

Mathematical Modeling of Vector-Borne Diseases

Roman Ullah¹, Sakhi Jan¹, Gul Zaman², Saleem Khan¹, Saeed Islam³, Muhammad Altaf Khan³,
Hakeem Ullah³

¹Department of Mathematics, Bacha Khan University Charsadda, Khyber Pakhtunkhwa, Pakistan

²Department of Mathematics, University of Malakand, Chakdara, Dir, Khyber Pakhtunkhwa, Pakistan

³Department of Mathematics, Abdul Wali Khan University, Mardan, Khyber Pakhtunkhwa, Pakistan

Received: August 6, 2015

Accepted: October 28, 2015

ABSTRACT

In this paper, we developed an outbreak form of vector-borne infection which has direct mode of transmission in supplement to the vector mediated transmission. The qualitative behavior of the proposed model is analyzed with the help of the threshold quantity R_0 . An iterative method is used to find the numerical solution.

KEYWORDS: vector-borne, threshold quantity, dynamical behavior.

INTRODUCTION

Vector-borne disease is carried by vectors, such as mosquitoes, tick and sand-is, that are organisms that transmit pathogens and parasites from one infected host to another one. The period vector mentions to a medium, an arthropod or some other agency through which a pathogenic micro-organism is conveyed from a contaminated person to another uninfected individual. Bacteria, virus and protozoa are the three main living organisms involved in the mechanism of the transmission. Developing countries, in particular the tropic and sub tropic regions of Africa and Asia are the main victims of vector borne diseases. Almost half the community of the world is contaminated by the vector-borne infections directing to high morbidity and mortality [1]. The increment in traveling to and from tropical regions has helped the circulation of diseases that are constantly being discovered. Several vector-borne infections have emerged in recent years as diseases of considerable and widespread importance, among which Lyme disease and West Nile virus.

Many controlled programs were applied globally to eradicate the vector-borne infections [3]. Most of these programs were thriving, but the achievement was short-lived. The components to blame for the resurgence of vector-borne infections are complex. A number of models have been devised in the literature to study the dynamics of a vector-borne infection that considers a direct mode of transmission in human host community [2-8] Mathematical modeling has verified to play a significant role in profiting some insights into the transmission dynamics of contagious infections and propose control strategies [9-24]. In this work, we show that appropriate mathematical models of vector-borne diseases can provide a qualitative assessment for the problem.

Model Formulation:

In this section, we formulate a vector-borne epidemic model in which the total human population is divided into three subclasses, susceptible, infectious and recovered which are denoted by $S_h(t)$, $I_h(t)$ and $T_h(t)$, respectively. Also the vector population is subdivided in susceptible and infectious vectors, denoted by $S_v(t)$ and $I_v(t)$, respectively. The vector population (mosquito) has no immune class, since their infective period ends with their death. Thus

$$N_h(t) = S_h(t) + I_h(t) + T_h(t)$$

and

$$N_v(t) = S_v(t) + I_v(t)$$

are respectively the total human and vector population at time t , so the model is given by the following differential equations.

$$\frac{dS_h(t)}{dt} = \alpha_n - \frac{\beta_1 S_h(t) I_v(t)}{N_v(t)} - \frac{\beta_2 S_h(t) I_h(t)}{N_h(t)} + \gamma_h I_h(t) - \mu_n S_h(t)$$

$$\begin{aligned}
\frac{dI_h(t)}{dt} &= \frac{\beta_1 S_h(t) I_v(t)}{N_v(t)} + \frac{\beta_2 S_h(t) I_{vh}(t)}{N_h(t)} - \mu_h I_h(t) - \rho_h I_h(t) \\
\frac{dT_h(t)}{dt} &= \rho_h I_h(t) - \mu_h T_h(t) - \gamma_h I_h(t) \\
\frac{dS_v(t)}{dt} &= \alpha_v - \frac{\beta_3 S_v(t) I_h(t)}{N_h(t)} - \mu_v S_v(t) \\
\frac{dI_v(t)}{dt} &= \frac{\beta_3 S_v(t) I_h(t)}{N_h(t)} - \mu_v I_v(t)
\end{aligned} \tag{1}$$

In model (1)

α_h is the recruitment rate of human into the population which is assumed to be susceptible.

