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Mathematical models to simulate the East African *trypanosomiasis* population dynamics.

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Abstract

This paper presents mathematical models for the East African *trypanosomiasis* or sleeping sickness. It is aimed at modelling the population dynamics for the human and domestic animal victims as well as the dynamics of the tsetse fly population that acts as the vector that spreads the parasite causing this disease. Since sleeping sickness is caused by two protozoan parasites that are morphologically similar but cause dramatically different diseases in humans and domestic animals, this paper examines the East African sleeping sickness only. An extended model is provided to show the significance of infectious contacts between the tsetse flies and animals that serve as the reservoir for the parasite that causes this disease. Steady states for the models are also presented and analysed.

Keywords: Mathematical model, steady state, trypanosomiasis.

1.0 Introduction.

African sleeping sickness is caused by two protozoan parasites that are morphologically similar but cause dramatically different diseases. In humans, the East African sleeping sickness 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 [11]. Trypanosomiasis in cattle is caused by the parasites *Trypanosoma congolense and Trypanosoma vivax* and is also carried by the tsetse fly. Details on the disease and its economic impact on humans and domestic animals can be found in [5, 6, 8].

This paper seeks to model mathematically the population dynamics of the East African sleeping sickness (EASS). The paper by Roger [3] 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. Moreover the role of the wild animals that serves as reservoir for the parasites was not discussed in the model.

Research [4] has shown that there actually exist two morphologically similar parasites that cause different diseases though both parasites are carried by the tsetse fly. Hence this paper will concentrate only on the East African sleeping sickness, caused by one of the parasites. More so, we formulate a mathematical model that takes into consideration the role of the wild animals that serve as reservoirs for the parasites, a significant and very important role not discussed in [3]. East African sleeping sickness 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 [4, 7].

For the EASS case, the initial bite leaves a distinctive sore spot called a chancre. Symptoms, which appear one to four weeks after infection, may include swollen lymph nodes, irritability, fever, severe headache, fatigue, muscle and joint pain, and a skin rash. During the second stage of the disease, the parasite crosses the blood-brain barrier and attacks the central nervous system. Neurological complications include slurred speech, confusion, and difficulty with walking.

Seven species of tsetse flies in the genus *Glossina* act as vectors of the disease to humans [9, 12, 13]. The cycle begins when a fly bites an infected mammal 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 [10].

The EASS variety lives within African wild animals particularly in antelope (in savannah and woodland areas), such as bushbuck and in wild pigs and warthogs and these wild populations act as a reservoir for the parasites. Tsetse flies bite these wild animals and then infect any domestic animals or humans that they subsequently bite. Usually they cause no problems with the antelope's health [5].

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.

2.0 The mathematical model

To model EASS, it is important to keep track of the disease status for the human population, domestic animal population (the wild animals having the protozoan do not have any problem with the disease) as well as the tsetse fly population.

The EASS will be modelled as a SIS disease. This is because the sickness typically occurs in waves, whereby the patient seems to recover, only to relapse a few days later. This succession of outs of sickness continues until the infected person dies. This series of recovery and relapse is due to the parasite changing its surface coating in an attempt to avoid the immune system [1].

For the purpose of this work, the human population will 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.

Steady state for total eradication of the disease will be examined and see if it is feasible in practical terms.

2.1. Derivation of Model

The following populations will be considered: the human population, domestic animal population and the tsetse fly population.

Each of the populations will be divided into 2 compartments containing susceptible and infected. Since we are dealing with the EASS, we shall omit an incubation class for the human and domestic animal populations; hence a person or domestic animal that catches the disease becomes infectious instantaneously.

Let $N_H(t)$ be the density of the total number of humans at time t. Also let $N_A(t)$ be the density of the total number of domestic animals at time $\boldsymbol{t}$. Likewise, let $N_T(t)$ be the total number of tsetse flies at time t.

Since we are having two compartments for each population, we similarly state the densities of the total number of susceptible and infected in each populations at time t. Let $S_H(t)$, $S_A(t)$ and $S_T(t)$ denotes the densities of susceptible humans, domestic animals and tsetse flies at time t, respectively. Also let $I_H(t)$, $I_A(t)$ and $I_T(t)$ denote the densities of infected humans, domestic animals and tsetse flies at time $\boldsymbol{t}$ respectively.

