2016

A two host species stage-structured model of West Nile virus transmission

Taylor A. Beebe
Virginia Commonwealth University

A TWO HOST SPECIES STAGE-STRUCTURED MODEL OF *West Nile virus* TRANSMISSION

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

by

Taylor Ann Beebe
Master of Science

Director: Dr. Suzanne Robertson, Assistant Professor
Department of Mathematics and Applied Mathematics

Virginia Commonwealth University
Richmond, Virginia
May 2016

Acknowledgments

A special thank you to my thesis advisor, Dr. Suzanne Robertson, without whom none of this would have been possible. Your guidance, encouragement, and patience were instrumental in this process.

To the other members of my committee, Dr. Rebecca Segal and Dr. Derek Johnson, thank you for being a part of this journey by offering your time and helpful advice in contribution to the improvement of this thesis.

To my family and friends, thank you for your unconditional support, this would not have been possible without you. Also to Christopher, thank you for your patience and support, and for reminding me that 'I can do this' when I needed it most.

Table of Contents

List of Figures

List of Tables

Abstract

We develop and evaluate a novel host-vector model of *West Nile virus* (WNV) transmission that incorporates multiple avian host species and host stage-structure (juvenile and adult stages), with both species-specific and stage-specific biting rates of vectors on hosts. We use this model to explore WNV transmission dynamics that occur between vectors and multiple structured host populations as a result of heterogeneous biting rates. Our analysis shows that increased exposure of juvenile hosts results in earlier, more intense WNV transmission when compared to the effects of differential host species exposure, regardless of other parameter values. We also find that, in addition to competence, increased juvenile exposure is an important mechanism for determining the effect of species diversity on the disease risk of a community.

Chapter 1

Introduction

1.1 Biological Background

The first case of *West Nile virus* (WNV) was identified in a woman in the West Nile district of Uganda in 1937 [26]. It wasn't introduced to the United States until 1999, and upon introduction it produced a large outbreak in New York that has continued to spread annually during the summer months and can now be found across the United States and most of North America [14, 26]. Since the time of its introduction through 2014, the Center for Disease Control and Prevention (CDC) has recorded over 41,000 cases of West Nile virus infection and over 1,700 have resulted in death. Annual transmission of WNV is highly seasonal, with the majority of infections reported in the summer months between July and September [6].

WNV persists in nature through a host-vector-host interaction cycle, with the principal vectors generally being mosquitoes of the genus *Culex*, specifically *Culex pipens*. Many types of hosts can become infected with WNV, however the most common reservoir hosts are birds, with WNV having been detected in more than 250 different species [26].

Studies have shown evidence of increased biting rates on some species of avian hosts

relative to their abundance [10]. This may be a result of either vector preference for certain species or increased availability of these hosts. Some species of birds may be more available, or exposed, to mosquitoes depending on nesting type (cavity or open nests) or nest height. Within a species, exposure may also vary with age. Increased juvenile stage exposure may occur as a result of low feather coverage, immobility, or lack of defense mechanisms [3, 8, 22]. Recent models have suggested that for vector borne diseases, feeding preferences both for species and stages play a very important role in WNV transmission, specifically the intensity and timing of WNV outbreaks [13, 20, 23].

Experimental infection studies have shown that many avian species vary in competence (the measure of a host's ability to contract and transmit the disease) [14]. Heterogeneity in competence and feeding preferences can play an important role in WNV transmission [19]. Other vector-borne diseases with similar interaction cycles have juvenile stages with increased competence relative to adult hosts, which is thought to drive disease risk [16, 24]. Despite disease similarities with WNV, this has not yet been shown for WNV.

1.2 Model Background

The first generalized SIR (susceptible-infected-recovered) model was proposed by W. O. Kermack and A. G. McKendrick in 1927 in order to explain fluctuations in the infected populations dynamics that occurred in epidemics such as the plague or cholera [12]. Their assumptions included a fixed population size, equal contact rates across all individuals, and omitted birth and death rates. Similarly, most early epidemic models assume a fixed population size with contact rates independent of population size [2]. Over time, many elaborations of the SIR model were created to model diseases with different life cycles or transmission characteristics.

Specifically, the SIR framework has been extended to model transmission of vector-

borne diseases, including *West Nile virus*. In 2001, Lord and Day developed a stage-structured model of *St. Louis Encephalitis virus* with a single recovered class for juveniles and adults with year round recruitment, later incorporating mortality from infection to model WNV [16, 17]. In 2004 Wonham et al. developed a single species WNV model with no stage-structure. They found that the form of the basic reproduction number R_0 (number of secondary infections from a single typical infected individual introduced into a completely susceptible population) suggests that host reduction may be ineffective for WNV control, and would actually increase WNV transmission by increasing the vector to susceptible host ratio [25]. In 2006 Unnasch et al. developed a two stage (young-of-year (YOY) and adult) model for *Eastern Equine Encephalitis virus* which included many stage specific parameters such as biting rate, susceptibility, and competence, and found that preferential feeding on YOY birds plays a key role in establishing and maintaining enzootic outbreaks [24]. In 2012 Simpson et al. developed a two species host-vector model of preferred and alternative avian hosts and found host preference-induced contact heterogeneity to be a driving factor in WNV enzootic transmission [23]. Finally, in 2013 Miller and Huppert created a host-vector model with two host species and examined the effects of host diversity and its implications on disease risk. They discussed the effects of differences in transmission related model parameters and vector feeding preferences and examined scenarios under which increased species diversity can dilute or amplify vector-borne disease transmission [19].

For WNV, there have been a number of models incorporating multiple host species [1, 19, 23, 25] as well as multiple host life stages [16, 17, 20], however to our knowledge there has not been any formal analysis done on a multiple host species, stage-structured model. The model proposed in this thesis is a two host species stage-structured model with variable species-specific and stage-specific biting rates of vectors on hosts. We use this model to investigate WNV transmission dynamics that occur between vectors and multiple structured host populations (specifically while including heterogeneous biting

rates, differential abundance, differential competence, and differential nesting parameters).

Chapter 2

Model Development

In this chapter, we develop a novel host-vector model for WNV (see Figure 2.1) that incorporates multiple host species and host stage-structure, as well as both species-specific and stage-specific biting rates of vectors on hosts. The modeling framework is based on that of Robertson and Caillouet [20]. We have modified their single species, three stage model to include two distinct host species each with two age classes, juvenile and adult. We use this model to explore the effect of both differential host species and host stage biting rates on WNV transmission. Traditionally, disease risk is measured by the basic reproduction number R_0, which is the number of secondary infections produced by introducing a single typical infected individual into a completely susceptible population [7]. Since our model is non-autonomous and the population is not at a disease-free steady-state, we do not use R_0. Intensity of transmission is also often measured by prevalence (number of infectious mosquitoes/1000 mosquitoes), however in this case we do not use prevalence as a measure of disease risk because our vector abundance is variable. Instead we measure disease risk by the number of peak infectious vectors (IV), the timing of the peak IV, and the total IV over the course of a single season. We look at these indicators in infected mosquitoes because they are among the greatest indicators for *West Nile virus* risk to humans (who can become infected by mosquitoes but cannot

transmit the disease). Throughout this thesis, we will refer to unequal biting rates on host species or stages as a vector "preference," though we acknowledge the biting rates may be due to other factors such as increased exposure or availability of certain host species or stages.

We will first discuss the development of the host equations and then conclude with the development of the vector equations. We condense the host life cycle into two stages, juvenile (J) and adult (A). Using differential equations, we model the number of susceptible, infected, and recovered birds over time in each stage for two different species of hosts. This results in twelve host differential equations. In order to differentiate between the two host species, anything pertaining to host species 2 (equation, function, or variable), will be denoted with a ˆ(hat) symbol.

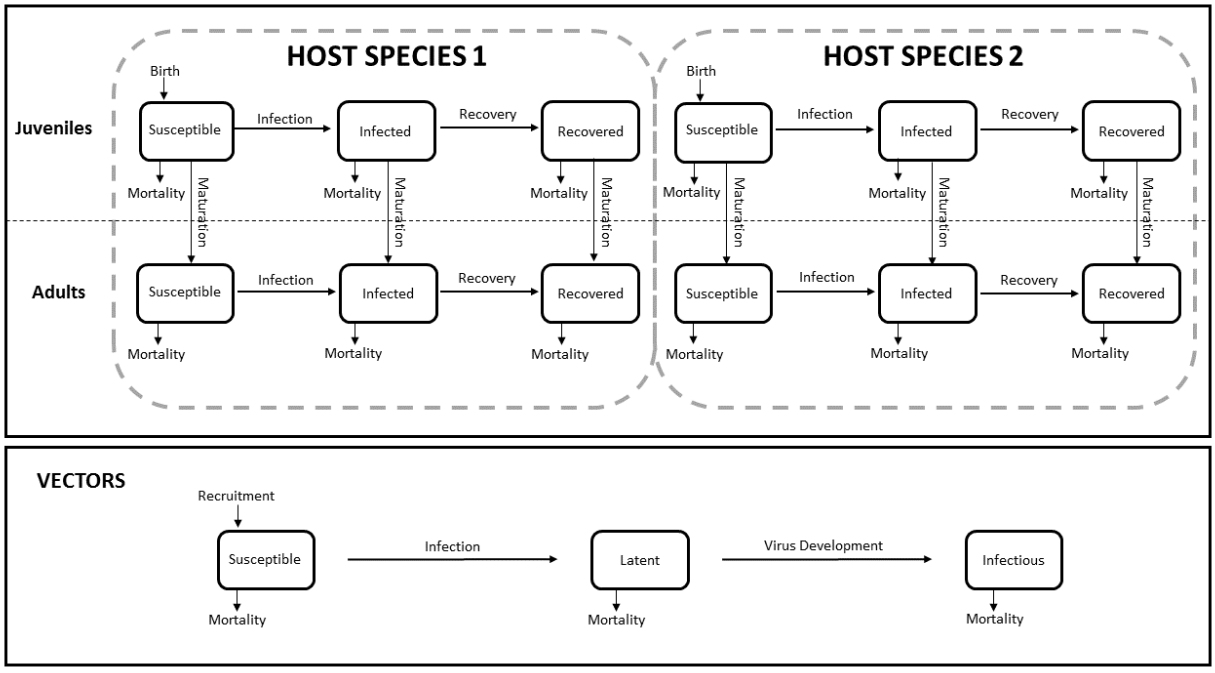

Figure 2.1: *West Nile virus* multi-host stage-structured model schematic. Shows all of the possible life cycles of both host species 1 and 2 as well as vectors.

The complete model is comprised of 15 differential equations (12 host equations and 3 vector equations) and defined as follows:

$$
\begin{aligned}
\frac{dJ_S}{dt} &= b(t) - a\alpha\alpha_J\beta_J M_I\frac{J_S}{J_T} - (m_J + \mu_J)J_S \\
\frac{dJ_I}{dt} &= a\alpha\alpha_J\beta_J M_I\frac{J_S}{J_T} - (m_J + \mu_J + \gamma_J + \nu_J)J_I \\
\frac{dJ_R}{dt} &= \gamma_J J_I - (m_J + \mu_J)J_R \\
\frac{dA_S}{dt} &= -a\alpha\alpha_A\beta_A M_I\frac{A_S}{A_T} + m_J J_S - \mu_A A_S \\
\frac{dA_I}{dt} &= a\alpha\alpha_A\beta_A M_I\frac{A_S}{A_T} + m_J J_I - (\mu_A + \gamma_A + \nu_A)A_I \\
\frac{dA_R}{dt} &= \gamma_A A_I - \mu_A A_R + m_J J_R \\
\frac{d\hat{J}_S}{dt} &= \hat{b}(t) - a\hat{\alpha}\alpha_{\hat{J}}\beta_{\hat{J}} M_I\frac{\hat{J}_S}{\hat{J}_T} - (m_{\hat{J}} + \mu_{\hat{J}})\hat{J}_S \\
\frac{d\hat{J}_I}{dt} &= a\hat{\alpha}\alpha_{\hat{J}}\beta_{\hat{J}} M_I\frac{\hat{J}_S}{\hat{J}_T} - (m_{\hat{J}} + \mu_{\hat{J}} + \gamma_{\hat{J}} + \nu_{\hat{J}})\hat{J}_I \\
\frac{d\hat{J_R}}{dt} &= \gamma_{\hat{J}}\hat{J}_I - (m_{\hat{J}} + \mu_{\hat{J}})\hat{J}_R \\
\frac{d\hat{A}_S}{dt} &= -a\hat{\alpha}\alpha_{\hat{A}}\beta_{\hat{A}} M_I\frac{\hat{A}_S}{\hat{A}_T} + m_{\hat{J}}\hat{J}_S - \mu_{\hat{A}}\hat{A}_S \\
\frac{d\hat{A}_I}{dt} &= a\hat{\alpha}\alpha_{\hat{A}}\beta_{\hat{A}} M_I\frac{\hat{A}_S}{\hat{A}_T} + m_{\hat{J}}\hat{J}_I - (\mu_{\hat{A}} + \gamma_{\hat{A}} + \nu_{\hat{A}})\hat{A}_I \\
\frac{d\hat{A}_R}{dt} &= \gamma_{\hat{A}}\hat{A}_I - \mu_{\hat{A}}\hat{A}_R + m_{\hat{J}}\hat{J}_R \\
\frac{dM_S}{dt} &= rM_T(1 - \frac{M_T}{K}) - a(\alpha\alpha_J\delta_J\frac{J_I}{J_T} + \alpha\alpha_A\delta_A\frac{A_I}{A_T} + \hat{\alpha}\alpha_{\hat{J}}\delta_{\hat{J}}\frac{\hat{J}_I}{\hat{J}_T} + \hat{\alpha}\alpha_{\hat{A}}\delta_{\hat{A}}\frac{\hat{A}_I}{\hat{A}_T})M_S - \mu_M(t)M_S \\
\frac{dM_L}{dt} &= a(\alpha\alpha_J\delta_J\frac{J_I}{J_T} + \alpha\alpha_A\delta_A\frac{A_I}{A_T} + \hat{\alpha}\alpha_{\hat{J}}\delta_{\hat{J}}\frac{\hat{J}_I}{\hat{J}_T} + \hat{\alpha}\alpha_{\hat{A}}\delta_{\hat{A}}\frac{\hat{A}_I}{\hat{A}_T})M_S - kM_L - \mu_M(t)M_L \\
\frac{dM_I}{dt} &= kM_L - \mu_M(t)M_I
\end{aligned}
\tag{2.1}
$$

Table 2.1: West Nile virus model baseline parameters. All parameter values are from Robertson and Caillouet [20]

Parameter	Description	Baseline
a	Biting rate on competent avian hosts	0.133 bites/mosquito/day
ϵ	Host species 1 exposure coefficient	varies
$\hat{\epsilon}$	Host species 2 exposure coefficient	varies
ϵ_J	Juvenile species 1 exposure coefficient	varies
ϵ_A	Adult species 1 exposure coefficient	varies
$\epsilon_{\hat{J}}$	Juvenile species 2 exposure coefficient	varies
$\epsilon_{\hat{A}}$	Adult species 2 exposure coefficient	varies
m_J	Juvenile species 1 maturation rate	$1/14$ days^{-1}
$m_{\hat{J}}$	Juvenile species 2 maturation rate	$1/14$ days^{-1}
μ_J	Juvenile species 1 natural mortality rate	0.0014 days^{-1}
μ_A	Adult species 1 natural mortality rate	0.0014 days^{-1}
$\mu_{\hat{J}}$	Juvenile species 2 natural mortality rate	0.0014 days^{-1}
$\mu_{\hat{A}}$	Adult species 2 natural mortality rate	0.0014 days^{-1}
β_J	Juvenile species 1 susceptibility	1
β_A	Adult species 1 susceptibility	1
$\beta_{\hat{J}}$	Juvenile species 2 susceptibility	1
$\beta_{\hat{A}}$	Adult species 2 susceptibility	1
γ_J	Juvenile species 1 recovery rate	$1/3$ days^{-1}
γ_A	Adult species 1 recovery rate	$1/3$ days^{-1}
$\gamma_{\hat{J}}$	Juvenile species 2 recovery rate	$1/3$ days^{-1}
$\gamma_{\hat{A}}$	Adult species 2 recovery rate	$1/3$ days^{-1}
δ_J	Juvenile species 1 infectivity	0.36
δ_A	Adult species 1 infectivity	0.36
$\delta_{\hat{J}}$	Juvenile species 2 infectivity	0.36
$\delta_{\hat{A}}$	Adult species 2 infectivity	0.36
ν_J	Juvenile species 1 virulence	0.1 days^{-1}
ν_A	Adult species 1 virulence	0.1 days^{-1}
$\nu_{\hat{J}}$	Juvenile species 2 virulence	0.1 days^{-1}
$\nu_{\hat{A}}$	Adult species 2 virulence	0.1 days^{-1}
q	Species 1 juvenile recruitment curve mean	Day 158.17
$\hat{q}$	Species 2 juvenile recruitment curve mean	Day 158.17
σ	Species 1 JRC standard deviation	11.4
$\hat{\sigma}$	Species 2 JRC standard deviation	11.4
f	Species 1 JRC scaling factor	285
$\hat{f}$	Species 2 JRC scaling factor	285
A_0	Number of adults of species 1 at start of season	150
$\hat{A}_0$	Number of adults of species 2 at start of season	150
r	Mosquito per capita birthrate	0.537 days^{-1}
K	Mosquito carrying capacity (larval)	15000
μ_M	Vector mortality rate	0.096 days^{-1}
k	Virus incubation period	0.106 days^{-1}
z	Start of Mosquito growth	Day 115

Hosts are born into the susceptible juvenile class at rate $b(t)$ for species 1, and rate $\hat{b}(t)$ for species 2. The functions $b(t)$ and $\hat{b}(t)$, also referred to as the the juvenile recruitment curves for host species 1 and host species 2, are modeled by the probability density functions of a Gaussian or Normal distribution, and given by

$$b(t) = \frac{f}{\sigma\sqrt{2\pi}} exp\left(\frac{-(q-t)^2}{2\sigma^2}\right) \tag{2.2}$$

$$\hat{b}(t) = \frac{\hat{f}}{\hat{\sigma}\sqrt{2\pi}} exp\left(\frac{-(\hat{q}-t)^2}{2\hat{\sigma}^2}\right). \tag{2.3}$$

The juvenile recruitment curves for species 1 and 2 have means of q and $\hat{q}$, variance σ and $\hat{\sigma}$, and are scaled by a factor f and $\hat{f}$.

Host juveniles can either move into another class within their stage (susceptible to infected, infected to recovered) or mature into the adult stage of their current class. The maturation rate (m_J or $m_{\hat{J}}$), is the inverse of the mean duration of the juvenile stage, i.e. $m_J = \frac{1}{14}\ days^{-1}$ indicates the species juvenile stage is expected to last 14 days.

For a susceptible host (juvenile or adult) to leave a susceptible class and move into an infected class it needs to become infected by being bitten by an infected mosquito (type I interaction, see Figure 2.2). Of all the bites by an infected mosquito on a susceptible host, only a fraction of them result in infection. The probability that infection will result from an infected mosquito biting a susceptible bird per bite is given by β_i, for $i = J, A, \hat{J}, \hat{A}$. The most prominent WNV vector in Virginia is *Culex pipens*, which feeds approximately once every three days. Of those meals, we assume that 80% of bites go to avian hosts, and approximately half of those avian hosts are competent (able to contract and spread the disease). The product of these terms give us the parameter a, the biting rate of mosquitoes on competent, avian hosts.

