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## BIFURCATION, SENSITIVITY AND OPTIMAL CONTROL ANALYSIS OF MODELLING ANTHRAX-LISTERIOSIS CO-DYNAMICS

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**Abstract.** In this paper, a deterministic model for the co-dynamics of Anthrax and Listeriosis diseases was formulated to investigate the qualitative and quantitative relationship of both diseases by incorporating prevention and treatment controls. The basic reproduction number, stability, existence and equilibria of each disease was investigated separately. The Anthrax-Listeriosis co-dynamics model was analysed and it the idea of backward bifurcation existed. The impact of Anthrax infection on the transmission of Listeriosis was determined. The Anthrax-Listeriosis co-dynamics model was extended and included time dependent control variables. Pontryagin's Maximum Principle was used to obtain the optimal control strategies needed for eradication of Anthrax-Listeriosis infections. We performed the numerical simulation of the co-dynamics model in order to give the quantitative implications of the results. It was established that Anthrax infection can be attributed to increased risk of Listeriosis but Listeriosis infection is not associate with the risk of Anthrax. Effective control of Anthrax means incorporating both the intervention strategies of Anthrax and Listeriosis.

**Keywords:** co-dynamics; optimal control; stability analysis; endemic equilibrium; sensitivity analysis.

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## 1. INTRODUCTION

Researchers in [1, 2] attempted to determine effectiveness of vaccination policies using SIR model. From the theoretical results of their study under constant vaccination, the dynamics of the disease model is similar to dynamics without vaccination. Several studies have used the methods of optimal control theory in the formulation of the models [3]. However, some of these studies focused on the effects of vaccination on the spread and transmission of the diseases as in the case of the authors in [4].

Moreover, authors in [5] studied a disease transmission model by considering the impact of a protective vaccine and came out with the optimal vaccine coverage threshold required for disease eradication. Also, in [6], optimal control was used to study a nonlinear SIR epidemic model with vaccination strategy. Some modelling techniques have been employed and established the role of optimal control using SIR epidemic model [7, 8]. [9], formulated an SIR epidemic model by considering vaccination as control measure.

Authors in [10] formulated a model for the transmission of Listeriosis in animal and human populations but never considered optimal control strategies in combating the disease. [11], also applied optimal control to investigate the impact of chemo-therapy on malaria disease with infection immigrants and [12] applied optimal control methods associated with preventing exogenous reinfection based on a exogenous reinfection tuberculosis model.

[13] researched on the identification and reservoirs of pathogens for effective control of sporadic disease and epidemics. *Listeria monocytogenes* is among the major zoonotic food borne pathogen that is responsible for approximately twenty eight percent of most food-related deaths in the United States annually and a major cause of serious product recalls worldwide. The dairy farm has been observed as a potential point and reservoir for *listeria monocytogenes*.

*Listeria monocytogenes* is the third major and common pathogen responsible for bacterial meningitis among neonates in North America. Factors that are responsible and can increase the risk of Listeriosis include acquired and induced immune suppression linked with HIV infection, hematologic malignancies, cirrhosis, diabetes, hemochromatosis and renal failure with hemodialysis [14].

Deterministic models are often used to study the dynamics of diseases in epidemiology. In recent times application of models in the study of disease transmission has increased. The availability of clinical data and electronic surveillance has facilitated the applications of mathematical models to critical examining of scientific hypotheses and the design of strategies of combating diseases[15, 16, 17].

## 2. MODEL FORMULATION

In this section, we divide the population into compartments. Total human and vector populations were represented by  $N_h$  and  $N_v$  respectively. Total human and vector populations expressed as;

$$(1) \quad N_h = S_h + I_a + I_l + I_{al} + R_a + R_l + R_{al}.$$

$$(2) \quad N_v = S_v + I_v.$$

Variable	Description
$S_h$	Susceptible humans
$I_a$	Anthrax Infected humans
$I_l$	Listeriosis Infected humans
$I_{al}$	Co-infected humans
$R_a$	Anthrax Recovered humans
$R_l$	Listeriosis Recovered humans
$R_{al}$	Co-infected Recovered humans
$S_v$	Susceptible animals
$I_v$	Infected animals
$C_p$	Population of carcasses

TABLE 1. Co-dynamics model variables with their interpretations.

Table 2 shows parameters used in the model formulation and their descriptions.

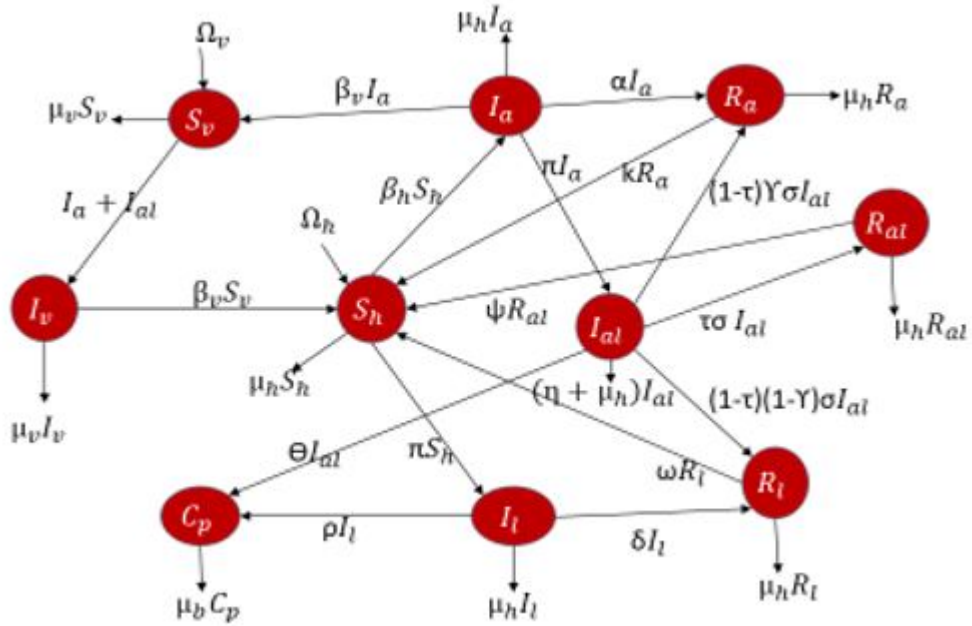


FIGURE 1. Flow chart for the co-infection model.

Parameter	Description	Value	Reference
$\phi$	Anthrax death rate	0.2	[18]
$m$	Listeriosis death rate	0.2	[10]
$q$	Co-infected Anthrax related death rate	0.04	assumed
$\eta$	Co-infected Listeriosis related death rate	0.08	assumed
$\beta_h$	Human transmission rate	0.01	[19]
$\beta_v$	Vector transmission rate	0.05	assumed
$k$	Anthrax waning immunity	0.02	assumed
$\mu_v$	Vector natural death rate	0.0004	[19]
$\Omega_h$	Human recruitment rate	0.001	assumed
$\Omega_v$	Vector recruitment rate	0.005	[19]
$\alpha$	Anthrax recovery rate	0.33	[20]
$\delta$	Listeriosis recovery rate	0.002	assumed
$\psi$	Co-infected waning immunity	0.07	assumed
$\rho$	Listeriosis contribution to environment	0.65	assumed
$\sigma$	Co-infected recovery rate	0.005	assumed
$\mu_b$	Bacteria death rate	0.0025	assumed
$\mu_h$	Human natural death rate	0.20	[21]
$\omega$	Listeriosis waning immunity	0.001	assumed
$\theta$	Modification parameter	0.45	assumed
$\varepsilon$	Co-infected Anthrax recovery only	0.025	assumed
$K$	Concentration of carcasses	10000	[20]
$\nu$	Bacteria ingestion rate	0.50	[22]

TABLE 2. Co-dynamics model parameters and their interpretations.

The variables used in the formulation of the model are described in Table 1.

Waning immunity rates are given by;  $\omega, k$  and  $\psi$ . Where  $\alpha, \delta$  and  $\sigma$  are the recovery rates respectively and  $\tau(1 - \sigma)$  are the co-infected persons who have recovered from Anthrax only.

The co-infected infected persons who have recovered Listeriosis is denoted by  $(1 - \tau)(1 - \sigma)$ .

This implies that;  $\sigma + \tau(1 - \sigma) + (1 - \tau)(1 - \sigma) = 1$ . Where,  $\pi = \frac{C_p \nu}{k + C_p}$ .

$$(3) \quad \left. \begin{aligned} \frac{dS_h}{dt} &= \Omega_h + kR_a + \omega R_l + \psi R_{al} - \beta_h I_v S_h - \pi S_h - \mu_h S_h \\ \frac{dI_a}{dt} &= \beta I_v S_h - \pi I_a - (\alpha + \mu_h + \phi) I_a \\ \frac{dI_l}{dt} &= \pi S_h - \beta_l I_v I_l - (\delta + \mu_h + m + \rho) I_l \\ \frac{dI_{al}}{dt} &= \beta_h I_v I_l + \pi I_a + (\sigma + \mu_h + \eta + \theta) I_{al} \\ \frac{dR_a}{dt} &= \alpha I_a - (k + \mu_h) R_a + (1 - \tau) \gamma \sigma I_{al} \\ \frac{dR_l}{dt} &= \delta I_l - (\omega + \mu_h) R_l + (1 - \tau)(1 - \gamma) \sigma I_{al} \\ \frac{dR_{al}}{dt} &= \tau \sigma I_{al} - (\psi + \mu_h) R_{al} \\ \frac{dC_p}{dt} &= \rho I_l + \theta I_{al} - \mu_b C_p \\ \frac{dS_v}{dt} &= \Omega_v - \beta_v (I_a + I_{al}) S_v - \mu_v S_v \\ \frac{dI_v}{dt} &= \beta_v (I_a + cI) S_v - \mu_v I_v \end{aligned} \right\}$$

### 3. ANALYSIS OF LISTERIOSIS ONLY MODEL

In this section, Listeriosis model only is considered.

