The Impact of Mosquito-Bird Interaction on the Spread of West Nile Virus to Human Populations

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Abstract

West Nile Virus (WNV) is a rapidly spreading and potentially fatal disease that presents a public health challenge. In this study, we develop a mixed Susceptible-Infected and Susceptible-Infected-Recovered model for WNV dynamics that includes mosquito, bird and human populations. We calculate the basic reproductive number (R_0) and establish the existence of possible multiple endemic equilibria. The sensitivity of R_0 to parameters is studied. The possibility of multiple equilibria when $R_0 < 1$ (backward bifurcations) implies that standard control measures are likely to be inadequate to control an epidemic.

1 Introduction

West Nile Virus (WNV) is an arbovirus of the flavivirus family that was first isolated in Uganda in 1937. Before 1999, human infections were found mostly in Africa, West

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Asia, and the Middle East [2]. It has spread to four continents [1]. Most human infections are asymptomatic; however, for those that are not, possible symptoms include headaches and neck aches to potentially lethal meningitis[1]. and encephalitis[2] [4]. In 1999, the first case of WNV in North America was identified in New York City. It has since spread to 46 states and infected over 9,800 people in 2003 alone [4]. Several deaths were reported in Southern California in 2004[15]. WNV is linked to high bird mortality [3]. In 2000, there were 71,332 birds infected in the state of New York, including 17,571 (24.6 %) American crows. Of the 3,976 dead birds tested, 1,263 (31.8 %) were positive for WNV [5].

Studies have shown that mosquitoes acquire the WNV in their salivary glands when they feed on infected birds with the virus [9]. The virus is transmitted when infected female mosquitos feed on birds and humans [9]. It is believed that handling infected bird carcasses that have died from WNV infections are also a source of human infections, although it has not been clinically proven [4]. Data exhibit a strong direct correlation between density of dead crows and human West Nile Virus cases [13].

Humans are secondary hosts [11]. However, recent growth in human infections naturally suggests a potential relevant role in transmission. Furthermore, since reducing human incidence is of great importance, the primary goal of our research is to create a model that incorporates humans, birds, and mosquitos as sources of WNV infections.

Our paper is organized as follows: Section 2 briefly reviews the ecology of the *Corvus Brachyrhynchos* (American crow) and the *Culex Pipiens* (mosquito), the primary reservoir and vector for WNV in the United States; Section 3 introduces a simplified model; Section 4 includes some partial analysis of the model of Section 3; Section 5 explains the sensitivity of R_0 (the basic reproductive number) to parameters; Section 6 illustrates some of our results to via numerical simulations; Section 7 reviews model results and outlines future work.

[1]Meningitis - Inflammation of one of the membranes covering the brain and spinal cord

[2]Encephalitis - Inflammation of the brain

2 Ecology of *Culex Pipiens* and *Corvus Brachyrhynchos*

2.1 Culex Pipiens

The primary vectors in WNV infection are mosquitos of the *Culex* family. *Culex Pipiens* is the only species that spreads WNV in urban and sylvan areas [5]. Each female (*Culex Pipiens*) lays 100-200 eggs every 2-3 days on the surface of stagnant water. Eggs generally hatch within two days in favorable temperature and humidity conditions. Eggs may be viable for up to five years [7]. Hatched eggs enter the larva stage (7-14 days) and then the pupa stage (2 days). Adult mosquitos have a maximal life-span of 20 days [7]. Female adults feed four times over their life-time [8]. Males feed primarily on plant nectar while females feed on birds and mammals. Birds provide the primary blood meals for female *Culex Pipiens* [7]. Once an uninfected mosquito bites an infected host, they will carry the disease for the rest of their lives, but will not die from the infection. When the mosquito infects a host, the virus replicates within the host's blood and during this process it may affect the central nervous system [9]. Disease symptoms typically last a few days, but there are exceptions [6]. Severe cases of encephalitis and meningitis may develop from 3 and 14 days after an infection [9]. Various studies support vertical transmission in vectors. However, since filial infection rates[3] were low (about 1 per 1,000 progeny tested [16]). Their impact is ignored on this preliminary study.

2.2 Corvus Brachyrhynchos

The maximal life-span of the American crow is 5 years [20]. A female can lay between 4-6 greenish eggs in the middle of April. Male and female take turns incubating them [20]. Eggs hatch around day 18^{th}, and fledglings are able to fly 35 days later [20]. Crows feed on insects, small vertebrates, bird eggs, nestlings, seeds, fruit and plant material [20].

Crow migration occurs in late summer or early fall in the Southeastern U.S. Route [20]. Evidence shows WNV spreading along this route. It is through this migration highway that American crows have had their biggest impact [21]. Dead bird surveillance has shown that the seasonal spread of the virus in the United States

[3]Filial infection rates - Percentage of offsprings of an infected female mosquito that are infected

is consistent with bird migration patterns [10]. WNV disease induced death on crows is 90% [18]. Hence, high levels of dead crow carcasses are detected wherever high increases in WNV incidence are reported [3]. Thus, the number of dead carcasses can be used to monitor, track, and possibly predict an outbreak of WNV in humans [3]. There is no evidence of vertical transmission among birds.

