POPULATION DYNAMICS OF THE EAST AFRICAN SLEEPING SICKNESS

D. Okuonghae and J. Osemwenkhae
Department of Mathematics,
University of Benin,
P.M.B 1154, Benin City, Nigeria.

ABSTRACT

Mathematical models of the East African sleeping sickness epidemiology are presented. This paper is aimed at modelling the dynamics of the disease as it affects the human and domestic animal populations. The mathematical model is extended to include the contact rate of the tsetse flies with the wild park animals that serve as the reservoir for the parasite that causes this disease. Steady states for the models are also presented as well as possible control strategy for the disease. Threshold conditions for the disease free equilibrium are presented for both models.

Keywords: *Mathematical model, steady state, trypanosomiasis, threshold conditions.*

INTRODUCTION

The purpose of this paper is to contribute to modelling in epidemiology. We wish to model mathematically the population dynamics of the East African *Trypanosomiasis* or sleeping sickness. The paper by Roger, (1988) provided a general model for the African sleeping sickness caused by the parasite *Trypasonomiasis brucei* or *T.brucei*, involving two vertebrate host species and the tsetse fly vector. In the paper, he generalized the disease and modelled the population dynamics of the vertebrates involved in the disease cycle. Medical research, however, has shown that there actually exist two morphologically similar parasites that cause different disease though both are caused by the tsetse fly.

In humans, the East African Sleeping Sickness (EASS) also known as the Rhodesian sleeping sickness is caused by the parasite *Trypanosoma brucei rhodesiense,* or *T. brucei rhodesiense* whereas West African sleeping sickness, also known as Gambian sleeping sickness, is caused by *Trypanosoma brucei gambiense* or *T.brucei gambiense.* Both diseases are transmitted by tsetse flies (The Tsetse Fly, 2001). Trypanosomiasis in cattle is caused by the parasites *Trypanosoma congolense and Trypanosoma vivax* and is also carried by the tsetse fly. See African Sleeping Sickness, 2004; African Trypanosomiasis, 2001; UNICEF-UNDP-WORLD BANK-WHO Special report, 2003 for more details. On

the rate of infection as well as the economic effect of the disease see Bolton College, 2002.

Descriptively, EASS is an acute disease that typically leads to death within weeks or months if not treated, unlike its West African counterpart that is chronic, since symptoms may not appear for months to years after the initial infection. Neurological complications include slurred speech, confusion, and difficulty with walking. See African Trypanosomiasis, 2002; Bolton College, 2002 for further details on the complications associated with the EASS.

Seven species of tsetse flies in the genus *Glossina* act as vectors of the disease to humans (Species of the tsetse fly, 2003; The Tsetse Fly, 2001; Tsetse Fly, 2004). The cycle begins when a fly bites an infected mammal (e.g. bushbuck, wild pig or warthog) and ingests the parasites. The protozoans multiply and develop over a series of weeks within the gut and salivary glands of the fly. When the fly bites another human or domestic animal host, the mature forms of the parasite enter the host, settling in the blood and spinal fluid (Health issues on the tsetse fly, 2003).

Game wardens and visitors as well as local villagers or farmers contract it as they monitor livestocks or collect firewood. Nevertheless, the level of occurrence varies in different localities.

The aim of this paper is to develop a mathematical model for better understanding of the dynamics of the disease as well as propose approaches for its control.

The remainder of this paper is structured as follows. In Section 2 we will give the formulation of the mathematical model for the EASS and carry out steady state analysis on the model. Section 3 will provide an extended model to the one formulated in the preceding section. We also do some steady state analysis on the extended model. Finally in Section 4 we will proceed with a discussion of our findings from the mathematical models considered and draw some conclusion.

THE MATHEMATICAL MODEL

In this section a model is derived consisting of a system of non-linear ordinary differential equations (ODEs). To model the EASS, we need to keep track of the disease status for the human population, domestic animal population though the wild animals having the protozoan do not have any problem with the disease as well as the tsetse fly population.

