The Impact of Infective Immigrants on the Spread and Dynamics of Zika Virus

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Abstract: In this paper, a compartment model has been built, presented and investigated the dynamics and spread of zika virus in both human and mosquito populations. It is focused to study the impact of symptomatic and asymptomatic infective immigrants on the spread of zika virus. A new mathematical model for human and model for vector population has been designed and presented. Here $I_1$ is symptomatic infective and $I_2$ is asymptomatic infective human populations. The present model is developed making some reasonable modifications in the corresponding epidemic SIR model by considering symptomatic and asymptomatic infective immigrants. Susceptible vectors get infection either from symptomatic or asymptomatic infected human populations. The basic reproduction number is derived using the next generation matrix method. Disease free equilibrium point is found and endemic equilibrium state is identified. It is shown that the disease free equilibrium point is locally and globally asymptotically stable if the reproduction number takes a value less than one unit and unstable if it is more than one unit. Simulation study is conducted using MATLAB ode45.

Keywords: $SI_1I_2R$ Model, Symptomatic Infected, Asymptomatic Infected, Zika Virus, Microcephally

1. Introduction

Zika is a viral infection that is usually spread in human population by the bite of an infected mosquito. Zika was first discovered in 1947 in Uganda [12]. During the period 1960 – 80, human infections, typically accompanied by mild illness, were found across Africa and Asia. The first large outbreak of disease causing Zika infection occurred in the Island of Yap, Federated States of Micronesia in 2007, indicating that the virus had moved from South – East Asia across the Pacific. During 2013 - 14 outbreaks in French Polynesia the neurological disorder of Guillain-Barré syndrome or GBS was found to have links to Zika infection. In South America, initially it was reported that the locally transmitted infection came from Brazil in 2015. In 2015 Brazil reported an association between Zika virus infection and GBS disease.

From 1 April 2015, to 31 March 2016 a total of 164,374 confirmed and suspected cases of Zika virus disease and 1474 cases of the GBS were reported in Bahia, Brazil, Colombia, the Dominican Republic, El Salvador, Honduras, Suriname, and Venezuela [16]. More cases of suspected Microcephally temporally linked with Zika outbreak [14]. In February 2016, as infection moved rapidly through the range occupied by Aedes mosquitoes in the Americas, WHO declared that Zika infection associated with Microcephally and other neurological disorders constitutes a Public Health Emergency of International Concern (PHEIC).

By the start of 2016, local transmission of Zika infection had been reported from more than 20 countries and territories in the Americas, and an outbreak numbering thousands of cases was under way in Cabo Verde, western Africa. Approximately 500 million people in Latin America and the Caribbean are at risk for Zika virus infection [16].

Aedes aegypti mosquitoes live in tropical, subtropical, and in some temperate climates. These mosquitoes live near and prefer to feed on people. They are considered highly efficient at spreading these diseases. There is no evidence that Zika spreads through touching, coughing, or sneezing. There is no vaccine or specific treatment for Zika virus infection. The most common
symptoms of Zika are fever, rash, joint pain, conjunctivitis or red eyes, muscle pain, and headache. Patients zika virus infected are advised to drink plenty of fluids to replenish fluid lost from sweating, vomiting and other insensible losses [13].

Many communities along with national agencies initiated the process to implement control measures that ranged from vector control and the use of repellents to the suggestion of avoiding pregnancies for two years [3]. In addition to Microcephaly the other problems found in pregnancies, among fetuses and infants infected with ZIka virus before birth such as: miscarriage, stillbirth, absent or poorly developed brain structures, defects of the eyes, hearing deficits, and impaired growths [3].

Due to association between Zika virus infection and Microcephaly, the epidemic trajectory of this viral infection poses a significant concern for nearly 15 million children born in Americas each year [6].

The impact of migration of population on the distribution and spread of zika virus disease is required to be analyzed properly and must be understood clearly. Migration and immigration of the people from one country to another country due to different reasons play a crucial role in the evolution and spread of zika virus.

The main objective of the present study is to understand the role of infective immigrants on the dynamics of zika virus, and also to develop effective solutions and strategies for its prevention and control and eventually to eradicate it.

Symptomatic infected human: The symptomatic infective humans are those who have already been infected by the disease and they show the symptoms of the disease and can transfer the disease to susceptible vector.

Asymptomatic infected human: The asymptotic infective humans have already been infected by the disease but they do not show any symptoms of the disease. However, they can transfer the disease to susceptible vector. It has been estimated that about 80% of persons infected with zika virus are asymptomatic [3].

2. Mathematical Formulation of the Model

The mathematical model is expected to help to understand better the transmission and spread of zika. The spread of the disease modeled using ordinary differential equations where humans and mosquitoes interact and infect each other.

