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A MATHEMATICAL MODEL FOR THE SPREAD OF WEST NILE VIRUS IN MIGRATORY AND RESIDENT BIRDS

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Abstract. We develop a mathematical model for transmission of West Nile virus (WNV) that incorporates resident and migratory host avian populations and a mosquito vector population. We provide a detailed analysis of the model's basic reproductive number and demonstrate how the exposed infected, but not infectious, state for the bird population can be approximated by a reduced model. We use the model to investigate the interplay of WNV in both resident and migratory bird hosts. The resident host parameters correspond to the American Crow (*Corvus brachyrhynchos*), a competent host with a high death rate due to disease, and migratory host parameters to the American Robin (*Turdus migratorius*), a competent host with low WNV death rates. We find that yearly seasonal outbreaks depend primarily on the number of susceptible migrant birds entering the local population each season. We observe that the early growth rates of seasonal outbreaks is more influenced by the the migratory population than the resident bird population. This implies that although the death of highly competent resident birds, such as American Crows, are good indicators for the presence of the virus, these species have less impact on the basic reproductive number than the competent migratory birds with low death rates, such as the American Robins. The disease forecasts are most sensitive to the assumptions about the feeding preferences of North American mosquito vectors and the effect of the virus on the hosts. Increased research on the these factors would allow for better estimates of these important model parameters, which would improve the quality of future WNV forecasts.

1. **Introduction.** West Nile virus (WNV) is a mosquito-borne arbovirus belonging to the *Flavivirus* genus [28] and is closely related to other viruses such as St. Louis encephalitis, Japanese encephalitis, Murray Valley encephalitis, and Kunjin. WNV is transmitted and perpetuated through a bird-mosquito-bird infection cycle. The *Culex* genus of mosquitoes is the main vector of the virus, although the different Culex species responsible for transmission vary greatly by region. The US Centers for Disease Control (CDC) has found WNV in 326 species of birds [19]. The ability of the virus to infect multiple bird species is a key factor in how it has spread so rapidly and extensively throughout the world. For example, migrating white storks introduced WNV to the Middle East [8]. The commercial transportation of infected birds can introduce the virus to new locations, as seen when Eastern

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Equine Encephalomyelitis was transported to Jamaica from the United States (US) via bird shipments [8]. Other animals are bitten by mosquitoes and infected by WNV as well, but in mammals the viral load is usually too low to easily reinfect other mosquitoes. Thus mammals, specifically humans and horses, are considered dead-end hosts and not a significant factor in the propagation of the virus.

Despite the lack of sufficient viral load in humans to reinfect mosquitoes, the medical effects on humans can be devastating. After an incubation period of about 3-14 days, about 60-80% of infections are entirely asymptomatic. The rest of the infected persons experience what is called West Nile Fever. West Nile Fever includes symptoms such as fever, headache, fatigue, skin rash, swollen lymph glands, and eye pain. If the disease progresses further it could become either West Nile meningitis, West Nile encephalitis, or West Nile poliomyelitis. About 1 in every 150 infected people die or have severe neurological damage, which can be permanent. Horses can also be infected with WNV and have a 40% mortality rate [20].

The CDC's mosquito WNV mitigation plans include: surveillance (monitoring levels of mosquito activity, and where virus transmission is occurring); reduction of mosquito breeding sites; community outreach and public education; and use of chemical and biological methods to control both mosquito larvae and adult mosquitoes [21]. These plans rely heavily on local surveillance, risk calculations of human disease, and a phased response at different levels of activity. Mathematical models can incorporate these factors and help optimize the timing and techniques to reduce the impact of a WNV epidemic.

The rapid emergence of WNV in North America spurred research on the transmission of the virus and new mathematical models to help understand, and mitigate, its spread. Thomas and Urena, 2001 [42] created a WNV model to examine the effect of culling the mosquito population on the rate of human infection. Their ordinary differential equation (ODE) transmission model used a frequency dependence biting rate that is independent of the densities of the bird and human populations. Wonham et al. [51] extended their model using an ODE suseptible-infected-recovered (SIR) model for the bird-mosquito interactions and neglected the human population in the transmission cycle. After the Wonham et al. benchmark paper, the main directions in modeling have been: incorporating humans into the model, expanding and refining the model to include more detailed aspects of the host or mosquito population, and focusing on the role of migration of the birds, which sometimes involves discrete patches of activity.

Chowell-Puente et al. [12] and Bowman et al. [6] coupled infected mosquito-bird models with a human population to estimate human cases. Cruz-Pacheco et al. (2004) investigated the effects of vertical transmission, from mother to offspring, in the mosquito population [14]. Vertical transmission of WNV could provide an explanation for the over-wintering of WNV. The Wonham et al. model has also been extended by adding multiple classes of populations. Unnasch et al. 2006 [46] incorporated the vulnerable young of the year or fledging bird classes. Adding this distinct group changes the dynamics of the virus spread and it appears that maintaining an outbreak relied heavily on the young of the year bird group. More recently, Cruz-Pacheco et al. [15] released a study focusing on the interactions between different species of birds and mosquitoes. The paper lays the groundwork for multi-species models of WNV and extends a model similar to Wonham et al.'s for multiple species of birds and mosquitoes. Abdelrazec et al. formulated and

analyzed a WNV model in multiple bird species in a single season and compared their results to outbreaks in Ontario, Canada [1].

Chatterjee et al. 2008 [8] investigated the importance of seasonal bird migration, and coupled the mosquito growth rate to the bird population. Qiu [35] employed discrete patches where the birds, but not the mosquitoes, migrate between the two patches. This model allows for two outbreaks to simultaneously run their course. Druand et al. [17] considered three distinct patches, each patch having local bird and mosquito populations, coupled with two migratory bird species. The parameters in their model were are defined for the patches to represent wet Africa, dry Africa, and the European Mediterranean basin. The migratory birds move the virus between areas starting new outbreaks or perpetuating the current outbreaks. Similar models have been used to study the interaction of migratory and resident birds in sustaining West Nile Virus [34] and avian influenza [6, 24, 49] infections.

Our ODE model and analysis continues this approach, incorporating migrating and resident birds into the model for one area by extending Wonham et al.'s [51] model and including seasonality using the approach described in Moore [32]. Infected bird populations are split into susceptible, infectious, and recovered classes, and the mosquito population among susceptible, exposed, and infectious classes.

After describing the ecological model parameters, we investigate the disease progression in a single bird population. We then analyze the difference in the model predictions when there are both migratory and resident bird populations. We derive the basic reproductive number, $\mathcal{R}_0$, and show simulations of the model predictions over multiple seasons. The model yields insights to the effects of preferred and alternate feeding hosts, the interplay between multi-species and single species of birds, and the conditions that are necessary for regular seasonal outbreaks to occur. We find that although the resident species may be good indicators of WNV activity because of high death rates, the migrant species is driving the epidemic. We also found that the return of a large number of susceptible migratory birds can cause yearly seasonal outbreaks in the model.

2. **Mathematical model.**

We start by defining and analyzing a model for the spread of WNV in one resident bird and mosquito population with a biting rate based on the Chitnis, Cushing, and Hyman (CCH) mosquito-borne disease model [9, 11, 30]. We then extend this model to include both a resident and migratory bird species and compare the reproductive numbers for the two models to analyze the relative importance of the migration, population sizes, difference in the biting preference of the mosquitoes for the two bird species, mortality, and other factors.

2.1. **Single resident bird model.**

The system of ODEs for the mosquito vectors and bird hosts are

$$\dot{S}_v = \psi_v N_v - \lambda_v S_v - \mu_v S_v \tag{1a}$$

$$\dot{E}_v = \lambda_v S_v - \nu_v E_v - \mu_v E_v \tag{1b}$$

$$\dot{I}_v = \nu_v E_v - \mu_v I_v \tag{1c}$$

$$\dot{S}_b = \psi_b H + \rho_b R_b - \lambda_b S_b - \mu_b S_b \tag{1d}$$

$$\dot{E}_b = \lambda_b S_b - \nu_b E_b - \mu_b E_b \tag{1e}$$

$$\dot{I}_b = \nu_b E_b - \gamma_b I_b - \mu_b I_b - \delta_b I_b \tag{1f}$$

$$\dot{R}_b = \gamma_b I_b - \rho_b R_b - \mu_b R_b \tag{1g}$$

TABLE 1. Model Variables and Parameters

Symbol	Interpretation	Units
S_v, S_b	Susceptible mosquitoes and resident birds	vectors, birds
E_v, E_b	Exposed vectors and birds	vectors, birds
I_v, I_b	Infected vectors and birds	vectors, birds
R_b	Recovered birds	birds
N_v, N_b	Total vectors or birds	vectors, birds
ψ_v, ψ_b	Recruitment rate of vectors or birds	day^{-1}
μ_i	Death rate of species i	day^{-1}
ν_i	Incubation rate for species i	day^{-1}
γ_i	Loss of infectivity rate of species i	day^{-1}
ρ_i	Loss of immunity rate of species i	day^{-1}
δ_i	Disease induced death rate of species i	day^{-1}
b	Total number of bites per day	bites day^{-1}
b_{bv}	Number of bites from vectors per bird per day	bites day^{-1}
b_{vb}	Number of bites on birds per vector per day	bites day^{-1}
β_{ij}	Probability of transmission per bite from j to i	dimensionless
σ_i	Max tolerance (hosts) or ideal feeding (mosquitoes)	bites day^{-1}
λ_i	Inoculation rate of species i	day^{-1}

The derivatives, $\dot{}$, are with respect to time, in days. The subscript v refers to the mosquito vector and b for the resident bird host. Birds and mosquitoes enter the susceptible class through a per-capita birth rate, ψ_j, and leave all compartments via a per-capita death rate, μ_j. Mosquitoes move from exposed to infectious at a constant per-capita rate, ν_v that is the inverse of the incubation period. Infected birds move from infected to infectious at a rate ν_b and recover at a constant per capita rate, γ_b, the inverse of the infectious period. Birds die from disease at a rate δ_b and lose immunity at a rate ρ_b. We normalize the bird recruitment rate

$$H = \frac{\mu_b}{\psi_b} K \tag{2}$$

to be limited by the carrying capacity, K.

