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Control of the Transmission of Chikungunya Fever Epidemic Through the use of Adulticide

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Abstract: Problem statement: Another newly emerging disease, chikungunya fever, has appeared in various places such as the Island of Réunion, Thailand and other India. To control an epidemic of disease once it has been established, the use of adulticide is proposed. **Approach:** To simulate the effects of different control measurement, a mathematical model incorporating the specific features of this disease has been proposed. Using the standard methods for analyzing the dynamical stability of the system, the role of the efficacy of the adulticide on the basic reproduction number of the disease is studied. **Results and Conclusion:** It was found that below a particular value of the efficacy which depends on the values of the parameters used in the model, adulticide would not be effective at controlling the epidemic.

Keywords: Mathematical model, chikungunya fever, basic reproduction number, equilibrium, stability analysis, skin rash, transmission, dengue fever, *aedes aegypti* mosquito, symptoms, adulticide, equilibrium state

INTRODUCTION

Chikungunya fever is an arboviral disease caused by a member of the genus: *alphavirus* (family: Togaviridae), which was first isolated in 1953 in Tanzania (Pioloux *et al*., 2007). The symptoms of this disease are a fever, skin rash and incapacitating arthralgia. In the major outbreak of this disease in 2005 on the island of Réunion, 244,000 out of a population of 775,000 inhabitants reported that they had experienced these symptoms (Moulay *et al*., 2011). A different genotype (Asian strain) of this virus was found in India and Thailand in the early sixties.

The probable vector in the recent outbreak on the Réunion Island was the *Aedes albopictus* mosquito, while the probable vector in the outbreak in India was the *Aedes aegypti* mosquito. In the ongoing outbreak in Thailand, a new strain of the Chikungunya virus was isolated in both mosquito serotypes (Thavara *et al.*, 2009). Studies have shown that *Aedes albopictus* is more susceptible to the virus and therefore a more effective transmitter. The virus was found in both sexes of these two species of mosquitoes, indicating a possible role for transovarial transmission of the virus in the field populations of the mosquitoes.

The only defense against the appearance of an epidemic due to this virus is the control of the vector by use of insecticides. This can be done by the applications of larvicides to kill the mosquitoes in their larval state or of adulticides to kill them in their adult stage. Since the use of adulticides is more detrimental to the environment, The Ministry of Public Health in Thailand has launched national mosquito larva eradication program for the purpose of controlling both a Chikungunya and a dengue epidemic (Buathong, 2009). This approach is consistent with the recommendations of the 35th Session of the Subcommittee of the Executive Committee on Planning and Programming of PAHO (SPP35/7, 2001). They also pointed out that the use of adulticide be considered as a last resort strategy when the epidemic is established. While their recommendations were for Dengue fever, the policies also apply to the control of Chikungunya fever. Massad *et al.* (2008) recently studied the risk of Chikungunya fever in a Dengue epidemic area. They found that the basic reproduction number for Chikungunya fever was 64.4 % of that for Dengue fever. They assumed that both viruses were being transmitted by the same mosquito, *Aedes aegypti.* As we have pointed out, the Chikungunya

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virus is also transmitted by the *Aedes albopictus* mosquito which is a more effective transmitter of this virus than is the *Aedes aegypti* mosquito. This specie of mosquitoes is present in Thailand.

Mathematical models have become important tools in analyzing the spread and control of diseases using simulation approaches as in Adetunde (2009) ; Koriko and Yusuf, (2008) proposed mathematical models of Tuberculosis disease, determined the disease free and endemic equilibrium point and analyzed the stability.

In this study, we are interested in the role of applying adulticide on the temporal evolution of this disease and how the efficacy of the adulticide relates to the control of the spread. This can be done by using a mathematical model for the transmission of Chikungunya fever. Dumont, Chiroleu and Domerg (2008) used a SEIR (susceptible, exposed, infected, recovered) model for the human population and a LSEI (larvae-susceptible-exposed-infected) model for the mosquito population to describe the transmission of this disease on the Réunion Island. They found that a quick and focus interventions may be effective for controlling the diseases. Dumont and Chiroleu (2010) recently studies in more detail the vector control of this disease. They took into account the fact that the mean life of a mosquito infected by the Chikungunya virus is nearly half of that of an uninfected mosquito. Their preliminary results showed that the early use of massive spraying and mechanical control can stop the propagation of the Chikungunya disease.

