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Numerical Simulations and Solutions of a Mathematical Model for Zika Virus Disease

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Abstract: In this work, a new model for Zika virus disease is formulated. The model incorporates the effects of treatment and use of sterile insect technique in controlling the spread of Zika in human and mosquito populations respectively. The existence and uniqueness of solutions to the model are shown. The model is also shown to be well-posed epidemiologically. The semi-analytical solutions of the model are obtained by differential transform methods and compare with the numerical solutions by Runge-Kutta method of order 4 and is seen to perform relatively well. Numerical simulations show how effective the various controls suggested help to reduce the spread of the disease. The analysis also highlights the health burdens associated with increasing cases of asymptomatic occurrences. The analyses show that combining control strategies produces a better result in controlling the spread of Zika virus than using any of the controls individually.

Keywords: Differential transform method; Modelling; Simulation; Sterile insect technique; Zika virus.

1. INTRODUCTION

Zika virus disease is a flavivirus disease transmitted amongst humans through the bites of infectious female aedes mosquitoes, through blood transfusion, during sex and from mother to child during pregnancy [1]. The first case of Zika virus was discovered in 1947 in Uganda while the first cases in humans were reported in 1954 in Nigeria [2, 3]. The first epidemic case in humans was reported in Yap, Federal States of Micronesia, Pacific region in 2007 with the second epidemic case coming up in 2013 in the same region [4]. The Zika virus infection as a significant vector transmitted disease has in the last few decades caused many cases in Latin America [5]. The European Centre for Disease Prevention and Control said that the first autochthonous cases in the region of the Americas were discovered in Brazil in 2015 and in December same year, the Ministry of Health reported about 440,000 – 1,300,000 estimated cases. Calvet *et al.* [6] said that Zika infection during pregnancy causes congenital abnormalities of the brain which microcephaly is inclusive. Zika virus also triggers the Guillain-Barre syndrome [7]. In humans, the Zika virus incubates within 3 to 14 days before symptoms appear [8]. Common symptoms include headache, maculopapular rash, mild fever, muscle and joint pain, malaise, arthralgia, and conjunctivitis [4, 9, 10]. From available data, adult humans are the most infectious. Zika virus infection, dengue and chikungunya are among the major causes of ill-health in the tropics and subtropics, and it causes significant health, economic and social burdens [11].

The sterile insect technology is one of the methods of biologically controlling insects and pests [12]. It involves releasing an overwhelming number of sterile insects preferably males into the target population to mate with the females [13]. In this method, females are not the preferred choice because they can start laying eggs and thus increase the population which is to be reduced further. In the case of controlling mosquitoes, using females as part of the control will be dangerous as they can feed on human blood and transmit plasmodium. When a female mosquito mates with a sterile male, it produces no offspring as they lay eggs without hatching thereby reducing the population of the next [14, 15]. The sterile insect cannot replicate by themselves, hence cannot become established in the environment. When sterile insects are repeatedly introduced into an environment, the target population is either reduced or eliminated with time.

There have been several works done on the mathematical modelling of the disease such as in [1] where the authors used Wolbachia mosquitoes as control measures for the mosquito population and obtained the approximate solution of their system by homotopy perturbation method. In [5], the authors studied the optimization of control methods against the spread of Zika virus in human and mosquito populations. The two control strategies considered in their work were awareness campaigns and the use of insecticides. They fitted their results with real data obtained from an outbreak in Colombia and explored the various control strategies for optimality. Their work showed that combining both control strategies performed better than adopting only one of the controls. Rezapour *et al.* [16] presented a new fractional-order model for Zika virus infections amongst humans and also between humans and mosquitoes. Approximate analytical solution was calculated using fractional Euler method and numerical experiment was performed to demonstrate the effect of fractional derivative on the system when compared with integer derivative. Agudelo and Ventresca [17] studied a mathematical model for Zika virus disease that addresses sexual transmission as well as transmission through mosquito bites. The model was fitted with data from Colombia between 2015 and 2017. Their work showed that focusing on mitigating the diseases by targeting mosquitoes is the best approach of controlling the disease. Alfwzan *et al.* [18] developed a mathematical model for Zika virus disease which was analyzed both numerically and dynamically. Nonstandard numerical methods are employed to check the stability and consistency of solutions. The numerical schemes preserved the behaviour and properties of the model. Ibrahim and Denes [19] proposed a model for Zika virus disease incorporating mother-to-child and sexual transmission. The model also included a compartment for infants with microcephaly and also showed the impact of asymptomatic humans, seasonality and infection by sexual transmission. They analyzed the model considering constant and variable time. The model was fitted to a data gotten from Colombia which showed the effect of transmission by sexual contact on women with Zika virus disease and infants with microcephaly. Their work showed that the best way to control the spread of Zika virus disease is to reduce the population of mosquitoes, offer adequate protection during sex and eliminating biting of mosquitoes.

Wang *et al.* [20] developed a new model for Zika virus disease transmission which incorporates transmission by sexual contacts, mosquito bites and through sewage. Their model was fitted with data from Brazil between 2015-2016. In their reproduction number, mosquito bites accounted for large effect of endemicity, followed by transmission through sex and then sewage. They also discovered that temperature and multiple transmission increased the prevalence of the disease in Brazil. In [21], the authors presented a deterministic model for Zika virus disease that incorporated vertical and direct transmissions. Their work investigated the dynamics of the disease transmission in the presence of mosquitoes, infectious pregnant women and infected children, Numerical simulations showed that human contact rates with mosquitoes affects the endemicity of the disease more than other forms of transmissions. Kouidere *et al.* [22] used fractional derivatives to model an optimal control problem of Zika virus disease. The controls employed were sensitization programs against the disease and treatment. Through numerical simulations, the proposed controls were seen to be effective. Helikumi *et al*. [23] also carried out an optimal control analysis of Zika virus disease with health education campaigns, use of insecticides and preventive measures as the controls. They discovered that fractional-order model fitted better than the classical integer-order model. The simulation results were seen to justify the use of the controls suggested.

In this work however, we considered a model with only mosquitoes as the transmission agent and proposed using sterile insect technology to control the vectors. Most of the literatures reviewed in this work stated that Zika virus endemicity is associated mostly with vector transmission, hence the focus of this work on transmission by mosquitoes only. It is also known that most Zika-infected humans are asymptomatic, hence we equally showed the effect of such an occurrence on the health burden of the disease which most literatures did not consider which is one of the motivations of this work. Asymptomatic occurrence ensures that people will be infected without knowing and can easily infect others hence diseases with high asymptomatic cases like Zika virus disease are better prevented than managed. This research was also motivated by the work in [1] where Wolbachia mosquitoes were used as control measures. Wolbachia is one of the biological means of controlling insects and pests just like sterile insect technique. Presently, Sterile insect technique (SIT) is targeted at eradicating Anopheles mosquitoes causing malaria, aedes aegypti and aedes albopictus mosquitoes causing dengue, yellow fever, filariasis, chikungunya and Zika virus diseases [24, 25]. The major aim of this work is to highlight the effect of using sterile insect technique as a control measure for mosquitoes while also incorporating the occurrence of asymptomatic cases which is a health concern. The rest of the work is arranged as follows: The model is presented in Section 2, and the basic model analysis is shown. In Section 3, the numerical solution was obtained while in Section 4, numerical simulations were carried out. The results were discussed in Section 5 and the work was concluded in Section 6.

2. ANALYSIS OF THE MATHEMATICAL MODEL

2.1 Model Development

Two populations were considered for this model, the human population and the aedes mosquito population with the sterile-insect mosquito population introduced to control the multiplication of the aedes mosquitoes. The total population of human and vector were divided into nine compartments; Susceptible humans S_h, Exposed humans E_{hz}, Symptomatic infectious humans I_{hzS}, Asymptomatic infectious humans I_{hzA}, Infectious humans undergoing treatment I_{hzT}, Recovered humans R_h, Susceptible mosquitoes S_{zv}, Exposed mosquitoes E_{zv} and Infectious mosquitoes I_{zv}. The mosquitoes responsible for causing Zika virus disease in humans are assumed to be introduced into the environment through births and migration. These mosquitoes bite and feed on human blood. During the blood meal, these mosquitoes can inject the Zika virus into the humans thereby infecting them with the virus. We considered the introduction of sterile male mosquitoes which will mate with female aedes mosquitoes. We assumed that as more sterile male mosquitoes interact with the wild females in the target environment, the population of the female aedes mosquitoes will reduce with time and possibly eliminated from the environment.

Table 1. Countries and territories with current or previous Zika virus transmission by WHO as in 2022.

