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Existence of Equilibrium points for the Mathematical Modeling of Yellow Fever Transmission Incorporating Secondary Host

[1]Somma S. A., [2]Akinwande N. I., [3]Jiya M. and [4]Abdulrahman S.*

[1,2,3]Department of Mathematics, Federal University of Technology, P. M. B., 65, Minna, Nigeria.
[4]Department of Mathematics, Federal University, Birni Kebbi, Nigeria.

Abstract

In this paper we, formulated a mathematical model of yellow fever transmission incorporating secondary host using first order ordinary differential equation. We verified the feasible region and the positivity of solution of the model. There exist two equilibria; disease free equilibrium (DFE) and endemic Equilibrium (EE). The disease free equilibrium (DFE) points were obtained.

Keywords: mathematical model; yellow fever; incorporating secondary host; feasible region; positivity

1. Introduction

Yellow fever, known historically as yellow jack, or yellow plague is an acute viral disease. In most cases symptoms include fever, chills, loss of appetite, nausea, muscle pains particularly in the back, and headaches. The disease is caused by the yellow fever virus and is spread by the bite of the female mosquito. It only infects humans, other primates and several species of mosquito [1]. In cities it is primarily spread by mosquitoes of the *Aedes aegypti* species. The virus is an RNA virus of the genus *Flavivirus* [2].

Yellow fever virus is mainly transmitted through the bite of the yellow fever mosquito *Aedes aegypti*, but other mosquitoes such as the tiger mosquito (*Aedes albopictus*) can also serve as a vector for this virus. Like other Arboviruses which are transmitted via mosquitoes, the yellow fever virus is taken up by a female mosquito when it ingests the blood of an infected human or other primate. Viruses reach the stomach of the mosquito, and if the virus concentration is high enough, the virus can infect epithelial cells and replicate there [3].

Vector-borne diseases (e.g. malaria, dengue, fever, yellow fever, lyme disease, trypanosomiases, and leishmania), amongst all the human infectious diseases, continue to remain a public health concern and a severe burden on economies, causing high human mortality in the world. These diseases have not only posed problems to national economies, but have also caused poverty and low living standards, especially in countries in the tropical and subtropical regions of the world [4].

Yellow fever virus (YFV) is the prototype species for the genus *Flavivirus* [5-9]. Historically, YFV is one of the most important human arboviral pathogens. It continues to cause large sporadic epidemics in Africa but typically emerges as epizootics among nonhuman primates in South America with or without associated human cases. YFV emergence is cyclical; outbreaks occur ≈7–10 years apart. Several phylogenetic studies have shown that YFV is locally maintained during these interepizootic periods in Peru and Brazil [9-11]. Yellow fever virus undergoes regionally independent evolution within some countries [10]. The sporadic emergence of YFV in the Americas has been strongly associated with infection of red howler monkeys *Alouatt aseniculus*, which are particularly susceptible to disease [12]. There are three types of transmission cycle:

(a) **Sylvatic (or jungle) yellow fever** occurs in tropical rainforests where monkeys, infected by sylvatic mosquitoes, pass the virus onto other mosquitoes that feed on them; these mosquitoes, in turn bite and infect humans entering the forest. Because of this sylvatic cycle, the yellow fever cannot be eradicated [13].

(b) **The intermediate cycle of yellow fever transmission** occurs in humid or semi-humid savannahs of Africa. Semi-domestic mosquitoes infect both monkey and human hosts and increased contact between man and infected mosquito leads to disease. In recent years, this has been the most common form of transmission of yellow fever in Africa.

(c) **Urban yellow fever** results in large explosive epidemics when travellers from rural areas introduce the virus into areas with high human population density. Domestic mosquitoes, most notably *Aedes aegypti*, carry the virus from person to person. It is well adapted to urban centres and can also transmit other diseases, including dengue fever and chikungunya. The urban cycle is responsible for the major outbreaks of yellow fever that occur in Africa.

