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Modeling And Simulation Study Of Anthrax Attack On Environment

Zerihun Mamo Sinkie[1] and Narasimha Murthy S.[2*]
School of Mathematical & Statistical Sciences, Hawassa University, Hawassa, Ethiopia
Email: zerihunnm@gmail.com[1] and simhamurthy44@gmail.com[2]
*Author for correspondence

***Abstract*—Anthrax is an acute febrile disease of warm blooded animals including humans. In this study an epidemiological model of anthrax in animal population is undertaken. We consider mathematical model for anthrax with four compartments, and the effectiveness of clinical signs on the infected animals is clearly brought out. Stability analysis for disease free equilibrium and endemic equilibrium points are studied. We find that increase in curing rate of infected animals by treatment and proper removal of carcasses decrease pathogens and increases the number of susceptible animals.**

Keywords—Anthrax model, mathematical modeling, simulation of model, equilibrium points, eigen values, stability analysis, disinfection measures, anthrax treatment and anthrax attack.

1. Introduction

Anthrax is an acute, febrile i.e., having or showing the symptoms of a fever or a great deal of nervous excitement disease of warm blooded animals, including humans. It is caused by Bacillus anthraces, a gram-positive, non-motile, spore-forming bacterium, and occurs most commonly as a rapidly fatal septicemia in animals [1]. These are extremely resistant to chemical and environmental factors, and have the ability to return into a vegetative or virulent state during the process of germination [3].

It also occurs in humans when they are exposed to infected animals or tissue from infected animals or when they are directly exposed to Bacillus anthraces. Depending on the root of infection, anthrax disease can occur in three forms: coetaneous, gastrointestinal, and inhalation [11]. The ways in which anthrax can be transmitted are through skin, the most common and most often occurs when a person butchers an animal infected with anthrax, by ingestion, transmission occurs when a person eats infected meat, and by inhalation. A person needs to inhale merely $8{,}000 - 10{,}000$ tiny, $1 - 5\,\mu m$ in diameter, anthrax spores to become infected [2]. The incubation which takes approximately 11 days, prodromal stage and are characterized by non-headache neurological symptoms such as fever, fatigue, and muscle aches and the fulminate stage. The individual is very ill and experiencing respiratory distress, death occurs within days of the emergence of symptoms are the three anthrax progression stages. Those that begin prophylaxis during the incubation stage and adhere to the entire 60 days of medication will not become ill. However, those who begin taking medication but stop in the middle of the course may progress to the prodromal and fulminate stages and possibly die [2].

To provide a comprehensive structure for understanding the transmission behavior as well as for evaluating of the effectiveness of different strategic actions against it the qualitative studies are crucial. In this direction mathematical modeling is very important for solving and understanding the dynamics of the spread of infectious disease. As the anthrax is zoonosis or that it can be transmitted from infected animals to human beings, which is naturally found in land covered mainly with grass, suitable for grazing cattle or sheep, Bacillus anthraces spores can persist in the environment for many years, especially in settings that are rich in soil nitrogen and organic content. If livestock infected with anthrax spores, human beings infection occur easily [3].

For the control of anthrax epidemics, it is important to understand not only the pathogenesis and interactions of Bacillus anthraces with host animals but also the ecology of the spores. There are numerous opportunities during an anthrax outbreak for spores to be dispersed over large geographic areas. Carnivores are less susceptible to anthrax than are herbivores. Consequently, carnivores may become sub clinical carriers and disperse ingested spores, Dragon and Rennie, 1995. Avian scavengers e.g., vultures, gulls and ravens, also may disperse widely the spores that adhere to their feathers as they feed on infected carcasses. Infected animals shed the spores in their fasces and urine. Mosquitoes and tabanids have been implicated in mechanical transmission of spores from animal to animal, and from animal to vegetation. Water and wind are other vehicles of transmission [1].

Anthrax primarily affects herbivorous livestock and wildlife species, but also poses serious public health risks in many parts of the world. Carnivores may also become infected by ingesting contaminated carcasses, but disease associated illness and deaths are rather than in herbivores. Anthrax outbreaks in animals in nearly 200 countries are recorded by The World Anthrax Data Site, a World Health Organization Collaborating Center for Remote Sensing and Geographic Information Systems for Public Health. Anthrax is a globally distributed disease, and has

been reported by all continents that are populated densely with animals and humans [4].

