Bull Math Biol (2016) 78:2228–2242
DOI 10.1007/s11538-016-0219-4

Society for Mathematical Biology

ORIGINAL ARTICLE

Sex, Mosquitoes and Epidemics: An Evaluation of Zika Disease Dynamics

David Baca-Carrasco[1] · **Jorge X. Velasco-Hernández**[1]

Received: 3 May 2016 / Accepted: 3 October 2016 / Published online: 14 October 2016

Abstract Since the first major outbreak reported on the island Yap in 2007, the Zika virus spread has alerted the scientific community worldwide. Zika is an arbovirus transmitted by *Aedes* mosquitoes; particularly in Central and South America, the main vector is the same mosquito that transmits dengue and chikungunya, *Aedes aegypti*. Seeking to understand the dynamics of spread of the Zika, in this paper, three mathematical models are presented, in which vector transmission of the virus, sexual contact transmission and migration are considered. Numerical analysis of these models allows us to have a clear view of the effects of sexual transmission and migration in the spread of the virus, showing that sexual transmission influences the magnitude of the outbreaks and migration generates outbreaks over time, each of lower intensity than the previous.

Keywords ZIKA virus · Sexual transmission · Migration · Basic reproductive number

1 Introduction

Zika is a arbovirus of the family *Flaviviridae*, genus *Flavivirus* (Dick et al. 1952). In the American continent,the most important *Flavivirus* are Dengue virus, Yellow fever virus, St Louis encephalitis virus, Japanese encephalitis virus, West Nile virus

✉ David Baca-Carrasco
davidbaca@im.unam.mx

Jorge X. Velasco-Hernández
jx.velasco@im.unam.mx

[1] Instituto de Matemáticas, UNAM, Campus Juriquilla, Boulevard Juriquilla 3001, Juriquilla, Queretaro 76230, Mexico

and Zika virus (ZIKV), transmitted by *Aedes* mosquitoes but could be also transmitted by other species, for example, *Culex* (Duffy et al. 2009; ECDC 2015; Hayes 2009; Tognarelli et al. 2015; Wikan 2016; Yakoba and Walker 2016).

Zika was discovered and isolated for the first time in a rhesus monkey in the Zika forest of Uganda in 1947. The studies showed that the virus circulates between primates and mosquitoes, particularly *Aedes africanus* (Dick et al. 1952; Fauci and Morens 2015).

The first report of human infection was in 1954 in Nigeria (Macnamara 1954); in 2007, the first large outbreak was reported in the Yap Island of the Federated States of Micronesia and the largest known outbreak before Brasil (Bogoch et al. 2016; Campos et al. 2015; ECDC 2015; Hennessey et al. 2016) started in the French Polynesia, South Pacific (Duffy et al. 2009; Musso et al. 2015; Tognarelli et al. 2015). Since then, the virus has spread very fast to large part of the Americas (Yakoba and Walker 2016).

Studies show that an Aedes-transmitted Zika epidemic is followed by an Aedes-transmitted Chikungunya epidemic (Fauci and Morens 2015). The ongoing pandemic confirms that Zika is predominantly a mild or asymptomatic dengue-like disease but the Guillain-Barré syndrome and other neurologic conditions may represent grave complications of Zika (ECDC 2015; Goorhuis et al. 2016; Oster et al. 2016).

Transmission of ZIKV to humans takes place through the bite of an infected female mosquito of the *Aedes* species (Petersen et al. 2016). ZIKV has been isolated from semen, confirming evidence that other nonvector means of transmission exist (however, further studies are needed to determine how long the virus can remain alive in semen). Recently ZIKV has been found in the female genital tract and the first case of transmission of ZIKV through sexual contact of female to male has been documented (Davidson et al. 2016; Prisant et al. 2016), also blood transfusions (problem that requires attention, since 80 % of people with ZIKV are asymptomatic) and perinatal transmission have been documented (ECDC 2015; Goorhuis et al. 2016; Musso et al. 2015; Oster et al. 2016). It is also suspected that the rapid spread of the virus is closely linked to people traveling to places where there are outbreaks and then return to their places of origin bringing virus with them (Ginier 2016; Goorhuis et al. 2016).

ZIKV is transmitted by mosquitoes of the family *Aedes*, such as *Ae. aegypti*, *Ae.albopictus*, *Ae.africanus*, *Ae.luteocephalus*, *Ae.vitattus*, *Ae.furcifer*, *Ae.hensilii* and *Ae.apicoargenteus* (Duffy et al. 2009; ECDC 2015; Hayes 2009; Tognarelli et al. 2015; Wikan 2016; Yakoba and Walker 2016). In the United States,the more widespread mosquito is *Ae.albopictus* constituting a risk for Zika spread. In this work, we construct and analyze mathematical models where the basic known characteristics of the disease are represented. The basic framework is that of a SEIR model coupled to a Ross-Macdonald type model. We introduce transmission of the virus through sexual contact and analyze the effect of migration in the dynamics of disease spread.

2 Mathematical Models

In this section, we develop two models for the problem described in the introduction. The first one is a very simple scenario in which sexual transmission is represented as if it were a directly transmitted disease; the second is somewhat more realistic in that two-sex dynamics is explicitly established.

2.1 A *SEIR*-Vector Model

Let S, E, I and R denote the susceptible, latent, infectious and immune individuals in a human population of total constant size N_H, and let M and V denote the susceptible and infected individuals of a mosquito population of total constant size $N_v = q/\delta$ where q is the recruitment rate and δ the mortality rate of the mosquitos; t represents time in days (see Fig. 1).

After Fig. 1 and a rescaling, the model is given by

$$\begin{aligned} E' &= \alpha V(1-E-I-R) + \phi I(1-E-I-R) - (\gamma+\mu)E, \\ I' &= \gamma E - (\eta+\mu)I, \\ R' &= \eta I - \mu R, \\ V' &= \beta I(1-V) - \delta V. \end{aligned} \tag{1}$$

In these equations, μ, γ and η represent the mortality, incubation and infection rates of the disease in the human while ϕ, α and β represent the contact rates for human to human, vector to human and human to vector, respectively.