3 Model Description

We use an *S-I (Susceptible-Infected)* model for the mosquito population, and since its members do not die from the disease, it is assumed that the mosquito population (N_M) is roughly constant. Specifically, $N_M = S_M + I_M$, where S_M and I_M denote the susceptible and infected female mosquito populations.

We model the impact of WNV on humans via a *S-I-R (Susceptible-Infected-Recovered)* model where the human population (N_H) is assumed constant. Specifically, $N_H = S_H + I_H + R_H$, where S_H, I_H, and R_H denote the susceptible, infected, and recovered human populations.

WNV dynamics on birds is modeled via a *S-I (Susceptible-Infected)* model. The total crow population (N_B) cannot be assumed to be constant since disease-induced deaths are high. If S_B, I_B denote the susceptible and infected crow populations then N_B is modeled by the following differential equation

$$N_B' = \Lambda - \mu_B N_B - \gamma I_B \tag{1}$$

where Λ represents an assumed constant recruitment rate, μ_B the natural per capita mortality rate and γ the per-capita disease induced rate for crows. A more realistic model may assume that $\Lambda = \Lambda(N)$, a situation that may be explored in the future. For simplicity we first consider the case when $\gamma = 0$,

$$N_B' = \Lambda - \mu_B N_B$$

in this case, $N(t)$ approaches $\frac{\Lambda}{\mu_B}$ as $t \to \infty$. For the rest of this manuscript, we set $N(0) = \frac{\Lambda}{\mu_B}$.

P_H and P_B denote the mosquito preference probability to biting humans or birds, respectively, with $0 < P_H < P_B \leq 1$ note that these preferences only appear in the equation for the mosquito population, because when a mosquito infects a human or a bird, it already exercised its choice to bite this host.

Transfer from the susceptible to infected class depends on various factors including the biting rate of the mosquitos, the probability of transmission per bite, host preference, disease prevalence and other factors [14]. $\beta_H b\frac{N_H}{N_M}\frac{P_H N_M}{N_H+N_B}\frac{I_H}{N_H}S_M$ denotes the incidence rate (new cases of infection per unit of time or force of infection) generated by the interactions between mosquitos and infected humans; where bS_M denotes the average number of bites of uninfected mosquitos per unit time; $P_H\frac{N_H}{N_M}\frac{N_M}{N_H+N_B}$ denotes the likelihood that the mosquito will contact a human; $\frac{I_H}{N_H}$ is the 'probability' that the bitten host is infected and β_H is the probability that this intersection leads to a new infection. By similar argument, $\beta_B b\frac{N_B}{N_M}\frac{P_B N_M}{N_H+N_B}\frac{I_B}{N_B}S_M$ denotes the incidence rate generated by the interaction between mosquitos and infected birds. $\beta_M b\frac{N_M}{N_B}\frac{N_B}{N_H+N_B}\frac{I_M}{N_M}$ denotes the incidence rate generated by the interaction between birds and infected mosquitos. Similarly, $\beta_M b\frac{N_M}{N_H}\frac{N_H}{N_H+N_B}\frac{I_M}{N_M}$ denotes the incidence rate generated by the interaction between humans and infected mosquitos.
Figure 1 highlights via a box-diagram the interaction between vector, birds and humans.

The following is the system of non-linear differential equations:

$$S'_M = \mu_M N_M - \beta_H b\frac{N_H}{N_M}\frac{P_H N_M}{N_H+N_B}\frac{I_H}{N_H}S_M - \beta_B b\frac{N_B}{N_M}\frac{P_B N_M}{N_H+N_B}\frac{I_B}{N_B}S_M - \mu_M S_M,$$

$$S'_B = \Lambda - \beta_M b\frac{N_M}{N_B}\frac{N_B}{N_H+N_B}\frac{I_M}{N_M}S_B - \mu_B S_B,$$

$$S'_H = \mu_H N_H - \beta_M b\frac{N_M}{N_H}\frac{N_H}{N_H+N_B}\frac{I_M}{N_M}S_H - \mu_H S_H,$$

$$I'_M = \beta_H b\frac{N_H}{N_M}\frac{P_H N_H}{N_H+N_B}\frac{I_H}{N_H}S_M + \beta_B b\frac{N_B}{N_M}\frac{P_B N_B}{N_H+N_B}\frac{I_B}{N_B}S_M - \mu_M I_M,$$

$$I'_B = \beta_M b\frac{N_M}{N_B}\frac{N_B}{N_H+N_B}\frac{I_M}{N_M}S_B - \mu_B I_B,$$

$$I'_H = \beta_M b\frac{N_M}{N_H}\frac{N_H}{N_H+N_B}\frac{I_M}{N_M}S_H - (\mu_H+\theta)I_H,$$

$$R'_H = \theta I_H - \mu_H R_H,$$

where $N_M = I_M + S_M$, $N_B = I_B + S_B$, and $N_H = S_H + I_H + R_H$
and $N'_M = 0$, $N'_B = \Lambda - \mu_B N_B - \gamma I_B$, and $N'_H = 0$.

