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MATHEMATICAL MODEL ON THE TRANSMISSION DYNAMICS OF ANTHRAX AND RABIES CO-INFECTION ON WILD HERBIVORES AND CARNIVORES

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Abstract: This study is to understand the dynamics of the co-infection of anthrax and rabies in wild herbivores and carnivores. A co-infection mathematical model is developed and its biological well-posedness is studied by proving properties of boundedness and non-negativity of the solutions. The basic reproduction numbers for the model is computed. The equilibrium points were identified. The qualitative analysis reveals that the disease free equilibrium of the sub-models were locally and globally asymptotically stable while the co-infection model is locally asymptotically stable but globally unstable. The dynamics of the model is studied using numerical approach and the impact of some of the parameters are studied.

Keywords: co-infection; wild animals; stability; equilibrium-point; transmission.

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1. INTRODUCTION

Anthrax is a bacterial infection that affects a number of animals and human. It is caused by spore-forming bacteria (*Bacillus anthracis*) which is usually acquired from contaminated vegetation, water, soil or infected animal tissues. Though animals are mainly thought to become infected when they ingest spores; however, inhalation could also play a role and entry through skin lesions may be possible (Spickler [46]). Primary host of Anthrax disease are ungulate herbivores such as sheep, goats, cattle, horses, pigs, camels, antelope, deer and buffaloes, but its occurs in wide range of omnivores, carnivores and other vertebrates (Kashyap et al [27], Mackey and Kribs [30]). Species of animals differ in susceptibility to anthrax: domesticated and wild herbivores tend to be very susceptible and often die rapidly while omnivores and carnivores are more resistant to developing clinical signs, and may recover without treatment if they become ill (Cleggy et al [18]).

Direct transmission between living animals is not significant but carcasses are important in contaminating the environment. The environment is contaminated by the fluids that may exude from the orifices. When the fluids are exposed to air, the bacteria form spores and contaminate soil, plant roots and nearby vegetation. Bacteria in the tissues also form spores if a carcass is opened. Sporulation does not seem to occur inside a closed carcass, where the bacteria is thought to be destroyed within a few days by putrefaction. Water might also become contaminated when large numbers of vultures wash their beaks and feathers after feeding on infected carcasses (Berry [12]). Signs of the illness usually appear 3 to 7 days after the spores are swallowed or inhaled. Once signs begin in animals, they usually die within two days. Anthrax spores can remain viable for long periods in the soil or animal products such as hides (including processed hides) and wool. In some cases, they have been reported to survive for decades (Galante and Fasanella [21]).

Rabies is a viral disease caused by Lyssa virus and this virus affects the central nervous system. It induces inflammation of the brain in mammals. Exposure to the virus occurs when saliva or neural tissue (brain or spinal cord tissues) of an infected animal is introduced into open cuts or wounds or come in contact with the mucous membranes (mouth, nose, eyes). Immediately after exposure, the virus enters an eclipse phase when the virus is not easily detected. During this phase, the virus replicates in non-nervous tissue such as the muscle. After several days or months, it enters into the peripheral nerves and is transported to the central nervous system (Barrosa et al [11]).

Rabies are usually spread by bites of infected animals. All mammals are susceptible to becoming

infected with rabies but not all are effective at transmitting the virus. Herbivores, rodents and other non-biting mammals are not significant in the spread of the disease but carnivores and bats maintain infection naturally (Adedeji et al [3]). Animals with rabies often have unusual behaviour. The early stage of infection include fever and tackling at the site of exposure and this is followed by other symptoms like wild movement, inability to control parts of the body, loss of consciousness, confusion, fear of water and uncontrolled excitement (Eze et al [20]). These signs or symptoms starts to show on an average of 1 to 3 months from the time of exposure. This time varies depending on the site of exposure, the amount of rabies virus transmitted, and the immune status of the exposed (Ojo et al [36]).

Many scholars have studied the dynamics of anthrax disease, rabies disease and the co-infection of diseases. Akande et al [4], Alfwzan et al [5], Ali et al [6], Baloba and Seidu [9], Baloba et al [10], Githire et al [22], Mackey and Kribs [30], Osman et al [38], Raza et al [41] and Raza and Abdella [42] studied the dynamics of anthrax disease in animals and or humans. And some of these scholars studied the optimal control of the anthrax disease in animals and or humans. Similarly, many rabies dynamical models are also studied to suggest its mitigation strategies and control strategies. Such Scholars are Abdulmajid and Hassan [1], Asamoah et al [7], Aydogan et al [8], Barrosa et al [11], Bornaa et al [13], Bukari et al [14], Charles et al [16], Eze et al [20], Hailemichael et al [24], Huang and Li [26], Musaili and Chepkwony [34], Pantha et al [39], Somma et al [45] and Zarin et al [49].

Studies on the dynamics of co-infection models are also studied in several literature. Osman [37] and Rekha et al [43] developed and analyzed a mathematical model of anthrax and listeriosis co-dynamics. Hayes et al [25] reviewed the mathematical models of disease transmission between livestock and wildlife. From their study, it was shown that of 118 diseases at the wildlife-livestock interface represented in literature only 14 have been explored through mathematical modelling. And this formed a motivational base for the study, as it would be good to understand the dynamics of diseases in the wild as some wild animals can be reservoir for disease affecting humans and other domestic animals.

2. PRELIMINARIES

2.1 MODEL FORMULATION

The co-infection model of anthrax and rabies diseases is formulated by subdividing the population into two, the herbivores and the carnivores. The herbivores population is later divided into 7 compartments: The susceptible herbivores (S_h), The anthrax exposed herbivores (E_{ha}), The rabies exposed herbivores (E_{hr}), The herbivores exposed to both anthrax and rabies disease (E_{har}), The anthrax infectious herbivores (I_{ha}), The rabies infectious herbivores (I_{hr}) and The co-infectious herbivores (I_{har}). While the carnivores population is subdivided into 8 compartments: The susceptible carnivores (S_c), The anthrax exposed carnivores (E_{ca}), The rabies exposed carnivores (E_{cr}), The carnivores exposed to both anthrax and rabies diseases (E_{car}), The anthrax infectious carnivores population (I_{ca}), The rabies infectious carnivores population (I_{cr}), The co-infectious carnivores population (I_{car}) and The anthrax Recovered carnivores population (R_{ca}). We also added the environmental pathogen in our model.

The susceptible class are those who are at risk of becoming infected, the exposed are those who are exposed to the disease but have no symptom of the disease, the infectious class are those showing the symptoms of the disease while the recovered class are those who have recovered from the diseases.

We assume the following:

- Any animals added to the population either by birth or migration are disease free.
- The anthrax and rabies diseases can affect both the herbivores and the carnivores (Adedeji et al [3], Kashyap et al [27], Mackey and Kribs [30]).
- The herbivores are infected with anthrax by coming in contact with the pathogen in the environment while the carnivores are infected by coming in contact with life or dead infectious herbivores or carnivores.
- The herbivores and carnivores are infected with rabies when bitten by a rabies infectious carnivore.
- The herbivores do not recover from the anthrax disease.
- Some carnivores can recover from the anthrax disease (Cleggy et al, [18])
- All recovered population from Anthrax are reintroduced to the susceptible population.
- There are no recoveries for rabies disease by either of the animals.

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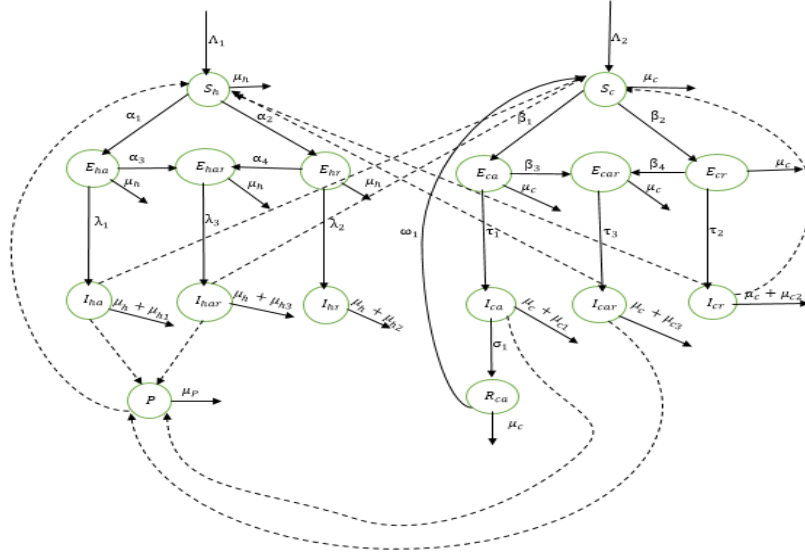


Figure 1: Flow diagram for the co-infection of anthrax and rabies diseases

Using the flow diagram in figure 1 and the dynamics of the disease as described above, we arrive at the following system of nonlinear ordinary differential equations for transmission of the co-infection of anthrax and rabies in the given population as:

$$\frac{dS_h}{dt} = \Lambda_1 - (\alpha_1 + \alpha_2 + \mu_h)S_h$$

$$\frac{dS_c}{dt} = \Lambda_2 - (\beta_1 + \beta_2 + \mu_c)S_c + \omega_1 R_{ca}$$

$$\frac{dE_{ha}}{dt} = \alpha_1 S_h - (\alpha_3 + \lambda_1 + \mu_h)E_{ha}$$

$$\frac{dE_{ca}}{dt} = \beta_1 S_c - (\beta_3 + \tau_1 + \mu_c)E_{ca}$$

$$\frac{dE_{hr}}{dt} = \alpha_2 S_h - (\alpha_4 + \lambda_2 + \mu_h)E_{hr}$$

$$\frac{dE_{cr}}{dt} = \beta_2 S_c - (\beta_4 + \tau_2 + \mu_c)E_{cr}$$

$$\frac{dE_{har}}{dt} = \alpha_3 E_{ha} + \alpha_4 E_{hr} - (\lambda_3 + \mu_h)E_{har}$$

$$\frac{dE_{car}}{dt} = \beta_3 E_{ca} + \beta_4 E_{cr} - (\tau_3 + \mu_c)E_{car}$$

$$\frac{dI_{ha}}{dt} = \lambda_1 E_{ha} - (\mu_h + \mu_{h1})I_{ha}$$

$$\frac{dI_{ca}}{dt} = \tau_1 E_{ca} - \sigma_1 I_{ca} - (\mu_c + \mu_{c1})I_{ca}$$

$$\frac{dI_{hr}}{dt} = \lambda_2 E_{hr} - (\mu_h + \mu_{h2}) I_{hr}$$

$$\frac{dI_{cr}}{dt} = \tau_2 E_{cr} - (\mu_c + \mu_{c2}) I_{cr}$$

$$\frac{dI_{har}}{dt} = \lambda_3 E_{har} - (\mu_h + \mu_{h3}) I_{har}$$

$$\frac{dI_{car}}{dt} = \tau_3 E_{car} - (\mu_c + \mu_{c3}) I_{car}$$

$$\frac{dR_{ca}}{dt} = \sigma_1 I_{ca} - (\omega_1 + \mu_c) R_{ca}$$

$$\frac{dp}{dt} = \delta_1 (I_{ha} + I_{ca} + I_{har} + I_{car}) - \mu_p P$$

where $\alpha_1 = a_1 P$, $\alpha_2 = c_1 (I_{cr} + I_{car})$, $\alpha_3 = e_1 (I_{cr} + I_{car})$, $\alpha_4 = g_1 P$, $\beta_1 = b_1 (I_{ha} + I_{ca} + I_{har} + I_{car})$,

$\beta_2 = d_1 (I_{cr} + I_{car})$, $\beta_3 = f_1 (I_{cr} + I_{car})$, $\beta_4 = h_1 (I_{ha} + I_{ca} + I_{har} + I_{car})$ and $a_1, b_1, c_1, d_1, e_1, f_1, g_1, h_1 \neq 0$.

