# Local Stability Analysis of a River Blindness Disease Model with Control

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# ABSTRACT

In this paper, a mathematical model to study the dynamics of River Blindness is presented. The existence and uniqueness of solutions of the model were examined by actual solution. The effective reproduction number was obtained using the next generation matrix. The Disease Free Equilibrium (DFE) State was obtained and analysed for stability. It was found that, the DFE State is Locally Asymptotically Stable (LAS) if the effective reproduction number  $R_0 < 1$  and

unstable if  $R_0 > 1$ .

(Keywords: basic reproduction number; disease free equilibrium; river blindness; stability)

# INTRODUCTION

River blindness is a parasitic disease that causes loss of vision and it is the second largest cause of blindness after trachoma. It mainly affects the eyes and the skin. River blindness is caused by the parasitic filarial nematode *Onchocera volulus*and and is transmitted by the *Simulium* black flies (CDC, 2013; WHO 1995). The blackfly usually lays its eggs attached to rocks and vegetation submerged in fast flowing and highly oxygenated rivers and streams where the larva and pupa stage develop before transforming to the adult stage. This causes high occurrence of the disease in villages located along riverine areas where blackflies breed (WHO, 1995).

Despite copious strategies employed by different national and international organizations to eliminate the disease, recent examination data from the World Health Organization (WHO) and Onchocerciasis Control Programme (OCP) show that more than 17.7 million are infected globally. River blindness is also prevalent in Western and Central Africa and also in the Central and Southern America. Most of the cases of blindness caused by river blindness are found in the sub-Sahara Africa (outer part of the areas covered by OCP (WHO, 1995).

Although river blindness is not a disease that leads to death, there is however a clear indication that blindness causes social and economic complications which may lead to death because of vision loss. It has been estimated that the average lifespan at the onset of blindness is between 7-9 years (Prost, 1986) and that blindness related excess-mortality varies between 3.3 in trachoma endemic areas (Taylor et al, 1991) and 4 times (Prost and Vaugelade, 1981) the mortality of non-blind persons.

River blindness is prevalent in tropical Africa and parts of tropical America (Basanez, 2001). Most recent estimates by WHO mention more than 17.7 million infected, 500,000 visually impaired, and another 270,000 blind. About 99% of infected persons are in Africa and 11% in Nigeria and it is more prevalent in Mubi village in Adamawa State where about 89% of the entire village suffers from different forms of blindness. The hyperendemic has earned the village the name "Village of the Blind". This occurrence is also found in Yemen and a few other countries in Southern America. River blindness is locally transmitted in thirty countries in Africa. 13 foci in America and in Yemen. In West Africa, the fear of the infection is one of the causes of migration from fertile riverine areas to sub-marginal lands, which results in over cultivation and low productivity (Basanez, et al., 2006).

The infection is transmitted from human to human by the black fly. Humans are the host of *Onchocera volulus*. When a black fly bites a person who has river blindness, microscopic worms (microfilariae) in an infected person's skin are then transmitted among persons in the population. Fertilized female worms which lived in subcutaneous human tissue produce microfilariae which are picked up from the skin by the black fly during its blood meals. Some of these microfilariae when picked die and others are digested together with blood meals, while a few that are successful in penetrating the wall of the black fly's stomach settle in the thoracic muscle (chest). After 7 days, the microfilariae has passed through the three larva stages, they become free larva capable of infecting the human host (Galadima, 1982). Only female black flies transmit Onchocera volvulus and the transmission time is usually between 6:00 am and 11:00 am. Most microfilariae do not reach the black fly and instead live in the human body thereby aggravating and inflammatory reaction due to the human's immune system reacting around dead microfilariae.

Blindness is caused by а progressive accumulation of this process when microfilariae invade the eye tissues, which leads to irreversible ocular lesions resulting first in visual impairment and eventually, total blindness (Hugo, 2013; Remme, 2006).

As mathematics is growing, the need for more realistic ways of communicating mathematical ideas is increasingly becoming more important. It is a known fact that mathematical models are used in comparing, planning, implementing, evaluating and optimizing various detection, prevention, therapy, and control programs. More so, model may help explain a system and to study the effects of different components and also to make predictions. The predictions are mainly done through the use of models or conceptual scheme of the test of time through criticism and skeptical appraisal which are integral parts of the scientific process. It is assumed that there is no new process under the sun, but man's various models have allowed him to transform his corner of the universe (Bolarin, 2011).

