Journal of Orissa Mathematical Society ISSN: 0975-2323
Vol. 37, No. 01-02, 2018, 37-54

A Stochastic Disease Models for Zika - Exploring the Probability of Pathogen Persistence

Donald D. Porchia*

Abstract

A stochastic epidemic model for the transmission dynamics of Zika is formulated as a continuous-time Markov chain. The stochastic model is derived from a deterministic compartmental disease model based on a coupled system of ordinary differential equations. The disease dynamics of the deterministic and stochastic disease models are compared in order to determine the effect of stochasticity on the transmission dynamics. The probability of disease extinction as well as that of a epidemic are numerically simulated from the stochastic model and compared to a multi-type Bienamye-Galton-Watson branching process approximation. Analytical and numerical results show significant differences between the stochastic and deterministic model predictions.

1 Introduction

The Zika virus (ZIKV) was first discovered in the blood of a rhesus monkey captured in the Zika forests of Uganda at the Yellow Fever Research Institute in 1947 [10]. The virus is named after

*Dept. of Mathematics, University of Central Florida, Orlando, USA, email: Donald.Porchia@ucf.edu

the forest where it it was first discovered. The following year, the virus was found in the Aedes africanus mosquito, [11]. By 1952, antibodies against Zika were found in the sera of individuals in Uganda and the republic of Tanzania [25]. In the following years, successive serosurveys found evidence of ZIKV antibodies in the human populations of Africa, India and Southeast Asia, [12, 18, 21, 25, 26, 27].

From the 1960s though the 1980s, a small number of human cases were detected along with widespread exposure to the virus [28]. The first confirmed transmission of the disease in humans was discovered when a researcher working with the virus was infected in 1964. Zika was also detected in mosquitoes in Asia, Indonesia, India, and Pakistan [23].

The disease was largely ignored until the first large scale outbreak in humans occurred on the pacific island of Yap in the Federated States of Micronesia in 2007. Until the 2007 Yap outbreak, only 14 cases had been reported world wide. After the Yap outbreak, it is estimated that 73% of the island's population has been infected. It is possible that regular exposure combined with mild or asymptomatic infections in Africa and Asia have prevented the large outbreaks seen on Yap or in the Americas [28].

In 2008, the first case of Zika spread by sexual transmission was detected. This is the first documented case of sexual transmission of a disease typically spread by insects [15]. In 2013 - 2014, four outbreaks occurred in French Polynesia, Easter Island, the Cook Islands and New Caledonia [24]. In the aftermath of these outbreaks, a possible association was found between Zika and congenital malformations such as microcephaly as well as severe neurological and autoimmune complications such as GuillainBarr syndrome [28].

The Zika virus (ZIKV) is a vector-borne disease transmitted to humans through the bites of infected mosquitoes of the genus Aedes. It can also be transmitted among humans by sexual contact, blood transfusions or vertically from mother to child. Although first discovered in 1947,

there were no large scale outbreaks of the disease until the 2008 outbreak in Micronesia. Until then, the symptoms of Zika where thought to be mild. It was only afterward that a correlation was discovered between Zika infection during pregnancy and microcephaly as well as other severe neurological conditions such as Guillian-Barre syndrome. Due to this fact and the large scale nature of recent outbreaks the World Health Organization has declared that the Zika virus and its associated complications are a public health emergency of international concern.

The organization of this paper is as follows: Section 2, we formulate the deterministic model; in Section 3 we present the deterministic model analysis; in Section 4, we formulate the stochastic model as a continuous time Markov chain (CTMC); in section 5, we discuss the branching process approximation to the CTMC. Section 6 concludes our work and provides a discussion.

2 The Deterministic Model

Zika is unique in that it is a vector transmitted disease that also allows for transmission by sexual contact. We will describe the disease transmission by a coupled system of differential equations. The host population will be described by a SIR model in which each individual may be classified as being in one of three epidemiological states at time t: Susceptible $S_h(t)$, Infected $I_h(t)$, or Removed $R_h(t)$. The total population is defined as $N_h(t) = S_h(t) + I_h(t) + R_h(t)$. The vector population will be described by a system in which each mosquito is in only one of two epidemiological states either susceptible $S_v(t)$ or infected $I_v(t)$. The total mosquito populations is defined as $N_v(t) = S_v(t) + I_v(t)$. Since we are interested in the probability of a epidemic starting and not the long term dynamics of the disease, we will assume that the birth and death rate for the human and mosquito population are in equilibrium. Hence the host and vector populations are constant.