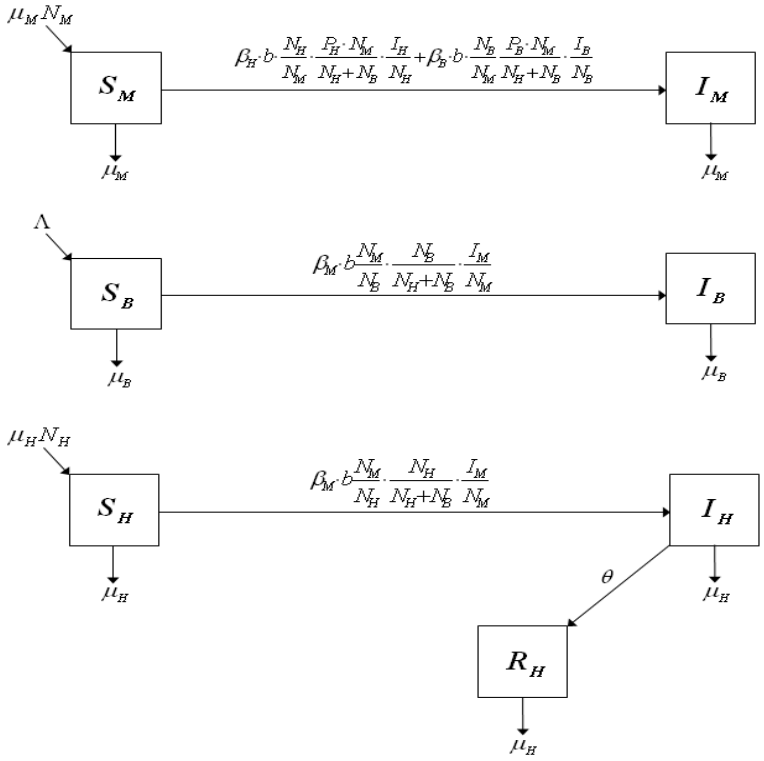

Figure 1: Model for WNV vector-host cross-infection among mosquitos, birds and humans.

Parameter	**Description**	**Value**
P_H	Mosquito preference probability to biting humans	0.3
P_B	Mosquito preference probability to biting birds	0.6
β_M	Transmission probability from mosquitos to hosts per bite	0.88
β_B	Transmission probability from birds to mosquitos per mosquito bite	0.16
β_H	Transmission probability from humans to mosquitos per mosquito bite	0.2
μ_M	Natural mortality rate for mosquitos	0.055
μ_B	Natural mortality rate for birds	0.00055
μ_H	Natural mortality rate for humans	0.000037
b	Number of bites per mosquito per unit time	0.2
θ	Recovery rate of humans	0.91

Table 1: Parameters definitions. parameters.

The value of β_B was taken from [11]; b from [8]; θ from [12]. μ_M was taken as the inverse of the average maximal lifespan of a vector (18 days). (similarly for μ_B and μ_H) were obtained from the inverse of the average lifespan of birds and humans, respectively, in days. By assumption, we choose $P_H = 0.3 < P_B = 0.6$. Numerical simulations used the values in Table 1.

4 Model Analysis

4.1 The Basic Reproductive Number (R_0)

The basic reproductive number (R_0) for vector transmitted diseases was introduced by Ross (1911) and it was heavily used and expanded by MacDonald (1952) in the context of malaria [29]. R_0 represents the average number of secondary infections generated by a typical infectious individual in a population of susceptible at demographic equilibria. It is computed by introducing an infected vector (host) and determining the average number of susceptible vectors (hosts)that would be infected over its infectious lifespan. The basic reproductive number (R_0) is computed[4] (see below)using the method found in Diekmann *et al.* [22].

In order to interpret it, we introduce the following quantities:

$$N_B^* \equiv \frac{\Lambda}{\mu_B}$$

$$R_0^{MH} \equiv \frac{bN_H}{N_B^*+N_H}\frac{\beta_M}{\mu_M}$$
$$R_0^{HM} \equiv \frac{bP_HN_M}{N_B^*+N_H}\frac{\beta_H}{\mu_H+\theta}$$
$$R_0^{MB} \equiv \frac{bN_B^*}{N_B^*+N_H}\frac{\beta_M}{\mu_M}$$
$$R_0^{BM} \equiv \frac{bP_BN_M}{N_B^*+N_H}\frac{\beta_B}{\mu_B},$$

$\sqrt{R_0^H} \equiv \sqrt{R_0^{MH} \cdot R_0^{HM}}$ represents secondary infections generated by humans (vectors) in humans (vectors) in a disease-free system and $\sqrt{R_0^B} \equiv \sqrt{R_0^{MB} \cdot R_0^{BM}}$ represents the secondary infections generated by birds (vectors) in birds (vectors) in a disease-free system.

[4] See Appendix, subsection 8.1.

