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Modeling the Effects of Passive Immunity in Birds for the Disease Dynamics of West Nile Virus

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Abstract

West Nile Virus (WNV) is a mosquito-borne virus that circulates among birds but also affects humans. Migrating birds carry these viruses from one place to another each year. WNV has spread rapidly across the continental United States resulting in numerous human infections and deaths. Several studies suggest that larval mosquito control measures should be taken as early as possible in a season to control the mosquito population size. Also, adult mosquito control measures are necessary to prevent the transmission of WNV from mosquitoes to birds and humans. To better understand the effective strategy for controlling affected larval mosquito population, we have developed a mathematical model using a system of first order differential equations to investigate the transmission dynamics of WNV in a mosquito-bird-human community. We also incorporated vertical transmission in mosquitoes and passive immunity in birds to more accurately simulate the spread of the disease.

Keywords: SIR model, passive immunity, West Nile Virus

1 Introduction

West Nile Virus is a vector transmitted disease transferred from infected mosquitoes to other animals such as birds and humans. From 1999 to 2012 the virus caused over 1,500 deaths [23] and the most dramatic outbreak of the virus in North America occurred during the summer of 2002 [25]. The virus crossed the Mississippi River and infected the Pacific Coast region of the United States, bringing the total number of infected human cases to 4,156 and resulting in 284 deaths [25]. The economic burden of West Nile Virus is significant [4, 9]; the cost of treating and maintaining patients with West Nile Virus was \$778 million from 1999 to 2012 [4]. Thus, it is imperative that we contain this disease.

Mathematicians have modeled the spread of West Nile Virus using SIR and SEIR models [9, 17, 19, 22]. Most notably, they have simulated the use of pesticides on mosquito populations to test the effect of West Nile Virus on birds and humans [22]. The CDC recommends using multiple abatement strategies to control the local mosquito population. One such strategy incorporates the use of two different insecticides: adulticides (targets mature mosquitoes) and larvicides (targets mosquito larvae) [7]. Adulticides on their own are shown to have a great effect on lowering the mosquito populations [22]. Naturally, when sprayed more often, such as weekly instead of monthly, the mosquito population falls more drastically

and is controlled faster [22]. Since mosquitoes are the primary carrier of the disease, when their populations are lowered, we see a lower spread of the virus [22, 24]. Birds circulate the disease amongst themselves and mosquitoes [22, 24]. An infected bird flies into a new area and susceptible mosquitoes bite the infected bird, transmitting the disease to the mosquito [22, 24]. Infected birds will also fly to other areas, spreading the disease across continents, while some will remain in the same area, infecting more mosquitoes in the community [5]. Those mosquitoes will go on to bite a susceptible bird, giving them the disease and the cycle repeats itself perpetually [22, 24]. Mosquitoes are also able to bite humans and transmit West Nile Virus to them [22, 24]. All of these elements are necessary to create a model of how the disease spreads. However, there are two important elements that previous models have overlooked: vertical transmission and passive immunity [1, 2, 17].

Most mathematical models of West Nile Virus have focused on how mosquitoes spread the virus themselves and how birds are quick to fall ill to the virus [9, 17, 19, 22]. Yet, mosquitoes can spread the disease not only by infecting birds and humans, but also by passing the disease onto their larvae, a process known as vertical transmission [2, 17, 18]. On the other hand, once a bird recovers from West Nile Virus, it is immune to the disease. The bird has a high chance of passing its immunity down to its young, a process known as passive immunity [1, 20]. Many bird species develop passive immunity including house sparrows [21], flamingos [3], rock pigeons [14], and Eastern

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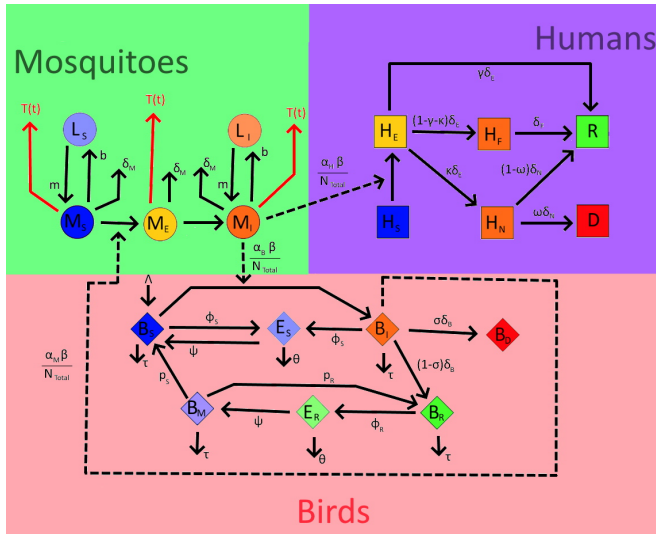