α_v is the recruitment rate in susceptible vector class.

β_1 is the transmission rate from vector to human.

β_2 is the probability from human to human transmission.

β_3 is transmission rate from human to vector.

μ_h and μ_v are death rate in human and vector classes respectively.

ρ_h is the fraction of infected people who get treatment.

γ_h is the rate at which the infected people become susceptible.

Dimension Formulation:

From system (1), we have, $\frac{dN_h}{dt} = \alpha_h - \mu_h N_h$.

Now, $\frac{dN_h}{dt} = 0$, because $N_h = 1$.

Therefore, $\alpha_h - \mu_h N_h = 0$, that is $\alpha_h = \mu_h N_h$.

Using the above, we transform system (1) and have

$$\begin{aligned}
\frac{ds_h(t)}{dt} &= \mu_h(1 - s_h(t)) - \beta_1 s_h(t) i_v(t) - \beta_2 s_h(t) i_h(t) + \gamma_h i_h(t) \\
\frac{di_h(t)}{dt} &= \beta_1 s_h(t) i_v(t) + \beta_2 s_h(t) i_h(t) - (\mu_h + \rho_h) i_h(t) \\
\frac{dt_h(t)}{dt} &= (\rho_h - \gamma_h) i_h(t) - \mu_h t_h \\
\frac{ds_v(t)}{dt} &= \mu_v(1 - s_v(t)) i_h(t) - \beta_3 s_v(t) i_h(t) \\
\frac{di_v(t)}{dt} &= \beta_3 s_v(t) i_h(t) - \mu_v i_v(t),
\end{aligned} \tag{2}$$

where solutions are restricted to $s_h + i_h + t_h = 1$ and $s_v + i_v = 1$. Before analyzing the normalized model (1), we consider the normalized model (2) by scaling and so we can study the following reduced system that describe the dynamics of the proportion of individual in each class.

$$\begin{aligned}
\frac{ds_h(t)}{dt} &= \mu_h(1 - s_h(t)) - \beta_1 s_h(t) i_v(t) - \beta_2 s_h(t) i_h(t) + \gamma_h i_h(t) \\
\frac{di_h(t)}{dt} &= \beta_1 s_h(t) i_v(t) + \beta_2 s_h(t) i_h(t) - (\mu_h + \rho_h) i_h(t) \\
\frac{di_v(t)}{dt} &= \beta_3(1 - i_v(t)) i_h(t) - \mu_v i_v(t).
\end{aligned} \tag{3}$$

Determining t_h from $s_h + i_h + t_h = 1$ and $s_v + i_v = 1$, respectively. The correlation between normalized and un-normalized model is explained. Throughout this work we study the reduced system (3) in the closed positively invariant set $\Gamma = \{(s_h, i_h, t_h) \in R_+^3, 0 \leq s_h + i_h \leq 1\}$, where R_+^3 denotes the non-negative cone.

Local stability:

We illustrate the local stability of the disease free and the endemic equilibrium of the system (3) by the following theorems 1 and 2.

Theorem 1: The Disease Free Equilibrium E_0 is locally asymptotically stable for $R_0 < 1$, otherwise unstable.

Proof: By linearizing the system (3) about an Endemic equilibrium E_0 we get the Jacobian matrix:

$$J(E_0) = \begin{bmatrix} -\mu_h - \beta_1 i_h - \beta_2 i_h & \beta_2 s_h - \gamma_h & -\beta_1 s_h \\ \beta_1 i_h + \beta_2 i_h & \beta_2 s_h - (\mu_h + \rho_h) & \beta_1 s_h \\ 0 & \beta_3(1 - i_v) & -\beta_3 i_h - \mu_v \end{bmatrix}$$

Now, we have analyze the stability of DFE, and for this we calculate the characteristic equation of $J(E_0)$ as follows.

$$J(E_0) = \begin{bmatrix} -\mu_h & -\beta_2 + \gamma_h & -\beta_1 \\ 0 & \beta_2 - (\mu_h + \rho_h) & \beta_1 \\ 0 & \beta_3 & -\mu_v \end{bmatrix}$$

