Available online at http://scik.org
J. Math. Comput. Sci. 8 (2018), No. 6, 654-672
https://doi.org/10.28919/jmcs/3798
ISSN: 1927-5307

MODELING THE TRANSMISSION DYNAMICS OF ANTHRAX DISEASE IN CATTLE AND HUMANS

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Abstract. Anthrax is a zoonotic disease caused by bacillus anthraces. In this study a deterministic mathematical model for transmission dynamics of anthrax in humans and animals is presented and quantitatively analyzed. To understand the dynamics of anthrax, the basic reproduction number R_0 which measures average new infections is computed using next generation matrix operator and analyzed by normalized forward sensitivity index. Analysis shows that anthrax transmission rate to animals, animals' natural death rate, anthrax natural death rate and animal's birth rate are the most sensitive parameters to the disease transmission dynamics. When animal recruitment and anthrax transmission rates increase, the basic reproduction number R_0 also increase proportionally. However, when animal natural and anthrax induced death rates increase, basic reproduction number R_0 decreases. Numerical simulations using Runge-Kutta method show that animals drive the dynamics of anthrax. The study suggests control strategies such as vaccination, fumigation and decomposition of carcasses to eradicate the disease.

Keywords: sensitivity analysis; zoonotic disease; mathematical model; humans; cattle.

2010 AMS Subject Classification: 00A71, 00A72, 92B05, 92D30 .

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Received July 12, 2018

1. Introduction

Anthrax is an acute infectious disease caused by bacterium Bacillus anthraces [10]. The disease is transmitted in four different forms that includes, inhalation, ingestion, contact and injection. Herbivorous are more vulnerable to infection when they eat spores in the soil or plants, [15]. Omnivorous as well are vulnerable to the disease and they catch the infection through eating meat which is contaminated with anthrax. There are four types of anthrax which are inhalation, ingestion or gastrointestinal, injection and cutaneous anthrax [12].

Grasses serve as exploited habitats of bacillus anthracis and grazing animals such as sheep, goat and cattle are predominantly victims [13]. The alkaline soil with a pH greater than 6.0, high nitrogen level caused by decaying vegetation in soil, balanced periods of rain, droughts and temperature higher than 15 degrees Celsius facilitate the occurrence of anthrax and influence the ecology and survival of the bacterium bacillus anthraces [9]. The life span of bacillus anthraces is approximated to be 200 years [10]. They die naturally and during rain season at a rate of 0.000014 day^{-1} [12].

Anthrax is a zoonotic disease which is transmitted from infected animal to human beings. Humans are vulnerable to cutaneous type of anthrax [14]. In the cycle of anthrax, the environment as the pathogen's reservoir serves as the central source of the infection. Cattle get the disease during grazing period especially in rain and dry season. An infected animal can cycle the spores or pathogens (bacillus anthraces) again back to the environment during excretion [12]. Humans catch the disease through contacting an infected animal's product during butchering and slaughtering and when eating raw meat from infected animal.

Anthrax has persisted in Africa where for example in Kruger National Park in South Africa, number of roan antelope declined from 450 to 45 animals [5]. In Tanzania, 109 black wildbeest, 21 grant's gazelle,10 cattles and 26 goats died, [8]. Also in Kilimanjaro region, three villages reported hospitalization of 36 who ate anthrax contaminate meat from a cow [11]. In Arusha as well it has been reported that out of 134 people infected with anthrax, 8 died. Also, in Dar es Salaam out of 22 infected people, 6 died [11].

To provide better understanding of anthrax dynamics and suggest control measures to eradicate the disease mathematical models are of paramount importance. Few mathematical models

have been developed. Keeling and Rohani [7] developed mathematical model for anthrax basing on environmental contamination and contact between uninfected animals and infected carcases. The analysis shows that environmental contamination determines the threshold value for the model.

Furniss and Hahn [3] formulated the mathematical model basing on environmental contamination and direct contact between infected animals and non-infected carcasses. The model exhibit the threshold value determined by an environmental contamination parameter. Friedman and Yakubu [2] used a mathematical model developed by Hahn and Furnish and study anthrax is transmitted through carcass ingestion, spread of carcasses to the environment and migration rates on the persistence of animal population. The analysis shows that the spread of carcasses to the environment increases the transmission of the disease. Though the problem is addressed, infection between animals and human being in the transmission dynamics of anthrax is not considered. By modifying the models developed by [14-10], this work is intending to study the dynamics of anthrax when infection between animals and human being is considered.

2. Materials and Methods

2.1 Model Development

The model for anthrax is formulated by extending the models which were developed by Mushayabasa [10] and Sinkie [14] to include human beings. Dynamics of anthrax divides human and animal population each into two classes: susceptible S_h and infected I_h humans, and susceptible S_a and infected I_a animals respectively. Carcasses' class is represented by C.