Figure 1: Schematic diagram of the model featuring all eighteen populations subdivided into three categories: mosquitoes represented by circles, birds represented by diamonds, and humans represented by squares. The dashed lines showcase interactions between species while the solid lines showcase interactions within the same species. Note the passive immunity cycle in the bird category.

screech-owls [15]. Both vertical transmission and passive immunity are essential parts of simulating the dynamics of West Nile Virus precisely. Vertical transmission depends on the overall mosquito population and increases the spread of disease while passive immunity depends on both the bird population and a bird’s chances of recovering from the disease, slowing disease spread. The goal of our work is to give insight to the importance of passive immunity in birds and to show how it is incorporated in the disease dynamics.

2 Model

Our model utilizes a system of eighteen ordinary differential equations (ODEs) based off of [22] that act as a modified SEIR model, explained in the schematic diagram in Figure 1. There are three main categories: birds, mosquitoes, and humans. The birds category highlights passive immunity, the mosquitoes category highlights vertical transmission, and these important additions to the model affect the humans category. Every category interacts with at least one other category in order to properly simulate the spread of West Nile Virus.

The bird category contains seven sections: susceptible eggs (E_S), immune eggs (E_R), immune birds (B_M), susceptible birds (B_S), infected birds (B_I), recovered

birds (B_R), and dead birds (B_D). Susceptible and infected birds lay susceptible eggs. These eggs hatch and grow into susceptible birds that are capable of developing West Nile Virus. Birds that have been infected with and subsequently become immune to the virus lay recovered eggs. The process of adult bird populations passing their immunity onto their young is passive immunity; recovered eggs hatch into the temporary state of immune birds [20]. This state makes the birds immune to West Nile Virus for a specific amount of time before they join the susceptible bird category. Susceptible birds are not infected with the disease but are capable of developing West Nile Virus [22]. Infected birds are currently suffering from the disease and are capable of spreading the virus to susceptible mosquitoes [22]. Birds move into the recovered category after they are no longer hosting the disease [22]. Once a bird is recovered, it is permanently immune to the disease [22]. Recovered birds are also able to pass their immunity onto their young if they reproduce. The dead birds category records only birds that have died from West Nile Virus [22].

The mosquito category contains five sections: susceptible larvae (L_S), infected larvae (L_I), susceptible mosquitoes (M_S), exposed mosquitoes (M_E), and infected mosquitoes (M_I). Susceptible and exposed mosquitoes lay susceptible larvae. These larvae have a chance of maturing into susceptible, healthy adult mosquitoes. Infected mosquitoes lay infected larvae [2]. Due to vertical transmission, infected mosquitoes have the ability to pass West Nile Virus onto their young [2]. In turn, these larvae will mature into infected mosquitoes. The difference between the categories of mature mosquitoes is that susceptible mosquitoes are healthy but biting infected birds will infect them, exposed mosquitoes bite an infected bird but are not yet infectious, and infected mosquitoes have West Nile Virus and are able to pass the disease onto birds and humans [22]. Infected mosquitoes are also able to vertically transmit their disease to their young, should they reproduce.

The human category contains six sections: susceptible humans (H_S), exposed humans (H_E), humans with West Nile Fever (H_F), humans with neuroinvasive disease (H_N), recovered humans (R), and dead humans (D). Susceptible humans are healthy, but able to catch West Nile Virus should they be bitten. Infected mosquitoes bite humans, exposing them to the disease. There is a chance that exposed humans recover before developing any symptoms of West Nile Fever or neuroinvasive disease. However, humans are considered dead-end hosts and are unable to infect mosquitoes even if they are infected [19]. Recovered humans have recovered from either West Nile Fever or neuroinvasive disease and are immune to catching West Nile Virus again. Dead humans refers to humans who have perished from neuroinvasive disease.