The characteristic equation of $J(E_0)$ is

$$\begin{aligned} Char(J(E_0)) = det(\lambda I - J(E_0)) &= \begin{vmatrix} \lambda + \mu_h & \beta_2 - \gamma_h & \beta_1 \\ 0 & \lambda - \beta_2 + (\mu_h + \rho_h) & -\beta_1 \\ 0 & -\beta_3 & \lambda + \mu_v \end{vmatrix} \\ &= (\lambda + \mu_h)[\lambda^2 + \lambda(\mu_h + \mu_v + \rho_h - \beta_2) + \mu_v(\mu_h + \rho_h - \beta_2)(1 - R_0)]. \end{aligned}$$

Where

$$R_0 = \frac{\beta_1 \beta_3}{\mu_v(\mu_h + \rho_h - \beta_2)}$$

A little calculation gives that all the eigenvalues are negative only if for $R_0 < 1$. Thus the Disease Free Equilibrium E_0 is locally asymptotically stable for $R_0 < 1$, otherwise unstable.

Endemic Equilibrium:

To find the endemic equilibria of the system (1) where at least one of the infected components is non-zero, we need to take the following steps:

Let $E^* = (s_h^*, i_h^*, i_v^*)$ represents endemic equilibrium of the system (3). By solving the equations of the system (1) at steady state, we get

$$\begin{aligned} s_h^* &= \frac{(\mu_h + \gamma_h i_h^*)(\beta_3 i_h^* + \mu_v)}{(\beta_3 i_h^* + \mu_v)(\mu_h + \beta_3 i_h^*)(\beta_1 \beta_3 i_h^*)}, & t_h^* &= \frac{(\rho_h - \gamma_h) i_h^*}{\mu_h}, \\ s_v^* &= \frac{\mu_v}{(\beta_3 i_h^* + \mu_v)}, & i_v^* &= \frac{\beta_3 i_h^*}{(\beta_3 i_h^* + \mu_v)}. \end{aligned}$$

Let The following theorem analyzes the local stability of the endemic equilibrium when $R_0 > 1$.

Theorem 2: The endemic equilibria E^* is locally asymptotically stable for $R_0 > 1$, $\beta_3 i_h^* + \mu_h > (\mu_h + \rho_h)(\mu_h + \beta_1 i_v^* + \beta_2 i_h^*)$ and $\mu_v(\mu_h + \rho_h - \beta_2) > M$.

Proof: By linearizing the system (3) about an Endemic equilibrium E^* we get the Jacobian matrix:

$$J(E^*) = \begin{bmatrix} -\mu_h - \beta_1 i_v^* - \beta_2 i_h^* & \beta_2 s_h^* - \gamma_h & -\beta_1 s_h^* \\ \beta_1 i_v^* + \beta_2 i_h^* & \beta_2 s_h^* - (\mu_h + \rho_h) & \beta_1 s_h^* \\ 0 & \beta_3(1 - i_v^*) & -\beta_3 i_h^* - \mu_v \end{bmatrix}$$

After some row operations, we get

$$J(E^*) = \begin{bmatrix} -\mu_h - \beta_1 i_v^* - \beta_2 i_h^* & \beta_2 s_h^* - \gamma_h & -\beta_1 s_h^* \\ 0 & -\frac{(\mu_h + \rho_h - \gamma_h)(\mu_h + \beta_1 i_v^* + \beta_2 i_h^*)(\gamma_h - \beta_2 s_h^*)\mu_h}{\mu_h + \beta_1 i_v^* + \beta_2 i_h^*} & \frac{\beta_1 \mu_h s_h^*}{\mu_h + \beta_1 i_v^* + \beta_2 i_h^*} \\ 0 & 0 & -\beta_3 i_h^* - \mu_v + M \end{bmatrix}$$

Where $M = \frac{\beta_1 \beta_2 \mu_h s_h^* (1 - i_v^*)}{(\mu_h + \rho_h - \gamma_h)(\mu_h + \beta_1 i_v^* + \beta_2 i_h^*)(\gamma_h - \beta_2 s_h^*)\mu_h}$.