This paper shall present the EASS as a SIS disease since the disease appears in a wave-like pattern with the recovered individual likely to fall again to the disease after another round of infection. This pattern of recovery and relapse is due to the parasite changing its surface coating in an attempt to avoid the immune system. It is said that the parasite has roughly 1000 different types of coats it can 'wear' (Frank, 1999).

The incubation period of the disease is neglected in the formulation of the model. This is because symptoms start to appear rather quickly (most times within a week) after initial infection unlike the West African sleeping sickness case where symptoms may not appear for months or even years after the initial infection. In this case (West African sleeping sickness), incubation period may have to be included in the modelling process. This is a major difference between the East African sleeping sickness and the West African sleeping sickness.

The human population shall be divided into two different classes, namely the susceptible and infective classes. Also the animal population as well as the tsetse fly population will be divided into similar set of classes. Modelling the flows between the human, animal as well as tsetse fly populations leads to a system of non-linear ordinary differential equations.

Equilibrium states are considered for the model, with emphasis on the disease free state. Numerical solutions are given and probable control strategies for the disease are proposed.

DERIVATION OF MODEL

We denote the number of susceptible in the human population by $\mathbf{S_H(t)}$ and infected persons by $\mathbf{I_H(t)}$. Likewise we shall represent the susceptible domestic animal population with $\mathbf{S_A(t)}$ and the infected domestic animals with $\mathbf{I_A(t)}$. In the case of the tsetse fly population, we let $S_T(t)$ be the population of susceptible flies while $I_T(t)$ stands for the infected flies i.e. flies that carry the parasites.

We shall ignore birth and death rates in both vertebrate populations and assume further that the humans, domestic animals and tsetse flies populations are closed.

Hence the model becomes

$$\frac{dS_H}{dt} = -\beta_H S_H I_T + \gamma_H I_H$$

$$\frac{dI_H}{dt} = \beta_H S_H I_T - \gamma_H I_H$$

$$\frac{dS_A}{dt} = -\beta_A S_A I_T + \gamma_A I_A$$

$$\frac{dI_A}{dt} = \beta_A S_A I_T - \gamma_A I_A$$

$$\frac{dS_T}{dt} = -(\beta_{1T} S_T I_H + \beta_{2T} S_T I_A) + \gamma_T I_T + b_T N_T - d_T S_T$$

$$\frac{dI_T}{dt} = \beta_{1T} S_T I_H + \beta_{2T} S_T I_A - \gamma_T I_T - d_T I_T \qquad \textbf{(1)}$$

The human, domestic animal and tsetse fly populations are represented by $\mathbf{N_H}$, $\mathbf{N_A}$ and $\mathbf{N_T}$ respectively. Hence in addition to the governing equation we shall also be having that

$$S_H + I_H = N_H$$

$$S_A + I_A = N_A$$

$$S_T + I_T = N_T \qquad \textbf{(2)}$$

We assume that the flies have equal birth and death rates. Let $\mathbf{b_T}$ and $\mathbf{d_T}$ be the birth and death rates of the tsetse fly population respectively.

From the model in (1), $\beta_H S_H I_T$, **where** $\beta_H = \frac{ap_H}{N_H}$ is the incidence of the disease for the human population. The parameter a is the biting rate of the tsetse flies. Using a similar argument in (Nicholas, 2003), the functional response of the tsetse flies to humans is assumed constant. A fraction $\frac{S_H}{N_H}$ of blood meals necessary for the transmission of the parasites are taken from susceptible

humans, and each leads to infection with probability p_H. For the domestic animals, the incidence of the disease is $\beta_A S_A I_T$, **where** $\beta_A = \frac{ap_A}{N_A}$. The explanation of the parameters is similar to the explanations for the human case. Here p_A is the probability that a susceptible domestic animal will become infected when bitten by a tsetse fly.

For the tsetse flies, the incidence 'function' is given by

$\beta_{1T} S_T I_H + \beta_{2T} S_T I_A$, **where** $\beta_{1T} = \frac{ap_{TH}}{N_H}, \beta_{2T} = \frac{ap_{TA}}{N_A}$. In this case, p_{TH} denotes the probability that a susceptible tsetse fly will become infected on biting an infectious human. Similarly, p_{TA} denotes the probability that a susceptible fly will become infected upon biting an infectious domestic animal.