$$\begin{aligned}
 \frac{dL_S}{dt} &= b(M_S + M_E) - mL_S - \delta_L L_S \\
 \frac{dL_I}{dt} &= bM_I - mL_I - \delta_L L_I \\
 \frac{dM_S}{dt} &= mL_S - \frac{\alpha_M \beta M_S B_I}{N_{\text{Total}}} - \delta_M M_S - T(t)M_S \\
 \frac{dM_E}{dt} &= \frac{\alpha_M \beta M_S B_I}{N_{\text{Total}}} - \eta M_E - \delta_M M_E - T(t)M_E \\
 \frac{dM_I}{dt} &= mL_I + \eta M_E - \delta_M M_I - T(t)M_I \\
 \frac{dE_S}{dt} &= \phi_S(B_S + B_I) + (1 - \mu)\phi_R B_R - \theta E_S - \psi E_S \\
 \frac{dE_R}{dt} &= \mu\phi_R B_R - \theta E_R - \psi E_R \\
 \frac{dB_S}{dt} &= \Lambda - \frac{\alpha_B \beta M_I B_S}{N_{\text{Total}}} + p_S B_M + \psi E_S - \tau B_S \\
 \frac{dB_I}{dt} &= \frac{\alpha_B \beta M_I B_S}{N_{\text{Total}}} - \delta_B B_I - \tau B_I \\
 \frac{dB_R}{dt} &= (1 - \sigma)\delta_B B_I + p_R B_M - \tau B_R \\
 \frac{dB_M}{dt} &= \psi E_R - (p_S + p_R)B_M - \tau B_M \\
 \frac{dB_D}{dt} &= \sigma\delta_B B_I \\
 \frac{dH_S}{dt} &= -\frac{\alpha_H \beta M_I H_S}{N_{\text{Total}}} \\
 \frac{dH_E}{dt} &= \frac{\alpha_H \beta M_I H_S}{N_{\text{Total}}} - \delta_E H_E \\
 \frac{dH_F}{dt} &= (1 - \gamma - \kappa)\delta_E H_E - \delta_F H_F \\
 \frac{dH_N}{dt} &= \kappa\delta_E H_E - \delta_N H_N \\
 \frac{dR}{dt} &= \delta_F H_F + (1 - \omega)\delta_N H_N + \gamma\delta_E H_E \\
 \frac{dD}{dt} &= \omega\delta_N H_N
 \end{aligned}$$

The parameter N_{Total} includes the total blood supply such that $N_{\text{Total}} = B_S(t) + B_I(t) + B_R(t) + B_M(t) + H_S(t) + H_E(t) + H_F(t) + H_N(t) + R(t)$. Also note that there is no birth nor natural death rate for humans like there is for birds and mosquitoes. Since the model takes places over a three month period, the death rate for humans is negligible. The other parameter values for this model are listed in Table 1.

The interaction between the mosquito, bird, and human populations is the base of our model of the spread of the virus. Infected mosquitoes lay eggs at a rate of b that become infected larvae. Once hatched, they mature at growth rate, m , into infected mosquitoes, continuing the cycle. Similarly, susceptible mosquitoes lay eggs at a rate of b which become susceptible larvae that grow

into susceptible mosquitoes at a rate of m . Once adults, susceptible mosquitoes bite at a rate of β , the average biting rate per day. We assume the mosquito biting rate for both humans and birds are the same [22]. Depending on the amount of infected birds, as $\alpha_M \beta / N_{\text{Total}}$ models, there is a chance that a susceptible mosquito bites an infected bird, exposing the mosquito to the virus. Once the virus enters their system, the susceptible mosquito moves into the exposed mosquito category. Exposed mosquitoes transition to infected mosquitoes at a rate of η and are then able to transmit West Nile Virus to birds and humans as well as participate in vertical transmission.

Infected mosquitoes are also able to bite susceptible birds and change them into infected birds as $\alpha_B \beta / N_{\text{Total}}$ models. After a bird is infected it has a chance to recover, which $(1 - \sigma)\delta_B$ models, and a chance to die from the virus, which $\sigma\delta_B$ models. Recovered birds pass their immunity down to their young, which $\mu\phi_R$ simulates. These eggs hatch and grow into birds at a rate of ψ . There is also a chance that they do not pass their immunity down to their young, which $(1 - \mu)\phi_R$ models. If this were to happen, they would lay susceptible eggs instead of recovered eggs. Susceptible eggs grow into susceptible birds at a rate of ψ . These birds lay susceptible eggs, which ϕ_S models.