Due to the short lifespan of the vector we will not consider recovery for the vector and assume the mosquito stays infected until death. Humans will recover from the disease at the rate, μ_h. The

contact rate from mosquitoes to susceptible humans β_{hv} is defined as the product of the number of bites received by a human per unit time (bite rate) and the probability that the bite could transmit the infection, ρ_{hv}. Thus, $\beta_{hv} = b\rho_{hv}$ Zika can also be transmitted through sexual contact so let β_{hh} be the transmission rate from infected human to susceptible human. Additionally, as a vector borne virus, infected humans transmit the disease to susceptible mosquitoes at a rate $\beta_{vh} = b\rho_{vh}$, where b is the bite rate and ρ_{vh} is the probability of a infected human transmitting the disease to a susceptible mosquito upon being bitten.

Using the above definitions, we have that a susceptible human receives β_{hv} mosquito bites capable of transmitting the disease per unit time. The fraction of those bites that come from a infected mosquito is given by $\frac{I_v}{N_v}$. Therefore the number of new infective humans per unit time from vector transmission is

$$\beta_{hv} S_h \frac{I_v}{N_v}.$$

Similarly, the number of new infective mosquitoes per unit time from host transmission is given by

$$\beta_{vh} S_v \frac{I_h}{N_h}.$$

However, Zika is the first example of a vector borne disease with a direct transmission route. Thus, the number of new infected humans per unit time from sexual contact is given by

$$\beta_{hh} S_h \frac{I_h}{N_h}.$$

The variables are integer values since they describe the population size of each compartment. However, if we assume the size is sufficiently large then we can treat them as continuously valued.

The $S_h I_h R_h - S_v I_v$ model description is given by the following system of equations,

$$
\begin{aligned}
S_h'(t) &= \mu_h N_h - \mu_h S_h(t) - \beta_{hh} S_h(t) \frac{I_h(t)}{N_h} - \beta_{hv} S_h(t) \frac{I_v(t)}{N_v} \\
I_h'(t) &= \beta_{hh} S_h(t) \frac{I_h(t)}{N_h} + \beta_{hv} S_h(t) \frac{I_v(t)}{N_v} - (\gamma_h + \mu_h) I_h(t) \\
R_h'(t) &= \gamma_h I_h(t) - \mu_h R_h(t) \\
S_v'(t) &= \mu_v N_v - \mu_v S_v(t) - \beta_{vh} S_v(t) \frac{I_h(t)}{N_h} \\
I_v'(t) &= \beta_{vh} S_v(t) \frac{I_h(t)}{N_h} - \mu_v I_v(t)
\end{aligned} \tag{2.1}
$$

Adding the first three equations, we have that $N_h'(t) = 0$ and $N_h(t)$ is a constant, which is to be expected since we built the model assuming that the number of births was equal to the number of deaths. Similarly, $N_v(t)$ is a constant for the vector populations as well.

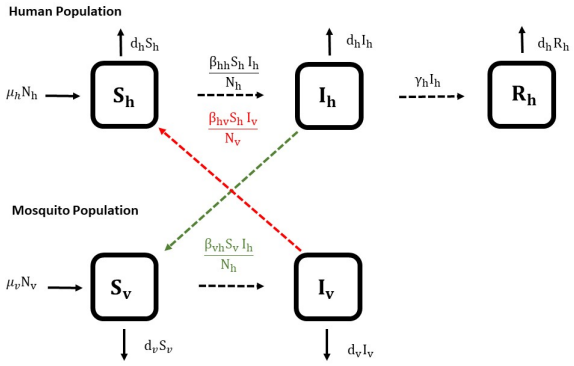

Figure 1: Schematic representation of the Zika epidemic model

3 The Deterministic Model Analysis

3.1 Existence and Stability of Model Equilibria

We find the equilibrium points by setting the right hand side of the model (2.1) equal to zero. In other words, assume that $S_h' = 0$, $I_h' = 0$, $R_h' = 0$, $S_v' = 0$, and $I_v' = 0$ then solve the following system of equations