Susceptible humans increase due to birth at a rate b_h and decrease due to anthrax infection after eating meat from infected animal and carcasses at a rate σ. They also can acquire the disease when they eat or comes into contact with infected carcasses at a rate δ and when they come into contact with environment which is contaminated with pathogen at a rate ϕ. However, infected humans increase at a rate σ when susceptible human eat meat from infected animal and carcasses. Infected humans also increase when susceptible human eat or come into contact with infected carcasses at a rate δ and also when they come into contact with environment which is

contaminated with pathogens at a rate ϕ . However, infected humans decrease due to anthrax and natural induced death at rates r and μ_h respectively.

Susceptible animals replenish due to birth at a rate b_a, however, they decrease due to anthrax infection following contact with pathogens at a rate β. Infected animals increase following infection of susceptible animal at a rate β, they suffer disease induced death at a rate π. All animal classes suffer natural mortality at a rate μ_a.

Infected animals die due to anthrax and become carcasses at a rate π. The carcasses increase at rate of π due to anthrax induced death rate and decrease due to decomposition at a rate of θ and shed pathogens to the environment at a rate θ. The pathogens increase at a rate of θ following carcass's decomposition and decrease at a rate of ω due to natural death and sometimes due to rain flushing off. The interaction between infected animals, environment and humans is well illustrated by the Figure 1

2.2 Model assumptions

The model assumes that all animals and humans are susceptible to the disease. The recruitment rate for new individual is through birth. No incubation period for both animals and humans. Huamans and animals suffer natural mortality at rates μ_h and μ_a respectively. Infected animals and environment are the main source of infections. Carcasses shed pathogens to environment through excretion[12].

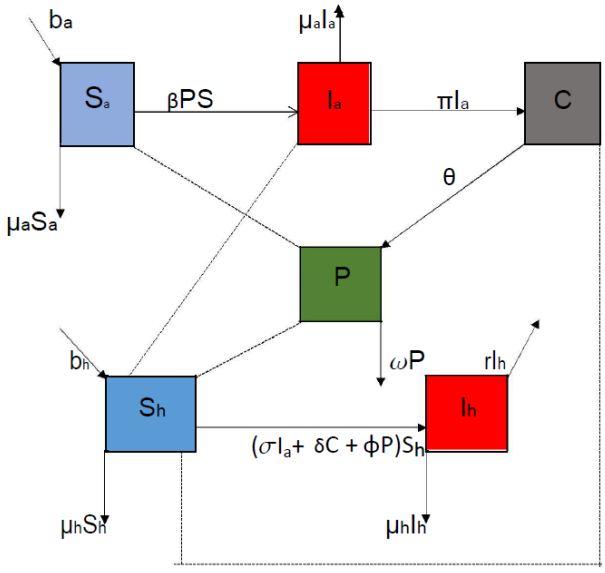


FIGURE 1. The model compartment-mental diagram

Placing together both formulation and assumptions, model which describes the interaction animals, humans and environment is given by:

$$\frac{dS_h}{dt} = b_h - (\sigma I_a + \delta C + \phi P + \mu_h)S_h \tag{1a}$$

$$\frac{dI_h}{dt} = (\sigma I_a + \delta C + \phi P)S_h - (\mu_h + r)I_h \tag{1b}$$

$$\frac{dP}{dt} = \theta C - \omega P \tag{1c}$$

$$\frac{dS_a}{dt} = b_a - (\beta P + \mu_a)S_a \tag{1d}$$

$$\frac{dI_a}{dt} = \beta P S_a - (\mu_a + \pi)I_a \tag{1e}$$

$$\frac{dC}{dt} = (\pi + \mu_a)I_a - \theta C \tag{1f}$$

$S_h(0) > 0; I_h(0) \geq 0; P(0) \geq 0; S_a(0) > 0; I_a(0) \geq 0; C(0) \geq 0$

2.3 Invariant region

Invariant region shows the boundedness of solutions. To determine the region, human, animal and pathogens' population are considered each separately. Humans, animal and pathogens' populations denoted by N_h, N_a and P respectively. Human population is given by:

$$N_h = S_h + I_h$$

$$\frac{dN_h}{dt} \leq b_h - \mu_h S_h \tag{2}$$

Solving the inequality (2) we get:

$$N_h(t) \leq \frac{b_h}{\mu_h} + \left(N_h(0) - \frac{b_h}{\mu_h}\right) e^{-\mu_h t} \tag{3}$$

but as t tends to infinity then: $N_h \leq \frac{b_h}{\mu_h}$ for $N_h(0) = S_h(0) + I_h(0)$.

