Hindawi
Abstract and Applied Analysis
Volume 2024, Article ID 1072681, 16 pages
https://doi.org/10.1155/2024/1072681

Research Article

Mathematical Modeling of Coccidiosis Dynamics in Chickens with Some Control Strategies

Yustina A. Liana [1] **and Mary C. Swai** [2]

[1]*Department of Mathematics and ICT, College of Business Education, P.O. Box 1968, Dar-es-Salaam, Tanzania*
[2]*Department of Mathematics and ICT, Open University of Tanzania, P.O. Box 23409, Dar-es-Salaam, Tanzania*

Correspondence should be addressed to Yustina A. Liana; lianayustina@yahoo.com

Received 25 August 2023; Revised 22 December 2023; Accepted 20 January 2024; Published 5 February 2024

Academic Editor: Alberto Fiorenza

Coccidiosis is an infectious disease caused by the *Eimeria* species. The species can infect a bird's digestive system, severely slow down its growth, and is a serious economic burden for chickens. A mathematical model for the transmission dynamics of coccidiosis disease in chickens in the presence of control interventions has been formulated and analyzed to gain insights into the dynamics of the disease in the population. Three control interventions, namely vaccination, sanitation, and treatment, are implemented. The study intends to assess the effects of these control interventions in coccidiosis transmission dynamics. Using the theory of differential equations, the invariant set of the model was derived, and the model's solution was found to be mathematically and biologically significant. Analytical methods are employed to establish equilibrium solutions and investigate the stability of the model system's equilibria, while numerical simulations illustrate the analytical results. The effective reproduction number is obtained using the next-generation matrix method, and the local stability of the equilibria of the model is established. The disease-free equilibrium is proved to be locally stable when the effective reproduction number is less than unity. Also, the nature of the bifurcation and its implications for disease prevention are investigated through the application of the center manifold theory. On the other hand, sensitivity analysis is carried out to investigate the parameters that impact the transmission of coccidiosis disease using the normalized forward sensitivity index. The parameters that have a greater influence on the effective reproduction number should be targeted for control purposes to lessen the spread of disease. Furthermore, numerical simulation is performed to investigate the contribution of each control intervention.

1. Introduction

Coccidiosis is a poultry disease that is triggered by a protozoan parasite of the genus *Eimeria* that invades a bird's digestive tract and significantly slows its growth. Worldwide, the poultry industry is among the main suppliers of animal protein as it provides both eggs and meat [1]. Any pathogen that undermines the effectiveness of a poultry production sector could endanger world food security. The chicken can be affected by seven species of the genus *Eimeria*, each with different pathogenic properties and targeting a particular intestinal location. The seven species are *Eimeria acervulina, Eimeria brunetti, Eimeria, maxima, Eimeria mitis, Eimeria necatrix, Eimeria praecox, and Eimeria tenella*. The model of transmission begins with the ingestion of sporulated oocysts (the infection form of the parasite). Depending on the species, the infection can result in nutrient deficiencies, reduced rates of growth, and, in the case of the most pathogenic species, increased mortality. According to the literature, though seven species can affect chicken, *E. tenella* is considered to be the most pathogenic [2, 3]. The infected chicken has ruffled feathers and symptoms of depression or drowsiness. Furthermore, feed and water consumption are reduced, and feces are watery, whitish, and occasionally bloody. This causes dehydration, impaired weight gain, and, in the absence of treatment, death may occur [4, 5]. The risk of developing clinical coccidiosis is greatest between 1 and 3 weeks of chicken's life. Mortality and morbidity rates for chickens between the ages of 10 and 30 days and 35 and 60 days may exceed 80% [6]. Coccidiosis control and prevention are based on the use of vaccines, natural feed additives, prophylactic anticoccidial drugs, and improved farm handling practices. Some beneficial

practices include facility cleaning and disinfection, proper ventilation, and safe drinking water, all of which helped to contribute of keeping litter conditions that limit oocyst sporulation [7, 8].

To help identify crucial intervention points and reduce disease-related mortality, mathematical modeling has emerged as a crucial tool. Researchers can forecast a phenomenon's future by using mathematical models. For example, we can obtain a wealth of knowledge regarding the transmission dynamics, eradication, and future spread of infectious or viral diseases in society through the use of mathematical models and simulations of these diseases [9, 10]. Recently, few mathematical models have been made in the area of coccidiosis. For example, Kachanova et al. [11] conducted an experimental model of coccidiosis by *E. tenella* in broiler chicken. The findings showed that the infected chicken gained much less weight each day than the noninfected bird. The outcomes also demonstrated that the dose of infection affects the quantity of oocysts expelled with feces. Also, Zou et al. [6] construct a principle component analysis-based model for assessing chicken coccidiosis resistance. The findings suggested that a better parameter for assessing an individual biological resistance to coccidiosis illness may be the infection index (II). Further, Johnston et al. [12] developed a model of the chicken infection caused by *E. maxima* and *E. praecox* within the host. Assumptions about the quantity of available host cells, the average life expectancy of these cells, and the age structure inside the host-cell population were made and the mathematical models combined with experimental data to test whether these conditions could reproduce the crowding effect in the two species were studied. The findings revealed that, at very low infectious doses, experimental data indicated that crowding occurred during in vivo infections. However, none of the models could accurately simulate crowding at the same dosages while preserving accurate estimations of the dynamics of the enterocyte pool. Despite the studies that have been conducted to curb coccidiosis, the disease remains one of the most common parasite infections in the poultry. Coccidiosis causes an annual loss to the poultry business of over $3 billion worldwide, of which more than 70% is attributable to chickens' stunted growth and lower feed conversion rates [6, 13]. To the best of our knowledge, no mathematical model has attempted to examine the role of treatment, vaccination, and sanitation or cleaning of the environment as a control strategy in the transmission dynamics of coccidiosis disease. Vaccination is administered to protect the susceptible chicken from coccidiosis for a sufficient period. The current vaccines available in the market for coccidiosis pathogens include live virulent, live attenuated, and subunit vaccines [1]. Environmental hygiene and sanitation involve raising people's awareness about biosecurity measures to free the environment from coccidiosis pathogens. This is attained by ensuring cleanliness in the chicken's yard and its surroundings to prevent indirect transmission of the pathogens to the chicken from the unhygienic environment [7]. Therefore, relying on several clinical and epidemiological studies conducted on the transmission of coccidiosis disease in chickens, in this study, we developed a mathematical model to examine the role of treatment, vaccination, and sanitation in the transmission dynamics of coccidiosis in chickens. The implementation of these three control strategies will contribute to the reduction and possible elimination of coccidiosis in the chicken population. The current study will add up knowledge to the existing literature and provide a cornerstone for further research on poultry infectious diseases.

2. Model Description and Formulation

The chicken population at any time t, denoted by $N(t)$ is divided into five subpopulations based on the disease status as follows; susceptible chickens who are at risk of acquiring coccidiosis disease, $S(t)$, vaccinated chicken, $V(t)$, chicken infected with a coccidiosis disease who are capable of transmitting the infection to susceptible chicken, $I(t)$, and the recovered chicken, $R(t)$. The number of coccidiosis viruses in a contaminated environment is denoted by $H(t)$. It is assumed that chickens are recruited at a constant rate Λ. Susceptible chickens are vaccinated at rate τ in which vaccinated chickens who did not respond to vaccination wane out at the rate η and become susceptible again. It is also assumed that a susceptible chicken can be infected with coccidiosis if it comes into effective contact with an infectious chicken at a force of infection $\lambda = \beta I + \alpha H$, where β denotes transmission rate for infectious chicken and α denotes ingestion rate of coccidiosis pathogen by chicken. Infected chicken shed pathogens into the environment at a rate ϵ. The term pathogens refers to infectious agents that course infection and illness in the chickens. These pathogens are shed from an infected chicken through its feces, containing oocysts. These oocysts can spread and contaminate the environment including water, food, and litter [14]. A newly infected chicken can progress to the infectious chicken class at rate γ. Sanitation is very important as it leads to the death of the coccidiosis pathogen. By so doing, the number of infections may significantly be reduced. Furthermore, it is assumed that the infected chicken in the $I(t)$ compartment receives treatment at a rate θ. Moreover, all recovered chickens acquire temporary immunity, which wanes out at the rate ψ and becomes susceptible again. The disease-induced mortality occurs in the compartment, $I(t)$ at rate δ. The pathogens deplete naturally at a rate μ_h or by sanitation measures at the rate ρ. All chickens in different subgroups experience natural death at a rate μ. The model flow diagram is in Figure 1.

Putting together all assumptions and model descriptions, we obtain the following system of nonlinear differential equations:

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \lambda S - (\tau + \mu)S + \eta V + \psi R,\\
\frac{dV}{dt} &= \tau S - (\eta + \mu)V,\\
\frac{dE}{dt} &= \lambda S - (\gamma + \mu)E,\\
\frac{dI}{dt} &= \gamma E - (\theta + \epsilon + \mu + \delta)I,\\
\frac{dR}{dt} &= \theta I - (\psi + \mu)R,\\
\frac{dH}{dt} &= \epsilon I - (\rho + \mu_h)H
\end{aligned} \quad , \qquad (1)$$

where $\lambda = \beta I + \alpha H$.

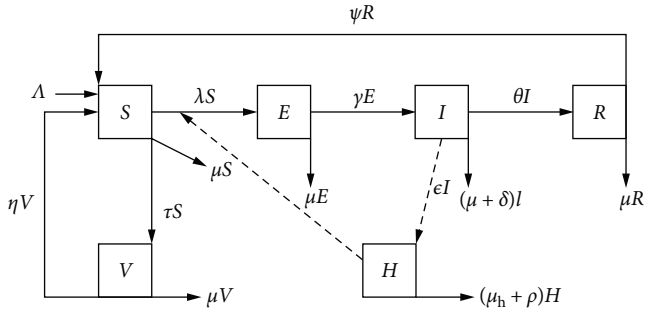

FIGURE 1: The schematic diagram for the transmission dynamics of coccidiosis in chicken population.

