Analysis of the dynamics of rabies in North Shewa, Ethiopia

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Abstract. In this paper, authors proposed and developed a deterministic model that explains the dynamics of rabies infection in human and dog populations in North Shewa, Ethiopia. The rabies model was analysed by determining a feasible region, positivity of the solution set, basic reproductive number, equilibrium points and their stability. Next Generation Matrix approach was employed in computing the basic reproduction number and its entirely dependent on the parameters of dog population. Its the threshold value that determines the dynamics of rabies transmission in North Shewa, Ethiopia. It was established that whenever $R_0 < 1$, rabies will die out from the population and whenever $R_0 > 1$, rabies persists in the population. Contribution each parameter was conducted using sensitivity analysis of the parameter values of the basic reproductive number. Some parameters were more sensitive and responsible for the spread of rabies in North Shewa region of Ethiopia. Moreover, stability analysis approach was employed to determine the disease free and endemic equilibrium of the rabies model. The results of the numerical simulation showed that as the contact rate and infecton rate increases, the disease spread faster. Moreover, as the removal rate and treatment rate increases, the disease spread decreases.

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1. Introduction

Rabies is an acute and fatal zoontic disease remains one of the most feared and important threats public health around the world [1]. Rabies virus also circulates among domesticated animals and among various bat species. Despite geographic and ecological overlap in the ranges of many of these species, the implications of multispecies host susceptibility and the community ecology of rabies has rarely been examined [2].

The Ethiopian Health and Nutrition Research Institute, the current Ethiopian Public Health Institute, indicated that human rabies has been reported in Ethiopia in 1903 for the first time. Ethiopia is among high burden African countries in regard to human rabies virus exposure since ancient times [3]. Among the first symptom of the rabies pain, burning or numbness at the site of the infections, the production of large quantities of saliva and tear coupled with an inability to speak or swallow are typical during the later stage of the diseases; this can be result in hydro phobia in which the animal difficulty swallowing because the throat and jaw become slowly paralyzed, show panic when presented liquid to drink and cannot quench it thirst [4].

The rabies is manly transmitted from animal to human through close contact with infected saliva via bite scratch and invariably results in death [5]. Domestic and wild animals can potentially transmit the disease to humans mainly through biting [5]. The transmission of rabies virus usually begins when infected saliva of a host is passed to an uninfected animal [6].The most common mode of rabies virus transmission is through the bite and virus containing saliva of an infected host.

China and India have the most and second most reported cases, respectively [7]. Despite, the preventable nature of the disease and existence of effective and economical control strategy [8], rabies remain as one of the major public health problem resulting in an estimate loss of approximate 60,000 lives in the worldwide each year which almost all of the case belong to Africa and Asia [5].

Dogs were the reasons for 97% of rabies related human death in Ethiopia [9]. Despite, the availability of vaccine both to human and dogs in the country, only 3.9% dogs were found vaccinated as the study done from 2009 - 2012 in Addis Ababa [9]. The widespread use of traditional medications and religious approach to treat rabies cases as evil spirit are also the challenge for prevention and control of rabies as shown in Figure 1.

[10] formulated a mathematical model to investigate rabies epidemics and found that vaccination rate is one of the key components determining rabies transmission. The work by [11] further extended the existing models on rabies transmission between dogs to include dog to human transmission and concluded that human post exposure prophylaxis (PEP) with a dog vaccination campaign was the more cost effective in controlling the disease in the long run.

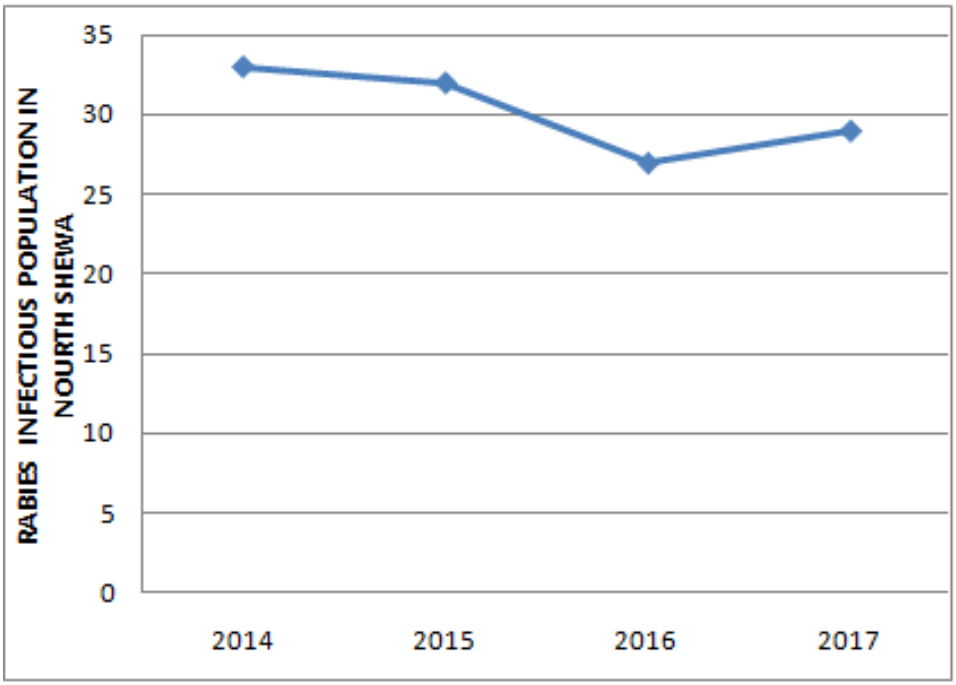

Figure 1: Rabies case reported in Nourth Shewa from 2014 to 2017

Generally, biological or epidemiological models explain the transmission dynamics of diseases and determine the persistence or die out of such diseases with time. They also determine the contribution of each parameter to the basic reproductive number and the best optimal control measure through sensitivity analysis and optimal control theory respectively [12, 13, 14].

1.1 Model description and formulation

1.1.1 Model formation

In this model we consider population of dogs and human. Each population is subdivided in to four compartments; susceptible, exposed, infected and recovers. Susceptible humans are recruited at a rate μ_h either by birth or immigration, and their number increase from individuals that come from sub-classes of rabies recovered by losing their temporary immunity with rate of σ and decrease by individuals that move to exposed compartments at a rate of β_h and natural death rate with rate θ_h. Some exposed human population move to infected compartment at the rate of δ_h and the remaining exposed human population who get the drug(vaccination) compartment join the recover compartment at a rate of τ. The infected human compartment decrease both by natural and rabbies induced death rate θ_h and ω_h respectively.

Susceptible dogs are recruited at a rate μ_d either by birth or immigration, and their number increases from individuals that come from sub-classes of rabies recover by losing their temporary immunity with rate of γ and their number decrease by individuals that move to exposed compartments with rate of β_d and natural death rate with rate θ_d. The exposed population of dog which get drug go to the recovery by rate of α and the remaining which not get the drug with time go to the infected class. The infected population of dog reduces by natural death (θ_d), diseased induce rate (ω_d) and the removing parameter(ω_2). Total human population is given by; $N(t) = S(t) + E(t) + I(t) + R(t)$

The dynamics of rabbies transmission in human and dogs population is represented in the schematic diagram as shown in Fig: 2

