A MATHEMATICAL MODEL OF THE TRANSMISSION DYNAMICS AND CONTROL OF TRYPANOSOMIASIS

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Abstract

A Mathematical model describing the transmission dynamics and control of Trypanosomiasis is developed and analyzed. The model involves three interacting populations; humans, livestock and tsetse-flies. We obtained the disease-free equilibrium state of the model and carried out local stability analysis using the effective reproduction number (R_c).

Keywords: Trypanosome, protozoa, Sleeping Sickness, hemo-lymphatic, meningo encephalitic

Introduction

African Trypanosomiasis (AT) commonly called sleeping sickness is an infectious disease of both human beings and animals. It is a vector–borne parasitic disease caused by an extracellular protozoa belonging to the genus, trypanosome, species brucei. The parasites are transmitted to humans by tsetse fly (Glossina genus) bite which have acquired their infection from human beings or animals harboring the pathogenic parasite (WHO,2015). There are two types of Human African Trypanosomiasis (HAT); West African sleeping sickness (WASS) which is the chronic type caused by Trypanosoma brucei gambiense (T.b.g) found only in humans, and East African sleeping-sickness (EASS), the acute type caused by Trypanosoma brucei rhodesiense (T.b.r) found in domesticated animals as well as in humans. According to World Health Organization (WHO,1998), ninety-five percent (95%) of the trypanosomiasis cases are chronic, with the victim suffering the disease for many years before eventual death. Acute infection can cause death within weeks. HAT (WASS) clinically evolves in two stages. First or early stage-known as the hemo-lymphatic phase, start with painful nodules/chancre with surrounding erythema and swelling 2-3 days after tsetse fly bite which erupts into a red sore, then invasion of the lymphatic system and blood stream 2-3weeks later characterized by non-specific symptoms like irregular bouts of fever, fatigue, headaches, aching muscles, increased sweating, and weight loss. (generally goes undiagnosed without sleeping sickness surveillance). Second or later stage-known as meningo-encephalitic phase is marked by involvement of the central nervous system with extensive neurological effects and can lead to serious sleep cycle disturbances(the disease earned its name from the hallmark of the 2nd stage classic symptoms, daytime slumber and nighttime insomnia) anxiety mood, confusion, slurred speech, paralysis, progressive mental deterioration, and ultimately results in death without effective treatment (Microbewiki, African Trypanosomiasis., WHO, 2013).

During the past decades, Akinwande (1995,2005),Adamu, *et al* (2011), Onyebiyuwa *et al* (2010), Nannyonga *et al* (2010), Damian *et al* (2014), Jose *et al* (2014), Abdulrahman (2014), Otieno *et al* (2014),Rachid *et al* (2015) have developed mathematical models of infectious diseases. Considering the work of the aforementioned authors, a new mathematical model is develop to complement and extend on their works by incorporating stage progression,screening and treatment in the proposed model.

Model Formulation

We formulated a model compartmentalizing the total population into nine epidemiological classes, with the following variables and parameters.

S_h (t) Susceptible humans at time t, I_h(t) Infected humans first stage at time t, I_m (t) Infected humans second stage at time t, R_h (t) Recovered humans at time t $N_v(t)$ Non-carrier vectors at time t, $C_v(t)$ Carrier vectors at time t , S_l(t) Susceptible livestock at time t, I_l(t) Infected livestock at time t, and $R_l(t)$ Recovered livestock at time t. where

Λ_h, Λ_v and Λ_l are the daily recruitment rates of human, vector and livestock respectively into the susceptible population. α, $\alpha\eta_1$, $\alpha\eta_2$ and $\alpha\eta_3$ are the effective transmission rates of AT from vector to human, human to vector, vector to livestock and livestock to vector while μ_h, μ_l and μ_v, δ_h, δ_l and δ_v are the natural and induced death rates for human, livestock and vector populations. γ_h is recovery rate of human due to natural healing, τ_m and τ_l are treatment rates of infected human and infected livestock respectively. ω_h, ω_l -waning rate of temporal immunity for human and livestock, while εp, the efficacy of protective clothing, ε_f efficacy of fumigation, φ_p human compliance with protective clothing and φ_f rate of usage of fumigation are the control strategies used.