After some calculations, we see that all the eigen values are negative if $R_0 > 1$, $\beta_3 i_h^* + \mu_h > (\mu_h + \rho_h)(\mu_h + \beta_1 i_v^* + \beta_2 i_h^*)$ and $\mu_v(\mu_h + \rho_h - \beta_2) > M$ and so the endemic equilibria is locally asymptotically stable for these conditions.

Global Dynamics

To show that the system (3) is globally asymptotically stable, we use the Lyapunov function theory for both the disease-free and the endemic equilibrium. First we present the global stability of the disease-free equilibrium.

Theorem 3: For $R_0 < 1$, $\mu_h + \rho_h > \beta_2$, the infected-free equilibrium E_0 is globally asymptotically stable in the interior of Γ .

Proof: To establish the global stability of the disease free equilibrium we construct the following Lyapunov function.

$$L(t) = \mu_v i_h + \beta_1 i_v$$

Calculating the time derivative of "L", we have

$$L'(t) = \mu_v i'_h + \beta_1 i'_v = \mu_v [\beta_1 S_h i_v + \beta_2 S_h i_h - (\mu_h + \rho_h) i_h] + \beta_1 [\beta_3 (1 - i_v) i_h - \mu_v i_v]$$

Putting $S_h = 1 - i_h$, we get

$$\begin{aligned} L'(t) &= \mu_v \beta_1 i_v - \mu_v \beta_1 i_v i_h + \mu_v \beta_2 i_h - \mu_v \beta_2 i_h^2 - \mu_v (\mu_h + \rho_h) i_h + \beta_1 \beta_3 i_h - \beta_1 \beta_3 i_h i_v - \beta_1 \mu_v i_v \\ &= -\mu_v \beta_1 i_v i_h - \mu_v \beta_2 i_h^2 - \beta_1 \beta_3 i_h i_v - \mu_v (\mu_h + \rho_h - \beta_2) \left[1 - \frac{\beta_1 \beta_3}{\mu_v (\mu_h + \rho_h - \beta_2)} \right] i_h \\ &= -\mu_v \beta_1 i_v i_h - \mu_v \beta_2 i_h^2 - \beta_1 \beta_3 i_h i_v - \mu_v (\mu_h + \rho_h - \beta_2) [1 - R_0] i_h, \end{aligned}$$

where

$$R_0 = \frac{\beta_1 \beta_3}{\mu_v (\mu_h + \rho_h - \beta_2)}$$

Here the time derivative of Lyapunov function is negative if $R_0 < 1$ and $L'(t) = 0$ if and only if $i_v = i_h = 0$. Consequently the largest compact invariant set in $\{(s_h, i_h, i_v) \in \Gamma, L'(t) = 0\}$ when $R_0 < 1$ is the singleton set E_0 . Hence E_0 is globally asymptotically stable.

Theorem 4: The endemic equilibrium E^* is globally asymptotically stable in the interior of Γ when $\mu_h = \frac{\rho_h i_h^* - \gamma_h i_h^*}{(1 - s_h - i_h)}$ and $\gamma_h < \rho_h$.

Proof: To establish the global stability of endemic equilibrium we construct the following Lyapunov function

$$L(t) = i_h(t) + s_h(t)$$

Calculating the time derivative of "L"

$$\begin{aligned} L'(t) &= i'_h(t) + s'_h(t) \\ &= \beta_1 s_h i_v + \beta_2 s_h i_h - (\mu_h + \rho_h) i_h + \mu_h (1 - s_h) \beta_1 s_h i_v + \beta_2 s_h i_h + \gamma_h i_h \\ &= \mu_h (1 - s_h - i_h) - (\rho_h - \gamma_h) i_h \end{aligned}$$

using $\mu_h = \frac{\rho_h i_h^* - \gamma_h i_h^*}{(1 - s_h - i_h)}$, we get

$$\begin{aligned} L'(t) &= \frac{\rho_h i_h^* - \gamma_h i_h^*}{(1 - s_h - i_h)} (1 - s_h - i_h) - (\rho_h - \gamma_h) i_h \\ &= (\rho_h - \gamma_h) (i_h - i_h^*) \\ &< 0 \end{aligned}$$

Thus the time derivative of Lyapunov function is negative and $L'(t) = 0$ for $i_h = i_h^*$. Hence by Lassalle's invariance principal E^* is globally asymptotically stable on Γ .