For the purpose of this study the whole human population is classified in to four compartments: Susceptible \( S(t) \), Symptomatic infected \( I_s(t) \), Asymptomatic infected \( I_a(t) \), and Recovered \( R(t) \). Similarly, the mosquito population is classified into two compartments: susceptible, and infected.

Many mathematical models did not consider symptomatic and asymptomatic infected immigrants on the dynamics of zika virus [1 – 3]. To fill the gap and to account its impact the present study considers it. Thus, a new model that combines a \( SI_sI_aR \) system for disease transmission in the human hosts and \( SI \) model for disease transmission in the mosquito vector has been constructed. The variables used in the model and their physical interpretations are as follows:

| \( S(t) \) | Number of susceptible human population at any time \( t \) |
| \( I_s(t) \) | Number of Symptomatic infected human population at any time \( t \) |
| \( I_a(t) \) | Number of asymptomatic infected human population at any time \( t \) |
| \( R(t) \) | Number of recovered human population at any time \( t \) |
| \( S_v(t) \) | Number of susceptible vector population at any time \( t \) |
| \( I_v(t) \) | Infected vector population at any time \( t \) |

The vector component of the model does not include an immune class as mosquitoes never recover from the infection. That is, their infective period ends with their death due to their relatively short lifecycle. Thus, the immune class in the mosquito population is negligible and death occurs equally in all classes.

The model can be used for diseases that persist in a population for a long period of time.

Susceptible human compartment: People will join the susceptible compartment by natural birth or by immigration. People go out from this compartment by natural death or go to the symptomatic or asymptomatic infected classes. The remaining people will stay in the compartment itself. Peoples in the susceptible compartment will get infection from infected vector.

Infected human compartments: Into infected compartment some people will enter from susceptible after getting infection. Some others will enter by immigrations from other places. Some people of the infected human compartment will die with natural reasons or go to recovered class after developing immunity.

Recovered compartment: Human populations in recovered class will go out of the compartment through natural death.

Susceptible vector compartment: Vectors will join the susceptible class by natural birth and leaves the compartment through natural death. They go to the infected class after getting infection from symptomatic or asymptomatic infected humans.

Infected vector compartment: The infected vector leaves the compartment through natural death because of short life cycle, but not of infection.

In these model \( A_hN_h \) and \( A_vN_v \) represents the total recruitment rate of human and mosquito population respectively.

The susceptible human population get infection by the rate \( b \), where \( b \) is the rate at which susceptible human becomes infected because of infected vector and \( b \) is the biting rate of infected mosquito at which susceptible human becomes infected because of infected vector. The symptomatic infected immigrants come to the symptomatic infected human population by the rate \( P_1 \) and the asymptomatic infected immigrants come to the asymptomatic infected human population by the rate \( P_2 \). The symptomatic and asymptomatic infected human population goes to the recovered class equally by the rate \( \delta \) and from the compartment due to natural death by the rate \( \mu_hI_1 \) and \( \mu_hI_2 \) respectively. The recovered human leave the compartment due to natural death by the rate \( \mu_hR_h \). The susceptible
mosquito population get infection either from symptomatic or asymptomatic infected humans by the rate $\theta$ and respectively, and leave the compartment due to natural death by the rate $\mu_v$, $S_v$ and goes to infected mosquito compartment. Note that in this model we consider $\mu_v > \theta$. That is, the probability of transferring the disease to susceptible vector by asymptomatic infected person is more than by symptomatic infected person. The infected mosquito population infects the susceptible human population by the rate $\Phi$ and leaves the compartment due to natural death by the rate $\mu_v f_v$.

Table 2. Description of variables and parameters.

| $\Lambda_h$ | Recruitment rate of human population |
| $\mu_h$ | Natural death rate of human population |
| $b$ | Biting rate of infected mosquito |
| $\Phi$ | Rate of infection of susceptible human because of infected vector |
| $P_1$ | Symptomatic infected human immigration rate |
| $P_2$ | Asymptomatic infected human immigration rate |
| $\delta$ | Recovered rate of symptomatic and asymptomatic infected human population |
| $\Lambda_v$ | Recruitment rate of mosquito population |
| $\mu_v$ | Natural death rate of mosquito population |
| $\theta$ | Rate at which susceptible vector get infection from symptomatic infected human |
| $\varepsilon$ | Rate at which susceptible vector get infection from asymptomatic infected human |
| $1 - \varepsilon$ | Rate at which susceptible human becomes asymptomatic infected |

2.1. Model Assumptions

The present model has been developed based on the following assumptions:

(i) Susceptible vectors can be infected by symptomatic or asymptomatic infected humans.