The variables are defined in Table 1 and the total populations are denoted $N_v = S_v + E_v + I_v$ and $N_b = S_b + E_b + I_b + R_b$. The equations for the total populations are $\dot{N}_v = \psi_v N_v - \mu_v N_v$ and $\dot{N}_b = \psi_b H - \mu_b N_b - \delta_b I_b$. The range of values for the model parameters (Table 1) will be defined later (Table 2).

2.1.1. *Biting rate and force of infection equations.* We assume the infectivity of the recovered or latently infected birds is minimal, so the recovered class is unable to infect mosquitoes. This assumption is based on the low level of virus found in the bloodstream of recovered birds in lab experiments (even though some virus was sometimes found in organs) [36]. It is therefore thought that after avian species have contracted WNV and it has run its course, there is not a high enough viral load left in the bird's blood to result in even a small likelihood of transmission to a mosquito [36]. In the context of the questions we are asking with our model, this assumption has only a small impact on the early disease progression, even though $\mathcal{R}_0$ is sensitive to the transmission rates.

The biting rate is defined as a function of the total number of bites per day by mosquitoes, [9, 11, 30]

$$b = \frac{\sigma_v N_v \sigma_b N_b}{\sigma_v N_v + \sigma_b N_b} \ . \tag{3}$$

The parameter σ_v is the number of bites per day a mosquito would ideally have if a host were always available and is estimated from the gonotrophic cycle of the mosquito. The σ_b term is the number of bites per day an avian host will accommodate before it takes measures to protect against additional bites (measures such as removing itself from the area). The total biting rate, b is defined by the total number of bites per day that the entire mosquito population would like, $\sigma_v N_v$, times the total number of bites per day the entire bird population will tolerate, $\sigma_b N_b$ divided by the sum of $\sigma_v N_v$ and $\sigma_b N_b$. This biting rate dependents on the size of both the host and mosquito populations. When there are sufficient mosquitoes and hosts available, then this nonlinear biting rate is well approximated by the commonly used frequency dependence or mass action (density dependent) biting rates [50]. When the mosquitoes or hosts become scarce, then (3) has the correct asymptotic limits [9].

The forces of infection, λ_v, and λ_b, for(4)

$$\lambda_v = b_{vb}\beta_{vb}\frac{I_b}{N_b}, \qquad \lambda_b = b_{bv}\beta_{bv}\frac{I_v}{N_v} \tag{4}$$

are the rates individuals are infected and move from the susceptible to the exposed class. For birds, the infection rate λ_b is the product of the average number of bites per bird per day ($b_{vb} = \frac{b}{N_b}$)*times* the probability that a mosquito bite will successfully transmit the virus to the bird (β_{bv}) *times* the probability ($\frac{I_v}{N_v}$) that a mosquito is infectious. (When there are multiple species of birds, then the model will include the proportion of mosquito bites for each bird species.) The mosquito infection rate, λ_v, is similar to λ_b, but includes two terms representing the sum of the virus transmitted by the infected birds and low level transmission by the recovered birds and the average number of times a mosquito bites a bird per day ($b_{bv} = \frac{b}{N_v}$).

2.1.2. *The basic reproductive number.* We derive the basic reproduction number for equations (1) using the next generation matrix method [47, 23]. In this approach, we need only consider the infected population classes $M = (E_v, I_v, E_b, I_b)^T$ since these are the only classes that play a role in creating and removing infections. The ODEs for the infected population, $\dot{M} = F - V$, can be defined as the rate of production of new infections, F, minus the removal of individuals from that population class, V, where

$$F = \begin{pmatrix} b_{vb}\beta_{vb}\frac{I_b}{N_b}S_v \\ 0 \\ b_{bv}\beta_{bv}\frac{I_v}{N_v}S_b \\ 0 \end{pmatrix} \ , \ V = \begin{pmatrix} \nu_v E_v + \mu_v E_v \\ -\nu_v E_v + \mu_v I_v \\ \nu_b E_b + \mu_b E_b \\ -\nu_b E_b + \gamma_b I_b + \mu_b I_b + \delta_b I_b \end{pmatrix} \ .$$

The Jacobians of each of these matrices, $\mathbb{J}_{\mathbb{F}}$ and $\mathbb{J}_{\mathbb{V}}$, evaluated at the disease free equilibrium ($S_v = N_v,\ S_b = N_b,\ E_v = I_v = I_b = R_b = 0$) are:

$$\mathbb{J}_{\mathbb{F}} = \begin{pmatrix} 0 & 0 & 0 & b_{vb}\beta_{vb} \\ 0 & 0 & 0 & 0 \\ 0 & b_{bv}\beta_{bv} & 0 & \\ 0 & 0 & 0 & 0 \end{pmatrix} \ , \ \mathbb{J}_{\mathbb{V}} = \begin{pmatrix} \nu_v + \mu_v & 0 & 0 & 0 \\ -\nu_v & \mu_v & 0 & 0 \\ 0 & 0 & \nu_b + \mu_b & 0 \\ 0 & 0 & -\nu_b & \gamma_b + \mu_b + \delta_b \end{pmatrix} \ .$$

The next generation matrix is easier to understand if we define the average time in E_v, I_v, E_b, and I_b as $\tau_{ev} = 1/(\nu_v + \mu_v)$, $\tau_{iv} = 1/\mu_v$, $\tau_{eb} = 1/(\nu_b + \mu_b)$, and $\tau_{ib} = 1/(\gamma_b + \mu_b + \delta_b)$, respectively. We also define the probability of an infected mosquito or bird surviving to the infected states as $p_{iv} = \nu_v/(\nu_v + \mu_v)$ and $p_{ib} = \nu_b/(\gamma_b + \mu_b + \delta_b)$. Note that the probability that an infected mosquito or bird enters the exposed state is $p_{ev} = p_{eb} = 1$. Using this notation, the inverse of the Jacobian of the removal matrix $\mathbb{J}_\mathbb{V}$ is

$$\mathbb{J}_\mathbb{V}{}^{-1} = \begin{pmatrix} \tau_{ev} & 0 & 0 & 0 \\ p_{iv}\tau_{iv} & \tau_{iv} & 0 & 0 \\ 0 & 0 & \tau_{eb} & 0 \\ 0 & 0 & p_{ib}\tau_{ib} & \tau_{ib} \end{pmatrix} . \tag{5}$$

The $(j,j) - th$ element of this matrix is the average time spent in state j. The $(j-1,j)$ element is the expected average time that a mosquito or bird in state $j-1$ will spend in state j. That is, it is the probability that the mosquito or bird will advance to state j *times* the time spent in state j.

The next generation matrix $\mathbb{J}_\mathbb{F}\mathbb{J}_\mathbb{V}{}^{-1}$ is

$$\mathbb{J}_\mathbb{F}\mathbb{J}_\mathbb{V}{}^{-1} = \begin{pmatrix} 0 & 0 & p_{ib}b_{vb}\beta_{vb}\tau_{iv} & b_{vb}\beta_{vb}\tau_{ib} \\ 0 & 0 & 0 & 0 \\ p_{iv}b_{bv}\beta_{bv}\tau_{iv} & b_{bv}\beta_{bv}\tau_{iv} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} . \tag{6}$$

The reproductive number $\mathcal{R}_0$ is given by the spectral radius of the next generation matrix $\mathbb{J}_\mathbb{F}\mathbb{J}_\mathbb{V}{}^{-1}$. This can be represented as the square root of the product of two dimensionless numbers representing the spread of the virus from a mosquito to susceptible birds ($\mathcal{R}_0^v$) times the spread from a bird to mosquitoes ($\mathcal{R}_0^b$). That is, $\mathcal{R}_0 = \sqrt{\mathcal{R}_0^v \times \mathcal{R}_0^b}$. The host-to-mosquito (bird) reproductive number $\mathcal{R}_0^b$ is the number of secondary mosquito infections created by one infected bird population and is the sum of two terms. The mosquito-to-bird (vector) reproductive number $\mathcal{R}_0^v$ is the number of secondary bird infections created by one infected mosquito in a fully susceptible bird population. These reproductive numbers are defined as

$$\mathcal{R}_0^v = p_{iv}b_{bv}\beta_{bv}\tau_{iv} \ , \tag{7a}$$

$$\mathcal{R}_0^b = p_{ib}b_{bv}\beta_{vb}\tau_{ib} \tag{7b}$$

and are the product of the probability (p_{i*}) that an infected mosquito or bird survives to the infectious state *times* the rate that they infect others ($b_{**}\beta_{**}$) while infectious *times* the length of time (τ_{i*}) they are in the infectious state.