The initial conditions of the model system (1) are $S(0) \geq 0$, $V(0) \geq 0$, $E(0) \geq 0$, $I(0) \geq 0$, $R(0) \geq 0$, and $H(0) \geq 0$.

3. Model Analysis

3.1. Invariant Region. Since the model system (1) deals with human beings, it is assumed that all parameters and variables in the model are nonnegative for all $t \geq 0$. The discussion on the invariant region involves the description of the region in which the solution of the system makes epidemiological sense. We state and prove the following theorem.

Theorem 1. *The region* $\Omega = \{(S, V, E, I, R, H) \in \mathscr{R}_+^5 : N(t) \leq \frac{\Lambda}{\mu}\}$ *is positively invariant under the flow induced by the model system (1).*

Proof. Consider the population size: $N(t) = S(t) + V(t) + E(t) + I(t) + R(t) + H(t)$ and the rate of change given by:

$$\begin{aligned} \frac{dN}{dt} &= \frac{dS}{dt} + \frac{dV}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dR}{dt} + \frac{dH(t)}{dt}, \\ \Rightarrow \frac{dN}{dt} &= \Lambda - N\mu - (\delta I + \rho H) - \mu_h. \end{aligned} \tag{2}$$

From Equation (2) we have:

$$\frac{dN}{dt} \leq \Lambda - N\mu. \tag{3}$$

Solving Equation (3) for N yields $N \leq \frac{\Lambda}{\mu} + ce^{-ut}$ implying that $N \leq \frac{\Lambda}{\mu}$ as $t \to \infty$, where c is the constant of integration. Therefore, the feasible region solution set of the system enters the region Ω. Thus, the region Ω is a positively invariant set under the flow induced by the model and the model is well-posed and also it is biologically meaningful. □

3.2. Positivity of the Solutions. In this section, we describe the positivity of the solution of the model system.

Theorem 2. *The solution set* $\Omega = \left\{(S(t), V(t), E(t), I(t), R(t), H(t)) \in \mathscr{R}_+^5 : (S(0), V(0), E(0), I(0), R(0)H(0)) > 0\right\}$ *of the model system (1) is positive for all* $t \geq 0$.

Proof. Consider the first equation of the model system (1):

$$\frac{dS}{dt} = \Lambda + \eta V + \psi R - (\beta I + \alpha H + \tau + \mu)S. \tag{4}$$

It follows that

$$\frac{dS}{dt} \geq -(\beta I + \alpha H + \tau + \mu)S. \tag{5}$$

Integrating Equation (5) by separation of variables, we obtain:

$$\ln S(t) \geq -(\beta I + \alpha H + \tau + \mu)t + K, \tag{6}$$

where K is the constant of integration.

Applying the initial conditions $S(0)$, in Equation (6) gives:

$$S(t) \geq S(0)e^{-(\beta I + \alpha H + \tau + \mu)t} > 0 \text{ provided } (\beta I + \alpha H + \tau + \mu) < \infty. \tag{7}$$

Similarly, the remaining other equations in the model system (1) give the following results:

$$\begin{aligned} &V(t) \geq V(0)e^{-(\eta+\mu)t} > 0 \text{ provided } (\eta + \mu) < \infty, \\ &E(t) \geq E(0)e^{-(\gamma+\mu)t} > 0 \text{ provided } (\gamma + \mu) < \infty, \\ &I(t) \geq I(0)e^{-(\theta+\epsilon+\mu+\delta)t} > 0 \text{ provided } ((\theta + \epsilon + \mu + \delta) < \infty, \\ &R(t) \geq R(0)e^{-(\psi+\mu)t} > 0 \text{ provided } (\psi + \mu) < \infty, \\ &H(t) \geq H(0)e^{-(\rho+\mu_h)t} > 0 \text{ provided } (\rho + \mu_h) < \infty. \end{aligned} \tag{8}$$

Therefore, the solutions of the model are positive for all values of $t > 0$. □

3.3. Existence of Equilibrium. In this part, we shall establish the equilibrium point. To find the existence equilibrium of the model, the right-hand side of the model system (1) is equated to zero. Thus, we have the following model system:

$$\begin{cases} 0 = \Lambda - \lambda S - (\tau + \mu)S + \eta V + \psi R, \\ 0 = \tau S - (\eta + \mu)V, \\ 0 = \lambda S - (\gamma + \mu)E, \\ 0 = \gamma E - (\theta + \epsilon + \mu + \delta)I, \\ 0 = \theta I - (\psi + \mu)R, \\ 0 = \epsilon I - (\rho + \mu_h)H, \end{cases} \tag{9}$$

given that

$$\lambda = \beta I + \alpha H. \tag{10}$$

The equilibrium of the model system is therefore given by $E_* = S^*, V^*, E^*, I^*, R^*, H^*$ as follows:

$$
\begin{aligned}
S^* &= -\frac{a_2a_6(-a_5\Lambda + I^*\theta\psi)}{a_5(I^*\alpha a_2\epsilon + I^*a_2a_6\beta + a_6(-\eta)\tau + a_1a_2a_6)},\\
V^* &= -\frac{a_6\tau(-a_5\Lambda + I^*\theta\psi)}{a_5(I^*\alpha a_2\epsilon + I^*a_2a_6\beta + a_6(-\eta)\tau + a_1a_2a_6)},\\
E^* &= -\frac{I^*a_2(a_6\beta + \alpha\epsilon)(-a_5\Lambda + I^*\theta\psi)}{a_3a_5(-a_6\eta\tau + a_2(a_6(a_1 + I^*\beta) + I^*\alpha\epsilon))}\\
I^* &= \frac{E^*\gamma}{a_4},\\
R^* &= -\frac{I^*\theta}{a_5},\\
H &= \frac{I^*\epsilon}{a_6},
\end{aligned}
\tag{11}
$$

where

$$
\begin{aligned}
a_1 &= \tau + \mu, a_2 = \eta + \mu, a_3 = \gamma + \mu, a_4 = \theta + \epsilon + \mu + \delta,\\
a_5 &= \psi + \mu, a_6 = \rho + \mu_h.
\end{aligned}
\tag{12}
$$

Then, substituting E^* into I^* from Equation (11), we obtain:

$$
I^*(AI^* + B) = 0, \tag{13}
$$

where

$$
\begin{aligned}
A &= \alpha a_2\gamma\theta\psi\epsilon + \alpha a_3a_4a_5a_2\epsilon + a_6a_2\beta\gamma\theta\psi + a_3a_4a_5a_6a_2\beta,\\
B &= -\alpha a_2a_5\gamma\Lambda\epsilon - a_2a_6a_5\beta\gamma\Lambda - a_3a_4a_6a_5\eta\tau + a_1a_2a_3a_4a_6a_5
\end{aligned}.
\tag{14}
$$

Now, solving Equation (13), we obtain one of the solution is $I^* = 0$, which represent the existence of disease-free equilibrium (DFE) and the remaining equation as follows:

$$
AI^* + B = 0, \tag{15}
$$

indicate the existence of the endemic equilibrium point.

3.4. Disease-Free Equilibrium (DFE) Point. DFE is obtained in the absence of infection: It always exists when $I = 0$. Substitute $I = 0$ into equations in model system (9) to get:

$$
\begin{aligned}
E^0 &= R^0 = H^0 = 0,\\
S^0 &= \frac{\Lambda a_2}{a_1a_2 - \eta\tau},\\
V^0 &= \frac{\tau\Lambda}{a_1a_2 - \eta\tau}.
\end{aligned}
\tag{16}
$$

Therefore, the DFE is given by:

$$
E^0 = (S^0, V^0, E^0, I^0, R^0, H^0) = \left(\frac{\Lambda a_2}{a_1a_2 - \eta\tau}, \frac{\tau\Lambda}{a_1a_2 - \eta\tau}, 0, 0, 0, 0\right). \tag{17}
$$

3.5. The Effective Reproduction Number. The reproduction number that is used to investigate whether an infection introduced into a population will be eliminated or become endemic is obtained. It is defined as the expected number of secondary cases produced in a completely susceptible population by a typical infectious individual during its period of infectiousness [15, 16].

Following the ideas in [15, 17–19], the effective reproduction number R_e, is obtained by using the next-generation matrix method, which is given by the next-generation matrix's spectral radius. We can rewrite the equations in the model system (1) starting with infectious classes first and then the rest follow. This leads to the following system:

$$
\begin{aligned}
\frac{dE}{dt} &= \lambda S - a_3E,\\
\frac{dI}{dt} &= \gamma E - a_4I,\\
\frac{dH}{dt} &= \epsilon I - a_6H,\\
\frac{dS}{dt} &= \Lambda - \lambda S - a_1S + \eta V + \phi R,\\
\frac{dV}{dt} &= \tau S - a_2V,\\
\frac{dR}{dt} &= \theta I - a_5R.
\end{aligned}
\tag{18}
$$

From system (18) F_i and V_i are obtained as follows:

$$
F_i = \begin{bmatrix} \lambda S\\ 0\\ 0 \end{bmatrix}, \tag{19}
$$

where $\lambda = \beta I + \alpha H$ and

$$
V_i = \begin{bmatrix} a_3E\\ a_4I - \gamma E\\ a_6H - \epsilon I \end{bmatrix}. \tag{20}
$$

The partial differentiation of F_i and V_i with respect to E, I, and H gives:

$$
DF_i = \begin{bmatrix} 0 & \beta S & \alpha S\\ 0 & 0 & 0\\ 0 & 0 & 0 \end{bmatrix}, DV_i = \begin{bmatrix} a_3 & 0 & 0\\ -\gamma & a_4 & 0\\ 0 & -\epsilon & a_6 \end{bmatrix}. \tag{21}
$$