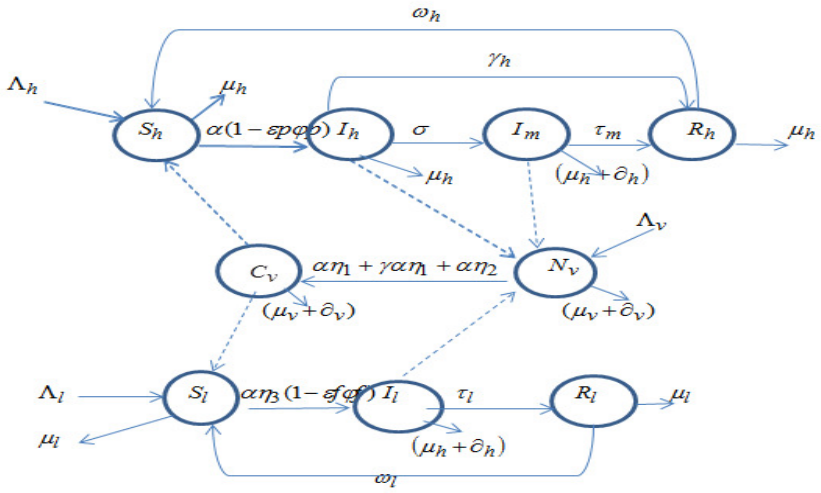

Figure1: Schematic diagram of AT transmission dynamics and control

The mathematical equations of the model can be described by a system of ordinary differential equations given below;

$$\frac{dS_h}{dt} = \Lambda_h + \omega_h R_h - \frac{\alpha C_v S_h (1-\varepsilon p\varphi p)}{P_h} - \mu_h S_h \qquad (1)$$

$$\frac{dI_h}{dt} = \frac{\alpha C_v S_h (1-\varepsilon p\varphi p)}{P_h} - (\sigma + \gamma_h + \mu_h) I_h \qquad (2)$$

$$\frac{dI_m}{dt} = \sigma I_h - (\tau_m + \mu_h + \delta_h) I_m \quad (3)$$

$$\frac{dR_h}{dt} = \gamma_h I_h + \tau_m I_m - (\mu_h + \omega_h) R_h \quad (4)$$

$$\frac{dN_v}{dt} = \Lambda_v - (\frac{\alpha\eta_1 I_h}{P_v} + \frac{\phi\alpha\eta_1 I_m}{P_v} + \frac{\alpha\eta_2 I_l}{P_v}) N_v - (\mu_v + \delta_v) C_v \quad (5)$$

$$\frac{dC_v}{dt} = (\frac{\alpha\eta_1 I_h}{P_v} + \frac{\phi\alpha\eta_1 I_m}{P_v} + \frac{\alpha\eta_2 I_l}{P_v}) N_v - (\mu_v + \delta_v) C_v \quad (6)$$

$$\frac{dS_l}{dt} = \Lambda_l + \omega_l R_l - \frac{\alpha\eta_3 C_v S_l (1 - \varepsilon f \varphi f)}{P_l} - \mu_l s_l \quad (7)$$

$$\frac{dI_l}{dt} = \frac{\alpha\eta_3 C_v S_l (1 - \varepsilon f \varphi f)}{P_l} - (\tau_l I_l + k_l + \delta_l) I_l \quad (8)$$

$$\frac{dR_l}{dt} = \tau_l I_l - (\omega_l + k_l) R_l \quad (9)$$

where

$$P_h(t) = S_h(t) + I_h(t) + I_m(t) + R_h(t)$$
$$P_l(t) = S_l(t) + I_l(t) + R_l(t)$$
$$P_{v(t)=N_v(t)+C_v(t)} \quad (10)$$

So that

$$\frac{dP_h}{dt} = \Lambda_h - \mu_h P_h - \delta_h I_h - \delta_h I_m$$
$$\frac{dP_l}{dt} = \Lambda_l - k_l P_l - \delta_l I_l \quad (11)$$
$$\frac{dP_v}{dt} = \Lambda_v - (\mu_v + \delta_v) C_v$$

In a biological region-feasible region:

$$\Omega = \left\{ S_h, I_h, I_m, R_h, N_v, C_{v,} S_l, I_l, R_l \right\} \in \Re_+^9 : N \le P_h + P_l + P_v \quad (12)$$

It can be shown to be positively invariant with respect to the system (1) –(9) The domain is invalid epidemiologically as the sub population $S_{h,} I_h, I_m, R_h, N_{v,} C_v, S_l, I_l, and, R_l$ are all non- negative and the sum of each population (P_h, P_l, P_v) is less than or equal to the total population.

