MATH. SCI. RES. J. 14(3) 2010 53-69

THE DYNAMICS OF AN AGE-STRUCTURED PEST MANAGEMENT MODEL

Hong Zhang*
Department of Mathematics, Jiangsu University,
ZhenJiang, JiangSu 212013, P.R. China
Department of Mathematics and Mathematical Statistics, Umeå University,
SE-901 87 Umeå, Sweden
hongzhang@ujs.edu.cn

Paul Georgescu†
Department of Mathematics, "Gh. Asachi" Technical University of Iaşi,
Bd. Copou 11, 700506 Iaşi, Romania
v.p.georgescu@gmail.com

Received: date / Accepted: date

Abstract

This paper investigates a compartmental pest management model which divides the pest population into a susceptible and an infective class, while also including a class of pathogenic viruses. The model is age-structured, in the sense that it accounts for the differences in the amounts of pathogenic viruses released by infective pests at various infection ages. First, the asymptotic behavior of the system is established via a monotonicity analysis which makes use of several integral inequalities. The linearized stability of the equilibria for the system is then discussed by means of Michailov criterion. As an outcome of our analysis, it is observed that the maximal length of the infective period plays an important role in the dynamics of the system. Several pest control strategies are further investigated by means of numerical simulations, the paper being concluded with a discussion on the biological significance of the mathematical findings.

Key words: Age structure, compartmental model, asymptotic behavior, transport equation, Michailov criterion 2000 MSC: 92D25, 92D40

*HZ's research was supported by the National Natural Science Foundation of China (grant ID: 10771104) and the high-level talent project of Jiangsu University(grant ID: 08JDG047)

†PG's research was supported by CNCSIS of Romania (grant ID: 342/2009)

1 Introduction

Pest control strategies were occasionally described 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. A passage in Homer's Iliad (eighth century BC) mentions the use of fire to drive locusts into the sea, and the ancient Egyptians organized long lines of human drovers to repel swarms of invading locusts.

Nowadays, to prevent pest outbreaks farmers often resort to pesticides, but pesticide overuse may have unwanted consequences including insect resistance, resurgence and outbreaks of secondary pests. Also, pesticide residues may affect human health and the environment.

Generally, insects, like humans and other animals, can be infected by disease-causing organisms such as bacteria, viruses and fungi. Under appropriate conditions, like high humidity or high pest abundance, these naturally occurring organisms may multiply to cause disease outbreaks or epizootics that can decimate an insect population. Hence, a wise alternative to chemical control is microbial control which is, generally, man's use of suitably chosen living entomopathogenic organisms, referred as microbial agents, to control pests. Control agents can be bacteria, fungi, viruses, nematodes and protozoa that either kill the harmful organism or interfere with its biological processes[7],[8],[10],[15],[13],[23],[14],[30]. The control of rabbit pests in Australia[9] by the viral disease called "myxomatosis" provides a spectacular example of a virus controlling a pest.

With the development of biotechnology and the advent of ecologically advisable policies for pest management, microbial control policies have achieved immense importance in agricultural protection programs. A large number of Baculoviruses, each one with sometimes very restricted but different host stages, can be used as microbial control agents of insects, the greatest potential being displayed by the Baculoviridae Nuclear Polyhedrosis Virus (NPV) and the Granulo Virus (GV) [24]. In this regard, since NPVs are often host specific, cause autoinfection and are usually fatal, their use is increasing gradually in agricultural management[28]. At this moment, there is a growing amount of literature on the usage of entomopathogens to suppress pests[14], [9],[6],[11],[12],[27], many models of pest-pathogen dynamics being formulated and analyzed[1],[2],[3],[4],[5],[16],[17],[25].

In India, the "army worm" *Spodoptera litura* has defoliated many economically important crops including cole, cotton, and soybean. Synthetic pesticides have been used against this pest, with unfortunate side effects. Currently, entomologists are trying to use the Spodoptera polyhedrosis virus (SlNPV) to control the fourth instar larvae, which is the most damaging evolutionary stage of pest.

The celebrated Kermack and McKendrick SI model with differential infectivity has been introduced by Kermack and McKendrick in [19, 20, 21], in the form

$$\begin{cases} S'(t) = S(t)\displaystyle\int_0^\infty A(\tau)S'(\tau)d\tau \\ I(t) = -\displaystyle\int_0^\infty \frac{A(\tau)}{A(0)}S'(t-\tau)d\tau \end{cases}, \tag{1.1}$$

where $S(t)$ and $I(t)$ represent the size of the susceptible and infective class at time t, respectively, and $A(\tau)$ represents the expected infectivity of an individual with age of infection τ. If $A(\tau) = \rho e^{-w\tau}$, Kermack and McKendrick's model simplifies to

$$\begin{cases} S' = -\rho SI, \\ I' = \rho SI - wI \end{cases}. \tag{1.2}$$

Goh [17] studied (1.2) from a numerical viewpoint, attempting to find sufficient conditions for the control of a given pest population. In order to describe with greater accuracy the effects of pathogens upon a target pest population, Anderson and May [2] formulated the system

$$\begin{cases} I' = \rho PS - (\theta + b + \mu)I, \\ H' = rH - \theta I, \\ P' = A + \omega I - (\gamma + \rho H)P, \end{cases} \tag{1.3}$$

In the above, H represents the total size of the host population, $H = S + I$, where S and I are as above, P is the size of the pathogen population, ω denotes the rate at which pathogens are produced by infective individuals, γ is the mortality rate of the host population in the infective stage, A is the release rate of the pathogens, b represents the death rate of the host if no pathogens are present, μ denotes the host recovery rate and θ is the pathogen-induced death rate. Anderson and May showed that if the release rate A exceeds a critical level, the host (pest) population tends to extinction. Brown [5] then proposed and investigated a modified SIRS model, understood as a framework for the study of epizootiological diseases in an insect-pathogen system. By means of a linearized stability analysis, it has been shown in [5] that the system with one immune and one susceptible host class can exhibit stable, periodic or unstable behavior depending on the values of the model parameters. To incorporate the effects of the (constant) maturation time, Moerbeek and Van Den Bosch[25] introduced several stage-structured models of insect-pathogen interactions with delay and conclusioned that their dynamics is heavily influenced by the stage-specific susceptibility. Zhang et al.[29] dealt with an impulsively controlled epidemic model with delay, stage-structure and a general form of the incidence rate. In Zhang et al. [29], the pest population was divided into three compartments: pest eggs, susceptible pests and infective pests and it has been assumed that infective pests do not damage crops. The main results of Zhang et al. [29] indicated that if the amount of infective pests released each time, the maturation delay and the period of impulsive period release satisfy a certain condition, then the so-called susceptible pest-eradication periodic solution is globally stable, while the system may also become permanent under certain hypotheses.