Corresponding Author: Somma, S.A., Email: sam.abu@futminna.edu.ng, +2348068037304

Yellow fever vaccine has been used since 1937 in the prevention of yellow fever disease with more than 600 million doses of the vaccine having been delivered worldwide. Currently all yellow fever vaccines in use are live attenuated viral vaccine from the 17D lineage. The vaccine has been proven to be highly immunogenic and a single dose provides long-term protection against yellow fever. Yellow fever vaccine is recommended for people aged ≥9 months who are traveling to or living in areas with risk for YFV transmission in South America and Africa. In addition, some countries require proof of yellow fever vaccination for entry.

In *17 May 2013*, according to an article published in [14] Weekly Epidemiological Record (WER) reveals that the Organization's Strategic Advisory Group of Experts on immunization (SAGE) has reviewed the latest evidence and concluded that a single dose of vaccination is sufficient to confer life-long immunity against yellow fever disease. Therefore, a booster dose of yellow fever vaccine is not needed to maintain immunity.

Since yellow fever vaccination began in the 1930s, only 12 known cases of yellow fever post-vaccination have been identified, after 600 million doses have been dispensed. Evidence showed that among this small number of "vaccine failures", all cases developed the disease within five years of vaccination. This demonstrates that immunity does not decrease with time.

The model of yellow fever epidemics, which involves the interactions of two principal communities of hosts (humans) and Vectors (*aedes aegypti* mosquitoes) was formulated by Akinwande [15]. In his work, the host community was divided into three compartments of Susceptible $S(t)$, Infected $I(t)$ and Recovered $R(t)$ while the vector community was partitioned into two compartments of Susceptible $N(t)$ and Infective or virus carriers $M(t)$ where $t \geq 0$ is the time. He analyzed the local stability of the model using Jacobian matrix and implicit function.

An epidemic model of a vector-borne disease which has direct mode of transmission in addition to the vector-mediated transmission was studied by Hui-Ming *et al.* [16]. They assumed incidence term to be of the bilinear mass-action form. They also include both a baseline ordinary differential equation (ODE) version of the model, and, a differential-delay model with a discrete time delay. The delay in the differential-delay model accounts for the incubation time the vectors need to become infectious. They investigated the impact of the delay parameter on the stability of the equilibria.

The transmission dynamics of yellow fever (YF) within two host populations was described by Kungaro *et al.* [17], and built up a deterministic Susceptible – Vaccination – Expose – Infected - Recovered (SVEIR) model with vaccination to the entire new born. They formulated a model for the spread of Yellow fever in humans, vector and primates populations. The human population was divided into five (5) classes, the vector population was divided into three classes and the primate population was divided into three classes. It was assumed that the vaccinated and recovered individuals lose their immunity after ten years and need to be vaccinated again. They considered the standard incidence of all the populations. They used Metzler Matrix to analyzed the global stability of diseases free equilibrium (DFE).

A mathematical model of yellow fever incorporating the biology of the urban vector, the mosquito *Aedes aegypti* and the stages of the disease in the host (humans) was formulated by Souza *et al* [18]. From the epidemiological point of view, the mosquito follows a SEI sequence (Susceptible, Exposed, Infective). In their model, the adult populations are subdivided according to their status with respect to the virus. They assume that there is no vertical transmission of the virus and eggs, larvae, pupae and non parous adults are always susceptible. The humans are subdivided in sub-populations according to their status with respect to the illness as: susceptible (S), exposed (E), infective (I), in remission (r), toxic (T) and recovered (R).

Mathematical model to address the transmission dynamics of an infectious agent in a homogeneous population in the presence of an imperfect vaccine was studied by Raimundo *et al.* [19]. Their equations include the human and the vector and their eggs-population. The egg-population includes the intermediate stages, such as larvae and pupae. In their model the human population was divided into four (4) compartments which are susceptible S_H, vaccinated V_H, infected I_H and recovered R_H. The vector population was also divided into four (4) compartments of: susceptible S_M, infected I_M, latent L_M and non-infected eggs S_E.

In this paper, we formulated a mathematical model of yellow fever transmission incorporating secondary host, we assumed in our model that the vaccinated susceptible humans will move to recovered class.