Let us suppose that the average infectious period is γ_H^{-1} for humans and γ_A^{-1} for the domestic animals (the $\gamma's$ therefore stands for the rate at which the infected recover and become susceptible again). Assume that β_H is the infectious contact rate between humans and the tsetse flies. This will depend on two parameters: the biting rate, a of the tsetse flies and p_H, the probability that the susceptible becomes infected after each bite. Hence we have that $\beta_H = ap_H$. Likewise, let $\beta_A = ap_A$ be the contact rate between the domestic animals and the flies, with a as explained above and $\boldsymbol{p_H}$, the probability that the domestic animal bitten will get infected.

Also susceptible tsetse flies can get infected (i.e. carry the parasite) when they bite humans and domestic animals that already infected. Hence, let β_{1T} be the contact rate between susceptible flies and infected humans and let β_{2T} be the infectious contact rate between the flies and infected domestic animals. Using a similar argument as above, we have that $\beta_{1T} = ap_{TH}$ and $\beta_{2T} = ap_{TA}$, where p_{TH} and p_{TA} are the probabilities that a susceptible fly are will become infected on biting an infectious human and domestic animal respectively. So the β's represents the

infectious contact rate between the susceptible and infected which depends on the biting rate of the tsetse flies and the probability of getting infected. Of course this again depends on the amount of blood meals obtained by the flies from the host. We assume that there are enough hosts for the required blood meals.

We assume that the flies do not die of trypanosomiasis and that once they get infected, they becomes infectious throughout their life span (approximately 6 months).We shall ignore human and domestic animal birth and death rates.

The rate of change of S_H will be determined by the movement of newly infected out of this compartment and the entry of recovered individuals into the compartment. Also the rate of change of I_H will be determined by the inward movement of infected individuals and the outward movement of those that have recovered into S_H. The same explanation holds for the rate of change for S_A and I_A.

Likewise the rate of change of S_T will depend on the outward movement of initially susceptible tsetse flies that are now infected (due to contacts with infected humans and animals) and their death rate as well as new susceptible tsetse flies due to birth. Also the rate of change of I_T will be determined by the inward movement of the infected flies into the compartment and the outward movement due to death.

Putting all of these together, the governing equations for all populations in the different compartments are:

$$\frac{dS_H}{dt} = -\beta_H \frac{S_H}{N_H} I_T + \gamma_H I_H \tag{2.1}$$

$$\frac{dI_H}{dt} = \beta_H \frac{S_H}{N_H} I_T - \gamma_H I_H \tag{2.2}$$

$$\frac{dS_A}{dt} = -\beta_A \frac{S_A}{N_A} I_T + \gamma_A I_A \tag{2.3}$$

$$\frac{dI_A}{dt} = \beta_A \frac{S_A}{N_A} I_T - \gamma_A I_A \tag{2.4}$$

$$\frac{dS_T}{dt} = -\beta_{1T} S_T \frac{I_H}{N_H} - \beta_{2T} S_T \frac{I_A}{N_A} + b_T N_T - d_T S_T \tag{2.5}$$

$$\frac{dI_T}{dt} = \beta_{1T} S_T \frac{I_H}{N_H} + \beta_{2T} S_T \frac{I_A}{N_A} - d_T I_T \tag{2.6}$$

Each equation represents the rate of change, with respect to time, t of the different compartments. The first terms in (2.1)-(2.4) is the incidence of the disease in the human (equation (2.1) and (2.2)) and domestic animal (equations. (2.3) and (2.4)) populations while the last terms is the rate at which infected recover and become susceptible again. In equation (2.5), the first two terms represents the incidence of the disease among the tsetse fly population while the third and fourth term represents the birth and death rates for the susceptible flies, respectively. In (2.6), again the first two terms is the incidence for the tsetse fly population while the last term is the death rate for the infected flies. We assumed that the human population, animal population and the tsetse fly population is closed. Hence in addition to the governing equation we shall also be having that

$S_H + I_H = N_H$

$S_A + I_A = N_A$

$S_T + I_T = N_T$

As was stated earlier, the incubation period of the disease is neglected as symptoms starts to appear after one to four weeks of infection unlike in 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.