Parameter	Description	Range	Value	Reference
α	Mosquito biting rate. (Number of bites per mosquito per day)	0.3 - 1.0	0.5	[1, 16]
β_{hh}	Transmission rate from symptomatically infected humans to susceptible humans. (Per day)	0.0010.10	0.05	Assumed
ρ_{hv}	Transmission probability from an infectious mosquito to a susceptible human per bite. (Dimensionless)	0.1 - 0.75	0.4	[1, 16]
ρ_{vh}	Transmission probability from a symptomatically infected human to a susceptible mosquito per bite. (Dimensionless)	0.30.75	0.5	[9, 16]
$1/\mu_h$	lifespan in the human population (days)	-	$(365)(76)$	reference
$1/\mu_v$	lifespan in the vector population (days)	4 - 35	14	[1, 9, 16]
N_h	Population of humans	range	1000000	Assumed
$N_{ratio} = \frac{N_v}{N_h}$	Average ratio of mosquitoes to humans. (mosquitoes per human)	1 - 10	5	[22]
N_v	Population of mosquitoes	-	5000000	calculated
$1/\gamma_h$	Duration of human infection (days)	2 - 7 days	5	[8, 16]

Table 1: Parameter description and ranges for the Zika epidemic model

$$\begin{aligned}
\mu_h N_h - \mu_h S_h(t) - \beta_{hh} S_h(t)\frac{I_h(t)}{N_h} - \beta_{hv} S_h(t)\frac{I_v(t)}{N_v} &= 0 \\
\beta_{hh} S_h(t)\frac{I_h(t)}{N_h} + \beta_{hv} S_h(t)\frac{I_v(t)}{N_v} - (\gamma_h + \mu_h) I_h(t) &= 0 \\
\gamma_h I_h(t) - \mu_h R_h(t) &= 0 \\
\mu_v N_v - \mu_v S_v(t) - \beta_{vh} S_v(t)\frac{I_h(t)}{N_h} &= 0 \\
\beta_{vh} S_v(t)\frac{I_h(t)}{N_h} - \mu_v I_v(t) &= 0
\end{aligned} \tag{3.1}$$

The Zika free equilibrium occurs when $I_h = I_v = 0$ and is given by

$$(S_h^o, I_h^o, R_h^o, S_v^o, I_v^o) = (N_h, 0, 0, N_v, 0).$$

The existence of the endemic equilibrium, $I_h \neq 0$, $I_v \neq 0$, was shown in [17].

The basic reproduction number, $\mathscr{R}_0$ is defined to be the number of individuals that one infective will infect in a completely susceptible population. In other words, the number of secondary cases caused by one infected individual assuming a completely susceptible population.

We may now calculate the basic reproduction number using the next generation method as described by Van den Driessche and Watmough in [14].

$$\mathbb{N} = \begin{bmatrix} \frac{\beta_{hh}}{\gamma_h + \mu_h} & \frac{N_h \beta_{hv}}{N_v \mu_v} \\ \frac{N_v \beta_{vh}}{N_h(\gamma_h + \mu_h)} & 0 \end{bmatrix}$$

Then the basic reproduction number is the spectral radius of the next generation matrix which is given by

$$\mathscr{R}_o = \frac{\beta_{hh}}{2(\gamma_h + \mu_h)} + \frac{1}{2}\sqrt{\left(\frac{\beta_{hh}}{\gamma_h + \mu_h}\right)^2 + \frac{4\beta_{hv}\beta_{vh}}{\mu_v(\gamma_h + \mu_h)}} \tag{3.2}$$

$$= \frac{\mathscr{R}_h + \sqrt{\mathscr{R}_h^2 + 4\mathscr{R}_v^2}}{2} \tag{3.3}$$

where $\mathscr{R}_h = \frac{\beta_{hh}}{\gamma_h+\mu_h}$ is the reproduction number assuming only infections due to sexual transmission and $\mathscr{R}_v = \sqrt{\frac{\beta_{hv}\beta_{vh}}{\mu_v(\gamma_h+\mu_h)}}$ is the reproduction number assuming only vector borne infections.

From [14], we may apply Theorem 2 to establish the following result,

Theorem 3.1 *The Zika free equilibrium of model (2.1) is locally asymptotically stable if* $\mathscr{R}_o \leq 1$ *and unstable if* $\mathscr{R}_o$¿ 1.

