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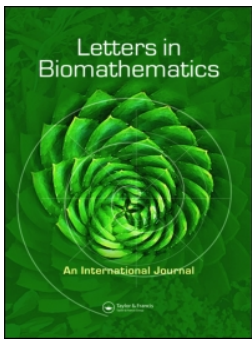
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A two-species stage-structured model for *West Nile virus* transmission

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ABSTRACT

We develop a host–vector model of *West Nile virus* (WNV) transmission that incorporates multiple avian host species as well as host stage-structure (juvenile and adult stages), allowing for both species-specific and stage-specific biting rates of vectors on hosts. We use this ordinary differential equation model to explore WNV transmission dynamics that occur between vectors and multiple structured host populations as a result of heterogeneous biting rates on species and/or life stages. Our analysis shows that increased exposure of juvenile hosts generally results in larger outbreaks of WNV infectious vectors when compared to differential host species exposure. We also find that increased juvenile exposure is an important mechanism for determining the effect of species diversity on the disease risk of a community.

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Epidemiology; vector-borne disease; ordinary differential equation; heterogeneity

1. Introduction

West Nile virus (WNV) was introduced to New York in 1999 and has subsequently spread throughout most of the United States and North America (Kilpatrick, LaDeau, & Marra, 2007; World Health Organization [WHO], 2011). While the majority of human infections are consistently reported in the summer months, the number of cases varies significantly both from year to year and with geographical location (Centers for Disease Control and Prevention [CDC], 2015). WNV persists through a host–vector–host interaction cycle, with the principal vectors being mosquitoes. While many types of hosts, including humans, can become infectious with WNV, the primary reservoir hosts are birds. WNV has been detected in more than 250 different avian species (WHO, 2011), and experimental studies have shown that these species vary widely in competence (a measure of a host’s ability to contract and transmit disease) (Kilpatrick et al., 2007).

There is also evidence of increased biting rates on some species of avian hosts relative to their abundance (Hamer et al., 2009; Kilpatrick, Kramer, Jones, Marra, & Daszak, 2006). This may be a result of a vector preference for certain species, but might also be due to increased availability or accessibility of these hosts. Some species of birds could be more exposed to mosquito bites depending on their nesting type (i.e. cavity vs. open nest) or

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nest height. Heterogeneity in competence and feeding preferences is known to play an important role in structuring WNV transmission (Ezenwa, Godsey, King, & Guptill, 2006; Kilpatrick et al., 2006; Simpson et al., 2012), and it can also affect the relationship between species diversity and disease risk (Cruz-Pacheco, Esteva, & Vargas, 2012; Miller & Huppert, 2013). Miller & Huppert (2013) developed a mathematical host–vector model with two host species and examined the effects of different assumptions on host competence and species-specific vector feeding preferences on transmission. They determined conditions for which increased species diversity can result in either dilution or amplification of vector-borne disease transmission.

Vector biting rates may also vary with host age. Increased exposure of younger birds, especially in the first week or two after hatching, may occur as a result of their minimal feather coverage or inability to defend against mosquitos (Blackmore & Dow, 1958; Edman & Scott, 1987; Scott & Edman, 1991). Increased vector biting rates on juvenile hosts relative to adults can result in more intense transmission even when competence is equal among stages (Robertson & Caillouët, 2016).

There have been a number of recent mathematical models for WNV incorporating either multiple host species (Abdelrazec, Lenhart, & Zhu, 2014; Bergsman, Hyman, & Manore, 2016; Miller & Huppert, 2013; Simpson et al., 2012) or multiple host life stages (Lord & Day, 2001a, 2001b; Robertson & Caillouët, 2016; Simpson et al., 2012; Unnasch et al., 2006). The model introduced in this paper incorporates both between-host and within-host heterogeneity. We model two avian host species each with juvenile and adult stages, allowing for both species-specific and stage-specific biting rates of vectors on hosts. We use this model to investigate how vector feeding preferences for host species compares to preferences for host stages, and explore the interaction of the two mechanisms. We also show how host stage-structure and stage-specific vector feeding preferences can alter how community composition affects disease risk.

2. Model development

In this section, we develop a novel host–vector model for WNV (Figure 1) that incorporates two host species each with stage-structure, and allows for both species-specific and stage-specific biting rates of vectors on hosts. We have modified the single-species, three-stage model of Robertson and Caillouët (2016) to include two distinct host species each with two age classes, juvenile and adult.

We use ordinary differential equations to model the number of susceptible, infectious, and recovered birds over time in each of the juvenile and adult stages for each species. In order to differentiate between the two host species, variables and parameters pertaining to the second host species will be denoted with a $\hat{}$ symbol. We also model susceptible, latent, and infectious mosquitoes over time. The complete model is composed of 15 differential equations (12 host equations and 3 vector equations) and defined as follows:

$$\begin{aligned}\frac{dJ_S}{dt} &= b(t) - \alpha\alpha_J\beta_J M_I \frac{J_S}{J_T} - (m_J + \mu_J)J_S \\ \frac{dJ_I}{dt} &= \alpha\alpha_J\beta_J M_I \frac{J_S}{J_T} - (m_J + \mu_J + \gamma_J + \nu_J)J_I\end{aligned}$$

$$\begin{aligned}
 \frac{dJ_R}{dt} &= \gamma_J J_I - (m_J + \mu_J) J_R \\
 \frac{dA_S}{dt} &= -a\alpha\alpha_A\beta_A M_I \frac{A_S}{A_T} + m_J J_S - \mu_A A_S \\
 \frac{dA_I}{dt} &= a\alpha\alpha_A\beta_A M_I \frac{A_S}{A_T} + m_J J_I - (\mu_A + \gamma_A + \nu_A) A_I \\
 \frac{dA_R}{dt} &= \gamma_A A_I - \mu_A A_R + m_J J_R \\
 \frac{d\hat{J}_S}{dt} &= \hat{b}(t) - a\hat{\alpha}\hat{\alpha}_j\beta_j M_I \frac{\hat{J}_S}{\hat{J}_T} - (m_j + \mu_j)\hat{J}_S \\
 \frac{d\hat{J}_I}{dt} &= a\hat{\alpha}\hat{\alpha}_j\beta_j M_I \frac{\hat{J}_S}{\hat{J}_T} - (m_j + \mu_j + \gamma_j + \nu_j)\hat{J}_I \\
 \frac{d\hat{J}_R}{dt} &= \gamma_j \hat{J}_I - (m_j + \mu_j)\hat{J}_R \\
 \frac{d\hat{A}_S}{dt} &= -a\hat{\alpha}\hat{\alpha}_A\beta_A M_I \frac{\hat{A}_S}{\hat{A}_T} + m_j \hat{J}_S - \mu_{\hat{A}} \hat{A}_S \\
 \frac{d\hat{A}_I}{dt} &= a\hat{\alpha}\hat{\alpha}_A\beta_A M_I \frac{\hat{A}_S}{\hat{A}_T} + m_j \hat{J}_I - (\mu_{\hat{A}} + \gamma_{\hat{A}} + \nu_{\hat{A}})\hat{A}_I \\
 \frac{d\hat{A}_R}{dt} &= \gamma_{\hat{A}} \hat{A}_I - \mu_{\hat{A}} \hat{A}_R + m_j \hat{J}_R \\
 \frac{dM_S}{dt} &= rM_T \left(1 - \frac{M_T}{K}\right) - a \left(\alpha\alpha_j\delta_j \frac{J_I}{J_T} + \alpha\alpha_A\delta_A \frac{A_I}{A_T} + \hat{\alpha}\hat{\alpha}_j\delta_j \frac{\hat{J}_I}{\hat{J}_T} + \hat{\alpha}\hat{\alpha}_A\delta_{\hat{A}} \frac{\hat{A}_I}{\hat{A}_T} \right) M_S \\
 &\quad - \mu_M(t)M_S \\
 \frac{dM_L}{dt} &= a \left(\alpha\alpha_j\delta_j \frac{J_I}{J_T} + \alpha\alpha_A\delta_A \frac{A_I}{A_T} + \hat{\alpha}\hat{\alpha}_j\delta_j \frac{\hat{J}_I}{\hat{J}_T} + \hat{\alpha}\hat{\alpha}_A\delta_{\hat{A}} \frac{\hat{A}_I}{\hat{A}_T} \right) M_S - kM_L - \mu_M(t)M_L \\
 \frac{dM_I}{dt} &= kM_L - \mu_M(t)M_I
 \end{aligned} \tag{1}$$