Table 1: parameters description

Parameters	Description
Λ_1	Recruitment level of herbivores into susceptible population
Λ_2	Recruitment level of carnivores into susceptible population
α_1	Proportion of the susceptible herbivores population that becomes exposed to anthrax disease
α_2	Proportion of the susceptible herbivores population that becomes exposed to rabies disease
α_3	Proportion of the anthrax exposed herbivores population that becomes exposed to both anthrax and rabies diseases
α_4	Proportion of the herbivores population that becomes exposed to both anthrax and rabies diseases
β_1	Proportion of the susceptible carnivores population that becomes exposed to anthrax disease
β_2	Proportion of the susceptible carnivores population that becomes exposed to rabies disease
β_3	Proportion of the anthrax carnivores population that becomes exposed to both anthrax and rabies diseases
β_4	Proportion of the rabies carnivores population that becomes exposed to both anthrax and rabies diseases
λ_1	Rate at which the exposed herbivores population becomes infectious with anthrax disease
λ_2	Rate at which the exposed herbivores population becomes infectious with rabies disease

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λ_3	Rate at which the exposed herbivores population becomes infectious with both anthrax and rabies diseases
τ_1	Rate at which the exposed carnivores population becomes infectious with anthrax disease
τ_2	Rate at which the exposed carnivores population becomes infectious with rabies disease
τ_3	Rate at which the exposed carnivores population becomes infectious with both anthrax and rabies diseases
σ_1	Rate at which the infectious carnivores population recover from anthrax disease
ω_1	Rate at which anthrax recovered carnivores becomes susceptible
μ_h	Natural death rate of herbivores
μ_c	Natural death rate of carnivores
μ_{h1}	Rate of death due to anthrax disease in herbivores
μ_{h2}	Rate of death due to rabies disease in herbivores
μ_{h3}	Rate of death due to co-infection of anthrax and rabies diseases in herbivores
μ_{c1}	Rate of death due to anthrax in carnivores
μ_{c2}	Rate of death due to rabies in carnivores
μ_{c3}	Rate of death due to co-infection on anthrax and rabies diseases in carnivores
δ_1	Pathogen shedding rate of anthrax-infectious animals
μ_p	Rate at which the anthrax pathogen decays in the environment
$a_1, b_1, c_1, d_1,$	Contact rates
e_1, f_1, g_1, h_1	

2.2 POSITIVITY AND BOUNDEDNESS OF SOLUTION

Theorem 1 (Positivity of solutions): All the populations of the anthrax and rabies co-infection model are non-negative with positive initial condition for all $t \geq 0$.

Proof: Assume $S_h(0) > 0, S_c(0) > 0, E_{ha}(0) > 0, E_{ca}(0) > 0, E_{hr}(0) > 0, E_{cr}(0) > 0, E_{har}(0) > 0, E_{car}(0) > 0, I_{ha}(0) > 0, I_{ca}(0) > 0, I_{hr}(0) > 0, I_{cr}(0) > 0, I_{har}(0) > 0, I_{car}(0) > 0, R_{ca}(0) > 0$ and $P(0) > 0$ at $t = 0$, then from Equation 1 of the system;

$$\frac{dS_h}{dt} = \Lambda_1 - (\alpha_1 + \alpha_2 + \mu_h)S_h \text{ and } \frac{dS_h}{S_h} \geq -(\alpha_1 + \alpha_2 + \mu_h)dt$$

By integration, $S_h \geq S_h(0)e^{-\int (\alpha_1 + \alpha_2 + \mu_h)dt} > 0$ for all $t \geq 0$.

Similar results are obtained for the equations of the system showing that all state variables will always be positive at all t .

Theorem 2 (Invariant region of existence): The dynamical system of the anthrax and rabies co-infection model is positively invariant in the closed variant set $\Omega = \{S_h, S_c, E_{ha}, E_{ca}, E_{hr}, E_{cr}, E_{har}, E_{car}, I_{ha}, I_{ca}, I_{hr}, I_{cr}, I_{har}, I_{car}, R_{ca}\} \in R_+^{15}$.

Proof: Suppose $\Omega = \{S_h, S_c, E_{ha}, E_{ca}, E_{hr}, E_{cr}, E_{har}, E_{car}, I_{ha}, I_{ca}, I_{hr}, I_{cr}, I_{har}, I_{car}, R_{ca}\} \in R_+^{15}$, $\Omega_1 = \{S_h, E_{ha}, E_{hr}, E_{har}, I_{ha}, I_{hr}, I_{har}\}$, $\Omega_2 = \{S_c, E_{ca}, E_{cr}, E_{car}, I_{ca}, I_{cr}, I_{car}, R_{ca}\}$ and $\Omega = \Omega_1 \times \Omega_2$ for all $t > 0$. Given that $N_h = S_h + E_{ha} + E_{hr} + E_{har} + I_{ha} + I_{hr} + I_{har}$ and $N_c = S_c + E_{ca} + E_{cr} + E_{car} + I_{ca} + I_{cr} + I_{car} + R_{ca}$ where N_h is the total herbivores population and N_c is the total carnivores population.

then

$$\frac{dN_h}{dt} = \frac{dS_h}{dt} + \frac{dE_{ha}}{dt} + \frac{dE_{hr}}{dt} + \frac{dE_{har}}{dt} + \frac{dI_{ha}}{dt} + \frac{dI_{hr}}{dt} + \frac{dI_{har}}{dt},$$

and

$$\frac{dN_c}{dt} = \frac{dS_c}{dt} + \frac{dE_{ca}}{dt} + \frac{dE_{cr}}{dt} + \frac{dE_{car}}{dt} + \frac{dI_{ca}}{dt} + \frac{dI_{cr}}{dt} + \frac{dI_{car}}{dt} + \frac{dR_{ca}}{dt}.$$

Hence,

$$\frac{dN_h}{dt} = \Lambda_1 - \mu_h N_h - \mu_{h1} I_{ha} - \mu_{h2} I_{hr} - \mu_{h3} I_{har},$$

which gives $\frac{dN_h}{dt} \leq \Lambda_1 - \mu_h N_h$ and $\frac{dN_h}{dt} + \mu_h N_h \leq \Lambda_1$.

Solving the differential equation, we have $N_h \leq \frac{\Lambda_1}{\mu_h} + C e^{-\mu_h t}$ and as $t \rightarrow \infty$, $C e^{-\mu_h t} \rightarrow 0$.

Hence, $N_h \leq \frac{\Lambda_1}{\mu_h}$.

Similarly,

$$\frac{dN_c}{dt} = \Lambda_2 - \mu_c N_c - \mu_{c1} I_{ca} - \mu_{c2} I_{cr} - \mu_{c3} I_{car},$$

and $N_c \leq \frac{\Lambda_2}{\mu_c} + B e^{-\mu_c t}$. As $t \rightarrow \infty$, $B e^{-\mu_c t} \rightarrow 0$, hence, $N_c \leq \frac{\Lambda_2}{\mu_c}$.

Therefore, the feasible solution of the model is positively invariant. The Proofs of theorem 1 and 2 shows that the system as described is well-posed epidemiologically and mathematically, hence can be analyzed.

3. MODEL ANALYSIS

3.1 Anthrax only model: When we exclude the rabies infection, we have the anthrax only model as:

$$\frac{dS_h}{dt} = \Lambda_1 - (\alpha_1 + \mu_h)S_h$$

$$\frac{dS_c}{dt} = \Lambda_2 - (\beta_1 + \mu_c)S_c + \omega_1 R_{ca}$$

$$\frac{dE_{ha}}{dt} = \alpha_1 S_h - (\lambda_1 + \mu_h)E_{ha}$$

$$\frac{dE_{ca}}{dt} = \beta_1 S_c - (\tau_1 + \mu_c)E_{ca}$$

$$\frac{dI_{ha}}{dt} = \lambda_1 E_{ha} - (\mu_h + \mu_{h1})I_{ha}$$

$$\frac{dI_{ca}}{dt} = \tau_1 E_{ca} - (\sigma_1 + \mu_c + \mu_{c1})I_{ca}$$

$$\frac{dR_{ca}}{dt} = \sigma_1 I_{ca} - (\omega_1 + \mu_c)R_{ca}$$

$$\frac{dP}{dt} = \delta_1 (I_{ha} + I_{ca}) - \mu_p P$$

where $\alpha_1 = a_1 P$, $\beta_1 = b_1 (I_{ha} + I_{ca})$ and $a_1, b_1 \neq 0$.

3.1.1 Disease free equilibrium (DFE) point of the Anthrax model (E_a^0)

The disease-free equilibrium is the equilibrium state in absence of infection (Peter et al [40]). This implies a state where we have absence of infection and it is denoted by E^0 . Disease free equilibrium is gotten by setting the infected classes that is the compartments that contributes to the spread of the disease in the population to zero and then equating all the equations of the model to zero and simplifying to find the values of the other classes (Duru et al [19]).

The infectious class for the anthrax model is: $\{E_{ha}, I_{ha}, E_{ca}, I_{ca}, P\}$, thus, $E_{ha} = I_{ha} = E_{ca} = I_{ca} = P = 0$.

Thus, the disease-free equilibrium (DFE) for the anthrax model is

$$E_a^0 = \{S_h^0, S_c^0, E_{ha}^0, E_{ca}^0, I_{ha}^0, I_{ca}^0, R_{ca}^0, P^0\} = \left(\frac{\Lambda_1}{\mu_h}, \frac{\Lambda_2}{\mu_c}, 0, 0, 0, 0, 0, 0\right).$$

3.1.2 Basic reproduction number of the Anthrax model (R_0^a)

In an epidemiological model, the basic reproduction number can be defined as the expected number of secondary cases directly generated by one case in a completely susceptible population and is denoted by R_0 , (Mahato and Mishra [32]).

The basic reproduction number is calculated using the Next-Generation matrix, $R_0 = \rho(FV^{-1})$, where ρ is the dominant eigenvalue of the matrix FV^{-1} , F and V are the matrices of new infection terms and the remaining transmission terms, respectively. Let

$$\frac{dx_i}{dt} = \mathcal{F}_i(x, y) - \mathcal{V}_i(x, y) \quad i = 1, 2, \dots, n \quad (\text{Disease class})$$

$$\frac{dy_i}{dt} = G_i(x, y) \quad i = 1, 2, \dots, m \quad (\text{Non-disease class})$$

F_i =Rate at which new infected enter compartment i .

V_i =Transfer of individuals into and out of i th compartment by other means.