At DFE point (by substituting E^0), we get:

$$F = \begin{bmatrix} 0 & \beta S^0 & \alpha S^0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix},$$
$$V = \begin{bmatrix} a_3 & 0 & 0 \\ -\gamma & a_4 & 0 \\ 0 & -\epsilon & a_6 \end{bmatrix}. \quad (22)$$
$$V^{-1} = \begin{bmatrix} \frac{1}{a_3} & 0 & 0 \\ \frac{\gamma}{a_3 a_4} & \frac{1}{a_4} & 0 \\ \frac{\gamma\epsilon}{a_3 a_4 a_6} & \frac{\epsilon}{a_4 a_6} & \frac{1}{a_6} \end{bmatrix}.$$

Thus, FV^{-1} is computed and given as follows:

$$FV^{-1} = \begin{bmatrix} \frac{(\beta a_6 + \alpha\epsilon)\gamma S^0}{a_3 a_4 a_6} & \frac{(\beta a_6 + \alpha\epsilon)S^0}{a_4 a_6} & \frac{\alpha S^0}{a_6} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}. \quad (23)$$

Therefore, the eigenvalues of matrix FV^{-1} are $\lambda_1 = 0$, $\lambda_2 = 0$, and $\lambda_3 = (\beta a_6 + \alpha\epsilon)\gamma S^0 / a_3 a_4 a_6$. Thus, the effective reproduction number is as follows:

$$R_e = \frac{(\beta a_6 + \alpha\epsilon)\gamma S^0}{a_3 a_4 a_6}, \quad (24)$$

where $S^0 = \Lambda a_2 / a_1 a_2 - \eta\tau$.

3.6. Endemic Equilibrium Point. The endemic equilibrium point is obtained when coccidiosis exists in the population. In this case, the endemic equilibrium point is obtained by solving Equation (15) and get:

$$I^* = \frac{a_1 a_2 a_3 a_4 a_5 a_6 (R_e - 1)}{(a_6\beta + \alpha\epsilon)(a_2\gamma\theta\psi + a_3 a_4 a_5)}. \quad (25)$$

Then, substituting Equation (28) to Equation (11) the endemic equilibrium $E_* = (S^*, V^*, E^*, I^*, R^*, H^*)$, is explicitly given as follows:

$$\begin{aligned}
S^* &= \frac{a_3 a_4 a_6}{a_6\beta\gamma + \alpha\gamma\epsilon}, \\
V^* &= \frac{a_3 a_4 a_6 \tau}{\alpha a_2 \gamma\epsilon + a_6 a_2 \beta\gamma}, \\
E^* &= \frac{a_1 a_2^2 a_4 a_5 (R_e - 1)(a_3 a_4 a_5 \Lambda(a_6\beta + \alpha\epsilon) + a_2\theta\psi(\alpha\gamma\Lambda\epsilon + a_6(\beta\gamma\Lambda - a_1 a_3 a_4)))}{(a_6\beta + \alpha\epsilon)(a_2\gamma\theta\psi + a_3 a_4 a_5)(a_2\gamma\theta\psi(a_1 a_2 - \eta\tau) + a_3 a_4 a_5(a_1 a_2 - \eta\tau))}, \\
I^* &= \frac{a_1 a_2 a_3 a_4 a_5 a_6 (R_e - 1)}{(a_6\beta + \alpha\epsilon)(a_2\gamma\theta\psi + a_3 a_4 a_5)}, \\
R^* &= -\frac{\theta(\alpha a_2\gamma\Lambda\epsilon + a_6 a_2\beta\gamma\Lambda + a_3 a_4 a_6\eta\tau - a_1 a_3 a_4 a_6 a_2)}{a_2(a_6\beta + \alpha\epsilon)(a_3 a_4 a_5 + \gamma\theta\psi)}, \\
H^* &= \frac{a_1 a_2 a_3 a_4 a_5 \epsilon (R_e - 1)}{(a_6\beta + \alpha\epsilon)(a_2\gamma\theta\psi + a_3 a_4 a_5)}.
\end{aligned} \quad (26)$$

Therefore, it can be noted that the model system (1) has a unique endemic equilibrium point when $R_e > 1$.

3.7. Local Stability of the DFE. Here, we investigate the local stability of the DFE of the model system (1). For local stability, the spread of infection depends on the initial size of the subpopulation. To prove the local stability of the DFE, the eigenvalues of the Jacobian matrix of the system computed at the DFE point are obtained. The Jacobian matrix is obtained from the linearization of the model system (1). The Jacobian matrix J evaluated at DFE point, (E_0) is given by:

$$J = \begin{bmatrix} -(a_1) & \eta & 0 & -\beta S^0 & \eta & -\alpha S^0 \\ \tau & -a_2 & 0 & 0 & 0 & 0 \\ 0 & 0 & -a_3 & \beta S^0 & 0 & 0 \\ 0 & 0 & \gamma & -a_4 & 0 & 0 \\ 0 & 0 & 0 & \theta & -a_5 & 0 \\ 0 & 0 & 0 & \epsilon & 0 & -a_6 \end{bmatrix}, \quad (27)$$

where a_1, a_2, a_3, a_4, a_5, and a_6 have the same meaning as in Equation (12) The DFE point is stable if all eigenvalues of the

Jacobian matrix at the DFE point are negative. The eigenvalues of the Jacobian matrix (27) are established from the characteristic equation $|J - \lambda I| = 0$. To obtain the eigenvalues of Equation (27):

$$J(E_0) = \begin{bmatrix} -(a_1) - \lambda & \eta & 0 & -\beta S^0 & \varphi & -\alpha S^0 \\ \tau & -a_2 - \lambda & 0 & 0 & 0 & 0 \\ 0 & 0 & -a_3 - \lambda & \beta S^0 & 0 & \alpha S^0 \\ 0 & 0 & \gamma & -a_4 - \lambda & 0 & 0 \\ 0 & 0 & 0 & \theta & -a_{5-\lambda} & 0 \\ 0 & 0 & 0 & \epsilon & 0 & -a_6 - \lambda \end{bmatrix}. \tag{28}$$

The model system (28) can be written as follows:

$$\begin{aligned} J(E_0) = (-a_1 - \lambda) & \begin{bmatrix} -a_2 - \lambda & 0 & 0 & 0 & 0 \\ 0 & -a_3 - \lambda & \beta S^0 & 0 & \alpha S^0 \\ 0 & \gamma & -a_4 - \lambda & 0 & 0 \\ 0 & 0 & \theta & -a_{5-\lambda} & 0 \\ 0 & 0 & \epsilon & 0 & -a_6 - \lambda \end{bmatrix} \\ -\tau & \begin{bmatrix} \eta & 0 & -\beta S^0 & \varphi & -\alpha S^0 \\ 0 & -a_3 - \lambda & \beta S^0 & 0 & \alpha S^0 \\ 0 & \gamma & -a_4 - \lambda & 0 & 0 \\ 0 & 0 & \theta & -a_{5-\lambda} & 0 \\ 0 & 0 & \epsilon & 0 & -a_6 - \lambda \end{bmatrix} \\ = [(-a_1 - \lambda)(-a_2 - \lambda)] & \begin{bmatrix} -a_3 - \lambda & \beta S^0 & 0 & \alpha S^0 \\ \gamma & -a_4 - \lambda & 0 & 0 \\ 0 & \theta & -a_{5-\lambda} & 0 \\ 0 & \epsilon & 0 & -a_6 - \lambda \end{bmatrix} \\ -\tau\eta & \begin{bmatrix} -a_3 - \lambda & \beta S^0 & 0 & \alpha S^0 \\ \gamma & -a_4 - \lambda & 0 & 0 \\ 0 & \theta & -a_{5-\lambda} & 0 \\ 0 & \epsilon & 0 & -a_6 - \lambda \end{bmatrix} \end{aligned}. \tag{29}$$

It follows that:

$$J(E_0) = [(-a_1 - \lambda)(-a_2 - \lambda) - \tau\eta](-a_5 - \lambda) \begin{bmatrix} -a_3 - \lambda & \beta S^0 & \alpha S^0 \\ \gamma & -a_4 - \lambda & 0 \\ 0 & \epsilon & -a_6 - \lambda \end{bmatrix}. \tag{30}$$

From system (30), we have negative eigenvalue:

$$\lambda_1 = -a_5, \tag{31}$$

the polynomial equation

$$P_1(\lambda) = (-a_1 - \lambda)(-a_2 - \lambda) - \tau\eta, \tag{32}$$

and matrix

$$J_2(E_0) = \begin{bmatrix} -a_3 - \lambda & \beta S^0 & \alpha S^0 \\ \gamma & -a_4 - \lambda & 0 \\ 0 & \epsilon & -a_6 - \lambda \end{bmatrix}. \quad (33)$$

Simplify the polynomial Equation (32) to obtain:

$$P_1(\lambda) = \lambda^2 + (a_1 + a_2)\lambda + a_1 a_2 - \tau\eta\ . \quad (34)$$

By Routh–Hurwitz criteria [16] the polynomial Equation (34) have negative root if $a_1 a_2 > \tau\eta$. Furthermore, the two eigenvalues λ_2 and λ_3 in Equation (34) can be rewritten as follows:

$$\begin{aligned} \lambda_2 &= \frac{-(a_1 + a_2) + \sqrt{a_1^2 + a_2^2 + 2(2\tau\eta - a_1 a_2)}}{2} \\ \lambda_3 &= \frac{-(a_1 + a_2) - \sqrt{a_1^2 + a_2^2 + 2(2\tau\eta - a_1 a_2)}}{2} \end{aligned}. \quad (35)$$

The rest of the eigenvalues are the roots of the polynomial obtained in Jacobian matrix (33) which is given by:

$$\lambda^3 + C_2\lambda^2 + C_1\lambda + C_0 = 0, \quad (36)$$

where

$$\begin{aligned} C_0 &= a_3 a_4 a_6 - (\epsilon\alpha + \beta a_6)\gamma S^0, \\ C_1 &= a_6(a_3 + a_4) + a_3 a_4, \\ C_2 &= a_3 + a_4 + a_6, \\ S^0 &= \frac{\Lambda a_2}{a_1 a_2 - \eta\tau}. \end{aligned} \quad (37)$$

By Routh–Hurwitz criteria the third-order polynomial Equation (36) has all roots in the open left half-plane if C_0, C_1, C_2 are positive and $C_1 C_2 > C_0$ [16]. It is clear that C_2 is positive, and C_1 is positive if $a_6(a_3 + a_4) + a_3 a_4 > \beta\gamma S^0$. Thus, we need to see the condition for $C_0 > 0$.

$$\begin{aligned} C_0 &= a_3 a_4 a_6 - (\epsilon\alpha + \beta a_6)\gamma S^0, \\ C_0 &= a_3 a_4 a_6 \left(1 - \frac{(\epsilon\alpha + \beta a_6)\gamma S^0}{a_3 a_4 a_6}\right), \\ C_0 &= a_3 a_4 a_6 \left(1 - \left[\frac{(\epsilon\alpha + \beta a_6)\gamma a_2 \Lambda}{a_3 a_4 a_6 (a_1 a_2 - \eta\tau)}\right]\right), \\ C_0 &= a_3 a_4 a_6 (1 - R_e). \end{aligned} \quad (38)$$

From Equation (38), C_0 is positive if $R_e < 1$. Hence, we have the following result.

