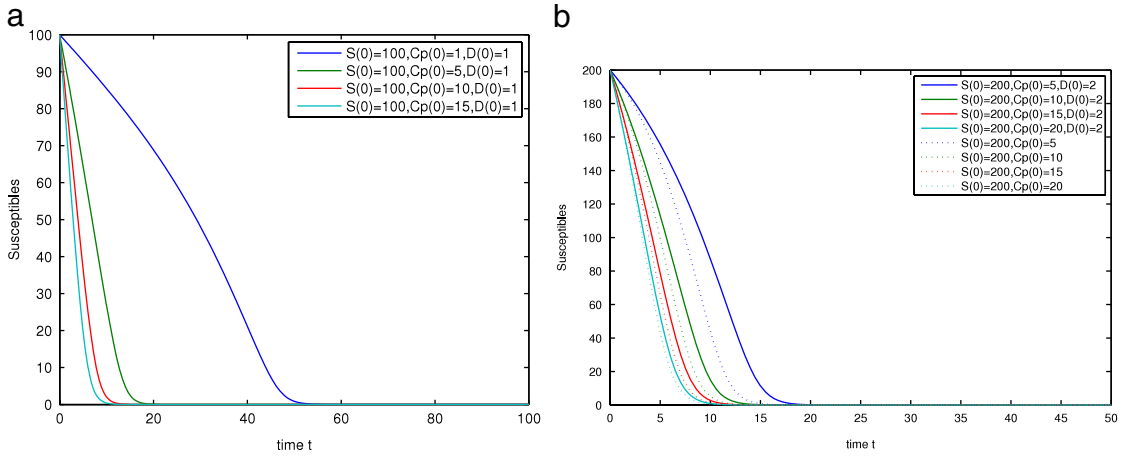

Fig. 4. $\alpha_1 = \alpha_3 = 0.3, \alpha_2 = \alpha_4 = 0.5, k_1 = k_3 = 0.2, k_2 = k_4 = 0.3, d_I(a) \equiv 1, a \in (0, h-\epsilon]$ $(d_I(a) \equiv +\infty, a > h-\epsilon)$, $\beta(a) \equiv 10/3, \gamma(a) \equiv \frac{1}{3}$, $d_{C_p} = 0.1, d_D = 1, \kappa = 0.01, h = 1.0001$ and $\epsilon = 0.0001$. (a) The time series for S with the same initial values $S(0)$ and $D(0)$, and different initial values $C_p(0)$. (b) The time series for S with defence immunity (continuous curves) versus S without defence immunity (dotted curves) $(\alpha_3 = \alpha_4 = 0, \gamma(a) \equiv 0)$.

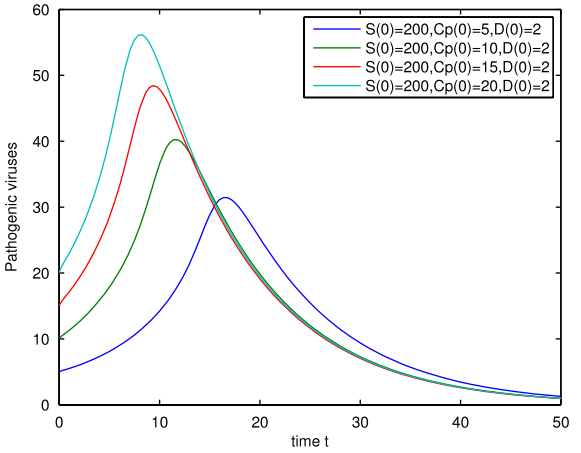

Fig. 5. $\alpha_1 = \alpha_3 = 0.3, \alpha_2 = \alpha_4 = 0.5, k_1 = k_3 = 0.2, k_2 = k_4 = 0.3, d_I(a) \equiv 1, a \in (0, h-\epsilon]$ $(d_I(a) \equiv +\infty, a > h-\epsilon)$, $\beta(a) \equiv 10/3, \gamma(a) \equiv \frac{1}{3}$, $d_{C_p} = 0.1, d_D = 1, \kappa = 0.01, h = 1.0001$ and $\epsilon = 0.0001$. The time series for C_p with the same initial values $S(0)$ and $D(0)$, and different initial values $C_p(0)$.

