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The effects of nicotine sequestration on the dynamics of hyperparasitism in a stage-structured model of Manduca sexta and its related parasitoid wasps

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The effects of nicotine sequestration on the dynamics of hyperparasitism in a stage-structured model of *Manduca sexta* and its related parasitoid wasps

A thesis submitted in partial fulfillment of the requirements for the degree of Master of Science at Virginia Commonwealth University.

by

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Abstract

Two proposed models will be used to help answer a long observed question in the dynamics of *Manduca sexta* and its related parasitoid wasps-Why is there a large difference in diversity in hyperparasitoid species between tobacco and other related plants such as tomato? Two stage structured differential equation models are presented. The first is a single patch model to study the changes in dynamics that occur between hosts, parasitoids, and hyperparasitoids as the amount of nicotine in the plant increases. The second is a two patch model that allows hyperparasitoids to choose between patches that are nicotine negative (i.e. tomato plants) and nicotine positive (i.e. tobacco plants). Both models will be used to investigate how host nicotine sequestration may impact hyperparasitoid diversity.

Chapter 1

Introduction

1.1 Biological Background

A parasite requires another organism to complete its life cycle and does not necessarily cause its host to die. However, a parasitoid will cause the death of its host. Hyperparasitoids are similar except that it uses a parasitoid for its host [22]. The dynamics of host, parasitoid, and hyperparasitoid interactions are extremely complex with connections between trophic levels making them challenging to model [10]. It is not trivial to predict how the dynamics in the parasitoid and hyperparasitoid populations will be affected when the host has a defense mechanism such as sequestration of allelochemicals. *Manduca Sexta*, the tobacco hornworm, sequesters nicotine from tobacco to protect itself from predators such as the parasitoid wasp, *Cotesia congregata*, a gregarious endoparasitoid [2]. *C. congregata* is not the only parasitoid in the system; there are also higher trophic level parasitoids known as hyperparasitoids, or secondary parasitoids, that will seek out and attack primary parasitoids [21]. There are many more species of hyperparasitoids than species of parasitoid. In some systems, in the order of *Lepidoptera*, there are as many as 16 hyperparasitoid species that will attack the primary parasitoid [22]. While *C. congregata* exclusively attacks the second and third instar of the host larvae, hyper-

parasitoids vary in their ovipositing strategies. There are several species that attack the prepupal and pupal stages of the primary parasitoid species while some specialize in attacking the larval stage.

There are four trophic levels in the system considered in this thesis. The first is either the tobacco plant that produces the nicotine to defend against pests or the nicotine free tomato plant. The second trophic level is the tobacco hornworm, *Manduca sexta*, a common pest in the east coast of the United States. *M. sexta* is unaffected by the presence of nicotine and releases most of the nicotine it consumes as a mist to fend off predators [11]. However, some of the chemical remains within the hemolymph of the host and can be transferred to higher trophic levels.

The third trophic level is the parasitoid species *Cotesia congregata*. *C. congregata* is a specialist species and has adapted to the presence of nicotine in the host [2]. While in the larval stage, *Manduca sexta* is susceptible to parasitism by parasitoids including *C. congregata*. A parasitic wasp in the braconidae family, *C. congregata* oviposits its eggs inside the body of *M. sexta*. A gregarious parasitoid, a single wasp will lay a large clutch of eggs that when matured will emerge from the host and ultimately cause its death [21]. The developing parasitoids will eat the mass of the host larva absorbing some nicotine. Studies show that this nicotine stays in the larva during development and then is lost once it pupates [2]. Therefore, hyperparasitoids that attack the larvae of the primary parasitoid will be exposed to nicotine and those that attack the pupae of the parasitoids will not.

The fourth trophic level has two competing hyperparasitoids. While developing as a larva within the host it is vulnerable to parasitism by the larvae-attacking hyperparasitoids, such as the ichneumonid wasp, *Mesochorus americanus*. When *C. congregata* emerges from the host and forms its cocoon, during a pre-pupal stage, it can be parasitized by pupae-attacking hyperparasitoids such as the ichneumonid wasp *Lysibia nana* and the pteromalid wasp *Hypopteramalus tabacum*. *L. nana* is a solitary parasitoid wasp

that will lay a single egg within the prepupae in the prepupal cocoon of the parasitoid [11].

Here we use mathematical models to investigate the dynamics of nicotine sequestration on the populations of the hosts, parasitoids and hyperparasitoids described above. Diversity of hyperparasitoids is lower on tobacco plants and the larvae-attacking species of hyperparasitoid do not emerge from any *C. congregata* cocoons (Karen Kester, Pers. Comm.). Mathematical models will be developed and used to investigate how host nicotine sequestration may impact hyperparasitoid diversity.

1.2 Model Background

Host and parasitoid systems were first modeled by Nicholson and Bailey in 1935 [17]. The inclusion of hyperparasitoids was first done by Beddington and Hammond [3], in discrete time with no developmental stage-structure. Later Briggs [4] developed a model for hosts and parasitoids using delay differential equations, incorporating host stage structure, and competition between multiple parasitoid species [4, 5]. The proposed model in this paper is a differential equation model incorporating stage-structure in each of the trophic levels involved: host, parasitoid, and hyperparasitoids.

Modeling host-parasitoid-hyperparasitoid populations is similar to modeling predator-prey interactions since parasitoids remove their host from the population as a predator would. This parallel allows for a continuous time model of the interactions of the different trophic levels with interesting dynamics not seen in the Beddington and Hammond discrete-time model [3, 16]. By removing the discrete time steps the populations of the host, parasitoid, and hyperparasitoid can change continuously with the change in population density. This allows us to see changing dynamics within a single generation [15, 14].

The first model proposed in this thesis will be a continuous time, stage-structured

model to study the hosts, parasitoids, and hyperparasitoids on a single "patch" where there is only one species of plant. The plants will either be a nicotine producing plant, such as tobacco, or a nicotine free plant, such as tomato. The single patch model will be analyzed for equilibria, the stability of those equilibria, and invasion criteria for competing hyperparasitoid species. By changing the plant species, we explore the effect of nicotine in the system on the stability of model equilibria and conditions for coexistence of competing hyperparasitoids. The second model proposed is a two patch model developed in the same manner as the single patch except that both species of plants will be present. Each patch will contain a different species of plant and independent populations of hosts and parasitoids. The hyperparasitoid adults will be able to move between patches and decide where to search for parasitoids. The two patch model will be used to optimize patch choice after developing equations for measuring individual hyperparasitoid fitness. Optimal patch choice will be determined for individual populations of hyperparasitoids and increasing effect of nicotine.

Chapter 2

Single Patch Model

In this chapter we develop a model to describe the dynamics of a system of hosts, parasitoids, and two competing hyperparasitoids on a single patch, or plant. We use this model to compare the differences in population dynamics as the amount of nicotine in a plant changes. The patch can be considered to be a non-nicotine producing plant or a nicotine producing plant in which the larvae-attacking hyperparasitoids will have a higher death rate than the pupae-attacking species. Without any consequences of nicotine the larvae-attackers will have a clear advantage over the pupae-attackers as they have the first opportunity to parasitize their host. However, the pupae-attackers have a survival advantage on nicotine-producing plants. There are three possible outcomes of the hyperparasitoid interactions: one species will persist and force exclusion of their competitor, both species will live in coexistence, or neither species will survive.

2.1 Host Only Model

We first consider a continuous time differential equation model for the host, *Manduca sexta*, alone. Like most insects *M. sexta* goes through four stages: egg, larval, pupal, and adult. We model all stages except the egg stage: larvae (L), pupae (P), and adults (A).

Eggs are laid by adults and hatch into larvae at a rate b times the density limiting

factor $1 - \frac{L}{K}$. Larvae can leave the stage by either maturing or dying naturally. Larvae mature at the rate g_L and die at the rate μ_L. The pupal population increases as larvae mature into pupae and decreases as pupae mature into adults at rate g_P. The pupal death rate is negligible and therefore not included in our model. The adult population increases as pupae mature into adults and decreases only through natural death at rate μ_A.

The host model equations are as follows:

$$\begin{aligned} \frac{dL}{dt} &= b\left(1 - \frac{L}{K}\right)A - \mu_L L - g_L L \\ \frac{dP}{dt} &= g_L L - g_P P \\ \frac{dA}{dt} &= g_P P - \mu_A A \end{aligned} \tag{2.1}$$

Parameter values for the host only model are given in Table 2.1. The egg stage lasts 2-8 days (on average 5 days). This is taken into account when determining the parameter b, the rate at which larvae are hatched. The larval stage has five or six instars. The first few instars rapidly grow while the final instars take about twice as long. The average total time in the larval stage is approximately 26 days. Maturation parameters were determined by the inverse of the mean lifespan in inverse days, i.e. $g_L = \frac{1}{26}$ days^{-1} is the maturation rate of host larvae into pupae. Mortality rates were calculated from data on the percent of individuals that successfully reached the next stage by solving for μ_L in the following equation,

$$P_L = \frac{g_L}{g_L + \mu_L},$$

where P_L is the percentage of host larvae that survive to reach the pupal stage. Other maturation and mortality rates were calculated in a similar manner from the values presented below.

Additionally, host population sizes are limited by larval density due to limited space and resources on the plant. The limitation on larval density is included in the model by

the parameter, K. The pupal stage lasts approximately 19 to 23 days (average of 21 days) until the adult moth emerges [18]. Adult females are able to lay eggs within a week of emerging. Adults are rather long lived with an estimated lifespan of 2 months.

Parameter	Parameter meaning	Estimated Value	Citation
b	rate of egg surviving to larva	3.3	Sasaki [20], Kingsolver [13]
K	limiting factor on larval density	$K >> 0$	
μ_L	mortality rate of larva	$\frac{1}{100}$	Kingsolver [13]
μ_A	mortality rate of adults	$\frac{1}{60}$	Covell [6]
g_L	maturation rate of larva	$\frac{1}{26}$	Harvey et al.[11], Reinecke et al. [18]
g_P	maturation rate of pupa	$\frac{1}{21}$	Reinecke et al. [18]

Table 2.1 Host parameters

2.1.1 Host Model Equilibria

The host only model has two equilibrium points: the extinction equilibrium, $(0, 0, 0)$, and non-extinction equilibrium, (L^*, P^*, A^*), where $L^* = \frac{K(bg_L - \mu_A\mu_L - \mu_A g_L)}{bg_L}$, $P^* = \frac{K(bg_L - \mu_A\mu_L - \mu_A g_L)}{bg_P}$, and $A^* = \frac{K(bg_L - \mu_A\mu_L - \mu_A g_L)}{b\mu_A}$. Since all parameters are positive in order for all components of the non-extinction equilibrium to be positive, we must have $bg_L - \mu_A\mu_L - \mu_A g_L > 0$, which can be rewritten as:

$$b\left(\frac{1}{\mu_A}\right)\left(\frac{g_L}{\mu_L + g_L}\right) > 1. \tag{2.2}$$

To determine the stability of each equilibrium we look at the eigenvalues of the host-only Jacobian matrix (A.1) evaluated at the equilibrium point. If all eigenvalues of the Jacobian are negative, then the equilibrium is stable. If there exists at least one positive eigenvalue than the equilibrium point is unstable.

The characteristic equation for the Jacobian matrix evaluated at the extinction equi-

librium (A.2) is:

$$\lambda^3 + (\mu_A + g_P + \mu_L + g_L)\lambda^2 + ((g_L + \mu_A + \mu_L)g_P + \mu_A(\mu_L + g_L))\lambda + ((\mu_L + g_L)\mu_A - g_L b)g_P = 0.$$

Using the Routh-Hurwitz criteria for stability the coefficients must all be the same sign. If any one coefficient sign differs, then there are roots, or eigenvalues, that are of different signs guaranteeing a positive eigenvalue and an unstable equilibrium. For the host only system the coefficients are $a_3 = 1$, $a_2 = (\mu_A + g_P + \mu_L + g_L)$, $a_1 = ((g_L + \mu_A + \mu_L)g_P + \mu_A(\mu_L + g_L))$, $a_0 = ((\mu_L + g_L)\mu_A - g_L b)g_P$. Since all parameters are positive, then it is clear that $a_3 > 0$, $a_2 > 0$, and $a_1 > 0$. If the criterion (2.2) for the non-extinction equilibrium to be positive holds, then $a_0 < 0$. Therefore, by the Routh-Hurwitz criteria, since one of the coefficients of the characteristic equation is negative and there is at least one positive coefficient, there exists at least one eigenvalue with zero or positive real parts. Therefore, the extinction equilibrium will be unstable when (2.2) is met. If (2.2) fails to hold and the non-extinction equilibrium is not biologically relevant, then all coefficients of the characteristic equation are positive. Under the Routh-Hurtwitz criteria, the next condition needed for stability is $a_1 a_2 > a_0$. If $a_2 > 0$, and $a_1 > 0$, then $a_1 a_2 > 0$. If $a_0 < 0$, then by substitution $a_1 a_2 > 0 > a_0$. Therefore the second Routh-Hurwitz criteria is met and the extinction equilibrium is stable under the criterion (2.2).

The characteristic equation for the Jacobian matrix evaluated at the non-extinction equilibrium (A.3) is given by:

$$\lambda^3 + \frac{b g_L + g_P \mu_A + \mu_A}{\mu_A}\lambda^2 + \frac{b g_L g_P + b g_L \mu_A + g_P \mu_A^2}{\mu_A}\lambda + g_P(b g_L - \mu_A g_L - \mu_A \mu_L) = 0$$

If condition (2.2) is met, then all coefficients are positive. If $a_1 a_2 > a_0$ is true, then the equilibrium point is stable. With $a_2 = \frac{b g_L + g_P \mu_A + \mu_A}{\mu_A}$, $a_1 = \frac{b g_L g_P + b g_L \mu_A + g_P \mu_A^2}{\mu_A}$, and

$a_0 = g_P(bg_L - \mu_A g_L - \mu_A \mu_L)$, this reduces to

$$\frac{g_P \mu_A{}^4 + \left(g_P{}^2 + (g_L + \mu_L) g_P + b g_L\right) \mu_A{}^3 + 2 b g_L g_P \mu_A{}^2 + \left(b^2 g_L{}^2 + b g_L g_P{}^2\right) \mu_A + b^2 g_L{}^2 g_P}{\mu_A{}^2} > 0$$

which is true since all parameters are positive. Thus, the non-extinction equilibrium in the host-only model is stable for all parameters sets for which (2.2) holds and the non-extinction equilibrium is positive.

