Research Article

Global Stability of an Anthrax Model with Environmental Decontamination and Time Delay

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Anthrax occurs worldwide and is associated with sudden death of cattle and sheep. This paper considers an epidemic model of anthrax in animal population, only. The susceptible animal is assumed to be infected, only, through ingestion of the disease causing pathogens. The proposed model incorporates time delay and environmental decontamination by humans. The time delay represents the period an infected animal needs to succumb to anthrax-related death. By constructing suitable Lyapunov functionals, we demonstrate that the global dynamics of this model fully hinges on whether the associated reproductive number is greater or less than unity. The effectiveness of environmental decontamination on eradication of anthrax in the community is explored through the reproductive number.

1. Introduction

Anthrax is an acute zoonotic disease caused by the spore-forming bacterium *Bacillus anthracis* [1]. The disease can infect all warm-blooded animals, including humans. However, ruminants, particularly cattle and sheep, are more susceptible [2]. The disease is associated with death of cattle and sheep, such that very few livestock producers or veterinarians have witnessed the disease or its signs [2]. Outbreaks often occur when livestock are grazing on neutral or slightly alkaline soils and have been exposed to the spores through ingestion of contaminated soil in endemic areas or forage is sparse because of overgrazing or drought or when soil has been disturbed by digging or other human activities [2]. Anthrax is one of the most dramatic diseases affecting wild animal in Africa. For instance, within Etosha National Park, anthrax has been attributed to the death of a variety of herbivores, from elephants to ostriches [3].

The use of mathematical models to explore the spread and control of infectious diseases has proved to be an important tool for the scientists and epidemiologists. In 1983, Hahn and Furniss [4] constructed a deterministic model to explore the impact of environmental contamination and animal carcasses on driving anthrax epidemic. Within their framework they

assumed that upon infection infected animals die immediately and there are no births or deaths from causes other than anthrax related [4]. More, recently, Friedman and Yakubu [3] extended the model of Hahn and Furniss [4] to study the effects of anthrax transmission, carcass ingestion, carcass induced environmental contamination, and migration rates on the persistence or extinction of animal population. Their work revealed among others that decreasing the levels of carcass ingestion by removal of carcasses in game reserves may not always lead to a reduction in anthrax cases.

Although anthrax infection in cattle is regarded often as a fatal disease with no signs [2], 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. So incorporating this reason we extend the work of Hahn and Furniss [4] to include a fixed delay. Apart from the delay, our new model incorporates the role of human effort on decontamination of the environment through the destruction of animal carcass and disinfection of the ground or area that contained the carcass by adding lime. The main goal of this paper is to explore the effectiveness of environmental decontamination on controlling anthrax outbreaks and to study the global stability of the anthrax model with time delay.

2. Model Formulation and Analysis

A framework to assess the dynamics of anthrax in livestock is proposed. Hahn and Furniss's [4] deterministic model of anthrax dynamics provides the starting point for our discussion on the effectiveness of environmental decontamination on controlling the spread of anthrax. The framework is governed by the following system of nonlinear ordinary differential equations:

$$\begin{aligned} \frac{dS}{dt} &= b - \beta P(t) S(t) - \mu S(t), \\ \frac{dC}{dt} &= \beta P(t) S(t) - (\phi + d) C(t), \\ \frac{dP}{dt} &= \alpha C(t) - (\phi + \epsilon) P(t). \end{aligned} \quad (1)$$

The first equation describes the dynamics of the susceptible animals. The parameter b denotes the entrance of new animals through birth and they are assumed to be susceptible to the disease; β is the disease transmission rate which occurs when a susceptible animal ingests the free-living spores while grazing in an endemic area; μ denote the permanent exit of the animals due to natural causes or other reasons not related to anthrax infection. Upon infection, we assume that an infected animal dies without displaying clinical signs of the disease. Thus, the second equation captures the dynamics of the carcasses of animals that may have succumbed to anthrax-induced death; d denote the decay rate of carcasses which is assumed to be constant; ϕ 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. The third equation describes dynamics of free-living spores or pathogens; α denote the rate at which carcasses of animals that would have died of anthrax and not properly destroyed shed the bacteria into the environment; ϵ^{-1} is 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 [5]; thus $\epsilon = 1/(365 \times 200) = 0.000014 \text{ day}^{-1}$.

