

Deterministic and stochastic CTMC models from Zika disease transmission

Mona Zevika, and Edy Soewono

Citation: [AIP Conference Proceedings](#) **1937**, 020023 (2018); doi: 10.1063/1.5026095

View online: <https://doi.org/10.1063/1.5026095>

View Table of Contents: <http://aip.scitation.org/toc/apc/1937/1>

Published by the [American Institute of Physics](#)

Articles you may be interested in

[Deterministic and stochastic models for middle east respiratory syndrome \(MERS\)](#)

[AIP Conference Proceedings](#) **1937**, 020017 (2018); 10.1063/1.5026089

[Dimensional reduction for a SIR type model](#)

[AIP Conference Proceedings](#) **1937**, 020005 (2018); 10.1063/1.5026077

[A Gompertz population model with Allee effect and fuzzy initial values](#)

[AIP Conference Proceedings](#) **1937**, 020002 (2018); 10.1063/1.5026074

[A deterministic model of nettle caterpillar life cycle](#)

[AIP Conference Proceedings](#) **1937**, 020019 (2018); 10.1063/1.5026091

[Application of differential transformation method for solving dengue transmission mathematical model](#)

[AIP Conference Proceedings](#) **1937**, 020012 (2018); 10.1063/1.5026084

[A model of immunomodulatory for dengue infection mm](#)

[AIP Conference Proceedings](#) **1937**, 020024 (2018); 10.1063/1.5026096

Deterministic and Stochastic CTMC Models from Zika Disease Transmission

Mona Zevika^{a)} and Edy Soewono^{b)}

Department of Mathematics, Institut Teknologi Bandung, Bandung, Indonesia 40132.

^{a)}monazevika@s.itb.ac.id

^{b)}esoewono@math.itb.ac.id

Abstract. Zika infection is one of the most important mosquito-borne diseases in the world. Zika virus (ZIKV) is transmitted by many Aedes-type mosquitoes including Aedes aegypti. Pregnant women with the Zika virus are at risk of having a fetus or infant with a congenital defect and suffering from microcephaly. Here, we formulate a Zika disease transmission model using two approaches, a deterministic model and a continuous-time Markov chain stochastic model. The basic reproduction ratio is constructed from a deterministic model. Meanwhile, the CTMC stochastic model yields an estimate of the probability of extinction and outbreaks of Zika disease. Dynamical simulations and analysis of the disease transmission are shown for the deterministic and stochastic models.

INTRODUCTION

Zika infection is one of the most important mosquito-borne diseases in the world. The emergence of ZIKV started with a first outbreak in the Pacific area in 2007, a second large outbreak occurred in the Pacific in 2013/2014 and subsequently, the virus spread in other Pacific islands [1]. Zika virus (ZIKV) is transmitted by many Aedes-type mosquitoes including Aedes aegypti [10]. In addition, there are also reports that infections could occur via the placenta, breast milk, saliva, blood transfusion and sex [20]. Pregnant women with the Zika virus are at risk of having a fetus or infant with a congenital defect and suffering from microcephaly and other neurological disorders [16].

Researchers on the dissemination of Zika have been widely practiced. Edy and Gleen have researched about the effect of postponing pregnancy in Zika transmission [4]. Kandasamy, et. al studied the control of dengue and Zika virus vector Aedes-aegypti using the predatory copepod Megacyclops formosanus [11]. F.B. Agosto, S. Bewick, and W.F. Fagan analyze a new system of ordinary differential equations which incorporate human vertical transmission of Zika virus, the birth of babies with microcephaly and asymptotically infected individuals [6]. F.B. Agosto et al also formulate and analyze a new system of ordinary differential equations which incorporate both vector and sexual transmission routes [7]. From the mobility aspect, Nicole et. al investigated factors that may contribute to travel avoidance to areas experiencing ZIKV transmission while also considering different levels of health concern and awareness among groups with varying demographics [17].

In this investigation, we extend some work on deterministic model to closely related stochastic model to account for the variability in the transmission and recovery behavior at the initiation of a Zika disease outbreak. We formulate stochastic models based on the assumptions of the ODE model. The stochastic model applied in this investigation is the continuous-time Markov chains (CTMC). A multitype branching process approximation of the nonlinear CTMC model is used to estimate the probability of disease extinction.