2.2 Host-Parasitoid Model

In this section we extend the model to include the four stages of parasitoids. All four stages of the parasitoid are explicitly modeled: eggs, larvae , pupae, and adults.

The density of parasitoid eggs, P_E, increases as eggs are successfully oviposited into host larvae. We assume the rate of successful oviposition, α, is jointly proportional to the density of parasitoid adults (P_A) and the density of host larvae (L). Parasitoid eggs mature into parasitoid larvae (P_L) at rate g_{P_E}. Parasitoid eggs are also subject to natural mortality at rate μ_{P_E}. Parasitoid larvae mature into pupae at the rate g_{P_L} and die at the rate μ_{P_L}. Parasitoid pupae mature into adults at rate g_{P_P} and die at the rate μ_{P_P} Parasitoid adults have a natural mortality rate of μ_{P_A}.

When the equations (2.1) are coupled with the parasitoid equations the host-parasitoid

model equations are as follows:

$$\begin{aligned}
\frac{dL}{dt} &= b\left(1-\frac{L}{K}\right)A - \mu_L L - g_L L - \alpha P_A L \\
\frac{dP}{dt} &= g_L L - g_P P \\
\frac{dA}{dt} &= g_P P - \mu_A A \\
\frac{dP_E}{dt} &= \alpha P_A L - \mu_{P_E} P_E - g_{P_E} P_E \\
\frac{dP_L}{dt} &= g_{P_E} P_E - g_{P_L} P_L - \mu_{P_L} P_L \\
\frac{dP_P}{dt} &= g_{P_L} P_L - g_{P_P} P_P - \mu_{P_P} P_P \\
\frac{dP_A}{dt} &= g_{P_P} P_P - \mu_{P_A} P_A
\end{aligned} \tag{2.3}$$

The parasitoid *C. congregata* attacks the second and third instar of the host larvae. For simplicity the model does not differentiate between instars and the parasitoid is allowed to attack any hosts in the larval stage. *C. congregata* is also gregarious which means it lays a large clutch of eggs rather than a single egg. The difference is taken into account by the parameter α which will be an order of magnitude larger than the successful oviposition rate of the hyperparasitoids. Once the eggs are oviposited within the host larvae they will hatch after 4 days. Following the same method used for host parameters the maturation rate of parasitoid eggs is $g_{P_E} = \frac{1}{4}$. Assuming fifty percent survival rate gives $\mu_{P_E} = \frac{1}{4}$. The larva of *C. congregata* will then mature in the host and after 10-12 days escape from the host and form cocoons, therefore $g_{P_L} = \frac{1}{11}$. The adults will emerge from the cocoons after 4-8 days, so we let $g_{P_P} = \frac{1}{6}$. Pupal mortality is low enough to be negligible, so we assume $\mu_{P_P} = 0$. [2, 23]. The adult parasitoids live about 4 and half days, so $\mu_{P_A} = \frac{2}{9}$ [8, 9].

2.2.1 Host-Parasitoid Model Equilibria

Setting the equations (2.3) to zero and solving yields three equilibria:

Parameter	Parameter meaning	Estimated Value	Citation
α	rate of successful oviposition of the primary parasitoid	$\alpha > 0$	
g_{P_E}	maturation rate of parasitoid eggs	$\frac{1}{4}$	Reinecke et al. [18]
g_{P_L}	maturation rate of parasitoid larvae	$\frac{1}{11}$	Barbosa et al. [2]
g_{P_P}	maturation rate of parasitoid pupae	$\frac{1}{6}$	Thorpe and Barbosa [2, 23]
μ_{P_E}	mortality rate of parasitoid eggs	$\frac{1}{4}$	set for 50% survival
μ_{P_L}	mortality rate of parasitoid larvae	$\frac{7}{600}$	Thorpe and Barbosa [23]
μ_{P_P}	mortality rate of parasitoid pupae	0	Thorpe and Barbosa [23]
μ_{P_A}	mortality rate of parasitoid adults	$\frac{2}{9}$	Dhammi[8], Fulton[9]

Table 2.2 Parasitoid parameters

$$(0, 0, 0, 0, 0, 0, 0) \tag{2.4}$$

$$(L^*, P^*, A^*, 0, 0, 0, 0) \tag{2.5}$$

$$(L^*, P^*, A^*, P_E^*, P_L^*, P_P^*, P_A^*) \tag{2.6}$$

The first equilibrium is the extinction equilibrium point (2.4) where all components are zero. This extinction equilibrium is stable exactly when the extinction equilibrium of the host only model is stable. Parasitoids cannot persist in the absence of the host and also must go extinct. The second is the host-only equilibrium point (2.5), where L^*, P^*, and A^* are identical to the equilibrium point in the host-only model and all stages of the parasitoid population are zero. The third equilibrium point is the coexistence equilibrium point (2.6) where both the host and the parasitoids coexist. The equilibrium values at this steady state are as follows:

$$L^* = \frac{\mu_{P_A}(g_{P_P} + \mu_{P_P})(g_{P_L} + \mu_{P_L})(g_{P_E} + \mu_{P_E})}{\alpha g_{P_E} g_{P_L} g_{P_P}},$$

$$P^* = \frac{g_L \mu_{P_A}(g_{P_P} + \mu_{P_P})(g_{P_L} + \mu_{P_L})(g_{P_E} + \mu_{P_E})}{\alpha g_P g_{P_E} g_{P_L} g_{P_P}},$$

$$A^* = \frac{g_L \mu_{P_A}(g_{P_P} + \mu_{P_P})(g_{P_L} + \mu_{P_L})(g_{P_E} + \mu_{P_E})}{\alpha g_{P_E} g_{P_L} g_{P_P} \mu_A},$$

$$P_E^* = \frac{\alpha L^* P_A^*}{g_{P_E} + \mu_{P_E}},$$

$$P_L^* = \frac{\mu_{P_A}(g_{P_P} + \mu_{P_P})}{g_{P_L} g_{P_P}} P_A^*$$

$$P_P^* = \frac{\mu_{P_A}}{g_{P_P}} P_A^*,$$

$$P_A^* = \left(\frac{1}{\alpha}\right)\left(\frac{b g_L - \mu_A(g_L + \mu_L)}{\mu_A} - \frac{b}{K} A^*\right).$$

When evaluated at the host-only equilibrium (2.5), the Jacobian matrix of model (2.3) has a block structure where the upper 3×3 block is the same as the original host-only model Jacobian evaluated at the non-extinction equilibrium which was determined to have all eigenvalues with negative real part as long as the equilibrium point was positive (A.4). The lower 4×4 block (A.5) of the Jacobian matrix evaluated at the second equilibrium point (2.5) can be tested using the same Routh-Hurwitz criteria.

Under the Routh-Hurwitz criteria, for a four dimensional system to have all negative eigenvalues, all the coefficients of the characteristic equation must be positive. For the lower block the characteristic equation has three coefficients that are strictly positive $a_3 > 0$, $a_2 > 0$, $a_1 > 0$. However, the constant coefficient is not always positive:

$$a_0 = ((\mu_{P_A}\,(g_{P_P} + \mu_{P_P})\,(gpl + \mu_{PL})\,(g_{P_E} + \mu_{P_E}) - \alpha\, g_{P_E}\, g_{P_L}\, g_{P_P}\, K)\, b + g_{P_P}\, g_{P_L}\, g_{P_E}\, \alpha\, K \mu_A)\, g_L$$
$$+ \mu_A\, \mu_L\, K \alpha\, g_{P_E}\, g_{P_L}\, g_{P_P}$$

Requiring $a_0 < 0$ results in the criteria:

$$\alpha L^* \left(\frac{g_{P_E}}{g_{P_E} + \mu_{P_E}}\right)\left(\frac{g_{P_L}}{g_{P_L} + \mu_{P_L}}\right)\left(\frac{g_{P_P}}{g_{P_P} + \mu_{P_P}}\right)\left(\frac{1}{\mu_{P_A}}\right) > 1. \tag{2.7}$$

This is the same as the invasion criteria, the criteria necessary for parasitoids to grow from low initial numbers and persist in the presence of a stable host population. The invasion criteria is can be interpreted as the total number of offspring of a parasitoid $\alpha L^* \left(\frac{1}{\mu_{P_A}}\right)$, multiplied by the probability that the larvae will survive to adulthood $\left(\frac{g_{P_E}}{g_{P_E}+\mu_{P_E}}\right)\left(\frac{g_{P_L}}{g_{P_L}+\mu_{P_L}}\right)\left(\frac{g_{P_P}}{g_{P_P}+\mu_{P_P}}\right)$. When the left hand side of (2.7) is greater than one each adult parasitoid produces more than one adult parasitoid and the parasitoid population persists. Therefore, if criterion (2.7) is met and the parasitoid can invade a population of hosts, then the host-only equilibrium (2.5) was unstable. The coexistence equilibrium (2.6) may be stable, or the hosts and parasitoids may enter into a limit cycle; this has been observed to occur in simulations for certain parameter values and has also been seen in previous models such as the Nicholson and Bailey model [17, 14, 10].

Due to the complexity of the model, further stability analysis will be done numerically using the parameter values in Tables 2.1 and 2.2. When the Jacobian is evaluated at the positive equilibrium point (2.6) with the parasitoid oviposition rate, $\alpha = 0.02$, then all real eigenvalues of the matrix are negative. Thus the equilibrium is stable.

2.3 Host-Parasitoid-Hyperparasitoid Model

In this section we include two competing hyperparasitoid species to produce the final model. Each hyperparasitoid species will have two stages: larvae and adults. H_L represents the density of the larvae of the larvae-attacking hyperparasitoids and H_A represents the density of the adults of the larvae-attacking hyperparasitoids. Similarly, $\hat{H}_L$ and $\hat{H}_A$ are the density of the larvae and adults of the pupae-attacking hyperparasitoids respectively. The parasitoid model does not include an explicit prepupal stage so for

the sake of simplicity the prepupal hyperparasitoid, *H. tabacum*, will be referred to as a pupae-attacking hyperparasitoid, $\hat{H}$. While *M. americanus* is a larval-pupal hyperparasitoid, the species will be referred to as the larvae-attacking hyperparasitoid species, H. Note that any parasitoid larvae that have been parasitized by H cannot be parasitized by the species $\hat{H}$ that attacks a later stage.

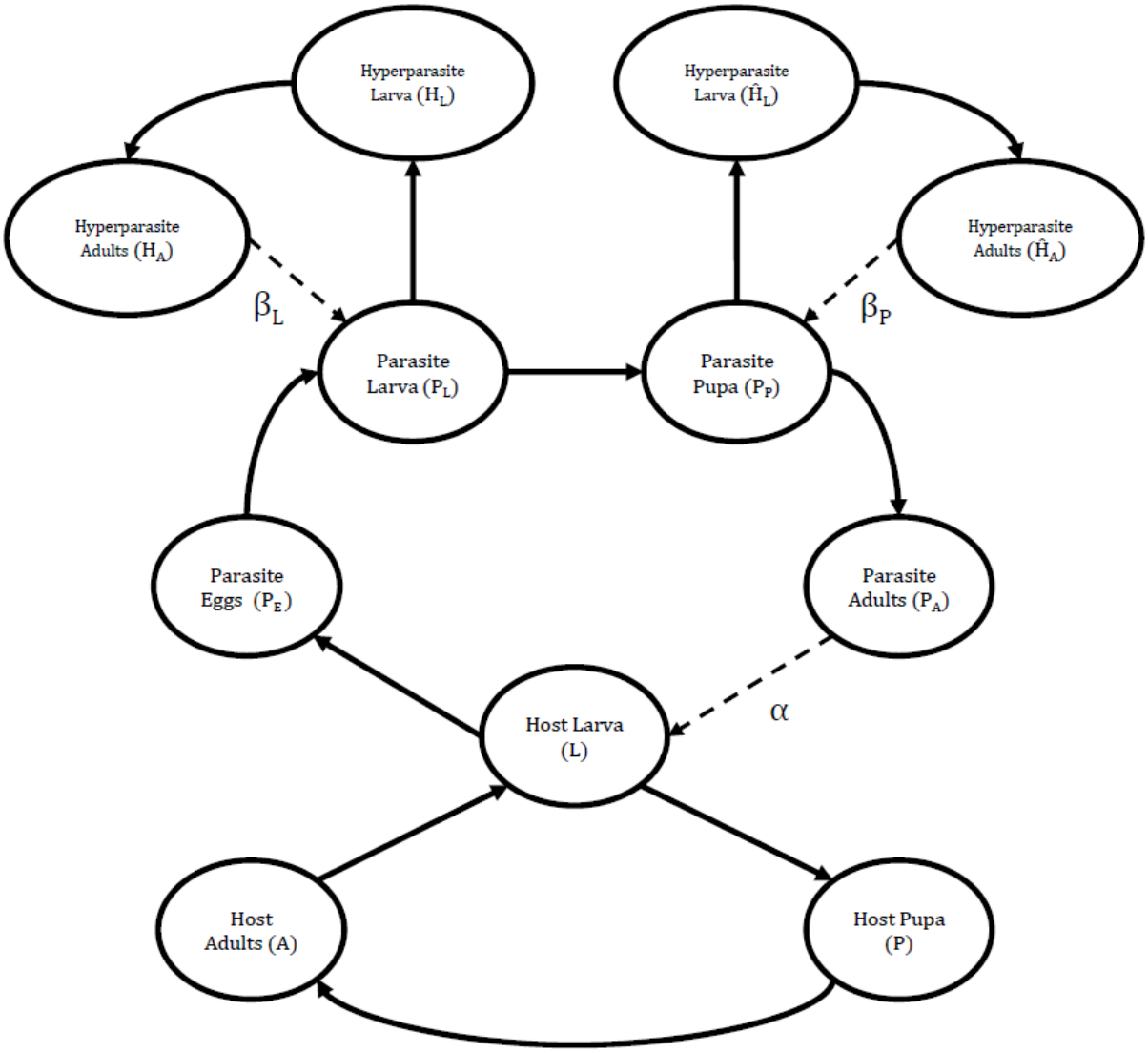

Figure 2.1 Flow diagram illustrating the movement of individuals in the population. Solid lines indicate maturation and dashed lines indicate parasitism.

