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A Mathematical Model of Dracunculiasis Epidemic and Eradication

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Abstract*: We present a mathematical model for the spread of dracunculiasis with focus on three populations; human, vector and parasite. The reproductive number is obtained from next generation matrix and the stability analyses of disease-free and endemic equilibria are conducted. Simulation of the model is presented by solving the systems of the differential equations to explore the behaviour of the model using maple 14. The paper also analyzes key parameters to determine the effective intervention. The result of this paper shows that reducing the parasite birth rate is more effective than water treatment.*
Keywords:*Copepod, dracunculiasis, reproductive number, stability.*

I. Introduction

Dracunculiasis (Dracontiasis), more generally called Guinea Worm Disease (GWD), is a serious problem in various countries in Africa. It is a parasitic infection acquired by drinking water from ponds contaminated by cyclopoid copepods infected with third stage larvae of the parasite Dracunculusmedinensis. Worm emergence is through the skin after a year of entering the infected person and this is usally associated with secondary bacterial infection. It has been called a neglected disease of neglected people since it strikes remote farming populations who have been passed over by national development efforts [1]. Intervention and prevention techniques have been implemented in endemic areas to significantly reduce outbreaks. Dracunculiasis is a disease of poor rural communities where the population often has to obtain drinking water from ponds infested with water fleas called Cyclops. Guinea worm eggs are not directly infective to humans. They can remain active in water for about three days and die unless they are swallowed by a cyclops. Inside the cyclops, the guinea-worm larvae develop over a period of about two weeks into a larval stage that is infective to humans [2]. The Cyclops become inactive after infection and die early [3].

There are no symptoms during the year long incubation period. High susceptibility to tetanus through ulcers caused by the emerging worms in addition to habitual abortion in some pregnant women [4] has been associated with guinea-worm diseases. When the adult worms are immersed in water, they release hundreds of thousands of mature larvae to begin the cycle anew. Each infection lasts only one year, but more than one guinea worm may emerge simultaneously or sequentially over the course of weeks, depending on the number and intensity of infection the preceding year. Humans do not develop immunity, and there is no cure or vaccine for the infection. However, the worm can be removed by physically pulling the worm out which may take up to two months to complete as worm can grow up to a meter in length and only 1-2cm can be removed per day [5]. Dracunculiasis can only be prevented by teaching persons to always filter drinking water from unsafe sources through a fine cloth and to avoid entering such sources when they have a worm emerging or about to emerge from their bodies, by treating contaminated water with ABATE larvicide (temephos; BASF, Mount Olive, NJ) or by providing safe drinking water from underground sources.

Considerable progress has been made since 1986 in reducing the annual numberof reported dracunculiasis cases. The 1991 world health assembly (WHA) goal to eradicate dracunculiasis globally by 1995 was not achieved because of the limited funding available from international organization for support of technical and financial assistance to countries with endemic disease, and the limited time (4years) to meet the WHA goal [6]. In 2004, WHA established a new target date of 2009 for global eradication [7] despite considerable progress, that target also was not met, nevertheless, progress towards eradication continues. The number of cases of dracunculiasis worldwide reported by disease endemic countries to WHO and partner organizations decreased by 41 percent, from 1,797 cases in 2010 to 1,058 in 2011. As of June 2012, dracunculiasis remained endemic in four countries (Chad, Ethiopia, Mali and South Sudan [7]. The 395 cases reported and 219 villages reporting cases globally during January to June 2012 represent reductions of 51% and 39%, respectively, from the 807 cases reported and 358 villages that reported cases during January to June 2011. Of the 395 cases reported during January to June 22012, 99% were from South Sudan [7].

Few researchers have contributed towards the mathematical study of the eradication of dracunculiasis (guinea worm) diseases.Adetunde [8] investigated the current pattern of dracunculiasis disease in the Northern region of Ghana. He analysed the data from the region and wrote a time series model for the purpose of prediction. From his analysis it was observed that the number of Guinean infection cases reduce with time and

concluded that if the trend continues then there is likelihood that the guinea worm disease will be completely eradicated.

Recently, KathryhLink [9] in her M.Scthesis, highlighted compartmental modelling of the biological description of the disease. The model provides the basis for examining the guinea worm diseases host-microparasite interaction. An algebraic solution to disease-free equilibrium was found and a numerical stability analysis of the solution was conducted. Using next generation matrix, she determined the reproductive number R_0, which enabled her to discover that the disease-free equilibrium is stable provided the people's visitation rate to the river/water body is reduced.