Since the above models cannot be used to assess the impact of the pathogenic virus density and infection age upon the spread of the infection, the aim of this paper is to formulate and discuss a new model of pest management which involves the release of pathogens and investigate its dynamics. In this model, the pest population is divided into a susceptible and an infective class, a class of pathogenic viruses being also included. An additional variable is used to keep track of the so-called age of infection. The dynamics of the system is discussed by means of the linearized stability analysis of a functional differential equation, which is equivalent to the original age-structured model. The linearized stability analysis is performed using Michailov criterion and it is found that the maximal length of the infective period influences the dynamics of the system decisively. The remaining part of the paper is organized as follows. The model to be studied is proposed in Section 2, where the main biological assumptions used to formulate the model are also stated. This model is then reformulated as a functional differential equation on a certain space of continuous functions. The influence of the viral particle self-regulation and age structure on the asymptotic behavior of the system is discussed in Section 3. The biological significance of the mathematical findings is presented in Section 4, while numerical simulations are performed in Section 5 in order to find the critical values for the model parameters.

2 The age-structured SI model

In what follows, we attempt to formulate an age-dependent compartmental pest-pathogen model which describes the propagation in a target pest population of a disease possessing a free-living stage in the environment. This model includes time-dependent state variables representing the densities of susceptible hosts $S(t)$ and of pathogenic viruses $C_p(t)$ together with a time and age-dependent state variable representing the density of infected hosts $I(t, a)$, the variable a denoting the so-called age of infection, that is, the time passed since the pest became infected. The general scheme of the transitions between different pest states is presented in Figure 1.

The model to be studied is formulated as follows

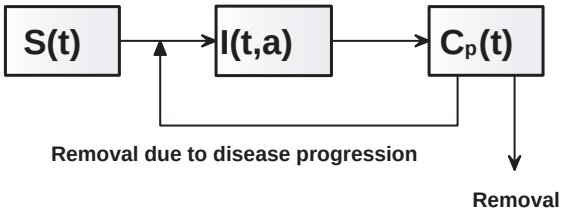

Figure 1: Flow chart of infection spread via pathogenic viruses

$$S'(t) = -\frac{\alpha_1 C_p(t) S(t)}{1 + k_1 S(t) + k_2 C_p(t)}, \quad t > 0 \tag{2.4}$$

$$\frac{\partial I(t,a)}{\partial t} + \frac{\partial I(t,a)}{\partial a} = -d_I(a) I(t,a), \quad t, a > 0 \tag{2.5}$$

$$C_p'(t) = -d_{C_p} C_p(t) + \int_0^h \beta(a) I(t,a) da - \frac{\alpha_2 C_p(t) S(t)}{1 + k_1 S(t) + k_2 C_p(t)} \quad t, a > 0 \tag{2.6}$$

with the boundary condition

$$I(t,0) = \frac{\alpha_1 C_p(t) S(t)}{1 + k_1 S(t) + k_2 C_p(t)}, \ t > 0 \tag{2.7}$$

and initial conditions

$$S(0) = S_0(0), \ C_p(0) = C_{p_0}(0), \tag{2.8}$$

$$I(0,a) = I_0(a), \ a \geq 0. \tag{2.9}$$

In the above, α_1 represents the amount of susceptible pests converted to infected pests per pathogen cell and unit of time, α_2 denotes the amount of pathogenic viruses consumed to propagate the infection per susceptible pest and unit of time, d_{C_p} represents the amount of pathogenic viruses removed per unit of time, $d_I(a)$ represents the amount of infected hosts with age of infection a which are removed per unit of time, k_1 and k_2 are nonnegative constants describing the effect of handling time on the incidence rate of infection and the magnitude of the interference of pathogen cells in host organisms, respectively.

The incidence rate of infection used in the model above, that is,

$$\lambda(S, C_p) = \frac{\alpha_1 C_p S}{1 + k_1 S + k_2 C_p},$$

degenerates into the bilinear incidence rate $g_1(S, C_p) = \alpha_1 C_p S$ if $k_1 = k_2 = 0$ and into saturated incidence rates if $k_1 = 0$ or $k_2 = 0$.

In the following, we shall assume that all initial conditions are nonnegative. The next assumptions are used to formulate the mathematical model:

(A_1) The epidemic occurs on a fast timescale, no natural mortality of susceptibles being considered. Susceptible hosts are not capable of reproduction once they are exposed to pathogenic organisms. Infective pests are not capable of damaging crops.

(A_2) Infected pests are parametrized by their age of infection, i.e., the time elapsed since the pest became infected, which is denoted by a. Infected pests release pathogenic viruses at a age-dependent rate $\beta(a)$.

(A_3) Pathogenic viruses are degraded at a constant rate d_{C_p}.

(A_4) The constants $\alpha_i (i = 1, 2)$ and d_{C_p} are all positive. Pathogenic viruses are produced by infected host only in some bounded interval of age of infection $[0, h)$, for some $h > 0$, which leads to $supp\,\beta(\cdot) \subset [0, h)$, where h is the maximal age of infection, as well as to $I(t, a) = 0$ for $a \geq h$. The mortality of infected hosts d_I is a nonnegative function of the infection age a and satisfies $\int_0^{h-} d_I(a) = +\infty$.