(ii) Infected mosquitoes cannot recover from the infection. Infected mosquitoes die naturally but not due to disease or infection and the birth rate of mosquito equals with its death rate.

(iii) Infected humans do not die due to infection, but due to natural death.

(iv) All the newborn ones are susceptible to infection.

(v) The model considers only mosquito as a transmission agent. That is, infected mosquito can infect a susceptible human; but, infected human cannot infect a susceptible human.

(vi) The development of the virus starts when the infected mosquito bites the human host.

(vii) The infection is propagated to vectors by asymptomatic infected hosts with more rates than by symptomatic infected hosts.

(viii) The recovered individuals are assumed to acquire permanent immunity and there is no transfer from the $R$ class back to the $S$ class.

(ix) Susceptible vectors can get infection, through direct contact with a symptomatic or asymptomatic infected individual.

(x) Natural death or due to other diseases occurs in each of the compartments.

(xi) Populations move from one compartment to another compartment with certain rates.

Based on these assumptions the compartmental structure of the model showing the compartments and flow of population has been represented by a flow diagram as follows:

![Figure 1. The compartmental structure describing Zika virus transmission.](image)

Considering the model assumptions and the flow diagram given in Figure 1 the dynamics of zika virus in human and mosquito populations can be described by a system of ordinary differential equations constructed in terms of notations of variables and parameters as follows:

$$
\frac{dS_h}{dt} = \Lambda_h N_h - b \Phi I_v \left( \frac{S_h}{N_h} \right) - \mu_h S_h 
$$

$$
\frac{dI_1}{dt} = b \in \Phi I_v \left( \frac{S_h}{N_h} \right) + P_1 I_1 - (\mu_h + \delta) I_1
$$

$$
\frac{dI_2}{dt} = b(1-\varepsilon)\Phi I_v \left( \frac{S_h}{N_h} \right) + P_2 I_2 - (\mu_h + \delta) I_2
$$

$$
\frac{dR_H}{dt} = \delta (I_1 + I_2) - \mu_v R_H
$$

$$
\frac{dS_v}{dt} = \Lambda_v N_v - b \theta I_1 \left( \frac{S_v}{N_v} \right) - b \gamma I_2 \left( \frac{S_v}{N_v} \right) - \mu_v S_v
$$

$$
\frac{dI_v}{dt} = b \theta I_1 \left( \frac{S_v}{N_v} \right) + b \gamma I_2 \left( \frac{S_v}{N_v} \right) - \mu_v I_v
$$

With initial conditions $S_h(0) = S_{h0}$, $I_1(0) = I_{10}$, $I_2(0) = I_{20}$.

$$
R_h(0) = R_{h0}, \quad S_v(0) = S_{v0}, \quad I_v(0) = I_{v0}
$$

The total population sizes $N_h$ and $N_v$ can be determined by

$$
N_h = S_h + I_1 + I_2 + R_h \text{ and } N_v = S_v + I_v
$$

2.2. Positivity of the Solution

The general model equations of system (1-6) are to be epidemiologically meaningful and well posed; we need to prove that all the state variables are non-negative.

Theorem 1: If $S_h(0) > 0$, $I_1(0) > 0$, $I_2(0) > 0$, $I_v(0)$ then the solutions $\{S_h(t), I_1(t), I_2(t), R_h(t), S_v(t), I_v(t)\}$ on the system of equation (1-6) are non-negative for all $t > 0$.

Proof: To show the positivity of the solution of the dynamical system comprising the equations (1) to (6) we have to consider and verify each differential equation and
show that their solution is positive.

First let us consider equation (1) of the dynamical system
\[
dS_h/dt = \lambda_h N_h - b \varnothing I_v (S_h/N_h) - \mu_h S_h,
\]
\[
dS_h/dt \geq -b \varnothing I_v (S_h/N_h) - \mu_h S_h, dS_h/dt \geq -\lambda S_h - \mu_h S_h \geq -(\lambda + \mu_h)S_h
\]

Where \( \lambda = b \varnothing (I_v/N_v) \). Up on integrating the inequality we obtain analytic solution as \( S_h(t) \geq S_{ho} e^{-(\lambda + \mu_h) t} \). Here \( S_{ho} \) is integral constant and represents initial population of the susceptible human compartment and hence it is a positive quantity. Now within the limit \( t \to \infty \) the analytical solution leads to \( S_h(t) > 0 \).

Hence the solution or the population size of the susceptible compartment \( S_h(t) \) is always positive.