4 Stochastic Epidemic Model

Stochastic modeling allows the random nature of the disease dynamics to be expressed in the model itself. When formulating a discrete or continuous Markov chain, instead of approximating the epidemiological classes as a continuum they are treated naturally as discrete positive integers. The transmission and the recovery of the disease is defined or governed as a probability, so there is always a chance that an individual will not infect another or that they will recover from the disease. That is, the disease can die out and there can be no further infection until the disease is reintroduced into the population. Whereas in a deterministic model, the disease can become infinitesimally small only to grow again. This is an artifact of the continuum approximation. The ability of the disease to become stochastically extinct is one of the major differences between the deterministic and stochastic models. In order to determine the probability of a major outbreak or disease extinction, we must consider a stochastic model. In this section we will consider a continuous time Markov chain (CTMC) model with a discrete number of hosts and vectors. We will apply the theory of multitype branching processes to estimate the probability of a major outbreak or disease extinction.

4.1 CTMC Model Formulation

Since time is continuous, we shall formulate the stochastic model as a continuous time Markov chain, although the dynamics for the a discrete time Markov chain will be similar. This model

can be thought of as a multitype birth and death process.

Let $S_h(t)$, $I_h(t)$, and $R_h(t)$ denote the discrete-valued random variables for the number of susceptible, infectious and recovered human hosts at time t. respectively. Let $S_v(t)$ and $I_v(t)$ denote the discrete-valued random variables for the number of susceptible and infectious mosquito vectors at time t. As before, let the total population for the hosts and vectors be given by $N_h(t) = S_h(t) + I_h(t) + R_h(t)$ and $N_v(t) = S_v(t) + I_v(t)$. For simplicity, the same notation is used for the discrete random variables and parameters as in the deterministic model.

For a CTMC model, we make the assumption that the transitions from one state to another may occur at any time t. The state transition rates for the CTMC model are presented in Table 2. It is assumed that in a any given interval Δt that at most only one event occurs. The multivariate stochastic process is defined as

$$X(t) = \{S_h(t), I_h(t), R_h(t), S_v(t), I_v(t), R_v(t) : t \in [0, \infty]\}$$

with joint probability distribution given by

$$p_{s_h,i_h,r_h,s_v,i_v,r_v}(t) = Prob\{S_h(t) = s_h, I_h(t) = i_h, R_h(t) = r_h, S_v(t) = s_v, I_v(t) = i_v, R_v(t) = r_v\}.$$

It is assumed that the this stochastic process is time homogeneous and satisfies the Markov property. The Markov property implies that the inter-event time is exponentially distributed with parameter

$$\omega(X(t)) = \mu_h N_h + \mu_v N_v + \beta_{hh} S_h(t) I_h(t) + \beta_{hv} S_h(t) I_v(t) + \beta_{vh} S_v(t) I_h(t) + \gamma_h I_h$$

5 Stochastic Threshold for Disease Extinction

In a stochastic epidemic model it is possible to make predictions concerning the probability of a disease outbreak or disease extinction. In a single group birth-death process, a birth can be

Table 2: State Transition rates for the simple model

Description	State Transition	Rate
Birth of a susceptible human	$(S_h, I_h, R_h, S_v, I_v) \to (S_h+1, I_h, R_h, S_v, I_v)$	$\mu_h N_h$
Death of a susceptible human	$(S_h, I_h, R_h, S_v, I_v) \to (S_h-1, I_h, R_h, S_v, I_v)$	$\mu_h S_h$
Death of a infected human	$(S_h, I_h, R_h, S_v, I_v) \to (S_h, I_h-1, R_h, S_v, I_v)$	$\mu_h I_h$
Death of a recovered human	$(S_h, I_h, R_h, S_v, I_v) \to (S_h, I_h, R_h-1, S_v, I_v)$	$\mu_h R_h$
Birth of a susceptible vector	$(S_h, I_h, R_h, S_v, I_v, R_v) \to (S_h+1, I_h, R_h, S_v, I_v)$	$\mu_v N_v$
Death of a susceptible vector	$(S_h, I_h, R_h, S_v, I_v) \to (S_h, I_h, R_h, S_v-1, I_v)$	$\mu_v S_v$
Death of a infected vector	$(S_h, I_h, R_h, S_v, I_v) \to (S_h, I_h, R_h, S_v, I_v-1)$	$\mu_v I_v$
Infection of a human	$(S_h, I_h, R_h, S_v, I_v) \to (S_h-1, I_h+1, R_h, S_v, I_v)$	$\beta_{hh} S_h(t) I_h(t) + \beta_{hv} S_h(t) I_v(t)$
Infection of a mosquito	$(S_h, I_h, R_h, S_v, I_v, R_v) \to (S_h, I_h, R_h, S_v-1, I_v+1)$	$\beta_{vh} S_v(t) I_h(t)$
Recovery of a infected human	$(S_h, I_h, R_h, S_v, I_v, R_v) \to (S_h+1, I_h-1, R_h, S_v, I_v)$	$\gamma_h I_h$

thought of as an infection and a death as a recovery. If $\mathscr{R}_0 > 1$ and if there are i_o initial infections in a wholly susceptible population then the probability of a major outbreak is approximated by $1-(\frac{1}{\mathscr{R}_0})^{i_o}$. The probability of disease extinction is $(\frac{1}{\mathscr{R}_0})^{i_o}$ [29]. However, this result is only valid when infections arise from a single infectious group. It is based on a single group branching process [2, 3, 4].

