SABA Publishing

Journal of Mathematical Analysis and Modeling
jmam.sabapub.com
ISSN 2709-5924

J Math Anal & Model
(2024)5(3) : 1-23
doi:10.48185/jmam.v5i3.1241

A GUMBORO-SALMONELLA CO-INFECTION MATHEMATICAL MODEL WITH OPTIMAL CONTROL

J.S. MUSAILI[a,*], I. CHEPKWONY[b] , W.N. MUTUKU[c]

[a] Department of Mathematics, Kenyatta University, Nairobi Kenya
[b] Department of Mathematics, Kenyatta University, Nairobi Kenya
[c] Department of Mathematics, Kenyatta University, Nairobi Kenya

• *Received: 10 July 2024* • *Accepted: 04 August 2024* • *Published Online: 29 October 2024*

Abstract

Poultry production contributes immensely to the economic growth of a country. For instance in Kenya, 20 tonnes of poultry meat worth 3.5 billion kenya shillings and 1.3 billion eggs worth 9.7 billion Kenya Shillings come from this sector. However, the sector is greatly threatened by poultry diseases among them, Gumboro(IBD) and Salmonella as inadequate knowledge exists of optimal control strategies for various poultry co-infections. In this research, a Gumboro-Salmonella co-infection mathematical model with optimal control is developed using a system of ODEs to perform an optimal control analysis. The analysis was done by formulating an optimal control problem and using Pontryagin's maximum principle to solve it. The Numerical simulation results showed that the best Gumboro-Salmonella co-infection control strategy involved combining all the interventions

Keywords: Gumboro, Salmonella, Optimal control, Numerical Simulation.
2010 MSC: 92B05, .

1. Introduction

Poultry production is an enterprise that plays a major role in the growth of an economy as well as in poverty reduction. The poultry population in Kenya is estimated to be 31 million birds of which the Indigenous chicken form 75%, broilers 22%, breeding stock 1%, and 2% of all other poultry species such as Turkey, Ostriches, Pigeons, Geese, Guinea fowl, and Ducks [1].

In Kenya each year, about 20 tonnes of poultry meat worth 3.5 billion Kenya shillings and 1.3 billion eggs worth 9.7 billion Kenya shillings come from the poultry sector,a production which has greatly increased due to increased demand for quality protein [2]. Indigenous chickens which form the largest of the chicken population are kept by the rural community

*Corresponding author: jane.smusaili@gmail.com

under a free range system by children and women since bird keeping is a less involving activity[3].

The commercialization of chicken is an emerging phenomenon that is rapidly gaining popularity. However, the sector is greatly threatened by diseases classified as biological, viral and parasitic poultry diseases with Gumboro and Salmonella forming a part of these diseases that lead to huge economic loss in the poultry industry [4].

Gumboro disease also called Infectious bursal disease(IBD) is a viral disease caused by IBD-virus transmitted through feacal-oral route(Horizontal transmission) [5]. The disease was reported in 1957 in Delmarva Pensulla leading to acute morbidity and mortality in broilers with first Kenya's Gumboro case reported in 1991 in commercial birds on the Kenyan coast and since then the disease has remained a great threat to the poultry industry[6].

Salmonella on the other hand is a bacterial disease caused by strains of bacteria [7]. The disease is transmitted both Horizontally and vertically with exposed animals(infected but symptoms haven't started showing) such as poultry and pigs being the main disease spreaders to other flocks, animals, and even human beings[8].

Mathematical modelling has been considered as a crucial tool in comprehending transmission dynamics for various diseases so as to manage them. Scientists have developed various mathematical models to investigate the transmission dynamics of various poultry diseases as well as their preventive and control strategies. For instance, mathematical models to investigate impacts of various control strategies on the transmission dynamics of Avian influenza disease in both human and poultry populations have been developed (see details in [9, 10, 11]), mathematical models for fowl pox disease were formulated and analyzed by [12, 13, 14], to investigate the transmission dynamics of above-mentioned disease in poultry with controls. Additionally, an optimal control model for newcastle disease was developed and analysed (see details in[15, 16]).

A number of models that describe the dynamics of Gumboro disease(disease in question) have been studied in the recent past (see[17, 18, 19]), However, few models have been proposed to study salmonella disease. For instance, a mathematical model to analyze the transmission dynamics of salmonella disease by[20].

It is important to note that scholars have developed co-infection mathematical models for various Human diseases to describe their transmission dynamics (see details in [21, 22, 23]), and as Co-infection affects the course of infection in human beings, so it does in plants and even poultry. However, co-infection in poultry is an overlooked area and therefore this research work aims to study the optimal control of Gumboro-Salmonella co-infection in poultry to establish an effective strategy so as to avoid endemicity of Gumboro-Salmonella co-infection in poultry.