Theorem 3. *The DFE point is locally asymptotically stable if $R_e < 1$ and unstable if the inequality is reversed.*

3.8. Global Stability of DFE. For global stability, the spread of the infection is independent of the initial size of the population. By using the comparison method [20], we establish the following theorem.

Theorem 4. *The DFE is globally asymptotically stable if $R_e < 1$ and unstable if the inequality is reversed.*

Proof. Let F and V be the Jacobian matrices of F_i and V_i defined as $F = [\frac{\partial F_i}{\partial x_j}(x_0)]$ and $V = [\frac{\partial V_i}{\partial x_j}(x_0)]$; $1 \le i, j \le m$, where x is the number of individuals in each compartment, x_0 is a DFE, F_i is the rate of appearance of new infections in compartment i, $V_i = V_i^- - V_i^+$, in which V_i^+ is the transfer rate of individuals into compartment i and V_i^- is the rate of transfer of individuals out of compartment i. The rate of change of variables representing the infected components of the model system (1) can be rewritten as follows:

$$\begin{bmatrix} E'(t) \\ I'(t) \\ H'(t) \end{bmatrix} = (F - V)\begin{bmatrix} E(t) \\ I(t) \\ H(t) \end{bmatrix} - \begin{bmatrix} (\beta I + \alpha H)S^0\left(1 - \frac{S}{S^0}\right) \\ 0 \\ 0 \end{bmatrix} \begin{bmatrix} E(t) \\ I(t) \\ H(t) \end{bmatrix}, \quad (39)$$

implying that

$$\begin{bmatrix} E'(t) \\ I'(t) \\ H'(t) \end{bmatrix} \le (F - V)\begin{bmatrix} E(t) \\ I(t) \\ H(t) \end{bmatrix}, \quad (40)$$

where the matrices F and V are defined as follows:

$$F = \begin{bmatrix} 0 & \beta S^0 & \alpha S^0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}, \quad (41)$$

$$V = \begin{bmatrix} a_3 & 0 & 0 \\ -\gamma & a_4 & 0 \\ 0 & -\epsilon & a_6 \end{bmatrix}, \quad (42)$$

where a_3, a_4, a_6, and S^0 have the same meaning as it is in Equations (12) and (37), respectively.

The matrix $(F - V)$ is given by:

$$J_2(G) = \begin{bmatrix} -a_3 & \beta S^0 & \alpha S^0 \\ \gamma & -a_4 & 0 \\ 0 & \epsilon & -a_6 \end{bmatrix}. \quad (43)$$

The eigenvalues of matrix (43) are established from the characteristic equation $|J_2(G) - \lambda I| = 0$.

$$J_2(G) - \lambda I = \begin{bmatrix} -a_3 - \lambda & \beta S^0 & \alpha S^0 \\ \gamma & -a_4 - \lambda & 0 \\ 0 & \epsilon & -a_6 - \lambda \end{bmatrix}. \quad (44)$$

The polynomial obtained in matrix (44) is given by:

$$\lambda^3 + D_2\lambda^2 + D_1\lambda + D_0 = 0, \quad (45)$$

where

$$\begin{aligned} D_2 &= a_3 + a_4 + a_6, \\ D_1 &= a_6(a_3 + a_6) + a_3a_4 - \beta\gamma S^0, \\ D_0 &= a_3a_4a_6 - (\epsilon\alpha + \beta a_6)\gamma S^0, \\ &= a_3a_4a_6\left(1 - \frac{(\epsilon\alpha + \beta a_6)\gamma S^0}{a_3a_4a_6}\right) \\ &= a_3a_4a_6(1 - R_e). \end{aligned} \quad (46)$$

By Routh–Hurwitz criteria, the third-order polynomial Equation (45) has all roots in the open left half-plane if D_0, D_1, D_2 are positive and $D_1D_2 > D_0$ [16]. It is clear that D_2 is positive, and D_1 is positive if $a_6(a_3 + a_4) + a_3a_4 > \beta\gamma S^0$. Also, from Equation (46) D_0 is positive if $R_e < 1$. This result signifies that the eigenvalues of matrix $(F - V)$ have negative real parts if $R_e < 1$. Therefore, system (1) is stable for $R_e < 1$. It follows that $(E^0, I^0, H^0) \to (0, 0, 0)$ as $t \to \infty$. Furthermore, examining the first, second, third, fourth, fifth, and sixth equations of system (9), gives $S = \Lambda a_2/a_1a_2 - \eta\tau$, and $V = \tau\Lambda/a_1a_2 - \eta\tau$ whenever $E^0 = I^0 = H^0 = 0$. Therefore, by employing the comparison method [21] it follows that $(S^0, V^0, E^0, I^0, R^0) \to (\Lambda a_2/a_1a_2 - \eta\tau, \tau\Lambda/a_1a_2 - \eta\tau, 0, 0, 0, 0)$ as $t \to \infty$ for $R_e < 1$. Hence, E_0 is globally asymptotically stable whenever $R_e < 1$. Thus, the system will come to a DFE point from any starting point. □

3.8.1. The Impact of Intervention Strategies. The effects of implementing intervention strategies are investigated. Since, the interventions have been invested in controlling pneumonia disease, it is very important to understand the benefits of implementing these interventions. The three intervention strategies' contribution is observed by comparing the basic reproduction number and the effective reproduction number.

Consider the reproduction number in the presence of all three interventions as presented in Equation (47):

$$R_e = \frac{(\beta a_6 + \alpha\epsilon)\gamma S^0}{a_3a_4a_6}, \quad (47)$$

where

$$\begin{aligned} S^0 &= \frac{\Lambda a_2}{a_1a_2 - \eta\tau} \\ a_1 &= \tau + \mu, \\ a_2 &= \eta + \mu, \\ a_3 &= \gamma + \mu, \\ a_4 &= \theta + \epsilon + \mu + \delta, \\ a_6 &= \rho + \mu_h. \end{aligned} \quad (48)$$

Substituting Equation (48) in Equation (47) gives:

$$\begin{aligned} R_e &= \frac{(\beta(\rho + \mu_h) + \alpha\epsilon)\gamma \frac{\Lambda(\eta+\mu)}{(\tau+\mu)(\eta+\mu)-\eta\tau}}{(\gamma + \mu)(\theta + \epsilon + \mu + \delta)(\rho + \mu_h)} \\ &= \frac{(\beta(\rho + \mu_h) + \alpha\epsilon)\gamma\Lambda(\eta + \mu)}{((\tau + \mu)(\eta + \mu) - \eta\tau)(\gamma + \mu)(\theta + \epsilon + \mu + \delta)(\rho + \mu_h)}, \\ &= \frac{\gamma\Lambda(\eta + \mu)(\beta(\rho + \mu_h) + \alpha\epsilon)}{\mu(\tau + \mu + \eta)(\gamma + \mu)(\theta + \epsilon + \mu + \delta)(\rho + \mu_h)}. \end{aligned} \quad (49)$$

The basic reproduction number (the reproduction number of the model in the absence of interventions) is given by:

$$R_0 = \frac{\gamma\Lambda(\beta\mu_h + \alpha\epsilon)}{\mu(\gamma + \mu)(\epsilon + \mu + \delta)\mu_h}. \quad (50)$$

Then, the reproduction number (R_e) in the presence of all three interventions can be presented as follows:

$$R_e = R_0k, \quad (51)$$

where

$$k = \frac{\mu_h(\eta + \mu)(\epsilon + \mu + \delta)(\beta(\rho + \mu_h) + \alpha\epsilon)}{(\rho + \mu_h)(\tau + \mu + \eta)(\theta + \epsilon + \mu + \delta)(\beta\mu_h + \alpha\epsilon)}. \quad (52)$$

In Equation (52),

$$\begin{aligned} \mu_h &< (\rho + \mu_h), \\ (\eta + \mu) &< (\tau + \mu + \eta), \\ (\epsilon + \mu + \delta) &< (\theta + \epsilon + \mu + \delta), \end{aligned} \quad (53)$$

This indicates that the value of the numerator is less than the value of the denominator. Consequently, it implies that $k < 1$. This demonstrates that vaccination, treatment, and sanitation effectively curb initial disease transmission and halt-epidemic spread.