2. Material and Methods

2.1 Model Formulation

The model equations are formulated using first order ordinary differential equation. Three populations were considered: human, vector (mosquito) and secondary host (monkey) populations. The populations are sub- divided into compartments with assumptions of the nature and rate of transfer from one compartment to another. We consider the total population sizes denoted by $N_h(t)$, $N_v(t)$ and $N_m(t)$ for the humans, mosquitoes (*Aedes aegypti*) and monkeys respectively. We use Susceptible, Infected and Recovered (SIR)model for the human population to describe the transmission dynamics of the disease. SIR model indicates that the passage of individuals is from the susceptible class, S_h, to infected class, I_h, and finally to the recovery class, R_h. $S_h(t)$ represents the number of individuals not yet infected with the yellow fever virus at time t. $I_h(t)$ denotes the number of individuals who have been infected with the yellow fever virus and is capable of spreading the disease to those in the susceptible category.

The transmission of the yellow fever virus between and among the population is driven by the mosquito bite. $I_h(t)$ is the compartment for individuals who have recovered from the disease through the treatment and natural healing. The humans in this class have permanent immunity and we assume that they cannot be re- infected by the yellow fever virus.

The transfer rates between the sub-classes are composed of several epidemiological parameters. The susceptible human population is increase by recruitment number Λ_h. When an *Aedes aegypti* mosquito bite a susceptible human, there is a probability, α_1 that the virus will be pass on to the human. The infected person moved to the infectious class. The susceptible individuals who are vaccinated and those who recovered through treatment or natural healing moved to recovered/immunity class at constant rates, ν and γ_h respectively. The recovered individuals have immunity to the disease and do not get clinically ill. In 2013, the WHO stated, "a single dose of vaccination is sufficient to confer life-long immunity against yellow fever disease.

The mosquito population is divided into two classes: Non-carrier vector, V_1 and carrier vector, $V_2(t)$. The Non-carrier vector population is increase by recruitment number Λ_v. Non-carrier vector become carrier by biting infectious human and infectious monkey. The virus enters the mosquito with probability $S_m(t)$ and t, when the mosquito bites an infectious human and infectious monkey respectively, the non-carrier vector becomes carrier. The mosquitoes are control at a rate δ_v. We assume that the infective period of the mosquito end with its death, and therefore the mosquito does not recover from being infected. The mosquitoes leave the population through natural death μ_v.

Monkey population is divided into two classes: susceptible, S_m and infectious, I_m. The susceptible monkey is increase by recruitment number Λ_m. Susceptible monkeys become infected with the mosquito bite, and there is a probability, α_4 that the virus will enter the monkey. The infected monkey moved to infectious class. We assume that, since monkeys are not immunized and treated; they don't have life-long immunity like human, hence the monkeys leave the population through the natural death and disease- induced death at the rates μ_m and δ_m respectively.

The model flow diagram is shown in figure 2. 1. The dash line from infected human class, I_h, to the non-carrier vector, V_1, shows that the infected human individuals infect the non-carrier vector population while the dash line from carrier vector, V_2, to the susceptible human population, α_4, shows the transfer of the virus from infected mosquito to susceptible human. So also, the dash line from infected monkey class, Λ_h, to the non-carrier vector, Λ_v, shows that the infected monkey infect the non-carrier vector population while the dash line from carrier vector, Λ_m, to the susceptible monkey population, S_m, shows the transfer of the virus from carrier vector to susceptible monkey.

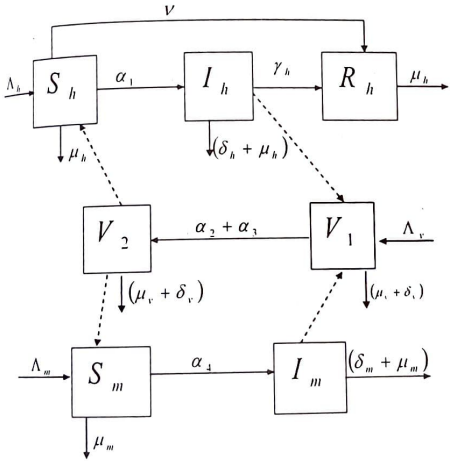

Figure 2.1: Model flowchart

2.2 Model Equations

$$\frac{dS_h}{dt} = \Lambda_h - \frac{\alpha_1 S_h V_2}{N_h} - (\nu + \mu_h)S_h \tag{2.1}$$