The total number of new infections from a single infected mosquito in the *two* (bird-to-vector and vector-to-bird) cycles is a branching process and grows geometrically. That is, a single infected mosquito in the vector-to-bird and bird-to-vector cycles would infect $\mathcal{R}_0^v\mathcal{R}_0^b$ mosquitoes. Similarly, a single infected bird would infect $\mathcal{R}_0^b\mathcal{R}_0^v$ birds over both cycles. The basic reproductive number measures the average number of new infected cases over a *single* infection cycle. The geometric average of the two reproductive numbers is the appropriate average and gives the same result over two cycles, independent of which animal is infected first.

2.1.3. *Reduced model with no exposed state for birds.* Our single bird model (1) is a susceptible-exposed-infected-recovered (SEIR) model for the birds that includes an exposed state for an infected bird. Most previous WNV models [14, 1] have a susceptible-infected-recovered (SIR) model for the bird population. Because the

exposed state can be important in some situations, we derive our reduced SIR model for the birds from (1) so that it still captures the role of the exposed state.

In particular, we will derive a reduced SIR bird model that has the same endemic solution as the full SEIR model and a very similar reproductive number. This is accomplished by defining the parameters for the reduced SIR model as an explicit function of the parameters for the full SEIR bird model (1) and combining the exposed and infectious states into a single infected state.

The reduced model eliminates the exposed state for the birds:

$$\dot{S}_v = \psi_v N_v - \lambda_v S_v - \mu_v S_v \tag{8a}$$
$$\dot{E}_v = \lambda_v S_v - \nu_v E_v - \mu_v E_v \tag{8b}$$
$$\dot{I}_v = \nu_v E_v - \mu_v I_v \tag{8c}$$
$$\dot{S}_b = \psi_b H - \lambda_b S_b - \mu_b S_b \tag{8d}$$
$$\dot{I}_b = \lambda_b S_b - \gamma_b I_b - \mu_b I_b - \delta_b I_b \tag{8e}$$
$$\dot{R}_b = \gamma_b I_b - \mu_b R_b \tag{8f}$$

where the force of infections have the same form as the SEIR model,

$$\lambda_b = b_{bv}\beta_{bv}\frac{I_v}{N_v}, \qquad \lambda_v = b_{vb}\beta_{vb}\frac{I_b}{N_b} \tag{9}$$

and the biting rate b is the same as in the full model.

Our goal is to define the parameters in (8) as a function of the parameters in (1). To make it clear which equation the parameters and variables are associated with, we use the hat symbol $\hat{}$ to denote the variables and parameters in the full SEIR model (1). That is, in our reduced model $S_b = \hat{S}_b$, $I_b = \hat{E}_b + \hat{I}_b$, $R_b = \hat{R}_b$, and $N_b = \hat{N}_b$. We will use the superscript * to denote the steady state solution and create our reduced model so that at equilibrium, the SIR and SEIR models have the same steady-state, $S_v^* = \hat{S}_v^*$, $E_v^* = \hat{E}_v^*$, $I_v^* = \hat{I}_v^*$, $S_b^* = \hat{S}_b^*$, $I_b^* = \hat{E}_b^* + \hat{I}_b^*$, $R_b^* = \hat{R}_b^*$.

The steady-state infectious population in (1f) satisfies $0 = \dot{\hat{I}}_b^* = \hat{\nu}_b\hat{E}_b^* - (\hat{\gamma}_b + \hat{\mu}_b + \hat{\delta}_b)\hat{I}_b^*$. This implies that at equilibrium the SEIR $\hat{E}_b^* = \hat{I}_b^*(\hat{\gamma}_b + \hat{\mu}_b + \hat{\delta}_b)/\hat{\nu}_b$. We define the scaling parameter $\phi = 1 + (\hat{\gamma}_b + \hat{\mu}_b + \hat{\delta}_b)/\hat{\nu}_b$ so that if models to agree at equilibrium, then the SIR infected population can be expressed as $I_b^* = \phi\hat{I}_b^* = \big(1 + (\hat{\gamma}_b + \hat{\mu}_b + \hat{\delta}_b)/\hat{\nu}_b\big)I_b^* = \hat{I}_b^* + \hat{E}_b^*$.

Adding equations (1e) and (1f) exposed and infectious bird populations, we have the equation for the total infected population in the SEIR equation. This can be compared with (8e) for the total infected population;

$$\dot{\hat{E}}_b + \dot{\hat{I}}_b = \hat{\lambda}_b\hat{S}_b - \hat{\gamma}_b\hat{I}_b - \hat{\mu}_b(\hat{E}_b + \hat{I}_b) - \hat{\delta}_b\hat{I}_b \tag{10a}$$
$$\dot{I}_b = \lambda_b S_b - \gamma_b I_b - \mu_b I_b - \delta_b I_b \ . \tag{10b}$$

Each term in these equations will be equal at steady-state, when $I_b^* = \phi\hat{I}_b^*$, if we define $\gamma_b = \hat{\gamma}_b/\phi$, and $\delta_b = \hat{\delta}_b/\phi$, $\lambda_b = \hat{\lambda}_b$, and $\mu_* = \hat{\mu}_*$. The infected populations, $\hat{I}_b$ and I_b, also appear in (1b) and (8b) in the definitions of $\hat{\lambda}_v$ and λ_v. If we define $\beta_{bv} = \hat{\beta}_{bv}/\phi$, then the steady-state forces of infection will be the same, $\hat{\lambda}_b^* = \hat{\lambda}_b^*$;

$$\lambda_v^* = b_{vb}\beta_{vb}\frac{I_b^*}{N_b^*} = \hat{b}_{vb}\big(\frac{\hat{\beta}_{vb}}{\phi}\big)\frac{(\phi\hat{I}_b^*)}{\hat{N}_b^*} = \hat{b}_{vb}\hat{\beta}_{vb}\frac{\hat{I}_b^*}{\hat{N}_b^*} = \hat{\lambda}_v^* \tag{11a}$$

We define all other parameters in equations (1) and (8) to be the same. With these scalings, it is a straightforward calculation to show that an equilibrium solution of (1) is also an equilibrium solution of (8) and visa versa.

The basic reproduction number for Eq. (8) can be derived using the next generation approach to give $\mathcal{R}_0 = \sqrt{\mathcal{R}_0^v \times \mathcal{R}_0^b}$, where

$$\mathcal{R}_0^v = b_{bv}\beta_{bv}\frac{\nu_v}{(\nu_v+\mu_v)}\frac{1}{\mu_v} = \hat{b}_{bv}\hat{\beta}_{bv}\frac{\hat{\nu}_v}{(\hat{\nu}_v+\hat{\mu}_v)}\frac{1}{\hat{\mu}_v} = \hat{\mathcal{R}}_0^v \tag{12a}$$

$$\mathcal{R}_0^b = b_{vb}\beta_{vb}\frac{1}{(\delta_b+\gamma_b+\mu_b)} = \hat{b}_{vb}\frac{\hat{\beta}_{vb}}{\phi}\frac{1}{(\hat{\delta}_b/\phi+\hat{\gamma}_b/\phi+\mu_b)} \tag{12b}$$

$$= \hat{b}_{vb}\hat{\beta}_{vb}\frac{1}{(\hat{\delta}_b+\hat{\gamma}_b+\phi\hat{\mu}_b)} \approx \hat{\mathcal{R}}_0^b \tag{12c}$$

The reduced and full model reproductive numbers are very close when one of two conditions hold: either $\phi \approx 1$, or $\phi\hat{\mu}_b$ is small compared to $\hat{\delta}_b+\hat{\gamma}_b$. When $\phi = 1$ then the parameters in the two models are the same; this is rarely the case. However $\hat{\mu}_b << \hat{\delta}_b + \hat{\gamma}_b$ and so the resulting in the bird SIR reproductive number is very close to the bird SEIR reproductive number.

Thus, the reduced bird model has almost the same basic reproductive number (hence the same early growth rate), and same endemic equilibrium as the full bird model with the exposed state. We observed that numerical solutions of these two models are very close in simulations for the parameter ranges in this study. Therefore, in the rest of our study, we use the SIR formulation for the two-bird model without explicitly including an exposed state, although we will implicitly account for it by defining the parameters appropriately.

We note that if we use the same approach to eliminate the exposed state in the mosquito model, then neither of these conditions would hold and the reproductive number the reduced models would be very different.

2.2. **Two-bird model.** We add a second migratory bird population (subscript "m") representing a competent migratory species with a lower death due to disease rate. The parameters for the migratory population are based on the migratory American Robin, although several bird species could fall into this category (e.g. Northern Cardinals, House Sparrows [27, 7, 4]). The American Robin is a preferred host for *Culex pipiens*, an important WNV mosquito species [25]. The mosquito preference for the different birds is captured in the model through the values for σ_m and σ_b, with mosquito preference for the migratory bird. The second bird population is denoted by subscript "b", representing a highly competent resident species that has a higher death due to disease rate for WNV. Previously σ_b represented the tolerance that a bird species had for mosquito bites, but now with two bird species and one mosquito species, the term will also incorporate a feeding preference of the mosquito. The higher the σ_i, $i \in (b, m)$, the higher the preference the mosquito has for that host.

