



A Dynamical Model for Transmission of West Nile Virus in Chicken-Mosquito Interaction

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Abstract. The West Nile virus (WNV) is transmitted through the bites of infected mosquitoes. The spread of WNV in chicken populations is quite unique. Although chickens can contract the virus through a mosquito bite, they immediately build immunity to the virus and do not show physical symptoms of illness and hence chickens are only temporary carriers of the virus. Recently, experimental results have shown that mosquitoes do not change fecundity behavior, yet results indicate that resistance to infection is associated with a fitness cost in terms of mosquito survival. We constructed a host-vector type transmission model for WNV in mosquito-chicken populations. The basic reproductive ratio, R_0 , was obtained. From sensitivity analysis of R_0 it was shown that under certain conditions this ratio decrease – with an increase of the lifetime of mosquito infection.

Keywords: *basic reproductive ratio; dynamical model; mosquito-chicken interaction; stability analysis; WNV transmission.*

1 Introduction

The spread of the West Nile Virus (WNV) has been reported in Africa, Asia, and Europe for decades, and recently it has been found in North America. It was first discovered in Uganda in 1937 and in Egypt and India in 1950. Causing millions of deaths in birds and a few thousand in humans, WNV has had a tremendous impact on public health policies [1,2].

A report from the US Center for Disease Control and Prevention indicates that there were 62 human cases of encephalitis, including seven deaths in 1999 [3]. WNV is most commonly transmitted to humans by mosquitoes. Until now there has been no medication to treat WNV infection, nor are there vaccines to prevent it. Unfortunately, most people infected with WNV will have no symptoms. About 1 in 5 people who are infected will develop a fever with other

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symptoms. Less than 1% of infected people develop a serious, sometimes fatal, neurologic illness [2,3].

WNV can infect many species of living beings, such as humans, horses, and also birds, especially chickens [4]. The symptoms of WNV in chickens are not easy to see, but according to Sanne, *et al.* in [2], most accurate information about WNV activity can be analyzed from infection in chickens. There is an incubation period before chickens can transmit WNV to mosquitoes, i.e. 3-5 days, while the incubation period for mosquitoes is 10-12 days.

Mathematical models have been developed by many different authors to understand the interaction of WNV in animals or humans [5]. A compartment model to describe the interaction between mosquitoes and birds is discussed in Wonham, *et al.* [6,7]. The basic reproductive ratio of the birds as endemic indicator was shown analytically, without including demographic factors. However, Bowman, *et al.* in [8] found that the basic reproductive ratio is not sufficient to characterize the dynamics of WNV, they also depend on the initial number of all state variables. A differential equations model with non-delay-autonomous approach was constructed by Lord and Day [9-10] in order to understand the impact of bird mortality in WNV transmission dynamics. Special interaction between female mosquitoes and birds was introduced by Cruz-Pacheco [5].

All of the mathematical models from the authors above have represented the spread of WNV between mosquitoes and birds. But according to [2], different from other birds, chickens have their incubation period after they are infected by WNV. Therefore, in this paper we introduce our mathematical model in order to understand the spread of WNV between chickens and mosquitoes. The immune effect in chicken populations is included in the model to give a better understanding of the problem.

This paper is constructed as follows. A host-vector model of WNV transmission is given in Section 2. The basic reproductive ratio and the numerical existence of an endemic equilibrium are discussed in Section 3. A dynamical analysis of the WNV transmission is performed in Section 4, to give a better understanding of the situation in the field. The conclusion is given in Section 5.

2 Host-Vector Model WNV Transmission

We have constructed a SEIR-SEI host-vector model for interaction between chicken and mosquito populations. The host population (chickens) consists of *susceptible* (S_h), *exposed* (E_h), *infected* (I_h), *temporary immune and less*

infectious (I_{hi}), and recovered (R_h) compartments, while the vector population (mosquitoes) is divided into three compartments: susceptible (S_v), exposed (E_v), and infected (I_v). The transmission scheme is given in Figure 1.