So, we obtain our $F = \frac{\partial \mathcal{F}}{\partial x_j}(E_a^0)$ and $V = \frac{\partial \mathcal{V}_i}{\partial x_j}(E_a^0)$ by differentiating $\mathcal{F}$ and $\mathcal{V}$ respectively

and evaluating them at the disease-free equilibrium E_a^0 . Then, we proceed to find $\rho(FV^{-1})$ with the dominant eigenvalue denoted by ρ as our basic reproduction number. The matrices $\mathcal{F}$ and $\mathcal{V}$ are given by

$$\mathcal{F} = \begin{bmatrix} a_1 P S_h \\ b_1 (I_{ha} + I_{ca}) S_c \\ 0 \\ 0 \\ 0 \end{bmatrix} \quad \text{and} \quad \mathcal{V} = \begin{bmatrix} (\lambda_1 + \mu_h) E_{ha} \\ (\tau_1 + \mu_c) E_{ca} \\ (\mu_h + \mu_{h1}) I_{ha} - \lambda_1 E_{ha} \\ (\sigma_1 + \mu_c + \mu_{c1}) I_{ca} - \tau_1 E_{ca} \\ \mu_P P - \delta_1 (I_{ha} + I_{ca}) \end{bmatrix},$$

while the Jacobian of $\mathcal{F}$ and $\mathcal{V}$ at E_a^0 is given by,

$$F = \begin{bmatrix} 0 & 0 & 0 & 0 & \frac{a_1 \Lambda_1}{\mu_h} \\ 0 & 0 & \frac{b_1 \Lambda_2}{\mu_c} & \frac{b_1 \Lambda_2}{\mu_c} & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix} \quad \text{and} \quad V = \begin{bmatrix} \lambda_1 + \mu_h & 0 & 0 & 0 & 0 \\ 0 & \tau_1 + \mu_c & 0 & 0 & 0 \\ -\lambda_1 & 0 & \mu_h + \mu_{h1} & 0 & 0 \\ 0 & -\tau_1 & 0 & \sigma_1 + \mu_c + \mu_{c1} & 0 \\ 0 & 0 & -\delta_1 & -\delta_1 & \mu_P \end{bmatrix}.$$

$$V^{-1} = \begin{bmatrix} \frac{1}{\lambda_1 + \mu_h} & 0 & 0 & 0 & 0 \\ 0 & \frac{1}{\tau_1 + \mu_c} & 0 & 0 & 0 \\ \frac{\lambda_1}{(\lambda_1 + \mu_h)(\mu_h + \mu_{h1})} & 0 & \frac{1}{\mu_h + \mu_{h1}} & 0 & 0 \\ 0 & \frac{\tau_1}{(\tau_1 + \mu_c)(\sigma_1 + \mu_c + \mu_{c1})} & 0 & \frac{1}{(\sigma_1 + \mu_c + \mu_{c1})} & 0 \\ \frac{\delta_1 \lambda_1}{(\lambda_1 + \mu_h)(\mu_h + \mu_{h1})\mu_P} & \frac{\delta_1 \tau_1}{(\tau_1 + \mu_c)(\sigma_1 + \mu_c + \mu_{c1})\mu_P} & \frac{\delta_1}{(\mu_h + \mu_{h1})\mu_P} & \frac{\delta_1}{(\sigma_1 + \mu_c + \mu_{c1})\mu_P} & \frac{1}{\mu_P} \end{bmatrix}$$

FV^{-1}

$$= \begin{bmatrix} \frac{a_1 \Lambda_1 \delta_1 \lambda_1}{(\lambda_1 + \mu_h)(\mu_h + \mu_{h1})\mu_h \mu_P} & \frac{a_1 \Lambda_1 \delta_1 \tau_1}{(\tau_1 + \mu_c)(\sigma_1 + \mu_c + \mu_{c1})\mu_h \mu_P} & \frac{a_1 \Lambda_1 \delta_1}{(\mu_h + \mu_{h1})\mu_h \mu_P} & \frac{a_1 \Lambda_1 \delta_1}{(\sigma_1 + \mu_c + \mu_{c1})\mu_h \mu_P} & \frac{a_1 \Lambda_1}{\mu_h \mu_P} \\ \frac{b_1 \Lambda_2 \lambda_1}{(\lambda_1 + \mu_h)(\mu_h + \mu_{h1})\mu_c} & \frac{b_1 \Lambda_2 \tau_1}{(\tau_1 + \mu_c)(\sigma_1 + \mu_c + \mu_{c1})\mu_c} & \frac{b_1 \Lambda_2}{(\mu_h + \mu_{h1})\mu_c} & \frac{b_1 \Lambda_2}{(\sigma_1 + \mu_c + \mu_{c1})\mu_c} & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

The eigenvalues of $FV^{-1} = \begin{bmatrix} 0 \\ 0 \\ 0 \\ \frac{a_1\Lambda_1\delta_1\lambda_1}{(\lambda_1+\mu_h)(\mu_h+\mu_{h1})\mu_h\mu_p} + \frac{b_1\Lambda_2\tau_1}{(\tau_1+\mu_c)(\sigma_1+\mu_c+\mu_{c1})\mu_c} \\ 0 \end{bmatrix}$.

Hence the dominate eigenvalue is $R_0^a = \frac{a_1\Lambda_1\delta_1\lambda_1}{(\lambda_1+\mu_h)(\mu_h+\mu_{h1})\mu_h\mu_p} + \frac{b_1\Lambda_2\tau_1}{(\tau_1+\mu_c)(\sigma_1+\mu_c+\mu_{c1})\mu_c}$.

3.1.3 Local stability of the disease-free equilibrium point

The Lyapunov's indirect method is used to analyze the local stability analysis of the disease-free equilibrium point by determining whether the eigenvalues of the Jacobian matrix of the model at DFE are negative or have a negative real part when $R_0 < 1$. The Jacobian of the system at the DFE point is gotten as

$$J = \begin{pmatrix} -\mu_h & 0 & 0 & 0 & 0 & 0 & 0 & \frac{-a_1\Lambda_1}{\mu_h} \\ 0 & -\mu_c & 0 & 0 & \frac{-b_1\Lambda_2}{\mu_c} & \frac{-b_1\Lambda_2}{\mu_c} & \omega_1 & 0 \\ 0 & 0 & -(\lambda_1 + \mu_h) & 0 & 0 & 0 & 0 & \frac{a_1\Lambda_1}{\mu_h} \\ 0 & 0 & 0 & -(\tau_1 + \mu_c) & \frac{b_1\Lambda_2}{\mu_c} & \frac{b_1\Lambda_2}{\mu_c} & 0 & 0 \\ 0 & 0 & \lambda_1 & 0 & -(\mu_h + \mu_{h1}) & 0 & 0 & 0 \\ 0 & 0 & 0 & \tau_1 & 0 & -(\sigma_1 + \mu_c + \mu_{c1}) & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \sigma_1 & -(\omega_1 + \mu_c) & 0 \\ 0 & 0 & 0 & 0 & \delta_1 & \delta_1 & 0 & -\mu_p \end{pmatrix}$$

Reducing the Jacobian matrix by removing the columns with one entry and its corresponding row, we have

$$J_1 = \begin{pmatrix} -(\lambda_1 + \mu_h) & 0 & 0 & 0 & \frac{a_1\Lambda_1}{\mu_h} \\ 0 & -(\tau_1 + \mu_c) & \frac{b_1\Lambda_2}{\mu_c} & \frac{b_1\Lambda_2}{\mu_c} & 0 \\ \lambda_1 & 0 & -(\mu_h + \mu_{h1}) & 0 & 0 \\ 0 & \tau_1 & 0 & -(\sigma_1 + \mu_c + \mu_{c1}) & 0 \\ 0 & 0 & \delta_1 & \delta_1 & -\mu_p \end{pmatrix}.$$

The elements $(-\mu_h, -\mu_c, -(\omega_1 + \mu_c))$ in the deleted columns of the Jacobian matrix, J and the eigenvalues of the Jacobian submatrix J_1 are the eigenvalues of J . The characteristic polynomial associated with the Jacobian submatrix J_1 is given by

$$P(W) = W_0\lambda^5 + W_1\lambda^4 + W_2\lambda^3 + W_3\lambda^2 + W_4\lambda + W_5,$$

where

$$W_0 = 1,$$

$$W_1 = \mu_p + B_1 + B_2 + B_3 + B_4,$$

$$W_2 = \mu_p(B_1 + B_2 + B_3 + B_4) + B_1(B_2 + B_3 + B_4) + B_3(B_2 + B_4) + B_2B_4(1 - R_0^a) + \frac{A_1B_2B_4\delta_1\lambda_1}{B_1B_3\mu_p},$$

$$W_3 = B_1B_2B_4(1 - R_0^a) + B_2B_3B_4(1 - R_0^a) + \mu_pB_2B_4(1 - R_0^a) + \mu_pB_1B_3(1 - R_0^a) + \mu_pB_4(B_1 + B_3)$$

$$+ \mu_pB_2(B_1 + B_3) + B_1B_3(B_2 + B_4) + \frac{A_2B_1B_3\tau_1\mu_p}{B_2B_4} + \frac{A_1B_2B_4\delta_1\lambda_1}{B_1B_3} + \frac{A_1B_2B_4\delta_1\lambda_1}{\mu_pB_1} + \frac{A_1B_2B_4\delta_1\lambda_1}{\mu_pB_3},$$

$$W_4 = \mu_pB_1B_2B_3(1 - R_0^a) + \mu_pB_1B_3B_4(1 - R_0^a) + \mu_pB_2B_3B_4(1 - R_0^a) + \mu_pB_1B_2B_4(1 - R_0^a) + B_1B_2B_3B_4(1 - R_0^a) + A_2B_1B_3\tau_1\mu_p \left[\frac{B_2+B_4}{B_2B_4} \right] + A_1B_2B_4\delta_1\lambda_1 \left[\frac{B_1+B_3}{B_1B_3} \right] + \frac{A_1B_2B_4\delta_1\lambda_1}{\mu_p},$$

$$W_5 = \mu_pB_1B_2B_3B_4(1 - R_0^a),$$

taking $A_1 = \frac{a_1\Lambda_1}{\mu_h}$, $A_2 = \frac{b_1\Lambda_2}{\mu_c}$, $B_1 = (\lambda_1 + \mu_h)$, $B_2 = (\tau_1 + \mu_c)$, $B_3 = (\mu_h + \mu_{h1})$ and $B_4 = (\sigma_1 + \mu_c + \mu_{c1})$.

By Descartes rules of signs (Eze et al[20], Wang [47]), there will be no sign changes in $P(W)$ if $R_0^a < 1$ and if there are no sign changes, then all the roots of the polynomial which corresponds to the eigenvalues of the Jacobian submatrix, J_1 will be negative. Thus, the DFE point of the system will be LAS if $R_0^a < 1$.

3.1.4 Global stability of the disease-free equilibrium

The Castillo-Chavez method is employed to check if the disease-free equilibrium is globally asymptotically stable. Let the model (system) be written in this form

$$\frac{dx}{dt} = F(x, y),$$

$$\frac{dy}{dt} = G(x, y), G(x, 0) = 0,$$

where $F(x, y)$ are the systems of differential equations satisfied by non-disease classes, $G(x, y)$ are the systems of differential equations satisfied by the disease classes. The conditions (C1) and (C2) must hold to guarantee global asymptotic stability at the disease-free equilibrium point,

$$(C1) \quad \text{For } \frac{dx}{dt} = F(x, 0), x^0 \text{ is globally asymptotically stable.}$$

$$(C2) \quad \hat{G}(x, y) = AI - G(x, y), \hat{G}(x, y) \geq 0 \text{ for } (x, y) \in \Omega.$$

where A is the jacobian matrix of $G(x, y)$ evaluated at the disease-free equilibrium point and Ω is the region where the model makes biological sense (Castillo-Chavez et al [15], Duru et al [19]).

$$\begin{aligned}
F(x, 0) &= \frac{dS_h}{dt} = \Lambda_1 - \mu_h S_h, \\
\frac{dS_c}{dt} &= \Lambda_2 - \mu_c S_c + \omega_1 R_{ca}, \\
\frac{dR_{ca}}{dt} &= -(\omega_1 + \mu_c) R_{ca}.
\end{aligned}$$

Solving the resulting equations at the DFE point $S_h(0) = S_h^0, S_c(0) = S_c^0, R_{ca}(0) = R_{ca}^0$ as $t \rightarrow \infty$ gives

$$\begin{aligned}
S_h &= \frac{\Lambda_1}{\mu_h} + c_1 e^{-\mu_h t} \rightarrow \frac{\Lambda_1}{\mu_h}, \quad S_c = \frac{\Lambda_2 + \omega_1 R_{ca}}{\mu_c} + c_2 e^{-\mu_c t} = \frac{\Lambda_2 + \omega_1 R_{ca}^0}{\mu_c} \rightarrow \frac{\Lambda_2}{\mu_c}, \quad R_{ca} = \\
&c_3 e^{-(\omega_1 + \mu_c)t} \rightarrow 0.
\end{aligned}$$

