

## Research Article

# Mathematical Modelling of the Transmission Dynamics of Contagious Bovine Pleuropneumonia with Vaccination and Antibiotic Treatment

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Received 18 September 2018; Revised 16 December 2018; Accepted 3 January 2019; Published 3 February 2019

Academic Editor: Urmila Diwekar

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In this paper we present a mathematical model for the transmission dynamics of Contagious Bovine Pleuropneumonia (CBPP) by considering antibiotic treatment and vaccination. The model is comprised of susceptible, vaccinated, exposed, infectious, persistently infected, and recovered compartments. We analyse the model by deriving a formula for the control reproduction number  $\mathcal{R}_c$  and prove that, for  $\mathcal{R}_c < 1$ , the disease free equilibrium is globally asymptotically stable; thus CBPP dies out, whereas for  $\mathcal{R}_c > 1$ , the unique endemic equilibrium is globally asymptotically stable and hence the disease persists. Thus,  $\mathcal{R}_c = 1$  acts as a sharp threshold between the disease dying out or causing an epidemic. As a result, the threshold of antibiotic treatment is  $\alpha_t^* = 0.1049$ . Thus, without using vaccination, more than 85.45% of the infectious cattle should receive antibiotic treatment or the period of infection should be reduced to less than 8.15 days to control the disease. Similarly, the threshold of vaccination is  $\rho^* = 0.0084$ . Therefore, we have to vaccinate at least 80% of susceptible cattle in less than 49.5 days, to control the disease. Using both vaccination and antibiotic treatment, the threshold value of vaccination depends on the rate of antibiotic treatment,  $\alpha_t$ , and is denoted by  $\rho_{\alpha_t}$ . Hence, if 50% of infectious cattle receive antibiotic treatment, then at least 50% of susceptible cattle should get vaccination in less than 73.8 days in order to control the disease.

## 1. Introduction

Contagious Bovine Pleuropneumonia (CBPP) is a major constraint to cattle production in the key pastoral regions of Africa (see [1–3] for more details). It is caused by *Mycoplasma mycoides* subspecies *mycoides* (Mmm) that attacks the lungs and the membranes of cattle and water buffalo. It is transmitted by direct contact between an infected and a susceptible animal which becomes infected by inhaling droplets disseminated by coughing. It causes high morbidity and mortality losses to cattle which leads to economic crisis (see [4–7] for more details). Cost of control of CBPP is also a major concern in African countries [6, 8]. Since some animals can carry the disease without showing signs of illness, controlling the spread is more difficult. In many countries in sub-Saharan Africa, CBPP control is based on vaccination alone, but this strategy does not eradicate the disease [9].

In [10] we presented and analysed a five-compartmental mathematical model of the transmission dynamics of CBPP, without any intervention, having the objective of identifying parameters that have significant role in changing the dynamics of the disease. As a result, from elasticity analysis, we found that the effective contact rate  $\beta$  and the rate of recovery  $\alpha_r$  are the top two parameters that control the dynamics of the disease in such a way that as the value of  $\beta$  decreases and the value of  $\alpha_r$  increases,  $\mathcal{R}_0$  decreases and can be made less than one; as a result the disease can be controlled. However, we know that vaccination is one of the ways of reducing the effective contact rate ( $\beta$ ) and antibiotic treatment is one way of reducing infection by increasing the recovery rate.

Thus, in this paper we consider vaccination and antibiotic treatment as a controlling tool of CBPP and present a compartmental model for the transmission dynamics of CBPP containing six compartments: susceptible, vaccinated,

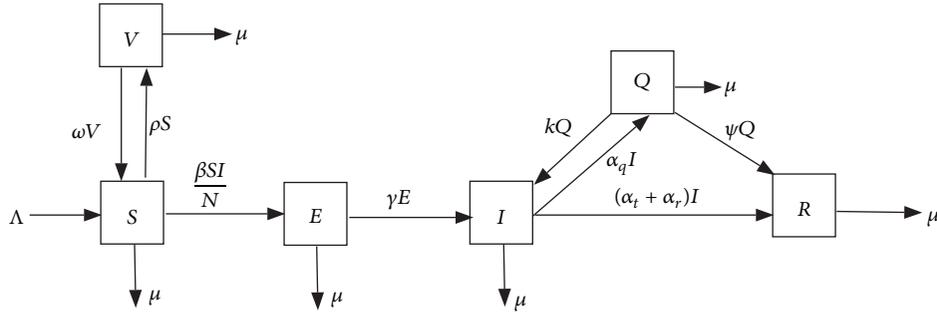


FIGURE 1: A compartmental model for the transmission dynamics of CBPP with antibiotic treatment and vaccination.

exposed, infectious, persistently infected, and recovered compartments. Antibiotic treatment is considered in the model by incorporating rate of recovery of treated cattle such that treated cattle move from infectious compartment to recovered compartment at a rate of  $\alpha_r$ .

The objective of this paper is to determine the better control method out of vaccination, antibiotic treatment, and a combination of both. We derive the formula for the control reproduction number  $\mathcal{R}_c$  and determine the number of cattle to be vaccinated and treated independently and in combination, which will enable us to choose the feasible and effective controlling method in our context. Numerical simulations are performed using MATLAB.

This paper is structured as follows. In Section 2, we present a mathematical model of the dynamics of CBPP, with vaccination and antibiotic interventions. In Section 3, we prove the well-posedness of the model. We calculate equilibria of the system and rigorously derive a formula of the control reproduction number  $\mathcal{R}_c$ , in Section 4. Stability analysis of the DFE and EE is presented in Section 5, we present parameter values and numerical simulations in Section 6, and lastly, we draw the conclusions and remarks in Section 7.

## 2. Mathematical Model

We model the transmission dynamics of Contagious Bovine Pleuropneumonia (CBPP). In this model we assume intervention by vaccination and antibiotic treatment. Thus, the compartmental model is consisting of susceptible, vaccinated, exposed, infectious, persistently infected, and recovered classes, as shown in Figure 1. We assume an open population, with a total number  $N$  at time  $t$ , where all newborn animals are born into susceptible class ( $S$ ) at rate  $b$ . Susceptible cattle move to vaccinal immune class ( $V$ ) at a rate  $\rho$ . Cattle in vaccinal immune class can lose vaccinal immunity and return back to susceptible class at a rate  $\omega$ . Susceptible animals move to the exposed compartment ( $E$ ) at a rate  $\beta(I/N)$ . Cattle in the exposed compartment move to the infectious compartment ( $I$ ) at a rate  $\gamma$ . Natural mortality occurs at a rate  $\mu$  and results in losses from all six compartments. However, we assume that death due to the disease does not occur. The infectious cattle either naturally heal or receive antibiotic treatment and enter directly into the recovered ( $R$ ) compartment at a rate  $\alpha_r$  and  $\alpha_t$ , respectively;

or they pass through a process of sequestration and enter into persistently infected ( $Q$ ) compartment at a rate  $\alpha_q$ . Cattle in persistently infected compartment are encapsulated and infected, but not infectious. As sequestra resolve and/or become noninfected, then the animals in persistently infected compartment move to the recovered ( $R$ ) compartment at a rate  $\psi$ . Recovered cattle remain recovered for life time. Infected sequestra can occasionally be reactivated and in this instance the animal will transition from the persistently infected ( $Q$ ) compartment back to the infectious ( $I$ ) compartment at a rate  $k$ . We assume random mixing of all individuals in the population. The total number of population at time  $t$ ,  $N$ , is given by  $N = S(t) + V(t) + E(t) + I(t) + Q(t) + R(t)$ . The flow diagram of the model is shown in Figure 1.