WHO Regional Office	Country / territory	Total
African Regional Office (AFRO)	Angola; Burkina Faso; Burundi; Cabo Verde; Cameroon; Central African Republic; Côte d'Ivoire; Ethiopia; Gabon; Guinea-Bissau; Kenya; Nigeria; Senegal; Uganda	14
American Regional Office (AMRO)/Pan American Regional Office (PAHO)	Anguilla; Antigua and Barbuda; Argentina; Aruba; Bahamas; Barbados; Belize; Bolivia (Plurinational State of); Bonaire, Sint Eustatius and Saba; Brazil; British Virgin Islands; Cayman Islands; Colombia; Costa Rica; Cuba; Curaçao; Dominica; Dominican Republic; Ecuador; El Salvador; French Guiana; Grenada; Guadeloupe; Guatemala; Guyana; Haiti; Honduras; Easter Island– Chile; Jamaica; Martinique; Mexico; Montserrat; Nicaragua; Panama; Paraguay; Peru; Puerto Rico; Saint Barthelemy; Saint Kitts and Nevis; Saint Lucia; Saint Martin; Saint Vincent and the Grenadines; Saint Maarten; Suriname; Trinidad and Tobago; Turks and Caicos; United States of America; United States Virgin Islands; Venezuela (Bolivarian Republic of)	49
SEARO	Bangladesh; India; Indonesia; Maldives; Myanmar; Thailand	6
Western Pacific Regional Office (WPRO)	American Samoa; Cambodia; Cook Islands; Fiji; French Polynesia; Lao People's Democratic Republic; Marshall Islands; Malaysia; Micronesia (Federated States of); New Caledonia; Palau; Papua New Guinea; Philippines; Samoa; Singapore; Solomon Islands; Tonga; Vanuatu; Viet Nam	19
EURO	France (Var department)	1
Total		89

WHO – World Health Organization, SEARO – Southeast Asian Regional Office, EURO – European Regional Office.

Table 2. Countries and territories with established aedes aegypti mosquito vectors, but no known cases of Zika virus transmission, by WHO regional office as in 2022.

WHO Regional Office	Country / territory	Total
AFRO	Benin; Botswana; Chad; Comoros; Congo; Democratic Republic of the Congo; Equatorial Guinea; Eritrea; Gambia; Ghana; Guinea; Liberia; Madagascar; Malawi; Mali; Mauritius; Mayotte; Mozambique; Namibia; Niger; Réunion; Rwanda; Sao Tome and Principe; Seychelles; Sierra Leone; South Africa; South Sudan; Togo; United Republic of Tanzania; Zambia; Zimbabwe	31
AMRO/PAHO	Uruguay	1
EMRO	Afghanistan; Djibouti; Egypt; Oman; Pakistan; Saudi Arabia; Somalia; Sudan; Yemen	9
SEARO	Bhutan; Nepal; Sri Lanka; Timor-Leste	4
WPRO	Australia; Brunei Darussalam; China; Christmas Island; Guam; Kiribati; Nauru; Niue; Northern Mariana Islands (Commonwealth of the); Tokelau; Tuvalu; Wallis and Futuna	12
EURO	Georgia; Região Autónoma da Madeira – Portugal; Russian Federation; Turkey	4
TOTAL		61

EMRO - Eastern Mediterranean Regional Office.

2.2 Basic Assumptions for the Zika Virus Disease Model

In order to set up our human and vector compartments to derive the model of the system, we made the following assumptions:

(a) The infectious human population was partitioned into two: those who are symptomatic and those who are asymptomatic. Most humans with Zika virus disease are usually asymptomatic than symptomatic [17, 26]. The effects of this asymptomatic occurrence are among the issues this study is meant to explore as most previous works on the disease have not highlighted it much.

(b) In the symptomatic human population, some of them accepted to go for treatment and some refused to go for treatment [26]. It is well-known that not all sick persons seek medical attention, those who do are moved to the class receiving treatment.

(c) Some of those who accepted to go for treatment recovered from the disease, some died naturally while others died due to the disease. Treatment does not guarantee recovery depending on the promptness and efficiency of the treatment measures as well as severity of the infection.

(d) It is assumed that those who accepted to go for treatment are also infectious with the disease as they have not fully recovered. Infectious humans undergoing treatment can also infect mosquitoes though at a rate minimal to those yet to begin treatment. Hence, this class contributes to the force of infection.

(e) The recovered humans are not re-introduced into the susceptible population since recovering from Zika virus disease grants permanent immunity to the disease. This is because those who recover from Zika virus disease become immunized against the disease [9, 10].

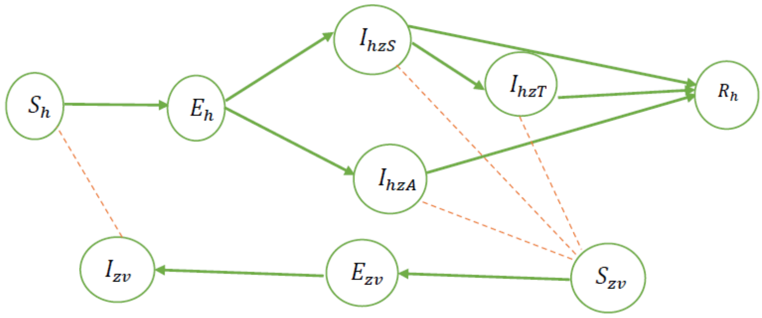


Figure 1. Flow diagram for Zika virus disease.

The susceptible human population, S_h is increased by level of recruitment through births and migration, and reduced by the rate of movement into the exposed class as well as natural death rate. The Exposed human population, E_{hz} is reduced by the rate of development of infectiousness and natural death rate. The Symptomatic human population, I_{hzS} is increased as more infectious humans become symptomatic and reduced by death rate due to the disease, proportion that are treated and proportion not undergoing treatment but recovers naturally with time. The Asymptomatic human population, I_{hzA} is increased as more infectious humans become asymptomatic and reduced by the proportion that recovers naturally with time, as well as natural and disease-induced death rates. The human population undergoing treatment, I_{hzT} is reduced by the proportion that recovers with time, and the different death rates. The susceptible mosquito population, S_{zv} is increased by the level of recruitment denoted and reduced by natural death rate, rate of reduction in mosquito population due to SIT as well as rate of exposure to malaria through contacts with infectious humans. The susceptible compartment is set up to investigate the number of SIT mosquitoes that should be interacting with a given number of female aedes mosquitoes at any given time to ensure proper control of the population of the female aedes mosquitoes. The exposed mosquito population is reduced by natural death rate, and rate of development of infectiousness.

The model is given by:

$$\begin{aligned}
\frac{dS_h}{dt} &= \Lambda_h - \alpha\eta_1 I_{zv} S_h - \tau_1 S_h \\
\frac{dE_{hz}}{dt} &= \alpha\eta_1 I_{zv} S_h - (\delta + \tau_1) E_{hz} \\
\frac{dI_{hzS}}{dt} &= \chi_1 \delta I_{hz} - (\tau_1 + \tau_2 + \psi + \omega_1) I_{hzS} \\
\frac{dI_{hzT}}{dt} &= \psi I_{hzS} - (\tau_1 + \tau_2 + \omega_2) I_{hzT} \\
\frac{dR_h}{dt} &= \omega_1 I_{hzS} + \omega_2 I_{hzT} + \omega_3 I_{hzA} - \tau_1 R_h \\
\frac{dS_{zv}}{dt} &= \Lambda_{zv} - \alpha(\eta_2 I_{hzS} + \eta_3 I_{hzT} + \eta_4 I_{hzA}) S_{zv} - (\kappa Z_{SIT} + \mu) S_{zv} \\
\frac{dE_{zv}}{dt} &= \alpha(\eta_2 I_{hzS} + \eta_3 I_{hzT} + \eta_4 I_{hzA}) S_{zv} - (\nu + \mu) E_{zv} \\
\frac{dI_{zv}}{dt} &= \nu E_{zv} - \mu I_{zv}
\end{aligned} \tag{1}$$

where $S_h(0) = S_h^0, E_{hz}(0) = E_{hz}^0, I_{hzS}(0) = I_{hzS}^0, I_{hzA}(0) = I_{hzA}^0, I_{hzT}(0) = I_{hzT}^0, R_h(0) = R_h^0, S_{zv}(0) = S_{zv}^0, E_{zv}(0) = E_{zv}^0$ *and* $I_{zv}(0) = I_{zv}^0$ are the initial conditions of the system. Tables 3 and 4 describe the state variables as well as the parameters used in the numerical simulations with their respective values and source.

Table 3. Description of variables used in the model for Zika only.

Variables	Description
S_h	Susceptible human population
E_{hz}	Exposed humans to Zika virus disease
I_{hzS}	Symptomatic infectious humans with Zika virus disease
I_{hzA}	Asymptomatic infectious humans with Zika virus disease
I_{hzT}	Infectious humans with Zika virus disease undergoing treatment
R_h	Recovered humans
S_{zv}	Susceptible aedes aegypti mosquito population
E_{zv}	Exposed aedes aegypti mosquito
I_{zv}	Infectious aedes aegypti mosquito population

Table 4. Description of parameters used in the Zika model.