For animal population:

$$N_a = S_a + I_a + C \tag{4}$$

$$\frac{dNa}{dt} \leq b_a - \mu_a N_a \tag{5}$$

Therefore the solution is given by:

$$N_a(t) \leq \frac{b_a}{\mu_a} + \left(N_a(0) - \frac{b_a}{\mu_a}\right) e^{-\mu_a t} \tag{6}$$

For $N_a(0) = S_a(0) + I_a(0) + C(0)$

Analysis of solution (6) considers two cases:

When $N_a(0) \geq \frac{b_a}{\mu_a}$ and $N_a(0) \leq \frac{b_a}{\mu_a}$. When $N_a(0) \geq \frac{b_a}{\mu_a}$

$$N_a(t) \leq \frac{b_a}{\mu_a} + \left(N_a(0) - \frac{b_a}{\mu_a}\right) e^{-\mu_a t}$$

Since:

$$\lim_{t\to\infty} \left(N_a(0) - \frac{b_a}{\mu_a}\right) e^{-\mu_a t} = \infty$$

then:

$$N_a(t) \leq \frac{b_a}{\mu_a}$$

From the definition of N_a, it follows that:

$$\lim_{t\to\infty} S_a \leq \frac{b_a}{\mu_a}, \lim_{t\to\infty} I_a \leq \frac{b_a}{\mu_a}, \lim_{t\to\infty} C \leq \frac{b_a}{\mu_a}$$

For pathogen population we have:

$$\frac{dP}{dt} = \theta C - \omega P, \text{ put } C \leq \frac{b_a}{\mu_a}$$
$$\frac{dP}{dt} \leq \theta \frac{b_a}{\mu_a} - \omega P \tag{7}$$

Solving the inequality (7) we get:

$$P(t) = \theta \frac{b_a}{\omega \mu_a} + \left(P(0) - \theta \frac{b_a}{\omega \mu_a}\right) e^{\omega \mu_a t} \tag{8}$$

It follows that, analysis of the solution (8) considers two cases, these are: $P(0) > \frac{\theta b_a}{\omega \mu_a}$ and $P(0) < \frac{\theta b_a}{\omega \mu_a}$. When: $P(0) > \frac{\theta b_a}{\omega \mu_a}$
we have:

$$P(0) \leq \theta \frac{b_a}{\omega \mu_a} \leq \theta \frac{b_a}{\omega \mu_a} + \left(P(0) - \theta \frac{b_a}{\omega \mu_a} \right) e^{\omega t}$$

and when:

$$P(0) < \frac{\theta b_a}{\omega \mu_a}$$

, we have:

$$P(0) \leq \theta \frac{b_a}{\omega \mu_a} \leq \theta \frac{b_a}{\omega \mu_a} + \left(P(0) - \theta \frac{b_a}{\omega \mu_a} \right) e^{\omega t}$$

Since:

$$\left(P(0) - \theta \frac{b_a}{\omega \mu_a} \right) e^{-\omega t} \to 0$$

then:

$$\lim_{t \to \infty} P < \frac{b_a}{\omega \mu_a}$$

Therefore the model system (1) is positive invariant in the region:
$\Gamma = \{(S_a, I_a, C, P, S_h, I_h) \in R_+^6 : 0 \leq S_a + I_a + C \leq \frac{b_a}{\mu_a}, 0 \leq P \leq \theta \frac{b_a}{\omega \mu_a}, 0 \leq S_h + I_h \leq \frac{b_h}{\mu_h}\}$.
Solution for the model system (1) which begins on the boundary of the boundary region Γ converge to the region and remain bounded. Therefore the model (1) is mathematically and epidemiologically meaningful and we can consider flow generated for analysis. This result is summarized in the following theorem;

Theorem: Solutions of the model system (1) enter the region:
$\Gamma = \{(S_a, I_a, C, P, S_h, I_h) \in R_+^6 : 0 \leq S_a + I_a + C \leq \frac{b_a}{\mu_a}, 0 \leq P \leq \theta \frac{b_a}{\omega \mu_a}, 0 \leq S_h + I_h \leq \frac{b_h}{\mu_h}\}$

2.4 Positivity of solutions

Theorem: *Let the initial value of variables of the model* (1) *be* $S_a(0) > 0, I_a(0) > 0, P(0) > 0, C(0) > 0, S_h(0) > 0$ *and* $I_h(0) > 0$. *Then the solution set* $\Gamma = \{S_a(0) > 0, I_a(0) > 0, P(0) > 0, C(0) > 0, S_h(0) > 0, I_h(0) > 0\}$ *is positive for all time t.*

Proof: Lets consider the equation number (1a) of the model system (1), we have:

$$\begin{aligned} \frac{dS_a}{dt} &= b_a - (\beta P + \mu_a) S_a \\ \frac{dS_a}{dt} &\geq -(\beta P + \mu_a) S_a \end{aligned} \tag{9}$$

By separating variables we get:

$$\frac{dS_a}{S_a} \geq -(\beta P + \mu_a) dt \tag{10}$$

By integrating both sides, we get;

$$\begin{aligned} \int \frac{dS_a}{S_a} &\geq \int_0^t (\beta P + \mu_a) dt \\ \ln S_a &\geq -\int_0^t (\beta P + \mu_a) dt + C \\ S_a(t) &\geq e^{\int_0^t -(\beta P + \mu_a) t} \end{aligned} \tag{11}$$