$$\frac{dI_h}{dt} = \frac{\alpha_1 S_h V_2}{N_h} - (\gamma_h + \mu_h + \delta_h)I_h \tag{2.2}$$

$$\frac{dR_h}{dt} = \nu S_h + \gamma_h I_h - \mu_h R_h \tag{2.3}$$

$$\frac{dV_1}{dt} = \Lambda_v - \frac{\alpha_2 V_1 I_h}{N_h} - \frac{\alpha_3 V_1 I_m}{N_m} - (\mu_v + \delta_v)V_1 \tag{2.4}$$

$$\frac{dV_2}{dt} = \frac{\alpha_2 V_1 I_h}{N_h} + \frac{\alpha_4 V_1 I_m}{N_m} - (\mu_v + \delta_v)V_2 \quad (2.5)$$

$$\frac{dS_m}{dt} = \Lambda_m - \frac{\alpha_4 S_m V_2}{N_m} - \mu_m S_m \quad (2.6)$$

$$\frac{dI_m}{dt} = \frac{\alpha_4 S_m V_2}{N_m} - (\mu_m + \delta_m)I_m \quad (2.7)$$

Where,

$$N_h = S_h + I_h + R_h \quad (2.8)$$

$$N_v = V_1 + V_2 \quad (2.9)$$

$$N_m = S_m + I_m \quad (2.10)$$

Table 2.1: Notation and definition of variables and parameter are represented as follows

Symbol	Description
$S_h(0)$	Number of susceptible humans at time t
$I_h(0)$	Number of infectious humans at time t
$R_h(0)$	Number of recovered/Immune human at time t
$V_1(0)$	Number of non-carrier vectors at time t
$V_2(0)$	Number of carrier vectors at time t
$S_m(0)$	Number of susceptible secondary host at time t
$I_m(0)$	Number of infectious secondary host at time t
N_h	Total human population at time t
N_v	Total vector population at time t
N_m	Total secondary vector population at time t
α_1	Effective virus Transmission rate from mosquito to humans
α_2	Effective virus Transmission rate from humans to mosquito
α_3	Effective virus Transmission rate from secondary host to mosquito
α_4	Effective virus Transmission rate from mosquito to secondary host
Λ_h	Recruitment number of human population
Λ_v	Recruitment number of mosquito population
Λ_m	Recruitment number of secondary vector population
δ_h	Disease-induced death rate of humans
δ_v	Death rate of mosquito due to application of insecticide
δ_m	Disease-induced death rate of secondary host
μ_h	Natural death rate of human population
μ_v	Natural death rate of mosquito population
μ_m	Natural death rate of secondary host population
γ_h	Recovery rate of human population due to drug administration
ν	vaccination rate for the human population

2.3 Invariant Region of the Model

The entire population size N_h, N_v and N_m can be determined by (2.8) to (2.10)

Adding equation (2 1), (2.2) and (2.3) gives

$$\frac{dN_h}{dt} = \Lambda_h - \mu_h N_h - \delta_h I_h \quad (2.11)$$

Adding equation (2.4) and (2.5) gives

$$\frac{dN_v}{dt} = \Lambda_v - (\mu_v + \delta_v)N_v \quad (2.12)$$

Adding equation (2.6) and (2.7) gives

$$\frac{dN_m}{dt} = \Lambda_m - \mu_m N_m - \delta_m I_m \quad (2.13)$$

In the absence of the disease $(\delta_h = \delta_m = 0)$, then, (2.11) and (2.13) gives

$\frac{dN_h}{dt} = \Lambda_h - \mu_h N_h - \delta_h I_h \leq \Lambda_h - \mu_h N_h$ (2.14)