The proportion of the total bites on the ith bird population is $\sigma_i N_i/(\sigma_m N_m + \sigma_b N_b)$ for $i = r, m$. The biting rate is modified to account for the two bird populations by changing the numerator from $\sigma_v N_v \sigma_b N_b$ to $\sigma_v N_v(\sigma_b N_b + \sigma_m N_m)$, and the denominator to $\sigma_v N_v + \sigma_b N_b + \sigma_m N_m$.

The resulting two-bird model equations are:

$$\dot{S}_v = \psi_v N_v - \lambda_v S_v - \mu_v S_v \tag{13a}$$
$$\dot{E}_v = \lambda_v S_v - \nu_v E_v - \mu_v E_v \tag{13b}$$
$$\dot{I}_v = \nu_v E_v - \mu_v I_v \tag{13c}$$

$$\dot{S}_b = \psi_b H - \lambda_b S_b - \mu_b S_b \tag{14a}$$
$$\dot{I}_b = \lambda_b S_b - \gamma_b I_b - \mu_b I_b - \delta_b I_b \tag{14b}$$
$$\dot{R}_b = \gamma_b I_b - \mu_b R_b \tag{14c}$$

$$\dot{S}_m = \psi_m H - \lambda_m S_m - \mu_m S_m \tag{15a}$$
$$\dot{I}_m = \lambda_m S_m - \gamma_m I_m - \mu_m I_m - \delta_m I_m \tag{15b}$$
$$\dot{R}_m = \gamma_m I_m - \mu_m R_m \tag{15c}$$

$$\lambda_v = b_{vb}\beta_{vb}\frac{I_b}{N_b} + b_{mv}\beta_{vm}\frac{I_m}{N_m}\ ,\ \lambda_b = b_{bv}\beta_{bv}\frac{I_v}{N_v}\ ,\ \lambda_m = b_{vm}\beta_{mv}\frac{I_v}{N_v}\ . \tag{16}$$

The biting rates in these equations are defined as

$$b_{bv} = \frac{b}{N_v}\frac{\sigma_b N_b}{\sigma_m N_m + \sigma_b N_b}\ ,\ b_{mv} = \frac{b}{N_v}\frac{\sigma_m N_m}{\sigma_m N_m + \sigma_b N_b}\ , \tag{17a}$$
$$b_{vb} = \frac{b}{N_b}\frac{\sigma_b N_b}{\sigma_m N_m + \sigma_b N_b}\ ,\ b_{vm} = \frac{b}{N_m}\frac{\sigma_m N_m}{\sigma_m N_m + \sigma_b N_b} \tag{17b}$$

where the total biting rate b is defined for the two-bird models

$$b = \frac{\sigma_v N_v(\sigma_b N_b + \sigma_m N_m)}{\sigma_v N_v + \sigma_m N_m + \sigma_b N_b} \tag{18}$$

For example, b_{vb} is the number of times the mosquito bites a resident bird per day and b_{mv} is the number of times that a migratory bird is bitten by a mosquitoes per day. Note that the two-bird model biting rate can account for different availability and attractiveness of the birds to mosquitoes.

The forces of infection, λ_b and λ_m in (16) are modified from (4) to include the migratory bird. Both λ_b and λ_m are the product of the proportion of infected mosquitoes I_v/N_v, the total number of bites per bird, the proportion of bites that are on the considered bird species, $\sigma_i N_i/(\sigma_m N_m + \sigma_b N_b)$, and the probability of transmission per bite, β_{iv}. The total mosquito force of infection, λ_v (13), is the sum of the force of infection from each of the two bird species/types.

When the model includes migration, the simulation starts with just resident bird present, or $S_m = N_m = 0$, in the winter cycle at day $t = 0$ in the fall with the departure of the migrating birds. At day $t = 180$ in the spring, the migrating birds return. This cycle repeats, to capture the summer and winter seasons. We consider the situation where the same mosquito population is present year round with no changes to behavior or density (parameters). Although we could have assumed that the mosquito population is greater in one of the seasons, we wanted to first isolate the effects of the bird distribution and migration on model output.

2.2.1. *Basic reproductive number for the two-bird model.* Following the same procedure to define $\mathcal{R}_0$ as in the one-bird case, we first define $M = (E_v,\ I_v,\ I_b,\ I_m)^T$, identify F and V vector, and define the ODEs $\dot{M} = F - V$:

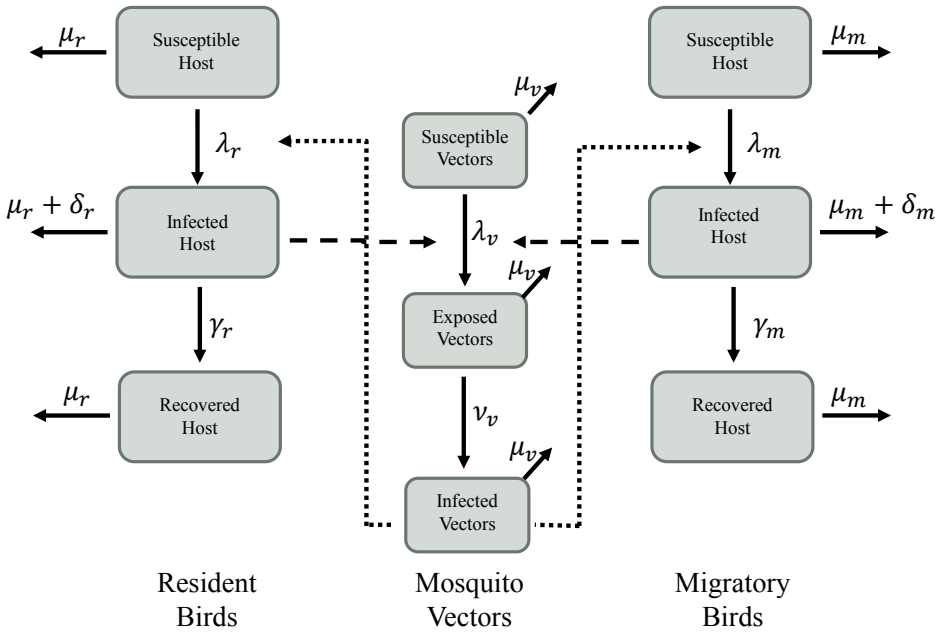

FIGURE 1. The mosquito infection is driven by both resident and migratory birds and one population infectious mosquitoes.

$$F = \begin{pmatrix} b_{vb}\beta_{vb}\frac{I_b}{N_b} + b_{mv}\beta_{vm}\frac{I_m}{N_m} \\ 0 \\ b_{bv}\beta_{bv}\frac{I_v}{N_v} \\ b_{mv}\beta_{mv}\frac{I_v}{N_v} \end{pmatrix}, \quad V = \begin{pmatrix} \nu_v E_v + \mu_v E_v \\ -\nu_v E_v + \mu_v I_v \\ \gamma_b I_b + \mu_b I_b + \delta_b I_b \\ \gamma_m I_m + \mu_m I_m + \delta_m I_m \end{pmatrix}$$

Computing the Jacobians of F and V and evaluating at the disease-free equilibrium, we have

$$\mathbb{J}_{\mathbb{F}} = \begin{pmatrix} 0 & 0 & b_{vb}\beta_{vb} & b_{mv}\beta_{vm} & 0 & 0 \\ 0 & 0 & 0 & 0 & & \\ 0 & b_{bv}\beta_{bv} & 0 & 0 & & \\ 0 & b_{mv}\beta_{mv} & 0 & 0 & & \end{pmatrix},$$

and

$$\mathbb{J}_{\mathbb{V}} = \begin{pmatrix} \nu_v + \mu_v & 0 & 0 & 0 \\ -\nu_v & \mu_v & 0 & 0 \\ 0 & 0 & \gamma_b + \mu_b + \delta_b & 0 \\ 0 & 0 & 0 & \gamma_m + \mu_m + \delta_m \end{pmatrix}.$$

Extending the notation for the probability of an infected mosquito or bird becoming infectious and the time that they are infectious, used in 5, we define the time spent in the E_m and I_m states as $\tau_{im} = 1/(\gamma_m + \mu_m + \delta_m)$. The then inverse of $\mathbb{J}_{\mathbb{V}}$ can be expressed as

$$\mathbb{J}_{\mathbb{V}}{}^{-1} = \begin{pmatrix} \tau_{ev} & 0 & 0 & 0 & 0 & 0 \\ p_{iv}\tau_{iv} & \tau_{iv} & 0 & 0 & 0 & 0 \\ 0 & 0 & \tau_{ib} & 0 & & \\ 0 & 0 & 0 & \tau_{im} & & \end{pmatrix}$$

giving a next generation matrix of $\mathbb{J}_{\mathbb{F}}\mathbb{J}_{\mathbb{V}}{}^{-1} =$

$$\begin{pmatrix} 0 & 0 & p_{ib}b_{vb}\beta_{vb}\tau_{iv} & b_{vb}\beta_{vb}\tau_{ib} & b_{mv}\beta_{vm}p_{im}\tau_{im} & b_{mv}\beta_{vm}\tau_{im} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ p_{iv}b_{bv}\beta_{bv}\tau_{iv} & b_{bv}\beta_{bv}\tau_{iv} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ p_{iv}b_{vm}\beta_{mv}\tau_{iv} & b_{vm}\beta_{mv}\tau_{iv} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}.$$

The reproductive number is the spectral radius of this matrix is given by

$$\mathcal{R}_0 = \sqrt{\mathcal{R}_0^{vb} \times \mathcal{R}_0^{bv} + \mathcal{R}_0^{vm} \times \mathcal{R}_0^{mv}} \tag{19}$$

where

$$\mathcal{R}_0^{mv} = p_{iv}b_{vm}\beta_{mv}\tau_{iv} \ , \ \mathcal{R}_0^{bv} = p_{ib}b_{vb}\beta_{bv}\tau_{ib} \ , \tag{20a}$$

$$\mathcal{R}_0^{vb} = p_{iv}b_{vb}\beta_{vb}\tau_{iv} \ , \ \mathcal{R}_0^{vm} = p_{iv}b_{mv}\beta_{vm}\tau_{im} \ . \tag{20b}$$

Note that the reproductive numbers all have the same form: probability an infected bird or mosquito becomes infectious (p_{**}) *times* the number of times an infectious bird is bitten, or an infectious mosquito bites per day (b_{**}) *times* the probability that those bites will transmit the infection (β_{**}) *times* the number of days the bird or mosquito is infectious (τ_{**}). The product of these terms is the number of secondary infections created by introducing one infectious bird or mosquito into a fully susceptible population.