We denote the recovery rate for the humans to be γ_H while the rate for which domestic animals leave the infective class is γ_A. The same explanation holds for γ_T

Next we dimensionalize the dependent variables in (1) using their respective population sizes i.e.

$$u_H = \frac{S_H}{N_H}, \quad v_H = \frac{I_H}{N_H}, u_A = \frac{S_A}{N_A}, \quad v_A = \frac{I_A}{N_A}, u_T = \frac{S_T}{N_T}, \quad v_T = \frac{I_T}{N_T}$$

Hence the governing equations (1) becomes,

$$\frac{du_H}{dt} = -\gamma_H(\beta_H u_H v_T N_T / \gamma_H - v_H)$$

$$\frac{dv_H}{dt} = \gamma_H(\beta_H u_H v_T N_T / \gamma_H - v_H)$$

$$\frac{du_A}{dt} = -\gamma_A(\beta_A u_A v_T N_T / \gamma_A - v_A)$$

$$\frac{dv_A}{dt} = \gamma_A(\beta_A u_A v_T N_T / \gamma_A - v_A)$$

$$\frac{du_T}{dt} = (\gamma_T + b_T)((-\beta_{1T} u_T v_H N_H - \beta_{2T} u_T v_A N_A)/(\gamma_T + b_T) + v_T)$$

$$\frac{dv_T}{dt} = (\gamma_T + b_T)((\beta_{1T} u_T v_H N_H + \beta_{2T} u_T v_A N_A)/(\gamma_T + b_T) - v_T) \qquad (3)$$

and

$$u_H + v_H = 1, \; u_A + v_A = 1, \; u_T + v_T = 1.$$

The system of 6 nonlinear ODEs will be reduced to a system of 5 nonlinear ODEs with one algebraic equation, leaving us with the following equations to solve:

$$\frac{du_H}{dt} = -\gamma_H(k_1 u_H v_T - v_H)$$

$$\frac{dv_H}{dt} = \gamma_H(k_1 u_H v_T - v_H)$$

$$\frac{du_A}{dt} = -\gamma_A(k_2 u_A v_T - v_A)$$

$$\frac{dv_A}{dt} = \gamma_A(k_2 u_A v_T - v_A)$$

$$\frac{dv_T}{dt} = (\gamma_T + b_T)((k_3(1-v_T)v_H + k_4(1-v_T)v_A - v_T) \qquad (4)$$

The system in (4) will be solved together with $u_T = 1 - v_T$.

The parameters in the equations are defined as:

$$k_1 = \frac{ap_H N_T}{\gamma_H N_H},\ k_2 = \frac{ap_A N_T}{\gamma_A N_A},\ k_3 = \frac{ap_{TH}}{(\gamma_T + b_T)},\ k_4 = \frac{ap_{TA}}{(\gamma_T + b_T)}.$$

STEADY STATE ANALYSIS

We shall investigate the equilibrium point for the disease free situation. This can be obtained from the solutions of the equations $\frac{dv_H}{dt} = 0,\ \frac{dv_A}{dt} = 0,\ \frac{dv_T}{dt} = 0$ i.e.

$$\gamma_H(k_1(1-v_H)v_T - v_H) = 0$$

$$\gamma_A(k_2(1-v_A)v_T - v_A) = 0$$

$$(\gamma_T + b_T)(k_3(1-v_T)v_H + k_4(1-v_T)v_A - v_T) = 0$$

The disease free state will have all the infected humans, domestic animals and tsetse flies return to zero after an initial introduction of (infected) tsetse flies into a susceptible population of humans and domestic animals.

In this case, the disease free equilibrium point is $(v_H, v_A, v_T) = (0, 0, 0)$ i.e. a state where there is no infectious human, domestic animal or even tsetse flies as $t \to \infty$ (after a long time has passed).