$\frac{dN_m}{dt} = \Lambda_m - \mu_m N_m - \delta_m I_m \leq \Lambda_m - \mu_m N_m$ (2.15)

Theorem 2.1

The system (2.1) to (2.7) has solutions which are contain in the feasible region $\Omega = \Omega_h \times \Omega_v \times \Omega_m$ for all $t > 0$

Proof

$\Omega = (S_h, I_h, R_h, V_1, V_2, S_m, I_m) \in$ be any solution of the system (2.1) to (2.7) with non-negative initial conditions

using theorem on differential inequality [20] on (2.12), (2.14) and (3.15) gives

$0 \leq N_h \leq \frac{\Lambda_h}{\mu_h}$ (2.16)

Hence,

$\Lambda_h - \mu_h N_h \geq ke^{-\mu_h t}$ where k is the constant (2.17)

Similarly

$0 \leq N_v \leq \frac{\Lambda_v}{(\mu_v + \delta_v)}$ (2.18)

$\Lambda_v - (\mu_v + \delta_v)N_v \geq ke^{-(\mu_v + \delta_v)t}$ where k is the constant (2.19)

$0 \leq N_m \leq \frac{\Lambda_m}{\mu_m}$ (2.20)

$\Lambda_m - \mu_m N_m \geq ke^{-\mu_m t}$ where k is the constant (2.21)

Therefore, all feasible solutions of the human, mosquito and monkey population of the model system are in the regions:

$$\Omega_h = \left\{ (S_h, I_h, R_h) \in \mathbb{R}^3 : S_h, I_h, R_h \geq 0, N_h \leq \frac{\Lambda_h}{\mu_h} \right\} \quad (2.22)$$

$$\Omega_v = \left\{ (V_1, V_2) \in \mathbb{R}^2 : V_1, V_2 \geq 0, N_v \leq \frac{\Lambda_v}{(\mu_v + \delta_v)} \right\} \quad (2.23)$$

$$\Omega_m = \left\{ (S_m, I_m) \in \mathbb{R}^2 : S_m, I_m \geq 0, N_m \leq \frac{\Lambda_m}{\mu_m} \right\} \quad (2.24)$$

Thus, the feasible set of the model is given by

$$(S_h, I_h, R_h, S_v, I_v, S_m, I_m) \in \begin{matrix} S_h, I_h, R_h, S_v, I_v, S_m, I_m \geq 0; \\ N_h \leq \frac{\Lambda_h}{\mu_h} \; N_v \leq \frac{\Lambda_v}{(\mu_v + \delta_v)} \; N_m \leq \frac{\Lambda_m}{\mu_m} \end{matrix} \quad (2.25)$$

Which is a positively invariant (i.e. solutions remain positive for all times, t) and the model is epidemiologically meaningful and mathematically well pose.

2.4 Positivity of solutions

Lemma 2.1

Let the initial data be

$\{(S_h(0), I_h(0), R_h(0), V_1(0), V_2(0), S_m(0), I_m(0)) \geq 0\} \in \Omega$ (2.26)

Then, the solution set

$\{S_h(t), I_h(t), R_h(t), V_1(t), V_2(t), S_m(t), I_m(t)\}$ (2.27)

Of the system (2.1) to (2.7) is positive for all $t > 0$

Proof

From (2.1)

$\frac{dS_h}{S_h} \geq -(\nu + \mu_h)dt$ (2.28)

Integrating (2.28) gives

$S_h(t) \geq S_h(0)e^{-(\nu + \mu_h)t}$ (2.29)