At initial condition, we get;

$$\begin{aligned} S_a(t) &\geq S_a(0) e^{\int_0^t (\beta P(s) + \mu_a) dt} \\ S_a(t) &\geq S_a(0) e^{\int_0^t (\beta P(s) + \mu_a) dt} \end{aligned} \tag{12}$$

Then, $S_a(t) \geq 0, \forall\, t \geq 0$

From the equation number (1b) of the model (1) we have;

$$\begin{aligned} \frac{dI_a}{dt} &= \beta P S_a - (\mu_a + \pi) I_a \\ \frac{dI_a}{dt} &\geq -(\mu_a + \pi) \\ \frac{dI_a}{I_a} &\geq -(\mu_a + \pi) dt \\ \int \frac{dI_a}{I_a} &\geq -\int_0^t (\mu_a + \pi) dt \\ I_a(t) &\geq I_a(0) e^{-(\mu_a + \pi) t} \geq 0 \end{aligned} \tag{13}$$

From the equation (1c) of the model (1), we have;

$$\begin{aligned} \frac{dC}{dt} &= \pi I_a - \theta C \\ \frac{dC}{dt} &\geq -\theta C \\ \int \frac{dC}{C} &= -\int_0^t \theta dt \\ C(t) &\geq C(0)e^{-\theta t} \geq 0. \end{aligned} \tag{14}$$

From the equation number (1d) of the model (1), we have;

$$\begin{aligned} \frac{dP}{dt} &= \theta C - \omega P \\ \frac{dP}{dt} &\geq -\omega P \\ \frac{dP}{P} &= \int_0^t -\omega dt \end{aligned} \tag{15}$$

Solving (15) we get:

$$P(t) \geq P(0)e^{-\omega t} \geq 0$$

Again from the equation (1e) of the model (1), we have;

$$\begin{aligned} \frac{dS_h}{dt} &= b_h - (\sigma I_h + \delta C + \mu_h + \phi P)S_h \\ \int \frac{dS_h}{S_h} &\geq -\int_0^t (\sigma I_h + \delta C + \mu_h + \phi P)dt \\ S_h(t) &\geq S_h(0)e^{-\int_0^t (\sigma I_h + \delta C + \mu_h + \phi P)dt} \geq 0 \end{aligned} \tag{16}$$

From the equation number (1f) of the model (1), we have;

$$\begin{aligned} \frac{dI_h}{dt} &= (\sigma I_h + \delta C + \phi P)S_h - (\mu_h + r)I_h \\ \frac{dI_h}{dt} &\geq -(\mu_h + r)I_h \\ \int \frac{dI_h}{I_h} &= \int_0^t -(\mu_h + r)dt \end{aligned} \tag{17}$$

$$I_h t \geq I_h(0)e^{-(\mu_h + r)t} \geq 0$$

2.5 The basic reproduction number R_0

The basic reproduction number is the average number of secondary infections generated by a single individual when introduced in an entirely susceptible population [1, 6]. It determines whether the disease persists or clears out. The disease clears out when $R_0 < 1$ and persists when $R_0 > 1$ [6].

To compute basic reproduction number, we adopt the next generation matrix method where new infections and transfer terms are considered. If the new infections are mathematically defined by f_i and transfer terms by v_i, then the matrices F and V are given by

$$F = \frac{\partial f_i}{\partial X_j}(x_0) \text{ and } V = \frac{\partial v_i}{\partial X_j}(x_0) \tag{18}$$

as defined by Van den Driessche and Watmough [16] . The basic reproduction number R_0 is therefore given by:

$$R_0 = \rho(FV^{-1}). \tag{19}$$

From the model equations (1) the new infections and transfer terms are given by,

$$f_i = \begin{bmatrix} (\sigma I_a + \delta C + \phi P)S_h \\ 0 \\ \beta P S_a \\ 0 \end{bmatrix} \tag{20}$$

and

$$v_i = \begin{bmatrix} (\mu_h + r)I_h \\ \theta C - \omega P \\ (\mu_a + \pi)I_a \\ \pi I_a - \theta C \end{bmatrix}. \tag{21}$$

Derivatives of f_i and v_i with respect to infected classes at disease free equilibrium are:

$$F = \begin{bmatrix} 0 & \sigma\frac{b_h}{mu_h} & \delta\frac{b_h}{\mu_h} & \phi\frac{b_h}{\mu_h} \\ 0 & 0 & 0 & 0 \\ 0 & \beta\frac{b_a}{\mu_a} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \tag{22}$$

and

$$V = \begin{bmatrix} \mu_h + r & 0 & 0 & 0 \\ 0 & -\omega & 0 & \theta \\ 0 & 0 & \mu_a + \pi & 0 \\ & 0 & 0 & \pi & -\theta \end{bmatrix}. \tag{23}$$

The inverse of the matrix V is:

$$V^{-1} = \begin{bmatrix} \frac{1}{\mu_h + r} & 0 & 0 & 0 \\ 0 & \frac{-1}{\omega} & \frac{\pi}{(\mu_a+\pi)\omega} & \frac{-1}{\omega} \\ 0 & 0 & \left(\frac{1}{\mu_a + \pi}\right) & 0 \\ 0 & 0 & \frac{\pi}{(\mu_a+\pi)\theta} & \frac{-1}{\theta} \end{bmatrix} \tag{24}$$