Secondly let’s consider equation (2) of the dynamical system \( dI_1/dt = b \in I_v (S_h/N_h) + p_1 I_1 - (\mu_h + \delta) I_1 \) which implies \( dI_1/dt \geq b \in I_v (S_h/N_h) - k I_1 \) where \( k = \mu_h + \delta - p_1 \). Integrating by using separation of variables we have the first order linear ordinary differential equation and can be solved to obtain a particular solution as \( I_1(t) \geq I_{10} e^{-k t} \) Since \( I_{10} > 0 \), and the exponential function always positive, it is clear that \( I_1(t) \geq 0 \). Similarly \( I_2(t) \geq I_{20} e^{-q t}, \) where \( q = \mu_h + \delta - p_2 \).

Thirdly, let’s consider equation (4) of the dynamical system \( dR_h/dt = \delta (I_1 + I_2) - \mu_h R_h \) which can be expressed as \( dR_h/dt \geq -\mu_h R_h \). Up on integrating the inequality we obtain an analytic solution as \( R_h(t) \geq R_{ho} e^{-\mu_h t} \). Since \( R_{ho} > 0 \), and the exponential function always positive, it is clear that \( R_h(t) \geq 0 \).

Fourthly consider equation (5) of the dynamical system \( dS_v/dt = \lambda_v N_v - b \gamma I_2 (S_v/N_v) - \mu_v S_v \) without the loss of generality this can be expressed as \( dS_v/dt - \gamma (I_2/N_v) \) where \( (t) = b \gamma (I_2/N_v) - b \gamma (I_2/N_v) \). Integrating by using separation of variable we have the first order linear ordinary differential equation and can be solved to obtain a particular solution as \( S_v(t) = S_{vo} e^{-(\gamma - \mu_v) t} \). Since \( S_{vo} > 0 \) and the exponential function always positive, it is clear that \( S_v(t) \geq 0 \).

Finally let’s consider equation (6) of the model equation which is \( dI_v/dt = b \theta I_1 (S_v/N_v) + b \gamma I_2 (S_v/N_v) - \mu_v I_v \) without the loss of generality this can be expressed as \( dI_v/dt \geq -\mu_v I_v \). Upon integrating the inequality we obtain the analytic solution as \( I_v(t) = I_{vo} e^{-\mu_v t} \). Here \( I_{vo} \) is integral constant and represent initial infective vector population and hence it is a positive quantity. Now within the limit \( t \to \infty \) the analytical solution leads to \( I_v(t) \geq 0 \). Hence the solution or the population size of the infected compartment \( I_v(t) \) is always non-negative.

Therefore the solution sets \( \{S_h(t), I_1(t), I_2(t), R_h(t), S_v(t), I_v(t)\} \) of the model equations (1) to (6) are all non-negative for all \( t \geq 0 \).

2.3. Boundedness of the Solution

The total human population \( N_h(t) = S_h(t) + I_1(t) + I_2(t) + R_h(t) \) implies using equations (1) to (4) \( dN_h/dt = dS_h/dt + dI_1/dt + dI_2/dt + dR_h/dt = \lambda_h N_h - \mu_h N_h + P_1 I_1 + P_2 I_2 \) (8) Here without loss of generality and physical meaning \( I_1 < N_h, I_2 < N_h \) and \( I_1 + I_2 \leq N_h \), which implies \( dN_h/dt \leq \lambda_h N_h - \mu_h N_h + P_1 N_h + P_2 N_h \) solving this we have the analytic solution of the form \( N_h(t) \leq (\lambda_h + P_1 + P_2)N_h/N_h - \mu_h N_h \) or equivalently it implies that \( 0 < N_h(t) \leq (\lambda_h + P_1 + P_2)N_h/N_h \) as \( t \to \infty \). Therefore the solution system of human population is bounded. Similarly the total vector population is \( N_v(t) = S_v(t) + I_v(t) \) implies using equations (5) and (6) we have \( dN_v/dt = dS_v/dt + dI_v/dt = \Lambda_v N_v - \mu_v N_v \). Since in our assumption the natural birth rate and the death rate of vector is equal then, \( \Lambda_v = \mu_v \) which implies the vector population is constant and equal with the initial vector population which is bounded. Therefore the feasible region is

\[\Omega = \{I_v X N_v = \{S_h I_1, I_2, R_h N_h, S_v, I_v \in R^2; N_h \leq (\lambda_h + P_1 + P_2)N_h, \mu_h \}
\]

2.4. Disease Free Equilibrium Point

Disease-free equilibrium (DFE) points are steady-state solutions where there is no Zika virus infection i.e. \( I_1 = I_2 = R_h = I_v = 0 \). This is obtained by setting the right hand side of the model equation equal to zero. Thus, the disease-free equilibrium point of the model equation is, \( E_0 = \{I_h I_1, I_2, R_h N_h, S_v, I_v \mid (\lambda_h N_h / \mu_h, 0, 0, 0, \lambda_v N_v / \mu_v, 0 \} \).

