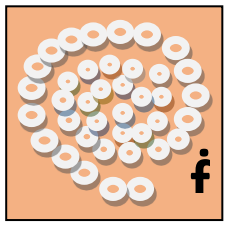

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Mathematical model of the transmission dynamics of Anthrax disease

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Abstract

Anthrax is a deadly disease,that occurs in ruminant animals such as sheep,cattle and others.The human can also contract anthrax when one comes in contact with infected animal or their product like hide,wool and so on.In this paper a system of non-linear differential equation is formulated to describe the transmission dynamics of the disease in human.The model was well formulated and epidemiologically meaningful by showing the positivity of the variables and obtaining the invariant region of the various compartment of the model.The equilibrium analysis[EEP] were also carried out and stability analysis based on the equilibrium points were also carried out. Finally, Numerical simulation was done using MATLAB which helped us to show in one of the graphs that with early treatment,the disease may eventually may be controlled

Keywords and phrases: Anthrax; mathematical model;endemic equilibrium point(EEP); numerical simulation

1 Introduction

Bacillus anthracis (anthrax) refers to the black,coal-like leision that is frequently seen on infected people's skin and also derived from Greek word anthracite which means coal [1].Anthrax was first mentioned in 1491 B.C in Mesopotamian writting and Book of Geenesis.The $5th$ and$6th$ Egyptian plagues shows the symptoms of anthrax.The disease is a spore-forming bacterium,a rare but serious illness which primarily affects livestock and game. Human become infected through either first hand or second hand interaction with sick animals [1].

The signs and symptoms are headache,Bulging of sore lymph glands surrounding it,swollen neck,shortness of breath,meningitis and spinal cord inflammation,failure of multiple organ and shock e.t.c

The mode of transmission are as follows; Cutaneous anthrax is gotten through skin wound or cut as the infection enters the body. It's the least dangerous because, with proper care, cutaneous anthrax seldom results in death. Secondly,Gastrointestinal anthrax is the type of anthrax infection,caused by consuming raw meat of an infected animal.Thirdly is the Pulmonary anthrax which happens when anthrax spores are inhaled.It is the most dangerous way to get the disease, and even with treatment, it is frequently fatal.Finally Injection anthrax which is the most recent method of anthrax infection that has been discovered. It is acquired through drug injection and has only been documented thus far in Europe.

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There are some risk factors for those who works in a laboratory, for example the veterinary especially for those that deal with livestock, Handles or dress game animals ,Inject illegal drugs like heroin and Handle animal by-product, examples wools, skins from areas with a high incidence of Anthrax and also in military especially those that are in the the area with high risk of anthrax exposure.

2 Prevention and control

The control measure as shown in Figure [1] above which is by breaking the infection cycle. The following procedure must be strictly followed:

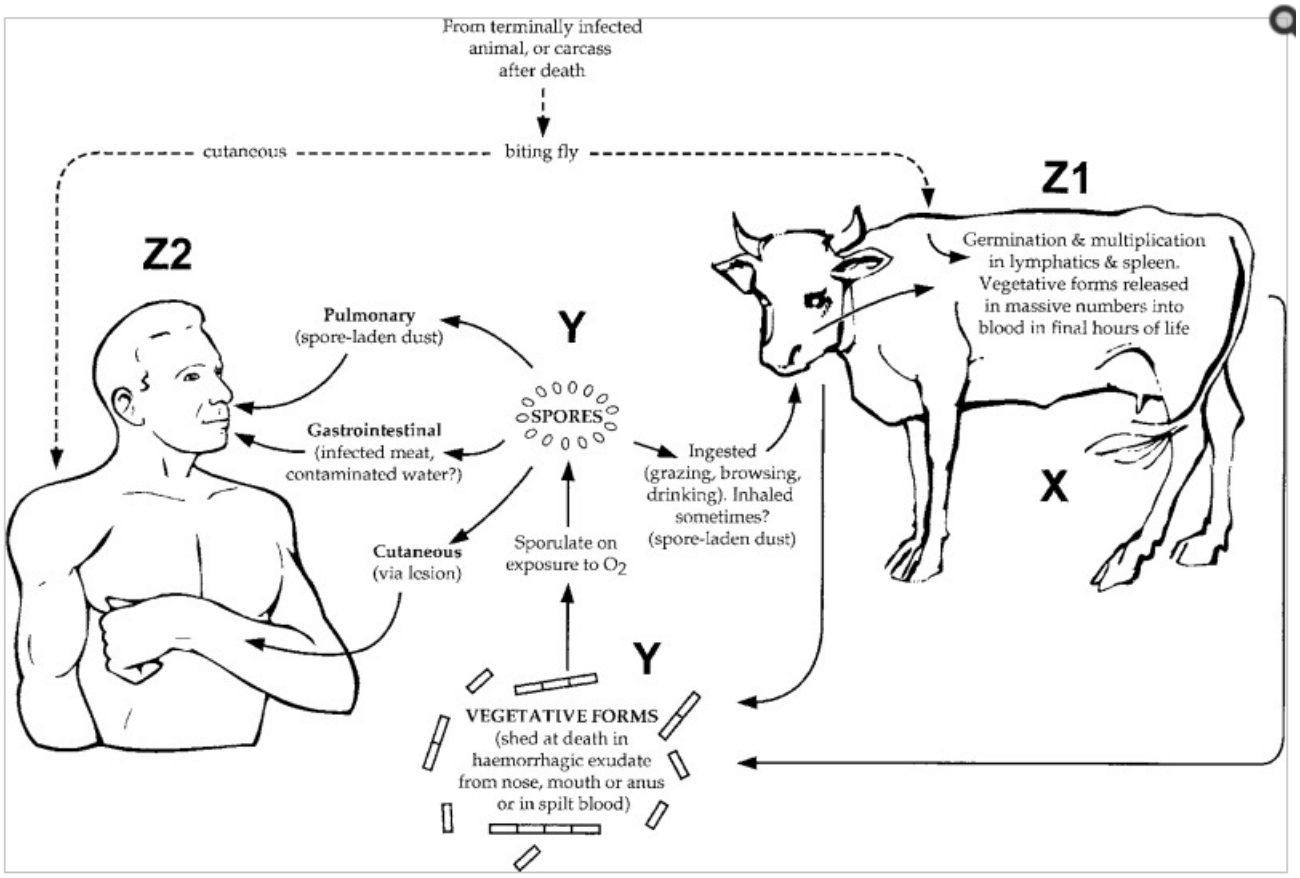

Figure 1: Breaking the cycle of infection

2.1 Disposal of anthrax (animal) carcasses

- Principles involved:Sporulation of anthrax does not occur inside an enclosed carcass. Most countries' policies do not involve postmortem examination of animals when anthrax is suspected. Without that, all vegetative B. anthracis cells in the carcass are killed in a few days by a putrefactive process(decay). The actual length of time after which no feasible B. anthracis remains in a carcass is unpredictable but depends immensely on the condition of the climate that is temperature.

- incineration: Incineration must be carried out with appropriate care to ensure complete burning from beneath. Usually, this involves raising the carcass off the ground before the process is started.

- Rendering:The sterilization of staple(raw materials) of animal rootage by heating method or treatment such that parts of carcasses are used later for commercial designs.

This procedure involves accurate performance at each of three stages: the collection stage, transport stage, and treatment stage of the carcass [5, 6].

2.2 Disinfection and decontamination by fumigation (formaldehyde or ethylene oxide)

Disinfection, decontamination, and proper disposal of infected/contaminated material are critical in preventing long-distance and international anthrax transmission, in addition to assisting in breaking the cycle of anthrax infection locally.

2.3 Infection control for human

1 If reasonable precautions are taken, Human-to-human communication poses no significant risk. For example, during the first $24-48$ hours post operative , cutaneous anthrax lesions should be dressed; and disposing of test samples or sterilizing equipment. Postmortem examinations should be discouraged in fatal cases; cremation is preferable for burial where local custom allows.

2 Antibiotics are advised to be used by anyone who has been exposed to spores. The Food and Drug Administration has approved ciprofloxacin (Cipro), doxycycline (Monodox, Vibramycin, and others), and levofloxacin (Levaquin) for the avoidance of diease in human.[2]

3 Visitors should wear protective clothing and footwear that can be sanitized or packaged for burning before entering infected properties.[2]

The study of Anthrax was based on the hypothesis that the disease is primarily spread by environmental contamination where the generation multiplication rate is defined as the number of new cases arising from a single isolated case.The effects of anthrax transmission via carcass ingestion,carcass-induced environmental contamination and migration rates leads to disease infection on animal population which may be persistence or lead to extinction.The basic reproduction number R_0 for the anthrax epizootic model was calculated with animal migration.It was observed that the removal of remains in the game reserves brings about a decrease in the level of carcass ingestion.As a result of that, the increase in level of carcass induced environmental contamination rate in an enzootic anthrax region can lead to the extinction of a persistent animal population[Friedman etall 2012].The researcher[Zerihun et al 2016] formulated a model of four compartment and studied the effectiveness of clinical signs on the infected animals.In 2018,Osman et al carried out a review study on the mathematcal modelling of transmission dynamics of anthrax in human and animal population indicating the quantitative and qualitative analyses of the model.Elijah et al. (2020) conducted a mathematical analysis of the Effects of Controls on the Anthrax Transmission Dynamics in ruminant and non-ruminant animals. The study proposed a nonlinear differential equation model to investigate the impact of vaccination on the dynamics of anthrax disease transmission in livestock and human populations. The model is shown to have only two equilibria, the disease-free and the endemic equilibrium points, both of which are locally stable if the Basic reproduction number (R0) is less than unity and greater than unity, respectively. According to local sensitivity analysis, the infection rate, pathogen-shedding rate, and vaccination rate of livestock are the parameters with the greatest positive impact on disease spread. The rate of disinfection, followed by the rate of vaccination, has the greatest negative impact on disease transmission. The numerical simulation demonstrated that implementing all control measures (vaccination, education, disinfection, and treatment) is the most effective strategy for preventing disease spread.Firstly, we considered the presence of Bacillius Anthracis in the environment through Air(aerosol).Secondly, an exposed treated class was included in the human compartment to checkmate the bacteria. Lastly, the model accommodated the class of infectious vectors without treatment in the animal compartment.