2. Model Formulation

This research builds upon a study conducted by [19] which described the Transmission dynamics of Gumboro disease. To describe the dynamics of Gumboro-Salmonella co-infection we developed a ten-compartmental model in which the chicken population

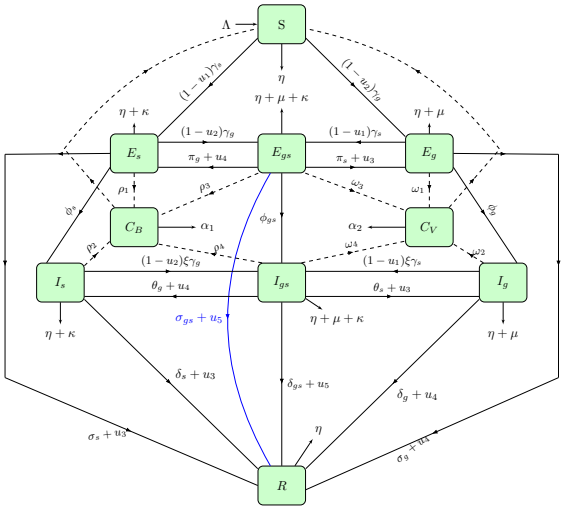

Figure 1: Flow chart of epidemic chicken population

was divided into eight classes namely; susceptible chicken S, birds at early stages of Gumboro infections E_g, birds at early stages of Salmonella infection E_s, birds at early stages of Gumboro-Salmonella co-infection E_{gs}, birds at acute stages of Gumboro infection I_g, at acute stages Salmonella infection I_s, birds at acute stages of Gumboro-Salmonella co-infection I_{gs} and birds Recovered from Gumboro, Salmonella, and Gumboro-Salmonella diseases in both early and acute stages of infections simultaneously R and pathogen population divided into two compartments namely Gumboro virus concentration in the environment C_V and Salmonella bacteria concentration in the environment C_B. Thus the total population at a given time (t) is $N(t) = N_c(t) + N_g(t) + N_s(t)$.The model was schematically described in Figure 1.

From Figure 1, the following equations were derived:

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - (1-u_1)\gamma_s S - (1-u_2)\gamma_g S - \eta S \\
\frac{dE_s}{dt} &= (1-u_1)\gamma_s S + (\pi_g + u_4)E_{gs} - (1-u_2)\gamma_g E_s - (\sigma_s + u_3)E_s - (\phi_s + \eta + \kappa)E_s \\
\frac{dE_g}{dt} &= (1-u_2)\gamma_g S + (\pi_s + u_3)E_{gs} - (1-u_1)\gamma_s E_g - (\sigma_g + u_4)E_g - (\phi_g + \eta + \mu)E_g \\
\frac{dE_{gs}}{dt} &= (1-u_1)\gamma_s E_g + (1-u_2)\gamma_g E_s - (\pi_s + u_3)E_{gs} - (\pi_g + u_4)E_{gs} - (\sigma_{gs} + u_5)E_{gs} \\
&\quad -(\phi_{gs} + \eta + \mu + \kappa)E_{gs} \\
\frac{dI_s}{dt} &= \phi_s E_s + (\theta_g + u_4)I_{gs} - \xi(1-u_2)\gamma_g I_s - (\delta_s + u_3)I_s - (\eta + \kappa)I_s \\
\frac{dI_g}{dt} &= \phi_g E_g + (\theta_s + u_3)I_{gs} - \xi(1-u_1)\gamma_s I_g - (\delta_g + u_4)I_g - (\eta + \mu)I_g \\
\frac{dI_{gs}}{dt} &= \xi(1-u_1)\gamma_s I_g + \xi(1-u_2)\gamma_g I_s + \phi_{gs} E_g - (\theta_s + u_3)I_{gs} - (\theta_g + u_4)I_{gs} - (\delta_{gs} + u_5)I_{gs}
\end{aligned}$$

$$
\begin{aligned}
& -(\eta+\mu+\kappa)I_{gs} \\
\frac{dR}{dt} &= (\sigma_s+u_3)E_s+(\sigma_g+u_4)E_g+(\sigma_{gs}+u_5)E_{gs}+(\delta_s+u_3)I_s+(\delta_g+u_4)I_g+(\delta_{gs}+u_5)I_{gs} \\
& -\eta R \\
\frac{dC_B}{dt} &= \rho_1 E_s+\rho_2 I_s+\rho_3 E_{gs}+\rho_4 I_{gs}-(\alpha_1+u_6)C_B \\
\frac{dC_V}{dt} &= \omega_1 E_g+\omega_2 I_g+\omega_3 E_{gs}+\omega_4 I_{gs}-(\alpha_2+u_7)C_V
\end{aligned}
\tag{2.1}
$$