Remark 2.1. *Since h is the maximal age of infection, it follows that the reasonable domain of the function d_I is the interval $[0, h)$ and all infected pests die before reaching infection age h.*

Remark 2.2. *The quantities $S_0(0)$ and $C_{p_0}(0)$ represent the initial amount of target pests and the initial amount of pathogens released by farmers, respectively.*

We now try to establish the well-posedness of the above model. From (2.5), (2.7) and (2.9), one obtains that $I(t, a)$ is given by

$$I(t,a) = \begin{cases} \frac{\alpha_1 C_p(t-a)S(t-a)}{1+k_1 S(t-a)+k_2 C_p(t-a)} e^{-\int_0^a d_I(\xi)d\xi}, & \text{for } t > a \geq 0; \\ I_0(a-t)e^{-\int_{a-t}^a d_I(\xi)d\xi}, & \text{for } a \geq t > 0. \end{cases} \tag{2.10}$$

Using classical existence and uniqueness results for functional differential equations (see Hale[18], Kuang[22]) it is seen that the integrodifferential system (2.4)-(2.6) in which $I(t, a)$ is substituted with its expression given by (2.10) has a unique solution, which is globally defined. We shall be concerned in the following with the positivity and the asymptotic behavior of the aforementioned solution.

3 The dynamical behavior of the pest-pathogen model

From a practical point of view, a pest management strategy is considered successful if the size of the susceptible pest class stabilizes in the long term under an economically significant threshold level (ET), defined as the lowest pest density that will cause economic damage, or the amount of pest injury which will justify the cost of using controls. Also, it is important to find out whether or not the size of the class of pathogenic viruses tends to 0, that is, whether or not the pathogenic viruses remain active in the long term.

To this purpose, let us denote by $N_I(t)$ the total size of the infected pest population at time t, defined as

$$N_I(t) = \int_0^h I(t, a)da.$$

Let us also define the total size of the pest population at time t by

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

3.1 The asymptotic behavior

The following result, apart from establishing the positivity of the solutions of (2.4)-(2.6), asserts that the total size of the infected pest class tends to 0, that is the infection disappears in the long term.

Theorem 3.1. *The following statements hold.*

(i) *S, C_p, I, N_I and N are nonnegative.*

(ii) *S and N are nonincreasing.*

(iii) *S, N_I, N and $\int_0^t \left(\int_0^h d_I(a) I(s, a)da \right) ds$ are bounded.*

(iv) *$\int_0^t N_I(s)ds$ is bounded and N_I tends to 0 as $t \to \infty$.*

Proof. By integrating (2.4) on $[0,t]$, one obtains

$$S(t) = S_0(0)e^{-\alpha_1 \int_0^t \frac{C_p(s)}{1+k_1 S(s)+k_2 C_p(s)} ds} \geq 0, \tag{3.11}$$

since $S_0(0)$ is nonnegative. Suppose that C_p becomes negative and define $t^* = \inf_{t\geq 0}\{t|C_p(t)=0\}$. Since $C_p'(t^*) = \int_0^h \beta(a)I(t^*,a)da$ and $I(t^*,a)$ is given by (2.10) with $t=t^*$, it follows that $C_p'(t^*) > 0$, which leads to a contradiction. Then C_p is nonnegative, which implies that S is nonincreasing (due to (3.11)) and that I is nonnegative (due to (2.10). Since I and S are nonnegative, it also follows that N and N_I are nonnegative. One notes that

$$\begin{aligned} N_I'(t) &= \frac{d}{dt}\Big(\int_0^h I(t,a)da\Big) \\ &= -\int_0^h d_I(a)I(t,a)da - \int_0^h \frac{\partial I(t,a)}{\partial a}da \\ &= -\int_0^h d_I(a)I(t,a)da + \frac{\alpha_1 C_p(t)S(t)}{1+k_1S(t)+k_2C_p(t)} \\ &= -\int_0^h d_I(a)I(t,a)da - S'(t) \end{aligned} \tag{3.12}$$

and consequently

$$N'(t) = -\int_0^h d_I(a)I(t,a)da, \tag{3.13}$$

which implies that N is nonincreasing. Integrating both sides of (3.13) on $[0,t]$, we then derive

$$S(t) + N_I(t) + \int_0^t \left(\int_0^h d_I(a)I(s,a)da\right) ds = S_0(0) + N_I(0), \tag{3.14}$$

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

$$\int_0^t \left(\int_0^h d_I(a)I(s,a)da\right) ds$$

follows. It is seen that

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

By denoting $a' = a+\xi$, $s' = s+\xi$, one obtains

$$\begin{aligned} \int_0^t N_I(s)ds &= \int_0^t \int_0^h \left(\int_\xi^h d_I(a')I(s+\xi,a')da'\right) d\xi ds \\ &= \int_0^h \left(\int_\xi^{t+\xi} \left(\int_\xi^h d_I(a')I(s',a')da'\right) ds'\right) d\xi \\ &\leq h\left(\int_0^{t+h} \int_0^h d_I(a')I(s',a')da'ds'\right) \end{aligned}$$

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

Remark 3.2. *The term* $\int_0^t \left(\int_0^h d_I(a)I(s,a)da \right) ds$ *gives the total amount of infected hosts of all infection ages which are removed during the time interval* $[0,t]$.

Next, to facilitate the further analysis of the asymptotical behavior, we shall construct a connection between the production of new pathogenic viruses and the degradation rate of infected pests.