This very simple model couples two very well-known epidemiological models, SEIR and Ross-Macdonald's, into a single system. Its basic reproduction number is

$$R_0 = \frac{1}{2}R_s + \sqrt{\left(\frac{1}{2}R_s\right)^2 + R_v^2}. \tag{2}$$

where

$$R_s = \frac{\phi\gamma}{(\gamma+\mu)(\eta+\mu)}, \quad R_v = \sqrt{\frac{\alpha\beta\delta}{q(\gamma+\mu)(\eta+\mu)}}$$

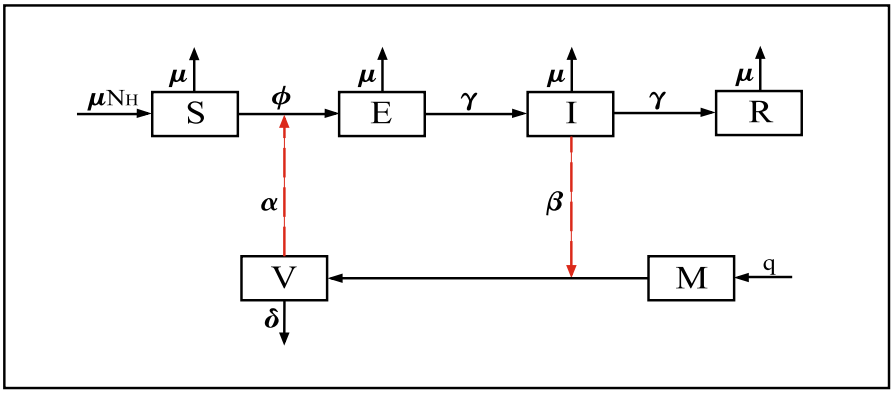

Fig. 1 Dynamics of virus spread. Each *arrow* points in direction of transmission and shows the relevant contact parameter

are the R_0's corresponding to the uncoupled human–human and vector–human transmission modes.

2.1.1 Existence of Disease Free Equilibrium

To continue the analysis, the equilibrium points (except the disease free equilibrium) are given by the solutions of the following two nonlinear algebraic equations

$$\begin{aligned} F_1(V) &= \beta\delta\mu A + (\alpha\beta\delta A + \phi\delta^2 A - \beta\delta\mu A)V - \alpha\beta\delta A V^2, \\ F_2(V) &= \alpha\beta^2 + \beta\delta\phi - (2\alpha\beta^2 + \beta\delta\phi)V + \alpha\beta^2 V^2, \\ A &= \frac{(\eta+\mu)(\gamma+\mu)}{\gamma\mu}. \end{aligned}$$

Note that both F_1 and F_2 are quadratic with opposite concavity, thus intersecting in the first quadrant for $0 < V < 1$.

To obtain F_1 and F_2 of the system (1), we solve equations $I' = 0$, $R' = 0$ and $V' = 0$ for E, R and I, respectively, and substitute these results in equation $E' = 0$. The condition for a unique nonnegative intersection is that $\beta\delta B < \alpha\beta^2 + \beta\delta\phi$, or equivalently

$$R_v^2 + R_s > 1, \tag{3}$$

which is satisfied if $R_0 > 1$. This condition guarantees the existence of an endemic equilibrium point, but provides no information on the prevalence level. Figure 2 shows the behavior of $F_1(V)$ and $F_2(V)$.

Given the geometry of the functions involved, the magnitude of endemic equilibrium depends on the slope of $F_1(V)$ given as:

$$F_1'(0) = (\alpha\beta\gamma + \phi\delta^2)\frac{B}{\mu} - \beta\delta B.$$

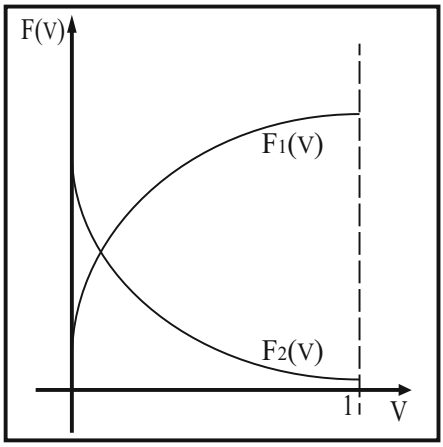

Fig. 2 Existence of the disease free equilibrium

Since $\mu \ll 1$, we see that $F_1'(0) \gg 1$, and thus the intersection is close to the origin. We can therefore conclude that if $R_0 > 1$ the endemic equilibrium exists but has a very low value (low prevalence). In essence,the behavior of the epidemic outbreak is one of very fast initial growth, high prevalence at the peak of the epidemic and then settlement to a very low but positive prevalence equilibrium state. Interestingly,the transient dynamics and equilibrium state of the model is firmly determined by the magnitude of $1/\mu$ (the length of sexual activity) and not by the contact parameter ϕ.

2.1.2 Numerical Analysis

We assign values to the parameters of the model, considering that ZIKV behaves similarly to dengue fever, e.g., (Adams and Boots 2010; Chowell et al. 2007; Feng and Velasco-Hernndez 1996; Pinho et al. 2010). To evaluate the sexual transmission effect on the disease spread,we show simulations for different values of ϕ. Table 1 shows the parameter values.

We use as benchmark the parameter values $\alpha = 0.2$, $\beta = 0.2$, $\gamma = 0.1$, $\eta = 0.125$, $\mu = 0.0001$, $\delta = 0.066$ and ϕ as shown in each figure. Figure 3 shows the behavior of solutions for varying ϕ. Increasing the value of this parameter, increases the value of R_0, but the epidemic tends to a low prevalence endemic state. The only observable change is in the intensity of the outbreak and the time it takes to dissipate.