To investigate the stability of the DFE (disease free equilibrium), we find the Jacobian of the system under consideration and obtain:

$$\begin{pmatrix} -k_1 v_T - 1 & 0 & k_1(1-v_H) \\ 0 & -k_2 v_T - 1 & k_2(1-v_A) \\ k_3(1-v_T) & k_4(1-v_T) & -k_3 v_H - k_4 v_A - 1 \end{pmatrix}$$

the point (0, 0, 0), J has the eigenvalues

$= -1,\ \lambda_2 = -1 + \sqrt{(k_1 k_3 + k_2 k_4)}$ **and** $\lambda_3 = -1 - \sqrt{(k_1 k_3 + k_2 k_4)}$.

The stability of this point depends crucially on $R_0 = k_1 k_3 + k_2 k_4$. If $0 < R_0 < 1$ the three eigenvalues will be negative, making the point (0, 0, 0) asymptotically stable otherwise the point is unstable. In other words, if $0 < R_0 < 1$, the disease dies out after enough time has passed. If $R_0 > 1$, it means that the point (0, 0, 0) will not be stable and there could be an epidemic of EASS in the community.

Now $R_0 = \dfrac{a^2 p_H p_{TH} N_T}{\gamma_H N_H (\gamma_T + b_T)} + \dfrac{a^2 p_A p_{TA} N_T}{\gamma_A N_A (\gamma_T + b_T)}$ is the basic reproductive ratio for the disease. We can write this as $R_0 = R_{0H} + R_{0T}$ where R_{0H} is the basic reproductive ratio for the human case while R_{0T} is the basic reproductive ratio for the domestic animal case. The East African sleeping sickness dies if and only if

$$R_0 < 1$$

From (5) and using the expression for R_0, we see that

$$N_T < \frac{\gamma_H N_H \gamma_A N_A (\gamma_T + b_T)^2}{a^2 (p_H p_{TH} \gamma_A N_A + p_A p_{TA} \gamma_H N_H)}$$

From (6) we now have a bound on the number of tsetse flies in the population that will not be enough in starting an epidemic of sleeping sickness. Once condition (6) is satisfied, then the disease free state is achievable. Examining (6) closely, one observes (rather obviously) that N_T is directly proportional to the rate at which the flies become less infectious and their birth rate. Our motive will be to make N_T as small as possible and that will mean to reduce the infectiousness of the tsetse flies and also reduce their birth rate.

Now it is assumed that most tsetse flies become infectious throughout their life span, hence making $\gamma_T \approx 0$. Therefore, our target will be controlling b_T. In (Eradicating the Tsetse Fly in Zanzibar Island, 2005), a technology called **Sterile Insect Technique (STI)** was proposed as a lasting solution to the sleeping sickness scourge. Since females tsetse flies usually mate only once, if they are mated by a sterile male they will not produce any offspring, hence reducing their birth rate. SIT relies on rearing large numbers of insects in purpose built "fly factories", sterilizing the males with carefully controlled doses of gamma radiation and finally releasing them by airplane over the target area.

The radiation induces sterility, but the treated male flies can still fly and mate with wild females. Mating between the sterile released males and wild female tsetse flies produces no offspring. When sufficient sterile males are released over a long enough period, fertile mating does not occur and the population is eliminated.

Hence, irrespective of the parameter values of the variables in the denominator of the fraction in the right hand side of the inequality in equation (6) (we are particularly referring to p_H, p_A and a), as we make b_T very small with $\gamma_T \approx 0$, we shall be making N_T small, hereby reducing the number of tsetse flies in the target community. As we reduce b_T, the probability of infective contacts of the flies with humans (p_H) and domestic animals (p_A) will be small as well as the biting rate of the flies. The idea is as we reduce the flies (by say the SIT technique), there will be fewer flies to bite the victims and hence pass on the parasite to them (human or animal victims). If there are no flies, there will be no biting rate and hence no probability of passing on the parasite into the human or domestic animal host.