Robert J. Smith et al., [5] developed a mathematical model of guinea worm disease. Impulsive differential equations were used to evaluate the effectiveness of chlorination. Latin Hypercube sampling was used to determine the practical effectiveness of three control parameters (education, filtration and chlorination). Despite the theoretical potential of chlorination to complete the eradication of the disease, education is far more effective.

In this paper we formulate a mathematical model to represent the spread of guinea worm disease among three different populations. We analyse the model for two intervention parameters, ξ_L (water treatment)andλ_0 (education) to check the more effective intervention.

The rest of this paper is organised as follows. Section 2 is devoted to model formulation. In section 3, we analyse the model and state the conditions that guarantee the stability of the disease-free and endemic equilibria, then the model is solved numerically. Conclusion is made in Section 4.

II. Model Formulation

Three different populations are considered in this paper, human, parasite and vector populations. The human population is divided into three compartments (or states) containing susceptible human S_H, exposed human $E_H$and infected human $I_H(N_H = S_H + E_H + I_H)$. When a man ingests an infected copepod, he becomes exposed and later (after about one year when the guinea worm larva has fully grown) becomes infected.

In this model we assume that more than one guinea worm can emerge simultaneously or sequentially over the course of the weeks, depending on the number and the intensity of infection the preceding year.

No human death occurs due to guinea worm disease. The susceptible human state S_H gains individuals through birth $\eta_H N_H\left(1-\frac{N_H}{K_H}\right)$, and recovery from infection κI_H. A loss of individual is as a result of death $\xi_H S_H\left(1-\frac{N_H}{K_H}\right)$ and infection $\varepsilon_C\beta\left(\frac{I_C}{\Phi_c}\right)S_H$. The exposed human state E_H gains individuals through infection $\varepsilon_C\beta\left(\frac{I_C}{\Phi_c}\right)S_H$ and loses individuals when they become infected αE_H and to natural death $\xi_H I_H\left(1-\frac{N_H}{K_H}\right)$. The infected human I_H gains individuals when exposed individuals become infected and loses individuals when they die $\xi_H I_H\left(1-\frac{N_H}{K_H}\right)$ or recover κI_H.

The susceptible copepod state S_C gains more individuals only through birth $\eta_C S_H\left(1-\frac{N_C}{K_C}\right)$. The population loses copepods through natural death$\xi_C S_C\left(1-\frac{N_C}{K_C}\right)$, consumption by human $\beta\left(\frac{S_C}{\Phi_C}\right)N_H$, and to infection by guinea worm larvae, $\varepsilon_L\gamma\left(\frac{L}{\Phi_L}\right)S_C$, $(N_C = S_C + I_C)$. The infected copepod state I_C loses individual through death $\xi_C' I_C\left(1-\frac{N_C}{K_C}\right)$and consumption by human$\beta\left(\frac{I_C}{\Phi_C}\right)N_H$. Copepod can never recover from infection. In this paper it is assumed that the amount of copepods consumed per time by maximum population of human is less than the saturation population of copepods per time$\left(i.e.\,0 < \beta K_H < \Phi_C(\eta_C - \xi_C')\right)$.

The guinea worm population is represented by both eggs and larvae. The egg state E gains more individual by the release of eggs from adult worms existing in human host $\lambda_0\lambda_1 I_H$. The population loses eggs through natural death $\xi_E E$ and hatching ωE into the larvae. It is assumed that $\omega > \xi_E$. The larvae state L gains more larvae by hatching of eggs $\omega\sigma E$. Losses occur due to natural death $\xi_L L$ and consumption of larvae by copepod$\gamma\left(\frac{L}{\Phi_L}\right)N_C$.

Thus the model is

$$\frac{dS_H}{dt} = (\eta_H N_H - \xi_H S_H)\left(1-\frac{N_H}{K_H}\right) + \kappa I_H - \varepsilon_C\beta\left(\frac{I_C}{\Phi_C}\right)S_H \qquad 2.1$$

$$\frac{dE_H}{dt} = \varepsilon_C\beta\left(\frac{I_C}{\Phi_C}\right)S_H - \alpha E_H - \xi_H E_H\left(1-\frac{N_H}{K_H}\right) \qquad 2.2$$

$$\frac{dI_H}{dt} = \alpha E_H - \kappa I_H - \xi_H I_H\left(1-\frac{N_H}{K_H}\right) \qquad 2.3$$