3.9. Bifurcation Analysis. The presence of forward or backward bifurcations has a significant impact on infectious disease epidemiological control efforts. The bifurcation analysis of the equilibrium points determines whether the disease can be entirely eradicated or whether it will remain in the population. The bifurcation analysis is carried out at the DFE using the center manifold theory proposed by Castillo-Chavez and Song [22]. This is accomplished by renaming the model's state variables of the model system (1) as follows: let $x_1 = S$, $x_2 = V$, $x_3 = E$, $x_4 = I$, $X_5 = R$, and $x_6 = H$ and the system can be written in the form of:

$$\frac{dX_i}{dt} = F(X_i), \tag{54}$$

where $X_i = (x_1, x_2, ..., x_6)^T$, $F = (f_1, f_2, ..., f_6)^T$, and $(.)^T$ denote a matrix transpose. The model system (1) takes the form:

$$\begin{aligned}
\frac{dx_1}{dt} &= f_1 = \Lambda - \beta x_1 x_4 - \alpha x_1 x_6 - a_1 x_1 - \eta x_3 + \psi x_5,\\
\frac{dx_2}{dt} &= f_2 = \tau x_1 - a_2 x_2,\\
\frac{dx_3}{dt} &= f_3 = \beta x_1 x_4 + \alpha x_1 x_6 - a_3 x_3,\\
\frac{dx_4}{dt} &= f_4 = \gamma x_3 - a_4 x_4,\\
\frac{dx_5}{dt} &= f_5 = \theta x_4 - a_5 x_5,\\
\frac{dx_6}{dt} &= f_6 = \epsilon x_4 - a_6 x_6.
\end{aligned} \tag{55}$$

Let β^* be the bifurcation parameter, then, system (55) is linearized at a DFE point when $\beta = \beta^*$ with $R_e = 1$. Hence, solving for β^* from $R_e = 1$ in Equation (47) gives:

$$\beta^* = \frac{a_3 a_4 a_6 (a_1 a_2 - \eta\tau)}{a_2 \Lambda \gamma (a_6 + \epsilon\alpha)}. \tag{56}$$

Then, the linearized system (55) is transformed with $\beta = \beta^*$ which has a simple zero eigenvalue, and center manifold theory is used to analyze the dynamics of Equation (55) near $\beta = \beta^*$. Thus, the Jacobian matrix of system (55) at DFE E_0 denoted by $J(\beta^*)$ is given by:

$$J(b^*) = \begin{pmatrix}
-a_1 & \eta & 0 & \beta^* k_1 & \theta & -\alpha k_1\\
\tau & -a_2 & 0 & 0 & 0 & 0\\
0 & 0 & -a_3 & \beta^* k_1 & 0 & \alpha k_1\\
0 & 0 & \gamma & -a_4 & 0 & 0\\
0 & 0 & 0 & \theta & -a_5 & 0\\
0 & 0 & 0 & \epsilon & -a_6 & 0
\end{pmatrix}, \tag{57}$$

where $k_1 = \Lambda a_2 / a_1 a_2 - \eta\tau$.

Then, the right and left eigenvectors corresponding with the zero eigenvalues are now computed. $\omega = [\omega_1, \omega_2, \omega_3, \omega_4, \omega_5, \omega_6]T$ gives the right eigenvector associated with the zero eigenvalue, and the following equations are obtained:

$$\begin{cases}
-a_1\omega_1 + \eta\omega_2 + \beta^* k_1 \omega_4 + \theta\omega_5 - \alpha k_1 \omega_6 = 0,\\
\tau\omega_1 - a_2\omega_2 = 0,\\
-a_3\omega_3 + \beta^* k_1 \omega_4 + \alpha k_1 \omega_6 = 0,\\
\gamma\omega_3 - a_4\omega_4 = 0,\\
\theta\omega_4 - a_5\omega_5 = 0,\\
\epsilon\omega_4 - a_6\omega_5 = 0.
\end{cases} \tag{58}$$

Solving system (58) for $\omega_{i's}$, $i = 1, 2, ..., 6$, gives the following right eigenvectors:

$$\begin{aligned}
\omega_1 &= \left[\frac{\alpha k_1 a_6}{\eta\alpha k_1 \epsilon}\left(\frac{a_3 a_4 - \gamma\beta^* k_1}{\gamma}\right) - \frac{\beta^* k_1 a_6}{\tau\epsilon} + \frac{\theta}{\eta}\right]\frac{\eta a_2 \omega_5}{\eta\tau - a_1 a_2},\\
\omega_2 &= \left[\frac{\alpha k_1 a_6}{\eta\alpha k_1 \epsilon}\left(\frac{a_3 a_4 - \gamma\beta^* k_1}{\gamma}\right) - \frac{\beta^* k_1 a_6}{\tau\epsilon} + \frac{\theta}{\eta}\right]\frac{\eta\tau\omega_5}{\eta\tau - a_1 a_2},\\
\omega_3 &= \frac{a_4 a_6 \omega_5}{\gamma\epsilon},\\
\omega_4 &= \frac{a_6\omega_5}{\epsilon},\\
\omega_6 &= \left[\frac{a_3 a_4}{\gamma} - \alpha k_1\right]\frac{a_6\omega_5}{\alpha k_1 \epsilon},\\
\omega_5 &= \omega_5 > 0.
\end{aligned} \tag{59}$$

Furthermore, to calculate the left eigenvectors given by $v = [v_1, v_2, v_3, v_4, v_5, v_6]^T$ which satisfy $v.\omega = 1$, the matrix $J(\beta^*)$ should be transposed and becomes:

$$J(b^*)^T = \begin{pmatrix}
-a_1 & \tau & 0 & 0 & 0 & 0\\
\eta & -a_2 & 0 & 0 & 0 & 0\\
0 & 0 & -a_3 & \gamma & 0 & 0\\
\beta^* k_1 & 0 & \beta^* k_1 & -a_4 & \theta & \epsilon\\
\theta & 0 & 0 & 0 & -a_5 & -a_6\\
-\alpha k_1 & 0 & \alpha k_1 & 0 & 0 & 0
\end{pmatrix}. \tag{60}$$

Then, solving $J(b^*)^T$, the following equations are obtained:

$$\begin{cases}
-a_1 v_1 + \tau v_2 = 0,\\
\eta v_1 - a_2 v_2 = 0,\\
-a_3 v_3 + \gamma v_4 = 0,\\
\beta^* k_1 v_1 + \beta^* k_1 v_2 - a_4 v_4 + \theta v_5 + \epsilon v_6 = 0,\\
-\alpha k_1 v_1 + \alpha k_1 v_3 = 0.
\end{cases} \tag{61}$$

Solving system (61) for $v_{i's}$, $i = 1, 2, ..., 9$, the following left eigenvectors are obtained:

$$\begin{aligned}
\nu_1 &= \nu_1 > 0,\\
\nu_2 &= \frac{\eta\nu_1}{a_2},\\
\nu_3 &= \nu_1,\\
\nu_4 &= \left[\frac{\theta}{a_5} - \left(\frac{\beta^* k_1\eta}{a_2} - \frac{a_4a_3}{\gamma} + \frac{\theta^2}{a_5}\right)\frac{a_6}{\theta a_6 - \epsilon a_5}\right]\nu_1,\\
\nu_5 &= \left[\beta^* k_1 + \frac{\beta^* k_1\eta}{a_2} - \frac{a_4a_3}{\gamma}\right]\frac{a_5\nu_1}{\theta a_6 - \epsilon a_5}.
\end{aligned} \tag{62}$$

To establish the conditions for the occurrence of forward or backward bifurcations, the Castillo-Chavez and Song Theorem 4.1 in [22] is used. The theorem is provided below for easy reference since it is important for providing the local stability of the endemic equilibrium point around $R_0 = 1$.

Theorem 5. *Consider the following general system of ordinary differential equations with a parameter ϕ such that*

$$\frac{dx}{dt} = f(x,\phi), f:\mathbb{R}^n\times\mathbb{R}\to\mathbb{R} \text{ and } f\in\mathbb{C}^2(\mathbb{R}^n\times\mathbb{R}), \tag{63}$$

where 0 is an equilibrium point of the system (that is, $f(0,\phi) \equiv 0$ for all ϕ) and

(1) *$A = D_x f(0,0) = (\frac{\partial f_i}{\partial x_i}(0,0))$ is the linearization matrix of system (63) around the equilibrium 0 with ϕ evaluated at 0.*
(2) *Zero is a simple eigenvalue of A, and all other eigenvalues of A have negative real parts.*
(3) *Matrix A has a nonnegative right eigenvector ω and a left eigenvector v corresponding to the zero eigenvalue.*

Let f_k be the k^{th} components of f and

$$\begin{aligned}
a &= \sum_{k,i,j=1}^{n} \nu_k\omega_i\omega_j \frac{\partial^2 f_k}{\partial x_i\partial x_j}(0,0),\\
b &= \sum_{k,i=1}^{n} \nu_k\omega_i \frac{\partial^2 f_k}{\partial x_i\partial\phi}(0,0).
\end{aligned} \tag{64}$$

The local dynamics of the system around the equilibrium point are totally determined by the signs of a and b for $|\phi| << 1$,

(i) *$a>0$, $b>0$. When $\phi<0$, 0 is locally asymptotically stable and there exists a positive unstable equilibrium; when $0<\phi<<1$, 0 is unstable and there exists a negative, locally asymptotically stable equilibrium.*
(ii) *$a<0, b<0$. When $\phi<0$, 0 is unstable; when $0<\phi<<1$, 0 is asymptotically stable equilibrium, and there exists a positive unstable equilibrium.*
(iii) *$a>0, b<0$. When $\phi<0$, 0 is unstable, and a positive unstable equilibrium appears.*
(iv) *$a<0, b>0$. When ϕ changes from positive to negative, 0 changes its stability from stable to unstable. Correspondingly, a negative unstable equilibrium becomes positive and locally asymptotically stable.*

Particularly, if $a>0$, and $b>0$, then, a subcritical (or backward) bifurcation occurs at $\phi = 0$.