We assume all hosts modelled are competent, or able to contract and spread WNV. Hosts are born into the susceptible juvenile class of species 1 and 2 at rate $b(t)$ and $\hat{b}(t)$, respectively, where

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

and

$$\hat{b}(t) = \frac{\hat{f}}{\hat{\sigma}\sqrt{2\pi}} \exp\left(\frac{-(\hat{q}-t)^2}{2\hat{\sigma}^2}\right). \tag{3}$$

We model the juvenile recruitment curves for species 1 and 2 by scaled Gaussian distributions (Lord & Day, 2001b; Robertson & Caillouët, 2016) with means of q and \hat{q} , variance σ and $\hat{\sigma}$, and scaling factors f and \hat{f} , respectively.

Juvenile hosts of either species can either move into another class (susceptible, infectious, recovered) within their stage or mature into the adult stage of their current class at rate m_j or m_j , where maturation rates are the inverse of the mean duration of the juvenile

Table 1. West Nile virus model (1) baseline parameters.

Parameter	Description	Baseline
a	Biting rate on competent avian hosts	.133 bites/mosquito/day
$\epsilon, \hat{\epsilon}$	Host species exposure coefficients	varies
ϵ_J, ϵ_J	Juvenile exposure coefficients	varies
$\epsilon_A, \epsilon_{\hat{A}}$	Adult exposure coefficients	varies
m_J, m_J	Juvenile maturation rates	1/14 days ⁻¹
μ_J, μ_J	Juvenile natural mortality rates	.0014 days ⁻¹
$\mu_A, \mu_{\hat{A}}$	Adult natural mortality rates	.0014 days ⁻¹
β_J, β_J	Juvenile susceptibilities	1
$\beta_A, \beta_{\hat{A}}$	Adult susceptibilities	1
γ_J, γ_J	Juvenile recovery rates	1/3 days ⁻¹
$\gamma_A, \gamma_{\hat{A}}$	Adult recovery rates	1/3 days ⁻¹
δ_J, δ_J	Juvenile infectivities	.36
$\delta_A, \delta_{\hat{A}}$	Adult infectivities	.36
ν_J, ν_J	Juvenile virulences	.1 days ⁻¹
$\nu_A, \nu_{\hat{A}}$	Adult virulences	.1 days ⁻¹
q, \hat{q}	Juvenile recruitment curve (JRC) means	Day 158.17
$\sigma, \hat{\sigma}$	JRC standard deviations	11.4
f, \hat{f}	JRC scaling factors	285
A_0, \hat{A}_0	Number of adults at start of season	150
r	Mosquito per capita birthrate	.537 days ⁻¹
K	Mosquito carrying capacity (larval)	15000
μ_M	Vector mortality rate	.096 days ⁻¹
k	Virus incubation period	.106 days ⁻¹
z	Start of mosquito growth	Day 115

Notes: For host-specific parameters, parameters without a ^{hat} symbol refer to species 1, and those with a ^{hat} refer to species 2. All parameter values are as in Robertson and Caillouët (2016).

stage. Susceptible hosts (either juvenile or adult) can leave the susceptible class and move into the infectious class, if bitten by an infectious mosquito (Figure 1). The parameter a gives the biting rate of mosquitoes on avian hosts. The probability that a bite from an infectious mosquito results in a susceptible bird becoming infectious may vary by species and/or stage, and is given by β_i , for $i = J, A, \hat{J}, \hat{A}$.

We assume that the total bites taken by a mosquito are first divided among the two host species, with a fraction $\alpha(t)$ on host species 1 and the remainder ($\hat{\alpha}(t) = 1 - \alpha(t)$) on host species 2 where:

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

and

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

The subscript T denotes total population (i.e. $J_T(t)$ is the total juvenile host species 1 population at time t) and is given by the sum of the susceptible, infectious, and recovered classes (i.e. $J_T(t) = J_S(t) + J_I(t) + J_R(t)$). The constants ϵ and $\hat{\epsilon}$ are the species exposure coefficients and determine how frequently one species is bitten relative to the other. For example, if $\epsilon = \hat{\epsilon}$ then both species are being bitten in proportion to their abundance in

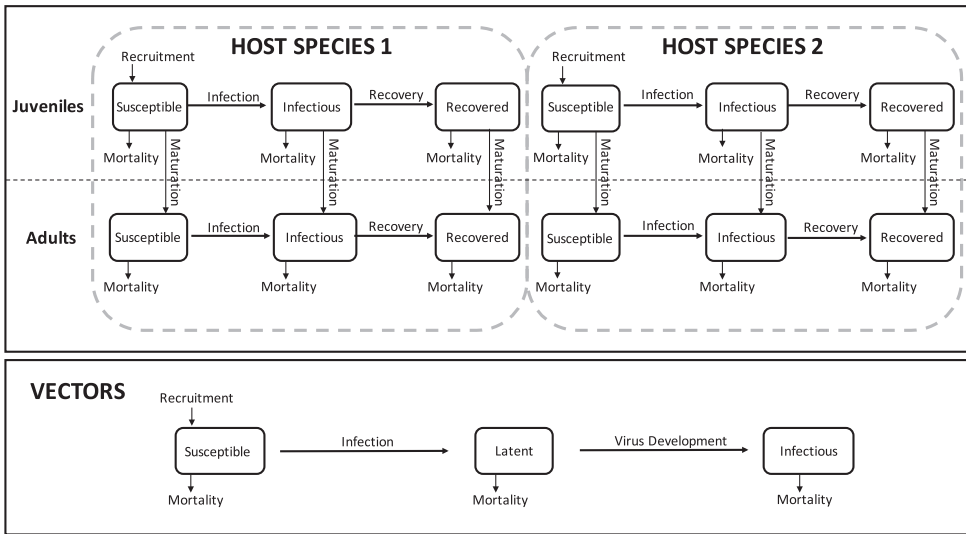


Figure 1. *West Nile virus* multi-host stage-structured model schematic. Note: Transmission of WNV occurs when an infectious vector bites a susceptible juvenile or adult host of species 1 or 2, or when a susceptible vector bites an infectious juvenile or adult host of species 1 or 2.

the population and the per capita biting rate is equal for all hosts. If $\epsilon > \hat{\epsilon}$, then the per capita biting rate is higher for species 1 than species 2; we call this a vector preference for species 1. If $\epsilon < \hat{\epsilon}$, then there is a vector preference for species 2. Note that while the exposure coefficients ϵ and $\hat{\epsilon}$ are constant, as they are assumed to be a characteristic of each species, the proportion of bites going to each species ($\alpha(t)$ and $\hat{\alpha}(t)$) changes throughout the season depending on the total population of each species at any given time.