#### MATERIALS AND METHODS

#### **Model Formulation**

A mathematical model for the dynamics of transmission and control of river blindness was developed. improving on existing models assuming that after being treated, the individual goes back to the susceptible class.

The model consists of the human and the vector (Black fly) population. The human population is

divided into three (3) compartments namely: the human  $S_{k}(t)$ , susceptible the infected human  $I_{\mu}(t)$ , and the treated human  $T_{\mu}(t)$ .

The vector population is divided into susceptible vector  $S_V(t)$  and infected vector  $I_V(t)$  Suppose that those infectious individuals are treated with Ivermectin/Mectizan drugs bear in mind that the treatment is partially effective. We have the following set of equations:

$$\frac{dS_h}{dt} = \Lambda_h - \frac{\beta_h I_s S_h}{N_h} + \delta T_h - \mu_h S_h \tag{1}$$

$$\frac{dI_h}{dt} = \frac{\beta_h I_s S_h}{N_h} - (\gamma + \mu_h) I_h$$
<sup>(2)</sup>

$$\frac{dT_h}{dt} = \gamma I_h - (\delta + \mu_h) T_h \tag{3}$$

$$\frac{dS_V}{dt} = \Lambda_V - \frac{\beta_V I_h S_V}{N_V} - (\sigma + \mu_V) S_V$$
(4)

$$\frac{dI_V}{dt} = \frac{\beta_V I_h S_V}{N_V} - (\sigma + \mu_V) I_V$$
(5)

Where,

$$N_{h} = S_{h} + I_{h} + T_{h}$$

$$N_{v} = S_{v} + I_{v}$$

$$(6)$$

As initial condition based on our assumptions, we choose:

$$S_{h}(0) = S_{h0}, I_{h}(0) = I_{h0}, T_{h}(0) = T_{h0}, S_{v}(0) = S_{v0}, I_{v}(0) = I_{v0}$$
(7)

#### Table 1: Variables of the Model

Symbols	Description
$S_h(t)$	Number of susceptible humans at time $t$
$I_h(t)$	Number of infectious humans at time $t$
$T_h(t)$	Number of treated humans at time t
$S_v(t)$	Number of non-carrier vectors at time t
$I_V(t)$	Number of carrier vectors at time t
$N_h(t)$	Total human population at time t
$N_v(t)$	Total vector population at time t

Table 2: Parameters of the Model.

Symbols	Description
$\Lambda_h$	Recruitment rate of human population
$\Lambda_{V}$	Recruitment rate of vector population
$\beta_h$	Contact rate of susceptible humans acquired
$\beta_{v}$	Contact rate of non-carrier vectors acquired
	infection with infected human
δ	Rate at which the recovery human move to
	susceptible human class
γ	Treatment rate of humans.
σ	Death of vector by insecticide
$\mu_h$	Natural death of human population
$\mu_{v}$	Natural death of vector population

## **Positivity of Solutions**

It is necessary to prove that all solutions of system (1) - (5) with positive initial data will remain positive for all times (t). This will be established by the following theorem.

Lemma 1: Let the closed set

$$\begin{bmatrix} S_h \\ I_h \\ T_h \\ S_v \\ I_v \end{bmatrix} \in \mathfrak{R}^5 \begin{array}{c} S_h(0) \ge 0 \\ I_h(0) \ge 0 \\ T_h(0) \ge 0 \\ I_v(0) \ge 0 \\ S_h + I_h + T_h \le \frac{\Lambda_h}{\mu_h} \\ S_V + I_V \le \frac{\Lambda_V}{\sigma + \mu_V} \end{bmatrix}$$

Then the solutions:

 $\{S_h(t), I_h(t), T_h(t), S_V(t), I_V(t)\} \text{ of the system}$ (1) – (5) are positive for all  $t \ge 0$ .