There have been several reports on the development of resistance of the mosquitoes to several of the insecticides being employed in Thailand. Jirakanjanakit *et al.* (2007) found that *Aedes aegypti* had developed resistance to two commonly used insecticides, permetrin and deltamethrin. In another study, Jirakanjanakit *et al.* (2007) found that while temephos, a larvicide, was effective against the *Aedes aegypti* mosquitoes in mainly provinces in Thailand, its efficacy against the mosquitoes in two provinces ranged between 50.5-71.4%. Perich *et al.* (2000) found that the resting behavior of *Aedes aegypti* lowered the efficacy of adulticide, making a spraying program to be ineffectual.

We are interested in this study on whether there is an efficacy value below which an epidemic will not occur. We do this by performing a stability analysis based on the Routh-Hurwitz criteria (Marsden and McCracken, 1976) of a mathematical model for the transmission of Chikungunya fever which takes into account the application of an adulticide. We will determine the basic reproduction number for the disease and see when it will be less than one. We present the model and its analysis, respectively. We follow this with the results of our numerical calculations. Finally we present our discussion and conclusions.

MATERIALS AND METHODS

Model formulation: Our model is based on the standard SIR model instead of the SEIR model since we are interested in the effects of applying adulticide on the transmission of Chikungunya disease and not on the effects of delay times. In our model, the human population is divided into the susceptible human ($\bar{S}_h$), infected human ($\bar{I}_h$) and recovered human ($\bar{R}_h$) compartments. The mosquito population is divided into the susceptible mosquito ($\bar{S}_m$) and infected mosquito ($\bar{I}_m$), the recovered mosquito does not exist, since an infected mosquito does not recover. The dynamics of the disease is depicted in the compartment diagram, Fig. 1,

Looking at Fig. 1, we see that the transmission dynamics of the Chikungunya fever are described by the following ordinary differential equations; (Note: the unbarred variables are the normalized variables, i.e., the human and mosquito population are divided by N_h and N_m, respectively: $N_h = \bar{S}_h + \bar{I}_h + \bar{R}_h$ and $N_m = \bar{S}_m + \bar{I}_m$):

$$\frac{dS_h}{dt} = \mu_h(1 - S_h) - b\beta_{mh}\frac{A}{\mu_m + p}\frac{I_m}{N_h}S_h \quad (1)$$

$$\frac{dI_h}{dt} = b\beta_{mh}\frac{A}{\mu_m + p}\frac{I_m}{N_h}S_h - (\gamma_h + \mu_h)I_h \quad (2)$$

$$\frac{dR_h}{dt} = \gamma_h I_h - \mu_h R_h \quad (3)$$

$$\frac{dS_m}{dt} = A - b\beta_{hm}\frac{S_m}{N_h}I_h - \mu_m S_m - pS_m \quad (4)$$

$$\frac{dI_m}{dt} = b\beta_{hm}\frac{S_m}{N_h}I_h - (\mu_m + p)I_m \quad (5)$$

We assumed that the total human population, N_h, remains constant, so that the sum of the normalized human populations is one, i.e., $1 = S_h + I_h + R_h$ and that the total mosquito population also remains constant, leading to $A/(\mu_m + p) = S_m + I_m$, where S_m and I_m are the normalized mosquito populations.