Parameters	Description	Values	Sources
Λ_h	Recruitment level of humans into susceptible population	50	[26]
Λ_{zv}	Recruitment level of aedes aegypti mosquitoes	100	[26]
τ_1	Natural death rate of humans	0.00004	[2]
τ_2	Death rate due to Zika virus disease	0.0003	[26]
μ	Natural death rate of mosquitoes	0.0556	[2]
α	Biting rate of mosquitoes with Zika virus	0.4	[26]
η_1	Effective rate of transmission of Zika virus from mosquitoes to humans	0.0009	[2]
η_2	Effective rate of transmission of Zika virus from symptomatic infectious humans to mosquitoes	0.07	[2]
η_3	Effective rate of transmission of Zika virus from asymptomatic infectious humans to mosquitoes	0.07	[2]
η_4	Effective rate of transmission of Zika virus from symptomatic infectious humans undergoing treatment to mosquitoes	0.05	[26]
δ_2	Rate at which exposed human with Zika virus become infectious	0.3333	[1]
ν	Rate of development of exposed aedes aegypti mosquitoes to infectious aedes aegypti mosquitoes	0.1111	[1]
χ_1	Proportion of infectious humans who are symptomatic	0.31	[26]
χ_2	Proportion of infectious humans who are asymptomatic	0.62	[26]
ω_1	Rate of recovery of symptomatic infectious humans	0.1429	[27]
ω_2	Rate of recovery of symptomatic infectious humans undergoing treatment	0.1667	[2]
ω_3	Rate at which asymptomatic infectious humans with Zika virus disease recover naturally	0.118	[1]
κ	Mating rate of SIT mosquitoes with aedes aegypti mosquitoes	0.5	Assumed
ψ	Rate at which symptomatic infectious humans accept treatment	0.85	[26]
Z_{SIT}	Population of Sterile males to control aedes aegypti population	300	Assumed

2.3 Positivity of Solutions

Lemma 1: Let the initial data set for the model be $\{S_h^0 > 0, E_{hz}^0(0) > 0, I_{hzS}^0 > 0, I_{hzA}^0 > 0, I_{hzT}^0 > 0, R_h^0 > 0, S_{zv}^0 > 0, E_{zv}^0 > 0, I_{zv}^0 > 0\}$ at $t = 0$. Then, the solution set $\{S_h(t), E_{hz}(t), I_{hzS}(t), I_{hzA}(t), I_{hzT}(t), R_h(t), S_{zv}(t), E_{zv}(t), I_{zv}(t)\}$ of the model with the given initial data will remain positive for all time $t > 0$.

Proof: Given the model of the system in Equation (1), we have that:

$$\frac{dS_h}{dt} \geq -(\alpha\eta_1 I_{zv} + \tau_1)S_h$$

$$\frac{dE_{hz}}{dt} \geq -((\chi_1 + \chi_2)\delta + \tau_1)E_{hz}$$

$$\vdots$$

$$\frac{dI_{zv}}{dt} \geq -\mu I_{zv}$$

Hence, solving the differential inequality with the given initial solution gives,

$$S_h \geq S_h^0 e^{-\int (\alpha\eta_1 I_{zv} + \tau_1)dt} > 0$$

$$E_{hz} \geq E_{hz}^0 e^{-\int ((\chi_1+\chi_2)\delta+\tau_1)dt} > 0$$

$$\vdots$$

$$I_{zv} \geq I_{zv}^0 e^{-\int \mu dt} > 0$$

Thus, all the solutions have been shown to be nonnegative at any $t > 0$ and the proof is complete.

2.3 Boundedness of Solutions

Lemma 2: The region, Ω is positively invariant and is an attractor of all positive solution of the system, where $\Omega = \Omega_1 \times \Omega_2$, $\Omega_1 = \left\{(S_h,\ E_{hz},\ I_{hzA},\ I_{hzS},\ I_{hzT},\ R_h): S_h + E_{hz} + I_{hzA} + I_{hzS} + I_{hzT} + R_h \leq \frac{\Lambda_h}{\tau_1}\right\} \in R^6$ and $\Omega_2 = \left\{(S_{zv},\ E_{zv},\ I_{zv}): S_{zv} + E_{zv} + I_{zv} \leq \frac{\Lambda_{zv}}{\mu}\right\} \in R^3$.

Proof: The total human population of the system denoted by N_h is given by

$$N_h = S_h + E_{hz} + I_{hzA} + I_{hzS} + I_{hzT} + R_h \tag{2}$$

where

$$\frac{dN_h}{dt} = \frac{dS_h}{dt} + \frac{dE_{hz}}{dt} + \frac{dI_{hzA}}{dt} + \frac{dI_{hzS}}{dt} + \frac{dI_{hzT}}{dt} + \frac{dR_h}{dt} \tag{3}$$

Thus,

$$\frac{dN_h}{dt} = \Lambda_h - \tau_1(N_h) - \tau_3(I_{hzA} + I_{hzS} + I_{hzT}) \leq \Lambda_h - \tau_1(N_h)$$

When solved using method of integrating factor for solving first-order ODEs, we have

$$N_h \leq \frac{\Lambda_h}{\tau_1} + ce^{-\tau_1 t}$$

As $t \to \infty$, we will have $0 < N_h \leq \frac{\Lambda_h}{\tau_1}$. This shows that the solution $N_h \to \frac{\Lambda_h}{\tau_1}$ as $t \to \infty$ and the set Ω_1 is positively invariant. Also, for the aedes mosquito population, we will have that

$$N_{zv} \leq \frac{\Lambda_{zv}}{\mu} + ce^{-\mu t}$$

Thus, as $t \to \infty$, we will have $0 < N_{zv} \leq \frac{\Lambda_{zv}}{\mu}$. This shows that the solution $N_{zv} \to \frac{\Lambda_{zv}}{\mu}$ as $t \to \infty$ and the set Ω_2 is positively invariant. But $N = N_{hz} + N_{zv}$, therefore, N which is the total population is also bounded. The Proofs in Lemmas 1 and 2 ensure that the system is mathematically and epidemiologically well-posed in the region Ω, hence can be studied and analyzed [26].

2.4 Existence and Uniqueness of solutions to the system

Theorem 1: *(Banach Fixed Point Theorem):* Let Ω be a complete metric space and let $f: \Omega \to \Omega$ be a contraction, that is, $\exists$ $c \in (0,1)$ such that for all $z_1, z_2 \in \Omega$, then

$$d\big(f(z_1), f(z_2)\big) \leq cd(z_1, z_2).$$

Then, f has a unique fixed point in Ω. That is, there exists a unique $z^* \in \Omega$ such that

$$f(z^*) = z^*.$$

Lemma 4.3 (*Lipschitz Continuity*): A function $f(t,z)$ is said to be Lipschitz continuous in z if there exist a constant $K \geq 0$ such that

$$|f(t,z_1) - f(t,z_2)| \leq K|z_1 - z_2|.$$

Here, K is called a Lipschitz constant and f is said to be K-Lipschitz $\forall\, z_1, z_2 \in \Omega$. In summary, to establish the existence and uniqueness of solutions to the system requires showing that the state variables satisfy Lipschitz condition and that the Lipschitz constant, say $K \in (0,1)$.

The Zika-only model can be expressed as

$$Y'(t) = f(Y(t)), \qquad Y(t_0) = Y_0$$

where $Y(t) = (S_h, E_{hz}, I_{hzS}, I_{hzA}, I_{hzT}, R_h, S_{zv}, E_{zv}, I_{zv})$ and

$$\mathrm{f(Y(t))} = \begin{bmatrix} \Lambda_{hz} - (\alpha\eta_1 I_{zv} + \tau_1)S_h \\ \alpha\eta_1 I_{zv} S_h - \big((\chi_1 + \chi_2)\delta + \tau_1\big)E_{hz} \\ \chi_1\delta E_{hz} - (\tau_1 + \tau_2 + \psi + \omega_1)I_{hzS} \\ \chi_2\delta E_{hz} - (\tau_1 + \tau_3 + \omega_3)I_{hzA} \\ \psi I_{hzS} - (\tau_1 + \tau_2 + \omega_2)I_{hzT} \\ \omega_1 I_{hzS} + \omega_3 I_{hzA} + \omega_2 I_{hzT} - \tau_1 R_h \\ \Lambda_{zv} - \alpha(\eta_2 I_{hzS} + \eta_3 I_{hzA} + \eta_4 I_{hzT})S_{zv} - (\kappa\, Z_{SIT} + \mu)S_{zv} \\ \alpha(\eta_2 I_{hzS} + \eta_3 I_{hzA} + \eta_4 I_{hzT})S_{zv} - (\nu + \mu)E_{zv} \\ \nu E_{zv} - \mu I_{zv} \end{bmatrix}$$

Let $||.||$ be the maximum norm in Ω taking to be the Banach domain for continuous functions where $\left||Y(t)|\right| = \sum \left||Y|\right|_\infty$. Let $\left||S_h|\right| \leq k_1, \left||E_{hz}|\right| \leq k_2, \left||I_{hzS}|\right| \leq k_3, \left||I_{hzA}|\right| \leq k_4, \left||I_{hzT}|\right| \leq k_5, \left||R_h|\right| \leq k_6, \left||S_{zv}|\right| \leq k_7, \left||E_{zv}|\right| \leq k_8,$ $\left||I_{zv}|\right| \leq k_9, \left||Z_{SIT}|\right| \leq k_{10}$ and $0 \leq w_{i=1,\ldots,9} < 1$.