From (2.2)

$$\frac{dI_h}{I_h} \geq -(\gamma_h + \mu_h + \delta_h)dt \tag{2.30}$$

Integrating (2.30) gives

$$I_h(t) \geq I_h(0)e^{-(\gamma_h + \mu_h + \delta_h)t} \tag{3.31}$$

From (2.3)

$$\frac{dR_h}{dt} \geq -\mu_h R_h \tag{2.32}$$

Integrating (2.32) gives

$$R_h(t) \geq R_h(0)e^{-\mu_h t} \tag{2.33}$$

From (2.4)

$$\frac{dV_1}{dt} \geq -(\mu_v + \delta_v)V_1 \tag{2.34}$$

Integrating (2.34) gives

$$V_1(t) \geq V_1(0)e^{-(\mu_v + \delta_v)t} \tag{2.35}$$

From (2.5)

$$\frac{dV_2}{dt} \geq -(\mu_v + \delta_v)V_2 \tag{2.36}$$

Integrating (2.36) gives

$$V_2(t) \geq V_2(0)e^{-(\mu_v + \delta_v)t} \tag{2.37}$$

From (2.6)

$$\frac{dS_m}{dt} \geq -\mu_m S_m \tag{2.38}$$

Integrating (2.38) gives

$$S_m(t) \geq S_m(0)e^{-\mu_m t} \tag{2.39}$$

From (2.7)

$$\frac{dI_m}{dt} \geq -(\mu_m + \delta_m)I_m \tag{2.40}$$

Integrating (2.40) gives

$$I_m(t) \geq I_m(0)e^{-(\mu_m + \delta_m)t} \tag{2.41}$$

Therefore, all the solution of equations of system (2.1) to (2.7) are positive for all $t > 0$

2.5 Existence of Equilibrium Points of the Model

At equilibrium

$$\frac{dS_h}{dt} = \frac{dI_h}{dt} = \frac{dR_h}{dt} = \frac{dV_1}{dt} = \frac{dV_2}{dt} = \frac{dS_m}{dt} = \frac{dI_m}{dt} = 0 \tag{2.42}$$

Let

$$(S_h, I_h, R_h, V_1, V_2, S_m, I_m) = (S_h^*, I_h^*, R_h^*, V_1^*, V_2^*, S_m^*, I_m^*) \tag{2.43}$$

be the arbitrary equilibrium point

Therefore, the system (2.1) - (2.7) becomes

$$\Lambda_h - \frac{\alpha_1 S_h^* V_2^*}{N_h} - (\nu + \mu_h)S_h^* = 0 \tag{2.44}$$

$$\frac{\alpha_1 S_h^* V_2^*}{N_h} - (\gamma_h + \mu_h + \delta_h)I_h^* = 0 \tag{2.45}$$

$$\nu S_h^* + \gamma_h I_h^* - \mu_h R_h^* = 0 \tag{2.46}$$

$$\Lambda_v - \frac{\alpha_2 V_1^* I_h^*}{N_h} - \frac{\alpha_3 V_1^* I_m^*}{N_m} - (\mu_v + \delta_v)V_1^* = 0 \tag{2.47}$$

$$\frac{\alpha_2 V_1^* I_h^*}{N_h} + \frac{\alpha_3 V_1^* I_m^*}{N_m} - (\mu_v + \delta_v)V_2^* = 0 \tag{2.48}$$

$$\Lambda_m - \frac{\alpha_4 S_m^* V_2^*}{N_m} - \mu_m S_m^* = 0 \tag{2.49}$$

$$\frac{\alpha_4 S_m^* V_2^*}{N_m} - (\mu_m + \delta_m)I_m^* = 0 \tag{2.50}$$

Let

$$A_1 = (\nu + \mu_h), \quad A_2 = (\gamma_h + \mu_h + \delta_h), \quad A_3 = (\mu_v + \delta_v) \text{ and } A_4 = (\mu_m + \delta_m) \tag{2.51}$$

Hence, equation (2.44) to (2.50) becomes

$$\Lambda_h - \frac{\alpha_1 S_h^* V_2^*}{N_h} - A_1 S_h^* = 0 \quad (2.52)$$

$$\frac{\alpha_1 S_h^* V_2^*}{N_h} - A_2 I_h^* = 0 \quad (2.53)$$

$$\nu S_h^* + \gamma_h I_h^* - \mu_h R_h^* = 0 \quad (2.54)$$

$$\Lambda_v - \frac{\alpha_2 V_1^* I_h^*}{N_h} - \frac{\alpha_3 V_1^* I_m^*}{N_m} - A_3 V_1^* = 0 \quad (2.55)$$