The basic reproductive number (R_0) is given by:

$$R_0 = \sqrt{R_0^H + R_0^B}$$

We observe that in R_0,

$\frac{1}{\theta+\mu_H}$, $\frac{1}{\mu_B}$, and $\frac{1}{\mu_M}$ are the infectious periods for humans, birds, and mosquitos; $\frac{bP_HN_M}{N_B^*+N_H}\frac{1}{\mu_H+\theta}$ represents the average number of mosquitos that a person will get bitten by; $R_0^{HM} \equiv \frac{bP_HN_M}{N_B^*+N_H}\frac{\beta_H}{\mu_H+\theta}$ represents the secondary infections generated by an infected human in vectors in a disease-free system; $\frac{bP_BN_M}{N_B^*+N_H}\frac{1}{\mu_B}$ represents the average number of mosquitos that a bird will get bitten by. Hence, $R_0^{BM} \equiv \frac{bP_BN_M}{N_B^*+N_H}\frac{\beta_B}{\mu_B}$, gives the secondary infections generated by an infected bird in a disease-free system; $\frac{bN_H}{N_B^*+N_H}\frac{1}{\mu_M}$ represents the average number of mosquito bites to humans. Hence, $R_0^{MH} \equiv \frac{bN_H}{N_B^*+N_H}\frac{\beta_M}{\mu_M}$ gives the secondary infections generated by an infected vector in humans; $\frac{bN_B^*}{N_B^*+N_H}\frac{1}{\mu_M}$ gives the number of mosquito bites to birds and thus, $R_0^{MB} \equiv \frac{bN_B^*}{N_B^*+N_H}\frac{\beta_M}{\mu_M}$ represents the secondary infections generated by an infected mosquito in birds in a disease-free system;

Therefore, the product of $\sqrt{R_0^{HM} \cdot R_0^{MH}}$ represents the contribution to new infections between mosquitos and humans. Likewise, $\sqrt{R_0^{BM} \cdot R_0^{MB}}$ represents the contributions to new infections between mosquitos and birds. R_0 is the number of secondary infections generated by an infectious individuals over his infection period in a disease-free system.

4.2 Equilibria

As an initial step, we analyze the model when $\gamma = 0$ and then proceed to study its dynamics numerically when $\gamma > 0$. We define $x_M = \frac{I_M}{N_M}$, $x_H = \frac{I_H}{N_H}$ and $x_B = \frac{I_B}{N_B}$ to be the proportions of the infected mosquitos, humans, and birds classes, respectively. For simplicity, we define $K_M = \frac{N_M}{N_B^*+N_H}$, $K_H = \frac{N_H}{N_B^*+N_H}$, and $K_B = \frac{N_B}{N_B^*+N_H}$. Then, we have

$$
\begin{aligned}
x'_M &= P_H\beta_H bK_H x_H[1-x_M] + P_B\beta_B bK_B x_B[1-x_M] - \mu_M x_M,\\
x'_B &= \beta_M bK_M x_M[1-x_B] - \tfrac{\Lambda}{N_B}x_B,\\
x'_H &= \beta_M bK_M x_M[1-x_H-y_H] - (\mu_H+\theta)x_H,\\
y'_H &= \theta x_H - \mu_H y_H.
\end{aligned}
$$

The equilibria satisfy:
$x_M^* = \frac{1}{\mu_M}(P_H\beta_H bK_H x_H^*[1-x_M^*] + P_B\beta_B bK_B x_B^*[1-x_M^*])$,

$x_H^* = \frac{\mu_H\beta_M bK_M x_M^*}{\beta_M bK_M x_M^*(\mu_H+\theta)+\mu_H(\mu_H+\theta)}$,

$x_B^* = \frac{N_B\beta_M bK_M x_M^*}{N_B[\beta_M bK_M x_M^*]+\Lambda}$.

Some algebra shows that x_M^* are the roots of:
$P(x_M^*) = Ax_M^{*3} + Bx_M^{*2} + Cx_M^*$, that is,

$P(x_M^*) = x_M^*[Ax_M^{*2} + Bx_M^* + C]$. Consequently we have three roots $x_M = 0$ or

$x_M = \frac{-B\pm\sqrt{B^2-4AC}}{2A}$, where:

$A = -[\mu_M\beta_M^2 b^2 K_M^2\frac{\Lambda}{\mu_B}(\mu_H+\theta) + b^3\beta_M^2 K_M^2\frac{\Lambda}{\mu_B}[P_H\beta_H\mu_H K_H + P_B\beta_B K_B(\mu_H+\theta)]]$

$B = \frac{\Lambda}{\mu_B}\beta_M^2 K_M^2[P_H\beta_H b^2 K_H\mu_H + P_B\beta_B b^3 K_B(\mu_H+\theta)] - \frac{\Lambda}{\mu_B}\mu_M\mu_H(\mu_H+\theta)\mu_B R_0^2$

$C = \mu_M\mu_H\Lambda(\mu_H+\theta)(R_0^2-1)$

If $x_M^* = 0$ then $x_H^* = 0$ and $x_B^* = 0$. This is the disease free equilibrium.