West Nile Virus similarly affects humans: $\alpha_H \beta / N_{\text{Total}}$ represents the infected mosquitoes that bite susceptible humans and expose them to the disease [22]. Once a human is exposed to the disease, there is a chance that they will recover immediately from the disease at a rate of $\gamma\delta_E$. However, they may suffer from West Nile Fever at a rate of $(1 - \gamma - \kappa)\delta_E$. Most people recover from West Nile Fever at a rate of δ_F while others develop neuroinvasive disease at a rate of $\kappa\delta_E$. Once somebody has neuroinvasive disease, they either recover at a rate of $(1 - \omega)\delta_N$ or they die at a rate of $\omega\delta_N$.

Several studies suggest the use of adulticides in order to control the mosquito population [7, 9, 12, 22]. $T(t)$ models the adulticides. Within this function s equals the effectiveness of the adulticide being used. Our model uses s -values of 0%, 20%, 40%, and 80% treatment effectiveness as given by example in Figure 3. Simulating spraying at certain intervals, such as every seven, fourteen, or twenty-four days, makes this function dependent on time. For example, if we were to treat weekly, $T(t) = s$ when $t \equiv 0 \pmod{7}$ and $T(t) = 0$ when $t \not\equiv 0 \pmod{7}$.

We also set our immune birds to lose or permanently maintain their immunity at various time intervals based on their species (i.e. every 14 days for chickens, 10 days for owls, and 9 days for house sparrows [20]). After each time interval, p_S percent of the immune bird population moves to the susceptible bird category and p_R percent of the immune bird population moves to the recovered bird category [20].

Table 1: Parameters and their units, values, and sources for the system of ordinary differential equations that models the disease dynamics.

Parameter	Definitions	Units	Value	Source
b	Mosquito Birth Rate	Larvae/(Day · Adults)	0.045	[22]
m	Mosquito Maturation Rate	Adults/(Larvae · Day)	0.07	[23, 27]
δ_L	Natural Larval Death Rate	Day ⁻¹	0.027	[11]
α_M	Probability of Transmission from Birds to Mosquitoes	—	0.23	[12]
β	Bite Rate	Day ⁻¹	2.5	[22]
δ_M	Natural Mosquito Death Rate	Day ⁻¹	0.031	[11]
$T(t)$	$T(t) = s$; Success Rate of Adulticides	Day ⁻¹	varies	[22]
η	Virus Incubation Rate in Mosquitoes	Day ⁻¹	0.1	[10]
ϕ_S	Egg Laying Rate for Susceptible Birds	Day ⁻¹	varies	[15, 19, 21]
μ	Percent of Eggs Receiving Passive Immunity	—	varies	[15, 19, 21]
ϕ_R	Egg Laying Rate for Recovered Birds	Day ⁻¹	varies	[15, 19, 21]
θ	Natural Death Rate of Bird Eggs	Day ⁻¹	0.45	[15, 19, 21]
ψ	Maturation Rate of Bird Eggs	Day ⁻¹	varies	[15, 19, 21]
Λ	Recruitment Rate of Birds	Birds/Day	varies	[22]
α_B	Probability of Transmission from Mosquitoes to Birds	—	0.27	[12]
τ	Natural Bird Death Rate	Day ⁻¹	varies	[26]
δ_B	Rate of Recovery in Birds	Day ⁻¹	1/(4.5)	[16]
σ	Fraction of WNV Infected Birds Dying from the Disease	Day ⁻¹	0.72	[16]
α_H	Probability of Transmission from Mosquitoes to Humans	—	0.06	[6]
δ_E	Incubation Period in Humans	Day ⁻¹	1/4	[22]
γ	Fraction of Human Population that is Asymptomatic	Day ⁻¹	0.75	[8]
κ	Fraction of Human Population that Can Develop Neuroinvasive Disease	Day ⁻¹	0.01	[8]
δ_F	Rate of Recovery for WNV Fever in Humans	Day ⁻¹	1/14	[6]
δ_N	Rate of Recovery for Neuroinvasive Disease in Humans	Day ⁻¹	1/(37.5)	[13]
ω	Fraction of Humans Dying from Neuroinvasive Disease	Day ⁻¹	0.1	[8]
p_S	Percent of Immune Birds that Lose their Immunity	—	varies	[15, 19, 20, 21]
p_R	Percent of Immune Birds that Keep their Immunity	—	varies	[15, 19, 20, 21]