Here, we assume that the total host population $N_h = S_h + E_h + I_h + I_{hi} + R_h$ and the total vector population $N_v = S_v + E_v + I_v$ are constant. We give different natural death rates of mosquitoes between susceptible and infected mosquitoes [2]. According to Figure 1 and the parameter descriptions in Table 1, the dynamical equations of WNV transmission are given by System (1).

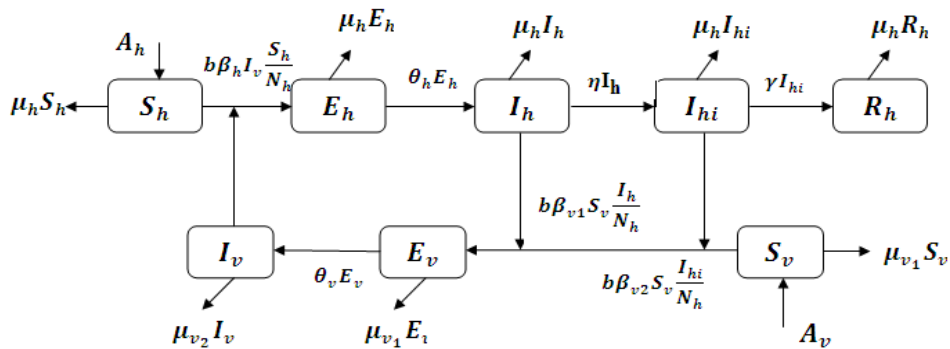


Figure 1 The scheme of host-vector WNV transmission.

WNV transmission model in Chicken-Mosquitoes interaction

$$\left. \begin{aligned}
 \frac{dS_h}{dt} &= \mu_h N_h - \frac{b\beta_h S_h I_v}{N_h} - \mu_h S_h & \frac{dS_v}{dt} &= A_v - \frac{bS_v(\beta_{v1} I_h + \beta_{v2} I_{hi})}{N_h} - \mu_{v1} S_v \\
 \frac{dE_h}{dt} &= \frac{b\beta_h S_h I_v}{N_h} - (\theta_h + \mu_h) E_h & \frac{dE_v}{dt} &= \frac{bS_v(\beta_{v1} I_h + \beta_{v2} I_{hi})}{N_h} - (\theta_v + \mu_{v1}) E_v \\
 \frac{dI_h}{dt} &= \theta_h E_h - (\eta + \mu_h) I_h & \frac{dI_v}{dt} &= \theta_v E_v - \mu_{v2} I_v \\
 \frac{dI_{hi}}{dt} &= \eta I_h - (\gamma + \mu_h) I_{hi} \\
 \frac{dR_h}{dt} &= \gamma I_{hi} - \mu_h R_h
 \end{aligned} \right\} \quad (1)$$

Table 1 Definition and Parameter Values.

Parameters in the models	Definition	Value(s)	Dimension
A_h	Recruitment rate of chickens	-	day ⁻¹
A_v	Recruitment rate of mosquitoes	-	day ⁻¹
b	Biting rate (average number of bites per day)	$0 \leq b \leq 1$	day ⁻¹
β_h	Transmission rate from mosquitoes to chickens	$0 \leq \beta_h \leq 1$	day ⁻¹
β_{v_1}	Transmission rate from chickens to mosquitoes	$0 \leq \beta_{v_1} \leq 1$	day ⁻¹
β_{v_2}	Transmission rate from immune chickens to mosquitoes	$0 \leq \beta_{v_2} \leq 1$	day ⁻¹
μ_h	Natural death rate of chickens	0.00014	day ⁻¹
μ_{v_1}	Natural death rate of mosquitoes	$0.011 \leq \mu_{v_1} \leq 0.05$	day ⁻¹
μ_{v_2}	Death rate of infected mosquitoes	$0.011 \leq \mu_{v_2} \leq 0.04$	day ⁻¹
θ_h	Transition rate from exposed chickens to infected chickens	$0 \leq \theta_h \leq 1$	day ⁻¹
η	Transition rate from infected chickens to immune chickens	$0 \leq \eta \leq 0.143$	day ⁻¹
θ_v	Transition rate from exposed mosquitoes to infected mosquitoes	$0 \leq \theta_v \leq 1$	day ⁻¹
γ	Recovery rate for chickens	$0.015 \leq \gamma \leq 0.1$	day ⁻¹
N_h	Population size of chickens N_h $= \frac{A_h}{\mu_h}$	-	-
N_v	Population size of mosquitoes $N_v = \frac{A_v}{\mu_{v_1}}$	-	-