Numerical Results

In the given section, we investigate the numerical solution of the model (3) by choosing the base line for susceptible human $S_h=800$, infected human $I_h=100$ and infected vector $I_v=100$. The parameter and their values used are given as

$\beta_1 = 0.000078$, $\beta_2 = 0.004$, $\beta_3 = 0.002$, $\gamma_h = 0.002$, $\mu_v = 0.002$, $\rho_h = 0.0002$, and $\mu_h = 0.002$.

Figure 1, shows the dynamical behavior of the model.

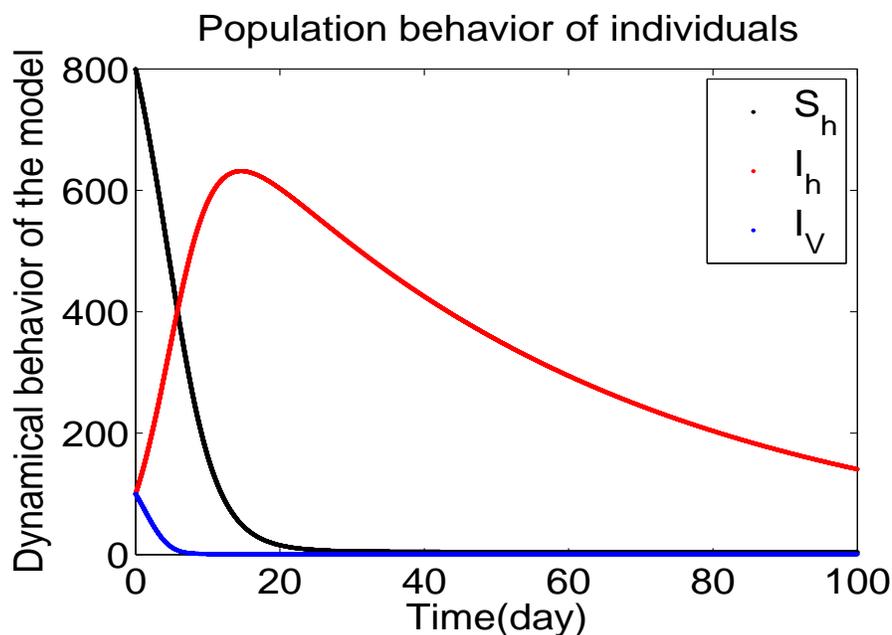


Figure 1: The dynamical behavior of the model.

Conclusion

In this work, we discussed the compartmental vector-borne disease model. As in epidemiological models, our model has two steady states, an uninfected steady state and endemically infected steady state. By establishing the stability results we found both the disease-free and the endemic equilibria. We also presented that for $R_0 < 1$, the locally asymptotically stable disease-free equilibrium of the proposed model co-exists with a locally asymptotically stable endemic equilibrium. Then to present the global stability of both the disease-free and endemic states, we developed Lyapunov functions. We believe that this new analysis is helpful in study of vector-borne diseases.

REFERENCES

- [1] H. Yang, H. Wei, X. Li; "Global stability of an epidemic model for vector-borne disease" *Journal of Systems Science & Complexity*, 2010 (23) 279--292.
- [2] Z. Feng, J. Velasco-Hernández; "Competitive exclusion in a vector-host model for the dengue fever" *Journal of Mathematical Biology*, 1997 (35) 523--544.
- [3] Z. Qiu; "Dynamical behavior of a vector-host epidemic model with demographic structure" *Computers & Mathematics with Applications*, 2008. vol.(56), no. 12, 3118--3129.
- [4] V. Wiwanitkit; "Unusual mode of transmission of dengue," *The Journal of Infection in Developing Countries*, 2009 (30) 51--54,
- [5] M. H. Wei Li, M. Martcheva; "An epidemic model of a vector-borne disease with direct transmission and time delay" *Journal of Mathematical Analysis and Applications*, 2008, (342), 2, 895--908.
- [6] L. Cai, X. Li; "Analysis of a simple vector-host epidemic model with direct transmission" *Discrete Dynamics in Nature and Society*, 2010.
- [7] A. A. Lashari, G. Zaman; "Global dynamics of vector-borne diseases with horizontal transmission in host population," *Computers & Mathematics with Applications*, 2011 (61), 4, 745-754.