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

$$FV^{-1} = \begin{bmatrix} 0 & -\frac{\phi\, b_h}{\mu_h\omega} & \frac{\sigma\, b_h}{\mu_h(\mu_a+\pi)} + \frac{\delta\, b_h\pi}{\mu_h(\mu_a+\pi)\theta} + \frac{\phi\, b_h\pi}{\mu_h(\mu_a+\pi)\omega} & -\frac{\delta\, b_h}{\mu_h\theta} - \frac{\phi\, b_h}{\mu_h\omega} \\ 0 & 0 & 0 & 0 \\ 0 & -\frac{\beta\, b_a}{\omega\,\mu_a} & \frac{\pi\beta\, b_a}{\mu_a(\mu_a+\pi)\omega} & -\frac{\beta\, b_a}{\omega\,\mu_a} \\ 0 & 0 & 0 & 0 \end{bmatrix} \tag{25}$$

From (19) the basic reproduction number R_0 is given by:

$$R_0 = \frac{\beta b_a \pi}{\mu_a(\mu_a + \pi)\omega} \tag{26}$$

The basic reproduction number R_0 depends on animal infection rate β, animal recruitment rate b_a, animal anthrax induced death rate π, animal natural mortality μ_a and pathogen's natural death rate ω. The basic reproduction number R_0 is directly proportion to β and b_a therefore

increasing β and b_a will also increase the basic reproduction number R_0. Parameters μ_a and ω which are animal and pathogen's natural death rates are inversely proportional to the basic reproduction number R_0. Increasing animal and pathogens' natural death rates will decrease the basic reproduction number R_0.

3. Sensitivity Analysis

Sensitivity analysis assists to understand the potentials of each parameter to disease transmission [4]. It identifies sensitive parameters which should be the target when designing disease interventions. We employ the Maximum Likelihood estimation, an inbuilt Matlab fminsearch function to estimate our parameters.

Table shows the model parameters' values. Parameter values from the related literature were used as initial values to fit the model from the data collected from the field. The data were collected at Ngorongoro district in Tanzania from 2006 to 2016 [11].

Parameter	Values &Units	Source	Fitted Parameters
b_a	$1.369 \times 10^{-}5\ day^{-1}$	[14, 10]	0.0002
β	$0.0001\ day^{-1}$	[14, 10]	0.0016
θ	$0.001125\ day_{-1}$	[14, 10]	0.0386
ω	$0.000014\ day^{-1}$	[14, 10]	0.0003
μ_a	$0.0001\ day^{-1}$	[14, 10]	0.0006
π	0.06	assumed	0.4141
μ_h	$0.00016\ day^{-1}$	assumed	0.0002
b_h	$0.015\ day^{-1}$	assumed	0.0005
δ	$0.000002\ day^{-1}$	Assumed	0.0001
ϕ	$0.000001219\ day^{-1}$	Assumed	0.0003

4. Sensitivity Analysis of R_0

In this section parameters' indices with respect to basic reproduction number R_0 are determined. The basic reproduction number R_0 depends on five parameters which are used to derive analytical expression for each parameters. If λ is a parameter in the basic reproduction number R_0 its sensitivity index with respect to R_0 is given by:

$$r_{\lambda}^{R_0} = \frac{\partial R_0}{\partial \lambda} x \frac{R_0}{\lambda} \tag{27}$$

Using equation (27), sensitivity index for each parameter is given in Table .

Parameter	Sensitivity Index
β	$+1.0000$
b_a	$+1.00000$
π	$+0.001446829127$
μ_a	-1.001446829
ω	-1.0000

From sensitivity indices we note that, positive indices imply proportional relationship with basic reproduction number R_0. Any percentage increase in parameters with positive indices will make the same percentage increase in basic reproduction number R_0. Parameters with positive indices are animal recruitment rate b_a and animal infection rate β.

On the other hand, negative indices imply inverse relationship with basic reproduction number R_0. Any percentage increase in parameters with negative indices will make the same percentage reduction in basic reproduction number R_0. Parameters with negative sensitivity indices are animal death rate μ_a and pathogens' life span ω.