In the next section, we conduct a dynamical analysis to study the dynamics and identify the critical parameters of WNV transmission.

3 Basic Reproductive Ratio

The disease-free equilibrium of the system is given by $E_0 = \left(S_h = \frac{A_h}{\mu_h}, E_h = 0, I_h = 0, I_{hi} = 0, R_h = 0, S_v = \frac{A_v}{\mu_{v_1}}, E_v = 0, I_v = 0 \right)$. Because of

N_h is constant, it means that $\frac{dS_h}{dt} + \frac{dE_h}{dt} + \frac{dI_h}{dt} + \frac{dI_{hi}}{dt} + \frac{dR_h}{dt} = 0$. Therefore, from Eq. (1) we obtain $N_h = \frac{A_h}{\mu_h}$. Meanwhile, the assumption that N_v is constant, one obtains $\frac{dS_v}{dt} + \frac{dE_v}{dt} + \frac{dI_v}{dt} = 0$ and the lifetime of infected mosquitoes $\frac{1}{\mu_{v2}}$ greater than the lifetime of susceptible mosquitoes $\frac{1}{\mu_{v1}}$ ($\mu_{v1} > \mu_{v2}$) obtains $N_v \geq \frac{A_v}{\mu_{v1}}$. Here, we choose $N_v = \frac{A_v}{\mu_{v1}}$. We obtain the next generation matrix (see [11],[12]), given as follows:

$$NGM = \begin{bmatrix} 0 & 0 & 0 & 0 & \frac{b\beta_h\theta_v}{(\theta_v+\mu_{v1})\mu_{v2}} & \frac{b\beta_h}{\mu_{v2}} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ \frac{b\beta_{v1}\theta_h N_v}{(\theta_h+\mu_h)(\mu_h+\eta)N_h} + \frac{b\beta_{v2}\theta_h\eta N_v}{(\theta_h+\mu_h)(\mu_h+\eta)(\mu_h+\gamma)N_h} & \frac{b\beta_{v1}N_v}{(\mu_h+\eta)N_h} + \frac{b\beta_{v2}\eta N_v}{(\mu_h+\eta)(\mu_h+\gamma)N_h} & \frac{b\beta_{v2}N_v}{(\mu_h+\gamma)N_h} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

The eigen values of NGM are zero of multiplicity 3 and

$$\pm \sqrt{\frac{b^2\beta_h\theta_h\theta_v((\gamma+\mu_h)\beta_{v1}+\eta\beta_{v2})}{\mu_{v2}(\theta_h+\mu_h)(\theta_v+\mu_{v1})(\eta+\mu_h)(\gamma+\mu_h)}} \frac{N_v}{N_h}$$

Therefore the basic reproductive ratio R_0 is given in the form

$$R_0^2 = \frac{b^2\beta_h\theta_h\theta_v((\gamma+\mu_h)\beta_{v1}+\eta\beta_{v2})}{\mu_{v2}(\theta_h+\mu_h)(\theta_v+\mu_{v1})(\eta+\mu_h)(\gamma+\mu_h)} \left(\frac{N_v}{N_h}\right). \tag{3}$$

As shown in Eq. (3), the basic reproductive ratio of System (1) depends on all parameters of System (1), such as the ratio between mosquito and chicken population, $\frac{N_v}{N_h}$, the biting rate, and other transition parameters. We obtain that

E_0 is locally asymptotically stable if and only if $R_0 < 1$ by using the diagonal and determinant method (see [13]) from the Jacobian matrix of System (1). Here, the value of $R_0 < 1$ represents that the new WNV infections produces less than one new infected individual during the infection period, therefore WNV infection cannot spread.