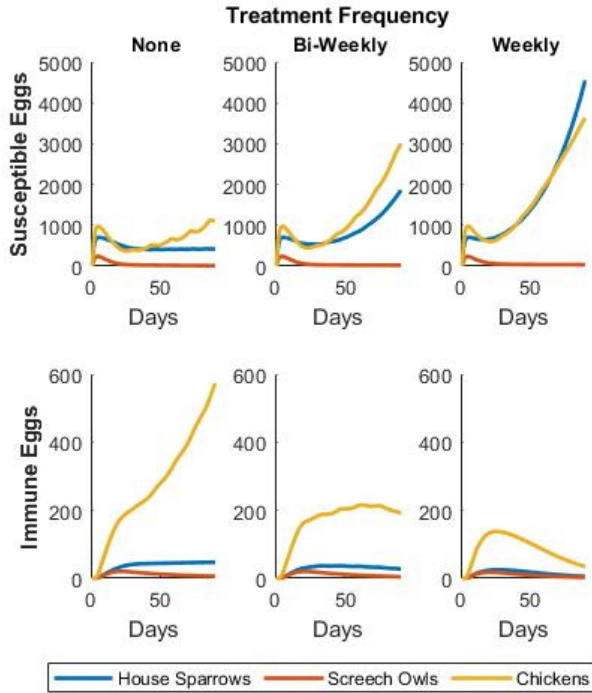


Figure 2: Susceptible and immune egg populations with an 80% treatment effectiveness against mosquitoes. Notice the treatment frequency increasing from no treatment to bi-weekly treatment to weekly treatment.

3 Results

3.1 Birds and Eggs

To show the effects of different hatching rates, growth rates, laying rates, and passive immunity rates, we selected three bird species (due to there being pre-existing literature of these species): house sparrows [21], screech owls [15], and chickens [19]. We then plotted them against different treatment effectiveness rates and treatment occurrence rates. Some differences of these species include chickens overall having the highest egg laying rate due to them being able to lay one egg per day. However, this means that their clutch size is only one, much smaller than both house sparrows which have a clutch size anywhere between four and six eggs and screech owls which have, on average, a clutch size of 3 eggs. Sparrows lay a clutch every 3–4 weeks while screech owls lay a clutch about once per month. The hatching rate for every bird species is also different. It is important to consider the average rates for birds in the specific community to obtain the most accurate results.

As seen in Figure 2, our simulation suggests that the amount of eggs that develop an immunity to West Nile

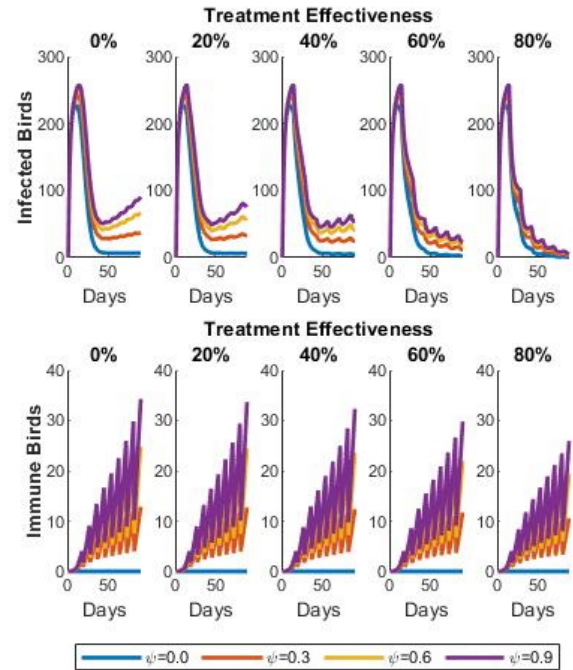


Figure 3: Infected and immune bird populations as affected by different ψ -values (0.0, 0.3, 0.6, 0.9) with treatment frequency being bi-weekly. The treatment effectiveness increases left to right at 20% increments. Note that these oscillations are from a bi-weekly treatment and from birds leaving the immunity category.

Virus through passive immunity is inversely proportional to the frequency of the treatment. Treating the mosquito population with adulticides leads to a smaller recovered egg population. This is due to the decrease in birds becoming infected with the virus in the first place, and thus leading to less birds that become immune because there are less recovered birds laying immune eggs. So, the more rampant West Nile Virus, the more of an effect passive immunity has on bird and egg populations.

The amount of susceptible eggs increases with the frequency of treatment. This is because of the lack of disease in the area, similar to how the lack of disease shows a decrease in the recovered egg category. Birds are less likely to become infected with West Nile Virus, and are therefore remaining in the susceptible category to lay susceptible eggs with no chance to develop an immunity to the disease. However, this is not the only cause. With the lack of disease, birds are less likely to die, creating an overall increase in the bird population [17].