$$(4) \quad \left. \begin{aligned} \frac{dS_h}{dt} &= \Omega_h + \omega R_l - \pi S_h - \mu_h S_h \\ \frac{dI_l}{dt} &= \pi S_h - (\delta + \mu_h + m) I_l \\ \frac{dR_l}{dt} &= \delta I_l - (\omega + \mu_h) R_l \\ \frac{dC_p}{dt} &= \rho I_l - \mu_b C_p \end{aligned} \right\}$$

**3.1. Disease Free Equilibrium (DFE).** We obtain the DFE of the Listeriosis only model by using system of equations in (4).

$$(5) \quad \Omega_h + \omega R_l - \pi S_h - \mu_h S_h = 0$$

$$(6) \quad S_h = \frac{\Omega_h}{\mu_h}$$

$$(7) \quad \xi_{0l} = (S_h^*, I_l^*, R_l^*, C_p^*) = \left( \frac{\Omega_h}{\mu_h}, 0, 0, 0 \right).$$

**3.2. Basic reproduction number.** In this section, we employ the concept of the Next Generation Matrix in computing  $\mathfrak{R}_{0l}$ . Using the theorem in [23] on the Listeriosis model only in (4). ( $\mathfrak{R}_{0l}$ ), is given by:

$$(8) \quad \mathfrak{R}_{0l} = \frac{v\rho\Omega_h}{\mu_b\mu_h K(\delta + \mu_h + m)}$$

**3.3. Existence of the disease free equilibrium.** The (*DFE*) of the Listeriosis model only was obtained using system of equations in (4). This was obtained as;

$$(9) \quad \xi_{0l} = \left( \frac{\Omega_h}{\mu_h}, 0, 0, 0 \right).$$

$\mathfrak{R}_{0l}$  of the Listeriosis only model was established as;

$$(10) \quad \mathfrak{R}_{0l} = \frac{v\rho\Omega_h}{\mu_b\mu_h K(\delta + \mu_h + m)}.$$

Using the next generation operator in [23, 24], the linear stability can be established on the system of equations in (4). The disease-free equilibrium, ( $\xi_{0l}$ ) is locally asymptotically stable whenever ( $\mathfrak{R}_{0l} < 1$ ) and unstable whenever ( $\mathfrak{R}_{0l} > 1$ ).

**3.4. Endemic equilibrium (*EE*).** The EE points are computed using system of equations in (4). The EE points are as follows:

$$S_h^* = \frac{\Omega_h + \omega R_l^*}{\mu_h + \pi^*}, I_l^* = \frac{\pi^* S_h^*}{(\delta + \mu_h + m)}, R_l^* = \frac{\delta I_l^*}{\omega + \mu_h}, C_p^* = \frac{\rho I_l^*}{\mu_b}.$$

$$\xi_{0l} = (S_h^*, I_l^*, R_l^*, C_p^*) = \left( \frac{\Omega_h + \omega R_l^*}{\mu_h + \pi^*}, \frac{\pi^* S_h^*}{(\delta + \mu_h + m)}, \frac{\delta I_l^*}{\omega + \mu_h}, \frac{\rho I_l^*}{\mu_b} \right).$$

$$(11) \quad \xi_{0l} = \left( \frac{\Omega_h + \omega R_l^*}{\mu_h + \pi^*}, \frac{\pi^* S_h^*}{(\delta + \mu_h + m)}, \frac{\delta I_l^*}{\omega + \mu_h}, \frac{\rho I_l^*}{\mu_b} \right)$$

$$(12) \quad \left. \begin{aligned} S_h^* &= \frac{\Omega_h + \omega R_l^*}{\mu_h + \pi^*} \\ I_l^* &= \frac{\pi^* S_h^*}{(\delta + \mu_h + m)} \\ R_l^* &= \frac{\delta I_l^*}{\omega + \mu_h} \\ C_p^* &= \frac{\rho I_l^*}{\mu_b} \end{aligned} \right\}$$

### 3.5. Existence of the endemic equilibrium (EE).

**Lemma 1.** *The Listeriosis only model has a unique EE if and only if  $\mathfrak{R}_{0l} > 1$ .*

*Proof.* Listeriosis force of infection;  $\left( \pi = \frac{C_p v}{K + C_p} \right)$ , satisfies the polynomial;

$$(13) \quad P(\pi^*) = A(\pi^*)^2 + B(\pi^*) = 0$$

Where;

$$A = \Omega_h \rho (\omega + \mu_h) + \mu_b K (m(\omega + \mu_h) + \mu_h (\delta + \mu_h + \omega))$$

and

$$B = (\omega + \mu_h) (1 - R_{0l}).$$

By mathematical induction,  $A > 0$  and  $B > 0$  whenever  $\mathfrak{R}_{0l} < 1$ . It implies that  $\pi^* = \frac{-B}{A} \leq 0$ .

In conclusion, the Listeriosis model has no EE any time  $\mathfrak{R}_{0l} < 1$ .  $\square$

The analysis illustrates the impossibility of backward bifurcation in the Listeriosis only model. Since there is no existence of EE whenever  $\mathfrak{R}_{0l} < 1$ .

## 4. ANALYSIS OF ANTHRAX ONLY MODEL

In this section, Anthrax only model is considered in the analysis of disease transmission.



$$(14) \quad \left. \begin{aligned} \frac{dS_h}{dt} &= \Omega_h + kR_a - \beta_h I_v S_h - \mu_h S_h \\ \frac{dI_a}{dt} &= \beta I_v S_h - (\alpha + \mu_h + \phi) I_a \\ \frac{dR_a}{dt} &= \alpha I_a - (k + \mu_h) R_a \\ \frac{dS_v}{dt} &= \Omega_v - \beta_v I_a S_v - \mu_v S_v \\ \frac{dI_v}{dt} &= \beta_v I_a S_v - \mu_v I_v \end{aligned} \right\}$$

**4.1. Disease free equilibrium (DFE).** We obtain the DFE of the Anthrax only model using system of equations in (14).

$$(15) \quad \Omega_h + kR_a - \beta_h I_v S_h - \mu_h S_h = 0$$

$$(16) \quad S_h = \frac{\Omega_h}{\mu_h}$$

$$(17) \quad \Omega_v - \beta_v I_a S_v - \mu_v S_v = 0$$

$$(18) \quad S_v = \frac{\Omega_v}{\mu_v}$$

$$(19) \quad \xi_{0a} = (S_h^*, I_a^*, R_a^*, S_v^*, I_v^*) = \left( \frac{\Omega_h}{\mu_h}, 0, 0, \frac{\Omega_v}{\mu_v}, 0 \right)$$

**4.2. Basic reproduction number ( $\mathfrak{R}_{0a}$ ).** In this section, We employed the concept of the Next Generation Matrix in computing ( $\mathfrak{R}_{0a}$ ). Using the theorem in [23] on the Anthrax model in equation (14),  $\mathfrak{R}_{0a}$  of the Anthrax only model is given by:

$$(20) \quad \mathfrak{R}_{0a} = \sqrt{\frac{\Omega_h \Omega_v \beta_h \beta_v}{\mu_h \mu_v^2 (\alpha + \mu_h + \phi)}}$$

**4.3. Stability of the disease-free equilibrium.** Using the next generation operator concept in [23] on the systems of equation in model (14), the linear stability of  $(\xi_{0a})$ , can be established. The DFE is locally asymptotically stable whenever  $\mathfrak{R}_{0a} < 1$  and unstable otherwise.

**4.4. Endemic equilibrium.** The EE points are computed using system of equations in (14).

The EE points are as follows:

$$S_h = \frac{\Omega_h + kR_a^*}{\mu_h + \beta_h I_v^*}, I_a^* = \frac{\beta_v S_h^* I_v^*}{(\alpha + \mu_h + \phi)}, R_a^* = \frac{\alpha I_a^*}{k + \mu_h}, S_v^* = \frac{\Omega_v}{\mu_v + \beta_v I_a^*}, I_v^* = \frac{\beta_v S_v^* I_a^*}{\mu_v}.$$

The EE of the Anthrax only model is given by;

$$\xi_{0a} = (S_h^*, I_a^*, R_a^*, S_v^*, I_v^*) = \left( \frac{\Omega_h + kR_a^*}{\mu_h + \beta_h I_v^*}, \frac{\beta_v S_h^* I_v^*}{(\alpha + \mu_h + \phi)}, \frac{\alpha I_a^*}{k + \mu_h}, \frac{\Omega_v}{\mu_v + \beta_v I_a^*}, \frac{\beta_v S_v^* I_a^*}{\mu_v} \right)$$

Hence;

$$(21) \quad \xi_{0a} = \left( \frac{\Omega_h + kR_a^*}{\mu_h + \beta_h I_v^*}, \frac{\beta_v S_h^* I_v^*}{(\alpha + \mu_h + \phi)}, \frac{\alpha I_a^*}{k + \mu_h}, \frac{\Omega_v}{\mu_v + \beta_v I_a^*}, \frac{\beta_v S_v^* I_a^*}{\mu_v} \right)$$

**4.5. Existence of the endemic equilibrium.**

**Lemma 2.** *The Anthrax only model has a unique EE whenever  $\mathfrak{R}_{0a} > 1$ . Considering the EE points of the Anthrax only model;*

$$Proof. \quad \xi_{0a} = \left( \frac{\Omega_h + kR_a^*}{\mu_h + \beta_h I_v^*}, \frac{\beta_v S_h^* I_v^*}{(\alpha + \mu_h + \phi)}, \frac{\alpha I_a^*}{k + \mu_h}, \frac{\Omega_v}{\mu_v + \beta_v I_a^*}, \frac{\beta_v S_v^* I_a^*}{\mu_v} \right).$$

The EE point satisfies the given polynomial;

$$(22) \quad P(I_a^*) = A_1 (I_a^*)^2 + B_1 (I_a^*) = 0$$

Where;

$$A_1 = \beta_v (\Omega_v \beta_h (k\phi + \mu_h (\alpha + k + \phi + \mu_h)) + \mu_h (k + \mu_h) (\alpha + \phi + \mu_h) \mu_v)$$

and

$$B_1 = (k + \mu_h) (1 - R_{0a}^2).$$

By induction,  $A_1 > 0$  and  $B_1 > 0$  whenever  $\mathfrak{R}_{0a} < 1$ . Hence,  $I_a^* = \frac{-B_1}{A_1} \leq 0$ . In conclusion, the Anthrax only model has no endemic any time  $\mathfrak{R}_{0a} < 1$ .  $\square$

The analysis shows the impossibility of backward bifurcation in the Anthrax only model. Because there is no existence of EE whenever  $\mathfrak{R}_{0a} < 1$ .