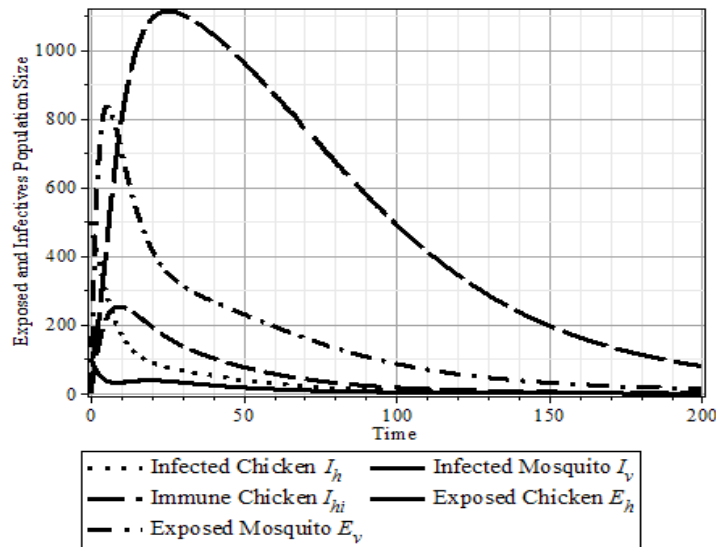


Figure 2 Existence and stability of endemic equilibrium with total population size and vital parameter data set $\{N_h = 400, N_v = 2000, b = 1, \eta = \frac{1}{7}, \gamma = \frac{1}{10}, \theta_v = \frac{1}{8}, \beta_h = 0.01, \mu_{v,2} = 0.04, \beta_{v,1} = 0.18, \beta_{v,2} = 0.1, \theta_h = \frac{1}{4}\}$.

From numerical simulation we found that $R_0 = 1.43$ and endemic equilibrium $E_1 = \{S_{h^*} = 201, E_{h^*} = 1.08, I_{h^*} = 1.9, I_{hi^*} = 2.7, R_{h^*} = 193.3, S_{v^*} = 1939, E_{v^*} = 16.9, I_{v^*} = 54.2\}$. We obtained $R_0 > 1$, it means that the WNV can invade and spread into the chicken and mosquito population. Note that it is not possible to express the endemic equilibrium analytically. The numerical results indicating the stability of the endemic equilibrium are shown in Figure 2.

4 Sensitivity Analysis

We have used the Maple software to show level sets of R_0 for variation parameters such as the lifetime of infected mosquitoes, the lifetime of susceptible mosquitoes, the recovery rate of the chickens, the transition rate from infected chickens to immune chickens, and the effect of different lifetimes of mosquitoes to the change the value of R_0 . The first level sets of R_0 in Figure 3(a) show relations between the transition rate from infected chickens to immune chickens η and the death rate of infected mosquitoes $\mu_{v,2}$ for $\gamma = 0.1$. The second level sets of R_0 in Figure 3(b) show relations between the mortality rate of infected mosquitoes $\mu_{v,2}$ and the recovery rate of the chickens γ for $\eta = 0.143$. Both figures were obtained from hypothetical data $A_h = 500$,

$A_v = 1000$, $b = 1$, and the values of the transition parameters from Sanne, *et al.* [2], namely $\mu_h = 0.0014$, $\beta_h = 0.01$, $\theta_h = 0.25$, $\mu_{v1} = 0.048$, $\beta_{v1} = 0.18$, $\beta_{v2} = 0.1$, and $\theta_v = 0.125$.