Hence, the solution tends to the disease-free equilibrium point irrespective of the size of the initial population. Therefore, C1 is satisfied.

$$\begin{aligned}
\frac{dI}{dt} &= G(x, I) \Rightarrow \frac{dE_{ha}}{dt} = \alpha_1 S_h - (\lambda_1 + \mu_h) E_{ha}, \\
\frac{dE_{ca}}{dt} &= \beta_1 S_c - (\tau_1 + \mu_c) E_{ca}, \\
\frac{dI_{ha}}{dt} &= \lambda_1 E_{ha} - (\mu_h + \mu_{h1}) I_{ha}, \\
\frac{dI_{ca}}{dt} &= \tau_1 E_{ca} - (\sigma_1 + \mu_c + \mu_{c1}) I_{ca}, \\
\frac{dp}{dt} &= \delta_1 (I_{ha} + I_{ca}) - \mu_p P,
\end{aligned}$$

where $\alpha_1 = a_1 P$, $\beta_1 = b_1 I_{ha} + b_1 I_{ca}$ and $a_1, b_1 \neq 0$.

$$\begin{aligned}
A &= \begin{pmatrix} -(\lambda_1 + \mu_h) & 0 & 0 & 0 & \frac{a_1 \Lambda_1}{\mu_h} \\ 0 & -(\tau_1 + \mu_c) & \frac{b_1 \Lambda_2}{\mu_c} & \frac{b_1 \Lambda_2}{\mu_c} & 0 \\ \lambda_1 & 0 & -(\mu_h + \mu_{h1}) & 0 & 0 \\ 0 & \tau_1 & 0 & -(\sigma_1 + \mu_c + \mu_{c1}) & 0 \\ 0 & 0 & \delta_1 & \delta_1 & -\mu_p \end{pmatrix} \text{ and} \\
\hat{G}(x, I) &= \begin{pmatrix} \frac{a_1 P (\Lambda_1 - \mu_h S_h)}{\mu_h} \\ b_1 I_{ha} + b_1 I_{ca} \left(\frac{\Lambda_2 - \mu_c S_c}{\mu_c} \right) \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}.
\end{aligned}$$

$\hat{G}(x, I) \geq 0$ since $\Lambda_1 \geq \mu_h S_h$ and $\Lambda_2 \geq \mu_c S_c$, satisfying the two conditions. Hence, the anthrax model at the DFE is globally asymptotically stable.

3.1.5 Endemic Equilibrium of the anthrax model (E_a^*)

The endemic disease equilibrium state is the state where the disease cannot be totally absent in the various compartments in the population (Duru et al [19]). The endemic equilibrium is gotten by setting the model equation to zero and then solving the system of equations.

The endemic equilibrium of the anthrax model is solved by equating the model to zero and solving simultaneously to obtain $E_a^* = \{S_h^*, S_c^*, E_{ha}^*, E_{ca}^*, I_{ha}^*, I_{ca}^*, R_{ca}^*, P^*\} = \left\{ \frac{\Lambda_1}{(\alpha_1 + \mu_h)}, \frac{\Lambda_2 + \omega_1 R_{ca}^*}{(\beta_1 + \mu_c)}, \frac{\alpha_1 \Lambda_1}{(\lambda_1 + \mu_h)(\alpha_1 + \mu_h)}, \frac{\beta_1(\Lambda_2 + \omega_1 R_{ca}^*)}{(\tau_1 + \mu_c)(\beta_1 + \mu_c)}, \frac{\lambda_1 \alpha_1 \Lambda_1}{(\mu_h + \mu_{h1})(\lambda_1 + \mu_h)(\alpha_1 + \mu_h)}, \frac{\tau_1 \beta_1(\Lambda_2 + \omega_1 R_{ca}^*)}{(\mu_c + \mu_{c1})(\tau_1 + \mu_c)(\beta_1 + \mu_c)}, \frac{\sigma_1 \tau_1 \beta_1 \Lambda_2}{(\omega_1 + \mu_c)(\mu_c + \mu_{c1})(\tau_1 + \mu_c)(\beta_1 + \mu_c) - \sigma_1 \tau_1 \beta_1 \omega_1}, \frac{\delta_1(I_{ha}^* + I_{ca}^*)}{\mu_p} \right\}$.

3.1.6 Existence and uniqueness of the endemic equilibrium

To show the existence and uniqueness of the endemic equilibrium, the conditions stated by (Massawe et al [33]) is used. For the endemic equilibrium point to exist, then $S_h^* > 0, S_c^* > 0, E_{ha}^* > 0, E_{ca}^* > 0, I_{ha}^* > 0, I_{ca}^* > 0$.

Adding up the equation of the system, we have

$$\Lambda_1 + \Lambda_2 - \mu_h(S_h^* + E_{ha}^* + I_{ha}^*) - \mu_c(S_{ca}^* + E_{ca}^* + I_{ca}^* + R_{ca}^*) - \mu_{h1}I_{ha}^* - \mu_{c1}I_{ca}^* - \delta_1(I_{ha}^* + I_{ca}^*) - \mu_p P^* = 0.$$

This can be rewritten as

$$\Lambda_1 + \Lambda_2 = \mu_h(S_h^* + E_{ha}^* + I_{ha}^*) + \mu_c(S_{ca}^* + E_{ca}^* + I_{ca}^* + R_{ca}^*) + \mu_{h1}I_{ha}^* + \mu_{c1}I_{ca}^* + \delta_1(I_{ha}^* + I_{ca}^*) - \mu_p P^*$$

Since $\Lambda_1 + \Lambda_2 > 0$, then $\mu_h(S_h^* + E_{ha}^* + I_{ha}^*) + \mu_c(S_{ca}^* + E_{ca}^* + I_{ca}^*) + \mu_{h1}I_{ha}^* + \mu_{c1}I_{ca}^* > 0$.

This implies $\mu_h(S_h^* + E_{ha}^* + I_{ha}^*) > 0, \mu_c(S_{ca}^* + E_{ca}^* + I_{ca}^*) > 0, \mu_{h1}I_{ha}^* > 0$ and $\mu_{c1}I_{ca}^* > 0$.

Hence, $\mu_h, \mu_c, \mu_{h1}, \mu_{c1} > 0$ and $S_h^*, E_{ha}^*, I_{ha}^*, S_c^*, E_{ca}^*, I_{ca}^*, R_{ca}^* > 0$.

This guarantees the existence of an endemic equilibrium since the state variables are all positive.

3.2 Rabies only model

Removing the anthrax disease from the co-infection model, we have the rabies only model as

$$\frac{dS_h}{dt} = \Lambda_1 - (\alpha_2 + \mu_h)S_h,$$

$$\frac{dS_c}{dt} = \Lambda_2 - (\beta_2 + \mu_c)S_c,$$

$$\frac{dE_{hr}}{dt} = \alpha_2 S_h - (\lambda_2 + \mu_h)E_{hr},$$

$$\frac{dE_{cr}}{dt} = \beta_2 S_c - (\tau_2 + \mu_c) E_{cr},$$

$$\frac{dI_{hr}}{dt} = \lambda_2 E_{hr} - (\mu_h + \mu_{h2}) I_{hr},$$

$$\frac{dI_{cr}}{dt} = \tau_2 E_{cr} - (\mu_c + \mu_{c2}) I_{cr},$$

where $\alpha_2 = c_1 I_{cr}$, $\beta_2 = d_1 I_{cr}$ and $c_1, d_1 \neq 0$.

3.2.1 Disease Free Equilibrium

The infectious class for the Rabies model are: $\{E_{hr}, I_{hr}, E_{cr}, I_{cr}\}$. Setting the infectious classes to zero gives

$E_{hr} = I_{hr} = E_{cr} = I_{cr} = 0$. Then, we equate all the equations in the model to zero, substitute the values of the infectious class as zero and solve to obtain the following;

Thus, the disease-free equilibrium (DFE) of the system is

$$E_r^0 = \{S_h^0, S_c^0, E_{hr}^0, E_{cr}^0, I_{hr}^0, I_{cr}^0\} = \left(\frac{\Lambda_1}{\mu_h}, \frac{\Lambda_2}{\mu_c}, 0, 0, 0, 0\right).$$

3.2.2 Basic Reproduction Number of the rabies model(R_0^r)

Using the Next-generation matrix approach as described for Anthrax, the matrices, $\mathcal{F}$ and $\mathcal{V}$ are

$$\mathcal{F} = \begin{bmatrix} c_1 I_{cr} S_h \\ d_1 I_{cr} S_c \\ 0 \\ 0 \end{bmatrix} \quad \text{and} \quad \mathcal{V} = \begin{bmatrix} (\lambda_2 + \mu_h) E_{hr} \\ (\tau_2 + \mu_c) E_{cr} \\ (\mu_h + \mu_{h2}) I_{hr} - \lambda_2 E_{hr} \\ (\mu_c + \mu_{c2}) I_{cr} - \tau_2 E_{cr} \end{bmatrix}.$$

Finding the Jacobian at disease free equilibrium $E_r^0 = \{S_h^0, S_c^0, E_{hr}^0, E_{cr}^0, I_{hr}^0, I_{cr}^0\} = \left(\frac{\Lambda_1}{\mu_h}, \frac{\Lambda_2}{\mu_c}, 0, 0, 0, 0\right)$

we have

$$F = \begin{bmatrix} 0 & 0 & 0 & \frac{c_1 \Lambda_1}{\mu_h} \\ 0 & 0 & 0 & \frac{d_1 \Lambda_2}{\mu_c} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \quad \text{and} \quad V = \begin{bmatrix} \lambda_2 + \mu_h & 0 & 0 & 0 \\ 0 & \tau_2 + \mu_c & 0 & 0 \\ -\lambda_2 & 0 & \mu_h + \mu_{h2} & 0 \\ 0 & -\tau_2 & 0 & \mu_c + \mu_{c2} \end{bmatrix}.$$

Taking the inverse of the matrix V gives

$$V^{-1} = \begin{bmatrix} \frac{1}{\lambda_2 + \mu_h} & 0 & 0 & 0 \\ 0 & \frac{1}{\tau_2 + \mu_c} & 0 & 0 \\ \frac{\lambda_2}{(\lambda_2 + \mu_h)(\mu_h + \mu_{h2})} & 0 & \frac{1}{\mu_h + \mu_{h2}} & 0 \\ 0 & \frac{\tau_2}{(\tau_2 + \mu_c)(\mu_c + \mu_{c2})} & 0 & \frac{1}{\mu_c + \mu_{c2}} \end{bmatrix},$$

where $FV^{-1} = \begin{bmatrix} 0 & \frac{c_1\Lambda_1\tau_2}{\mu_h(\tau_2+\mu_c)(\mu_c+\mu_{c2})} & 0 & \frac{c_1\Lambda_1}{\mu_h(\mu_c+\mu_{c2})} \\ 0 & \frac{d_1\Lambda_2\tau_2}{\mu_c(\tau_2+\mu_c)(\mu_c+\mu_{c2})} & 0 & \frac{d_1\Lambda_2}{\mu_c(\mu_c+\mu_{c2})} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}$ and the eigenvalues of $FV^{-1} = \begin{bmatrix} 0 \\ 0 \\ 0 \\ \frac{d_1\Lambda_2\tau_2}{\mu_c(\tau_2+\mu_c)(\mu_c+\mu_{c2})} \end{bmatrix}$.

Hence the dominate eigenvalue is $R_0^r = \frac{d_1\Lambda_2\tau_2}{\mu_c(\tau_2+\mu_c)(\mu_c+\mu_{c2})}$.