The differential equation model is given by system (1)–(7)

$$\frac{dS}{dt} = \mu N + \omega V - \frac{\beta SI}{N} - \rho S - \mu S \tag{1}$$

$$\frac{dV}{dt} = \rho S - \omega V - \mu V \tag{2}$$

$$\frac{dE}{dt} = \frac{\beta SI}{N} - \gamma E - \mu E \tag{3}$$

$$\frac{dI}{dt} = \gamma E + kQ - (\alpha_r + \alpha_t) I - \alpha_q I - \mu I \tag{4}$$

$$\frac{dQ}{dt} = \alpha_q I - kQ - \psi Q - \mu Q \tag{5}$$

$$\frac{dR}{dt} = (\alpha_r + \alpha_t) I + \psi Q - \mu R. \tag{6}$$

with initial condition

$$(S(0), V(0), E(0), I(0), Q(0), R(0)) = (S_0, V_0, E_0, I_0, Q_0, R_0). \tag{7}$$

## 3. Well-Posedness of the System

Let  $X(t) = (S(t), V(t), E(t), I(t), Q(t), R(t))$  and

$$f : \Omega \rightarrow \mathcal{Y} \tag{8}$$

$$X \mapsto X'$$

provided that  $\mathcal{Y} \subseteq R^6$ ,  $\Omega$  is a compact subset of  $R^6$  such that  $\Omega = \{x = (S(t), V(t), E(t), I(t), Q(t), R(t)) \in R^6 : S(t) + V(t) + E(t) + I(t) + Q(t) + R(t) \leq N_0 = N(0)\}$ , and  $f = (f_1, f_2, f_3, f_4, f_5, f_6)$ , where

$$f_1(X) = \frac{dS}{dt} = \mu N + \omega V - \frac{\beta SI}{N} - \rho S - \mu S \tag{9}$$

$$f_2(X) = \frac{dV}{dt} = \rho S - \omega V - \mu V \tag{10}$$

$$f_3(X) = \frac{dE}{dt} = \frac{\beta SI}{N} - \gamma E - \mu E \tag{11}$$

$$f_4(X) = \frac{dI}{dt} = \gamma E + kQ - (\alpha_r + \alpha_t) I - \alpha_q I - \mu I \tag{12}$$

$$f_5(X) = \frac{dQ}{dt} = \alpha_q I - kQ - \psi Q - \mu Q. \tag{13}$$

$$f_6(X) = \frac{dR}{dt} = (\alpha_r + \alpha_t) I + \psi Q - \mu R. \tag{14}$$

Then, (9)–(14) can be written of the form

$$\begin{aligned} X'(t) &= f(X(t)); \\ X(0) &= (S_0, V_0, E_0, I_0, Q_0, R_0) \in \Omega. \end{aligned} \tag{15}$$

**Theorem 1.** *System (1)–(6) has a unique solution  $X(t)$  which is positive and bounded if  $f$  is given by (15) and the initial conditions  $X(0) = (S_0, V_0, E_0, I_0, Q_0, R_0)$  is nonnegative.*

*Proof.* See [10]. □

### 4. Equilibria and Control Reproduction Number

#### 4.1. Equilibria of the System

**Proposition 2.** *Model (1)–(6) has at least two equilibrium points, the disease free equilibrium, and at least one endemic equilibrium.*

*Proof.* The equilibria of the system are obtained by solving equations:

$$\frac{dS}{dt} = \frac{dV}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dQ}{dt} = \frac{dR}{dt} = 0. \tag{16}$$

From (9)–(14), we have

$$\mu N + \omega V - S \left( \frac{\beta I}{N} + \rho + \mu \right) = 0 \tag{17}$$

$$\rho S - V(\omega + \mu) = 0 \tag{18}$$

$$\frac{\beta SI}{N} - E(\gamma + \mu) = 0 \tag{19}$$

$$\gamma E + kQ - I(\alpha_r + \alpha_t + \alpha_q + \mu) = 0 \tag{20}$$

$$\alpha_q I - Q(k + \psi + \mu) = 0 \tag{21}$$

$$(\alpha_r + \alpha_t) I + \psi Q - \mu R = 0. \tag{22}$$

From (21),

$$Q = \frac{\alpha_q I}{k}; \quad \text{where, } \bar{k} = k + \psi + \mu. \tag{23}$$

Putting (23) into (20) yields

$$E = \frac{(\bar{k}\bar{\alpha} - k\alpha_q) I}{\gamma \bar{k}}; \quad \text{where, } \bar{\alpha} = \alpha_r + \alpha_t + \alpha_q + \mu. \tag{24}$$

And, putting (23) into (22), we find that

$$R = \frac{((\alpha_r + \alpha_t)\bar{k} + \psi\alpha_q) I}{\mu \bar{k}}. \tag{25}$$

Similarly, putting (24) into (19) gives

$$\frac{\beta SI}{N} - \frac{\bar{\gamma}(\bar{k}\bar{\alpha} - k\alpha_q) I}{\gamma \bar{k}} = 0. \iff \tag{26}$$

$$\beta I \left( \frac{S}{N} - \frac{\bar{\gamma}(\bar{k}\bar{\alpha} - k\alpha_q)}{\beta \gamma \bar{k}} \right) = 0. \tag{27}$$

Then, we have the following two cases for solution of (27).

*Case 1.* If  $\bar{\gamma}(\bar{k}\bar{\alpha} - k\alpha_q)/\beta\gamma\bar{k} \geq 1$ , then  $I = 0$  is the only solution of (27) and

*Case 2.* If  $\bar{\gamma}(\bar{k}\bar{\alpha} - k\alpha_q)/\beta\gamma\bar{k} < 1$ , then  $I = 0$  or  $S = \bar{\gamma}(\bar{k}\bar{\alpha} - k\alpha_q)N/\beta\gamma\bar{k}$  are the solutions of (27).

For Case 1, when  $I = 0 = I^0$ , let  $S = S^0, V = V^0, E = E^0, Q = Q^0$ , and  $R = R^0$  for (17)–(22). Then, from (23)–(25), we have

$$Q^0 = E^0 = R^0 = 0. \tag{28}$$

And, from (17) and (18),

$$V^0 = \frac{\rho N}{\rho + \bar{\omega}} \tag{29}$$

and

$$S^0 = \frac{\bar{\omega} N}{\rho + \bar{\omega}}. \tag{30}$$

Therefore, from (28)–(30),  $X^0 = (S^0, V^0, E^0, I^0, Q^0, R^0) = (\bar{\omega}N/(\rho + \bar{\omega}), \rho N/(\rho + \bar{\omega}), 0, 0, 0, 0)$  is the disease free equilibrium (DFE).

For Case 2, we are done when  $I = 0$ . And, when  $S = \bar{\gamma}(\bar{k}\bar{\alpha} - k\alpha_q)N/\beta\gamma\bar{k} = S^*$ , let  $V = V^*, E = E^*, I = I^*, Q = Q^*$  and  $R = R^*$  for (17)–(22). Then (18) gives that

$$V^* = \frac{\rho \bar{\gamma}(\bar{k}\bar{\alpha} - k\alpha_q) N}{\bar{\omega} \beta \gamma \bar{k}}. \tag{31}$$

Finally, putting  $S^*$  and  $V^*$  into (17), we get

$$I^* = \frac{(\beta\gamma\bar{k}\bar{\omega} + \bar{\gamma}(\rho + \bar{\omega})(\bar{k}\bar{\alpha} - k\alpha_q))\mu N}{\beta\bar{\omega}\bar{\gamma}(\bar{k}\bar{\alpha} - k\alpha_q)}. \quad (32)$$

Hence,  $X^* = (S^*, V^*, E^*, I^*, Q^*, R^*)$  is an endemic equilibrium (EE), where,  $E^*, I^*, Q^*, R^*$  are as in (24), (32), (23), and (25), respectively.  $\square$