3 Methodology/model formulation

In this Section, we developed a Mathematical model describing the transmission dynamics of Anthrax. The definitions and representations of the model parameters and variables declaration are highlighted,

flow diagram describing the dynamics of the disease was shown, basic assumptions are all clearly stated.

3.1 Assumptions of the model

In this study, the following assumptions were made;

1. There is adquate contact of a susceptible individual with an infected individual,carcasses and spores in air,so that transmission may occur,and the susceptible individual may join the exposed class at the rate $\beta = (\alpha_1 C + \alpha_2()I_H + I_V) + \alpha_3(e^{\theta} - 1)$
2. We assumed in vector compartment that the infectious population,I_V is classified into 2 stages as the one undergoing treatment I_{VT} at the rate sigma (σ) and the second stage is the infectious vector without treatment,I_{VN} and recover at the rate rho (ρ) due to immunity.
3. In the infectious human class,there is inhalation i.e spores in air at the rate of Ae^{θ}. Due to this there may be no disease free equilibrium (DFE)and therefore, the Basic reproduction number R_0 is not calculated.
4. We also assumed that recovered human and vector are permanently immuned to the disease.

Model flow diagram is seen in Figure ??:

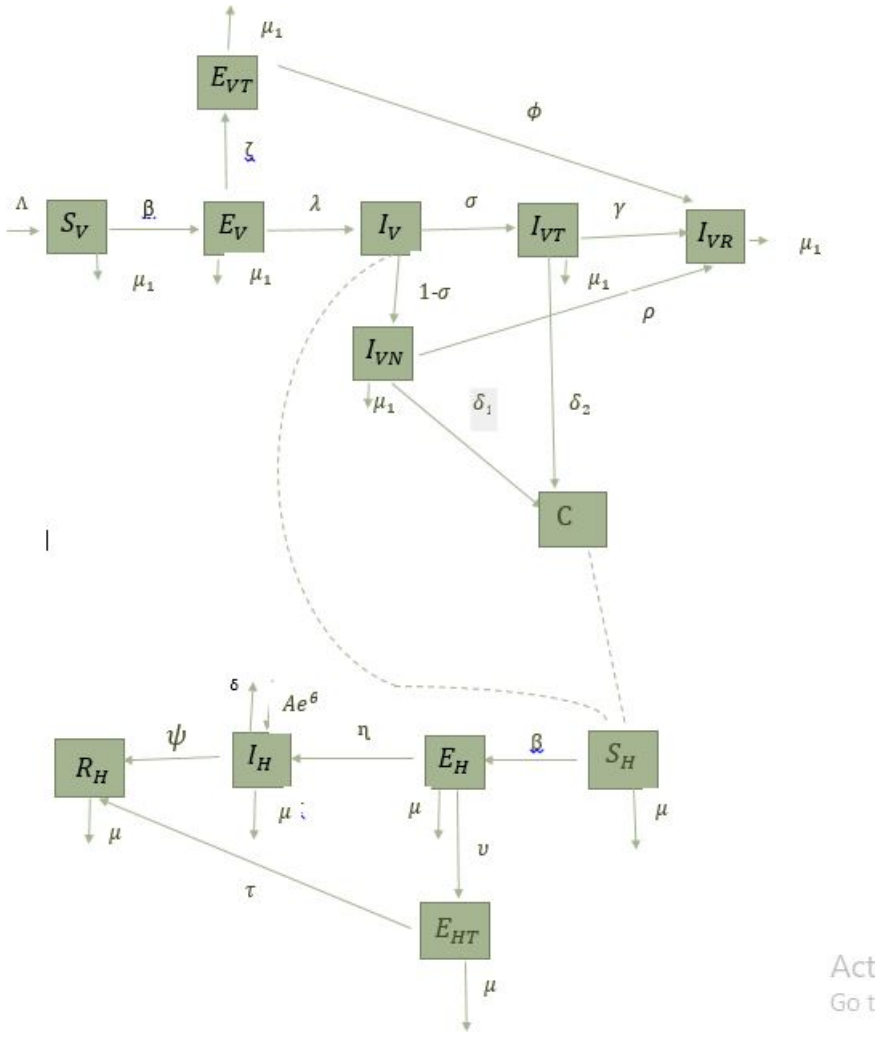

Figure 2: Flow diagram for transmission dynamics of Anthrax model.