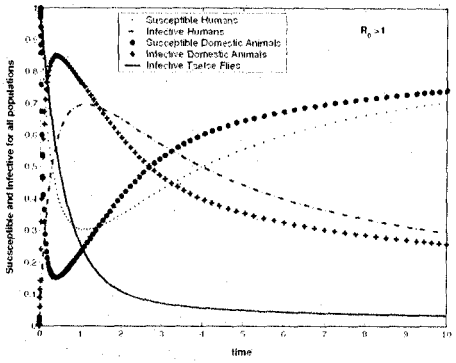

Fig. 1: Numerical solution for the dimensionalize equation (4) with $R_0 > 1$

Fig. 1 and 2 show the numerical solution of the dimensionalize equations (4) with $R_0 > 1$ and $R_0 < 1$ respectively.

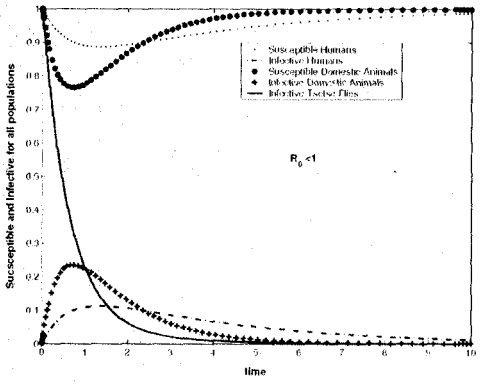

Fig. 2: Numerical solution of the nondimensional equation (4) with $R_0 < 1$

We introduce infective flies into a susceptible population of humans and domestic animals. We observe from figure (1) that the disease free state was not achieved since $R_0 > 1$. The population dynamics of the humans and domestic animals, whether susceptible or infective, depended critically on the population of infectious tsetse flies. As we reduce the number of infected flies in the system, the less infectives we have in both vertebrates' populations. Figure 2 helps confirm our earlier mathematical fact that as $R_0 << 1$ the infected human and domestic animal population reduces to zero.

EXTENDED MODEL

We shall be extending the model (1) we have just considered. In the model, we simply introduced infected insects into the population of humans and domestic animals. However, we recall that the East African *Trypanosomiasis* has wild game mammals as the main reservoir for the disease. Hence contacts between these animals and the tsetse flies will definitely play a role in the number of infectives in the human population and the domestic animal population. It is after the contacts with these wild animals that the cycle of cross-transmission starts.

Let M be the number of such wild games in any given locality and assume that the susceptible tsetse flies acquire infection from these wild animals at the rate $c\varphi MS_T$. We define c to be the number of potentially infective contacts that a susceptible tsetse fly has per day with any of the wild animals; φ is the probability that a tsetse fly will actually get an infection after a potentially infective contact with a wild animal. Assume, also that M is constant for that period when the contact is made and the cycle of infection begins.

The extended model becomes:

$$\frac{dS_H}{dt} = -\beta_H S_H I_T + \gamma_H I_H$$

$$\frac{dI_H}{dt} = \beta_H S_H I_T - \gamma_H I_H$$

$$\frac{dS_A}{dt} = -\beta_A S_A I_T + \gamma_A I_A$$

$$\frac{dI_A}{dt} = \beta_A S_A I_T - \gamma_A I_A$$

$$\frac{dS_T}{dt} = -(\beta_{1T} S_T I_H + \beta_{2T} S_T I_A) + \gamma_T I_T + b_T N_T - d_T S_T - c\varphi MS_T$$

$$\frac{dI_T}{dt} = (\beta_{1T} S_T I_H + \beta_{2T} S_T I_A) - \gamma_T I_T - d_T I_T + c\varphi MS_T$$

The variables and parameters remain as explained in Section 2. Also the human, domestic animals and tsetse fly population remains constant. Dimensionalizing the state variables as was done in Section 2 gives;

$$\frac{du_H}{dt} = -\gamma_H(k_1 u_H v_T - v_H)$$

$$\frac{dv_H}{dt} = \gamma_H(k_1 u_H v_T - v_H)$$

$$\frac{du_A}{dt} = -\gamma_A(k_2 u_A v_T - v_A)$$

$$\frac{dv_A}{dt} = \gamma_A(k_2 u_A v_T - v_A)$$

$$\frac{dv_T}{dt} = (\gamma_T + b_T)((k_3(1-v_T)v_H + k_4(1-v_T)v_A - v_T + k_5(1-v_T)) \quad (8)$$

where the parameters $k_1, k_2, k_3, \text{and } k_4$ remains as they are in Section 2. The additional parameter is $k_5 = \dfrac{c\varphi M}{(\gamma_T + b_T)}$. Hence we shall solve (8) together with $u_T = 1 - v_T$.