4.2. *The Control Reproduction Number ( $\mathcal{R}_c$ ).* Due to the presence of control measures, we will use the term control reproduction number ( $\mathcal{R}_c$ ) instead of the commonly used basic reproduction number ( $\mathcal{R}_0$ ). As explained in [12], we use the next generation matrix to calculate the control reproduction number. Compartments  $E, I,$  and  $Q$  are considered to be the disease compartments and  $S, V,$  and  $R$  are the nondisease compartments. We set  $\mathcal{F} = (\mathcal{F}_1, \mathcal{F}_2, \mathcal{F}_3)^T$  and  $\mathcal{V} = (\mathcal{V}_1, \mathcal{V}_2, \mathcal{V}_3)^T$ , where  $\mathcal{F}_i$  represents the rate of new infections in the  $i^{th}$  disease compartment,  $\mathcal{V}_i^+$  being the transfer rate of individuals into compartment  $i$  by all other means while  $\mathcal{V}_i^-$  represents the transfer rate of individual out of compartment  $i$ . Assuming  $X_0$  to be the DFE, we have

$$\begin{aligned} \mathcal{F} &= \begin{bmatrix} \frac{\beta SI}{N} \\ 0 \\ 0 \end{bmatrix}, \\ \mathcal{V} &= \mathcal{V}^- - \mathcal{V}^+ = \begin{bmatrix} \bar{\gamma}E \\ \bar{\alpha}I - \gamma E - kQ \\ \bar{k}Q - \alpha_q I \end{bmatrix}, \\ F &= \left[ \frac{\partial \mathcal{F}_i}{\partial x_j} (X_0) \right] = \begin{bmatrix} 0 & \frac{\beta S^0}{N} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}, \\ V &= \left[ \frac{\partial \mathcal{V}_i}{\partial x_j} (X_0) \right] = \begin{bmatrix} \bar{\gamma} & 0 & 0 \\ -\gamma & \bar{\alpha} & -k \\ 0 & -\alpha_q & \bar{k} \end{bmatrix} \text{ and} \\ V^{-1} &= \begin{bmatrix} \frac{1}{\bar{\gamma}} & 0 & 0 \\ \frac{\bar{\gamma}}{\gamma\bar{k}} & \frac{\bar{k}}{\bar{k}\bar{\alpha} - k\alpha_q} & \frac{k}{\bar{k}\bar{\alpha} - k\alpha_q} \\ \frac{\alpha_q\bar{\gamma}}{\bar{\gamma}(\bar{k}\bar{\alpha} - k\alpha_q)} & \frac{\alpha_q}{\bar{k}\bar{\alpha} - k\alpha_q} & \frac{\bar{\alpha}}{\bar{k}\bar{\alpha} - k\alpha_q} \end{bmatrix}. \end{aligned} \quad (33)$$

Therefore,

$$FV^{-1} = \frac{\beta S^0}{N} \begin{bmatrix} \frac{\gamma\bar{k}}{\bar{\gamma}(\bar{k}\bar{\alpha} - k\alpha_q)} & \frac{\bar{k}}{\bar{k}\bar{\alpha} - k\alpha_q} & \frac{k}{\bar{k}\bar{\alpha} - k\alpha_q} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}. \quad (34)$$

Therefore,  $\mathcal{R}_c = \rho(FV^{-1}) = T/2 + \sqrt{(T/2)^2 - D}$ , where  $T$  and  $D$  are trace and determinant of the matrix  $FV^{-1}$ . Since  $D = 0$ ,

$$\mathcal{R}_c = T = \frac{\beta\bar{\omega}}{\rho + \bar{\omega}} \left( \frac{\gamma\bar{k}}{\bar{\gamma}(\bar{k}\bar{\alpha} - k\alpha_q)} \right) \quad (35)$$

Equivalently,

$$\mathcal{R}_c = \left( \frac{\bar{\omega}}{\rho + \bar{\omega}} \right) \left( \frac{\bar{k}(\bar{\alpha} - \alpha_t) - k\alpha_q}{\bar{k}\bar{\alpha} - k\alpha_q} \right) \mathcal{R}_0, \quad (36)$$

where  $\mathcal{R}_0 = \beta\gamma\bar{k}/\bar{\gamma}(\bar{k}(\bar{\alpha} - \alpha_t) - k\alpha_q)$  is the basic reproduction number as derived in [10] and  $\bar{\omega}/(\rho + \bar{\omega})$  is the proportion of cattle that survive the vaccination class and the control reproduction number,  $\mathcal{R}_c$ , is the average number of secondary cases caused by an infected individual over the course of infectious period in the presence of vaccination and antibiotic treatment. We observe that  $\mathcal{R}_c < \mathcal{R}_0$ .

## 5. Stability Analysis

### 5.1. Stability Analysis of the Disease Free Equilibrium (DFE)

#### 5.1.1. Local Stability Analysis of the DFE

**Theorem 3** (see [12]). *If  $X^0$  is a DFE of the model given by (1)–(7), then  $X^0$  is locally asymptotically stable if  $\mathcal{R}_c < 1$ , and unstable if  $\mathcal{R}_c > 1$ , where  $\mathcal{R}_c$  is defined by (36).*

*Proof.* See [12].  $\square$

#### 5.1.2. Global Stability Analysis of the DFE

**Theorem 4.** *If  $\mathcal{R}_c < 1$ , then disease free equilibrium  $(\bar{\omega}N/(\bar{\omega} + \rho), \rho N/(\bar{\omega} + \rho), 0, 0, 0, 0)$  is globally asymptotically stable in  $\Omega$ . If  $\mathcal{R}_c > 1$ , then the DFE is unstable, the system is uniformly persistent and there is at least one equilibrium in interior of  $\Omega$ , where  $\Omega = \{(S, V, E, I, Q, R) \in R_+^6 : S \geq 0, V \geq 0, E \geq 0, I \geq 0, Q \geq 0, R \geq 0, S + V + E + I + Q + R \leq N_0 = N(0)\}$ .*

*Proof.* We use matrix-theoretic method as explained in [13]. We assume  $x = (E, I, Q)^T$  and  $y = (S, V, R)^T$ . And, considering  $F, V,$  and  $V^{-1}$  as in Section 4.2, we set

$$\begin{aligned} f(x, y) &= (F - V)x - \mathcal{F}(x, y) + \mathcal{V}(x, y) \\ &= \begin{bmatrix} \frac{\beta S^0 I}{N} - \frac{\beta SI}{N} \\ 0 \\ 0 \end{bmatrix} = \frac{\beta I}{N} \begin{bmatrix} \left( \frac{\bar{\omega}N}{\bar{\omega} + \rho} - S \right) \\ 0 \\ 0 \end{bmatrix}; \end{aligned} \quad (37)$$

where  $S^0 = \bar{\omega}N/(\bar{\omega} + \rho)$  is as in (30).

And,

$$V^{-1}F = \frac{\beta S^0}{N} \begin{bmatrix} 0 & \frac{1}{\bar{\gamma}} & 0 \\ \frac{\bar{\gamma}}{\gamma k} & 0 & 0 \\ \frac{\bar{\gamma}(\bar{k}\bar{\alpha} - k\alpha_q)}{\alpha_q \bar{\gamma}} & 0 & 0 \end{bmatrix} \quad (38)$$

$$= \frac{\beta \bar{\omega}}{\bar{\omega} + \rho} \begin{bmatrix} 0 & \frac{1}{\bar{\gamma}} & 0 \\ \frac{\bar{\gamma}}{\gamma k} & 0 & 0 \\ \frac{\bar{\gamma}(\bar{k}\bar{\alpha} - k\alpha_q)}{\alpha_q \bar{\gamma}} & 0 & 0 \end{bmatrix} \quad (39)$$

$$= \frac{\beta \gamma \bar{\omega}}{\bar{\gamma}(\bar{\omega} + \rho)} \begin{bmatrix} 0 & \frac{1}{\bar{\gamma}} & 0 \\ \frac{\bar{\gamma}}{k} & 0 & 0 \\ \frac{\alpha_q}{(\bar{k}\bar{\alpha} - k\alpha_q)} & 0 & 0 \end{bmatrix} \quad (40)$$