3.2.3 Local stability of the disease-free equilibrium point

The Jacobian of the rabies model at DFE E_r^0 is

$$J_2 = \begin{pmatrix} -\mu_h & 0 & 0 & 0 & 0 & \frac{-c_1\Lambda_1}{\mu_h} \\ 0 & -\mu_c & 0 & 0 & 0 & \frac{-d_1\Lambda_2}{\mu_c} \\ 0 & 0 & -(\lambda_2 + \mu_h) & 0 & 0 & \frac{c_1\Lambda_1}{\mu_h} \\ 0 & 0 & 0 & -(\tau_2 + \mu_c) & 0 & \frac{d_1\Lambda_2}{\mu_c} \\ 0 & 0 & \lambda_2 & 0 & -(\mu_h + \mu_{h2}) & 0 \\ 0 & 0 & 0 & \tau_2 & 0 & -(\mu_c + \mu_{c2}) \end{pmatrix}$$

This can be reduced further to obtain

$$J_{22} = \begin{pmatrix} -(\tau_2 + \mu_c) & \frac{d_1\Lambda_2}{\mu_c} \\ \tau_2 & -(\mu_c + \mu_{c2}) \end{pmatrix}.$$

The elements $(-\mu_h, -\mu_c, -(\lambda_2 + \mu_h), -(\mu_h + \mu_{h2}))$ in the columns deleted from the Jacobian matrix, J_2 and the eigenvalues of the Jacobian submatrix J_{22} are the eigenvalues of J_2 . The characteristic polynomial associated with the Jacobian submatrix J_{22} is given by

$$P(\lambda) = \lambda^2 + ((\tau_2 + \mu_c) + (\mu_c + \mu_{c2}))\lambda + (\tau_2 + \mu_c)(\mu_c + \mu_{c2})(1 - R_0^r).$$

By Descartes rules of signs [47], there will be no sign changes in $P(\lambda)$ if $R_0^r < 1$ and if there are no sign changes, then all the roots of the polynomial which corresponds to the eigenvalues of the Jacobian submatrix, J_1 will be negative. Thus, the DFE point of the system will be LAS if $R_0^r < 1$.

3.2.4 Global stability of the disease-free equilibrium

Using the Castillo-Chavez theorem on global stability, Let the model (system) be written in this form

$$\frac{dx}{dt} = F(x, 0) = \frac{dS_h}{dt} = \Lambda_1 - \mu_h S_h,$$

$$\frac{dS_c}{dt} = \Lambda_2 - \mu_c S_c$$

Solving the resulting equations by using integrating factor with initial conditions $S_h(0) = S_h^0, S_c(0) = S_c^0$ as $t \rightarrow \infty$ gives

$$S_h = \frac{\Lambda_1}{\mu_h} + c_1 e^{-\mu_h t} \rightarrow \frac{\Lambda_1}{\mu_h},$$

$$S_c = \frac{\Lambda_2}{\mu_c} + c_2 e^{-\mu_c t} \rightarrow \frac{\Lambda_2}{\mu_c}.$$

Hence, the solution tends to the disease-free equilibrium point irrespective of the size of the initial population. Therefore, C1 is satisfied.

$$\frac{dI}{dt} = G(x, I) \Rightarrow \frac{dE_{hr}}{dt} = c_1 I_{cr} S_h - (\lambda_2 + \mu_h) E_{hr},$$

$$\frac{dE_{cr}}{dt} = d_1 I_{cr} S_c - (\tau_2 + \mu_c) E_{cr},$$

$$\frac{dI_{hr}}{dt} = \lambda_2 E_{hr} - (\mu_h + \mu_{h2}) I_{hr},$$

$$\frac{dI_{cr}}{dt} = \tau_2 E_{cr} - (\mu_c + \mu_{c2}) I_{cr},$$

$$A = \begin{pmatrix} -(\lambda_2 + \mu_h) & 0 & 0 & \frac{c_1 \Lambda_1}{\mu_h} \\ 0 & -(\tau_2 + \mu_c) & 0 & \frac{d_1 \Lambda_2}{\mu_c} \\ \lambda_2 & 0 & -(\mu_h + \mu_{h2}) & 0 \\ 0 & \tau_2 & 0 & -(\mu_c + \mu_{c2}) \end{pmatrix} \text{ and } \hat{G}(x, I) = \begin{pmatrix} \frac{c_1 I_{cr} (\Lambda_1 - \mu_h S_h)}{\mu_h} \\ \frac{d_1 I_{cr} (\Lambda_2 - \mu_c S_c)}{\mu_c} \\ 0 \\ 0 \end{pmatrix}$$

$\hat{G}(x, I) \geq 0$ since $\Lambda_1 \geq \mu_h S_h$ and $\Lambda_2 \geq \mu_c S_c$, therefore the rabies model at the DFE will be globally asymptotically stable. The global asymptotical stability of the disease-free equilibrium point shows that no matter the initial sizes of the infected population, rabies will be controlled in the system when the $R_0^r < 1$.

3.3 Co-infection model

3.3.1 Disease Free Equilibrium

The infectious class for the Co-infection model are:

$$\{E_{ha}, E_{ca}, E_{hr}, E_{cr}, E_{har}, E_{car}, I_{ha}, I_{ca}, I_{hr}, I_{cr}, I_{har}, I_{car}, P\}.$$

Then, we equate all the equations in the model to zero to obtain the disease-free equilibrium (DFE) of the system is

3.3.2 Basic Reproduction Number (R_0)

$$\mathcal{F} = \begin{bmatrix} a_1 P S_h \\ b_1(I_{ha} + I_{ca} + I_{har} + I_{car})S_c \\ c_1(I_{cr} + I_{car})S_h \\ d_1(I_{cr} + I_{car})S_c \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix} \quad \text{and} \quad \mathcal{V} = \begin{bmatrix} (e_1 I_{cr} + e_1 I_{car} + \lambda_1 + \mu_h)E_{ha} \\ (f_1 I_{cr} + f_1 I_{car} + \tau_1 + \mu_c)E_{ca} \\ (g_1 P + \lambda_2 + \mu_h)E_{hr} \\ (h_1 I_{ha} + h_1 I_{ca} + h_1 I_{har} + h_1 I_{car} + \tau_2 + \mu_c)E_{cr} \\ -(e_1 I_{cr} + e_1 I_{car})E_{ha} - g_1 P E_{hr} + (\lambda_3 + \mu_h)E_{har} \\ -(f_1 I_{cr} + f_1 I_{car})E_{ca} - (h_1 I_{ha} + h_1 I_{ca} + h_1 I_{har} + h_1 I_{car})E_{cr} + (\tau_3 + \mu_c)E_{car} \\ -\lambda_1 E_{ha} + (\mu_h + \mu_{h1})I_{ha} \\ -\tau_1 E_{ca} + \sigma_1 I_{ca} + (\mu_c + \mu_{c1})I_{ca} \\ -\lambda_2 E_{hr} + (\mu_h + \mu_{h2})I_{hr} \\ -\tau_2 E_{cr} + (\mu_c + \mu_{c2})I_{cr} \\ -\lambda_3 E_{har} + (\mu_h + \mu_{h3})I_{har} \\ -\tau_3 E_{car} + (\mu_c + \mu_{c3})I_{car} \\ -\delta_1(I_{ha} + I_{ca} + I_{har} + I_{car}) + \mu_p P \end{bmatrix}$$
$$E_{ar}^0 = \{S_h^0, S_c^0, E_{ha}^0, E_{ca}^0, E_{har}^0, E_{hr}^0, E_{cr}^0, E_{car}^0, I_{ha}^0, I_{ca}^0, I_{hr}^0, I_{cr}^0, I_{har}^0, I_{car}^0, R_{ca}^0, P\}$$

$$= \left(\frac{\Lambda_1}{\mu_h}, \frac{\Lambda_2}{\mu_c}, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0 \right)$$

we have

[illegible]

and

$$V = \begin{bmatrix} \lambda_1 + \mu_h & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \tau_1 + \mu_c & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \lambda_2 + \mu_h & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \tau_2 + \mu_c & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \lambda_3 + \mu_h & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \tau_3 + \mu_c & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ -\lambda_1 & 0 & 0 & 0 & 0 & 0 & \mu_h + \mu_{h1} & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & -\tau_1 & 0 & 0 & 0 & 0 & 0 & \sigma_1 + \mu_c + \mu_{c1} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -\lambda_2 & 0 & 0 & 0 & 0 & 0 & \mu_h + \mu_{h2} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -\tau_2 & 0 & 0 & 0 & 0 & 0 & \mu_c + \mu_{c2} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\lambda_3 & 0 & 0 & 0 & 0 & 0 & \mu_h + \mu_{h3} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -\tau_3 & 0 & 0 & 0 & 0 & 0 & \mu_c + \mu_{c3} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -\delta_1 & -\delta_1 & 0 & 0 & -\delta_1 & -\delta_1 & \mu_p \end{bmatrix}$$

$$\text{Eigen values of } FV^{-1} = \begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ \frac{a_1 \Lambda_1 \delta_1 \lambda_1}{(\lambda_1 + \mu_h)(\mu_h + \mu_{h1})\mu_h \mu_P} + \frac{b_1 \Lambda_2 \tau_1}{(\tau_1 + \mu_c)(\sigma_1 + \mu_c + \mu_{c1})\mu_c} \\ \frac{d_1 \Lambda_2 \tau_2}{\mu_c(\tau_2 + \mu_c)(\mu_c + \mu_{c2})} \end{bmatrix}$$

Hence the dominate Eigen value is $R_0^{ar} = a + b$ or rs

That is $R_0^{ar} = \max\{R_0^a, R_0^r\}$

3.3.3 Local stability of the disease-free equilibrium

Finding the Jacobian of the co-infection model at disease free equilibrium and finding the Eigen values

$$\begin{pmatrix} -(\tau_3 + \mu_c) \\ -(\mu_c + \mu_{c3}) \\ -\frac{1}{2}(\tau_2 + \mu_c) - \frac{1}{2}(\mu_c + \mu_{c2}) + \frac{1}{2}\sqrt{\frac{4d_1\Lambda_2\tau_2}{\mu_c} + (\tau_2 + \mu_c)^2 - 2(\tau_2 + \mu_c)(\mu_c + \mu_{c2}) + (\mu_c + \mu_{c2})^2} \\ -\frac{1}{2}(\tau_2 + \mu_c) - \frac{1}{2}(\mu_c + \mu_{c2}) - \frac{1}{2}\sqrt{\frac{4d_1\Lambda_2\tau_2}{\mu_c} + (\tau_2 + \mu_c)^2 - 2(\tau_2 + \mu_c)(\mu_c + \mu_{c2}) + (\mu_c + \mu_{c2})^2} \\ -(\lambda_3 + \mu_h) \\ -(\mu_h + \mu_{h3}) \\ \text{Roots of } Z^5 + KZ^4 + LZ^3 + MZ^2 + NZ + Q \\ -\mu_h \\ -(\omega_1 + \mu_c) \\ -\mu_c \\ -(\lambda_2 + \mu_h) \\ -(\mu_h + \mu_{h2}) \end{pmatrix}$$

Where $K = l + m + n + q + \mu_p$

$$L = lm + ln + lq + l\mu_p + mn + mq + nq + m\mu_p + n\mu_p + q\mu_p - f\tau_1$$

$$M = lm\mu_p + lmn + lmq + ln\mu_p + lq\mu_p + lnq + mn\mu_p + mq\mu_p + mnq + nq\mu_p - e\lambda_1\delta_1 - fl\tau_1 - f\tau_1\mu_p - fn\tau_1$$

$$N = lmn\mu_p + lmq\mu_p + lmnq + lnq\mu_p + mnq\mu_p - e\lambda_1\delta_1m - e\lambda_1\delta_1q - l\tau_1\mu_p - fln\tau_1 - fn\mu_p\tau_1$$

$$Q = lmnq\mu_p - emq\lambda_1\delta_1 - lnf\mu_p\tau_1$$

$$\text{And } e = \frac{a_1\Lambda_1}{k\mu_h}, \quad f = \frac{b_1\Lambda_2}{\mu_c}, \quad l = \lambda_1 + \mu_h, \quad m = \tau_1 + \mu_c$$

$$n = \mu_h + \mu_{h1}, \quad q = \sigma_1 + \mu_c + \mu_{c1}$$

Every other eigenvalues have negative real part. Then we investigated the stability of the characteristics equation $Z^5 + KZ^4 + LZ^3 + MZ^2 + NZ + Q$ using the Descartes rules of signs. Descartes rule of signs states that, let $P(Z) = Z^5 + KZ^4 + LZ^3 + MZ^2 + NZ + Q$ be a polynomial with real co-efficient:

- The number of positive zeros of P is either equal to the number of variations in sign of $P(Z)$ or less than this by an even number.
- The number of negative zeros in P is either equal to the number of variations in sign of $P(-Z)$ or less than this by an even number, [20].