Model Analysis

Existence of Disease-Free Equilibrium, E^0

The disease –free equilibrium states are steady- state solutions where there is no disease. Hence, all the infected classes will be zero. The entire population comprises of susceptible individuals. **Theorem1**: A disease –free equilibrium state of the model (1) exists at the point

$$E^0 = 0\left(S_h^o, I_h^o, I_m^o, R_h^o, N_v^o, C_v^o, S_l^o, I_l^o, R_l^o \right) = \left(\frac{\Lambda_h}{\mu_h}, 0, 0, 0, \frac{\Lambda_v}{K_4}, 0, \frac{\Lambda_l}{k_l}, 0, 0 \right)$$

Proof:

At the equilibrium state, the rate of change of each variable is equal to zero. i.e

$$\frac{dS_h}{dt}=\frac{dI_h}{dt}=\frac{dI_m}{dt}=\frac{dR_h}{dt}=\frac{dN_v}{dt}=\frac{dC_v}{dt}=\frac{dS_l}{dt}=\frac{dI_l}{dt}=\frac{dR_l}{dt}=0 \tag{13}$$

Let; $(S_h, I_h, I_m, N_v, C_v, S_l, I_l, R_l)=\left(S_h^*, I_h^*, I_m^*, N_v^*, C_v^*, S_l^*, I_l^*, R_l^*\right)$

at equilibrium state. Thus we have from system (1) –(9)

$$\Lambda_h+\omega_h R_h\frac{\alpha C_h^* S_h^*(1-\varepsilon p\varphi p)}{P_h^*}-\mu_h S_h^*=0 \tag{14}$$

$$\frac{\alpha C_h^* S_h^*(1-\varepsilon p\varphi p)}{P_h^*}-K_1 I_h^*=0 \tag{15}$$

$$\sigma I_h^*-K_2 I_m^*=0 \tag{16}$$

$$\gamma_h I_h^*+\tau_m I_m^*-K_3 R_h^*=0 \tag{17}$$

$$\Lambda_v-\left(\frac{\alpha\eta_1 I_h^* N_v^*}{P_v^*}+\frac{\phi\alpha\eta_1 I_m^* N_v^*}{P_v^*}+\frac{\alpha\eta_2 I_l^* N_v^*}{P_{*v}}\right)-K_4 N_v^*=0 \tag{18}$$

$$\frac{\alpha\eta_1 I_h^* N_v^*}{P_v^*}+\frac{\phi\alpha\eta_1 I_m^* N_v^*}{P_v^*}+\frac{\alpha\eta_2 I_l^* N_v^*}{P_v^*}-K_4 C_v^*=0 \tag{19}$$

$$\Lambda_l+\omega_l R_l^*-\frac{\alpha\eta_3 C_v^* S_l^*(1-\varepsilon f\varphi f)}{P_l^*}-u_l S_l^*=0 \tag{20}$$

$$\frac{\alpha\eta_3 C_v^* S_l^*(1-\varepsilon f\varphi f)}{P_l^*}-K_5 I_l^*=0 \tag{21}$$

$$\tau_l I_l^*-K_6 R_l^*=0 \tag{22}$$

where

$$\left.\begin{aligned} &K_1=(\sigma+\tau_h+\mu_h), K_2=(\tau_m+\mu_h+\delta_h), K_3=(\mu_h+\omega_h)\\ &K_4=(\mu_v+\delta_v), K_5=(\tau_l+k_l+\delta_l), K_6=(k_l+\omega_l)\end{aligned}\right\}$$

From (16),we have

$$I_m^*=\frac{\sigma I_h^*}{K_2} \tag{23}$$

Substituting (23) into (17) yields

$$R_h^*=\left(\frac{\gamma_h K_2+\tau_m\sigma}{K_2 K_3}\right)I_h^* \tag{24}$$

Substituting (24) into (14) gives

$$S_h^*=\left[\frac{\Lambda_h K_2 K_3+\omega_h(K_2\gamma_h+\tau_m\sigma)I_h^*}{K_2 K_3\{\alpha C_v^*(1-\varepsilon p\varphi p)+\mu_h P_h^*\}}\right]P_h^* \tag{25}$$