$$\frac{dE}{dt} = \lambda_0\lambda_1 I_H - (\omega + \xi_E)E \qquad 2.4$$

$$\frac{dL}{dt} = \omega\sigma E - \xi_L L - \gamma\left(\frac{L}{\Phi_L}\right)N_C \quad 2.5$$

$$\frac{dS_C}{dt} = (\eta_C N_C - \xi_C S_C)\left(1 - \frac{N_C}{K_C}\right) - \varepsilon_L\gamma\left(\frac{L}{\Phi_L}\right)S_C - \beta\left(\frac{S_C}{\Phi_C}\right)N_H \quad 2.6$$

$$\frac{dI_C}{dt} = \xi_L\gamma\left(\frac{L}{\Phi_L}\right)S_C - \beta\left(\frac{I_C}{\Phi_C}\right)N_H - \xi_C' I_C\left(1 - \frac{N_C}{K_C}\right) \quad 2.7$$

Since the model monitors changes in the human, cyclops and parasite populations, the variables and the parameters are assumed to be non-negative for all $t \geq 0$. Therefore (2.1) – (2.7) will be analysed is a suitable feasible region $\Re$ of biological interest. We have the following lemma on the region system (2.1) – (2.7) are resisted to.

Lemma 2.1: The Feasible region $\Re$ defined by

$$\Re = \{(S_H(t), E_H(t), I_H(t), E(t), L(t), S_C(t), I_C(t)) \in R_+^7 : N_H(0) \leq N_H(t) \leq K_H, \; 0 \leq N_H(t) < K_C\}$$

with initial conditions

$S_H(0) \geq 0, E_H(0) \geq 0, I_H(0) \geq 0, E(0) \geq 0,\ L(0) \geq 0, S_C(0) \geq 0,\ I_C(0) \geq 0$is positive invariant for system (2.1) – (2.7).

Proof: Adding (2.1) – (2.3) we obtain

$$\frac{dN_H}{dt} = \left(1 - \frac{N_H(t)}{K_H}\right)(\eta_H - \xi_H)N_H(t)$$

The assumption that $N_H(0) \leq K_H$ implies

$$N_H(t) = \left\{\begin{matrix} \frac{K_H N_{H(0)}}{(K_H - N_{H(0)})\exp(-(\eta_H - \xi_H)t) + N_H(0)} if N_H(0) < K_H \\ K_H if N_H(0) = K_H \end{matrix}\right\} \leq K_H$$

Using the facts that $N_H(t) \leq K_H$ and $\eta_H \geq \xi_H$ in (2.8) we have

$$\frac{dN_H}{dt} \geq 0 \implies N_H(t) \geq N_H(0)$$

thus, $N_H(0) \leq N_H(t) \leq K_H$.

Similarly, adding (2.6) and (2.7) we obtain

$$\frac{dN_C}{dt} = \left(1 - \frac{N_C}{K_C}\right)(\eta_C N_C - \xi_C S_C - \xi_C' I_C) - \beta\left(\frac{N_C}{\Phi_C}\right)N_H$$

Since $\xi_C' > \xi_C$ and $\beta\left(\frac{N_C}{\Phi_C}\right)N_H \geq 0$ we have

$$\frac{dN_C}{dt} < \left(1 - \frac{N_C(t)}{K_C}\right)(\eta_C - \xi_C)N_C$$

which follows that

$$N_C(t) < \frac{K_C N_C(0)}{(K_C - N_C(0))\exp(-(\eta_C - \xi_C)t) + N_C(0)} < K_C$$

Since $N_C(0) < K_C$.

Table 1: State variables

S_H	Number of susceptible humans
E_H	Number of exposed
I_H	Number of infected humans
E	Number of guinea worm eggs
L	Number of guinea worm larvae
S_C	Number of susceptible copepod
I_C	Number of infected copepod