Where Λ is the recruitment rate of birds to the susceptible class through Hatching and migration, the Susceptible birds are infected with Gumboro and salmonella diseases at the rate of $\gamma_g = \frac{\omega C_V}{\tau_2+C_V}$ and $\gamma_s = \frac{\rho C_B}{\tau_1+C_B}$ respectively. Where, ω is the contact rate of susceptible birds with an infectious bursal disease(IBD) virus-contaminated environment, τ_2 is the IBD-virus concentration in the environment with a 50 percent probability of Gumboro infections, ρ is the contact rate of susceptible birds with a salmonella typhi bacteria in the environment and τ_1 is the salmonella typhi concentration in the environment that have 50 percent probability of salmonella disease infections. All bird populations experience natural death at the rate η and they also die from Gumboro and salmonella at the rates μ and κ respectively. The Gumboro-infected birds in both stages of infection shed the virus to the environment at the rates ω_{1-4} which die at the rate α_2, while the Salmonella infected birds in both stages of infection shed the bacteria to the environment at the rates ρ_{1-4} which die at the rate α_1. $\xi\gamma_g$ and $\xi\gamma_s$ are the infection rates of I_s and I_g with Gumboro and Salmonella diseases respectively. While π_s, θ_s, π_g, θ_g, σ_g, σ_s, σ_{gs}, δ_g, δ_s, δ_{gs} are recovery rates. Birds at the early stages of Gumboro, Salmonella, and Gumboro–Salmonella infections move to the acute stages of infection at the rates ϕ_g, ϕ_s and ϕ_{gs} respectively. Additionally, the time-dependent control measures ($u_i(t), i = 1,2,3.....7$) defined as;

(i) u_1 -Vaccination of susceptible birds against Salmonella disease to boost their immunity hence preventing salmonella infections.

(ii) u_2 Vaccination of susceptible birds against Gumboro disease to boost their immunity hence preventing Gumboro disease infections.

(iii) u_3-Treatment of Salmonella-infected birds by applying supportive measures such as administration of mild antibiotics.

(iv) u_4-Treatment of Gumboro disease by use of supportive measures such as proper ventilation, increased intake of clean and quality water, and antistress multivitamins.

(v) u_5-treatment of birds with Gumboro-Salmonella co-infection using Supportive measures.That is combining the treatment of Salmonella and Gumboro diseases.

(vi) u_6- Elimination of Salmonella bacteria from the environment using relevant disinfectants and detergents such as TH4, Norocleanse, and ultrawide.

(vii) u_7- Eradication of Gumboro virus from the environment through curling and proper disposal of critical Gumboro cases.

3. Basic properties of the model

Here the positivity and boundedness of the model solutions is discussed.

3.1. Positivity of the solutions of the co-infection model

Lemma 3.1. *Let* $S(0) > 0,\ E_s(0) \geqslant 0,\ E_g(0) \geqslant 0,\ E_{gs}(0) \geqslant 0,\ I_s(0) \geqslant 0,\ I_g(0) \geqslant 0,\ I_{gs}(0) \geqslant 0,\ R(0) \geqslant 0,\ C_B(0) \geqslant 0,\ C_V(0) \geqslant 0$ *be the initial conditions of the system then the solution set* $\{S,\ E_s,\ E_g,\ E_{gs},\ I_s,\ I_g,\ I_{gs},\ R,\ C_B,\ C_V\}(t)$ *of model (2.1) is non-negative for all* $t > 0$

Proof. From the first equation of system (2.1)
$\frac{ds}{dt} = \Lambda - ((1-u_1)\gamma_s + (1-u_2)\gamma_g + \eta)S$
we have

$$\frac{ds}{dt} > -((1-u_1)\gamma_s + (1-u_2)\gamma_g + \eta)S. \tag{3.1}$$

Separating the variables we obtain

$$\frac{ds}{s} > -((1-u_1)\gamma_s + (1-u_2)\gamma_g + \eta)dt \tag{3.2}$$

Integrating on both sides of equation (3.2) we have
$\ln S > -((1-u_1)\gamma_s + (1-u_2)\gamma_g + \eta)t + c_1$
or

$$S(t) > ce^{-((1-u_1)\gamma_s + (1-u_2)\gamma_g + \eta)t} \tag{3.3}$$

From equation (3.3), it is clear that $S(0) = c$ for $t = 0$.
Therefore, $S(t) > S(0)e^{-((1-u_1)\gamma_s + (1-u_2)\gamma_g + \eta)t}$ and as $t \longrightarrow \infty$ we have $S(t) > 0\ \forall\ t > 0$.
Also from the second equation of system (2.1);
$\frac{dE_s}{dt} = (1-u_1)\gamma_s S + (\pi_g + u_4)E_{gs} - (1-u_2)\gamma_g E_s - (\sigma_s + u_3)E_s - (\phi_s + \eta + \kappa)E_s$
we have
$\frac{dE_s}{dt} \geqslant -((1-u_2)\gamma_g + \sigma_s + u_3 + \phi_s + \eta + \kappa)t$
solving the above equation by separation of variables we have
$\ln E_s(t) \geqslant -((1-u_2)\gamma_g + \sigma_s + u_3 + \phi_s + \eta + \kappa)t + c$
which implies that

$$\ln E_s(t) > ce^{-((1-u_2)\gamma_g + \sigma_s + u_3 + \phi_s + \eta + \kappa)t} \tag{3.4}$$