In section 2, we construct the deterministic model of Zika transmission, and in section 3 we formulate the continuous-time Markov chains (CTMC) model for Zika transmission. In section 4, we show the numerical simulation of deterministic and stochastic models.

DETERMINISTIC MODEL

In this section, we construct a deterministic model for Zika disease transmission by developing the SIR-SI dengue model [5]. The human population is divided into five compartments, namely vulnerable humans, infected humans, recovered humans, infected congenital infants, and congenital inborn defects. Meanwhile, the mosquito population is divided into two compartments, vulnerable mosquitoes, and infected mosquitoes. Let $S_h(t)$, $I_h(t)$, and $R_h(t)$ be the number of susceptible, infectious, and recovered individuals of the human at time t , respectively. We also denote the number of infected congenital infants and congenital inborn defects as $I_c(t)$, and $R_c(t)$, respectively. Denote the total population size of human $N_h(t) = S_h(t) + I_h(t) + R_h(t) + I_c(t) + R_c(t)$. Let $S_v(t)$ and $I_v(t)$ be the number of susceptible and infectious mosquitoes at time t , respectively. The total population size of mosquitoes is given by $N_v(t) = S_v(t) + I_v(t)$. The formulation of deterministic model is as follows

$$\begin{aligned}
 \dot{S}_h(t) &= A_h - p\mu_h(I_h + R_h) - \frac{\beta_h S_h I_v}{N_h} - \mu_h S_h + \alpha R_h, \\
 \dot{I}_h(t) &= \frac{\beta_h S_h I_v}{N_h} - \gamma_h I_h - \mu_h I_h, \\
 \dot{R}_h(t) &= \gamma_h I_h - \mu_h R_h - \alpha R_h, \\
 \dot{I}_c(t) &= p\mu_h(I_h + R_h) - \gamma_h I_c(t) - \mu_h I_c, \\
 \dot{R}_c(t) &= \gamma_h I_c - \mu_h R_c, \\
 \dot{S}_v(t) &= A_v - \frac{\beta_v S_v (I_h + I_c)}{N_h} - \mu_v S_v, \\
 \dot{I}_v(t) &= \frac{\beta_v S_v (I_h + I_c)}{N_h} - \mu_v I_v.
 \end{aligned} \tag{1}$$

The parameters A_h and A_v represent recruitment rates of humans and mosquitoes populations, whereas the parameters μ_h and μ_v represent the rates of natural death of humans and mosquitoes, respectively. The parameters γ_h and p are the transition rate related to the infection process (recovery) and the proportion of humans born with a congenital defect. The parameter α represents the period of a recovered human becomes a susceptible human. The parameters β_h and β_v represent the infection rate of humans and mosquitoes, respectively. Humans and mosquitoes populations in this model are kept constant with $N_h = \frac{A_h}{\mu_h}$ and $N_v = \frac{A_v}{\mu_v}$.

TABLE 1. Variables and Parameters related to the dynamical model.

Description	Parameter/Variables	Dimension
Susceptible humans	S_h	humans
Infected humans	I_h	humans
Recovered humans	R_h	humans
Infected congenital humans	I_c	humans
Recovered congenital humans	R_c	humans
Susceptible mosquitoes	S_v	mosquitoes
Infected mosquitoes	I_v	mosquitoes
Human recruitment rate	A_h	humans·day ⁻¹
Proportion of congenital birth	p	dimensionless
Human death rate	μ_h	day ⁻¹
Human transmission rate	β_h	humans·mosquitoes ⁻¹ ·day ⁻¹
Human latent period	γ_h	day ⁻¹
Human recovered period	α	day ⁻¹
Mosquitoes recruitment rate	A_v	mosquitoes·day ⁻¹
Mosquitoes death rate	μ_v	day ⁻¹
Mosquitoes transmission rate	β_v	mosquitoes·humans ⁻¹ ·day ⁻¹

From (1), the disease free endemic point (DFE) is given by

$$(S_h, I_h, R_h, I_c, R_c, S_v, I_v) = \left(\frac{A_h}{\mu_h}, 0, 0, 0, 0, \frac{A_v}{\mu_v}, 0 \right). \quad (2)$$