Next we nondimensionalise the dependent variables in equations (2.1) to (2.6). We scale quantities representing the susceptible and infective in each category with their respective total population size. Hence we shall be having that

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

Hence the governing equations (2.1) becomes,

$$\frac{du_H}{dt} = -\gamma_H (k_1 u_H v_T - v_H) \tag{2.7}$$
$$\frac{dv_H}{dt} = \gamma_H (k_1 u_H v_T - v_H) \tag{2.8}$$
$$\frac{du_A}{dt} = -\gamma_A (k_2 u_A v_T - v_A) \tag{2.9}$$
$$\frac{dv_A}{dt} = \gamma_A (k_2 u_A v_T - v_A) \tag{2.10}$$
$$\frac{du_T}{dt} = b_T (-k_3 u_T v_H - k_4 u_T v_A - u_T - 1) \tag{2.11}$$
$$\frac{dv_T}{dt} = b_T (k_3 u_T v_H + k_4 u_T v_A - v_T) \tag{2.12}$$

where $k_1 = \frac{\beta_H N_T}{\gamma_H N_H}$, $k_2 = \frac{\beta_A N_T}{\gamma_A N_A}$, $k_3 = \frac{\beta_{1T}}{b_T}$ and $k_4 = \frac{\beta_{2T}}{b_T}$, and $u_H + v_H = 1$, $u_A + v_A = 1$, $u_T + v_T = 1$.

We have a system of 6 coupled nonlinear ordinary differential equations to solve. However this can be reduced to a system of 5 coupled nonlinear ordinary differential equations with one algebraic equation. Hence we have the equations for $\frac{du_H}{dt}, \frac{dv_H}{dt}, \frac{du_A}{dt}, \frac{dv_A}{dt}$ and $\frac{dv_T}{dt} = b_T((k_3(1-v_T)v_H \quad + k_4(1-v_T) \quad v_A - v_T)$ to solve with $u_T = 1 - v_T$.

2.2. **Analytical Results**

To study the behaviour of the system of differential equations that describes the dynamics of the infected (i.e. equations (8), (10) and (12)), we find the equilibrium solutions, and linearize about these to examine their stability.

2.2. Equilibrium Solutions.

We now set each of the three differential equations to zero i.e. $\frac{dv_H}{dt} = 0$, $\frac{dv_A}{dt} = 0$, $\frac{dv_T}{dt} = 0$ and solve for v_H, v_A and v_T for which the system will no longer change. So we have that

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

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

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

There is a steady state (equilibrium point) of the system at $(v_H, v_A, v_T) = (0, 0, 0)$ 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). By intuition, there is also a non-trivial equilibrium point at $(v_H, v_A, v_T) = (v_H^*, v_A^*, v_T^*)$ which is rather complicated to write down in simple algebraic form.

2.3 Linearization of the system

To determine the behaviour of the infected populations near each of the equilibrium solutions, we need to compute the linearization of the system, which is obtained from the Jacobian matrix of the system under consideration. For the system of equations (2.8), (2.10) and (2.12), the Jacobian is the following:

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

If we substitute a set of equilibrium values for v_H, v_A and v_T in the matrix J, then the matrix J will represent the linearization of the system of differential equations under consideration about that equilibrium solution. We will now examine the two equilibrium solutions.

2.3.1. Equilibrium 1

For the solution (0,0,0), the eigenvalues of J was found to be $\lambda_1 = -1$, $\lambda_2 = -1 + \sqrt{(k_1 k_3 + k_2 k_4)}$ and $\lambda_3 = -1 - \sqrt{(k_1 k_3 + k_2 k_4)}$ which are all real numbers. Hence the stability of this point depends crucially on $R_0 = k_1 k_3 + k_2 k_4$. If $R_0 < 1$ the three eigenvalues will be negative, making the equilibrium solution (0, 0, 0) asymptotically stable otherwise the point is unstable. In other words, if $R_0 < 1$, 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. This means that obtaining a disease free situation will not possible. Hence the disease remains endemic.