The parameter ψ is the maturation rate from eggs to

birds. From Figure 3, we can see that as ψ increases, so does the overall bird populations since more birds are in the population. Also, as treatment goes up, both the infected and immune bird populations decrease because fewer birds are becoming infected since the infected mosquitoes are being killed off. Immune birds decrease overall since there are fewer birds becoming infected that recover and are laying eggs. This growth rate for eggs can be adjusted based on the species of birds in one's local community.

The parameter p_R is the percent of immune birds that retain their passive immunity throughout their entire adulthood. This varies based on the species of birds. From Figure 4, infected bird population increases as p_R decreases. When there is a smaller percent of birds retaining their immunity, there are more birds that become susceptible after their temporary period of being an immune bird. Thus, there are more birds that can become infected with the disease. The population of immune birds increases as the percent of birds retaining their immunity increases. This is attributed to an increase in recovered birds that will lay eggs that will eventually hatch into immune birds.

As expected, the less treatment available, the more infected birds there are, as we see in Figure 5. We can also see that as treatment effectiveness increases, the more dramatically the infected bird populations drop because the treatment kills of a larger population of infected mosquitoes, drastically lowering the chance of birds becoming infected with the disease later on.

3.2 Mosquitoes and Larvae

The bird population also affects the mosquitoes. From Figure 6, the less effective the treatment is, the more the population of birds affects the mosquito population. The infected mosquito and larvae populations grow as ψ increases because there is an increase of birds that the disease can infect. We can also see from Figure 6 that the infected mosquito population does indeed decrease as the treatment becomes more effective.

The different rates that different bird species have impacts the mosquito populations. So, in Figure 7, we compare the infected mosquito population with different bird species that have different hatching rates, clutch sizes, passive immunity rates, and growth rates.

Infected mosquitoes decrease with more treatment, as do all mosquito populations. The more often the treatment, the more the graph oscillates. This is due to spraying in different intervals and killing a large portion of mosquitoes off at a time. However, as seen in the control case, if we do not treat at all, the infected mosquito population will actually increase, posing a health hazard.

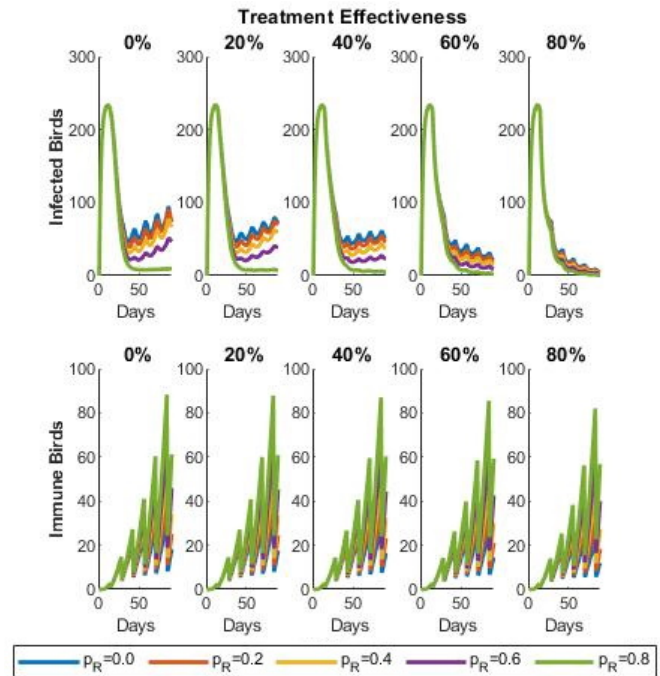


Figure 4: Infected and immune bird populations as affected by different p_R -values (0.0, 0.2, 0.4, 0.6, 0.8) with treatment frequency being bi-weekly. The treatment effectiveness increases left to right at 20% increments. Note that these oscillations are from a bi-weekly treatment and birds leaving the immunity category.

3.3 Humans

Infected humans are composed of both humans with neuroinvasive disease and humans with West Nile Fever. In Figure 8, we see that infected humans diminish as fewer humans are exposed to the disease. As expected, the more we treat, the fewer humans that become infected with the disease.