Lemma 3.3. *Suppose that there exists a nonnegative integrable function* g *defined on* $[0,h)$ *for the given* h *such that for every* $a \in [0,h)$

$$\beta(a) \leq \int_0^{h-a} g(\xi) d_I(\xi + a) e^{-\int_a^{a+\xi} d_I(\tau)d\tau} d\xi. \tag{3.15}$$

Then

$$\begin{aligned} &\int_0^t \int_0^h \beta(a) I(s,a) da ds \\ &\quad \leq \left(\int_0^h g(\xi) d\xi \right) \left(\int_0^{t+h} \int_0^h d_I(a) I(s,a) da ds \right). \end{aligned} \tag{3.16}$$

Proof. According to (2.10) and (3.15), one obtains that

$$\begin{aligned} \int_0^t \int_0^h \beta(a) I(s,a) da ds &\leq \int_0^t \int_0^h \int_0^{h-\xi} g(\xi) d_I(\xi + a) I(s+\xi, a+\xi) da d\xi ds \\ &< \int_0^h g(\xi) d\xi \int_0^{t+h} \int_0^h d_I(a) I(s,a) da ds. \end{aligned}$$

□

Subsequently, ones directly obtain the following results

Theorem 3.4. *Assume that the condition (3.15) holds. Then*

(i) C_p *and* $\int_0^t C_p(s)ds$ *are bounded.*

(ii) S *tends to a positive limit* S^* *as* $t \to \infty$.

Proof. By integrating (2.6), one obtains

$$\begin{aligned} &C_p(t) + d_{C_p} \int_0^t C_p(s)ds + \alpha_2 \int_0^t \frac{C_p(s)S(s)}{1 + k_1 S(s) + k_2 C_p(s)} ds \\ &\quad = C_{p_0}(0) + \int_0^t \int_0^h \beta(a) I(s,a) da ds, \end{aligned} \tag{3.17}$$

and, due to Lemma 3.3, it follows that $C_p t)$, $\int_0^t C_p(s)ds$ and $\int_0^t \frac{C_p(s)S(s)}{1+k_1 S(s)+k_2 C_p(s)} ds$ are bounded. Since S is nonincreasing, it has a limit for $t \to \infty$ and, due to (3.11) and to the boundedness of $\int_0^t \frac{C_p(s)S(s)}{1+k_1 S(s)+k_2 C_p(s)} ds$, this limit is positive. □

Remark 3.5. *Condition (3.15) is to be understood as a boundedness condition for the release rate of patogenic viruses. In this regard, it is natural to expect that if this rate is majorized by a suitable function of the virus removal rate, then the total amount of pathogenic viruses released within t time units will stay bounded and the susceptible pests will persist in the long term.*

Next, we shall consider the case $d_{C_p} \equiv 0$ (no removal of pathogenic viruses, that is). In this setting, the pathogenic viruses will remain active in the long term, as the following result asserts.

Theorem 3.6. *Assume that $d_{C_p} \equiv 0$. Further, assume that*

$$\alpha_1\beta(a) - \alpha_2 d_I(a) \geq 0. \tag{3.18}$$

Then $C_p(t)$ converges to a positive limit $C_p^* \geq C_{p_0}(0)$ and $S(t)$ tends to 0 as $t \to \infty$.

Proof. From (3.12) and (3.17), we obtain that

$$\begin{aligned} C_p(t) = C_{p_0}(0) + \frac{1}{\alpha_1}\int_0^t \int_0^h (\alpha_1\beta(a) - \alpha_2 d_I(a))I(s,a)dads \\ + \frac{\alpha_2}{\alpha_1}(N_I(0) - N_I(t)). \end{aligned}$$

Due to (3.18) and (iv) of Theorem 3.1, $C_p(t)$ has a limit C_p^* for $t \to \infty$ and $C_p^* \geq C_{p_0}(0) + \frac{\alpha_2}{\alpha_1}N_I(0)$. The asymptotic behavior of $S(t)$ for $t \to \infty$ follows now from (3.11). □

Remark 3.7. *The product terms $\alpha_1\beta(a)$ and $\alpha_2 d_I(a)$ describe the practical infection efficiency and the practical removal efficiency when the infection-age of infected pests is a. In this regard, Theorem 3.6 can be interpreted as if the pathogenic viruses are not naturally degraded and are produced fast enough, compensating infection-stage mortality, then the susceptible pest population S will become extinct in the long term.*

3.2 The linearized stability analysis

Let us recall equations (2.4)-(2.6) and consider the following reduced system

$$\begin{cases} S'(t) = -\frac{\alpha_1 C_p(t)S(t)}{1+k_1 S(t)+k_2 C_p(t)}, \\ C_p'(t) = -d_{C_p}C_p(t) + \int_0^h \frac{A(a)C_p(t-a)S(t-a)}{1+k_1 S(t-a)+k_2 C_p(t-a)}da - \frac{\alpha_2 C_p(t)S(t)}{1+k_1 S(t)+k_2 C_p(t)}, \end{cases} \tag{3.19}$$

where $A(a) = \alpha_1\beta(a)e^{-\int_0^a d_I(\xi)d\xi}$. 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 $(0,0)$ and a semi-trivial infected pest-free equilibrium $(\overline{S},0)$, where $\overline{S} = S(0)$. In the following, we shall analyze the local stability of the equilibria by analyzing the characteristic equation of system (3.19).

The characteristic equations at $(0,0)$ and $(\overline{S},0)$ have the following forms

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

and, respectively,

$$det\begin{pmatrix} \lambda & \alpha_1\overline{S} \\ 0 & \lambda + d_{C_p} + \frac{\alpha_2\overline{S}}{1+k_1\overline{S}} - \frac{\overline{S}}{1+k_1\overline{S}}\int_0^h A(a)e^{-\lambda a}da \end{pmatrix} = 0.$$

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

$$\lambda + d_{C_p} + \frac{\alpha_2\overline{S}}{1+k_1\overline{S}} - \frac{\overline{S}}{1+k_1\overline{S}}\int_0^h A(a)e^{-\lambda a}da = 0. \tag{3.20}$$

Obviously, the stability of the infected pest-free solution is determined by the sign of the real part for the complex roots of (3.20). The following preliminary result is taken into account by the argument principle known as Michailov criterion.