2.5. Basic Reproduction Number

The basic reproduction number denoted by \( R_0 \), is defined as the number of secondary infections that result from the introduction of a single infectious individual into a completely susceptible population during its entire period of infectiousness, will be calculated by using the next generation matrix technique. It is an important parameter in epidemiology as it sets the threshold in the study of a disease both for predicting its outbreak as well as evaluating its control strategies. Persistent and die out of the disease in a community depend on the size of the reproduction number, \( R_0 \). If the reproductions number \( R_0 > 1 \) the disease break out and if the reproduction number \( R_0 < 1 \), the disease dies out over the period of time. When \( R_0 = 1 \) then the disease becomes endemic, meaning the disease remains in the population at a constant rate as one infected human transmits the disease to one susceptible vector and one infected vector transmits the disease to one susceptible human.

Now by considering the infective compartments in human and mosquito population we can compute the reproduction number by using next generation approach. The infected compartments are \( I_1, I_2, I_v \) . From those infected compartments we can compute the infection state \( F \) and transfer state \( V \). The reproduction number is the dominant eigenvalue of the matrix \( FV^{-1} \). The rate of change of population in this compartments are give in the dynamic model equation (2, 3) and equation(6). From this we can find \( f_i \) and \( v_i \).
3. Analysis of the Model

In this section, the equilibrium points for the general model are identified and their stability analysis is made. The model exhibits two types of equilibrium points viz., disease free equilibrium points and endemic equilibrium points.

3.1. Scaling of the Model

In order to simplify and analyze the Zika model in system (1-6) we work with fractional quantities instead of actual populations by scaling the population of each class by the total species population as papers [4, 8, 11]. The model can be simplified by assuming the following fractions with respect to time\(t\).

\[
F = \begin{bmatrix}
0 & 0 & \text{be} \Phi \\
0 & 0 & \alpha(1-\varepsilon) \Phi \\
b \theta u & b & 0
\end{bmatrix}
\]

The eigenvalues of the matrix \(FV^{-1}\) are the solution of the characteristic equation \(|FV^{-1} - \lambda I| = 0\) where I is an identity matrix and \(\lambda\) is the Eigen value of the matrix \(FV^{-1}\).

\[
FV^{-1} = \begin{bmatrix}
0 & 0 & \frac{\text{be}\Phi}{u} \\
0 & 0 & \frac{\alpha(1-\varepsilon)\Phi}{v} \\
\frac{b\theta u}{k} & \frac{b\theta u}{q} & \frac{1}{u}
\end{bmatrix}
\]

Hence the characteristic equation of the matrix \(FV^{-1}\) is given by

\[
\det (FV^{-1} - \lambda I) = 0
\]

Differentiating the fractions with respect to time \(t\) and simplifying as follows and the scaled version of the model becomes

\[
dS_h/dt = \Lambda - \alpha s_h i_v - \mu_h s_h
\]

\[
di_v/dt = \alpha \varepsilon i_v s_h - \mu_h i_1 - \delta i_1 + P_1 i_1
\]

\[
di_2/dt = \alpha(1-\varepsilon) i_v s_h - \mu_h i_2 - \delta i_2 + P_2 i_2
\]

\[
di_1/dt = \beta i_1 s_v + b i_2 s_v - \mu_v i_v
\]

3.2. Existence and Stability of Disease Free Equilibrium

Disease-free equilibrium (DFE) points are steady-state solutions where there is no Zika virus infection and the equilibrium points are obtained by setting the right hand sides of the model equations to zero.

Theorem 1: The disease free equilibrium point is locally asymptotically stable if and only if the reproduction number \(R_0 < 1\) and unstable if \(R_0 > 1\).

Proof: The local stability of \(E_0\) is then determined from the signs of the eigenvalues of the Jacobian matrix. At the disease-free equilibrium, \(E_0\), the Jacobian matrix is given by

\[
J_{E_0} = \begin{bmatrix}
-\mu_h & 0 & -\alpha \frac{s_h}{\mu_h} \\
0 & -k & \alpha \varepsilon \frac{s_h}{\mu_h} \\
0 & -q & \alpha(1-\varepsilon) \frac{s_h}{\mu_h}
\end{bmatrix}
\]

and

\[
\det (J_{E_0} - \lambda I) = 0
\]

Where, \(s_h = \frac{\mu_h}{\mu_v}\) solving \(Q(\lambda) = \det (J_{E_0} - \lambda I) = 0\) we have

\[
-\mu_h + \lambda = 0 \Rightarrow \lambda = -\mu_h
\]