In the case of multiple infectious groups, the probability of disease extinction or outbreak may be approximated using a multitype branching process theory. The probability for extinction in the multigroup case depends on both the number of initially infected individuals in each group along with the extinction probability for each group. Further, unlike in the deterministic model, persistence is not guaranteed simply because $\mathscr{R}_0 > 1$. One of the most profound differences between a deterministic and a stochastic model is the fact that disease extinction is possible immediately following the initial infectious even when the deterministic model would assume a endemic equilibrium. During the initial stages of the infection, the invasion probabilities are approximated by assuming that the whole population is susceptible using a Bienamye-Galton-Watson branching process [6, 7].

5.1 The Bienamye-Galton-Watson branching process (BGWbp)

The branching process theory can be used to find the disease invasion and extinction probabilities for a multigroup disease model. The general theory for a multitype branching process will be presented and then it will be applied to the Zika model in order to calculate the probability of a major outbreak and disease extinction. We begin by defining a Bienamye-Galton-Watson branching process as in [2, 3, 4],

Definition 5.1 *A multitype BGWbp $I(t)_{t=0}^{\infty}$ is a collection of vector random variables $I(t)$, where each vector consists of k different types, $I(t) = (I_1(t), I_2(t), \cdots, I_k(t))$ and each random variable $I_i(t)$ has k associated offspring random variables for the number of offsprings of type $j = 1, 2, \cdots, k$ from a parent of type i.*

We assume that there i types of infectious group with $I_i(t)$ members at any time t. Each member can give birth to an individual of type j, $I_j(t)$. If $i = j$ then this would be a within group transmission (sexual transmission in the case of Zika) and if $i \neq j$ then it would be between group transmission (vector to host or host to vector transmission in the case of Zika). The number of offspring generated by any type i individual is assumed to be independent. We also assume that the offspring generating function for any individual of the same type is identical.

Let $Z_{ji}{}_{j=i}^{n}$ be a sequence of random variables representing the number of offspring of type j generated by a type i individual. The offspring probability generating function (pgf) for the infectious population, I_i is then defined for a single infectious individual at the start of the epidemic, $I_i(0) = 1$ for some $i = 1, \cdots, n$ and all other groups $I_j(0) = 0$ for all $j \neq i$. Therefore, the offspring pgf, $f_i : [0, n]^n \to [0, 1]$ is defined as

$$f_i(\zeta_1, \cdots, \zeta_n) = \sum_{\kappa_n=0}^{\infty} \cdots \sum_{\kappa_1=0}^{\infty} P_i(\kappa_1, \cdots, \kappa_n) \zeta_1^{\kappa_1} \cdots \zeta_n^{\kappa_n}$$

where

$$P_i(\kappa_1,\cdots,\kappa_n) = Prob\{Z_{1i}=\kappa_1,\cdots,Z_{ni}=\kappa_n\}$$

is the probability that one infected individual of type i infects or "gives birth" to κ_j individuals of type j. There is always a fixed point of the pfg, $f_i(1,\cdots,1)=1$ [2, 3, 4, 19, 5]. Note that $f_i(0,\cdots,0)$ denotes the extinction probability $I_i(0)$. We can define a stochastic threshold analogous to the spectral radius of the next generation matrix in the deterministic case as the expectation matrix of the offspring probability function. Define $\mathbb{M}=[m_{ji}]$ as an *nxn*, nonnegative, irreducible matrix where the m_{ji} entry is the expected number of offsprings of type j individuals produced by an infected type i individual. The elements of the matrix $\mathbb{M}$ are calculated as follows,

$$m_{ji} = \left.\frac{\partial f_i}{\partial u_j}\right|_{\kappa=1}$$

The stochastic threshold for disease extinction or persistance of the multitype BGWbp is determined by the size of the spectral radius of the expectation matrix $\mathbb{M}$. Thus if the spectral radius, $\rho(\mathbb{M})\leq 1$, then the probability of ultimate disease extinctions as $t\to\infty$ is one. However, if $\rho(\mathbb{M})>1$ then there is a positive probability that the disease may persist. Following the work of Allen, van den Driessche, and Lahodny in [2, 3, 4, 5, 13, 19, 20], we summarize the results in the following theorem,