In (1), there are three infectious compartments, I_h, I_c , and I_v . We linearize the differential equations for these infectious variables at the DFE, the jacobian of (1) is given by

$$J = \begin{bmatrix} -\mu_h - \gamma_h & 0 & \beta_h \\ p\mu_h & -\mu_h - \gamma_h & 0 \\ \hat{\beta}_v & \hat{\beta}_v & -\mu_v \end{bmatrix}, \quad (3)$$

where $\hat{\beta}_v = \beta_v \frac{N_v}{N_h}$. Following [18], where $J = F - V$, we write F and V as

$$F = \begin{bmatrix} 0 & 0 & \beta_h \\ 0 & 0 & 0 \\ \hat{\beta}_v & \hat{\beta}_v & 0 \end{bmatrix} \quad (4)$$

$$V = \begin{bmatrix} -\mu_h - \gamma_h & 0 & 0 \\ p\mu_h & -\mu_h - \gamma_h & 0 \\ 0 & 0 & -\mu_v \end{bmatrix} \quad (5)$$

The next generation matrix of the system is

$$K = F \cdot V^{-1} = \begin{bmatrix} 0 & 0 & -\frac{\beta_h}{\mu_v} \\ 0 & 0 & 0 \\ -\frac{\beta_h \hat{\beta}_v (\gamma_h + \mu_h + p\mu_h)}{\mu_v (\gamma_h + \mu_h)^2} & -\frac{\hat{\beta}_v}{\mu_h + \gamma_h} & 0 \end{bmatrix} \quad (6)$$

The basic reproduction number is the spectral radius of K , we obtain the basic reproduction number in the form

$$R_0 = \sqrt{\frac{\beta_h \hat{\beta}_v (\gamma_h + \mu_h + p\mu_h)}{\mu_v (\gamma_h + \mu_h)^2}}. \quad (7)$$

STOCHASTIC MODEL

CTMC Model

For a small number of infectious individuals, the predictions of the ODE model do not necessarily agree with the MC model. If $R_0 > 1$, there is a possibility in the MC model that infectious individuals die or recover before an outbreak occurs. In this case, an MC model with a discrete number of individuals is more realistic than an ODE model [8]. To formulate a continuous-time Markov chain (CTMC) model, let

$$\mathbf{X}(t) = (S_h(t), I_h(t), R_h(t), I_c(t), R_c(t), S_v(t), I_v(t)) \quad (8)$$

denote a discrete-valued random vector where $\mathbf{X}(t) \in (\mathbb{Z}^+ \cup \{0\})^7$. We use the same notation for the discrete random variables as for the deterministic model (1) for simplicity. The random variables $S_h, I_h, R_h, I_c, R_c, S_v$, and I_v are discrete and the process is time-homogeneous. State transitions and rate for CTMC model given by Table 2. The expression $r\Delta t + o(\Delta t)$ is the infinitesimal transition probability for the change $\Delta \mathbf{X} = \mathbf{X}(t + \Delta t) - \mathbf{X}(t)$. The dynamic of the nonlinear CTMC model can be approximated near the disease-free equilibrium by multitype (Galton-Watson) branching process [3],[9],[19].

TABLE 2. State transitions and rates describing the CTMC model.

Description	Changes	Rate, r
Birth of S_h	$S_h \longrightarrow S_h + 1$	$A_h - p\mu_h(I_h + R_h)$
Birth of I_c	$I_c \longrightarrow I_c + 1$	$p\mu_h(I_h + R_h)$
Death of S_h	$S_h \longrightarrow S_h - 1$	$\mu_h S_h$
Death of I_h	$I_h \longrightarrow I_h - 1$	$\mu_h I_h$
Death of R_h	$R_h \longrightarrow R_h - 1$	$\mu_h R_h$
Death of I_c	$I_c \longrightarrow I_c - 1$	$\mu_h I_c$
Death of R_c	$R_c \longrightarrow R_c - 1$	$\mu_h R_c$
Infection of S_h	$(S_h, I_h) \longrightarrow (S_h - 1, I_h + 1)$	$\frac{\beta_h S_h I_v}{N_h}$
Recovered of I_h	$(I_h, R_h) \longrightarrow (I_h - 1, R_h + 1)$	$\gamma_h I_h$
Recovered of I_c	$(I_c, R_c) \longrightarrow (I_c - 1, R_c + 1)$	$\gamma_h I_c$
Birth of I_c	$I_c \longrightarrow I_c + 1$	A_v
Death of I_v	$I_v \longrightarrow I_v - 1$	$\mu_v I_v$
Death of R_v	$R_v \longrightarrow R_v - 1$	$\mu_v R_v$
Infection of S_c	$(S_c, I_c) \longrightarrow (S_c - 1, I_c + 1)$	$\frac{\beta_h S_v (I_h + I_c)}{N_v}$