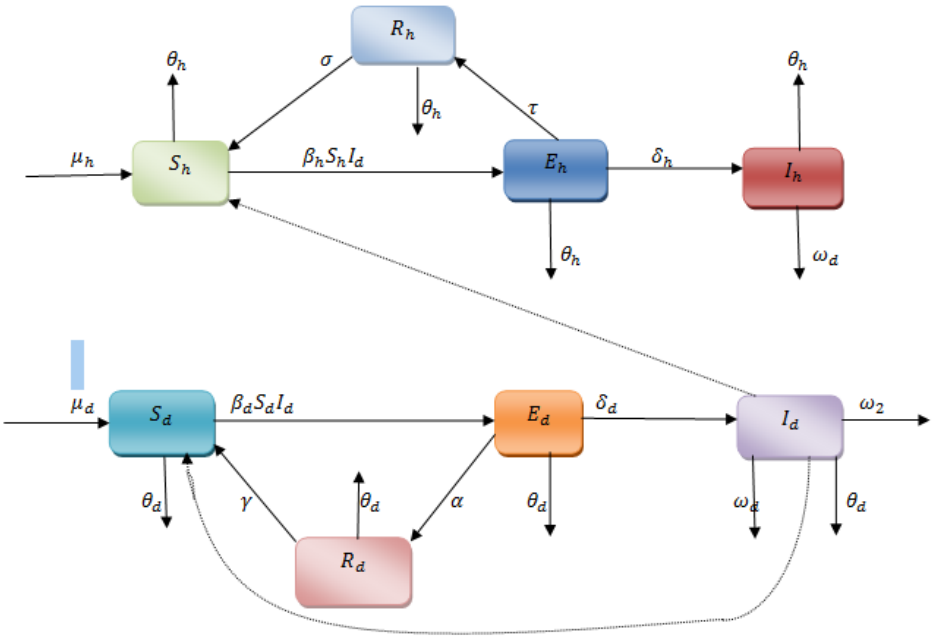

Figure 2: Schematic diagram of the model

1.1.2 Model equations

Based on the assumptions and interrelation between the variables and parameters in figure 2, the following system of ordinary differential equation generated.

$$(1)\qquad \begin{cases} \frac{dS_h}{dt} = \mu_h + \sigma R_h - (\beta_h I_d + \theta_h) S_h, \\ \frac{dE_h}{dt} = \beta_h S_h I_d - (\delta_h + \tau + \theta_h) E_h, \\ \frac{dI_h}{dt} = \delta_h E_h - (\theta_h + \omega_h) I_h, \\ \frac{dR_h}{dt} = \tau E_h - (\sigma + \theta_h) R_h, \\ \frac{dS_d}{dt} = \mu_d + \gamma R_d - (\beta_d I_d + \theta_d) S_d, \\ \frac{dE_d}{dt} = \beta_d S_d I_d - (\delta_d + \theta_d + \alpha) E_d, \\ \frac{dI_d}{dt} = \delta_d E_d - (\theta_d + \omega_2 + \omega_d) I_d, \\ \frac{dR_d}{dt} = \alpha E_d - (\theta_d + \gamma) R_d. \end{cases}$$

1.2 Model analysis

1.2.1 Invariant region

The region in which solutions of rabbies model system is uniformly bounded is the proper subset $\Omega \in \mathbb{R}^8$ and $\Omega = N_H \cup N_D \in \mathbb{R}^4 \times \mathbb{R}^4$.

Considering the human population at any time t; $N_H = S_h + E_h + I_h + R_h$.

The feasible solution of human population of model system in equation (1);

$$(2)\qquad D_h = \left\{ (S_h,\, E_h,\, I_h,\, R_h) \in \mathbb{R}^4,\, 0 \le N_H \le \frac{\mu_h}{\theta_h} \right\}.$$

Moreover, considering dogs population, denoted by N_D; $N_D = S_d + E_d + I_d + R_d$.

The feasible solution of the human population of model system in equation (1) the region $D_d = \left\{ (S_d,\, E_d,\, I_d,\, R_d) \in \mathbb{R}^4 : 0 \le N_D \le \frac{\mu_d}{\theta_d} \right\}$

Total dog and human population is given by; $N_M = N_D + N_H$

By considering;

$$\begin{aligned} N_H &= S_h + E_h + I_h + R_h, \\ N_D &= S_d + E_d + I_d + R_d, \\ N_M &= S_h + E_h + I_h + R_h + S_d + E_d + I_d + R_d. \end{aligned}$$

Feasible solution of human population of model system in equation (1);

$$(3)\quad D_d = \left\{ (S_d, E_d, I_d, R_d, S_h, E_h, I_h, R_h) \in \mathbb{R}^4 \times \mathbb{R}^4 = \mathbb{R}^8 : 0 \le N_Z \le \frac{\mu_N}{\theta_Y} \right\}.$$

1.2.2 Positivity of solutions

Theorem 1. *Let $S_h(0) > 0$, $E_h(0) \ge 0$, $I_h(0) \ge 0$, $R_h(0) \ge 0$, $S_d(0) \ge 0$, $E_d(0) \ge 0$, $I_d(0) \ge 0$ and $R_d(0) \ge 0$ then the solution set S_h, E_h, I_h, R_h, S_d, E_d, I_d and R_d of the system of the equation (1) are positive for all $t > 0$.*

Proof.

$$(4)\quad \frac{dS_h}{dt} = \mu_h + \sigma R_h - \beta_h S_h I_h - \theta_{,h}\, S_h, \frac{dS_h}{dt} + (\beta_h I_h + \theta_h) S_h = \mu_h + \sigma . R_h.$$

Let $A(t) = e^{-\int(\beta_h I_h + \theta_h)dsh}$ be the integrating factor

$$A(t) = e^{-\int(\beta_h I_h+\theta_h)dsh}(\int(\mu_h + \sigma R_h)dsh + C) \geq 0,$$

$$\frac{dE_h}{dt} = \beta_h S_h I_h - \delta_h E_h - \tau E_h - \theta_h E_h, \tag{5}$$

$$\frac{dE_h}{dt} + (\delta_h + \tau + \theta_h)E_h = \beta_h S_h I_h. \tag{6}$$

Let $B(t) = e^{-\int(\delta_h+\tau+\theta_h)deh}$ be the integrating factor

$$\frac{dB(t)}{dt} = B(t)\beta_h S_h I_h, \tag{7}$$

$$\int \frac{dB(t)}{dt} = \int (B(t)\beta_h S_h I_h)deh, \tag{8}$$

$$B(t) = e^{-\int(\delta_h+\tau+\theta_h)de}(\int \beta_h S_h I_h deh + c) \geq 0. \tag{9}$$

By apply in same approach for; $I_h(t), R_h(t), S_d(t), E_d(t), I_d(t), R_d(t)$.

Hence, $S_h(t), E_h(t), I_h(t), R_h(t), S_d(t), E_d(t), I_d(t), R_d(t)$ are positive from equation (1). □

1.2.3 Diseases free equilibrium point

Disease free equilibrium point of the system in equation (1) in the absence of rabbies infections is determined. $E_h = I_d = E_d = I_h = 0$. From equation (1)

$$\frac{dS_d}{dt} = \mu_d - \beta_d S_d I_d - \theta_d S_d + \gamma R_d = 0, \tag{10}$$

$$S_d^* = \frac{\mu_d}{\theta_d}, \tag{11}$$

$$\frac{dS_h}{dt} = \mu_h + \sigma R_h - \beta_h S_h I_d - \theta_{,h}\, S_h = 0 \tag{12}$$

$$S_h^* = \frac{\mu_h}{\theta_h}, \tag{13}$$

$$DFE = \left(\frac{\mu_h}{\theta_h}, 0, 0, 0, \frac{\mu_d}{\theta_d}, 0, 0, 0\right). \tag{14}$$

1.2.4 Basic reproductive number

This is a threshold value that governs the dynamics of rabbies. By employing the "Next Generation Matrix" [15, 16]. Considering;

$$\frac{dE_d}{dt} = \beta_d S_d I_d - \delta_d E_d - \theta_d E_d - \alpha E_d, \tag{15}$$

$$\frac{dI_d}{dt} = \delta_d E_d - \theta_d I_d - \omega_2 I_d - \omega_d I_d, \tag{16}$$

$$\frac{dE_h}{dt} = \beta_h S_h I_d - \delta_h E_h - \tau E_h - \theta_h E_h, \tag{17}$$

$$\frac{dI_h}{dt} = \delta_h E_h - \theta_h I_h - \omega_h I_h. \tag{18}$$

This can be written as;

$$F = \begin{bmatrix} \beta_d S_d I_d \\ 0 \\ \beta_h S_h I_d \\ 0 \end{bmatrix} \quad V = \begin{bmatrix} -\delta_d E_d - \theta_d E_d - \alpha E_d \\ \delta_d E_d - \theta_d I_d - \omega_2 I_d - \omega_d I_d \\ -\delta_h E_h - \tau E_h - \theta_h E_h \\ \delta_h E_h - \theta_h I_h - \omega_h I_h \end{bmatrix}.$$