The density of larvae-attacking hyperparasitoid larvae, H_L, increases as eggs are suc-

cessfully oviposited into parasitoid larvae. We assume the rate of successful oviposition, β_L, is jointly proportional to the density of the larvae-attacking hyperparasitoid adults, H_A, and the density of the parasitoid larvae, P_L. The larvae will mature into adults of the same species at rate g_{H_L} and will be lost due to mortality by the rate μ_{H_L}.

The density of pupae-attacking hyperparasitoid larvae, $\hat{H}_L$, increases as eggs are successfully oviposited into parasitoid pupae. We assume the rate of successful oviposition, β_L, is jointly proportional to the density of larvae-attacking hyperparasitoid adults, $\hat{H}_A$, and the density of parasitoid pupae, P_P. The larvae will mature into adults of the same species at rate $g_{\hat{H}_L}$ and will be lost due to mortality by the rate $\mu_{\hat{H}_L}$.

Once the hyperparasitoid equation are coupled with (2.3) we obtain the complete host-parasitoid-hyperparasitoid model:

$$\begin{aligned}
\frac{dL}{dt} &= b\left(1-\frac{L}{K}\right)A - \mu_L L - g_L L - \alpha P_A L \\
\frac{dP}{dt} &= g_L L - g_P P \\
\frac{dA}{dt} &= g_P P - \mu_A A \\
\frac{dP_E}{dt} &= \alpha P_A L - \mu_{P_E} P_E - g_{P_E} P_E \\
\frac{dP_L}{dt} &= g_{P_E} P_E - g_{P_L} P_L - \mu_{P_L} P_L - \beta_L H_A P_L \\
\frac{dP_P}{dt} &= g_{P_L} P_L - g_{P_P} P_P - \mu_{P_P} P_P - \beta_P \hat{H}_A P_P \\
\frac{dP_A}{dt} &= g_{P_P} P_P - \mu_{P_A} P_A \\
\frac{dH_L}{dt} &= \beta_L H_A P_L - g_{H_L} H_L - \mu_{H_L} H_L \\
\frac{dH_A}{dt} &= g_{H_L} H_L - \mu_{H_A} H_A \\
\frac{d\hat{H}_L}{dt} &= \beta_P \hat{H}_A P_P - g_{\hat{H}_L} \hat{H}_L - \mu_{\hat{H}_L} \hat{H}_L \\
\frac{d\hat{H}_A}{dt} &= g_{\hat{H}_L} \hat{H}_L - \mu_{\hat{H}_A} \hat{H}_A
\end{aligned} \tag{2.8}$$

While there is little data collected for rates of hyperparasitoid maturation, much of their biology is similar to that of their hosts, the primary parasitoids [12]. Hyperpar-

asitoids live longer than their parasitoid hosts. In the model the parameters for the hyperparasitoids are kept close to that of the parasitoids because an accurate lifespan could not be determined. All hyperparasitoids are considered obligate and solitary. Obligate hyperparasitoids are exclusively at a higher trophic level and cannot act as primary parasitoids. Solitary means that they only lay one egg per oviposition [22, 11]. Nicotine will only affect the survival of the larvae-attacking hyperparasitoid larvae, increasing their death rate, μ_{H_L}, relative to a nicotine-free environment. We assume that larvae of the hyperparasitoids that attack the pre-pupal/pupal stage are not affected by higher levels of nicotine [11]. This is because nicotine levels in parasitoids are higher in the larval stage before the cocoon is formed and most of the nicotine is removed due to the formation of the cocoon as the parasitoid enters the pupal stage [2].

Hyperparasitoid Parameters			
Parameter	Parameter meaning	Estimated Value	Citation
β_L	rate of successful oviposition of larval-attacking hyperparasitoids	$\beta_L > 0$	
β_P	rate of successful oviposition of pupae-attacking hyperparasitoids	$\beta_P > 0$	
g_{H_L}	maturation rate of hyperparasitoid larva	$\frac{4}{45}$	Harvey et al.[11]
$g_{\hat{H}_L}$	maturation rate of hyperparasitoid larva	$\frac{4}{45}$	Harvey et al. [11]
μ_{H_L}	mortality rate hyperparasitoid larva	varying	$\mu_{H_L} \leqslant \mu_{\hat{H}_L}$
μ_{H_A}	mortality rate of hyperparasitoid adults	$\frac{2}{9}$	$\mu_{H_A} \leqslant \mu_{P_A}$
$\mu_{\hat{H}_L}$	mortality rate of hyperparasitoid larva	$\frac{1}{45}$	Harvey et al. [11]
$\mu_{\hat{H}_A}$	mortality rate of hyperparasitoid adults	$\frac{2}{9}$	$\mu_{H_A} \leqslant \mu_{P_A}$

Table 2.3 Hyperparasitoid parameters

2.3.1 Host, Parasitoid, and Hyperparasitoid Model Equilibria

There are six equilibrium points for this model:

Extinction:

$$(0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0) \tag{2.9}$$

Hosts only:

$$(L^*, P^*, A^*, 0, 0, 0, 0, 0, 0, 0, 0) \tag{2.10}$$

Hosts and Parasitoids:

$$(L^*, P^*, A^*, P_E^*, P_L^*, P_P^*, P_A^*, 0, 0, 0, 0) \tag{2.11}$$

Hosts, Parasitoids, and Larvae-attacking Hyperparasitoids:

$$(L^*, P^*, A^*, P_E^*, P_L^*, P_P^*, P_A^*, H_L^*, H_A^*, 0, 0) \tag{2.12}$$

Hosts, Parasitoids, and Pupae-attacking Hyperparasitoid:

$$(L^*, P^*, A^*, P_E^*, P_L^*, P_P^*, P_A^*, 0, 0, \hat{H}_L^*, \hat{H}_A^*) \tag{2.13}$$

Coexistence of hosts, parasitoids, and both hyperparasitoids:

$$(L^*, P^*, A^*, P_E^*, P_L^*, P_P^*, P_A^*, H_L^*, H_A^*, \hat{H}_L^*, \hat{H}_A^*) \tag{2.14}$$

There is never an equilibrium point where parasitoids and/or hyperparasitoids are present at positive levels in the absence of the host, since the host is necessary for these populations to survive. Also there are no equilibrium points where hyperparasitoids exist without parasitoids.

We focus our analysis of this model on the stability of the three equilibria containing hyperparasitoids (2.12), (2.13), (2.14). There are two exclusion equilibria where only one hyperparasitoid species exists and the coexistence equilibrium where both hyperparasitoid populations exist. We hypothesize that the presence of nicotine may cause the coexistence equilibria to go unstable and lead to the exclusion of the larvae-attackers.

2.4 Invasion Criteria

We are interested in the conditions that change the two competing hyperparasitoids' coexistence. In this section we derive criteria for when one type of hyperparasitoid can invade the system if the other type is present at equilibrium. The species that is

already present is referred to as the 'resident' while the species that is being introduced is referred to as the 'invader.' If a population without the invader is at an equilibrium that is unstable, then when a small number of invaders is introduced their population will grow. This may result in coexistence or extinction of the resident [16].

Invasion criteria for pupae-attacking hyperparasitoids

If larvae-attacking hyperparasitoids are the resident species and are at equilibrium (2.12), the criterion for pupae-attacking hyperparasitoids to invade is:

$$\beta_P \left(\frac{g_{\hat{H}_L}}{g_{\hat{H}_L} + \mu_{\hat{H}_L}} \right) \left(\frac{1}{\mu_{\hat{H}_A}} \right) P_P^* > 1 \tag{2.15}$$

where P_P^* is the population density of the parasitoid pupae at the stable equilibrium and,

$$P_P^* = \frac{g_{P_L} \mu_{H_A} (g_{H_L} + \mu_{H_L})}{\beta_L g_{H_L} (g_{P_P} + \mu_{P_P})}$$

If an individual from the invading species can produce more than one adult in its lifetime than it is possible for the species to invade. This is determined by the total number of offspring for the parasitoid which is proportional to the successful attack rate, β_P, times the lifespan of the individual, $\left(\frac{1}{\mu_{\hat{H}_A}} \right)$, and the density of the parasitoid pupae at equilibrium, P_P^*. The total number of offspring is then multiplied by the probability they will survive to adulthood, $\left(\frac{g_{\hat{H}_L}}{g_{\hat{H}_L} + \mu_{\hat{H}_L}} \right)$. The total number of offspring for the invading species is dependent on the density of their host at the stage they attack, for this case it is the parasitoid pupal stage, P_P^*. However, that density is dependent on the resident species and its ability to suppress their hosts.

If all parameters between the hyperparasitoid populations were equal and P_P^* is substituted into (2.15) results in:

$$g_{P_L} \left(\frac{1}{g_{P_P} + \mu_{P_P}} \right) > 1$$

This means that the pupal stage attacking hyperparasitoid can invade if the maturation rate of the primary parasitoid larva, g_{P_L}, is greater than the rate at which parasitoid pupa are disappearing, $g_{P_P} + \mu_{P_P}$, either through maturation or death. Since the pupae-attacking hyperparasitoids are trying to invade into a population in which the competitor with the advantage already exists it would follow that in order to be successful they would require their resource to be more available than for the larvae-attacking hyperparasitoids.

The effects of nicotine are assumed to increase the rate of mortality of the larvae-attacking hyperparasitoid larva, μ_{H_L}. The only location of this parameter in the invasion criteria (2.15) is in the numerator of P_P^*, the parasitoid pupae population density. By lowering or raising μ_{H_L} we are respectively lowering or raising the invasion criteria (2.15). Thus the effects of nicotine increase the pupae-attackers' ability to invade.

Invasion criteria for larvae-attacking hyperparasitoids:

If pupae-attacking hyperparasitoids are the resident species and are at equilibrium, the criterion for larvae-attacking hyperparasitoids to be able to invade is:

$$\beta_L \left(\frac{g_{H_L}}{g_{H_L} + \mu_{H_L}} \right) \left(\frac{1}{\mu_{H_A}} \right) P_L^* > 1 \tag{2.16}$$

with the equilibrium point of the parasitoid larva in the pupae-attacking hyperparasitoid only system:

$$P_L^* = \frac{\left(\left((b g_L - \mu_A (g_L + \mu_L)) \beta_P \mu_{P_A} - \mu_{\hat{H}_A} g_{P_P} \alpha \mu_A \right) g_{\hat{H}_L} - \mu_{\hat{H}_A} g_{P_P} \alpha \mu_A \mu_{\hat{H}_L} \right) K g_{P_E} \left(g_{\hat{H}_L} + \mu_{\hat{H}_L} \right) \mu_{\hat{H}_A} g_{P_P} \alpha}{\beta_P{}^2 b g_L \, g_{\hat{H}_L}{}^2 \mu_{P_A}{}^2 (g_{P_L} + \mu_{P_L}) (g_{P_E} + \mu_{P_E})}.$$

With substitutions from the equilibrium point (2.13) the equilibrium of the pupae-attacking hyperparasitoid only system:

$$P_L^* = \left(\frac{g_{P_E}}{g_{P_E} + \mu_{P_E}} \right) \left(\frac{1}{g_{P_L} + \mu_{P_L}} \right) (\alpha P_A^* L^*)$$

If all parameters were equal between the two hyperparasitoid species the determining criteria would reduce to:

$$\left(\frac{g_{P_E}}{g_{P_E}+\mu_{P_E}}\right)\left(\frac{1}{g_{P_L}+\mu_{P_L}}\right)\left(\frac{1}{\mu_{P_A}}\right)\alpha g_{P_P} L^* > 1$$

The criteria is heavily dependent on the abundance of parasitoids in the particular stage that the invader requires for reproduction. In (2.15) the resource was the pupae of the parasitoid species. So long as the pupae were around longer than the larvae it allows the pupae-attacking hyperparasitoid to invade. When resident and invader are reversed and the invader is now dependent on the abundance of larvae the criteria changes to suit that resource. The criteria (2.16) depends on the total eggs laid by parasitoids, $\alpha L^*\left(\frac{1}{\mu_{P_A}}\right)$, multiplied by the probability they survive to the larval stage, $\frac{g_{P_E}}{g_{P_E}+\mu_{P_E}}$, and further multiplied by time spent as larva proportional to pupa, $\frac{g_{P_P}}{g_{P_L}+\mu_{P_L}}$.

If there are no differences in advantages between the different hyperparasitoid species such as higher attack rate success, then invasibility relies entirely on the availability of resources. Note that the location and effects of μ_{H_L} are reversed in the larval-attacking invaders criteria (2.16). The mortality rate of the larvae-attacking hyperparasitoids, μ_{H_L}, is in the denominator and when the parameter is raised to simulate the effect of nicotine it will lower the criteria for invasion and therefore lowers the chances that H, the larvae-attacking hyperparasitoids, have to invade a system where $\hat{H}$, the pupae-attacking hyperparasitoids, resides.

2.5 Bifurcations and Stability

There are certain parameters that, when varied, cause a change in the dynamics of the competing hyperparasitoid species. The first parameter that we investigate is the mortality rate of the larvae-attacking hyperparasitoid larvae, μ_{H_L}. If nicotine has no effect on the mortality of the larvae-attacking hyperparasitoid larvae, then pupae-attacking

hyperparasitoids will be excluded. Increasing μ_{H_L} may cause the host, parasitoids, and larvae-attacking hyperparasitoid equilibrium (2.12) to lose stability. The second parameter that we investigate is the rate of successful oviposition by the pupae-attacking hyperparasitoid, β_P. At high values of β_P (or β_L) the parasitoid population goes extinct. If β_P is too low the hyperparasitoid population cannot persist.

