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Global Stability Analysis of the Disease-Free Equilibrium State of a Mathematical Model of Trypanosomiasis

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ABSTRACT: The global stability analysis represents a compound failure, mechanism which provides lower calculated factors of safety. In this research, the global stability analysis was used to propose a mathematically model of the transmission dynamics and control of Trypanosomiasis, known as African sleeping sickness. We obtained the Disease-free equilibrium state and present graphical profile of some of the compartments.

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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. World Health Organization (2013, 2015) reported that approximately 60 million people and 48 million cattle out of the estimated 172 million cattle are at risk of sleeping sickness in 36 countries in sub-Saharan Africa, In 1998 almost 40,000 cases were reported, but estimates were that between 300,000 to 500,000 were undiagnosed and therefore not treated.17, 600 cases were reported in 2004, below 10,000 cases in 2009, and it dropped to 6,314 cases in 2012.The disease ranked 3rd in economic importance (after malaria and schistosomiasis) of all vector-borne human diseases and it is also the second greatest cause of mortality in affected communities, countries such as Angola, the Democratic Republic of Congo and Sudan had 50 percent occurrence in several of their villages. A number of mathematical models both simulation and analytical have been proposed to describe infectious diseases and African Trypanosomiasis. Otieno *et al*., (2014) describes an analytical model for trypanosomiasis in a cattle population. Damian *et al*., (2014) model the use of insecticide-treated cattle to control tsetse and trypanosome bruceirhodesiense, Madsen et al., (2012) described the effect of seasonal fluctuation in tsetse fly population and human African Trypanosomiasis, John *et al.,*(2012), model the Control of Trypanosomiasis using Trypanocides or Insecticide-Treated Livestock, Nannyonga *et al*., (2010) model on co-infection of malaria and trypanosomiasis. Akinwande (1995), Diekman (1990, 2000), Abdulrahman (2014) formulated models on infectious diseases. In this study, we describe a model for trypanosomiasis in human and cattle populations, which incorporates stage progression, screening and treatment of the population.

MODEL FORMULATION

A mathematical model of the dynamics of Trypanosomiasis incorporating stage progression, screening and treatment of the population, as control strategies was formulated. The model involved three interacting populations, humans, vectors and livestock. The total populations are compartmentalized intonine epidemiological classes, with the following variables: 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)

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= Infected livestock at time t, and $R_l(t)$ = Recovered livestock at time t.

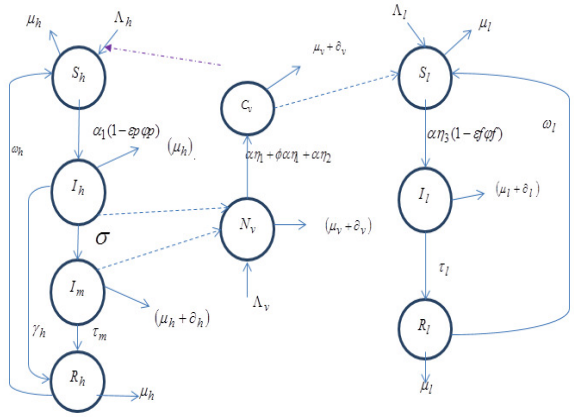

Fig 1: Schematic diagram of AT transmission dynamics and control.

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

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

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

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

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

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

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

$$\frac{dI_l}{dt} = \frac{\alpha \eta_3 C_v S_l (1-\varepsilon f \varphi f)}{P_l} - (\tau_l I_l + \mu_l + \delta_l) I_l \quad 7$$

$$\frac{dR_l}{dt} = \tau_l I_l - (\omega_l + \mu_l) R_l \quad 8$$

Where

$$\left.\begin{aligned} P_h(t) &= S_h(t) + I_h(t) + I_m(t) + R_h(t) \\ P_l(t) &= S_l(t) + I_l(t) + R_l(t) \\ P_v(t) &= N_v(t) + C_v(t) \end{aligned}\right\} \quad 9$$

$$\frac{dS_l}{dt} = \Lambda_l + \omega_l R_l - \frac{\alpha \eta_3 C_v S_l (1-\varepsilon f \varphi f)}{P_l} - \mu_l s_l \quad 10$$

So that

$$\left.\begin{aligned} \frac{dP_h}{dt} &= \Lambda_h - \mu_h P_h - \delta_h I_h - \delta_h I_m \\ \frac{dP_l}{dt} &= \Lambda_l - \mu_l P_l - \delta_l I_l \\ \frac{dP_v}{dt} &= \Lambda_v - (\mu_v + \delta_v) C_v \end{aligned}\right\} \quad 11$$

In a biological region-feasible region

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

Where the parameters 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, εp is 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. .