Theorem 5.1 *Let $I(t)$ be a BGWbp and its associated expectation matrix $\mathbb{M}$ defined as above be nonnegative and irreducible.*

(i) If $\rho(\mathbb{M})\leq 1$ then the BGWbp is subcritical or critical with

$$\lim_{t\to\infty} Prob\{I(t)=0\}=1.$$

(ii) If $\rho(\mathbb{M})>1$ then the BGWbp is supercritical with

$$\lim_{t\to\infty} Prob\{I(t)=0\}=q_1^{i_1}\cdots q_n^{i_n}<1.$$

where $(q_1, \cdots, q_n)$ is the unique fixed point of n offspring pgfs and $(i_1, \cdots, i_n)$ is the vector of initial infectives for the n groups. The $q_{i's}$ are the probabilities of disease extinction for infectives of type i and the probability of disease extinction is approximately

$$q_1^{i_1} \cdots q_n^{i_n}.$$

5.2 Extinction Probabilities for Zika

In this CTMC model of Zika virus, there are two infectious classes: the infected humans (hosts) and the infected mosquitoes (vectors). Let the humans be type 1 and the mosquitoes type 2. The stochastic threshold can then be approximated by a two-type BGWbp. Let the infected human hosts be denoted as type 1 and let the mosquito vectors be denoted as type 2.

In order to create the offspring probability generating function for the branching process, we will use the state transitions for the CTMC model in Table 2. Define the offspring probability generating function for type 1 humans as f_i. Assume that the population is at the Zika free equilibrium with one infected human. The rate at which one infected human can infected a susceptible human is covered by sexual transmission and is give by $\beta_{hh}N_h$ which results in two infected humans in the population. However, this is not the only infection can arise as there is also the possibility that the human will infect a vector at the rate $\beta_{vh}N_v$ which will result in one infected human and one infected mosquito. Finally, the infected human may die or recover from the disease at the rate $\mu_h + \gamma_h$. In order to form the offspring generating function, these rates must be converted into probabilities by dividing by the sum of the rates of infection, death and recovery which is equal to $\beta_{hh}N_h + \beta_{vh}N_v + \mu_h + \gamma_h$. Therefore, the offspring pgf for type 1 humans is given by

$$f_1(\zeta_1,\zeta_2) = P_1(2,0)\zeta_1^2 + P_1(1,1)\zeta_1\zeta_2 + P_1(0,0) \tag{5.1}$$

$$= \frac{\beta_{hh}N_h\zeta_1^2 + \beta_{vh}N_v\zeta_1\zeta_2 + \mu_h + \gamma_h}{\beta_{hh}N_h + \beta_{vh}N_v + \mu_h + \gamma_h}, \quad \zeta_1,\zeta_2 \in [0,1] \tag{5.2}$$

Similarly, for the case of type 2 mosquitoes, we have

$$f_2(\zeta_1,\zeta_2) = P_2(1,1)\zeta_1\zeta_2 + P_2(0,0) \tag{5.3}$$

$$= \frac{\beta_{hv}N_h\zeta_1\zeta_2 + \mu_v}{\beta_{hv}N_h + \mu_h}, \quad \zeta_1,\zeta_2 \in [0,1] \tag{5.4}$$

We may now calculate the expectation matrix from the offspring probabilities. The individual matrix elements, m_{ji}, are given by

$$m_{11} = \left.\frac{\partial f_1(\zeta_1,\zeta_2)}{\partial\zeta_1}\right|_{\zeta_1=1,\zeta_2=1} = \frac{2\beta_{hh}N_h + \beta_{vh}N_v}{\beta_{hh}N_h + \beta_{vh}N_v + \mu_h + \gamma_h} \tag{5.5}$$

$$m_{12} = \left.\frac{\partial f_2(\zeta_1,\zeta_2)}{\partial\zeta_1}\right|_{\zeta_1=1,\zeta_2=1} = \frac{\beta_{hv}N_h}{\beta_{hv}N_h + \mu_h} \tag{5.6}$$