All mosquito bites on the modeled hosts are first distributed among the two host species with a fraction going to host species 1 and the remainder going to host species 2. The fraction of bites going to species 1 and species 2 are given by α and $\hat{\alpha}$, respectively,

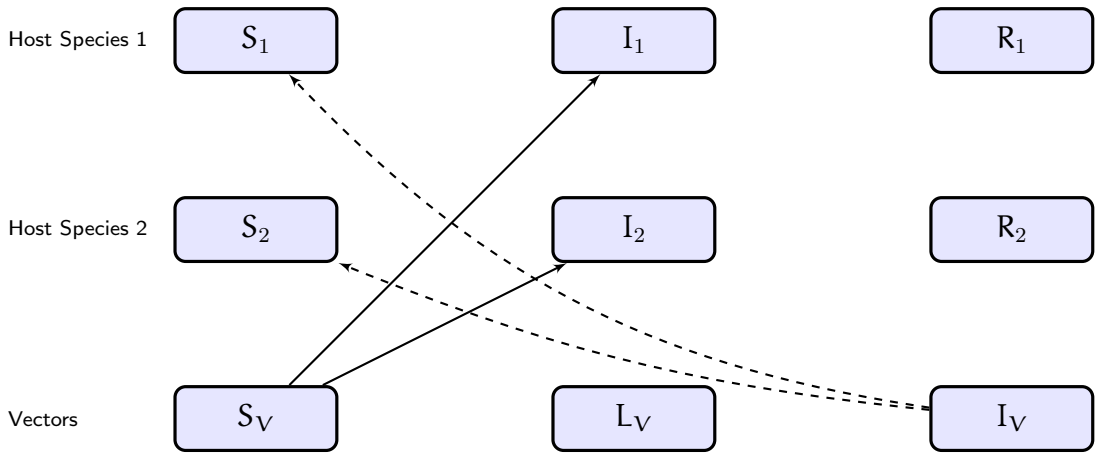

Figure 2.2: There are two possible interactions between host and vector that may result in infection. Type I, an infected vector biting a susceptible host is represented by the dashed line and results in host infection. Type II, a susceptible vector biting an infected host, is represented by the solid line and results in vector infection.

which are calculated by:

$$\alpha(t) = \frac{\epsilon(J_T(t) + A_T(t))}{\epsilon(J_T(t) + A_T(t)) + \hat{\epsilon}(\hat{J}_T(t) + \hat{A}_T(t))}$$
$$\hat{\alpha}(t) = \frac{\hat{\epsilon}(\hat{A}_T(t) + \hat{J}_T(t))}{\epsilon(J_T(t) + A_T(t)) + \hat{\epsilon}(\hat{J}_T(t) + \hat{A}_T(t))}$$

where the subscript T denotes total population (i.e., $J_T(t)$ is the total juvenile host species 1 population at time t). Since the total population for a stage is given by the sum of all of the classes (Susceptible, Infected, and Recovered), then $J_T(t) = J_S(t) + J_I(t) + J_R(t)$, $A_T(t) = A_S(t) + A_I(t) + A_R(t)$, $\hat{J}_T(t) = \hat{J}_S(t) + \hat{J}_I(t) + \hat{J}_R(t)$, and $\hat{A}_T(t) = \hat{A}_S(t) + \hat{A}_I(t) + \hat{A}_R(t)$. The constants ϵ, and $\hat{\epsilon}$ are the exposure coefficients for each species and determine how one species is bitten in proportion to the other. For example, if $\epsilon = \hat{\epsilon} = 1$ then both species are being bitten in proportion to their abundance in the population and all hosts receive the same number of bites. If $\epsilon > \hat{\epsilon}$ then species 1 is receiving more bites than species 2; we call this a species preference (species 1 preferred). If $\epsilon < \hat{\epsilon}$, then species 2 is receiving more bites than species 1; this is also a species preference (species 2 preferred). In this thesis, exposure coefficients usually vary between 1 and 15. We

consider a ratio of exposure coefficients of 10 or greater to be a strong preference, less than 5 to be a slight preference, and 5 to 9 an intermediate preference. It is important to note that these exposure coefficients are constant. They are assumed to be a characteristic of each species, while the proportion of bites going to each species ($\alpha(t)$ and $\hat{\alpha}(t)$) is a function that may change throughout the season depending on the total population of each species at a given time, t.

All mosquito bites on a host species are then distributed among each of the avian stages with a fraction going to juveniles ($J, \hat{J}$) and adults ($A, \hat{A}$) of each host species. The fraction of bites going to juveniles of species 1, adults of species 1, juveniles of species 2 and adults of species 2 is given by $\alpha_J, \alpha_A, \alpha_{\hat{J}}, \alpha_{\hat{A}}$ respectively, which are calculated by:

$$\begin{aligned}
\alpha_J(t) &= \frac{\epsilon_J J_T(t)}{\epsilon_J J_T(t) + \epsilon_A A_T(t)} \\
\alpha_A(t) &= \frac{\epsilon_A A_T(t)}{\epsilon_J J_T(t) + \epsilon_A A_T(t)} \\
\alpha_{\hat{J}}(t) &= \frac{\epsilon_{\hat{J}} \hat{J}_T(t)}{\epsilon_{\hat{J}} \hat{J}_T(t) + \epsilon_{\hat{A}} \hat{A}_T(t)} \\
\alpha_{\hat{A}}(t) &= \frac{\epsilon_{\hat{A}} \hat{A}_T(t)}{\epsilon_{\hat{J}} \hat{J}_T(t) + \epsilon_{\hat{A}} \hat{A}_T(t)}.
\end{aligned}$$

The constants ϵ_J, ϵ_A, $\epsilon_{\hat{J}}$, and $\epsilon_{\hat{A}}$ are the exposure coefficients for each stage and determine how frequently a stage is bitten relative to the other stage for that species. For example, if $\epsilon_J = \epsilon_A = \epsilon_{\hat{J}} = \epsilon_{\hat{A}} = 1$ then all stages are being bitten in proportion to their abundance in the population and all hosts receive the same number of bites. Or, if $\epsilon_J = \epsilon_{\hat{J}} > \epsilon_A = \epsilon_{\hat{A}}$ then the juveniles (of both species 1 and 2) are being bitten more than the adults (of species 1 and 2), and we call this a stage preference (i.e., juveniles as a whole are preferred over the adults). Similar to species preference values, stage preference values usually vary between 1 and 15. We consider a stage preference of 10 or greater to be a strong preference, less than 5 to be a slight preference, and 5 to 9 an intermediate preference. Again, it is important to note that these exposure coefficients

are constants, as they are assumed to be a characteristic of each stage, while the proportion of bites going to each stage ($\alpha_i(t)$ for $i = J, A, \hat{J}, \hat{A}$) is a function that may change throughout the season depending on the total population of each stage at a given time, t.

For a susceptible juvenile of host species 1 to become infected it must be bitten by an infectious mosquito and that bite must transmit the virus. To model this, we consider the following interaction: an infected mosquito (M_I) bites a competent avian juvenile host of host species 1. The bitten host is susceptible, and the bite results in infection. The product of the parameters associated with each of these terms gives us the rate at which juveniles of host species 1 become infected (i.e., leave the susceptible class and enter into the infected class). A similar term can be found in each of the other 3 susceptible equations ($\frac{dA_s}{dt}$, $\frac{d\hat{J}_s}{dt}$, $\frac{d\hat{A}_s}{dt}$).

The competence of a host is a measure of how likely that host is to contract and transmit the disease. Competence is calculated using the susceptibility (β_i for $i = J, A, \hat{J}, \hat{A}$), recovery rate ($\gamma_i$ for $i = J, A, \hat{J}, \hat{A}$) and the infectivity rate (δ_i for $i = J, A, \hat{J}, \hat{A}$) of each stage [15]. Recall, that we are assuming that all bites by infectious mosquitoes on hosts will result in transmission of WNV to hosts ($\beta_i = 1$ for $i = J, A, \hat{J}, \hat{A}$). Then, if a species or stage has a shorter recovery rate and a higher infectivity rate than other hosts, then we say that it is more competent. For example, if $\gamma_J = \gamma_A < \gamma_{\hat{J}} = \gamma_{\hat{A}}$, and $\delta_J = \delta_A > \delta_{\hat{J}} = \delta_{\hat{A}}$, then species 1 (juveniles and adults) is more competent than species 2 (juveniles and adults).

Another way that a host can leave a class is through death. In this model we consider both natural mortality, whose rates are given by μ_i for $i = J, A, \hat{J}, \hat{A}$, as well as mortality due to disease (virulence), which has rates ν_i for $i = J, A, \hat{J}, \hat{A}$. Infected hosts move into the recovered class at rate γ_i for $i = J, A, \hat{J}, \hat{A}$.

For the vectors, we model the susceptible (M_S), latent (M_L), and infectious (M_I) mosquitoes over time. Upon contracting WNV, susceptible vectors move from the sus-

ceptible class into the latent class. While in this class, vectors have WNV but they cannot transmit the diease to hosts. After the disease incubation period is over, vectors transition into the infectious class where they remain for the duration of their lifespan.

We assume that all mosquitoes are born susceptible. Mosquitoes are born into the susceptible class at rate r times a density limiting factor 1-$\frac{M_T}{K}$, where K is the larval mosquito carrying capacity. One way that vectors can leave the susceptible class is by becoming infected. This type of infection can only result from a susceptible vector biting an infected host, which then results in vector infection (type II infection, see Figure 2.2).

One possible interaction that could result in vector infection is a susceptible vector becoming infected by an infected juvenile of host species 1. To model this, we consider the following interaction: a susceptible mosquito (M_S) bites an infected juvenle host of species 1. The juvenile host is already infected and the bite results in infection for the mosquito. The product of the parameter values associated with each these terms gives the number of mosquito infections resulting from a susceptible mosquito biting an infected juvenile of host species 1. There are also similar terms for an infected adult of host species 1 infecting a susceptible mosquito, and infected juvenile and adults from host species 2 infecting a susceptible mosquito. The sum of these 4 terms gives the total number of new mosquito infections.

A vector can leave any of the classes through mortality. The death rate is density independent and is given by the term $\mu_M(t)$ which is a piecewise function defined as:

$$\mu_M(t) = \begin{cases} \mu_M & t \leqslant 240 \\ \mu_M + 0.001(t - 240) & t > 240 \end{cases} \tag{2.4}$$

which tells us that vector mortality is constant until day 240 and then increases linearly after day 240. This late season increase in mosquito mortality is necessary to ensure the mosquito population declines at the end of the season as observed in nature.

All model analysis is done through simulations in MATLAB. Each simulation will

simulate a single season beginning at Julian day 100. Each simulation will return 150 adult birds for each species, 70% of whom are susceptible and 30% recovered for each species. Mosquitoes are introduced at Julian day 115, with 99 susceptible and 1 infectious. Unless otherwise specified all parameter values are as in Table 2.1.

In this thesis we will answer the following questions:

- Chapter 3 - Is the common simplifying assumption of using weighted average parameter values in a single species model an adequate representation of the dynamics that occur in model 2.1? What are the consequences of this simplifying assumption?
- Chapter 4 - What are the effects of individually varying certain parameters (recovery rate, infectivity, competence, the standard deviation of the juvenile recruitment curve, and the mean of the juvenile recruitment curve) on the intensity and timing of WNV transmission in model 2.1?
- Chapter 5 - What are the effects of incorporating heterogeneous species specific and/or stage specific biting rates on the intensity and timing of WNV transmission in model 2.1?
- Chapter 6 - What are the differences in the intensity and timing of WNV transmission in model 2.1 when comparing increased species exposure with increased stage exposure in addition to certain species specific parameters (competence, standard deviation of the juvenile recruitment curve, and the mean of the juvenile recruitment curve)?
- Chapter 7 - Can we use model 2.1 to reproduce the results of dilution and diversity amplification found by Miller and Huppert [19]? What is the effect of incorporating increased biting rates on juvenile hosts on these results?

Chapter 3

Comparison of WNV Transmission Models with One and Two Host Species

Previous WNV models have included either stage-structure [16, 17, 20] or multiple species [19, 23, 25] but to our knowledge none have incorporated both. A common simplifying assumption is to model a single species, representing a typical host by using average (or weighted average) parameter values. Our goal in this chapter is to investigate the consequences of this simplifying assumption and show that the WNV transmission dynamics resulting from a single species model do not adequately represent the dynamics that occur in a two species model.

The first thing we do is reduce our model of WNV (model 2.1) to a single species model (see Figure 3.1). We show that using a two species model allows us to look at scenarios that we cannot consider with a single species model, and we get dynamics in a two species model that cannot be achieved using a single species model.

In the two species model we incorporate community composition, differential host competence, and heterogeneous biting rates on juveniles, and compare these dynamics with those from single species model. It is important to know whether a model will be able to accurately model these transmission dynamics because all three of these host

characteristics occur regularly in nature. These host characteristics are all things that we can clearly model with a two species model however we would not traditionally attempt to do so with a single species model.

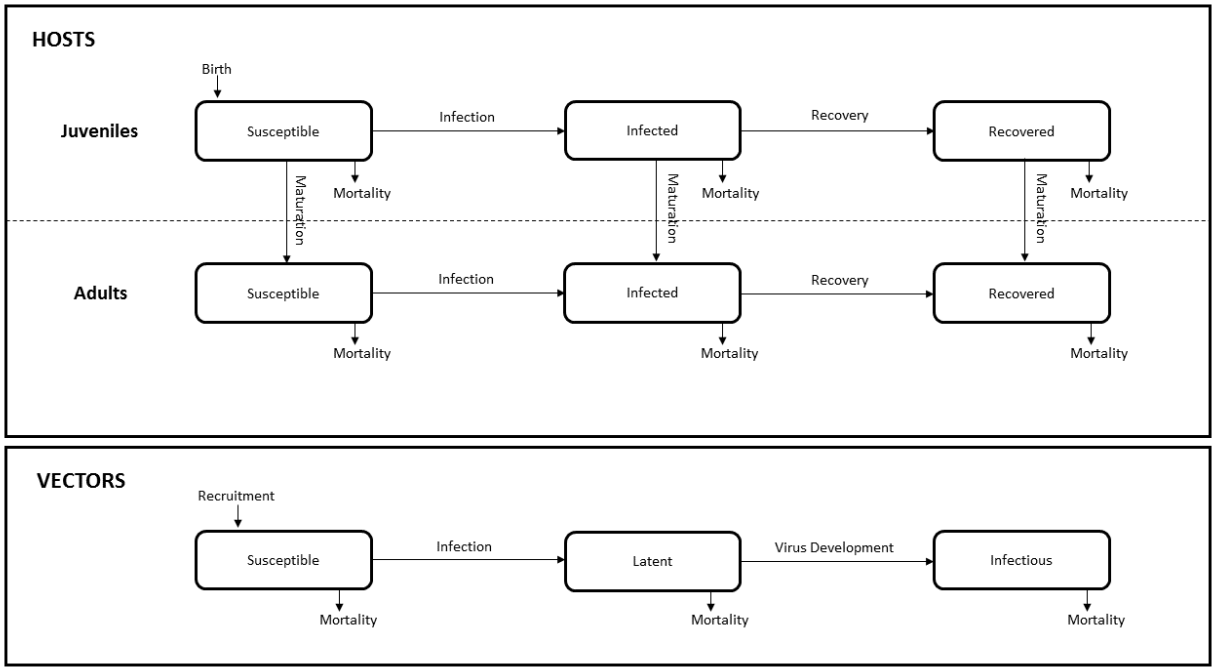

Figure 3.1: *West Nile virus* single host stage-structured model schematic. Shows all of the possible life cycles of the hosts as well as the vectors.

3.1 Single Species Model

We can reduce model 2.1 from a two host species model to a single host species. The single species model is defined as follows and all baseline parameter values can be found in Table 3.1:

$$\begin{aligned}
\frac{dJ_S}{dt} &= b(t) - a\alpha_J\beta_J M_I\frac{J_S}{J_T} - (m_J + \mu_J)J_S \\
\frac{dJ_I}{dt} &= a\alpha_J\beta_J M_I\frac{J_S}{J_T} - (m_J + \mu_J + \gamma_J + \nu_J)J_I \\
\frac{dJ_R}{dt} &= \gamma_J J_I - (m_J + \mu_J)J_R \\
\frac{dA_S}{dt} &= -a\alpha_A\beta_A M_I\frac{A_S}{A_T} + m_J J_S - \mu_A A_S \\
\frac{dA_I}{dt} &= a\alpha_A\beta_A M_I\frac{A_S}{A_T} + m_J J_I - (\mu_A + \gamma_A + \nu_A)A_I \\
\frac{dA_R}{dt} &= \gamma_A A_I - \mu_A A_R + m_J J_R \\
\frac{dM_S}{dt} &= rM_T(1 - \frac{M_T}{K}) - a(\alpha_J\delta_J\frac{J_I}{J_T} + \alpha_A\delta_A\frac{A_I}{A_T})M_S - \mu_M(t)M_S \\
\frac{dM_L}{dt} &= a(\alpha_J\delta_J\frac{J_I}{J_T} + \alpha_A\delta_A\frac{A_I}{A_T})M_S - kM_L - \mu_M(t)M_L \\
\frac{dM_I}{dt} &= kM_L - \mu_M(t)M_I
\end{aligned} \tag{3.1}$$

where

$$b(t) = \frac{f}{\sigma\sqrt{2\pi}}exp\left(\frac{-(q-t)^2}{2\sigma^2}\right)$$

is the juvenile recruitment curve for the hosts, with variance σ, mean q, and scaled by a factor of f,

$$\begin{aligned}
\alpha_J(t) &= \frac{\epsilon_J J_T(t)}{\epsilon_J J_T(t) + \epsilon_A A_T(t)} \\
\alpha_A(t) &= \frac{\epsilon_A A_T(t)}{\epsilon_J J_T(t) + \epsilon_A A_T(t)}
\end{aligned}$$

are the fraction of bites going to juvenile hosts and adult hosts respectively, and the vector mortality rate $\mu_M(t)$ is as in equation 2.4.

For a more in depth discussion on the parameters and how each of the equations were created, refer back to Chapter 2: Model Development.

Table 3.1: West Nile virus model parameters. All parameter values are from Robertson and Caillouet [20]

Parameter	Description	Baseline
a	Biting rate on competent avian hosts	0.133 bites/mosquito/day
ϵ	Host exposure coefficient	varies
ϵ_J	Juvenile exposure coefficient	varies
ϵ_A	Adult exposure coefficient	varies
m_J	Juvenile maturation rate	$1/14$ days^{-1}
μ_J	Juvenile natural mortality rate	0.0014 days^{-1}
μ_A	Adult natural mortality rate	0.0014 days^{-1}
β_J	Juvenile susceptibility	1
β_A	Adult susceptibility	1
γ_J	Juvenile recovery rate	$1/3$ days^{-1}
γ_A	Adult recovery rate	$1/3$ days^{-1}
δ_J	Juvenile infectivity	0.36
δ_A	Adult 1 infectivity	0.36
ν_J	Juvenile virulence	0.1 days^{-1}
ν_A	Adult virulence	0.1 days^{-1}
q	Juvenile recruitment curve mean	Day 158.17
σ	JRC standard deviation	11.4
f	JRC scaling factor	285
A_0	Number of adults at start of season	150
r	Mosquito per capita birthrate	0.537 days^{-1}
K	Mosquito carrying capacity (larval)	15000
μ_M	Vector mortality rate	0.096 days^{-1}
k	Virus incubation period	0.106 days^{-1}
z	Start of Mosquito growth	Day 115

3.2 Results

By reducing to a single species model, we have effectively taken away all possibility of any type of host species comparisons. In the single species model, we are left solely with looking at the effects of different parameter values for the two stages, juveniles and

adults.

Now that we have a single species model, we have to decide what would be the best way to directly translate the two species attributes to a single species. For example, how might we convey differential host species competence in a model that only has one host? In order to do this, we will use the same simplifying assumption that is generally used; average parameter values. Since one of the characteristics we incorporate in the two species model is host species abundance in the host community, we incorporate this into the process and use these values to get weighted averages.

This chapter is broken into two main sections: (1) equal host competence, and (2) differential host competence. For both of these situations, in the two host species model we will examine the effects of host species 1 being more abundant than host species 2, equal host species abundance, and host species 2 being more abundant than host species 1, and then incorporate 4 scenarios of differential biting rates on juvenile hosts for each. For each of these scenarios, we will compare the transmission dynamics from the two species model with those from a single species model with weighted average parameter values.

3.2.1 Equal Host Competence

When considering our two species model with equal host competence (i.e., $\gamma_1 = \gamma_2$ and $\delta_1 = \delta_2$) there are three possibilities that may occur when we allow abundance to differ: (1) both species are equally abundant in the host community, (2) species 1 is more abundant than species 2, or (3) species 2 is more abundant than species 1. For each of these cases we will consider four scenarios of juvenile stage preference: (1) no stage preference, (2) preference for juveniles of host species 1, (3) preference for juveniles of host species 2, and (4) an overall juvenile preference. For each, we will compare the dynamics of the two species model to the dynamics of the single species model, and explore if it is possible for the single species model to capture the key WNV transmission

dynamics.

Equal Host Abundance (50/50)

For the first part of our analysis, we compare a few possible scenarios that can occur with each species being of equal competence and equal abundance in the two species model with a single species model that has average parameter values. To show equal abundance in the two species model we let the host species 1 population make up 50% of the total host community and the host species 2 population make up the remaining 50% of the community. We then give each host species equal and increased competence by shortening both species' recovery rates (which increases the duration of infection) to $\gamma_1 = \gamma_2 = \frac{1}{6}$, and increasing both species' infectivity to $\delta_1 = \delta_2 = 0.72$. To translate this into our single species model, we calculate a new recovery rate and infectivity for the single host species by using weighted averages. Since the species are equally abundant and equally competent, we get competence parameters for the single species model of $\gamma = \frac{1}{6}$, and $\delta = 0.72$.

No Stage Preference

Using these competence and abundance values, we first look at the base case of no preference (Figure 3.2). Our analysis shows that when using these average parameters the infectious vector (IV) curve from the single species model is identical to the infectious vector (IV) curve of the two species model. This is not surprising, because all competence and abundance values are equal in the two species model, which is the same as only having a single species. This is a situation where we would be able to use a single species model in place of a two species model.