Table 2: List of parameters

η_H	Human birth rate $\left(\frac{1}{\text{time}}\right)$
ξ_H	Human death rate $\left(\frac{1}{\text{time}}\right)$
κ	Recovery rate $\left(\frac{1}{\text{time}}\right)$
ε_H	Human infection fraction $\left(\frac{\text{human infected}}{\text{copepod}}\right)$
β	Copepod consumption rate $\left(\frac{\text{copepod consumed}}{\text{human} \times \text{time}}\right)$
α	Worm emergence rate $\left(\frac{1}{\text{time}}\right)$
λ_0	Visitation rate $\left(\frac{\text{visits to the water}}{\text{time}}\right)$
λ_1	Egg release rate $\left(\frac{\text{egg released}}{\text{human} \times \text{rate}}\right)$
ξ_E	Natural death rate of eggs $\left(\frac{1}{\text{time}}\right)$
ω	Hatching rate $\left(\frac{1}{\text{time}}\right)$
σ	Ratio of matured larvae to the number of eggs hatched $\left(\frac{\text{larvae survived}}{\text{egg}}\right)$
ξ_L	Natural death rate of larvae $\left(\frac{1}{\text{time}}\right)$
γ	Larvae consumption rate $\left(\frac{\text{larvae consumed}}{\text{copepod} \times \text{time}}\right)$
ε_L	Copepod infection fraction $\left(\frac{\text{copepod infected}}{\text{larvae}}\right)$
ξ_C	Natural death rate of copepod $\left(\frac{1}{\text{time}}\right)$
$\xi_C^{'}$	Death rate of infected copepod $\left(\frac{1}{\text{time}}\right)$
η_C	Copepod birth rate $\left(\frac{1}{\text{time}}\right)$
Φ_C	Copepod saturation constant (copepod)
Φ_L	Larvae saturation constant (larvae)

III. Equilibrium and Stability Analysis

In this section, equilibrium and stability analysis of the model are discussed. When modelling infectious diseases, the most important issue that arises is whether the disease spread could attain pandemic level or it could be wiped out. To have a better understanding of the dynamics of the disease, equilibrium and stability analysis is performed.

3.1 Disease-Free Equilibrium

For the disease-free equilibrium, we set the disease states and the left-hand side of (2.1)-(2.7) to zero. The resulting system is solved which is given to $\pi_0 = (S_H^*, 0,0,0,0, S_C^*, 0)$

$$S_H^* = K_H$$

$$S_C^* = K_C\left(1 - \frac{\beta K_H}{\Phi_C\left(\eta_C - \xi'_C\right)}\right) < K_C$$

The last inequality holds since $0 < \beta K_H < \Phi_C\left(\eta_C - \xi_C^{'}\right)$

We obtain the reproductive number R_0 by expressing (2.1) – (2.7) as the difference between the rate of new infection in each infected compartment F and the rate of transfer between each infected compartment G.

$$\begin{bmatrix} \frac{dE_H}{dt} \\ \frac{dI_H}{dt} \\ \frac{dE}{dt} \\ \frac{dL}{dt} \\ \frac{dI_C}{dt} \end{bmatrix} = F - G = \begin{bmatrix} \varepsilon_C\beta\left(\frac{I_C}{\Phi_C}\right)S_H \\ 0 \\ \lambda_0\lambda_1 I_H \\ 0 \\ \varepsilon_L\gamma\left(\frac{L}{\Phi_L}\right)S_C \end{bmatrix} - \begin{bmatrix} \alpha E_H - \xi_H E_H\left(\frac{E_H+I_H}{K_H}\right) \\ -\alpha E_H + \kappa I_H + \xi_H I_H\left(\frac{E_H+I_H}{K_H}\right) \\ (\omega + \xi_E)E \\ -\omega\sigma E + \xi_L L + \gamma\left(\frac{L}{\Phi_L}\right)N_C \\ \beta\left(\frac{I_C}{\Phi_C}\right)N_H + \xi_C^{'}I_C\left(1 - \frac{N_C}{K_C}\right) \end{bmatrix}$$

The Jacobian matrices J_F and J_G of F and G are found about π_0.

$$T = J_F J_G^{-1} = \begin{bmatrix} 0 & 0 & 0 & 0 & \frac{\varepsilon_C\beta S_C^* K_C}{\beta S_C^* K_C + \xi_C^{'}\Phi_C S_C^* - \xi_C^{'}\Phi_C S_C^*} \\ 0 & 0 & 0 & 0 & 0 \\ \frac{\lambda_0\lambda_1}{K} & \frac{\lambda_0\lambda_1}{K} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{\xi_L\gamma S_C^*\omega\sigma}{(\omega+\xi_E)(\xi_L\Phi_L+\gamma S_C)} & \frac{\xi_L\gamma S_C^*}{\xi_L\Phi_L+\gamma S_C} & 0 \end{bmatrix}$$

$R_0$is the maximum eigenvalue of T given as

$$R_0 = \sqrt[3]{\frac{\varepsilon_L \gamma S_C^* \omega \sigma \lambda_0 \lambda_1 \xi_C \beta K_H K_C}{\kappa(\omega+\xi_E)(\xi_L \Phi_L + \gamma S_C^*)(\beta S_H^* K_C + \xi_c' \Phi_C S_C^* - \xi_c' \Phi_C S_C^*)}}$$

Clearly, $R_0 \geq 0$ since$K_C \geq S_C^*$

Theorem 3.1: If one of the diseased classes of an equilibrium point of the system is zero, then all the diseased classes are zero.