Disease-free Equilibrium E^0**:** At disease –free equilibrium state there is no disease. Hence, the infected classes are zero. From (1-9)

$$\Lambda_h + \omega_h R_h \frac{\alpha C_h^* S_h^* (1-\varepsilon p \varphi p)}{P_h^*} - \mu_h S_h^* = 0 \quad 13$$

$$\frac{\alpha C_h^* S_h^* (1-\varepsilon p \varphi p)}{P_h^*} - K_1 I_h^* = 0 \quad 14$$

$$\sigma I_h^* - K_2 I_m^* = 0 \quad 15$$

$$\gamma_h I_h^* + \tau_m I_m^* - K_3 R_h^* = 0 \quad 16$$

$$\frac{\alpha\eta_1 I_h^* N_v^*}{P_v^*}+\frac{\phi\alpha\eta_1 I_m^* N_v^*}{P_v^*}+\frac{\alpha\eta_2 I_l^* N_v^*}{P_v^*}-K_4 C_v^*=0 \quad 17$$

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

$$\Lambda_l+\omega_l R_l^*-\frac{\alpha\eta_3 C_v^* S_l^*(1-\varepsilon f\varphi f)}{P_l^*}-k_l S_l^*=0 \quad ..19$$

$$\frac{\alpha\eta_3 C_v^* S_l^*(1-\varepsilon f\varphi f)}{P_l^*}-K_5 I_l^*=0 \quad 20$$

$$\tau_l I_l^*-K_6 R_l^*=0 \quad 21$$

Where

$$\left.\begin{aligned}K_1=(\sigma+\tau_h+\mu_h),K_2=(\tau_m+\mu_h+\delta_h),K_3=(\mu_h+\omega_h)\\K_4=(\mu_v+\delta_v),K_5=(\tau_l+k_l+\delta_l),K_6=(k_l+\omega_l)\end{aligned}\right\} \quad 22$$

From (15),we have

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

Substituting (23) into (16) yields

$$R_h^*=\left(\frac{\gamma_h K_2+\tau_m\sigma}{K_2K_3}\right)I_h^* \quad 24$$

Substituting (24) into (13) gives

$$S_h^*=\left[\frac{\Lambda_h K_2K_3+\omega_h(K_2\gamma_h+\tau_m\sigma)I_h^*}{K_2K_3\{\alpha C_v^*(1-\varepsilon p\varphi p)+\mu_h P_h^*\}}\right]P_h^* \quad 25$$

From (16) , (23)and (24) 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

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

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

Put (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 putting (19) into (17) 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 (20)

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

Put (30) into (21) 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 (30), (19) becomes

$$S_l^* = \frac{K_5 K_6 P_l^* \Lambda_l}{\alpha\eta_3 C_v^* (1-\varepsilon f\varphi f) K_5 K_6 - \omega_l \tau_l \alpha\eta_3 C_v^* (1-\varepsilon f\varphi f) + K_5 K_6 P_l^* k_l} \quad 32$$

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

$$R_l^* = \frac{\tau_l \alpha\eta_3 C_v^* (1-\varepsilon f\varphi f)}{K_5 K_6 P_l^*} \left[\frac{K_5 K_6 P_l^* \Lambda_l}{K_5 K_6 \alpha\eta_3 C_v^* (1-\varepsilon f\varphi f) - \omega_l \tau_l \alpha\eta_3 C_v^* (1-\varepsilon f\varphi f) + K_5 K_6 P_l^* k_l} \right] \quad 34$$

Also (32) into (30) yields

$$I_l^* = \frac{\alpha\eta_3 C_v^* (1-\varepsilon f\varphi f)}{K_5 P_l^*} \left[\frac{K_5 K_6 P_l^* \Lambda_l}{K_5 K_6 \alpha\eta_3 C_v^* (1-\varepsilon f\varphi f) - \omega_l \tau_l \alpha\eta_3 C_v^* (1-\varepsilon f\varphi f) + K_5 K_6 P_l^* k_l} \right] \quad 35$$