4 Discussion and Conclusion

Modeling West Nile Virus is crucial to simulating the spread of the disease in local communities to prepare for disease outbreaks in both human and bird populations. Since these models are used to improve public health, it is paramount to give the most precise model. Since passive immunity and vertical transmission are both phenomena observed in the natural world, it is necessary to include them within the disease dynamics [1, 2]. Our model improved upon past models by exploring the importance of passive immunity in birds, showing that passive immunity

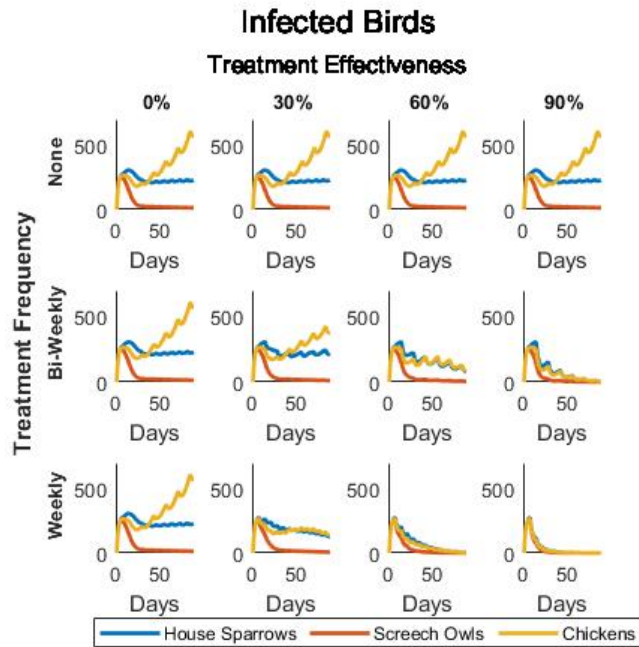


Figure 5: The infected bird population represented by three species of birds: house sparrows (blue), screech owls (red), and chickens (yellow). From left to right treatment effectiveness increases in 20% increments and from top to bottom treatment frequency increases from no treatment to bi-weekly treatment to weekly treatment. Note that these oscillations are from various treatment frequencies and birds leaving the immunity category.

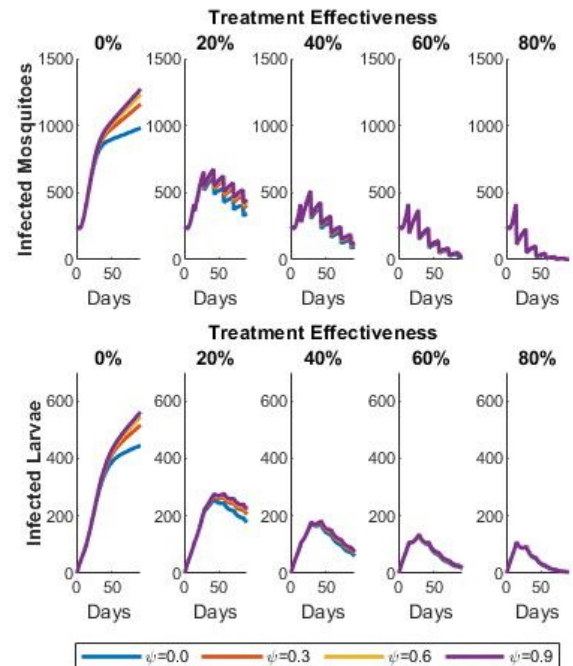


Figure 6: Infected mosquitoes and larvae populations as affected by different ψ -values (0.0, 0.3, 0.6, 0.9) with treatment frequency being bi-weekly. The treatment effectiveness increases left to right at 20% increments. Note that these oscillations are from a bi-weekly treatment and from birds leaving the immunity category.

affects both the bird and mosquito populations. Passive immunity increases bird immunity and lowers the disease spread overall.

The more factors that are considered, the more accurate our estimations of disease spread will be. Using passive immunity requires a new category of birds, also known as immune birds. These are different from recovered birds in that after the small period of immunity, the birds will be able to get infected again. This is essential knowledge to apply to mathematical modeling as it changes the outcome and results of other mathematical models. Vertical transmission increases the rate of infection and thus, is a potential threat to human and bird health. Thus, it is important to utilize these in mathematical models. Our work has laid the foundation of utilizing both of passive immunity and vertical transmission in West Nile Virus modeling, yet there is still more to be done. For instance, it would be interesting to look at the stability analysis of the model as well as a mix of bird populations. This would provide accurate results for a specific area when considering the makeup of the bird population by species. It would also be compelling to

consider temperature patterns in future work as temperature affects a multitude of parameters such as the rate mosquitoes lay their eggs, the rate birds enter and leave the community, and the mosquito biting rate.