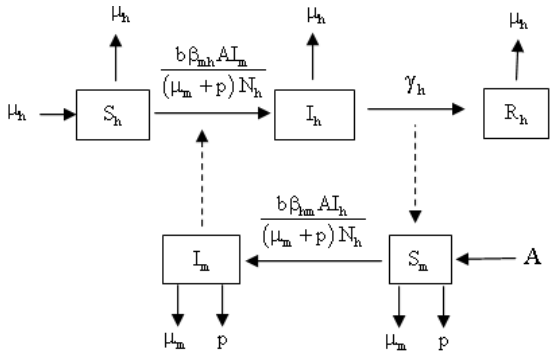


Fig. 1: Flow chart for the transmission of Chikungunya fever

μ_h is the birth (death) rate of human population, γ_h is the recovery rate of the infected human, A is the recruitment rate of the mosquito, b is the biting rate of mosquito population, β_{hm} is the probability that the infected mosquito transmits the virus to susceptible human by one bite, β_{hm} is the probability that the infected human transmits the virus to susceptible mosquito and μ_h is the death rate of mosquito population. Most importantly, p is the efficacy of adulticide chemical in killing the mosquito.

Analysis of the model: The model will be analyzed to investigate the equilibrium points and its stability. The conditions that the total mosquito and human populations are constants reduce the number of dependent variables to three, which we pick to be S_h, I_h and I_m. We find the total mosquito population at equilibrium to be $N_m = A/(\mu_m + p)$. This last relation also means that the number of mosquitoes can be controlled by changing the efficacy of the adulticide or by altering the recruitment rate of the adult mosquitoes, i.e., by removing the sites in which the larvae can breed.

Equilibrium points: The equilibrium points which is obtained by setting the LHS of Eq. 1, 2 and 5 to zero, i.e.:

$$\frac{dS_h}{dt} = 0, \quad \frac{dI_h}{dt} = 0, \quad \frac{dI_m}{dt} = 0 \tag{6}$$

Doing this, we obtain:

$$S_h = \frac{\mu_h N_h (L + b\beta_{hm} I_h)}{QKI_h + \mu_h N_h (L + b\beta_{hm} I_h)}, \quad I_m = \frac{KI_h}{L + b\beta_{hm} I_h} \tag{7}$$

And two values of I_h:

$$I_h = 0 \text{ and } I_h^* = \frac{-Y + \sqrt{Y^2 - 4XZ}}{2X} \tag{8}$$

Where:

$$X = TQK + VQK + VT + V^2$$

$$Y = DQK + UQK + UT + 2UV + VD - VQKN_h$$

$$Z = UD + U^2 - UQKN_h$$

$$\text{with } Q = \frac{b\beta_{mh}}{N_h}, K = b\beta_{hm}(A/(\mu_m + p)), D = L\gamma_h, \quad U = L\mu_h, V = \mu_h b\beta_{hm} \tag{9}$$

The first value leads to a disease free equilibrium state while the second value leads to an endemic equilibrium state.

Disease free state (E_0): In the absence of any infected mosquitoes, the equilibrium state will be the disease free state:

$$E_0(S_h, I_h, I_m) = (1, 0, 0) \tag{10}$$

Endemic disease state (E_1): This occurs when the $I_h^* = \frac{-Y + \sqrt{Y^2 - 4XZ}}{2X}$ is positive. The values of the susceptible humans and infected mosquitoes at equilibrium will be:

$$S_h^* = \frac{\mu_h N_h (L + b\beta_{hm} I_h^*)}{QKI_h^* + \mu_h N_h (L + b\beta_{hm} I_h^*)}, I_m^* = \frac{KI_h^*}{L + b\beta_{hm} I_h^*} \tag{11}$$

Thus the equilibrium endemic state becomes:

$$E_1(S_h^*, I_h^*, I_m^*) = \left(\frac{\mu_h N_h (L + b\beta_{hm} I_h^*)}{QKI_h^* + \mu_h N_h (L + b\beta_{hm} I_h^*)}, I_h^*, \frac{KI_h^*}{L + b\beta_{hm} I_h^*} \right) \tag{12}$$

Local asymptotically stability of equilibrium state: To determine local asymptotical stability of the equilibrium state, we first expand the solutions of Eq. 1, 2 and 5 about their equilibrium values, i.e., $\bar{X} = \overline{X^*} + \delta\bar{X}$ where $\overline{X^*}$ is the equilibrium state given by either Eq. 10 or 12. Performing this expansion, we get:

$$\left.\frac{d\delta\bar{X}}{dt}\right|_{\bar{X}=\overline{X^*}} = \text{Jacobian } (J)_{\bar{X}=\overline{X^*}} \cdot \delta\bar{X} \tag{13}$$