Differentiating F and V with respect E_d, I_d, E_h and I_h.

$$F = \begin{bmatrix} 0 & \beta_d S_d & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & \beta_h S_h & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix},$$

$$V = \begin{bmatrix} \delta_d + \theta_d + \alpha & 0 & 0 & 0 \\ -\delta_d & \theta_d + \omega_2 + \omega_d & 0 & 0 \\ 0 & 0 & \delta_d + \tau + \theta_h & 0 \\ 0 & 0 & -\delta_h & \theta_h + \omega_h \end{bmatrix},$$

where V^{-1} is given by

$$V^{-1} = \begin{bmatrix} \frac{1}{(\delta_d+\alpha+\theta_d)} & 0 & 0 & 0 \\ \frac{\delta_d}{(\delta_d+\theta_d+\alpha)(\theta_d+\omega_2+\omega_d)} & \frac{1}{(\theta_d+\omega_2+\omega_d)} & 0 & 0 \\ 0 & 0 & \frac{1}{(\delta_d+\tau+\theta_h)} & 0 \\ 0 & 0 & \frac{\delta_h}{(\delta_d+\theta_d+\alpha)(\theta_d+\omega_2+\omega_d)} & \frac{1}{(\theta_h+\omega_h)} \end{bmatrix}. \tag{19}$$

Determining the product of F and V^{-1};

$$FV^{-1} = \begin{bmatrix} \frac{\beta_d\mu_d\delta_d}{\theta_d(\delta_d+\theta_d+\alpha)(\theta_d+\omega_2+\omega_d)} & \frac{\beta_d\mu_d}{\theta_d(\theta_d+\omega_2+\omega_d)} & 0 & 0 \\ 0 & 0 & 0 & 0 \\ \frac{\beta_h\mu_h\delta_d}{\theta_h(\delta_d+\theta_d+\alpha)(\theta_d+\omega_2+\omega_d} & \frac{\beta_h\mu_h}{\theta_h(\theta_d+\omega_2+\omega_d)} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \tag{20}$$

Let $K = FV^{-1}$, the eigenvalues of FV^{-1} can be obtained

$$|K| = \begin{vmatrix} \frac{\beta_d\mu_d\delta_d}{\theta_d(\delta_d+\theta_d+\alpha)(\theta_d+\omega_2+\omega_d)} - \lambda & \frac{\beta_d\mu_d}{\theta_d(\theta_d+\omega_2+\omega_d)} & 0 & 0 \\ 0 & 0-\lambda & 0 & 0 \\ \frac{\beta_h\mu_h\delta_d}{\theta_h(\delta_d+\theta_d+\alpha)(\theta_d+\omega_2+\omega_d} & \frac{\beta_h\mu_h}{\theta_h(\theta_d+\omega_2+\omega_d)} & 0-\lambda & 0 \\ 0 & 0 & 0 & 0-\lambda \end{vmatrix} = 0,$$

$$\left(\frac{\beta_d\mu_d\delta_d}{\theta_d(\delta_d+\theta_d+\alpha)(\theta_d+\omega_2+\omega_d)}-\lambda\right)(\lambda)(\lambda)(\lambda)=0, \tag{21}$$

$$\frac{\beta_d\mu_d\delta_d}{\theta_d(\delta_d+\theta_d+\alpha)(\theta_d+\omega_2+\omega_d)}=0 \quad \text{or} \quad \lambda=0. \tag{22}$$

Hence, eigenvalues,

$$\lambda_1=0 \quad \text{or} \quad \lambda_2=\frac{\beta_d\mu_d\delta_d}{\theta_d(\delta_d+\theta_d+\alpha)(\theta_d+\omega_2+\omega_d)}.$$

The dorminant eigenvalue is the spectral radius (basic reproductive number).

$$R_O=\frac{\beta_d\mu_d\delta_d}{\theta_d(\delta_d+\theta_d+\alpha)(\theta_d+\omega_2+\omega_d)}. \tag{23}$$

1.2.5 Endemic equilibrium point

Endemic equilibrium points are steady state situations where the disease persists in the population. To determine the endemic equilibrium point we put the right side of equation (1) equal to zero.

The endemic equilibrium point of the model is written below;

$$E^*=(S_h^*,\,E_h^*,\,I_h^*,\,R_h^*,\,S_d^*,\,E_d^*,\,I_d^*,\,R_d^*)\,, \tag{24}$$

where

$$\begin{aligned}
S_h^*&=\frac{(\delta_h+\tau+\theta_h)\mu_h\beta_h(\sigma+\theta_h)}{\beta_h[(\delta_h+\tau+\theta_h)(\sigma+\theta_h)(\beta_hI_d^*+\theta_h)-\sigma\tau\beta_hI_d^*]},\\
E_h^*&=\frac{\mu_h\beta_hI_d^*(\sigma+\theta_h)}{[(\delta_h+\tau+\theta_h)(\sigma+\theta_h)(\beta_hI_d^*+\theta_h)-\sigma\tau\beta_hI_d^*]},\\
I_h^*&=\frac{\delta_h\mu_h\beta_hI_d^*(\sigma+\theta_h)}{(\theta_h+\omega_h)[(\delta_h+\tau+\theta_h)(\sigma+\theta_h)(\beta_hI_d^*+\theta_h)-\sigma\tau\beta_hI_d^*]},\\
R_h^*&=\frac{\tau\mu_h\beta_hI_d^*}{[(\delta_h+\tau+\theta_h)(\sigma+\theta_h)(\beta_hI_d^*+\theta_h)-\sigma\tau\beta_hI_d^*]},\\
S_d^*&=\frac{(\delta_d+\theta_d+\alpha)(\theta_d+\omega_2+\omega_d)}{\beta_d\delta_d},\\
E_d^*&=\frac{(\theta_d+\gamma)[\theta_d(\delta_d+\theta_d+\alpha)(\theta_d+\omega_2+\omega_d)-\mu_d\beta_d\delta_d]}{(\theta_d+\omega_2+\omega_d)\beta_d(\gamma\alpha-(\delta_d+\theta_d+\alpha)E_d)(\theta_d+\gamma)},\\
I_d^*&=\frac{(\theta_d+\gamma)[\theta_d(\delta_d+\theta_d+\alpha)(\theta_d+\omega_2+\omega_d)-\mu_d\beta_d\delta_d]}{(\theta_d+\omega_2+\omega_d)\beta_d(\gamma\alpha-(\delta_d+\theta_d+\alpha)E_d)(\theta_d+\gamma)},\\
R_d^*&=\frac{\alpha[\theta_d(\delta_d+\theta_d+\alpha)(\theta_d+\omega_2+\omega_d)-\mu_d\beta_d\delta_d]}{\beta_d\delta_d(\gamma\alpha-(\delta_d+\theta_d+\alpha)E_d)(\theta_d+\gamma)}.
\end{aligned}$$

1.3 Stability analysis of disease free equilibrium

1.3.1 Local stability of disease free equilibrium

Local stability of an equilibrium point means that if you put the system somewhere nearby the point then it will move itself to the equilibrium point in some time.

Theorem 2. *The disease free equilibrium point is locally asymptotically stable if $R_0 < 1$ otherwise it is unstable.*

Proof. To prove local stability of disease free equilibrium, we obtained the Jacobean's matrix of the system (1) at the disease free equilibrium (DFE).