Thus let's investigate the sign change in $P(Z) = Z^5 + KZ^4 + LZ^3 + MZ^2 + NZ + Q$

Proof:

- Check if $K = l + m + n + q + \mu_p > 0$. Since $l, m, n, q, \mu_p > 0$ then $K > 0$.
- Check if $L = lm + ln + lq + l\mu_p + mn + mq + nq + m\mu_p + n\mu_p + q\mu_p - f\tau_1 > 0$.

$$L = lm + ln + lq + l\mu_p + mn + nq + m\mu_p + n\mu_p + q\mu_p + \frac{mqe\lambda_1\delta_1}{ln\mu_p} + mq(1 - R_0^a)$$

Thus $L > 0$ if $(1 - R_0^a) > 0$. Hence completing the proof.

- Check if $M = lm\mu_p + lmn + lmq + ln\mu_p + lq\mu_p + lnq + mn\mu_p + mq\mu_p + mnq + nq\mu_p - e\lambda_1\delta_1 - lf\tau_1 - f\tau_1\mu_p - fn\tau_1 > 0$

$$\begin{aligned} M &= lm\mu_p + lmn + lmq + ln\mu_p + lq\mu_p + lnq + mn\mu_p + mq\mu_p + mnq + nq\mu_p - ln\mu_p R_0^a + \frac{ln\mu_p f\tau_1}{mq} \\ &\quad - lmqR_0^a + \frac{mqe\lambda_1\delta_1}{n\mu_p} - mq\mu_p R_0^a + \frac{mqe\lambda_1\delta_1}{ln} - nmqR_0^a + \frac{mqe\lambda_1\delta_1}{l\mu_p} \\ &= lm\mu_p + lmn + lq\mu_p + lnq + mn\mu_p + nq\mu_p \\ &\quad + (lmq + mnq + mq\mu_p + ln\mu_p)(1 - R_0^a) + \frac{ln\mu_p f\tau_1}{mq} + \left(\frac{1}{n\mu_p} + \frac{1}{ln} + \frac{1}{l\mu_p}\right)mqe\lambda_1\delta_1 \end{aligned}$$

Thus, $M > 0$ if $(1 - R_0^a) > 0$. This is true for disease free, thus completing the proof.

- Check if $N > 0$ and $N = lmn\mu_p + lmq\mu_p + lmnq + lnq\mu_p + mnq\mu_p - e\lambda_1\delta_1 m - e\lambda_1\delta_1 q - lf\tau_1\mu_p - fln\tau_1 - fn\mu_p\tau_1$

$$\begin{aligned} N &= lmn\mu_p + lmq\mu_p + lmnq + lnq\mu_p + mnq\mu_p - lmn\mu_p R_0^a + \frac{ln\mu_p f\tau_1}{q} - lnq\mu_p R_0^a + \frac{ln\mu_p f\tau_1}{m} \\ &\quad - lmq\mu_p R_0^a + \frac{mqe\lambda_1\delta_1}{n} - lmnqR_0^a + \frac{mqe\lambda_1\delta_1}{\mu_p} - mnq\mu_p R_0^a + \frac{mqe\lambda_1\delta_1}{l} \\ &= (1 - R_0^a)(lmn\mu_p + lmq\mu_p + lmnq + lnq\mu_p + mnq\mu_p) + mqe\lambda_1\delta_1 \left(\frac{1}{l} + \frac{1}{n} + \frac{1}{\mu_p}\right) \\ &\quad + ln\mu_p f\tau_1 \left(\frac{1}{m} + \frac{1}{q}\right) \end{aligned}$$

$$(1 - R_0^a)(lmn\mu_p + lmq\mu_p + lmnq + lnq\mu_p + mnq\mu_p) + mqe\lambda_1\delta_1 \left(\frac{1}{l} + \frac{1}{n} + \frac{1}{\mu_p}\right) +$$

$ln\mu_p f\tau_1 \left(\frac{1}{m} + \frac{1}{q}\right) > 0$ if $(1 - R_0^a)$. This is true for disease free population, thus completing the proof.

- Check if $Q > 0$ and $Q = lmnq\mu_p - (emq\lambda_1\delta_1 + lnf\mu_p\tau_1) = lmnq\mu_p - lmnq\mu_p R_0^a = lmnq\mu_p(1 - R_0^a)$. Thus, $Q > 0$ if $(1 - R_0^a) > 0$.

Since there is no sign change and $K, L, M, N, Q > 0$, by Descartes rules of signs, the polynomial have no positive Eigen value. Hence the disease free equilibrium of the co-infection model is locally asymptotically stable.

3.3.4 Global stability of the disease-free equilibrium

Using the Castillo-Chavez theorem on global stability, Let the model (system) be written in this form

$$\frac{dx}{dt} = F(x, I) \Rightarrow \frac{dS_h}{dt} = \Lambda_1 - (a_1P + c_1I_{cr} + c_1I_{car} + \mu_h)S_h$$

$$\frac{dS_c}{dt} = \Lambda_2 - (b_1I_{ha} + b_1I_{ca} + b_1I_{har} + b_1I_{car} + d_1I_{cr} + d_1I_{car} + \mu_c)S_c + \omega_1R_{ca}$$

$$\frac{dR_{ca}}{dt} = \sigma_1I_{ca} - (\omega_1 + \mu_c)R_{ca}$$

$$F(x, 0) = \frac{dS_h}{dt} = \Lambda_1 - \mu_hS_h$$

$$\frac{dS_c}{dt} = \Lambda_2 - \mu_cS_c + \omega_1R_{ca}$$

$$\frac{dR_{ca}}{dt} = -(\omega_1 + \mu_c)R_{ca}$$

Solving the resulting equations by using integrating factor with initial conditions $S_h(0) = S_h^0, S_c(0) = S_c^0$ as $t \rightarrow \infty$

$$S_h = \frac{\Lambda_1}{\mu_h} + c_1e^{-\mu_h t} \rightarrow \frac{\Lambda_1}{\mu_h}$$

$$S_c = \frac{\Lambda_2}{\mu_c} + c_2e^{-\mu_c t} \rightarrow \frac{\Lambda_2}{\mu_c}$$

$$R_{ca} = c_3e^{-(\omega_1 + \mu_c)t} \rightarrow 0$$

Hence, the solution tends to the disease-free equilibrium irrespective of the size of the initial population. Therefore, C1 is satisfied.

$$\frac{dI}{dt} = G(x, I) \Rightarrow \frac{dE_{ha}}{dt} = a_1PS_h - (e_1I_{cr} + e_1I_{car}) + \lambda_1 + \mu_h)E_{ha}$$

$$\frac{dE_{ca}}{dt} = b_1(I_{ha} + I_{ca} + I_{har} + I_{car})S_c - (f_1I_{cr} + f_1I_{car} + \tau_1 + \mu_c)E_{ca}$$

$$\frac{dE_{hr}}{dt} = c_1(I_{cr} + I_{car})S_h - (g_1P + \lambda_2 + \mu_h)E_{hr}$$

$$\frac{dE_{cr}}{dt} = d_1(I_{cr} + I_{car})S_c - (h_1I_{ha} + h_1I_{ca} + h_1I_{har} + h_1I_{car} + \tau_2 + \mu_c)E_{cr}$$

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$$\frac{dE_{har}}{dt} = e_1(I_{cr} + I_{car})E_{ha} + g_1PE_{hr} - (\lambda_3 + \mu_h)E_{har}$$

$$\frac{dE_{car}}{dt} = f_1(I_{cr} + I_{car})E_{ca} + h_1(I_{ha} + I_{ca} + I_{har}I_{car})E_{cr} - (\tau_3 + \mu_c)E_{car}$$

$$\frac{dI_{ha}}{dt} = \lambda_1E_{ha} - (\mu_h + \mu_{h1})I_{ha}$$

$$\frac{dI_{ca}}{dt} = \tau_1E_{ca} - \sigma_1I_{ca} - (\mu_c + \mu_{c1})I_{ca}$$

$$\frac{dI_{hr}}{dt} = \lambda_2E_{hr} - (\mu_h + \mu_{h2})I_{hr}$$

$$\frac{dI_{cr}}{dt} = \tau_2E_{cr} - (\mu_c + \mu_{c2})I_{cr}$$

$$\frac{dI_{har}}{dt} = \lambda_3E_{har} - (\mu_h + \mu_{h3})I_{har}$$

$$\frac{dI_{car}}{dt} = \tau_3E_{car} - (\mu_c + \mu_{c3})I_{car}$$

$$\frac{dp}{dt} = \delta_1(I_{ha} + I_{ca} + I_{har} + I_{car}) - \mu_pP$$

$$A = \begin{bmatrix} -(\lambda_1 + \mu_h) & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \frac{a_1\Lambda_1}{\mu_h} \\ 0 & -(\tau_1 + \mu_c) & 0 & 0 & 0 & 0 & \frac{b_1\Lambda_2}{\mu_c} & \frac{b_1\Lambda_2}{\mu_c} & 0 & 0 & \frac{b_1\Lambda_2}{\mu_c} & \frac{b_1\Lambda_2}{\mu_c} & 0 \\ 0 & 0 & -(\lambda_2 + \mu_h) & 0 & 0 & 0 & 0 & 0 & 0 & \frac{c_1\Lambda_1}{\mu_h} & 0 & \frac{c_1\Lambda_1}{\mu_h} & 0 \\ 0 & 0 & 0 & -(\tau_2 + \mu_c) & 0 & 0 & 0 & 0 & 0 & \frac{d_1\Lambda_2}{\mu_c} & 0 & \frac{d_1\Lambda_2}{\mu_c} & 0 \\ 0 & 0 & 0 & 0 & -(\lambda_3 + \mu_h) & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -(\tau_3 + \mu_c) & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ \lambda_1 & 0 & 0 & 0 & 0 & 0 & -(\mu_h + \mu_{h1}) & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \tau_1 & 0 & 0 & 0 & 0 & 0 & -(\sigma_1 + \mu_c + \mu_{c1}) & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \lambda_2 & 0 & 0 & 0 & 0 & 0 & -(\mu_h + \mu_{h2}) & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \tau_2 & 0 & 0 & 0 & 0 & 0 & -(\mu_c + \mu_{c2}) & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \lambda_3 & 0 & 0 & 0 & 0 & 0 & -(\mu_h + \mu_{h3}) & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \tau_3 & 0 & 0 & 0 & 0 & 0 & -(\mu_c + \mu_{c3}) & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \delta_1 & \delta_1 & 0 & 0 & \delta_1 & \delta_1 & -\mu_p \end{bmatrix}$$

$$\hat{G}(x, I) = \begin{pmatrix} \frac{a_1\Lambda_1 + (e_1I_{cr} + e_1I_{car})\mu_hE_{ha} - a_1P\mu_hS_h}{\mu_h} \\ \frac{(\Lambda_2 - \mu_cS_c)(b_1I_{ha} + b_1I_{ca} + b_1I_{har} + b_1I_{car}) + (f_1I_{cr} + f_1I_{car})\mu_cE_{ca}}{\mu_c} \\ \frac{(\Lambda_1 - \mu_hE_{hr})(c_1I_{cr} + c_1I_{car}) + g_1\mu_hP}{\mu_h} \\ \frac{(\Lambda_2 - \mu_cS_c)(d_1I_{cr} + d_1I_{car}) + (h_1I_{ha} + h_1I_{ca} + h_1I_{har} + h_1I_{car})\mu_cE_{cr}}{\mu_c} \\ \frac{-(e_1I_{cr}E_{ha} + e_1I_{car}E_{ha} + g_1PE_{hr})}{-(f_1I_{cr} + f_1I_{car})E_{ca} - (h_1I_{ha} + h_1I_{ca} + h_1I_{har} + h_1I_{car})E_{cr}} \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}$$

$\hat{G}(x, I) \not\geq 0$, the conditions are not satisfied. Hence, the co-infection model at the DFE is not globally asymptotically stable.