Where, the Jacobian is given:

$$J(S_h^*, I_h^*, I_m^*) = \begin{bmatrix} -(b\beta_{mh}\frac{I_m^*}{N_h} + \mu_h) & 0 & -b\beta_{mh}\frac{S_h^*}{N_h} \\ b\beta_{mh}\frac{I_m^*}{N_h} & -(\gamma_h + \mu_h) & b\beta_{mh}\frac{S_h^*}{N_h} \\ 0 & b\beta_{hm}\frac{((A/(\mu_m + p)) - I_m^*)}{N_h} & -(b\beta_{hm}\frac{I_h^*}{N_h} + \mu_m + p) \end{bmatrix} \quad (14)$$

with the equilibrium values substituted in.

Assuming that $\delta\overline{X}(t) = \overline{X_o}e^{\lambda t}$, the eigenvalues are obtained by solving:

$$\det(J_{\overline{X}=\overline{X^*}} - \lambda I) = 0 \quad (15)$$

Evaluating the determinant, we obtain what is known as the characteristic equation. Since we are looking for local asymptotically stable states, the eigenvalues should be negative and satisfy the Routh-Hurwitz criteria (Marsden and McCracken, 1976).

Local asymptotically stability of disease free state: Evaluating the Jacobian at the disease free state $E_0 = (1,0,0)$, we find that:

$$J = \begin{bmatrix} -\mu_h & 0 & -b\beta_{mh} \\ 0 & -(\gamma_h + \mu_h) & 0 \\ 0 & b\beta_{hm}\frac{(A/(\mu_m + p))}{N_h} & -(\mu_m + p) \end{bmatrix}_{E_0} \quad (16)$$

Substituting this Jacobian into Eq. 15, we arrive at the following characteristic equation:

$$(\mu_h + \lambda)(\lambda^2 + G\lambda + H) = 0 \quad (17)$$

Where:

$$G = p + \mu_m + \mu_h + \gamma_h, H = \gamma_h\mu_h + \gamma_h p + \mu_h\mu_m + \mu_h p - b^2\beta_{hm}\beta_{mh}\frac{(A/(\mu_m + p))}{N_h} \quad (18)$$

Looking at Eq. 17, we see that one of the eigenvalues is $\lambda_1 = -\mu_h$. The other two are the solutions of $\lambda^2 + G\lambda + H = 0$. They will be negative when G > 0, H > 0 or when:

$$b^2\beta_{hm}\beta_{mh}(A/(\mu_m + p)) < N_h(\mu_m + p)(\gamma_h + \mu_h) \quad (19)$$

The Routh-Hurwitz criteria for all the eigenvalues to be negative will be satisfied when $R_0 < 1$ where:

$$R_0 = \frac{b^2\beta_{hm}\beta_{mh}(A/(\mu_m + p))}{N_h(\mu_m + p)(\gamma_h + \mu_h)} \quad (20)$$

Local asymptotically stability of endemic equilibrium state: Evaluating the Jacobian at the endemic equilibrium state:

$$E_1(S_h^*, I_h^*, I_m^*) = \left(\frac{\mu_h N_h(L + \beta_{hm}I_h^*)}{QKI_h^* + \mu_h N_h(L + \beta_{hm}I_h^*)}, I_h^*, \frac{KI_h^*}{L + b\beta_{hm}I_h^*}\right) \quad (21)$$

where, $\overline{I_h^*}$ is the solution of $I_h^* = \frac{-Y + \sqrt{Y^2 - 4XZ}}{2X}$ where the definitions of X, Y and Z are given in Eq. 9, we get:

$$J = \begin{bmatrix} -\left(b\beta_{mh}\frac{I_m^*}{N_h} + \mu_h\right) & 0 & -\frac{b\beta_{mh}S_h^*}{N_h} \\ b\beta_{mh}\frac{I_m^*}{N_h} & -(\gamma_h + \mu_h) & \frac{b\beta_{mh}S_h^*}{N_h} \\ 0 & b\beta_{hm}\frac{(A/(\mu_m + p)) - I_m^*}{N_h} & -\left(b\beta_{hm}\frac{I_h^*}{N_h} + \mu_m + p\right) \end{bmatrix}_{E_1} \quad (22)$$