2.2 A Two-Sex *SIR*-Vector Model

We now simplify our model assuming that there is no latency period for the disease in humans. We also consider that the human to human contact occurs between heterosexual couples, distinguishing between men and women in their respective rates of infection. Let S_f, I_f and R_f denote the susceptible, infectious and immune individuals in a *female* human population of total constant size N_f and S_m, I_m and R_m denote the susceptible, infectious and immune individuals in a *male* human population of total constant size N_m, such that $N_f + N_m = N$ the total human population and consider $N_f = N_m$; let M and V denote the susceptible and infected individuals of

Table 1 Parameter values

Notation	Description	
μ	Average age spam of sexually activity	$[\frac{1}{50\times365}, \frac{1}{18\times365}]$
α	Infection rate between susceptible human and infective vector	[0.1, 0.8]
ϕ	Infection rate between humans by sexual contact	Free
γ	Proportion of human exposed that become infective	$[\frac{1}{15}, \frac{1}{7}]$
η	Recovered rate of human host	$[\frac{1}{12}, \frac{1}{6}]$
δ	Average life of the vector	$[\frac{1}{20}, \frac{1}{10}]$
β	Infection rate between susceptible vector and infective human	[0.1, 1]

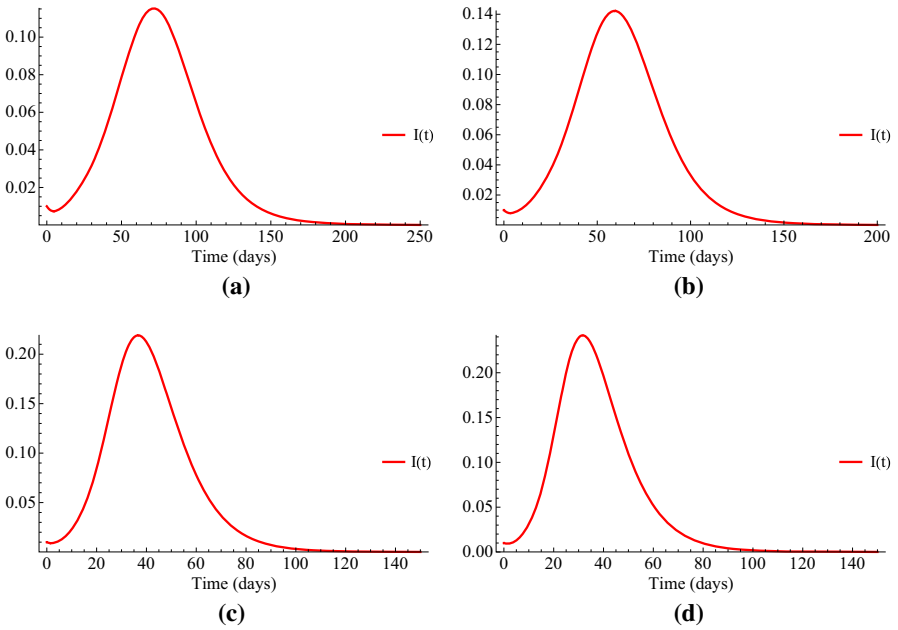

Fig. 3 Behavior of the infected population in system (1) for different values of the infection rate ϕ (a): $\Phi = 0$, $R_0 = 2.199$, (b): $\Phi = 0.1$, $R_0 = 2.635$, (c): $\Phi = 0.5$, $R_0 = 4.967$, (d): $\Phi = 0.7$, $R_0 = 6.352$

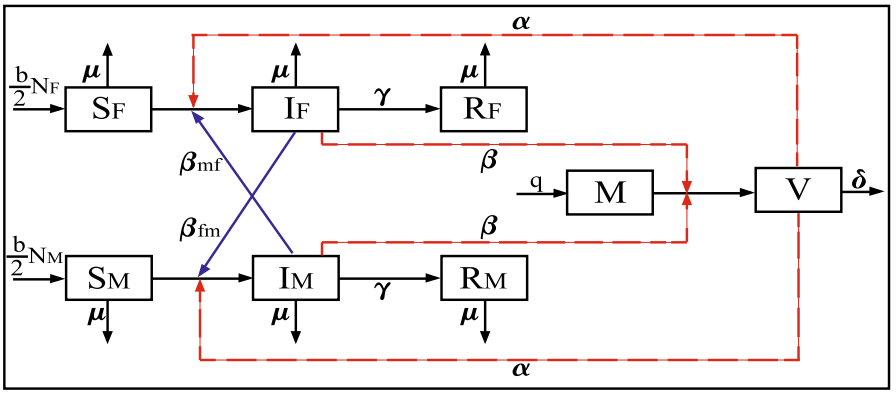

Fig. 4 Dynamics of virus spread considering two sexes

a mosquito population of total constant size $N_v = \frac{q}{\delta}$, where q is the recruitment rate and δ the mortality rate of the mosquitos. Figure 4 shows the dynamics of the model.

Considering Fig. 4 and normalizing the system, the SIR-vector model is given by

$$
\begin{aligned}
S_f' &= \mu - \mu S_f - \beta_{mf} S_f I_m - \frac{\alpha}{2} S_f V, \\
I_f' &= \beta_{mf} S_f I_m + \frac{\alpha}{2} S_f V - (\gamma + \mu) I_f, \\
R_f' &= \gamma I_f - \mu R_f,
\end{aligned}
$$

$$
\begin{aligned}
S_m' &= \mu - \mu S_m - \beta_{fm} S_m I_f - \frac{\alpha}{2} S_m V, \\
I_m' &= \beta_{fm} S_m I_f + \frac{\alpha}{2} S_m V - (\gamma + \mu) I_m, \\
R_m' &= \gamma I_m - \mu R_m, \\
M' &= \delta - \beta M \left(I_f + I_m\right) - \delta M, \\
V' &= \beta M \left(I_f + I_m\right) - \delta V
\end{aligned} \tag{4}
$$

where, b and μ represent human birth and mortality rates; β_{mf}, β_{fm}, α and β represent the contact rate for male to female, female to male, vector to human and human to vector, respectively.