The bites on each host species are subsequently distributed among each of the juvenile and adult stages of that species with a fraction going to juveniles ($\alpha_J, \alpha_{\hat{J}}$) and the remainder going to adults ($\alpha_A, \alpha_{\hat{A}}$) according to:

$$\alpha_J(t) = \frac{\epsilon_J J_T(t)}{\epsilon_J J_T(t) + \epsilon_A A_T(t)} \tag{4}$$

$$\alpha_A(t) = \frac{\epsilon_A A_T(t)}{\epsilon_J J_T(t) + \epsilon_A A_T(t)} \tag{5}$$

$$\alpha_{\hat{J}}(t) = \frac{\epsilon_{\hat{J}} \hat{J}_T(t)}{\epsilon_{\hat{J}} \hat{J}_T(t) + \epsilon_{\hat{A}} \hat{A}_T(t)} \tag{6}$$

$$\alpha_{\hat{A}}(t) = \frac{\epsilon_{\hat{A}} \hat{A}_T(t)}{\epsilon_{\hat{J}} \hat{J}_T(t) + \epsilon_{\hat{A}} \hat{A}_T(t)}. \tag{7}$$

The constants $\epsilon_J, \epsilon_A, \epsilon_{\hat{J}}$ and $\epsilon_{\hat{A}}$ are the exposure coefficients for each stage of each species and determine how frequently a stage is bitten relative to the other stage for that species. If $\epsilon_J = \epsilon_A$ and $\epsilon_{\hat{J}} = \epsilon_{\hat{A}}$ then all stages are bitten in proportion to their abundance in the population and all hosts receive the same number of bites. If $\epsilon_J > \epsilon_A$ or $\epsilon_{\hat{J}} > \epsilon_{\hat{A}}$, then the juveniles of species 1 or 2, respectively, are being bitten more than the adults of their species. We will refer to increased juvenile exposure coefficients as a juvenile stage

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 .

Therefore, $a\alpha\alpha_J$ is the rate at which juvenile hosts of species 1 are bitten by infectious vectors (M_I). A fraction $\frac{J_S}{J_T}$ of these hosts are susceptible, and a bite results in disease transmission with probability β_J . Thus $a\alpha\alpha_J\beta_J\frac{J_S}{J_T}M_I$ is the rate at which juveniles of host species 1 (J_S) become infectious (J_I). Similar terms describe the rate at which the other types of susceptible hosts become infectious. While all hosts have a natural mortality rate μ_i , $i = J, A, \hat{J}, \hat{A}$, infectious hosts are subject to increased disease-related mortality at rate ν_i , $i = J, A, \hat{J}, \hat{A}$. Infectious hosts move into the recovered class at rate γ_i , $i = J, A, \hat{J}, \hat{A}$.

For the vectors, we model susceptible (M_S), latent (M_L) and, infectious (M_I) mosquitoes over time. We assume that all mosquitoes are born susceptible (no vertical transmission). Mosquitoes are born into the susceptible class at density-dependent rate $r\left(1 - \frac{M_T}{K}\right)$. Vectors can move from the susceptible to latent class by becoming infected upon biting any infectious host. Vectors move from the latent to the infectious class (where they can infect hosts) at rate k (the inverse of the duration of the extrinsic incubation period), where they remain until death. All vectors are subject to the density-independent, time-dependent mortality rate $\mu_M(t)$ defined as:

$$\mu_M(t) = \begin{cases} \mu_M & t \leq 240 \\ \mu_M + 0.01(t - 240) & t > 240. \end{cases} \quad (8)$$

Mortality is constant until day 240 and then increases linearly to account for the observed late-season decline in mosquito populations. Note model (1) is non-autonomous, with seasonal juvenile reproduction and mosquito mortality rates. Therefore, there is no disease-free equilibrium with both stages present in the population.

3. Comparison of single and two species stage-structured models

When modelling the spread of disease in a population consisting of multiple host species, a common simplifying assumption is to ignore species diversity and instead model a single representative host species by using parameter values averaged over all hosts. Recall that there is a great deal of diversity in competence among avian species, where competence is calculated as the product of susceptibility (per bite probability of transmission from mosquito to bird), infectivity (per bite probability of transmission from bird to mosquito), and the expected duration of infection (given by the inverse of the recovery rate) (Komar et al., 2003).

In this section, we investigate the consequences of averaging across host species when species may also have within-host heterogeneity in the form of stage-structure and stage-dependent exposure to mosquitoes. We compare the WNV transmission dynamics of our two-species model (1), where each species has stage-specific exposure coefficients, to those resulting from a single-species model where the juvenile and adult exposure coefficients are given by the average values weighted by species abundance in the community.

The single-species model is defined as follows:

$$\begin{aligned}
 \frac{dJ_S}{dt} &= b(t) - a\alpha_J\beta_J M_I \frac{J_S}{J_T} - (m_J + \mu_J)J_S \\
 \frac{dJ_I}{dt} &= a\alpha_J\beta_J M_I \frac{J_S}{J_T} - (m_J + \mu_J + \gamma_J + \nu_J)J_I \\
 \frac{dJ_R}{dt} &= \gamma_J J_I - (m_J + \mu_J)J_R \\
 \frac{dA_S}{dt} &= -a\alpha_A\beta_A M_I \frac{A_S}{A_T} + m_J J_S - \mu_A A_S \\
 \frac{dA_I}{dt} &= a\alpha_A\beta_A M_I \frac{A_S}{A_T} + m_J J_I - (\mu_A + \gamma_A + \nu_A)A_I \\
 \frac{dA_R}{dt} &= \gamma_A A_I - \mu_A A_R + m_J J_R \\
 \frac{dM_S}{dt} &= rM_T \left(1 - \frac{M_T}{K}\right) - a \left(\alpha_J \delta_J \frac{J_I}{J_T} + \alpha_A \delta_A \frac{A_I}{A_T}\right) M_S - \mu_M(t)M_S \\
 \frac{dM_L}{dt} &= a \left(\alpha_J \delta_J \frac{J_I}{J_T} + \alpha_A \delta_A \frac{A_I}{A_T}\right) M_S - kM_L - \mu_M(t)M_L \\
 \frac{dM_I}{dt} &= kM_L - \mu_M(t)M_I
 \end{aligned} \tag{9}$$

where the juvenile recruitment curve $b(t)$ is as in Equation (2), $\alpha_J(t)$ and $\alpha_A(t)$ are as in Equations (4) and (5), and $\mu_M(t)$ is given by Equation (8). All baseline parameter values are equivalent to those of the corresponding parameters in the two-species model given in Table 1, except for $A_0 = 300$ and $f = 570$ to keep the initial bird population and nestlings per adult equal across models.

All model analysis is done through simulations in MATLAB. Each simulation will simulate a single season beginning at Julian day 100. Each model simulation will start with 300 total adult birds, 70% of whom are susceptible and 30% recovered. Mosquitoes are introduced at Julian day 115, with 99 susceptible and 1 infectious. Since our models are non-autonomous, we will use the peak number of infectious vectors over the course of a season as model output. We begin by considering the case when the two host species differ only in juvenile exposure coefficients. Note that if juvenile preferences are equal the two species are identical and the two-species model simply reduces to the single-species model. We set $\epsilon_A = \epsilon_{\hat{A}} = \epsilon_J = 1$ and without loss of generality incorporate a preference for juveniles of species 1 ($\epsilon_J > 1$); since species 1 and species 2 are otherwise identical, if they are also equal in abundance then a preference for juveniles of species 2 will yield the same results. If $\epsilon_J = 5$, the weighted average juvenile exposure coefficient for the single-species model is $\bar{\epsilon}_J = 3$, and if $\epsilon_J = 15$, $\bar{\epsilon}_J = 8$. The infectious mosquito curve for the single and two-species models are shown for these parameter sets in Figure 2. The single-species model underestimates the peak infectious vectors compared to the two-species model when juvenile preference is low, but overestimates for larger values of ϵ_J .

Next we consider the case where the two host species differ in competence, specifically infectivity (probability of bird-mosquito transmission per bite) and recovery rate. We leave species 1 at its baseline recovery rate ($\gamma_J = \gamma_A = \frac{1}{3}$) and infectivity ($\delta_J = \delta_A = .36$), and increase the competence of all stages of host species 2 by shortening the recovery rate

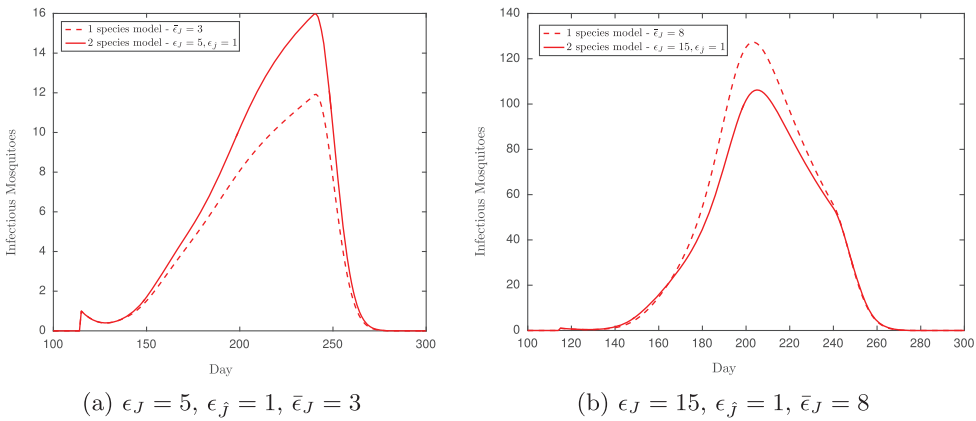


Figure 2. Comparison of infectious mosquito temporal dynamics for models (1) and (9).