**Proof:** From Equation (1) we have:

$$\frac{dS_h}{dt} = \Lambda_h - \frac{\beta_h I_s S_h}{N_h} + \delta T_h - \mu_h S_h$$
$$\frac{dS_h}{dt} \ge -\mu_h S_h$$
$$\frac{dS_h}{S_h} \ge -\mu_h dt$$
$$\int_0^t \frac{1}{S_h} dS_h \ge -\mu_h \int_0^t dx$$
$$S_h(t) \ge S_h(0) e^{-\mu_h t} \ge 0$$
Similarly,

$$I_{h}(t) \ge I_{h}(0)e^{-(\gamma+\mu_{h})t} \ge 0$$
$$T_{h}(t) \ge T_{h}(0)e^{-(\delta+\mu_{h})t} \ge 0$$
$$S_{y}(t) \ge S_{y}(0)e^{-(\sigma+\mu_{y})t} \ge 0$$

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$$I_{v}(t) \ge I_{v}(0)e^{-(\sigma+\mu_{v})t} \ge 0$$

Hence, the solution of  $\{S_h(t), I_h(t), T_h(t), S_V(t), I_V(t)\}$  of the system (1) – (5) are positive for all  $t \ge 0$ 

## **Existence and Uniqueness of Solution**

**Theorem 1**: Let the Equations (1) - (5) with initial conditions (7) has a unique Solution for all  $t \ge 0$ .

**Proof:** Let 
$$\phi(t) = S_h(t) + I_h(t) + T_h(t)$$
 and  $\psi(t) = S_V(t) + I_V(t)$ 

We obtain:

$$\frac{d\phi}{dt} = \Lambda_h - \mu_h \phi, \phi(0) = S_{h(0)} + I_{h(0)} + T_{h(0)} = \phi_0$$

$$\frac{d\psi}{dt} = \Lambda_V - (\sigma + \mu_V)\psi,$$
  
$$\psi(0) = S_{V(0)} + I_{V(0)} = \psi_0$$
(9)

By direct integration, we obtain the solution of Problem (8) and (9) as:

$$\phi(t) = \frac{\Lambda_h}{\mu_h} + \left(\phi_0 - \frac{\Lambda_h}{\mu_h}\right) e^{-\mu_h t}$$
(10)

$$\psi(t) = \frac{\Lambda_V}{(\sigma + \mu_V)} + \left(\psi_0 - \frac{\Lambda_V}{(\sigma + \mu_V)}\right) e^{-(\sigma + \mu_V)t}$$
(11)

Then, we obtain:

$$S_{h}(t) = \left[\frac{\Lambda_{h}}{\mu_{h}}(1 - e^{-\mu_{h}t}) + \phi_{0}e^{-\mu_{h}t}\right] - (I_{h}(t) + T_{h}(t))$$
(12)

$$I_{h}(t) = \left[\frac{\Lambda_{h}}{\mu_{h}}(1 - e^{-\mu_{h}t}) + \phi_{0}e^{-\mu_{h}t}\right] - (S_{h}(t) + T_{h}(t))$$
(13)

$$T_{h}(t) = \left[\frac{\Lambda_{h}}{\mu_{h}}(1 - e^{-\mu_{h}t}) + \phi_{0}e^{-\mu_{h}t}\right] - (S_{h}(t) + I_{h}(t))$$
(14)

$$S_{V}(t) = \left[\frac{\Lambda_{V}}{(\sigma + \mu_{V})} \left(1 - e^{-(\sigma + \mu_{V})t}\right) + \psi e^{-(\sigma + \mu_{V})t}\right] - \left(I_{V}(t)\right)$$
(15)

$$I_{V}(t) = \left[\frac{\Lambda_{V}}{(\sigma + \mu_{V})} \left(1 - e^{-(\sigma + \mu_{V})t}\right) + \psi e^{-(\sigma + \mu_{V})t}\right] - (S_{v}(t)) \quad (8) \quad (3.12)(16)$$

Hence, the model equation (1) to (5) has a unique solution.

#### **Steady State Solutions**

At equilibrium:

$$\frac{dS_{h}}{dt} = \frac{dI_{h}}{dt} = \frac{dT_{h}}{dt} = \frac{dS_{v}}{dt} = \frac{dI_{v}}{dt} = 0$$
(17)
(3.14)

And at any arbitrary equilibrium state, let:

$$E^{*} = \begin{bmatrix} S_{h} \\ I_{h} \\ T_{h} \\ S_{V} \\ I_{V} \end{bmatrix} = \begin{bmatrix} S_{h}^{0} \\ I_{h}^{0} \\ T_{h}^{0} \\ S_{V}^{0} \\ I_{V}^{0} \end{bmatrix}$$
(18)

The model analysis above yields the disease free equilibrium point given by:

$$\begin{bmatrix} S_{h} \\ I_{h} \\ T_{h} \\ S_{V} \\ I_{V} \end{bmatrix} = \begin{bmatrix} \frac{\Lambda_{h}}{\mu_{h}} \\ 0 \\ 0 \\ \frac{\Lambda_{V}}{(\sigma + \mu_{V})} \\ 0 \end{bmatrix}$$
(19)