Now $R_0 = \frac{\beta_H N_T}{\gamma_H N_H} . \frac{\beta_{1T}}{b_T} + \frac{\beta_A N_T}{\gamma_A N_A} . \frac{\beta_{2T}}{b_T}$ is the basic reproduction rate of the infection. 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. We see that R_{0H} is the number of secondary cases in humans that are expected to be produced by a single primary case (in humans) introduced into a wholly susceptible population (of tsetse flies and humans). The human case leads to $\frac{\beta_H N_T}{\gamma_H N_H}$ cases in the tsetse flies, each of which leads to $\frac{\beta_{1T}}{b_T}$ cases in humans. Also R_{0T} is the number of secondary cases in domestic animals that are expected to be produced by a single primary case (in domestic animals) introduced into a wholly susceptible population (of tsetse flies and domestic animals). The domestic animal case leads to $\frac{\beta_A N_T}{\gamma_A N_A}$ cases in the tsetse flies, each of which leads to $\frac{\beta_{2T}}{b_T}$ cases in the domestic animals.

Therefore the East African sleeping sickness dies if and only if $R_0 < 1$.

2.3.2. Equilibrium 2

By intuition, we know there exist a non-trivial steady state at $(v_H, v_A, v_T) = (v_H^*, v_A^*, v_T^*)$ which is rather complicated to write in simple algebraic form. However, the stability will again depend crucially on R_0. For the point to be stable (which implies endemicity since the infective in the human, domestic animals and tsetse fly population does not tend to zero as 'time' tends to infinity), clearly and obviously $R_0 > 1$. Hence the disease remains endemic in both the human and domestic animal populations.

3.0 Extended model

This work will now consider an extension of the model (2.1)-(2.6) just considered. In the model, infected insects were simply introduced into the population of humans and domestic animals. Recall that the East African sleeping sickness 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. Let us assume that the susceptible tsetse flies acquire infection from these wild animals at the rate $c\varphi MS_T$. Define $\boldsymbol{c}$ 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. Let us assume that M is constant for that period when the contact is made and the cycle of infection begins.

Hence the governing equation for the extended model becomes:

$$\frac{dS_H}{dt} = -\beta_H \frac{S_H}{N_H} I_T + \gamma_H I_H \tag{3.1}$$

$$\frac{dI_H}{dt} = \beta_H \frac{S_H}{N_H} I_T - \gamma_H I_H \tag{3.2}$$

$$\frac{dS_A}{dt} = -\beta_A \frac{S_A}{N_A} I_T + \gamma_A I_A \tag{3.3}$$

$$\frac{dI_A}{dt} = \beta_A \frac{S_A}{N_A} I_T - \gamma_A I_A \tag{3.4}$$

$$\frac{dS_T}{dt} = -\beta_{1T} S_T \frac{I_H}{N_H} - \beta_{2T} S_T \frac{I_A}{N_A} + b_T N_T - d_T S_T - c\varphi M S_T \tag{3.5}$$

$$\frac{dI_T}{dt} = \beta_{1T} S_T \frac{I_H}{N_H} + \beta_{2T} S_T \frac{I_A}{N_A} - d_T I_T + c\varphi M S_T \tag{3.6}$$

The equations remain as explained in Section 2. Also the human, domestic animals and tsetse fly population remains constant. If we nondimensionalise the system as done in Section 2, using the same non-dimensional variables while leaving the time scale unchanged, we shall have the non-dimensional problem (to be solved) for the extended model to be:

$$\frac{du_H}{dt} = -\gamma_H (k_1 u_H v_T - v_H) \tag{3.7}$$

$$\frac{dv_H}{dt} = \gamma_H (k_1 u_H v_T - v_H) \tag{3.8}$$

$$\frac{du_A}{dt} = -\gamma_A (k_2 u_A v_T - v_A) \tag{3.9}$$

$$\frac{dv_A}{dt} = \gamma_A (k_2 u_A v_T - v_A) \tag{3.10}$$

$$\frac{du_T}{dt} = b_T (-k_3 u_T v_H - k_4 u_T v_A - u_T - 1 + k_5 u_T) \tag{3.11}$$

$$\frac{dv_T}{dt} = b_T (k_3 u_T v_H + k_4 u_T v_A - v_T) \tag{3.12}$$

where the parameters $k_1, k_2, k_3,$ and k_4 remains as they are in Section 2. The additional parameter k_5. $= \frac{c\varphi M}{b_T}$ We have a system of 6 coupled nonlinear ordinary differential equations to solve. However this can be reduced to a system of 5 coupled nonlinear ordinary differential equations with one algebraic equation. Hence we have the equations for $\frac{du_H}{dt}, \frac{dv_H}{dt}, \frac{du_A}{dt}, \frac{dv_A}{dt}$ and $\frac{dv_T}{dt} = b_T((k_3(1-v_T)v_H + k_4(1-v_T)v_A - v_T + k_5(1-v_T))$ to solve with $u_T = 1 - v_T$.