Substituting and simplifying (17)

$$N_v = \frac{\Lambda_v P_v^* (C_v^* + B)}{\alpha^2 \eta_1 \Lambda_h K_3 (K_2 + \phi\sigma) C_v^*} + \frac{\Lambda_v P_v^*}{K_4 P_v^*} + \frac{\Lambda_v P_v^* \left\{ K_5 P_l^* [\alpha\eta_3 (K_5 K_6 - \omega_l \tau_l)] C_v^* (1-\varepsilon f\varphi f) + K_5 K_6 P_l^* k_l \right\}}{\alpha^2 \eta_2 \eta_3 C_v^* (1-\varepsilon f\varphi f) K_5 K_6 P_l^* \Lambda_l} \quad 36$$

From (18), we have

$$C_v^* \left\{ \frac{\alpha^2 \eta_1 \Lambda_h K_3 (K_2 + \phi\sigma) N_v^*}{(AC_v^* + B) P_v^*} + \frac{\alpha^2 \eta_3 \eta_4 K_5 K_6 P_l^* \Lambda_l N_v^*}{P_v^* K_5 P_l^* [\alpha\eta_3 [(K_5 K_6 - \omega_l \tau_l) C_v^*] + K_5 K_6 P_l^* \mu_l} - K_4 \right\} = 0 \quad 37$$

Thus

$C_v^* = 0$ or 37

$$\frac{\alpha^2 \eta_1 \Lambda_h K_3 (K_2 + \phi\sigma) N_v^*}{(AC_v^* + B) P_{*_v}} + \frac{\alpha^2 \eta_2 \eta_3 K_5 K_6 P_l^* \Lambda_l N_v^*}{P_v^* K_5 P_l^* [\alpha\eta_3 [(K_5 K_6 - \omega_l \tau_l) C_v^*] + K_5 K_6 P_l^* \mu_l} - K_4 = 0 \quad 38$$

Substituting (37) into (27),(28), (33),(34),(35) we obtain

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

$$C_v^* > 0$$

when

$$\frac{\alpha^2 \eta_1 \Lambda_h K_3 (K_2 + \phi\sigma) N_v^*}{K_4 (AC_v^* + B) P_v} + \frac{\alpha^2 \eta_2 \eta_3 K_5 K_6 P_l \Lambda_l N_v^*}{K_4 P_v K_5 P_l [\alpha\eta_3 [(K_5 K_6 - \omega_l \tau_l) C_v^*] + K_5 K_6 P_l \mu_l} > 1 \quad 40$$

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 consider (37), substituting into (13),(17),and (19),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

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

stable if $R_c < 1$.

Effective Reproduction number, R_c: We apply the next generation matrix operator as used by Diekmann and Heesterbeek (2000), and improved upon by Van de 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

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

Global Stability of Disease-Free equilibrium,E^0:

Theorem 1: The DFE E^o of model equation (1) is globally asymptotically stable (GAS) in Ω if $R_c < 1$ and unstable if $R_c > 1$.

Proof: - To establish the global stability of the DFE, the two conditions for the global stability of DFE as in Castillo-Chavel *et al* (2002), for $R_c<1$was used for the equations of model system (1-9). It is possible to re – write the model system (1-9) in the following way

$$\left.\begin{aligned} \frac{dX_s}{dt} &= A(X_s - X_{DFE,S}) + A_1X_i \\ \frac{dX_i}{dt} &= A_2X_i \end{aligned}\right\} \quad 43$$