## 5. ANTHRAX-LISTERIOSIS CO-INFECTION MODEL

We consider the dynamics of the Anthrax-Listeriosis co-infection of system of equations in equation (3).

**5.1. Disease free equilibrium (DFE).** The DFE of the Anthrax-Listeriosis model is obtained using system of equations in (3).

$$(23) \quad \Omega_h + kR_a + \omega R_l + \psi R_{al} - \beta_h I_v S_h - \pi S_h - \mu_h S_h = 0$$

$$(24) \quad S_h = \frac{\Omega_h}{\mu_h}$$

$$(25) \quad \Omega_v - \beta_v (I_a + cI_{al}) S_v - \mu_v S_v = 0$$

$$(26) \quad S_v = \frac{\Omega_v}{\mu_v}$$

DFE is given by;

$$\xi_{0al} = (S_h^*, I_l^*, I_a^*, I_{al}^*, R_l^*, R_a^*, R_{al}^*, C_p^*, S_v^*, I_v^*)$$

Hence;

$$(27) \quad \xi_{0al} = \left( \frac{\Omega_h}{\mu_h}, 0, 0, 0, 0, 0, 0, 0, \frac{\Omega_v}{\mu_v}, 0 \right)$$

**5.2. Basic reproduction number.** The concept of the next generation operator method in [23] was employed on the system of equations in (3) to compute  $\mathfrak{R}_{al}$  of the Anthrax-Listeriosis co-infection model. The  $\mathfrak{R}_{al}$  given by;

$$(28) \quad \mathfrak{R}_{al} = \max \{ \mathfrak{R}_a, \mathfrak{R}_l \}$$

Where,  $\mathfrak{R}_a$  and  $\mathfrak{R}_l$  are the reproduction numbers of Anthrax and Listeriosis respectively.

$$(29) \quad \mathfrak{R}_a = \sqrt{\frac{\Omega_h \Omega_v \beta_h \beta_v}{\mu_h \mu_v^2 (\alpha + \mu_h + \phi)}}$$

and

$$(30) \quad \mathfrak{R}_l = \frac{v\rho\Omega_h}{\mu_b\mu_h K} \left( \frac{(\sigma + \mu_h + \eta + \theta) + \theta(\delta + \mu_h + m)}{(\delta + \mu_h + m)(\sigma + \mu_h + \eta + \theta)} \right)$$

**Theorem 3.** *The  $\xi_{0al}$  is locally asymptotically stable whenever  $\mathfrak{R}_{al} < 1$  and unstable otherwise.*

**5.3. Impact of Listeriosis on Anthrax.** In this section, We analysed the impact of Listeriosis on Anthrax and the vice versa. This was done by expressing the reproduction number of one in terms of the other. By expressing the basic reproduction number of Listeriosis on Anthrax, that is expressing  $\mathfrak{R}_l$  in terms of  $\mathfrak{R}_a$ ;

$$\text{From; } \mathfrak{R}_a = \sqrt{\frac{\Omega_h \Omega_v \beta_h \beta_v}{\mu_h \mu_v^2 (\alpha + \mu_h + \phi)}}$$

Solving for  $\mu_h$  in the above,

$$(31) \quad \mu_h = \frac{-G_1 \mathfrak{R}_a + \sqrt{G_1^2 \mathfrak{R}_a^2 + 4G_2}}{2\mu_v \mathfrak{R}_a}$$

where,

$$G_1 = \mu_v (\alpha + \phi) \text{ and } G_2 = \Omega_h \Omega_v \beta_h \beta_v$$

Given;

$$\sqrt{G_1^2 \mathfrak{R}_a^2 + 4G_2} = G_3 \mathfrak{R}_a + G_4,$$

Implies;

$$(32) \quad \mu_h = \frac{\mathfrak{R}_a (G_3 - G_1) + G_4}{2\mu_v \mathfrak{R}_a}$$

By substituting  $\mu_h$  into  $\mathfrak{R}_l$ ;

$$\mathfrak{R}_l = \frac{\mathfrak{R}_{0l} (G_4 + (G_3 - G_1) \mathfrak{R}_a + 2(\sigma + \eta + \theta) \mu_v \mathfrak{R}_a + \theta (G_4 + (G_3 - G_1) \mathfrak{R}_a + 2(m + \delta) \mu_v \mathfrak{R}_a))}{G_4 + (G_3 - G_1) \mathfrak{R}_a + 2(\sigma + \eta + \theta) \mu_v \mathfrak{R}_a}$$

where  $\mathfrak{R}_{0l}$  is given the relation;

$$\mathfrak{R}_{0l} = \frac{v\rho\Omega_h}{\mu_b\mu_h K (\delta + \mu_h + m)}.$$

Now, taking the partial derivative of  $\mathfrak{R}_l$  with respect to  $\mathfrak{R}_a$ ;

$$(33) \quad \frac{\partial \mathfrak{R}_l}{\partial \mathfrak{R}_a} = \frac{2G_4\theta(m + \delta - (\sigma + \eta + \theta))\mu_v\mathfrak{R}_{0l}}{[G_4 + (G_3 - G_1 + 2(\sigma + \eta + \theta)\mu_v\mathfrak{R}_a)]^2}.$$

If  $(m + \delta) \geq (\sigma + \eta + \theta)$ , the derivative  $\left(\frac{\partial \mathfrak{R}_l}{\partial \mathfrak{R}_a}\right)$ , is strictly positive. Two scenarios can be deduced from the derivative  $\left(\frac{\partial \mathfrak{R}_l}{\partial \mathfrak{R}_a}\right)$ , depending on the values of the parameters;

$$\frac{\partial \mathfrak{R}_l}{\partial \mathfrak{R}_a} = 0, \text{ and } \frac{\partial \mathfrak{R}_l}{\partial \mathfrak{R}_a} \geq 0$$

- (1) If  $\frac{\partial \mathfrak{R}_l}{\partial \mathfrak{R}_a} = 0$ , it implies that  $(m + \delta) = (\sigma + \eta + \theta)$  and the biological implications is that Anthrax has no significance effect on the spread of Listeriosis.
- (2) If  $\frac{\partial \mathfrak{R}_l}{\partial \mathfrak{R}_a} > 0$ , it implies that  $(m + \delta) \geq (\sigma + \eta + \theta)$ , and the biological implications is that an increase in Anthrax cases would result in an increase Listeriosis cases in the environment. That is Anthrax enhances Listeriosis infections in the environment.

However, by expressing the basic reproduction number of Anthrax on Listeriosis, that is expressing  $\mathfrak{R}_a$  in terms of  $\mathfrak{R}_l$ ;

$$(34) \quad \mu_h = \frac{H_1 - H_2\mathfrak{R}_l + \sqrt{H_3\mathfrak{R}_l^2 + H_4\mathfrak{R}_l + H_5}}{2\mathfrak{R}_l}.$$

where;

$$H_1 = (1 + \theta)\mathfrak{R}_{0l}, H_2 = (m + \delta + \sigma + \eta + \theta)$$

$$H_3 = (\sigma + \eta + \theta - m - \delta), H_4 = 2(\theta - 1)(m + \delta - \sigma - \eta - \theta)\mathfrak{R}_{0l}$$

$$H_5 = (1 + \theta)^2\mathfrak{R}_{0l}^2.$$

By letting,

$$\sqrt{H_3\mathfrak{R}_l^2 + H_4\mathfrak{R}_l + H_5} = H_6\mathfrak{R}_l + H_7.$$

It implies that;

$$(35) \quad \mu_h = \frac{(H_6 - H_2)\mathfrak{R}_l + H_7 + H_1}{2\mathfrak{R}_l}$$

Therefore,

$$(36) \quad \mathfrak{R}_a^2 = \frac{4\Omega_h\Omega_v\beta_h\beta_v\mathfrak{R}_l^2}{[(H_6 - H_2)\mathfrak{R}_l + H_7 + H_1][H_7 + H_1 + 2(\alpha + \phi)\mathfrak{R}_l + (H_6 - H_2)\mathfrak{R}_l]\mu_v}$$

Now, taking the partial derivative of  $\mathfrak{R}_a$  with respect to  $\mathfrak{R}_l$  in equation (36), gives;

$$(37) \quad \frac{\partial \mathfrak{R}_a}{\partial \mathfrak{R}_l} = \frac{4(H_7 + H_1)[H_7 + H_1 + (\alpha + \phi + H_6 - H_2)\mathfrak{R}_l]\Omega_h\Omega_v\beta_h\beta_v\mathfrak{R}_l}{[(H_6 - H_2)\mathfrak{R}_l + H_7 + H_1]^2[H_7 + H_1 + (2(\alpha + \phi) + H_6 - H_2)\mathfrak{R}_l]^2\mu_v}$$

If the partial derivative of  $\mathfrak{R}_a$  with respect to  $\mathfrak{R}_l$  is greater than zero,  $\left(\frac{\partial \mathfrak{R}_a}{\partial \mathfrak{R}_l} > 0\right)$ , the biological implication is that an increase in the number of cases of Listeriosis would result in an increase in the number of cases of Anthrax in the environment. Moreover, the the impact of Anthrax treatment on Listeriosis can also be analysed by taking the partial derivative of  $\mathfrak{R}_a$  with respect to  $\alpha$ ,  $\left(\frac{\partial \mathfrak{R}_a}{\partial \alpha}\right)$ .

$$(38) \quad \frac{\partial \mathfrak{R}_a}{\partial \alpha} = -\frac{\alpha}{\alpha + \phi + \mu_h}$$

Clearly,  $\mathfrak{R}_a$  is a decreasing function of  $\alpha$ , the epidemiological implication is that the treatment of Listeriosis would have an impact on the transmission dynamics of Anthrax.