Lemma 3.8. *Let*

$$\Phi(\lambda) = \lambda + d_{C_p} + \frac{\alpha_2 \overline{S}}{1 + k_1 \overline{S}} - \frac{\overline{S}}{1 + k_1 \overline{S}} \int_0^h A(a) e^{-\lambda a} da$$

and let $\Delta_C Arg\Phi(\lambda)$ 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 $Arg\gamma(\mu)$) as μ varies from 0 to ∞ for a curve γ in C, i.e.,

$$\Delta = \Delta_{\mu \in [0,\infty)} Arg\gamma(\mu). \tag{3.21}$$

Suppose that

(a) *(3.20) has no pure imaginary roots;*

(b) *there exists a positive constant k^* such that $|\lambda| < k^*$ for all complex roots of (3.20) with $\operatorname{Re}\lambda > 0$.*

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

The proof of this result is given in the Appendix.

To apply Lemma 3.8, we need to prove that $\Phi(\lambda)$ satisfies conditions (a) and (b) in Lemma 3.8.

Lemma 3.9. *Suppose that one of the following conditions hold*

(a) $d_{C_p} + \frac{\alpha_2 \overline{S}}{1+k_1 \overline{S}} > \frac{\overline{S}}{1+k_1 \overline{S}} \int_0^h A(a) da$;

(b) $d_{C_p} + \frac{\alpha_2 \overline{S}}{1+k_1 \overline{S}} \neq \frac{\overline{S}}{1+k_1 \overline{S}} \int_0^h A(a) da$ *and* $\frac{\overline{S}}{1+k_1 \overline{S}} \int_0^h A(a) da < \pi$.

Then Φ has no zeros on the imaginary axis.

The proof of this result is given in the Appendix.

Lemma 3.10. *There exists a positive constant k^* such that $|\lambda| < k^*$ for all complex roots of (3.20) with $\operatorname{Re}\lambda > 0$.*

The proof of this result is given in the Appendix.

According to Lemmas 3.8–3.10, we directly obtain the following result.

Theorem 3.11. *If*

$$d_{C_p} + \frac{\alpha_2 \overline{S}}{1 + k_1 \overline{S}} > \frac{\alpha_1 \overline{S}}{1 + k_1 \overline{S}} \int_0^h \beta(a) e^{-\int_0^a d_I(\xi) d\xi} da, \tag{3.22}$$

then all possible roots of (3.20) lie in the left half-plane.

Remark 3.12. *Condition (3.22) guarantees that all real parts of eigenvalues are negative. That is, (3.22) has a stabilizing effect.*

Theorem 3.13. *If*

$$d_{C_p} + \frac{\alpha_2 \overline{S}}{1 + k_1 \overline{S}} < \frac{\alpha_1 \overline{S}}{1 + k_1 \overline{S}} \int_0^h \beta(a) e^{-\int_0^a d_I(\xi) d\xi} da < \pi, \tag{3.23}$$

then a single root lies in the right half-plane.

Proof. In fact, $\Delta = Arg\Phi(\infty i) - Arg\phi(0) = \frac{\pi}{2} - \pi = -\frac{\pi}{2}$. Hence $N^\infty = 1$. This completes the proof. □

Remark 3.14. *Condition (3.23) guarantees that the infected pest-free equilibrium is unstable. That is, (3.23) has a destabilizing effect.*

The following result demonstrates that, under some circumstances, the supremum infection age h plays an important role in dynamics of structure.

Corollary 3.15. *Suppose that the following parameters take the form*

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

for some $\epsilon>0$. Then

(a) *The equilibrium $(\overline{S},0)$ is always stable for all $h>0$, if $d_{C_p}+\frac{\alpha_2\overline{S}}{(1+k_1\overline{S})}\geq\frac{\alpha_1\overline{S}\beta}{(1+k_1\overline{S})d_I}$;*

(b) *If $\frac{\alpha_1\overline{S}\beta}{d_I(1+k_1\overline{S})}>d_{C_p}+\frac{\alpha_2\overline{S}}{(1+k_1\overline{S})}$ and $h-\epsilon<\frac{1}{d_I}\ln\frac{\alpha_1\overline{S}\beta}{\alpha_1\overline{S}\beta-d_I(d_{C_p}(1+k_1\overline{S})+\alpha_2\overline{S})}$, the equilibrium $(\overline{S},0)$ is stable;*

(c) *If $\frac{\alpha_1\overline{S}\beta}{d_I(1+k_1\overline{S})}>\max\{\pi,d_{C_p}+\frac{\alpha_2\overline{S}}{(1+k_1\overline{S})}\}$ and $\frac{1}{d_I}\ln\frac{\alpha_1\overline{S}\beta}{\alpha_1\overline{S}\beta-d_I(d_{C_p}(1+k_1\overline{S})+\alpha_2\overline{S})}<h-\epsilon<\frac{1}{d_I}\ln\frac{\alpha_1\overline{S}\beta}{\alpha_1\overline{S}\beta-d_I(1+k_1\overline{S})\pi}$, the equilibrium $(\overline{S},0)$ is unstable.*

4 Pest control strategies

Field surveys are outlined by Prasad and Wadhwani[26] 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 leaves of 6 cm diameter being 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. The histomicrograph clearly revealed that various midgut cells, fat bodies, connective tissues and integument either lost their identity or became highly disorganized. Also, at maximum period of treatment, i.e. 96 h, mortality increased from 5.43% to 78.91%, from the lowest dose (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 body is 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.

To confirm our mathematical findings and facilitate their interpretation, we proceed to investigate further by using numerical simulations (See Table 1).

Let $S(t)$ denote the number of pests in the larvae and adult stages. In each generation there is a burst

Table 1: Threshold values for the stability and instability of the equilibrium $(\overline{S}(=S(0)),0)$

$\alpha_1=0.3,\alpha_2=0.5,k_1=0.2,$ $k_2=0.3,d_I(a)\equiv1,$ $\beta(a)\equiv10/3,d_{C_p}=0.1$	*Stability of the equilibrium* $(\overline{S},0)$	*Instability of the equilibrium* $(\overline{S},0)$
$\overline{S}=100$	$h-\epsilon<0.736$	$0.736<h-\epsilon<1.077$
$\overline{S}=500$	$h-\epsilon<0.734$	$0.734<h-\epsilon<1.006$
$\overline{S}=1000$	$h-\epsilon<0.734$	$0.734<h-\epsilon<0.997$
$\overline{S}=2000$	$h-\epsilon<0.734$	$0.734<h-\epsilon<0.993$

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, suitable for describing processes within a generation([17]).