Proof: At equilibrium, the left sides of (2-1) – (2.7) are set to zero. Suppose $I_H = 0$ in (2.4), $(\omega + \xi_E) > 0$implies $E = 0$. In (2.5) we have $\left(\xi_L + \frac{\gamma N_C}{\Phi_L}\right) L = 0$, but $\left(\xi_L + \frac{\gamma N_C}{\Phi_L}\right) > 0$ by our assumption, therefore $L = 0$. It follows from (2.7) that $I_C = 0$since β, I_C, Φ_C, N_H, ξ_c',N_C and K_C are positive. Finally, we have from (2.2) that $E_H = 0$if $I_C = 0$.

Table 3: Parameter values for the disease-free area (Ghana) and endemic area (Ethiopia) with time in days

Parameter	Diseases free area	Endemic area	Reference	Parameter	Diseases free area	Endemic area	Reference
η_H	8.767×10^{-5}	1.055×10^{-1}	[10]	ξ_H	2.11×10^{-5}	2.548×10^{-5}	[10]
κ	0.0222	0.0222	calculated	ε_C	0.004	0.024	calculated
β	0.5	2	Calculate	α	0.00274	0.00274	calculated
λ_0	0.002	0.02	Assumed	λ_1	3000000	3000000	[2]
ξ_E	0.333	0.333	[2]	ω	0.072	0.072	[9]
σ	0.4	0.4	Calculate	ξ_L	0.0333	0.0333	[3]
γ	3	3	[3]	ε_L	0.1	0.1	assumed
ξ_C	0.005	0.005	[2]	η_C	0.75	0.75	[2]
ξ'_C	0.125	0.125	[3]	Φ_C	200000	200000	
K_H	1000	1000	assumed	K_C	100000	100000	assumed
Φ_L	500000	5000000					

3.2 Bifurcation

We consider two parameters λ_0 and ξ_L as means of intervention. Education is the intervention represented by λ_0 and continuous water treatment with the use of larvicide by ξ_L. By reducing the visitation rate, our model reveals that π_0 is stable if $0 < \lambda_0 < 0.00115$.

Water treatment with the use of larvicide increases the death rate of guinea worm larva. Our model describes that π_0 is stable if ξ_L>0.1016.

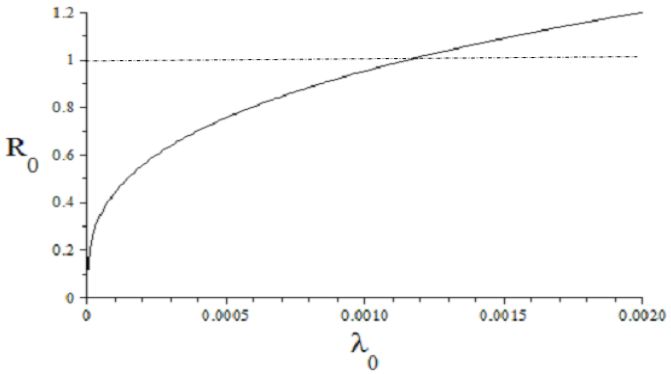

Figure 1: Intervention parameter λ_0 (education)

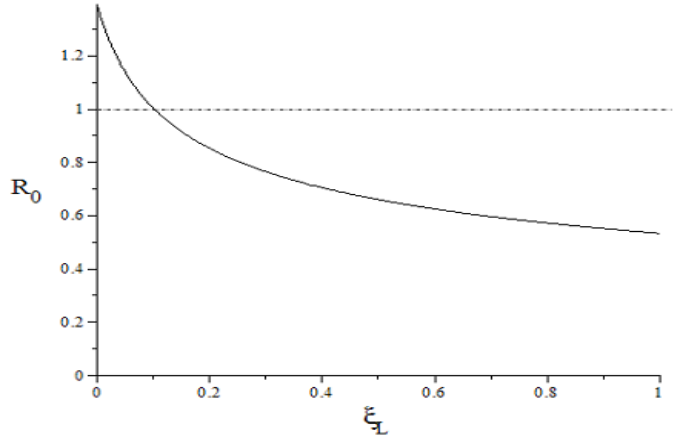

Figure 2: Intervention parameter ξ_L (water treatment)

3.3 Endemic Equilibrium

Theorem 3.2: If $\lambda_0\lambda_1 I_H < (\omega + \xi_E)E, \quad t \geq 0$, there exist three equilibria

$\pi_1 = (K_H, 0,0,0,0,0,0)$

$\pi_2 = (0,0,0,0,0,K_C,0)$ and

$\pi_3 = (K_H, 0,0,0,0, S_C^*, 0)$

where $S_C^* = K_C\left(1 - \frac{\beta K_H}{\Phi_C(\eta_C - \xi'_C)}\right)$. π_1 is unstable while π_2 and π_3 are locally stable.