**5.4. Analysis of backward bifurcation.** In this section, the phenomenon of backward bifurcation was carried out by aplying the centre manifold theory on the sysyem of equations in (3) as outlined in [25]. Considering  $\beta_h$  and  $v$  as bifurcation parameters, it implies that  $\mathfrak{R}_a = 1$  and  $\mathfrak{R}_l = 1$  if and only if,

$$(39) \quad \beta_h = \beta_h^* = \frac{\mu_h\mu_v^2(\alpha + \phi + \mu_h)}{\Omega_h\Omega_v\beta_v}$$

and

$$(40) \quad v = v^* = \frac{\mu_b\mu_h K(\delta + \mu_h + m)(\sigma + \mu_h + \eta + \theta)}{\rho\Omega_h(\sigma + \mu_h + \eta + \theta + \theta(m + \delta + \mu_h))}$$

Considering the following change of variables;

$$S_h = x_1, I_a = x_2, I_l = x_3, I_{al} = x_4, R_a = x_5,$$

$$R_l = x_6, R_{dl} = x_7, C_p = x_8, S_v = x_9, I_v = x_{10}.$$

This would give the total population as;

$$N = x_1 + x_2 + x_3 + x_4 + x_5 + x_6 + x_7 + x_8 + x_9 + x_{10}.$$

By applying vector notation;

$$X = (x_1, x_2, x_3, x_4, x_5, x_6, x_7, x_8, x_9, x_{10})^T.$$

The Anthrax-Listeriosis co-infection model can be expressed as;

$$(41) \quad \frac{dX}{dt} = F(X),$$

where

$$F = (f_1, f_2, f_3, f_4, f_5, f_6, f_7, f_8, f_9, f_{10})^T.$$

The following system of equation is obtained;

$$(42) \quad \left. \begin{aligned} \frac{dx_1}{dt} &= \Omega_h + kx_5 + \omega x_6 + \psi x_7 - \beta_h x_{10} x_1 - \pi x_1 - \mu_h x_1 \\ \frac{dx_2}{dt} &= \beta_h x_{10} x_1 - \pi x_2 - (\alpha + \mu_h + \phi) x_2 \\ \frac{dx_3}{dt} &= \pi x_1 - \beta_l x_{10} x_3 - (\delta + \mu_h + m + \rho) x_3 \\ \frac{dx_4}{dt} &= \beta_l x_{10} x_3 + \pi x_2 + (\sigma + \mu_h + \eta + \theta) x_4 \\ \frac{dx_5}{dt} &= \alpha x_2 - (k + \mu_h) x_5 + (1 - \tau) \gamma \sigma x_4 \\ \frac{dx_6}{dt} &= \delta x_3 - (\omega + \mu_h) x_6 + (1 - \tau) (1 - \gamma) \sigma x_4 \\ \frac{dx_7}{dt} &= \tau \sigma x_4 - (\psi + \mu_h) x_7 \\ \frac{dx_8}{dt} &= \rho x_3 + \theta x_4 - \mu_b x_8 \\ \frac{dx_9}{dt} &= \Omega_v - \beta_v (x_2 + x_4) x_9 - \mu_v x_9 \\ \frac{dx_{10}}{dt} &= \beta_v (x_2 + x_4) x_9 - \mu_v x_{10} \end{aligned} \right\}$$

Backward bifurcation was carried out by employing the centre manifold theory on the system of equations in (3). This concept involves computation of the Jacobian of the system of equations in (42) at DFE. The Jacobian matrix at DFE is given by;

$$(43) \quad J(\xi_0) = \begin{bmatrix} -\mu_h & 0 & 0 & J_1 & k & \omega & \psi & J_2 & 0 & J_3 \\ 0 & -J_4 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & J_3 \\ 0 & 0 & -J_5 & J_1 & 0 & 0 & 0 & J_2 & 0 & 0 \\ 0 & 0 & 0 & -J_6 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \alpha & 0 & J_7 & -J_8 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \delta & J_9 & 0 & -J_{10} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \sigma & 0 & 0 & -J_{11} & 0 & 0 & 0 \\ 0 & 0 & \rho & \theta & 0 & 0 & 0 & -\mu_b & 0 & 0 \\ 0 & -J_{12} & 0 & -J_{12} & 0 & 0 & 0 & 0 & -\mu_v & 0 \\ 0 & J_{12} & 0 & J_{12} & 0 & 0 & 0 & 0 & 0 & -\mu_v \end{bmatrix}$$

where;  $J_1 = \frac{\rho\Omega_h}{\mu_h}$ ,  $J_2 = \frac{\mu_b(\delta + \mu_h + m)(\sigma + \mu_h + \eta + \theta)}{\rho(\sigma + \mu_h + \eta + \theta + \theta(\delta + \mu_h + m))}$ ,  
 $J_3 = \frac{\mu_v^3(\alpha + \phi + \mu_h)}{\Omega_v\beta_v}$ ,  $J_4 = (\alpha + \phi + \mu_h)$ ,  
 $J_5 = (\delta + \mu_h + m)$ ,  $J_6 = (\sigma + \mu_h + \eta + \theta)$ ,  $J_7 = (1 - \tau)\gamma\sigma$ ,  
 $J_8 = (k + \mu_h)$ ,  $J_9 = (1 - \tau)(1 - \gamma)\sigma$ ,  $J_{10} = (\omega + \mu_h)$ ,  
 $J_{11} = (\psi + \mu_h)$  and  $J_{12} = \frac{\Omega_v\beta_v}{\mu_v}$ .

Clearly, the Jacobian matrix at DFE has a case of simple zero eigenvalue as well as other eigenvalues with negative real parts. This is a clear indication that the centre manifold theorem is applicable. By applying the centre manifold theorem in [22, 25], the left and right eigenvectors of the Jacobian matrix  $J(\xi_0)$  is computed first. Letting the left and right eigenvector represented by:

$$y = \begin{bmatrix} y_1, & y_2, & y_3, & y_4, & y_5, & y_6, & y_7, & y_8, & y_9, & y_{10} \end{bmatrix} \text{ and} \\ w = \begin{bmatrix} w_1, & w_2, & w_3, & w_4, & w_5, & w_6, & w_7, & w_8, & w_9, & w_{10} \end{bmatrix}^T \text{ respectively.}$$

The following were obtained;

$$w_1 = \frac{Kw_5}{\mu_h} + \frac{w_2\mu_v^2(\alpha + \phi + \mu_h)}{\mu_h}, \quad w_2 = \frac{\mu_v^2}{\Omega_v\beta_v}, \\ w_3 = w_4 = w_6 = w_7 = w_8 = 0, \quad w_5 = \frac{\alpha\mu_v^2}{\Omega_v\beta_v(k + \mu_h)}, \\ w_9 = -w_{10}, \quad w_{10} = 1.$$

and



$$y_1 = y_3 = y_5 = y_6 = y_7 = y_8 = y_9 = 0, y_2 = \frac{v_{10}\Omega_v\beta_v}{\mu_v(\alpha + \phi + \mu_h)},$$

$$y_2 = y_4, y_{10} = \frac{-\mu_v(\sigma + \mu_h + \eta + \theta)}{\Omega_v\beta_v}.$$

By further simplifications, it can be shown that;

$$a = \frac{\tau w_{10}\mu_v^3(\sigma + \mu_h + \eta + \theta)}{\Omega_v\beta_v} - 2w_{10}\beta_v \left[ \frac{\mu_v^2(\sigma + \mu_h + \eta + \theta)}{\mu_h\Omega_v\beta_v} + \frac{\alpha K\mu_v^2(\sigma + \mu_h + \eta + \theta)}{\mu_h\Omega_v\beta_v(k + \mu_h)(\alpha + \phi + \mu_h)} \right]$$

and

$$b = y_2 w_{10} \frac{\Omega_h}{\mu_h} > 0.$$

It can be deduced that the coefficient  $b$  would always be positive. Backward bifurcation will take place in the system of equations in (3) if the coefficient  $a$  is positive. In conclusion, the DFE is not globally stable.

This phenomenon only exists in situations where DFE and EE coexists. Epidemiological implication is that the idea that whenever  $\mathfrak{R}_0 < 1$ , the disease can be controlled is no longer a sufficient condition.

## 6. SENSITIVITY ANALYSIS OF THE CO-INFECTION MODEL

We performed sensitivity analysis of  $\mathfrak{R}_{al}$  of the co-dynamics model. This is to determine the significance each parameter on  $\mathfrak{R}_{al}$  [26, 27]. The sensitivity index of  $\mathfrak{R}_0$  to a parameter  $x$  is given by the relation:

$$(44) \quad \Pi_x^{\mathfrak{R}_0} = \left( \frac{\partial \mathfrak{R}_0}{\partial x} \right) \left( \frac{x}{\mathfrak{R}_0} \right)$$

The sensitivity analysis of  $\mathfrak{R}_{0a}$  and  $\mathfrak{R}_{0l}$  were determined separately, since the basic reproduction number of the co-infection model is usually;

$$(45) \quad \mathfrak{R}_0 = \max\{ \mathfrak{R}_{0a}, \mathfrak{R}_{0l} \}$$

**6.1. Sensitivity indices of  $\mathfrak{R}_{0a}$ .** In this section, we derive the sensitivity of  $\mathfrak{R}_{0a}$  to each of the parameters. Detailed sensitivity indices of  $\mathfrak{R}_{0a}$  are shown in Table 3. The values in Table 3, showed that the most sensitive parameters are human recruitment rate, vector recruitment rate, human transmission rate and vector transmission rate. In creasing or decreasing the human

recruitment rate by 10% would increase or decrease  $\mathfrak{R}_{0a}$  by 12.164%. However, increasing or decreasing human and vector transmission rates by 10% would increase or decrease  $\mathfrak{R}_{0a}$  by 1.216% and 0.243% respectively.