Notes: Both species are at baseline (equal) competence with no species feeding preferences. There is no stage feeding preference for species 2. Model (9) underestimates transmission when juveniles of species 1 are 5 times as exposed as adults, and overestimates transmission when the juveniles of species 1 are 15 times more exposed than adults.

to $\gamma_j = \gamma_A = \frac{1}{6}$, doubling the mean duration of infection, and increasing infectivity to $\delta_j = \delta_A = .72$. Assuming each host species comprises 50% of the total host community, the competence parameter values for the single-species model are $\bar{\gamma}_J = \bar{\gamma}_A = .25$ and $\bar{\delta}_J = \bar{\delta}_A = .54$. We compare the predictions of the two-species and single-species models for different scenarios of juvenile preferences.

The single-species model underestimates transmission when neither species has a juvenile preference, when both species have a juvenile preference, and when the more competent species has a juvenile preference (Figure 3). However, when only the less competent species has a juvenile preference, the single-species model now overestimates the intensity of disease transmission.

Comparing the WNV transmission dynamics from the two-species model with those of the single-species model, we see that unless the two species are identical, the single-species model with weighted average parameter values will either over or underestimate the intensity of WNV transmission. When there are no stage preferences or equal stage preferences, so species differ only in competence, peak transmission from the single-species model underestimates that of the heterogeneous two-species model. This is consistent with many studies finding that heterogeneity in transmission often increases levels of infection (Adler, 1992; Diekmann & Heesterbeek, 2000; Dushoff & Levin, 1995; Dye & Hasibeder, 1986; Robertson, Eisenberg, & Tien, 2013; Yorke, Hethcote, & Nold, 1978). Robertson and Caillouët (2016) found that heterogeneous biting rates on avian stage classes also resulted in more intense WNV outbreaks.

However, when the only difference between species is juvenile exposure, the averaged single-species model can actually overestimate transmission. When only one host species has a juvenile preference, the juveniles of only that species are bitten at an increased rate while both stages of the other equally competent species are being bitten in proportion to their abundance. This reduces the intensity of WNV transmission compared to the single-species model where juveniles of both species receive bites at increased rates compared to adults. When we have differential competence and an increased biting rate on the juveniles of the more competent species, the single-species model again underestimates the intensity

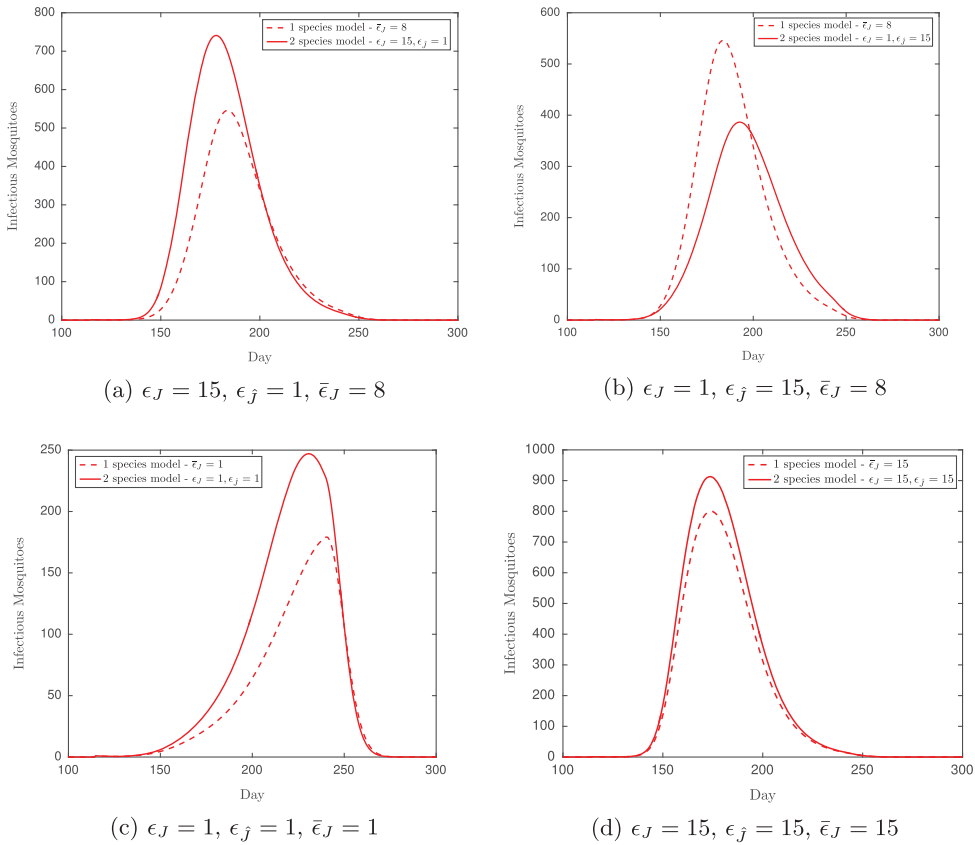


Figure 3. Comparison of infectious mosquito temporal dynamics for models (1) and (9). Notes: Species 1 has twice the infectivity and duration of infection as species 2 ($\gamma_1 = \gamma_A = \frac{1}{6}, \gamma_2 = \gamma_A = \frac{1}{3}, \bar{\gamma}_1 = \bar{\gamma}_A = \frac{1}{4}, \delta_1 = \delta_A = .72, \delta_2 = \delta_A = .36, \bar{\delta}_1 = \bar{\delta}_A = .54$) and there are no species feeding preferences. Results are shown for 4 stage preference scenarios: (a) 15x juvenile preference for species 1 and no stage preferences for species 2, (b) No stage preference for species 1 and 15x juvenile preference for species 2, (c) No stage preference for either species, and (d) 15x preference for both species.

of WNV transmission from the two-species model as it does when juvenile exposure coefficients are equal among species. However, increased biting rates on the juveniles of the less competent species results in the single-species model overestimating the intensity of transmission from the two-species model. We note that for each case where the single-species model with weighted average parameter values does not accurately describe the transmission dynamics that occur in the two-species model, we can adjust the average preference values to come close to the two-species model results but we can never match the infectious mosquito curve exactly.

4. Comparing species and stage preferences

A vector feeding preference for certain species has been shown to increase transmission if the preferred host is at least as competent as the alternative host (Miller & Huppert, 2013). A feeding preference for juveniles also increases transmission when juveniles are equal to adults in competence (Robertson & Caillouët, 2016). In this section, we compare

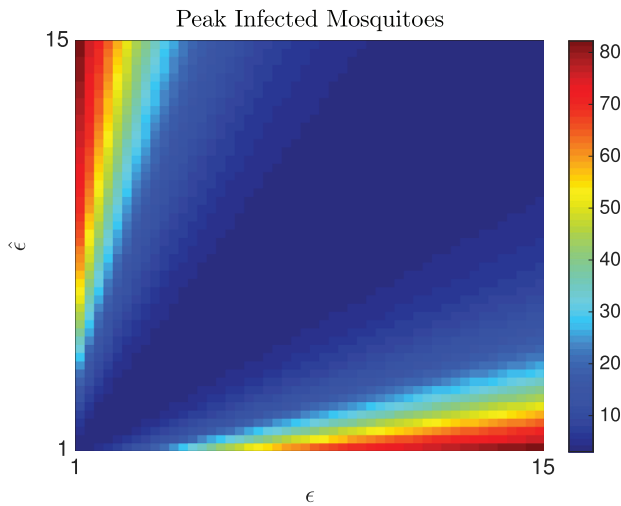


Figure 4. Shown is the peak number of infectious mosquitoes as a function of species 1 exposure coefficient ϵ and species 2 exposure coefficient $\hat{\epsilon}$.

