STABILITY ANALYSIS OF THE DISEASE-FREE EQUILIBRIUM STATE FOR YELLOW FEVER DISEASE

M. Bawa[1], S. Abdulrahman[2*], S. Abubakar[2] and Y.B. Aliyu[1]

[1]Department of Mathematics, Ibrahim Badamasi Babangida University, Lapai, Niger State, Nigeria
[2]Department of Mathematics and Statistics, Federal University of Technology, Minna, Niger State, Nigeria
*Corresponding E-mail.: sirajo.abdul@futminna.edu.ng

Abstract

In this paper, we developed and analysed the disease-free equilibrium state of a new mathematical model for the dynamics of yellow fever infection in a population with vital dynamics, incorporating vaccination as control measure. We obtained the effective basic reproduction number, R_c which can be used to control the transmission of the disease and hence, established the conditions for local and global stability of the disease free equilibrium.

Keywords: Yellow fever, Disease-free equilibrium state, Effective basic reproduction number, Stability.

1.0. Introduction

Yellow Fever, non contagious, infectious disease, caused by a virus, and characterized in severe cases by high fever and jaundice. Originally yellow fever was believed to be exclusively a disease of humans, but research has revealed that it also affects monkeys and other animals. It is believed that diseased monkeys of Africa and tropical America are the primary source of infection and that carrier mosquitoes transmit the infection to humans. This type of the disease, which occurs only sporadically in human beings, is known as jungle yellow fever. If infected individuals move into a populated area, they may be bitten by a semidomestic species of mosquito, such as *Aëdes aegypti,* which lives close to human habitations. These feed on the blood of humans and are the chief transmitting agents in epidemics of urban yellow fever. The World Health Organization (WHO) estimates that there are 200,000 cases of yellow fever worldwide each year, causing about 30,000 deaths annually.

In order to find an efficient way to control an infection, it is of great importance to establish its transmission dynamics. One main goal of mathematical epidemiology is to understand how to control and eradicate diseases (Ma and Ma, 2006). Mathematical models are used extensively in the study of ecological and epidemiological phenomena (Kaplan and Brandeau, 1994). They are particularly helpful as experimental tools with which to evaluate and compare control procedures and preventive strategies, and to investigate the relative effects of various sociological, biological and environmental factors on the spread of diseases. This is so because they can help in figuring out decisions that are of significance importance on the outcomes and provide comprehensive examinations that enter into decisions in a way that human reasoning and debate cannot.

Even though yellow fever is endemic in Africa, mathematicians have not taken their time to study the disease dynamics. Except for the work of Akinwande (1995) and Akinwande (1996), to the best of our knowledge no work has been published on the disease. In this work, we therefore complement and extend the work of Akinwande (1995) and Akinwande (1996) by incorporating vital dynamics, immunization, standard incidence, and disease induced death due to yellow fever infection.

Model Formulation

Model variables and parameters

divided the human population into 4 compartments and the vector population into 2 mpartments as described below:

(t) Susceptible humans at time t

(t) Vaccinated humans at time t

(t) Infected humans at time t

(t) Recovered humans at time t

(t) Susceptible vectors at time t

(t) Infected vectors at time t

$N_H(t)$ Total number of human population at time t

$N_V(t)$ Total number of vector population at time t

And the model has the following parameters.

b_H Per capital birth rate of humans

b_V Per capital birth rate of the vectors

μ_H Per capital natural death rate of humans

μ_V Per capital natural death rate of humans

$j = \frac{N_V}{N_H}$ Number of vector per human host

δ Yellow fever-induced death rate

β_H Effective contact rate for humans

β_V Effective contact rate for vectors

ε Vaccine efficacy

c Immunization coverage rate for S and therefore $\rho = \varepsilon c$ is the effective immunization rate for S

ω Loss (waning) of vaccine immunity

σ_S Rate of moving from S_U to S_F

σ_A Rate of moving from acutely infected classes to chronically infected / removed classes