Note: $A < 0 \quad \forall\ x_M^* > 0$, then
If $R_0 > 1$ then $C > 0$. In this case, there is only one endemic equilibrium, namely

$x_M^* = \frac{-B-\sqrt{B^2-4AC}}{2A}$,

$x_H^* = \frac{\mu_H\beta_M bK_M(B+\sqrt{B^2-4AC})}{\beta_M bK_M B\mu_H+\beta_M bK_M B\theta+\beta_M bK_M\sqrt{B^2-4AC}\mu_H+\beta_M bK_M\sqrt{B^2-4AC}\theta-2\mu_H^2 A-2\mu_H A\theta}$,

$x_B^* = \frac{N_B\beta_M bK_M(B+\sqrt{B^2-4AC})}{N_B\beta_M bK_M(B+\sqrt{B^2-4AC})-2A\Lambda}$.

If $R_0 < 1$ then, $C < 0$. In this case we can have multiple endemic equilibria. In fact, if $B^2 - 4AC > 0$ and $B > 0$ then there are two endemic equilibria. They are given by

$$x_M = \frac{-B \pm \sqrt{B^2 - 4AC}}{2A},$$

$$x_H = \frac{\mu_H \beta_M b K_M (B \pm \sqrt{B^2 - 4AC})}{\beta_M b K_M B \mu_H + \beta_M b K_M B \theta + \beta_M b K_M \sqrt{B^2 - 4AC} \mu_H + \beta_M b K_M \sqrt{B^2 - 4AC} \theta - 2\mu_H^2 A - 2\mu_H A \theta},$$

$$x_B = \frac{N_B \beta_M b K_M (B \pm \sqrt{B^2 - 4AC})}{N_B \beta_M b K_M (B + \sqrt{B^2 - 4AC}) + \Lambda)}.$$

We observe that if $B^2 - 4AC < 0$ or $B < 0$ then there is no endemic equilibrium with $0 \leq x_M \leq 1$, $0 \leq x_H \leq 1$ and $0 \leq x_B \leq 1$.

5 Simulations When $\gamma > 0$ and $\phi > 0$

The introduction of a secondary host (humans) appears to be capable of generating outbreaks when $R_0 < 1$ (not the case in models without humans [11]). We ran simulations of our model and varied β_B while choosing different initial conditions. Figures 2, 3, and 4 show that reducing $R_0 < 1$ is not enough to guarantee that the disease will die out in any of the three populations. The levels of infection in mosquitos can vary anywhere from 0 to 60% when $0.18 < R_0 < .35$.

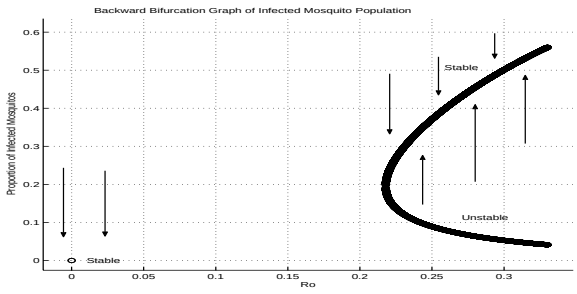

Figure 2: Backward Bifurcation for Infected Mosquitos, varying β_B and with the following parameter values: $\mu_M = 0.055; \mu_B = 0.00055, \mu_H = 0.000037, b = 0.2, \gamma = 0.443, \theta = 0.91, \phi = 0.09, P_H = 0.4; P_B = 0.6, N_B = 30000, \Lambda = N_B * \mu_B, N_H = 20000, N_M = 5000, \beta_H = 0.8, \beta_M = 0.9 and \beta_B varies from 0.1 to 20 with 1000 points.$

Figure 3 shows that the level of endemicity for bird populations also range from 0 to 3.5% when $0.18 < R_0 < .35$.

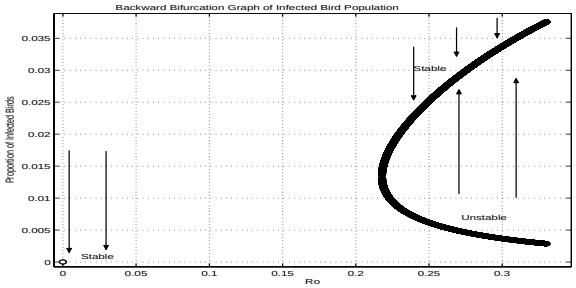

Figure 3: Backward Bifurcation for Infected Birds, varying β_B and with the following parameter values: $\mu_M = 0.055$; $\mu_B = 0.00055$, $\mu_H = 0.000037$, $b = 0.2$,$\gamma = 0.443$,$\theta = 0.91$,$\phi = 0.09$,$P_H = 0.4$; $P_B = 0.6$, $N_B = 30000$, $\Lambda = N_B * \mu_B$, $N_H = 20000$, $N_M = 5000$, $\beta_H = 0.8$, $\beta_M = 0.9$, and β_B varies from 0.1 to 20 with 1000 points.