Parameter	Description	Sensitivity Index
$\Omega_h$	Human recruitment rate	1.2164
$\Omega_v$	Vector recruitment rate	0.2433
$\beta_h$	Human transmission rate	0.1216
$\beta_v$	Vector transmission rate	0.0243
$\alpha$	Anthrax recovery rate	-0.0037
$\mu_h$	Human natural death rate	-0.0122
$\mu_v$	Vector natural death rate	-0.0061
$\phi$	Anthrax related death rate	-0.0065
$\theta$	Modification parameter	$3.42913 * 10^{-6}$

TABLE 3. Sensitivity indices of  $\mathfrak{R}_{0a}$  to each of the parameter values.

**6.2. Sensitivity indices of  $\mathfrak{R}_{0l}$ .** In this section, we derive the sensitivity of  $\mathfrak{R}_{0l}$  to each of the parameters. Detailed sensitivity indices of  $\mathfrak{R}_{0l}$  are shown in Table 4. The values in Table 4 showed that the most sensitive parameters are the human recruitment rate, Listeriosis contribution to environment, bacteria ingestion rate and Listeriosis related death. Increasing or decreasing the human recruitment rate by 10% would increase or decrease  $\mathfrak{R}_{0l}$  by 0.201487%. Moreover, increasing or decreasing Listeriosis contribution to environment and bacteria ingestion rate by 10% would increase or decrease  $\mathfrak{R}_{0l}$ .

Parameter	Description	Sensitivity Index
$\Omega_h$	Human recruitment rate	0.0201487
$\sigma$	Co-infected human recovery rate	$-5.41441 * 10^{-6}$
$\mu_h$	Human natural death rate	-0.00014638
$\eta$	Listeriosis death rate among co-infected	$-5.41441 * 10^{-6}$
$\theta$	Modification parameter	$3.42913 * 10^{-6}$
$\nu$	Bacteria ingestion rate	0.0000402975
$\rho$	Listeriosis contribution to environment	0.0000309981
$K$	Concentration of carcasses	$-2.01487 * 10^{-10}$
$\delta$	Listeriosis recovery rate	-0.0000402218
$\mu_b$	Carcasses mortality rate	-0.0080595
$m$	Listeriosis related death	-0.0000402218

TABLE 4. Sensitivity indices of  $\mathfrak{R}_{0l}$  to each of the parameter values.

## 7. EXTENSION OF THE MODEL TO OPTIMAL CONTROL

In this section, we extended model analysis to optimal control. This was carried out to determine the impact of intervention schemes. The optimal control problem is derived by incorporating the following control functions into the Anthrax-Listeriosis co-infection model in Figure 1 and the introduction of an objective functional that seeks to minimise:  $(u_1, u_2, u_3, u_4, u_5)$ . The controls  $u_1(t)$  and  $u_2(t)$  denotes the efforts on preventing Anthrax and Listeriosis respectively. The controls  $u_3(t)$  and  $u_4(t)$  denotes the treatment of Anthrax and Listeriosis infected persons respectively. Moreover,  $u_3(t)$  satisfies  $0 \leq u_3 \leq f_3$  and  $u_4(t)$  satisfies  $0 \leq u_4 \leq f_4$  where  $f_3$  and  $f_4$  denote drug efficacies use in the treatment of Anthrax and Listeriosis infections respectively. Also,  $u_5(t)$  is the treatment control on co-infected and it satisfies  $0 \leq u_5 \leq f_5$ . Where  $f_5$  is the drug efficacy use in the treatment of Anthrax-Listeriosis co-infected. The following system of equations are obtained as a result of incorporating the controls in the model;

$$(46) \quad \left. \begin{aligned} \frac{dS_h}{dt} &= \Omega_h + kR_a + \omega R_l + \psi R_{al} - (1 - u_1) \beta_h I_v S_h - (1 - u_2) \pi S_h - \mu_h S_h \\ \frac{dI_a}{dt} &= (1 - u_1) \beta_h I_v S_h - (1 - u_2) \pi I_a - (u_3 \alpha + \mu_h + \phi) I_a \\ \frac{dI_l}{dt} &= (1 - u_2) \pi S_h - (1 - u_1) \beta_l I_v I_l - (u_4 \delta + \mu_h + m + \rho) I_l \\ \frac{dI_{al}}{dt} &= (1 - u_1) \beta_h I_v I_l + (1 - u_2) \pi I_a + (u_5 \sigma + \mu_h + \eta + \theta) I_{al} \\ \frac{dR_a}{dt} &= u_3 \alpha I_a - (k + \mu_h) R_a + (1 - \tau) \gamma u_5 \sigma I_{al} \\ \frac{dR_l}{dt} &= u_4 \delta I_l - (\omega + \mu_h) R_l + (1 - \tau) (1 - \gamma) u_5 \sigma I_{al} \\ \frac{dR_{al}}{dt} &= \tau u_5 \sigma I_{al} - (\psi + \mu_h) R_{al} \\ \frac{dC_p}{dt} &= \rho I_l + \theta I_{al} - \mu_b C_p \\ \frac{dS_v}{dt} &= \Omega_v - (1 - u_1) \beta_v (I_a + I_{al}) S_v - \mu_v S_v \\ \frac{dI_v}{dt} &= (1 - u_1) \beta_v (I_a + I_{al}) S_v - \mu_v I_v \end{aligned} \right\}$$

In biological modelling, the objective of optimal control is to minimise spread or infections, cost of treatment and cost of prevention. The objective functional that can be used to achieve this is given by:

$$(47) \quad J(u_1, u_2, u_3, u_4, u_5) = \int_0^{t_f} (A_1 I_a + A_2 I_l + A_3 I_{al} + A_4 I_v + A_5 u_1^2 + A_6 u_2^2 + A_7 u_3^2 + A_8 u_4^2 + A_9 u_5^2) dt.$$

subject to the system of equations in (46).

Where,  $A_1, A_2, A_3, A_4, A_5, A_6, A_7, A_8, A_9$  referred to as the weight constants to aid balance the terms in the integral in order to avoid the dominance. They are termed as the balancing cost factors.  $A_1 I_a, A_2 I_l$  are the costs associated with infected persons with Anthrax and Listeriosis respectively.  $A_3 I_{al}, A_4 I_v$  are the cost associated with co-infected persons and infected vectors respectively.  $A_5 u_1^2, A_6 u_2^2$  are the cost associated with efforts of prevention of Anthrax and Listeriosis respectively.  $A_7 u_3^2, A_8 u_4^2$  are the cost associated with the treatment of Anthrax and Listeriosis infected persons respectively.  $A_9 u_5^2$  is the cost associated with the treatment of infected with Anthrax-Listeriosis simultaneously.

Where  $t_f$  is the final period of the intervention. This implies that  $(A_1 I_a, A_2 I_l, A_3 I_{al}, A_4 I_v)$ , represents a linear function for the cost associated with infections

and  $(A_5u_1^2, A_6u_2^2, A_7u_3^2, Au_4^2, A_9u_5^2)$ , represents a quadratic function for the cost associated with preventions and treatments.

The model control efforts is by linear combination of  $u_i^2(t)$ ,  $(i = 1, 2)$ . The quadratic in nature of the control efforts are as a result of the assumption that costs are generally non-linear in nature. Thus, our aim is to minimise the number of infectives and reduce cost of treatment. The objective is finding the optimal functions  $(u_1^*(t), u_2^*(t), u_3^*(t), u_4^*(t))$ , such that;

$$(48) \quad J(u_1^*, u_2^*, u_3^*, u_4^*, u_5^*) = \{J(u_1, u_2, u_3, u_4, u_5) \mid u_1, u_2, u_3, u_4, u_5 \in \cup\}$$

where,  $\cup = \{(u_1, u_2, u_3, u_4, u_5) : u_1, u_2, u_3, u_4, u_5 \text{ measurable},$

$$(0 \leq u_1 \leq 1, 0 \leq u_2 \leq 1, 0 \leq u_3 \leq f_2, 0 \leq u_4 \leq f_3, 0 \leq u_5 \leq f_5 \forall t \in [0, t_f])\}$$

are the control set.

**7.1. Pontryagin's Maximum Principle.** The Pontryagin's Maximum Principle provides the necessary conditions that an optimal must satisfy [28]. The principle changes the system of equations in (46) and (47) into minimisation problem point-wise Hamiltonian ( $H$ ), with respect to;

$$(u_1, u_2, u_3, u_4, u_5).$$

$$(49) \quad \left. \begin{aligned} H = & A_1I_a + A_2I_l + A_3I_{al} + A_4I_v + A_5u_1^2 + A_6u_2^2 + A_7u_3^2 + A_8u_4^2 + A_9u_5^2 \\ & + \lambda_1 \{ \Omega_h + kR_a + \omega R_l + \psi R_{al} - (1 - u_1) \beta_h I_v S_h - (1 - u_2) \pi S_h - \mu_h S_h \} \\ & + \lambda_2 \{ (1 - u_1) \beta_h I_v S_h - (1 - u_2) \pi I_a - (u_3 \alpha + \mu_h + \phi) I_a \} \\ & + \lambda_3 \{ (1 - u_2) \pi S_h - (1 - u_1) \beta_l I_v I_l - (u_4 \delta + \mu_h + m + \rho) I_l \} \\ & + \lambda_4 \{ (1 - u_1) \beta_h I_v I_l + (1 - u_2) \pi I_a + (u_5 \sigma + \mu_h + \eta + \theta) I_{al} \} \\ & + \lambda_5 \{ u_3 \alpha I_a - (k + \mu_h) R_a + (1 - \tau) \gamma u_5 \sigma I_{al} \} \\ & + \lambda_6 \{ u_4 \delta I_l - (\omega + \mu_h) R_l + (1 - \tau) (1 - \gamma) u_5 \sigma I_{al} \} \\ & + \lambda_7 \{ \tau u_5 \sigma I_{al} - (\psi + \mu_h) R_{al} \} \\ & + \lambda_8 \{ \rho I_l + \theta I_{al} - \mu_b C_p \} \\ & + \lambda_9 \{ \Omega_v - (1 - u_1) \beta_v (I_a + I_{al}) S_v - \mu_v S_v \} \\ & + \lambda_{10} \{ (1 - u_1) \beta_v (I_a + I_{al}) S_v - \mu_v I_v \} \end{aligned} \right\}$$

where  $\lambda_1, \lambda_2, \lambda_3, \lambda_4, \lambda_5, \lambda_6, \lambda_7, \lambda_8, \lambda_9, \lambda_{10}$  are referred to as the co-state variables (adjoint variables).