For biological reasons we will study the dynamics of system (1) in the closed set

$$\begin{aligned} \Gamma = \left\{ (S, C, P) \in \mathbb{R}_+^3 : 0 \leq S + C \leq \frac{b}{\omega}, 0 \leq P \leq \frac{\alpha b}{(\phi + \epsilon)\omega} \right\}, \end{aligned} \quad (2)$$

where $\mathbb{R}_+^3$ denotes the nonnegative cone of $\mathbb{R}^3$ including its lower dimensional faces and $\omega \leq \min(\mu, \phi, d)$. It can easily be verified that Γ is positively invariant with respect to (1).

2.1. Equilibria and the Reproductive Number. In the absence of anthrax infection in the community, system (1) admits

an infection-free equilibrium given by $\mathcal{E}^0 = (b/\mu, 0, 0)$. The Jacobian matrix of system (1) evaluated about $\mathcal{E}^0$ is

$$J = \begin{pmatrix} -\mu & 0 & -\frac{\beta b}{\mu} \\ 0 & -(\phi + d) & \frac{\beta b}{\mu} \\ 0 & \alpha & -(\phi + \epsilon) \end{pmatrix}. \quad (3)$$

It is clear that $-\mu$ is an eigenvalue of matrix J . The other two are determined from matrix A . Consider

$$A = \begin{pmatrix} -(\phi + d) & \frac{\beta b}{\mu} \\ \alpha & -(\phi + \epsilon) \end{pmatrix}. \quad (4)$$

The trace and determinant of A are, respectively, given by

$$\begin{aligned} \text{tr}(A) &= -(2\phi + \epsilon + d), \\ \det(A) &= (\phi + d)(\epsilon + \phi) \left[1 - \frac{\beta \alpha b}{\mu(\phi + d)(\phi + \epsilon)} \right]. \end{aligned} \quad (5)$$

Let the reproductive number of system (1) be

$$\mathcal{R}_a = \frac{\beta \alpha b}{\mu(\phi + d)(\phi + \epsilon)}. \quad (6)$$

Since $\text{tr}(A) < 0$ and $\det(A) > 0$ (when $\mathcal{R}_a < 1$) it follows that the equilibrium point $\mathcal{E}^0$ is locally asymptotically stable if and only if $\mathcal{R}_a < 1$.

Biologically, the term $\mathcal{R}_a$ represents the average number of new anthrax cases generated when susceptible animals ingest disease causing pathogens. It gives a measure of the power of anthrax to invade the cattle population in the presence of environmental decontamination.

Direct calculation can easily show that system (1) has two possible equilibrium points, namely, the infection-free $\mathcal{E}^0$ and the endemic equilibrium point $\mathcal{E}^*$, given by

$$\begin{aligned} \mathcal{E}^* &= \left\{ S^* = \frac{b}{\mu \mathcal{R}_a}, C^* = \frac{\mu(\epsilon + \phi)}{\alpha \beta} (\mathcal{R}_a - 1), P^* = \frac{\mu}{\beta} (\mathcal{R}_a - 1) \right\}. \end{aligned} \quad (7)$$

From (7) it is evident that $\mathcal{E}^*$ makes biological sense whenever $\mathcal{R}_a > 1$.

2.2. Effectiveness of Environmental Decontamination. In this section we explore the strength of environmental decontamination on reducing or eliminating new anthrax infections. In the absence of environmental decontamination in the community ($\phi = 0$) the average number of new anthrax infections generated through spores ingestion is modeled by the threshold quantity $\mathcal{R}_0$ which is given by

$$\mathcal{R}_0 = \frac{\beta \alpha b}{\mu \epsilon d}. \quad (8)$$

FIGURE 1: Graphical illustration showing the strength of environmental decontamination on controlling the spread of anthrax. We consider $\epsilon = 0.00014 \text{ day}^{-1}$ [5] and $d = 0.06 \text{ day}^{-1}$ [4].