Branching Process Aproximation

Based on [14], for the offspring probability generator function (pgf) of $x_i(0) = 1, x_j(0) = 0$, for $i \neq j, f_i : [0, 1]^n \longrightarrow [0, 1]$, is define as

$$f_i(x_1, x_2, \dots, x_n) = \sum_{k_n=0}^{\infty} \dots \sum_{k_1=0}^{\infty} P_i(k_1, k_2, \dots, k_n) x_1^{k_1} x_2^{k_2} \dots x_n^{k_n} \quad (9)$$

where $P_i(k_1, k_2, \dots, k_n)$ is the probability that one individual of type i gives birth to k_j individual of type j . For the branching process approximation this model, assumed that infected humans (I_h) as x_1 , infected congenital humans (I_c) as x_2 , and infected mosquitoes (I_v) as x_3 .

The offspring of pgf for I_h , given $I_h(0) = 1, I_c(0) = 0$, and $I_v(0) = 0$, is

$$f_1(x_1, x_2, x_3) = \frac{\mu_h + \gamma_h + \hat{\beta}_v x_1 x_3 + p\mu_h x_1 x_2}{\mu_h + \gamma_h + \hat{\beta}_v + p\mu_h} \quad (10)$$

The terms $\frac{\mu_h}{\mu_h + \gamma_h + \hat{\beta}_v + p\mu_h}$ and $\frac{\gamma_h}{\mu_h + \gamma_h + \hat{\beta}_v + p\mu_h}$ represent the probability of human dies and recovers before causing a secondary infection, respectively. The term $\frac{\hat{\beta}_v}{\mu_h + \gamma_h + \hat{\beta}_v + p\mu_h}$ represents the probability of a susceptible mosquito becomes infected because of contact with an infected human. The term $\frac{p\mu_h}{\mu_h + \gamma_h + \hat{\beta}_v + p\mu_h}$ represents the probability of birth of a congenital human from an infected human.

The offspring of pgf for I_c , given $I_c(0) = 1, I_h(0) = 0$, and $I_v(0) = 0$, is

$$f_2(x_1, x_2, x_3) = \frac{\mu_h + \gamma_h + \hat{\beta}_v x_2 x_3}{\mu_h + \gamma_h + \hat{\beta}_v} \quad (11)$$

The term $\frac{\mu_h + \gamma_h}{\mu_h + \gamma_h + \hat{\beta}_v}$ represents the probability of an infected congenital human dies or recovers before causing secondary infection. Meanwhile, the term $\frac{\hat{\beta}_v}{\mu_h + \gamma_h + \hat{\beta}_v}$ represents the probability of a susceptible mosquito becomes infected because contact with an infected congenital human.

The offspring of pgf for I_v , given $I_v(0) = 1, I_h(0) = 0$, and $I_c(0) = 0$, is

$$f_3(x_1, x_2, x_3) = \frac{\mu_v + \beta_h x_1 x_3}{\mu_v + \beta_h} \quad (12)$$

The term $\frac{\mu_v}{\mu_v + \beta_h}$ represents the probability of an infected mosquito dies before causing secondary infection. The term $\frac{\beta_h}{\mu_v + \beta_h}$ represents the probability of a susceptible human becomes infected because contact with an infected mosquito.

The expectation matrix $M = [m_{kj}]$ of the offspring pgfs is a nonnegative nm matrix whose entry m_{kj} gives the expected number of infectious offspring in patch k produced by an infectious individual in patch j [8]. That is,

$$m_{kj} = \frac{\partial f_j}{\partial x_k} \Big|_{\mathbf{x}=\mathbf{1}} \quad (13)$$