γ Rate of recovery from I to R

2.2. Model equations

The model equation equations are given below

$$\frac{dS}{dt} = b_H N_H - \frac{\beta_H I_V}{N_H} S + \omega V - (\rho + \mu_H) S \qquad (1)$$

$$\frac{dI}{dt} = \frac{\beta_H I_V}{N_H} S - (\gamma + \mu_H + \delta) I \qquad (2)$$

$$\frac{dV}{dt} = \rho S - (\omega + \mu_H) V \qquad (3)$$

$$\frac{dR}{dt} = \gamma I - \mu_H R \tag{4}$$

$$\frac{dS_V}{dt} = b_V N_V - \frac{\beta_V I}{N_H} S_V - \mu_V S_V \tag{5}$$

$$\frac{dI_V}{dt} = \frac{\beta_V I}{N_H} S_V - \mu_V I_V \tag{6}$$

where,

$$N_H(t) = S(t) + I(t) + V(t) + R(t) \tag{7}$$

and

$$N_V(t) = S_V(t) + I_V(t) \tag{8}$$

So that

$$\frac{dN_H}{dt} = (b_H - \mu_H) N_H - \delta I \tag{9}$$

and

$$\frac{dN_V}{dt} = (b_V - \mu_V) N_V \tag{10}$$

Consider equations (1 – 6) for the normalised quantities. Since, it is better and easier (convenient) to analyze our model in terms of proportions of quantities instead of actual populations as described in Busenberg et al. (1990), Akinwande (1996), Li et al. (1999), Hethcote (2000), Tumwiine et al. (2007), Capasso (2008) and Benyah (2008). This can be done by scaling the population of each class by the total populations N. We let

$$s = \frac{S}{N_H}, i = \frac{I}{N_H}, v = \frac{V}{N_H}, r = \frac{R}{N_H}, s_v = \frac{S_V}{N_V}, i_v = \frac{I_V}{N_V}$$

denote the fractions of the classes S, I, V, R, S_V and I_V in the human and vector population respectively. This is done by differentiating the fractions (using quotient rule) with respect to time, t.

then simplifying, we have from (1 – 10)

$$\frac{ds}{dt} = b_H - \beta_H j i_v s + \omega v - (\rho + b_H) s + \delta s i \tag{11}$$

$$\frac{di}{dt} = \beta_H j i_v s - (\gamma + \mu_H + \delta) i + \delta i^2 \tag{12}$$

$$\frac{dv}{dt} = \rho s - (\omega + b_H) v + \delta v i \tag{13}$$

$$\frac{dr}{dt} = \gamma i - b_H r + \delta r i \tag{14}$$

$$\frac{ds_v}{dt} = b_V - \beta_V i s_v - b_V s_v \tag{15}$$

$$\frac{di_v}{dt} = \beta_V i s_v - b_V i_v \tag{16}$$

in the biological - feasible region:

$$\Omega = \left\{ \begin{matrix} (s,i,v,r,s_v,i_v) \in \Re_+^6 : 0 \le s, 0 \le i, 0 \le v, 0 \le r, 0 \le s_v, 0 \le i_v \\ s+i+v+r=1, s_v+i_v=1 \end{matrix} \right\} \qquad (17)$$

This can be shown to be positively invariant with respect to the system (11)–(16). We note that the total human and vector population size N_H and N_V does not appear in (11)–(16); this is as a direct result of the homogeneity of the equations in (1)–(6).

3.0. Model Analysis

We now determine the existence of equilibria points; computing the effective basic reproduction number; and establishing the conditions for stability of the equilibria points.

3.1. Existence of disease free equilibrium state, E_f

At the disease free equilibrium state we have absence of infection. Thus, all the infected classes will be zero and the entire population will comprise of susceptible.
At equilibrium state the rate of change of each variable is equal to zero. i.e.

$$\frac{ds}{dt} = \frac{di}{dt} = \frac{dv}{dt} = \frac{dr}{dt} = \frac{ds_v}{dt} = \frac{di_v}{dt} = 0$$

let $(s,i,v,r,s_v,i_v) = (s^*,i^*,v^*,r^*,s_v^*,i_v^*)$ at equilibrium state. Thus, substituting into (11)–(16) with $i^* = r^* = i_v^* = 0$, we obtained the disease – free equilibrium state given by:

$$(s^*,i^*,v^*,r^*,s_v^*,i_v^*) = \left(\frac{\omega + b_H}{\omega + b_H + \rho}, 0, \frac{\rho}{\omega + b_H + \rho}, 0, 1, 0 \right) \qquad (18)$$

3.2. Effective basic reproduction number, R_c

Consideration of stability of a disease-free equilibrium gives certain conditions under which disease will die out or stay in the population called the Basic reproduction number, R_0. Using the next generation operator technique described by Diekmann and Heesterbeek (2000) and subsequently analyzed by Van den Driessche and Watmough (2002), we obtained the effective basic reproduction number, R_C of the equations (11)-(16) which is the spectral radius (ρ) of the next generation matrix, K.
i.e.