Table 1: Variable declaration

Variable	Description
S_V	Total number of susceptible vectors at time t
E_v	The number of Exposed vectors at time t
E_{vT}	The number of Exposed vectors undergoing treatment at time t
I_v	The number of infectious vectors
I_{vT}	The number of infectious vectors undergoing treatment at time,t
I_{vN}	The number of infectious vectors without treatment at time,t
I_{vR}	The number of recovered vectors at time,t
C	The number of infected / infectious Carcasses at time,t
S_H	The number of Susceptible humans at time,t
E_H	The number of Exposed humans at time, t
E_{HT}	The number of Exposed humans undergoing treatment at time,t
I_v	The number of infectious humans at time ,t
R_H	The number of recovered humans at time,t

Table 2: Parameter declaration

parameter	Description
Λ	recruitment level of the vectors
β	force of infection , $\beta = \alpha_1 C + \alpha_2(I_H + I_v) + \alpha_3(e^{\theta} - 1)$
β_1	force of infection , $\beta_1 = \alpha_{11} C + \alpha_{21}(I_v) + \alpha_{31}(e^{\theta} - 1)$
ζ	progression from exposed to exposed undergoing treatment
λ	progression rate from exposed to infectious
σ	rate at which the infectious go for treatment
1-σ	rate of recovery without treatment
ϕ	progression rate from exposed treated to recovered
γ	rate of recovery from infectious vector that are treated
ρ	rate of recovery from infectious vectors without treatment
δ_1, δ_2	death rate due to disease
η	progression rate from exposed humans to infectious humans
υ	rate at which exposed human go for treatment
θ	density of the particles in the Air
τ	recovery rate of treated exposed humans
Ψ	recovery rate of treated infectious humans
ϵ	recovery rate of infectious humans without treatment
μ	natural death unrelated to the disease

3.1.1 Subsubsection

4 Analysis of the model

Endemic Equilibrium Point

The endemic equilibrium point (EEP) E^* i.e where $R_0 > 1$ is a steady state solution,where the disease persists in the population.The existence and uniqueness of endemic equilibrium point E^* should satisfy the conditions: $E^* = (S_V^*, E_V^*, E_{VT}^*, I_{VT}^*, I_{VN}^*, I_{VR}^*, S_H^*, E_H^* E_{HT}^*, I_H^*, R_H^*)$ and
$E^* = (S_V^*, E_V^*, E_{VT}^*, I_{VT}^*, I_{VN}^*, I_{VR}^*, S_H^*, E_H^* E_{HT}^*, I_H^*, R_H^*, C^*) \neq 0$.It is now needed to find the endemic equilibrium point of the system (4.1) -(4.12)and setting right hand to zero gives

$$\begin{aligned}
\Lambda_v - \beta_1 S_v - \mu_1 S_v &= 0 && (1)\\
\beta_1 S_v - (\zeta + \lambda + \mu_1) E_v &= 0 && (2)\\
\zeta E_v - \Phi E_{VT} - \mu_1 E_{vT} &= 0 && (3)\\
\lambda\sigma E_v - (\gamma + \delta_2) I_{VT} &= 0 && (4)\\
\lambda(1-\sigma) E_v - (\rho + \delta_1) I_N &= 0 && (5)\\
\Phi E_{vT} + \gamma I_{vT} + \rho I_{vN} - \mu_1 I_{VR} &= 0 && (6)\\
\Lambda_H - \beta S_H - \mu S_H &= 0 && (7)\\
\beta S_H - (\eta + \upsilon + \mu) E_H &= 0 && (8)\\
\upsilon E_H - (\tau + \mu) E_{HT} &= 0 && (9)\\
\eta E_H - (\Psi + \mu) I_H + (e^{\theta} - 1) S_H &= 0 && (10)\\
\Psi I_H + \tau E_{HT} - \mu R_H &= 0 && (11)\\
\delta_1 I_{VN} + \delta_2 I_{VT} &= 0 && (12)
\end{aligned}$$