**Theorem 4.** Given optimal controls  $(u_1^*, u_2^*, u_3^*, u_4^*, u_5^*)$  and solutions

$S_h, I_a, I_l, I_{al}, R_a, R_l, R_{al}, C_p, S_v, I_v$  of the corresponding state systems

(46) and (47) that minimise the objective functional  $J(u_1, u_2, u_3, u_4, u_5)$

over  $\cup$ . Then there exists adjoint variables

$\lambda_1, \lambda_2, \lambda_3, \lambda_4, \lambda_5, \lambda_6, \lambda_7, \lambda_8, \lambda_9, \lambda_{10}$  satisfying;

$$(50) \quad \frac{d\lambda_i}{dt} = - \frac{\partial H}{\partial x(t)}$$

where

$i = 1, 2, 3, 4, 5, 6, 7, 8, 8, 9, 10$

and

$\dot{x} = S_h, I_a, I_l, I_{al}, R_a, R_l, R_{al}, C_p, S_v, I_v$

with transversality conditions,

$$\lambda_1(tf) = \lambda_2(tf) = \lambda_3(tf) = \lambda_4(tf) = \lambda_5(tf) = \lambda_6(tf) = \lambda_7(tf) = \lambda_8(tf) = \lambda_9(tf) = \lambda_{10}(tf) = 0,$$

and ;

$$(51) \quad \left. \begin{aligned} u_1^* &= \min \left\{ 1, \max \left( 0, \frac{\beta_h I_v S_h (\lambda_2 - \lambda_1) + \beta_l I_v I_l (\lambda_4 - \lambda_3) + \beta_v S_v (I_a + I_{al}) (\lambda_{10} - \lambda_9)}{2A_5} \right) \right\} \\ u_2^* &= \min \left\{ 1, \max \left( 0, \frac{\pi S_h (\lambda_3 - \lambda_1) + \pi I_a (\lambda_4 - \lambda_2) + \rho (I_l + \theta I_{al}) \lambda_8}{2A_6} \right) \right\} \\ u_3^* &= \min \left\{ 1, \max \left( 0, \frac{\alpha I_a (\lambda_2 - \lambda_5)}{2A_7} \right) \right\} \\ u_4^* &= \min \left\{ 1, \max \left( 0, \frac{\delta I_l (\lambda_3 - \lambda_6)}{2A_8} \right) \right\} \\ u_5^* &= \min \left\{ 1, \max \left( 0, \frac{\sigma I_{al} [\lambda_4 + (1 - \tau) \gamma \lambda_5 + (1 - \tau) (1 - \gamma) \lambda_6 - \tau \lambda_7]}{2A_9} \right) \right\} \end{aligned} \right\}$$

*Proof.* There exists an optimal control due to the convexity of the integrand of the objective functional  $J$  with respect to  $u_1, u_2, u_3, u_4, u_5$ , a priori boundedness of the state solutions and the Lipschitz property of the state system with respect to the state variables [28]. The differential equations governing the adjoint variables are obtained by differentiating the Hamiltonian function, evaluated at the optimal control.

From the relation;

$$(52) \quad \frac{d\lambda_i}{dt} = -\frac{\partial H}{\partial \dot{x}(t)}$$

where  $i = 1, 2, 3, 4, 5, 6, 7, 8, 8, 9, 10$  and  $\dot{x} = S_h, I_a, I_l, I_{al}, R_a, R_l, R_{al}, C_p, S_v, I_v$ . The system of equations obtained as a result of taking the partial derivatives of the Hamiltonian with respect to the associated state variables are the solutions of the adjoint systems. The following adjoint or co-state variables are solutions of adjoint systems obtained below;

$$(53) \quad \left. \begin{aligned} -\frac{d\lambda_1}{dt} &= \mu_h \lambda_1 + (1 - u_1) \beta_h I_v (\lambda_1 - \lambda_2) - (1 - u_2) \left( \frac{C_p v}{K + C_p} \right) (\lambda_1 - \lambda_3) \\ -\frac{d\lambda_2}{dt} &= -A_1 - u_3 \alpha \lambda_5 + (1 - u_2) \left( \frac{C_p v}{K + C_p} \right) (\lambda_2 - \lambda_4) + (-u\alpha + \mu_h + \phi) \lambda_2 \\ &\quad + (1 - u_1) \beta_v S_v (\lambda_9 - \lambda_{10}) \\ -\frac{d\lambda_3}{dt} &= -A_2 + (1 - u_1) \beta_v I_v (\lambda_3 - \lambda_4) - u_4 \delta \lambda_6 - (1 - u_2) \rho \lambda_8 + (u_4 \delta + \mu_h + m) \lambda_3 \\ -\frac{d\lambda_4}{dt} &= -A_3 + (u_5 \sigma + \mu_h + \eta + \theta) \lambda_4 - (1 - \tau) \gamma u_5 \sigma \lambda_5 + (1 - \tau) (1 - \gamma) u_5 \sigma \lambda_6 \\ &\quad + \tau u_5 \sigma \lambda_7 + \theta \lambda_8 + (1 - u_1) \beta_v S_v (\lambda_{10} - \lambda_9) \\ -\frac{d\lambda_5}{dt} &= -k \lambda_1 + (k + \mu_h) \lambda_5 \\ -\frac{d\lambda_6}{dt} &= -\omega \lambda_1 + (\omega + \mu_h) \lambda_6 \\ -\frac{d\lambda_7}{dt} &= -\psi \lambda_1 + (\psi + \mu_h) \lambda_7 \\ -\frac{d\lambda_8}{dt} &= (1 - u_2) S_h \frac{Kv}{(K + C_p)^2} (\lambda_2 - \lambda_3) + (1 - u_2) I_a \frac{Kv}{(K + C_p)^2} (\lambda_2 - \lambda_4) + \mu_b \lambda_8 \\ -\frac{d\lambda_9}{dt} &= (1 - u_1) \beta_v (I_a + I_{al}) (\lambda_9 - \lambda_{10}) - \mu_v \lambda_9 \\ -\frac{d\lambda_{10}}{dt} &= -A_4 + (1 - u_1) \beta_h S_h (\lambda_1 - \lambda_2) + (1 - u_1) \beta_h I_l (\lambda_3 - \lambda_4) \end{aligned} \right\}$$

The system of equations in (7.7) satisfies the transversality conditions;  $\lambda_1(tf) = \lambda_2(tf) = \lambda_3(tf) = \lambda_4(tf) = \lambda_5(tf) = \lambda_6(tf) = \lambda_7(tf) = \lambda_8(tf) = \lambda_9(tf) = \lambda_{10}(tf) = 0$ .

Now, combining the Pontryagin's Maximum Principle and the existence result of the optimal control [29, 28].

Moreover, the characterisation of the optimal control is obtained by solving the partial derivative of the Hamiltonian function with respect to the control sets and equating the derivatives to zero.

$$(54) \quad \frac{\partial H}{\partial u_i} = 0$$

where;  $u_i = u_i^*$ , and  $i = 1, 2, 3, \dots, n$ . The following are obtained;

$$(55) \quad \left. \begin{aligned} \frac{\partial H}{\partial u_1} = 0 &= 2A_5 u_1 + \beta_h I_v S_h (\lambda_1 - \lambda_2) + \beta_l I_v I_l (\lambda_4 - \lambda_3) + \beta_v S_v (I_a + I_{al}) (\lambda_9 - \lambda_{10}) \\ \frac{\partial H}{\partial u_2} = 0 &= 2A_6 u_2 + \pi S_h (\lambda_1 - \lambda_3) + \pi I_a (\lambda_2 - \lambda_4) - \rho (I_l + \theta I_{al}) \lambda_8 \\ \frac{\partial H}{\partial u_3} = 0 &= 2A_7 u_3 + \alpha I_a (\lambda_5 - \lambda_2) \\ \frac{\partial H}{\partial u_4} = 0 &= 2A_8 u_4 + \delta I_l (\lambda_6 - \lambda_3) \\ \frac{\partial H}{\partial u_5} = 0 &= 2A_9 u_5 + \sigma I_{al} [\lambda_4 + (1 - \tau) \gamma \lambda_5 + (1 - \tau) (1 - \gamma) \lambda_6 + \tau \lambda_7] \end{aligned} \right\}$$

By re-arranging and simplification;

$$(56) \quad \left. \begin{aligned} u_1^* &= \frac{\beta_h I_v S_h (\lambda_2 - \lambda_1) + \beta_l I_v I_l (\lambda_4 - \lambda_3) + \beta_v S_v (I_a + I_{al}) (\lambda_{10} - \lambda_9)}{2A_5} \\ u_2^* &= \frac{\pi S_h (\lambda_3 - \lambda_1) + \pi I_a (\lambda_4 - \lambda_2) + \rho (I_l + \theta I_{al}) \lambda_8}{2A_6} \\ u_3^* &= \frac{\alpha I_a (\lambda_2 - \lambda_5)}{2A_7} \\ u_4^* &= \frac{\delta I_l (\lambda_3 - \lambda_6)}{2A_8} \\ u_5^* &= \frac{\sigma I_{al} [\lambda_4 + (1 - \tau) \gamma \lambda_5 + (1 - \tau) (1 - \gamma) \lambda_6 - \tau \lambda_7]}{2A_9} \end{aligned} \right\}$$