3.4 Impact of anthrax disease on the rabies disease

Using the methods described by (Acheneje et al [2]) we examined the relationship between the basic reproduction number of the anthrax only model and the rabies only model in order to investigate the effect of anthrax on the spread of rabies pandemic.

Let's recall that, $R_0^a = \frac{a_1 \Lambda_1 \delta_1 \lambda_1}{(\lambda_1 + \mu_h)(\mu_h + \mu_{h1})\mu_h \mu_P} + \frac{b_1 \tau_1 R_0^r \mu_c (\tau_2 + \mu_c)(\mu_c + \mu_{c2})}{(\tau_1 + \mu_c)(\sigma_1 + \mu_c + \mu_{c1})d_1 \tau_2 \mu_c}$ and

$$R_0^r = \frac{d_1 \Lambda_2 \tau_2}{\mu_c (\tau_2 + \mu_c)(\mu_c + \mu_{c2})},$$

We expressed Λ_2 in terms of either of the basic reproduction numbers as both reproduction number has this parameter, we have

$$R_0^r = \frac{d_1 \Lambda_2 \tau_2}{\mu_c (\tau_2 + \mu_c)(\mu_c + \mu_{c2})}$$

$$\Lambda_2 = \frac{R_0^r \mu_c (\tau_2 + \mu_c)(\mu_c + \mu_{c2})}{d_1 \tau_2}, \text{ thus } b = \frac{b_1 \tau_1 R_0^r \mu_c (\tau_2 + \mu_c)(\mu_c + \mu_{c2})}{(\tau_1 + \mu_c)(\sigma_1 + \mu_c + \mu_{c1})d_1 \tau_2 \mu_c}$$

$$\text{Hence, } R_0^a = \frac{a_1 \Lambda_1 \delta_1 \lambda_1}{(\lambda_1 + \mu_h)(\mu_h + \mu_{h1})\mu_h \mu_P} + \frac{b_1 \tau_1 R_0^r \mu_c (\tau_2 + \mu_c)(\mu_c + \mu_{c2})}{(\tau_1 + \mu_c)(\sigma_1 + \mu_c + \mu_{c1})d_1 \tau_2 \mu_c}$$

$$\frac{\partial R_0^a}{\partial R_0^r} = \frac{b_1 \tau_1 \mu_c (\tau_2 + \mu_c)(\mu_c + \mu_{c2})}{(\tau_1 + \mu_c)(\sigma_1 + \mu_c + \mu_{c1})d_1 \tau_2 \mu_c}, \text{ thus } \frac{\partial R_0^a}{\partial R_0^r} > 0.$$

A positive partial derivative of R_0^a with respect to R_0^r indicates that an increase in anthrax diseases will positively affect the spread of the rabies outbreak.

Variables	Values	Source	Variables	Values	Source
S_h	500	Assumed	I_{ha}	50	Assumed
S_c	100	Assumed	I_{ca}	10	Assumed
E_{ha}	100	Assumed	I_{hr}	50	Assumed
E_{ca}	20	Assumed	I_{cr}	10	Assumed
E_{hr}	100	Assumed	I_{har}	10	Assumed
E_{cr}	20	Assumed	I_{car}	5	Assumed
E_{har}	20	Assumed	R_{ca}	5	Assumed
E_{car}	10	Assumed	P	10	Assumed
Λ_1	125	Assumed	τ_1	0.01	Estimated
Λ_2	25	Assumed	τ_2	0.0078	Ndendya et al [35], Zainab et al [48]
a_1	0.005	Raza and Abdella [42]	σ_1	0.01	Kashyap et al [27]

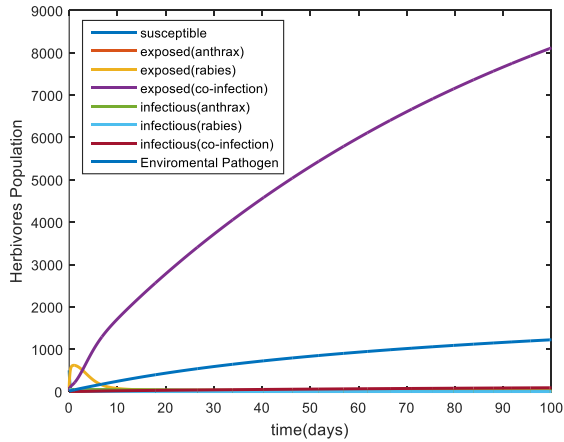
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Parameters	Values	Source	Parameters	Values	Source
b_1	0.005	Mackey [29]	ω_1	0.02	Kashyap et al [27]
c_1	0.7	Estimated	μ_h	0.00012	Mackey [29]
d_1	0.878	Ndendya et al[35]	μ_c	0.00068	Mackey [29]
e_1	0.7	Estimated	μ_{h1}	0.015	Kashyap et al[27]
f_1	0.878	Ndendya et al[35]	μ_{c1}	0.02	Raza and Abdella [42]
g_1	0.005	Raza and Abdella [42]	μ_{h2}	0.8	Ndendya et al [35], Zainab et al [48]
h_1	0.005	Mackey [29]	μ_{c2}	0.2	Mackey [29]
λ_1	0.001	Estimated	μ_{h3}	0.9	Estimated
λ_2	0.0078	Ndendya et al [35], Zainab et al [48]	μ_{c3}	0.5	Estimated
δ_1	0.45	Akande et al [4]	μ_p	0.035	Akande et al [4], Raza and Abdella [42]

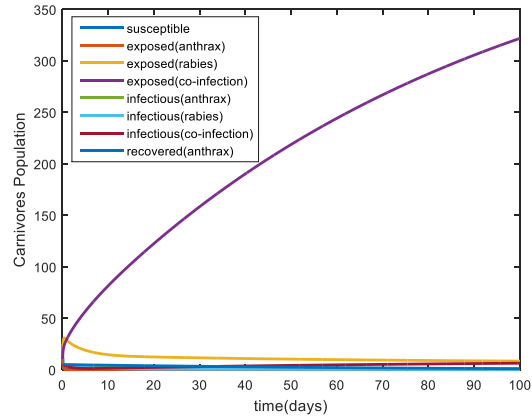
Table 2: Values for the variables and parameters used in the co-infection model

4. NUMERICAL SIMULATION

The numerical simulation of the proposed co-infection model is done using the ode45 package of MATLAB software. In doing so, the value of the variables and parameters in table 2 is used.



(a) Herbivores



(b) carnivores

Figure 2: the numerical solutions of the co-infected model with parameter values from table 2.

Figure 2 is the plots of the numerical solution of co-infection model with parameter values from table 2. It shows the population of each component against time for the herbivores and carnivores

populations.

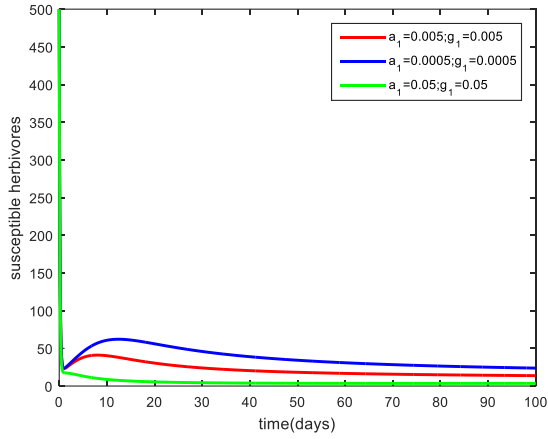


Figure 3(a)

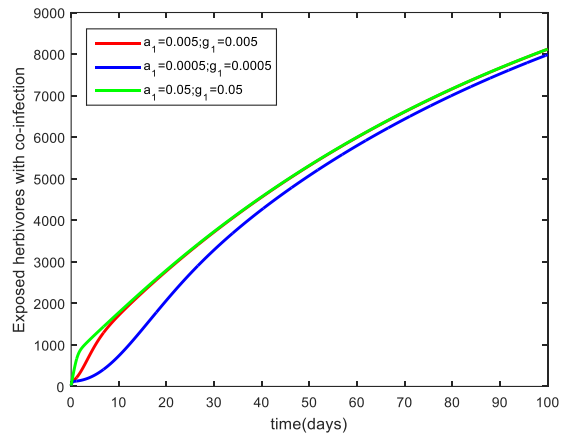


Figure 3(b)

Figure 3 shows the impact of varying the contact rate herbivores has with the anthrax pathogen. We see that increasing the contact herbivores has with the anthrax pathogen will increase the population of herbivores exposed to anthrax only disease and the co-infection of anthrax and rabies disease. It decreases the susceptible population of herbivores and the herbivores exposed to rabies disease

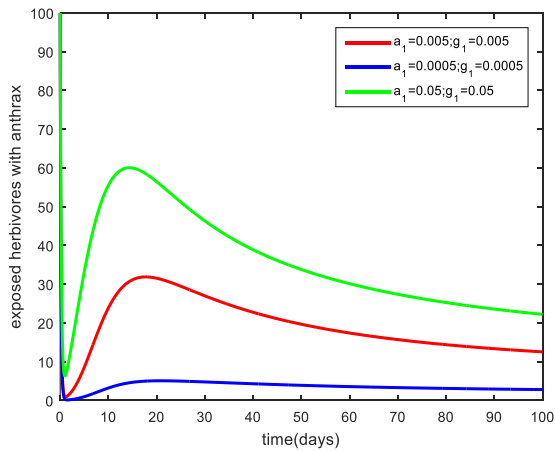


Figure 3(c)

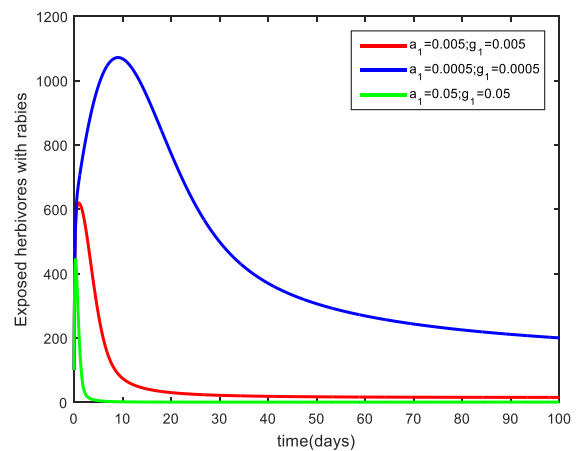


Figure 3(d)

Figure 3: graphical representation of the impact of contact with the environmental pathogen on the population of the herbivores and carnivores for the co-infection model.