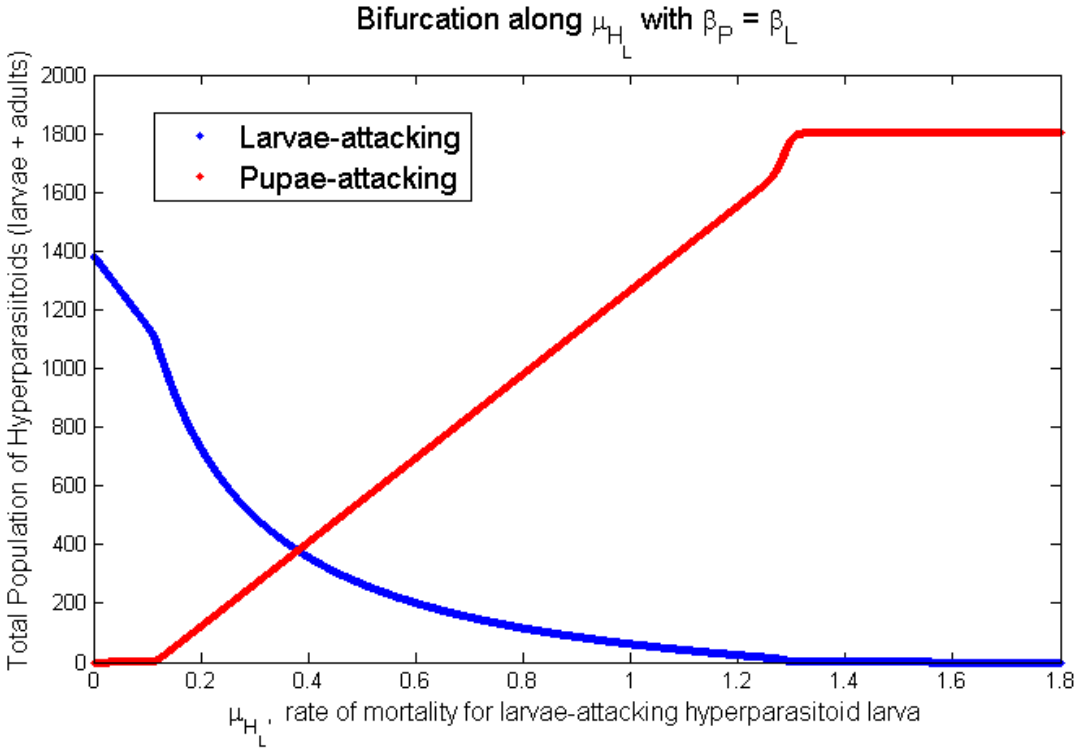

Figure 2.2 The total population (larvae plus adults) of larvae-attacking and pupae-attacking hyperparasitoids is plotted against the bifurcation parameter μ_{H_L}, the mortality rate of larvae-attacking hyperparasitoid larvae. Here $\alpha = 0.02$, $\beta_L = \beta_P = 0.002$.

With an increased effect of nicotine on μ_{H_L} the dynamics shift away from the exclusion of the pupae-attacking hyperparasitoids. As μ_{H_L} approaches 0.2, the model dynamics change from larvae-attackers only to coexistence of both hyperparasitoid species. When $\mu_{H_L} \approx 1.3$ pupae-attackers become the only hyperparasitoid species (see Figure 2.2). In order for larvae-attacking hyperparasitoids to be unable to persist, nicotine must have a high effectiveness for killing the hyperparasitoid larva or be at high concentrations in the parasitoid larvae.

Different rates of successful oviposition is another scenario that could result in exclusion of a competing hyperparasitoid species. Let k be the ratio between the successful attack rates of the pupae-attacking hyperparasitoids to the larvae-attacking hyperparasitoids,

$$k = \frac{\beta_P}{\beta_L}.$$

Setting $\beta_L = 0.002$ and increasing k results in pupae-attackers exclusion equilibrium (2.12) going unstable for k just below 2, or an oviposition success rate of the pupae-attackers less than twice that of the larvae-attackers (see Figure 2.3). There is a large range of k for which all equilibria are unstable and the population exhibits stable limit cycles with coexistence of all species. However, increasing k further eventually leads to the exclusion of the larvae-attackers. This level of success can also have a very detrimental effect on total population size for the pupae-attackers. Even on a plant that produces no nicotine the difference in oviposition success rates shows changes in equilibria stability. However, unlike the change in μ_{H_L}, there are values of k that cause complex oscillation patterns, such as when $3 < k < 9$ (see Figure 2.3).

The bifurcation diagram for larvae-attacking hyperparasitoid larvae mortality, μ_{H_L}, in Figure 2.2 shows it is possible for nicotine to cause the exclusion of the larvae-attacking hyperparasitoids. However, it occurs when μ_{H_L} is highly detrimental to the larvae-attacking hyperparasitoids. The change in the ratio of the attack rates, β, in Figure 2.3 shows that it can explain the coexistence of the two parasitoid species on a plant that does not produce nicotine. However, high success rates result in low population density of the parasitoid species which in turn results in low population density of the hyperparasitoid species. By changing $\beta_P = 0.004$ this places parameters into a regime that allows both species to coexist on the nicotine-free patch. Varying μ_{H_L} under the new conditions for β_P the previous dynamics are shifted such that exclusion of the larvae-attacking hyperparasitoid occurs at a lower $\mu_{H_L} \approx 0.8$ (see Figure 2.4).

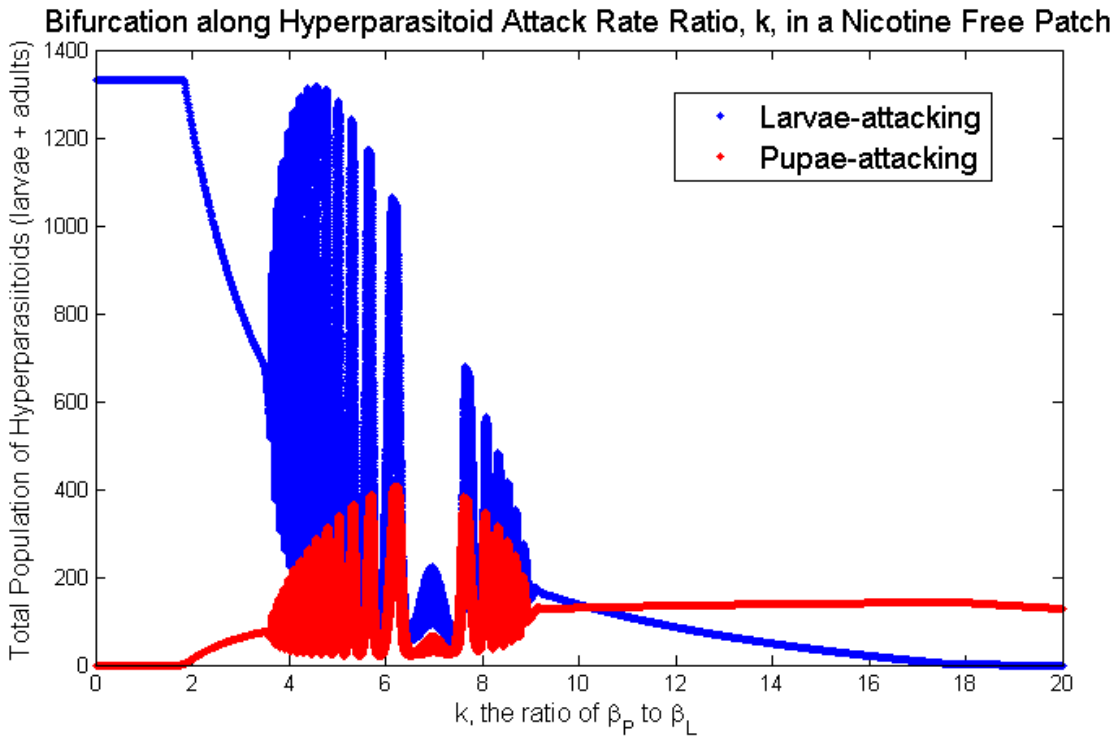

Figure 2.3 The total population (larvae plus adults) of larvae-attacking and pupae-attacking hyperparasitoids is plotted against the bifurcation parameter, k, the ratio of successful oviposition by pupae-attacking hyperparasitoid adults to the successful oviposition by larvae-attacking hyperparasitoid adults. Here $\alpha = 0.02$, $\mu_{H_L} = \mu_{\hat{H}_L}$, and $\beta_L = 0.002$.

From Figure 2.5, the change to a nicotine-positive patch removes the oscillations seen in the nicotine-free patch. While the mortality rate, $\mu_{H_L} = \frac{1}{3}$, is set for a nicotine-positive patch the difference in successful attack rates needed for exclusion of the larvae-attacking parasitoids is lowered than from the nicotine-free patch from $k \approx 18$ to k between 4 and 5.

Further increasing the mortality rate of larvae-attacking hyperparasitoid larvae, $\mu_{H_L} = \frac{2}{3}$, and varying the ratio, k, shows exclusion of the larvae-attacking hyperparasitoids at k just above 2 (see Figure 2.6). With an increase in the effect of nicotine on μ_{H_L} the pupae-attacking hyperparasitoids can exclude the competing larvae-attacking hyperparasitoids with a lower rate of successful oviposition. Both shifts μ_{H_L} and β_P combine to give the pupae-attacking hyperparasitoids a higher advantage.

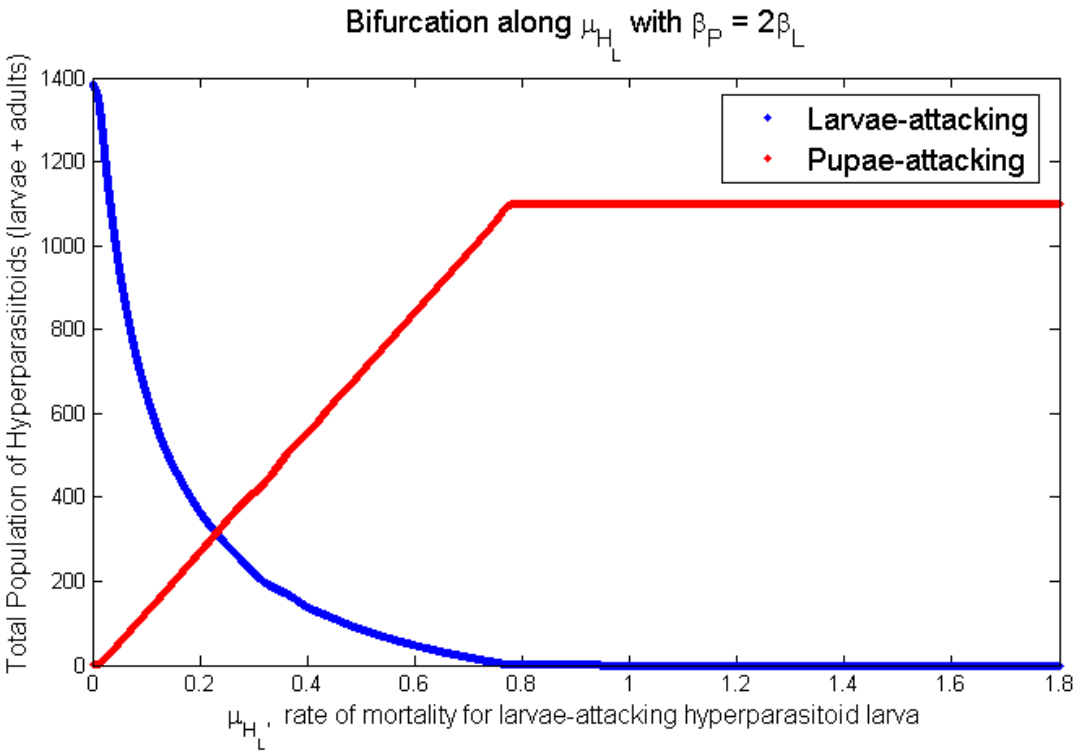

Figure 2.4 The total population (larvae plus adults) of larvae-attacking and pupae-attacking hyperparasitoids is plotted agasint the bifurcation parameter μ_{H_L}, the mortality rate of larvae-attacking hyperparasitoid larvae. Here $\alpha = 0.02$, $\beta_L = 0.002$, and $\beta_P = 0.004$.

A change in the effect of nicotine on μ_{H_L} does not independently causes extinction of the larvae-attacking hyperparasitoids. A high mortality rate will lower the larvae-attacking hyperparasitoids' population density, but it is only when competing with the pupae-attacking hyperparasitoids that results in the their extinction.

2.6 Discussion

The larvae-attacking hyperparasitoids have the benefit of attacking an earlier stage of their resource the parasitoid, but suffer from the presence of nicotine. The pupae-attacking hyperparasitoids must wait and attack a later stage of the parasitoid, but do not suffer from nicotine. These trade-offs for the competing species affect the dynamics of the system. When on a nicotine-free plant such as a tomato this advantage is enough

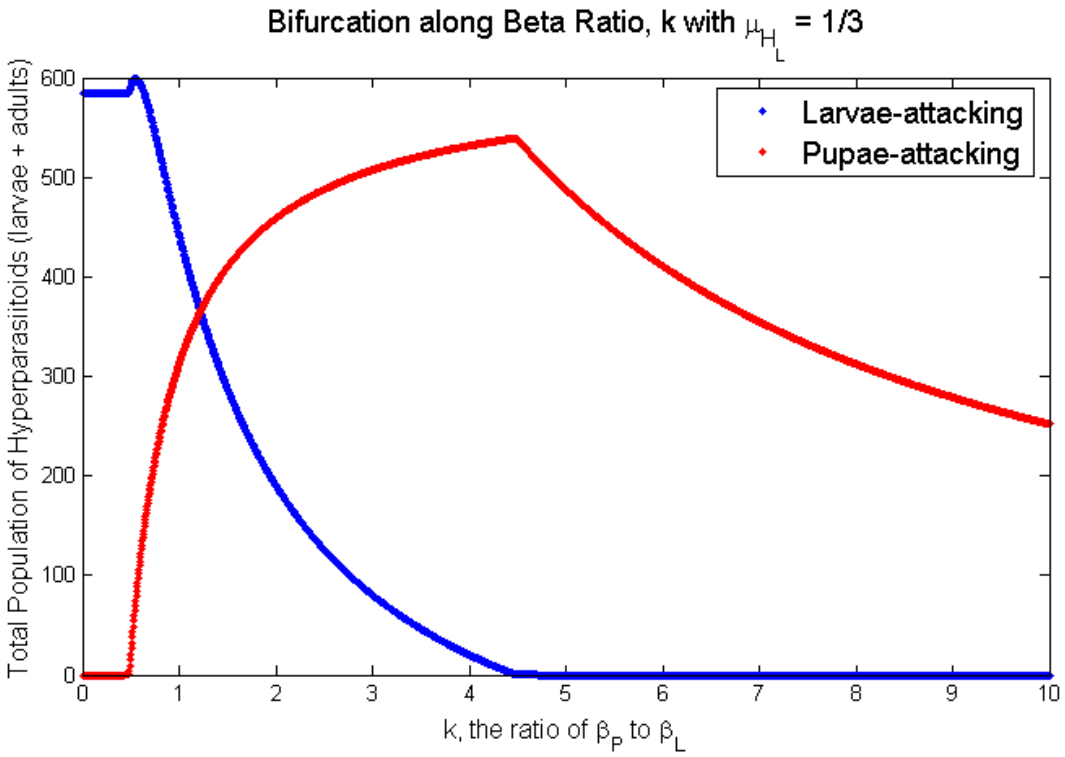

Figure 2.5 The total population (larvae plus adults) of larvae-attacking and pupae-attacking hyperparasitoids is plotted against the bifurcation parameter, k, the ratio of β_P to β_L. Here $\alpha = 0.02$, $\mu_{H_L} = \frac{1}{3}$, and $\beta_L = 0.002$.

to force the pupae-attackers out the population. This follows what is seen in Lotka-Volterra models; the competitor who reduces the limiting resource the most will force out other competitors [1, 4].