STEADY STATE ANALYSIS

Again we carry out the same analysis as was done for the first model and dealing with the equations for $\dfrac{dv_H}{dt}, \dfrac{dv_A}{dt} \text{ and } \dfrac{dv_T}{dt}$. We shall investigate the DFE point which can be obtained from the solutions of the equations $\dfrac{dv_H}{dt} = 0, \dfrac{dv_A}{dt} = 0, \dfrac{dv_T}{dt} = 0$.

There is a steady state of the system at $(v_H, v_A, v_T) = (0, 0, 0)$. To investigate its stability, the Jacobian of the system under consideration is

$$\begin{pmatrix} -k_1 v_T - 1 & 0 & k_1(1-v_H) \\ 0 & -k_2 v_T - 1 & k_2(1-v_A) \\ k_3(1-v_T) & k_4(1-v_T) & -k_3 v_H - k_4 v_A - k_5 - 1 \end{pmatrix}$$

For the point (0, 0, 0), J has the eigenvalues

$$\lambda_1 = -1,\ \lambda_2 = -1 - \frac{1}{2}k_5 + \frac{1}{2}\sqrt{k_5^2 + 4k_2k_4 + 4k_1k_3},\ \lambda_3 = -1 - \frac{1}{2}k_5 - \frac{1}{2}\sqrt{k_5^2 + 4k_2k_4 + 4k_1k_3}$$

In this case, the eigenvalues will all be negative if and only if $R_0 = k_1k_3 + k_2k_4 - k_5 < 1$, where R_0 is the basic reproduction ratio for the disease. Therefore the point (0, 0, 0) will be asymptotically stable if and only if $0 < R_0 < 1$. In this case the disease dies out after enough time has elapsed. For this model we see a much tighter condition on R_0. However, if $R_0 > 1$, then an introduction of

infected flies into the human and domestic animal population will trigger an outbreak of the East African sleeping sickness and that will lead to an epidemic.

In the case of this model, $R_0 = \frac{a^2 p_H p_{TH} N_T}{\gamma_H N_H (\gamma_T + b_T)} + \frac{a^2 p_A p_{TA} N_T}{\gamma_A N_A (\gamma_T + b_T)} - \frac{c\varphi M}{\gamma_T + b_T}$. Since the EASS dies if and only if $0 < R_0 < 1$ and using the expression for R_0, we observe that

$$N_T < \frac{\gamma_H N_H \gamma_A N_A (\gamma_T + b_T) - c\varphi M \gamma_H N_H \gamma_A N_A}{a^2 (p_H p_{TH} \gamma_A N_A + p_A p_{TA} \gamma_H N_H)} \qquad (9)$$

From (9) we have a bound on the number of tsetse flies in the target population that will not be enough in starting an epidemic of sleeping sickness. Comparing this to the bound in (6), we observe that apart from trying to place a severe control on b_T (as explained in Section 2), we must also place control on the contact rate of the tsetse flies with the animals that serve as reservoir for the parasites. One way this can be achieved is by targeting the animals for culling (Strategic Review of Traps and Targets for Tsetse, (2005) hereby reducing M. We need to ensure that the numerator in (9) is nonnegative, which will imply that

$$\gamma_T + b_T \geq c\varphi M .$$

Hence a strategy of eliminating the tsetse flies (apart from reducing their birth rate as explained in Section 2) will be restricting their contact with the wild animals that serve as reservoir to the parasites which invariably will mean making **c** as small as possible. This will be achieved by either spraying the entire park with insecticides that are not harmful to humans as well as the animals or culling these animals (reducing M). This is in harmony with strategies suggested in (Strategic Review of Traps and Targets for Tsetse, (2005).