$$\frac{\alpha_2 V_1^* I_h^*}{N_h} + \frac{\alpha_3 V_1^* I_m^*}{N_m} - A_3 V_2^* = 0 \quad (2.56)$$

$$\Lambda_m - \frac{\alpha_4 S_m^* V_2^*}{N_m} - \mu_m S_m^* = 0 \quad (2.57)$$

$$\frac{\alpha_4 S_m^* V_2^*}{N_m} - A_4 I_m^* = 0 \quad (2.58)$$

From (2.53) and (2.58) we have

$$I_h^* = \frac{\alpha_1 S_h^* V_2^*}{A_2 N_h^*} \quad (2.59)$$

And

$$I_m^* = \frac{\alpha_4 S_m^* V_2^*}{A_4 N_m^*} \quad (2.60)$$

Substituting (2.59) and (2.60) into (2.56) gives

$$\left[\alpha_1 \alpha_2 A_4 N_m^{*2} S_h^* V_1^* + \alpha_3 \alpha_4 A_2 N_h^{*2} S_m^* V_1^* - A_2 A_3 A_4 N_h^{*2} N_m^{*2}\right] V_2^* = 0 \quad (2.61)$$

From (2.61)

$$V_2^* = 0 \quad (2.62)$$

Or

$$\alpha_1 \alpha_2 A_4 N_m^{*2} S_h^* V_1^* + \alpha_3 \alpha_4 A_2 N_h^{*2} S_m^* V_1^* - A_2 A_3 A_4 N_h^{*2} N_m^{*2} = 0 \quad (2.63)$$

Thus, equation (2.61) gives the existence of two different equilibria; one satisfying (2.62) and the other satisfying (2.63).
Substituting (2.62) into (2.59) and (2.60) gives

$$I_h^* = V_2^* = I_m^* = 0 \quad (2.64)$$

3. Results and Discussion

Equations (2.62) and (2.63) are the Disease Free Equilibrium (DFE) and Endemic Euilibrium (EE) respectively.

3.1 Disease Free Equilibrium (DFE) Points

Let,

$$E^0 = (S_h, I_h, R_h, V_1, V_2, S_m, I_m) = (S_h^0, I_h^0, R_h^0, V_1^0, V_2^0, S_m^0, I_m^0) \quad (2.65)$$

be the DFE point

Substituting (2.64) into (2.52) to (2.58) gives

$$\Lambda_h - A_1 S_h^0 = 0 \quad (2.66)$$

$$\nu S_h^0 - \mu_h R_h^0 = 0 \quad (2.67)$$

$$\Lambda_v - A_3 V_1^0 = 0 \quad (3.68)$$

$$\Lambda_m - \mu_m S_m^0 = 0 \quad (2.69)$$

From (2.66) to (2.69), thus, a DFE exist at the point

$$(S_h^0, I_h^0, R_h^0, V_1^0, V_2^0, S_m^0, I_m^0) = \left(\frac{\Lambda_h}{A_1}, 0, \frac{\Lambda_h \nu}{\mu_h A_1}, \frac{\Lambda_v}{A_3}, 0, \frac{\Lambda_m}{\mu_m}, 0\right) \quad (2.70)$$

4. Conclusion

The feasible set Ω of the model (2.1) to (2.7) is a positively invariant (i.e. solutions remain positive for all times, t) and the model is epidemiologically meaningful and mathematically well pose. Equation (2.61) showed the existence of two equilibria in the model; equation (2.62) showed the disease free equilibrium (DFE) and equation (2.63) showed the endemic-equilibrium (EE). We went further to obtained the disease free equilibrium points.

References

[1] Oldstone, Michael (2009). *Viruses, Plagues, and History: Past, Present and Future.* Oxford University Press. pp. 102–4.

[2] Bazin, Hervé (2011). *Vaccination : A History From Lady Montagu To Genetic Engineering.* Montrouge: J. LibbeyEurotext. 407.