Proof: Set the left side of (2.1)-(2.7) to zero. Putting $\lambda_0\lambda_1 I_H = c(\omega + \xi_E)E, 0 \leq c < 1$, for $t \geq 0$, in (2.4), we have $E = 0$. This implies, by Lemma 2.1 and Theorem 3.1, that $E_H = I_H = L = I_C = 0$, thus, (2.1) – (2.7) is reduced to

$$0 = (\eta_H - \xi_H)S_H\left(1 - \frac{S_H}{K_H}\right) \qquad 3.1$$

$$0 = (\eta_C - \xi_C)S_C\left(1 - \frac{S_C}{K_C}\right) - \beta\left(\frac{S_C}{\Phi_C}\right)S_H \qquad 3.2$$

Neglecting the trivial case, π_1, π_2 and π_3 are obtained from (3.1) and (3.2).

We check the stabilities of π_1 and π_3 (because $\pi_3 = \pi_2$ when $K_H = 0$) by finding the eigenvalues of the Jacobian matrices of system (2.1)-(2.7) evaluated at π_1 and π_2 respectively [11,12].

To check the stability of π_1, we have the Jacobian matrix

$$J_{\pi_1} = \begin{bmatrix} -\eta_H + \xi_H & -\eta_H + \xi_H & \Gamma & 0 & 0 & 0 & -\frac{\varepsilon_C \beta K_H}{\Phi_C} \\ 0 & -\alpha & 0 & 0 & 0 & 0 & \frac{\varepsilon_C \beta K_H}{\Phi_C} \\ 0 & \alpha & -\kappa & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \beth & 0 & 0 & 0 \\ 0 & 0 & 0 & \omega\sigma & -\xi_L & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \daleth & \eta_C \\ 0 & 0 & 0 & 0 & 0 & 0 & -\frac{\beta K_H}{\Phi_C} - \xi'_C \end{bmatrix}$$

where

$\Gamma = -\eta_H + \xi_H + \kappa$

$\beth = (c-1)(\omega + \xi_E)$

$\daleth = \eta_C - \frac{\beta K_H}{\Phi_C} - \xi_C$

and the eigenvalues are

$$y_1 = \begin{pmatrix} -\kappa \\ -\alpha \\ -\frac{\beta K_H}{\Phi_C} - \xi'_C \\ \eta_C - \frac{\beta K_H}{\Phi_C} - \xi_C \\ -\xi_L \\ (c-1)(\omega + \xi_E) \\ -\eta_H + \xi_H \end{pmatrix}$$

π_1 is not stable since $\eta_C - \frac{\beta K_H}{\Phi_C} - \xi_C > 0$

For π_3, we have the Jacobian matrix

$$J_{\pi_3} = \begin{bmatrix} -\eta_H + \xi_H & -\eta_H + \xi_H & \Gamma & 0 & 0 & 0 & -\frac{\varepsilon_C \beta K_H}{\Phi_C} \\ 0 & -\alpha & 0 & 0 & 0 & 0 & \frac{\varepsilon_C \beta K_H}{\Phi_C} \\ 0 & \alpha & -\kappa & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \beta & 0 & 0 & 0 \\ 0 & 0 & 0 & \omega\sigma & -\xi_L - \frac{\gamma S_C^*}{\Phi_L} & 0 & 0 \\ -\frac{\beta S_C^*}{\Phi_C} & -\frac{\beta S_C^*}{\Phi_C} & -\frac{\beta S_C^*}{\Phi_C} & 0 & -\frac{\varepsilon_L \gamma S_C^*}{\Phi_L} & \Theta & \Psi \\ 0 & 0 & 0 & 0 & \frac{\varepsilon_L \gamma S_C^*}{\Phi_L} & 0 & \Xi \end{bmatrix}$$

where

$$\Theta = (\eta_C - \xi_C)\left(1 - \frac{2S_C^*}{K_C}\right) - \frac{\beta K_H}{\Phi_C}$$