For any S_{h1} and $S_{h2} \in \Omega$, then

$$\left||f(t,S_{h1}) - f(t,S_{h2})|\right| = \left||(\Lambda_{hz} - (\alpha\eta_1 I_{zv} + \tau_1)S_{h1}) - (\Lambda_{hz} - (\alpha\eta_1 I_{zv} + \tau_1)S_{h2})|\right|$$
$$= \left||(\alpha\eta_1 I_{zv} + \tau_1)(S_{h1} - S_{h2})|\right| \leq (\alpha\eta_1 k_9 + \tau_1)\left||S_{h1} - S_{h2}|\right| \leq w_1\left||S_{h1} - S_{h2}|\right|$$

The Lipschitz continuity in S_h is established with w_1 as the Lipschitz constant. Similarly, we can establish the Lipschitz continuity in other state variables as follows:

$$\left||f(t,E_{hz1}) - f(t,E_{hz2})|\right| \leq w_2\left||E_{hz1} - E_{hz2}|\right|$$
$$\left||f(t,I_{hzS1}) - f(t,I_{hzS2})|\right| \leq w_3\left||I_{hzS1} - I_{hzS2}|\right|$$
$$\left||f(t,I_{hzA1}) - f(t,I_{hzA1})|\right| \leq w_4\left||I_{hzA1} - I_{hzA2}|\right|$$
$$\left||f(t,I_{hzT1}) - f(t,I_{hzT2})|\right| \leq w_5\left||I_{hzT1} - I_{hzT2}|\right|$$
$$\left||f(t,R_{h1}) - f(t,R_{h2})|\right| \leq w_6\left||R_{h1} - R_{h2}|\right|$$
$$\left||f(t,S_{zv1}) - f(t,S_{zv2})|\right| \leq w_7||S_{zv1} - S_{zv2}||$$
$$\left||f(t,E_{zv1}) - f(t,E_{zv2})|\right| \leq w\_8m||E_{zv1} - E_{zv2}||$$
$$\left||f(t,I_{zv1}) - f(t,I_{zv2})|\right| \leq w_9||I_{zv1} - I_{zv2}||$$

where $w_1 = \alpha\eta_1 k_9 + \tau_1,\ w_2 = (\chi_1 + \chi_2)\delta + \tau_1,\ w_3 = \tau_1 + \tau_2 + \psi + \omega_1, w_4 = \tau_1 + \tau_2 + \omega_3, w_5 = \tau_1 + \tau_2 + \omega_2, w_6 = \tau_1, w_7 = \alpha(\eta_2 k_3 + \eta_3 k_4 + \eta_4 k_5) + (\kappa k_{10} + \mu), w_8 = \nu + \mu, w_9 = \mu$. The terms appearing in the $w_i's$ represents fractional outflow from each compartment whose sum must be less than one. It can be shown that the infectious classes, $(E_{hz}, I_{hzS}, I_{hzA}, I_{hzT}, E_{zv}, I_{zv})$ tend to zero as $t \to \infty$ by solving for them from the system (1). Hence, $k_3, k_4, k_5, k_9, k_{10}$ all tend to zero guaranteeing that all the Lipschitz constants, $w_i's \in (0,1)$ and by Banach fixed point theorem, the solutions to the system exist and is unique.

2.5 Control Reproduction Number

The Zika-free equilibrium (ZFE) is the steady state of the system, (1) where there is no Zika virus disease in the system [27]. To obtain the ZFE for the system, each equation in the system is set to zero, the disease class, $Y = (E_{hz}, I_{hzS}, I_{hzA}, I_{hzT}, E_{zv}, I_{zv})$ is equated to zero and the values of other state variables are obtained from the resulting equations. Thus, the ZFE of the system will be

$$E_0 = \left(\frac{\Lambda_h}{\tau_1}, 0,0,0,0,0, \frac{\Lambda_{zv}}{\mu}, 0,0\right)$$

Using the ZFE, the Zika control number is obtained by employing the Next-Generation Matrix (NGM) approach [28]. The control reproduction number is a threshold parameter used to measure the rate of endemicity of the disease. It is defined as the average number of secondary infections that can be caused by one infectious index case introduced into an entirely susceptible population [29]. It is a threshold parameter such that when it is less than one, the disease will be expected to die out and when it is greater than one, the disease will persist in the system [30]. For our Zika virus disease model, the infectious class consists of the following equations:

$$\frac{dE_{hz}}{dt} = \alpha\eta_1 I_{zv} S_h - \big((\chi_1 + \chi_2)\delta + \tau_1\big)E_{hz}$$
$$\frac{dI_{hzS}}{dt} = \chi_1 \delta E_{hz} - (\tau_1 + \tau_2 + \psi + \omega_1)I_{hzS}$$
$$\frac{dI_{hzT}}{dt} = \psi I_{hzS} - (\tau_1 + \tau_2 + \omega_2)I_{hzT}$$
$$\frac{dE_{zv}}{dt} = \alpha(\eta_2 I_{hzS} + \eta_3 I_{hzT} + \eta_4 I_{hzA})S_{zv} - (\nu + \mu)E_{zv}$$
$$\frac{dI_{zv}}{dt} = \nu E_{zv} - \mu I_{zv}$$

From the NGM method, the rate of appearance of new infection in the system is denoted by $\mathcal{F}$ while the rate of movement into and out of the system through any other means is denoted by $\mathcal{V}$. Both are obtained from the system in Equation (1) to be:

$$\mathcal{F} = \begin{bmatrix} \alpha\eta_1 I_{zv} S_h \\ 0 \\ 0 \\ 0 \\ \alpha(\eta_2 I_{hzS} + \eta_3 I_{hzA} + \eta_4 I_{hzT})S_{zv} \\ 0 \end{bmatrix} \quad \text{and } \mathcal{V} = \begin{bmatrix} \big((\chi_1 + \chi_2)\delta + \tau_1\big)E_{hz} \\ -\chi_1\delta E_{hz} + (\tau_1 + \tau_2 + \psi + \omega_1)I_{hzS} \\ -\chi_2\delta E_{hz} + (\tau_1 + \tau_2 + \omega_3)I_{hzA} \\ -\psi I_{hzS} + (\tau_1 + \tau_2 + \omega_2)I_{hzT} \\ (\nu + \mu)E_{zv} \\ -\nu E_{zv} + \mu I_{zv} \end{bmatrix}.$$

Thus, $$F = \frac{\partial \mathcal{F}}{\partial Y} = \begin{bmatrix} 0 & 0 & 0 & 0 & 0 & C_1 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & C_2 & C_3 & C_4 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix} \quad \text{and} \quad V = \frac{\partial \mathcal{V}}{\partial Y} = \begin{bmatrix} D_1 & 0 & 0 & 0 & 0 & 0 \\ -\chi_1\delta & D_2 & 0 & 0 & 0 & 0 \\ -\chi_2\delta & 0 & D_3 & 0 & 0 & 0 \\ 0 & -\psi & 0 & D_4 & 0 & 0 \\ 0 & 0 & 0 & 0 & D_5 & 0 \\ 0 & 0 & 0 & 0 & -\nu & \mu \end{bmatrix}$$

where $C_1 = \frac{\alpha\eta_1\Lambda_h}{\tau_1},\ C_2 = \frac{\alpha\eta_2\Lambda_{zv}}{\mu},\ C_3 = \frac{\alpha\eta_3\Lambda_{zv}}{\mu}, C_4 = \frac{\alpha\eta_4\Lambda_{zv}}{\mu},\ D_1 = (\chi_1 + \chi_2)\delta + \tau_1,\ D_2 = \tau_1 + \tau_2 + \psi + \omega_1,\ D_3 = \tau_1 + \tau_2 + \omega_3,\ D_4 = \tau_1 + \tau_2 + \omega_2,\ D_5 = \nu + \mu$. Finding the inverse of the matrix, V gives

$$V^{-1} = \begin{bmatrix} \frac{1}{D_1} & 0 & 0 & 0 & 0 & 0 \\ \frac{\chi_1\delta}{D_1 D_2} & \frac{1}{D_2} & 0 & 0 & 0 & 0 \\ \frac{\chi_2\delta}{D_1 D_3} & 0 & \frac{1}{D_3} & 0 & 0 & 0 \\ \frac{\psi\chi_1\delta}{D_1 D_2 D_4} & \frac{\psi}{D_2 D_4} & 0 & \frac{1}{D_4} & 0 & 0 \\ 0 & 0 & 0 & 0 & \frac{1}{D_5} & 0 \\ 0 & 0 & 0 & 0 & \frac{\nu}{D_5\mu} & \frac{1}{\mu} \end{bmatrix}$$

Then, the product of F and V^{-1} given by FV^{-1} becomes

$$FV^{-1} = \begin{bmatrix} 0 & 0 & 0 & 0 & \frac{C_1\nu}{D_5\mu} & \frac{C_1}{\mu} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ \frac{C_2\chi_1\delta}{D_2D_1} + \frac{C_3\chi_2\delta}{D_1D_3} + \frac{C_4\psi\chi_1\delta}{D_2D_1D_4} & \frac{C_2}{D_2} + \frac{C_4\psi}{D_2D_4} & \frac{C_3}{D_3} & \frac{C_4}{D_4} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

and the eigenvalues of the matrix, FV^{-1} will be

$$\rho = \begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \\ \sqrt{\frac{\delta(C_2\chi_1D_3D_4 + C_3\chi_2D_2D_4 + C_4\chi_1\psi D_3)C_1\nu}{D_1D_2D_3D_4D_5\mu}} \\ -\sqrt{\frac{\delta(C_2\chi_1D_3D_4 + C_3\chi_2D_2D_4 + C_4\chi_1\psi D_3)C_1\nu}{D_1D_2D_3D_4D_5\mu}} \end{bmatrix}$$

with the dominant eigenvalue of FV^{-1} denoted by $\rho(FV^{-1})$ taken to be

$$\rho(FV^{-1}) = \sqrt{\frac{\delta(C_2\chi_1D_3D_4 + C_3\chi_2D_2D_4 + C_4\chi_1\psi D_3)C_1\nu}{D_1D_2D_3D_4D_5\mu}}$$

gives us the value of our control reproduction number for the Zika virus disease model as

$$R_{0z} = \left(\frac{C_1\nu\delta(C_2\chi_1D_3D_4 + C_3\chi_2D_2D_4 + C_4\chi_1\psi D_3)}{D_1D_2D_3D_4D_5\mu}\right)^{\frac{1}{2}}$$

The first term in the summation is the total expected population of humans that will be infected at the ZFE by one newly symptomatic infectious human before entering treatment, the second term is the total expected number of humans that will be infected at the ZFE by one newly asymptomatic infectious human while the third term is the total expected number of humans that will be infected at the ZFE by one newly infectious human whilst they are undergoing treatment for Zika.