In Ethiopia the anthrax occurred in Wabessa village in the Dessie Zuria district, during May and June, and it occurred in the 2002, the mortality rates were 7.7%, 32.7% and 47.1% in cattle, goats and donkeys, respectively [8]. In SNNPR from March to April 2007 E.C., 225 cattle died where as 2 people cured by treatment and 114 cattle died where as 54 people cured by treatment in SEGEN and DEBUB OMO zones respectively [14]. The mathematical model of anthrax developed by Mushayabasa [9] considered only the susceptible animals, the environmental contamination and the number of carcasses of animals that may have died of anthrax. Hahn and Furniss, in [12] and [13] also used before the mathematical model of anthrax contamination of environment and the contact between animal and infected carcasses. However if there is early and effective treatment the infected animals can be cured and hence be the susceptible. Hence, we in this study are interested to model and simulate the anthrax attack by considering infectious and treatment.

Mathematical modeling and simulation studies are successfully applied to population growths [16 – 17], breast cancer [18], Prey – predator system [19] and HIV/AIDS [20].

Recently, Steady Mushayabasa [9] developed an anthrax model with environmental decontamination and time delay. He assumed that an infected animal dies without displaying clinical signs of the disease. Therefore the following questions arise: How, the anthrax infected animals at incubation period can be protected from anthrax induced death? The model, Steady Mushayabasa which ignores the infective compartment or the model assuming the infected compartment, has smaller basic reproductive number? What happen to the Mushayabasa [9] model if treatment on the infective animals considered? And which of the basic reproductive number is more effective at equilibrium points? Hence, we develop a mathematical model for anthrax attack with four compartments by assuming some infected animals can also display clinical signs of disease and can be cured by treatment under the assumption that there is no natural exit of infected and susceptible animals.

2. Methodology

2.1 Construction of the Model

Although anthrax infection in cattle is regarded often as a fatal disease with no signs biologically the infection should take a period before an infected animal succumbs to anthrax-induced mortality, and this period may play an important role in controlling anthrax outbreaks. Because of this reason we extend the work of Steady Mushayabasa [9], by including the infective compartment. We consider the four compartments and the system of ordinary differential equations represent the change of rate of each compartment. The four compartments yield system of odes form SICP model which is the modification of the model SCP developed by Steady Mushayabasa, June, 2015, where $s(t), I(t), C(t)$ and $P(t)$ represent susceptible animals, infected animals, carcasses and free living spores or pathogens respectively.

2.1.1. Model

The present model is as shown in Figure1.

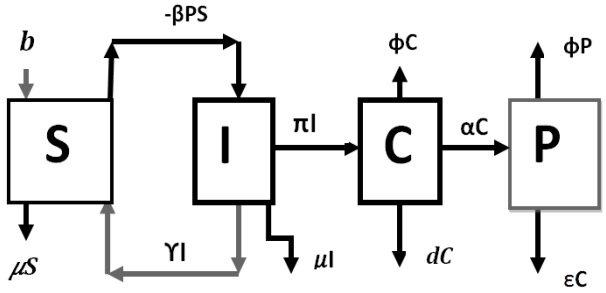

Figure 1: Diagram of mathematical model of anthrax attack on environment

2.1.2. Model formulation

The mathematical model of anthrax attack from the above diagram yields the following differential equations:

$$\frac{dS}{dt} = b + \gamma I(t) - \beta P(t)S(t) - \mu S(t) \quad (1)$$

$$\frac{\mathrm{dI}}{\mathrm{dt}} = \beta P(t)S(t) - (\mu + \Upsilon + \pi)I(t) \quad (2)$$

$$\frac{dC}{dt} = \pi I(t) - (\phi + d)C(t) \quad (3)$$

$$\frac{\mathrm{dP}}{\mathrm{dt}} = \alpha C(t) - (\phi + \varepsilon)P(t) \quad (4)$$