Figure 4 shows that the disease levels in humans are very small. In other words, very few cases will be found in humans. The results of these three bifurcation diagrams highlight the importance of initial conditions. In particular it is worth noticing the potential role of infected flocks of birds (dynamic changes) in the generation of WNV outbreaks in regions where effective vector control measures may be in place ($R_0 < 1$).

6 Sensitivity Analysis

Although reducing R_0 may not be the key to controlling WNV, it is still of interest to look at the sensitivity of R_0 to variations in parameters. The sensitivity indexes of R_0 with respect to each of its parameters is calculated. Most of these indexes are non-constant as they depend on other parameters. Table 2 collects the sensitivity indexes.

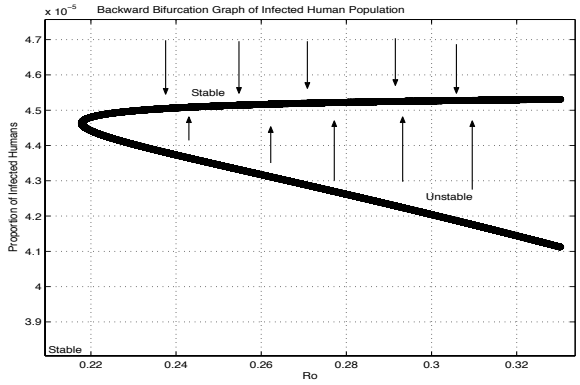

Figure 4: Backward Bifurcation for Infected Humans, varying β_B and with the following parameter values: $\mu_M = 0.055$; $\mu_B = 0.00055$, $\mu_H = 0.000037$, $b = 0.2$,$\gamma = 0.443$, $\theta = 0.91$, $\phi = 0.09$, $P_H = 0.4$; $P_B = 0.6$, $N_B = 30000$, $\Lambda = N_B * \mu_B$, $N_H = 20000$, $N_M = 5000$, $\beta_H = 0.8$, $\beta_M = 0.9$, and β_B varies from 0.1 to 20 with 1000 points.

Parameter	**Sensitivity Index**
b	1
P_B	$\frac{1}{2}\frac{A}{A+B}$
P_H	$\frac{1}{2}\frac{B}{A+B}$
β_M	$\frac{1}{2}$
β_B	$\frac{1}{2}\frac{A}{A+B}$
β_H	$\frac{1}{2}\frac{B}{A+B}$
μ_M	$-\frac{1}{2}$
μ_B	$\frac{1}{2}\frac{1}{\mu_B}\frac{2\Lambda B\mu_B+\Lambda A-\mu_B A[2\mu_B N_H+N_H]}{A+B}$
μ_H	$-\frac{1}{2}\frac{\mu_H}{\mu_H+\theta}\frac{B}{A+B}$
N_M	$\frac{1}{2}$
N_H	$\frac{1}{2}\frac{N_H\mu_B}{N_H\mu_B+\Lambda+m\mu_B}\frac{-B-2A}{A+B}$
Λ	$-\frac{1}{2}\frac{(\mu_B+\Lambda)A+2\Lambda B}{A+B}$
θ	$-\frac{1}{2}\frac{\theta B}{\mu_H+\theta A+B}$

Table 2: Parameter sensitivity indexes with $A \equiv P_B\Lambda\beta_B(\mu_H+\theta)$ and $B \equiv P_H\mu_B N_H\beta_H(\mu_B)$

The sensitivity indexes for the parameters b, μ_M, N_M, and β_M are independent of any changes in all other parameters. They hold the greatest value in understanding

how to reduce the value of R_0.
The greatest (constant) sensitivity index belongs to the biting rate b. If the biting rate were to decrease by 1% then the value of R_0 would also decrease by 1%. Humans can reduce this biting rate by wearing appropriate clothing or using mosquito repellent. However, a bird's only protection from being bitten is the thickness of its feathers. There is no good method for decreasing the number of mosquito bites in birds.

The sensitivity index for the natural mortality rate of mosquitos μ_M equals $-\frac{1}{2}$. This implies that a 1% decrease in μ_M gives a $\frac{1}{2}$% increase in R_0. In short, if the average lifespan of a mosquito were to increase then the chances of an epidemic occurring increase. It is well documented that WNV cases are highest during the summer months. It is also known that for some WNV-carrying Culex mosquitos, lifespan tends to decrease as temperature increases [24]. This result cannot be addressed well with this model as the role of temperature is not included. Our model result cannot explain known patterns of WNV occurrence during summer months[24].

The total number of mosquitos N_M has a sensitivity index of 0.5, which suggests that a 1% decrease in N_M would cause a $\frac{1}{2}$% decrease in R_0. This suggests that mosquito control may have a significant impact in reducing the chances of an outbreak.