Then, the Jacobean matrix become

(25)
$$\begin{pmatrix} -\theta_h & 0 & 0 & \sigma & 0 & 0 & -\beta_h\frac{\mu_h}{\theta_h} & 0 \\ 0 & -(\delta_h+\tau+\theta_h) & 0 & 0 & 0 & 0 & \beta_h\frac{\mu_h}{\theta_h} & 0 \\ 0 & \delta_h & -(\theta_h+\omega_h) & 0 & 0 & 0 & 0 & 0 \\ 0 & \tau & 0 & -(\sigma+\theta_h) & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\theta_d & 0 & -\beta_d\frac{\mu_d}{\theta_d} & \gamma \\ 0 & 0 & 0 & 0 & 0 & -(\delta_d+\theta_d+\alpha) & \beta_d\frac{\mu_d}{\theta_d} & 0 \\ 0 & 0 & 0 & 0 & 0 & \delta_d & -(\theta_d+\omega_2+\omega_d) & 0 \\ 0 & 0 & 0 & 0 & 0 & \alpha & 0 & -(\theta_d+\gamma) \end{pmatrix}$$

(26)
$$J(DFE) = \begin{pmatrix} C1 & 0 & 0 & \sigma & 0 & 0 & -b3 & 0 \\ 0 & C2 & 0 & 0 & 0 & 0 & b3 & 0 \\ 0 & \delta_h & c3 & 0 & 0 & 0 & 0 & 0 \\ 0 & \tau & 0 & C4 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & c5 & 0 & -b4 & \gamma \\ 0 & 0 & 0 & 0 & 0 & C6 & b4 & 0 \\ 0 & 0 & 0 & 0 & 0 & \delta_d & b1 & 0 \\ 0 & 0 & 0 & 0 & 0 & \alpha & 0 & b2 \end{pmatrix}$$

where

(27) $C1 = -\theta_h$, $C6 = -(\delta_d + \theta_d + \alpha)$,

(28) $C2 = -(\delta_h + \tau + \theta_h)$, $b1 = -(\theta_d + \omega_2 + \omega_d)$,

(29) $C3 = -(\theta_h + \omega_h)$, $b2 = -(\theta_d + \gamma)$,

(30) $C4 = -(\sigma + \theta_h)$, $b3 = \beta_h\dfrac{\mu_h}{\theta_h}$,

(31) $C5 = -\theta_d$, $b4 = \beta_d\dfrac{\mu_d}{\theta_d}$

(32)
$$|J(DFE) - \lambda| = \begin{vmatrix} C1-\lambda & 0 & 0 & \sigma & 0 & 0 & -b3 & 0 \\ 0 & C2-\lambda & 0 & 0 & 0 & 0 & b3 & 0 \\ 0 & \delta_h & C3-\lambda & 0 & 0 & 0 & 0 & 0 \\ 0 & \tau & 0 & C4-\lambda & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & C5-\lambda & 0 & -b4 & \gamma \\ 0 & 0 & 0 & 0 & 0 & C6-\lambda & b4 & 0 \\ 0 & 0 & 0 & 0 & 0 & \delta_d & b1-\lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & \alpha & 0 & b2-\lambda \end{vmatrix} = 0$$

$$(C1-\lambda)\begin{vmatrix} C2-\lambda & 0 & 0 & 0 & 0 & b3 & 0 \\ \delta_h & C3-\lambda & 0 & 0 & 0 & 0 & 0 \\ \tau & 0 & C4-\lambda & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & C5-\lambda & 0 & -b4 & \gamma \\ 0 & 0 & 0 & 0 & C6-\lambda & b4 & 0 \\ 0 & 0 & 0 & 0 & \delta_d & b1-\lambda & 0 \\ 0 & 0 & 0 & 0 & \alpha & 0 & b2-\lambda \end{vmatrix} = 0, \tag{33}$$

$$\begin{aligned} &(C1-\lambda)(C2-\lambda)(C3-\lambda)(C4-\lambda) \\ &\cdot \begin{vmatrix} C5-\lambda & 0 & -b_4 & \gamma \\ 0 & C6-\lambda & b_4 & 0 \\ 0 & \delta_d & b_1-\lambda & 0 \\ 0 & \alpha & 0 & b_2-\lambda \end{vmatrix} = 0 \end{aligned} \tag{34}$$

$$\begin{aligned} &(C1-\lambda)(C2-\lambda)(C3-\lambda)(C4-\lambda)(C5-\lambda) \\ &\cdot \begin{vmatrix} C6-\lambda & b_4 & 0 \\ \delta_d & b_1-\lambda & 0 \\ \alpha & 0 & b_2-\lambda \end{vmatrix} = 0, \\ &(C1-\lambda)(C2-\lambda)(C3-\lambda)(C4-\lambda)(C5-\lambda)(b_2-\lambda) \\ &\cdot [(C6-\lambda)(b_1-\lambda)-b_4\delta_d] = 0, \\ &(C1-\lambda)(C2-\lambda)(C3-\lambda)(C4-\lambda)(C5-\lambda)(b_2-\lambda) = 0, \end{aligned}$$

$$\begin{aligned} \lambda_1 &= C1 & \lambda_4 &= C4 \\ \lambda_2 &= C2 & \lambda_5 &= C5 \\ \lambda_3 &= C3 & \lambda_6 &= b_2 \end{aligned}$$

or

$$\begin{aligned} &(C6-\lambda)(b_1-\lambda)-b_4\delta_d = 0, \\ &\lambda^2-(C6+b_1)\lambda+C6b_1-b_4\delta_d. \end{aligned} \tag{35}$$

Therefore, from the Routh-Hurwitz criterion of order two, it implies that the conditions, $-(c_6+b_1)>0$ and $C6\ b_1-b_4\delta_d>0$. From $C6\ b_1-b_4\delta_d>0$

$$-(\delta_d+\theta_d+\alpha)(-(\theta_d+\omega_2+\omega_d))-\beta_d\frac{\mu_d}{\theta_d}\delta_d>0, \tag{36}$$

$$(\delta_d+\theta_d+\alpha)(\theta_d+\omega_2+\omega_d)-\beta_d\frac{\mu_d}{\theta_d}\delta_d>0, \tag{37}$$

$$(\delta_d+\theta_d+\alpha)(\theta_d+\omega_2+\omega_d)>\beta_d\frac{\mu_d}{\theta_d}\delta_d, \tag{38}$$

$$(\delta_d+\theta_d+\alpha)(\theta_d+\omega_2+\omega_d)>\frac{\beta_d\mu_d\delta_d}{\theta_d}, \tag{39}$$

$$1>\frac{\beta_d\mu_d\delta_d}{\theta_d(\delta_d+\theta_d+\alpha)(\theta_d+\omega_2+\omega_d)}. \tag{40}$$

$R_0<1$ therefore DFE is locally asymptotically stable. The prove is completed. □

1.3.2 Global stability of disease free equilibrium

According to Livingstone.et.al. 2015 to get the global stability of disease free equilibrium point of system (1) we write our system as follows:

$$\begin{cases} \frac{dX_1}{dt} = B(X_1 - X_1(E_0)) + B_1 X_2 \\ \frac{dX_2}{dt} = B_2 X_2 \end{cases} \tag{41}$$

$$X_1 = \text{ transmitting compartments}, \tag{42}$$

$$X_2 = \text{ non - transmitting compartments}, \tag{43}$$

$$X_1 = (S_h,\ R_h,\ S_d,\ R_d), \tag{44}$$

$$X_2 = (E_h,\ I_h,\ E_d,\ I_d). \tag{45}$$

The DFE is denoted by $DFE = \left\{ \frac{\mu_h}{\theta_h}, 0, \frac{\mu_d}{\theta_d}, 0 \right\}$

$$X_1 - X_1(DFE) = \begin{cases} S_h - \frac{\mu_h}{\theta_h} \\ R_h \\ S_d - \frac{\mu_d}{\theta_d} \\ R_d \end{cases} \tag{46}$$

For the global stability of DFE we need to prove the following.