3. NUMERICAL SOLUTIONS BY DIFFERENTIAL TRANSFORM METHOD

Differential transformation method (DTM) is one of the approximate solution methods used to solve both linear and non-linear equations. The technique was first introduced by Zhou [31, 32] in 1986 for solving linear and nonlinear initial value problems in electrical circuit analysis before being extended to other works like [33] which used it to solve linear and nonlinear ODEs. In [33], it was demonstrated that using DTM does not require linearization, perturbation or discretization which showed a major advantage of the method over others. It was also shown that the method can reduce the number of computational works required and still accurately provide the series solution with fast convergence rate [33].

The differential transform of a function $f(x)$ is defined as follows by [34]

$$F(k) = \frac{1}{k!}\left[\frac{d^kf(x)}{dx^k}\right]_{x=0} \tag{4}$$

where $f(x)$ is the original function and $F(k)$ is the transformed function. The differential inverse transform of $F(k)$ is given by

$$y(x) = \sum_{k=0}^{\infty} F(k)x^k \tag{5}$$

Substituting Equations (4) into (5) gives

$$f(x) = \sum_{k=0}^{\infty} \frac{x^k}{k!}\left[\frac{d^kf(x)}{dx^k}\right]_{x=0} \tag{6}$$

Equation (6) shows that differential transform derived its concept from Taylor series expansion. The standard operations by differential transform method is given in [33-35]. From the standard operations of DTM, we will have for the system in Equation (1)

$$S_h(K+1) = \frac{1}{(K+1)}\left[\Lambda_h - \alpha\eta_1 \sum_{m=0}^{k} S_h(m)\, I_{zv}(\mathrm{k}-\mathrm{m}) - \tau_1 S_h\right]$$
$$E_{hz}(k+1) = \frac{1}{(K+1)}\left[\alpha\eta_1 \sum_{m=0}^{k} S_h(m)\, I_{zv}(\mathrm{k}-\mathrm{m}) + (\delta+\tau_1)E_{hz}(k)\right]$$
$$I_{hzS}(k+1) = \frac{1}{(K+1)}[\chi_1\delta I_{hz}(\mathrm{k}) - (\tau_1+\tau_2+\psi+\omega_1)I_{hzS}(k)]$$
$$I_{hzA}(k+1) = \frac{1}{(K+1)}[\chi_2\delta I_{hz}(\mathrm{k}) - (\tau_1+\tau_2+\omega_3)I_{hzA}(k)]$$
$$I_{hzT}(k+1) = \frac{1}{(K+1)}[\psi I_{hzS}(\mathrm{k}) - (\tau_1+\tau_2+\omega_2)I_{hzT}(k)]$$
$$R_h(k+1) = \frac{1}{(K+1)}[\omega_1 I_{hzS}(k) + \omega_2 I_{hzT}(k) + \omega_3 I_{hzA}(\mathrm{k}) - \tau_1 R_h(k)]$$
$$S_{zv}(k+1) = \frac{1}{(K+1)}\left[\Lambda_{zv} - \left[\alpha\left[\eta_2 \sum_{m=0}^{k} I_{hzS}(\mathrm{k}-\mathrm{m}) + \eta_3 \sum_{m=0}^{k} I_{hzA}(\mathrm{k}-\mathrm{m}) + \eta_4 \sum_{m=0}^{k} I_{hzT}(\mathrm{k}-\mathrm{m})\right] - (\kappa Z_{SIT}+\mu)\right] S_{zv}(k)\right]$$
$$E_{zv}(k+1) = \frac{1}{(K+1)}\left[\alpha\left[\eta_2 \sum_{m=0}^{k} I_{hzS}(\mathrm{k}-\mathrm{m}) + \eta_3 \sum_{m=0}^{k} I_{hzA}(\mathrm{k}-\mathrm{m}) + \eta_4 \sum_{m=0}^{k} I_{hzT}(\mathrm{k}-\mathrm{m})\right] S_{zv}(k) - (\nu+\mu)E_{zv}(k)\right]$$
$$I_{zv}(k+1) = \frac{1}{(K+1)}[\nu E_{zv}(k) - \mu I_{zv}(k)]$$

Using these assumed initial values for the state variables; $S_h = 500, E_{hz} = 110, I_{hzS} = 20, I_{hzA} = 40, I_{hzT} = 15, R_h = 10, S_{zv} = 1000, E_{zv} = 95, I_{zv} = 70, Z_{SIT} = 300$ and the parameter values from Table 4. Thus, the series solution for the Zika-only model by DTM with three-term approximation becomes

$$\begin{gathered} S_h(t) = 500 + 37.38t + 23.9286t^2 + 13.1997t^3 \\ E_{hz}(t) = 110 - 21.5001t + 4.4033t^2 + 2.9783t^3 \\ I_{hzS}(t) = 20 - 8.4993t + 3.1102t^2 - 0.8781t^3 \\ I_{hzA}(t) = 40 + 17.9975t - 3.2864t^2 + 0.4329t^3 \\ I_{hzT}(t) = 15 + 14.4964t - 4.8230t^2 + 1.1498t^3 \\ R_h(t) = 10 + 11.2956t + 1.7479t^2 - 0.2491t^3 \\ S_{zv}(t) = 500 - 75{,}890.02t + 5{,}766{,}795.18t^2 - 292{,}131{,}843.4t^3 \\ E_{zv}(t) = 95 + 974.1635t - 75{,}073.3563t^2 + 3{,}796{,}178.593t^3 \\ I_{zv}(t) = 70 + 6.6625t + 53.9296t^2 - 2{,}781.2161t^3 \end{gathered} \tag{7}$$

The solutions obtained by the DTM is simulated against the solutions by Runge-Kutta method of order 4 obtained with MATLAB 2015b and the results are shown in Figures 2-10. The plots showed that both solutions are similar and relatively comparable within some interval of t. The DTM is an approximation method and in this case was truncated after four terms. A further study will require obtaining higher terms of the DTM and comparing what happens with time, t.

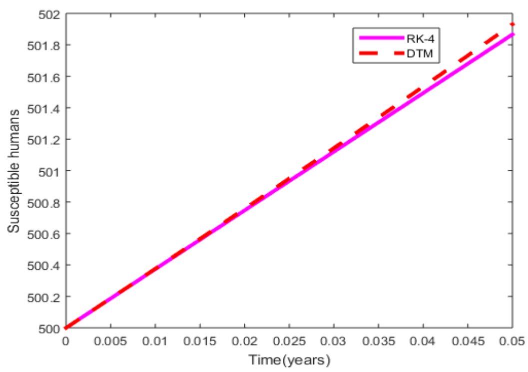


Figure 2. Susceptible humans.

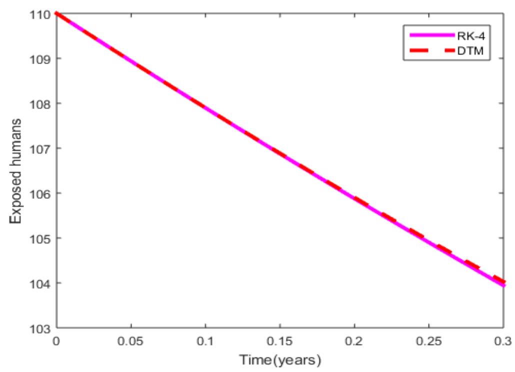


Figure 3. Exposed humans.

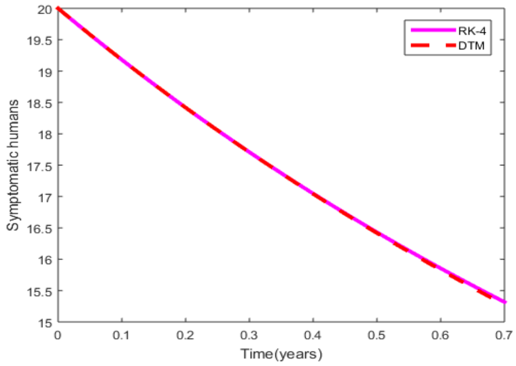


Figure 4. Symptomatic humans.

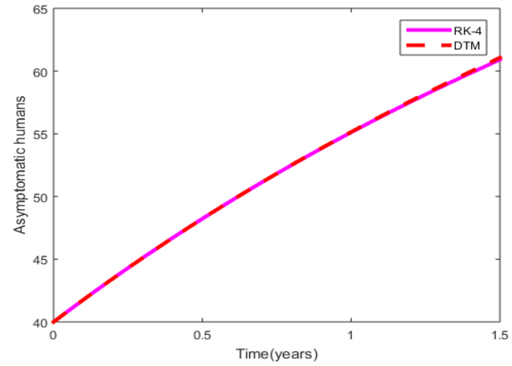


Figure 5. Asymptomatic humans.