Differential Stage Preference

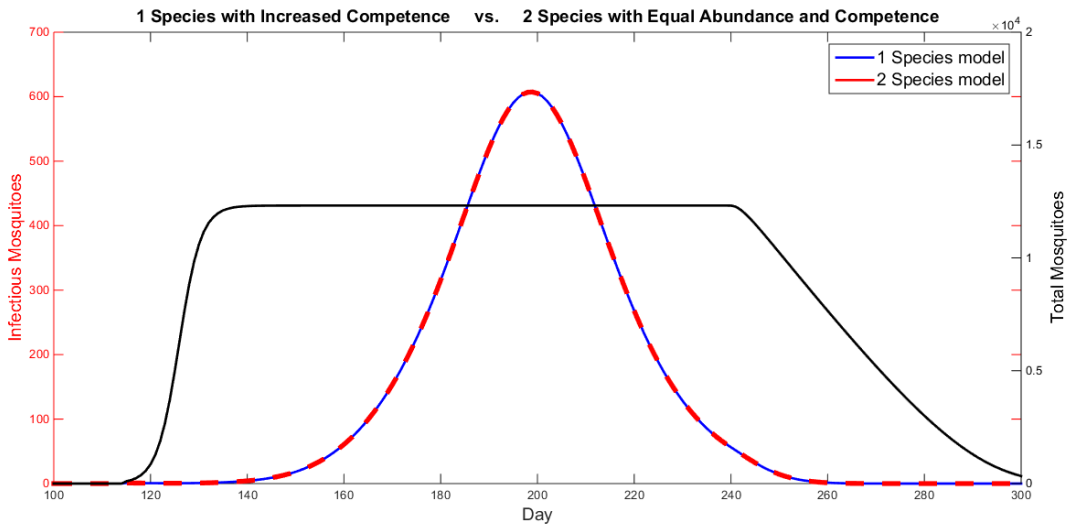

Figure 3.2: With equal competence, equal abundance, and no stage preferences the single host species model with weighted average parameter values gives the same dynamics as the two host species model. In the two species model, competence values are $\gamma_1 = \gamma_2 = \frac{1}{6}, \delta_1 = \delta_2 = 0.72$, with the fraction of species 1 = 0.5, and the fraction of species 2 = 0.5. In the single species model, parameter values are given by $\gamma = \frac{1}{6}, \delta = 0.72$.

Next we consider the same situation of equal host competence and equal host abundance, but begin to incorporate vector stage preferences. The first preferences we incorporate are a strong preference for juveniles of species 1 ($\epsilon_J = 10$), or a strong preference for juveniles of species 2 ($\epsilon_{\hat{J}} = 10$) (Figure 3.3 and Table 3.2). Since species 1 and species 2 are identical, both of these preferences will yield equivalent results. Analysis shows that the weighted average juvenile preference used in the single species model of ϵ_J (or $\epsilon_{\hat{J}}$) $= 5.5$ is an overestimate of the IV curve given by the two species model. The preference value in the single species model that best fits the infectious vector curve given by the two species model is $\epsilon_J = 4.72$. However, while the peak IV is close to that of the two species model the curve is not an exact match.

Equal Stage Preferences

The final situation that we consider for equal competence and equal abundance is an overall juvenile preference (i.e., there is a preference for juveniles of host species 1 and

juveniles of host species 2). To convey this in the two species model we set $\epsilon_J = \epsilon_{\hat{J}} = 10$. Analysis shows that the average preference value of $\epsilon_J = 10$ in the single species model yields an exact match in vector outputs (total IV, peak IV, and day of peak IV) as the two species model with an overall juvenile preference. Similar to the no preference case, in the two species model both host species have equal competence and equal abundance. Giving both of these species the same increased preference is the same as only having a single species. This is another situation where the single species model can be used to model a multiple species situation.

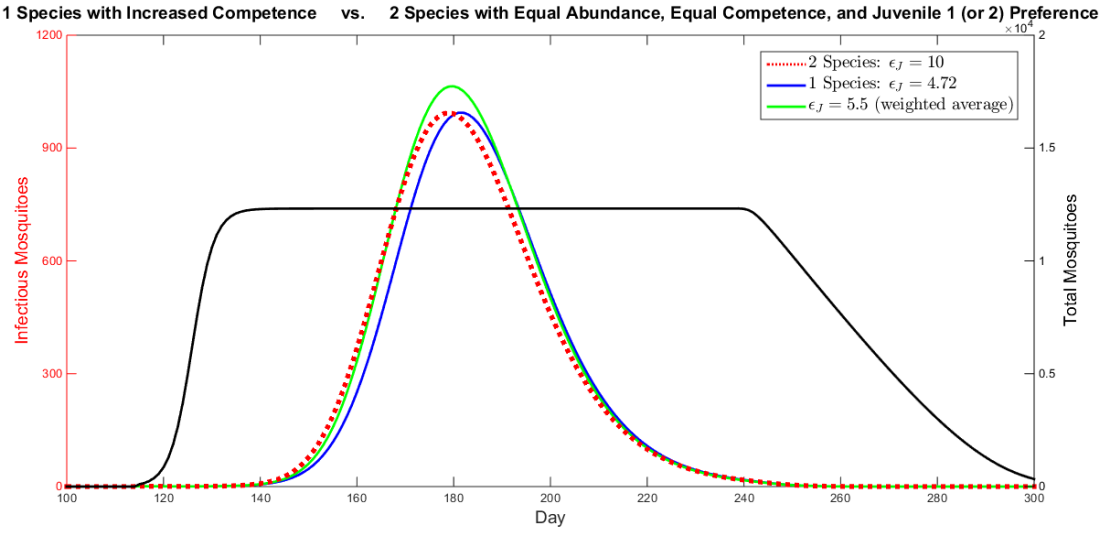

Figure 3.3: With equal competence, equal abundance, and a strong juvenile preference for either species 1 or species 2 (ϵ_J or $\epsilon_{\hat{J}} = 10$), the single host species with weighted average parameter values is an overestimate of the two species model. The preference value that best fits the two species model is $\epsilon_J = 4.72$. In both models, parameter values are as in Figure 3.2.

Table 3.2: Vector totals for Figure 3.3. The weighted average of $\epsilon_J = 5.5$ in the single species model is an overestimate of the two species model.

Model	Peak IV	Day of Peak IV	Total IV
2 Species:$\epsilon_J = 10$ or $\epsilon_{\hat{J}} = 10$	993.6	179	38,905
1 Species:$\epsilon_J = 5.5$ (Weighted Avg.)	1064.2	180	40,606
1 Species:$\epsilon_J = 4.72$	993.9	182	37,724

Differential Host Abundance (75/25 and 25/75)

We now consider the case where two host species with equal competence are present at different abundances in the host community and compare the results to those of a single species model with weighted average parameter values. In the two species model, we let the host species 1 population make up 75% of the host community, while the host species 2 population makes up the remaining 25%. Taking the weighted averages of the two species model parameters to find the competence for the single species model we get $\gamma = \frac{1}{6}$ and $\delta = 0.72$.

No Stage Preference

Using these competence and abundance values we first consider the base case of no species or stage biting preference. Analysis shows that when using these average parameters, the IV curve from the single species model is identical to the IV curve from the two species model (Figure 3.4). Even though there are different host species abundances, since the two species have all of the same parameter values it is as though there is only a single species present, which is why the single species model can accurately model this two species situation.

Differential Stage Preferences

Next we will consider the effects of incorporating a strong juvenile preference for species 1 or species 2, but not both. For the single species model,

$$\epsilon_J = 0.75(10) + 0.25(1) = 7.75$$

$$\epsilon_J = 0.75(1) + 0.25(10) = 3.25$$

are the weighted averages of the preference values for the cases where $\epsilon_J = 10$, and $\epsilon_{\hat{J}} = 10$ respectively. In both cases these weighted averages are overestimates of both

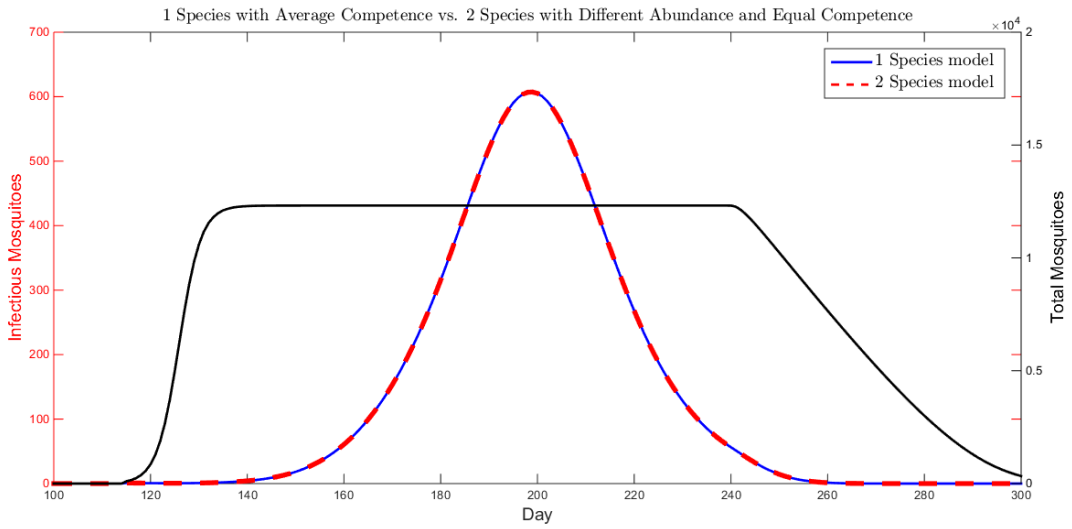

Figure 3.4: With equal competence, different abundances, and no stage preferences, the single host species model with weighted average parameter values gives the same dynamics as the two host species model. In both models competence values as in Figure 3.2. For the two species model the fraction of species 1 = 0.75 and the fraction of species 2 = 0.25.

the peak IV as well as the total IV given by the two species model (Figure 3.5 and Table 3.3). For the case where $\epsilon_J = 10$, the preference value for the single species model that best fits the two species IV curve is given by $\epsilon_J = 6.95$. In the case where $\epsilon_{\hat{J}} = 10$, the preference value for the single species model that best fits the two species IV curve is given by $\epsilon_J = 2.88$. While the best fit preferences are extremely close in peak IV given by the two species model, they both severely underestimate the total IV.

Table 3.3: Vector totals for Figure 3.5. The weighted averages of $\epsilon_J = 7.75$ and $\epsilon_J = 3.25$ are overestimates of the two species model.

Model	**Peak IV**	**Day of Peak IV**	**Total IV**
2 Species:$\epsilon_J = 10$	1,174.4	175	46,829
1 Species:$\epsilon_J = 7.75$ (Weighted Avg.)	1,224.9	176	48,305
1 Species:$\epsilon_J = 6.95$	1,174.1	177	45,697
2 Species:$\epsilon_{\hat{J}} = 10$	795	185	31,672
1 Species:$\epsilon_J = 3.25$ (Weighted Avg.)	838.4	187	32,284
1 Species:$\epsilon_J = 2.88$	795.2	188	30,979

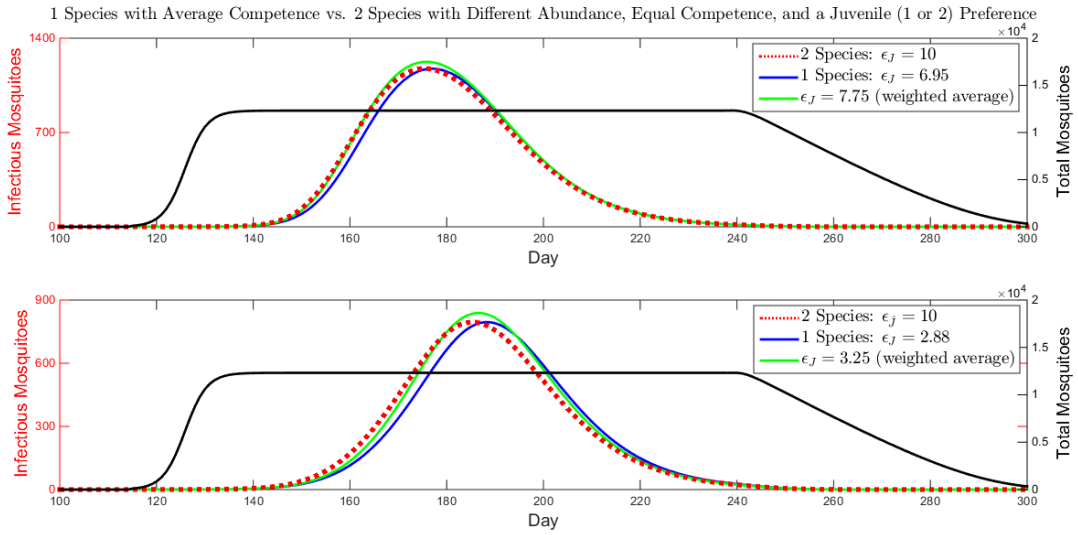

Figure 3.5: With equal competence, different abundance, and a strong juvenile preference for either species 1 or species 2 ($\epsilon_J = 10$ or $\epsilon_{\hat{J}} = 10$), the single host species with weighted average parameter values is an overestimate of the two species model. The preference values that best fit the two species model are $\epsilon_J = 6.95$ and $\epsilon_J = 2.88$ respectively. In both models, parameter values are as in Figure 3.4.

Equal Stage Preferences

The final situation that we consider for equal competence and different host abundance is a preference for both juveniles of host species 1 and juveniles of host species 2. To convey this in the two species model, we set $\epsilon_J = \epsilon_{\hat{J}} = 10$. Similar to the other cases when we have equal host competence, if there is an overall preference for one stage then the two species model becomes a single species model regardless of community composition (i.e., all parameter values for both species are equal except abundance), in which case the single species model can model these situations exactly.

Switched Abundance

The other possible scenario that we could consider would be switching the two species abundances. In which case the host species 1 population would make up 25% of the total host community and the host species 2 population would make up the other

75% of the community. Since we are currently only considering equal competence we need not consider this situation (the results would be the same as our previous analysis where host species 1 makes up 75% and host species 2 makes up 25% of the total population).

3.2.2 Differential Host Competence

Now that we have examined the case of equal host competence we can incorporate differential host competence in our two species model and see how the results compare to those of a single species model with weighted average parameter values. Considering the same scenarios as with equal host competence, we again examine each scenario under varying assumptions for juvenile preferences. For this section, we will leave species 1 competence at its baseline recovery rate ($\gamma_1 = \frac{1}{3}$) and infectivity ($\delta_1 = 0.36$), and we will increase the competence of host species 2 by shortening its recovery rate to $\gamma_2 = \frac{1}{6}$, and increasing its infectivity to $\delta_2 = 0.72$.

Equal Host Abundance (50/50)

In this section we consider differential competence and equal abundance (each host species comprises 50% of the total host community). Using the competence values described above, we calculate a new recovery rate and infectivity by using weighted averages. Therefore, for our single species model,

$$\gamma = 0.5(\frac{1}{3}) + 0.5(\frac{1}{6}) = \frac{1}{4},$$
$$\delta = 0.5(0.36) + 0.5(0.72) = 0.54.$$

No Stage Preference

Using these competence values in the single species model we first consider the base case of no preference. Our analysis shows that while the day of the peak IV in each of the curves and the overall shapes of the IV curves are similar, the single species model with weighted average competence values and no preference is a severe underestimate of the two species model with different host species competence, equal abundance, and no preference (Figure 3.6). Recall that previously when we had equal competence (regardless of abundance) these curves were exactly the same. Therefore, anytime host species differ in competence the single species model is not an adequate representation of the WNV transmission dynamics that occur in the two species model.

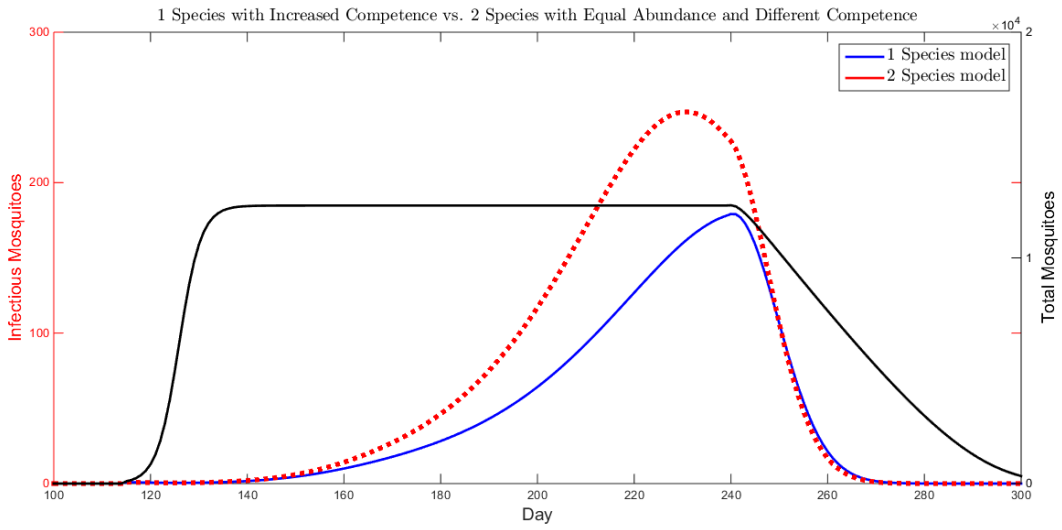

Figure 3.6: With equal host abundance, no stage preferences, and differential host competence, the single host species model with weighted average parameter values is an underestimate of peak IV and total IV in the two species model. In the two species model parameters are $\gamma_1 = \frac{1}{3}, \gamma_2 = \frac{1}{6}, \delta_1 = \frac{1}{3}, \delta_2 = \frac{1}{4}$, with fraction of host species 1 = 0.5 and fraction of host species 2 = 0.5. In the single species model competence parameters are a weighted average and given by $\gamma = \frac{1}{4}, \delta = 0.54$.

Differential Stage Preferences

Next we consider the effect of incorporating differential biting rates on juveniles. In the two species model we will either increase the juvenile preference for the less competent host species, species 1 ($\epsilon_J = 10$), or we will increase the juvenile preference for the more competent host species, species 2 ($\epsilon_{\hat{J}} = 10$).

Simulations show that when the less competent species has an increased juvenile preference in the two species model, the weighted average of $\epsilon_J = 5.5$ in the single species model is a overestimate of the intensity of disease transmission. The preference value which gives the closest (but not exact) fit in the single species model is $\epsilon_J = 4.46$. On the other hand, when the more competent species has an increased juvenile preference, the weighted average of $\epsilon_J = 5.5$ in the single species model is an underestimate of the intensity of disease transmission. The switch from an overestimate to an underestimate is because we have switched the preference from the less competent species to the more competent species. With equal host abundance this switch always results in increased WNV transmission. The preference value that gives the closest (but not exact) fit in the single species model is $\epsilon_J = 9.27$. In both cases, the single species model with weighted parameters is not an adequate substitution for the two species model.

The final scenario we consider with equal abundance and differential competence is an increased biting rate on the juveniles of both host species. To show this in the two species model we set $\epsilon_J = \epsilon_{\hat{J}} = 10$. Using average competence parameters in the single species model, the weighted average of $\epsilon_J = 10$ is an underestimate of the intensity of WNV transmission resulting from the two species model (Figure 3.7 and Table 3.4). The preference value that best fits the two species output is given by $\epsilon_J = 13.15$ and even that is not an exact match.

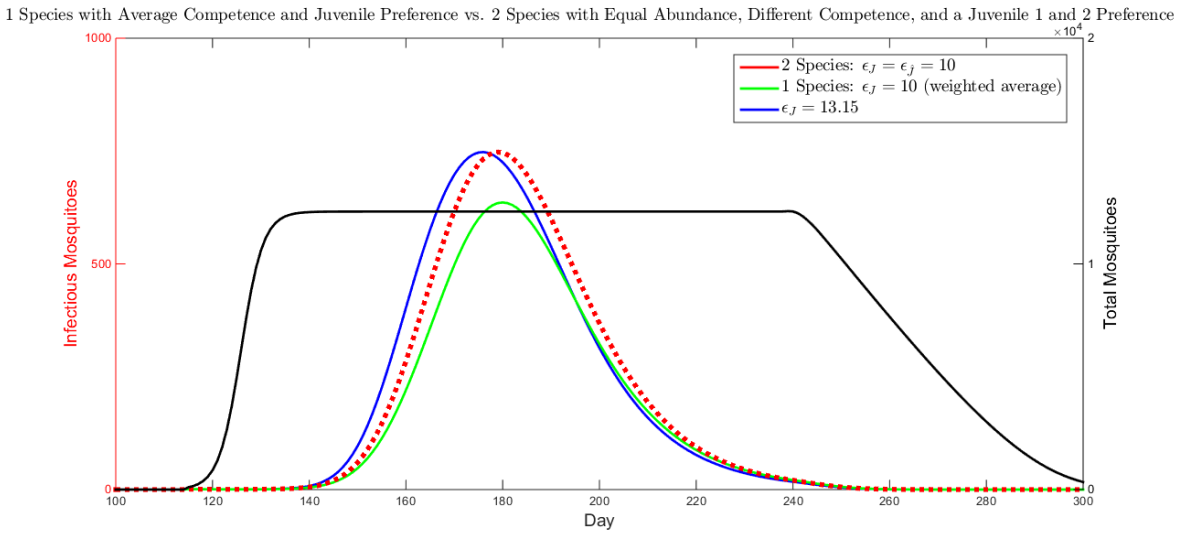

Figure 3.7: With equal host abundance, different host competence and an overall juvenile preference, the single host species model with weighted average parameter values is an underestimate of the peak IV and total IV from the two species model. In both models, parameter values are as in Figure 3.6

Table 3.4: Vector totals for Figure 3.7. The weighted average of $\epsilon_J = 10$ in the single species model is an underestimate of the two species model.

Model	Peak IV	Day of Peak IV	Total IV
2 Species:$\epsilon_J = \epsilon_{\hat{J}} = 10$	747.6	179	30,421
1 Species:$\epsilon_J = 10$ (Weighted Avg.)	636.1	180	25,839
1 Species:$\epsilon_J = 13.15$	747.7	176	30,789

Differential Host Abundance (75/25 and 25/75)

Now that we have discussed and compared the transmission dynamics from a single species model with weighted average parameter values to those of a two species model with equal host abundance and differential competence, we can consider the effect of differential host abundance. When incorporating differential host abundance and differential host competence there are two cases that can occur: (1) the more abundant species is less competent, or (2) the more abundant species is more competent. With each case, we will also incorporate and examine various scenarios of juvenile stage preferences.