We observe that  $F \geq 0$ ,  $V^{-1} \geq 0$ , and  $f(x, y) \geq 0$  when  $\rho = 0$  and  $f(x, (\bar{\omega}N/(\bar{\omega} + \rho), \rho N/(\bar{\omega} + \rho), 0)^T) = 0$  in  $\Omega$ . Since matrix  $V^{-1}F$  is reducible, we use Theorem 2.1 of [13] to construct a Lyapunov function. Let  $\omega^T = (v_1, v_2, v_3) \geq 0$  be the left eigenvector of nonnegative matrix  $V^{-1}F$  corresponding to the eigenvalue  $\mathcal{R}_c$ . Then

$$(v_1, v_2, v_3) V^{-1}F = \mathcal{R}_c (v_1, v_2, v_3) \quad (41)$$

such that

$$(v_1, v_2, v_3) V^{-1}F = \left( \frac{\beta \bar{\omega} \gamma}{(\bar{\omega} + \rho) \bar{\gamma}} \right) (0, x, 0); \text{ where,} \quad (42)$$

$$x = \frac{v_1}{\gamma} + \frac{\bar{k} v_2}{(\bar{k}\bar{\alpha} - k\alpha_q)} + \frac{\alpha_q v_3}{(\bar{k}\bar{\alpha} - k\alpha_q)} \quad (43)$$

and

$$\mathcal{R}_c (v_1, v_2, v_3) = \frac{\beta \bar{\omega} \gamma}{(\bar{\omega} + \rho) \bar{\gamma}} \left( \frac{\bar{k}}{\bar{\alpha}\bar{k} - k\alpha_q} \right) (v_1, v_2, v_3). \quad (44)$$

Thus, from (41)-(44), we find that  $v_1 = v_3 = 0$  and  $v_2 \in \mathbb{R}^+$ . Hence,  $\omega^T = (0, v_2, 0)$ . By Theorem 2.1 of [13],

$$\begin{aligned} L &= \omega^T V^{-1} x \\ &= v_2 \left( \frac{\gamma \bar{k} E}{\bar{\gamma}(\bar{k}\bar{\alpha} - k\alpha_q)} + \frac{\bar{k} I}{\bar{k}\bar{\alpha} - k\alpha_q} + \frac{k Q}{\bar{k}\bar{\alpha} - k\alpha_q} \right) \end{aligned} \quad (45)$$

is the Lyapunov function for the system when  $\mathcal{R}_c < 1$ . Since  $L' = (\mathcal{R}_c - 1)\omega^T x - \omega^T V^{-1} f(x, y) = 0$  implies that  $x = 0$  and  $y = (\bar{\omega}N/(\bar{\omega} + \rho), \rho N/(\bar{\omega} + \rho), 0)^T$ , it follows that

$(\bar{\omega}N/(\bar{\omega} + \rho), \rho N/(\bar{\omega} + \rho), 0, 0, 0, 0)$  is the only invariant set in  $\Omega$  when  $x = 0$  and  $y = (\bar{\omega}N/(\bar{\omega} + \rho), \rho N/(\bar{\omega} + \rho), 0)^T$ . Thus, by LaSalle's invariance principle, the DFE  $(\bar{\omega}N/(\bar{\omega} + \rho), \rho N/(\bar{\omega} + \rho), 0, 0, 0, 0)$  is globally asymptotically stable in  $\Omega$  when  $\mathcal{R}_c < 1$ . If  $\mathcal{R}_c > 1$ , then  $L' > 0$  for  $x = 0$  and  $y = (\bar{\omega}N/(\bar{\omega} + \rho), \rho N/(\bar{\omega} + \rho), 0)^T$ . Hence, by continuity,  $L' > 0$  in the neighbourhood of the DFE, implies that the DFE is unstable when  $\mathcal{R}_c > 1$ . Instability of the DFE implies uniform persistence of (1)-(7). Uniform persistence and the positive invariance of the compact set  $\Omega$  imply the existence of a unique EE of (1)-(7).  $\square$

### 5.2. Global Stability Analysis of the Endemic Equilibrium (EE)

**Theorem 5.** *If  $\mathcal{R}_c > 1$ , then the endemic equilibrium  $X^*$  of (1)-(7) is unique and globally asymptotically stable in the interior of  $\Omega$ .*

*Proof.* We use a graph-theoretic method as explained in [13]. Thus, for construction of a Lyapunov function, set  $D_1 = S - S^* - S^* \ln(S/S^*)$ ,  $D_2 = V - V^* - V^* \ln(V/V^*)$ ,  $D_3 = E - E^* - E^* \ln(E/E^*)$ ,  $D_4 = I - I^* - I^* \ln(I/I^*)$ ,  $D_5 = Q - Q^* - Q^* \ln(Q/Q^*)$ , and  $D_6 = R - R^* - R^* \ln(R/R^*)$ . And, putting  $(S^*, V^*, E^*, I^*, Q^*, R^*)$  into (17)-(22), we find that  $\mu N = \beta S^* I^*/N + S^*(\rho + \mu) - \omega V^*$ ,  $\omega + \mu = \rho S^*/V^*$ ,  $\gamma + \mu = \beta S^* I^*/NE^*$ ,  $\alpha_r + \alpha_q + \mu = (\gamma E^* + kQ^*)/I^*$ ,  $k + \psi + \mu = \alpha_q I^*/Q^*$ , and  $\mu = (\alpha_r I^* + \psi Q^*)/R^*$ . We use these equalities and the inequality  $1 - x + \ln x \leq 0$  in differentiation of  $D_1, D_2, D_3, D_4, D_5$ , and  $D_6$ , with respect to  $t$ , as follows:

$$\begin{aligned} D_1' &= \left(1 - \frac{S^*}{S}\right) \left(\mu N + \omega V - \frac{\beta SI}{N} - S(\rho + \mu)\right) \\ &= \left(1 - \frac{S^*}{S}\right) \left(\frac{\beta S^* I^*}{N} + S^*(\rho + \mu) - \omega V^* + \omega V - \frac{\beta SI}{N} - S(\rho + \mu)\right) \\ &= \omega V^* \left(1 - \frac{S^*}{S}\right) \left(\frac{V}{V^*} - 1\right) \\ &\quad + S^*(\rho + \mu) \left(1 - \frac{S^*}{S}\right) \left(1 - \frac{S}{S^*}\right) + \frac{\beta S^* I^*}{N} \left(1 - \frac{S^*}{S}\right) \left(1 - \frac{S}{S^*}\right) \\ &= \omega V^* \left(\frac{V}{V^*} - \frac{S^* V}{SV^*} + \frac{S^*}{S} - 1\right) + \frac{(\rho + \mu)(\omega + \mu)V^*}{\rho} \left(1 - \frac{S}{S^*} - \frac{S^*}{S} + 1\right) \\ &\quad + \frac{\beta S^* I^*}{N} \left(1 - \frac{SI}{S^* I^*} - \frac{S^*}{S} + \frac{I}{I^*}\right) \\ &\leq \frac{(\rho + \mu)(\omega + \mu)V^*}{\rho} \left(\frac{V}{V^*} - \frac{S^* V}{SV^*} - \frac{S}{S^*} + 1\right) \\ &\quad + \frac{\beta S^* I^*}{N} \left(1 - \frac{SI}{S^* I^*} - \frac{S^*}{S} + \frac{I}{I^*}\right) \\ &\leq \frac{(\rho + \mu)(\omega + \mu)V^*}{\rho} \left(\frac{V}{V^*} - \frac{S}{S^*} - \ln\left(\frac{S^*}{S}\right)\right) \end{aligned}$$