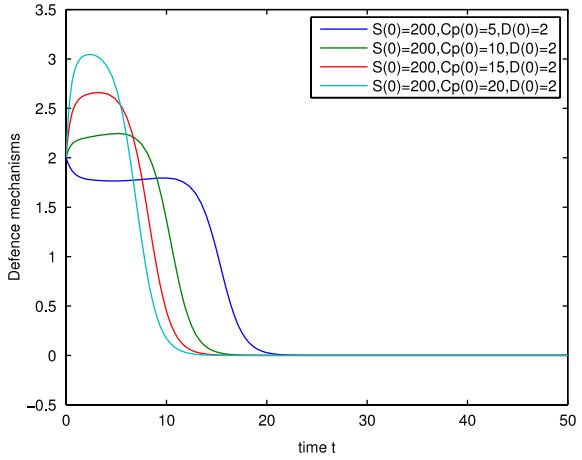

Fig. 6. $\alpha_1 = \alpha_3 = 0.3, \alpha_2 = \alpha_4 = 0.5, k_1 = k_3 = 0.2, k_2 = k_4 = 0.3, d_I(a) \equiv 1, a \in (0, h-\epsilon]$ $(d_I(a) \equiv +\infty, a > h-\epsilon)$, $\beta(a) \equiv 10/3, \gamma(a) \equiv \frac{1}{3}$, $d_{C_p} = 0.1, d_D = 1, \kappa = 0.01, h = 1.0001$ and $\epsilon = 0.0001$. The time series for D with the same initial values $S(0)$ and $D(0)$, and different initial values $C_p(0)$.

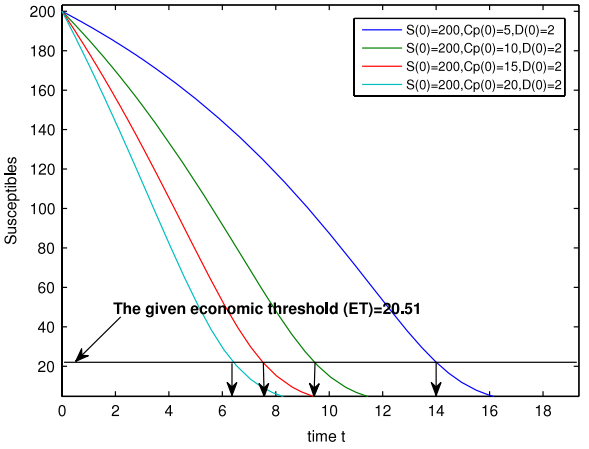

Fig. 7. The magnified view of Fig. 5(a).

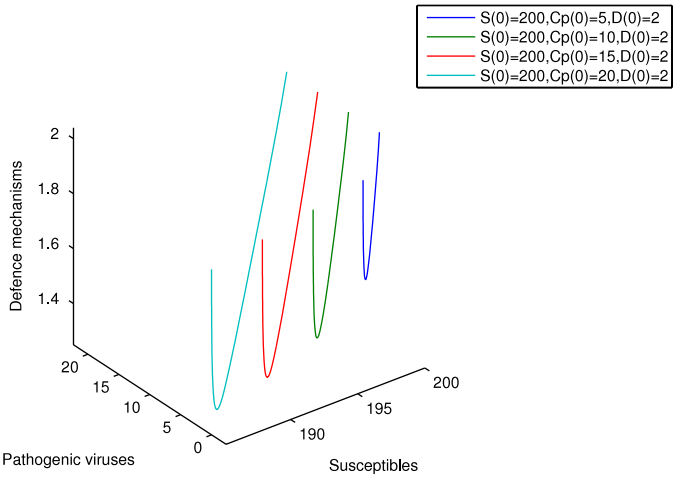

Fig. 8. $\alpha_1 = \alpha_3 = 0.3, \alpha_2 = \alpha_4 = 0.5, k_1 = k_3 = 0.2, k_2 = k_4 = 0.3, d_I(a) \equiv 1, a \in (0, h-\epsilon]$ $(d_I(a) \equiv +\infty, a > h-\epsilon)$, $\beta(a) \equiv 10/3, \gamma(a) \equiv \frac{1}{3}$, $d_{C_p} = 0.1, d_D = 1, \kappa = 0.01, h = 0.9$ and $\epsilon = 0.0001$. The trajectories with the same initial values $S(0)$ and $D(0)$, and different initial values $C_p(0)$.