Equation (1) describes the dynamics of the susceptible animals. Here β, b, γ, and μ denote the transmission rate of pathogens to susceptible animals due to contact or ingesting pathogens, the rate of input/inflow of animals due to different cases which maybe one of the natural situations, the curing rate of infected animals by treatment or diagnosis and the rate of natural exit or outflow of animals due to different cases which maybe one of the natural situations respectively. Equation (2) describes the dynamics of infected animals. Here π stands for the rate of anthrax induced death. Equation (3) describes the dynamics of carcasses of animals that are died by anthrax, here ϕ and d denote the rate of disinfection or decontamination of infected areas through the removal or destruction of animal carcasses and adding of lime to the ground where a decomposing animal carcass would have been identified and decaying rate of carcasses which are died due to anthrax attack. Equation (4) describes the dynamics of free living spores or pathogens. Here α and ε stand for rate of shedding of free spores or pathogens into the ground, from carcasses that are not properly removed or from carcasses that are decaying at that environment, and the average life span of free-living pathogens. The length of survival of anthrax free-living spores in the environment is estimated to be around 200 years [9] e.g., $\epsilon = [1/(365 \times 200)] = 0.000014$ per day.

2.1.3. Model analysis

We now find the equilibrium points of the model. There are two equilibrium points such as

anthrax free equilibrium point and endemic equilibrium point.

I. Anthrax free equilibrium point

This is found by considering the right hand side of each equation (1), (2), (3) and (4) equating to zero.

$$b + \gamma I(t) - \beta P(t)S(t) - \mu S(t) = 0 \quad (5)$$
$$\beta P(t)S(t) - (\mu + \Upsilon + \pi)I(t) = 0 \quad (6)$$
$$\pi I(t) - (\phi + d)C(t) = 0 \quad (7)$$
$$\alpha C(t) - (\phi + \varepsilon)P(t) = 0 \quad (8)$$

From (7) we obtain $\pi I(t) - (\phi + d)C(t) = 0$. At the state of disease free, no animals are infected. Hence, $I(0) = 0$ this implies $\pi I(t) - (\phi + d)C(t) = 0$ is true if and only if $I = 0$ and $C = 0$. From (8) we have $[\alpha C(t) - (\phi + \varepsilon)P(t)] = 0$. Hence $C = 0$ and $P = 0$. From (5) we have $[b + \gamma I(t) - \beta P(t)S(t) - \mu S(t)] = 0$. This gives $[b - \mu S(t)] = 0$ or equivalently $S = (b/\mu)$. Therefore the anthrax free equilibrium point is $(S, I, C, P)' = (b/\mu, 0, 0, 0)$. At this anthrax free equilibrium point no infection means no animals die with anthrax disease. And no free spores or pathogens are shed to the ground.

II. The basic reproductive number R_i

Before calculating the basic reproductive number, R_i, we find the Jacobin matrix $J(b/\mu, 0, 0, 0)$. The Jacobin matrix for the present situation is

$J(b/\mu, 0, 0, 0) =$

$$\begin{pmatrix} -\mu & \Upsilon & 0 & -\frac{b\beta}{\mu} \\ 0 & -(\Upsilon + \pi + \mu) & 0 & \frac{b\beta}{\mu} \\ o & \pi & -(\phi + d) & 0 \\ 0 & 0 & \alpha & -(\phi + \varepsilon) \end{pmatrix} \quad (a)$$

We calculate the basic reproductive number by finding the determinant of J.

$\det(J) =$

$$\begin{vmatrix} -\mu & \Upsilon & 0 & -\frac{b\beta}{\mu} \\ 0 & -(\Upsilon + \pi + \mu) & 0 & \frac{b\beta}{\mu} \\ o & \pi & -(\phi + d) & 0 \\ 0 & 0 & \alpha & -(\phi + \varepsilon) \end{vmatrix} \quad (b)$$

The trace and determinant of the matrix J are, respectively, given by $Trace(J) = -(2\phi + \epsilon + d + \Upsilon + \pi + \mu)$, which is a negative quantity and $det(J) = (\mu)(\Upsilon + \pi + \mu)(\phi + d)(\phi + \varepsilon)\{1 - \{b\beta\pi\alpha/a_{11}\}\}$. Here we have used the notation $a_{11} = [\mu(\Upsilon + \pi + \mu)(\phi + d)(\phi + \varepsilon)]$.