In order to more accurately simulate the disease spread in a specific location, scientists need data for birds in that location, such as: the average bird egg laying rates, the average egg growth rates, and the average rates of passive immunity for the specific bird species in that area. This will give the most accurate results for the disease spread in any given community. Instead of using specific birds in the model, these values can be replaced for the average values for passerine birds in a local area.

Since West Nile Virus mainly pertains to the summer months in terms of disease spread, it is also important to consider the variance of bird populations over several years. An expansion of this model could consider the long-term ramifications of West Nile Virus in a certain area. Overall, our work serves as a template for modeling West Nile Virus in any of the diverse environments in which West Nile Virus is extant.

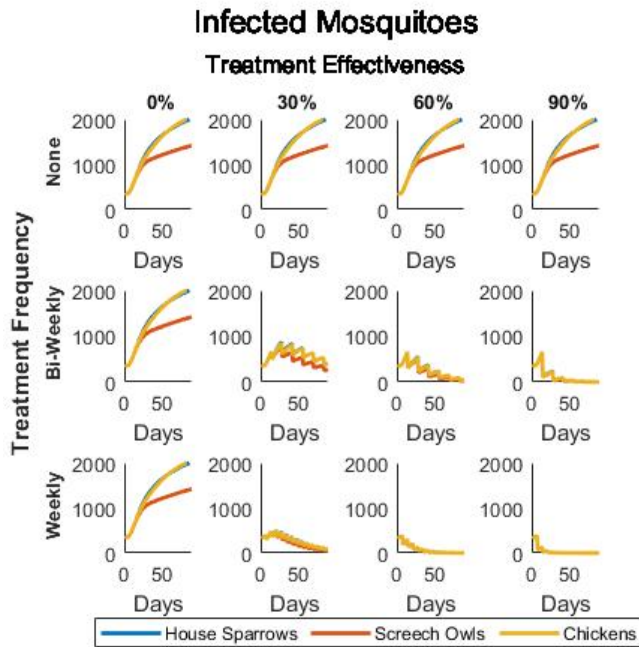


Figure 7: The infected mosquito population using three different species of birds: house sparrows (blue), screech owls (red), and chickens (yellow). From left to right treatment effectiveness increases in 20% increments and from top to bottom treatment frequency increases from no treatment to bi-weekly treatment to weekly treatment.

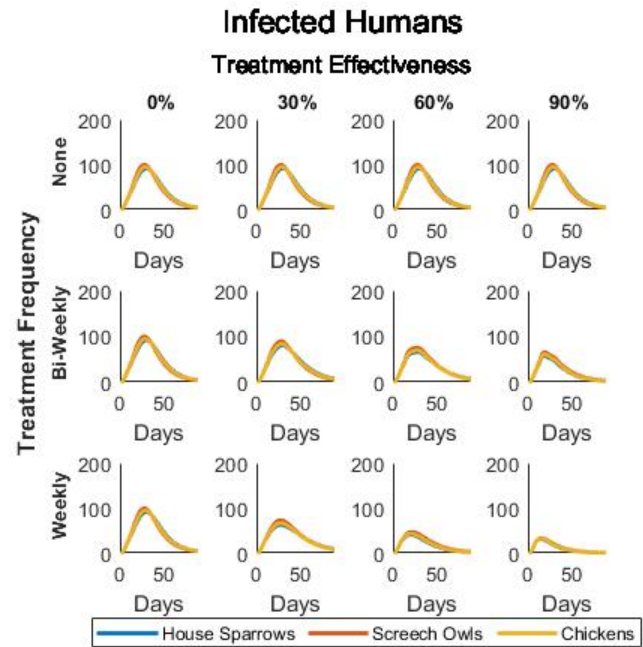


Figure 8: The infected human (humans with WNV Fever and humans with neuroinvasive disease) population using three different species of birds: house sparrows (blue), screech owls (red), and chickens (yellow). From left to right treatment effectiveness increases in 20% increments and from top to bottom treatment frequency increases from no treatment to bi-weekly treatment to weekly treatment.

Author Contributions

Noelle West (undergraduate) and Vinodh Chellamuthu (faculty) designed the model, performed the numerical simulations, and analyzed model output. Noelle West (undergraduate) implemented the model using MatLab and did the literature review.

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