5. Numerical Simulation

In this section long and short terms behavior of anthrax dynamics in both humans and animal by considering sensitive parameter is analyzed. Parameter values from the data collected from the field are used. The general dynamics of anthrax with no controls is demonstrated in Figure 2

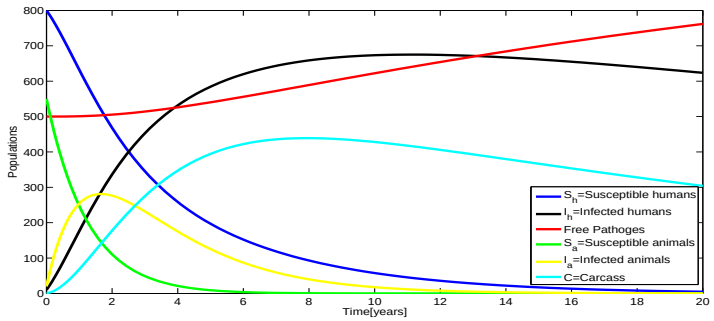
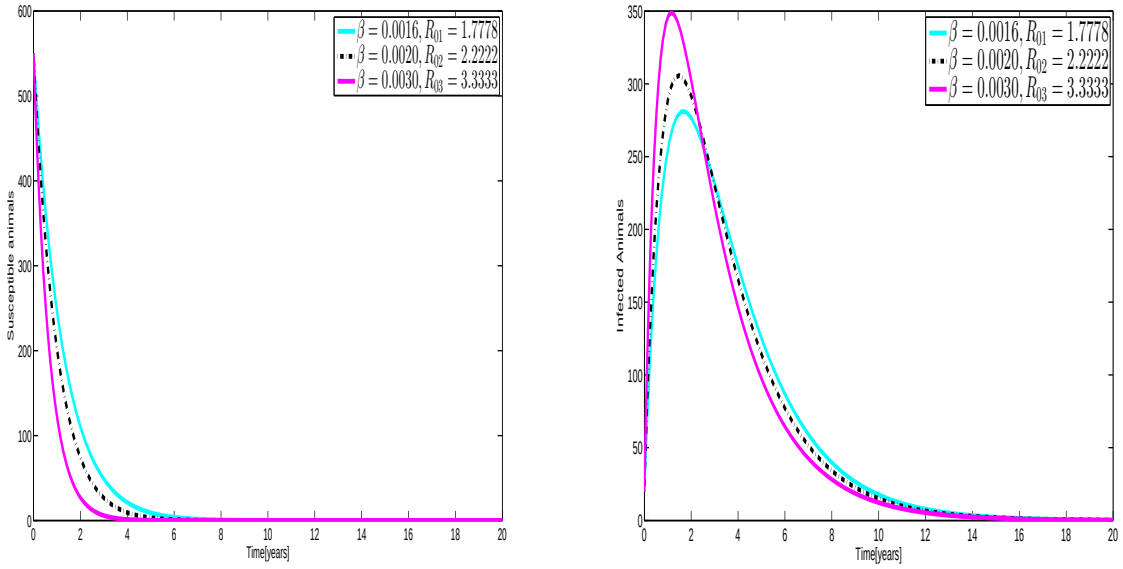


FIGURE 2. The general dynamics of anthrax in humans and animals

Susceptible humans and animals decrease due to anthrax infection rate as shown in Figure 2. Susceptible animals decrease exponentially as they acquire bacillus anthraces during grazing [3, 12]. Humans decrease as they acquire anthrax from environment or by eating meat from infected animals or by contacting infected carcasses or coming into contact with pathogen in the environment. The anthrax infection appear to be critical between five to fifteen years as demonstrated by Figure 2. However, pathogens are increasing due to shedding and decomposition of carcasses.

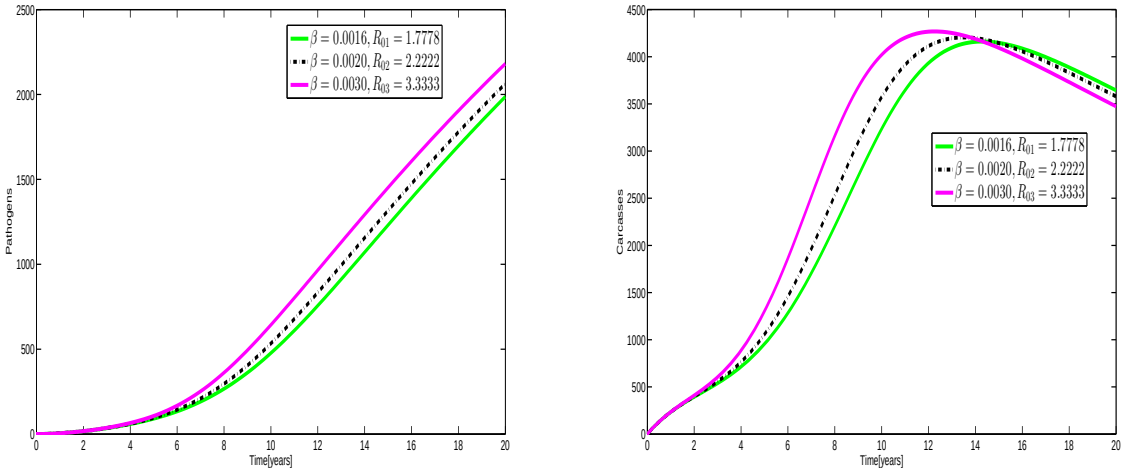


(A) Anthrax in susceptible animals (B) Anthrax in infected animals

FIGURE 3. Variation of anthrax transmission dynamics with animal infection rate

Figure 3 shows the effect of varying animal transmission rate to the dynamics of anthrax in both susceptible and infected animals. It is found that as animal infection rate increases,