Here we now assume the basic reproductive number of the system as

$R_i = \{b\beta\pi\alpha/[\mu(\Upsilon + \pi + \mu)(\phi + d)(\phi + \varepsilon)]\}$ (9)

Hence, $R_i = [\pi / (\Upsilon + \pi + \mu)] R_a < 1$, where we have used the notation $R_a = \{b\beta\pi/[\mu(\phi + d)(\phi + \varepsilon)]\}$. Since, $[\pi/(\Upsilon + \pi + \mu)] < 1$ we have $R_a < 1$ as required, Mushayabasa [9]. Here R_i is the product of R_a and $[\pi/(\Upsilon + \pi + \mu)]$, where R_a stand for the reproductive number assumed by Mushayabasa [9] and R_i is the basic reproductive number of the present model. This implies $R_i < R_a$ which shows the relative merit of present model over Mushayabasa [9] model. Also $Det(J) > 0$ and $Trace(J) < 0$. Hence, the anthrax free equilibrium point ε^0 is locally asymptotically stable and the disease cannot invade the population.

Biologically, the term R_i represents the average number of new anthrax cases generated when susceptible animals ingest disease causing pathogens which is decreased due to the treatment during incubation period. In this case this is also true that the death of animals due to anthrax infection decreases as R_i decreases. But, if $R_i > 1$ then the attack of anthrax increases and the measure to eradicate it must be planed.

III. Endemic equilibrium points

The endemic equilibrium point is given by $E^* = \{S^*, I^*, C^*, P^*$ where susceptible population is $S^* = (b/\mu R_i)$, infected population is $I^* = [1 - (1/R_i)][b(\Upsilon + \pi + \mu)/((\Upsilon + \pi + \mu) - \gamma\mu)\pi\alpha]$, carcasses population is $C^* = [1 - (1/R_i)]\{b(\Upsilon + \pi + \mu)/[(\phi + d)[(\Upsilon + \pi + \mu) - \gamma\mu]\alpha]\}$ and the pathogen population is $P^* = [1 - (1/R_i)]\{b(\Upsilon + \pi + \mu)/[(\phi + d)(\phi + \varepsilon)((\Upsilon + \pi + \mu) - \gamma\mu)]\}$.

Here, $\Upsilon + \mu + \pi > \mu$ or equivalently $[\Upsilon + \mu + \pi - \mu] > 0$. Hence, $R_i > 1$ so that $P^*, I^*, C^* > 0$. The basic reproductive number must be greater than one. Hence we observe that R_i is greater than one so that S^*, I^*, C^* and P^* are nonnegative. Therefore the present model is stable at endemic equilibrium.

IV. Eigenvalues

From the determinant of (b) we have the eigenvalues $\lambda_1, \lambda_2, \lambda_3,$ and λ_4. These are calculated up on evaluating the determinant

$$(-\mu)\begin{vmatrix} -(\Upsilon + \pi + \mu) - \lambda & 0 & b\beta/\mu \\ \pi & -(\phi + d) - \lambda & 0 \\ 0 & \alpha & -(\phi + \varepsilon) - \lambda \end{vmatrix}$$

From this it is clear that one of the eigenvalues $\lambda_1 = -\mu$ and the other three eigenvalues are determined. They are $\lambda_2 = -(\Upsilon + \pi + \mu)$, $\lambda_3 = -(\phi + d)$, and $\lambda_4 = -(\phi + \varepsilon)$. Thus, we conclude that as all the four eigenvalues are real and negative, and hence the system is stable at anthrax free equilibrium point. Thus this model is preferable than the one that ignores infectious treatment [9].