Where $X_s = (S_h^0, R_h^0, N_v^0, S_l^0, R_l^0)^T$ denote the different compartments of non – infected individuals, and $X_i = (I_h^0, I_m^0, C_v^0, I_l^0)^T$ denotes the compartments of different infected individuals. The disease – free equilibrium is denoted as $E^o = (X_S^*, O)$. where $X_S^* = (P_h^o + P_l^o + P_v^o, 0)$

$$\frac{dX_s}{dt} = F(X_s, 0) = \begin{cases} \Lambda_h + \omega_h R_1^0 - \mu_h S_h^0 \\ -(\mu_h + \omega_h) R_h^0 i.e. - K_3 R_h^0 \\ \Lambda_v - K_4 N_v^0 \\ \Lambda_l + \omega_l R_l^0 - k_l S_l^0 \\ -K_6 R_l^0 \end{cases} \quad 44$$

a linear differential equation which on solving gives the following:

$$S_h^0(t) = \frac{(\Lambda_h + \omega_h R_h^0)}{\mu_h} - \frac{(\Lambda_h + \omega_h R_h^0) e^{-\mu_h t}}{\mu_h} + S_h^0(0) e^{-\mu_h t} \quad 45$$

$$R_h^0(t) = R_h^0(0) e^{-k3t} \quad 46$$

$$N_v^0(t) = \frac{\Lambda_v}{4} - \frac{\Lambda_v e^{-k4t}}{4} + N_v^0(0) e^{-k4t} \quad 47$$

$$S_l^0(t) = \frac{(\Lambda_l + \omega_l R_l^0)}{k_l} - \frac{(\Lambda_l + \omega_l R_l^0) e^{-k_l t}}{k_l} + S_l^0(0) e^{-k_l t} \quad 48$$

$$R_l^0(t) = R_l^0(0) e^{-K6t} \quad 49$$

Now from (10) gives

$S_h^0(t) + R_h^0(t) + N_v^0(t) + S_l^0(t) + R_l^0(t) \to N^0(t)$, as t $\to$ 0.regardless of the value of $S_h^o(o), R_h^o(o), N_v^o(o), S_l^o(o), R_l^o(o)$ .Thus $X_S^* = (P_h^o + P_l^o + P_v^o, 0)$ is globally and asymptotically stable.

Next, $\bar{G}(X_s, X_i) = AX_i - G(X_s, X_i)$,

$$AXi = \begin{bmatrix} -K_1 & 0 & \alpha(1-\varepsilon p \varphi p) & 0 \\ \sigma & -K_2 & 0 & 0 \\ \alpha\eta_1 & \phi\alpha\eta_1 & -K_4 & \alpha\eta_2 \\ 0 & 0 & \alpha\eta_3(1-\varepsilon f \varphi f) & -K_5 \end{bmatrix} \quad 50$$

It is obvious that this is an M-matrix (Metzler also called quasi-positive matrix whose diagonal Elementsare non-negative). The off diagonal elements of A are non-negative.

$$G(X_s, X_i) = \begin{bmatrix} \frac{\alpha C_v^0 S_h^0 (1-\varepsilon p \varphi p)}{P_h} & -K_1 I_h^0 \\ \sigma I_h^0 & -K_2 I_m^0 \\ \frac{\alpha\eta_1 I_h^0}{P_v} + \frac{\phi\alpha\eta_1 I_m^0}{P_v} + \frac{\alpha\eta_2 I_l^0}{P_v} & -K_4 C_v^0 \\ \frac{\alpha\eta_3 C_v^0 S_l^0 (1-\varepsilon f \varphi f)}{P_l} & -K_5 I_l^0 \end{bmatrix} \quad 51$$

Then,

$$\hat{G}(X_{s,}X_i) = AX_s - G(X_s, X_i) = \begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}^T \quad 52$$

i.e.

$$\hat{G}(X_{s,}X_i) = [0 \quad 0 \quad 0 \quad 0]^T \quad 53$$

Thus, $\hat{G}(X_{s,}X_i) = 0$,hence the proof is complete.

Numerical Simulations: We give the numerical simulation for the model system (1-9) for the purpose of verifying some of the analytic results. We give the numerical simulation for the model system (1) for the purpose of verifying some of the analytic results.