The probability of transmission per contact β_M has a sensitivity index of .5. This result adds to the previous one as b and β_M appear together (multiplicative). This further suggests that mosquito control may have an impact in reducing the chances of an outbreak. Because the sensitivity indexes for all other parameters are nonconstant and because we do not have exact values for most of them, we pursue a sensitivity analysis of them via simulations. We focus on the study of the sensitivity of two parameters P_H and P_B via simulations. For our purposes, we assume $P_H = 0.3$ and $P_B = 0.7$. According to statistical data on birds [25] and census data on humans [26], it is estimated that there are 3 billion crows in North America and 294 million humans in the United States. If we assume a uniform distribution of humans and crows across North America and that the US occupies 1/3 of the total land area, then the number of crows in the USA may be approximately 1 billion crows. Thus we can assume for our sensitivity analysis that there is a 3:1 ratio of crows to humans in the United States. After some simulations under these conditions, we arrive at the results in table 3.

Parameter	Sensitivity Index
b	1
P_B	0.5
P_H	0.00005
β_M	0.5
β_B	0.5
β_H	0.000054
μ_M	-0.5
μ_B	-0.298
μ_H	-2.1910^{-9}
N_M	0.5
N_H	-0.2499
Λ	-0.25
θ	-0.000054

Table 3: Sensitivity index values under a bird-biased system.

β_H, P_H, μ_H and θ have negligible sensitivity indexes to R_0. In general, these parameters make less than $< 0.05\%$ change in R_0. In Figure 2, we can see that adding 0.4 to β_H, the infected human population raise by 12 infected individuals.

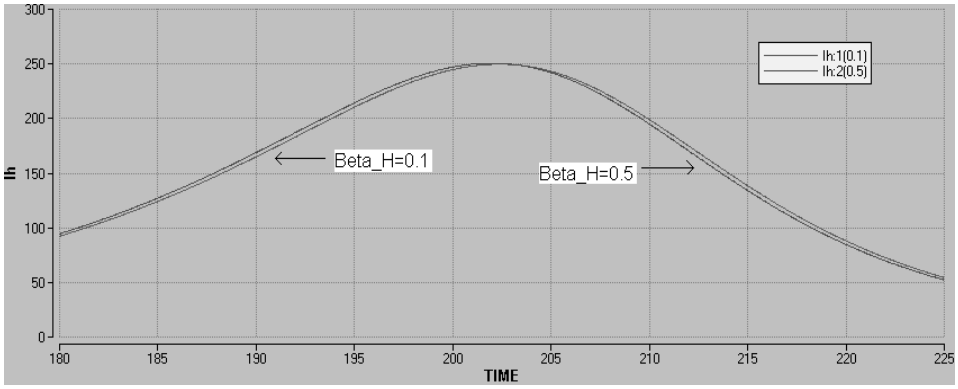

Figure 5: Time series for infected humans, varying β_H

Apart from the parameters mentioned previously, the next greatest positive sensitivity indexes are each approximately equal to $\frac{1}{2}$ and belong to the parameters P_B

and β_B. This result is verified numerically in Figure 3.Similarly, the next greatest negative sensitivity indexes are approximately equal to $-\frac{1}{4}$ and belong to μ_B and N_H.

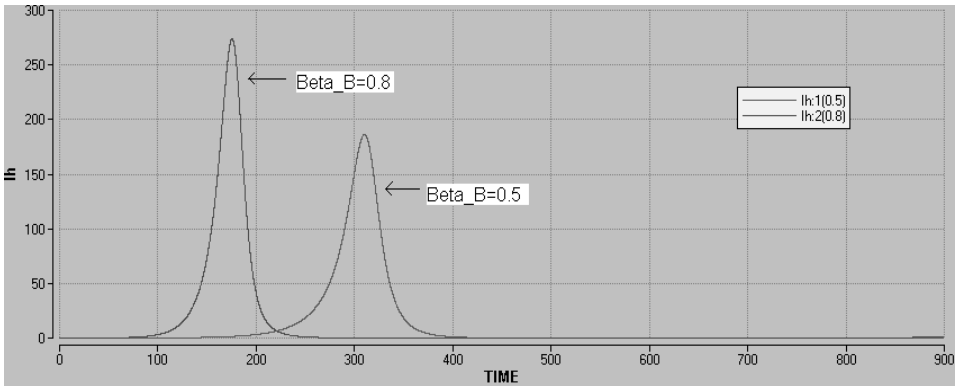

Figure 6: Time series for infected humans, varying β_B.

Biologically speaking, β_B is a natural constant and, in general, cannot be manipulated. Thus, knowledge of their high sensitivity indexes may not imply any suggestions in preventing the outbreak of the virus in any populations. The current assumption about WNV is that high mortality in birds is a significant indicator of an outbreak. However, under the assumptions above, as the natural mortality rate (μ_B) increases, R_0 decreases by a factor of 0.298%. This would imply that high levels of bird deaths would imply less chance of an outbreak. This result is consistent with the current assumptions about the dynamic of bird deaths as a forewarning of outbreaks in human populations. However, as the recruitment rate Λ increases by 1%, the value of R_0 decreases by .25%. Theoretically, this would mean that the possibility of an outbreak decreases when there are more birds entering a population. This result contradicts the hypothesis that an influx of infected birds into an area can produce an outbreak.