The expectation matrix for the offspring pgf of Zika cases is

$$M = \begin{bmatrix} \frac{\hat{\beta}_v + p\mu_h}{\hat{\beta}_v + p\mu_h + \mu_h + \gamma_h} & \frac{p\mu_h}{\hat{\beta}_v + p\mu_h + \mu_h + \gamma_h} & \frac{\hat{\beta}_v}{\hat{\beta}_v + p\mu_h + \mu_h + \gamma_h} \\ 0 & \frac{\hat{\beta}_v}{\hat{\beta}_v + \mu_h + \gamma_h} & \frac{\hat{\beta}_v}{\hat{\beta}_v + \mu_h + \gamma_h} \\ \frac{\beta_h}{\beta_h + \mu_v} & 0 & \frac{\beta_h}{\beta_h + \mu_v} \end{bmatrix} \quad (14)$$

The characteristic polynomial for expectation matrix is

$$\begin{aligned} p(\lambda) &= a_3 \lambda^3 + a_2 \lambda^2 + a_1 \lambda + a_0 \\ a_3 &= (\hat{\beta}_v + \gamma_h + \mu_h)(\hat{\beta}_v + p\mu_h + \gamma_h + \mu_h)(\mu_v + \beta_h) \\ a_2 &= -(3\hat{\beta}_v^2 + (3p\mu_h + 4\gamma_h + 4\mu_h)\hat{\beta}_v + (\gamma_h + \mu_h)(2p\mu_h + \gamma_h + \mu_h)\beta_h \\ &\quad 2\hat{\beta}_v^2 \mu_v + 2\mu_v(p\mu_h + \gamma_h + \mu_h)\hat{\beta}_v + p\mu_h \mu_v (\gamma_h + \mu_h)) \\ a_1 &= (2\hat{\beta}_v^2 + (3p\mu_h + \gamma_h + \mu_h)\hat{\beta}_v + p\mu_h(\gamma_h + \mu_h))\beta_h + \hat{\beta}_v^2 * \mu_v + p\hat{\beta}_v \mu_h \mu_v \\ a_0 &= -2p\beta_h \hat{\beta}_v \mu_h \end{aligned} \quad (15)$$

The spectral radius of M cannot be explicitly calculated. For $\lambda = 1$, we get the expression

$$p(1) = -\hat{\beta}_v(p\mu_h + \gamma_h + \mu_h)\beta_h + (\gamma_h + \mu_h)^2 \mu_v. \quad (16)$$

$p(1) < 0$ if $R_0 > 1$, so (15) have a unique $\rho(M)$ at $[0, 1]$.

If $\rho(M) \leq 1 \Leftrightarrow R_0 \leq 1$ (critical and subcritical process), then the probability of extinction of the process is 1 [8]

$$\lim_{t \rightarrow \infty} \text{Prob}\{\mathbf{I}(t) = 0\} = 1. \quad (17)$$

If $\rho(M) > 1 \Leftrightarrow R_0 > 1$ (supercritical case), then the probability of extinction is

$$\mathbb{P}_0 = \lim_{t \rightarrow \infty} \text{Prob}\{\mathbf{I}(t) = 0\} = q_1^{i_1} q_2^{i_2} \dots q_n^{i_n}, \quad (18)$$

where $i_j = I_j(0)$ and q_j is the unique fix point lying in $(0,1)$ of the pgf, $f_j(q_1, q_2, \dots, q_n) = q_j$ for $j \in [1, n]$, [2, 8, 9, 13, 19].

For this Zika case, let $I_h(0) = h_0$, $I_c(0) = c_0$, and $I_v(0) = v_0$, the probability extinction define

$$\mathbb{P}_0 = \lim_{t \rightarrow \infty} \text{Prob}\{I_h(t) + I_c(t) + I_v(t) = 0\} = q_1^{h_0} q_2^{c_0} q_3^{v_0}, \quad (19)$$

for fix point $(q_1, q_2, q_3) \in (0, 1)^3$ of the offspring pgfs can be calculated by

$$\begin{aligned} q_1 &= \frac{\beta_h q_3 - \mu_v(1 - q_3)}{\beta_h q_3} \\ q_2 &= \frac{\gamma_h + \mu_h}{\gamma_h + \mu_h + (1 - q_3)\hat{\beta}_v} \\ r(q_3) &= b_2 q_3^2 + b_1 q_3 + b_0 \\ b_2 &= \hat{\beta}_v^2 (\mu_v + \beta_h) \\ b_1 &= ((\beta_h + 2\mu_v)\hat{\beta}_v + (p\mu_h + \gamma_h + \mu_h)\beta_h + \mu_v(p\mu_h + 2\gamma_h + 2\mu_h))\hat{\beta}_v \\ b_0 &= (\hat{\beta}_v^2 + (p\mu_h + 2\gamma_h + 2\mu_h)\hat{\beta}_v + (\gamma_h + \mu_h)^2)\mu_v \end{aligned} \quad (20)$$

The term q_1 is the probability of disease extinction for infected humans population, the term q_2 is the probability of disease extinction for infected congenital humans population, and the term q_3 is the probability of disease extinction for infected mosquitoes population.