The characteristic equation obtained from $\det(J - \lambda I) = 0$ is:

$$\lambda^3 + \gamma\lambda^2 + \mu\lambda + \eta = 0 \quad (23a)$$

Where:

$$\gamma = w + k + q, \quad \mu = kw + qw + kq - vl, \quad \eta = kql + lmv - kvl \quad (23b)$$

With:

$$k = b\beta_{mh}\frac{I_m^*}{N_h} + \mu_h, \quad l = b\beta_{mh}\frac{S_h^*}{N_h}, \quad m = b\beta_{mh}\frac{I_m^*}{N_h}, \quad q = \gamma_h + \mu_h, \quad v = \frac{b\beta_{hm}((A/(\mu_m + p)) - I_m^*)}{N_h}, \quad w = b\beta_{hm}\frac{I_h^*}{N_h} + \mu_m + p \quad (23c)$$

The coefficients of the characteristic equation will be positive if the following Routh-Hurwitz criteria:

$$\gamma\mu > \eta \quad (24a)$$

Where:

$$\gamma\mu = kw^2 + qw^2 + 3kqw - wvl + k^2w + k^2q - kvl + q^2w + kq^2 - qvl \quad \text{and} \quad \eta = kql + lmv - kvl \quad (24b)$$

Is satisfied. For values meeting these criteria, E_1 will be locally asymptotically stable.