1. B should be a matrix with real negative Eigen values.
2. B_2 Should be a Metzler matrix

Using system (1) together with the representation in (41) the two equations can be written as follows:

$$\begin{bmatrix} \mu_h + \sigma R_h - \beta_h S_h I_d - \theta_h S_h \\ \tau E_h - \sigma R_h - \theta_h R_h \\ \mu_d - \beta_d S_d I_d - \theta_d S_d + \gamma R_d \\ \alpha E_d - \theta_d R_d - \gamma R_d \end{bmatrix} = B \begin{bmatrix} S_h - \frac{\mu_h}{\theta_h} \\ R_h \\ S_d - \frac{\mu_d}{\theta_d} \\ R_d \end{bmatrix} + B_1 \begin{bmatrix} E_h \\ I_h \\ E_d \\ I_d \end{bmatrix}$$

and

$$\begin{bmatrix} \beta_h S_h I_d - \delta_h E_h - \tau E_h - \theta_h E_h \\ \delta_h E_h - \theta_h I_h - \omega_h I_h \\ \beta_d S_d I_d - \delta_d E_d - \theta_d E_d - \alpha E_d \\ \delta_d E_d - \theta_d I_d - \omega_2 I_d - \omega_d I_d \end{bmatrix} = B_2 \begin{bmatrix} E_h \\ I_h \\ E_d \\ I_d \end{bmatrix}.$$

Matrices B, B_1 and B_2 are order 4×4 matrix Using non - transmitting elements of the Jacobean matrix of system (1) and representation in (**??**) we

get

$$B = \begin{bmatrix} -\theta_h & \sigma & 0 & 0 \\ 0 & -(\sigma+\theta_h) & 0 & 0 \\ 0 & 0 & -\theta_d & \gamma \\ 0 & 0 & 0 & -(\theta_d+\gamma) \end{bmatrix}, \tag{47}$$

$$B_1 = \begin{bmatrix} 0 & 0 & 0 & -\beta_h S_h \\ \tau & 0 & 0 & 0 \\ 0 & 0 & 0 & -\beta_d S_d \\ 0 & 0 & \alpha & 0 \end{bmatrix}, \tag{48}$$

$$B_2 = \begin{bmatrix} -\delta_d-\theta_d-\alpha & \beta_d S_d & 0 & 0 \\ \delta_d & -\theta_d-\omega_2-\omega_d & 0 & 0 \\ 0 & \beta_h S_h & -\delta_d-\tau-\theta_h & 0 \\ 0 & 0 & \delta_h & -\theta_h-\omega_h \end{bmatrix} \tag{49}$$

$$\begin{vmatrix} -\theta_h-\lambda & \sigma & 0 & 0 \\ 0 & -(\sigma+\theta_h)-\lambda & 0 & 0 \\ 0 & 0 & -\theta_d-\lambda & \gamma \\ 0 & 0 & 0 & -(\theta_d+\gamma)-\lambda \end{vmatrix} = 0,$$

$$(-\theta_h-\lambda)(-(\sigma+\theta_h)-\lambda)(-\theta_d-\lambda)(-(\theta_d+\gamma)-\lambda) = 0, \tag{50}$$

$$\lambda_1 = -\theta_h, \tag{51}$$

$$\lambda_2 = -(\sigma+\theta_h), \tag{52}$$

$$\lambda_3 = -\theta_d, \tag{53}$$

$$\lambda_4 = -(\theta_d+\gamma). \tag{54}$$

The Eigen value of matrix B is negative and the off diagonal elements of matrix B_2 are non - negative which is Metzler matrix. This proves that the DFE point of system (1) globally asymptotically stable in the region $\mathbb{R}^8$ and $R_0 < 1$.

1.4 Stability analysis of endemic equilibrium point

In this section we focus on the stability of the endemic equilibrium point. The stability of endemic equilibrium point is divided into two local and global.

1.4.1 Local stability analysis of endemic equilibrium point

Theorem 3. *The endemic equilibrium E^* of model (1) is globally asymptotically stable whenever $R_0 > 1$.*

Proof. To determine the local stability of endemic equilibrium point from the differential equation (1) first we determine the Jacobean matrix at E^*.

$$J(E^*) = \begin{pmatrix} C1 & 0 & 0 & \sigma & 0 & 0 & -b3 & 0 \\ b5 & C2 & 0 & 0 & 0 & 0 & b3 & 0 \\ 0 & \delta_h & C3 & 0 & 0 & 0 & 0 & 0 \\ 0 & \tau & 0 & C4 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & C5 & 0 & -b4 & \gamma \\ 0 & 0 & 0 & 0 & b6 & C6 & b4 & 0 \\ 0 & 0 & 0 & 0 & 0 & \delta_d & b1 & 0 \\ 0 & 0 & 0 & 0 & 0 & \alpha & 0 & b2 \end{pmatrix} \tag{55}$$

where

$$\begin{aligned} C1 &= -\beta_h I_d^* - \theta_h & C6 &= -(\delta_d + \theta_d + \alpha) \\ C2 &= -(\delta_h + \tau + \theta_h) & b1 &= -(\theta_d + \omega_2 + \omega_d) \\ C3 &= -(\theta_h + \omega_h) & b2 &= -(\theta_d + \gamma) \\ C4 &= -(\sigma + \theta_h) & b3 &= \beta_h S_h^* \\ C5 &= -\beta_d I_d^* - \theta_d & b4 &= \beta_d S_d^* \\ b6 &= -\beta_d I_d^* & b5 &= -\beta_h I_d^* \end{aligned}$$

$$|J(E^*) - \lambda| = \begin{vmatrix} (C1-\lambda) & 0 & 0 & \sigma & 0 & 0 & -b3 & 0 \\ b5 & C2-\lambda & 0 & 0 & 0 & 0 & b3 & 0 \\ 0 & \delta_h & C3-\lambda & 0 & 0 & 0 & 0 & 0 \\ 0 & \tau & 0 & C4-\lambda & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & C5-\lambda & 0 & -b4 & \gamma \\ 0 & 0 & 0 & 0 & b6 & C6-\lambda & b4 & 0 \\ 0 & 0 & 0 & 0 & 0 & \delta_d & b1-\lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & \alpha & 0 & b2-\lambda \end{vmatrix} = 0 \tag{56}$$

From the above, we get

$$\lambda^7 + m_1\lambda^6 + m_2\lambda^5 + m_3\lambda^4 + m_4\lambda^3 + m_5\lambda^2 + m_6\lambda + m_7 = 0. \tag{57}$$

From the above $\lambda_1 = C3$ it is negative and to see the remains value of λ we use Routh Hurwitz criterion. To obtain the precise number of roots with nonnegative real part, proceed as follow arrange the coefficient of polynomial and values subsequently calculated form them as below:

$$m_0\lambda^n + m_1\lambda^{n-1} + \cdots + m_{n-1}\lambda + m_n = 0, \tag{58}$$

$$m_0\lambda^n + m_1\lambda^{n-1} + \cdots + m_{n-1}\lambda + m_n = 0$$

λ^7	1	m_2	m_4	m_6
λ^6	m_1	m_3	m_5	m_7
λ^5	d_1	d_2	d_3	0
λ^4	e_1	e_2	e_3	
λ^3	f_1	f_2	0	
λ^2	h_1	h_2		
λ^1	n_1	0		
λ^0	x_1			

$$d_1 = -\frac{1}{m_1}\begin{vmatrix} -1 & m_2 \\ m_1 & m_3 \end{vmatrix} = -\frac{1}{m_1}(-m_3 + m_1m_2) = \frac{1}{m_1}(-m_3 + m_1m_2), \tag{59}$$

$$d_2 = -\frac{1}{m_1}\begin{vmatrix} -1 & m_4 \\ m_1 & m_5 \end{vmatrix} = -\frac{1}{m_1}(-m_5 + m_1m_4) = \frac{1}{m_1}(-m_5 + m_1m_4), \tag{60}$$

$$d_3 = -\frac{1}{m_1}\begin{vmatrix} -1 & m_6 \\ m_1 & m_7 \end{vmatrix} = -\frac{1}{m_1}(-m_7 + m_1m_6) = \frac{1}{m_1}(-m_7 + m_1m_6) \tag{61}$$

$$e_1 = -\frac{1}{d_1}\begin{vmatrix} m_1 & m_3 \\ d_1 & d_2 \end{vmatrix} = -\frac{1}{d_1}(m_1d_2 - m_3d_1) \tag{62}$$

$$= -\frac{1}{d_1m_1}(m_1(-m_5 + m_1m_4) - m_3(-m_3 + m_1m_2)) \tag{63}$$

$$= \frac{1}{d_1m_1}[m_3^2 + m_1^2m_4 - m_1m_5 + m_3m_1m_2] \tag{64}$$

$$= \frac{1}{m_5 - m_1m_2}[-m_3^2 - m_1^2m_4 + m_1m_5 + m_3m_1m_2], \tag{65}$$

$$e_2 = -\frac{1}{d_1}\begin{vmatrix} m_1 & m_5 \\ d_1 & d_3 \end{vmatrix} = -\frac{1}{d_1}(m_1d_3 - m_5d_1) \tag{66}$$