Notes: WNV transmission increases with the ratio of $\epsilon/\hat{\epsilon}$ and $\hat{\epsilon}/\epsilon$. All parameters are as in Table 1 with no stage preferences.

the impact of differential feeding rates of vectors on host species and stages on WNV transmission.

First, we explore the effect of vector feeding preferences on different host species, keeping all other parameters besides species exposure coefficients equal. When examining the effect of host-species preferences, we vary the values of ϵ and $\hat{\epsilon}$ from 1 to 15. The peak number of infectious vectors are shown in Figure 4. There is symmetry about the diagonal since the two host populations are otherwise equal in parameters and abundance. If $\epsilon = \hat{\epsilon}$, species are bitten in proportion to their abundance in the community. We find that the most intense WNV transmission occurs when the ratio of exposure coefficients is maximized. ($\epsilon = 1, \hat{\epsilon} = 15$ or $\epsilon = 15, \hat{\epsilon} = 1$).

We next consider the effect of a vector feeding preference for a host life stage (either juvenile or adult) for both species with no species feeding preferences and all other parameter values kept at their respective baselines (Figure 5). We no longer see symmetry about the diagonal, as juveniles and adults differ in their relative abundance in the population as well as community susceptibility. Juveniles are born susceptible whereas adults may start the season recovered, and infectious or recovered juveniles may also mature into adults. Our analysis shows that increased adult exposure has little to no effect on WNV transmission when stages are equal in competence. As the ratio of juvenile to adult exposure coefficients increases, WNV transmission increases and peak transmission moves earlier in the season (not shown). Peak transmission occurs for $\epsilon_J/\epsilon_A = \epsilon_{\hat{J}}/\epsilon_{\hat{A}} = 15$; the peak number of infectious vectors for these parameters is over 4 times larger than the peak number of infectious vectors when there is a 15-fold difference in species exposure coefficients.

We now examine the consequences of both species and stage preferences in a community. We assume there is a feeding preference for one species, and we consider the case where the preferred species has a juvenile stage preference and the case where the non-preferred species has a juvenile stage preference. Recall the assumption of our model is that vectors first choose between the two species and then the two stages. Figure 6

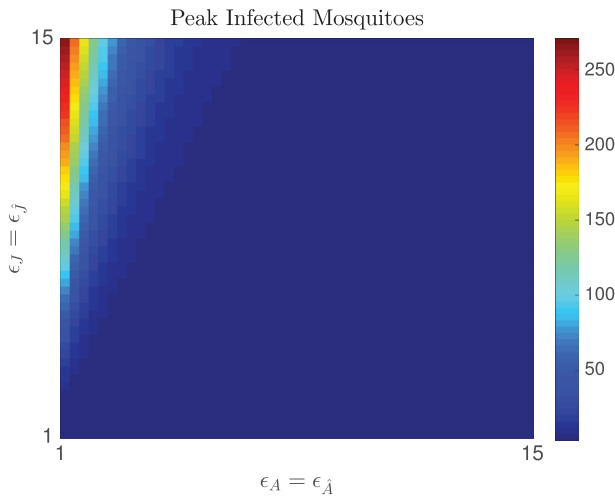


Figure 5. Shown is the peak number of infectious mosquitoes as a function of overall adult exposure coefficients $\epsilon_A = \epsilon_{\hat{A}}$ and overall juvenile exposure coefficients $\epsilon_J = \epsilon_{\hat{J}}$. Notes: Maximum transmission occurs when there is no adult preference ($\epsilon_A = \epsilon_{\hat{A}} = 1$) and a strong juvenile preference ($\epsilon_J = \epsilon_{\hat{J}} = 15$). All other parameters are as in Table 1 with no species preferences.

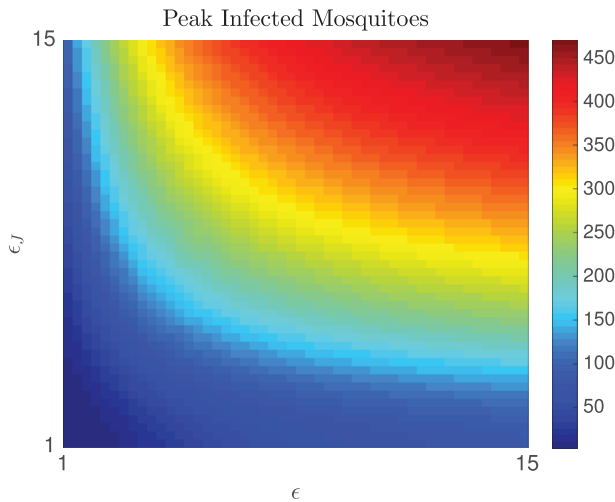


Figure 6. Shown is the peak number of infectious mosquitoes as a function of species 1 exposure coefficient ϵ and species 1 juvenile exposure coefficient ϵ_J . Notes: Maximum transmission occurs when both ϵ and ϵ_J are maximized. All other parameters are as in Table 1 with all other exposure coefficients set to 1.

shows how transmission intensity depends on the magnitude of the preferred species' and juvenile stage exposure coefficients, when the preferred species has a juvenile preference. Again we see disease transmission increases as either ϵ and ϵ_J increases, but transmission reaches its maximum when ϵ and ϵ_J are both at their maximum values. When species 1 is strongly preferred with highly exposed juveniles, most bites are on a single host group (when present): juveniles of species 1. Peak numbers of infectious vectors are almost 10 times higher when $\epsilon = \epsilon_J = 15$ than when only $\epsilon = 15$ or $\epsilon_J = 15$.

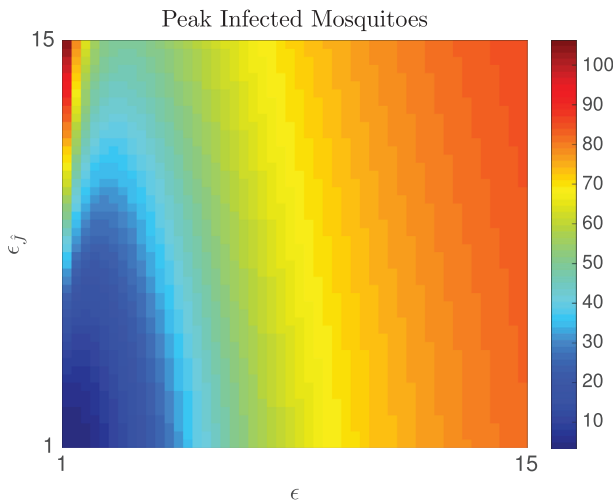


Figure 7. Shown is the peak number of infectious mosquitoes as a function of species 1 exposure coefficient ϵ and species 2 juvenile exposure coefficient ϵ_j .

Notes: When there is a feeding preference for species 1, the exposure coefficient of species 2 has little effect on overall transmission. All other parameters are as in Table 1 with all other exposure coefficients set to 1.

Next we examine the effects of the non-preferred species having differential stage exposure. When the non-preferred species has increased juvenile exposure, then that preference has less effect on the transmission of WNV than when the preferred species has increased juvenile exposure because that species is already getting fewer bites (Figure 7).

Figures 4 and 5 shows that having overall increased juvenile exposure results in a much greater intensity of disease transmission than increased exposure of one species. In Figure 8, we compare the temporal dynamics of the host and vector populations for no feeding preferences, species preference ($\epsilon = 15$), and overall juvenile preference ($\epsilon_j = \epsilon_j = 15$). Species are otherwise identical, with all other parameters are held at their respective baseline values.