From (15) and (25) we have

$$I_h^* = \frac{\alpha C_v^*(1-\varepsilon p\varphi p)K_2K_3\Lambda_h}{\{\alpha[K_1K_2K_3 - \omega_h(K_2\gamma_h + \tau_m\sigma)]C_v^*(1-\varepsilon p\varphi p) + K_1K_2K_3\mu_h P_h^*\}} \quad (26)$$

Let

$$\left.\begin{aligned} A &= \alpha[K_1K_2K_3 - \omega_h(K_2\gamma_h + \tau_m\sigma)](1-\varepsilon p\varphi p) \\ B &= K_1K_2K_3\mu_h P_h^* \end{aligned}\right\} \quad \text{then,}$$

$$I_h^* = \frac{K_2K_3\alpha\Lambda_h C_v^*}{AC_v^* + B} \quad (27)$$

substituting (27) into (24) gives

$$R_h^* = (\gamma_h K_2 + \tau_m\sigma)\left(\frac{\alpha\Lambda_h C_v^*}{AC_v^* + B}\right) \quad (28)$$

Also by substituting (27) into (25) gives

$$S_h^* = \left[\frac{K_2K_3[\Lambda_h(AC_v^* + B) + \omega_h(K_2\gamma_h + \tau_m\sigma)\alpha\Lambda_h C_v^*}{K_2K_3(\alpha C_v^* + \mu_h P_h^*)(AC_v^* + B)}\right]P_h^* \quad (29)$$

From (21)

$$I_l^* = \frac{\alpha\eta_3 C_v^* S_l^*(1-\varepsilon f\varphi f)}{K_5 P_l^*} \quad (30)$$

subtitute (30) into (22) gives

$$R_l^* = \frac{\tau_l\alpha\eta_3 S_l^*(1-\varepsilon f\varphi f)C_v^*}{K_5K_6P_l^*} \quad (31)$$

From (31), (20) becomes

$$S_l^* = \frac{K_5K_6P_l^*\Lambda_l}{\alpha\eta_3 C_v^*(1-\varepsilon f\varphi f)K_5K_6 - \omega_l\tau_l\alpha\eta_3 C_v^*(1-\varepsilon f\varphi f) + K_5K_6P_l^*\mu_l} \quad (32)$$

Similarly substituting (27) into (23) gives

$$I_m^* = \frac{\sigma\alpha\Lambda_h K_3 C_v^*}{AC_v^* + B} \quad (33)$$

Also (32) into (31) gives

$$R_l^* = \frac{\tau_l\alpha\eta_3 C_v^*(1-\varepsilon f\varphi f)}{K_5K_6P_l^*}\left[\frac{K_5K_6P_l^*\Lambda_l}{K_5K_6\alpha\eta_3 C_v^*(1-\varepsilon f\varphi f) - \omega_l\tau_l\alpha\eta_3 C_v^*(1-\varepsilon f\varphi f) + K_5K_6P_l^*\mu_l}\right] \quad (34)$$

Also (32) into (30) yields

$$I_l^* = \frac{\alpha\eta_3 C_v^*(1-\varepsilon f\varphi f)}{K_5 P_l^*}\left[\frac{K_5K_6P_l^*\Lambda_l}{K_5K_6\alpha\eta_3C_v^*(1-\varepsilon f\varphi f)-\omega_l\tau_l\alpha\eta_3C_v^*(1-\varepsilon f\varphi f)+K_5K_6P_l^*\mu_l}\right] \quad (35$$

simplifying (18) gives

$$N_v = \frac{\Lambda_v P_v^*(C_v^*+B)}{\alpha^2\eta_1\Lambda_h K_3(K_2+\phi\sigma)C_v^*} + \frac{\Lambda_v P_v^*}{K_4 P_v^*} + \frac{\Lambda_v P_v^*\left\{K_5P_l^*[\alpha\eta_3(K_5K_6-\omega_l\tau_l)]C_v^*(1-\varepsilon f\varphi f)+K_5K_6P_l^*\mu_l\right\}}{\alpha^2\eta_2\eta_3C_v^*(1-\varepsilon f\varphi f)K_5K_6P_l^*\Lambda_l} \quad (36)$$

From (19), we have

$$C_v^*\left\{\frac{\alpha^2\eta_1\Lambda_h K_3(K_2+\phi\sigma)N_v^*}{(AC_v^*+B)P_v^*} + \frac{\alpha^2\eta_3\eta_4K_5K_6P_l^*\Lambda_l N_v^*}{P_v^*K_5P_l^*\left[\alpha\eta_3[(K_5K_6-\omega_l\tau_l)C_v^*]+K_5K_6P_l^*\mu_l\right.} - K_4\right\} = 0$$