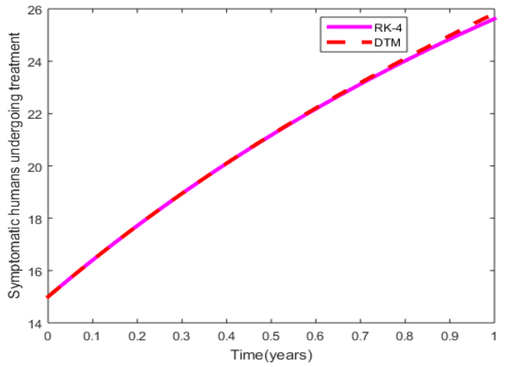


Figure 6. Treated humans.

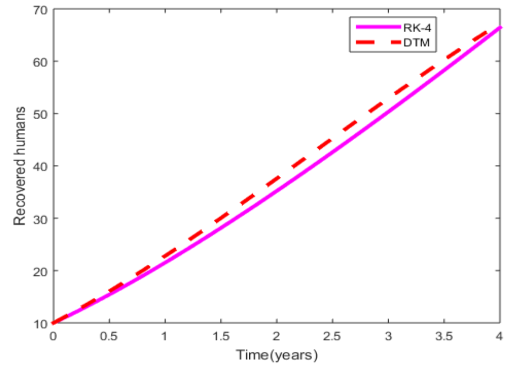


Figure 7. Recovered humans.

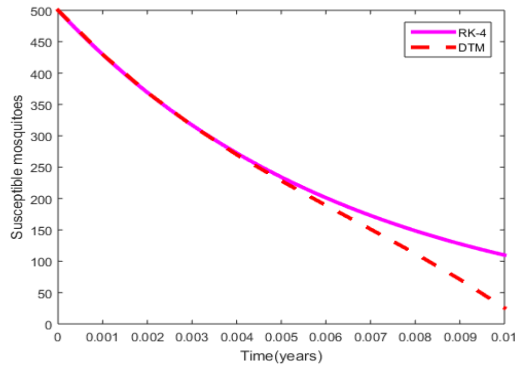


Figure 8. Susceptible mosquitoes.

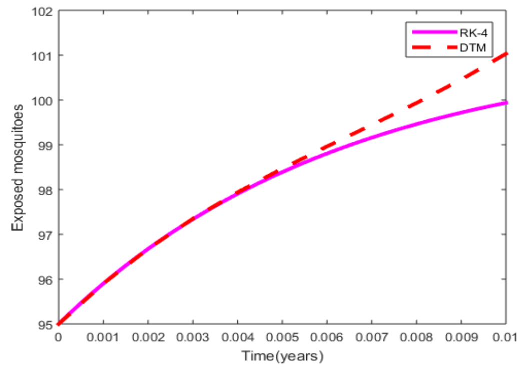


Figure 9. Exposed mosquitoes.

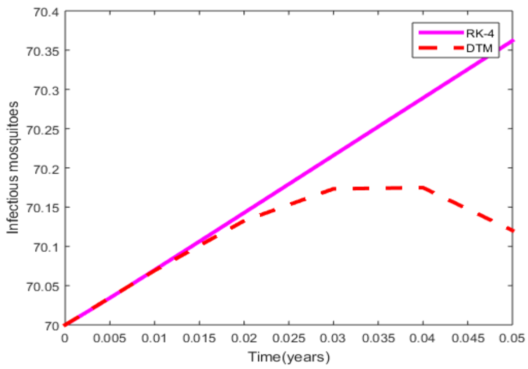


Figure 10. Infectious mosquitoes.

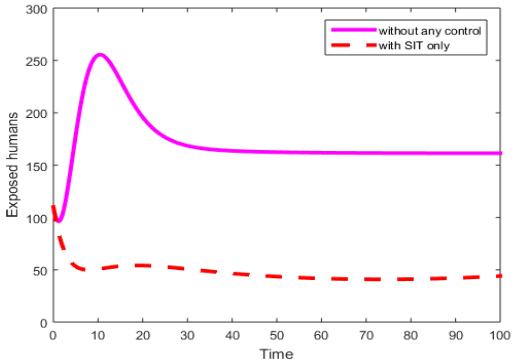


Figure 11. Exposed humans under SIT.

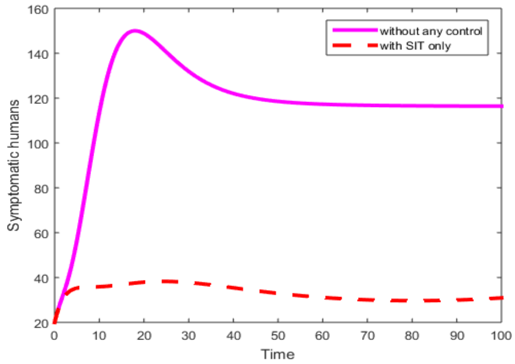


Figure 12. Symptomatic humans under SIT.

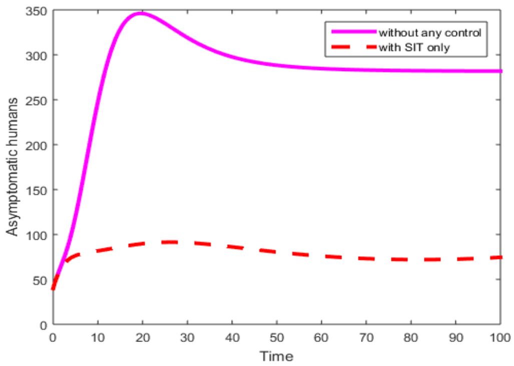


Figure 13. Asymptomatic humans under SIT.

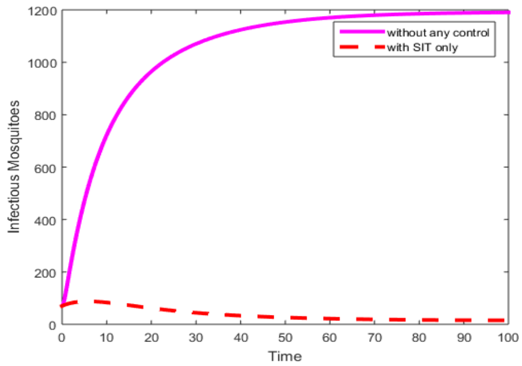


Figure 14. Infectious mosquitoes under SIT.

4. NUMERICAL SIMULATION

In this section, numerical simulation with MATLAB 2015b on the system is performed to highlight the effects of the controls incorporated in the model. The numerical solution was done using the same parameter values from Table 4 and initial values of the state variables in section 2.

4.1 Effects of SIT Only

The effects of the interaction between the sterile mosquitoes and the females in the wild are shown in Figures 11-14. The results of the simulation show that as sterile mosquitoes mate with the female mosquitoes in the wild, the population of the infectious class in both the human and mosquito populations reduced greatly with time. This occurrence is associated with the fact that when the females in the wild mates with the sterile males and become pregnant, the lay eggs without hatching them. This causes an overall depletion of the number of mosquitoes with time as further procreation has been drastically reduced.

4.2 Effects of Treatment Only

The effects of employing only treatment as a control measure is shown in Figures 15-18. The simulations showed that while treatment reduced the population of symptomatic humans significantly, it did not have much effect on the other infectious compartments. The symptomatic humans reduced as expected since they are the only population undergoing treatment. However, the occurrence of asymptomatic cases who are not treated ensure that Zika virus disease will continue to spread in the system explaining why the other infectious compartment did not reduce significantly. This observation underlines the need for preventive measures to be adopted in the control of the disease since treatment offers little help in this regard.

4.3 Effects of Treatment and SIT

Here, the effect of combining treatment and SIT as control measures are simulated and shown in Figures 19-22. This control measure performed better than using either treatment only or sterile insect technique only to control the spread of the disease. Both the infectious human and infectious mosquito populations reduced more significantly in this scenario than when any of the controls are employed independently. This shows that it is preferable to employ measures that affect both humans and mosquitoes in the control of Zika virus disease than using a measure that affects only either of them.

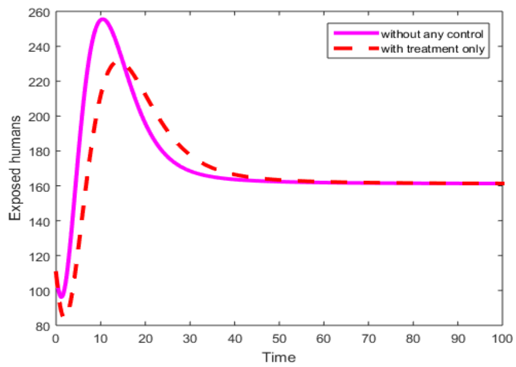


Figure 15. Exposed humans under treatment.

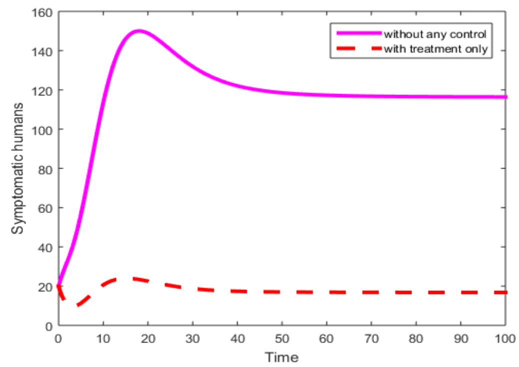


Figure 16. Symptomatic humans under treatment.