On solving (1) -(12), the variables were obtained as
$C^* = \frac{\Lambda_V - \alpha_2 S_V (I_H + I_V - \alpha_3 (e^{\theta}-1) - \mu S_V}{\alpha_1 S_V}, E_H^* = \frac{\beta \Lambda_H}{(\beta+\mu)(\eta+\upsilon+\mu)}, E_{HT}^* = \frac{\upsilon \beta \Lambda_H}{(\beta+\mu)(\tau+\mu)(\eta+\upsilon+\mu)}, E_V = \frac{\beta S_V}{(\beta+\mu)(\eta+\lambda+\mu)}, E_{VT}^* = \frac{\eta \beta \Lambda_V}{(\phi+\mu)(\beta+\mu)(\eta+\lambda+\mu)}, I_H^* = \frac{\eta \beta \Lambda_H}{(\beta+\mu)(\tau+\mu)(\eta+\upsilon+\mu) - a(e^{\theta}-1)}, I_{VN} = \frac{\lambda(1-\sigma)\beta \Lambda_V}{(\rho+\delta_1+\mu)(\beta+\mu)(\zeta+\lambda+\mu)}, I_{VT} = \frac{\lambda\sigma)\beta \Lambda_V}{(\gamma+\delta_2+\mu)(\beta+\mu)(\zeta+\lambda+\mu)}, I_{VR}^* = \frac{\phi Q_1 + \gamma Q_2 + \rho Q_3}{\mu}, R_H^* = \frac{\psi Q_5 + \tau Q_4}{\mu}, S_H^* = \frac{\Lambda_H}{\beta+\mu}, S_V^* = \frac{\Lambda_V}{\beta_1+\mu_1}$

4.0.1 Invariant region and positivity of solution

For the Anthrax transmission dynamics model(??)-(??) to be epidemiological meaningful,it is necessary to prove that all solutions with non-negative initial data will remain non-negative or positive for all time and therefore is mathematically well posed.

4.0.2 Positivity of solution

Here now it is to be shown that all state variables remain non-negative since they represent human population and the presence of the disease in the population.
The invariant set
For $\Omega = \Omega_H + \Omega_V$
Let$\Omega_H = (S_H, E_H, E_{HT}, I_H, R_H) and$
$\Omega_V = (S_V, E_V, E_{VT}, I_{VN}, I_{VT}, I_{VR})$
Then let $\Omega = (S_H, E_H, E_{HT}, I_H, R_H, S_V, E_V, E_{VT}, I_{VN}, I_{VT}, I_{VR}) \in \Re_+^{11}$
So at initial time $S_H(0) > 0, E_H(0) > 0, E_{HT}(0) > 0, I_H(0) > 0, R_H(0) > 0, S_V(0) > 0, E_V(0) > 0, E_{VT}(0) > 0, I_{VN}(0) > 0, I_{VT}(0) > 0, I_{VR}(0) > 0$.This is because at the initial time of study of the disease transmission,there is the presence of the disease in the population and so each compartment of the disease population is non negative.

the equation (??)

$$\begin{aligned}
\frac{dS_V}{dt} &= \Lambda_V - \beta_1 S_V - \mu_1 S_V \\
\frac{dS_V}{dt} &\geq -\mu_1 S_V \\
\int \frac{dS_V}{S_V} &\geq -\mu_1 \int dt \\
lnS_V &\geq -\mu_1 t + C \\
e^{lnS_V} &\geq e^{-\mu_1 t} * e^c \\
S_V(t) &\geq Ae^{-\mu_1 t} \quad where \qquad A = e^C \qquad (13) \\
at \quad t = 0 & \\
S_V(0) &= S_{V_0} \geq A \qquad (14)
\end{aligned}$$

Substitute equation (14) into (13) we have

$$S_V(t) \geq S_{V_0} e^{-\mu t}$$

as $t \to \infty$,

$$S_V(t) \to 0$$

from equation (??)

$$\begin{aligned}
\frac{dE_V}{dt} &= \beta_1 S_V - (\zeta + \lambda + \mu_1 + \lambda + \mu) E_V \\
\frac{dE_V}{dt} &\geq -(\zeta + \lambda + \mu_1) E_V \\
\int \frac{dE_V}{E_V} &\geq -\int (\zeta + \lambda + \mu_1) dt \\
lnE_V &\geq -(\zeta + \lambda + \mu_1)t + c \\
e^{lnE_V} &\geq e^(\zeta + \lambda + \mu_1)t * e^c \\
E_V(t) &\geq Be^{(\zeta+\lambda+\mu_1)t} \qquad where \qquad B = e^C \qquad (15)
\end{aligned}$$

at t = 0

$$E_V(0) = E_{V_0} \geq B \qquad (16)$$

Put equation (16) into (15)

$$E_V(t) = E_{V_0} e^{-[\zeta+\lambda+\mu_1]t} \qquad (17)$$

as $t \to \infty$, $E_V(t) \to 0$
Similarly, the same argument holds for the remain variables.