The sum is under the square root is because the mosquito bites are distributed among multiple bird populations. We take the square root of this sum because this is a branching process and the geometric average over the two infections stages (bird-to-mosquito and mosquito-to-bird) is the appropriate average.

This formula can generalized to include J bird species as

$$\mathcal{R}_0 = \sqrt{\sum_{j=1}^{J} \mathcal{R}_0^{vj} \times \mathcal{R}_0^{jv}} \ , \text{ where } \mathcal{R}_0^{vj} = p_{ij}b_{vj}\beta_{vj}\tau_{ij} \text{ and } \mathcal{R}_0^{jv} = p_{iv}b_{jv}\beta_{jv}\tau_{iv} \ .$$

This basic reproductive number reduces to the $\mathcal{R}_0$ found similar studies by Cruz-Pacheco et al. [14] and Abdelrazec et al. [1], if instead of using the nonlinear biting rate model 13, we use a simplier linear biting rate used in their studies and if we assume there is no mosquito biting preference.

2.3. **Parameters.** The ranges for the parameter values (Table 2) were estimated from the referenced papers, research involving the laboratory- and field-based experimental infections of live birds and mosquitoes. When published estimates for parameters were not available, they were estimated for the model to be consistent with the population being modeled. For example, ψ_v, the recruitment (birth) rate of the mosquito population was chosen to equal the death rate of the mosquito population resulting in a constant mosquito population, an assumption that simplifies the model at the expense of making it less realistic. We assume that WNV infection does not change the mortality of the short-lived mosquitoes. After initial analysis, we also make the simplifying assumption that recovered birds are immune for life ($\rho_m = \rho_b = 0$). We leave these parameters in the model to allow investigating the sensitivity of this assumption in situations where the immunity may wain in the hosts. A bird's tolerance for mosquito bites (σ_m and σ_b) represent a preferred and alternative host situation. These values were varied to test different hypotheses within this study.

TABLE 2. Parameter Baseline Values and Ranges

Parameter	Value	Range	Units	Reference(s)
β_{vb}	0.9738	0.65-1	bite^{-1}	[44, 43, 45, 13, 22]
β_{vm}	0.8875	0.65-1	bite^{-1}	[44, 43, 45, 13, 22]
β_{iv}	0.468	0.32-0.58	bite^{-1}	[44, 45]
ψ_v	0.0666	-	day^{-1}	Assumption*
μ_v	0.0666	0.05-0.33	day^{-1}	[48]
ν_v	0.1	0.07-0.14	day^{-1}	[39]
δ_v	0	-	day^{-1}	Assumption**
σ_v	0.25	0.125-0.33	bites $\times$ day^{-1}	[48, 37]
ψ_m	0.0014	0-0.033	day^{-1}	[33]
μ_m	0.0014	0-0.01	day^{-1}	[18, 5]
ν_m	0.5	0.33-1	day^{-1}	[26]
γ_m	0.2222	0.2-0.25	day^{-1}	[26]
ρ_m	0	-	day^{-1}	Assumption**
δ_m	0	0-0.125	day^{-1}	[26]
σ_m	30	0-∞	bites $\times$ day^{-1}	Assumption***
ψ_b	0.0164	0-0.022	day^{-1}	[40]
μ_b	0.0014	0-0.01	day^{-1}	[5, 40]
ν_b	0.5	0.33-1	day^{-1}	[26]
γ_b	0.1	0-0.2	day^{-1}	[26]
ρ_b	0	-	day^{-1}	Assumption**
δ_b	0.26	0.125-0.33	day^{-1}	[26, 31]
σ_b	5	0-∞	bites $\times$ day^{-1}	Assumption***

Subscripts m and b refer to migratory and resident bird species, v refers to the mosquito vector. We assume that the susceptible mosquito population size is stable (*) and ρ_b is approximated based on the local ecology [36] of the environment (**), and the biting rates of the resident and migratory birds, σ_b and σ_m, are defined to test specific dynamics of preferred and alternate host preferences for mosquitoes (***).

We analyze $\mathcal{R}_0$ for the baseline values in Table 2 with a ratio of 10 mosquitoes for each bird, and find that $\mathcal{R}_0 = 1.78$ when the resident bird alone is present ($N_m = 0$); $\mathcal{R}_0 = 2.99$ when only the migratory bird population is present; and $\mathcal{R}_0 = 3.46$ when we have both resident and migratory birds present in equal numbers. The basic reproduction number for both present is higher than for either alone because the number of bites per bird, b, is modified by the relative number of bites tolerated by each species when they are both present, incorporating mosquito preference for the migratory bird into the reproduction number for both species. Note that in the two-bird Winter and Summer models, the basic reproductive numbers are different since the populations changes with the seasons. The disease-free equilibrium is unstable ($\mathcal{R}_0 > 1$) for each bird population alone.

We use forward sensitivity analysis [3, 2] to determine the relative importance of model parameters on $\mathcal{R}_0$ for both low and high ranges of a parameter into the $\mathcal{R}_0$ expression. The most important factor in the model is the average number of bites from an infectious mosquito. This is a function of the lifetime of the mosquito $1/\mu_v$ and the mosquito's biting rate σ_v. Many infected mosquitoes die before they become infectious, hence a small decrease in the average lifetime of a mosquito can

TABLE 3. Sensitivity analysis of $\mathcal{R}_0$ with respect to the parameters in the single bird population cases for resident (left) and migratory (right) bird parameter ranges from Table 2. The ratio $\Delta\mathcal{R}_0/\Delta P$ measures that rate of change (approximate derivative) of $\mathcal{R}_0$ with respect to the parameter P over the range of allowed values. We see that $\mathcal{R}_0$ is most sensitive to the biting rate and death rate of mosquitoes

Parameter	$\mathcal{R}_0$ Range	$\Delta\mathcal{R}_0/\Delta P$	Parameter	$\mathcal{R}_0$ Range	$\Delta\mathcal{R}_0/\Delta P$
β_{vb}	1.452–1.800	0.99	β_{vm}	2.555–3.169	1.75
β_{bv}	1.469–1.978	1.96	β_{mv}	2.469–3.324	3.29
μ_v	0.469–2.161	5.94	μ_v	0.835–3.632	9.99
ν_v	1.642–1.888	3.51	ν_v	2.756–3.173	5.96
σ_v	1.066–2.119	5.14	σ_v	1.553–3.847	11.19
μ_b	1.756–1.78	2.4	μ_m	2.930–2.995	6.5
γ_b	1.573–2.089	2.58	γ_m	2.816–3.146	6.6
δ_b	1.626–2.245	3.02	δ_m	2.391–2.986	4.76

have a big impact on $\mathcal{R}_0$. These results agree with detailed sensitivity analysis studies of this model for malaria, dengue, and chikungunya [10, 30].

3. Simulations and analysis.

3.1. One-bird model simulations.

We start by introducing one exposed mosquito into a single bird population. We use the baseline values (Table 2), the initial conditions $S_v = 9,999$, $E_v = 1$, $I_v = 0$, $S_b = 1,000$, $I_b = 0$, and $R_b = 0$, and did not include seasonality (time dependence) in the parameter values.

For the resident bird parameters, $\mathcal{R}_0 = 1.78$ and the outbreak reaches its maximum at day $t = 147$ when there are $I_v = 907$ infected mosquitoes. The resident bird population, in contrast, sees its highest infection levels at time $t = 130$ days when $I_b \approx 64$. Despite what may seem like a relatively small outbreak size, the resident population declined from an initial population of 1000 to 294 at time $t = 173$ days. After this, the bird populations recovers slightly and after some oscillation stays at a size of 356 (the oscillations occur after the 400th day). A low-level endemic infection persists and the recovered class of resident birds will remain higher than the susceptible class.

For the migrant bird parameters, $\mathcal{R}_0 = 2.99$, as expected, the outbreak grows faster than before. The infectious mosquito population peaks on day 84 with a total of 1519 infectious mosquitoes. The infectious migrant bird population reaches its peak of 160 infectious birds at time 70. The migrant population stays constant for the duration because there is no disease-induced death and the birth rate equals the natural death rate. Eventually most of these birds become immune (recovered class) so transmission is minimal while the susceptible class grows. In multi-year simulations, there are initially minor outbreaks around days 389 and 706, but eventually the simulation reaches an endemic equilibrium with 5.5 infectious birds, 114 susceptible birds, and 880 recovered birds.