We now verify the effectiveness of the disinfection rate: From equation (9) if the disinfection rate and $\phi = 0$, the basic reproductive number $R_p = [b\beta\pi\alpha/\mu\varepsilon d(\Upsilon + \pi + \mu)]$, then the eigenvalues are $\lambda_1 = -\mu$, $\lambda_2 = -(\Upsilon + \pi + \mu)$, $\lambda_3 = -d$, and $\lambda_4 = -\varepsilon$. On the other hand if curing rate of infected animals is $\Upsilon = 0$, then $R_g = [b\beta\pi\alpha/\mu(\phi + \varepsilon)(\phi + d)(\pi + \mu)]$. Similarly, if both disinfection rate $\phi = 0$ and curing rate $\Upsilon = 0$ then $R_{pg} = [b\beta\pi\alpha/\mu\varepsilon d(\pi + \mu)]$.

3. Simulation of the model

By using Matlab software and parameter values from appendix given at the end of this paper we simulate the model and obtain the following

results: In the initial period from 0 to 10 years there is rapid variable in the anthrax attack and with increase in the period the anthrax reaches stable state in all the four cases as shown in Figure 2. If there are no disinfection measures, then there is rapid decrease of susceptible animals in the period from 0 to 10 and then the pathogen invades the environment that causes all animals die out as shown in Fig. 3. This shows that even if treatment given to the infected population is a factor to tolerate anthrax, the disinfection measures play great advantage. When the curing rate is $\Upsilon = 0$ then the number of susceptible animals approaches to 2000 from initial value of 5500 as shown by Fig. 4. From this we notice that due to lack of treatment the number of susceptible animals decrease at least by half in the same period of time as can be observed from Figure 2 and Figure 4. When both the disinfection rate ϕ and the curing rate Υ are zero, then the susceptible animals die out rapidly and pathogens grow constantly as seen in Figure 5.

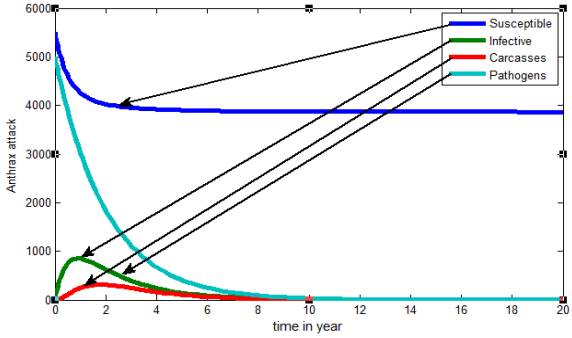

Figure 2: Graphical illustration for parameter values $\beta = 0.0001, \alpha = 0.001125$, $b = 1.369e-5$, $\square = 0.99$, $\mu = 0.0001$, $\pi = 0.6$, $\phi = 0.5$, $d = 0.8$ and $\varepsilon = 0.000014$

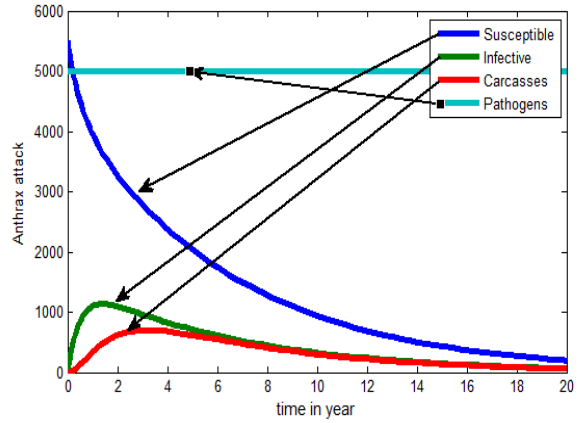

Figure3: Graphical illustration for parameter values $\beta = 0.0001$, $\alpha = 0.001125$, $b = 1.369e-5$, $\square = 0.99$, $\mu = 0.0001$, $\pi = 0.6$, $d = 0.8$, $\varepsilon = 0.000014$ and, $\phi = 0$. Here disinfection measure has not been considered.