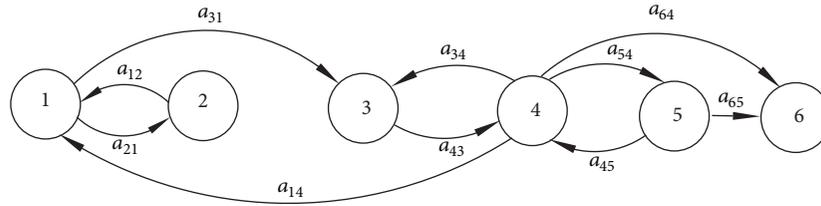


FIGURE 2: The weighted digraph  $(G, A)$  constructed for model (1)–(6).

$$\begin{aligned}
 & -\ln\left(\frac{V}{V^*}\right) + \frac{\beta S^* I^*}{N} \left(1 - \frac{SI}{S^* I^*} - \frac{S^*}{S} + \frac{I}{I^*}\right) \\
 & =: a_{12}G_{12} + a_{14}G_{14} \\
 D'_2 & = \left(1 - \frac{V^*}{V}\right) (\rho S - (\omega + \mu)V) = \left(1 - \frac{V^*}{V}\right) \left(\rho S - \frac{\rho S^* V}{V^*}\right) \\
 & = \rho S^* \left(1 - \frac{V^*}{V}\right) \left(\frac{S}{S^*} - \frac{V}{V^*}\right) \\
 & = \rho S^* \left(\frac{S}{S^*} - \frac{V}{V^*} - \frac{V^* S}{VS^*} + 1\right) \leq \rho S^* \left(\frac{S}{S^*} - \frac{V}{V^*} + \ln \frac{V}{V^*} + \ln \frac{S^*}{S}\right) =: a_{21}G_{21} \\
 D'_3 & = \left(1 - \frac{E^*}{E}\right) \left(\frac{\beta SI}{N} - (\gamma + \mu)E\right) = \left(1 - \frac{E^*}{E}\right) \cdot \left(\frac{\beta SI}{N} - \frac{\beta S^* I^* E}{NE^*}\right) \\
 & = \left(1 - \frac{E^*}{E}\right) \left(\frac{\beta S^* I^*}{N}\right) \left(\frac{SI}{S^* I^*} - \frac{E}{E^*}\right) \\
 & = \frac{\beta S^* I^*}{N} \left(\frac{SI}{S^* I^*} - \frac{E}{E^*} - \frac{E^* SI}{ES^* I^*} + 1\right) \\
 & \leq \frac{\beta S^* I^*}{N} \left(\frac{SI}{S^* I^*} - \frac{E}{E^*} + \ln \frac{E}{E^*} - \ln \frac{SI}{S^* I^*}\right) \\
 & = \frac{\beta S^* I^*}{N} \left(\frac{SI}{S^* I^*} - \ln \frac{SI}{S^* I^*} - \frac{I}{I^*} + \ln \frac{I}{I^*}\right) \\
 & + \frac{\beta S^* I^*}{N} \left(-\frac{E}{E^*} + \ln \frac{E}{E^*} + \frac{I}{I^*} - \ln \frac{I}{I^*}\right) =: a_{31}G_{31} \\
 & + a_{34}G_{34} \\
 D'_4 & = \left(1 - \frac{I^*}{I}\right) (\gamma E + kQ - I(\alpha_r + \alpha_q + \mu)) = \left(1 - \frac{I^*}{I}\right) \left(\gamma E + kQ - \frac{I(\gamma E^* + kQ^*)}{I^*}\right) \\
 & = \gamma E^* \left(\frac{E}{E^*} - \frac{I}{I^*} - \frac{I^* E}{IE^*} + 1\right) + kQ^* \left(\frac{Q}{Q^*} - \frac{I}{I^*} - \frac{I^* Q}{IQ^*} + 1\right) \\
 & \leq \gamma E^* \left(\frac{E}{E^*} - \frac{I}{I^*} + \ln \frac{I}{I^*} - \ln \frac{E}{E^*}\right) + kQ^* \left(\frac{Q}{Q^*} - \frac{I}{I^*} + \ln \frac{I}{I^*} - \ln \frac{Q}{Q^*}\right) =: a_{43}G_{43} + a_{45}G_{45}
 \end{aligned}$$

$$\begin{aligned}
 D'_5 & = \left(1 - \frac{Q^*}{Q}\right) (\alpha_q I - Q(k + \psi + \mu)) = \left(1 - \frac{Q^*}{Q}\right) \cdot \left(\alpha_q I - Q\left(\frac{\alpha_q I^*}{Q^*}\right)\right) \\
 & = \alpha_q I^* \left(\frac{I}{I^*} - \frac{Q}{Q^*} - \frac{IQ^*}{I^* Q} + 1\right) \leq \alpha_q I^* \left(\frac{I}{I^*} - \frac{Q}{Q^*} + \ln \frac{Q}{Q^*} - \ln \frac{I}{I^*}\right) \\
 & =: a_{54}G_{54} \\
 D'_6 & = \left(1 - \frac{R^*}{R}\right) (\alpha_r I + \psi Q - \mu R) = \left(1 - \frac{R^*}{R}\right) \left(\alpha_r I + \psi Q - \frac{R(\alpha_r I^* + \psi Q^*)}{R^*}\right) \\
 & = \alpha_r I^* \left(\frac{I}{I^*} - \frac{R}{R^*} - \frac{R^* I}{RI^*} + 1\right) + \psi Q^* \left(\frac{Q}{Q^*} - \frac{R}{R^*} - \frac{R^* Q}{RQ^*} + 1\right) \\
 & \leq \alpha_r I^* \left(\frac{I}{I^*} - \frac{R}{R^*} + \ln \frac{R}{R^*} - \ln \frac{I}{I^*}\right) + \psi Q^* \left(\frac{Q}{Q^*} - \frac{R}{R^*} + \ln \frac{R}{R^*} - \ln \frac{Q}{Q^*}\right) =: a_{64}G_{64} + a_{65}G_{65}
 \end{aligned} \tag{46}$$

where  $a_{12} = ((\rho + \mu)(\omega + \mu)/\rho)V^* = (\rho + \mu)S^*$ ,  $a_{14} = a_{31} = a_{34} = \beta S^* I^*/N$ ,  $a_{21} = \rho S^*$ ,  $a_{43} = \gamma E^*$ ,  $a_{45} = kQ^*$ ,  $a_{54} = \alpha_q I^*$ ,  $a_{64} = \alpha_r I^*$ ,  $a_{65} = \psi Q^*$ , and all other  $a_{ij} = 0$  such that the weight matrix is  $A = [a_{ij}]_{6 \times 6}$ , where  $a_{ij} > 0$  is the weight of arc  $(j, i)$ . Thus, the associated weighted digraph  $(G, A)$  for the model given by system of (1)–(6) is presented in Figure 2.

Along each directed cycle,  $G_{12} + G_{21} = 0$ ,  $G_{34} + G_{43} = 0$ , and  $G_{45} + G_{54} = 0$ . Therefore, by Theorem 3.5 of [13], there exists  $c_i, 1 \leq i \leq 6$ , such that  $V = \sum_{i=1}^6 c_i D_i$  is a Lyapunov function for (1)–(6), where the relations between  $c_i$ 's can be derived from Theorems 3.3 and 3.4 of [13] that  $d^+(2) = 1$  implies  $c_1 a_{12} = c_2 a_{21}$ ,  $d^+(3) = 1$  implies  $c_4 a_{43} = c_3 (a_{31} + a_{34})$ , and  $d^+(5) = 1$  implies  $c_5 a_{54} = c_4 a_{45} + c_6 a_{65}$ . Hence,  $c_2 = (a_{12}/a_{21})c_1$ ,  $c_4 = ((a_{31} + a_{34})/a_{43})c_3$ , and  $c_6 = ((a_{54}a_{43}c_5 - a_{45}(a_{31} + a_{34})c_3)/a_{65}a_{43})$ . And,  $V' = \sum_{i=1}^6 c_i D'_i = 0$  implies  $X = X^*$ . Hence the largest invariance set for (1)–(7) where  $V' = 0$  is the singleton set  $\{X^*\}$ .