To govern the local dynamics of the transformed system (55), the values of a and b are determined to explore whether the model system (55) exhibits forward or backward bifurcation. For system (55), the associated nonzero second-order partial derivatives at DFE and $\beta = \beta^*$ are given by:

$$\begin{aligned}
&\frac{\partial^2 f_1}{\partial x_1\partial x_4} = -\beta, \frac{\partial^2 f_1}{\partial x_1\partial x_6} = -\alpha, \frac{\partial^2 f_3}{\partial x_1\partial x_4} = \beta,\\
&\frac{\partial^2 f_3}{\partial x_1\partial x_6} = \alpha.
\end{aligned} \tag{65}$$

Therefore,

$$\begin{aligned}
a &= -\nu_1\omega_1\omega_4\beta - \nu_1\omega_1\omega_6\alpha + \nu_3\omega_1\omega_4\beta + \nu_3\omega_1\omega_6\alpha\\
&= (\omega_1\omega_4\beta + \omega_1\omega_6\alpha)[\nu_3 - \nu_1] \text{ and}
\end{aligned}, \tag{66}$$

$a>0$ if $\nu_1<0$ and $a<0$ if $\nu_1>0$ as a is positive if $(\omega_1\omega_4\beta + \omega_1\omega_6\alpha)\nu_3 > (\omega_1\omega_4\beta + \omega_1\omega_6\alpha)\nu_1$ and a is negative if $(\omega_1\omega_4\beta + \omega_1\omega_6\alpha)\nu_3 < (\omega_1\omega_4\beta + \omega_1\omega_6\alpha)\nu_1$.

For the sign of b, it can be shown that the associated nonvanishing partial derivatives are as follows:

$$\frac{\partial^2 f_1}{\partial x_4\partial\beta^*} = \frac{-\Lambda a_2}{a_1a_2 - \eta\tau}, \frac{\partial^2 f_3}{\partial x_4\partial\beta^*} = \frac{\Lambda a_2}{a_1a_2 - \eta\tau}. \tag{67}$$

Then, it follows from the above expression that:

$$\begin{aligned}
b &= \frac{-\nu_1\omega_4\Lambda a_2}{a_1a_2 - \eta\tau} + \frac{\nu_3\omega_4\Lambda a_2}{a_1a_2 - \eta\tau}\\
&= \frac{\Lambda a_2\omega_4}{a_1a_2 - \eta\tau}[\nu_3 - \nu_1]
\end{aligned}, \tag{68}$$

$b>0$ if $\nu_1<0$ and $a_1a_2>\eta\tau$ otherwise b will be <0.

Theorem 6. *The unique endemic equilibrium is ensured, and by Theorem 5, the model is locally asymptotically stable for $R_0>1$. Moreover, according to Theorem 5 item (i), the model experiences backward bifurcation when $a>0$. This is true only if $\nu_1<0$, and $a_1a_2<\eta\tau$ are present; otherwise, forward bifurcation occurs.*

The model system (1) exhibits forward bifurcation at $R_0 = 1$ when $a<0$ and $b>0$. Biologically, this scenario demonstrated that the model system (1) is globally asymptotically stable at a DFE point when $R_0<1$, and has a distinct endemic equilibrium point for $R_0>1$. When R_0 is close to one the

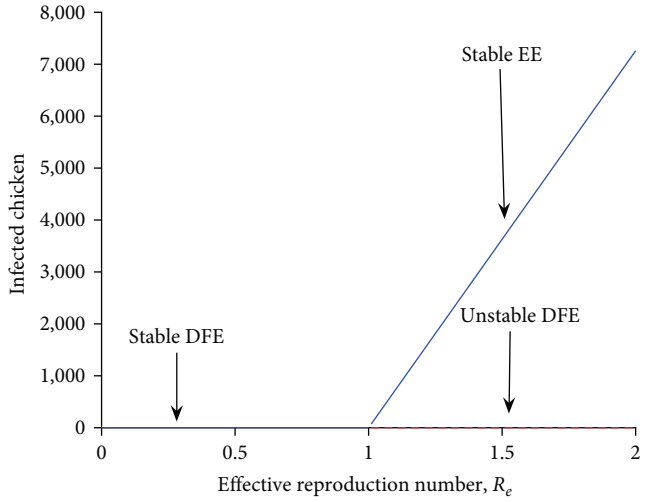

FIGURE 2: Forward bifurcation diagram for the model system (1) which shows that the disease-free equilibrium and endemic equilibria exchange stability when $Re=1$ using parameter values in Table 2. The blue curve depicts stable equilibria and the dashed red curve depicts unstable equilibria.

unique endemic equilibrium point is locally asymptotically stable. When R_0 falls below one, that is $R_0<1$ no endemicity exists and the disease continues to decline, and as R_0 increases to above one, that is $R_0>1$, the disease invades the population. Furthermore, when $a>0$ and $b>0$, the model system (1) exhibits a backward bifurcation, as R_0 approaches one, the disease prevalence rapidly increases giving rise to a situation in which the DFE co-exists with the endemic equilibrium. The biological implication of backward bifurcation is that the classical requirements of having the reproduction number less than unity, although necessary, is no longer sufficient for disease eradication. Based on Equation (15), The model system (1) exhibit a forward bifurcation as shown in Figure 2.

3.10. Global Stability of the Endemic Equilibrium Point. This section applies the Korobeinikov technique, as employed in the work of [23], to examine the global stability of the endemic equilibrium point E_*. Using this method, an appropriate Lyapunov function is created in the form of:

$$L=\sum A_i(x_i-x_i^*\ln x_i)\ , \tag{69}$$

where A_i is a nonnegative constant, x_i is the population of the i^{th} subpopulation, and x_i^* is the endemic equilibrium point E_*. Therefore, the following Lyapunov function is developed as follows:

$$\begin{aligned}L=\ & A_1(S-S^*\ln S)+A_2(V-V^*\ln V)+A_3(E-E^*\ln E)\\ & +A_4(I-I^*\ln I)+A_5(R-R^*\ln R)+A_6(H-H^*\ln H).\end{aligned} \tag{70}$$

Given that $A_1, A_2, A_3 \ldots A_6$ are nonnegative constants whereby V is Lyapunov function. The selection of the function V and its constant ensures that it is continuous and differentiable in space at all times. Differentiating V with respect to time gives:

$$\begin{aligned}\frac{dL}{dt}=\ & A_1\left(1-\frac{S^*}{S}\right)\frac{dS}{dt}+A_2\left(1-\frac{V^*}{V}\right)\frac{dV}{dt}\\ & +A_3\left(1-\frac{E^*}{E}\right)\frac{dE}{dt}+A_4\left(1-\frac{I^*}{I_h}\right)\frac{dI}{dt}\\ & +A_5\left(1-\frac{R^*}{R}\right)\frac{dR}{dt}+A_6\left(1-\frac{H^*}{H}\right)\frac{dH}{dt}.\end{aligned} \tag{71}$$

The following is therefore obtained by substituting equations of the model system (1)–(71).

$$\begin{aligned}\frac{dL}{dt}=\ & A_1\left(1-\frac{S^*}{S}\right)[\Lambda-(\beta I+\alpha H)S-(\tau+\mu)S+\eta V+\psi R]\\ & +A_2\left(1-\frac{V^*}{V}\right)[\tau S-(\eta+\mu)V]+A_3\left(1-\frac{E^*}{E}\right)[(\beta I+\alpha H)S-(\gamma+\mu)E]\\ & +A_4\left(1-\frac{I^*}{I}\right)[\gamma E-(\theta+\epsilon+\mu+\delta)I]+A_5\left(1-\frac{R^*}{R}\right)[\theta I-(\psi+\mu)R]\\ & +A_6\left(1-\frac{H^*}{H}\right)[\epsilon I-(\rho+\mu_h)H].\end{aligned} \tag{72}$$

The following are obtained at the endemic equilibrium point:

$$\begin{aligned}\Lambda\ & =\beta S^*I^*+\alpha H^*S^*+(\tau+\mu)S^*-\eta V^*-\psi R^*, \eta+\mu\\ & =\frac{\tau S^*}{V^*}, \gamma+\mu=\frac{\alpha I^*S^*}{E^*}+\frac{\alpha H^*S^*}{E^*},\\ & \theta+\varepsilon+\mu+\delta=\frac{\gamma E^*}{I^*}, \psi+\mu=\frac{\theta I^*}{R^*}, \rho+\mu=\frac{\varepsilon I^*}{H^*}.\end{aligned} \tag{73}$$

Then, by substituting Equation (73) to Equation (72) and doing some simplifications, we obtain:

$$\frac{dL}{dt}=-A_1\left(1-\frac{S^*}{S}\right)^2(\tau+\mu)S+F(S,V,E,R,H), \tag{74}$$

given that

Table 1: Values of sensitivity indices.