$$\Psi = \eta_C - \frac{(2\eta_C - \xi_C)S_C^*}{K_C}$$

$$\Xi = -\frac{\beta K_H}{\Phi_C} - \xi_C'\left(1 - \frac{S_C^*}{K_C}\right)$$

and the eigenvalues are

$$y_2 = \begin{pmatrix} -\kappa \\ -\alpha \\ -(\eta_C - \xi_C) + \frac{\beta K_H}{\Phi_C} \\ -\frac{\beta K_H(\eta_C - \xi_C + \xi_C')}{\Phi_C(\eta_C - \xi_C)} \\ (c-1)(\omega + \xi_E) \\ -\eta_H + \xi_H \\ -\xi_L - \frac{\gamma K_C(\Phi_C(\eta_C - \xi_C) - \beta K_H)}{\Phi_L \Phi_C(\eta_C - \xi_C)} \end{pmatrix}$$

Therefore, π_3 is locally stable when $c < 1$.

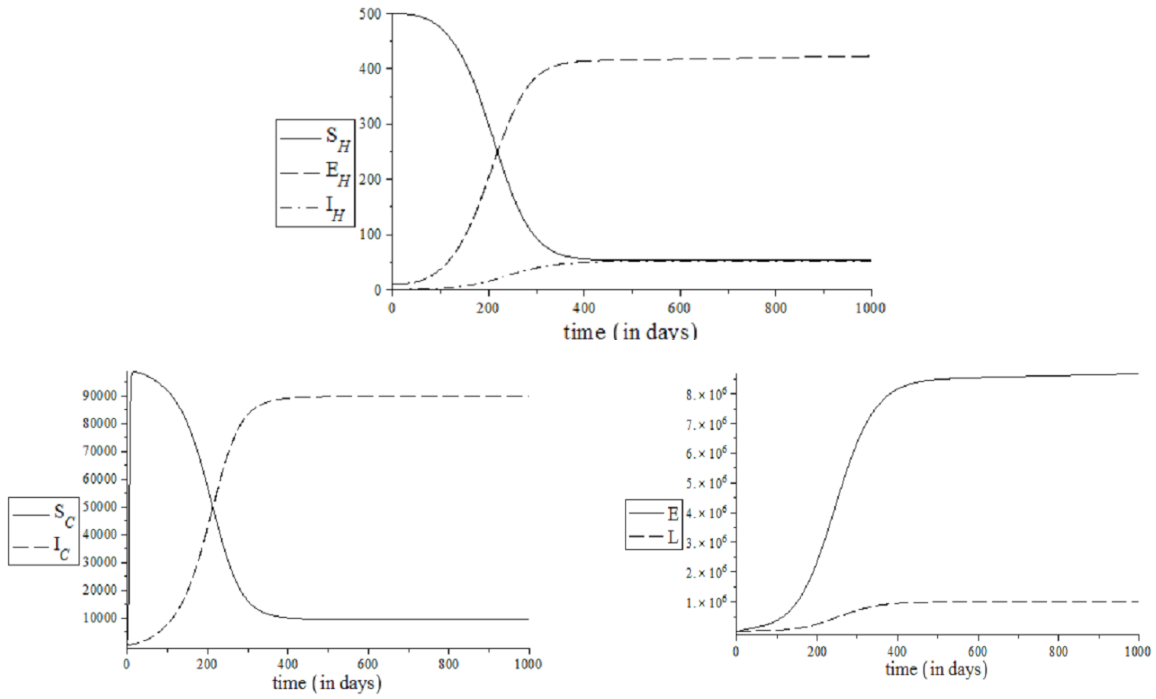

Figure 3: $S_H(0) = 500, E_H(0) = 10, I_H(0) = 500, E(0) = 10000, L(0) = 5000, S_C(0) = 1000, I_C(0) = 500$

We stimulated the endemic equilibrium using the parameter values in Table 3. The results show that the disease is present in all the population classes.

To check Theorem 3.2, we stimulated the endemic equilibrium with $\lambda_0 = 0$. The results agree with Theorem 3.2. This means that if the education about the disease is effective and the visitation rate is reduced, the disease will be wiped out.

Using the data in Table 3, we stimulated the case when the intervention parameter ξ_L (i.e. water treatment) is effective. It was observed that if the water treatment is effective to the level that the life span of larvae is reduced to 147minutes (i.e. ξ_L=9.796 per day), the population of guinea worm egg remains in the system but other disease classes decrease as time increases.