By employing the phenomenon of standard control arguments involving the bounds on the controls, it can be concluded that;

$$(57) \quad u_i^* = \begin{cases} 0 & \text{if } \Phi_i^* \leq 0 \\ \Phi_i^* & \text{if } 0 < \Phi_i^* < 1 \\ 1 & \text{if } \Phi_i^* \geq 1 \end{cases}$$

for  $i = 1, 2, 3, 4, 5$

where;

$$(58) \quad \left. \begin{aligned} \Phi_1^* &= \frac{\beta_h I_v S_h (\lambda_2 - \lambda_1) + \beta_l I_v I_l (\lambda_4 - \lambda_3) + \beta_v S_v (I_a + I_{al}) (\lambda_{10} - \lambda_9)}{2A_5} \\ \Phi_2^* &= \frac{\pi S_h (\lambda_3 - \lambda_1) + \pi I_a (\lambda_4 - \lambda_2) + \rho (I_l + \theta I_{al}) \lambda_8}{2A_6} \\ \Phi_3^* &= \frac{\alpha I_a (\lambda_2 - \lambda_5)}{2A_7} \\ \Phi_4^* &= \frac{\delta I_l (\lambda_3 - \lambda_6)}{2A_8} \\ \Phi_5^* &= \frac{\sigma I_{al} [\lambda_4 + (1 - \tau) \gamma \lambda_5 + (1 - \tau) (1 - \gamma) \lambda_6 - \tau \lambda_7]}{2A_9} \end{aligned} \right\}$$



□

## 8. NUMERICAL RESULTS

Numerical solutions of the optimal system are illustrated using Runge-Kutta fourth order scheme. These were obtained by solving the state systems, adjoints equations and the transversality conditions. This is a two-point boundary-value problem with two boundary conditions at initial time,  $t = 0$  and final time,  $t = t_f$ . The objective is to solve this optimal problem at time,  $t_f = 120$  days. This period represents the time at which prevention and treatment strategies should end.

Following optimal control strategies were considered; prevention of Anthrax infection  $u_1$ , prevention of Listeriosis infection  $u_2$ , control efforts  $u_3$  and  $u_4$  on treatment of Anthrax and Listeriosis respectively. Control efforts  $u_5$  on the treatment of Anthrax-Listeriosis co-infection. The four most effective were selected.

The description of the variables and parameters used in the simulation of the co-dynamics model is shown in Table 2.

**8.1. Strategy 1: Prevention, ( $u_2$ ) and treatment, ( $u_4$ ) of Listeriosis.** Objective functional was optimised by using Listeriosis prevention control, ( $u_2$ ) and Listeriosis treatment control, ( $u_4$ ) and setting the Anthrax prevention control, ( $u_1$ ), Anthrax treatment control, ( $u_3$ ) and Anthrax-Listeriosis co-infection control, ( $u_5$ ) to zero. Figure 2: showed decrease in Listeriosis infected persons but not as much as Anthrax infected persons. However, Figure 3: showed reduction in Anthrax-Listeriosis co-infected individuals. Case without control is indicated with black line and case with control is indicated with blue line.

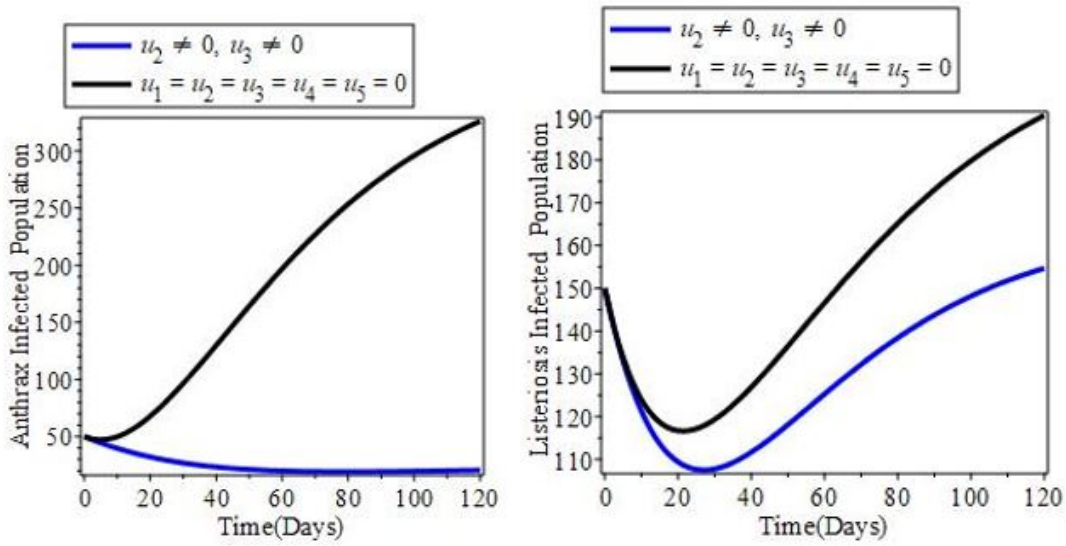


FIGURE 2. Effects of prevention and treatment on Anthrax and Listeriosis infected population.

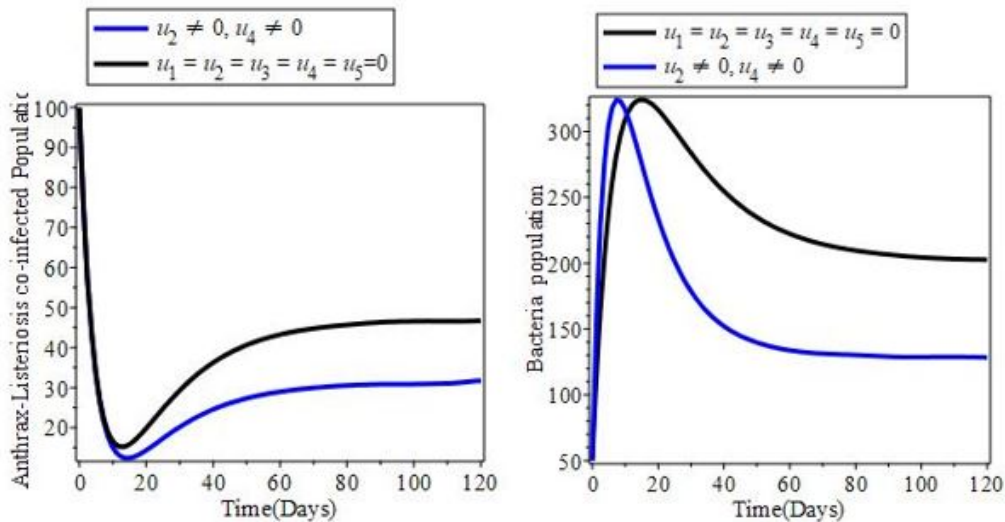


FIGURE 3. Effects of prevention and treatment on Anthrax-Listeriosis infected population and bacteria population.

**8.2. Strategy 2: Prevention of Anthrax, ( $u_1$ ) and Listeriosis, ( $u_2$ ).** Objective functional is optimised by using Anthrax prevention control, ( $u_1$ ) and Listeriosis prevention control, ( $u_2$ ) by setting the Anthrax treatment control, ( $u_3$ ), treatment control of Listeriosis, ( $u_4$ ) and treatment control of co-infected, ( $u_5$ ) to zero. Observations; Figure 4: showed reduction of Anthrax

infections and reduction of Listeriosis infection. Much reduction of Anthrax infections than Listeriosis infections. Figure 5: showed reduction in Anthrax-Listeriosis co-infection and reduction in bacteria population. Case without control is indicated with black line and case with control is indicated with blue line.

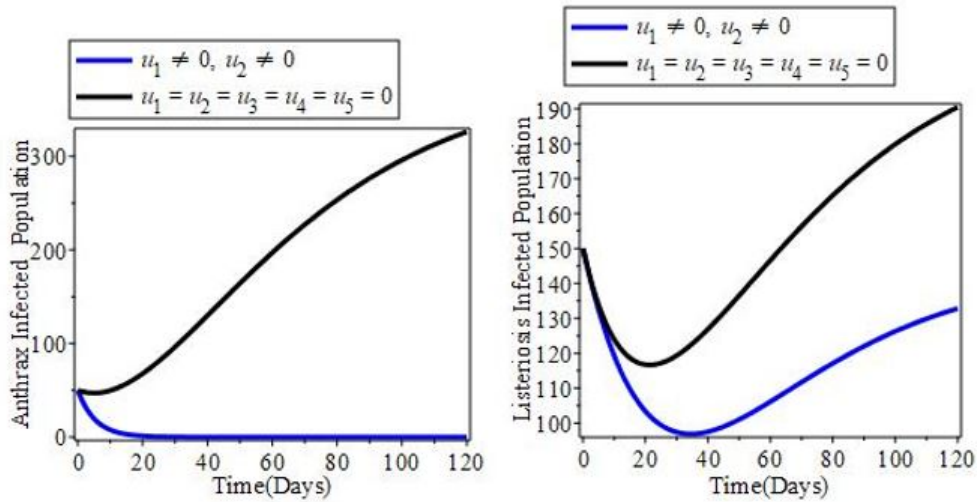


FIGURE 4. Effects of Anthrax and Listeriosis prevention on Anthrax and Listeriosis infected population.