Parameters	Sensitivity index for $R_e<1$	Sensitivity index for $R_0>1$
Λ	+1	+1
β	+0.0805	+0.7820
α	+0.2180	+0.9195
μ	−0.04383	−0.0944
γ	+0.3733	+0.3733
θ	−0.1068	—
δ	−0.2137	−0.2392
η	+0.0042	—
τ	−0.0118	—
ϵ	+0.1111	+0.7999
ρ	−0.2127	—
μ_h	−0.0053	−0.9195

$$
\begin{aligned}
&F(S,V,E,R,H)=\\
&A_1\left(1-\frac{S^*}{S}\right)\left[(R_R^*)\psi+(V-V^*)\eta+\left(1-\frac{IS}{I^*S^*}\right)\beta I^*S^*+\left(1-\frac{HS}{H^*I^*}\right)\alpha H^*S^*\right]\\
&+A_2\left(1-\frac{V^*}{V}\right)\left[\left(1-\frac{S^*V}{V^*S}\tau S\right)\right]+A_3\left(1-\frac{E^*}{E}\right)\left[\left(1-\frac{I^*S^*E}{E^*SI}\right)\beta IS+\left(1-\frac{H^*S^*E}{E^*HS}\right)\alpha HS\right]\\
&A_4\left(1-\frac{I^*}{I}\right)\left[\left(1-\frac{E^*I}{EI^*}\right)\gamma E\right]+A_5\left(1-\frac{R^*}{R}\right)\left[\left(1-\frac{I^*R}{R^*I}\right)\theta I\right]\\
&+A_6\left(1-\frac{H^*}{H}\right)\left[\left(1-\frac{I^*H}{H^*I}\right)\varepsilon I\right].
\end{aligned}
\tag{75}
$$

Then, if $S=S^*, V=V^*, E=E^*, I=I^*, R=R^*, H=H^*$, the function (75) becomes less than or equal to zero and $\frac{dL}{dt}\leq 0$ for all $S,V,E,I,R,H>0$, following the approach of Korobeinikov [23]. Hence, the largest compact invariant set in $\{S,V,E,I,R,H\}$ where $\frac{dL}{dt}=0$ is the singleton $\{S^*, V^*, E^*, I^*, R^*, H^*\}$ which is the endemic equilibrium point of the model system (1). It is implied that $\{S^*, V^*, E^*, I^*, R^*, H^*\}$ is globally asymptotically stable in the interior region of $\{, V, E, I, R, H\}$ by Lasalle's invariant principle [24]. As a result, the following theorem is provided:

Theorem 7. *If $R_0>1$, then the coccidiosis model (1) has a unique endemic equilibrium point $S^*, V^*, E^*, I^*, R^*, H^*$ which is globally asymptotically stable in S, V, E, I, R, H.*

3.11. Sensitivity Analysis. Sensitivity indices measure the relative change in a state variable when a parameter change [25]. Initial disease transmission is directly related to the reproduction number. The sensitivity analysis is performed to determine which model parameters are the most important to disease transmission and prevalence (the parameters that are the most sensitive with respect to the initial transmission of the disease). In this study, we use it to discover parameters that have a high impact on reproduction number, and should be targeted by intervention strategies [26]. If a variable is a differentiable function of the parameter, the sensitivity indices may be alternatively defined using partial derivatives [25]. We intend to know how each parameter affects the effective reproduction number R_e. We use the formula of the normalized sensitivity index of a variable for R_e to achieve our goal.

The normalized forward sensitivity index of a variable, Z, depends differentiability on index of a parameter, ϕ is defined as follows:

$$\gamma_\phi^Z=\left(\frac{\partial Z}{\partial \phi}\right)\left(\frac{\phi}{Z}\right). \tag{76}$$

Applying the formula for R_0 and R_e, we have:

$$\gamma_\phi^{R_0}=\left(\frac{\partial R_0}{\partial \phi}\right)\left(\frac{\phi}{R_0}\right), \tag{77}$$

$$\gamma_\phi^{R_e}=\left(\frac{\partial R_e}{\partial \phi}\right)\left(\frac{\phi}{R_e}\right). \tag{78}$$

Applying the normalized forward sensitivity index defined in Equations (77) and (78) yields to the results in Table 1. The parameter values in Table 2 are used to determine the sensitivity indices.

3.11.1. Interpretation of Sensitivity Indices. The sensitivity indices are presented in Table 1, when $R_0>1$ (in the absence of intervention) and when $R_e<1$ (in the presence of interventions). Parameters that have positive sensitivity indices in Table 1, indicated that if their values are increased while

Table 2: Parameters and their descriptions for the coccidiosis dynamic model Equation (1).

Parameter	Descriptions	Values	Source
Λ_h	Chicken recruitment rate	0.001/day	Assumed
β	Transmission rate for infectious chicken	0.7/day	Assumed
α	*Eimeria* transmission rate due to the chicken-to-environment interaction	0.8/day	Assumed
μ	Nature death rate of a chicken	$1.37 - 5.48 \times 10^{-4}$/day	[27]
γ	Chicken incubation rate	0.09/day	Assumed
θ	Chicken recover rate/treatment rate	0.01/day	Assumed
ψ	Immunity waning rate for the recovered chicken	0.09/day	Assumed
δ	Chicken death rate due to coccidiosis	0.02/day	Assumed
η	Waning rate of vaccinated chicken	0.03/day	Assumed
τ	Rate at which susceptible chicken are vaccinated	0.001/day	Assumed
μ_h	Parasite natural death rate	0.001/day	[13]
ρ	*Eimeria* death rate due to sanitation measures	0.04/day	Assumed
ϵ	Parasite shedding rate to the environment by infectious chicken	0.4/day	[13]

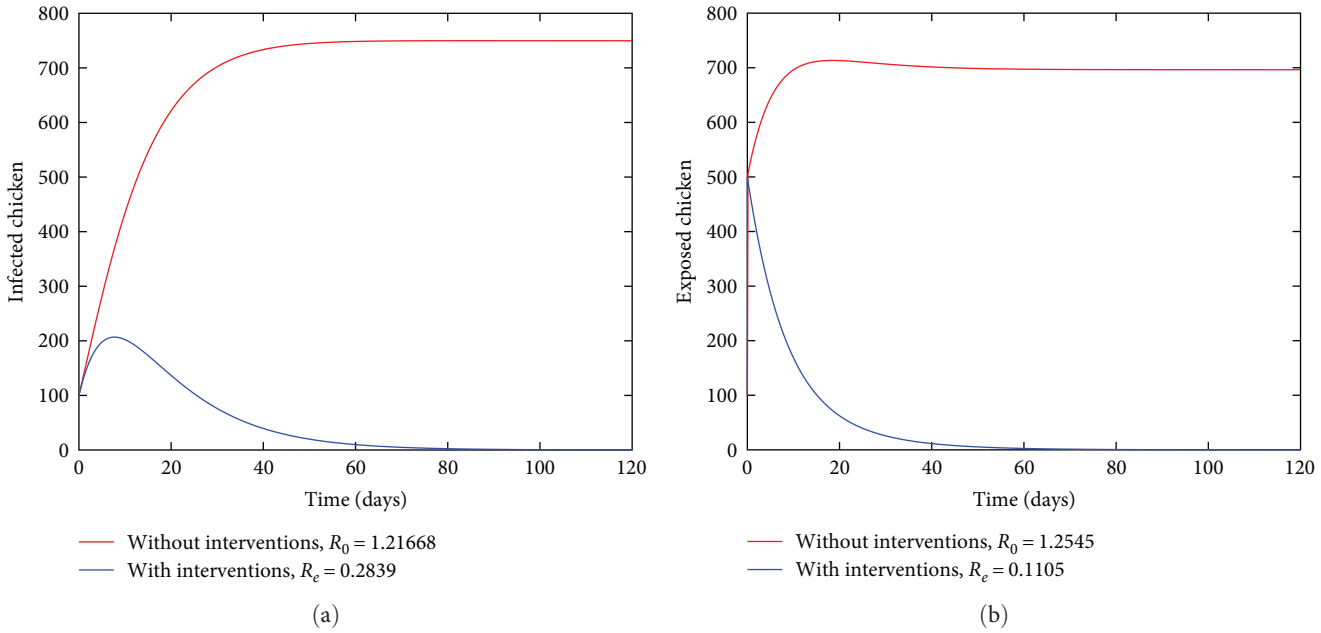

Figure 3: (a, b) Effects of interventions on coccidiosis dynamics over time.

keeping other values constant, reproduction number increases. These parameters have a greater influence on spreading the coccidiosis infection. But the parameters with negative sensitivity indices, show that if their values are increased while keeping the rest parameters fixed, reproduction number decreases. The most sensitive parameter is Λ with positive sensitivity indices. Other parameters with greater positive sensitivity indices are $\beta, \alpha,$ and γ. These parameters have an impact on reproduction number, and should be targeted for intervention strategies. These findings can be used to inform the development of intervention strategies to control coccidiosis infection. For example, interventions that target reducing the rate of transmission, such as vaccination or improved hygiene, are likely to be most effective.

3.12. Numerical Analysis. In this section, the model system (1) was numerically solved using MATLAB software and the Runge–Kutta fourth-order approach. To support previously analytical findings, various graphical representations are provided and discussed. Since many parameters were not publicly available, we collected a few from the literature and others were assumed for demonstration. For simulation, the study employed the initial values of the parameters from Table 2. The following initial values for the subpopulation were assumed as follows: $S(0) = 400$, $V = 300$, $E = 100$, $I = 100$, $R = 80$, and $H = 5,000$.

3.13. Effects of Interventions on Infected Chickens. Figure 3 depicts how the number of exposed and infected chickens

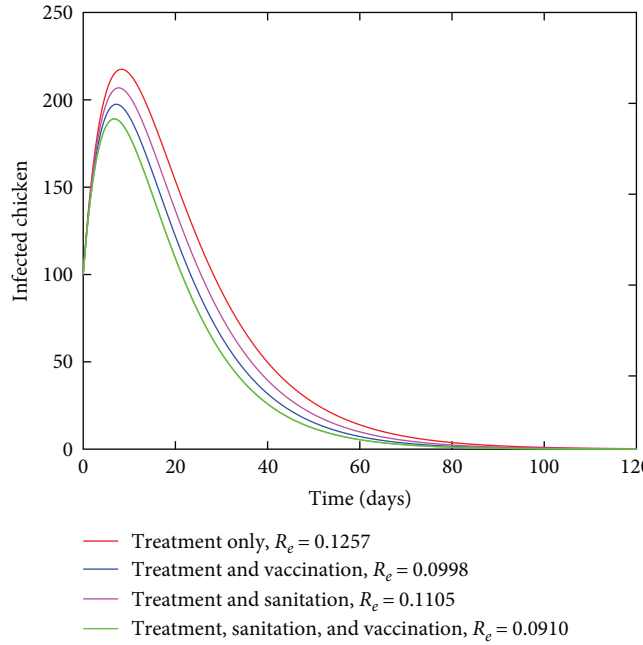

FIGURE 4: Impacts of various intervention on infected chicken population.