3.1. **Analytical results**

The same analysis as was carried out in the first model will also be carried out for the extended model. Again the equations for analysis will be those of $\frac{dv_H}{dt}, \frac{dv_A}{dt}$ and $\frac{dv_T}{dt}$. The work shall investigate equilibrium solutions for, $\frac{dv_H}{dt} = 0,\ \frac{dv_A}{dt} = 0,\ \frac{dv_T}{dt} = 0$ i.e.

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

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

$b_T(k_3(1-v_T)v_H + k_4(1-v_T)v_A - v_T + k_5(1-v_T)) = 0$

There is a steady state of the system at $(v_H, v_A, v_T) = (0, 0, 0)$. Again by intuition, there is also a non-trivial equilibrium point at $(v_H, v_A, v_T) = (v_H^*, v_A^*, v_T^*)$ which is rather complicated to write down in simple algebraic form.

2.3. Linearization of the system

To determine the behaviour of the infected populations near each of the equilibrium solutions, we need to compute the linearization of the system, which is obtained from the Jacobian matrix of the system under consideration.

For the system of equations (3.8), (3.10) and (3.12), the Jacobian is the following:

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

If we substitute a set of equilibrium values for v_H, v_A and v_T in the matrix J, then the matrix J will represent the linearization of the system of differential equations under consideration about that equilibrium solution.

3.3.1. Equilibrium 1

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 as explained in Section 2. Therefore the point (0, 0, 0) will be asymptotically stable if and only if $R_0 < 1$. In this case the disease dies out after enough time has elapsed. For this model we see a much tighter condition on R_0. This though may really be practically unrealistic as it would mean carrying out the near impossible task of eradicating the tsetse flies totally and/or killing the entire wild, park animals that serve as reservoirs for the parasites causing the disease, leading to no contacts between the flies and these animals!

3.3.2. Equilibrium 2

Again by intuition, a non-trivial steady state exists at the point (v_H^*, v_A^*, v_T^*) where the stability will again depend crucially on R_0. Obviously the point will be stable if and only if $R_0 > 1$, hence the disease remains endemic.

4.0 Summary and conclusion

In this work, two models for the dynamics of populations affected by the acute disease, the African sleeping sickness (East African variant) are presented. The first model looks at the complete picture of the disease by examining the effect of the tsetse fly on both human population and domestic animal population in any given settlement where the flies exist. The second model is an extension of the first which includes the contact rate between the tsetse flies and the animals serving as reservoirs of the parasites. Here the infectious contact rate of tsetse flies to the animal plays a huge role in the whole disease cycle.

How then do the society/government at least control the disease, knowing that achieving a disease free state is almost impossible, suggesting endemicity? One of the reasons for making models of infectious diseases is to design policies aimed at eradicating or at least controlling them. Currently, there is neither a vaccine (so that vaccination could be a means of controlling or even eradicating the disease) nor a drug available to *prevent* infection with sleeping sickness. Also there is no immunity to the disease [7]. 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.

The most significant control policy is to reduce R_0, the basic reproductive ratio, below 1 for both models. This basically entails reducing N_T (the tsetse fly population) by the spraying of insecticides or chemicals in areas (villages, parks and so on) where the insects are rife. By implication this will also lead to a decrease in b_T.

Vector control still remains the best strategy against the disease. Restricting human and animal movements to regions where there are many wild animals that could have interactions with the tsetse flies and then pass on the parasites to the flies could be adopted. Also, people are encouraged to put on protective garments that will prevent the tsetse flies from biting them. In the main time further research may go into discovering drugs or vaccine to stop this deadly scourge.

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