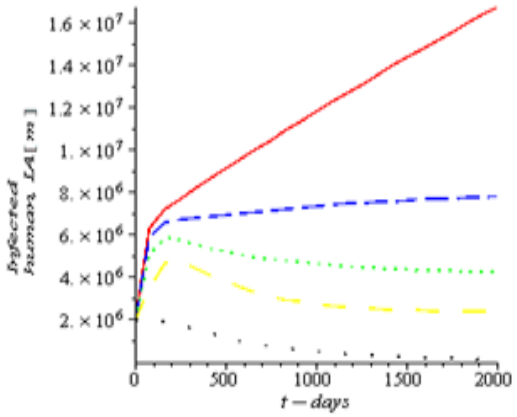

Fig 2: Graph of infected human IA(t) against time t when
$\varphi p = \varphi f = \delta_v = \tau_m = \tau_l = 0, \varphi p = \varphi f = \delta_v = \tau_m = \tau_l = 0.25,$
$\varphi p = \varphi f = \delta_v = \tau_m = \tau_l = 0.50, \varphi p = \varphi f = \delta_v = \tau_m = \tau_l = 0.75$
$\varphi p = \varphi f = \delta_v = \tau_m = \tau_l = 0.95$

Conclusion: In this paper, a non-linear mathematical model of AT is developed and analyzed incorporating the treatment of the infectious second stage human population, basic reproduction number R_o was used to establish the conditions for Global Stability of the Disease-Free Equilibrium (DFE) showing that the Disease-Free Equilibrium will be logically asymptotically stable if $R_o < 1$. Numerical simulation shows that the disease can be eradicated when both preventive and treatment strategies are adopted.

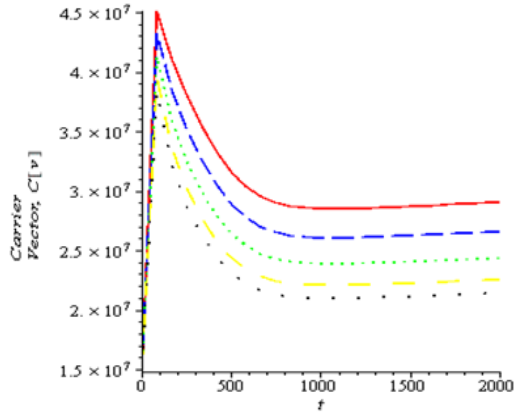

Fig 3: Graph of carrier vector at time t when
$\delta_v = 0, \delta_v = 0.25, \delta_v = 0.50, \delta_v = 0.75, v = 0.95$

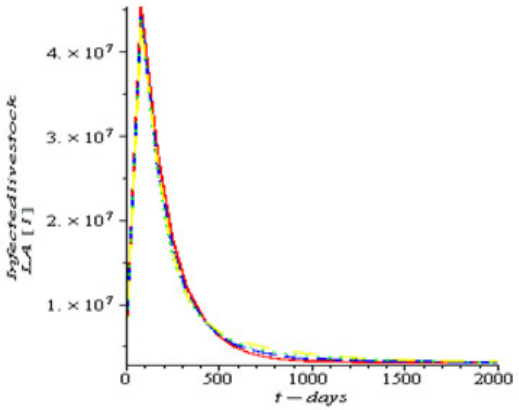

Fig.4: Graph of infected livestock I(t) against time when
$\varphi p = \varphi f = \delta_v = \tau_m = \tau_l = 0, \varphi p = \varphi f = \delta_v = \tau_m = \tau_l = 0.25$
$\varphi p = \varphi f = \delta_v = \tau_m = \tau_l = 0.50, \varphi p = \varphi f = \delta_v = \tau_m = \tau_l = 0.75$

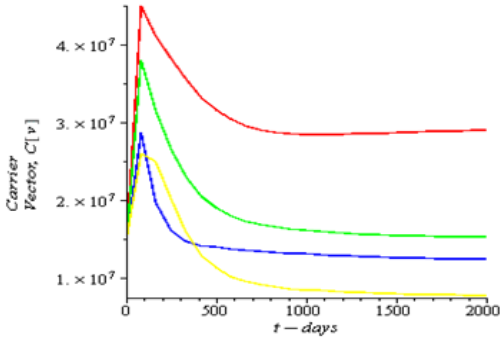

Fig 5: Graph of Carrier vector using combined strategies when

$\varphi p = \varphi f = \delta_v = \tau_m = \tau_l = 0, \varphi p = \varphi f = \delta_v = \tau_m = \tau_l = 0.25$

$\varphi p = \varphi f = \delta_v = \tau_m = \tau_l = 0.50, \varphi p = \varphi f = \delta_v = \tau_m = \tau_l = 0.75$

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