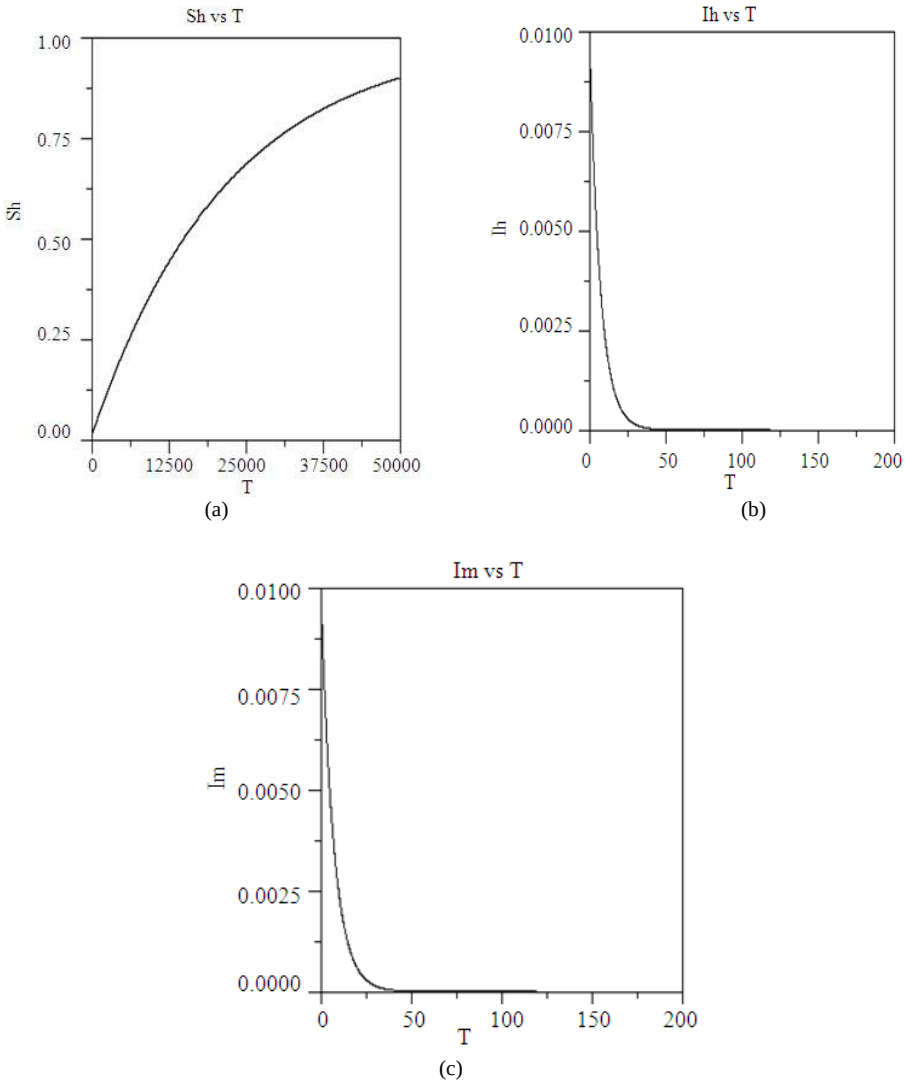


Fig. 2: Time series evolution of the population compartments for the case where the disease free state is the equilibrium state. (a) Susceptible human proportion (S_h). (b) Infected human proportion (I_h). (c) Infected mosquito proportion (I_m). The time evolutions of the three populations were obtained by solving Eq. 1, 2 and 5 using the following values taken from Table 1; A = 5000, b = 1, μ_m = 0.25, p =1, γ_h = 0.1428, μ_h = 0.0000457, β_{mh} = 0.5, β_{lm} = 0.7, N_h = 10,000. These values lead to a basic reproduction number R_0 = 0.784063 < 1. As we see, the equilibrium state is the disease free state E_0 = (1, 0, 0)

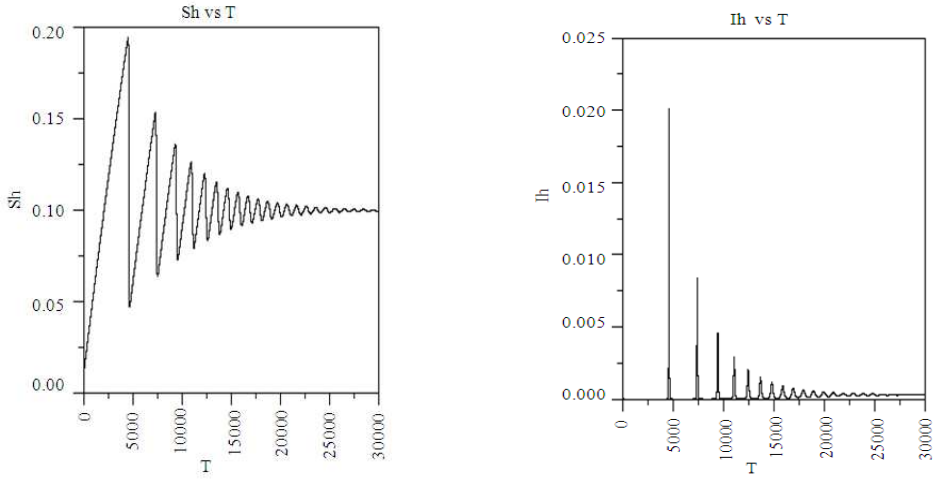
RESULTS AND DISCUSSION

Stability of disease free state: For the numerical values of the parameters listed in Table 1, the calculated eigenvalues and basic reproduction numbers are:

$$\lambda_1 = -0.0000457, \quad \lambda_2 = -1.14033, \quad \lambda_3 = -0.00251 \quad \text{and} \quad R_0 = 0.784063 \tag{25}$$

We have numerical solved Eq. 1, 2 and 5 using the values of the parameters listed in Table 1. Since these values leads to all of the eigenvalues to be negative and the basic reproduction number to be less than one, the equilibrium state will be the disease free state E_0 given by Eq. 10. Looking at Fig. 2, we see that this is true.