$$= -\frac{1}{d_1m_1}(m_1(-m_7 + m_1m_6) - m_5(-m_3 + m_1m_2)) \tag{67}$$

$$= \frac{1}{d_1m_1}[-m_1^2m_6 + m_1m_7 + m_5m_1m_2 - m_5m_3] \tag{68}$$

$$= \frac{1}{m_3 - m_1m_2}[-m_1^2m_6 + m_1m_7 + m_5m_1m_2 - m_5m_3], \tag{69}$$

$$e_3 = -\frac{1}{d_1}\begin{vmatrix} m_1 & m_7 \\ d_1 & 0 \end{vmatrix} = -\frac{1}{d_1}(-d_1m_7) = m_7, \tag{70}$$

$$f_1 = -\frac{1}{e_1}\begin{vmatrix} d_1 & d_2 \\ e_1 & e_2 \end{vmatrix}, \tag{71}$$

$$f_2 = -\frac{1}{e_1}\begin{vmatrix} d_1 & d_3 \\ e_1 & e_3 \end{vmatrix} = -\frac{1}{e_1}(d_1e_3 - e_1d_3), \tag{72}$$

$$h_1 = -\frac{1}{f_1}\begin{vmatrix} e_1 & e_2 \\ f_1 & f_2 \end{vmatrix} = -\frac{1}{f_1}(e_1f_2 - f_1e_2), \tag{73}$$

$$h_2 = -\frac{1}{f_1}\begin{vmatrix} e_1 & e_3 \\ f_1 & 0 \end{vmatrix}, \tag{74}$$

$$h_2 = -\frac{1}{f_1}(0 - e_3f_1) = e_3 = m_7, \tag{75}$$

$$n_1 = -\frac{1}{f_1}\begin{vmatrix} f_1 & f_2 \\ h_1 & h_2 \end{vmatrix}, \tag{76}$$

$$n_1 = -\frac{1}{f_1}(f_1h_2 - h_1f_2), \tag{77}$$

$$n_1 = \frac{1}{f_1}(h_1f_2 - f_1h_2). \tag{78}$$

Let $h_1 = z$, , $f_1 = r$, $f_2 = y$ then, $m_1{=}\frac{1}{f_1}(zy - rm_7)$

$$n_1 = -\frac{1}{n_1}\begin{vmatrix} h_1 & h_2 \\ n_1 & 0 \end{vmatrix} = -\frac{1}{n_1}(-n_1h_2) = h_2 = e_3 = m_7.$$

By Using the Routh Hurwitz criterion it can be seen that all the Eigen values of the characteristic equation have negative real part if and only if $m_1 > 0$, $m_2 > 0$, $m_3 > 0$, $m_4 > 0$, $m_5 > 0$, $m_6 > 0$, $m_7 > 0$, $d_1 > 0$, $d_2 > 0$, $d_3 > 0$, $e_1 > 0$, $e_2 > 0$, $e_3 > 0$, $f_1 > 0$, $f_2 > 0$, $h_1 > 0$, $h_2 > 0$, $n_1 > 0$.

Since $R_0 = \frac{\mu_d\beta_d\delta_d}{\theta_d(\delta_d+\theta_d+\alpha)(\theta_d+\omega_2+\omega_d)}$. Hence, $R_0 > 1$. □

This implies that the endemic equilibrium point (E^*) is local asymptotical stable.

1.4.2 Global stability analysis of endemic equilibrium

Global stability means that the system will come to the equilibrium point from any possible starting point.

Theorem 4. *If $R_0 > 1$, E^* of the model (1) is globally asymptotically stable.*

Proof. Using the Lyapunov approach in [17], global asymptotic stability of E^*: Define

$$
\begin{aligned}
&V(S_h^*, E_h^*, I_h^*, R_h^*, S_d^*, I_d^*, I_d^*, R_d^*)\\
&= (S_h - S_h^* - S_h^* \ln \frac{S_h^*}{S_h}) + (E_h - E_h^* - E_h^* \ln \frac{E_h^*}{E_h})\\
&+ (I_h - I_h^* - I_h^* \ln \frac{I_h^*}{I_h}) + (R_h - R_h^* - R_h^* \ln \frac{R_h^*}{R_h}) + (S_d - S_d^* - S_d^* \ln \frac{S_d^*}{S_d})\\
&+ (E_d - E_d^* - E_d^* \ln \frac{E_d^*}{E_d}) + (I_d - I_d^* - I_d^* \ln \frac{I_d^*}{I_d}) + (R_d - R_d^* - R_d^* \ln \frac{R^*}{R_d}).
\end{aligned} \tag{79}
$$

By direct calculating the derivative of V along the solution of (1), we have;

$$
\begin{aligned}
&\frac{dV}{dt} = \left(1 - \frac{S_h^*}{S_h}\right) \frac{dS_h}{dt} + \left(1 - \frac{E_h^*}{E_h}\right) \frac{dE_h}{dt} + \left(1 - \frac{I_h^*}{I_h}\right) \frac{dI_h}{dt} + \left(1 - \frac{R_h^*}{R_h}\right),\\
&\frac{dR_h}{dt} + \left(1 - \frac{S_d^*}{S_d}\right) \frac{dS_d}{dt} + \left(1 - \frac{E_d^*}{E_d}\right) \frac{dE_d}{dt} + \left(1 - \frac{I_d^*}{I_d}\right) \frac{dI_d}{dt} + \left(1 - \frac{R_d^*}{R_d}\right) \frac{dR_d}{dt}
\end{aligned} \tag{80}
$$

$$
\begin{aligned}
\frac{dV}{dt} &= \mu_h + \sigma R_h + \beta_h S_h^* I_d + \theta_h S_h^* + \beta_h S_h I_d\\
&+ \delta_h E + \tau E^* + \theta_h E^* + \delta_h E_h\\
&+ \theta_h I_h^* + \omega_h I_h^* + \tau E_h + \sigma R_h^* + \theta_h R_h^*\\
&+ \mu_d + \gamma R_d + \beta_d S_d^* I_d + \theta_d S_d^* + \beta_d S_d I_d\\
&+ \delta_d E_d^* + \theta_d E_d^* + \alpha E_d^* + \delta_d E_d + \theta_d I_d^* + \omega_2 I_d^*\\
&+ \omega_d I_d^* + \alpha E_d + \theta_d R_d^* + \gamma R_d^*\\
&- \Big(- \beta_h S_h I_d - \theta_h S_h - \frac{S_h^*}{S_h} \mu_h - \frac{S_h^*}{S_h} \sigma R_h - \delta_h E_h\\
&- \tau E_h - \theta_h E_h - \frac{E^*}{E_h} \beta_h S_h I_d - \theta_h I_h - \omega_h I_h\\
&- \frac{I_h^*}{I_h} \delta_h E_h - \sigma R_h - \theta_h R_h - \frac{R_h^*}{R_h} \tau E_h - \beta_d S_d I_d - \theta_d S_d\\
&- \frac{S_d^*}{S_d} \mu_d - \frac{S_d^*}{S_d} R_d - \delta_d E_d,
\end{aligned} \tag{81}
$$

$$
\begin{aligned}
&\theta_d E_d - \alpha E_d - \frac{E_d^*}{E_d} \beta_d S_d I_d - \theta_d I_d - \omega_2 I_d - \omega_d I_d\\
&- \frac{I_d^*}{I_d} \delta_d E_d - \theta_d R_d - \gamma R_d - \frac{R_d^*}{R_d} \alpha E_d \Big),
\end{aligned} \tag{82}
$$

$$
\frac{dV}{dt} = A - B, \tag{83}
$$

where

$$
\begin{aligned}
A = {} & \mu_h + \sigma R_h + \beta_h S_h^* I_d + \theta_h S_h^* + \beta_h S_h I_d + \delta_h E^* + \tau E^* + \theta_h E^* + \delta_h E_h \\
& + \theta_h I_h^* + \omega_h I_h^* + \tau E_h + \sigma R_h^* + \theta_h R_h^* + \mu_d + \gamma R_d + \beta_d S_d^* I_d \\
& + \theta_d S_d^* + \beta_d S_d I_d + \delta_d E_d^* + \theta_d E_d^* + \alpha E_d^* + \delta_d E_d \\
& + \theta_d I_d^* + \omega_2 I_d^* + \omega_d I_d^* + \alpha E_d + \theta_d R_d^* + \gamma R_d^*,
\end{aligned} \tag{84}
$$