4.1 Invariant Region

The total population size for human and animal are

$$N = N_H + N_V \qquad (18)$$

$$\text{let} N_H = S_H + E_H + E_{HT} + I_H + R_H \qquad (19)$$

$$\Rightarrow \frac{dN_H}{dt} = \frac{dS_H}{dt} + \frac{dE_H}{dt} + \frac{dE_{Ht}}{dt} + \frac{dI_H}{dt} + \frac{dR_H}{dt} \qquad (20)$$

$$\frac{dN_H}{dt} = \Lambda_H - \beta S_H - \mu S_H + \beta S_H - \eta E_H - \upsilon E_H - \mu E_H + \upsilon E_E H \tag{21}$$

$$-\tau E_{HT} - \mu E_{HT} + \eta E_H - \Psi I_H - \mu I_H + a(e^{\theta} - 1) + \Psi I_H + \tau E_{HT} - \mu R_H \tag{22}$$

$$\frac{dN_H}{dt} = \Lambda_H - (S_H + E_H + E_{HT} + I_H + R_H)\mu + a(e^{\theta - 1}) \tag{23}$$

$$\frac{dN_H}{dt} = \Lambda_H - N_H\mu + a(e^{\theta} - 1) \tag{24}$$

$$\rightarrow \frac{dN_H}{dt} + \mu N_H = \Lambda_H + a(e^{\theta} - 1) \tag{25}$$

$\rightarrow (I.F) = e^{\int \mu dt} = e^{\mu t}$
we integrate

$$N_H(t)e^{\mu}t = \int (\Lambda_H + a(e^{\theta} - 1))e^{\mu t}dt + C \tag{26}$$

$$= (\frac{\Lambda_H + a(e^{\theta} - 1)}{\mu})e^{\mu t} + C \tag{27}$$

$$\rightarrow N_H(t) = \frac{\Lambda_H + a(e^{\theta} - 1)}{\mu} + Ce^{-\mu t} \tag{28}$$

$$\tag{29}$$

at $t = 0, N_H = N_H(0) = N_{H0}$

$$\rightarrow N_{H0}(t) = \frac{\Lambda_H + a(e^{\theta} - 1)}{\mu} + C \tag{30}$$

$$\rightarrow C = N_{H_0} - \frac{\Lambda_H + a(e^{\theta} - 1)}{\mu} \tag{31}$$

$\Rightarrow$

$$N_H(t) = \Lambda_H + a(e^{\theta} - 1) + \left[N_{H_0} - \frac{\Lambda_H + a(e^{\theta} - 1)}{\mu}\right] e^{-\mu t} \tag{32}$$

$$= \frac{\Lambda_H + a(e^{\theta} - 1)}{\mu} \left[1 - e^{-\mu t}\right] + N_{H_0}e^{-\mu t} \tag{33}$$

As $t \longrightarrow \infty, e^{-\mu t} \longrightarrow 0$ and so we have

$$N_H(t) = \frac{\Lambda_H}{\mu} \tag{34}$$

when $e^{\theta} = e^0 = 1$ so that $a(e^0 - 1) = 0$

The total population size of animal

$$N_V = S_V + E_V + E_{VT} + I_{VT} + I_{VN} + I_{VR} \tag{35}$$

$$\Rightarrow \frac{dN_V}{dt} = \frac{S_V}{dt} + \frac{E_V}{dt} + \frac{E_{VT}}{dt} + \frac{I_{VT}}{dt} + \frac{I_{VN}}{dt} + \frac{I_{VR}}{dt} + \frac{C}{dt} \tag{36}$$

$$\frac{N_V}{dt} = \Lambda_V - \beta S_V - \mu S_V + \beta S_V - \zeta E_V - \lambda E_V - \mu E_V \zeta E_V - \phi E_{VT} - \mu_1 E_{VT} + \lambda\sigma E_V - \gamma I_{VT} - \delta_2 I_{VT} \tag{37}$$

$$\lambda E_V - \lambda\sigma E_V - \rho I_{VN} - \delta_1 I_{VN} + \phi E_{VT} + \gamma I_{VT} + \rho I_{VN} - \mu_1 I_{VR} + \delta_1 I_{VN} + \delta_2 I_{VT} + \mu_1 I_{VN} \tag{38}$$

$$\frac{dN_V}{dt} = \Lambda_V - (S_V + E_V + E_{VT} + I_{VN} + I_{VT} + I_{VR})mu_1 \tag{39}$$

In the absence of the disease (Anthrax) we are left with

$$\frac{N_V}{dt} = \Lambda_V - N_V\mu_1 \tag{40}$$

$$\text{integrating factor (I.F)} = e^{\int \mu_1 dt} = e^{\mu_1 t} \tag{41}$$

$$\frac{dN_V}{dt} + \mu_1 N_V e^{\mu_1 t} = \Lambda_V e^{\mu_1 t} \tag{42}$$

$$dN_V(t)e^{\mu_1}(t) = \Lambda_V e^{\mu_1 t} \tag{43}$$

we integrate

$$N_V(t)e^{\mu} t = \frac{1}{\mu}\Lambda_V e^{\mu} t + C \tag{44}$$

$$N_V(t) = \frac{\Lambda_V}{\mu} + \frac{C}{e^{\mu} t} \tag{45}$$

$$N_V(t) = \frac{\Lambda_V}{\mu} + Ce^{\mu_1} t \tag{46}$$

At the initial condition $N_V(0) = N_{H_0}$ for $t = 0$

$$N_{V_0} = \frac{\Lambda}{\mu_1} + C \tag{47}$$

$$C = N_{V_0} - \frac{\Lambda_V}{\mu_1} \tag{48}$$

substitute equation (48) into equation (46)