To explore the effectiveness of environmental decontamination on controlling new anthrax infections we define the efficacy function

$$\begin{aligned} E(\phi) &= \left[\frac{\mathcal{R}_0 - \mathcal{R}_a}{\mathcal{R}_0} \right] \times 100\% \\ &= \left[1 - \frac{\epsilon d}{(\epsilon + \phi)(\phi + d)} \right] \times 100\%. \end{aligned} \quad (9)$$

From (9) it is clear that the effectiveness of environmental decontamination depends on the length of survival of free-living pathogens in the environment and the decay rate of undestroyed carcasses of animals that have succumbed to anthrax infection. In Figure 1 we numerically explore the effectiveness of different environmental decontamination levels.

Numerical results in Figure 1 suggest that environmental decontamination rate 0.05 day^{-1} can be effective in reducing the generation of new anthrax infections by 50%. Further, we note that environmental decontamination rate greater than or equal to 0.01 day^{-1} can be effective in attaining 100% control on the spread of anthrax in the community.

2.3. Anthrax Model with Time Delay. Although anthrax infection in cattle is often a fatal disease with no clinical signs displayed by an infected animal, it is worth noting that the infection should take a period before an infected animal succumbs to anthrax-induced death and the size of this period may play an important role in controlling the outbreak of this disease. So incorporating this reason we introduce a time delay into system (1) to represent the aforementioned period:

$$\begin{aligned} \frac{dS}{dt} &= b - \beta P(t) S(t) - \mu S(t), \\ \frac{dC}{dt} &= \beta P(t - \tau) S(t - \tau) - (\phi + d) C(t), \\ \frac{dP}{dt} &= \alpha C(t) - (\phi + \epsilon) P(t). \end{aligned} \quad (10)$$

System (10) satisfies the following initial conditions:

$$\begin{aligned} S(\theta) &= \phi_1(\theta), \\ C(\theta) &= \phi_2(\theta), \\ P(\theta) &= \phi_3(\theta), \end{aligned} \quad (11)$$

$\theta \in [-\tau, 0],$

where $\phi_i(\theta)$ for $i = 1, 2, 3$ denote the nonnegative continuous functions on $\theta \in [-\tau, 0]$. All the parameters in system (10) are the same as in system (1) except for the positive constant τ which represents the length of the delay. It can be verified that Γ is positively invariant with respect to system (10). We denote by $\partial\Gamma$ and $\hat{\Gamma}$ the boundary and the interior of Γ in $\mathbb{R}_+^3$, respectively. With the same motivation as before, we introduce the reproductive number of differential-delay model (10) which is given by a similar expression

$$\mathcal{R}_a = \frac{\beta \alpha b}{\mu(\phi + d)(\phi + \epsilon)}. \quad (12)$$

2.3.1. Anthrax-Free Equilibrium Point and Its Stability. Direct calculation shows that system (10) has the same disease-free equilibrium $\mathcal{E}^0 = (b/\mu, 0, 0)$ as in system (1). The Jacobian matrix of system (10) about $\mathcal{E}^0$ is

$$J_w = \begin{pmatrix} -\mu & 0 & -\frac{\beta b}{\mu} \\ 0 & -(\phi + d) & \frac{\beta b}{\mu} e^{-\lambda\tau} \\ 0 & \alpha & -(\phi + \epsilon) \end{pmatrix}. \quad (13)$$

From (13) it follows that the characteristic equation is

$$\begin{aligned} (\lambda + \mu) \left[\lambda^2 + (2\phi + \epsilon + d)\lambda + (\epsilon + \phi)(\phi + d) \right. \\ \left. - \frac{\beta \alpha b}{\mu} e^{-\lambda\tau} \right] = 0. \end{aligned} \quad (14)$$