Dynamics of spread of Zika is complex and the structure of its basic reproductive number is also complex. The next-generation matrix method and decomposition into subsystems used by Olmos et al. (2015) were used for calculation. Thus, the basic reproductive number is given by:

$$
R_0 = \sqrt[3]{\frac{R_{fvm}^3 + R_{mvf}^3}{2} + \sqrt{\left(\frac{R_{fvm}^3 + R_{mvf}^3}{2}\right)^2 - \left(\frac{R_{mf}^2 + R_{fv}^2 + R_{mv}^2}{3}\right)^3}} + \frac{R_{mf}^2 + R_{fv}^2 + R_{mv}^2}{\sqrt[3]{\frac{R_{fvm}^3 + R_{mvf}^3}{2} + \sqrt{\left(\frac{R_{fvm}^3 + R_{mvf}^3}{2}\right)^2 - \left(\frac{R_{mf}^2 + R_{fv}^2 + R_{mv}^2}{3}\right)^3}}},
$$

were R_{fvm}, R_{mvf}, R_{mf}, R_{fv} and R_{mv} are the basic reproductive numbers corresponding to the decoupled systems $Female \to Vector \to Male$, $Male \to Vector \to Female$, $Male \to Female$, $Female \to Vector$ and $Male \to Vector$ and they are given by

$$
R_{fvm} = \sqrt{\frac{\alpha\beta\beta_{mf}}{\delta(\gamma+\mu)^2}}, \quad R_{mvf} = \sqrt{\frac{\alpha\beta\beta_{fm}}{\delta(\gamma+\mu)^2}},
$$

$$
R_{mf} = \sqrt{\frac{\beta_{mf}\beta_{fm}}{\delta(\gamma+\mu)^2}}, \quad R_{fv} = R_{mv} = \sqrt{\frac{\alpha\beta}{\delta(\gamma+\mu)}}.
$$

A numerical analysis of system (4) shows that the dynamics of the model solutions are similar to those shown by system (1): sexual transmission does not determine the endemic level in the population, only affecting the magnitude of the outbreak. As before the benchmark for the numerical analysis is given by the set of values for the parameters: $\alpha = 0.2$, $\beta = 0.2$, $\gamma = 0.125$, $\mu = 0.000136$ and $\delta = 0.066$, leaving β_{mf} and β_{fm} free to explore their influence on the behavior of solutions. In Fig. 5, the behavior of solutions of the system (4) for different values of β_{mf} and β_{fm} is shown.

We conclude that although Zika transmission can occur through sexual contact, it has a minor effect on the overall population dynamics and, moreover, it does not

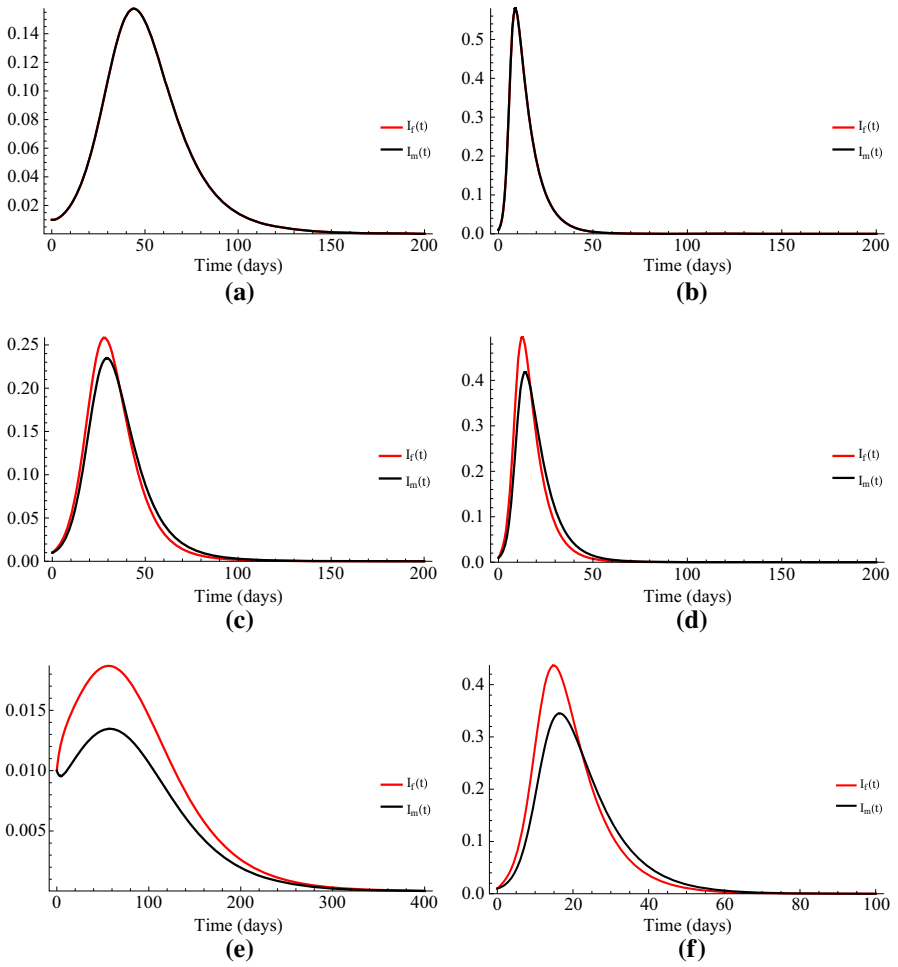

Fig. 5 (Color figure online) Behavior of the infected population in system (4) for different values of infection rates β_{mf} and β_{fm}. **e** and **f** show the dynamics of the disease is simulated considering sexually transmission only. **a** $\beta_{fm} = \beta_{mf} = 0.01$, $R_0 = 2.2$, **b** $\beta_{fm} = \beta_{mf} = 0.8$, $R_0 = 7.07$, **c** $\beta_{fm} = 0.1$, $\beta_{mf} = 0.2$, $R_0 = 2.8$, **d** $\beta_{fm} = 0.3$, $\beta_{mf} = 0.8$, $R_0 = 4.9$, **e** $\beta_{fm} = 0.1$, $\beta_{mf} = 0.2$, $R_0 = 1.1$, **f** $\beta_{fm} = 0.3$, $\beta_{mf} = 0.8$, $R_0 = 3.9$