NUMERICAL SIMULATION

For numerical simulation of deterministic and stochastic model, we use parameter value as $N_h(0) = 1000, N_v(0) = 700, A_h = \frac{N_h}{365.70}, A_v = \frac{N_v}{30}, p = 0.5, \beta_h = 0.15, \beta_v = 0.3, \gamma_h = \frac{1}{10}, \mu_h = \frac{1}{365.70}, \mu_v = \frac{1}{30}$. The numerical simulation of deterministic and stochastic models show in figure 1. Using the same parameter value and initial condition $S_h(0) = N_h(0) - I_h(0) - I_c(0)$ and $S_v(0) = N_v(0) - I_v(0)$, we simulate 10,000 sample for getting probability of extinction approximation. The result show in table 3. The level set of R_0 show at figure 2.

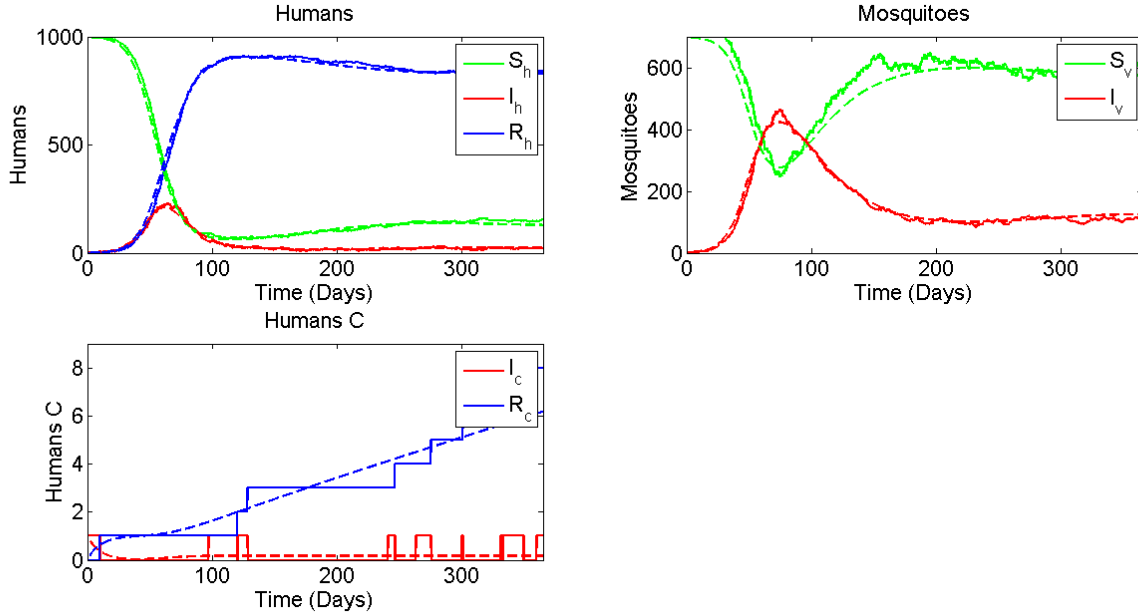


FIGURE 1. Deterministic (dash) and stochastic (line) simulation, $R_0 = 3.0734, P_0 = 0.0417$ with $N_h = 1000, N_v = 700, \mu_h = \frac{1}{365.70}, \mu_v = \frac{1}{30}, \beta_h = 0.15, \beta_v = 0.3, \gamma_h = \frac{1}{10}, p = 0.5$.

TABLE 3. Probability of disease extinction, P_0 , and numerical approximation (Approx.) based on 10,000 sample paths of the CTMC model.