The preference parameter P_B has a smaller, positive sensitivity indexes (in comparison to those that have 0.5 sensitivity indexes). This implies that if mosquitos tend to prefer to bite birds more than humans, then as our estimated value of their preference to birds increases, the possibility of an outbreak increases as well. This result is consistent with the assumption that mosquitos tend to prefer to bite birds

Finally, the parameter N_H gives us a more clearer picture of the dynamic of the disease within different sizes of human populations. Though the value of the

sensitivity index of N_H is not as large as the value of other parameters, the fact that it is positive implies that there is less of a chance of an outbreak in a large cities and more of a chance of outbreak in smaller communities.

Due to time constraints, further sensitivity analysis was not computed for other situations such as varying bird to human ratios and mosquito preferences. Future research will include these situations included in the sensitivity analysis.

7 Conclusions and Future Work

We derived a model which not only includes the interaction of WNV among mosquitos, birds and humans, but also accounts for natural preference in which mosquitos choose hosts. As a result of assuming preferences in our model, we derived a basic reproductive number. We found from numerical simulations, varying $\gamma > 0$ and $\phi > 0$, that if the conditions $B > 0$, $\sqrt{B^2 - 4AC} > 0$ and $R_0 < 1$ are satisfied for the $P(x_M)$ polynomial, we have enough conditions for backward bifurcations in all three populations. This result contradicts the popular trend in WNV that human populations have no importance in WNV. The implications of this result are that if the disease were to become endemic, it would be much more difficult to eradicate the disease.

Though sensitivity analysis may suggest that the most plausible method of eradication of WNV in a closed population would be to reduce the mosquito population or reduce the biting rate, it is unclear whether either of these two methods will result in permanent eradication of the disease. However, sensitivity of this model hints that epidemics may occur during the winter, when various Culex mosquitos have lower mortality rates. However, because of the backward bifurcation, initial conditions become extremely important to the system, meaning that even if control measures are in place, a sudden migration of infected birds could produce an outbreak.

In future studies, we we hope to add a latent class to birds, humans, and mosquitos. We will also explore the possibility of adding additional hosts into the model and view their impact upon the system's dynamics. We would also like to explore more of the possibilities when $\gamma > 0$.

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9 Appendix

9.1 Finding R_0 in the Disease Free Equilibria (DFE)

To obtain R_0 we use the method by Diekmann *et al.* [22].

We find the Jacobian in DFE $(N_H, \frac{\Lambda}{\mu_B}, N_M, 0, 0, 0, 0, 0, 0)$ of the differential equations of infectious classes (I'_M, I'_B, I'_H)

$$J(DFE)=\begin{pmatrix} -\mu_M & \beta_H b\frac{P_H N_M}{N_B+N_H} & \beta_B b\frac{P_B N_M}{N_B+N_H} \\ \beta_M b\frac{N_H}{N_B+N_H} & -(\mu_H+\theta) & 0 \\ \beta_M b\frac{N_B}{N_B+N_H} & 0 & -(\mu_B+\gamma) \end{pmatrix}$$

- According to the method defined by Diekmann *et al.* [22], we now take the dot product between M and D^{-1}

$$MD^{-1}=\begin{pmatrix} 0 & \beta_H b\frac{P_H N_M}{N_B+N_H(\mu_H+\theta)} & \beta_B b\frac{P_B N_M}{N_B+N_H(\mu_B+\gamma)} \\ \beta_{M_2}\frac{P_H N_H}{(P_B N_B+P_H N_H+P_m m)\mu_M} & 0 & 0 \\ \beta_{M_1 b}\frac{P_B N_B}{(P_B N_B+P_H N_H+P_m m)\mu_M} & 0 & 0 \end{pmatrix}$$

where

$$A = \beta_H b \frac{P_H N_M}{P_B N_B + P_H N_H + P_m m(\mu_H + \theta)}$$

$$B = \beta_B b \frac{P_B N_M}{P_B N_B + P_H N_H + P_m m(\mu_B + \gamma)}$$

$$C = \beta_{M_2 b} \frac{P_H N_H}{(P_B N_B + P_H N_H + P_m m)\mu_M}$$

$$D = \beta_{M_1 b} \frac{P_B N_B}{(P_B N_B + P_H N_H + P_m m)\mu_M}$$

$$MD^{-1} = \begin{pmatrix} 0 & A & B \\ C & 0 & 0 \\ D & 0 & 0 \end{pmatrix}$$

now, we get

$$R_0 = \sqrt{CA + DB}$$

hence,

$R_0 =$

$$R_0 = \sqrt{\frac{bN_H}{N_B^* + N_H} \frac{\beta_M}{\mu_M} \cdot \frac{bP_H N_M}{N_B^* + N_H} \frac{\beta_H}{\mu_H + \theta} + \frac{bN_B^*}{N_B^* + N_H} \frac{\beta_M}{\mu_M} \cdot \frac{bP_B N_M}{N_B^* + N_H} \frac{\beta_B}{\mu_B}}$$