explain the rapid spread of the disease. Therefore, the factor that allows the virus to spread fast must be the migration of people infected with Zika. To determine the role of migration in the spread of Zika, in the next section, a model that includes this factor is proposed.

2.3 Two-Sex *SIR*-Vector Model: The Role of Migration

Here besides considering transmission of the virus through sexual contact, we introduce human migration in a metapopulation-type model. Consider two places or patches

(A and B) inhabited by humans who can travel from their patch of residence to the other patch. The human population in each patch is divided into classes S_{FA}, I_{FA}, R_{FA}, S_{MA}, I_{MA} and R_{MA} representing susceptible, infected and recovered populations of female and male individuals, respectively, of patch A; similary, S_{FB}, I_{FB}, R_{FB}, S_{MB}, S_{MB} and S_{MB} representing susceptible, infected and recovered populations of female and male individuals of patch B. We also assume that $S_{FA} + I_{FA} + R_{FA} = N_{FA}$, $S_{MA} + I_{MA} + R_{MA} = N_{MA}$, $S_{FB} + I_{FB} + R_{FB} = N_{FB}$, $S_{MB} + I_{MB} + R_{MB} = N_{MB}$, $N_{FA} + N_{MA} = N_A$, $N_{FB} + N_{MB} = N_B$ and that $N_{FA} = N_{MA}$ and $N_{FB} = N_{MB}$.? On the other hand, in both patches A and B there is a local mosquito population that transmits the virus, where M_A, V_A, M_B and V_B, represent susceptible and infected individuals of the respective mosquito populations and such that $M_A + V_A = N_{VA}$, $M_B + V_B = N_{VB}$ and $N_{VA} = N_{VB} = \frac{q}{\delta}$, where q and δ are the recruitment and mortality rates of the mosquitos, respectively (see Fig. 6).

In Fig. 6, b and μ represent human birth and mortality rates; β_{MF}, β_{FM}, α and β represent the contact rate for male to female, female to male, vector to human and human to vector, respectively; e_{AB} and e_{BA} represent migration rate of A to B and B to A, respectively. See Appendix for the model equations.

2.3.1 Numerical Analysis

We now present a series of simulations for certain sets of values of the parameters. Each of the Figures represents one possible scenario that may develop under the presence

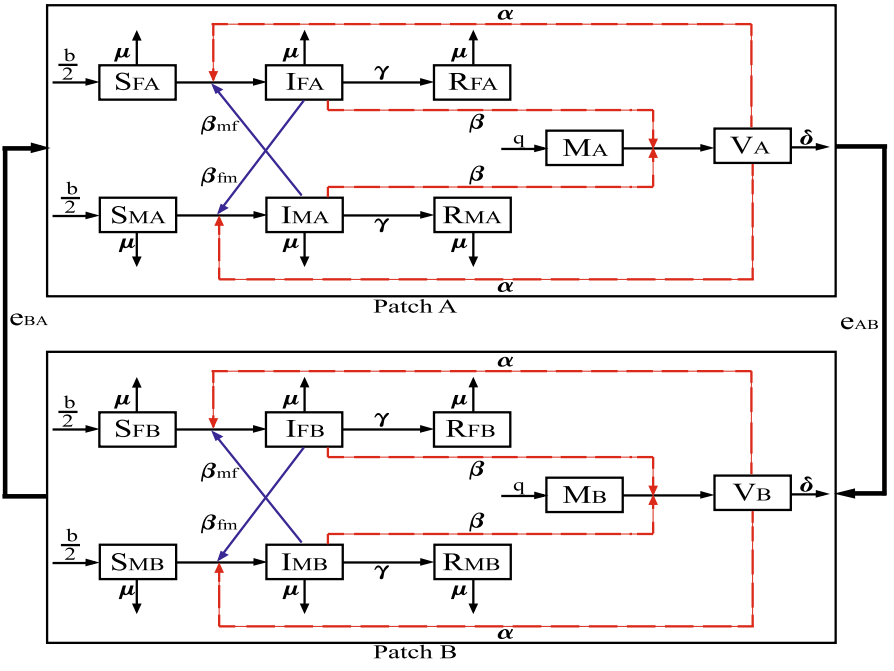

Fig. 6 (Color figure online) Diagramatic model for Zika spread considering two sex and migration