We first consider the case when the more abundant species is less competent. In the two species model we let the host species 1 population make up 75% of the total host

community, and the host species 2 population make up the remaining 25% of the host community. The competence parameters are the same as before with host species 1 at the baseline of $\gamma_1 = \frac{1}{3}$ and $\delta_1 = 0.36$, and host species 2 at an increased competence of $\gamma_2 = \frac{1}{6}$ and $\delta_2 = 0.72$. Of our two host species, we now have one who is more abundant but less competent (1) and another who is more competent but less abundant (2). To translate this into our single species model we calculate a new recovery rate and infectivity by using weighted averages. For the single species model

$$\gamma = 0.75(\frac{1}{3}) + 0.25(\frac{1}{6}) = \frac{7}{24}, \text{ and}$$
$$\delta = 0.75(0.36) + 0.25(0.72) = 0.45.$$

No Stage Preference

Following our previous pattern, using the competence and abundance values described above we first look at the base case of no preference. We found that while similar in shape and day of peak occurrence, the infectious mosquitoes peak in the two host species model is severely underestimated by the infectious mosquito peak in the singe host species model.

Differential Stage Preferences

Next, we consider the effect of incorporating heterogeneous biting rates on juvenile hosts. In the two species model we consider the two scenarios of host species 1 having a strong juvenile preference, or host species 2 having a strong juvenile preference. To convey these two situations of heterogeneous biting rates in our two species model we set either $\epsilon_J = 10$ or $\epsilon_{\hat{J}} = 10$.

When the juveniles of the more abundant but less competent species (species 1) are preferred in the two species model, the weighted average preference of $\epsilon_J = 7.75$ in

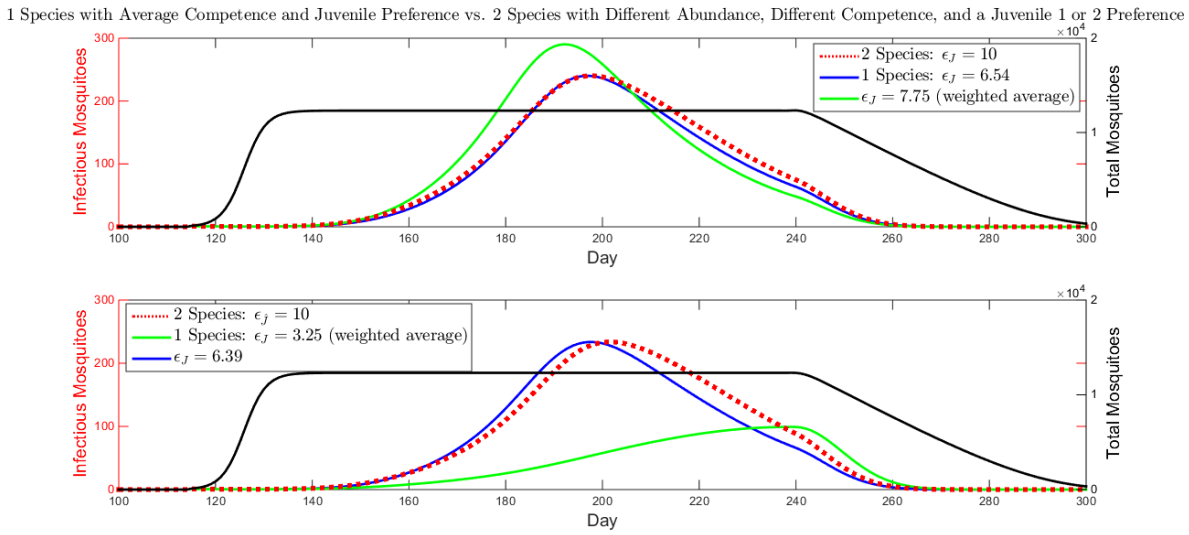

Figure 3.8: With different species competence, different species abundance, and a strong juvenile preference for either species 1 (top) or species 2 (bottom) ($\epsilon_J = 10$ or $\epsilon_{\hat{J}} = 10$), the single host species with weighted average competence parameter values is either an overestimate (top graph) or an underestimate (bottom graph) of the two species model. The preference values that best fit the two species model are $\epsilon_J = 6.54$ (top) and $\epsilon_J = 6.39$ (bottom). In the two species model, parameter values are given by $\gamma_1 = \frac{1}{3}, \gamma_2 = \frac{1}{6}, \delta_1 = 0.36, \delta_2 = 0.72$, with species 1 fraction of the host community = 0.75, species 2 fraction of the host community = 0.25. The single species competence parameters are $\gamma = \frac{7}{24}, \delta = 0.45$.

Table 3.5: Vector totals for Figure 3.8. For increased biting rates on juveniles of species 1, the weighted average of $\epsilon_J = 7.75$ in the single species model is an overestimate of the two species model. For increased biting rates of juveniles of species 2, the weighted average of $\epsilon_J = 3.25$ is an underestimate of the two species model.

Model	**Peak IV**	**Day of Peak IV**	**Total IV**
2 Species:$\epsilon_J = 10$	240.2	198	13,044
1 Species:$\epsilon_J = 7.75$ (Weighted Avg.)	280.3	193	13,356
1 Species:$\epsilon_J = 6.54$	240.2	197	12,222
2 Species:$\epsilon_{\hat{J}} = 10$	233.6	202	12,785
1 Species:$\epsilon_J = 3.25$ (Weighted Avg.)	99.2	239	5,816.3
1 Species:$\epsilon_J = 6.39$	233.6	198	12,030

the single species model overestimates the intensity of WNV transmission (Figure 3.8 and Table 3.5). However, when the juveniles of the less abundant but more competent species (species 2) have a juvenile preference in the two species model, the weighted average preference of $\epsilon_J = 3.25$ in the single species model underestimates the intensity

of WNV transmission (Figure 3.8 and Table 3.5).

Equal Stage Preferences

The final case that we consider is the effect of having an overall juvenile preference. To do this in the two species model we set $\epsilon_J = \epsilon_{\hat{J}} = 10$, and in the single species model give the hosts weighted average competence parameters. In this case, the average preference is $\epsilon_J = 10$, which is an underestimate of the intensity of WNV transmission, while the best fit preference value to the two species model is given by $\epsilon_J = 12.5$ which still underestimates the total number of IV (See Figure 3.9 and Table 3.9).

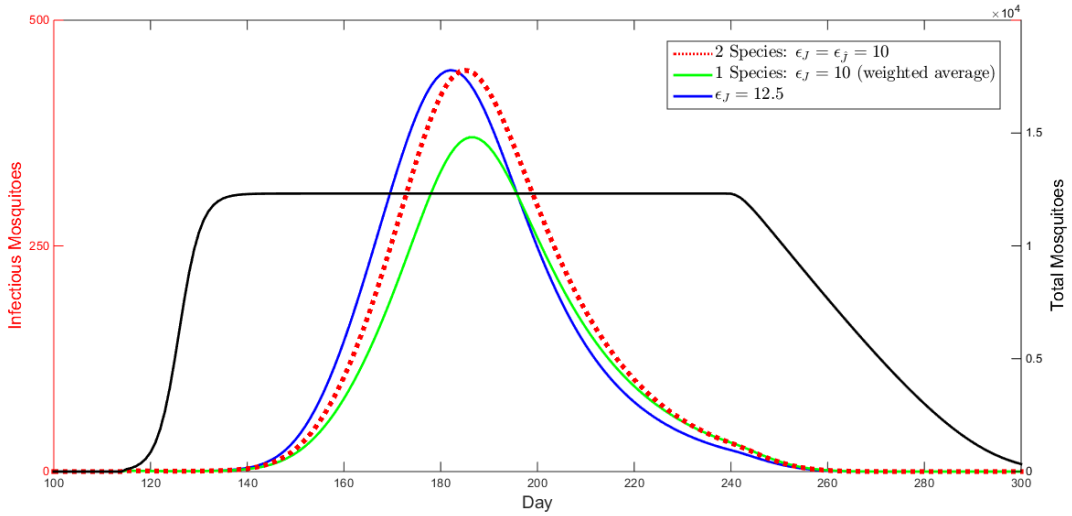

Figure 3.9: With differential competence and abundance and a strong overall juvenile preference ($\epsilon_J = \epsilon_{\hat{J}} = 10$), the single host species with weighted average competence parameters is an underestimate of the two species model. The preference value that best fits the two species model is $\epsilon_J = 12.5$. For both models competence and abundance parameter values are as in Figure 3.8.

Table 3.6: Vector totals for Figure 3.9. The weighted average of $\epsilon_J = 10$ in the single species model is an underestimate of the two species model.

Model	Peak IV	Day of Peak IV	Total IV
2 Species:$\epsilon_{\hat{J}} = \epsilon_J = 10$	444.5	185	19,014
1 Species:$\epsilon_J = 10$ (Weighted Avg.)	370.3	186	16,053
1 Species:$\epsilon_J = 12.5$	444.7	182	18,717

Switched Abundance

In our analysis we also considered a final scenario of switching the two species abundance. In this case, competence would remain the same and the host species 1 population would make up 25% of the total host community, while host species 2 would make up 75% of the total host community. This gives us the situation where the more competent species is more abundant, and the less competent species is less abundant. Calculating the weighted averages for the single species model,

$$\gamma = 0.25(\frac{1}{3}) + 0.75(\frac{1}{6}) = \frac{5}{24}, \text{ and}$$
$$\delta = 0.25(0.36) + 0.75(0.72) = 0.63.$$

Using these values and examining the base case of no preference, we find that while similar in shape the single species model with average competence values is an overall underestimate of disease transmission from the two species model. We also found that when looking at a juvenile preference for the less abundant, less competent host species ($\epsilon_J = 10$), the weighted average of $\epsilon_J = 3.25$ is an overestimate of WNV transmission with the best fit being $\epsilon_J = 2.84$. When looking at a juvenile preference for the more abundant more competent host species ($\epsilon_{\hat{J}} = 10$), the weighted average of $\epsilon_J = 7.75$ is an underestimate of WNV transmission, with the best fit being $\epsilon_J = 10.55$. In the final case of an overall juvenile preference ($\epsilon_J = \epsilon_{\hat{J}} = 10$), the weighted average of $\epsilon_J = 10$ in the single species model was an underestimate of WNV transmission, with the best fit given by a juvenile preference of $\epsilon_J = 12.25$.

3.3 Discussion

Comparing the WNV transmission dynamics from the two species model with the transmission dynamics from the single species model with weighted average parameter values, we see some distinct patterns beginning to form. In some situations, the single species model with weighted average parameter values is an accurate substitution for the two species model, however in others it is not.

When we have equal competence, in all situations where the two species model has equal parameter values for host species 1 and host species 2 (i.e., no host stage preferences with equal host competence), the single species model is able to capture the dynamics perfectly regardless of abundance. If host species 1 and host species 2 have all equal parameters then there are no differences between the two species, making it the same as only having a single species in the two species model. In these situations, the single species model can replace the two species model.

When there is equal competence but host species 1 and host species 2 do not have equal parameter values (i.e., juvenile preference for either host species 1 or host species 2), then the single species model with weighted average parameter values overestimates disease transmission. Even though all competence and abundance values are equal in the two species model, having a juvenile preference for one host or the other means that the juveniles of one of the host species are getting more bites while the other equally competent species is being bitten in proportion to its abundance, which reduces the intensity of WNV transmission compared to the case where all juveniles receive bites at increased rates and causes the weighted average values in the single species model to overestimate the intensity of transmission. In these situations, the single species model is not an adequate representation of the two species model.

When we do not have equal competence we see a different pattern. With either no stage preference or an overall juvenile preference, regardless of host abundance, the single species model with weighted averages will always underestimate the intensity of

WNV transmission of the two species model. In these situations, the weighted average competence parameters do not accurately represent the effect of having one species of higher competence and one species of lower competence. The more competent species is infecting mosquitoes at a very high rate, while the low competent species are still getting bitten and infecting mosquitoes. Combined, this results in an increased intensity of WNV transmission. In these situations, the single species model is not an adequate replacement for the two species model.

When we have differential competence and an increased biting rate on the juveniles of the more competent species, the single species model will always underestimate the intensity of WNV transmission from the two species model. Average parameter values cannot account for this because in the two species model, the juveniles of the more competent species are so much more preferred that they are receiving almost all of the bites which results in very high disease transmission, while the non-preferred species is contributing little to none to the IV. The single species model cannot accurately account for this bite distribution and therefore results in an underestimate of disease transmission intensity.

On the other hand, when we have an increased biting rate on the juveniles of the on the less competent species the single species model will always overestimate the intensity of transmission from two species model, regardless of abundance. Almost all of the bites are now going to the juveniles of the less competent species with a small portion going to juveniles of the more competence species, which results in decreased disease transmission. The more competent species is receiving nearly none of the bites and therefore not resulting in a large number of IV. Again, the average competence parameters cannot account for this distribution of bites and therefore will always result in an overestimate of disease transmission intensity.

For each case where the single species model with weighted average parameter values can not accurately describe the transmission dynamics that occur in the two species

model, we can adjust the preference values to come close to the the two species model results but we can never match the two species results exactly. While there must exist some combination of parameters to match single species model to the two species model exactly, it would not be a direct translation of what is occurring in the two species model, and would be impossible to do so without already having the two species model to compare it to which would therefore completely defeat the purpose.

Table 3.7: A summary of all comparisons of the single species model with weighted parameters against the two species model. The abundance ratio gives the percent of each species in the total host community (species 1/species 2), the preference tells us the type of juvenile preference from the two species model, and the outcome of the single species model tells us how the single species IV curve fits with the two species IV curve.

Equal Competence			Differential Competence		
Abundance Ratio	Preference	Outcome of Single Species Model	Abundance Ratio	Preference	Outcome of Single Species Model
50/50	None	Equal	50/50	None	Underestimate
50/50	$\epsilon_J = 10$	Overestimate	50/50	$\epsilon_J = 10$	Overestimate
50/50	$\epsilon_{\hat{J}} = 10$	Overestimate	50/50	$\epsilon_{\hat{J}} = 10$	Underestimate
50/50	$\epsilon_J = \epsilon_{\hat{J}} = 10$	Equal	50/50	$\epsilon_J = \epsilon_{\hat{J}} = 10$	Underestimate
75/25	None	Equal	75/25	None	Underestimate
75/25	$\epsilon_J = 10$	Overestimate	75/25	$\epsilon_J = 10$	Overestimate
75/25	$\epsilon_{\hat{J}} = 10$	Overestimate	75/25	$\epsilon_{\hat{J}} = 10$	Underestimate
75/25	$\epsilon_J = \epsilon_{\hat{J}} = 10$	Equal	75/25	$\epsilon_J = \epsilon_{\hat{J}} = 10$	Underestimate
25/75	None	Equal	25/75	None	Underestimate
25/75	$\epsilon_J = 10$	Overestimate	25/75	$\epsilon_J = 10$	Overestimate
25/75	$\epsilon_{\hat{J}} = 10$	Overestimate	25/75	$\epsilon_{\hat{J}} = 10$	Underestimate
25/75	$\epsilon_J = \epsilon_{\hat{J}} = 10$	Equal	25/75	$\epsilon_J = \epsilon_{\hat{J}} = 10$	Underestimate

Therefore, we can confidently conclude that while under certain scenarios a single species model can replicate the outcome of a two host species and vector interaction, in general, if the two host species have any differences other than abundance, we cannot use a single species model to accurately depict the two host species interactions with the vector population and must use a two species model. For a summary of all the results from this section, refer to table 3.7.

Chapter 4

A WNV Transmission Model with Two Host Species and No Vector Biting Preferences

Now that we have established why we created a two species stage-structured model, and that a two species model can model scenarios that a one species model cannot account for, we can begin to look more closely at the two species model.

Avian species exhibit a great deal of variability in transmission related parameters such as infectivity and duration of viremia, as well as nesting parameters such as the mean and variance of the juvenile recruitment curve. Within a species, juveniles and adults may also differ in parameters related to competence [5]. During the analysis of this section, we only vary one parameter at a time while holding all others constant in order to observe that parameter's direct effect. Furthermore, we are only looking at situations where there are no species or stage feeding preferences present, and as such all preference parameter values are kept at the baseline of $\epsilon = \hat{\epsilon} = 1$, $\epsilon_J = \epsilon_A = 1$, and $\epsilon_{\hat{J}} = \epsilon_{\hat{A}} = 1$.

4.1 Results

4.1.1 Effect of Recovery Rate on WNV Transmission

The recovery rate (γ) is estimated by 1/(number of days infected), and tells us the fraction of the infected population that recovers every day. Therefore, as the number of days spent infected (duration of viremia) increases, the recovery rate decreases.

Looking at the effect of recovery rate on model 2.1 with no vector feeding preferences, there are two possible scenarios we want to examine: (1) varying recovery rates between host species 1 and host species 2, and (2) varying recovery rates between stages (juveniles and adults).

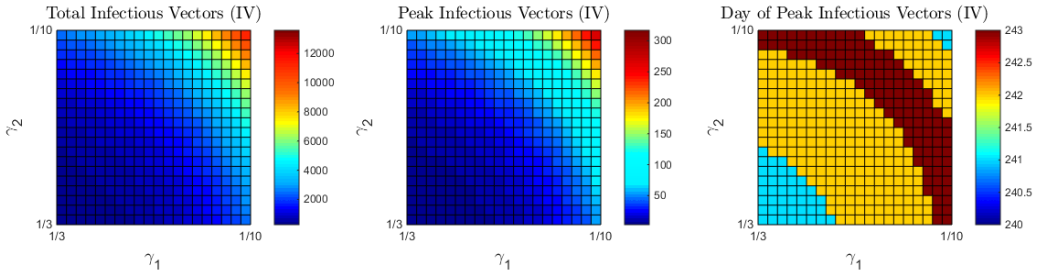

Figure 4.1: Host recovery rate (γ) is symmetric about the diagonal. As the recovery rate decreases (the duration of infection increases) for both host species the total IV and peak IV increase. Therefore, maximum WNV transmission occurs when both host species recovery rates are at their minimum of $\gamma = \frac{1}{10}$.

To explore the effect of differential recovery rate among species, we allow each species' recovery rate to vary from the baseline value of $\frac{1}{3}$ (3 days infected) to $\frac{1}{10}$ (10 days infected). Figure 4.1 shows the model outputs of total IV, peak IV, and day of peak IV for each pair of species' recovery rates. Figure 4.1 shows that host recovery rate is symmetric about the diagonal. Also, if one species is at the maximum recovery rate of $\gamma = \frac{1}{3}$, then the recovery rate of the other species has little effect on WNV transmission. Furthermore, as the recovery rate decreases in both species the number of total IV and the peak IV increases monotonically. The longer a host spends infectious, the longer it

is able to spread WNV to mosquitoes, thus increasing disease transmission. The final thing that we notice from Figure 4.1 is that recovery rate has little effect on the timing of WNV transmission. Increasing or decreasing γ will not move the timing of the peak IV drastically in one direction or the other.

Now that we have seen the effect of varying recovery rates between host species, we explore the effect of varying recovery rates between host stages. Our analysis shows that the effect of decreasing the juvenile recovery rate from $\gamma = \frac{1}{3}$ to $\gamma = \frac{1}{10}$ while the adult recovery rate is at the base of $\gamma = \frac{1}{3}$ does not have a large impact on the timing and intensity of disease transmission (Figure 4.2). Decreases in the adult recovery rate have a much larger impact on the total and peak IV than decreasing the juvenile recovery rate. Overall, the intensity and timing of WNV transmission seems to only depend on the adult recovery rate, and changes very little depending on the juvenile recovery rate. This is not surprising since hosts only remain in the juvenile stage for 14 days before transitioning into the adult stage. Since juveniles make up such a small, fleeting portion of the population, changing their recovery rate does not have a great effect on overall disease transmission. Also, similar to the case of differential host species recovery rates, differential host stage recovery rates do not have a large impact on the timing of disease transmission.

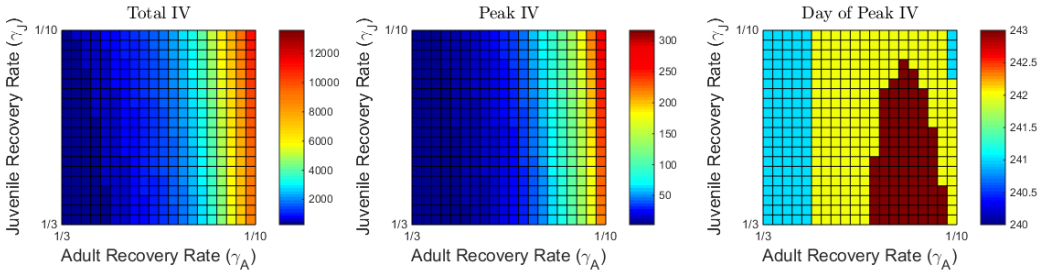

Figure 4.2: When the juvenile and adult recovery rates of both species are varied, the adult recovery rate has a much larger impact on the intensity of WNV transmission than the juvenile recovery rate.

4.1.2 Effect of Infectivity on WNV Transmission

Infectivity is the probability of bird-mosquito WNV transmission per bite [15]. For example, $\delta_J = 0.36$ tells us that if a susceptible mosquito bites an infected juvenile host of species 1, then there is a 36% chance that host-vector interaction will result in infection for the mosquito. When looking at the effect of infectivity on WNV transmission, we want to examine both the effect of differential host species infectivity rates as well as the effect of differential host stage recovery rates.