$$\Rightarrow N_V(t) = \frac{\Lambda_V}{\mu_1} + e^{-\mu_1 t}(N_{V_0} - \frac{\Lambda_V}{\mu_1}) \tag{49}$$

$$\Rightarrow N_V(t) \leq \frac{\Lambda_V}{\mu} + N_{V_0}e^{-\mu t} - \frac{\Lambda_V}{\mu_1}e^{-\mu_1 t} \tag{50}$$

At t $\longrightarrow \infty, N_V(t) \longrightarrow \frac{\Lambda}{\mu_1}$

Thus,the feasible set for the model system (??)-(??) is given by

$$\left\{ \begin{bmatrix} S_H \\ E_H \\ E_{HT} \\ I_H \\ R_H \\ S_V \\ E_V \\ E_{VT} \\ I_V \\ I_{VT} \\ I_{VN} \\ I_{VR} \end{bmatrix} \in R_+^{12} \middle| \begin{array}{l} S_H \geq 0 \\ E_H \geq 0 \\ E_{HT} \geq 0 \\ I_H \geq 0 \\ R_H \geq 0 \\ S_V \geq 0 \\ E_V \geq 0 \\ E_{VT} \geq 0 \\ I_V \geq 0 \\ I_{VT} \geq 0 \\ I_{VN} \geq 0 \\ I_{VR} \geq 0 \\ N_H \leq 0 \\ N_V \leq 0 \end{array} \right\}$$

It implies that model equations are positively invariant.Hence the model is mathematically well posed and biologically meaningful (Gumel & Niger 2008).

5 Numerical simulations/sensitivity analysis

In this section, the numerical simulation of the model equations is conducted to find out the dynamics of the disease in the human population and afterwards the animal population.The simulations were conducted using MATLAB.

S/N	Parameter	Values	Source
1	Λ_H	1000	O.Sharomi et al,2007
2	Λ_V	2000	O.Sharomi et al,2007
3	α_1	0.0033	Pantha Buddhi Raj,2016
4	α_1	0.0022	Assumed
5	α_1	0.02	Assumed
6	λ	0.001	Pantha Buddhi Raj,2016
7	σ	0.45	O.Sharomi et al,2007
8	γ	0.0034	O.Sharomi et al,2007
9	δ_1	0.0043	O.Sharomi et al,2007
9	δ_2	0.0036	O.Sharomi et al,2007
10	ρ	0.003	O.Sharomi et al,2007
11	ϵ	0.03	Assumed
12	μ	0.0005	Pantha Buddhi Raj,2016
13	ϕ	0.05	Assumed
14	υ	1	Assumed
15	τ	0.065	Pantha Buddhi Raj,2016
16	Ψ	0.045	Assumed
17	e	0.23	Estimated
18	a	0.2	Estimated

Variable @ initial condition	Value
S_V	1000
E_V	800
E_{VT}	700
I_{VT}	690
I_{VN}	650
I_{VR}	600
S_H	2000
E_H	400
E_{HT}	300
I_H	150
R_H	130

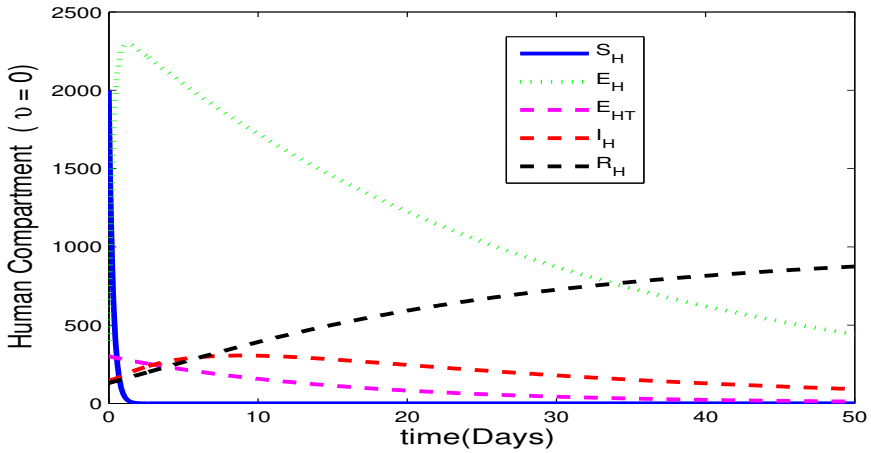

Figure 3: Graph of the dynamic of Human population when $\upsilon = 0$

It is shown in the above graph that after some time,all the people in susceptible class will become infected.Since there is no treatment the infected population E_H continues to develop infectious fully and thus reduces the population of the exposed class.The only few people that recovered is due to strong immune system.