$I_h(0)$	$I_c(0)$	$I_v(0)$	P_0	Approx.
1	0	0	0.3943	0.3869
0	1	0	0.3943	0.3936
0	0	1	0.2684	0.2641
1	1	0	0.1555	0.1584
0	1	1	0.1058	0.1084
1	0	1	0.1058	0.1067
2	0	0	0.1555	0.1537
0	2	0	0.1555	0.1548
0	0	2	0.0720	0.0707
1	1	1	0.0417	0.0397

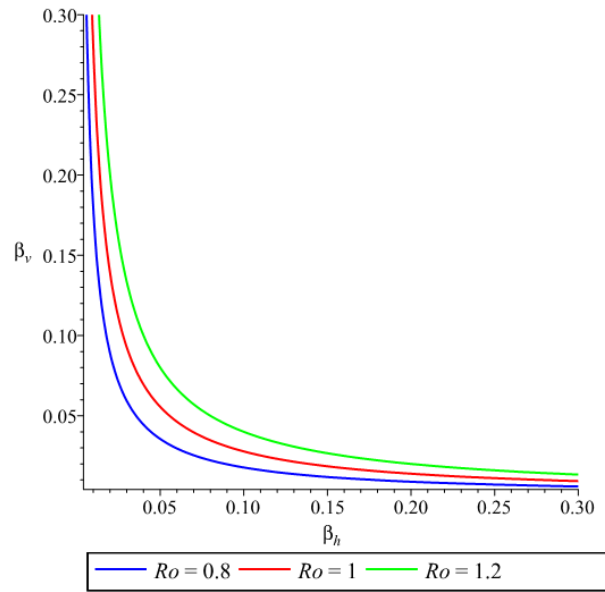


FIGURE 2. Level set of R_0 , with $N_h = 1000, N_v = 700, \mu_h = \frac{1}{365 \cdot 70}, \mu_v = \frac{1}{30}, \gamma_h = \frac{1}{10}, p = 0.5$.

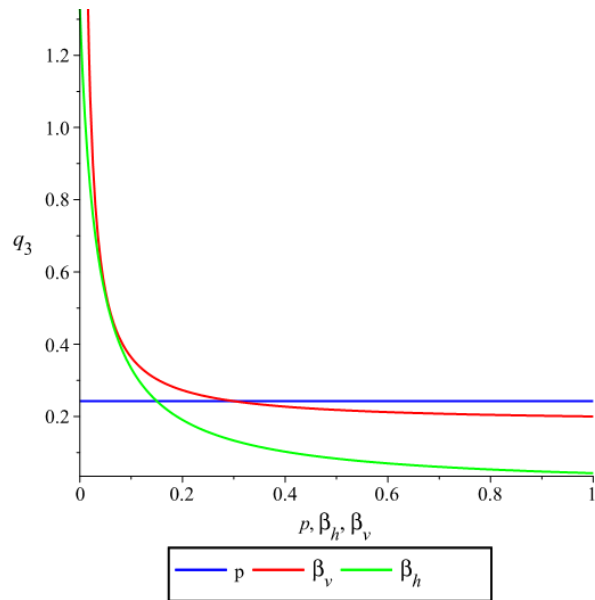


FIGURE 3. Sensitivity of $q_3 : p, \beta_h, \beta_v$

CONCLUSION

The thresholds for deterministic model is $R_0 = \sqrt{\frac{\beta_h \beta_v (\gamma_h + \mu_h + p \mu_h)}{\mu_v (\gamma_h + \mu_h)^2}}$. The threshold for stochastic model is equivalent with R_0 . Threshold theorem in Allen and Driessche (2013) gives relationship between threshold deterministic and stochastic follow by $R_0 > 1 (= 1, < 1) \Leftrightarrow \rho(M) > 1 (= 1, < 1)$ [15]. With multitype branching process approximation of the nonlinear CTMC model near the disease-free equilibrium, probability of disease extinction can be predicted (20). Probability of Zika disease outbreak is

$$1 - q_1^{h_0} q_2^{c_0} q_3^{v_0} \quad (21)$$

where q_1, q_2 , and q_3 show in (20). We demonstrate the usefulness of this probability in epidemic model and indicate a good agreement between the probability of disease extinction \mathbb{P}_0 and numerical estimation based on computational samples. In addition, the new relationship is derived between the deterministic and stochastic thresholds.