$$
\begin{aligned}
B = {} & -\beta_h S_h I_d - \theta_h S_h - \frac{S_h^*}{S_h}\mu_h - \frac{S_h^*}{S_h}\sigma R_h \\
& - \delta_h E_h - \tau E_h - \theta_h E_h - \frac{E^*}{E_h}\beta_h S_h I_d \\
& - \theta_h I_h - \omega_h I_h - \frac{I_h^*}{I_h}\delta_h E_h - \sigma R_h - \theta_h R_h - \frac{R_h^*}{R_h}\tau E_h - \beta_d S_d I_d \theta_d S_d - \frac{S_d^*}{S_d}\mu_d \\
& - \frac{S_d^*}{S_d} R_d - \delta_d E_d - \theta_d E_d - \alpha E_d - \frac{E_d^*}{E_d}\beta_d S_d I_d - \theta_d I_d \omega_2 I_d - \omega_d I_d \\
& - \frac{I_d^*}{I_d}\delta_d E_d - \theta_d R_d - \gamma R_d - \frac{R_d^*}{R_d}\alpha E_d.
\end{aligned} \tag{85}
$$

Thus, if $A < B$, then $\frac{dV}{dt} \leq 0$; Noting that $\frac{dV}{dt} = 0$. If and only if $S_d = S_d^*$, $E_d = E_d^*$, $I_d = I_d^*$, $R_d = R_d^*$, $S_h = S_h^*$, $E_h = E_h^*$, $I_h = I_h^*$, $R_h = R_h^*$. Therefore, the largest compact invariant set in $\{(S_h^*, E_h^*, I_h^*, R_h^*, S_d^*, I_d^*, I_d^*, R_d^*) \in \mathbb{R}^8 : \frac{dV}{dt} = 0\}$ is the singleton E^*, where E^* is the endemic equilibrium of the system (1) by invariance principle (is criterion for the asymptotic stability of autonomous (possibly nonlinear) dynamical system), it implies that E^* is globally asymptotically stable in $\mathbb{R}^8$ if $A < B$. This implies that $R_0 > 1$ [18, 19, 20]. □

1.5 Sensitivity analysis

The sensitivity analysis of the model parameters was carried out in order to determine parameters in the model that have a high transmission influence on the disease [21, 22, 23, 24]. We analysed the reproduction number to determine whether or not treatment of infective and mortality can lead to the effective elimination or control the rabies disease in the population.

We determine the most sensitive parameter by using the relation;
$P_{mi} = \frac{mi}{R_0} \times \frac{\partial R_0}{\partial mi}$, where mi are parameter, and R_0 is the reproductive number.
If $P_{mi} < 0$, then mi have an effect of controlling the disease
If $P_{mi} > 0$, then mi have an effect on expanding the disease

$$P_{\beta_d} = \frac{\beta_d}{R_0} \times \frac{\partial R_0}{\partial \beta_d}$$

The results of the sensitivity analysis as shown in Table 1 indicates that some parameters are more sensitive to the reproduction number than others. The following parameters; $\theta_d, \alpha, \omega_2, \omega_d$ help reduce the spread of the infection. However, the following parameters μ_d, β_d, δ_d increases the spread of the infection whenever their values increases.

Table 1: Sensitivity indices of model parameters to R_0

Sensitivity indices of R_0		
No	Parameter	Sensitivity index
1.	μ_d	$+ve$
2.	β_d	$+ve$
3.	δ_d	$+ve$
4.	θ_d	$-ve$
5.	α	$-ve$
6.	ω_2	$-ve$
7.	ω_d	$-ve$

1.6 Model validation and validity

Model validation is an important and yet controversial aspect of any model-based methodology in general, and system dynamics in particular. Validity of the results in a model-based study is crucially dependent on the validity of the model. The validation process is undertaken in order to ensure that the model developed is sufficiently accurate for the purpose at hand.

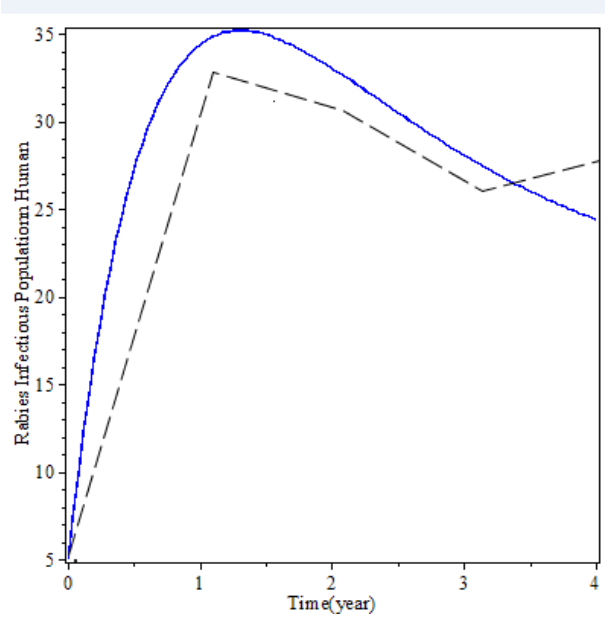

Figure 3: The comparison between the reported human rabies cases in North Shewa zone of Health Bearaua from 2014 to 2017 and the simulation of $I_h(t)$ from the model

The dashed curve represents the data reported by the North Shewa Ministry of Health while the solid curve is simulated by using our model. The initial values

used in the stimulations were $s[h](0) = 50$,$e[h](0) = 25$, $i[h](0) = 5$, $r[h](0) = 5$, $s[d](0) = 250$, $e[d](0) = 500$, $i[d](0) = 1000$, $r[d](0) = 250$.

The numerical simulation of human rabies cases in North Shewa from 2014 to 2017 is shown in Fig. 4, indicating that our model provides a good match to the reported data. The awareness of rabies for people in recent years has been enhanced gradually.

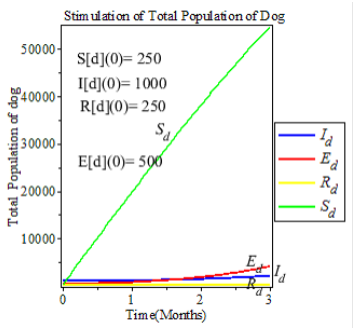

Figure 4: Simulation of total human population with initial value

2. Numerical simulation

Numerical simulations are required to study the behaviour of a systems whose mathematical model is too complex to provide analytical solution as in most non linear systems.

Table 2 shows the values of the parameters used in the various simulations.

Table 2: parameter value

Parameter	**Value**	**Source**
μ_h	200	Assumption
θ_h	0.0008	Assumption
δ_h	0.17	[25]
ω_h	1	[26]
β_h	2.29×10^{-2}	[25]
τ	0.1	[27]
σ	1	[27]
μ_d	2×10^{4}	[26]
θ_d	0.083	[26]
δ_d	2.571×10^{-2}	[4]
ω_d	0.005	Assumption
β_d	3.776×10^{-6}	Assumption
α	0.005	Assumption
γ	0.5	[26]
ω_2	0.001	Assumption