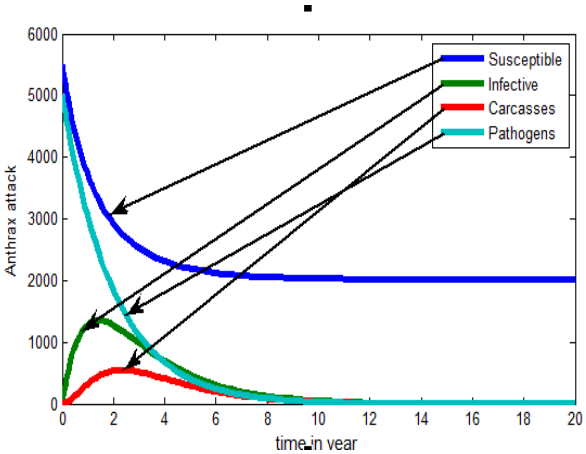

Figure 4: Graphical illustration for parameter values $\beta = 0.0001$, $\alpha = 0.001125$, $b = 1.369e-5$, $\mu = 0.0001$, $\pi = 0.6$, $\phi = 0.5$, $d = 0.8$ and $\varepsilon = 0.000014$, $\square = 0$. Here treatment is not considered.

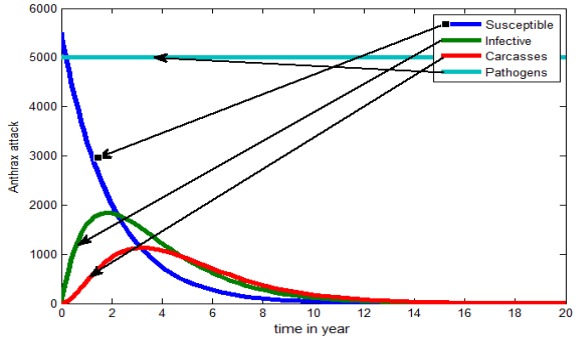

Figure 5: Graphical illustration for parameter values $\beta = 0.0001$, $\alpha = 0.001125$, $b = 1.369e-5$, $\mu = 0.0001$, $\pi = 0.6$, $d = 0.8$ and $\varepsilon = 0.000014$, $\phi = 0$, $\square = 0$. Here treatment and disinfection measures are not considered.

4. Conclusions

Anthrax epidemic is recognized as the result of lack of awareness against its symptoms, no readiness to treat the infected animals, lack of focus to eradicate the carcasses in effective methods. In our study we found the Jacobin matrix, basic reproductive number R_i , eigen values λ_i , and equilibrium points to show whether or not the disease is locally asymptotically stable to the environment and globally asymptotically stable. We have seen that due to treatment of infective animals and increase of disinfection measures, the pathogen dies out in short period of time. This may be important information in controlling the spread of anthrax. Another vital problem is lack of attention about the properties of anthrax like its viability for long period, ways of transmission and damage that it causes. We would like to take up study on this in future.

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References

[1]. Reginald Johnson, Epizootiology and ecology of anthrax, United States department of agriculture, Animal and Plant Health Inspection Service, 970-494-7326, reginald.a.johnson@aphis.usda.gov.

[2]. Michelle Lee Houck, Master of Science, Compartmental and simulation models for evaluating Medkit prepositioning strategies for anthrax Attack Response, University of Maryland, 2011.

[3]. Paola A. Pinzón-Arango, Terri A. Camesano, Nancy A. Burnham, George D. Pins, Maria M. Santore, Robert W. Thompson, Investigation of the Biological and Physicochemical properties of Bacillus anthracic spores during germination, Virulence, and Killing January 2012.

[4]. Avner Friedman, Abdul Aziz Yakubu, Mathematical Biosciences, 241, 2013, 137–144 www.elsevier.com/locate/mbs.

[5]. Diane C. Jamrog, Michael P. Shatz, and Cassandra Smith. Modeling responses to anthrax and Smallpox attacks, Lincoln Laboratory Journal, Vol. 17, No. 1, 2007.

[6]. Kumar R., Chow C. C., Bartels J. D., Clermont G. and Vodovotz Y. A mathematical simulation of the inflammatory response to anthrax infection, Jan 2008, Vol. 29, No. 1, 104-11.

[7]. Yeshigeta Gelaw and Tsedeke Asaminew. Periocular cutaneous anthrax in Jimma zone, Southwest Ethiopia: a case series, http://www.biomedcentral.com/1756-0500/6/313, 2013.