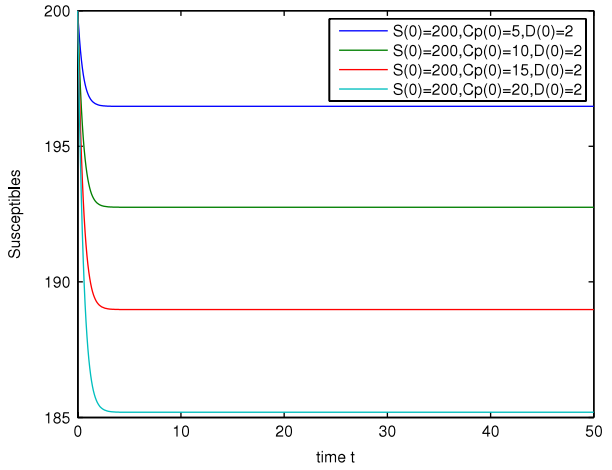

Fig. 9. $\alpha_1 = \alpha_3 = 0.3, \alpha_2 = \alpha_4 = 0.5, k_1 = k_3 = 0.2, k_2 = k_4 = 0.3, d_I(a) \equiv 1, a \in (0, h-\epsilon]$ $(d_I(a) \equiv +\infty, a > h-\epsilon)$, $\beta(a) \equiv 10/3, \gamma(a) \equiv \frac{1}{3}$, $d_{C_p} = 0.1, d_D = 1, \kappa = 0.01, h = 0.9$ and $\epsilon = 0.0001$. The time series for S with the same initial values $S(0)$ and $D(0)$, and different initial values $C_p(0)$.

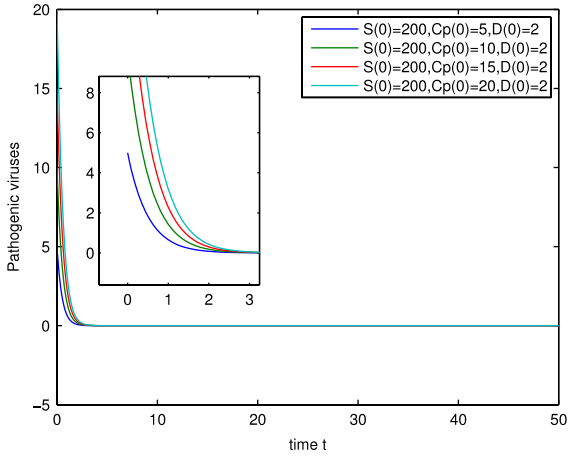

Fig. 10. $\alpha_1 = \alpha_3 = 0.3, \alpha_2 = \alpha_4 = 0.5, k_1 = k_3 = 0.2, k_2 = k_4 = 0.3, d_I(a) \equiv 1, a \in (0, h-\epsilon]$ $(d_I(a) \equiv +\infty, a > h-\epsilon)$, $\beta(a) \equiv 10/3, \gamma(a) \equiv \frac{1}{3}$, $d_{C_p} = 0.1, d_D = 1, \kappa = 0.01, h = 0.9$ and $\epsilon = 0.0001$. The time series for C_p with the same initial values $S(0)$ and $D(0)$, and different initial values $C_p(0)$.