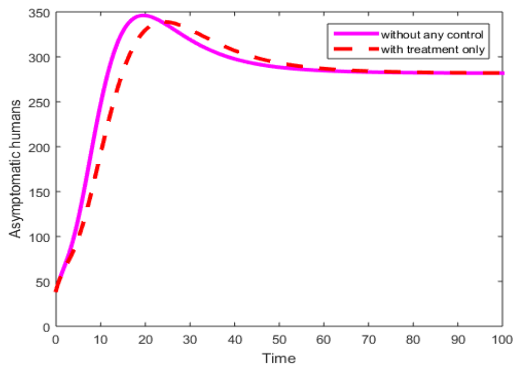


Figure 17. Asymptomatic humans under treatment.

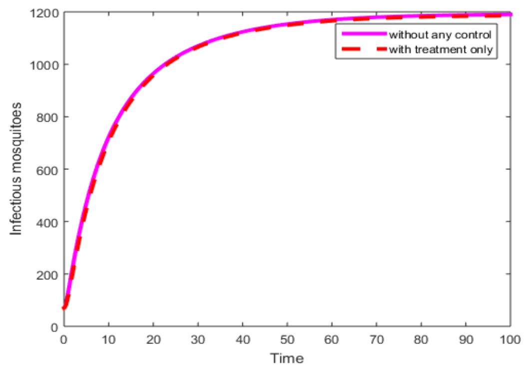


Figure 18. Infectious mosquitoes under treatment.

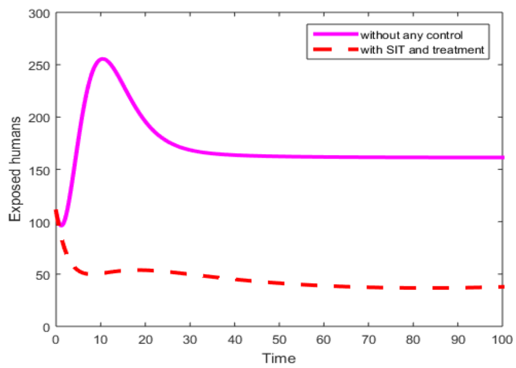


Figure 19. Exposed humans under both controls.

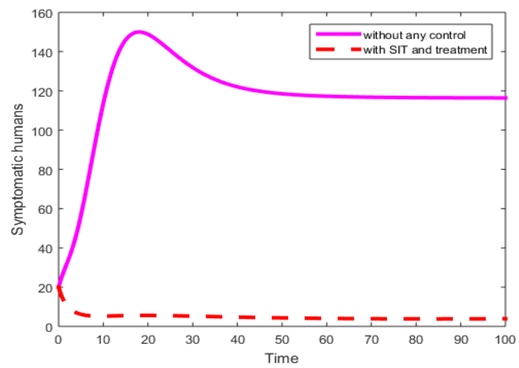


Figure 20. Symptomatic humans under both controls.

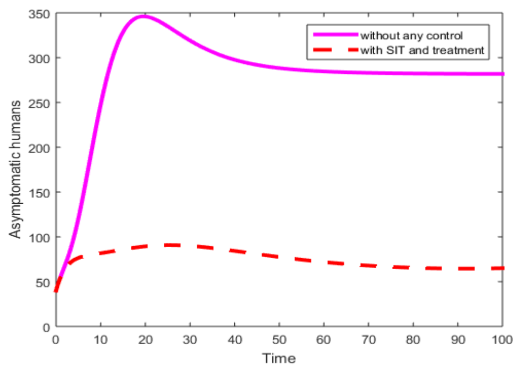


Figure 21. Asymptomatic humans under both controls.

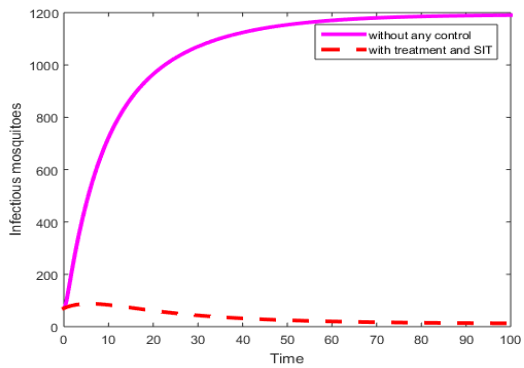
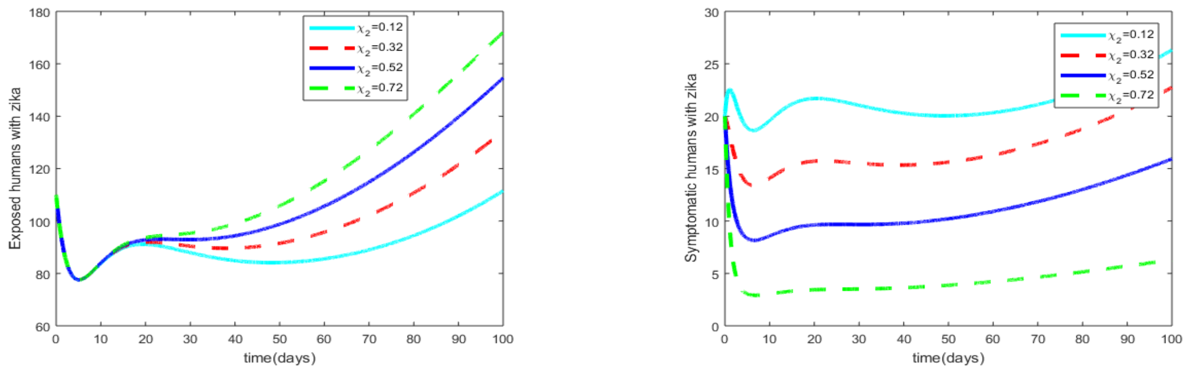
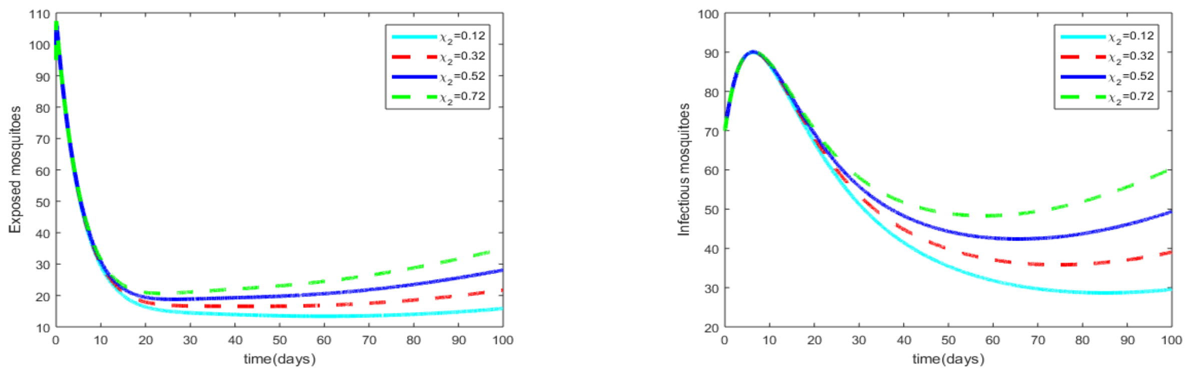


Figure 22. Infectious mosquitoes under both controls.



(a) (b)

Figure 23. Effect of asymptomatic occurrence on: (a) Exposed humans, (b) Symptomatic humans.



(a) (b)

Figure 24. Effect of asymptomatic occurrence on: (a) Exposed mosquitoes, (b) Infectious mosquitoes.

4.4 Effects of Asymptomatic Occurrence

The effect of the occurrence of asymptomatic cases was also investigated using the values in Table 4. The results are shown in Figures 23 and 24. The results showed that an increase in the occurrence of asymptomatic cases will lead to an increase in the number of exposed humans and mosquitoes to Zika virus disease. This occurs because asymptomatic humans are not treated and can transfer viruses to mosquitoes unhindered. The more mosquitoes are infected, the higher the probability of infecting more humans. Consequently, the number of infectious humans and mosquitoes also increased due to the increase in the occurrence of asymptomatic cases. The occurrence of asymptomatic cases cannot be stopped or controlled as long as Zika virus is in circulation. Hence, the best option is to apply measures that will eradicate Zika virus disease from the system.

5 DISCUSSION

In the numerical simulations carried out in this work in section 4, the effects of the various controls proposed were shown. Firstly, the effect of sterile males interacting with the females in the wild was shown. The plots showed that SIT is a good technique in controlling the spread of malaria. All the infectious populations were greatly reduced under this measure. As the sterile male's mate with the females in the environment, the females become pregnant, lay eggs but are not able to hatch them thus reducing the mosquito population with time. Also, the effect of treatment of infectious humans was also shown to have minimal effect in reducing the spread of the disease. Adopting only the treatment of infectious humans with Zika virus disease as a control measure will not produce much result as there are cases of asymptomatic humans who are not known and treated yet infectiously. Thereafter, both treatment and use of sterile insect technique were combined as a control measure and was seen to produce a better result than when each of the controls were used separately. The occurrence of asymptomatic cases shows that any effort at successfully controlling the disease must incorporate preventive measures such as vaccination, protection against mosquito bites, mosquito repletion methods like SIT, Wolbachia, etc. However, we have only considered SIT and treatment in this work as there are no known vaccines yet to prevent Zika virus disease and other control measures have been widely explored by other authors.