The other three Eigenvalues are the roots of the characteristic equation of the matrix formed by excluding the first row and first column of the Jacobian matrix and has the characteristic equation of the form

\[
\lambda^3 + a_1 \lambda^2 + a_2 \lambda + a_3 = 0
\]

Where,

\[
a_1 = k + q + \mu_v > 0, \quad a_2 = kq + k\mu_v + q\mu_v - \alpha b \mu \eta \gamma(1-\varepsilon) - \alpha b \mu \eta \theta \varepsilon \quad \text{and} \quad a_3 = kq \mu_v(1 - R_0^2) \quad \text{if and only if} \quad R_0 < 1
\]

Due to the complexity in determining the signs of the remaining eigenvalues, we employ Routh–Hurwitz conditions for stability. The roots of the characteristic equation have negative real parts if and only if all the principal diagonal minors of the Hurwitz matrix are positive. For our case of a third order system, the stability criterion is defined by the inequalities

\[
a_1 > 0, a_2 > 0 \quad \text{and} \quad a_1a_2 - a_3 > 0.
\]

Clearly \(a_1 > 0, a_3 > 0\) and \(a_1a_2 - a_3 > 0\).
where $kq + \mu_{\nu} + \mu_{\psi} > \alpha b \psi u \gamma (1 - \epsilon) + \alpha b \eta \mu \theta \epsilon$ and also $a_1 \alpha_2 - a_3 > 0$.

Hence, by Routh–Hurwitz criterion, all the eigenvalues have negative real parts if $R_0 < 1$. Hence from Routh–Hurwitz criterion we have all the real parts of eigenvalues of the Hurwitz matrix being negative, hence the disease free equilibrium point is stable if $R_0 < 1$.

Theorem 2: The disease free equilibrium point $E_0$ of the system of ordinary differential equations is globally stable if $R_0 < 1$.

Proof: The proof is based on using a comparison theorem. The equation of the infected components can be written in terms of

$$
\begin{bmatrix}
\frac{dI_1}{dt} \\
\frac{dI_2}{dt} \\
\frac{dI_3}{dt}
\end{bmatrix} = (F - V)
\begin{bmatrix}
I_1 \\
I_2 \\
I_3
\end{bmatrix} -
\begin{bmatrix}
0 & 0 & 0 \\
0 & (b \theta I_1 + b \gamma I_2)I_0 & 0 \\
0 & 0 & (b \theta I_1 + b \gamma I_2)I_0
\end{bmatrix}
\begin{bmatrix}
I_1 \\
I_2 \\
I_3
\end{bmatrix}
$$

(15)

And also all the eigenvalues of the matrix $[F - V]$ have negative real parts. It follows that the system of linear differential inequalities (15) is stable whenever $R_0 < 1$ [19].

3.3. The Endemic Equilibrium Point

Endemic equilibrium point is a steady-state solution, where the disease persists in human and mosquito population. This equilibrium implies that if the carrier population is present in the system, then the infection will be transmitted to the human and mosquito population. Endemic equilibrium $E_1$ of the system is obtained by setting right hand side of the model system of equation equal to zero. For the existence and uniqueness of endemic equilibrium $E_1 = (S^*_h, I^*_h, I^*_m, I^*_v)$ where $= (S^*_h, I^*_h, I^*_m, I^*_v)$ and solving system above by setting the right hand side of the equation equals to zero we have the following $E_1 = (S^*_h, I^*_h, I^*_m, I^*_v)$

$$
S^*_h = \frac{\lambda_h (r R_0^2 + 1)}{R_0^2 \mu_h (r + 1)}
$$

$$
I^*_h = \frac{\lambda_h (r R_0^2 - 1)}{R_0^2 \mu_h (r + 1)}
$$

$$
I^*_v = \frac{\lambda_h (1 - \epsilon) + a b \lambda_h u \theta \epsilon - \mu_{\psi} k q}{a b \lambda_h k \mu \gamma (1 - \epsilon) + a b \lambda_h q u \theta \epsilon + a \mu_{\psi} k q}
$$

where $r = \mu_h / \alpha$

Here $I^*_v$ is positive if and only if $R_0 > 1$ and therefore all endemic equilibrium points can be expressed in terms of $R_0$ and all are positive. We apply the linearization technique in the system to determine the stability of the endemic equilibrium.

Theorem 3: The endemic equilibrium point of system of equation is locally asymptotically stable if and only if the reproduction number $R_0 > 1$ and unstable if $R_0 < 1$.