Clearly for $t = 0$, $c = E_s(0)$.
Thus, equation (3.4) becomes;
$E_s(t) \geqslant E_s(0)e^{-((1-u_2)\gamma_g + \sigma_s + u_3 + \phi_s + \eta + \kappa)t}$
and as $t \longrightarrow \infty$,we have
$E_s(t) \geqslant 0\ \forall\ t > 0$
Applying the same method to other equations of the system (2.1) we get
$E_g(t) \geqslant E_g(0)e^{-((1-u_1)\gamma_s + \sigma_g + u_4 + \phi_g + \eta + \mu)E_g} \geqslant 0$;
$E_{gs}(t) \geqslant E_{gs}(0)e^{-(u_4 + \pi_g + u_3 + \pi_s + u_5\sigma_{gs} + \eta + \mu + \kappa)} \geqslant 0$;
$I_s(t) \geqslant I_s(0)e^{-(\xi(1-u_2)\gamma_g + \delta_s + u_3 + \eta + \kappa)t} \geqslant 0$
$I_g(t) \geqslant I_g(0)e^{-(\xi(1-u_1)\gamma_s + \delta_g + u_4 + \eta + \mu)t} \geqslant 0$
$I_{gs}(t) \geqslant I_{gs}(0)e^{-(\theta_s + u_3 + \theta_g + u_4 + \delta_{gs} + u_5 + \eta + \mu + \kappa)t} \geqslant 0$

$R(t) \geqslant R(0)e^{-\eta t} \geqslant 0$
$C_B(t) \geqslant C_B(0)e^{-\alpha_1 t} \geqslant 0$
$C_V(t) > C_V(0)e^{-\alpha_2 t} \geqslant 0$ hence proof of the theorem. □

3.2. Boundedness of the solutions of the co-infection model

Let $\Omega = (\Omega_C \cup \Omega_{C_B} \cup \Omega_{C_V}) \subset \mathbb{R}^{10}_+$ be a feasible region in which the solutions of the total population are bounded. Where Ω_C is the feasible region of the solutions of birds population and that of Salmonella and Gumboro pathogens population is given by Ω_{C_B} and Ω_{C_V} respectively. We show that the solutions of the system (2.1) are bounded in the feasible region.

The total bird population is N_C given by
$N_C = S(t) + E_s(t) + E_g(t) + E_{gs}(t) + I_s(t) + I_g(t) + I_{gs}(t) + R(t)$
and
$\frac{dN_C}{dt} = \frac{dS}{dt} + \frac{dE_s}{dt} + \frac{dE_g}{dt} + \frac{dE_{gs}}{dt} + \frac{dI_s}{dt} + \frac{dI_g}{dt} + \frac{dI_{gs}}{dt} + \frac{dR}{dt}$
Thus, from system (2.1) we have

$$\begin{aligned}\frac{dN_C}{dt} &= \Lambda - \eta(S(t) + E_s(t) + E_g(t) + E_{gs}(t) + I_s(t) + I_g(t) + I_{gs}(t) + R(t)) \\ &\quad - (\kappa E_s + \mu E_g + \mu E_{gs} + \kappa E_{gs} + \kappa I_s + \mu I_g + \mu I_{gs} + \kappa I_{gs}).\end{aligned} \tag{3.5}$$

When the birds are not infected, equation (3.5) reduces to

$$\frac{dN_C}{dt} \leqslant \Lambda - \eta N_c \tag{3.6}$$

Upon solving the equation (3.6), we get

$$N_c(t) \leqslant \frac{\Lambda}{\eta} + (N_c(0) - \frac{\Lambda}{\eta})e^{-\eta t} \tag{3.7}$$

and taking limits as $t \longrightarrow \infty$ we have

$$N_c \leqslant \frac{\Lambda}{\eta} \tag{3.8}$$

Thus the birds population is bounded in

$$\Omega_C = \left\{(S(t),\ E_s(t),\ E_g(t),\ E_{gs}(t),\ I_s(t),\ I_g(t),\ I_{gs}(t),\ R(t)) \in \mathbb{R}^8_+ : N_c \leqslant \frac{\Lambda}{\eta}\right\}$$

Considering the Salmonella pathogens population compartment, that is the ninth equation of system (2.1),we have;

$$\frac{dC_B}{dt} = \rho_1 E_s + \rho_2 I_s + \rho_3 E_{gs} + \rho_4 I_{gs} - \alpha_1 C_B, \tag{3.9}$$

equation (3.9) reduces to

$$\frac{dN_s}{dt} \leqslant \frac{\Lambda(\rho_1 + \rho_2 + \rho_3 + \rho_4)}{\eta} - \alpha_1 N_s. \tag{3.10}$$