The third and the fourth level sets of R_0 in Figure 4 are relations between the transition rate from exposed chickens to infected chickens θ_h and the transition rate from exposed mosquitoes to infected mosquitoes θ_v . The level sets of R_0 for $\mu_{v1} = 0.049 > 0.039 = \mu_{v2}$ and the level sets of R_0 for $\mu_{v1} = 0.039 < 0.049 = \mu_{v2}$ are shown in Figure 4(a) and Figure 4(b), respectively. Both figure use hypothetical data $A_h = 10$, $A_v = 1000$, $b = 1$ and the values of the transition parameters from Sanne, *et al.* [2], namely $\mu_h = 0.0014$, $\beta_h = 0.01$, $\gamma = 0.1$, $\eta = 0.143$, $\mu_{v1} = 0.048$, $\mu_{v2} = 0.039$, $\beta_{v1} = 0.18$, and $\beta_{v2} = 0.1$.

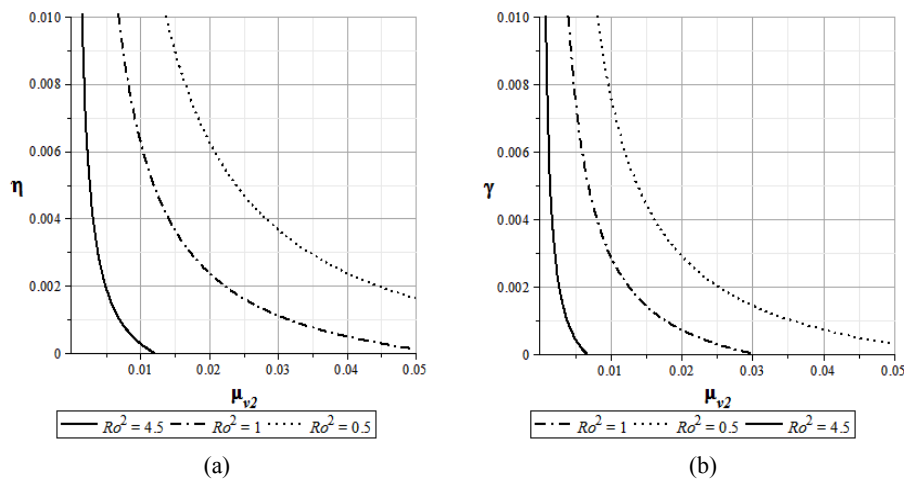


Figure 3 Level sets of R_0 with respect to transition rate from infected chickens to immune chickens η (a), and to recovery rate of chickens to death rate of infected mosquitoes μ_{v2} (b).

Figure 3 shows that R_0 decreased faster with the increase of η (see Figure 3(a)) as well as with the increase of γ (see Figure 3(b)). Suitable, from the experimental data in Sanne, *et al.* [2] shown that the average lifetime of

mosquitoes increases due to infection ($\mu_{v1} > \mu_{v2}$). Also, we conclude that the basic reproductive ratio may decrease, as shown in Figure 3.

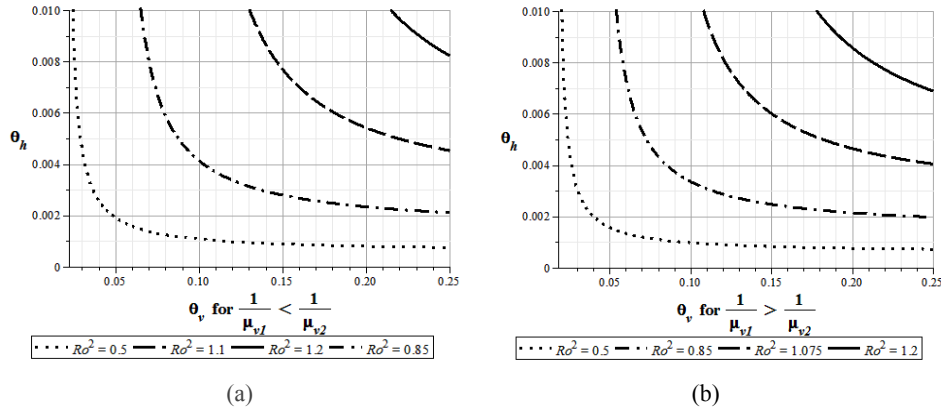


Figure 4 Relation between transition rate from exposed chickens to infected chickens θ_h , and transition rate from exposed mosquitoes to infected mosquitoes θ_v .