Proof: At the steady states of the model, the Jacobean matrix is given by

$$
J_{E1} =
\begin{bmatrix}
-(a I^*_v + \mu_h) & 0 & 0 & -a S^*_h \\
ace I^*_v & -k & 0 & a e S^*_v \\
\alpha (1 - \epsilon) I^*_v & 0 & -q & a (1 - \epsilon) S^*_h \\
b u \theta S^*_v & b u & S^*_v & -\mu_v
\end{bmatrix}
$$

The characteristic polynomial at the endemic equilibrium point is given by $P(\lambda) = \det (J_{E1} - \lambda I) = 0$, and therefore

$$
P(\lambda) = \lambda^4 + a_1 \lambda^3 + a_2 \lambda^2 + a_3 \lambda + a_4 = 0,
$$

(16)

Where

$$
a_1 = a I^*_v + \mu_h + k + q \mu_v > 0 \text{ if and only if } R_0 > 1,
$$

$$
a_2 = a k I^*_v + a q I^*_v + a \mu_v I^*_v + \mu_h k + \mu_h q + \mu_h \mu_v
$$

$$
+ k q + k \mu_v + q \mu_v
$$

$$
- a b \psi u \gamma (1 - \epsilon) S^*_h S^*_v - a b \theta u \epsilon S^*_h S^*_v
$$

$$
a_3 = a k q + a k I^*_v + a q \mu_v I^*_v + \mu_h q + \mu_h \mu_v k
$$

$$
- a b \psi u \gamma (1 - \epsilon) S^*_h S^*_v
$$

$$
- a b \theta u \epsilon S^*_h S^*_v + k q \mu_v
$$

$$
- k a b \psi u \gamma (1 - \epsilon) S^*_h S^*_v - ab q u \theta \epsilon S^*_h S^*_v
$$

$$
a_4 = a k q + a k I^*_v + a q \mu_v I^*_v + \mu_h \mu_v k + \mu_h \psi u \gamma (1 - \epsilon) S^*_h S^*_v
$$

Since solving the above characteristic polynomial for eigenvalues is tedious we will use the Routh–Hurwitz criterion to determine whether all roots have negative real parts and establish the stability of the system without solving the characteristic equation itself. By Routh–Hurwitz criteria the determinant of Hurwitz matrix becomes positive if the following conditions hold true $a_1 > 0, a_2 > 0$ and $a_4 > 0$ and $a_1 a_2 a_3 > a^2 a_4 + a^2$. It required that all these requirements should hold true in our present model. Therefore, after a certain manipulation we have the following results

$$
a_2 > 0 \text{ if and only if } R_0 > 1 \text{ and } a b \psi u \gamma (1 - \epsilon) (R_0^2 + 1) + \theta \epsilon (R_0^2 - 1) > a b \lambda_h (r R_0^2 + 1)(u \psi (1 - \epsilon) + u \theta \epsilon),
$$

$$
a_3 > 0 \text{ if and only if } R_0 > 1 \text{ and } a \lambda_h (b (R_0^2 - 1)) (u \psi (1 - \epsilon) - q u \theta \epsilon) > a b \lambda_h (r R_0^2 + 1)(u \psi (1 - \epsilon) + u \theta \epsilon)
$$

$$
a_4 > 0 \text{ if and only if } R_0 > 1 \text{ and } a b \lambda_h (R_0^2 - 1)(u \psi (1 - \epsilon) - q u \theta \epsilon) > a b \lambda_h (r R_0^2 + 1)(u \psi (1 - \epsilon) + u \theta \epsilon)
$$

Hence all the requirements on the parameters are found hold well.

Similarly, after exercising the same procedure it can be further observed that $a_1 a_2 a_3 > a_4$ and $a_1 a_2 a_3 > a_2^2 a_4 + a_2^2$ holds with a certain conditions. Hence all roots of the characteristic polynomial are negative and this verifies that the system of equation (1) to (6) is locally asymptotically stable.

4. Simulation Study of the Model.

Our numerical simulations examine the effect of infective symptomatic and asymptomatic immigrants on the transmission of the disease using MATLAB ode45 and show the sensitive parameters on the dynamics of the disease. Some of the parameter values are taken from literatures and the others are by assumptions. The parametric values are given in the following table.
Table 3. Model parameters and their values.