$$m_{21} = \left.\frac{\partial f_1(\zeta_1,\zeta_2)}{\partial\zeta_2}\right|_{\zeta_1=1,\zeta_2=1} = \frac{\beta_{vh}N_v}{\beta_{hh}N_h + \beta_{vh}N_v + \mu_h + \gamma_h} \tag{5.7}$$

$$m_{22} = \left.\frac{\partial f_2(\zeta_1,\zeta_2)}{\partial\zeta_2}\right|_{\zeta_1=1,\zeta_2=1} = \frac{\beta_{hv}N_h}{\beta_{hv}N_h + \mu_h} \tag{5.8}$$

and therefore the expectation matrix is given by

$$\mathbb{M} = \begin{bmatrix} \frac{2\beta_{hh}N_h+\beta_{vh}N_v}{\beta_{hh}N_h+\beta_{vh}N_v+\mu_h+\gamma_h} & \frac{\beta_{hv}N_h}{\beta_{hv}N_h+\mu_h} \\ \frac{\beta_{vh}N_v}{\beta_{hh}N_h+\beta_{vh}N_v+\mu_h+\gamma_h} & \frac{\beta_{hv}N_h}{\beta_{hv}N_h+\mu_h} \end{bmatrix} = \begin{bmatrix} A+B & C \\ B & C \end{bmatrix}$$

where

$$A = \frac{2\beta_{hh}}{\beta_{hh}N_h + \beta_{vh}N_v + \mu_h + \gamma_h} \quad B = \frac{\beta_{vh}N_v}{\beta_{hh}N_h + \beta_{vh}N_v + \mu_h + \gamma_h} \quad C = \frac{\beta_{hv}N_h}{\beta_{hv}N_h + \mu_h}$$

The spectral radius of the expectation matrix, $\rho(\mathbb{M})$, is the solutions to the following

$$\lambda = \frac{1}{2}(A+B+C) + \frac{1}{2}\sqrt{(A+B+C)^2 - 4BC}$$

In order to find the extinction probabilities, we must find the fixed points of the nonlinear equations,

$$\frac{\beta_{hh}N_h\zeta_1^2+\beta_{vh}N_v\zeta_1\zeta_2+\mu_h+\gamma_h}{\beta_{hh}N_h+\beta_{vh}N_v+\mu_h+\gamma_h}=\zeta_1 \tag{5.9}$$

$$\frac{\beta_{hv}N_h\zeta_1\zeta_2+\mu_v}{\beta_{hv}N_h+\mu_h}=\zeta_2 \tag{5.10}$$

6 Discussion

Validation of the numerical models and simulations will be conducted in a future paper. However, qualitatively we have seen that the expectation of the stochastic model behaves similarly to the deterministic model. Moreover, for any given stochastic realization there is a distinct possibility of stochastic extinction. The Bienamye-Galton-Watson branching process provides a mechanism for calculating the probability of that event. Further, it is possible to quantify the effect that the direct sexual selection has on the disease. Because of the low transmission possibility, in this model its effects on the extinction probability is negligible.

References

1. Andraud M, Hens N, Marais C and Beutels P (2012) Dynamic epidemiological models for dengue transmission: a systematic review of structural approaches. PLoS One 7(11)

2. Allen LJS (2008) An introduction to stochastic epidemic models. In: Brauer F, van den Driessche P, Wu J (eds) Mathematical epidemiology. Springer, Berlin, pp 77128

3. Allen LJS (2010) An introduction to stochastic processes with applications to biology, 2nd edn. Chapmanand Hall/CRC Press, Boca Ratton

4. Allen LJS (2012) Branching processes. Encyclopedia of theoretical ecology. University of California Press, Berkeley

5. Allen LJS (2015) Stochastic population and epidemic models: persistence and extinction. Mathematical biosciences institute lecture series, stochastics in biological systems, vol 1.3. Springer International Publishing, Berlin

6. Ball F (1983) The threshold behaviour of epidemic models. J Appl Probab 20:227241

7. Ball F, Donnelly P (1995) Strong approximations for epidemic models. Stoch Process Appl 55:121

8. Bearcroft WGC (1956) Zika virus infection experimentally induced in a human volunteer, Trans. R. Soc. Trop. Med. Hyg. 50(5), 442448

9. Chikaki E, and Ishikawa H (2009) A dengue transmission model in Thailand considering sequential infections with all four serotypes, J. Infect. Dev. Countr. 3(9), 711722