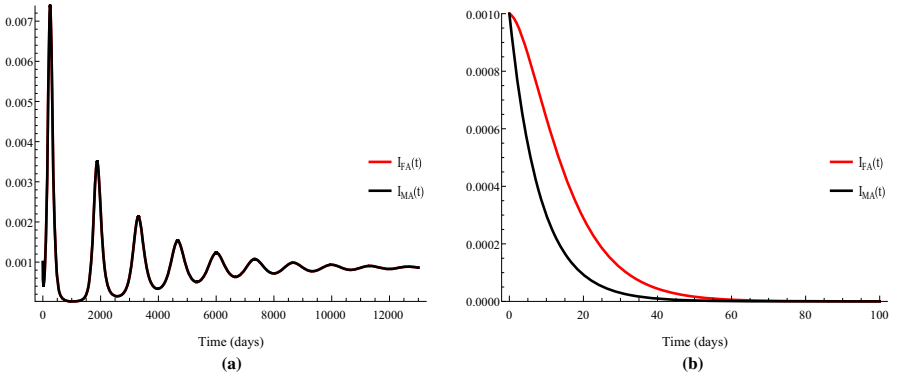

Fig. 7 (Color figure online) Solutions of system (5) for parameter values: **a** vector transmission only ($\beta_{FM} = \beta_{MF} = e_{AB} = e_{BA} = 0$, $p_A = p_B = k_1 = k_2 = 1$, $R_0 = 2.2$) and **b** sexual transmission only ($\beta_{FM} = 0.01$, $\beta_{MF} = 0.4$, $\alpha = \beta = e_{AB} = e_{BA} = 0$, $p_A = p_B = k_1 = k_2 = 1$ and $R_0 = 0.5$). Observe that sexual transmission cannot to maintain the spread of the disease

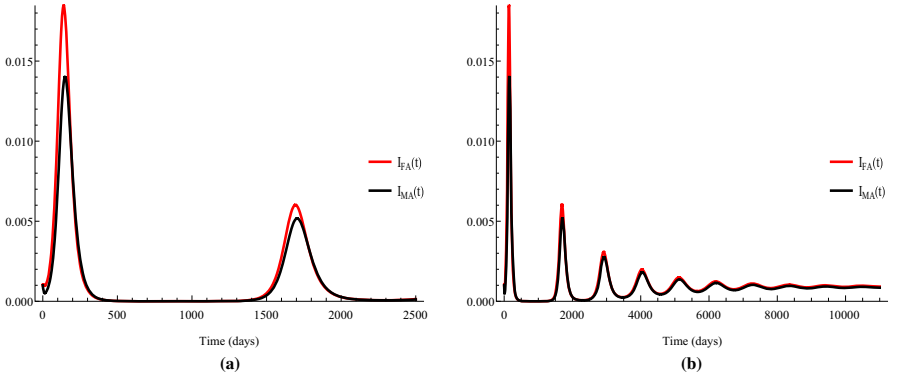

Fig. 8 (Color figure online) Solutions of system (5) for parameter values: $e_{AB} = e_{BA} = 0$, $p_A = p_B = k_1 = k_2 = 1$, that is, without migration and $R_0 = 2.81$; **a** 2500 days and **b** 10,000 days

of migration and Zika infection. For this model, our benchmark parameter values are: $\alpha = 0.2$, $\beta = 0.2$, $\gamma = 0.125$, $\mu = 0.000136$, $\delta = 0.066$.

Since there are very few documented cases of infection through sexual contact female to male, the rate of infection through sexual contact is low for $F \to M$ transmission and higher for $M \to F$ transmission: $\beta_{FM} = 0.01$ and $\beta_{MF} = 0.4$. The parameters for the migration rates are free to explore the effect on the dynamics of system. Figure 7 shows the behavior of solutions for the case when there is no migration, for two cases (a) only vector transmission and (b) only sexual transmission.

Figure 8 shows the behavior of the system in the absence of migration; the behavior, as expected, is the same for both patches (A and B).

Figure 9 shows the behavior of solutions when there is migration from A to B only and the entire population in B is susceptible. Observe that migration favors the disease eradication in patch A, which tends to die out after a short time, while in patch B a very pronounced outbreak occurs, that tends later to disappear but it is followed by

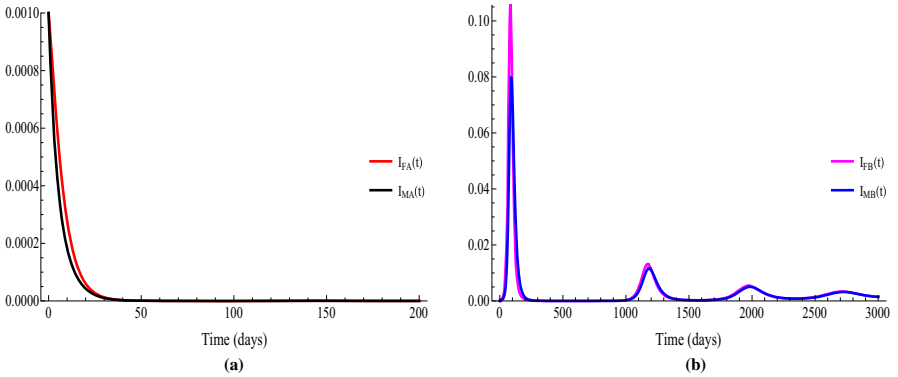

Fig. 9 (Color figure online) Solutions of system (5) for parameter values: $e_{AB} = 0.1$, $e_{BA} = 0$, $p_A = 0.998$, $p_B = k_1 = k_2 = 1$ and $R_0 = 4.25$. Behavior of solutions when there is migration from A to B only and the entire population in B is susceptible

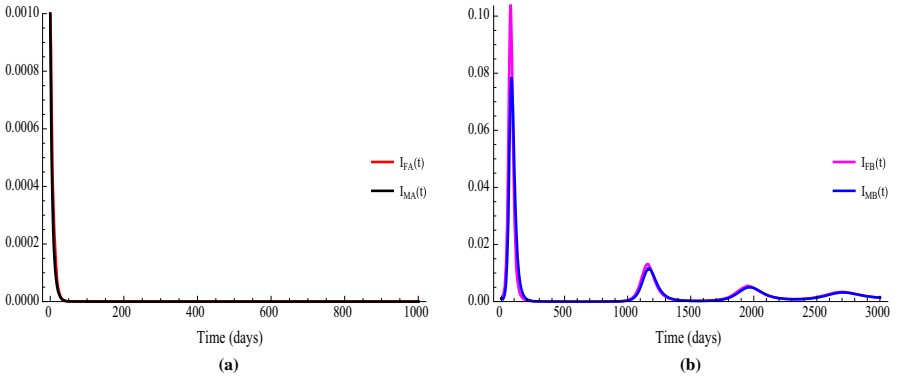

Fig. 10 (Color figure online) Solutions of the system (5) for parameter values: $e_{AB} = 0.1$, $e_{BA} = 0$, $p_A = 0.998$, $p_B = k_1 = k_2 = 1$ and $R_0 = 4.25$. Migration from A to B only and equal initial infection levels in both patches

smaller periodic outbreaks that eventually tend to stabilize at a very small endemic level.