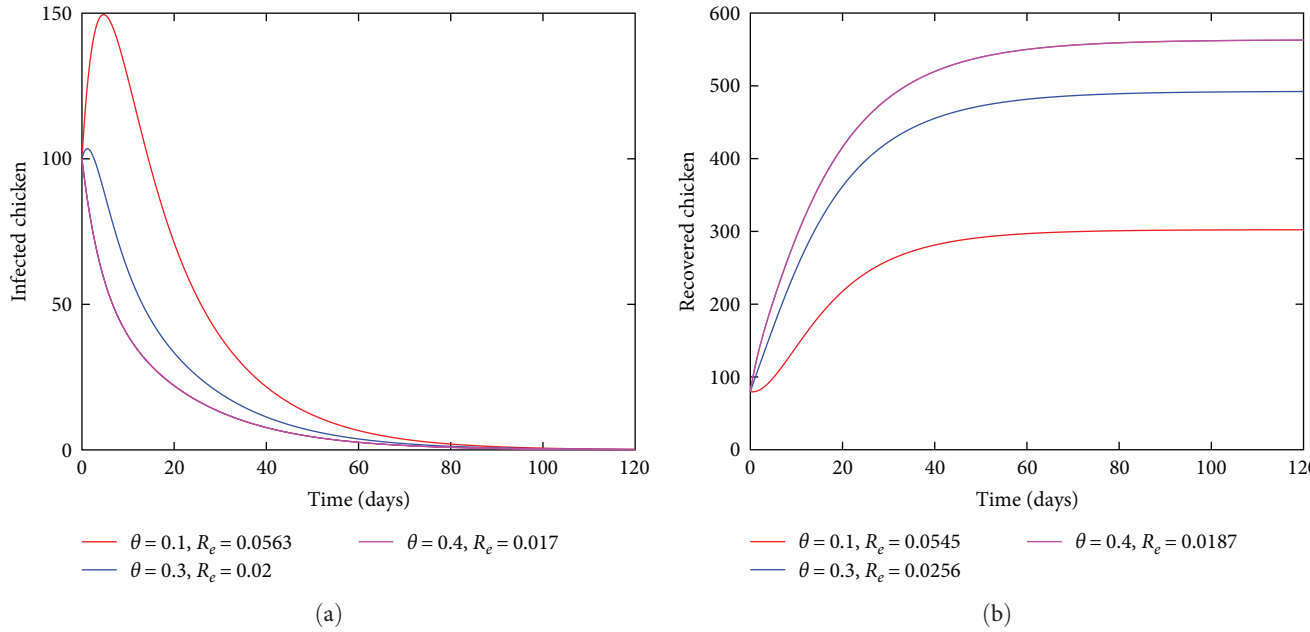

FIGURE 5: (a, b) Effects of treatment on coccidiosis dynamics over time.

changes over time. It can be observed from Figures 3(a) and 3(b) that the number of exposed and infected chickens decreases as time increases when there are interventions and increases when there are no interventions. This finding implies that the prevention measures of treatment, vaccination, and sanitation have a positive impact on reducing the spread of coccidiosis in the population.

Figure 4 depicts how the number of exposed and infected chickens changes over time. It can be observed from Figures 4(a) and 4(b) that the number of exposed and infected chickens decreases as time increases when there are interventions and increases when there are no interventions. This finding implies that the prevention measures of treatment, vaccination, and sanitation have a positive impact on reducing the spread of coccidiosis in the population.

3.14. Effect of Varying Some Parameter Values. Figure 5 illustrates the effects of varying treatment rates. As the rate of

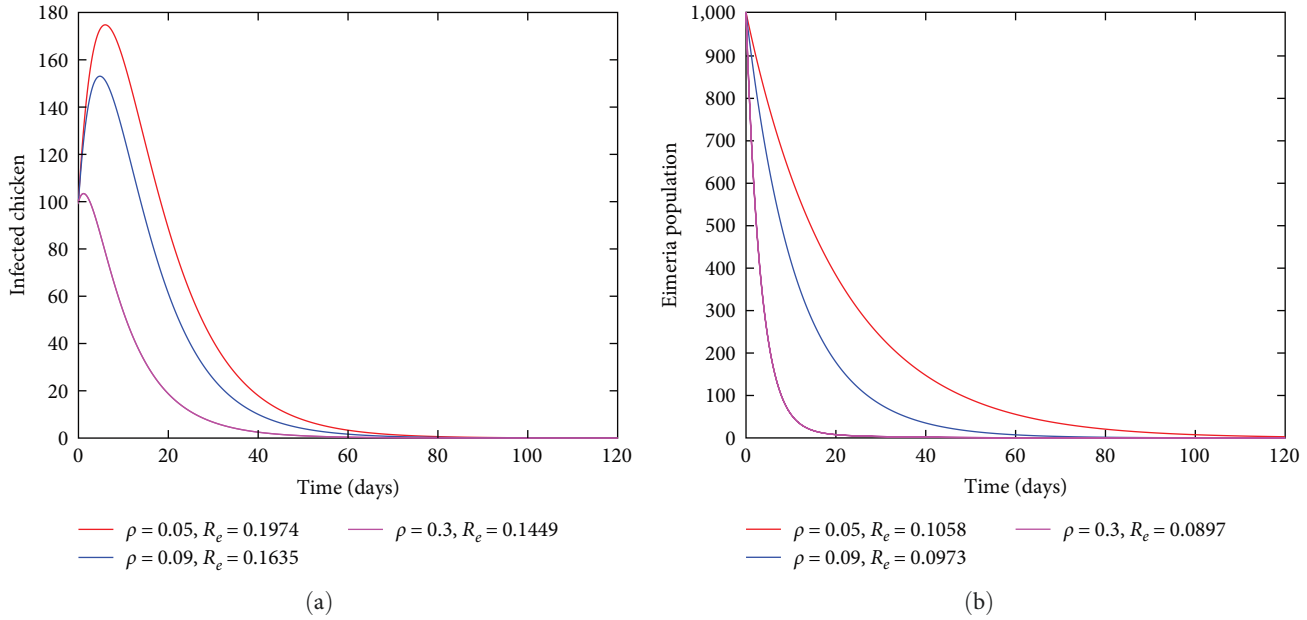

FIGURE 6: Variation of sanitation rate on (a) infected chicken and (b) pathogen population.

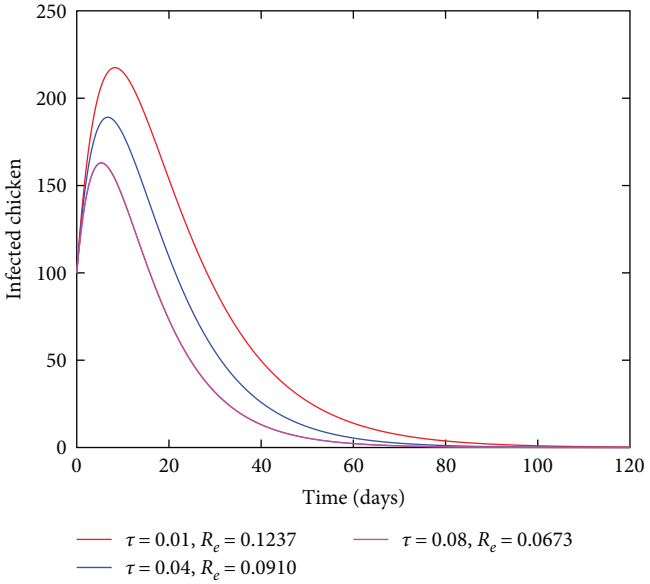

FIGURE 7: Variation of vaccination rate on infected chicken population.

treating the infected chicken increases, the number of infected chickens also reduces, as shown in Figure 5(a). On the other hand, by increasing the treatment rates, the number of recovered chickens increases with time as shown in Figure 5(b). This result indicates that treating the affected chicken has a significant effect on curbing coccidiosis as it reduces the number of sick chickens and increases the number of recovered individuals in the population.

Figure 6 shows the impact of sanitation on infected chickens and *Eimeria* pathogen population. The findings show that when sanitation rates increase, the number of both infected chickens and pathogens decreases as time passes as shown in Figures 6(a) and 6(b). This finding demonstrated that cleanliness has a major role in the complete eradication of coccidiosis disease in the population.

In this instance, the focus was to evaluate the contribution made by the sanitation strategy toward eradicating the coccidiosis epidemic in the community. Figure 7 findings demonstrate that when sanitation rates increase, the number of infected chickens significantly decreases. In this strategy, efforts like adequate waste disposal and hygienic practice follow-up may be practiced with the main objective of limiting the spread of this disease in the community.

4. Conclusion

A deterministic model with control interventions for chicken coccidiosis disease is derived and analyzed to investigate the effect of each control intervention for controlling this disease. The effective reproduction number and equilibria of the model are derived. The stability analysis of the DFE is derived, and it is stable when the reproduction number is less than unity. Also, the contribution of the control interventions is assessed by comparing the basic reproduction number (R_0) and effective reproduction number (R_e), whereby it is observed that $R_e < R_0$. This result implies that the control interventions (vaccination, sanitation, and treatment) reduce the transmission rate of the disease. On the other hand, sensitivity analysis carried out on the model parameters shows that: the control parameters (θ (treatment), ρ (sanitation), and τ (vaccination)) are the essential parameters for effective control of the disease, as they have negative indices. Numerical analysis of the model system (1) is carried out to illustrate the effectiveness of the control interventions. The results show that as the rate of the control parameters increases, the number of infected chicken and

coccidiosis pathogens decreases. The results further show that applying the control interventions singly or in combination reduces the infection rate, though the combination has the most significant results. The model discussed in this paper is not exhaustive. As a result, the model's underlying assumptions can be adjusted to include the aspect of the age structure of the chicken, the role of seasonal variations on the transmission dynamics of coccidiosis in the chicken population, or optimal and cost-effectiveness.

Data Availability

All data and materials that support the findings of this research study are included in the paper.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

Acknowledgments

Y. Liana would like to appreciate the support provided by the College of Business Education, Tanzania. M. Swai is grateful for the support provided by the Open University of Tanzania.

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