Since $\lambda = -\mu < 0$ is a root of (14), we only need to consider

$$\lambda^2 + a_{11}\lambda + a_{12} + b_{11}e^{-\lambda\tau} = 0, \quad (15)$$

where

$$\begin{aligned} a_{11} &= (2\phi + \epsilon + d) > 0, \\ a_{12} &= (\epsilon + \phi)(\phi + d) > 0, \\ b_{11} &= -\frac{\beta \alpha b}{\mu} < 0. \end{aligned} \quad (16)$$

Equation (14) with $\tau = 0$ is

$$\lambda^2 + a_{11}\lambda + a_{12} + b_{11} = 0. \quad (17)$$

If $\tau = 0$, we have already deduced that all roots of system (15) have negative real parts when $\mathcal{R}_a < 1$, and only one root of (15) has positive real part when $\mathcal{R}_a > 1$.

For $\tau > 0$, we assume that $\lambda = i\omega$ ($\omega > 0$) is a root of (15). This is the case if and only if

$$-\omega^2 + ia_{11}\omega + a_{12} + b_{11}(\cos \omega\tau - i \sin \omega\tau) = 0. \quad (18)$$

Separating the real and imaginary parts yields

$$\begin{aligned} -\omega^2 + a_{12} &= -b_{11} \cos \omega\tau, \\ -a_{11}\omega &= -b_{11} \sin \omega\tau. \end{aligned} \quad (19)$$

Adding up the squares of both equations, we obtain

$$\omega^4 + (a_{11}^2 - 2a_{12})\omega^2 + a_{12}^2 - b_{11}^2 = 0. \quad (20)$$

Let $z = \omega^2$, so that (21) reduces to

$$z^2 + (a_{11}^2 - 2a_{12})z + a_{12}^2 - b_{11}^2 = 0. \quad (21)$$

Substituting for a_{11} , a_{12} , and b_{11} gives

$$\begin{aligned} &z^2 + (\epsilon + \phi)^2 (\phi + d)^2 z \\ &+ (\epsilon + \phi)^2 (\phi + d)^2 \left[1 - \left(\frac{\beta\alpha b}{\mu(\epsilon + \mu)(\phi + d)} \right)^2 \right] \end{aligned} \quad (22)$$

$$= 0.$$

Substituting $\mathcal{R}_a$ into (22) gives

$$\begin{aligned} &z^2 + (\epsilon + \phi)^2 (\phi + d)^2 z + (\epsilon + \phi)^2 (\phi + d)^2 [1 - \mathcal{R}_a^2] \\ &= 0. \end{aligned} \quad (23)$$

Whenever $\mathcal{R}_a < 1$, (23) has two roots which have a positive product implying that they are complex or they are real but they have the same sign. In addition, they have negative sum which implies that they are either real and negative or complex conjugates with negative real parts. Consequently, (23) does not have positive real roots which lead to the conclusion that there is no ω such that $i\omega$ is a solution of (14). Therefore, it follows from Lemma 2.4 of Ruan and Wei [6] that the real parts of all eigenvalues of characteristic equation (14) are negative for all values of the delay $\tau \geq 0$. Thus, we have the following theorem.

Theorem 1. *The equilibrium point $\mathcal{E}^0$ of system (10) is locally asymptotically stable for all time delay $\tau \geq 0$ if $\mathcal{R}_a < 1$.*

In the next theorem we establish the global stability of the infection-free equilibrium for system (10).

Theorem 2. *The equilibrium point $\mathcal{E}^0$ of system (10) is globally asymptotically stable when $\mathcal{R}_a \leq 1$ for all $\tau \geq 0$.*

Proof. We denote by x_t the translation of the solution of system (10); that is, $x_t = (S(t + \theta), C(t + \theta), P(t + \theta))$, where $\theta \in [-\tau, 0]$. Let us define a Lyapunov functional

$$\begin{aligned} U(x_t) &= \alpha C(t) + \alpha\beta \int_{t-\tau}^t P(\theta) S(\theta) d\theta \\ &+ (\phi + d) P(t). \end{aligned} \quad (24)$$