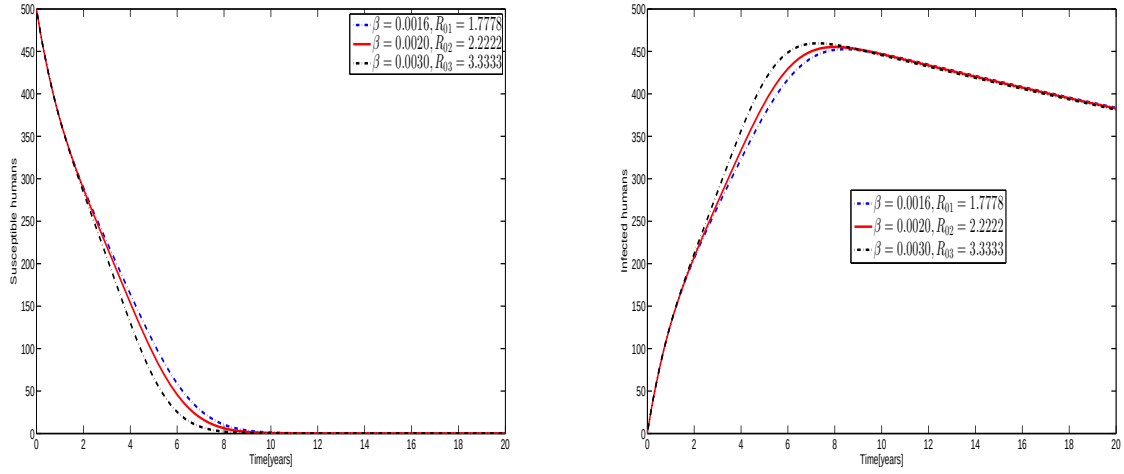
susceptible animals decrease while infected animals flourish. When animals are infected at a rate 0.003 day^{-1}, all susceptible animals may acquire anthrax in twelve years as demonstrated in Figure 3a.



(A) Anthrax in pathogens population (B) Anthrax in carcasses population

FIGURE 4. Variation of animal infection rate in carcass and pathogen Population

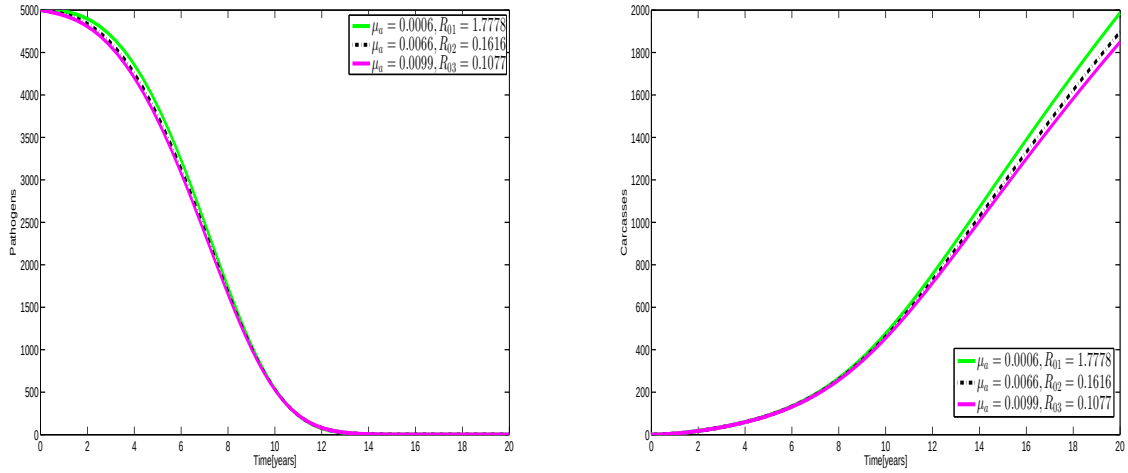
Figure 4a shows the effect of varying animal transmission rate in both pathogens and carcasses population. The results show that as the animal transmission rate increases both carcasses and pathogens increase correspondingly.



(A) Anthrax dynamics in susceptible humans population (B) Anthrax dynamics in infected humans population

FIGURE 5. Variation of animal infection rate in both susceptible and infected humans