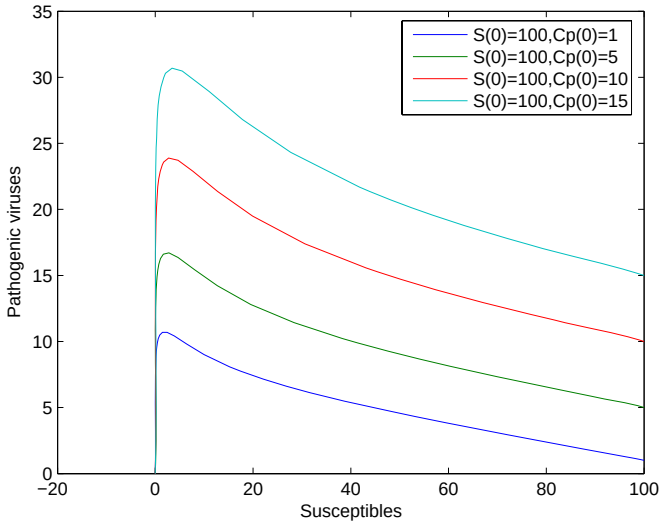

Figure 2: $\alpha_1 = 0.3$,$\alpha_2 = 0.5$,$k_1 = 0.2$,$k_2 = 0.3$, $d_I(a) \equiv 1$,$\beta(a) \equiv 10/3$,$d_{C_p} = 0.1$, $h = 1$ and $\epsilon = 0.1$. The trajectories with the same initial value $S(0)$ and different initial values $C_p(0)$.

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 Figure 2 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, if we set the economic threshold (ET)= 40, from Figure 4 (the magnified view of Figure 3), we may choose the highest applied dose of 15 to control the amount of the pest population below the given ET under the assumption that the period of treatment is 4. To make the management more economically viable, it is recommended to use the dose of 10 to control the amount of the pest population below the given ET under the assumption that the period of treatment is 6. On the other hand, Figures 3 and 4 reveal that different doses of the virus bring about significant mortality (it is fated that infected hosts should die out), which was both dose and time dependent.

From Figures 5 and 6, it is seen that a small amount of pathogenic viruses may be introduced into a target pest population in order to generate an epidemic which will subsequently lead to the death of all infected pests.

5 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, a class of pathogenic viruses 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 partial differential equation of transport type 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 2-dimensional functional differential equation with distributed delay. The asymptotic behavior of the system is 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 rate of the pathogenic viruses is 0, then the susceptible pests become extinct as well.

Next, a linearized stability analysis is performed via the use of Michailov criterion and it is found out

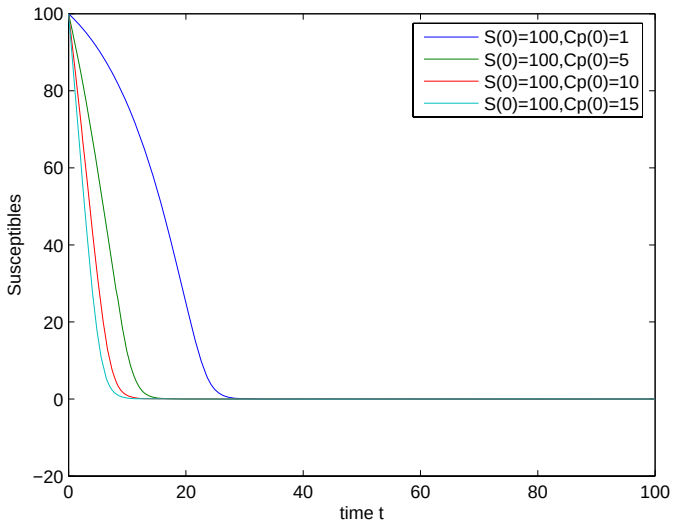

Figure 3: $\alpha_1 = 0.3$,$\alpha_2 = 0.5$,$k_1 = 0.2$,$k_2 = 0.3$, $d_I(a) \equiv 1$,$\beta(a) \equiv 10/3$,$d_{C_p} = 0.1$, $h = 1$ and $\epsilon = 0.1$. The time series for S with the same initial value $S(0)$ and different initial values $C_p(0)$.

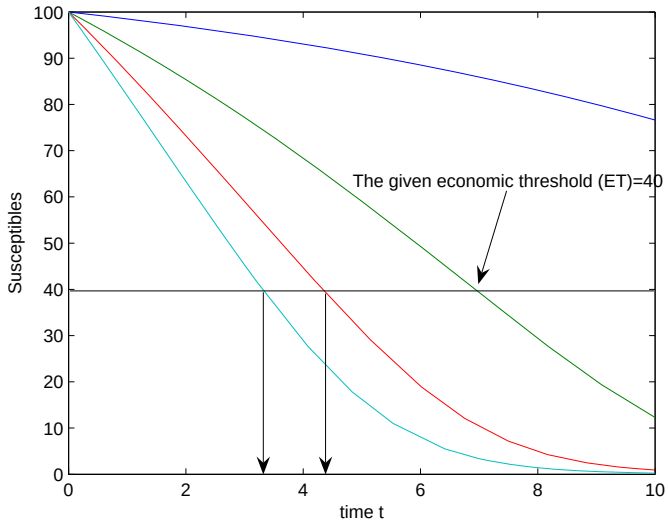

Figure 4: $\alpha_1 = 0.3$,$\alpha_2 = 0.5$,$k_1 = 0.2$,$k_2 = 0.3$, $d_I(a) \equiv 1$,$\beta(a) \equiv 10/3$,$d_{C_p} = 0.1$, $h = 1$ and $\epsilon = 0.1$. The magnified view of the time series for S with the same initial value $S(0)$ and different initial values $C_p(0)$.