- [8] M. de la Sen, R. P. Agarwal, A. Ibeas, S. Alonso-Quesada; "On the existence of equilibrium points, boundedness, oscillating behavior and positivity of a SVEIRS epidemic model under constant and impulsive vaccination," *Advances in Difference Equations* 2011 (2011), 32.
- [9] M. Sen, S. Alonso-Quesada; "Vaccination strategies based on feedback control techniques for a general SEIR-epidemic model," *Applied Mathematics and Computation*, 2011 (218), 7, 3888--3904.
- [10] T. Zhang, J. Liu, Z. Teng; "Dynamic behavior for a nonautonomous SIRS epidemic model with distributed delays," *Applied Mathematics and Computation*, 2009, (214), 2, 624--631.
- [11] J. L. Aron, R. M. May; *The Population Dynamics of Infectious Diseases*, Chapman & Hall, London, UK, 1982.
- [12] H. W. Hethcote; "The mathematics of infectious diseases," *SIAM Review*, 2000. (42), 4, 599--653.
- [13] G. A. Ngwa, W. S. Shu; "A mathematical model for endemic malaria with variable human and mosquito populations," *Mathematical and Computer Modelling*, 2000 (32), 7-8, 747--763.
- [14] L. Esteva, C. Vargas; "A model for dengue disease with variable human population," *Journal of Mathematical Biology*, 1999 (38), 3, 220--240.
- [15] O. Ozair, A. A. Lashari, I. H. Jung, K. O. Okosun, "Stability analysis and optimal control of a vector-borne disease with nonlinear incidence," *Discrete Dynamics in Nature and Society*, 2012 (2012), 21.
- [16] J. P. LaSalle; *The Stability of Dynamical Systems*, Regional Conference Series in Applied Mathematics, SIAM, Philadelphia, Pa, USA, 1976.
- [17] M. Li, J. S. Muldowney; "A geometric approach to global-stability problems," *SIAM Journal on Mathematical Analysis*, 1996 (27), 4, 1070--1083.
- [18] R. Ullah, G. Zaman, S. Islam, Prevention of influenza Pandemic by Multiple control strategies, *J. Appl. Math.*, vol 2012, Article ID 294275, 14 pages, 2012. doi:10.1155/2012/294275.
- [19] R. Ullah, G. Zaman, S. Islam, Multiple Control Strategies for Prevention of Avian Influenza Pandemic, *The Scientific World Journal*, Volume 2014, Article ID 949718, 9 pages, <http://dx.doi.org/10.1155/2014/949718>.
- [20] M. A. Khan, S. Islam, S. A. Khan, I. Khan, S. Shafie, T. Gul, Prevention of Leptospirosis Infected Vector and Human Population by Multiple Control Variables, *Abstract and Applied Analysis*, Volume 2014 (2014), Article ID 619035, 9 pages <http://dx.doi.org/10.1155/2014/619035>.
- [21] R. Ullah, G. Zaman, S. Islam, Muhammad Raheel Muhyuddin, Global dynamics of the avian-human influenza with horizontal transmission in human population, *Life Science Journal*, 9 (4) (2012) 5747-5753.
- [22] R. Ullah, G. Zaman, S. Islam, I. Ahmad, Dynamical features and vaccination strategies in an SEIR epidemic model, *Research Journal of Recent Sciences*, 2 (10) (2013) 48-56.
- [23] X. Q. Zhao; *Dynamical Systems in Population Biology*, vol. 16 of CMS Books in Mathematics, Springer, New York, NY, USA, 2003.
- [24] G. Butler, H. I. Freedman, P. Waltman; "Uniformly persistent systems," *Proceedings of the American Mathematical Society*, 1986 (96), 3, 425--430.