6 CONCLUSION

In this work, we presented a new mathematical model for Zika virus disease which incorporated treatment and using sterile insect technique to control the vectors. The model was shown to be mathematically well-posed by establishing that all solutions to the model system are positive and will remain positively bounded as time, t tends to infinity. Thereafter, the ZFE of the system was obtained and used to obtain the zika control number, a threshold for measuring the endemicity of Zika virus disease in the system. The numerical solutions of the system were obtained by differential transform method while the numerical simulations were also shown. The results of the simulations showed that combining treatment and use of sterile insect technique as control measures performed better than when any of the strategies was employed independently. The effects of increasing occurrence of asymptomatic cases were also shown. It was shown that the more cases of asymptomatic occurrences are recorded, the more the disease persists and remains endemic. This result highlights the need for more efforts in preventing the occurrence of the disease than in managing their presence. In conclusion, Zika virus disease will be better controlled in any environment where it is existing if the control measures simultaneously focus on both humans and the mosquito populations. Also, since there are no known drugs for effective treatment of the disease coupled with the danger associated with increasing cases of asymptomatic occurrence, efforts should also be made in preventing the spread of the disease than in treatment. In further studies, optimal control analysis and bifurcation analysis can be carried out to gain more insight into the disease dynamics. Also, other forms of force of infection and different control measures can be incorporated, analyzed and compared with this study.

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DECLARATION OF CONFLICTING INTERESTS

The authors declare no potential conflicts of interest with respect to the research and publication of this article.

REFERENCES

[1] M. C. Anyanwu, G. C. E. Mbah and E. C. Duru, On mathematical model for Zika virus disease control with Wolbachia-infected mosquitoes. *Abacus,* 47, 2020, 35-54.
[2] J. Lamwong and P. Pongsumpun. Age structural model of Zika virus, *International Journal of Modelling and Optimization*, 8, 2018, 17-23.
[3] R. Rakkiyappan, V. P. Latha and F. A. Rihan. A fractional-order model for Zika virus infection with multiple delays, *Complexity*, 20, 2019, 1-21.
[4] F. Y. Eguda, J. Andrawus, I. M. Stephen and A. Sale. Modeling the spread of Zika virus with vaccination and vector reduction as control strategies, *Dutse Journal of Pure and Applied Sciences,* 5, 2019, 273-284.
[5] G. Gonzalez-Parra and T. Benincasa. Mathematical Modeling and numerical simulations of Zika in Colombia considering mutation, *Journal of Mathematical Computation and Simulation*, 163, 2019, 1-18.
[6] G. Calvet, R. S. Aguiar, A. S. O. Melo et al. Detection and sequencing of Zika virus from amniotic fluid of fetuses with microcephaly in Brazil: A case study, *The Lancet: Infectious Diseases*, 16(6), 2016, 653-660.
[7] F. Krauer, M. Riesen, L, Reveiz, et al., Zika virus infection as a cause of congenital brain abnormalities and Guillain–Barré's syndrome: Systematic review, *PLOS Medicine*, 14, 2016, 1-27.
[8] D. Gao, Y. Lou, D. He, T. C. Porco, Y. Kuang, G. Chowell and S. Ruan. Prevention and control of Zika as a mosquito-borne and sexually transmitted disease: A mathematical modeling analysis, *Scientific Reports*, 6, 2016, 1-17.
[9] World Health Organization, The History of Zika virus, https://www.who.int/news-room/feature-stories/detail/the-history-of-Zika-virus, 2016 (accessed 17.11.2022).
[10] CDC, Zika virus disease in the United States. Centre for Disease Control and Prevention, https://www.cdc.gov/zika/geo/united-states.html, 2016 (accessed 30.11.2022).
[11] S.C. Weaver and M. Lecuit, Chikungunya virus and the global spread of a mosquito-borne disease, *New England Journal of Medicine*, 372, 2015, 1231-1239.
[12] M. J. B. Vreysen, A. S. Robinson and J. Hendrichs. *Area-wide Control of Insect Pests, from Research to Field Implementation*, Springer, Dordrecht, Netherlands, 2007.
[13] V. A. Dyck, J. Hendrichs and A. S. Robbinson. *Sterile Insect Technique: Principles and Practice in Area-wide Integrated Pest Management.* CRC Press, Boca Raton, USA, 2021.
[14] R. Anguelov, Y. Dumont and J. Lubuma, Mathematical modeling of sterile insect Technology for control of Anopheles mosquito, *Computers and Mathematics with Applications*, 64, 2012, 3.
[15] W, Atokolo and G. C. E. Mbah. Modeling the control of Zika virus vector population using the sterile insect technology, *Journal of Applied Mathematics,* 20, 2020, 1-12.
[16] S. Rezapour, H, Mohammadi and A. Jajarmi, A new mathematical model for Zika virus transmission, *Advances in Difference Equations,* 7, 2020, 589-603.
[17] S. Agudelo and M. Ventresca, Modeling the spread of the Zika virus by sexual and mosquito transmission, *PLoS One*, 17, 2022, e0270127.
[18] W. F. Alfwzan, A. Raza, J. Martin-Vaquero, D. Baleanu, M. Rafiq, N. Ahmed and Z. Iqbal, Modeling and transmission dynamics of Zika virus through efficient numerical method, *AIP Advances,* 13, 2023, 1-12.
[19] M. A. Ibahim and A. Denes. A mathematical model for Zika virus infection and microcephaly risk considering sexual and vertical transmission, *Axioms*, 12, 2023, 263-288.
[20] L. Wang, Q. Jia, G. Zhu, G., Ou and T. Tang. Transmission dynamics of Zika virus with multiple infection routes and a case study in Brazil, *Scientific Reports*, 14, 2024, 1-15.

[21] A. K. Dotia, M. O. Ibrahim, A. Gambo, A. B. Musa, and A. O. Lawani, Deterministic model of Zika-virus with carrier mother and reservoirs: A mathematical analysis approach, *Federal University Dutsin-Ma Journal of Sciences*, 8, 2024, 316-331.

[22] Kouidere, A. El Bhih, I. Minifi, O. Balatif and K. Adnaoui, Optimal control problem for mathematical modeling of Zika virus transmission using fractional order derivatives, *Frontiers in Applied Mathematics and Statistics*, 2024, 1-9.

[23] M. Helikumi, P. O. Lolika, K. A. Makau, M. C. Ndambuki and A. Mhlanga, Modeling Zika virus disease dynamics with control strategies, *Informatics*, 85, 2024, 1-28.

[24] W. Klassen, Introduction: Development of the sterile insect technique for African malaria vectors, *Malaria Journal,* 8, 2009, 11-14.

[25] L. H. Chen and D. H. Hamer., Zika virus: Rapid spread in the Western Hemisphere, *Annals of Internal Medicine,* 164, 2016, 613-615.

[26] E. C. Duru, G. C. E. Mbah, M. C. Anyanwu and T. N. Nnamani, Modelling the co-infection of malaria and Zika virus disease, *Journal of Nigerian Society for Physical Sciences*, 6, 2024, 1-20.

[27] P. Andayani, L. R. Sari, A. Suryanto and I. Darti. Numerical study for Zika virus transmission with Beddington-Deangelis incidence rate, *Far East Journal of Mathematical Sciences,* 111, 2019, 145-157.

[28] E. D. Gurmu, B. K. Bole and P. R. Koya. Mathematical model for transmission dynamics of HIV/ADS and HSV-II coinfection, *Applications of Modelling and Simulation*, 4, 2020, 217-236.

[29] E. D. Gurmu, B. K. Bole and P. R. Koya, Optimal control strategy on the transmission dynamics of Herpes Simplex Virus-II (HSV-II), *Applications of Modelling and Simulation*, 5, 2021, 22-34.

[30] P. Van Den Driessche and J. Watmough, Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission, *Mathematical Biosciences,* 180, 2002, 29-48.

[31] J. M. W. Munganga, J. N. Mwambakana, R. Maritz, T. A. Batubenge and G. M. Moremedi. Introduction of the differential transform method to solve differential equations at undergraduate level, *International Journal of Mathematical Education in Science and Technology,* 45, 2014, 781-794.

[32] A. Khatib, *Differential Transform Method for Differential Equations*, M.Sc. Thesis, Department of Mathematics at Palestine Polytechnic University, 2016.

[33] F. S. Akinboro, S. Alao and F. O. Akinpelu. Numerical solution of SIR model using differential transformation method and variational iteration method, *General Mathematics Notes*, 22, 2014, 82-92.

[34] F. Astuti, A. Suryanto and I. Darti, Multi-step differential transform method for solving the influenza virus model with disease resistance, *IOP Conference Series: Materials Science and Engineering,* 546, 2019, 1-11.

[35] R. Singh and A. Kumar, A comparative study of homotopy perturbation method with differential transformation method to solve a reaction diffusion equation, *Indian Journal of Science and Technology,* 10, 2017, 1-9.