Solving equation (3.10) for N_s, yields

$$N_s(t) \leqslant \frac{\Lambda(\rho_1+\rho_2+\rho_3+\rho_4)}{\eta\alpha_1} + \left(N_s(0) - \frac{\Lambda(\rho_1+\rho_2+\rho_3+\rho_4)}{\eta\alpha_1}\right) e^{-\alpha_1 t} \tag{3.11}$$

and taking limits as $t \longrightarrow \infty$ we obtain

$$N_s(t) \leqslant \frac{\Lambda(\rho_1+\rho_2+\rho_3+\rho_4)}{\eta\alpha_1}$$

Hence, the Salmonella population is bounded in the region

$$\Omega_{C_B} = \left\{ C_B(t) \in \mathbb{R}^1_+ : N_s(t) \leqslant \frac{\Lambda(\rho_1+\rho_2+\rho_3+\rho_4)}{\eta\alpha_1} \right\}$$

considering the last equation of system (2.1),that is equation ten,we have;

$$\frac{dC_V}{dt} = \omega_1 E_g + \omega_2 I_g + \omega_3 E_{gs} + \omega_4 I_{gs} - \alpha_2 C_V. \tag{3.12}$$

Upon reduction of equation (3.12), we have

$$\frac{dN_g}{dt} \leqslant \frac{\Lambda(\omega_1+\omega_2+\omega_3+\omega_4)}{\eta} - \alpha_2 N_g. \tag{3.13}$$

By use of integrating factor we solve equation (3.13) to get

$$N_g \leqslant \frac{\Lambda(\omega_1+\omega_2+\omega_3+\omega_4)}{\eta\alpha_2} + \left(N_g(0) - \frac{\Lambda(\omega_1+\omega_2+\omega_3+\omega_4)}{\eta\alpha_2}\right) e^{-\alpha_2 t} \tag{3.14}$$

Taking the limit of equation (3.14), as t tends to infinity, gives $N_g \leqslant \frac{\Lambda(\omega_1+\omega_2+\omega_3+\omega_4)}{\eta\alpha_2}$. Thus the Gumboro population is bounded in the region

$$\Omega_{C_V} = \left\{ C_V(t) \in \mathbb{R}^1_+ : N_g \leqslant \frac{\Lambda(\omega_1+\omega_2+\omega_3+\omega_4)}{\eta\alpha_2} \right\}$$

Since the birds population, Gumboro pathogen population and the Salmonella pathogen population are bounded, then the model will be analysed in a suitable feasible region

$$\begin{aligned}\Omega = \{&(S, E_g, E_s, E_{gs}, I_s, I_g, I_{gs}, R) \in \mathbb{R}^8_+;\ C_B \in \mathbb{R}_+;\ C_V \in \mathbb{R}_+;\ S > 0;\\ &E_g, E_s, E_{gs}, I_s, I_g, I_{gs}, R, C_B, C_V \geqslant 0;\ N_c \leqslant \frac{\Lambda}{\eta};\\ &N_g \leqslant \frac{\Lambda(\omega_1+\omega_2+\omega_3+\omega_4)}{\eta\alpha_2};\ N_s(t) \leqslant \frac{\Lambda(\rho_1+\rho_2+\rho_3+\rho_4)}{\eta\alpha_1}\}\end{aligned}$$

4. Optimal Control Problem

Optimization of disease prevention and control is one of the branches of Mathematical modeling which helps in determining the effective strategies for disease prevention and control. The model aims at minimizing the number of infections as well as the costs for the control strategies. The objective function to be minimized is formulated as follows

$$J = \min_{u_1,u_2,u_3,u_4,u_5,u_6,u_7} \int_{t_0}^{t_f} [a_1 E_s(t) + a_2 E_g(t) + a_3 E_{gs}(t) + a_4 I_s(t) + a_5 I_g(t) + a_6 I_{gs}(t) + a_7 C_B(t) + a_8 C_V(t) + \frac{1}{2}\sum_{i=1}^{7} w_i u_i^2]dt$$

where the coefficients $a_1, a_2, a_3, a_4, a_5, a_6, a_7, a_8$ and $\sum_{i=1}^{7} w_i$ are the constant weights. The optimal control model is taken into consideration on the full-time horizon $[t_0, t_f]$. Therefore, the optimal control model's solution is $(u_1^*, u_2^*, u_3^*, u_4^*, u_5^*, u_6^*, u_7^*)$, in which $J(u_1^*, u_2^*, u_3^*, u_4^*, u_5^*, u_6^*, u_7^*) = \min\{J(u_1, u_2, u_3, u_4, u_5, u_6, u_7) | u_1, u_2, u_3, u_4, u_5, u_6, u_7 \in U\}$ Where $U = \{(u_1^*, u_2^*, u_3^*, u_4^*, u_5^*, u_6^*, u_7^*)\}$ such that $u_1^*, u_2^*, u_3^*, u_4^*, u_5^*, u_6^*, u_7^*$ are measurable with $0 \leqslant u_1^* \leqslant 1$, $0 \leqslant u_2^* \leqslant 1$, $0 \leqslant u_3^* \leqslant 1$, $0 \leqslant u_4^* \leqslant 1$, $0 \leqslant u_5^* \leqslant 1$, $0 \leqslant u_6^* \leqslant 1$, $0 \leqslant u_7^* \leqslant 1$ for $t \in [0, t_f]$ is the control set.