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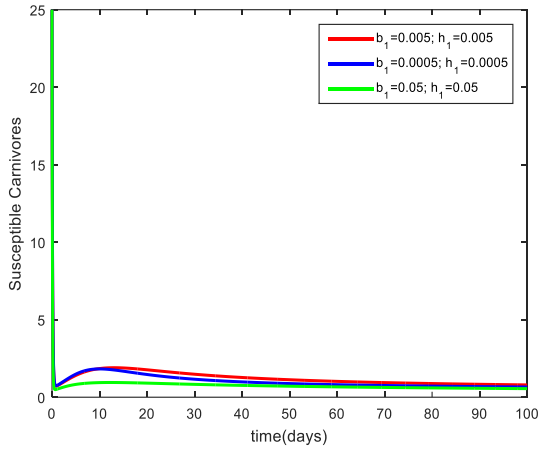


Figure 4(a)

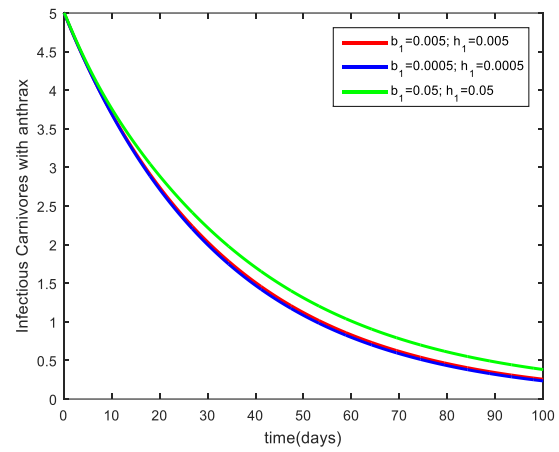


Figure 4(b)

Figure 4 shows the impact of varying the contact rate carnivores has with animal's infected with anthrax pathogen. We see that increasing the contact carnivores has with infectious animals with the anthrax pathogen will increase the population of carnivores infected with anthrax only disease and the co-infection of anthrax and rabies disease. It decreases the susceptible population of carnivores and the carnivores exposed to the rabies disease.

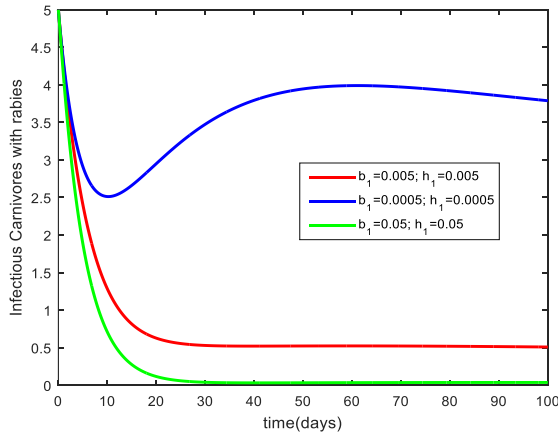


Figure 4(c)

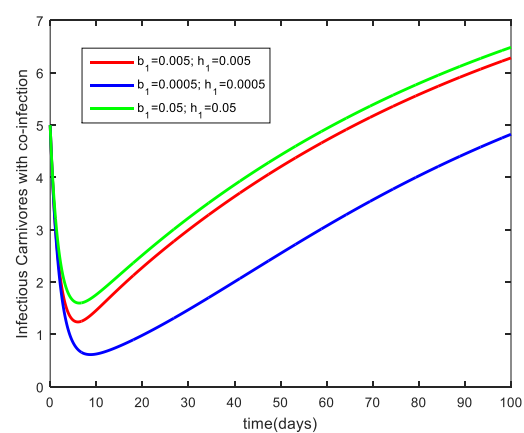


Figure 4(d)

Figure 4: graphical representation of the impact of contact with the anthrax infectious animal on the population of the herbivores and carnivores for the co-infection model.

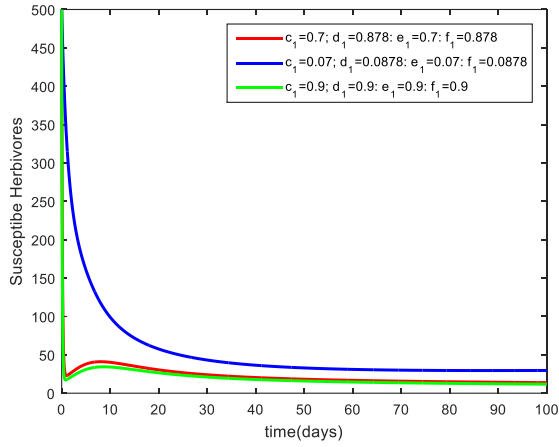


Figure 5(a)

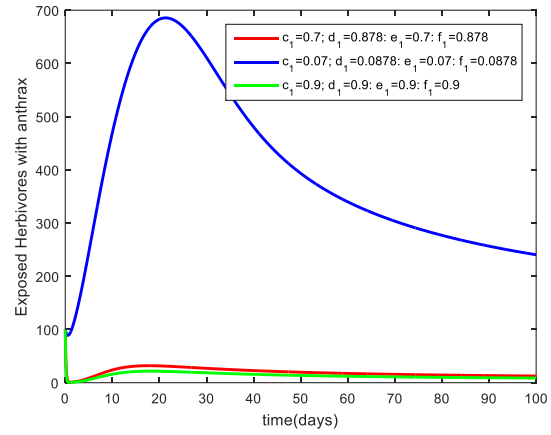


Figure 5(b)

Figure 5 shows the impact of the contact with infectious rabies animals when the contact rate is varied. We see that increasing the contact the animals has with rabied infectious animals will increase the population of the exposed herbivores and carnivores with rabies (figure 5(c) and 5(g).

Also increasing the contact rate of the animals with the rabied infectious animals will decrease the population of the susceptible herbivores (fig. 5(a)), exposed herbivores with anthrax disease (fig. 5(b)), Susceptible carnivores (fig. 5(e)) and exposed carnivores with anthrax disease (fig. 5(f)). It increases the population of herbivores and carnivores exposed to the both disease (anthrax and rabies) slightly, which later tends to the same population despite the value of the contact rate.

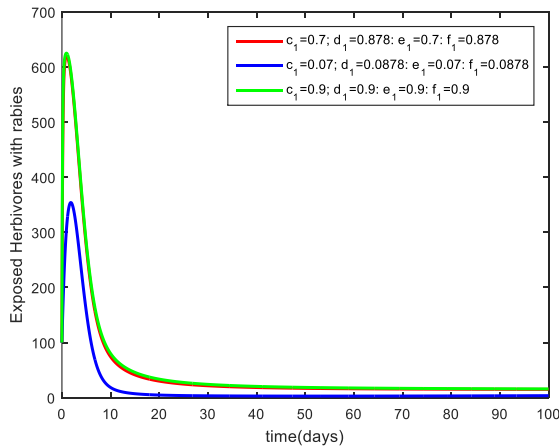


Figure 5(c)

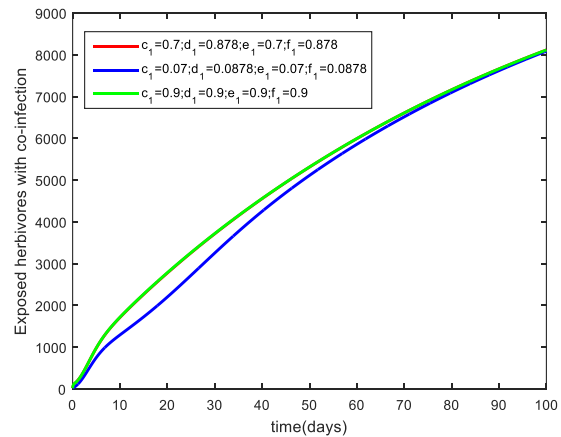


Figure 5(d)

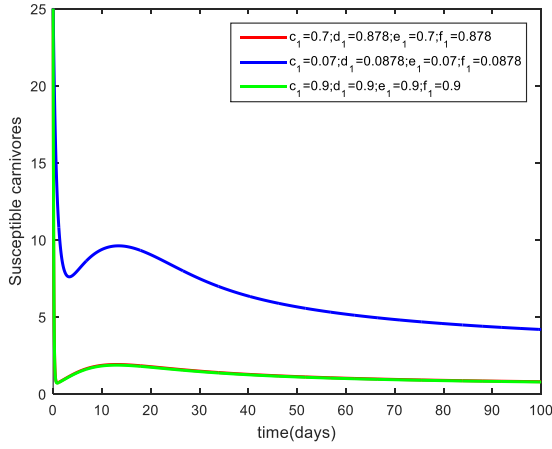


Figure 5(e)

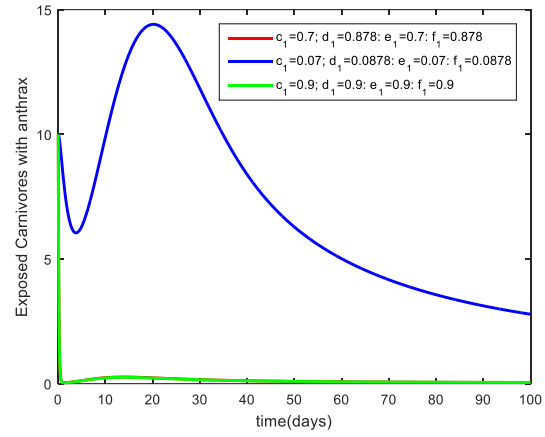


Figure 5(f)

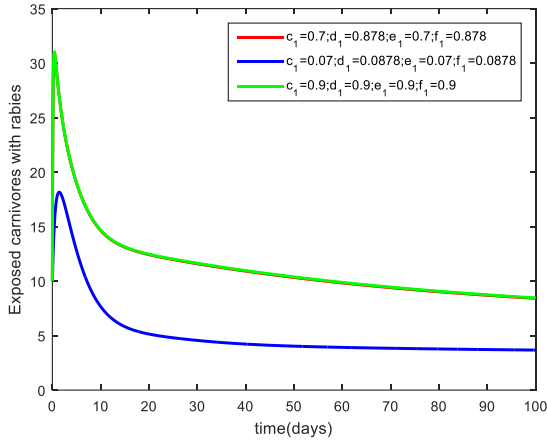


Figure 5(g)

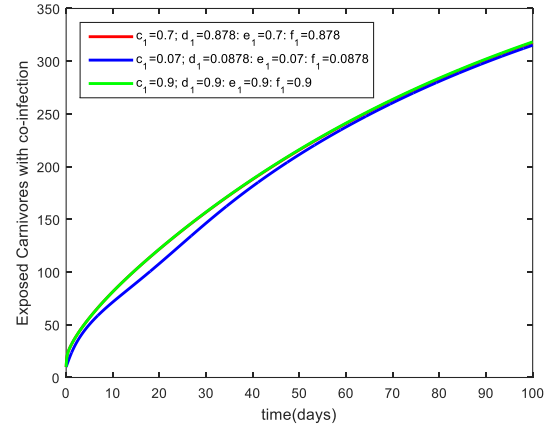


Figure 5(h)

Figure 5: graphical representation of the impact of contact with the rabies infectious animal on the population of the herbivores and carnivores for the co-infection model.

5. CONCLUSION

This study developed a model to study the transmission dynamics of anthrax and rabies co-infection on wild herbivores and carnivores. It is established that the model is well posed and the solutions are positive within a biologically meaningful range using the positivity of solution and the invariant region of existence theorems. The equilibrium points for the model of the separate diseases as well as the equilibrium point of the co-infection model are established and analyzed. The local stability of the disease free equilibrium of the models is discovered by establishing that there is no sign change using the Descartes rule of signs while the global stability of the models

was established using the Castillo-Chavez method. It is discovered that the anthrax only model and the rabies only model are both locally and globally stable while the co-infection model is only locally stable and globally unstable. We prove the existence of the endemic equilibrium of the models. The basic reproduction numbers for the models are calculated using the next-generation matrix approach. We also checked the impact of varying the contact rates on the model using Numerical simulations.

CONFLICT OF INTERESTS

The authors declare that there is no conflict of interests.

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