Figure 10 shows the behavior of the system solutions when there is migration from A to B only and the infection levels are the same in both patches. The behavior of the disease in this scenario is very similar to that shown in Fig. 9, except for the outbreak magnitude in patch B which is slightly smaller, since the disease was already present in it.

Figure 11 shows the behavior of solutions considering an equal migration in both directions and setting the entire population in B susceptible. Note that the behavior of the disease in both patches is the same, except at the beginning, but in a short time both dynamics become coupled.

In Fig. 12, the behavior of the solutions is shown with equal migration rates in both directions and the initial infection levels equal in both patches.

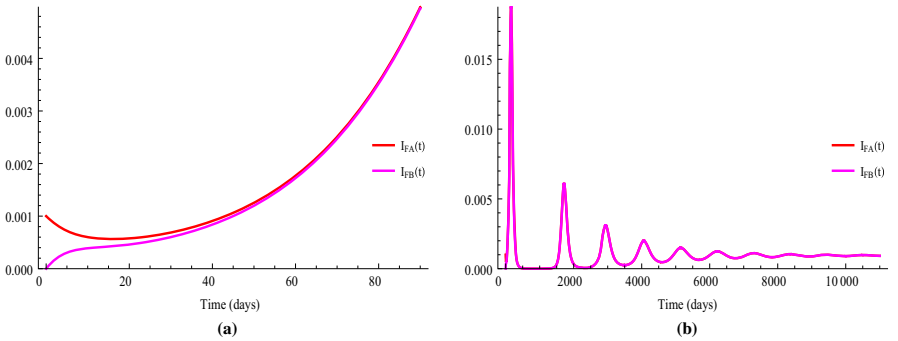

Fig. 11 (Color figure online) Solutions of the system (5) for parameter values: $e_{AB} = e_{BA} = 0.1$, $p_A = p_B = 0.998$, $k_1 = k_2 = 1$, $R_0 = 2.26$. Equal migration in both directions and the entire population in B susceptible

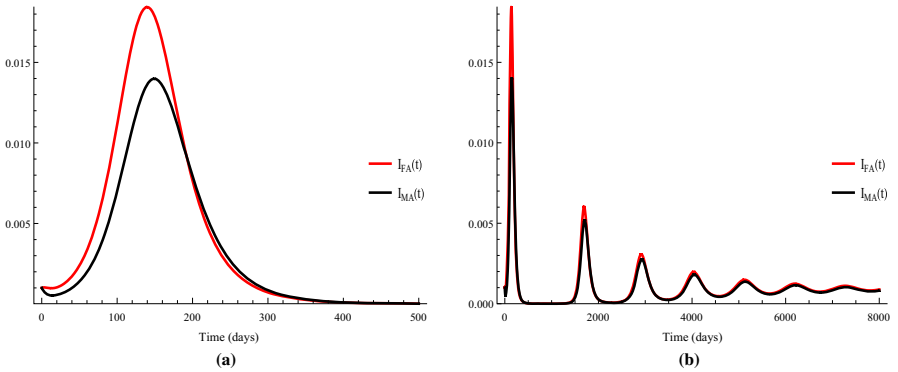

Fig. 12 (Color figure online) Solutions of the system (5) for parameter values: $e_{AB} = e_{BA} = 0.1$, $p_A = p_B = 0.998$, $k_1 = k_2 = 1$ and $R_0 = 2.26$. Equal migration rates in both directions and initial infection levels equal in both patches

Model (5) shows that migration is a determining factor for the spread of the disease. However, we can also see that the disease progresses through outbreaks, that, after a certain time practically go extinct, later appearing with less intensity, which it is consistent with the evolution of spread presented by the virus, appearing by outbreaks in different places (Duffy et al. 2009; Macnamara 1954; Musso et al. 2015; Tognarelli et al. 2015; Yakoba and Walker 2016). The model allows us to predict what is expected of the disease in the future and underline the important role of migration.

3 Conclusions

Our models predict some interesting features regarding the dynamics of spread of Zika and also suggest what we can expect in the future for the epidemic. One common result in the three models is that the endemic level subsequent to an outbreak is very low and that the transmission of the disease through sexual contact, only influences the magnitude of disease outbreak. The disease does not go away completely after the

outbreak, remaining endemic but at very low numbers. Our models are deterministic in nature but one can expect that at the very low densities at which Zika persist demographic stochasticity is a very important factor, probably the driving factor for the appearance of the unexpected epidemic outbreaks we observe in nature, same ones that do not appear in the same periods of time between one and another , as suggested by our model. We also find that transmission through sexual contact is insufficient for the disease to spread at the speed that has been observed these past months. Sexual transmission only affects the magnitude and duration of outbreaks, even when gender distinction is made. On the other hand, migration is decisive for the disease rapid spread of Zika. The time evolution of the disease shows successive outbreaks of decreasing intensity until stabilization at a very low endemic equilibrium. This may explain the presence of the virus, sporadically, in different parts of the world and the time between successive outbreaks.