$R_C = \rho K$, where $K = FV^{-1}$

Now,

$$F = \begin{pmatrix} 0 & \beta_H j s^* \\ \beta_V s_v^* & 0 \end{pmatrix} \quad \text{and} \quad V = \begin{pmatrix} (\gamma + \mu_H + \delta) & 0 \\ 0 & b_V \end{pmatrix}$$

Thus,

$$R_C = \sqrt{\frac{\beta_V \beta_H j s^*}{b_V (\gamma + \mu_H + \delta)}} \qquad (s_v^* = 1) \qquad (19)$$

3.3. Local stability of disease free equilibrium, E_f

We used the Jacobian stability approach to prove the stability of the disease free equilibrium state. Using the relation

and $r = 1 - s - i - v \qquad (20)$

$$s_v = 1 - i_v \tag{21}$$

allows us as explained in Hethcote (2000), Benyah (2008) to attack (11)-(16) by studying the subsystem:

$$\frac{ds}{dt} = b_H - \beta_H j i_v s + \omega v - (\rho + b_H)s + \delta s i \tag{22}$$

$$\frac{di}{dt} = \beta_H j i_v s - (\gamma + \mu_H + \delta) i + \delta i^2 \tag{23}$$

$$\frac{dv}{dt} = \rho s - (\omega + b_H) v + \delta v i \tag{24}$$

$$\frac{di_v}{dt} = \beta_V i (1 - i_v) - b_V i_v \tag{25}$$

Linearization of the equations (22)-(25) at E_f gives the Jacobian matrix

$$J(E_f) = \begin{pmatrix} -(\rho + b_H) & 0 & \omega & -\beta_H j s^* \\ 0 & -(\gamma + \mu_H + \delta) & 0 & \beta_H j s^* \\ \rho & 0 & -(\omega + b_H) & 0 \\ 0 & \beta_V & 0 & -b_V \end{pmatrix} \tag{26}$$

Using elementary row-transformation, we have

$$J(E_f) = \begin{pmatrix} -(\rho + b_H) & 0 & \omega & -\beta_H j s^* \\ 0 & -(\gamma + \mu_H + \delta) & 0 & \beta_H j s^* \\ 0 & 0 & -(\omega + b_H) + \dfrac{\rho\omega}{(\rho + b_H)} & \dfrac{-\rho\beta_H j s^*}{(\rho + b_H)} \\ 0 & 0 & 0 & -b_V + \dfrac{\beta_V \beta_H j s^*}{(\gamma + \mu_H + \delta)} \end{pmatrix} \tag{27}$$

Thus, the eigenvalues are

$$\lambda_1 = -(\rho + b_H) < 0,\ \lambda_2 = -(\gamma + \mu_H + \delta) < 0,\ \lambda_3 = -\frac{b_H(\omega + \rho + b_H)}{(\rho + b_H)} < 0$$

and

$$\lambda_4 = \frac{-b_V(\gamma + \mu_H + \delta) + \beta_V \beta_H j s^*}{(\gamma + \mu_H + \delta)}$$

now, for λ_4 to be negative, we must have

$$\frac{-b_V(\gamma + \mu_H + \delta) + \beta_V \beta_H j s^*}{(\gamma + \mu_H + \delta)} < 0$$

Simplifying, gives

$$\sqrt{\frac{\beta_V \beta_H j s^*}{b_V(\gamma + \mu_H + \delta)}} < 1$$

Thus, if $R_C < 1$, λ_7 is negative, implying all the eigenvalues have negative real parts, we thus, established the following result.

Theorem 1: The disease- free equilibrium E_f of the model is locally asymptotically stable (LAS) if $R_C < 1$.

3.4. Global stability of disease free equilibrium, E_f

The epidemiological implication of the theorem is that yellow fever can be eliminated (control) from the population when $R_C < 1$, if the initial size of the sub-populations of the model are in the basin of attraction of the DFE.

In order to ensure that the disease is independent of the initial size of the sub-populations of the model (1)-(6), it is necessary to show that the DFE is globally- asymptotically stable (GAS). One common approach in studying the global asymptotic stability of the DFE is to construct an appropriate Lyapunov function (Li et al., 1999, Fall et al., 2007, Huo et al., 2010, Garba and Gumel, 2010). However, we applied the result introduced by Castillo-Chavez et al. (2002).

Theorem 2: The disease- free equilibrium E_f of (1)-(6) is globally asymptotically stable (GAS) in Ω if $R_C < 1$.