Fig. 3 shows the numerical solution of the dimensionalize problem (8). Clearly the rate of contact of the tsetse flies with the animals that serve as reservoir of the disease is quite significant in affecting the disease cycle.

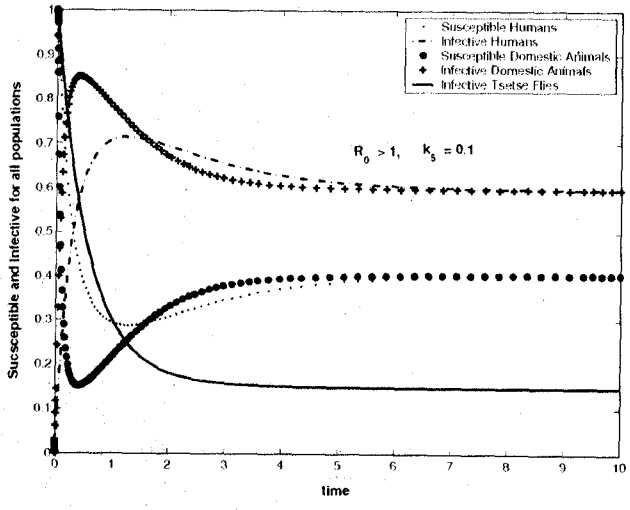

Fig. 3. The effect of infectious contacts between tsetse flies and wild animals that serve as reservoir for the disease with $R_0 > 1$.

That there exists a steady state that is stable when $R_0 > 1$ is obvious from Fig. 3 and Fig. 4. Endemicity is implied since $R_0 > 1$. In other words, once $R_0 > 1$, there will be an outbreak of the disease which will later settle into an endemic state after a long time has passed. In fact even when we decrease k_1, k_2, k_3 *and* k_4 significantly, and left k_5 unchanged, the result is what we see in Fig. 4. We see that endemicity is still there although on a much smaller scale than in Fig. 3. This clearly shows the significance of the contact rate between the flies and the animals serving as reservoirs for the parasite.

Fig. 5 shows that the infected humans as well as domestic animal population reduce to zero as $0 < R_0 << 10$. With R_0 very small, the introduction of the infected tsetse flies was not enough to start an epidemic. In fact only very little infectives were observed before their number reduced to zero.

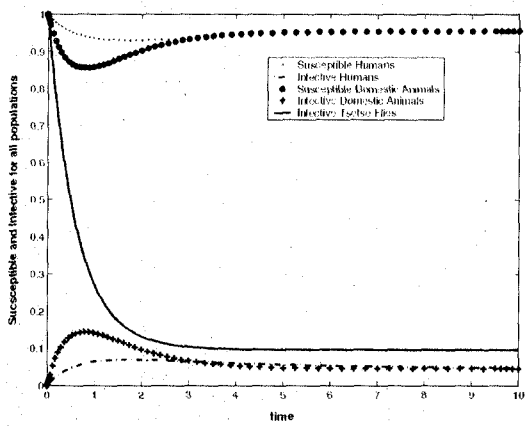

Fig. 4: Effects of contact rate between flies and reservoir animals with $R_0 > 1$.

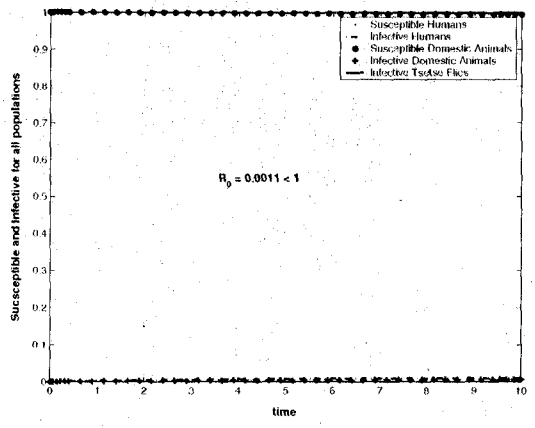

Fig. 5: Effects of contact rate between flies and reservoir animals with $0 < R_0 < 1$