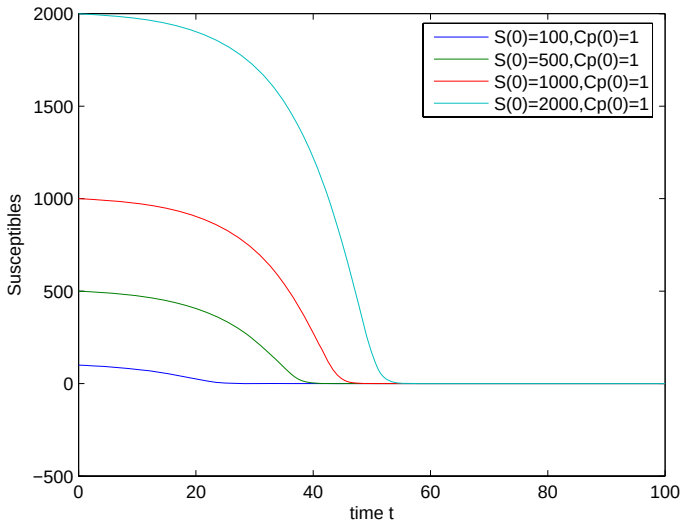

Figure 5: $\alpha_1 = 0.3$,$\alpha_2 = 0.5$,$k_1 = 0.2$,$k_2 = 0.3$, $d_I(a) \equiv 1$,$\beta(a) \equiv 10/3$,$d_{C_p} = 0.1$, $h = 1$ and $\epsilon = 0.1$. The time series for S with the same initial value $C_p(0)$ and different initial values $S(0)$.

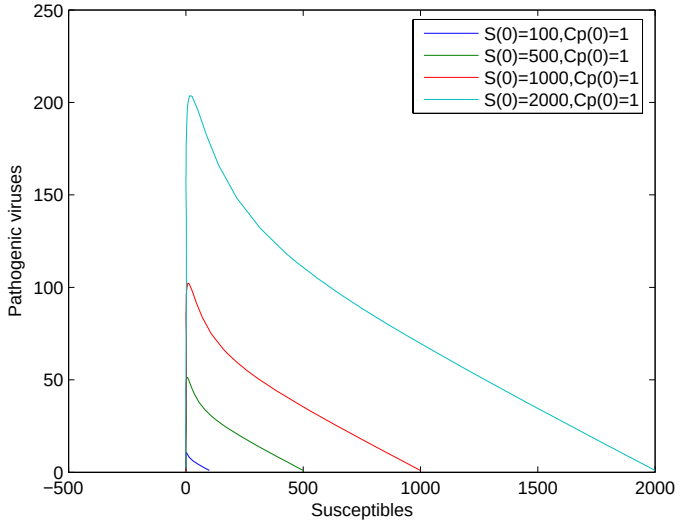

Figure 6: $\alpha_1 = 0.3$,$\alpha_2 = 0.5$,$k_1 = 0.2$,$k_2 = 0.3$, $d_I(a) \equiv 1$,$\beta(a) \equiv 10/3$,$d_{C_p} = 0.1$, $h = 1$ and $\epsilon = 0.1$. The trajectories with the same initial value $C_p(0)$ and different initial values $S(0)$.

that the maximal age of infection plays an important role important role in the dynamics of the system. In this regard, the purpose of the paper is to investigate the impact of the maximal infection age upon the stability of the system. A condition which assures the stability of the semitrivial susceptibles-only equilibrium regardless of the value of the maximal age of infection is found, while being observed that if the converse inequality is satisfied, then the susceptibles-only equilibrium may still be stable provided that the maximal age of infection is less than a certain value.

The mathematical results which are obtained in this paper may be useful for many agricultural researchers, as the use of viral pathogens 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.

References

[1] R.M. Anderson, R.M. May, *Regulation and stability of host-parasite population interactions. I. Regulatory Processes*, J. Anim. Ecol. 47 (1978), 219–247.

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

[3] R.M. Anderson, R.M. May, *Population Ecology of Infectious Disease Agents*, Theoretical Ecology Principles and Applications, 2nd Edn, 318–355, Sinaur Association, Sunderland, 1981.

[4] C.J. Briggs, H.C.J. Godfray, *The dynamics of insect-pathogen interactions in stage-structured populations*, Am. Nat. 145 (1995), 855–887.

[5] G.C. Brown, *Stability in an insect-pathogen model incorporating age-dependent immunity and seasonal host reproduction*, Bull. Math. Biol. 46 (1984), 139–153.

[6] H.D. Burgess, N.W. Hussey, *Microbial Control of Insects and Mites*, Academic Press, New York, 1971.

[7] A.J. Cherry, C.J. Lomer, D. Djegui, F. Schulthess, *Pathogen incidence and their potential as microbial control agents in IPM of maize stemborers in West Africa*, Biocontrol, 44(a) (1999), 301–327.

[8] J.S. Cory, M.L. Halis, T. Williams, *Field trial of a genetically improved baculovirus insecticide*, Nature, 370 (1994), 138–140.

[9] P.E. Davis, K. Myers, J.B. Hoy, *Biological Control Among Vertebrates*, Theory and Practice of Biological Control, 501–519, Plenum Press, New York, 1976.

[10] P. Debach, D. Rosen, *Biological Control by Natural Enemies*, 2nd edn, Cambridge University Press, Cambridge, 1991.

[11] L.A. Falcon, *Problems associated with the use of arthropod viruses in pest control*, Annu. Rev. Entomol. 21 (1976), 305–324.

[12] F. Fenner, F.N. Ratcliffe, *Myxomatosis*, Cambridge University Press, Cambridge, 1965.

[13] P. Ferron, *Pest Control Using the Fungi Beauveria and Metarhizium*, Microbial Control in Pests and Plant Diseases, 1970–1980, Academic Press, London, 1981.

[14] M.L. Flint, R. Van den Bosch, *Introduction to Integrated Pest Management*, Plenum Press, New York and London, 1981.