Proof: To establish the global stability of the disease free equilibrium, the two conditions (H1) and (H2) as in Castillo-Chavez et al. (2002) must be satisfied for $R_C < 1$. We rewrite the model (3.11) in the form:

$$\frac{dX_1}{dt} = F(X_1, X_2), \quad \frac{dX_2}{dt} = G(X_1, X_2); G(X_1, 0) = 0 \qquad (3.15)$$

where $X_1 = (s^*, v^*)$ and $X_2 = (i^*, i_v^*)$, with the components of $X_1 \in \Re^2$ denoting the uninfected population and the components of $X_2 \in \Re^2$ denoting the infected population.

The disease-free equilibrium is now denoted as

$$E_f = (X_1^*, 0), \; X_1^* = \left(\frac{\omega + b_H}{\omega + b_H + \rho}, \frac{\rho}{\omega + b_H + \rho} \right)$$

Now, for the first condition, that is globally asymptotically stability of X_1^*, we have

$$\frac{ds}{dt} = b_H - \beta_H j i_v s + \omega v - (\rho + b_H) s + \delta s i \qquad (11)$$

$$\frac{di}{dt} = \beta_H j i_v s - (\gamma + \mu_H + \delta) i + \delta i^2 \qquad (12)$$

$$\frac{dv}{dt} = \rho s - (\omega + b_H) v + \delta v i \qquad (13)$$

$$\frac{dr}{dt} = \gamma i - b_H r + \delta r i \qquad (14)$$

$$\frac{ds_v}{dt} = b_V - \beta_V i s_v - b_V s_v \qquad (15)$$

$$\frac{di_v}{dt} = \beta_V i s_v - b_V i_v \;\; \beta_H j i_v s - (\gamma + \mu_H + \delta) i + \delta i^2 \qquad (16)$$

$$\frac{dX_1}{dt} = F(X_1, 0) = \begin{bmatrix} b_H + \omega v^* - (\rho + b_H)s^* \\ \rho s^* - (\omega + b_H)v^* \end{bmatrix}$$

a linear differential equations.

Solving, we have

$$s^*(t) = \frac{b_H + \omega v^*}{(\rho + b_H)} - \frac{b_H + \omega v^*}{(\rho + b_H)} e^{-(\rho + b_H)t} + s^*(0)e^{-(\rho + b_H)t}$$

$$v^*(t) = \frac{\rho s^*}{(\omega + b_H)} - \frac{\rho s^*}{(\omega + b_H)} e^{-(\omega + b_H)t} + v^*(0)e^{-(\omega + b_H)t}$$

Now, clearly we have $s^*(t) + v^*(t) \to 1$ as $t \to \infty$, regardless of the value of $s^*(0)$ and $v^*(0)$.

Thus $X_1^* = \left(\frac{\omega + b_H}{\omega + b_H + \rho}, \frac{\rho}{\omega + b_H + \rho} \right)$ is globally asymptotically stable.

Next, for the second condition, that is $\hat{G}(X_1, X_2) = AX_2 - G(X_1, X_2)$, we have

$$A = \begin{bmatrix} -(\gamma + \mu_H + \delta) & \beta_H j s^* \\ \beta_V s_v^* & -b_V \end{bmatrix}$$

This is clearly an M-matrix (the off-diagonal elements of A are non-negative).

$$G(X_1, X_2) = \begin{bmatrix} \beta_H j i_v s - (\gamma + \mu_H + \delta) i + \delta i^2 \\ \beta_V i s_v - b_V i_v \end{bmatrix}$$

then,

$$\hat{G}(X_1, X_2) = AX_2 - G(X_1, X_2) = \begin{bmatrix} \delta i^2 \\ 0 \end{bmatrix}$$

i.e.

$$\hat{G}(X_1, X_2) = \left[\delta i^2, 0 \right]^T$$

Since all parameters are assumed non-negative, we have

$$\delta i^2 \geq 0$$

It is thus obvious that $\hat{G}(X_1, X_2) \geq 0$. Hence, the proof is complete.

4.0. Conclusions

In this paper, we have presented a mathematical model which incorporated some important factors that play significant role in the transmission dynamics and control of yellow fever. These factors are: vital dynamics, immunization, standard incidence, and disease induced death due to yellow fever infection. Our analysis reveals that the disease can be control if the effective basic reproduction number, R_C is less than one regardless of the initial population profile. This means that every effort must be put in place by all concerned to prevent the virus infection by reducing R_C strictly less than unity.

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