With the assumption that nicotine is causing an increase in the rate of mortality for the larvae-attacking hyperparasitoid larvae, μ_{H_L}, coexistence can be attained in an environment that contains nicotine. Through the rise of μ_{H_L} the advantage of attacking an earlier stage of the parasitoid is no longer enough for the larvae-attackers to exclude the pupae-attacking hyperparasitoids and coexistence is possible. If the effects of nicotine are preventing the larvae-attacking hyperparasitoids from entering that environment, then the effects must be relatively severe and cause nearly complete mortality. This situation might occur if the larvae-attackers are not specialists and rarely encounter nicotine, and are therefore not adapted to its effects.

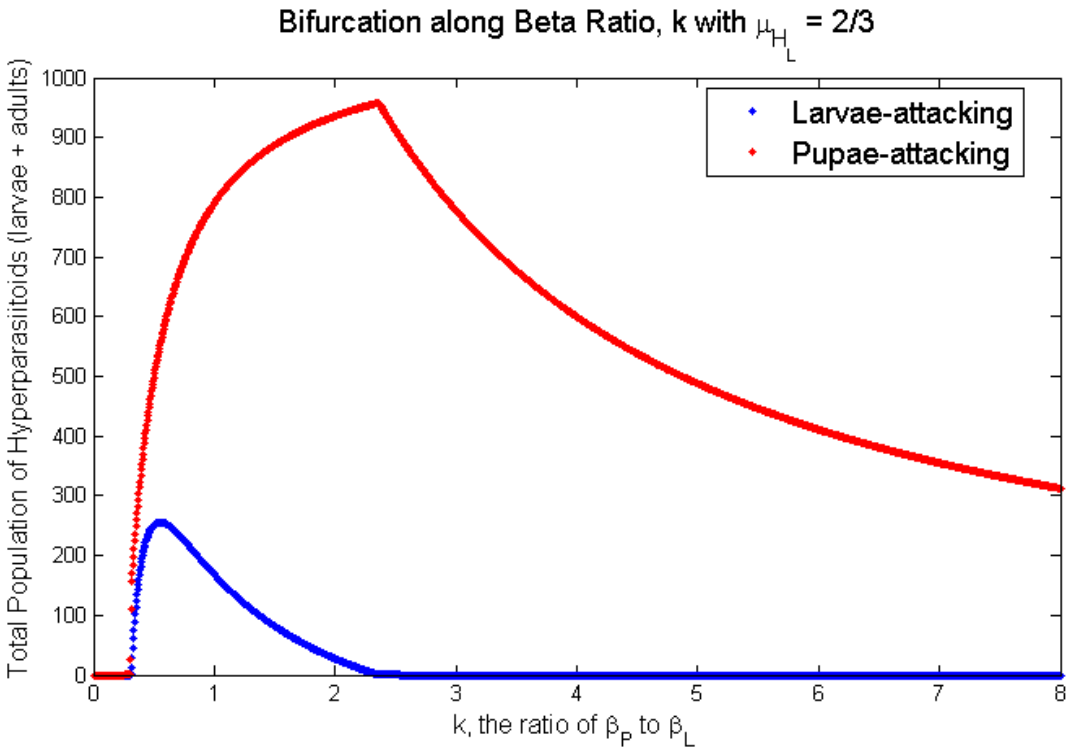

Figure 2.6 The total population (larvae plus adults) of larvae-attacking and pupae-attacking hyperparasitoids is plotted against the bifurcation parameter, k, the ratio of β_P to β_L. Here $\alpha = 0.02$, $\mu_{H_L} = \frac{2}{3}$, and $\beta_L = 0.002$.

Based on observations coexistence of both hyperparasitoid species occurs in a nicotine free environment while exclusion of the larvae attackers occurs in the nicotine positive environment. The advantage for larvae-attacking hyperparasitoids by attacking an earlier stage of the parasitoid would normally prevent the coexistence in the nicotine free environment except when the pupae-attacking species has a higher success rate of oviposition, $\beta_P > \beta_L$. The attack rates β_L and β_P are based on the success of the hyperparasitoids both in search and handling time. Ovipositing into the larva of the parasitoid through the cuticle of the host is a relatively rare trait among hyperparasitoids. It could be that handling time and search time are higher for the larvae-attacker. When both the successful attack rate of the pupae-attacking hyperparasitoids, β_P, is increased in a nicotine positive patch the exclusion of the larvae-attackers occurs with a weaker effect of nicotine.

When the attack rates are equal, $\beta_L = \beta_P$, in a nicotine-free environment, there was exclusion of the pupae-attackers. Thus the effect of nicotine is not enough to explain the observation. When the effect of nicotine on mortality of the larvae-attacking species' larva is combined with a higher successful attack rate for the pupae-attacking species both observations can occur in the model.

Chapter 3

Two Patch Model

In this chapter we develop a model with two patches, one where the plant may produce nicotine for the host to sequester, N^+, and one where the plant does not produce nicotine, N^-. The hosts and parasitoids have separate populations for each plant, so the hyperparasitoids are the only ones able to move between plants. We let the parameters p and q denote the probability an individual of the larvae-attacking and pupae-attacking hyperparasitoids, respectively, choose to attack on the nicotine-free patch N^-. We will refer to p and q as the strategies of each species, and we assume these strategies are heritable. Based on replicator equation concepts in evolutionary game theory, the frequency of a strategy in the population will increase if the expected fitness for an individual using that strategy is greater than the average fitness of the population [7].

We define individual fitness to be the expected total number of offspring surviving to adulthood produced over the course of its lifetime. Hyperparasitoids will want to change their strategy if doing so results in increased fitness. The equilibrium strategy for each individual will therefore be a Nash equilibrium where changing strategies will result in a loss of fitness on that patch. [7, 19]. We will focus on the changes made to the equilibrium strategy as the effect of nicotine increases the rate of mortality of the larvae-attacking hyperparasitoid larvae in the N^+ patch .

3.1 Two Patch Model Equations

The model equations for hosts, parasitoids, and hyperparasitoid larvae on the N^- patch are identical to the the equations (2.8). The equations for hosts and parasitoids on the N^+ patch are similar to the equations in (2.8) with the difference that they are identified with a $^+$ for purpose of differentiating from the population on the N^- patch. Larvae-attacking hyperparasitoid larvae enter N^- proportional to the probability of an individual adult choosing the patch, p. Conversely, the larvae-attacking hyperparasitoid larvae enter N^+ proportional to the probability of an individual adult choosing the patch, $1-p$. Similarly pupae-attacking hyperparasitoid larvae enter proportionally by q and $1-q$.

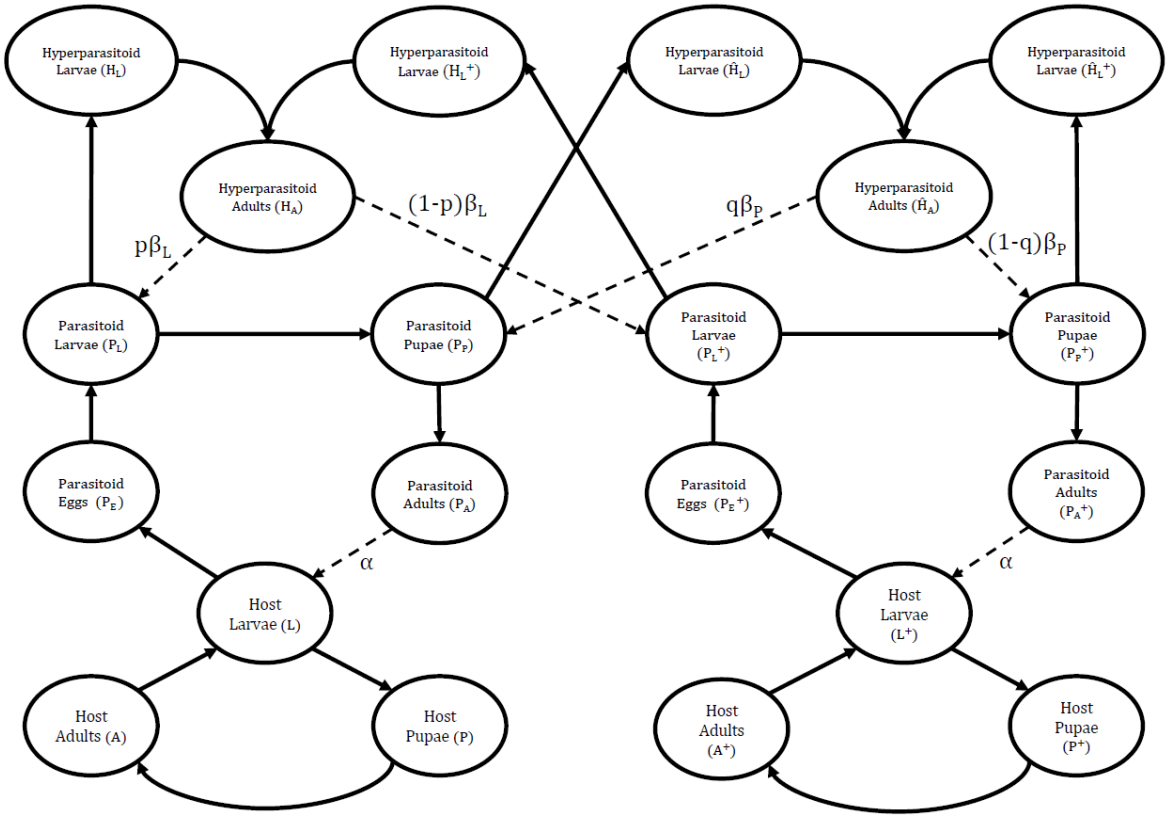

Figure 3.1 Flow diagram illustrating the movement of individuals in each population in the two patch system. Solid lines indicate maturation and dashed lines indicate parasitism.

When all the equations for the N^- patch, N^+ patch, and hyperparasitoid adults are

coupled together the equations for the two patch model are:

$$
\begin{aligned}
\frac{dL}{dt} &= b\left(1-\frac{L}{K}\right)A - \mu_L L - g_L L - \alpha_1 P_A L \\
\frac{dP}{dt} &= g_L L - g_P P \\
\frac{dA}{dt} &= g_P P - \mu_A A \\
\frac{dP_E}{dt} &= \alpha P_A L - \mu_{P_E} P_E - g_{P_E} P_E \\
\frac{dP_L}{dt} &= g_{P_E} P_E - g_{P_L} P_L - \mu_{P_L} P_L - p\beta_L H_A P_L \\
\frac{dP_P}{dt} &= g_{P_L} P_L - g_{P_P} P_P - \mu_{P_P} P_P - q\beta_P \hat{H}_A P_P \\
\frac{dP_A}{dt} &= g_{P_P} P_P - \mu_{P_A} P_A \\
\frac{dH_L}{dt} &= p\beta_L H_A P_L - g_{H_L} H_L - \mu_{H_L} H_L \\
\frac{d\hat{H}_L}{dt} &= q\beta_P \hat{H}_A P_P - g_{\hat{H}_L} \hat{H}_L - \mu_{\hat{H}_L} \hat{H}_L \\
\frac{dL^+}{dt} &= b\left(1-\frac{L^+}{K}\right)A^+ - \mu_L L^+ - g_L L^+ - \alpha_2 P_A^+ L^+ \\
\frac{dP^+}{dt} &= g_L L^+ - g_P P^+ \\
\frac{dA^+}{dt} &= g_P P^+ - \mu_A A^+ \\
\frac{dP_E^+}{dt} &= \alpha P_A^+ L^+ - \mu_{P_E} P_E^+ - g_{P_E} P_E^+ \\
\frac{dP_L^+}{dt} &= g_{P_E} P_E^+ - g_{P_L} P_L^+ - \mu_{P_L} P_L^+ - (1-p)\beta_L H_A P_L^+ \\
\frac{dP_P^+}{dt} &= g_{P_L} P_L^+ - g_{P_P} P_P^+ - \mu_{P_P} P_P^+ - (1-q)\beta_P \hat{H}_A P_P^+ \\
\frac{dP_A^+}{dt} &= g_{P_P} P_P^+ - \mu_{P_A} P_A^+ \\
\frac{dH_L^+}{dt} &= (1-p)\beta_L H_A P_L^+ - g_{H_L} H_L^+ - \mu_{H_L^+} H_L^+ \\
\frac{d\hat{H}_L^+}{dt} &= (1-q)\beta_P \hat{H}_A P_P^+ - g_{\hat{H}_L} \hat{H}_L^+ - \mu_{\hat{H}_L} \hat{H}_L^+ \\
\frac{dH_A}{dt} &= g_{H_L}(H_L + H_L^+) - \mu_{H_A} H_A \\
\frac{d\hat{H}_A}{dt} &= g_{\hat{H}_L}(\hat{H}_L + \hat{H}_L^+) - \mu_{\hat{H}_A} \hat{H}_A
\end{aligned}
\tag{3.1}
$$

Parameters for the host and parasitoid populations do not vary between the patches. $\mu_{H_L^+}$ is the mortality rate of larvae-attacking hyperparasitoid larvae in the N^+ patch.