The derivative of V along the solutions of (10) is given by

$$\begin{aligned} &\frac{dU(x_t)}{dt} \\ &= \alpha \frac{dC(t)}{dt} + \frac{d}{dt} \left(\alpha\beta \int_{t-\tau}^t P(\theta) S(\theta) d\theta \right) \\ &+ (\phi + d) \frac{dP(t)}{dt} \\ &= \beta\alpha P(t) S(t) - (\phi + d)(\phi + \epsilon) P(t) \\ &\leq (\phi + d)(\epsilon + \phi) \left[\frac{\beta\alpha b}{\mu(\phi + d)(\epsilon + \phi)} - 1 \right] P(t) \\ &= (\phi + d)(\epsilon + \phi)(\mathcal{R}_a - 1) P(t) \leq 0. \end{aligned} \quad (25)$$

Therefore, $\dot{U} \leq 0$ holds for all $P \geq 0$ and $\mathcal{R}_a \leq 1$. Furthermore, $\dot{U} = 0$ if and only if $P = 0$, or $\mathcal{R}_a = 1$. Therefore, the largest compact invariant set in $\{(S, C, P) \in \Gamma : \dot{U} = 0\}$, when $\mathcal{R}_a \leq 1$, is the singleton $\{\mathcal{E}^0\}$. Thus, LaSalle's invariance principle [7] implies that $\mathcal{E}^0$ is globally asymptotically stable in Γ . This proves the theorem. $\square$

2.3.2. Endemic Equilibrium Point and Its Stability

Theorem 3. *Suppose $\mathcal{R}_a > 1$; then the equilibrium point $\mathcal{E}^0$ is unstable.*

Proof. From the discussion in Section 2.3.1, we have deduced that the characteristic equation associated with $\mathcal{E}^0$ is given by (14), and we only need to consider (15). From the above computations it can easily be verified that if $\tau = 0$, (15) has a positive root when $\mathcal{R}_a > 1$. Now, let $i\omega$ ($\omega > 0$) be a root of (15). Then solving (23) gives

$$\begin{aligned} \omega_+^2 &= \frac{1}{2} (\epsilon + \phi)^2 (\phi + d)^2 \\ &\cdot \left[-1 + \sqrt{1 - 4 \frac{(1 - \mathcal{R}_a^2)}{(\epsilon + \phi)^2 (\phi + d)^2}} \right], \end{aligned} \quad (26)$$

$$\begin{aligned} \omega_-^2 &= \frac{1}{2} (\epsilon + \phi)^2 (\phi + d)^2 \\ &\cdot \left[-1 - \sqrt{1 - 4 \frac{(1 - \mathcal{R}_a^2)}{(\epsilon + \phi)^2 (\phi + d)^2}} \right]. \end{aligned} \quad (27)$$

For $\mathcal{R}_a > 1$ it is evident that (26) makes sense while (27) is meaningless.

Define

$$\begin{aligned} \tau_j &= \frac{1}{\omega_+} \left[\arcsin \left(-\frac{\mu(2\phi + \epsilon + d)\omega_+}{\beta\alpha b} \right) + 2n\pi \right], \\ &j = 0, 1, 2, \dots \end{aligned} \quad (28)$$

Then (14) has a pair of purely imaginary roots $\pm i\omega$ when $\tau = \tau_j$ and has no roots appearing on the imaginary axis when $\tau \neq \tau_j$ for $j = 0, 1, 2, \dots$

Further, let $\lambda(\tau) = \psi(\tau) + i\omega(\tau)$ be the root of (14) satisfying $\psi(\tau_j) = 0$ and $\omega(\tau_j) = \omega_+$.

Differentiating both sides of (14) with respect to τ we get

$$(2\lambda + a_{11} - b_{11}\tau e^{-\lambda\tau}) \frac{d\lambda}{d\tau} = b_{11}\tau e^{-\lambda\tau}. \quad (29)$$

This gives

$$\begin{aligned} \left(\frac{d\lambda}{d\tau}\right)^{-1} &= \frac{2\lambda + a_{11}}{b_{11}\lambda e^{-\lambda\tau}} - \frac{\tau}{\lambda} \\ &= \frac{-\mu(\epsilon + 2(\lambda + \phi) + d)}{\beta\alpha b} e^{\lambda\tau} - \frac{\tau}{\lambda}. \end{aligned} \quad (30)$$