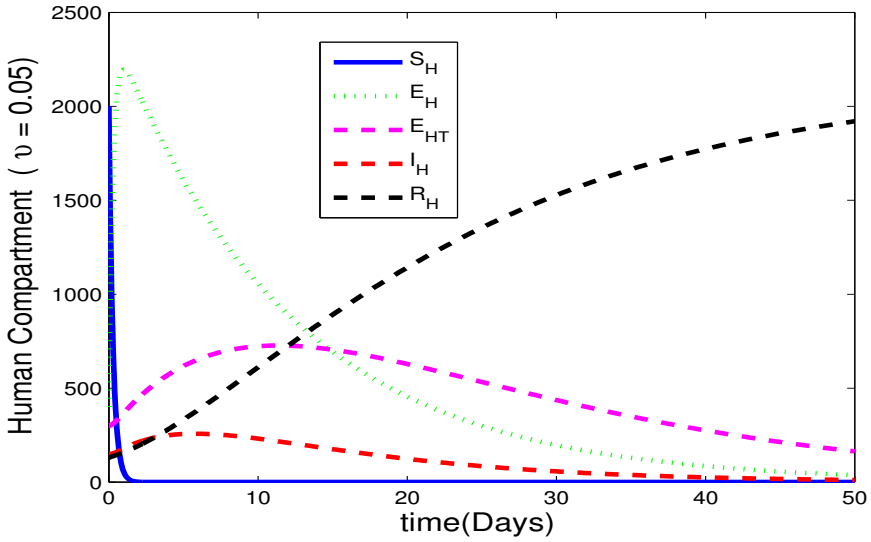

Figure 4: Effect of when $\upsilon = 0.05$ on the human population

The graph above showed the effect of prophylaxis as we can see the sharp drop in the curve of exposed Human and sharp increase in recovered class

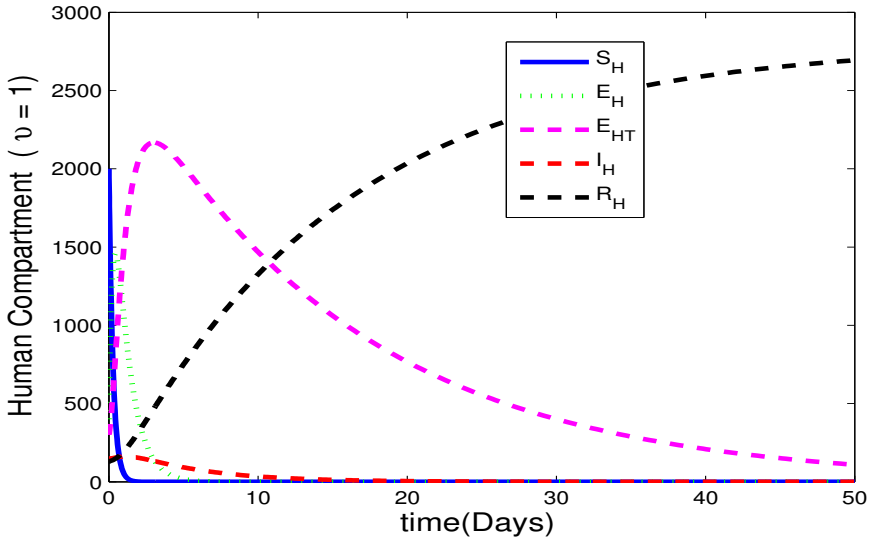

Figure 5: Effect of $\upsilon = 1$ on the human population

In this figure,the exposed and infectious population reduce drastically leading to large increase in recovered population due to treatment and can be seen also all the susceptible population became infected and thus exposed due lack of treatment at the initial time.

6 Discussions and conclusion

We have derived and analyzed a mathematical model for the transmission and control of Anthrax.A flow diagram was drawn showing the transmission,stratagies for control and treatment.The basic reproduction number R_0 and disease free equilibrium point (DFE) is not computered due to the presence of the (inhalation ,Ae^0) Bacillus anthracis spore in the air.Then, we perform the stability analysis of the model and showed that the endemic equilibrium is stable.

The Numerical Stimulation was carried out using MATLAB and we analyzed that $fig4.1$ and $fig4.4$ shows no effect of treatment on both human and vector population. $fig4.2$ and $fig4.5$ shows that Human and vector exposed to the disease are being treated and more human and vector enter the recovering class while $fig4.6$ and $fig4.7$ depicts the rate at which exposed Human and vector population decreases at the increase in treatment .

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