The migrant bird outbreak is much larger and occurs earlier, peaking on day 84 instead of day 148 (based on maximum infectious mosquitoes) and reaches a maximum of 1519 infectious mosquitoes as opposed to 907 in the resident bird

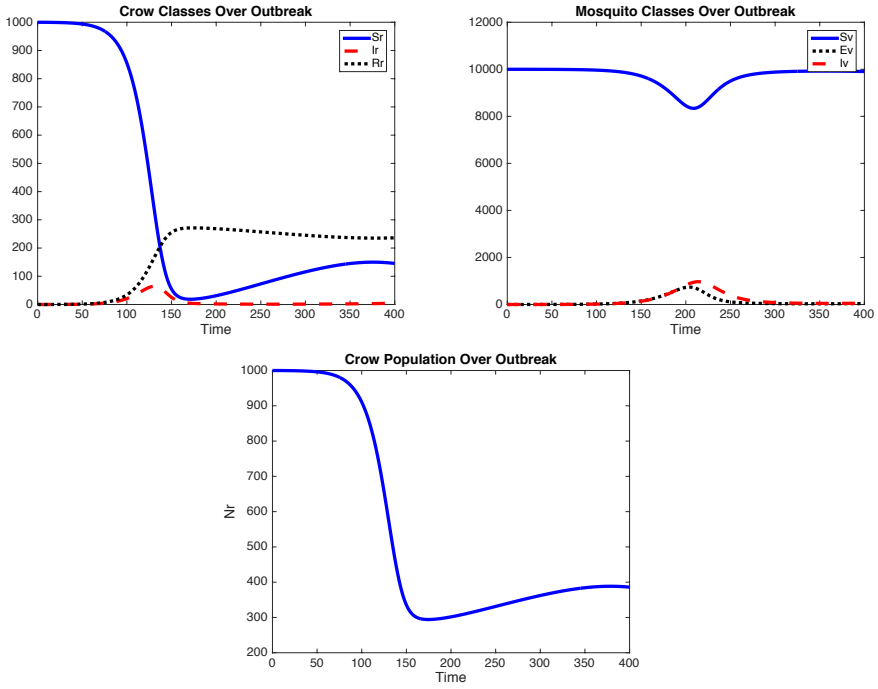

FIGURE 2. In this resident only bird (Crow) simulation, the evolution of S_r (solid blue line), I_r (dashed red line), and R_r (dotted black line) show that the bird deaths due to disease are high, as characterized by the die off at near $t = 100$ days. The outbreak peaks at time 148 with 907 infectious mosquitoes. We used initial conditions of $10,000$ mosquitoes, with one mosquito in the exposed class and 1000 susceptible resident birds. The die off observed in simulations is qualitatively consistent with deaths and reduced populations seen in crows due to WNV infection.

simulation. The migrant bird population is free of disease-induced death so it never decreases from its initial population of 1000, whereas the resident bird population is significantly reduced due to disease and reaches an equilibrium value of 356, roughly a third of its original size.

3.2. **Two-bird species model simulations.** In the next simulation, we evaluate the effect of introducing one exposed mosquito into a disease free two-bird population. We begin with a simulation with both resident and migratory birds present year round to separate the effects of the differences between the birds from the migration cycle. We use the baseline parameters from Table 2 and maintain a $10:1$ mosquito-to-bird population ratio, with 500 birds of each species and $10,000$ mosquitoes. Specifically, the initial conditions are $S_v = 9,999$, $E_v = 1$, $I_v = 0$, $S_b = 500$, $I_b = 0$, $R_b = 0$, $S_m = 500$, $I_m = 0$, and $R_m = 0$. See Figure 4 for plots of the results.

When two bird species are present, $\mathcal{R}_0^s = 3.46$, which is higher than either of the one-bird populations alone. As expected, the higher $\mathcal{R}_0^s$ simulation has an earlier outbreak. The infected mosquito I_v population peaks at time 71 with 1472 infectious mosquitoes. The total migrant bird population stays constant throughout the

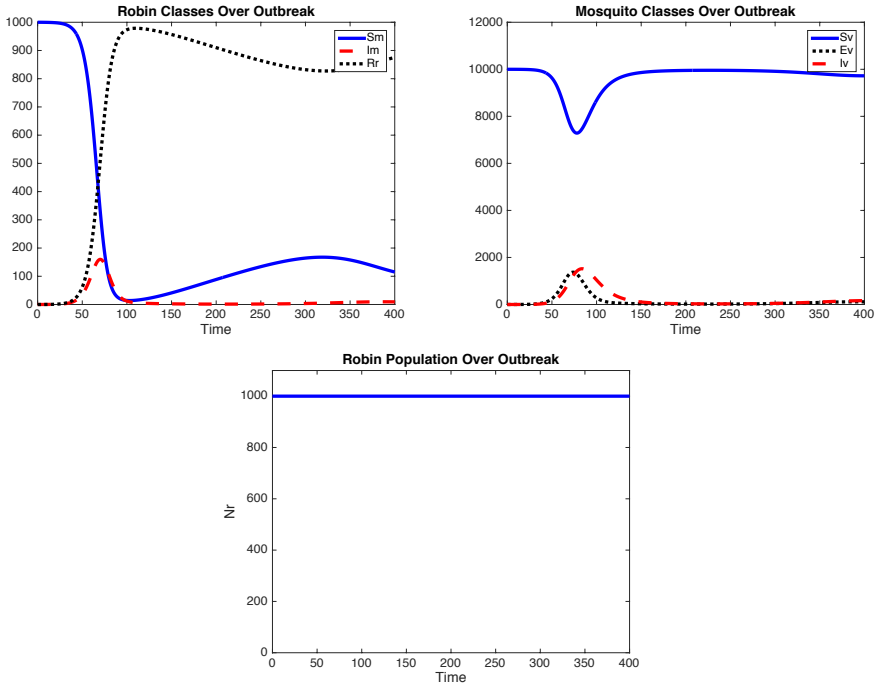

FIGURE 3. In this migratory only bird (Robin) simulation, with no deaths due to disease, the evolution of S_r (solid blue line), I_r (dashed red line), and R_r (dotted black line) has a larger outbreak that peaks faster than in the resident only bird case in Fig. 2. The outbreak peaks at time 84 with 1519 infectious mosquitoes. We used initial conditions of $1,000$ susceptible migratory birds, and $10,000$ mosquitoes,with 1 in the exposed class.

outbreak and after the outbreak itself. By time 80 or so, the migratory population becomes mostly recovered. The endemic state steadies with values of $S_m = 460$, $R_m = 37$, and $I_m = 3$. In contrast, the resident population faces high levels of disease-induced death from the virus. From an initial population size of 500 it decreases to a low of 208 before eventually reaching equilibrium at 252.

The reproductive number is higher in this case than it was in either single bird case. Because both species are highly competent, the epidemic does not suffer from the dilution effect [41] and has a higher reproductive number. Consequently, the outbreak happens sooner than in either single bird case. Furthermore, the outbreak has more infectious mosquitoes than a resident bird population alone has, but slightly less than in a solely migrant bird population. Also, despite the higher reproductive number and the larger magnitude of the outbreak, the resident population's relative decrease is less than in the simulation where there is only one bird class. In the two bird simulation, residents decreased to 252 from 500 originally, or about a 49.5% decrease. When there are only resident birds, then they decreased to 294 from $1,000$, or about a 70.6% decrease. So although increased biodiversity in these scenarios does not decrease the magnitude of the outbreak, it may help the highly susceptible resident population survive outbreaks.

3.2.1. *The importance of the mosquito to bird ratio.* In the previous simulations, we maintained a high mosquito to bird population ratio of $10:1$. We now compare

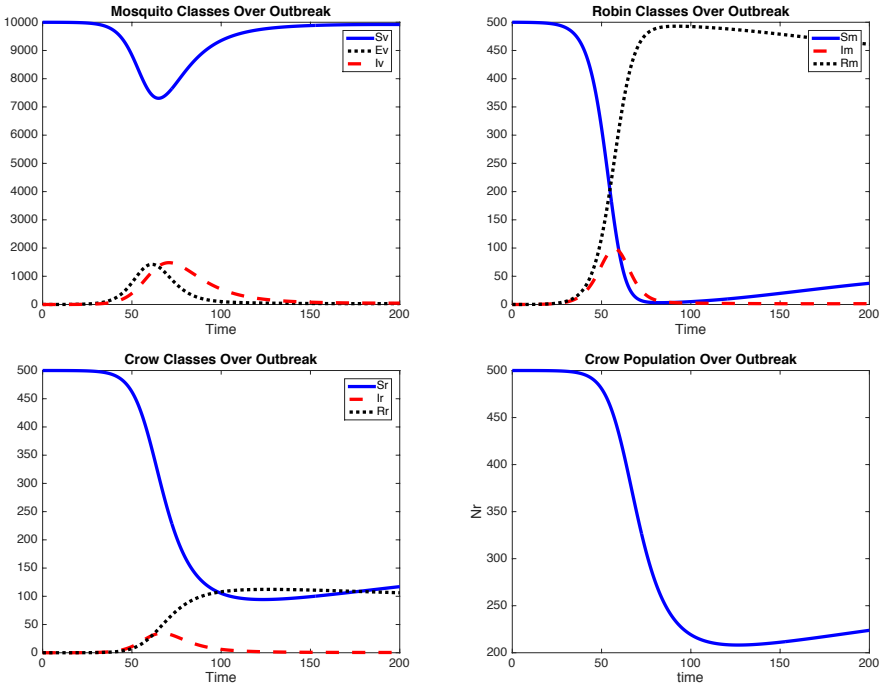

FIGURE 4. When both migratory and resident species are present, the resident bird deaths are less pronounced than when the migratory birds were not present and the outbreak is larger and peaks earlier. The outbreak peaks at time 71 with 1,472 infectious mosquitoes. We used initial conditions of 10,000 mosquitoes, 1 of which was in the exposed class the rest susceptible, 500 susceptible resident birds, and 500 susceptible migrant birds.