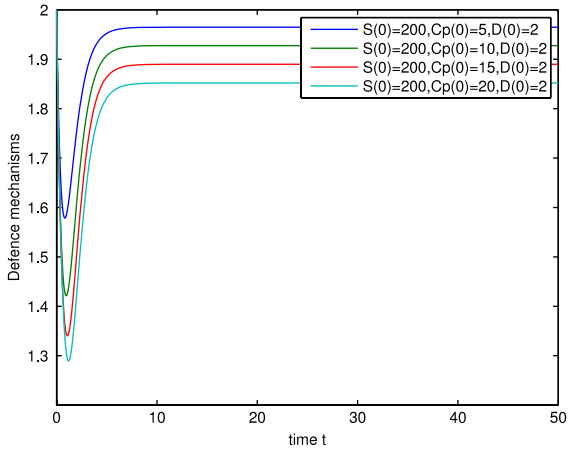

Fig. 11. $\alpha_1 = \alpha_3 = 0.3, \alpha_2 = \alpha_4 = 0.5, k_1 = k_3 = 0.2, k_2 = k_4 = 0.3, d_I(a) \equiv 1, a \in (0, h-\epsilon]$ $(d_I(a) \equiv +\infty, a > h-\epsilon)$, $\beta(a) \equiv 10/3, \gamma(a) \equiv \frac{1}{3}$, $d_{C_p} = 0.1, d_D = 1, \kappa = 0.01, h = 0.9$ and $\epsilon = 0.0001$. The time series for D with the same initial values $S(0)$ and $D(0)$, and different initial values $C_p(0)$.

Next, a linearized stability analysis is performed via the use of Michailov criterion and it is found out that the maximal age of infection and inherent non-specific resistance mechanisms play important roles in the dynamics of the system. Graphically, the "alert response" feature of the immune response is found via simulation of pathogenic viruses. Let us note that the system has multiple equilibria and, in addition, their attracting properties strongly depend on the initial conditions.

The mathematical results which are obtained in this paper may be useful for many agricultural researchers, as the feasible use of viral pathogens in order to avoid or weaken the defence immunity of infected hosts is a viable method of pest control, its characteristic feature being that it usually does not need to be reapplied each time a pest outbreak occurs.

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Appendix A. Proof of Lemma 4.3

Proof. According to (A_5), (8) and (14), one obtains that

$$\begin{aligned}\int_0^t\int_0^h \beta(a)I(s,a)\mathrm{d}a\mathrm{d}s &\le \int_0^t\int_0^h\int_0^{h-a} g(\xi)d_I(\xi+a)\mathrm{e}^{-\int_a^{a+\xi} d_I(\tau)\mathrm{d}\tau} I(s,a)\mathrm{d}\xi\mathrm{d}a\mathrm{d}s\\ &\le \int_0^t\int_0^h\int_0^{h-a} g(\xi)d_I(\xi+a)I(s+\xi,a+\xi)\mathrm{d}\xi\mathrm{d}a\mathrm{d}s. \end{aligned}\tag{28}$$

With the change of variables given by $a' = a+\xi$ and $s' = s+\xi$, one obtains that

$$\begin{aligned}\int_0^t\int_0^h \beta(a)I(s,a)\mathrm{d}a\mathrm{d}s &\le \int_0^t\int_0^h\int_0^{h-\xi} g(\xi)d_I(\xi+a)I(s+\xi,a+\xi)\mathrm{d}a\mathrm{d}\xi\mathrm{d}s\\ &= \int_0^h g(\xi)\left(\int_0^t\int_0^{h-\xi} d_I(\xi+a)I(s+\xi,a+\xi)\mathrm{d}a\mathrm{d}s\right)\mathrm{d}\xi\\ &= \int_0^h g(\xi)\left(\int_\xi^{t+\xi}\int_\xi^h d_I(a')I(s',a')\mathrm{d}a'\mathrm{d}s'\right)\mathrm{d}\xi\\ &\le \left(\int_0^h g(\xi)\mathrm{d}\xi\right)\left(\int_0^{t+h}\int_0^h d_I(a)I(s,a)\mathrm{d}a\mathrm{d}s\right)\end{aligned}$$

and consequently (15) holds. This completes the proof. □

Appendix B. Proof of Lemma 4.12

Proof. Let $C \doteq C^1 \cup C^2$ denote a closed semicircle into the right half-plane, in which C^1 denotes the straight line on the imaginary axis from ri to $-ri$ and C^2 the semicircle from $-ri$ to ri with radius r into the right half-plane. It is easy to note that

$$N - P = \frac{1}{2\pi}\Delta_C Arg\Phi(\lambda).$$

Here, N and P represent the number of zeros and, respectively, poles in C. In view of the analyticity of $\Phi(\lambda)$ in C, one obtains that $P = 0$. It is then noted that

$$N = \frac{1}{2\pi}\left(\Delta_{C^1} Arg\Phi(\lambda) + \Delta_{C^2} Arg\Phi(\lambda)\right).$$