Parameter	Parameter meaning	Restrictions
p	probability of attacking nicotine free larva	$0 \leqslant p \leqslant 1$
q	probability of attacking nicotine free pupae	$0 \leqslant q \leqslant 1$
$\mu^+_{H_L}$	mortality rate of larvae-attacking hyperparasitoids in presence of nicotine	$\mu^+_{H_L} \geqslant \mu_{H_L}$

Table 3.1 New parameters for two patch model

3.2 Fitness Functions

Total lifetime fecundity for a larvae-attacking hyperparasitoid choosing the nicotine-free patch, N^-, is determined by multiplying the number of larva produced by a hyperparasitoid adult in its lifetime, $\beta_L P^*_L \frac{1}{\mu_{H_A}}$, by the probability the larvae survive to adulthood, $\frac{g_{H_L}}{g_{H_L} + \mu_{H_L}}$. The parasitoid population equilibrium depends on the strategies, $P^*_L = P^*_L(p, q)$. Fitness for the pupae-attacking hyperparasitoids is determined in a similar manner. The fitness of the larvae-attacking hyperparasitoids in the nicotine free patch (F^-_H) and nicotine positive patch (F^+_H) are given by:

$$\begin{aligned} F^-_H &= \beta_L P^*_L \left(\frac{g_{H_L}}{g_{H_L} + \mu_{H_L}}\right)\left(\frac{1}{\mu_{H_A}}\right) \\ F^+_H &= \beta_L P^{+*}_L \left(\frac{g_{H_L}}{g_{H_L} + \mu^+_{H_L}}\right)\left(\frac{1}{\mu_{H_A}}\right) \end{aligned} \tag{3.2}$$

The fitness of the pupae-attacking hyperparasitoids in each patch is given by:

$$\begin{aligned} F^-_{\hat{H}} &= \beta_P P^*_P \left(\frac{g_{\hat{H}_L}}{g_{\hat{H}_L} + \mu_{\hat{H}_L}}\right)\left(\frac{1}{\mu_{\hat{H}_L}}\right) \\ F^+_{\hat{H}} &= \beta_P P^{+*}_P \left(\frac{g_{\hat{H}_L}}{g_{\hat{H}_L} + \mu_{\hat{H}_L}}\right)\left(\frac{1}{\mu_{\hat{H}_L}}\right) \end{aligned} \tag{3.3}$$

The Nash equilibrium occurs when fitness is equal in both patches for both hyperparasitoid species. it is stable if no individual can improve its fitness by a small change in strategy p or q [7].

Optimal strategy can be determined in several different contexts. We can examine optimal patch choice for each hyperparasitoid species in isolation, or we can consider the case where both species are present. In the latter case, the hyperparasitoid species (or "players") in this patch selection game can choose their strategy either consecutively or simultaneously. Consecutive decisions require that one of the players stick to their strategy regardless of the changes the other player is making [7]. The following results will show the optimal strategy for both hyperparasitoid species choosing alone, and also when they choose simultaneously.

3.3 Results

First we find the Nash equilibrium for each hyperparasitoid species individually by finding the p or q that solve the equations: $F_H^- = F_H^+$, for larvae-attackers, or $F_{\hat{H}}^- = F_{\hat{H}}^+$, for pupae-attackers.

Pupae-attacking hyperparasitoids:

Since the pupae-attackers are assumed to never suffer from the effects of nicotine, intuitively we would guess their optimal strategy would be to use both patches equally. Setting $F_{\hat{H}}^- = F_{\hat{H}}^+$ gives

$$\beta_P P_P^* \left(\frac{g_{\hat{H}_L}}{g_{\hat{H}_L} + \mu_{\hat{H}_L}} \right) \left(\frac{1}{\mu_{\hat{H}_L}} \right) = \beta_P P_P^{+*} \left(\frac{g_{\hat{H}_L}}{g_{\hat{H}_L} + \mu_{\hat{H}_L}} \right) \left(\frac{1}{\mu_{\hat{H}_L}} \right)$$

Since all host and parasitoid parameters are the same on both patches this reduces to

$$P_P^* = P_P^{+*}.$$

Parasitoid pupae equilibria will be equal in both patches when there are equal numbers of hyperparasitoids. This occurs when $q = 1 - q$ or $q = \frac{1}{2}$. Therefore the optimal strategy for the pupae-attackers while they are alone is $q = \frac{1}{2}$. However, their optimal strategy will change with any change in density of parasitoid pupae, P_P^*. If the larvae-attacking hyperparasitoids are present in the system they will affect this density due to their parasitism of the parasitoid larvae and the optimal strategy for the pupae-attackers will not remain at $q = \frac{1}{2}$.

Equilibrium population sizes for the pupae-attacking hyperparasitoid larvae in each patch as well as the total adults are given in Table 3.2 for $q = \frac{1}{2}$ (optimal q) as well as the boundary values $q = 0$ and $q = 1$. We see that population sizes are greatest for the optimal strategy and the population total on the boundaries is half of the total for the optimized strategy.

q	$(\hat{H}_L, \hat{H}_L^+, \hat{H}_A)$ at optimal q	$(\hat{H}_L, \hat{H}_L^+, \hat{H}_A)$ at $q = 1$	$(\hat{H}_L, \hat{H}_L^+, \hat{H}_A)$ at $q = 0$
0.5	(202,202,143)	(0,202,72)	(202,0,72)

Table 3.2 Optimal strategy q for pupae-attackers alone.

Larvae-attacking hyperparasitoids:

The optimal strategy of the larvae-attacking hyperparasitoids will vary with the effect of nicotine on their larval mortality rate, $\mu_{H_L}^+$, in the N^+ patch. The optimal strategy p increases as $\mu_{H_L}^+$ increases (Table 3.3. For comparison, population sizes at the optimal value of p are compared to $p = 1$, which corresponds to using only the N^- patch, and $p = 0$, which corresponds to only using the N^+ patch.

As $\mu_{H_L}^+$ increases, the equilibrium value of p increases, meaning the larvae-attackers are choosing the N^- patch more often than the N^+. This strategy results in higher population sizes than only using a single patch (see Table 3.3).

Competing hyperparasitoid species

Here we allow players to choose their strategies simultaneously. Finding the equilib-

$\mu^+_{H_L}$	p	(H_L, H^+_L, H_A) at optimal p	(H_L, H^+_L, H_A) at $p = 1$	(H_L, H^+_L, H_A) at $p = 0$
$\frac{1}{45}$	0.5	(126,126,90)	(126,0,45)	(0,126,45)
$\frac{1}{3}$	0.5138	(126,119,87)	(126,0,45)	(0,119,42)
$\frac{2}{3}$	0.5294	(126,112,85)	(126,0,45)	(0,112,40)
2	0.6027	(126,83,74)	(126,0,45)	(0,83,30)

Table 3.3 Optimal strategy p, for larvae-attackers alone, with increasing effects of nicotine. Also shown are equilibrium population sizes for hyperparasitoid larvae in each patch and total adults for the optimal value of p as well as $p = 1$ and $p = 0$.

rium values of p and q requires solving $F^-_H = F^+_H$ and $F^-_{\hat{H}} = F^+_{\hat{H}}$ simultaneously noting all equilibrium densities are dependent upon p and q. By setting $\beta_P = 0.004$ and $\beta_L = 0.002$ allows for a baseline of coexistence in N^-. The equilibrium strategies are found to move towards segregation as $\mu^+_{H_L}$ increases. With increasing $\mu^+_{H_L}$ the equilibrium strategy of the larvae-attackers moves closer to 1, so they are choosing the n^- patch more often than they N^+ patch. The equilibrium strategy of the pupae-attackers moves closer to 0, so they are spending more time in the N^+ patch. We note that since the attack rates used for simulations resulted in population oscillations rather than equilibria, the results in Table 3.4 are calculated from fitness values based on the approximated average population size over one period.

$\mu^+_{H_L}$	p	q	(H_L, H^+_L, H_A)	$(\hat{H}_L, \hat{H}^+_L, \hat{H}_A)$
$\frac{1}{45}$	0.5	0.5	(114,114,81)	(1,1,1)
$\frac{1}{3}$	0.8544	0.0281	(114,20,48)	(1,37,13)
$\frac{2}{3}$	0.9619	0.0140	(115,5,42)	(1,75,27)
1	1	0	(126,0,44)	(0,201,71)

Table 3.4 Optimal strategy p and q choosing simultaneously, $\beta_L = 0.002$, $\beta_P = 0.004$

3.4 Discussion

Pupae-attackers do not suffer any disadvantages without competitors present, therefore the best strategy for them is to evenly divide their population between the patches. If

they were to use the patches unequally, an individual could improve their fitness by moving to the less populated patch. In the absence of the pupae-attackers, the larvae-attackers will optimize their strategy based on their mortality rates in the N^+ patch leading to higher proportions of them choosing the N^- patch.

It was shown that the larvae-attackers will exclude the pupae-attackers on the N^- patch without the pupae-attackers having some additional advantage. Choosing simultaneously leads to the optimized strategy for both hyperparasitoid populations to move towards segregation of the two species as the effect of nicotine increases. The pupae-attackers choose the N^+ patch while the larvae-attackers choose the N^- patch. This is shown to lead to lower populations than when they are alone; however, it does lead to higher populations than when they are competing on the patches Table 3.4.

Chapter 4

Conclusion

Using a continuous time and stage-structured model of host, parasitoid, and hyperparasitoid populations we investigate the changes in dynamics in the presence and absence of nicotine. We assume nicotine moves up the trophic levels from plant to host to parasitoid to hyperparasitoid, eventually causing a higher rate of mortality in hyperparasitoid larvae on tobacco plants. We find that increasing the negative effects of nicotine makes it easier for pupae-attacking hyperparasitoids to invade a larvae-attacking hyperparasitoid population. When the effect of nicotine is large enough the pupae-attackers can exclude the larvae-attackers, resulting in lower diversity on tobacco and also supports the observation that larvae-attacking hyperparasitoids are not found on tobacco plants. However, when on a nicotine-free tomato plant, the larvae-attackers have the advantage of attacking an earlier stage of the parasitoid and will exclude the pupae-attackers which also results in lower diversity and contradicts the observation of increased diversity on nicotine free plants.

We propose that the pupae-attacking hyperparasitoid population may have an additional advantage that allows them to coexist with larvae-attackers in the absence of nicotine. If pupae-attackers have a higher rate of successful oviposition relative to the larvae-attacking hyperparasitoids coexistence is possible without any negative effects

of nicotine on the larvae-attackers. Differences in oviposition rates could be due to many reasons, but the primary reason is attacking larvae inside a host might require more search and handling time. By increasing the successful oviposition rate of pupae-attackers, both populations of hyperparasitoids can persist in either a stable equilibrium or stable oscillations. The effects of nicotine combined with a higher attack success rate for the pupae-attacking hyperparasitoids can explain the observations of higher hyperparasitoid diversity on nicotine free plants, and the absence of larvae-attackers on plants containing nicotine.

The two patch model introduces a population of hosts and parasitoids on both tobacco and tomato plants with hyperparasitoids choosing between plants to search for parasitoids. In the absence of their larvae-attacking competitors, the pupae-attackers choose each patch equally often. In the absence of pupae-attackers, larvae-attacking hyperparasitoids will choose the tomato more often as the effect of nicotine on mortality of their larvae increases in the tobacco patch. With both hyperparasitoid species present optimal strategies shifted towards segregation with the pupae-attacking hyperparasitoids on tobacco plants and larvae-attacking hyperparasitoids on tomato plants. The complexity of the system makes it difficult to produce any conclusions for the questions on hyperparasitoid diversity. However, the model lays groundwork for study into learning and search strategies of hyperparasitoids.

Bibliography

[1] R. A. Armstrong and R. McGehee. *Competitive exclusion*. American Naturalist, pages 151–170, 1980.

[2] P. Barbosa, J. A. Saunders, J. Kemper, R. Trumbule, J. Olechno, and P. Martinat. *Plant allelochemicals and insect parasitoids effects of nicotine on Cotesia congregata (Say)(Hymenoptera: Braconidae) and Hyposoter annulipes (Cresson)(Hymenoptera: Ichneumonidae)*. Journal of Chemical Ecology, 12(6):1319–1328, 1986.

[3] J. R. Beddington and P. S. Hammond. *On the dynamics of host-parasite-hyperparasite interactions*. The Journal of Animal Ecology, pages 811–821, 1977.

[4] C. J. Briggs. *Competition among parasitoid species on a stage-structured host and its effect on host suppression*. American Naturalist, pages 372–397, 1993.

[5] C. J. Briggs and T. R. Collier. *Autoparasitism, interference, and parasitoid-pest population dynamics*. Theoretical Population Biology, 60(1):33–57, 2001.

[6] C. V. Covell Jr et al. *A field guide to the moths of eastern North America.* Houghton Mifflin Co., 1984.

[7] R. Cressman and Y. Tao. *The replicator equation and other game dynamics*. Proceedings of the National Academy of Sciences, 111(Supplement 3):10810–10817, 2014.

[8] A. Dhammi. *Effect of Imidacloprid on Cotesia congregata, an endoparasitoid of Manduca sexta, and its translocation from host to endoparasitoid.* 2010.

[9] B. B. Fulton. *The hornworm parasite, Apanteles congregatus (Say) and the hyperparasite, Hypopteromalus tabacum (Fitch)*. Annals of the Entomological Society of America, 33(2):231–244, 1940.

[10] H. C. J. Godfray and M. Shimada. *Parasitoids as model organisms for ecologists*. Researches on population ecology, 41(1):3–10, 1999.

[11] J. A. Harvey, N. M. Van Dam, L. Witjes, R. Soler, and R. Gols. *Effects of dietary nicotine on the development of an insect herbivore, its parasitoid and secondary hyperparasitoid over four trophic levels*. Ecological Entomology, 32(1):15–23, 2007.

[12] J. A. Harvey, L. E. M. Vet, L. Witjes, and T. M. Bezemer. *Remarkable similarity in body mass of a secondary hyperparasitoid Lysibia nana and its primary parasitoid host Cotesia glomerata emerging from cocoons of comparable size*. Archives of Insect Biochemistry and Physiology, 61(3):170–183, 2006.

[13] J. G. Kingsolver. *Variation in growth and instar number in field and laboratory Manduca sexta*. Proceedings of the Royal Society B: Biological Sciences, 274(1612):977–981, 2007.

[14] N. J. Mills and W. M. Getz. *Modelling the biological control of insect pests: a review of host-parasitoid models*. Ecological modelling, 92(2):121–143, 1996.

[15] W. W. Murdoch. *Relevance of pest-enemy models to biological control*. Critical issues in biological control/edited by Manfred Mackauer and Lester E. Ehler, Jens Roland, 1990.