#### **Basic Reproduction Number,** $R_0$

Applying next generation matrix operator to compute the Basic Reproduction Number of the model as used by (Diekmann, *et al.*, 1990) and improved by Driessche, (2002). The basic reproduction number is the largest eigenvalue or spectral radius of  $FV^{-1}$ .

$$FV^{-1} = \left[\frac{\partial F_i(E^0)}{\partial x_i}\right] \left[\frac{\partial V_i(E^0)}{\partial x_i}\right]^{-1}$$
(20)

$$F = \begin{pmatrix} 0 & 0 & \frac{\beta_h S_h}{\mu_h} \\ 0 & 0 & 0 \\ \frac{\beta_V S_V}{\mu_V} & 0 & 0 \\ \mu_V & & & \end{pmatrix}$$
(21)

and

$$V = \begin{pmatrix} \left(\gamma + \mu_h\right) & 0 & 0\\ -\gamma & \left(\delta + \mu_h\right) & 0\\ 0 & 0 & \left(\sigma + \mu_V\right) \end{pmatrix}$$
(22)

$$V^{-1} = \begin{pmatrix} \frac{1}{\gamma + \mu_h} & 0 & 0\\ \frac{\gamma}{(\gamma + \mu_h)(\delta + \mu_h)} & \frac{1}{\delta + \mu_h} & 0\\ 0 & 0 & \frac{1}{\delta + \mu_V} \end{pmatrix} (23)$$

$$FV^{-1} = \begin{pmatrix} 0 & 0 & \frac{\beta_h S_h}{(\sigma + \mu_V)} \\ 0 & 0 & 0 \\ \frac{\beta_V S_V}{(\gamma + \mu_h) \mu_V} & 0 & 0 \end{pmatrix}$$
(24)

The effective reproduction number of the model is the dominant eigenvalue or spectral radius of the matrix  $FV^{-1}$ , thus:

$$R_0 = \sqrt{\frac{\beta_h \beta_V}{(\sigma + \mu_V)(\gamma + \mu_h)}}$$
(25)

Equation (25) is the Basic Reproduction Number of the model.

#### **RESULTS AND DISCUSSION**

# **Local Stability of Disease Free Equilibrium** $E^0$

**Theorem 2:** The disease free equilibrium  $E^{\circ}$  of the model Equations (1) – (5) is locally asymptotically stable (LAS) if  $R_0 < 1$  and unstable if  $R_0 > 1$ 

**Proof:** Linearization of the model Equations (1) – (5) at any arbitrary equilibrium point ( $E^*$ ) gives the Jacobian.

$$J(E^{*}) = \begin{bmatrix} -\left(\frac{\beta_{k}I_{V}}{\mu_{h}} + \mu_{h}\right) & 0 & \delta & 0 & \frac{-\beta_{k}S_{h}}{\mu_{h}} \\ \frac{\beta_{k}I_{V}}{\mu_{h}} & -(\gamma + \mu_{h}) & 0 & 0 & \frac{\beta_{k}S_{h}}{\mu_{h}} \\ 0 & \gamma & -(\delta + \mu_{h}) & 0 & 0 \\ 0 & \frac{-\beta_{V}S_{V}}{\mu_{V}} & 0 & -\left(\frac{\beta_{V}I_{h}}{\mu_{V}} + (\sigma + \mu_{V})\right) & 0 \\ 0 & \frac{\beta_{V}S_{V}}{\mu_{V}} & 0 & \frac{\beta_{V}I_{h}}{\mu_{V}} & -(\sigma + \mu_{V}) \end{bmatrix}$$
(26)

We evaluate the Jacobian at the disease free equilibrium to determine the local stability of the system. We obtain:

$$J(E^{\circ}) = \begin{bmatrix} -\mu_{h} & 0 & \delta & 0 & -\frac{\beta_{h}S_{h}}{\mu_{h}} \\ 0 & -(\gamma + \mu_{h}) & 0 & 0 & \frac{\beta_{h}S_{h}}{\mu_{h}} \\ 0 & \gamma & -(\delta + \mu_{h}) & 0 & 0 \\ 0 & -\frac{\beta_{V}S_{V}}{\mu_{V}} & 0 & -(\sigma + \mu_{V}) & 0 \\ 0 & \frac{\beta_{V}S_{V}}{\mu_{V}} & 0 & 0 & -(\sigma + \mu_{V}) \end{bmatrix}$$
(27)