Stability of endemic state: Next we change the value of p to be equal to 0.1 and keep the other values of the parameters the same. With these values, we obtain:



(a) (b)

Fig. 3: Time series evolution of the population compartments for the case where the endemic state is the equilibrium state. (a) Susceptible human proportion (S_h). (b) Infected human proportion (I_h). The time evolutions of the two populations were obtained by solving Eq. 1, 2 and 5 using the same values used to obtain Fig. 2 except that the efficacy is now set to 0.1. Using p = 0.1 instead of p = 1.0, we get a basic reproduction number R_0 = 10.0008 > 1. The equilibrium state becomes the endemic state E_1(0.100107, 0.000576, 0.00115)

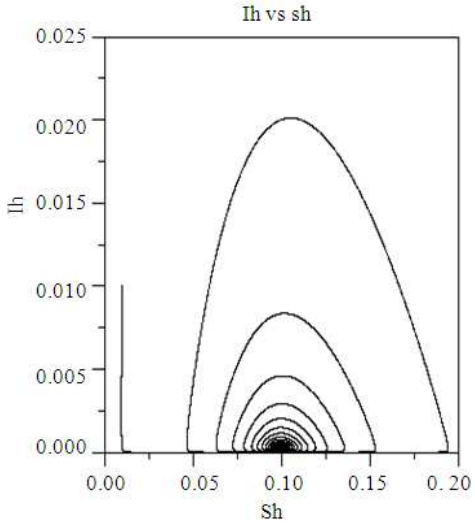


Fig. 4: Trajectory of the human population compartments in the S_h-I_h plane for the same set of parameter values used to obtain the time evolution behaviors shown in Fig. 3. As we see, the trajectory spirals into the endemic equilibrium state; E_1(0.100107, 0.000576, 0.00115)

$$\lambda_1 = -0.000015, \quad \lambda_2 = -0.436135+0.1636251i, \quad \lambda_3 = -0.4361350-0.1636251i \tag{26}$$

These values will lead the basic reproduction number R_0 to be 10.0008. This along with the fact that the real parts of the eigenvalues are all negative leads the equilibrium states to be the endemic state E_1(0.100107, 0.000576, 0.00115) and that this state will be locally asymptotically stable. The fact the λ_2 and λ_3 are complex conjugates means that the temporal behavior of the populations will exhibit oscillatory behaviors.

Table 1: Parameter values leading to disease free state

Parameters	value	References
b	1.0000000 (per day)	Rodrigues *et al*. (2010)
γ_h	0.1428000 (per day)	
β_{hm}	0.7000000	Pongsumpan (2006)
β_{mh}	0.5000000	
μ_h	0.0000457 (per day)	Nishiura (2006)
N_h	10,000.0000000	
μ_m	0.2500000 (per day)	
A	5,000.0000000	
p	1.0000000	

This is indeed seen in Fig. 3 where the time series solutions for susceptible and infected human populations are shown. As we see, as $t\rightarrow\infty$, the two populations converge to the endemic population levels. In Fig. 4, we see the trajectory of the solutions in the S_h-I_h plane. There we see, the trajectory spiraling into the equilibrium endemic state.

Our results show that the use of adulticide to control a Chikungunya fever epidemic once it develops depends on the efficacy of the adulticide to kill the mosquitoes. We have look to see what the value of the efficacy is needed for the adulticide to be effective. For the values of the parameters used, we find that the basic reproduction number R_0 is 0.726 for an efficacy value p = 0.9 and is 1.111 for p = 0.8. The need for such a high value of the efficacy is caused by our choice of 0.7 and 0.5 for the values of β_{hm} and β_{mh}, respectively. Using lower values of these two parameters would lead the required efficacy of the adulticide to be effective to be lower.

CONCLUSION

The values of the parameters for the mosquitoes used in the previous section are for the *Aedes aegypti* mosquitoes. A genetic change at position 226 in the gene for the glycolprotein E1/E2 created a mutated Chikungunya virus strain which had an increased capability for replication in the *Aedes albopictus* mosquito (Tsetsarkin, 2009). This change in the vector for transmission of the virus has made this disease more of a treat to the public health since *Aedes albopictus* has developed a capability to adapt to non tropical regions (Moulay *et al*., 2011), is aggressive day time biter (30-46 times per hour) (Cancrini, *et al*., 2003), lives a long life (4-8 weeks) and has long flight radius (400-600 meters) (Pioloux *et al*., 2007). These changes will change the numerical results but not the conclusion that control of the Chikungunya epidemics by means of spraying of adulticide must take into account the changing efficacy of the adulticide and when to switch to a new adulticide.

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