With equal exposure of all hosts, peak transmission occurs on day 240 when the vector population begins to decline due to increased mortality. When there is a species preference ($\epsilon = 15$), the peak in transmission occurs slightly (9 days) earlier and is over 26 times greater. All susceptible, infectious and recovered hosts in the population are bitten in proportion to their abundance (assuming both species start the season with the same percentage of adult hosts in the recovered class; Figure 9). However, since there is an increased biting rate on hosts of species 1 relative to species 2, there are fewer adult hosts of species 1 at the end of the season due to increased disease mortality relative to species 2. When there is an overall juvenile stage preference ($\epsilon_j = \epsilon_j = 15$), peak transmission is over 3 times greater than for the species 1 preference ($\epsilon = 15$) and occurs much earlier in the season, on day 187. Since the entire juvenile population is initially susceptible while some adults are initially recovered, there is an increased biting rate on susceptible hosts relative to their abundance in the population early in the season when juveniles are present in the population (Figure 9). Once these juveniles become infectious, we see an increase in bites on infectious 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

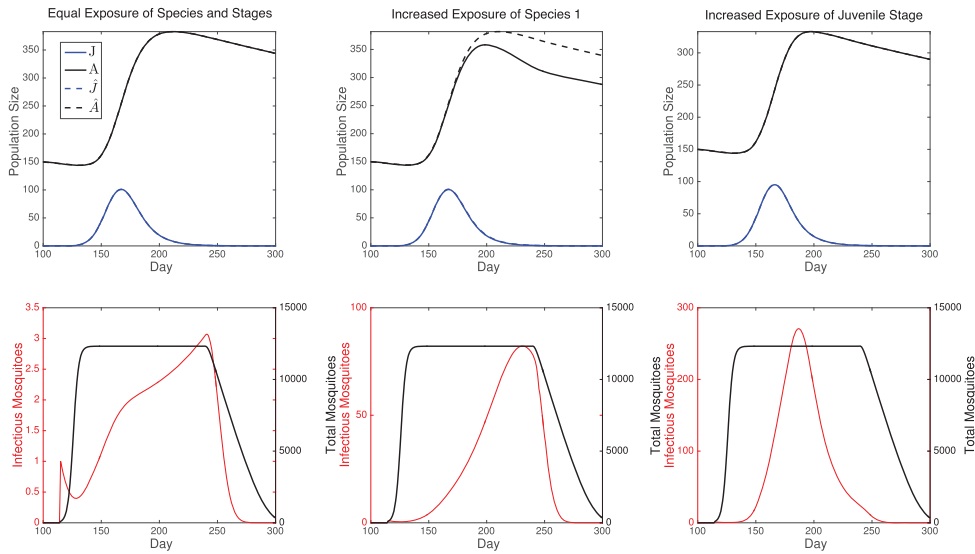


Figure 8. Shown is a comparison of the abundance of each host type (top row) as well as infectious and total mosquitoes (bottom row) over the course of the season, for equal exposure of all species and stages (left column), increased exposure of species 1 ($\epsilon = 15$; middle column) and overall increased juvenile exposure ($\epsilon_J = \epsilon_j = 15$; right column).

Notes: A juvenile stage preference of $\epsilon_J = \epsilon_j = 15$ results in an increased number of *West Nile virus* infectious mosquitoes and much earlier peak transmission than a species preference of $\epsilon = 15$. A species preference still results in an increased, earlier peak transmission compared to the case where all species and stages have equal exposure. The host population sizes for species 1 and 2 are identical except for the case where there is a vector feeding preference for species 1 (middle column); in this case, the population size of species 1 is reduced relative to species 2 due to disease-related mortality. Parameters are as in Table 1 and unless otherwise specified, all exposure coefficients are equal to 1.

death or mature into adults, all hosts are again bitten in proportion to their abundance in the community. When host species differ in competence ($\gamma_J = \gamma_A = \frac{1}{6}$, $\gamma_j = \gamma_{\hat{A}} = \frac{1}{3}$, $\delta_J = \delta_A = .72$, $\delta_j = \delta_{\hat{A}} = .36$), a species preference for the more competent species ($\epsilon = 15$) results in similarly timed, early season peak transmission (day 174) as when there is an overall juvenile preference ($\epsilon_J = \epsilon_j = 15$), with peak transmission for the overall juvenile preference only 8% higher. The only time when a strong species preference will surpass a strong juvenile stage preference and result in more intense WNV transmission is if we increase the competence of the preferred host species to its maximum infectivity ($\delta_J = \delta_A = 1$) with a very long infectious period ($\gamma_J = \gamma_A = \frac{1}{10}$).

5. The effect of host community composition on WNV transmission

Avian community composition is known to be very important in determining the vector-borne disease risk of an area (Ezenwa et al., 2006; Miller & Huppert, 2013). The dilution effect assumes that increased species diversity includes the presence of lower competency hosts that receive bites otherwise allocated to more competent hosts, therefore ‘diluting’ disease risk (Miller & Huppert, 2013; Schmidt & Ostfeld, 2001; Swaddle & Calos, 2008). In a community with two host species, this would mean that as the less competent host becomes more abundant in the host population, disease risk decreases, and as the more competent host becomes more abundant in the host population, disease risk increases.

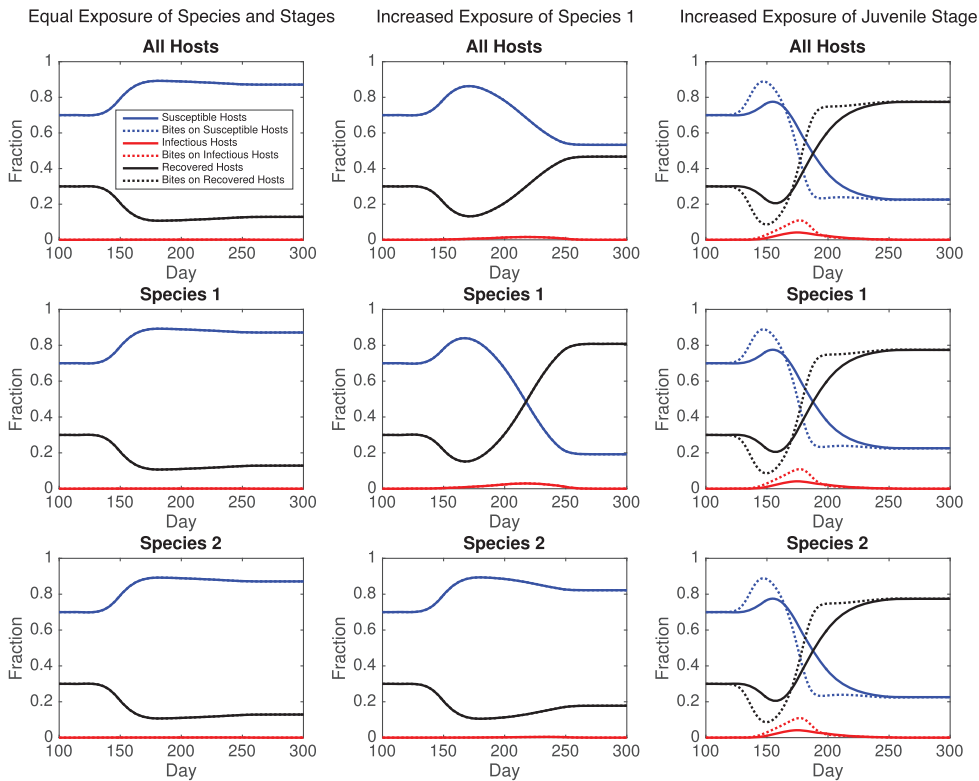


Figure 9. Shown are the percentage of hosts in each of the susceptible, infectious, and recovered classes throughout the season, as well as the percentage of bites on these host types, for the entire community population (first row), species 1 (middle row) and species 2 (bottom row).