Using elementary row transformation, the matrix (27) becomes:

$$J(E^{\circ}) = \begin{bmatrix} -\mu_{h} & 0 & \delta & 0 & -\frac{\beta_{h}S_{h}}{\mu_{h}} \\ 0 & -(\gamma + \mu_{h}) & 0 & 0 & \frac{\beta_{h}S_{h}}{\mu_{h}} \\ 0 & 0 & -(\delta + \mu_{h}) & 0 & \frac{\beta_{h}S_{h}}{(\gamma + \mu_{h})\mu_{h}} \\ 0 & 0 & 0 & -(\sigma + \mu_{v}) & -\frac{\beta_{v}S_{h}\beta_{h}S_{v}}{(\gamma + \mu_{h})\mu_{h}\mu_{v}} \\ 0 & 0 & 0 & 0 & -(\sigma + \mu_{v}) & -\frac{\beta_{v}S_{h}\beta_{h}S_{v}}{(\gamma + \mu_{h})\mu_{h}\mu_{v}} \end{bmatrix}$$
(28)

where,

$$A = -\frac{(\sigma\gamma + \sigma\mu_h + \gamma\mu_v + \mu_h\mu_v)N_hN_v + \beta_h\beta_vS_hS_v}{(\gamma + \mu_h)N_hN_v}$$

The characteristics equation of the upper triangular Jacobian is:

$$J(E^{*}) = \begin{bmatrix} -(\mu_{h} + \lambda) & 0 & 0 & 0 & -\frac{\beta_{h}S_{h}}{N_{h}} \\ 0 & -(\gamma + \mu_{h} + \lambda) & 0 & 0 & \frac{\beta_{h}S_{h}}{N_{h}} \\ 0 & 0 & -(\delta + \mu_{h} + \lambda) & 0 & \frac{\beta_{h}\gamma S_{h}}{(\gamma + \mu_{h})N_{h}} \\ 0 & 0 & 0 & -(\sigma + \mu_{v} + \lambda) & -\frac{\beta_{h}\beta_{v}S_{h}S_{v}}{(\gamma + \mu_{h})N_{h}N_{v}} \\ 0 & 0 & 0 & 0 & (A - \lambda) \end{bmatrix}$$
(29)

Therefore, the eigenvalues are,

$$\lambda_1 = \mu_h < 0 \tag{30}$$

$$\lambda_2 = -(\gamma + \mu_h) < 0 \tag{31}$$

$$\lambda_3 = -(\delta + \mu_h) < 0 \tag{32}$$

$$\lambda_4 = -(\sigma + \mu_\nu) < 0 \tag{33}$$

$$\lambda_{5} = A = \frac{\left(\sigma\gamma + \sigma\mu_{h} + \gamma\mu_{v} + \mu_{h}\mu_{v}\right)N_{h}N_{v} - \beta_{h}\beta_{v}S_{h}S_{v}}{(\gamma + \mu_{h})N_{h}N_{v}}$$

For  $\lambda_5$  to be negative, then,

$$\frac{-(\sigma\gamma + \sigma\mu_h + \gamma\mu_v + \mu_h\mu_v)N_hN_v + \beta_b\beta_vS_hS_v}{(\gamma + \mu_h)N_hN_v} < 0$$

$$\frac{\beta_{h}\beta_{v}S_{h}S_{v}}{(\sigma\gamma + \sigma\mu_{h} + \gamma\mu_{v} + \mu_{h}\mu_{v})N_{h}N_{v}} < 1$$

$$\frac{\beta_{h}\beta_{v}}{(\sigma + \mu_{v})(\gamma + \mu_{h})} < 1$$

$$R_{\circ} < 1$$
(34)

This implies that  $\lambda_5 < 0$  if  $R_c < 1$ , hence, the disease free equilibrium  $E^{\circ}$  of the equation is locally asymptotically stable (LAS) if  $R_c < 1$ .

#### CONCLUSION

A mathematical model to study the dynamics of River Blindness is presented and we determined the conditions for existence and stability of equilibrium states characterized in terms of the effective reproduction number. The study showed that there is a disease free equilibrium which is locally asymptotically stable if  $R_0 < 1$  and unstable if otherwise.

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