Thus, proving uniqueness and global asymptotic stability of  $X^*$  in interior of  $\Omega$  provided that  $\mathcal{R}_c > 1$ .  $\square$

TABLE 1: Description of model parameters and their values, indicating baselines, ranges, and references. Units are days<sup>-1</sup> unless otherwise defined. \* Proportions.

Parameter	Description	Baseline value	Value range and references
$\beta$	Effective contact rate	0.126	0.07 to 0.13 [11]
$p_e$	Vaccination efficacy*	0.65	0.5 to 0.8 [11]
$p_v$	Proportion vaccinated*	0.5	[11]
$\epsilon$	Vaccination efficiency*	0.8	[11]
$p$	Proportion immunized*	$\epsilon \times p_v \times p_e$	[11]
$\rho$	Rate of vaccination	$\frac{p}{73\text{days}}$	assumed
$\omega$	Rate of loss of vaccinal immunity	$\frac{1}{3 \times 365}$	0.00078 to 0.0011 [11]
$\gamma$	Transition rate from exposed to infectious compartment	$\frac{1}{6 \times 7}$	$\frac{1}{8 \times 7}$ to $\frac{1}{4 \times 7}$ [11]
$\alpha_r$	Natural recovery rate of infectious cattle	$\frac{1}{4 \times 56}$	$\frac{1}{4} \times \frac{1}{42}$ to $\frac{1}{4} \times \frac{1}{70}$ [11]
$\alpha_q$	Rate of sequestrum formation of infectious cattle	$3\alpha_r$	[11]
$\alpha_t$	Rate of recovery of treated cattle	$\frac{1}{28} - \frac{1}{56}$	$\frac{1}{28} - \frac{1}{42}$ to $\frac{1}{28} - \frac{1}{70}$ assumed
$k$	Rate of sequestrum reactivation	0.00009	0.00007 – 0.00011[11]
$\psi$	Rate of sequestrum resolution	0.0075	0.0068 to 0.0079 [11]
$\mu$	Mortality rate	$\frac{1}{5 \times 365}$	$\frac{1}{6 \times 365}$ to $\frac{1}{4.5 \times 365}$ [11] and estimated guess
$b$	Birth rate	$\frac{1}{5 \times 365}$	$\frac{1}{6 \times 365}$ to $\frac{1}{4.5 \times 365}$ [11] and estimated

## 6. Parameter Values and Numerical Simulations

6.1. *Parameter Values.* Most of the parameter values used in this paper are explained in Table 1, Sections 2.2 and 2.3 of [14], and Table 1 of [1]. We assume that the life expectancy of cattle is in average 5 years, then the value of  $\mu$  and  $b$  is taken to be  $1/(5 \times 365)$ ,  $\beta = 0.126$ , the incubation period between 4 and 8 weeks with mean value of 6 weeks yields  $\gamma = 1/(6 \times 7)$ , without applying antibiotic treatment, the infection period is between 6 and 10 weeks with mean value of 8 weeks and  $\alpha_q = 3\alpha_r$ , then  $\alpha_r = 1/(4 \times 56)$ , the persistently infected period given in a range of 18–21 weeks with an average period of 19 weeks with 4 months  $\times$  2 reactivations per month for 582 cases gives  $k = 0.0009$  and  $\psi = 0.0075$ , the rate of vaccination,  $\rho = \epsilon p_v p_e / t$ , where  $\epsilon$  is the efficiency of vaccine,  $p_v$  is the proportion vaccinated,  $p_e$  is efficacy of the vaccine, and  $t$  is the period of vaccination and vaccinal immunity lasts for 3

years which implies that  $\omega = 1/(3 \times 365)$ . When we introduce antibiotic treatment at a rate of  $\alpha_t$ , the period of infection (56 days) will be reduced to some new period  $P$  such that  $\alpha_r + \alpha_t + \alpha_q = 1/P$  implies  $\alpha_t = 1/P - 1/56$ . Since  $\mathcal{R}_c = 1$  acts as a sharp threshold between the disease dying out or causing an epidemic, we find that the threshold of antibiotic treatment is given by  $\alpha_t^* = \beta\gamma(\mathcal{R}_0 - 1)/(\gamma + \mu)\mathcal{R}_0 = 0.1049$ , where  $\mathcal{R}_0$  is the basic reproduction number as in [10]. This implies that, without using vaccination, more than 85.45% of the infectious cattle should receive antibiotic treatment or the period of infection should be reduced to less than 8.15 days to control the disease. And, threshold of vaccination is also given by  $\rho^* = (\epsilon \times p_v \times p_e)/t = (\omega + \mu)(\mathcal{R}_0 - 1) = 0.0084$ , where  $t$  is period of vaccination, which can be interpreted that at least 80% of susceptible cattle should get vaccination in less than 49.5 days in order to control the disease; however, since the proportion to be vaccinate  $p_v$  depends on  $t$ , a single value of  $\rho$  can have many practical interpretation. For the last

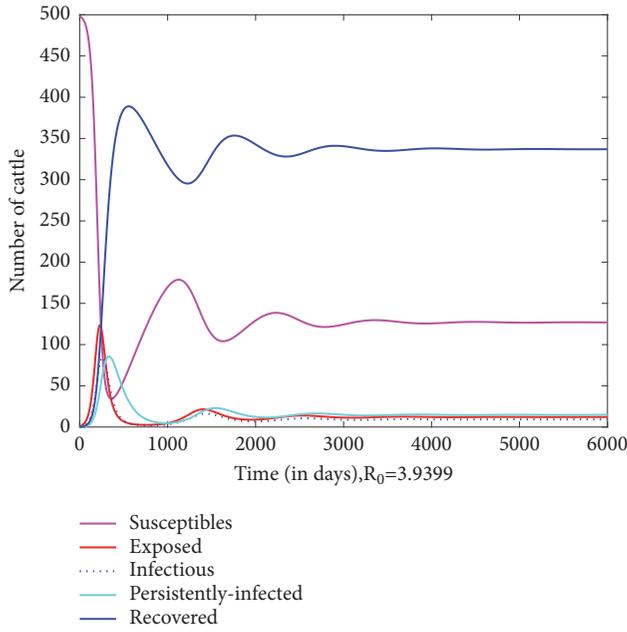


FIGURE 3: Number of cattle in each compartment for parameter values in Table 1 with  $\alpha_r = 1/56$  (as in [10]) and  $I_0 = 1, S_0 = 499, E_0 = Q_0 = R_0 = 0$ , giving approximate equilibrium values  $S^* = 127, E^* = 12, I^* = 9, Q^* = 15, R^* = 337$ , and  $\mathcal{R}_0 = 3.9399$ .