[3] Fontenille D, Diallo M, Mondo M, Ndiaye M, and Thonnon J., (1997). "First Evidence of Natural Vertical Transmission of Yellow Fever Virus in *Aedesaegypti*, its Epidemic

[4] Nourridine S., Teboh-Ewungkem M. I. and Ngwa G. A., (2011).A Mathematical Model of the Population Dynamics of Disease-transmitting Vectors with Spatial Consideration, *Journal of Biological Dynamics*; 5(4): 335–365.

[5] Monath TP, (1989). Yellow Fever. In: Monath TP, editor. The arboviruses: Ecology and Epidemiology. Boca Raton (FL): CRC Press.; 5: 139–231.

[6] Van der Stuyft P, Gianella A, Pirard M, Cespedes J, Lora J, Peredo C, (1999) Urbanisation of Yellow Fever in Santa Cruz, Bolivia. *The Lancet;* 353(1964):1558–1562.

[7] Figueiredo LT, (2000). The Brazilian Flaviviruses.*Microbes and Infections;* 2(13):1643–1649.

[8] Souza R P, Foster P G, Sallum M A, Coimbra T L, Maeda A Y, Silveira V R, Moreno E S, Silva F G, Rocco I M, Suzuki A, Ferreira I B, Oshiro F M, Petrella S M, Pereira L E, Katz G, Tengan C H, Siciliano M M and Santos C L, (2010). Detection of a New Yellow Fever Virus Lineage within the South American Genotype I in Brazil .*Journal of Medical Virology*; 82(1):175–185.

[9] Auguste A. J, Lemey P, Pybus O. G., Suchard M. A., Salas R. A., Adesiyun A. A., (2010). Yellow Fever Virus Maintenance in Trinidad and its Dispersal throughout the Americas.*Journalof Virology;* 84(19): 9967–9977.

[10] Bryant J, Wang H, Cabezas C, Ramirez G, Watts D, and Russell K, (2003). Enzootic Transmission of Yellow Fever Virus in Peru.*Emerging Infectious Disease,* 9(8): 926–933.

[11] Vasconcelos P F, Bryant J E, da Rosa T P, TeshR B, Rodrigues S G, Barrett AD, (2004). Genetic Divergence and Dispersal of Yellow Fever Virus, Brazil. *Emerging Infectious Disease;* 10(9): 1578–1584.

[12] Auguste A. J., Lemey P., Bergren N. A., Giambalvo D., Moncada M., Morón D., Rosa Hernandez, Hernandez R., Navarro J., and Weaver S. C., (2015). Enzootic Transmission of Yellow Fever Virus, Venezuela, *Emerging Infectious Disease;* 21(1): 99-102.

[13] Barrett A. D., Higgs S. (2007). "Yellow Fever: a Disease that has yet to be conquered". *Annual Revolution. Entomology,* 52: 209–29.

[14] "Yellow fever Fact sheet N°100". *World Health Organization*. May 2013. Retrieved 23 February 2014.

[15] Akinwande, N. I., (1996), A Mathematical Model of Yellow Fever Epidemics, AfrickaMathematika, 6: 50-59.

[16] Hui-Ming W., Xue-Zhi L., and Maia M., (2008). An Epidemic Model of a Vector-borne Disease with Direct Transmission and Time Delay, *Journal of Mathematical Analysis and Applications*; 342(2): 895-908.

[17] Kung'aro, M., Luboobi, L. S. and Shahada, F. (2015).Modeling and Stability Analysis of SVEIRS Yellow Fever Two Host Model.*Gulf Journal of Mathematics*, 3(3): 106-129.

[18] Fernandez M L, Otero M, Schweigmann N and Solari H G, (2013), A Mathematically Assisted Reconstruction of the Initial Focus of the Yellow Fever Outbreak in Buenos Aires (1871). Papers in Physics, vol. 5, art. 050002.

[19] Raimundo, S. M., Amaku, M. & Massad, E. (2015). Equilibrium Analysis of a Yellow Fever Dynamics Model with Vaccination, *Computational and Mathematical Methods in Medicine*. 1: 1-12.

[20] Birkhoff, G. & Rota, G. C. (1982).Ordinary differential equations, Ginn.