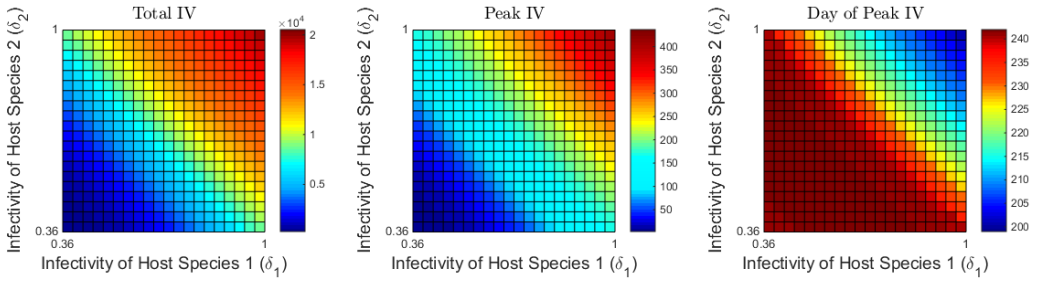

Figure 4.3: Infectivity is symmetric between host species 1 and host species 2. As infectivity increases, either in both species or only 1, the intensity of disease transmission increases. Also, as both species infectivity increases, the timing of increased disease transmission moves earlier in the season.

Figure 4.3 shows that differential host species infectivity is symmetric about the diagonal, which is unsurprising. If you set all parameter values of the two species equal and vary only one, then we will always see this symmetry. We also see that as the probability of WNV transmission from bird-mosquito increases, overall WNV transmission increases. Unlike differential recovery rates, here we also see that host infectivity has a direct effect on the timing of the peak IV. As infectivity increases in both species, we see the peak IV moving earlier in the season. For this to occur, both species must be highly competent, not only one or the other. This movement in the timing of peak disease transmission occurs because an increase in infectivity results in a higher probability of vector infection which means that it takes fewer bites on infected hosts for a vector to contract WNV. If it takes less interactions to result in infection then the vectors are

becoming infected quicker, which means that they are becoming infected earlier in the season.

We now look at the effect of incorporating different host stage infectivity on our model. Simulations show that increasing the juvenile infectivity from the base of $\delta = 0.36$ to its max of $\delta = 1$ has little to no effect on the timing and intensity of WNV transmission, regardless of the value of the adult infectivity (Figure 4.4). However if you increase the adult infectivity from $\delta = 0.36$ to its max of $\delta = 1$, regardless of the juvenile infectivity, we see and increased and earlier disease transmission. This is similar to the effect we see with differential host stage recovery rate. The juveniles are only around for a short period of the season before transitioning to adults and make up a small portion of the population, so increasing their infectivity without any stage preference has very little effect on the intensity of WNV transmission.

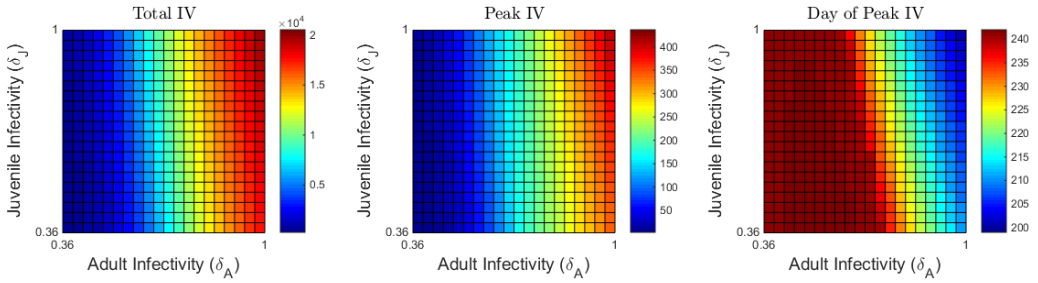

Figure 4.4: As the overall juvenile infectivity and the overall adult infectivity are varied, the juvenile recovery rate has little effect on the strength and timing of transmission while the adult recovery rate has a large effect on both the timing and intensity of WNV transmission.

4.1.3 Effect of Competence on WNV Transmission

Recall that competence is a measure of how likely a host is to become infected and transmit infection to a vector. Competence is found by multiplying infectivity (δ), sus-

ceptibility (β), and recovery rate (γ):

$$Competence = \beta(\frac{1}{\gamma})\delta = (\frac{1}{\gamma})\delta$$

since we assume β is equal to 1. Recovery rate γ is estimated by 1/(number of days infected), so we can consider competence to be the product of infectivity and number of days infected [15].

First we examine the overall effect on WNV transmission of increasing infectivity along with and decreasing recovery rate (i.e., increasing the competence of both host species). From Figure 4.5 we see that that there is not symmetry about diagonal. This is not surprising since we are now considering a combination of two different parameters. We see that as competence increases (infectivity increases and recovery rate decreases), overall WNV transmission increases. Therefore the maximum disease transmission occurs when the recovery rate is at its minimum of $\gamma = \frac{1}{10}$ and infectivity is at its maximum of $\delta = 1$. This would give the maximum competence of $\frac{1}{\frac{1}{10}} = 10(1) = 10$.

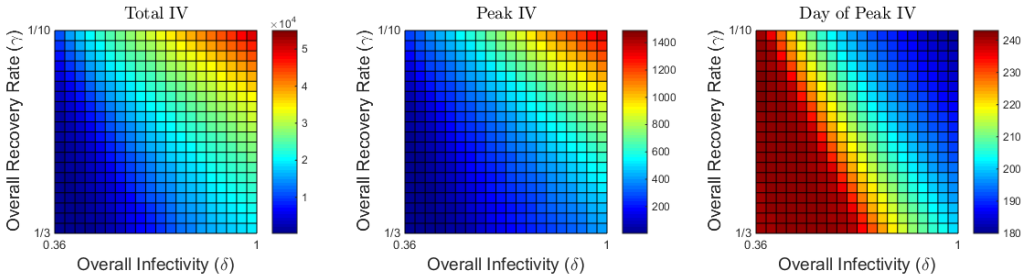

Figure 4.5: As competence increases (infectivity increases and recovery rate decreases) the overall WNV transmission increases. The non-symmetric plot shows that an increased infectivity has a larger impact on the intensity of WNV transmission than a short recovery rate.

When recovery rate is at its minimum of $\gamma = \frac{1}{10}$ and infectivity is at its minimum of $\delta = 0.36$, the strength of disease transmission is less then when infectivity and recovery rate are both at their respective maximums. From this we can conclude that when considering competence an increased infectivity is more of an indication of increased WNV

transmission then a low recovery rate. Similarly, we see that when recovery rate and infectivity are at their minimums, the timing of increased WNV transmission is later in the season then when they are both at their respective maximums. Again, this allows us to conclude that with respect to timing of increased WNV transmission a high infectivity is a greater indication of early WNV transmission then a fast recovery rate.

Now that we have considered the overall effect of competence, we can examine scenarios where a difference in competence may occur: between species and between stages. We first look at the effects of varying competence between the two host species. The minimum competence that can be achieved occurs at the baseline values of $\gamma = \frac{1}{3}$ and $\delta = 0.36$, and yields competence = $0.36(\frac{1}{\frac{1}{3}}) = 0.36(3) = 1.08$, while the maximum competence that can be achieved occurs at an infectivity rate of $\delta = 1$ and a recovery rate of $\gamma = \frac{1}{10}$, which results in competence = $1(\frac{1}{\frac{1}{10}}) = 1(10) = 10$.

We find that as the competence of one or both species increases so does the strength of disease transmission, and the timing of increased transmission moves earlier in the season. After seeing the individual effects of the parameters that make up competence (δ and γ) this is not surprising. Also, with no preferences, increasing competence yields the strongest and earliest WNV transmission.

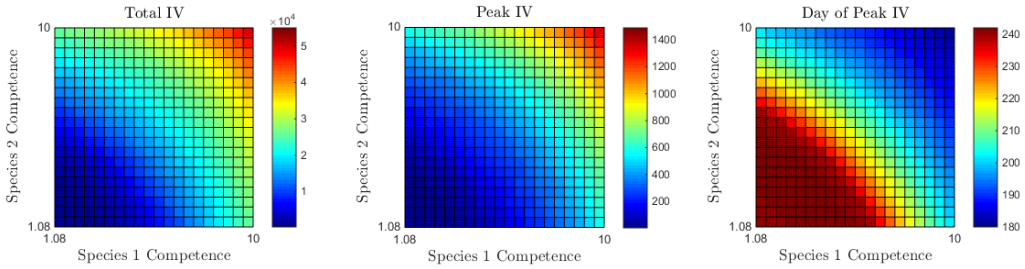

Figure 4.6: As the competence of both species increases, disease transmission increases achieving its maximum when both species are at maximum competence, while the day of peak IV moves earlier in the season.

However, when examining the effects of differential host stage competence we see a similar result as when we vary γ and δ by stage only (Figure 4.7). The competence of juveniles has a very small impact on the strength of WNV transmission regardless of the

competence of the adults. On the other hand, as the competence of the adults increases WNV transmission increases and peak WNV transmission moves earlier in the season.

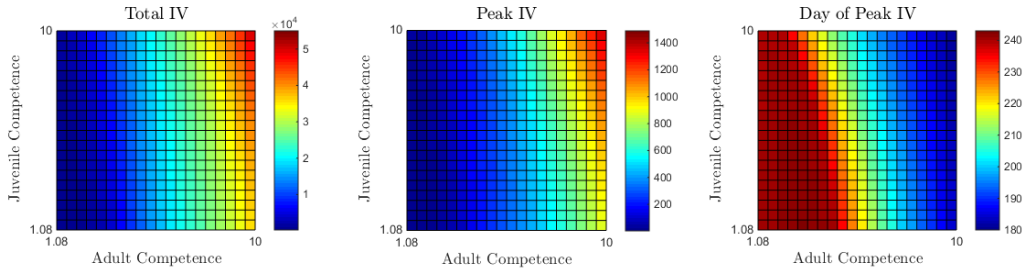

Figure 4.7: An increase in adult competence has a much larger effect on the strength of WNV transmission than an increase in juvenile competence. The maximum total and peak IV are reached when both juvenile and adult competences are at their maximum.

4.1.4 Effect of the Standard Deviation of Juvenile Recruitment on WNV Transmission

Nesting patterns may vary by species and climate largely due to resource availability. In warmer climates nesting can be less pulsed and clutch initiation date more variable. As the standard deviation of the recruitment curve is increased, we see a juvenile population curve with a smaller peak that is spread out over an extended period of time. This results in juveniles being around much longer during the season. With a small standard deviation, we see a juvenile population curve that is much more concentrated with a larger peak. This results the juveniles only being around for a short time during the season (see Figure 4.8).

As we vary σ and $\hat{\sigma}$ (Figure 4.9) we find that we get a symmetric response about the diagonal. When increasing σ and $\hat{\sigma}$ together, maximum WNV transmission occurs when they are both maximized. Increasing the standard deviation of the juvenile recruitment curves allows the juveniles to be around for an extended period of time. Without any preferences this simply yields an influx of susceptible hosts for a longer duration of the season. The final thing that we notice is that σ and $\hat{\sigma}$ have very little influence on the

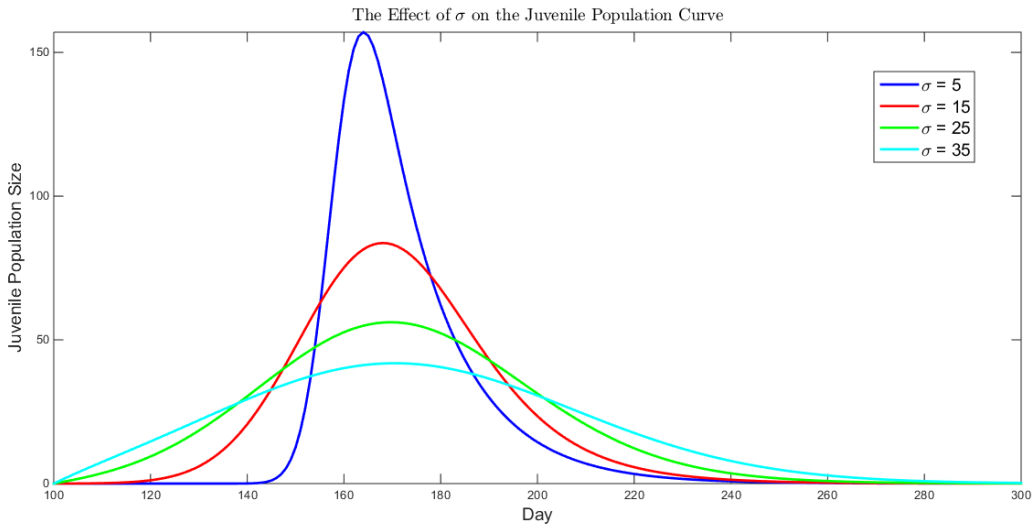

Figure 4.8: With a small juvenile recruitment curve standard deviation the juvenile population curve is condensed over a short period of the season with a very high peak. As the standard deviation gets larger, the juvenile population curve becomes more spread out over the season with a much lower peak. All parameter values are as in Table 2.1 with preference parameters at their baseline of 1.

timing of peak WNV transmission. So while the intensity of transmission is increasing, there is not a large change in the timing.

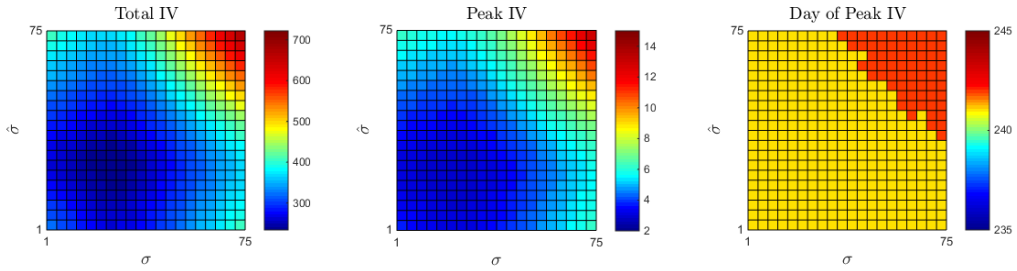

Figure 4.9: As σ and σ̂ increase, the IV and peak IV increase. However, there is not a large effect on the day of the peak IV. All parameters are as in Figure 4.8.

4.1.5 Effect of the Timing of Juvenile Recruitment on WNV Transmission

The mean of the juvenile recruitment curves give us an idea of what day the juveniles are being integrated into the total host population. As the mean of a juvenile recruitment

curve gets larger, juveniles are being introduced into the population later in the season. Similarly, as it gets smaller, juveniles are being introduced into the population earlier in the season.

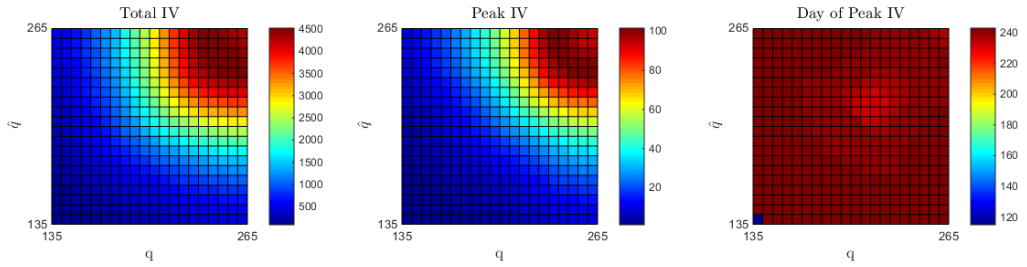

Figure 4.10: Varying q and $\hat{q}$, we see that maximum WNV transmission occurs when the juvenile recruitment curve is moved later in the season. With the juveniles being around only at the end of the season, during the season there is a much smaller host population (adults only) that is receiving all the bites and is therefore amplifying WNV transmission. All parameters values are as in Table 2.1 with all exposure coefficients equal to 1.

We found that the outputs of total and peak IV result in the same patterns (see Figure 4.10), with low WNV transmission occurring when the juveniles are introduced into the population earlier in the season and high WNV transmission occurring when the juveniles are introduced into the population later in the season. We also found that changing the juvenile recruitment curve mean had very little effect on the day of peak WNV transmission.

When q and $\hat{q}$ are large, the juveniles are being introduced into the system towards the end of the season. As a result, for a large majority of the season there is a smaller host population (consisting of adult hosts only) that is receiving all of the bites, therefore increasing transmission as a result of an increased bite to bird ratio. When q and $\hat{q}$ are smaller this increased bite to bird ratio does not exist as the total host population is around for the entirety of the season and therefore decreases WNV transmission.

4.2 Discussion

From our analysis on the effect of varying different parameters in our model with no preference present, we have found a number of different results.

When examining recovery rate (γ) and infectivity (δ) separately, looking at situation of differential species values we find that as you increase the recovery rate or infectivity the intensity of WNV transmission will also increase. However, when looking at varying these parameters between stages, the juvenile recovery rate and infectivity play little to no role in the intensity of disease transmission. We also found that when comparing these two parameters against each other, an increased infectivity more of an indication of increased WNV transmission than a short recovery rate.

We can also conclude that as host competence increases, the intensity of WNV transmission increases and peak WNV transmission moves earlier in the season. When we have an increase in stage competence, the juvenile competence plays little role in the strength of disease transmission or the timing of peak disease transmission, and WNV transmission is almost solely determined by the competence of the adult host population.

Also, increasing the standard deviation of the host juvenile recruitment curves in one or both species results in increased WNV transmission but has no effect on the day of peak transmission. As the standard deviation of the juvenile recruitment curve increases, it spreads the juvenile population out throughout the entire season resulting in a continuous new supply of susceptible hosts for the mosquitoes to infect.

Finally, we found that when both juvenile populations are not introduced until late in the season, there is an increase in WNV transmission. This increase occurs as a result of the decrease in the total host population (no juveniles). A smaller portion of hosts are receiving an increased number of bites and therefore amplifying disease transmission.

Chapter 5

A WNV Transmission Model with Two Host Species Incorporating Vector Biting Preferences

Since we have considered the effect of differential parameter values on our model with all preference parameter values at the baseline of $\epsilon = \hat{\epsilon} = 1$ and $\epsilon_J = \epsilon_A = \epsilon_{\hat{J}} = \epsilon_{\hat{A}} = 1$, we will now examine the effects of only having different vector feeding preferences with all other parameters at their respective baselines. When considering heterogeneous vector feeding preferences, there are a few scenarios that we want to investigate and compare: different host species feeding preferences, different host stage feeding preferences, a feeding preference for a stage in the preferred species, and a feeding preference for a stage in the non-preferred species.

Recall that although we refer to increased biting rates on certain hosts as a vector preference for those hosts, these hosts may also just be receiving more bites because they are more readily available (or more exposed). A parameter value of $\epsilon = 10$ accounts for the situation where host species 1 is more preferred by the vector and therefore is receiving 10x as many bites as host species 2, while it also accounts for the situation where host

species 1 is more exposed to the vector, perhaps as a result of nesting characteristics, and as such is receiving 10x as many bites as host species 2. Similarly, having a parameter value of $\epsilon_J = 10$ accounts for the situation where juveniles of host species 1 are more preferred by the vectors and therefore are receiving 10x as many bites as adults, as well as the situation where juveniles of host species 1 are more exposed to the vectors, perhaps as a result of low feather covering or weak defensive behavior [3, 11] and are therefore receiving 10x as many bites as adults.

5.1 Results

First we consider the effect of vector feeding preferences on different host species. Differential species preferences often occur in nature; for example, *West Nile virus* research indicates a strong vector feeding preference for the American robin (*Turdus migratorius*) [23]. When examining the effect of host species preferences, we vary the values of ϵ and $\hat{\epsilon}$ from 1 to 15 (no feeding preference to a very strong feeding preference) and measure the effects in terms of total and peak IV which are indicators for the intensity of WNV transmission, as well as the day of peak IV which tells us the timing of peak WNV transmission. Through simulation, our analysis shows a symmetric relationship about the diagonal (Figure 5.1), which occurs because the two host populations are otherwise equal. We also find that the most intense WNV transmission occurs when one host species does not have a vector feeding preference (ϵ or $\hat{\epsilon} = 1$), and the other host species is has the maximum vector feeding preference (ϵ or $\hat{\epsilon} = 15$). When one host species has a strong preference, if the other also has a preference it is only taking bites away from the more preferred host. As the species' preferences increase together, strength of disease transmission stays constant because it is the ratio of $\epsilon/\hat{\epsilon}$ that determines the preference. So regardless of the magnitude of exposure coefficients (ϵ and $\hat{\epsilon}$), if they are equal for both species then both species are being bitten at equal rates and it is as though there is

no vector preference.

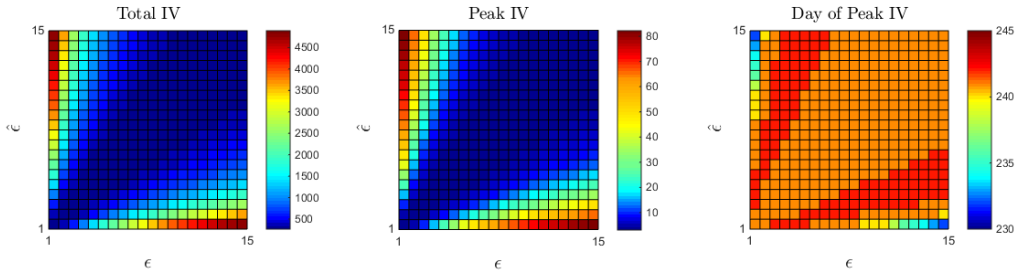

Figure 5.1: Increasing the host species preference increases WNV transmission only if there is a differential species preference. This is because it is the ratio of $\epsilon/\hat{\epsilon}$ that determines the preference (if $\epsilon = \hat{\epsilon}$ there is no preference). All parameters are as in 2.1 with K = 15000 and stage preference parameters at 1.