[16] W. W. Murdoch, C. J. Briggs, and R. M. Nisbet. *Consumer-Resource Dynamics (MPB-36)*. Princeton University Press, 2013.

[17] A. J. Nicholson and V. A. Bailey. *The Balance of Animal Populations.—Part I.* In *Proceedings of the Zoological Society of London*, volume 105, pages 551–598. Wiley Online Library, 1935.

[18] J. P. Reinecke, J. S. Buckner, and S.R. Grugel. *Life cycle of laboratory-reared tobacco hornworms, Manduca sexta, a study of development and behavior, using time-lapse cinematography*. The Biological Bulletin, 158(1):129–140, 1980.

[19] S. L. Robertson and I. M. Hamilton. *Habitat selection under the risk of infectious disease*. Evolutionary Ecology Research, 14(1):51–72, 2012.

[20] M. Sasaki and L. M. Riddiford. *Regulation of reproductive behaviour and egg maturation in the tobacco hawk moth, Manduca sexta*. Physiological Entomology, 9(3):315–327, 1984.

[21] D. J. Sullivan. *Insect hyperparasitism*. Annual review of entomology, 32(1):49–70, 1987.

[22] D. J. Sullivan and W. Völkl. *Hyperparasitism: multitrophic ecology and behavior*. Annual review of entomology, 44(1):291–315, 1999.

[23] K. W. Thorpe and P. Barbosa. *Effects of consumption of high and low nicotine tobacco by Manduca sexta (Lepidoptera: Sphingidae) on survival of gregarious endoparasitoid Cotesia congregata (Hymenoptera: Braconidae)*. Journal of chemical ecology, 12(6):1329–1337, 1986.

Appendices

Appendix A

Jacobian Matrices

Jacobian matrix of the hosts only system (2.1):

$$J = \begin{bmatrix} -\frac{bA}{K} - \mu_L - g_L & 0 & b(1-\frac{L}{K}) \\ g_L & -g_P & 0 \\ 0 & g_P & -\mu_A \end{bmatrix} \tag{A.1}$$

Evaluating the Jacobian at the extinction equilibrium of the host model: $(0,0,0)$.

$$J(0,0,0) = \begin{bmatrix} -\mu_L - g_L & 0 & b \\ g_L & -g_P & 0 \\ 0 & g_P & -\mu_A \end{bmatrix} \tag{A.2}$$

Evaluating the Jacobian at nonextinction equilibrium of the host only model: (L^*, P^*, A^*).

$$J(L^*, P^*, A^*) = \begin{bmatrix} -\frac{b g_L}{\mu_A} & 0 & \frac{\mu_A(g_L+\mu_L)}{g_L} \\ g_L & -g_P & 0 \\ 0 & g_P & -\mu_A \end{bmatrix} \tag{A.3}$$

The Jacobian for the host and parasitoid system (2.3) is:

$$J = \begin{bmatrix} -\frac{bA}{K} - \mu_L - g_L - \alpha P_A & 0 & b(1-\frac{L}{K}) & 0 & 0 & 0 & -\alpha L \\ g_L & -g_P & 0 & 0 & 0 & 0 & 0 \\ 0 & g_P & -\mu_A & 0 & 0 & 0 & 0 \\ \alpha P_A & 0 & 0 & -\mu_{P_E} - g_{P_E} & 0 & 0 & \alpha L \\ 0 & 0 & 0 & g_{P_E} & -\mu_{P_L} - g_{P_L} & 0 & 0 \\ 0 & 0 & 0 & 0 & g_{P_L} & -\mu_{P_P} - g_{P_P} & 0 \\ 0 & 0 & 0 & 0 & 0 & g_{P_P} & -\mu_{P_A} \end{bmatrix} \tag{A.4}$$

Lower block of the host-parasitoid Jacobian matrix evaluated at the host-only equilibrium point (2.5):

$$J(L^*, P^*, A^*, 0, 0, 0, 0)_{lower} = \begin{bmatrix} -\mu_{P_E} - g_{P_E} & 0 & 0 & \frac{\alpha K(bg_L - \mu_A\mu_L - \mu_A g_L)}{bg_L} \\ g_{P_E} & -\mu_{P_L} - g_{P_L} & 0 & 0 \\ 0 & g_{P_L} & -\mu_{P_P} - g_{P_P} & 0 \\ 0 & 0 & g_{P_P} & -\mu_{P_A} \end{bmatrix} \tag{A.5}$$

Appendix B

Matlab m-files

Function files

```
%The functional m-file for the single patch model.

function[Y] = hyperparasite(t,y,p);

Y = zeros(11,1); %change to total number of variables

b = p(1); %rate of eggs that hatch from adults
muL = p(2); %mortality rate of host larva
gL = p(3);%growth rate of host larva
alpha = p(4); %rate of successful oviposition on host by parasite
gP = p(5); %growth rate of host pupae
muA = p(6); %mortality rate of host adults
K = p(7); %limiting factor
muPE = p(8); %mortality rate of parasite eggs
gPE = p(9); %growth rate of parasite eggs
gPL = p(10); %growth rate of parasite larva
gPP = p(11); %growth rate of parasite pupae
gHL = p(12); %growth rate of early attacking hyperparasite larva
```

```
gHhatL = p(13); %growth rate of later attacking hyperparasite larva
muPL = p(14); %mortality rate of parasite larva
muPP = p(15); %mortality rate of parasite pupae
muPA = p(16); %mortality rate of parasite adults
muHL = p(17); %mortality rate of early h.parasite larva
muHA = p(18); %mortality rate of early h.parasite adults
muHhatL = p(19); %mortality rate of late h.parasite larva
muHhatA = p(20); %mortality rate of late h.parasite adults
betaL = p(21); %rate of successful oviposition on parasite larva by h.parasites
betaP = p(22); %rate of successful oviposition on parasite pupae by h.parasites
c = p(23); %average clutch size




L = y(1); %host larva
P = y(2); %host pupae
A = y(3); %host adults
PE = y(4); %parasite eggs
PL = y(5); %parasite larva
PP = y(6); %parasite pupae
PA = y(7); %parasite adults
HA = y(8); %hyperparasite adults (early attackers)
HL = y(9); %hyperparasite larva (early attackers)
HhatA = y(10); %hyperparasite adults (late attackers)
HhatL = y(11); %hyperparasite larva (late attackers)



%Single patch model equations
Y(1)=b*A*(1-L/K)-(muL+gL)*L-alpha*PA*L;
Y(2)=gL*L-gP*P;
Y(3)=gP*P-muA*A;
Y(4)=alpha*c*PA*L-(muPE+gPE)*PE;
```

```
Y(5)=gPE*PE-(gPL+muPL)*PL-betaL*HA*PL;
Y(6)=gPL*PL-(gPP+muPP)*PP-betaP*HhatA*PP;
Y(7)=gPP*PP-muPA*PA;
Y(8)=gHL*HL-muHA*HA;
Y(9)=betaL*HA*PL-(gHL+muHL)*HL;
Y(10)=gHhatL*HhatL-muHhatA*HhatA;
Y(11)=betaP*HhatA*PP-(gHhatL+muHhatL)*HhatL;


%---------------% Fitness Function %----------------------%
% Use this function to find fitness with an inputed strategies p and q for
% the early and late attackers respectively.

%To find the optimal strategy this file can be called in the command window
%using the "fminsearch" command

%i.e. fminsearch(@(minip) fitness(minip), [guess])
% where [guess] is a vector of the minip values [p;q] that you believe are
% near to the optmized fitness strategies



function RSS = fitness(minip)
r=minip(1); %r = p, the probability of attack on either patch for H (since p is already be
%q = 0.5; %-- use if optimizing for just r
q=minip(2); %Include if both are being used simulataenously

%q=minip(1); %comment out if running for both populations of hyperparasites
%r = 0.5138; %-- use if optmizing for just q
%---------------------% Running twopatch %--------------------------
%parameters
p = zeros(27,1);
```

```
p(1)=3.3; %b
p(2)=1/100; %muL
p(3)=1/26; %gL
p(4)=0.02; %alpha1 (variable)
p(5)=0.02; %alpha2
p(6)=1/21; %gP
p(7)=1/60; %muA
p(8)=250; %K (determined by author)
p(9)=1/4;%muPE
p(10)=1/4; %gPE
p(11)=1/11; %gPL
p(12)=1/6; %gPP
p(13)=4/45; %gHL (early)
p(14)=4/45; %gHhatL (late)
p(15)=7/600; %muPL
p(16)=0; %muPP
p(17)=2/9;%muPA
p(18)=1/45; %muHL
p(19)=1/4; %muHA
p(20)=1/45; %muHhatL
p(21)=1/4; %muHhatA
p(24)=1; %c
p(26)=r; %r
p(27)=q; %q


%----------------------------
p(22)=0.002; %betaL (variable)
p(23)=0.004; %betaP (variable)
%----------------------------
p(25)=1/45; %(1/45 = no nicotine effect) mortality of larva-attackers in N+ patch
%----------------------------
```

```
%time span
tspan = [0  7500];

%initial conditions
y0 = zeros(20,1);

y0(1) = 100; %host larva on N-
y0(3) = 100; %host adults on N-
y0(4) = 100; %host larva on N+
y0(6) = 100; %host adults on N+
y0(10) = 100; %parasite adults in N-
y0(14) = 100; %parasite adults in N+
y0(17) = 100; %early attacker, hyperparasites %r
y0(20) = 100; %late attacker, hyperparasites %q

options=['reltol',1e-14,'abstol',1e-14];
[T,Y] = ode45(@twopatch,tspan,y0,options,p);
%-------------------------------------------------------------------------

%Finding Fitness-----------------------

%Average of last 100 points in the two patch simulation.  Due to
%oscillations occuring frequently
eqPL = mean2(Y(end-99:end,8)); %take the equilibrium point for Parasite Larva in N-
eqPLp = mean2(Y(end-99:end, 12));%equilibrium point for parasite larva in N+
eqPP = mean2(Y(end-99:end, 9)); %eq for parasite pupa, N-
eqPPp = mean2(Y(end-99:end, 13)); %eq for parasite pupa, N+

%Fitness vector
Fit = zeros(4,1);
%FHp = Fit(1); %fitness of early attacker in N+
```

```
%Fhatp = Fit(2); % late attacker N+
%FHn = Fit(3);
%Fhatn = Fit(4);

%parameters
beta1 = p(22);
beta2 = p(23);
gHL = p(13);
gHhatL = p(14);
muHLplus = p(25);
muHL = p(18);
muHhatL = p(20);
muHA = p(19);
muHhatA = p(21);

%Find fitness for each group of hyperparasites
Fit(1) = beta1*eqPLp*(gHL/(gHL+muHLplus))*(1/muHA); %N+, early attackers
Fit(2) = beta2*eqPPp*(gHhatL/(gHhatL+muHhatL))*(1/muHhatA); %N+, late attackers
Fit(3) = beta1*eqPL*(gHL/(gHL+muHL))*(1/muHA); %early attacker N-
Fit(4) = beta2*eqPP*(gHhatL/(gHhatL+muHhatL))*(1/muHhatA); %late attacker N-

RSS = abs(Fit(1)-Fit(3))+abs(Fit(2)-Fit(4)); %both
%RSS = abs(Fit(1)-Fit(3));  %r, remove y0(20)
%RSS = abs(Fit(2)-Fit(4));  %q, remove y0(17)


% comment them out during fminsearch----------------------
%Fit
%Fit(1)+Fit(3)
%Fit(2)+Fit(4)


%--------------------% twopatch.m %---------------------------%
```

```
%Functional file for the two patch system.

function[Y] = twopatch(t,y,p);

Y = zeros(20,1); %nineteen groups/variables in the two patch model


b = p(1); %rate of eggs that hatch from adults
muL = p(2); %mortality rate of host larva
gL = p(3);%growth rate of host larva
alpha1 = p(4); %rate of attack on nicotine free by parasite
alpha2 = p(5); %rate of attack on nicotine positive by parasite
gP = p(6); %growth rate of host pupae
muA = p(7); %mortality rate of host adults
K = p(8); %limiting factor
muPE = p(9); %mortality rate of parasite eggs
gPE = p(10); %growth rate of parasite eggs
gPL = p(11); %growth rate of parasite larva
gPP = p(12); %growth rate of parasite pupae
gHL = p(13); %growth rate of early attacking hyperparasite larva
gHhatL = p(14); %growth rate of later attacking hyperparasite larva
muPL = p(15); %mortality rate of parasite larva
muPP = p(16); %mortality rate of parasite pupae
muPA = p(17); %mortality rate of parasite adults
muHL = p(18); %mortality rate of early h.parasite larva
muHA = p(19); %mortality rate of early h.parasite adults
muHhatL = p(20); %mortality rate of late h.parasite larva
muHhatA = p(21); %mortality rate of late h.parasite adults
beta1 = p(22); %rate of successful oviposition on parasite larva by h.parasites
beta2 = p(23); %rate of successful oviposition on parasite pupae by h.parasites
c = p(24); %average clutch size
muHLplus=p(25); %mortality rate for larval-hypers in the nicotine patch
r = p(26); %placeholder for 'p', probability of attack by H to N-
```

```
q = p(27); %placeholder for q, probability  of attack by H-hat to N-


%a little 'p' will denote those in the nicotine (+) positive patch
L = y(1); %host larva in nicotine (-) free patch
P = y(2); %host pupa in nicotine (-) free patch
A = y(3); %host adults in N-
Lp = y(4); % host larva, nicotine (+) positive patch
Pp = y(5); %host pupa, nicotine (+) positive patch
Ap = y(6); %host adults in N+
PE = y(7); %parasite eggs in nicotine (-) free patch
PL = y(8); %parasite larva in nicotine (-) free patch
PP = y(9); %parasite pupa in nicotine (-) free patch
PA = y(10); %parasite adults in nicotine (-) free patch
PEp = y(11); %parasite eggs, nicotine (+) positive patch
PLp= y(12); %parasite larva, nicotine (+) positive patch
PPp= y(13); %parasite pupa, nicotine (+) positive patch
PAp= y(14); %parasite adults, nicotine (+) positive patch
HL= y(15); %larva-attacking hyperparasite larva, nicotine (-) free patch
HLp= y(16); %larva-attacking hyperparasite larva, nicotine (+) positive patch
HA= y(17); %larva-attacking hyperparasite adults, chooser
HhatL= y(18); %pupa-attacking h.parasite larva nicotine (-) free patch
HhatLp= y(19); %pupa-attacking h.parasite larva, nicotine (+) positive patch
HhatA= y(20); %pupa-attacking h.parasite adults, chooser

%Hosts on nic free
Y(1) = b*(1-L/K)*A-muL*L-gL*L-alpha1*PA*L;
Y(2) = gL*L-gP*P;
Y(3) = gP*P-muA*A;

%Hosts on nic pos
Y(4) = b*(1-Lp/K)*Ap-muL*Lp-gL*Lp-alpha2*PAp*Lp;
Y(5) = gL*Lp-gP*Pp;
```

```
Y(6) = gP*Pp-muA*Ap;

%Parasitoids on nic free
Y(7) = c*alpha1*PA*L-(muPE+gPE)*PE;
Y(8) = gPE*PE-(gPL+muPL)*PL-r*beta1*HA*PL; %L-attacking H.Paras. in N(-) go to Y(14)
Y(9) = gPL*PL-(gPP+muPP)*PP-q*beta2*HhatA*PP;
Y(10) = gPP*PP-muPA*PA;

%Parasitoids on nic pos
Y(11) = c*alpha2*PAp*Lp-(gPE+muPE)*PEp;
Y(12) = gPE*PEp-(gPL+muPL)*PLp-(1-r)*beta1*HA*PLp;
Y(13) = gPL*PLp-(gPP+muPP)*PPp-(1-q)*beta2*HhatA*PPp;
Y(14) = gPP*PPp-muPA*PAp;

%Early attacking hyperparasitoids
Y(15) = r*beta1*HA*PL-(gHL+muHL)*HL;
Y(16) = (1-r)*beta1*HA*PLp-(gHL+muHLplus)*HLp;
Y(17) = gHL*(HL+HLp)-muHA*HA;

%Late attacking hyperparasitoids
Y(18) = q*beta2*HhatA*PP-(gHhatL+muHhatL)*HhatL;
Y(19) = (1-q)*beta2*HhatA*PPp-(gHhatL+muHhatL)*HhatLp;
Y(20) = gHhatL*(HhatL+HhatLp)-muHhatA*HhatA;
```