[8]. K. G. Shiferaw, Anthrax in Wabessa village in the Dessie Zuria district of Ethiopia, 7 September 2004 (Report).

[9]. Steady Mushayabasa, Global stability of an anthrax model with environmental decontamination and time delay, Hindawi publishing corporation, Discrete dynamics in nature and society, Vol. 2015, Article ID 573146, 6 pages http://dx.doi.org/$10.1155/2015/573146.

[10]. Rukmini Kumar, Carson C. Chow, John D. Bartels, Gilles Clermont, and Yoram Vodovotz. A Mathematical simulation of the Inflammatory response to anthrax infection, shock, Vol. 29, No. 1, pp. 104, Y111, (2008).

[11]. John F. Modlin, M. D., Dixie E. Snider, Jr., M. D., M. P. H. Use of Anthrax vaccine in the United States, U.S. Department of health & human services, Center for disease control and prevention CDC, October 2000.

[12]. B. D. Hahn and P. R. Furniss. A deterministic model of an anthrax epizootic: threshold results, 1983.

[13]. B. D. Hahn and P. R. Furniss. A mathematical model of an anthrax epizootic in the Kruger national park, Appl. Math. Model. 5 1981.

[14]. Alemayehu Yohannes and Zenebe Zerihun. The Anthrax outbreak field study report in SNNPR Debub Omo and Segen people's zones, 2015.

[15]. Mushayabasa S Modeling schistosomiasis and HIV/AIDS Codynamics, 2011.

[16]. **Mohammed Yiha Dawed, Purnachandra Rao Koya**, **Ayele Taye Goshu**. Mathematical Modelling of Population Growth: The Case of Logistic and Von Bertalanffy Models. Open Journal of Modelling and Simulation (OJMSi). Vol. 2, 2014, pp 113-126. http://dx.doi.org/10.4236/ojmsi.2014.24013

[17]. **Ayele Taye Goshu, Purnachandra Rao Koya**. Predator Population Dynamics Involving Exponential Integral Function when Prey follows Gompertz Model. Open Journal of Modelling and Simulation (OJMSi). Vol. 3, 2015, pp 70 – 80. http://dx.doi.org/10.4236/ojmsi.2015.33008

[18]. **Abdulsamad Engida Sado, Purnachandra Rao Koya.** Application of Brody Growth Function to Describe Dynamics of Breast Cancer Cells. American Journal of Applied Mathematics (AJAM). Vol. 3, No. 3, 2015, pp. 138-145. Doi:10.11648/j.ajam.20150303.20

[19]. Mohammed Yiha Dawed, Purnachandra Rao Koya, Temesgen Tibebu. Analysis of prey – predator system with prey population experiencing critical depensation growth function. American Journal of applied Mathematics. Vol. 3, No. 6, 2015, Pp. 327 – 334. Doi: 10.11648/j.ajam.20150306.23

[20]. Tadele Tesfa Tegegne, Purnachandra Rao Koya, Temesgen Tibebu Mekonen. Modeling the Combined Effect of Vertical Transmission and Variable inflow of infective immigrants on the dynamics of HIV/AIDS. American Journal of Applied Mathematics. Vol. 4, No. 1, 2016, pp. 11-19. Doi: 10.11648/j.ajam.20160401.12

Appendix

Model parameters and their baseline values are chosen from the available literature and used in the simulation study. They are the following:

Parameter and description	Baseline	Value & Units
α : Pathogen shedding rate	0.001125 to 0.1	0.001125 C per day
d **:** Decay rate of carcasses	0.4 to 1.2	0.4 Per day
β : Transmission rate	$1.3X10^{-10}$ to 0.0001	0.0001 Per day
ε : Life span of pathogens	$1.369x10^{-5}$ to $3.92x10^{-5}$	0.000014 Per day
Φ: Rate of disinfection of environment	0 to 1	0.5 percent
Rate of anthrax induced death	0.12 to 1	0.6 Per C per day
Natural exit/ outflow rate		0.0001 Per animal per day
Curing rate of infectious	0 to 1	0.99 Per infectious
Inflow rate/ including birth	$1.369x10^{-5}$ to $5.479x10^{-5}$	$1.369x10^{-5}$ Per day per animal