DISCUSSION AND CONCLUSION

In this work, two models for the dynamics of populations affected by the acute disease, the East African Trypasonomiasis, or sleeping sickness are presented. The first model looks at the disease by examining the effect of the tsetse fly on both human and domestic animal populations. Obviously, reduction in the

infectious flies reduces infections in both humans and animals. The second model included the contact rate between the flies and the wild animals that serves as reservoir for the parasites. Here contact rate played a huge role in the whole disease cycle.

One of the reasons for formulating mathematical models of infectious diseases is to enable us design policies aimed at eradicating or at least controlling the spread of the disease. At the moment, there is neither a vaccine against the disease nor a drug available to *prevent* infection h sleeping sickness. There are however drugs for the *treatment* of the disease, but at present, the drugs are scarce, difficult to administer and sometimes dangerous and harmful to both human and domestic animal populations. Worst of all is the fact that there is no immunity to the disease (African Trypanosomiasis, 2002). Hence prevention of sleeping sickness requires avoiding contact with the tsetse fly. That invariably will mean controlling the vectors (tsetse flies) that cause the disease.

As was seen from the models, reducing N_T, the tsetse fly population, remains the best option for controlling the spread of the disease. This can be achieved by spraying insecticides in areas where there are significant amounts of the flies. Such chemicals should not be harmful to humans or domestic animals. Techniques like the STI described earlier and detailed in (Eradicating the Tsetse Fly in Zanzibar Island, 2005) can be used in controlling the birth rate of the tsetse flies in order to reduce their population. Also drugs should be made available to treat those having EASS.

Conclusively, vector control still remains the best strategy against the disease. People especially park attendants and villagers should be encouraged to put on protective wears that will not allow the flies get to their skins. In the main time we still need to understand more fully the dynamics of the flies and study their relationships with the wild animals. As we get a better understanding of the ecology and epidemiology of the tsetse flies this will definitely assist in improving mathematical models for the East African sleeping sickness.

REFERENCES

African Sleeping Sickness (2004). www. emedicine. com/med/topic2140.htm

African Trypanosomiasis (2001). www. Astdh pphe.org/infect/trypanosomiasis/default.

African Trypanosomiasis (2002). Available at www.cdc.gov/ncidod/dpd/parasites/trypanosomiasis/ default.

Bolton College. African Sleeping Sickness (Insect Science at Bolton College)

Webpage (2002). www.bc.edu/schools/cas/biology/research/insect/tsetse/.

Eradicating the Tsetse Fly in Zanzibar Island: A model project (2005). Department of Technical Co-operation, International Atomic Energy Agency. Available at www.iaea.or.at/worldatom/program/tc/.

Frank, S. A. (1999). A model for the sequential dominance of antigenic variants in African trypanosome infections. Proceedings of the Royal Society of London B 266:1397- 1401.

Health issues on the tsetse fly (2003). Available at www.cnn.com/health/ 9802/28/sudan. sleeping.sickness.

Nicholas F.B. (2003). Essential Mathematical Biology: Springer-Valerg London Limited.

Roger D.J. (1988). A general model for the African trypanosomiases.

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Species of the tsetse fly (2003). www.uen.org/ utahlink/activities/view-activities

Strategic Review of Traps and Targets for Tsetse and African Trypanosomiasis Control (2005). Available at www.who.int/tdr.

The Tsetse Fly (2001). www.encyclopedia. com/html/t1/tsetsefl.asp.

The tsetse fly (2001).www.robert.u-net.com/tsetse.htm.

Tsetse Fly (2004). www.biosci.ohio-state.edu/~parasite/glossina.html.

The UNICEF-UNDP-WORLD BANK-WHO Special report on the African

Trypanosomiasis Webpage (2003). Available at www.who.int/tdr/diseases/tryp.