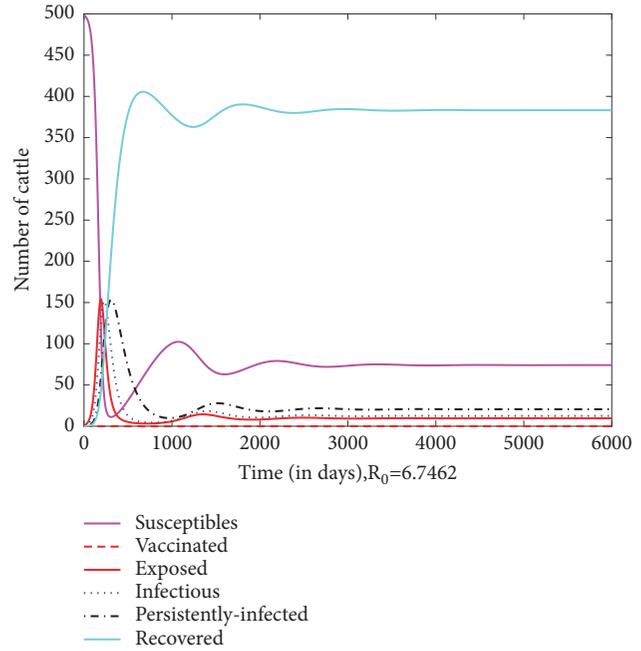


FIGURE 4: Number of cattle in each compartment with baseline parameter values in Table 1 with  $\rho = \alpha_r = 0, I_0 = 1, S_0 = 499$ , and  $V_0 = E_0 = T_0 = Q_0 = R_0 = 0$ , giving approximate equilibrium values  $S^* = 74, E^* = 10, I^* = 12, Q^* = 21, R^* = 383$ , and  $\mathcal{R}_0 = 6.7462$ .

option, in applying both vaccination and antibiotic treatment, the threshold value of vaccination depends on the rate of antibiotic treatment,  $\alpha_t$ , and is given by  $\rho_{\alpha_t} = (\omega + \mu) \left( \frac{\bar{k}(\bar{\alpha} - \alpha_t) - k\alpha_q}{(\bar{k}\bar{\alpha} - k\alpha_q)} \right) \mathcal{R}_0 - 1$ . Thus, if we introduce both antibiotic treatment and vaccination in the population such that 50% of infectious cattle receive antibiotic treatment or the period of infection is reduced to 28 days, then at least 50% of susceptible cattle should get vaccination in less than 73.8 days in order to control the disease. Mathematically, it means that, for  $\alpha_t = 1/28 - 1/56 = 1/56$ , we should take  $\rho = (0.65 \times 0.5 \times 0.8)/73$  to make  $\mathcal{R}_c < 1$ , to control the disease. Parameter values considering both antibiotic treatment and vaccination are summarized in Table 1.

- (i) All parameter values used in this paper and in [10] are the same except the value of  $\alpha_r$ , which is taken in [10] as  $1/56$  instead of  $1/(4 \times 56)$ .

6.2. Numerical Simulations. Initially we consider a herd size of 500 cattle population which is consisting of an infectious cattle and 499 susceptible cattle with individual animals as the epidemiological units of interest. For the same assumption and parametric values, the result obtained in this paper coincides with the result obtained in [10]; in this case,  $\mathcal{R}_0 = 3.9399$  as shown in Figure 3. Using parameter values in Table 1, model (1)-(6) is numerically solved. If no intervention is considered, the population goes extinct with  $\mathcal{R}_0 = 6.7462$  as shown in Figure 4. Figures 5 and 6 show the number of cattle in each compartment when we consider intervention by antibiotic treatment without vaccination and vice versa, respectively; in both cases,  $\mathcal{R}_c < 1$ . Lastly, considering both antibiotic treatment and vaccination, the number of cattle in

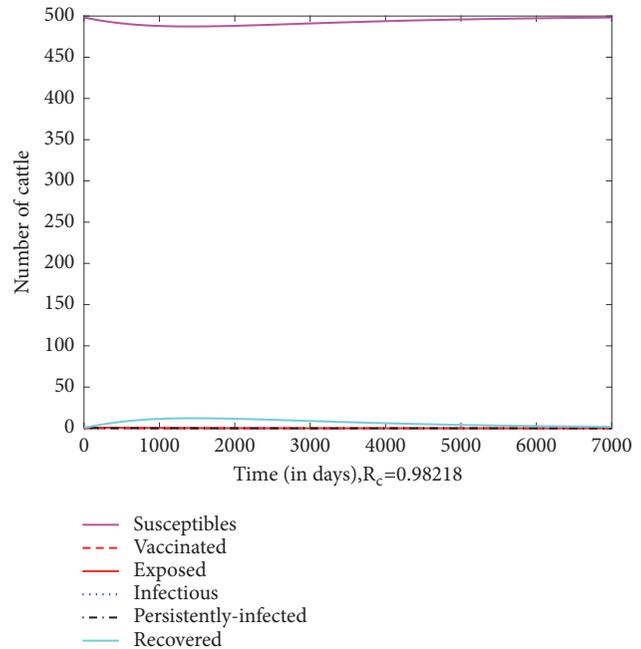


FIGURE 5: Number of cattle in each compartment with baseline parameter values in Table 1 with the assumption that 85.7% of infectious cattle receive antibiotic treatment within 8 days ( $\alpha_t = 1/8 - 1/56$ ) without vaccinating healthy cattle ( $\rho = 0$ ),  $I_0 = 1, S_0 = 499$ , and  $V_0 = E_0 = Q_0 = R_0 = 0$ , giving approximate equilibrium values  $S^* = 498, E^* = I^* = Q^* = 0, R^* = 2$ , and  $\mathcal{R}_c = 0.9822$ .

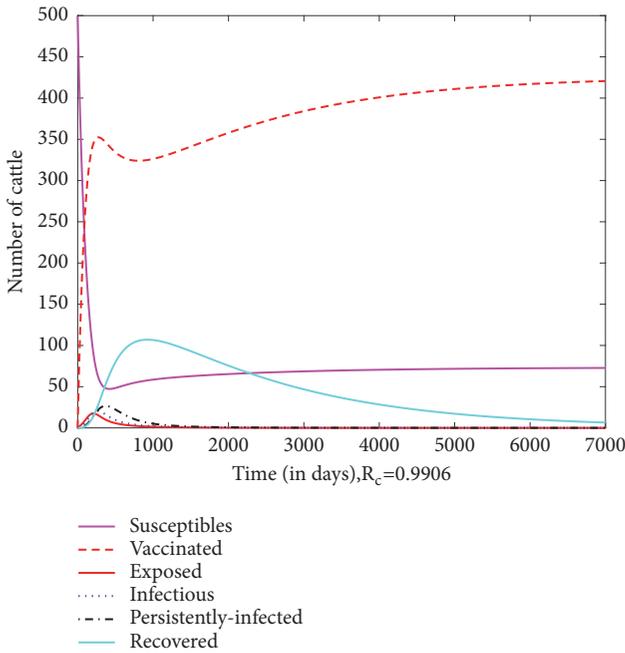


FIGURE 6: Number of cattle in each compartment with baseline parameter values in Table 1 with the assumption that 80% of susceptible cattle are vaccinated within 49 days ( $\rho = (0.65 * 0.8 * 0.8)/49$ ) without treating infectious cattle ( $\alpha_t = 0$ ),  $I_0 = 1$ ,  $S_0 = 499$ , and  $V_0 = E_0 = Q_0 = R_0 = 0$ , giving approximate equilibrium values  $S^* = 73$ ,  $V^* = 421$ ,  $E^* = I^* = Q^* = 0$ ,  $R^* = 6$ , and  $\mathcal{R}_c = 0.9906$ .