Thus

$$\begin{aligned} \text{sign} \left\{ \frac{d(\text{Re}(\lambda(\tau_j)))}{d\tau} \right\} &= \text{sign} \left\{ \text{Re} \left(\frac{d\lambda}{d\tau} \right)^{-1} \right\}_{\tau=\tau_j} \\ &= \text{sign} \left\{ \frac{\mu^2(2\omega_+^2 + 2\phi^2 + \epsilon^2 + d^2 + 2\phi(\epsilon + d))}{(\beta\alpha b)^2} \right\} \\ &> 0. \end{aligned} \quad (31)$$

By applying Lemma 2.4 in Ruan and Wei [6] and observing that (15) has a positive real root when $\tau = 0$, we obtain that characteristic equation (14) has a positive root at least for all $\tau \geq 0$. Therefore, the equilibrium point $\mathcal{E}^0$ of system (10) is unstable when $\mathcal{R}_a > 1$. This completes the proof. $\square$

Now, we investigate the effect of the time delay on the local stability of $\mathcal{E}^*$. The characteristic equation of system (10) at the endemic equilibrium $\mathcal{E}^*$ takes the following form:

$$\lambda^3 + a_{21}\lambda^2 + a_{22}\lambda + a_{23} = e^{-\lambda\tau} (b_{21}\lambda + b_{22}), \quad (32)$$

where

$$\begin{aligned} a_{21} &= 2\phi + \epsilon + d + \mu\mathcal{R}_a, \\ a_{22} &= (2\phi + \epsilon + d)\mu\mathcal{R}_a + (\phi + d)(\epsilon + \phi), \\ a_{23} &= \mu(\phi + d)(\epsilon + \phi)(2\mathcal{R}_a - 1), \\ b_{21} &= (\phi + d)(\epsilon + \phi), \\ b_{22} &= \mu(\phi + d)(\epsilon + \phi)\mathcal{R}_a. \end{aligned} \quad (33)$$

When $\tau = 0$, (32) becomes

$$\begin{aligned} \lambda^3 + a_{21}\lambda^2 + \mu(2\phi + \epsilon + d)\mathcal{R}_a\lambda \\ + \mu(\phi + d)(\epsilon + \phi)(\mathcal{R}_a - 1) = 0. \end{aligned} \quad (34)$$

By the Hurwitz criteria, all the roots of (34) have only negative real parts. Thus $\mathcal{E}^*$ is locally asymptotically stable when $\tau = 0$. Now, we consider the case $\tau \neq 0$. If $i\omega$ ($\omega > 0$) is a solution of (32), we have

$$\begin{aligned} -i\omega^3 - a_{21}\omega^2 + ia_{22}\omega + a_{23} \\ = (\cos \omega\tau - i \sin \omega\tau) (ib_{21}\omega + b_{22}). \end{aligned} \quad (35)$$

Separating the real and imaginary parts we have

$$\begin{aligned} \omega^3 - a_{22}\omega &= -b_{21}\omega \cos \omega\tau + b_{22} \sin \omega\tau, \\ a_{21}\omega^2 - a_{23} &= -b_{21}\omega \sin \omega\tau - b_{22} \cos \omega\tau. \end{aligned} \quad (36)$$

Squaring and adding the two equations of (36) yields

$$\begin{aligned} \omega^6 + (a_{21}^2 - 2a_{22})\omega^4 + (a_{22}^2 - 2a_{21}a_{23} - b_{21}^2)\omega^2 + a_{23}^2 \\ - b_{22}^2 = 0, \end{aligned} \quad (37)$$

where

$$\begin{aligned} a_{21}^2 - 2a_{22} &= (\phi + d)^2 + (\epsilon + \phi)^2 + \mu^2\mathcal{R}_a^2 > 0, \\ a_{22}^2 - 2a_{21}a_{23} - b_{21}^2 &= \mu^2(\epsilon + d)^2\mathcal{R}_a^2 + 2\mu(\epsilon + \phi) \\ &\cdot (\phi + d)(2\phi + \epsilon + d) \\ &\cdot \left[1 + \mathcal{R}_a \left(\frac{\mu}{(2\phi + \epsilon + d)} - 1 \right) \right] > 0, \\ a_{23}^2 - b_{22}^2 &= \mu^2(\phi + d)^2(\epsilon + \phi)^2(\mathcal{R}_a - 1)(3\mathcal{R}_a - 1) \\ &> 0. \end{aligned} \quad (38)$$