We now examine the effect of having a vector feeding preference on a host life stage (juvenile or adult) while all other parameter values are kept at their respective baselines. Differences in biting rates between stages may occur for a number of reasons. It is likely that the preference (or exposure) parameter values for juveniles will be higher than adults because juveniles have little feather coverage and are unable to defend themselves [3, 8, 22]. Our analysis shows that adult preference alone has little to no effect on WNV transmission (see Figure 5.2). Once the adult preference gets large enough, WNV transmission remains constant and at a minimum value, while the timing of peak WNV transmission also remains constant but at its maximum value (late in the season). If we keep the adult preference low, as the biting rates on juveniles increase, WNV transmission increases and peak transmission moves earlier in the season correlating closely with the timing of the increase in juvenile population. If there is a strong juvenile preference, then before the juveniles are around the mosquitoes are going to be attacking the susceptible adult mosquitoes resulting in infected adult hosts as well as infected mosquitoes. By the time the juveniles are introduced into the population, the number of infectious vectors will be elevated and they will all have a strong feeding preference for the completely susceptible and very small juvenile population. Since a small portion of the population will be receiving a majority of the bites, the juveniles will quickly become

infected and in turn infect a large number of vectors. Once the juveniles have matured into adults, the mosquitoes will then turn their attention back to the adult population (many of whom are still susceptible). As a result of this interaction, disease transmission will increase and the timing of peak disease transmission will move earlier in the season to correlate closer to the timing of the increase in the juvenile population.

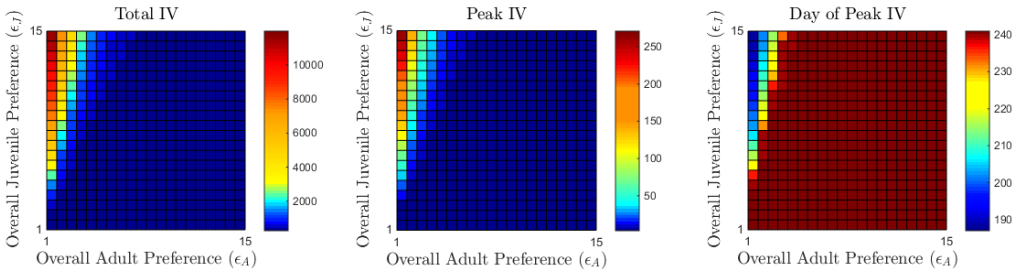

Figure 5.2: Examining the effect of differential stage preference we see that an adult preference has little to no effect on the timing or intensity of WNV transmission. However, an increased juvenile preference indicates an increase in disease transmission with the maximums transmission occurring when there is no adult preference ($\epsilon_A = 1$) and a strong juvenile preference ($\epsilon_J = 15$).

We now examine the effects of the interaction of species preference and stage preference. When combining a species preference and a stage preference, there are four possible cases we consider: (1,2) the preferred species has a stage preference (juvenile or adult), (3,4) or the non-preferred species has a stage preference (juvenile or adult).

By creation of our model, vectors first choose between the two species and then the two stages. Since the species choice is first, an increased preference for a species means an increased number of bites on that species. If the preferred species also has a juvenile preference, then that means an increased biting rate on the juveniles of the preferred species. As one or both preferences increase, the intensity of WNV transmission increases and the timing of peak transmission decreases (Figure 5.3). Disease transmission reaches its maximum when both ϵ and ϵ_J are at their maximum values of 15. When this occurs host species 1 has a very strong preference. Before the juveniles are around all bites are going to the adults of host species 1 which results in an increase in IV. Once the

strongly preferred juveniles are introduced into the community, almost all of the bites on hosts will be directed to a very small, very susceptible fraction of the population which causes an increase in WNV transmission and moves the timing earlier in the season to correspond with the availability of the juvenile population.

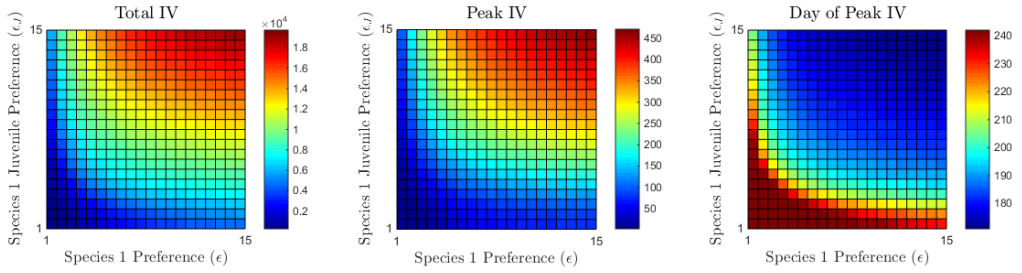

Figure 5.3: If the preferred species has a juvenile preference, then as one or both biting rates increase WNV transmission increases while the timing of peak WNV transmission moves earlier in the season. In this situation, the two preferences are working together resulting in an increased number of total and peak IV.

If the preferred species instead has an adult preference then we get different dynamics. The adult preference has little effect on WNV transmission and instead the species preference is doing a large portion of the work. As we increase the adult preference we do not see much of a change in intensity of disease transmission, however as we increase the species preference we will always see an increase in disease transmission, regardless of the adult stage preference. As we increase the two together, we see an increase WNV transmission. While the adult preference does not contribute much, the highest WNV transmission occurs when both the species and stage preferences are at their maximum values of ($\epsilon = \epsilon_A = 15$). Comparing this to the results of a juvenile stage preference for the preferred species, WNV transmission is decreased when the preferred species has an adult stage preference. This tells us that when a species preference exists, having an increased biting rate on juveniles if that species is a mechanism of increased WNV transmission than having an increased biting rate on adults.

Next we examine the effects of the non-preferred species having a stage preference. Again by construction species are chosen before stages. If the non-preferred species has

a stage preference (juvenile or adult), then that preference has little effect on the transmission of WNV because the species with the stage preference is getting very few bites (Figure 5.4). This is shown in Figure 5.4 because when increasing the species preference we get similar values regardless of the strength of the stage preference. Looking at cases when there is a stage preference for the non-preferred species, the species preference alone will determine the intensity of WNV transmission.

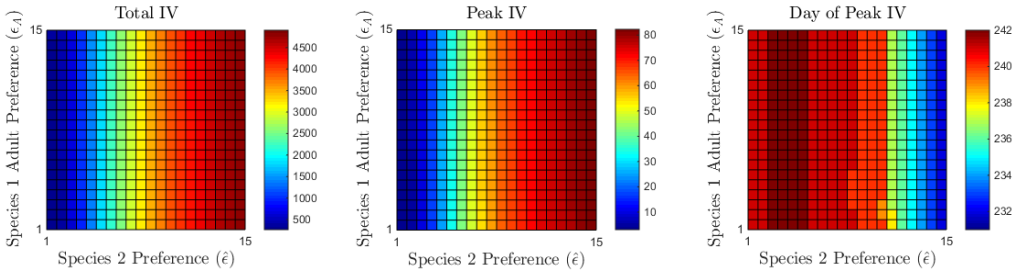

Figure 5.4: When the non-preferred species has a stage preference (juvenile or adult), the stage preference has little effect on the intensity and timing of WNV transmission. As the species preference increases, we get the close to the same values, regardless of the strength of the stage preference.

5.2 Discussion

Through the analysis of the effect of differential species and/or stage preferences (or exposure) on our model, we have been able to identify different scenarios where homogeneous vector preference yields maximum WNV transmission, as well as scenarios where heterogeneous vector preference yields maximum WNV transmission.

When comparing the effects of heterogeneous biting rates on host species 1 and host species 2, whenever both species have equal preference it is the same as neither species having any preference regardless of preference strength. Going back to chapter 2 and the α and $\hat{\alpha}$ functions (proportion of bites going to species 1 and 2 respectively), anytime ϵ and $\hat{\epsilon}$ are equivalent they will cancel each other out and each species will be bitten in proportion to its abundance in the population and all hosts will receive the same number

of bites. Similarly, when we are comparing the effects of preferences on the overall host stages (juvenile and adult) if we look at our $\alpha_J, \alpha_{\hat{J}}, \alpha_A, \alpha_{\hat{A}}$ functions (proportion of bites going to juveniles of host species 1 and 2, and adults of host species 1 and 2 respectively), if the preferences for each stage are equal to each other (regardless of their magnitude) ($\epsilon_J = \epsilon_{\hat{J}} = \epsilon_A = \epsilon_{\hat{A}}$) then all the preference terms will cancel each other out and each stage will be bitten in proportion to its abundance in the population and all hosts will receive the same number of bites. In these cases we see the maximums occurring with heterogeneous vector preference. To increase the disease transmission, the preferences for each stage or each species cannot be equal.

When combining the effects of species preference with stage preference, this is not the case. In these situations regardless of whether we are looking at stages of the preferred or non-preferred host species, as we increase both preferences together we are always increasing the intensity of WNV transmission. When looking at an increased biting rate for either stage of the preferred host species, the species preference and stage preference are both contributing to the increase in WNV transmission. Not only is the species receiving an increased number of bites, but when the preferred stage is around it is also receiving an increase in bites. This results in the majority of the bites being concentrated on a small fraction of the population which increases disease transmission. When looking at a preference for the stages of the non-preferred host species, the stage preference has very little effect on the intensity of WNV transmission are it is almost completely dictated by the species preference. Therefore, as the species preference increases so does disease transmission, regardless of the stage preference. In these cases we see the maximum WNV transmission occurring with homogeneous vector preferences.

Chapter 6

Comparing the Effect of Unequal Vector Biting Rates on Host Species Versus Host Life Stages

In Chapter 4, we discussed the effects of varying certain parameters (infectivity, recovery rate, competence, juvenile recruitment curve standard deviation, and juvenile recruitment curve mean) on our model. In Chapter 5, we discussed the different effects of incorporating species and/or stage preferences into our model. From the previous chapter, we know that there is a difference between having a species preference and having a stage preference in terms of the intensity and timing of WNV transmission. Our next step is to combine the two previous chapters and examine the difference between having a species preference or a stage preference while also varying a second parameter. Since juvenile preferences often occur in nature due to lack of defense mechanisms, low feather coverage, inability to leave the nest, etc., we are going to specifically examine the effect of a species preference against a juvenile stage preference with differential species parameter values for a single parameter. The parameters that we are going to focus on varying are competence, standard deviation of juvenile recruitment, and timing of

juvenile recruitment.

6.1 Results

6.1.1 Comparing the Effect of Species and Stage Preferences on WNV Transmission for Two Identical Host Populations

Before we can incorporate different host species parameter values, we first want to look at the base case (all non-preference parameters at their respective baselines) of comparing a increased species preference ($\epsilon = 15$) with an increased overall stage preference ($\epsilon_J = \epsilon_{\hat{J}} = 15$). Figure 5.3 shows that having an overall juvenile preference results in a much larger intensity of disease transmission than a species preference, and with a juvenile preference the timing of peak disease transmission is much earlier in the season then with a species preference (Figure 6.1).

We get increased WNV transmission with an increased biting rate on juveniles because when the juveniles (the preferred host) show up they are receiving an increased number of bites. The infectious mosquitoes are concentrating their bites on a small, susceptible portion of the population which results in an increase in disease transmission. This is also why the timing of peak transmission moves up so early in the season, because it is directly correlated with the presence of juvenile hosts.

Another thing that occurs with an increased juvenile stage preference that does not occur with an increased species preference is differential biting rates on infection classes (see the bottom row of graphs in Figure 6.1). When we have a species preference all classes get bitten in proportion to their abundance in the overall host population. This is because there is no preference between the stages of either host species. However, incorporating a juvenile preference results in differential biting rates on classes because the juveniles are being bitten at an increased rate when present in the population (Figure

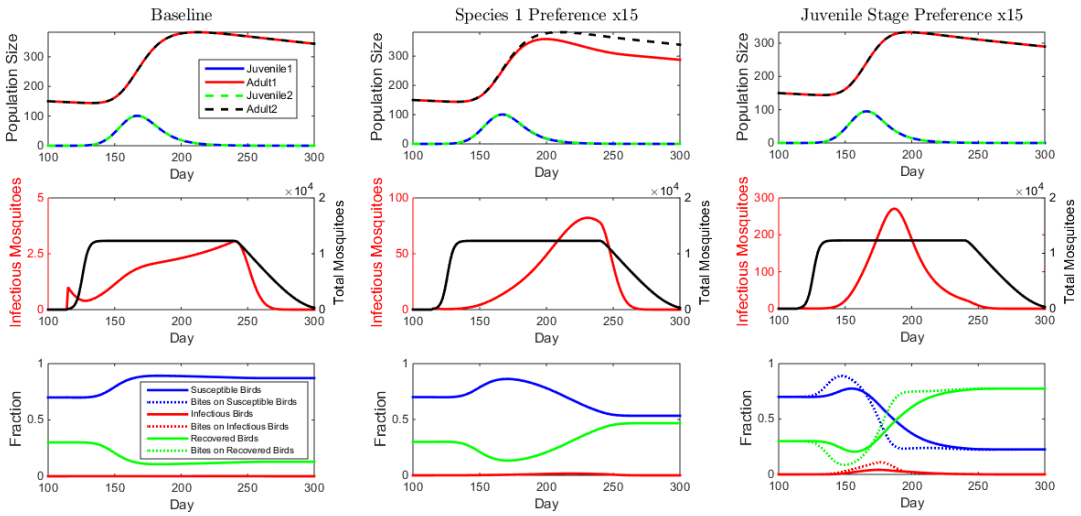

Figure 6.1: An increased juvenile stage preference of $\epsilon_J = \hat{\epsilon} = 15$ results in an increased number of *West Nile virus* infectious mosquitoes and much earlier peak transmission than a species preference of $\epsilon = 15$. The top row shows the abundance of each of the host stages over the course of the season. The middle row shows the number of infectious mosquitoes as well as total mosquito abundance throughout the season. The bottom row shows the percentage of the population in each class (susceptible, infected, recovered) throughout the season as well the percentage of mosquito bites on birds in each of these classes. Parameters are as in 2.1 and all other preferences equal to 1 [20].

6.2). Initially, the entire juvenile population is susceptible, so we see susceptible host begin to receive an increased number of bites relative to their abundance. Once these juveniles become infected, we see an increase in bites on infected hosts, and once the juveniles recover from infection, we see an increase in bites on recovered hosts. Finally, when the juveniles leave the system through death or mature into adults, all hosts are again bitten in proportion to their abundance in the community.

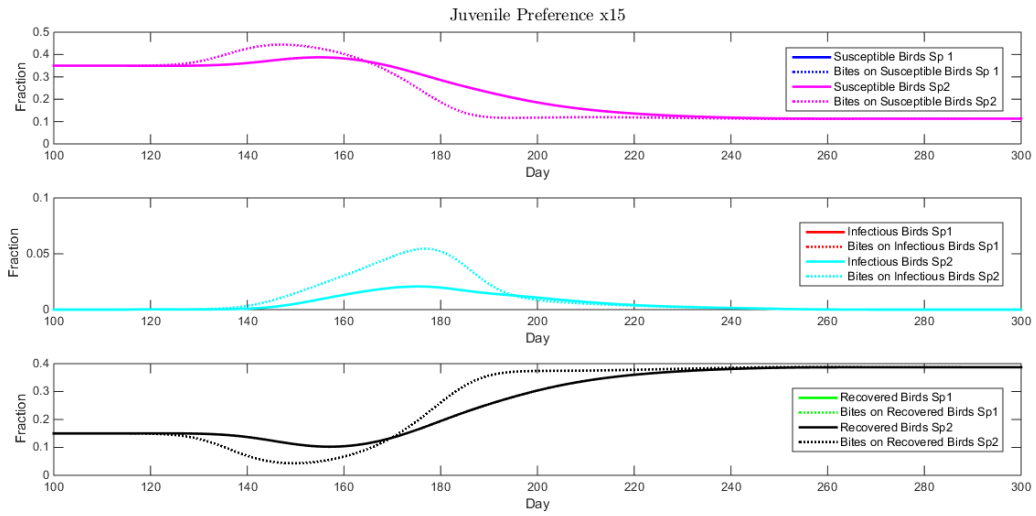

Figure 6.2: Distribution of bites to each host class (susceptible, infected, recovered) with a juvenile preference of $\epsilon_J = \epsilon_{\hat{J}} = 15$. During the period of time when the juveniles are present in the population we get differential biting rates as a result of the strong juvenile preference. Host species 1 and 2 are both modeled, however because each species are equally abundant (each make up 50% of the population), their curves are exactly the same, and so we can only see one of the species curves. All parameters are as in Figure 6.1.

6.1.2 Comparing the Effect of Species and Stage Preferences on WNV Transmission for Two Host Populations Differing in Competence

In Chapter 4 our analysis showed that if you increase competence and keep all other parameter values at their respective baselines, then disease transmission increases while the timing of peak disease transmission moves earlier in the season. Now, we are going to examine and compare the consequence of adding a species preference and juvenile stage preference onto host populations with difference competencies. For species preference there are two cases that must consider: (1) an increased vector feeding preference for the more competent species, and (2) an increased vector feeding preference for the less competent species. We do not have to consider multiple scenarios for an overall juvenile preference because both species have the juvenile preference.

Examining the differences between a species preference for the more competent species and an overall juvenile preference, we find that while the timing of peak transmission is similar the intensity of disease transmission is much smaller with the species preference (Figure 6.3). Intuitively, if there is a species preference for the less competent species, this peak will be even smaller (6.3). In fact, we found that when comparing an increased species preference with an increased juvenile stage preference, the juvenile stage preference will nearly always result in more intense WNV transmission.

There is only one scenario where this is not true. The only time when a strong species preference will surpass a strong juvenile stage preference and result in more intense WNV transmission occurs when we increase the competence of the more preferred host species to its maximum (Figure 6.4). Again, as a result of the highly increased competence levels, we see that in the cases of no preference and juvenile stage preference, by the end of the season almost 100% of the population is recovered.

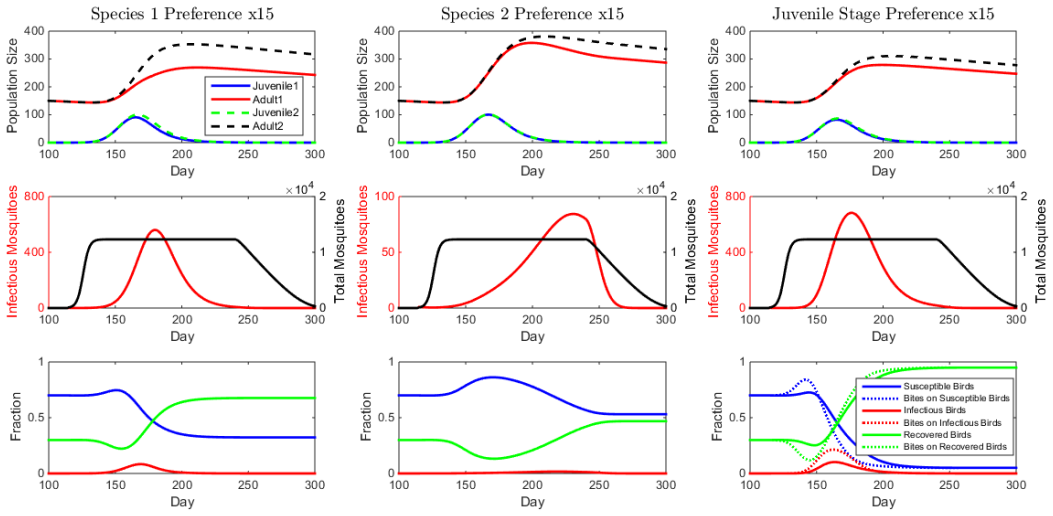

Figure 6.3: Examining the difference between species preference ($\epsilon = 15$) and juvenile stage preference ($\epsilon_J = \epsilon_{\hat{j}=15}$) with differential host competence. The intensity of WNV transmission is higher with a juvenile stage preference than with a species preference. Also, in the bottom graph, we see that the combination of a juvenile stage preference and increased competence results in nearly 100% of the susceptible host population becoming infected by the end of the season. Competence parameters are set at $\gamma_1 = \frac{1}{5}, \gamma_2 = \frac{1}{3}, \delta_1 = 0.6, \delta_2 = 0.36$, and all other parameters are as in Figure 6.1.

Another interesting thing that occurs with an increased competence of one host species and an overall juvenile preference that doesn't occur with only a species preference, is that by the end of the season almost 100% of the host population is recovered. This means that the combination of an increased biting rate on juveniles combined with an increased species competence results in nearly 100% of all susceptible hosts (from both the preferred stages and non-preferred stages) becoming infected by the end of the season (bottom graph for juvenile preference in Figure 6.3).