Simulation files

```
%-----------------% gohyperparasite.m %------------------------------%
% Used to simulate the single patch model.  Calls the hyperparasite.m file
% but contains the parameters and initial conditions needed to simulate it.
clear all

p = zeros(22,1);
```

```
p(1)=3.3; %b
p(2)=1/100; %muL
p(3)=1/26; %gL
p(4)=0.002; %alpha (variable)
p(5)=1/21; %gP
p(6)=1/60; %muA
p(7)=250; %K (determined by author)
p(8)=1/4;%muPE
p(9)=1/4; %gPE
p(10)=1/11; %gPL
p(11)=1/6; %gPP
p(12)=4/45; %gHL (early)
p(13)=4/45; %gHhatL (late)
p(14)=7/600; %muPL
p(15)=0; %muPP (1/1000 in Thorpe and Barbosa, ignored as a negligable change)
p(16)=2/9;%muPA
%-------------------------
p(17)=1/3; %muHL (early) (changes with Nicotine)
%-------------------------
p(18)=1/8; %muHA
p(19)=1/45; %muHhatL (late)
p(20)=1/8; %muHhatA
p(21)=0.002; %betaL (variable)
p(22)=0.008; %betaP (variable)
p(23)=10; %c


tspan = [0 3500]; %time span

y0 = zeros(11,1);%initial conditions
y0(1)=100; %host larva
y0(3)=100; %host adults
y0(7)=100; %parasite adults
```

```
y0(8)=100; %larva-attacking parasite adults
y0(10)=100; %pupa-attacking parasite adults

options=['reltol',1e-10,'abstol',1e-10];
[T,Y] = ode45(@hyperparasite,tspan,y0,options,p);

%Graphs Host population vs time
subplot(2,2,1);
plot(T,Y(:,1),T,Y(:,2),T,Y(:,3),'linewidth',2);
title('Hosts')
xlabel('T')
legend('Larvae','Pupae','Adult')

%Graphs the parasitoid population vs time
subplot(2,2,2);
plot(T,Y(:,4),T,Y(:,5),T,Y(:,6),T,Y(:,7),'linewidth',2);
title('Primary Parasitoids')
xlabel('T')
legend('Eggs','Larva','Pupae','Adult')

%Graphs the early attacking hyperparsitoids
subplot(2,2,3);
plot(T,Y(:,8),T,Y(:,9),'linewidth',2);
title('Larvae-attacking Hyperparasitoid')
xlabel('T')
legend('Adults','Larva')

%graphs the late attacking hypers
subplot(2,2,4);
plot(T,Y(:,10),T,Y(:,11),'linewidth',2);
title('Pupae-attacking hyperparasitoids')
xlabel('T')
legend('Adults','Larva')
```

```
%-------------Two patch model for hosts-parasitoids-hyparasitoids---------
%simulates the two patch system for populations of hosts and parsitoids in
%different patches with hyperparasitoids moving and choosing between
%patches
clear all


% Set parameters
p = zeros(27,1);

p(1)=3.3; %b
p(2)=1/100; %muL
p(3)=1/26; %gL
p(4)=0.02; %alpha1
p(5)=0.02; %alpha2
p(6)=1/21; %gP
p(7)=1/60; %muA
p(8)=125; %K (determined by author)
p(9)=1/4;%muPE
p(10)=1/4; %gPE
p(11)=1/11; %gPL
p(12)=1/6; %gPP
p(13)=4/45; %gHL (early)
p(14)=4/45; %gHhatL (late)
p(15)=7/600; %muPL
p(16)=0; %muPP (1/1000 in Thorpe and Barbosa, ignored as a negligable change)
p(17)=2/9;%muPA
p(18)=1/45; %muHL
p(19)=2/9; %muHA
p(20)=1/45; %muHhatL (late)
p(21)=2/9; %muHhatA
p(24)=1; %c
%----------------------------
```

```
p(22)=0.002; %betaL (variable)
p(23)=0.006; %betaP (variable)
%----------------------------
%--------------------------------
p(25)=1; %mortality of larva-attackers in N+ patch
p(26)=0.5; %r---probability of early attackers choosing N-
p(27)=0.5; %q---probability of early attackers choosing N+
%--------------------------------

%time span
tspan = [0  7500];

%initial conditions
y0 = zeros(20,1);

y0(1) = 100; %host larva on N-
y0(3) = 100; %host adults on N-
y0(4) = 100; %host larva on N+
y0(6) = 100; %host adults on N+
y0(10) = 100; %parasite adults in N-
y0(14) = 100; %parasite adults in N+
y0(17) = 100; %early attacker, hyperparasites, %r
y0(20) = 100; %late attacker, hyperparasites, %q

options=['reltol',1e-10,'abstol',1e-10];
[T,Y] = ode45(@twopatch,tspan,y0,options,p);

%Uncomment to allow program to output the hyperparasitoid populations.
%Y(end,15)
%Y(end,16)
%Y(end,17)
%Y(end,18)
%Y(end,19)
```

```
%Y(end,20)


%Plotting for each species individually
%Hosts both on N- and N+
subplot(2,2,1);
plot(T,Y(:,1),T,Y(:,2),T,Y(:,3),T,Y(:,4),T,Y(:,5),T,Y(:,6),'linewidth',2);
title('Hosts')
legend('L','P','A','L+','P+','A+')

%Parasitoids on N-
subplot(2,2,2);
plot(T,Y(:,7),T,Y(:,8),T,Y(:,9),T,Y(:,10),'linewidth',2);
title('Parasitoids in N-')
legend('PE','PL','PP','PA')

%Parasitoids on N+
subplot(2,2,3);
plot(T,Y(:,11),T,Y(:,12),T,Y(:,13),T,Y(:,14),'linewidth',2);
title('parasitoids in N+')
legend('PE+','PL+','PP+','PA+')

%All hyperparasitoid populations
subplot(2,2,4);
plot(T,Y(:,15),T,Y(:,16),T,Y(:,17),T,Y(:,18),T,Y(:,19),T,Y(:,20),'linewidth',2)
title('Hyperparasitoids')
legend('HL','HL+','HA','HhatL','HhatL+','Hhata')
```

Bifurcation diagram files

```
%Used to create bifurcation diagrams across the muHL parameter (mortality
%rate of the early attackers.  It simulates using the gohyperparasite.m
```

```
%file
clear all

p = zeros(22,1);
p(1)=3.3; %b (from Briggs)
p(2)=1/100; %muL
p(3)=1/26; %gL
p(4)=0.02; %alpha (variable)
p(5)=1/21; %gP
p(6)=1/60; %muA
p(7)=250; %K (determined by author)
p(8)=1/4;%muPE
p(9)=1/4; %gPE
p(10)=1/11; %gPL
p(11)=1/6; %gPP (Thorpe and Barbosa)
p(12)=4/45; %gHL (early)
p(13)=4/45; %gHhatL (late)
p(14)=7/600; %muPL (Thorpe and Barbosa)
p(15)=0; %muPP
p(16)=2/9;%muPA
%p(17)=1/7; %muHL (early) %parameter being bifurcated across
p(18)=2/9; %muHA
p(19)=1/45; %muHhatL (late)
p(20)=2/9; %muHhatA
p(21)=0.002; %betaL (variable)
p(22)=0.006; %betaP (variable)
p(23)=1; %c

%muVec = zeros(200,1);
%Htotal = zeros(50,200);
%Hhattotal = zeros(50,200);
tick = 0;
for i=1 : 1200;
```

```
    tick = tick + 1;
p(17)= 0.0005*i;
muVec(tick,1) = p(17);

tspan = [0 5000]; %time span

y0 = zeros(11,1);%initial conditions
y0(1)=100; %host larva
y0(3)=100; %host adults
y0(7)=100; %parasite adults
y0(8)=100; %larva-attacking parasite adults
y0(10)=100; %pupa-attacking parasite adults


options=['reltol',1e-10,'abstol',1e-10];
[T,Y] = ode45(@hyperparasite,tspan,y0,options,p);

%save last 50 points to a matrix for H and Hhat
  Htotal(:,tick) = Y(end-49:end,8)+Y(end-49:end,9);
  Hhattotal(:,tick) = Y(end-49:end, 10) + Y(end-49:end,11);

end

%graphing the hyperparasitoid populations against the parameter muHL
plot(muVec,Htotal,'b.',muVec,Hhattotal,'r.')
legend('Larvae-attacking','b.','Pupae-attacking','r.')
title('Bifurcation along \mu_{H_L}')
xlabel('\mu_{H_L},  rate of mortality for larvae-attacking hyperparasitoid larva')
ylabel('Hyperparasitoid Population Total')


%Used to creat bifurcation diagrams of k, the ratio of betaP to betaL.
% k = betaL / betaP
```

```
clear all

p = zeros(22,1);
p(1)=3.3; %b (from Briggs)
p(2)=1/100; %muL
p(3)=1/26; %gL
p(4)=0.02; %alpha (variable)
p(5)=1/21; %gP
p(6)=1/60; %muA
p(7)=250; %K (determined by author)
p(8)=1/4;%muPE
p(9)=1/4; %gPE
p(10)=1/11; %gPL
p(11)=1/6; %gPP (Thorpe and Barbosa)
p(12)=4/45; %gHL (early)
p(13)=4/45; %gHhatL (late)
p(14)=7/600; %muPL (Thorpe and Barbosa)
p(15)=0; %muPP (1/1000 in Thorpe and Barbosa, ignored as a negligable change)
p(16)=2/9;%muPA
p(18)=2/9; %muHA
p(19)=1/45; %muHhatL (late)
p(20)=2/9; %muHhatA
p(23)=1; %c

%---------------------
p(17)=2/3; %muHL (early) %change with nicotine
%---------------------
p(21)=0.002; %betaL (variable)
%p(22)=k*p(21); %betaP (variable)
%---------------------
```

```
%muVec = zeros(200,1);
%Htotal = zeros(50,200);
%Hhattotal = zeros(50,200);

tick = 0;
for i=1 : 1600;
    tick = tick + 1;
    k = 0.005*tick; %change for a finer mesh
p(22)= k*p(21);
betaratio(tick,1) = k;

tspan = [0 3000]; %time span

y0 = zeros(11,1); %initial conditions
y0(1)=100; %host larva
y0(3)=100; %host adults
y0(7)=100; %parasite adults
y0(8)=100; %larva-attacking parasite adults
y0(10)=100; %pupa-attacking parasite adults


options=['reltol',1e-10,'abstol',1e-10];
[T,Y] = ode45(@hyperparasite,tspan,y0,options,p);

%save last 100 points to a matrix for H and Hhat
  Htotal(tick,:) = Y(end-99:end,8)+Y(end-99:end,9);
  Hhattotal(tick,:) = Y(end-99:end, 10) + Y(end-99:end,11);

end

%graph the bifurcation of k vs the total population of hyperparasitoids
plot(betaratio,Htotal,'b.',betaratio,Hhattotal,'r.')
title('Bifurcation along Beta Ratio, k')
```

```
xlabel('k')
ylabel('Hyperparasite Population Total')
legend('Larval-attacking','Pupal-attacking')
```

Vita

Mark Patrick Zimmerman was born September 9, 1988 in Charlottesville, Virginia. Raised in Virginia, he knew at an early age he wanted to become a scientist. He graduated valedictorian with honors from William Monroe High School and the Blue Ridge Virtual Governor's School. Mark was offered the Provost scholarship and attended Virginia Commonwealth University in Richmond, Virginia for his Bachelor's degree.

While at VCU, Mark worked as a math and science tutor and interned in the Department of Human and Molecular Genetics under Dr. Joe Landry. Majoring in biology with minors in chemistry and mathematics he graduated Magna Cum Laude in 2011.

He worked at the Virginia state lab in Richmond and as an SAT tutor before reentering VCU in 2013 to earn his Master's Degree in Applied Mathematics from VCU. He was a graduate teaching assistant in pre-calculus and taught contemporary mathematics to freshman and sophomore. Mark is engaged to Anna Dillon with the wedding set for May of 2016. He is moving to Raleigh, North Carolina with Anna, his fiancée, and Pip, their dog, to pursue a doctoral degree in biomathematics at North Carolina State University where he has accepted an offer of an RTG/NFS Fellowship.