these simulations to situations with the lower mosquito bird ratio of $(4:1)$. The infected mosquitoes are shown in Figure 5 during an outbreak for four different cases.:

- The *baseline case* for our simulations when $S_v = 9,999$, $E_v = 1$, $I_v = 0$, $S_b = 500$, $I_b = 0$, $R_b = 0$, $S_m = 500$, $I_m = 0$, and $R_m = 0$. and the cases we will refer to as migrant dominant, resident dominant, and even.
- The *migrant dominant case* has the same baseline parameter values but initial populations of $S_v = 9,999$, $E_v = 1$, $I_v = 0$, $S_b = 500$, $I_b = 0$, $R_b = 0$, $S_m = 2,000$, $I_m = 0$, and $R_m = 0$.
- The *resident dominant case* has the same baseline parameter values and initial conditions $S_v = 9,999$, $E_v = 1$, $I_v = 0$, $S_b = 2,000$, $I_b = 0$, $R_b = 0$, $S_m = 500$, $I_m = 0$, and $R_m = 0$.
- The *even scenario case* has the same baseline parameter values and initial conditions $S_v = 9,999$, $E_v = 1$, $I_v = 0$, $S_b = 1,250$, $I_b = 0$, $R_b = 0$, $S_m = 1,250$, $I_m = 0$, and $R_m = 0$.

Figure 5 illustrates scenarios when reproductive numbers for the migrant dominant case ($\mathcal{R}_0 = 2.12$), the resident dominant case ($\mathcal{R}_0 = 2.59$), and when both bird populations are equal ($\mathcal{R}_0 = 2.39$), are all less than our baseline value ($\mathcal{R}_0 = 3.46$). As expected, the higher the $\mathcal{R}_0$ value of the system, the earlier the outbreak occurs,

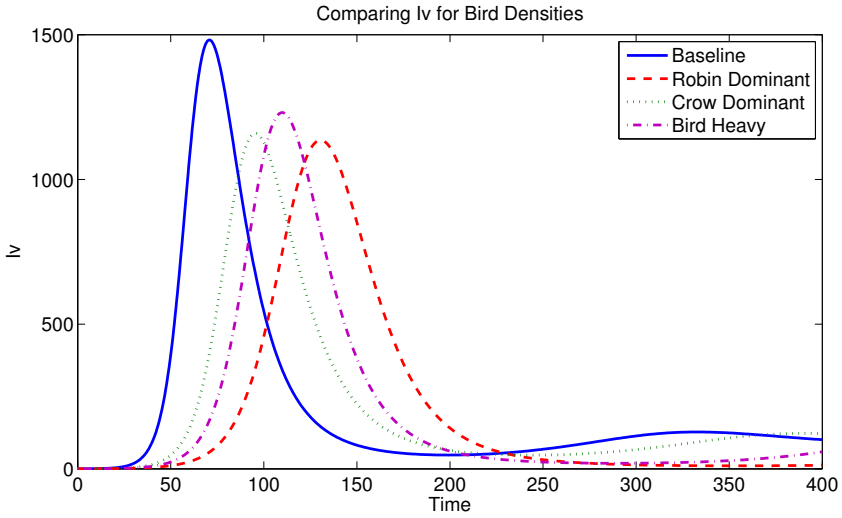

FIGURE 5. The baseline case with higher mosquito-to-host ratio has a much higher, and earlier, peak than the cases with larger bird populations. Surprisingly, the resident dominant, migrant dominant, and even cases behave similarly to each other for the lower mosquito-to-host ratio (bird heavy) scenarios.

but the same rule does not apply to the magnitude of the outbreak. It appears that in heavy bird (lower vector-to-host ratio) cases, a large population with evenly mixed migrants and residents has a larger outbreak size than the resident dominant or the migrant dominant cases.

When only one bird population is present, and there is a high mosquito-to-host ratio, then migrant birds have a much higher $\mathcal{R}_0$ value than residents. All of the cases with a high bird population have later, more prolonged, and smaller outbreak peaks than the baseline case. In all of our simulations, regardless of the mixture of bird populations, a decreased mosquito to bird ratio limited the magnitude of an outbreak and delayed the outbreak. This finding supports reducing the mosquito population, and hence decreasing the mosquito-to-host ratio.

3.3. **Two birds with migration.** The simulation is initialized with both bird populations present, referred to as "summer" in the figures. The migratory birds leave the simulation after the 6 summer months for 6 months "winter" migration. The infected migratory birds that leave for the winter return as recovered and immune the following year. The other parameters and initial conditions are set to the baseline conditions used in the previous simulations, $S_v = 9,999$, $E_v = 1$, $I_v = 0$, $S_b = 500$, $I_b = 0$, $R_b = 0$, $S_m = 500$, $I_m = 0$, and $R_m = 0$.

The first season's dynamics (Figure 6) are identical to the two-bird simulation without migration and $\mathcal{R}_0$ at this time is 3.46. There is an early outbreak, at around time $t = 70$ days, where the majority of migrant birds become immune and the resident population decreases by about half. At time $t = 180$ days, the migrant birds are removed, reducing their total population from N_m to 0. At this time, the simulation has only 50 infectious mosquitoes and the disease incidence declines. (Remark: The effective reproduction number, $\mathcal{R}_e$ measures that number

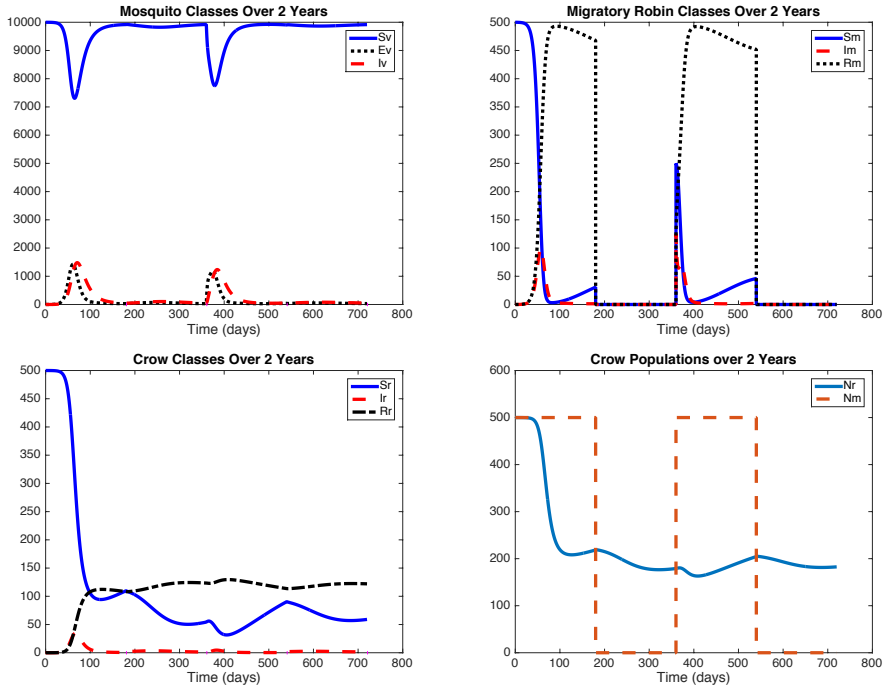

FIGURE 6. When the migratory birds arrive and stay for six months each year, the outbreak itself is the same as in the previous simulation and the low levels of the virus never build up to another large outbreak. We used initial conditions of 10,000 mosquitoes, with 1 mosquito in the exposed class, 500 susceptible residents, and 500 susceptible migrant birds. We assumed that half of the the migratory birds were susceptible when they arrived.

of secondary infections caused by introducing one additional infected mosquito and is calculated by studying the next generation matrix for the equations linearized about the current state. When the migratory birds leave, $\mathcal{R}_e = 0.643 < 1$.)

Even though the disease incidence decreases during the winter months, the disease continues to circulate at low levels and does not die out completely. Also, resident mortality continues, even with the low levels of infection. During this time, the resident birds are the only food source for the same number of mosquitoes. The migratory birds return each year and all previously infectious migrant birds are advanced to the recovered class. This has little impact since there were few infectious migratory birds at the time of migration. The addition of migratory birds as feeding targets for mosquitoes decreases the resident bird mortality-due-to-disease rates and their total population increases to the pre-winter levels of 220 individuals. This pattern quickly settles into a periodic annual oscillation. The mosquito and migrant bird populations behave similarly to the two-bird case without migration.