Thus

. C_v^* =0 or (37)

$$\frac{\alpha^2\eta_1\Lambda_h K_3(K_2+\phi\sigma)N_v^*}{(AC_v^*+B)P_{*_v}} + \frac{\alpha^2\eta_2\eta_3K_5K_6P_l^*\Lambda_l N_v^*}{P_v^*K_5P_l^*\left[\alpha\eta_3[(K_5K_6-\omega_l\tau_l)C_v^*]+K_5K_6P_l^*\mu_l\right.} - K_4 = 0 \quad (38)$$

Substituting (37) into (27),(28), (32),(33),(34) we obtain

$$I_h^* = I_m^* = I_l^* = R_h^* = R_l^* = 0 \quad (39)$$

$$C_v^* > 0$$

When

$$\frac{\alpha^2\eta_1\Lambda_h K_3(K_2+\phi\sigma)N_v^*}{K_4(AC_v^*+B)P_v} + \frac{\alpha^2\eta_2\eta_3K_5K_6P_l\Lambda_l N_v^*}{K_4P_vK_5P_l\left[\alpha\eta_3[(K_5K_6-\omega_l\tau_l)C_v^*]+K_5K_6P_l\mu_l\right.} > 1 \quad (40)$$

Thus giving two different equilibrium state, DFE state where

$$I_h^* = I_m^* = I_l^* = C_v^* = 0$$

And endemic equilibrium where all the compartments are greater than zero

Now, substituting (39) into (14),(18),and (20),we have

$$S_h^* = \frac{\Lambda_h}{\mu_h}, \qquad N_v^* = \frac{\Lambda_v}{K_4}, \qquad S_l^* = \frac{\Lambda_l}{\mu_l}$$

Thus a DFE state of the model exists at the point

$$\left(\left(S_h^*, I_h^*, I_m^*, R_h^*, N_v^*, C_v^*, S_l^*, I_l^*, R_l^*\right) = \left(\frac{\Lambda_h}{\mu_h}, 0, 0, 0, \frac{\Lambda_v}{K_4}, 0, \frac{\Lambda_l}{\mu l}, 0, 0\right)\right.$$

Effective Reproduction number, R_c

We apply the next generation matrix operator as used by Diekmann and Heesterbeek (1990,2000), and improved upon by Driessche and Watmough (2002), we obtained the effective reproduction number $R_c = FV^{-1}$ where F is the matrix of new infection terms and V is

the matrix of the transmission terms formed from the coefficient of the infected classes (I_m, I_h ,I_l,C_v).

$$F=\begin{bmatrix} 0 & 0 & \alpha(1-\varepsilon p\varphi p) & 0 \\ 0 & 0 & 0 & 0 \\ \alpha\eta_1 & \phi\alpha\eta_1 & 0 & \alpha\eta_2 \\ 0 & 0 & \alpha\eta_3(1-\varepsilon f\varphi f) & 0 \end{bmatrix} \text{ and } V^{-1}=\begin{bmatrix} \frac{1}{k_1} & 0 & 0 & 0 \\ \frac{\alpha}{K_1K_2} & \frac{1}{K_2} & 0 & 0 \\ 0 & 0 & \frac{1}{K_4} & 0 \\ 0 & 0 & 0 & \frac{1}{K_5} \end{bmatrix} \quad (41)$$

$$FV^{-1}=\begin{bmatrix} 0-\lambda & 0 & \frac{\alpha(1-\varepsilon p\varphi p)}{K_4} & 0 \\ 0 & 0-\lambda & 0 & 0 \\ \frac{\alpha\eta_1}{K_1}+\frac{\phi\sigma\alpha\eta_1}{K_1K_2} & \frac{\phi\alpha\eta_1}{K_2} & 0-\lambda & \frac{\alpha\eta_2}{K_5} \\ 0 & 0 & \frac{\alpha\eta_3(1-\varepsilon f\varphi f)}{K_4} & 0-\lambda \end{bmatrix} \quad (42)$$