If we denote by $\Delta_{C^i}^{\infty} Arg\Phi(\lambda)$, $i = 1, 2$, the changes of the argument as r tends to ∞, one obtains

$$\Delta_{C^1}^{\infty} Arg\Phi(\lambda) = -2\Delta.$$

Next, let N^{∞} be the number of zeros inside the semicircle as r tends to ∞, which is well defined by condition (b). Therefore, we get that

$$N^{\infty} = \frac{1}{2\pi}\Delta_{C^2}^{\infty} Arg\Phi(\lambda) - \frac{\Delta}{\pi}.$$

In the following we shall compute $\Delta^{\infty}_{C^2} Arg\Phi(\lambda)$. Recalling the equality

$$\frac{1}{2\pi}\Delta^{\infty}_{C^2} Arg\Phi(\lambda) = \frac{1}{2\pi \mathrm{i}}\int_{C^2}\frac{\Phi'(\lambda)}{\Phi(\lambda)}\mathrm{d}\lambda.$$

Let $\lambda = r\mathrm{e}^{\mathrm{i}\theta}, \theta \in [-\frac{\pi}{2}, \frac{\pi}{2}]$ and $r \in (0, \infty)$. We then get

$$\begin{aligned}
\int_{C^2}\frac{\Phi'(\lambda)}{\Phi(\lambda)}\mathrm{d}\lambda &= \int_{-\frac{\pi}{2}}^{\frac{\pi}{2}}\frac{\mathrm{i}r\mathrm{e}^{\mathrm{i}\theta}\Phi'(r\mathrm{e}^{\mathrm{i}\theta})}{\Phi(r\mathrm{e}^{\mathrm{i}\theta})}\mathrm{d}\theta \\
&= \int_{-\frac{\pi}{2}}^{\frac{\pi}{2}}\frac{\mathrm{i}r\mathrm{e}^{\mathrm{i}\theta}\left(1+\frac{\bar{S}}{1+k_1\bar{S}}\int_0^h aA(a)\mathrm{e}^{-ar(\cos\theta+\mathrm{i}\sin\theta)}\mathrm{d}a\right)}{r\mathrm{e}^{\mathrm{i}\theta}+d_{C_p}+\frac{\alpha_2\bar{S}}{1+k_1\bar{S}}+\frac{\alpha_3\kappa\bar{S}}{d_D+k_3\kappa\bar{S}}-\frac{\bar{S}}{1+k_1\bar{S}}\int_0^h A(a)\mathrm{e}^{-ar(\cos\theta+\mathrm{i}\sin\theta)}\mathrm{d}a}\mathrm{d}\theta \\
&= \int_{-\frac{\pi}{2}}^{\frac{\pi}{2}}\frac{\mathrm{i}\mathrm{e}^{\mathrm{i}\theta}\left(1+\frac{\bar{S}}{1+k_1\bar{S}}\int_0^h aA(a)\mathrm{e}^{-ar(\cos\theta+\mathrm{i}\sin\theta)}\mathrm{d}a\right)}{\mathrm{e}^{\mathrm{i}\theta}+\frac{d_{C_p}}{r}+\frac{\alpha_2\bar{S}}{r(1+k_1\bar{S})}+\frac{\alpha_3\kappa\bar{S}}{r(d_D+k_3\kappa\bar{S})}-\frac{\bar{S}}{r(1+k_1\bar{S})}\int_0^h A(a)\mathrm{e}^{-ar(\cos\theta+\mathrm{i}\sin\theta)}\mathrm{d}a}\mathrm{d}\theta.
\end{aligned}$$

Hence, it follows from Lebesgue's dominated convergence theorem that

$$\int_{C^2}\frac{\Phi'(\lambda)}{\Phi(\lambda)}\mathrm{d}\lambda \to \mathrm{i}\pi \quad \text{as } r \to \infty,$$

that is,

$$\frac{1}{2\pi}\Delta^{\infty}_{C^2} Arg\Phi(\lambda) = \frac{1}{2}.$$

Consequently, $N^{\infty} = \frac{1}{2} - \frac{\Delta}{\pi}$. This completes the proof. □

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