Next we studied the effect of the ratio between mosquitoes and chickens to the change the value of R_0 . Here, we have used parameter values $\beta_h = 0.01$, $\beta_{v_2} = 0.18, b = 1$, while the others parameter values from Table 1. Substituting these values in Eq. (3), we obtained the value of

$$R_0^2 = 0.64 \frac{N_v}{N_h}. \tag{4}$$

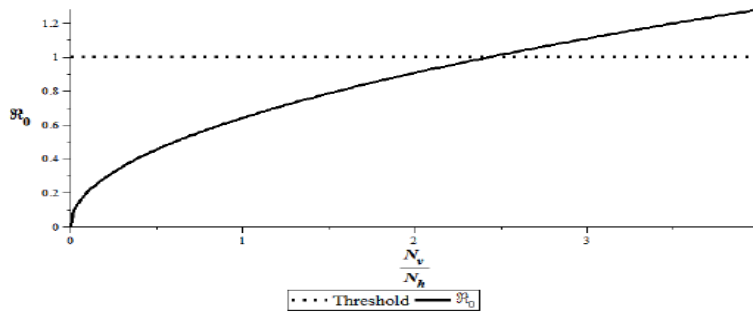


Figure 5 Magnitude of R_0 against ratio $\frac{N_v}{N_h}$

From Figure 4, R_0^2 is proportional to the ratio between mosquito and chicken population sizes. From Figure 5, we have $R_0 \leq 1$ for $0 < \frac{N_v}{N_h} \leq 2.24$ in Eq. (5).

It shows that the WNV infection cannot spread. In other words, the WNV will die out if the number of mosquitoes is smaller than 2.24 times the number of chickens. Furthermore, $\frac{N_v}{N_h} > 2.24$ show the WNV is endemic if the number of mosquitoes is more than 2.24 times the number of chickens. The simulation of the dynamics of I_h, I_{hi} , and I_v is shown in Figure 6.

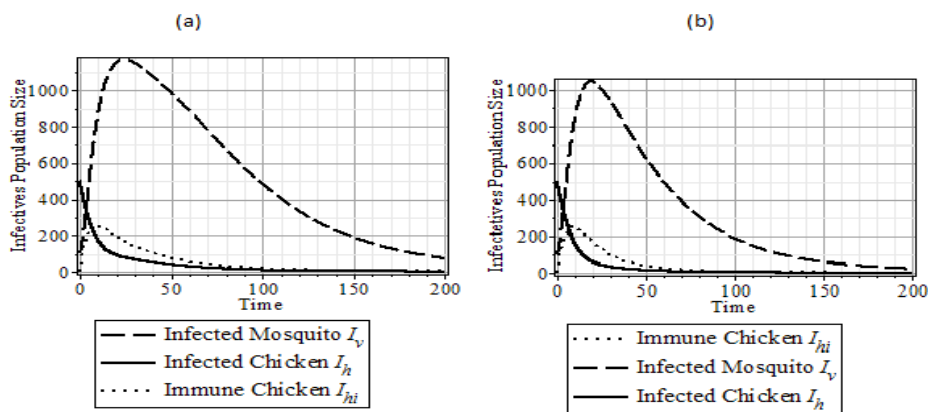


Figure 6 The dynamics of I_h, I_{hi} , and I_v population with the following parameter data set: $\{N_h = 400, N_v = 2000, b = 1, \eta = \frac{1}{7}, \gamma = \frac{1}{10}, \theta_v = \frac{1}{8}, \mu_{v2} = 0.04, \beta_h = 0.01, \beta_{v1} = 0.18, \beta_{v2} = 0.1, \theta_h = \frac{1}{4}\}$. (a) $\mu_{v1} = 0.049 > 0.039 = \mu_{v2}$, and $R_0 = 1.43$. (b) $\mu_{v1} = 0.039 < 0.049 = \mu_{v2}$, and $R_0 = 1.32$.