Notes: The first column shows results for equal species and stage exposure ($\epsilon = \epsilon_j = \epsilon_j = 1$), the middle column is for a vector feeding preference for species 1 ($\epsilon = 15$), and the right column is for an overall juvenile preference with no species preference ($\epsilon_j = \epsilon_j = 15$). Parameters are as in Table 1 and unless otherwise specified, all exposure coefficients are equal to 1.

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 (Brooks & Zhang, 2010; Loss et al., 2009; Simpson et al., 2012). Miller and Huppert (2013) used a model for two host species to explore how host community composition, host competence, and vector feeding preferences for host species can affect the disease risk of an area (as measured by the basic reproduction number, R_0). They found that under certain circumstances R_0 is maximized when both species are present in the community and not just the more competent species. They call this situation ‘diversity amplification.’

Miller and Huppert found that there are two cases when dilution will occur, with disease risk (R_0) increasing monotonically in proportion to the abundance of the higher competence host: (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. They also proved that there are two scenarios 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.

Here we use model 1 to explore how incorporating avian stage-structure and stage-specific feeding preferences affects the conditions for dilution or diversity amplification. We will measure disease risk using peak infectious vectors rather than R_0 for our non-autonomous model.

While Miller and Huppert's model incorporates multiple species, it 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 feeding preferences (with disease risk measured by peak infectious vectors rather than R_0 for our non-autonomous model), here we explore how incorporating stage preferences may change how species diversity affects WNV transmission.

If vectors have no species (or stage) preference and host species differ in competence, disease risk increases monotonically with the abundance of the higher competence species (Figure 10(a)). If we incorporate a juvenile preference for the less competent species (species 1), we find that although disease risk is still maximized for a single species community, which species that is depends on the juvenile exposure coefficient of species 1 (Figure 10). If the juvenile preference of species 1 is high enough, it can outweigh the increased competence of species 2 (Figure 10(b)). Here, a species having a strong juvenile stage preference is functionally similar to having increased competence.

Miller and Huppert also found dilution when there is a feeding preference for the less competent species (Figure 11(a)). We found this scenario can switch from dilution to diversity amplification by incorporating a juvenile preference for the less competent species (Figure 11(b)). As the juvenile exposure coefficient of the less competence species increases, 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 non-monotonic curve where the maximum is achieved when both host species are present (Figure 11). Since a strong juvenile stage preference is functionally similar to increased competence, the combination of a feeding preference for the less competent species and a strong juvenile preference for the less competent species would be functionally similar to the situation where the preferred species is also more competent, which results in diversity amplification (Figure 13(a)).

With no stage preferences, diversity amplification can occur if both host species have equal competence and one species is preferred by vectors (Figure 12(a)). By incorporating a juvenile preference for the non-preferred species, we can change diversity amplification to dilution (Figure 12(b)). If the juvenile exposure coefficient of the non-preferred species is high enough, disease risk increases with the proportion of the non-preferred species. Again, a strong juvenile preference is functionally similar to having an increased species competence. Therefore, the scenario of equal host competence, a feeding preference for one host species, and a juvenile stage preference for the non-preferred species is functionally similar to the case where there is a preference for the less competent species, which results in disease risk increasing monotonically with the abundance of the more competent (non-preferred) species (Figure 11(a)).

The second case where Miller and Huppert found diversity amplification (with no stage preferences) was when the preferred host species is also more competent (Figure 13(a)). We found diversity amplification can also be converted to dilution by incorporating a juvenile

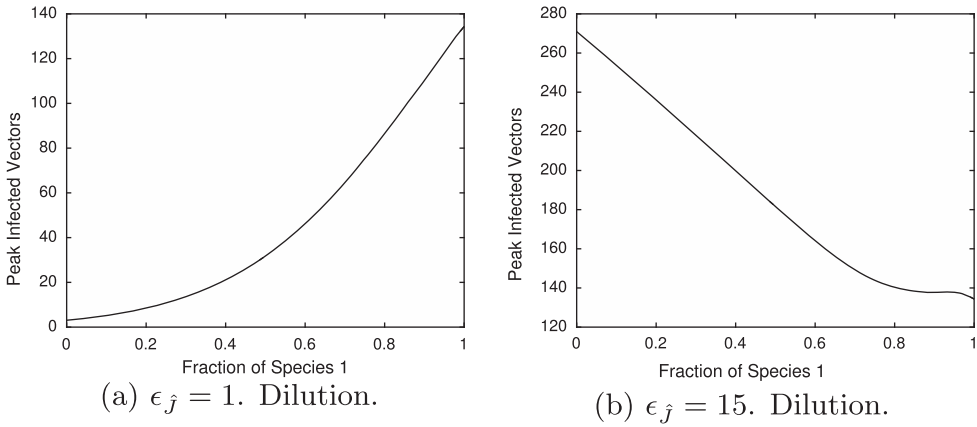


Figure 10. Species 1 is more competent, no species feeding preferences ($\gamma_j = \gamma_A = \gamma_{\hat{j}} = \gamma_{\hat{A}} = \frac{1}{3}$, $\delta_j = \delta_A = .62$, $\delta_{\hat{j}} = \delta_{\hat{A}} = .36$). (a) With no stage preferences, the peak number of infectious vectors is maximized when the community is composed only of species 1. (b) If there is a strong feeding preference for juveniles of species 2, the less competent species ($\epsilon_j = 15$), the peak number of infectious vectors is maximized when the community is composed only of species 2.

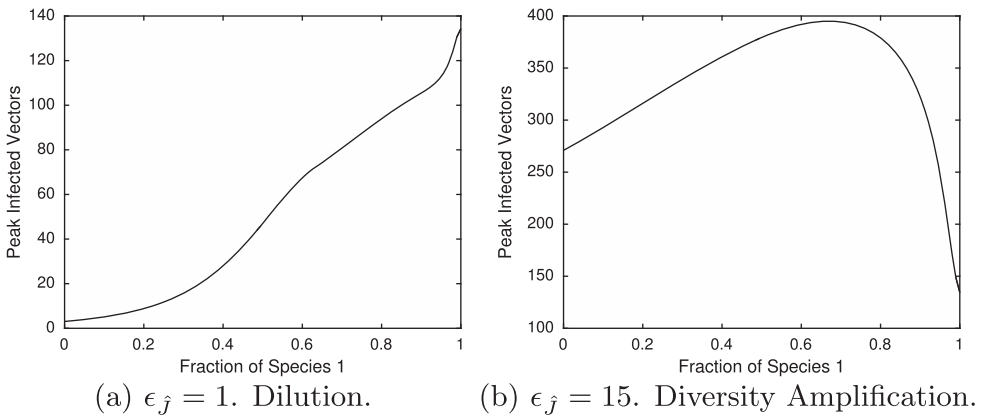
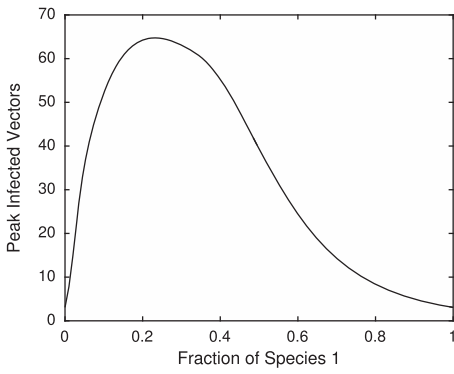
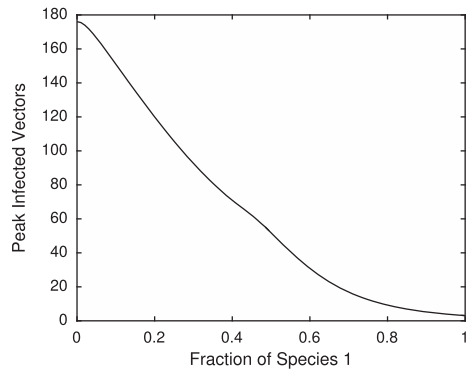


Figure 11. Species 1 is more competent, and species 2 is preferred ($\hat{\epsilon} = 5$, $\gamma_j = \gamma_A = \gamma_{\hat{j}} = \gamma_{\hat{A}} = \frac{1}{3}$, $\delta_j = \delta_A = .62$, $\delta_{\hat{j}} = \delta_{\hat{A}} = .36$). (a) With no stage preferences, the peak number of infectious vectors is maximized when the community is composed only of species 1. (b) If there is a strong feeding preference for juveniles of species 2, the preferred but less competent species ($\epsilon_j = 15$), the peak number of infectious vectors is maximized when both species are present in the community.

stage preference for the less preferred species. As we increase the juvenile preference, the disease risk curves go from being non-monotonic, with a maximum at an interior point, to monotonically increasing with the proportion of the species that has the juvenile preference (Figure 13). Here the combination of 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, resulting in a monotonic increase in disease risk as the abundance of the more competent species increases (Figure 11(a)).