Hence, if $\mathcal{R}_a > 1$, (32) has no positive roots. Accordingly, if $\mathcal{R}_a > 1$, the endemic equilibrium $\mathcal{E}^*$ exists and is locally asymptotically stable for all $\tau \geq 0$. Further, using a Lyapunov functional, we can obtain the following theorem.

Theorem 4. *Whenever $\mathcal{R}_a > 1$, then the unique equilibrium $\mathcal{E}^*$ of system (10) is globally asymptotically stable in $\hat{\Gamma}$ for all $\tau \geq 0$.*

Proof. We consider the following Lyapunov functional:

$$\begin{aligned} V_a(x_t) &= S(t) - S^* \ln S(t) + C(t) - C^* \ln C(t) \\ &+ \frac{\beta P^* S^*}{\alpha C^*} (P(t) - P^* \ln P(t)) \\ &+ \int_{t-\tau}^t \beta [P(\theta)S(\theta) - P^* S^* \ln P(\theta)S(\theta)] d\theta. \end{aligned} \quad (39)$$

Differentiating V_a along the solution (S, C, P) of system (10) and using the identities

$$\begin{aligned} b &= \beta P^* S^* + \mu S^*, \\ (\phi + d) &= \frac{\beta P^* S^*}{C^*}, \\ (\phi + \epsilon) &= \frac{\alpha C^*}{P^*}, \end{aligned} \quad (40)$$

one gets

$$\begin{aligned} \frac{dV_a}{dt} = & \mu S^* \left(2 - \frac{S(t)}{S^*} - \frac{S^*}{S(t)} \right) + \beta P^* S^* \left(3 - \frac{S^*}{S(t)} \right. \\ & - \frac{C(t) P^*}{C^* P(t)} - \frac{S(t - \tau) P(t - \tau) C^*}{S^* C(t) P^*} \\ & \left. + \ln \frac{S(t - \tau) P(t - \tau)}{S(t) P(t)} \right). \end{aligned} \quad (41)$$

Here,

$$2 \leq \frac{S(t)}{S^*} + \frac{S^*}{S(t)}, \quad (42)$$

for all $S(t) \geq 0$, because the arithmetic mean is greater than or equal to the geometric mean. Further, note that $3 - a - b - c + \ln(abc) \leq 0$ for any $a > 0$, $b > 0$, and $c > 0$; and the equality is satisfied if and only if $a = b = c = 1$, and, consequently, $\dot{V}_a(t) \leq 0$. Moreover, the largest invariant set of $\dot{V}_a(t) = 0$ is a singleton where $S(t) \equiv S^*$, $C(t) = C^*$, and $P(t) \equiv P^*$. By the Lyapunov-LaSalle invariance principle [7], we obtain global asymptotic stability of the endemic equilibrium (S^*, C^*, P^*) under the condition $\mathcal{R}_a > 1$. $\square$

3. Concluding Remarks

Anthrax epidemic is now recognized among other factors as the leading cause of species extinctions. In this paper, we propose and analyze an anthrax epidemic model. The model focuses on anthrax transmission in animal population only. Our model is based on the assumption that the disease transmission occurs only when a susceptible animal ingests the disease causing pathogen from contaminated soil in endemic areas when forage is sparse because of overgrazing or drought or when soil has been disturbed by digging or excavations. The proposed model incorporates time delay and environmental decontamination effort. The time delay represents the period that is needed for an infected animal to succumb to anthrax-induced death. We determine the reproductive number and establish that the global dynamics depends on whether the reproductive number is greater than one.

Conflict of Interests

The author declares no conflict of interests.

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