The migration simulations in Section 3.3 do not result in repeated outbreaks every year. We next considered the case when all returning migrating birds are susceptible to WNV, Figure 7. In these simulations, all the initial conditions, and parameter values all are identical to the previous simulation except that when the migratory birds return they all return as susceptible. Although this is not necessarily realistic, it provides a contrast to the previous simulation and may approximate both death

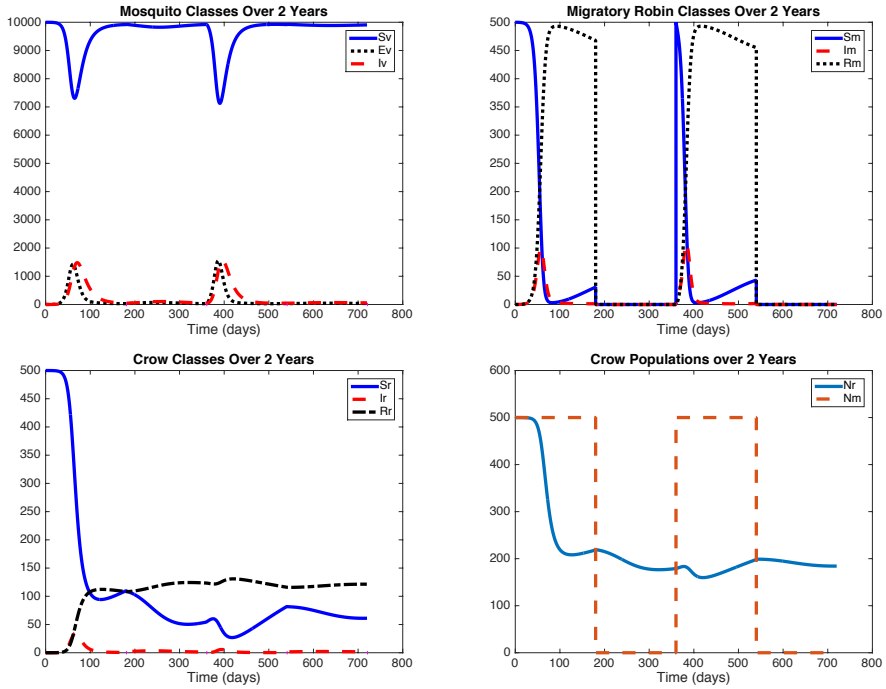

FIGURE 7. When all the migrants return susceptible every year, seasonal outbreaks of WNV occur as expected and observed in North America. The initial conditions were 10,000 mosquitoes, 1 of which was in the exposed class the rest susceptible, 500 susceptible residents, and 500 susceptible migrants.

during migration and during the "winter" season as well as a pulse of susceptible young of year at the beginning of the "summer" season.

The first season of the simulation is identical to the previous simulations. There is an early outbreak of roughly 1400 infectious mosquitoes, the resident population decreases dramatically and the migratory birds become mostly recovered and immune. When the migrant birds are removed at 6 months, the resident population decreases even further as in the previous simulation. The migratory birds are repopulated each year as fully susceptible. Interestingly, this leads to another outbreak slightly smaller than the initial one. The resident population decreases before starting to recover again after the outbreak ends and almost all the migrants gain immunity. Every year this same dynamic occurs creating a seasonal outbreak pattern. This pattern is similar to what actually occurs in North America each year, yet the critical assumption that the migratory birds start each new summer season susceptible is not entirely realistic and it is rare for an entire bird population to become immune by the end of a high transmission season. It does, however, suggest one mechanism driving seasonal outbreaks.

3.3.1. *Including mammals in the simulations.* Most mammals are considered to be dead-end hosts in the WNV system, meaning that although they can contract the virus, they do not reinfect mosquitoes that bite them. The previous simulations included only competent bird host species. *Culex pipiens* and other WNV mosquitoes are often opportunistic feeders and while they frequently bite birds, they also bite

mammals when available. We also considered second dead-end host species, such as humans or horses, that do not infect mosquitoes. In these simulations, not reported here, the timing of magnitude of outbreak was very similar to our two-bird simulation in Figure 4, even though there are no reinfections from the dead-end host population. This implies that while the resident species may be an indicator of disease prevalence, these cases have little effect on the disease dynamics as a whole.

4. **Summary and conclusions.** There have been annual outbreaks of WNV since it was first observed in North American in 1999. Despite the efforts of mosquito control programs and public health campaigns, these outbreaks have not diminished. Variations in the ecology, seasons, bird distributions and mosquito density all contribute the the complexity of the North American WNV transmission system.

Our model is an idealized representation of WNV transmission in a single area to give insight into how changes in bird migration patterns, distributions, and mosquito density affect WNV dynamics. We extended the Wonham et al. [51] and Abdelrzec et al. [1] models to examine the effect of the migratory and resident bird populations on the transmission dynamics over multiple seasons. The modified model includes a seasonal carrying capacity and the Chitnis et al. biting rate [9] to accurately account for situations when the bird to mosquito ratio was very large, or small.

After analyzing a simplified one-bird population model, we extended the model and analysis to include a second migratory bird population that is a preferred host for the mosquitos. Our sensitivity analysis of the basic reproduction number, $\mathcal{R}_0$, showed that the two most important parameters are the mosquito feeding rate, the mosquito death rate, and the vector-to-host ratio. Our results agree with the analysis of Abdelrazec et al. [1] that the size of the output is very sensitive to the host diversity and vector competence in transmitting the virus.

We first considered the situations where there were just resident (e.g. American Crows) or migratory (e.g. American Robin) birds. The migrants had a higher $\mathcal{R}_0$ and thus the disease spread more quickly, causing an earlier outbreak and a larger outbreak when compared with the resident simulation. We found that the resident population's effect on $\mathcal{R}_0$ is relatively small when a preferred host is present.

An increase in dead crows and other highly susceptible birds is often one of the first indicators of the presence of WNV in a new area. However, we found that the high disease-induced death rate of the resident species reduced their ability to reinfect mosquitoes and helped mitigate the spread. That is, our model suggests that although the resident birds are often the first indicator that WNV is entering a new area, the migrant hosts with lower death rates might be a more important driver for sustaining an outbreak.

We studied the impact of reducing the mosquito population, e.g through spraying, and found that decreasing the mosquito-to-bird ratio from $10:1$ to $4:1$ significantly slowed and diminished the summer outbreak. Since climate and other environmental factors can significantly affect mosquito density and thus biting intensity, changes in the mosquito-to-bird ratio could play a large role in variation in outbreak size and timing. This is one explanation for correlations found between winter temperatures (which affect mosquito population dynamics) and WNV risk [29, 38].

We examined the complementary roles of migrating and resident birds in one area with mosquito feeding preference for the migrant bird species. We estimated the equation parameters based on model ecology, the mosquito species, the seasonal effects, and included a biting rate that can capture both high and low mosquito densities. The resident population decreases during the high transmission season, and

then recovers during low transmission season. In the two-bird migration scenarios, we observed that if the recovered/immune population stays consistently high then there will be no large annual outbreaks. However, if many of the returning migratory birds are susceptible, then they can provide the virus the needed susceptible hosts to produce seasonal outbreaks. This occurs when there is a high turnover in the migratory bird population caused by the death of adult birds and a significant population of susceptible young birds each season.

Most of our parameter values are similar to Abdelrzec et al. The one exception is that they assume the death rate of non-corvids over a much wider range of values, while we assume that the non-corvid migratory species has a negligent death rate due to WNV. Although the Abdelrzec et al. [1] model only considered one season, our analysis agreed with their assessment that bird biodiversity needs to be a key component models estimate human risk for WNV and the effects of WNV on bird populations.

Our threshold conditions agree with the results of Cruz-Pacheco et al. [14], although they defined the basic reproduction number as the square of the R_0^2 defined in our analysis. Our basic reproduction number for crows is $(1.78)^2 = 3.16$, which is on the lower end of R_0 estimates for corvids in [14], since we assume β_v is smaller. We observe, as did [14] that although crows are better transmitters they produce fewer secondary infections than robins because their death rate from disease is so high. We include multiple bird species in our model simultaneously as well as seasonal migration. The seasonal results confirm assertions that considering dead crow numbers alone will not accurately reflect the outbreak size. [16] also includes seasonality in the mosquito species in their WNV model for one bird species. We consider discrete seasonality in the bird populations with only one species migrating while another is a resident all year.

We used American Crows and American Robins as representative competent species. The model could be extended to include other species with varying competence and to account for nestlings and young birds. Our model would be more realistic if we matched the abundance of multiple bird species to their actual relative abundances for specific regions. In the interest of isolating the effects of migrating and resident birds, the mosquito population was kept constant. Also, even though the mosquito biting rates and death rates, birth rates, and WNV transmission rates are temperature sensitive, they were kept constant in the model. The model can be extended to include mosquito population seasonality and temperature dependence, although this would add additional layers of complexity in understanding the competing forces.

The model forecasts are sensitive to the parameters for the susceptibility of birds to WNV. The avian experimental infection research used to define these parameters relied on small sample sizes. More comprehensive data on viral levels over the course of infection, loss of immunity, and natural population densities and behavior of the main North American avian species would lead to much more accurate modeling efforts. Additionally, since feeding preference had such a strong impact in the simulations, research further detailing the feeding preferences of the main North American WNV mosquito species would be valuable. WNV is, and will continue to be, a pressing topic for epidemiology and public health professionals.

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