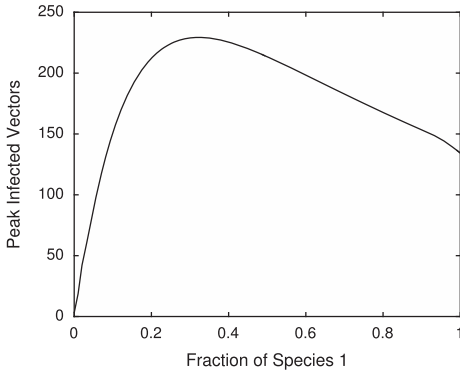


(a) $\epsilon_j = 1$. Diversity Amplification.

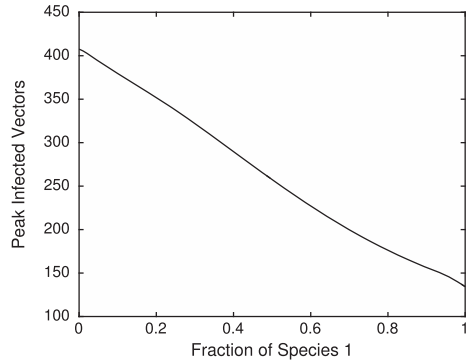


(b) $\epsilon_j = 10$. Dilution.

Figure 12. Species 1 and 2 are equal in competence and species 1 is preferred ($\epsilon = 5$, $\gamma_j = \gamma_A = \gamma_j = \gamma_{\hat{A}} = \frac{1}{3}$, $\delta_j = \delta_A = \delta_j = \delta_{\hat{A}} = .36$). (a) With no stage preferences, the peak number of infectious vectors is maximized when both species are present in the community. (b) If there is a strong feeding preference for juveniles of species 2, the non-preferred species ($\epsilon_j = 10$), the peak number of infectious vectors is maximized when the community is composed only of species 2.



(a) $\epsilon_j = 1$. Diversity Amplification.



(b) $\epsilon_j = 25$. Dilution.

Figure 13. Species 1 is more competent and species 1 is preferred ($\epsilon = 5$, $\gamma_j = \gamma_A = \gamma_j = \gamma_{\hat{A}} = \frac{1}{3}$, $\delta_j = \delta_A = .62$, $\delta_j = \delta_{\hat{A}} = .36$). (a) With no stage preferences, the peak number of infectious vectors is maximized when both species are present in the community. (b) If there is a strong enough feeding preference for juveniles of species 2, the non-preferred and less competent species ($\epsilon_j = 25$), the peak number of infectious vectors is maximized when the community is composed only of species 2.

6. Discussion

The dynamics of WNV are extremely complex, with transmission occurring most often not between one species of host and one species of vector, but a network of multiple host and vector species exhibiting both between and within species heterogeneity (Cruz-Pacheco et al., 2012; Diaz, Flores, Quaglia, & Contigiani, 2013). To our knowledge, our mathematical model presented here is the first to incorporate both species and stage specific biting rates of vectors on hosts. To show the consequences of ignoring species diversity for structured populations, we compared our two-species model to a single-species model that would result from averaging parameters over the community. Our results demonstrate that the WNV transmission dynamics in a community with two host

species are not adequately represented by a simplified model with one representative species if the host species differ in either competence or the exposure of their juvenile stage to vectors (for example, one species has highly exposed nestlings while the nestlings of the other species are protected from mosquitoes). A single-species model may either overestimate or underestimate transmission, depending on competence parameters and stage exposure coefficients. When species differ only in competence, or parameters related to their ability to transmit disease, a single-species model underestimates transmission levels compared to a two-species model incorporating between species heterogeneity. However, when species differ in the relative exposure of their juvenile and adult stages to vectors (i.e. the degree of within-species heterogeneity), the single-species model can overestimate transmission compared to a two-species model. This occurs when there is a large difference in the relative exposure of juveniles between equally competent species, or when species differ in competence and the less competent species has increased juvenile exposure. In the latter scenario, the competence parameters of the single-species model are higher than those of the less competent species, with all juveniles and receiving bites at an increased rate relative to adults (instead of just juveniles of the less competent species).

We also explored the relative importance of species and stage vector feeding preferences, comparing the effects of heterogeneity in species exposure coefficients (while holding stage exposure equal) to heterogeneous stage exposure for both species. Unless one species is extremely competent, we find increased juvenile stage exposure will result in higher levels of transmission relative to increased exposure of one species. It is important to note that both species are present throughout the entire season, while juveniles are only present in the middle of the season, from the time they hatch until they mature into adults. However, when present they are a small group of preferred, initially susceptible hosts and result in early season amplification of transmission in the vector population. Once all juveniles have matured into adults, hosts are again bitten in proportion to their abundance. When there is a species preference, the preferred host is present throughout the entire season, and bites are distributed to juveniles and adults of that species in proportion to their abundance. The other species continues to receive bites at a reduced rate throughout the entire season resulting in lower levels of infection in that species.

Between and within-species heterogeneity in exposure to vectors can also play a critical role in how species diversity in a community affects disease risk. [Miller and Huppert \(2013\)](#) showed that vector feeding preferences for certain host species, along with the competence of those species, is very important for determining if disease risk is maximized for a community composed of a single species (dilution) or more than one species (diversity amplification). We found that by incorporating stage structure and juvenile stage preferences, we were able to change cases of dilution to diversity amplification and vice versa, where disease risk is measured by peak infectious vectors during a season. When host species differ only in competence and there are no vector feeding preferences, disease risk is maximized when a community is composed exclusively of the more highly competent species. However, if the juvenile exposure coefficient of the less competent species is high enough, then disease risk is maximized when the community is composed only of the lower competence species. Here increased juvenile stage preference acts similarly to increased competence in the sense that both are mechanisms for increased disease amplification. When there is a vector feeding preference for the less competent species, disease risk is also maximized when only the more competent species is present. However, if there is a vector

feeding preference for juveniles of the less competent species, disease risk can be maximized when both species are present in the population. With no stage preferences, diversity amplification can occur when species are equal in competence but one species is preferred, or when there is a preference for the more competent species. We show both scenarios can be converted to dilution by incorporating a strong enough feeding preference for juveniles of the non-preferred species. Therefore, competence, species feeding preferences, and stage feeding preferences are all important mechanisms for determining the effect of increased species diversity on the disease risk of a community.

7. Conclusions and future work

The work presented here illustrates the need to consider both between and within species heterogeneity in models for vector-borne disease such as WNV. While we have allowed species to differ in exposure, competence and stage exposure coefficients, we note that species may also vary in many other ways, including their relative abundance in the community or percentage of the adult population that is susceptible at the beginning of the season. Avian species are also likely to differ in reproductive factors such as clutch initiation date, clutch size and length of the nestling stage (Baicich & Harrison, 2005). While we have used a unimodal Gaussian distribution to model nestling production, some species may produce multiple clutches over the course of a single season (Bulluck, Huber, Viverette, & Blem, 2013) and a bimodal juvenile recruitment curve may be appropriate for these species. Within a species, juveniles may potentially differ from adults in competence and also mortality rates, either from disease or increased predation.

Disclosure statement

No potential conflict of interest was reported by the authors.

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