5. The Hamiltonian and optimality system

By applying the Pontryagins Maximum Principle [24], the Hamiltonian (H), is defined as:

$$\begin{aligned} H =& a_1 E_s(t) + a_2 E_g(t) + a_3 E_{gs}(t) + a_4 I_s(t) + a_5 I_g(t) + a_6 I_{gs}(t) + a_7 C_B(t) + a_8 C_V(t) \\ &+ \frac{1}{2}w_1 u_1^2 + \frac{1}{2}w_2 u_2^2 + \frac{1}{2}w_3 u_3^2 + \frac{1}{2}w_4 u_4^2 + \frac{1}{2}w_5 u_5^2 + \frac{1}{2}w_6 u_6^2 + \frac{1}{2}w_7 u_7^2 + p_1(t)\frac{dS}{dt} \\ &+ p_2(t)\frac{dE_s}{dt} + p_3(t)\frac{dE_g}{dt} + p_4(t)\frac{dE_{gs}}{dt} + p_5(t)\frac{dI_s}{dt} + p_6(t)\frac{dI_g}{dt} + p_7(t)\frac{dI_{gs}}{dt} \\ &+ p_8(t)\frac{dR}{dt} + p_9(t)\frac{dC_B}{dt} + p_{10}(t)\frac{dC_V}{dt} \end{aligned}$$

where $p_1(t)$, $p_2(t)$, $p_3(t)$, $p_4(t)$, $p_5(t)$, $p_6(t)$, $p_7(t)$, $p_8(t)$, $p_9(t)$, $p_{10}(t)$ are adjoint variables associated with the state variable S, E_s, E_g, E_{gs}, I_s, I_g, I_{sg}, R, C_B, C_V respectively.

Theorem 5.1. *Given an optimal control set* $u_1, u_2, u_3, u_4, u_5, u_6, u_7$ *that minimizes* J *over* U *, there exist adjoint variables,* $p_1(t)$, $p_2(t)$, $p_3(t)$, $p_4(t)$, $p_5(t)$, $p_6(t)$, $p_7(t)$, $p_8(t)$, $p_9(t)$, $p_{10}(t)$ *satisfying*

$$\begin{aligned}
\frac{dp_1}{dt} &= -\frac{\partial H}{\partial S}; & \frac{dp_6}{dt} &= -\frac{\partial H}{\partial I_g};\\
\frac{dp_2}{dt} &= -\frac{\partial H}{\partial E_s}; & \frac{dp_7}{dt} &= -\frac{\partial H}{\partial I_{gs}};\\
\frac{dp_3}{dt} &= -\frac{\partial H}{\partial E_g}; & \frac{dp_8}{dt} &= -\frac{\partial H}{\partial R};\\
\frac{dp_4}{dt} &= -\frac{\partial H}{\partial E_{gs}}; & \frac{dp_9}{dt} &= -\frac{\partial H}{\partial C_B};\\
\frac{dp_5}{dt} &= -\frac{\partial H}{\partial I_s}; & \frac{dp_{10}}{dt} &= -\frac{\partial H}{\partial C_V};
\end{aligned} \tag{5.1}$$

and with transversality conditions $p_i(t_f) = 0$ *where* $i = 1, 2, \cdots, 10$. *Furthermore,*

$$u_1(t)^* = \max\left\{0, \min\left\{1, \frac{-\gamma_s S p_1 + \gamma_s S p_2 - \gamma_s E_g p_3 + \gamma_s E_g p_4 - \xi\gamma_s I_g p_6 + \xi\gamma_s I_g p_7}{w_1}\right\}\right\}$$

$$u_2(t)^* = \max\left\{0, \min\left\{1, \frac{-\gamma_g S p_1 + \gamma_g S p_3 - \gamma_g E_s p_2 + \gamma_g E_s p_4 - \xi\gamma_g I_s p_5 + \xi\gamma_g I_s p_7}{w_2}\right\}\right\}$$

$$u_3(t)^* = \max\left\{0, \min\left\{1, \frac{E_s p_2 - E_{gs} p_3 + E_{gs} p_4 + I_s p_5 - I_{gs} p_6 + I_{gs} p_7 - E_s p_8 - I_s p_8}{w_3}\right\}\right\}$$

$$u_4(t)^* = \max\left\{0, \min\left\{1, \frac{E_g p_3 - E_{gs} p_2 + E_{gs} p_4 + I_g p_6 - I_{gs} p_5 + I_{gs} p_7 - E_g p_8 - I_g p_8}{w_4}\right\}\right\}$$

$$u_5(t)^* = \max\left\{0, \min\left\{1, \frac{E_{gs} p_4 - E_{gs} p_8 + I_{gs} p_7 - I_{gs} p_8}{w_5}\right\}\right\}$$

$$u_6(t)^* = \max\left\{0, \min\left\{1, \frac{C_B p_9}{w_6}\right\}\right\}$$

$$u_7(t)^* = \max\left\{0, \min\left\{1, \frac{C_V p_{10}}{w_7}\right\}\right\}$$