[15] H.J. Freedman, *Graphical stability, enrichment, and pest control by a natural enemy*, Math. Biosci. 31 (1976), 207–225.

[16] H.C.J. Godfray, C.J. Briggs, N.D. Barlow, M. O'Callaghan, T.R. Glare and T.A. Jackson, *A model of insect-pathogen dynamics in which a pathogenic bacterium can also reproduce saprophytically*, Proc. R. Soc. Lond. B 266 (1999), 233-240.

[17] B.S. Goh, *Management and Analysis of Biological Populations*, Elsevier Scientific Publishing Company, Amsterdam-Oxford-New York, 1980.

[18] J.K. Hale, *Theory of Functional Differential Equations*, Springer-Verlag, New York, 1977.

[19] W.O. Kermack, A.G. McKendrick, *A contribution to the mathematical theory of epidemics*, Proc. Roy. Soc. A 115 (1927), 700–721.

[20] W.O. Kermack, A.G. McKendrick, *Contributions to the mathematical theory of epidemics. II- The problem of endemicity*, Proc. Roy. Soc. A 138 (1932), 55–83.

[21] W.O. Kermack, A.G. McKendrick, *Contributions to the mathematical theory of epidemics. III. Further studies on the problem of endemicity*, Proc. Roy. Soc. A 141 (1933), 94–122.

[22] Y. Kuang, *Delay Differential Equations with Application in Population Dynamics*, Academic Press, San Diego, 1993.

[23] M.L. Luff, *The potential of predators for pest control*, Agri. Ecos. Environ. 10 (1983), 159–181.

[24] M.E. Martignoni, P.J. Iwai, *A catalogue of viral diseases of insects and mites*, USDA Forest Service General Technical Report PNW-40, 1977.

[25] M. Moerbeek, F. Van den Bosch, *Insect-pathogen dynamics: stage-specific and insect density dependence*, Math. Biosci. 141 (1997), 115–148.

[26] A. Prasad, Y. Wadhwani, *Pathogenic virus and insect tissues: an effective way of pest control*, Current Science 91 (2006), 803–807.

[27] Y. Tanada, *Epizootiology of Insect Disease*, Biological Control of Insect Pests and Weeds, 548–578, Chapman and Hall, London, 1964.

[28] J.M. Vlak, *Genetic engineering of baculoviruses for insect control*, Molecular approaches to Fundamental and Applied Entomology, 90–127, Springer, Berlin, 1993.

[29] H. Zhang, L.S. Chen, J.J. Nieto, *A delayed epidemic model with stage-structure and pulses for management strategy*, Nonlinear Anal.: Real World Appl. 9 (2008), 1714–1726.

[30] Y. Zhang, B. Liu, L.S. Chen, *Dynamical behavior of Volterra model with mutual interference concerning IPM*, ESAIM Math. Mod. Numer. Anal. 38 (2004), 143–155.

6 Appendix

6.1 Proof of Lemma 3.8

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

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

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

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

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

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

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

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

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

$$\begin{aligned}
\int_{C^2} \frac{\Phi'(\lambda)}{\Phi(\lambda)} d\lambda &= \int_{-\frac{\pi}{2}}^{\frac{\pi}{2}} \frac{ire^{i\theta}\Phi'(re^{i\theta})}{\Phi(re^{i\theta})} d\theta \\
&= \int_{-\frac{\pi}{2}}^{\frac{\pi}{2}} \frac{ire^{i\theta}\big(1 + \frac{\overline{S}}{1+k_1\overline{S}} \int_0^h aA(a)e^{-ar(\cos\theta + i\sin\theta)} da\big)}{re^{i\theta} + d_{C_p} + \frac{\alpha_2\overline{S}}{1+k_1\overline{S}} - \frac{\overline{S}}{1+k_1\overline{S}} \int_0^h A(a)e^{-ar(\cos\theta + i\sin\theta)} da} d\theta \\
&= \int_{-\frac{\pi}{2}}^{\frac{\pi}{2}} \frac{ie^{i\theta}\big(1 + \frac{\overline{S}}{1+k_1\overline{S}} \int_0^h aA(a)e^{-ar(\cos\theta + i\sin\theta)} da\big)}{e^{i\theta} + \frac{d_{C_p}}{r} + \frac{\alpha_2\overline{S}}{r(1+k_1\overline{S})} - \frac{\overline{S}}{r(1+k_1\overline{S})} \int_0^h A(a)e^{-ar(\cos\theta + i\sin\theta)} da} d\theta.
\end{aligned}$$

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

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

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. □

6.2 Proof of Lemma 3.9

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

$$\begin{cases} d_{C_p} + \frac{\alpha_2\overline{S}}{1+k_1\overline{S}} - \frac{\overline{S}}{1+k_1\overline{S}} \int_0^h A(a)\cos\omega a da = 0, \\ \omega + \frac{\overline{S}}{1+k_1\overline{S}} \int_0^h A(a)\sin\omega a da = 0 \end{cases}$$

Condition (a) guarantees that $\mathrm{Re}\,\Phi(i\omega) > 0$, so clearly $\Phi(i\omega) \neq 0$. The second condition ensures $\mathrm{Im}\,\Phi(i\omega) > 0$ for all $\omega > 0$ since $sin\omega a \in [-1, 1]$. Hence 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. □

6.3 Proof of Lemma 3.10

Proof. Letting $\lambda = x + yi(x > 0)$ in (3.20), we have

$$\begin{cases} x + d_{C_p} + \alpha_2 \frac{\overline{S}}{1+k_1\overline{S}} - \frac{\overline{S}}{1+k_1\overline{S}} \int_0^h \big(A(a)e^{-xa}\cos ay\big)da = 0, \\ y + \frac{\overline{S}}{1+k_1\overline{S}} \int_0^h \big(A(a)e^{-xa}\sin ay\big)da = 0 \end{cases}$$

We may choose k^* as

$$k^* = 2\big(d_{C_p} + \alpha_2\overline{S} + \overline{S}\int_0^h A(a)da\big),$$

which completes the proof. □