From which we obtained the effective reproduction number as

$$R_c=\sqrt{\frac{\alpha^2\eta_1K_5(1-\varepsilon p\varphi p)[K_2+\phi\sigma]+K_1K_2\alpha^2\eta_2\eta_3(1-\varepsilon f\varphi f)}{K_1K_2K_4K_5}}$$

Local Stability of Disease-free Equilibrium State

Linearization of the model system (1) to (9) at any arbitrary point (E*) gives the Jacobian matrix (43), used in the local stability analysis. of the disease-free equilibrium state

$$J(E^0)=\begin{pmatrix} -\mu_h & 0 & 0 & \omega_h & 0 & -\alpha(1-\varepsilon p\varphi p) & 0 & 0 & 0 \\ 0 & -K_1 & 0 & 0 & 0 & -\alpha(1-\varepsilon p\varphi p) & 0 & 0 & 0 \\ 0 & \sigma & -K_2 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \gamma_h & \tau_m & -K_3 & 0 & 0 & 0 & 0 & 0 \\ 0 & -\alpha\eta_1 & -\phi\alpha\eta_1 & 0 & -K_4 & 0 & 0 & -\alpha\eta_2 & 0 \\ 0 & \alpha\eta_1 & \phi\alpha\eta_1 & 0 & 0 & -K_4 & 0 & \alpha\eta_2 & 0 \\ 0 & 0 & 0 & 0 & 0 & -\alpha\eta_3(1-\varepsilon f\varphi f) & -k_l & 0 & \omega_l \\ 0 & 0 & 0 & 0 & 0 & \alpha\eta_3(1-\varepsilon f\varphi f) & 0 & -K_5 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \tau_l & -K_6 \end{pmatrix} \quad (43)$$

Using elementary row-transformation gives

$$J(E^0)=\begin{pmatrix} -\mu_h & 0 & 0 & \omega_h & 0 & -\alpha(1-\varepsilon p\varphi p) & 0 & 0 & 0 \\ 0 & -K_1 & 0 & 0 & 0 & -\alpha(1-\varepsilon p\varphi p) & 0 & 0 & 0 \\ 0 & 0 & -K_2 & 0 & 0 & \dfrac{\sigma\alpha}{K_1} & 0 & 0 & 0 \\ 0 & 0 & 0 & -K_3 & 0 & H_1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -K_4 & -H_2 & 0 & -\alpha\eta_2 & 0 \\ 0 & 0 & 0 & 0 & 0 & -H_4 & 0 & \alpha\eta_2 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -k_l & -H_5 & \omega_l \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -H_6 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -K_6 \end{pmatrix} \quad (44)$$

$$\left.\begin{aligned} H_1=\frac{\alpha\left(\gamma_h K_2+\sigma\tau_m\right)}{K_1K_2}, H_2=\left(\frac{\alpha^2\eta_1}{K_1}+\frac{\phi\sigma\alpha^2\eta_1}{K_1K_2}\right), H_3=\left(K_4-\frac{\alpha^2\eta_1}{K_1}\right), \\ H_4=\left(H_3-\frac{\phi\sigma\alpha^2\eta_1}{K_1K_2}\right), H_5=\left(\frac{\alpha^2\eta_2\eta_3}{K_4}\right), H_6=\left(K_5-\frac{\alpha^2\eta_2\eta_3}{K_4}\right) \end{aligned}\right\} \quad (45)$$

The characteristic equation of the row transformed Jacobian matrix(43) is given as

$$J(E^0)=\begin{pmatrix} -(\mu_h+\lambda) & 0 & 0 & \omega_h & 0 & -\alpha & 0 & 0 & 0 \\ 0 & -(K_1+\lambda) & 0 & 0 & 0 & -\alpha & 0 & 0 & 0 \\ 0 & 0 & -(K_2+\lambda) & 0 & 0 & \dfrac{\sigma\alpha}{K_1} & 0 & 0 & 0 \\ 0 & 0 & 0 & -(K_3+\lambda) & 0 & H_1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -(K_4+\lambda) & -H_2 & 0 & -\alpha\eta_2 & 0 \\ 0 & 0 & 0 & 0 & 0 & -(H_4+\lambda) & 0 & \alpha\eta_2 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -(k_l+\lambda) & -H_5 & \omega_l \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -(H_6+\lambda) & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -(K_6+\lambda) \end{pmatrix} \quad (46)$$