Proof. Suppose $U = \{(u_1^*, u_2^*, u_3^*, u_4^*, u_5^*, u_6^*, u_7^*)\}$ is an optimal control and $S(t)$, $E_s(t)$, $E_g(t)$, $E_{gs}(t)$, $I_s(t)$, $I_g(t)$, $I_{gs}(t)$, $R(t)$, $C_B(t)$, $C_V(t)$ are the corresponding state solutions. Applying the Pontryagin's Maximum Principle [24], there exist adjoint variables satisfying:

$$\begin{aligned}
\frac{dp_1}{dt} = -\frac{\partial H}{\partial S} &= (1-u_1)\gamma_s p_1 + (1-u_2)\gamma_g p_1 + \eta p_1 - (1-u_1)\gamma_s p_2 - (1-u_2)\gamma_g p_3\\
\frac{dp_2}{dt} = -\frac{\partial H}{\partial E_s} &= -a_1 + (1-u_2)\gamma_g p_2 + (\sigma_s + u_3)p_2 + (\phi_s + \eta + \kappa)p_2 - (1-u_2)\gamma_g p_4 - \phi_s p_5\\
&\quad -(\sigma_s + u_3)p_8 - \rho_1 p_9\\
\frac{dp_3}{dt} = -\frac{\partial H}{\partial E_g} &= -a_2 + (1-u_1)\gamma_s p_3 + (\sigma_g + u_4)p_3 + (\phi_g + \eta + \mu)p_3 - (1-u_1)\gamma_s p_4 - \phi_g p_6
\end{aligned}$$

$$
\begin{aligned}
& & & -(\sigma_g + u_4)p_8 - \omega_1 p_{10} \\
\frac{dp_4}{dt} = -\frac{\partial H}{\partial E_{gs}} &= & & -a_3 - (\pi_s + u_3)p_2 - (\pi_g + u_4)p_3 + (\pi_s + u_3)p_4 + (\pi_g + u_4)p_4 + (\sigma_{gs} + u_5)p_4 \\
& & & +(\phi_{gs} + \eta + \mu + \kappa)p_4 - \phi_{gs}p_7 - (\sigma_{gs} + u_5)p_8 - \rho_3 p_9 - \omega_3 p_{10} \\
\frac{dp_5}{dt} = -\frac{\partial H}{\partial I_s} &= & & -a_4 + \xi(1-u_2)\gamma_g p_5 + (\delta_s + u_3)p_5 - (\eta + \kappa)p_5 - \xi(1-u_2)\gamma_g p_7 - (\delta_s + u_3)p_8 \\
& & & -\rho_2 p_9 \\
\frac{dp_6}{dt} = -\frac{\partial H}{\partial I_g} &= & & -a_5 + \xi(1-u_1)\gamma_s p_6 + (\delta_g + u_4)p_6 + (\eta + \mu)p_6 - \xi(1-u_1)\gamma_s p_7 - (\delta_g + u_4)p_8 \\
& & & -\omega_2 p_{10} \\
\frac{dp_7}{dt} = -\frac{\partial H}{\partial I_{gs}} &= & & -a_6 + (\theta_s + u_3)p_7 + (\theta_g + u_4)p_7 + (\delta_{gs} + u_5)p_7 + (\eta + \mu + \kappa)p_7 - (\theta_s + u_3)p_6 \\
& & & -(\theta_g + u_4)p_5 - (\delta_{gs} + u_5)p_8 - \rho_4 p_9 - \omega_4 p_{10} \\
\frac{dp_8}{dt} = -\frac{\partial H}{\partial R} &= & & \eta p_8 \\
\frac{dp_9}{dt} = -\frac{\partial H}{\partial C_B} &= & & -a_7 + (\alpha_1 + u_6)p_9 + \frac{(1-u_1)\rho\tau_1}{(\tau_1 + C_B)^2}(Sp_1 - Sp_2 + E_g p_3 - E_g p_4 + \xi I_g p_7 - \xi I_g p_8) \\
\frac{dp_{10}}{dt} = -\frac{\partial H}{\partial C_V} &= & & -a_8 + (\alpha_2 + u_7)p_{10} + \frac{(1-u_2)\omega\tau_2}{(\tau_2 + C_V)^2}(Sp_1 - Sp_3 + E_s p_2 - E_s p_4 + \xi I_s p_6 - \xi I_s p_8)
\end{aligned}
\tag{5.2}
$$