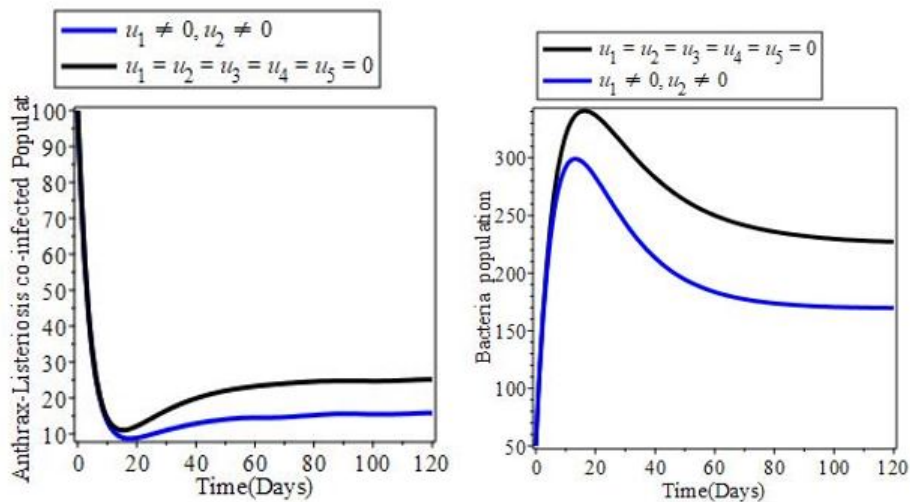


FIGURE 5. Effects of Anthrax and Listeriosis prevention on co-infected population and bacteria population.

**8.3. Prevention, ( $u_1$ ) and treatment, ( $u_3$ ) of Anthrax.** Objective functional was optimised by using anthrax prevention control, ( $u_1$ ) and anthrax treatment control, ( $u_3$ ) by setting Listeriosis prevention controls, ( $u_2$ ), treatment control of Listeriosis, ( $u_4$ ) and treatment control of co-infected, ( $u_5$ ) to zero. Figure 6: showed reduction of Anthrax infection and reduction in Listeriosis infections. Figure 7: showed reduction of Anthrax-Listeriosis co-infection and reduction of bacteria population responsible for the diseases in the environment. Case without control is indicated with black line and case with control is indicated with blue line.

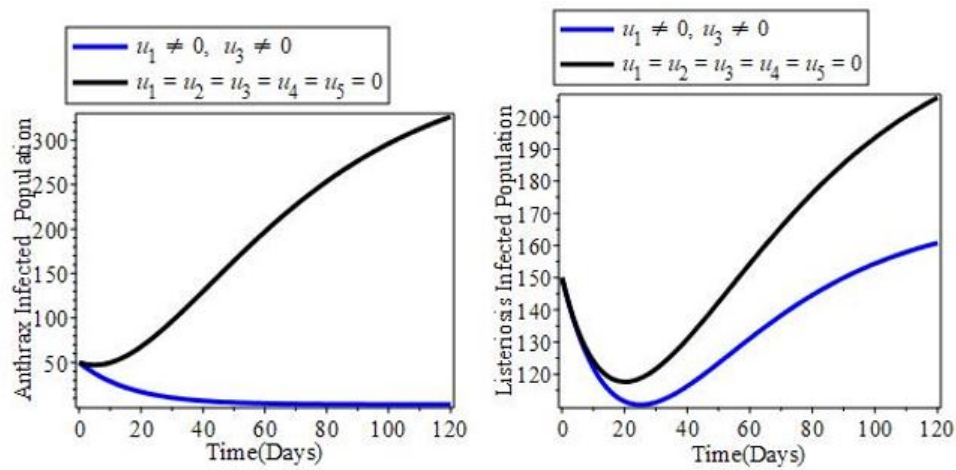


FIGURE 6. Effects of prevention and treatment on Anthrax and Listeriosis infected population.

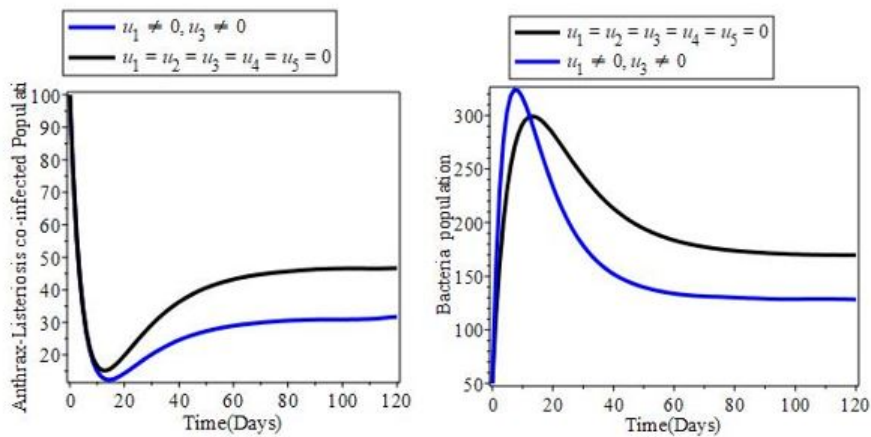


FIGURE 7. Effects of prevention and treatment on co-infected population and bacteria population.

**8.4. Treatment of Anthrax, ( $u_3$ ) and Listeriosis, ( $u_4$ ).** Objective functional was optimised by using anthrax treatment control, ( $u_3$ ) and Listeriosis treatment control, ( $u_4$ ) by setting the Anthrax prevention control, ( $u_1$ ), Listeriosis control, ( $u_2$ ) and treatment control of co-infection, ( $u_5$ ) to zero. Figure 8: showed complete reduction of Anthrax infections and reduction of Listeriosis infected persons. Figure 9: showed reduction of Anthrax-Listeriosis co-infected and reduction of bacteria population responsible for the diseases in the environment. Case without control is indicated with black line and case with control is indicated with blue line.

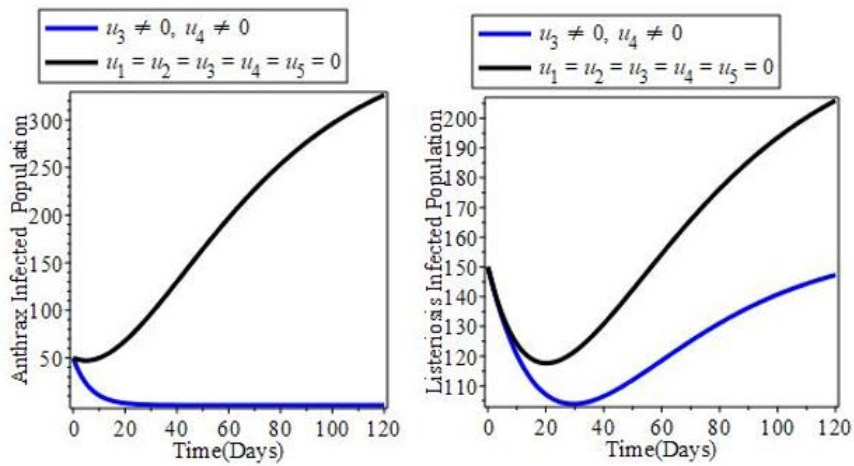


FIGURE 8. Effects of Anthrax and Listeriosis treatment on Anthrax and Listeriosis infected population.

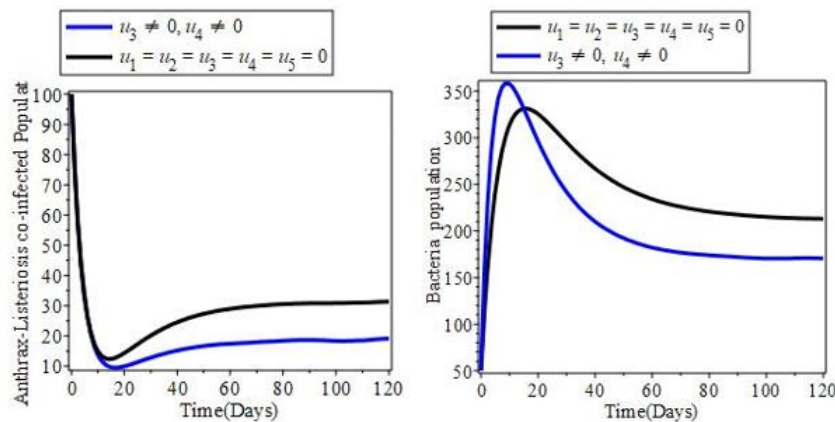


FIGURE 9. Effects of Anthrax and Listeriosis treatment on co-infected population and bacteria population.

## 9. CONCLUSION

Anthrax-Listeriosis co-infection model was formulated and incorporated the following control strategies; prevention of persons, prevention of vectors, treatment of infected persons, treatment of infected vectors and treatment of Anthrax-Listeriosis co-infected persons. The Co-dynamics model was qualitatively and quantitatively analysed for understanding of the transmission mechanism of Anthrax and Listeriosis co-infection.

Observations: the disease free equilibrium of the Anthrax only model was locally stable whenever  $\mathfrak{R}_{0a}$  was less one and a unique endemic equilibrium whenever  $\mathfrak{R}_{0a}$  was greater than one. Moreover, we observe that the disease free equilibrium of the Listeriosis only model was locally stable whenever  $\mathfrak{R}_{0l}$  was less one and a unique endemic equilibrium whenever  $\mathfrak{R}_{0l}$  was greater than one.

Our model analysis also revealed that the disease free equilibrium of the Anthrax-Listeriosis co-infection model is locally stable whenever  $\mathfrak{R}_{al}$  was less one and unstable otherwise. Our model exhibited the phenomenon of backward bifurcation. Epidemiological implications: the idea of Co-dynamics model been locally stable whenever  $\mathfrak{R}_{al}$  was less than one and unstable otherwise does not fully apply. Hence, the Anthrax-Listeriosis co-dynamics model showed a case of co-existence of the disease free equilibrium and endemic equilibrium whenever  $\mathfrak{R}_{al}$  was less than one.

We observed that the impact of Listeriosis on Anthrax infections showed that Anthrax infections can be linked with increased risk of Listeriosis but the reverse was not the case. Moreover, prevention and treatment of Anthrax without keeping Listeriosis under control was not the best strategy of combating either of these diseases. Prevention and treatment of Listeriosis can only be the effective way of combating Listeriosis if only Anthrax is kept under control. Anthrax infections can be linked to increased bacteria growth as shown in Figure 3 and Figure 5.

## CONFLICT OF INTERESTS

The author(s) declare that there is no conflict of interests.

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