Analyzing different scenarios generated by system (5) allows us to observe that, when migration occurs from patch A, where the disease is present, to patch B, where the total population is completely susceptible, note that the outbreak in patch A tends to its endemic level very quickly without the appearance of subsequent outbreaks, while in patch B there is a sequence of outbreaks of relatively low amplitude. When the patch B is infected and migration only occurs from A to B, the behavior is similar, except that the outbreak occurs earlier. When migration occurs in both directions with equal intensity migration rates, and one patch is fully susceptible there is an initial difference in the levels of infection in both patches for a short period of time, after which both patches synchronize. When the disease is present in both patches, the epidemic synchronizes very fast. For different migration rates, the disease dynamics is the same in both patches, except that the magnitude of the outbreaks is different, being higher in the population with the lowest rate of migration.

Finally, we conclude that the most vulnerable population sector are women, since the rate of infection through sexual contact is higher male to female and the magnitude of the outbreaks is higher in women than in men. Population size does not modify the above pattern.

Acknowledgments JXVH acknowledges support form PAPIIT (UNAM) Grant IA101215. DBC acknowledges support from CONACyT, UNAM and UNISON.

Appendix: Two-Sex and Migration Mathematical Model for ZIKV

In Sect. 2.3 analysis of a model in which, in addition to transmission through sexual contact, it considers the factor of migration between locations is presented. Figure 6 shows the dynamics to be studied and which is governed by the normalize system of ordinary differential equations

$$
\begin{aligned}
S'_{FA} &= \mu + k_1 e_{BA} S_{FB} - (\mu - e_{AB}) S_{FA} - \lambda_{FA} S_{FA}, \\
I'_{FA} &= \lambda_{FA} S_{FA} + k_1 e_{BA} I_{FB} - (\gamma + \mu + e_{AB}) I_{FA}, \\
R'_{FA} &= \gamma I_{FA} + k_1 e_{BA} R_{FB} - (\mu + e_{AB}) R_{FA}, \\
S'_{MA} &= \mu + k_2 e_{BA} S_{MB} - (\mu - e_{AB}) S_{MA} - \lambda_{MA} S_{MA},
\end{aligned}
$$

$$
\begin{aligned}
I'_{MA} &= \lambda_{MA} S_{MA} + k_2 e_{BA} I_{MB} - (\gamma + \mu + e_{AB}) I_{MA},\\
R'_{MA} &= \gamma I_{MA} + k_2 e_{BA} R_{MB} - (\mu + e_{AB}) R_{MA},\\
S'_{FB} &= \mu + \frac{1}{k_1} e_{AB} S_{FA} - (\mu + e_{BA}) S_{FB} - \lambda_{FB} S_{FB},\\
I'_{FB} &= \lambda_{FB} S_{FB} + \frac{1}{k_1} e_{AB} I_{FA} - (\gamma + \mu + e_{BA}) I_{FB},\\
R'_{FB} &= \gamma I_{FB} + \frac{1}{k_1} e_{AB} R_{FA} - (\mu + e_{BA}) R_{FB},\\
S'_{MB} &= \mu + \frac{1}{k_2} e_{AB} S_{MA} - (\mu + e_{BA}) S_{MB} - \lambda_{MB} S_{MB},\\
I'_{MB} &= \lambda_{MB} S_{MB} + \frac{1}{k_2} e_{AB} I_{MA} - (\gamma + \mu + e_{BA}) I_{MB},\\
R'_{MB} &= \gamma I_{MB} + \frac{1}{k_2} e_{AB} R_{MA} - (\mu + e_{BA}) R_{MB},\\
M'_A &= \delta - \beta M_A \left[\frac{P_A (I_{FA} + I_{MA}) + (1 - P_B)(I_{FB} + I_{MB})}{P_A + (1 - P_B)} \right] - \delta M_A,\\
V'_A &= \beta M_A \left[\frac{P_A (I_{FA} + I_{MA}) + (1 - P_B)(I_{FB} + I_{MB})}{P_A + (1 - P_B)} \right] - \delta V_A,\\
M'_B &= \delta - \beta M_B \left[\frac{P_B (I_{FB} + I_{MB}) + (1 - P_A)(I_{FA} + I_{MA})}{P_B + (1 - P_A)} \right] - \delta M_B,\\
V'_B &= \beta M_B \left[\frac{P_B (I_{FB} + I_{MB}) + (1 - P_A)(I_{FA} + I_{MA})}{P_B + (1 - P_A)} \right] - \delta V_B,
\end{aligned}
\tag{5}
$$

where,

$$
\begin{aligned}
\lambda_{FA} &= \frac{P_A \beta_{MF} I_{MA}}{P_A + (1 - P_B)} + \frac{\alpha P_A V_A}{2[P_A + (1 - P_B)]} + \frac{\alpha (1 - P_A) V_B}{2[P_B + (1 - P_A)]},\\
\lambda_{MA} &= \frac{P_A \beta_{FM} I_{FA}}{P_A + (1 - P_B)} + \frac{\alpha P_A V_A}{2[P_A + (1 - P_B)]} + \frac{\alpha (1 - P_A) V_B}{2[P_B + (1 - P_A)]},\\
\lambda_{FB} &= \frac{P_B \beta_{MF} I_{MB}}{P_B + (1 - P_A)} + \frac{\alpha P_B V_B}{2[P_B + (1 - P_A)]} + \frac{\alpha (1 - P_B) V_A}{2[P_B + (1 - P_A)]},\\
\lambda_{MB} &= \frac{P_B \beta_{FM} I_{FB}}{P_B + (1 - P_A)} + \frac{\alpha P_B V_B}{2[P_B + (1 - P_A)]} + \frac{\alpha (1 - P_B) V_A}{2[P_B + (1 - P_A)]},
\end{aligned}
$$

In equation, P_A is the proportion of time an individual of A remains in A and P_B is the proportion of time an individual of B remains in B, thus $1 - P_A$ is the proportion of time an individual of A remains in B and a similar interpretation has $1 - P_B$; $k_1 = \frac{N_{FB}}{N_{FA}}$ is the reason of the total of female of B and A, and $k_2 = \frac{N_{MB}}{N_{MA}}$ is the ratio of the total of male population in both patches.

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