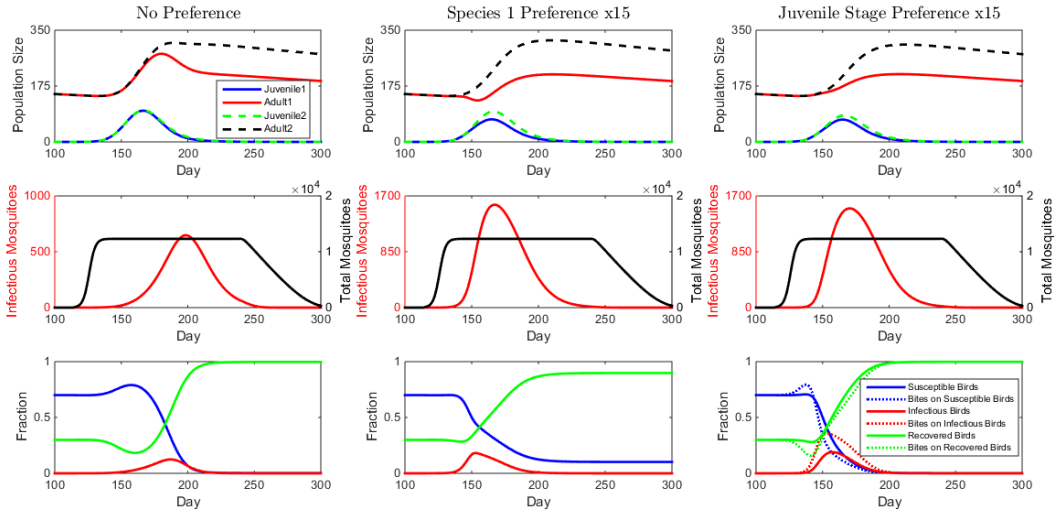

Figure 6.4: The only time when an increased biting rate on one host species can result in more intense WNV transmission than an increased biting rate on juveniles of both species occurs when the infectivity and recovery rate of the more preferred species are increased to their maximum of $\gamma = \frac{1}{10}$ and $\delta = 1$. All other parameter values are as in Figure 6.3.

6.1.3 Comparing the Effect of Species and Stage Preferences on WNV Transmission for Two Host Populations Differing in Standard Deviation of Juvenile Recruitment

Analysis from Chapter 4 showed that increasing the standard deviation of the juvenile recruitment curve of either host species 1 or host species 2, or both, results in increased

WNV transmission and has little to no effect on the day of peak transmission. Now we examine and compare the differences between incorporating a host species preference and a host juvenile stage preference with different species values of σ and $\hat{\sigma}$. When considering different values of the standard deviation of the juvenile recruitment curve along with a species preference, there are two cases we consider: (1) the preferred species has a larger standard deviation of juvenile recruitment, and (2) the preferred species has a smaller standard deviation of juvenile recruitment.

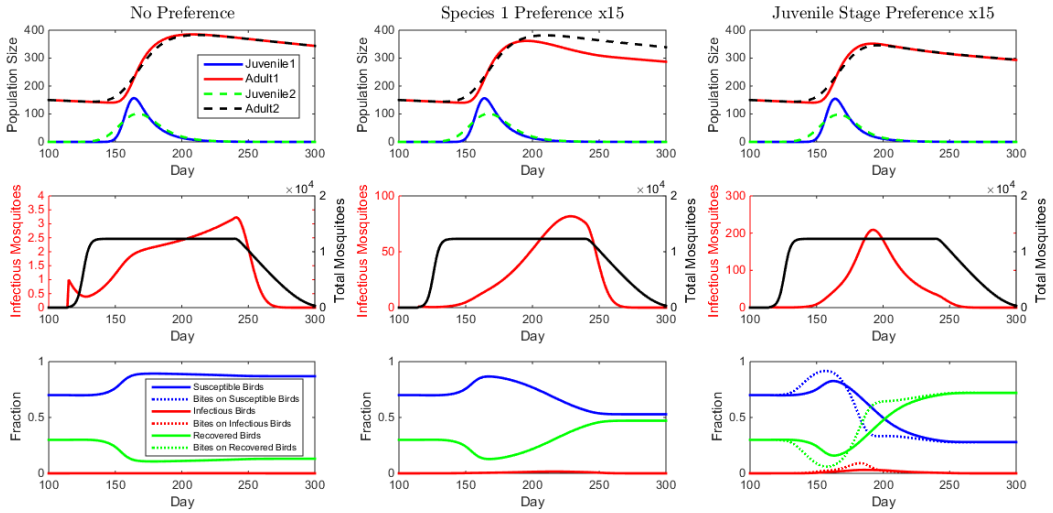

Figure 6.5: The effect of a host species preference and a host juvenile stage preference combined with differential standard deviations of the juvenile recruitment curves. The juvenile stage preference will always result in more intense WNV transmission than the host stage preference. Also the juvenile preference forces the timing of the peak infectious mosquito curve to move up in the season to correlate closer with the juvenile population (middle graph). Host juvenile recruitment standard deviations are set at $\sigma = 5, \hat{\sigma} = 11.4$. All other parameters are as in Figure 6.1.

With different values of σ and $\hat{\sigma}$, an increased juvenile stage preference will always result in higher disease transmission than an increased host species preference, regardless of whether the preferred species has a larger standard deviation or a smaller standard deviation compared to the non-preferred species (Figure 6.5). Recall that as σ increases the total juvenile population becomes much more concentrated over a very short period of time, while as σ decreases the total juvenile population is less concentrated and

spans over an extended period of time. Therefore, if there is any type of juvenile preference, having one of the preferred hosts around for a longer period of time will certainly increase WNV transmission.

6.1.4 Comparing the Effect of Species and Stage Preferences on WNV Transmission for Two Host Populations Differing in Timing of Juvenile Recruitment

The analysis conducted in Chapter 4 revealed that increased WNV transmission occurs when the juveniles of both species are introduced into the host population late in the season (large q and $\hat{q}$). When considering different values of the timing of juvenile recruitment along with a species preference, there are again two scenarios that we account for: (1) the preferred species has a later juvenile recruitment in the season, and (2) the preferred species has an earlier juvenile recruitment in the season.

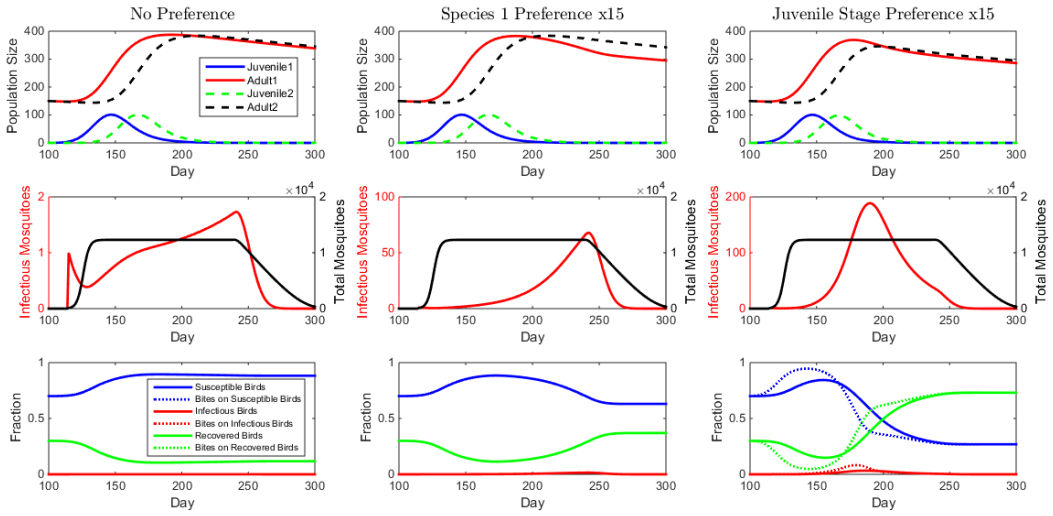

Figure 6.6: The effect of a host species preference and a host juvenile stage preference on differential mean juvenile recruitment curve values. The juvenile stage preference results in a larger number of peak IV than the host species preference because offsetting the juvenile recruitment curves offsets the two host juvenile populations, which means the preferred host is going to be around longer during the season. Host juvenile recruitment curve means are set at $q = 138$, $\hat{q} = 158.17$. All other parameters are as in Figure 6.1.

In both situations the juvenile stage preference results in a more increased disease transmission than having a host species preference (Figure 6.6). Recall that the juvenile recruitment curve mean moves the timing of the juvenile presence in the host population. If the juvenile population curves are offset, then it results in juveniles being around longer during the season. With the preferred host population around for a larger portion of the season and receiving an increased number of bites, we will see a significant increase in the intensity of WNV transmission.

6.2 Discussion

In this chapter, we explored and compared the differences between an increased host juvenile stage preference and an increased host species preference while also varying a second parameter. Aside from the preferences, the parameters we focused on were competence, juvenile recruitment curve standard deviation, and juvenile recruitment curve mean. In almost all cases, a strong juvenile stage preference resulted in a more increased disease transmission than a strong species preference. This is interesting because the juveniles are only around for a very short period of time during the season before they mature or leave the system through death, yet may play a major role in amplifying the disease risk for vectors and other hosts.

The one case where a host species preference can result in a more increased disease transmission occurs when we increase the competence of the preferred species to its maximum of $\gamma = \frac{1}{10}$ and $\delta = 1$. Even in this case there is not a large difference between the intensity of disease transmission.

We also found that incorporating a juvenile preference results in differential biting rates on host stages. When this occurs, hosts are not bitten with respect to their abundance in the community (which is different than what happens with a species or no preference). Hosts are no longer being bitten in proportion to their population because

there is such a strong juvenile stage preference that they are receiving a large quantity of bites from vectors in all juvenile classes (susceptible, infected, recovered) but only making up a small proportion of the host population.

Chapter 7

The Effect of Host Community Composition on WNV Transmission

Community composition is very important in determining the disease risk of an area for vector borne diseases [9, 19]. It has been shown that when a second host with low competence is present in the population, it will receive bites otherwise allocated to the more competent host and will "dilute" the disease risk; this is referred to as the dilution effect [19, 21]. Using this logic, as the less competent host becomes more abundant in the host population, the dilution effect increases and the disease risk decreases. Similarly, as the more competent host becomes more abundant in the host population, the dilution effect decreases and the disease risk increases. Maximum disease risk would therefore be obtained when the population is composed of solely the more competent species.

Many studies have questioned the universality of the dilution effect [4, 18, 23]. Recently, Miller and Huppert created a dual host model (with no stage-structure) and explored how a combination of host diversity, host competence, and vector preference can affect the disease risk of an area. They discuss a new mechanism which they dubbed "diversity amplification", where under certain circumstances the presence of multiple hosts can actually increase the disease risk (as measured by the basic reproduction num-

ber, R_0) [19]. This is important because between dilution and diversity amplification, they are able to account for the conflicting patterns that occur in nature.

In this chapter we examine the effect of changing host abundance in our model with differential competence and host preferences. We will examine scenarios under which Miller and Huppert found one should observe either the dilution effect or diversity amplification, and see if the same results hold in our model that incorporates stage-structure. We will then incorporate stage preference and examine the effects on diversity amplification and dilution. Note that we will measure disease risk using total IV and peak IV rather than R_0, for reasons discussed in Chapter 2.

7.1 Conditions for Dilution

In literature, the dilution effect is described as occurring if an increase in the host diversity results in a decreased probability that a vector will come across a highly competent host [19, 23]. Therefore, if there is a high abundance of a low competence host it will decrease the chance of a vector coming across a highly competent host. By this reasoning, we can see that the maximum and minimum disease risk would occur when the community is composed of a single host species.

Miller and Huppert found that there are two cases when dilution will occur, and in accordance with dilution theory logic, disease risk (R_0) increases monotonically in proportion to the abundance of the higher competence host. They find dilution occurs either (1) when there is no species preference and host species differ in competence, or (2) when there is a feeding preference for the less competent species.

While we have not proven this to be true for our model, large numbers of simulations indicate that these conditions also result in dilution for our model (Figures 7.1 and 7.2). In accordance with dilution theory logic, in both cases the peak IV and IV (disease risk) increase monotonically as the more competent species increases in abundance. As

such, the maximum disease risk occurs when only a single species is present in the host population.

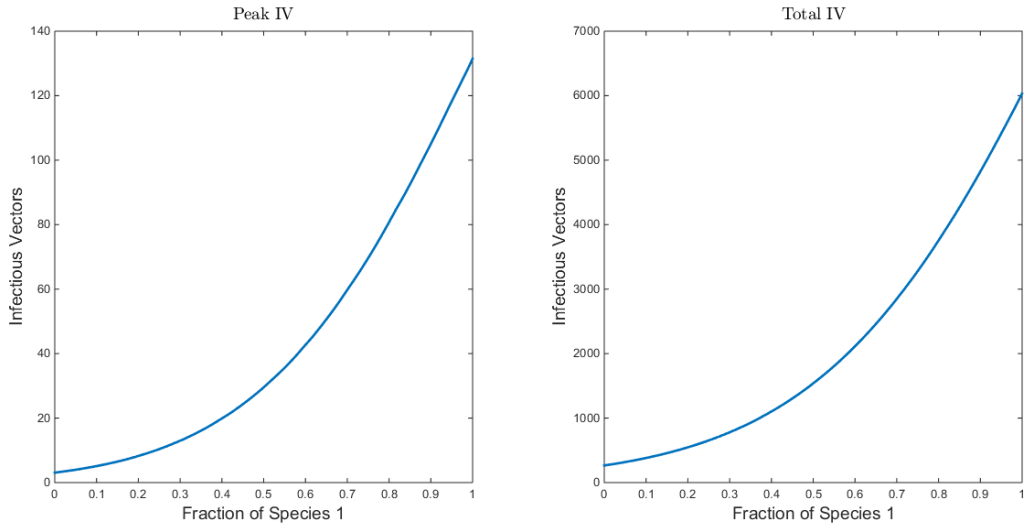

Figure 7.1: Peak IV and total IV curves decrease monotonically as the fraction of the less competent species (species 1) in the population increases (dilution). Here there is no species feeding preferences, and species 1 is more competent than species 2 ($\gamma_1 = \frac{1}{4}, \gamma_2 = \frac{1}{3}, \delta_1 = 0.50, \delta_2 = 0.36$)

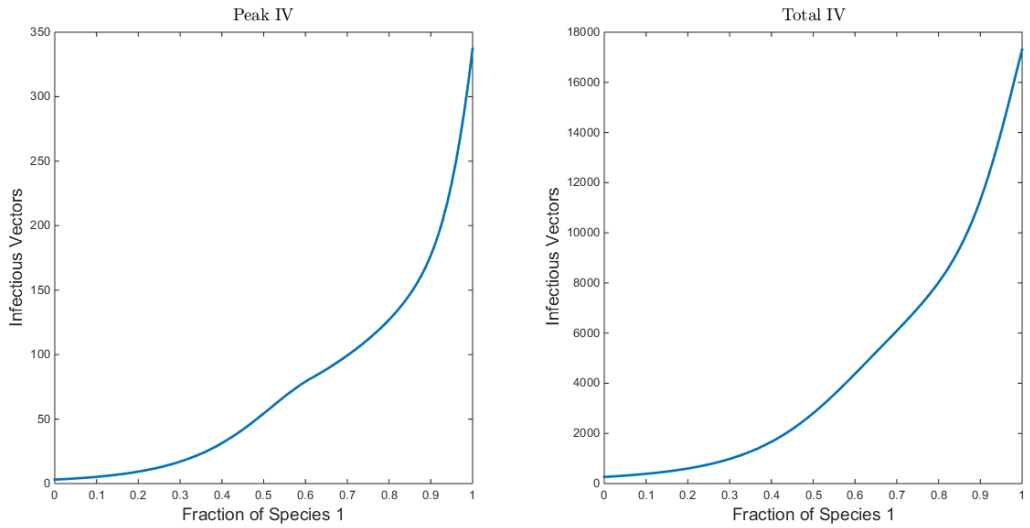

Figure 7.2: Peak IV and total IV curves increase monotonically as the fraction of the more competent species (species 1) in the population increases (dilution). Here there is a preference for the less competent species (species 2; $\hat{\epsilon} = 5$), and species 1 is more competent than species 2 ($\gamma_1 = \frac{1}{5}, \gamma_2 = \frac{1}{3}, \delta_1 = 0.60, \delta_2 = 0.36$)

7.2 Conditions for Diversity Amplification

While the dilution effect is widely accepted, there have been articles that question its universality by presenting evidence that maximum disease risk actually occurs when the community is composed of multiple host species, not a single host as the dilution effect suggests [18, 19, 23]. Miller and Huppert dubbed this mechanism diversity amplification, because in these cases communities of multiple host species have amplified disease risk compared to those comprised of a single species. They showed that there are also two cases when diversity amplification will occur: (1) when one host species is preferred but both are of equal competence, and (2) when the species that is preferred is also more competent.

Again, while we have not proven this to be true for our model, large numbers of simulations indicate that when these conditions are satisfied in our model, diversity amplification occurs and the maximum disease risk occurs when both species are present in the community (Figures 7.3 and 7.4). Since the maximum disease risk occurs when both species are present, we find the maximum values of peak IV and total IV at interior points and thus we get non-monotonic ”hump-shaped” curves, unlike the monotonically increasing or decreasing curves that result from dilution.

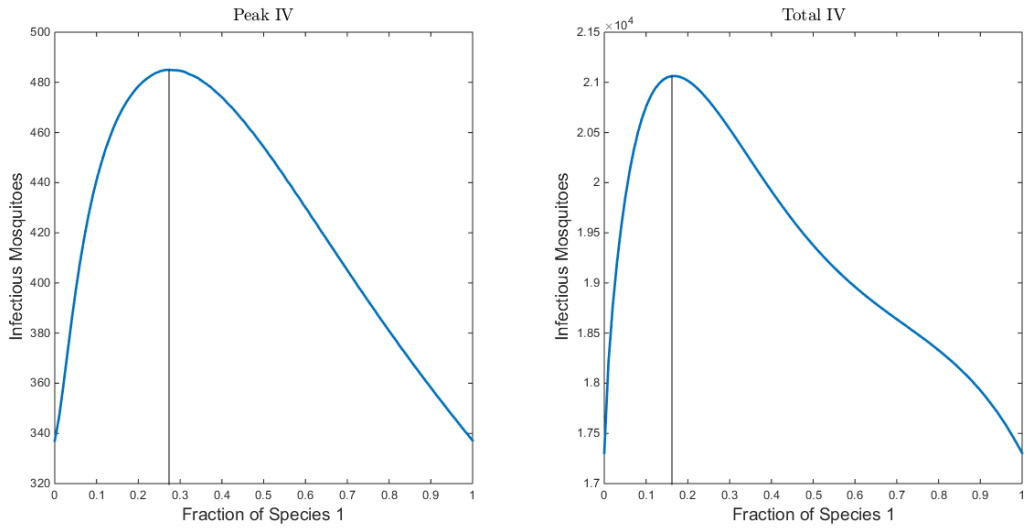

Figure 7.3: Disease risk is a non-monotonic function of species proportion. Therefore maximum disease risk occurs when both species are present in the community (diversity amplification). Here there is a feeding preference for species 1 ($\epsilon = 5$), but both species have equal competence ($\gamma_1 = \gamma_2 = \frac{1}{5}, \delta_1 = \delta_2 = 0.60$).

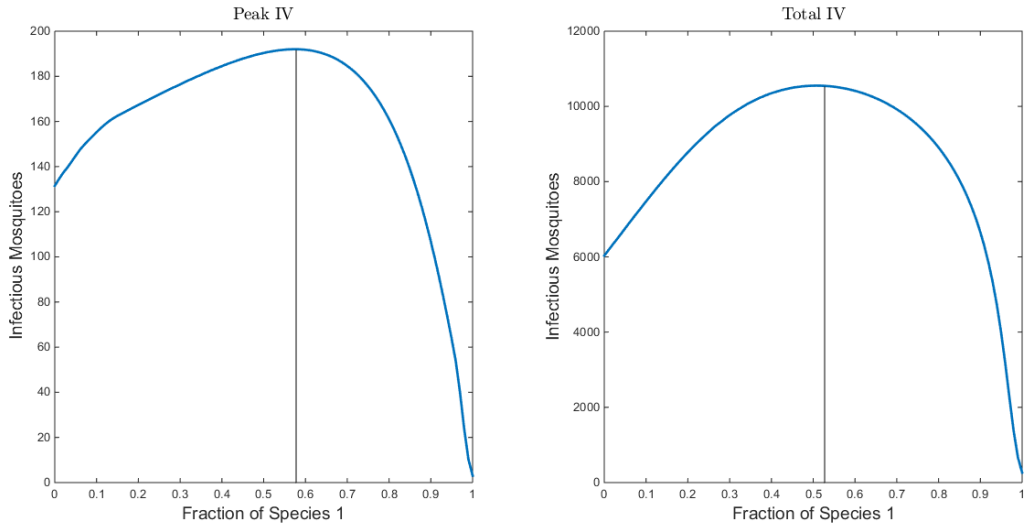

Figure 7.4: Disease risk is a non-monotonic function of species proportion. Therefore maximum disease risk occurs when both species are present in the community (diversity amplification). Here there is a feeding preference for the more competent species, species 2 ($\hat{\epsilon} = 4$), with competence parameters set at $\gamma_1 = \frac{1}{3}, \gamma_2 = \frac{1}{4}, \delta_1 = 0.36, \delta_2 = 0.50$.