From sensitivity of q_3 and p on figure 3, we conclude that the parameter p , the proportion of congenital birth, does not significantly affect the parameter value q_3 , the probability of extinction of infected mosquito. Furthermore, since the probability of extinction of infected humans and the probability of extinction of infected congenital humans, q_1 and q_2 depend on q_3 , we can convey that the proportion of congenital births has no significant effect on the probability of extinction of infected humans and infected congenital humans.

REFERENCES

- [1] Amjad Ali, Braira Wahid, Shazia Raque, and Muhammad Idrees. *Advances in research on Zika virus*. Asian Pacic Journal of Tropical Medicine. 2017. 10(4): 321331
- [2] Athreya, K. B., & Ney, P. E. (1972). *Branching processes*. New York: Springer.
- [3] Dorman, K. S., Sinsheimer, J. S., & Lange, K. *In the garden of branching processes*. *SIAM Rev.*(2004) 46, 202229.
- [4] Edy Soewono and G. Lahodny Jr., *On the Effect of Posptoning Pregnancy in a Zika Transmission Model*. Report Research Project. 2017.
- [5] Esteva L. and Vargas C. *Analysis of a dengue disease transmission model*. *Math Biosci.* 1998. 150(2):131-51.
- [6] F.B. Agosto, S. Bewick, and W.F. Fagan. *Mathematical model of Zika virus with vertical transmission*. *Infectious Disease Modelling*. 2017. 1-24.
- [7] F.B. Agosto, S. Bewick, and W.F. Fagan. *Mathematical model for Zika virus dynamics with sexual transmission route*. *Ecological Complexity* 29. 2017. 61-81.
- [8] Glenn E. Lahodny Jr. and Linda J.S. Allen, *Probability of a Disease Outbreak in Stochastic Multipatch Epidemic Models*. *Bull Math Biol* (2013) 75 : 11571180.
- [9] Harris, T. E. (1963). *The theory of branching processes*. Berlin: Springer.
- [10] Julien Riou, Chiara Poletto, and Pierre-Yves Bolle. *A comparative analysis of Chikungunya and Zika transmission*. *Epidemics* 19. 2017. 4352.
- [11] Kandasamy Kalimuthu, et al. *Control of dengue and Zika virus vector Aedes aegypti using the predatory copepod Megacyclops formosanus: Synergy with Hedychium coronarium-synthesized silver nanoparticles and related histological changes in targeted mosquitoes*. *Process Safety and Environmental Protection* 109. 2017. 8296
- [12] L.I. Zambrano et al., *Estimating and mapping the incidence of dengue and chikungunya in Honduras during 2015 using Geographic Information Systems (GIS)*, *J. Infect. Public Health*, 2016.
- [13] L.J.S. Allen. *An Introduction to Stochastic Processes with Applications to Biology, Second Edition*. Texas : A Chapman & Hall Book. 2010.
- [14] L.J.S. Allen and G. Lahodny Jr., *Extinction thresholds in deterministic and stochastic epidemic models*, *J. Biol. Dyn.* 6 (2012), 590-611.
- [15] L.J.S. Allen and P. van den Driessche. *Relations between deterministic and stochastic thresholds for disease extinction in continuous and discrete-time infectious disease models*, *Math. Biosci.* (2013). 243 pp. 99-108.
- [16] Mary Kay Kindhauser, Tomas Allen, Veronika Frank, Ravi Santhana, and Christopher Dye. *Zika: The Origin and Spread of a mosquito-borne virus*. Bulletin of the World Health Organization. 2016.
- [17] Nicole J. Olynk Widmar, S.R.Dominick, Audrey Ruple, and Wallace E. Tyner. *The inuence of health concern on travel plans with focus on the Zika virus in 2016*. *Preventive Medicine Reports* 6. 2017. 162-170.

- [18] O Diekmann, J.A.P Heesterbeek. *Mathematical Epidemiology of Infectious Diseases*. John Wiley & Son. New York. 2000.
- [19] Pénnison, S. *Conditional limit theorems for multitype branching processes and illustration in epidemiological risk analysis*. Ph.D. diss., Institut fr Mathematik der Universitt Postdam, Germany. 2010.
- [20] Yitades Gebre, Nikkiah Forbes, and Teshome Gebre. *Zika virus infection, transmission, associated neurological disorders and birth abnormalities: A review of progress in research, priorities and knowledge gaps*. [Asian Pac J Trop Biomed](#). 2016. 6(10): 815824