IV. Conclusion

We have highlighted a compartmental modelling approach for three different populations-human, vector and parasite. It is assumed that the human and vector populations grow logistically and parasite population increases by infected human's visitation to the river. The model is analysed for disease-free and endemic equilibria. Local stability of the non-trivialequilibria in both cases is guaranteed only under certain conditions.

For disease-free equilibrium, keeping the intervention parameter values in the value intervals given in section 3.2 allows the system to remain disease-free, the endemic equilibriaπ_2 and π_3 are stable when $\lambda_0\lambda_1 I_H(t) \leq (\omega + \xi_E)E(t)$. It is concluded from the analysis and simulation that if the intervention parameter (visitation rate) $\lambda_0 \leq \frac{(\omega+\xi_E)E}{\lambda_1 I_H}(I_H \neq 0)$, the spread of disease decreases and could be wiped out. By computer simulation it is shown that if the larvicide is effective (i.e. ξ_L is increased) to the point when $\frac{dL}{dt} \leq 0$, then the endemic equilibrium is locally stable.

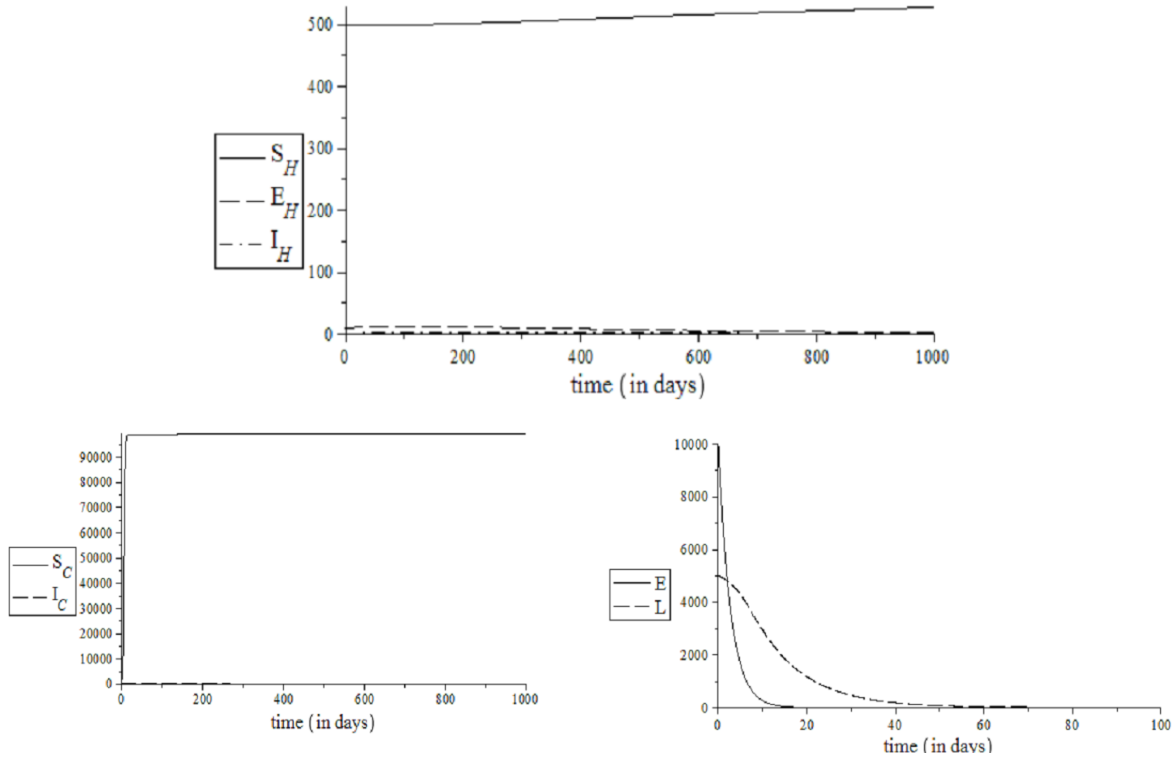

Figure 4: $\lambda_0 = 0, S_H(0) = 500, E_H(0) = 10, I_H(0) = 500, E(0) = 10000, L(0) = 5000, S_C(0) = 1000, I_C(0) = 500$

It is also found by simulation that reducing the parasite birth rate (which can be achieved by educating people not to put infected limbs into the drinking water) is more effective than water treatment. This model can be a useful tool in the control of the spread of dracunculiasis and the understanding of the model formulation can help in modelling any other water-borne disease.

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