7.3 The Effect of Stage Preference on the Conditions for Dilution and Diversity Amplification

The Miller and Huppert model incorporates multiple species but does not take into account any stage-structure. As such, all of their conclusions regarding the situations in which dilution and diversity amplification will occur are only in terms of species preference, competence, and abundance. While their conclusions regarding these situations also hold true in our stage-structured model (with no stage preference), it is of interest for us to see if incorporating stage preferences can change the situations that result in diversity amplification or dilution (we first consider the conditions where we will get dilution, then the conditions where we will get diversity amplification).

If there is no species preference and host species differ in competence we get dilution, where disease risk increases monotonically as the more competent species increases in abundance (see Figure 7.1). If we incorporate a juvenile preference for the less competent species (species 1), as we increase the juvenile preference, dilution does not turn into diversity amplification. However, we find that instead of the disease risk decreasing with the proportion of the less competent species, it begins to increase with the proportion of the less competent species (see Figure 7.5). This tells us that in this situation the juvenile preference of species 1 is outweighing the increased competence of species 2. As the strength of the juvenile stage preference increases, a concentrated number of bites are going to a small fraction of the population which is resulting in an increase in disease risk.

The second case where Miller and Huppert found dilution was a species preference for the less competent species (see Figure 7.2). We found dilution turned into diversity amplification when we incorporate a juvenile preference for the less competent species. As we increase this juvenile preference, the disease risk curves go from increasing with the proportion of the more competent species and achieving its maximum when only the

more competent species is present, to being a hump-shaped curve where the maximum is achieved when both host species are present (see Figure 7.6). Here, having a strong juvenile stage preference is functionally similar to having increased stage competence. So a biting preference for the less competent species and a strong juvenile preference for the less competent species would be functionally similar to the case of diversity amplification where the more preferred species is also more competent (see Figure 7.4).

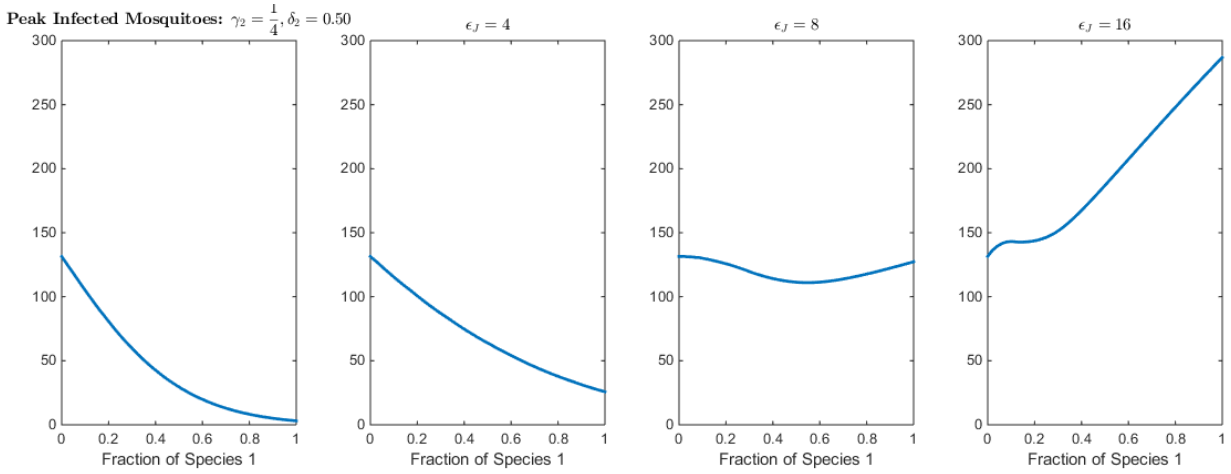

Figure 7.5: As we increase the strength of the juvenile preference for the less competent species (species 1), disease risk beings to increase (rather than decrease) as the proportion of host species 1 increases. In this case, the strong juvenile preference for species 1 overcomes the effects of the increased competence of species 2. All competence parameters are assumed to be as in Figure 7.1.

Diversity amplification can occur when there is a preferred host species and both species have equal competence (see Figure 7.3). By incorporating a juvenile preference for the non-preferred species, we can get diversity amplification to change to dilution (see Figure 7.7). As we increase the juvenile preference, we start to get an increase in the disease risk as the proportion of the non-preferred species increases. Again, a strong juvenile preference is functionally similar to having an increased species competence. Having equal host competence, a biting preference for one host species, and a juvenile stage preference for the non-preferred species is functionally similar to the case of dilution when there is a preference for the less competent species and the disease risk

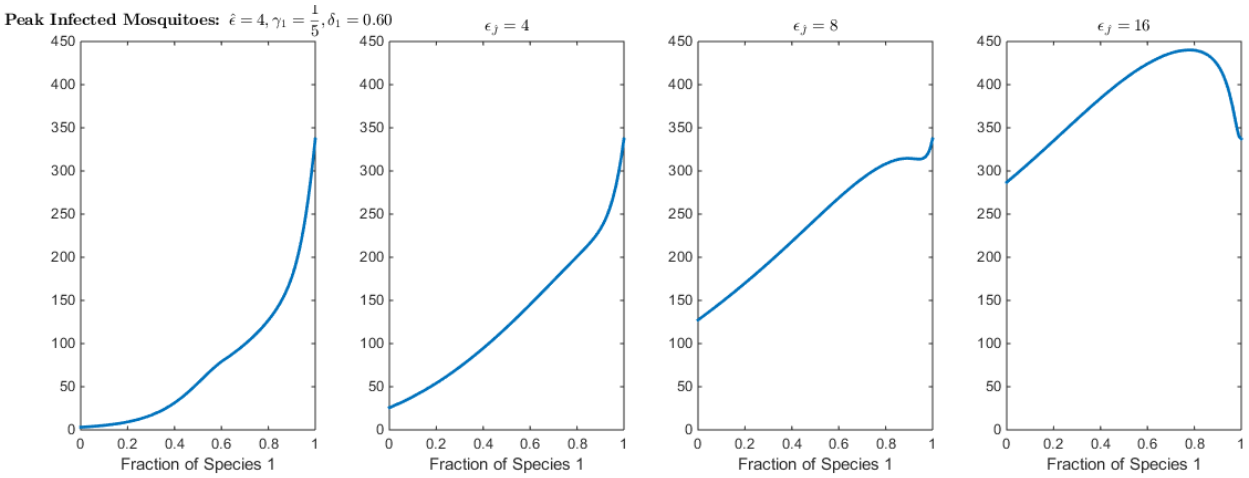

Figure 7.6: As we increase the strength of the juvenile preference for the preferred, less competent species (species 2), disease risk turns from dilution to diversity amplification. The strong juvenile preference for species 2 is functionally similar to an increase in host competence. All competence parameters are assumed to be as in Figure 7.2.

increases monotonically with as the abundance of the more competent species increases (see Figure 7.2).

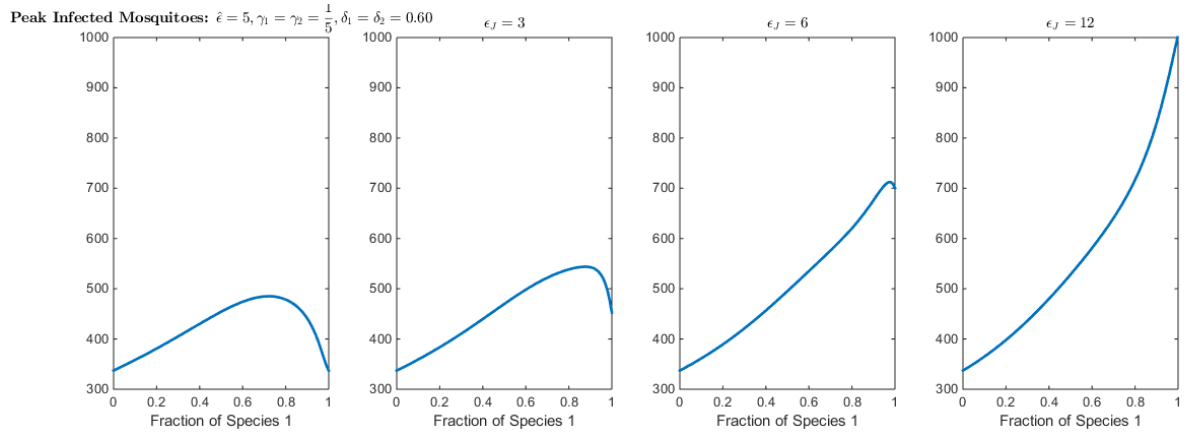

Figure 7.7: As we increase the juvenile preference for the non-preferred species (species 1), diversity amplification becomes dilution. In this case, juvenile preference is acting functionally similar to competence. Rather than disease risk being a non-monotonic function of species proportion, disease risk now increases monotonically as the proportion of the species with the juvenile preference increases. All competence parameters are assumed to be as in Figure 7.3.

The second case where Miller and Huppert found diversity amplification was when the preferred host species is also more competent (see Figure 7.4). We found diversity

amplification turned into dilution when we incorporated a juvenile stage preference for the less preferred species. As we increase the juvenile preference, the disease risk curves go from being non-monotonic and hump-shaped with a maximum at an interior point, to monotonically increasing with the proportion of the species that has the juvenile preference (see Figure 7.8). Here an increased juvenile stage preference is again functionally similar to increased competence. A biting preference for the more competent species and a strong juvenile preference for the less competent species is functionally similar to the case of dilution where there is a preference for the less competent species and disease risk increases monotonically as the abundance of the more competent species increases (see Figure 7.2).

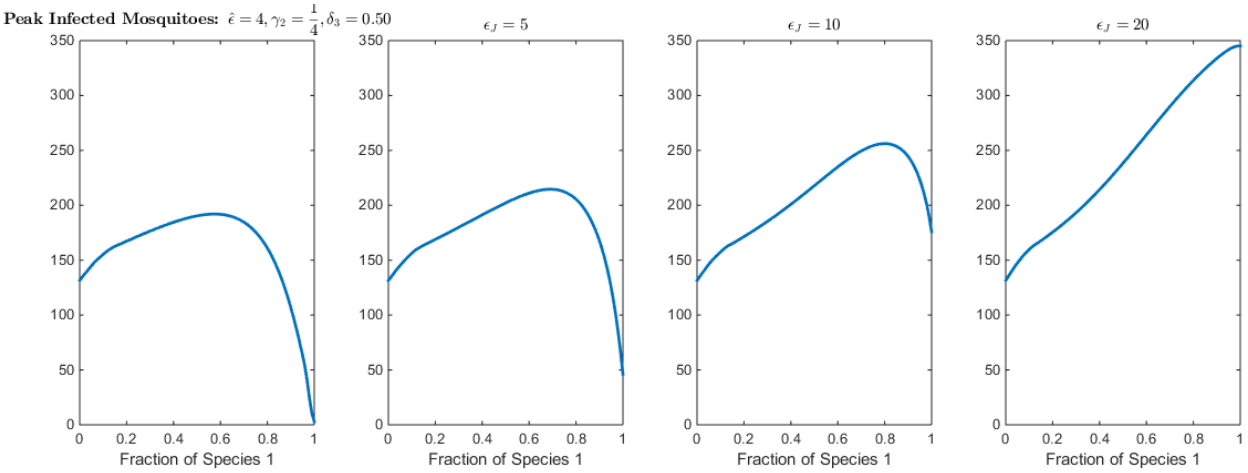

Figure 7.8: As we increase the juvenile preference for the non-preferred species (species 1), diversity amplification becomes dilution. Juvenile preference is acting functionally similar to competence. A strong enough juvenile preference for the non-preferred species (species 1) is outweighing the increased competence for the preferred species (species 2). Rather than disease risk being a non-monotonic function of species proportion, disease risk now increases monotonically as the proportion of the species with the juvenile preference (species 1) increases. All competence parameters are assumed to be as in Figure 7.4.

7.4 Discussion

While the dilution effect has been widely studied, Miller and Huppert were the first to analyze and create a mechanism for diversity amplification [19]. This is important because between dilution and diversity amplification they are able to account for the conflicting patterns that occur in nature. The model they used was a two species model with no stage-structure, and as such all of the conditions resulting in dilution and diversity amplification are given in terms of species preference and competence. Using our stage-structured, two species model, we were able to reproduce their results and verify that in cases of dilution, disease risk (for us, measured in total infectious and peak infectious vectors) increases with the proportion of the more competent host, achieving its maximum when only one host species is present, whereas in cases of diversity amplification, disease risk is a non-monotonic function of species proportion and achieves a maximum at an interior point where both host species are present.

Since Miller and Huppert's model did not incorporate stage-structure, we examined the effects of incorporating juvenile stage preferences on their results. We found that by incorporating a juvenile stage preference, we were able to change situations from dilution to diversity amplification and vice versa. We were able to conclude that increased juvenile stage preference acts similarly to increased competence in the sense that both are mechanisms for increased disease transmission, and as such both are important mechanisms for determining the effect of increased species diversity on the disease risk of a community.

Chapter 8

Conclusions and Future Work

Previous studies of WNV have examined the dynamics of single species stage-structured models [16, 17, 20], and models with two host species and no stage-structure [19, 23, 25]. To our knowledge our model is the first to incorporate multiple host species and stages.

We also incorporate variable species and stage specific biting rates of vectors on hosts. We show the differences between a single species model with weighted average parameters and a two species model, we investigate the difference between increased biting rates on host species and increased biting rates on host stages, and the effect of incorporating community composition and stage preference on the mechanisms of dilution and diversity amplification as proposed by Miller and Huppert.

We find that the general assumption of weighted average parameters on a single species model is an inadequate representation of the dynamics that can occur using a two species model. The only cases where it is appropriate are the cases in which both host species parameters are equal, regardless of community composition. In all other cases, this assumption results in either an overestimate or underestimate of the intensity of WNV transmission.

Investigation of the two species model shows that regardless of species competence and nesting parameters, an increased biting rate on juveniles always results in the most

intense WNV transmission except for one case. The one case where increased host species biting rates results in a more intense WNV transmission than increased juvenile biting rates is when the preferred species is at its maximum competence with recovery rate $\gamma_1 = \frac{1}{10}$ and infectivity $\delta_1 = 1$. However, even in this case there is only a slight increase in WNV transmission over the juvenile preference.

Further investigation showed that by incorporating community composition into our stage-structured, two species model, we were able to reproduce the results of Miller and Huppert. We verify that in cases of dilution, disease risk increases with the proportion of the more competent species, achieving its maximum when only a single host species is present, while in the cases of diversity amplification, disease risk is a non-monotonic function of species proportion and achieves its maximum at an interior point when both host species are present in the community. Incorporating stage-structure onto their results, we found that by adding increased biting rates on juveniles, in almost every case we were able to change the outcome from diversity amplification to dilution and vice versa. We conclude that increased biting rates on juveniles is functionally similar to competence and both are important mechanisms for determining the effect of species diversity on the disease risk of a community.

The possible future work for this model is extensive. Our model only simulates a single season. However, the percentage of susceptible verus recovered hosts at the end of a season will likely influence initial conditions for the next season. Also, in our model we assume that vector bites are first distributed among host species and then host stages (juveniles and adults). It would be of interest to see the effects of a simultaneous choice, where bites can be distributed to any stage of either species. Future directions also include a more mathematically rigorous description of the conditions for dilution and diversity amplification.

Bibliography

[1] Ahmed Abdelrazec, Suzanne Lenhart, and Huaiping Zhu. Transmission dynamics of *West Nile virus* in mosquitoes and corvids and non-corvids. *Journal of Mathematical Biology*, 68(6):1553–1582, 2014.

[2] Norman TJ Bailey. *The mathematical theory of infectious diseases and its applications*. Charles Griffin & Company Ltd, 5a Crendon Street, High Wycombe, Bucks HP13 6LE., 1975.

[3] John S Blackmore and Richard P Dow. Differential feeding of *Culex tarsalis* on nestling and adult birds. *Mosq News*, 18(1):15–17, 1958.

[4] Christopher P Brooks and Haimeng Zhang. A null model of community disassembly effects on vector-borne disease risk. *Journal of theoretical biology*, 264(3):866–873, 2010.

[5] KA Caillouët, AE Riggan, LP Bulluck, JC Carlson, and RT Sabo. Nesting bird host funnel increases mosquito-bird contact rate. *Journal of Medical Entomology*, 50(2):462–466, 2013.

[6] Centers for Disease Control and Prevention (CDC): *West Nile virus*. http://www.cdc.gov/westnile, 2015.

[7] Klaus Dietz. The estimation of the basic reproduction number for infectious diseases. *Statistical methods in medical research*, 2(1):23–41, 1993.

[8] John D Edman and Thomas W Scott. Host defensive behaviour and the feeding success of mosquitoes. *International Journal of Tropical Insect Science*, 8(4-5-6):617–622, 1987.

[9] VO Ezenwa, MS Godsey, RJ King, and SC Guptill. Avian diversity and *West Nile virus*: testing associations between biodiversity and infectious disease risk. *Proceedings of the Royal Society B: Biological Sciences*, 273(1582):109–117, 2006.

[10] Gabriel L Hamer, Uriel D Kitron, Tony L Goldberg, Jeffrey D Brawn, Scott R Loss, Marilyn O Ruiz, Daniel B Hayes, and Edward D Walker. Host selection by *Culex pipiens* mosquitoes and *West Nile virus* amplification. *The American journal of tropical medicine and hygiene*, 80(2):268–278, 2009.

[11] HW Kale, JD Edman, and LA Webber. Effect of behavior and age of individual ciconiiform birds on mosquito feeding success. *Mosq News*, 32(3):343–350, 1972.

[12] William O Kermack and Anderson G McKendrick. A contribution to the mathematical theory of epidemics. In *Proceedings of the Royal Society of London A: mathematical, physical and engineering sciences*, volume 115, pages 700–721. The Royal Society, 1927.

[13] A. Marm Kilpatrick, Laura D Kramer, Matthew J Jones, Peter P Marra, and Peter Daszak. *West Nile virus* epidemics in North America are driven by shifts in mosquito feeding behavior. *PLoS Biol*, 4(4):606–610, 2006.

[14] A Marm Kilpatrick, Shannon L LaDeau, and Peter P Marra. Ecology of *West Nile virus* transmission and its impact on birds in the western hemisphere. *The Auk*, 124(4):1121–1136, 2007.

[15] N Komar, S Langevin, S Hinten, N Nemeth, E Edwards, D Hettler, B Davis, R Bowen, and M Bunning. Experimental infection of North American birds with the New York 1999 strain of *West Nile virus*. *Emerging Infectious Diseases*, 9(3):311, 2003.

[16] CC Lord and JF Day. Simulation studies of *St. Louis encephalitis* and *West Nile virus*: the impact of bird mortality. *Vector Borne and Zoonotic Diseases*, 1(4):317–329, 2001.

[17] CC Lord and JF Day. Simulation studies of *St. Louis encephalitis virus* in South Florida. *Vector Borne and Zoonotic Diseases*, 1(4):299–315, 2001.

[18] Scott R Loss, Gabriel L Hamer, Edward D Walker, Marilyn O Ruiz, Tony L Goldberg, Uriel D Kitron, and Jeffrey D Brawn. Avian host community structure and prevalence of *West Nile virus* in chicago, illinois. *Oecologia*, 159(2):415–424, 2009.

[19] E Miller and A Huppert. The effects of host diversity on vector-borne disease: the conditions under which diversity will amplify or dilute the disease risk. *PloS one*, 8(11):e80279, 2013.

[20] Suzanne L Robertson and Kevin A Caillouët. A host stage-structured model of enzootic *West Nile virus* transmission to explore the effect of avian stage-dependent exposure to vectors. *Journal of Theoretical Biology*, 399:33–42, 2016.

[21] Kenneth A Schmidt and Richard S Ostfeld. Biodiversity and the dilution effect in disease ecology. *Ecology*, 82(3):609–619, 2001.

[22] Thomas W Scott and John D Edman. Effects of avian host age and arbovirus infection on mosquito attraction and blood-feeding success. *Bird-Parasite Interactions: Ecology, Evolution and Behaviour*, pages 179–204, 1991.

[23] JE Simpson, PJ Hurtado, J Medlock, G Molaei, TG Andreadis, AP Galvani, and MA Diuk-Wasser. Vector host-feeding preferences drive transmission of multi-host pathogens: *West Nile virus* as a model system. *Proceedings of the Royal Society B: Biological Sciences*, 279(1730):925–933, 2012.

[24] RS Unnasch, T Sprenger, CR Katholi, EW Cupp, GE Hill, and TR Unnasch. A dy-

namic transmission model of *Eastern Equine Encephalitis virus*. *Ecological Modelling*, 192(3):425–440, 2006.

[25] MJ Wonham, T de Camino-Beck, and MA Lewis. An epidemiological model for *West Nile virus*: invasion analysis and control applications. *Proceedings of the Royal Society of London. Series B: Biological Sciences*, 271(1538):501–507, 2004.

[26] Word Health Organization (WHO): *West Nile virus*. http://www.who.int/mediacentre/factsheets/fs354/en/, 2011.

Vita

Taylor Ann Beebe was born August 15, 1990 in Olympia, Washington. After obtaining her High School diploma from Capital High School she moved to Emmitsburg, Maryland to attend Mount Saint Mary's University on an NCAA Division I softball scholarship, where she received a Bachelor of Science Degree in Mathematics in 2012.

After taking some time off to work, she enrolled at Virginia Commonwealth University to earn her Master's Degree in Applied Mathematics. While a student at VCU she was a graduate teaching assistant in differential equations and taught algebra with applications to undergraduates. She will be moving to Washington, D.C. after graduation to pursue a career in industry.