Figure 6 shows that the effect of the increased lifetime of WNV infected mosquitoes accelerates the increase of the number of WNV infections at the beginning of the epidemic growth and extends the outbreak period.

5 Conclusion

We have obtained a model for WNV transmission for chicken-mosquito interaction, taking into account two stages of infection before the recovered chickens and the effect of the prolongation of the lifetime of the infected chickens. The numerical results indicate that the increase of lifetime of infected mosquitoes may reduce the basic reproductive ratio.

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References

- [1] Compton, J., Feldman D., Orizaga, S. & Stein, M., *A Model for West Nile Virus Transmission VIGRE Summer Program-2007*, PhD Dissertation. Mathematics Department, Texas A & M University, Texas, 2007.
- [2] Senne, D.A., Pedersen, J.C., Hutto, D.L., Taylor, W.D., Schmitt B.J. & Panigrahy, B., *Pathogenicity of West Nile Virus in Chickens*, *Avian Disease*, **44**, pp. 642-649, 2000.
- [3] Centers for Disease Control (CDC), *West Nile Virus*. Division of Vector-Borne Infectious Diseases, <http://www.cdc.gov/ncidod/dvbid/westnile/>, (20 March 2014).
- [4] Tabler, T., Wells, J. & Zhai, W. *Farmers, Chickens, and West Nile Virus*, Mississippi State University, Publication 2735B, 2013.
- [5] Cruz-Pacheco, G., Esteva, L., Montaña-Hirose, J.A. & Vargas C., *Modelling the Dynamics of West Nile virus*, *Bull. Math. Biol.*, **67**(6), p. 1157-1163, 2005.
- [6] Langevian, S.A., Bunning, M., Davis, B. & Komar, N., *Experimental Infection of Chickens as Candidate for West Nile Virus*, *Emerging Infectious Diseases*, **7**(4), pp. 726-729, 2001.
- [7] Wonham, M.J., De-Camino-Beck, T. & Lewis, M., *An Epidemiological Model for West Nile Virus: Invasion Analysis and Control Applications*, *Proc. Roy. Soc. London, Series B* 1538, pp. 501-507, 2004.
- [8] Bowman, C., Gumel, A.B., Wu, J., Van-den Driessche, P. & Zhu, H., *A Mathematical Model for Assessing Control Strategies Against West Nile Virus*, *Bull. Math. Biol.*, **67** (5), pp. 1107-1116, 2005
- [9] Lord, C. & Day, J.F., *Simulation Studies of St. Louis Encephalitis Virus in South Florida*, *Vector Borne Zoonotic Diseases*, **1**(4), pp. 299-306, 2001.
- [10] Lord, C. & Day, J.F., *Simulation Studies of St. Louis Encephalitis and West Nile Viruses: the Impact of Bird Mortality*, *Vector Borne Zoonotic Diseases*, **1**(4), pp. 317-323, 2001.
- [11] Diekmann, O. & Heesterbeek, J.A.P., *Mathematical Epidemiology of Infectious Diseases: Model Building, Analysis and Interpretation*, Editor in Chief Simon Levin, Princeton University, USA, Wiley Series in Mathematical and Computational Biology, 2000.

- [12] Van den Driessche, P. & Watmough, J., *Reproduction Numbers and Subthreshold Endemic Equilibria for Compartmental Models of Disease Transmission*, Math. Bioscience, **180**, pp. 29-48, 2000.
- [13] Horn, R.A. & Johnson, C.R., *Topics in Matrix Analysis*, Corrected Reprint of The 1991 Original, Cambridge University Press, Cambridge, 1994.