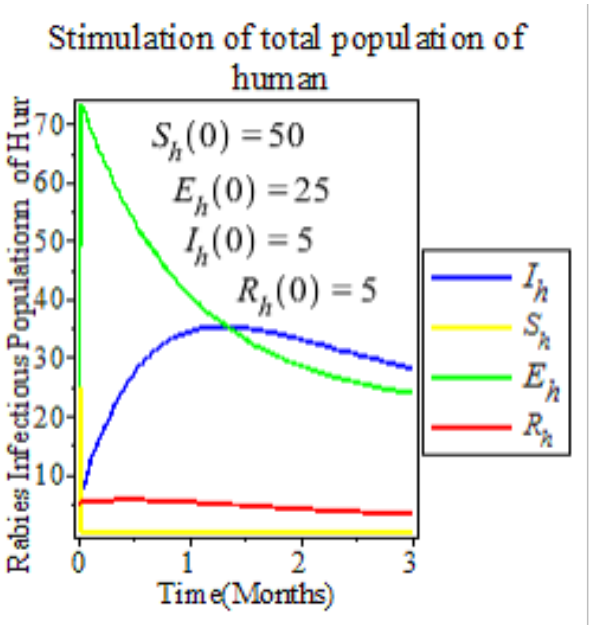

Figure 5: The stimulation of total population dogs with initial value

2.1 Effect of removing rate on rabies infectious population

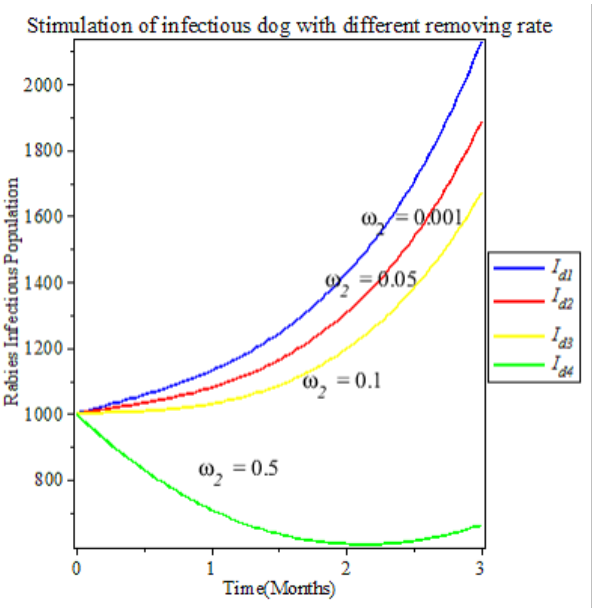

Figure 6: Simulation of infectious dogs with different removing rates.

In Fig. 6, we have experimented on the effect of ω_2 in decreasing the number of rabies infectious population. The figure shows that when the values of ω_2 increase, the number of rabies infectious population is going down (decreasing).

2.2 Effect of contact rate on exposed dogs population

In this section, as we see in Fig. 7, below the figure reflects that as the value of contact rate of rabies is increased, the exposed population increases.

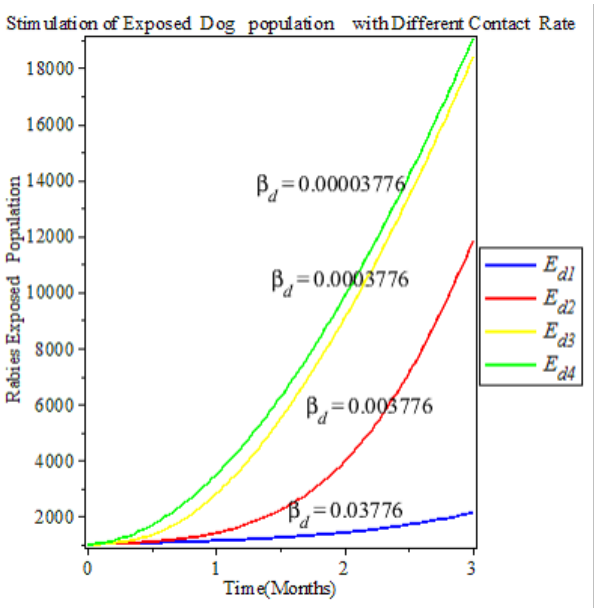

Figure 7: Simulation of infectious dogs with different removing rates.

2.3 Effects of treatment rate on recovered dogs population

We simulated the effects of treatment on recovered dogs population as shown in Fig: 8.

2.4 Effects of rate of infection on infectious dogs population

In this section, we simulated the effects of rate of infection on infectious dogs population as shown in Fig 9.

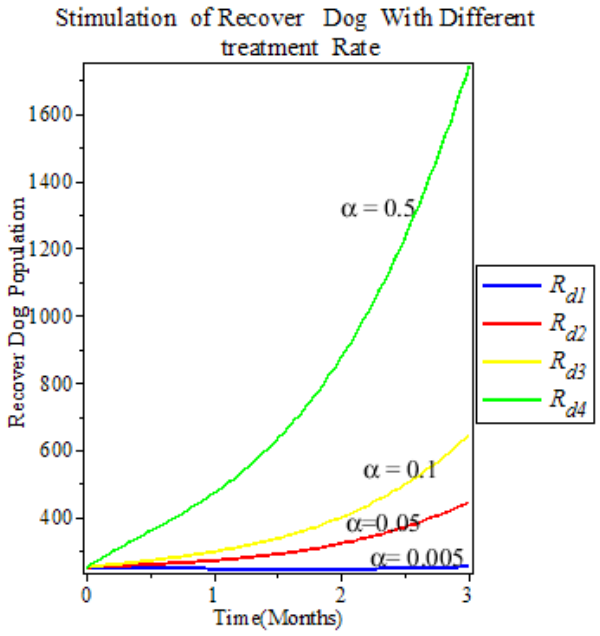

Figure 8: Simulation of infectious dogs with different removing rates.

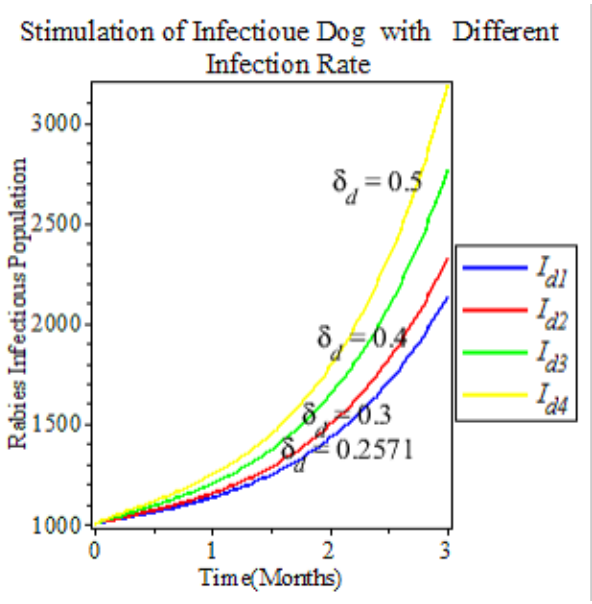

Figure 9: Simulation of infectious dogs with different infectious rates.

3. Conclusion

In this study we have formulated mathematical model of rabies. The model contains dog and human population. We defined the reproduction number in

terms of the parameters and computed it by using next generation operator. The results are depending only on the parameter of dog population. It was also established that for the basic reproduction number, $R_0 < 1$, the disease free equilibrium point is asymptotically stable so that the disease dies out after some period of time and if $R_0 > 1$, the disease free equilibrium is unstable and the disease prissiest. We also established that when $R_0 > 1$ then the endemic equilibrium is locally asymptotically stable, and unstable if $R_0 < 1$. The local stability theorems of disease free equilibrium and endemic equilibrium points of the model are proved by using Jacobean matrix and Routh-Hurwitz criterion. Further more global stability analysis of endemic equilibrium point was computed by using invariance principle. Sensitivity analysis of basic parameters and interpretation of the sensitivity index is also computed. Depending on the value of the sensitivity analysis of parameter the natural death rate, diseases induce rate, removing parameter and vaccination rate (the rate of exposed population of dog join to the recovered population) have an effects on controlling the rabies disease in the community and natural birth rate, the rate susceptible population infection by infected animal and the rate of exposed dog infected (incubation period) have an effect on expansion the rabies disease in the community. Moreover, numerical stimulation is performed in order to check the effect of each parameter in the expansion as well as in the controlling of rabies. Depending on numerical stimulation, if the removing rate and treatment rate are reduced (decrease) the disease spread increases and if the other rate like contact rate and infective rate are increase the disease spread increases.

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Data Availability Statement

The data supporting this Tuberculosis model analysis is from the Ethiopia National Institute of Health, a division of the National Health. Some of the parameter values are assumed and others are taken from published articles and are cited in this paper. These published articles are also cited at relevant places within the text as references.

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