| Parameters | Case 1 | Case 2 | Case 3 | Reference |
|------------|--------|--------|--------|-----------|
| $\Lambda$  | 0.06   | 0.06   | 0.06   | [17]      |
| $\mu_h$    | 0.003  | 0.003  | 0.003  | [19]      |
| $\mu_v$    | 0.029  | 0.029  | 0.029  | [11]      |
| $\beta$    | 0.3    | 0.3    | 0.3    | [20]      |
| $\phi\Phi$ | 0.75   | 0.65   | 0.4    | Assumption|
| $\theta$   | 0.25   | 0.63   | 0.1333 | Assumption|
| $\delta$   | 0.4    | 0.7    | 0.2    | Assumption|
| $P_1$      | 0.2    | 0.2    | 0.2    | [21]      |
| $P_2$      | 0.2    | 0.3    | 0.1    | Assumption|
| $\varepsilon$ | 0.2  | 0.2    | 0.2    | Assumption|
| $\sigma$   | 0.4218 | 0.3656 | 0.225  | Assumption|

Figure 2. Numerical simulation of zika virus with $\theta = 0.25, \alpha = 0.4, \sigma = 0.4218, P_1 = 0.1, P_2 = 0.2$.

In figure 2 the fractions of the populations $s_h$, $I_1$, $I_2$, and $I_v$ are plotted vs. time. With increasing time, the susceptible fraction of human population and symptomatic infected human populations are decrease and the fractions of infected vector populations increase.

Figure 3. Numerical simulation of zika virus with $\theta = 0.63, \alpha = 0.7, \sigma = 0.365, P_1 = 0.2, P_2 = 0.3$.

In figure 3 the parameters are changed and with increasing time, the fractions of susceptible humans and the fraction of asymptomatic infected humans decrease and infected vectors are increases very fast because of the values of $P_2$ and $\gamma$.

Figure 4. Numerical simulation of zika virus with $\theta = 0.1333, \alpha = 0.225, P_1 = 0.1, P_2 = 0.1$.

In figure 4 the fraction of symptomatic and asymptomatic infected human populations are increase initially and decrease after a certain time because of $P_1, P_2$ and the values of the interaction rates $\theta$ and $\sigma$. Here the fraction of susceptible human population is decreases with increase time because the interaction coefficients of symptomatic and asymptomatic infected human populations. In all figures the value of $\alpha$ plays a crucial role on the dynamics and spread of zika virus.

Figure 5. The effect of contact rates from infected human to vector on infected vector population.

In this figure as the contact rates from symptomatic and asymptomatic infected human populations to susceptible vector increases proportionally infected vector populations increase rapidly.
In figure (6) as the rate of asymptomatic infected immigrants increase the infected human population becomes increase and this leads to increase the chance on infection for susceptible vector from those infected humans. In our model, the interaction coefficient $\theta$ from asymptomatic infective to susceptible vector, $\gamma$ from infected vector to susceptible human are more sensitive parameters. $P_1$ the symptomatic immigration rate, $P_2$ asymptomatic immigration rates and $\alpha$ the transmission probability from infected vector to susceptible human also sensitive parameters.

In general, we could observe that increasing the contact rates, human to mosquito and mosquito to human, leads to the outbreak of the disease and results an increasing zika virus prevalence. However, controlling these parameters with different control strategies allow the reproduction number to become less than 1, and then the disease dies out.

5. Conclusions

Intervention measures, to prevent or reduce the transmission of vector born, are currently being used with a degree of success in some parts of the world. Some of the methods used include house spraying with residual insecticides and most recently the use of mosquito bed nets. These methods operate by reducing the contact rates between the mosquitoes and humans. Spraying reduces mosquito longevity (and perhaps also fertility). This strategy is also likely to kill mosquitoes so it would increase the chances of killing infected mosquitoes and increase the mosquito death rate $\mu_m$. Preventing mosquito-human contacts should reduce the number of bites per mosquito. Controlling the symptomatic and asymptomatic infected immigrants also reduce the contact rate between the infected host and susceptible vector.

In this study, we have derived and analyzed a mathematical model in order to understand the transmission and spread of the zika virus disease. The model is modified by eliminating the recovery human and susceptible mosquito population from the system and by considering symptomatic and asymptomatic infective immigrants. We defined a reproductive number $R_0$ which provides the expected number of new infections from one infectious individual over the duration of the infectious period given that all other members of the population are susceptible. We showed the existence and stabilities of equilibrium points of the model. In the model, we demonstrated that the disease-free equilibrium point $E_0$ is stable is $R_0 < 1$, and unstable if $R_0 > 1$.

Numerical simulation of the model shows the dynamic properties of human and vector compartments versus time and the stabilities of the equilibrium points. One can observe from the simulations that the infected human population increases with larger values of the contact rates from mosquito to human population and human to mosquito population and with rates of infective immigrants. We notice that in order to reduce the basic reproduction number below 1, focus on reduction of the contact between mosquito vector and human host and controlling infective immigrants. Thus, using treated bed nets, and insecticides that would reduce the mosquito population and keep the human population stable.

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