the eigenvalues are

$$\lambda_1=-\mu_h<0$$

$$\lambda_2=-K_1<0=-(\sigma+\gamma_h+\mu_h)<0$$

$$\lambda_3=-K_2=-(\gamma_h+\mu_h+\delta_h)<0$$

$$\lambda_4=-K_3=-(\mu_h+\omega_h)<0$$

$$\lambda_5=-K_4=-(\mu_v+\delta_v)<0$$

$$\lambda_6=-H_4=-(H_3-\frac{\phi\alpha^2\eta_1\sigma}{K_1K_2})<0$$

$$\lambda_7=-u_l<0$$

$$\lambda_8=-H_6=-\left(K_5-\frac{\alpha^2\eta_2\eta_3(1-\varepsilon f\varphi f)}{K_4}\right) \quad (47)$$

$\lambda_9 = -K_6 = -(\omega_l + \mu_l) < 0$

For

$\lambda_6 = -\left[\frac{K_4}{1} - \frac{\alpha^2\eta_1}{K_1} - \left(\frac{\alpha^2\eta_1\phi\sigma}{K_1K_2}\right)\right] \Rightarrow \lambda_6 = -\left(\frac{K_1K_2K_4 - K_2\alpha^2\eta_1 - \alpha^2\eta_1\phi\sigma}{K_1K_2}\right)$

$\lambda_6 = \left(\frac{\alpha^2\eta_1(1-\varepsilon p\varphi p)[K_2+\phi\sigma] - K_1K_2K_4}{K_1K_2}\right)$ Dividing through by $K_1K_2K_4$ we have ,

$\lambda_6 = K_4\left(\frac{\alpha^2\eta_1(1-\varepsilon p\varphi p)[K_2+\phi\sigma]}{K_1K_2K_4} - 1\right)$, but $R_{hv} = \frac{\alpha^2\eta_1(1-\varepsilon p\varphi p)[K_2+\phi\sigma]}{K_1K_2K_4}$

$\lambda_6 = K_4(R_{hv} - 1)$, if R_{hv} <1, then $\lambda_6 < 0$

$\lambda_8 = -H_6 = -\left(K_5 - \frac{\alpha^2\eta_2\eta_3(1-\varepsilon f\varphi f)}{K_4}\right) \Rightarrow \lambda_8 = \left(\frac{\alpha^2\eta_2\eta_3(1-\varepsilon f\varphi f)}{K_4K_5} - 1\right)$,

$R_{lv} = \left(\frac{\alpha^2\eta_2\eta_3(1-\varepsilon f\varphi f)}{K_4K_5}\right) \Rightarrow \lambda_8 = (R_{lv} - 1)$

by dividing through by K_5, but $then \lambda_8 < 0, if, R_{lv} < 1$

$\lambda_6 = \frac{\alpha^2\eta_1(1-\varepsilon p\varphi p)[K_2+\sigma\phi]}{K_1K_2K_4} < 0$,and $\lambda_8 = \frac{\alpha^2\eta_2\eta_3(1-\varepsilon f\varphi f)}{K_4K_5} < 0$

if R_c<1. Simplifying gives

$R_c = \sqrt{\frac{\alpha^2\eta_1K_5(1-\varepsilon p\varphi p)[K_2+\phi\sigma] + K_1K_2\alpha^2\eta_2\eta_3(1-\varepsilon f\varphi f)}{K_1K_2K_4K_5}} < 1$

Thus, λ_6 and $\lambda_8 < 0$ if $R_c < 1$. Hence the disease – equilibrium, E^0 is locally asymptotically stable if $R_c < 1$.. thus, determining the local stability of the system.

Conclusion

In this paper, a non-linear mathematical model of African Trypanosomiasis is developed, incorporating screening and treatment of the infectious second stage human population, the effective reproduction number R_c was obtained which was used to establish the conditions for Local Stability of the Disease-Free Equilibrium (DFE).The results showed that the Disease-Free Equilibrium will be locally asymptotically stable if $R_c < 1$.

Recommendations

Sensitization of the public on the danger of trypanosomiasis and the need for its prevention, since no vaccine exists for immunity against the disease. Generating a vaccine for the disease for both human and livestock to reduce infections, and can probably eradicate the disease if more than 70% of the population are vaccinated with a vaccine whose efficacy does not wane after injection. also combined control strategies that have great effect in eradicating the disease should be adopted.(preventive clothing, fumigation, screening and treatment of the infected individuals, insecticides-reducing the vector population)

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