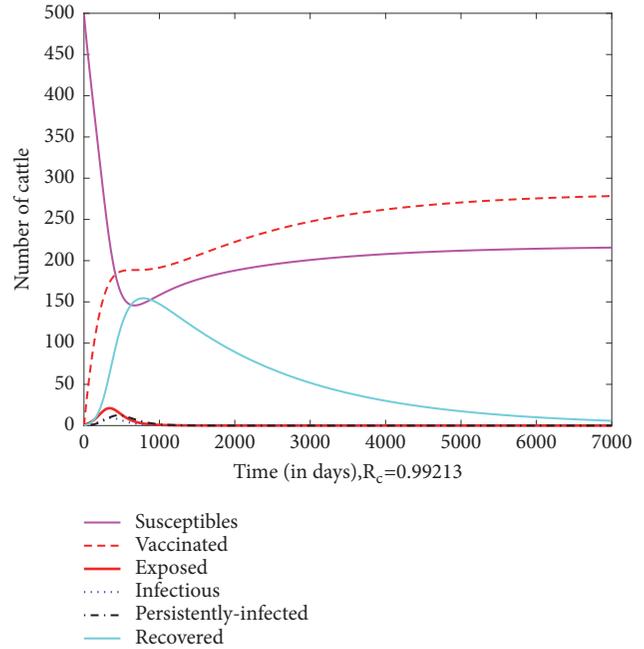


FIGURE 7: Number of cattle in each compartment with baseline parameter values in Table 1 with the assumption that 50% of infectious cattle receive antibiotic treatment or the period of infection is reduced to 28 days ( $\alpha_t = 1/28 - 1/56$ ), 50% of susceptible get vaccination within 73 days ( $\rho = (0.5 \times 0.8 \times 0.65)/73$ ),  $I_0 = 1$ ,  $S_0 = 499$  and  $V_0 = E_0 = T_0 = Q_0 = R_0 = 0$ , giving approximate equilibrium values  $S^* = 144$ ,  $V^* = 350$ ,  $E^* = I^* = Q^* = 0$ ,  $R^* = 6$ , and  $\mathcal{R}_c = 0.9921$ .

each compartment at time  $t$  is plotted in Figure 7; in this case,  $\mathcal{R}_c = 0.99213$  for parametric values in Table 1.

### 7. Conclusion and Remarks

In this paper we presented compartmental model and differential equations for the transmission dynamics of CBPP with intervention. We calculated the equilibrium of the system and to study the behaviour of the disease, we derived a formula for the control reproduction number  $\mathcal{R}_c$ . We proved that, for  $\mathcal{R}_c < 1$ , the DFE is globally asymptotically stable, thus CBPP dies out, whereas for  $\mathcal{R}_c > 1$ , the EE is globally asymptotically stable and hence the disease persists in all the populations. Hence  $\mathcal{R}_c = 1$  acts as a sharp threshold between the disease dying out or causing an epidemic. Without considering any intervention, the model in this paper coincides with the model studied in [10]; in both papers,  $\mathcal{R}_0 = 3.9399$  when  $\alpha_r = 1/56$ ; see Figure 3. Similarly, when  $\alpha_r = 1/(4 \times 56)$ , which is the right value, and without any intervention,  $\mathcal{R}_0 = 6.7462$ ; see Figure 4. Hence, without any intervention the disease persists in all the population. However, we can control the disease by giving antibiotic treatment to 85.7% of infectious cattle, without vaccinating any of healthy cattle; see Figure 5. As a second option, we can also control the disease by vaccinating 80% of susceptible cattle within a period of 49 days, without treating any of infectious cattle; see Figure 6. Finally, for parametric values in Table 1, with the assumption that 50% of susceptible are vaccinated within

a period of 73 days and 50% of infectious cattle are treated,  $\mathcal{R}_c$  can be made less than one and hence we can control the disease; see Figure 7. In all the above three intervention methods,  $\mathcal{R}_c < 1$  and hence the disease can be controlled by properly applying the methods as explained above; however, due to lack of awareness, time, and financial and logistic constraint, the first two methods do not look feasible in the context of developing countries. Therefore, we recommend that vaccination with antibiotic treatment is the best way to control the disease which is in agreement with the result of [1]. Since the proportion to be vaccinated  $p_v$  and  $t$  are independent variables of  $\rho$ , a given value of  $\rho$  can have many practical interpretation. Therefore, practical implementation of the value of  $\rho$  can be adjusted based on availability, cost of control, and time value.

### Data Availability

Data are available in the literature.

### Disclosure

An earlier version of this paper was presented at the International Conference in Mathematical Methods and Models in Biosciences and Young Scientists, Biomath 2017, Skukuza Camp, Kruger Park, South Africa.

## Conflicts of Interest

The authors declare that they have no conflicts of interest.

## References

- [1] J. C. Mariner, J. McDermott, J. A. P. Heesterbeek, G. Thomson, P. L. Roeder, and S. W. Martin, "A heterogeneous population model for contagious bovine pleuropneumonia transmission and control in pastoral communities of East Africa," *Preventive Veterinary Medicine*, vol. 73, no. 1, pp. 75–91, 2006.
- [2] J. O. Onono, B. Wieland, and J. Rushton, "Estimation of impact of contagious bovine pleuropneumonia on pastoralists in Kenya," *Preventive Veterinary Medicine*, vol. 115, no. 3-4, pp. 122–129, 2014.
- [3] E. M. Vilei and J. Frey, "Detection of *Mycoplasma mycoides* subsp. *mycoides* SC in bronchoalveolar lavage fluids of cows based on a TaqMan real-time PCR discriminating wild type strains from an lppQ- mutant vaccine strain used for DIVA-strategies," *Journal of Microbiological Methods*, vol. 81, no. 3, pp. 211–218, 2010.
- [4] N. Abdela and N. Yune, "Seroprevalence and distribution of contagious bovine pleuropneumonia in ethiopia: update and critical analysis of 20 years (1996-2016) reports," *Frontiers in Veterinary Science*, vol. 4, article 100, 2017.
- [5] M. J. Otte, R. Nugent, and A. Mcleod, "Transboundary animal diseases: assessment of socio-economic impacts and institutional responses food and agriculture organization," Technical report, Transboundary animal diseases: Assessment of socio-economic impacts and institutional responses, FAO, Rome, Italy, 2004.
- [6] A. Ssematimba, J. Jores, and J. C. Mariner, "Mathematical modelling of the transmission dynamics of contagious bovine pleuropneumonia reveals minimal target profiles for improved vaccines and diagnostic assays," *PLoS ONE*, vol. 10, article e0116730, no. 2, 2015.
- [7] N. E. Tambi, W. O. Maina, and C. Ndi, "An estimation of the economic impact of contagious bovine pleuropneumonia in Africa," *Revue Scientifique Et Technique*, vol. 25, no. 3, pp. 999–1011, 2006.
- [8] M. Lesnoff, G. Laval, P. Bonnet, and A. Workalemahu, "A mathematical model of contagious bovine pleuropneumonia (CBPP) within-herd outbreaks for economic evaluation of local control strategies: An illustration from a mixed crop-livestock system in Ethiopian highlands," *Animal Research*, vol. 53, no. 5, pp. 429–438, 2004.
- [9] V. Dupuy, L. Manso-Silvan, V. Barbe et al., "Evolutionary History of Contagious Bovine Pleuropneumonia Using Next Generation Sequencing of *Mycoplasma mycoides* Subsp. *mycoides* "Small Colony"," *PLoS ONE*, vol. 7, no. 10, article e46821, 2012.
- [10] A. A. Aligaz and J. M. W. Munganga, "Analysis of a mathematical model of the dynamics of contagious bovine pleuropneumonia," in *Mathematical Methods and Models in Biosciences*, vol. 1, pp. 64–80, Biomath Forum, Sofia, Bulgaria, 2017.
- [11] J. C. Mariner, A. Araba, and S. Makungu, "Consultancy on the dynamics of CBPP endemism and the development of effective control/ eradication strategies for pastoral communities: final data collection report," in *The Community Animal Health and Participatory Epidemiology Unit of the African Union inter*, Africa Bureau for Animal Resources, Nairobi, Kenya, 2003.
- [12] P. van den Driessche and J. Watmough, "Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission," *Mathematical Biosciences*, vol. 180, pp. 29–48, 2002.
- [13] Z. Shuai and P. van den Driessche, "Global stability of infectious disease models using Lyapunov functions," *SIAM Journal on Applied Mathematics*, vol. 73, no. 4, pp. 1513–1532, 2013.
- [14] J. C. Mariner, J. McDermott, J. A. P. Heesterbeek, G. Thomson, and S. W. Martin, "A model of contagious bovine pleuropneumonia transmission dynamics in East Africa," *Preventive Veterinary Medicine*, vol. 73, no. 1, pp. 55–74, 2006.