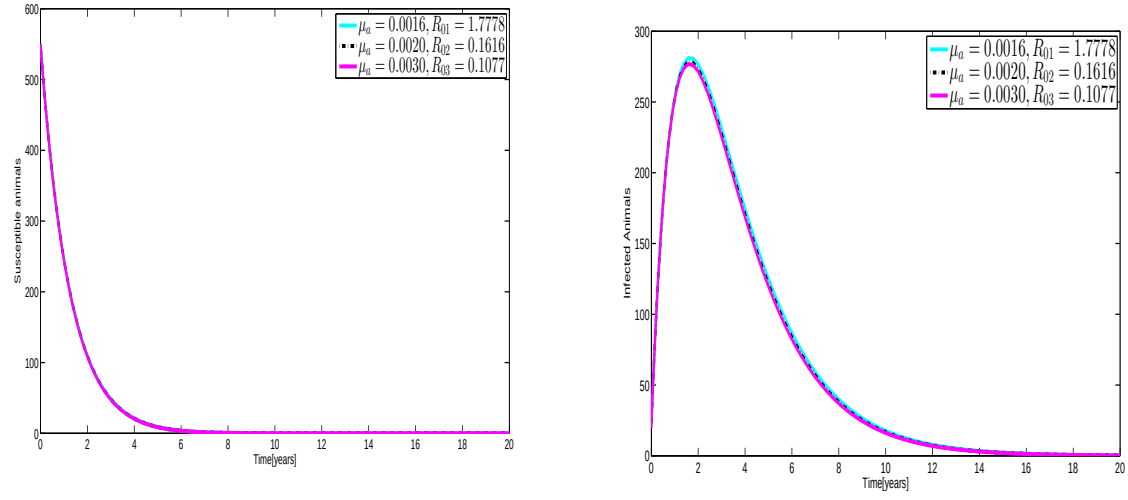
Susceptible and infected humans are demonstrated in Figure 5. The results show that when the animal transmission rate increases, susceptible humans decrease. The rise and fall of the graph are due to the nature of the occurrence of the disease whereby the disease erupts in seasonally during the rainy and high drought period.



(A) Anthrax dynamics in pathogens population (B) Anthrax dynamics in carcasses population

FIGURE 6. Variation of animal natural death rate in both pathogens and carcasses

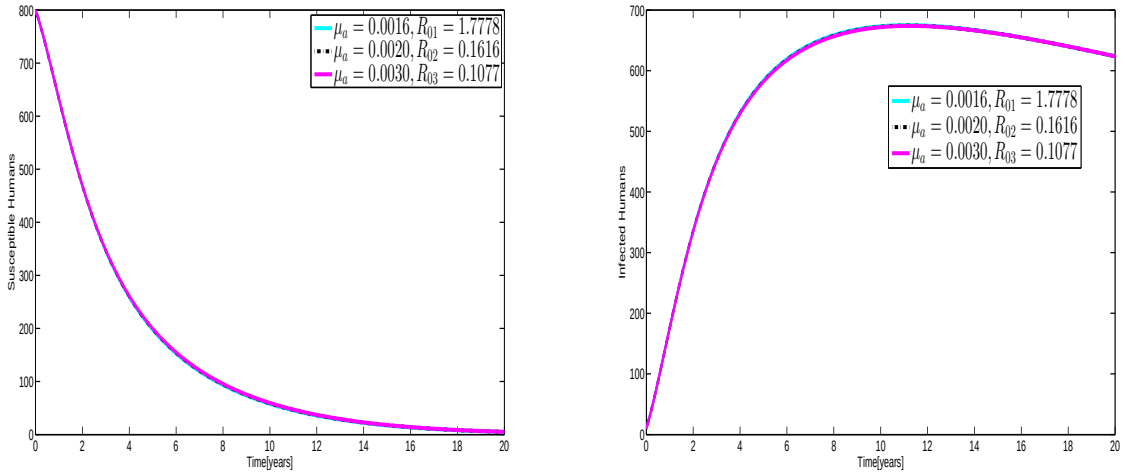
Figure 6 shows the effect of varying the animal natural death rate in both pathogens and carcasses. The results shows pathogen and carcasses decrease as animal death rate increases.



(A) Anthrax dynamics in susceptible animal population (B) Anthrax dynamics in infected animal population

FIGURE 7. Variation of animal natural death rate in both susceptible and infected animal

Figure 7 shows the effect of varying animal natural death rate in both susceptible and infected animals. The result shows that when the animal natural death rate increases both susceptible and infected animal decrease.



(A) Anthrax dynamics in susceptible humans population (B) Anthrax dynamics in infected humans population

FIGURE 8. Variation of animal natural death rate in both susceptible and infected humans

Figure 8 shows the effect of varying animals natural death rate to both susceptible and infected humans. The results show that when animal natural death rate increases susceptible humane increases and infected humans decreases.

6. Conclusion, Discussion and Recommendation

A simple anthrax model in humans and animals is developed and analyzed to determine which parameters drive the transmission dynamics of the disease. The basic reproduction number R_0 is computed and sensitivity index for each parameter in the basic reproduction number R_0 is derived. The analysis shows that animal recruitment and infection rates are more sensitive to the disease transmission. Anthrax infection increases as more animals are recruited and it decreases as animals suffer natural mortality. These results are demonstrated by numerical simulation. To eradicate the disease, this study proposes control strategies such as animal vaccination, fumigation and decomposition of carcasses.

Acknowledgement

The author thanks the Nelson Mandela African Institution of Science and Technology,Germany Academic Exchange and Supervisors for their support.

Conflict of Interests

The authors declare that there is no conflict of interests.

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