10. Dick GWA, Kitchen SF, Haddow AJ (1952) Zika Virus (I). Isolations and serological specificity, Transactions of The Royal Society of Tropical Medicine and Hygiene, Volume 46, Issue 5, 1 September 1952, Pages 509-520, https://doi.org/10.1016/0035-9203(52)90042-4

11. Dick GWA (1952) Zika virus (II). Pathogenicity and physical properties, Transactions of The Royal Society of Tropical Medicine and Hygiene, Volume 46, Issue 5, 1 September 1952, Pages 521-534, https://doi.org/10.1016/0035-9203(52)90043-6

12. Dick GWA (1953) Epidemiological notes on some viruses isolated in Uganda; Yellow fever, Rift Valley fever, Bwamba fever, West Nile, Mengo, Semliki forest, Bunyamwera, Ntaya, Uganda S andZika viruses. Trans. R. Soc. Trop. Med. Hyg. 47, 13-48

13. Allen LJS, van den Driessche P (2013) Relations between deterministic and stochastic thresholds for disease extinction in continuous- and discrete-time infectious disease models. Math Biosci 243:99108

14. van den Driessche P and Watmough J (2001). Reproductive numbers and sub-threshold endemic equilibria for compartment models of disease transmission, Math. Biosci., 180:2948

15. Foy BD, Kobylinski KC, Foy JL, et al. (2011) Probable Non-Vector-borne Transmission of Zika Virus, Colorado, USA. Emerging Infectious Diseases. 2011;17(5):880-882. doi:10.3201/eid1705.101939.

16. Gao D, Lou Y, He D, et al. (2016) Prevention and control of Zika as a mosquito-borne and sexually transmitted disease: a mathematical modeling analysis. Sci Rep 2016;6:28070-28070.

17. Kibona IE and Yang CH (2017) SIR Model of Spread of Zika Virus Infections: ZIKV Linked to Microcephaly Simulations. Health, 9, 1190-1210.

18. Kokernot RH, Casaca VM, Weinbren MP, McIntosh BM, (1965) Survey for antibodies against arthropod-borne viruses in the sera of indigenous residents of Angola. Trans. R. Soc. Trop. Med. Hyg. 59, 563-570

19. Lahodny GE Jr, Allen LJS (2013) Probability of a disease outbreak in stochastic multipatch epidemic models. Bull Math Biol. doi:10.1007/s11538-013-9848-z

20. Lahodny GE, Gautam R, Ivanek R (2015) Estimating the probability of an extinction or major outbreak for an environmentally transmitted infectious disease. J Biol Dyn 9:128155

21. MacNamara FN (1954) Zika virus: A report on three cases of human infection during an epidemic of jaundice in Nigeria. Trans. R. Soc. Trop. Med. Hyg. 48, 139-145

22. de Castro Medeiros LC et al. (2011) Modeling the dynamic transmission of dengue fever: investigating disease persistence, PLoS. Negl. Trop. Dis. 5(1), e942

23. Olson JG, Ksiazek TG, Suhandiman, Triwibowo (1981) Zika virus, a cause of fever in Central Java, Indonesia, Transactions of The Royal Society of Tropical Medicine and Hygiene, Volume 75, Issue 3, 1 January 1981, Pages 389-393, https://doi.org/10.1016/0035-9203(81)90100-0

24. Roth A, Mercier A, Lepers C, Hoy D, Duituturaga S, Benyon E, Guillaumot L, Souares Y. (2014) Concurrent outbreaks of dengue, chikungunya and Zika virus infections - an unprecedented epidemic wave of mosquito-borne viruses in the Pacific 2012-2014. Euro Surveill. https://doi.org/10.2807/1560-7917.ES2014.19.41.20929

25. Smithburn KC (1952) Neutralizing antibodies against certain recently isolated viruses in the sera of human beings residing in East Africa. J Immunol 1952;69:223-234

26. Smithburn KC (1952) Studies on certain viruses isolated in the tropics of Africa and South America; immunological reactions as determined by cross-neutralization tests. J. Immunol. 68, 441-460

27. Smithburn KC, Kerr JA, Gatne PB (1954) Neutralizing antibodies against certain viruses in the sera of residents of India. J. Immunol. 72, 248-257

28. World Heath Organization. (2018) The History of the Zika Virus. http://www.who.int/emergencies/zika-virus/history/en/

29. Whittle P (1995) The outcome of a stochastic epidemic: a note on Bailey's paper. Biometrika 42: 116-122