with transversality conditions $p_1(t_f) = p_2(t_f) = p_3(t_f) = p_4(t_f) = p_5(t_f) = p_6(t_f) = p_7(t_f) = p_8(t_f) = p_9(t_f) = p_{10}(t_f) = 0$. In a manner similar to that described in [24], we solve the equation $\frac{\partial H}{\partial u_i} = 0$ at u_i for $i = 1, 2, \cdots, 7$ to obtain the controls:

$$
\begin{aligned}
0 = \frac{\partial H}{\partial u_1} &= \gamma_s S p_1 - \gamma_s S p_2 + \gamma_s E_g p_3 - \gamma_s E_g p_4 + \xi\gamma_s I_g p_6 - \xi\gamma_s I_g p_7 + w_1 u_1 \\
0 = \frac{\partial H}{\partial u_2} &= \gamma_g S p_1 - \gamma_g S p_3 + \gamma_g E_s p_2 - \gamma_g E_s p_4 + \xi\gamma_g I_s p_5 - \xi\gamma_g I_s p_7 + w_2 u_2 \\
0 = \frac{\partial H}{\partial u_3} &= -E_s p_2 + E_{gs} p_3 - E_{gs} p_4 - I_s p_5 + I_{gs} p_6 - I_{gs} p_7 + E_s p_8 + I_s p_8 + w_3 u_3 \\
0 = \frac{\partial H}{\partial u_4} &= -E_g p_3 + E_{gs} p_2 - E_{gs} p_4 - I_g p_6 + I_{gs} p_5 - I_{gs} p_7 + E_g p_8 + I_g p_8 + w_4 u_4 \\
0 = \frac{\partial H}{\partial u_5} &= -E_{gs} p_4 + E_{gs} p_8 - I_{gs} p_7 + I_{gs} p_8 + w_5 u_5 \\
0 = \frac{\partial H}{\partial u_6} &= -C_B p_9 + w_6 u_6 \\
0 = \frac{\partial H}{\partial u_7} &= -C_V p_{10} + w_7 u_7
\end{aligned}
$$

Therefore, we have

$$
u_1 = \frac{-\gamma_s S p_1 + \gamma_s S p_2 - \gamma_s E_g p_3 + \gamma_s E_g p_4 - \xi\gamma_s I_g p_6 + \xi\gamma_s I_g p_7}{w_1}
$$

$$
\begin{aligned}
u_2 &= \frac{-\gamma_g S p_1 + \gamma_g S p_3 - \gamma_g E_s p_2 + \gamma_g E_s p_4 - \xi\gamma_g I_s p_5 + \xi\gamma_g I_s p_7}{w_2} \\
u_3 &= \frac{E_s p_2 - E_{gs} p_3 + E_{gs} p_4 + I_s p_5 - I_{gs} p_6 + I_{gs} p_7 - E_s p_8 - I_s p_8}{w_3} \\
u_4 &= \frac{E_g p_3 - E_{gs} p_2 + E_{gs} p_4 + I_g p_6 - I_{gs} p_5 + I_{gs} p_7 - E_g p_8 - I_g p_8}{w_4} \\
u_5 &= \frac{E_{gs} p_4 - E_{gs} p_8 + I_{gs} p_7 - I_{gs} p_8}{w_5} \\
u_6 &= \frac{C_B p_9}{w_6} \\
u_7 &= \frac{C_V p_{10}}{w_7}
\end{aligned}
$$

then

$$
\begin{aligned}
u_1(t)^* &= \max\left\{0, \min\left\{1, \frac{-\gamma_s S p_1 + \gamma_s S p_2 - \gamma_s E_g p_3 + \gamma_s E_g p_4 - \xi\gamma_s I_g p_6 + \xi\gamma_s I_g p_7}{w_1}\right\}\right\} \\
u_2(t)^* &= \max\left\{0, \min\left\{1, \frac{-\gamma_g S p_1 + \gamma_g S p_3 - \gamma_g E_s p_2 + \gamma_g E_s p_4 - \xi\gamma_g I_s p_5 + \xi\gamma_g I_s p_7}{w_2}\right\}\right\} \\
u_3(t)^* &= \max\left\{0, \min\left\{1, \frac{E_s p_2 - E_{gs} p_3 + E_{gs} p_4 + I_s p_5 - I_{gs} p_6 + I_{gs} p_7 - E_s p_8 - I_s p_8}{w_3}\right\}\right\} \\
u_4(t)^* &= \max\left\{0, \min\left\{1, \frac{E_g p_3 - E_{gs} p_2 + E_{gs} p_4 + I_g p_6 - I_{gs} p_5 + I_{gs} p_7 - E_g p_8 - I_g p_8}{w_4}\right\}\right\} \\
u_5(t)^* &= \max\left\{0, \min\left\{1, \frac{E_{gs} p_4 - E_{gs} p_8 + I_{gs} p_7 - I_{gs} p_8}{w_5}\right\}\right\} \\
u_6(t)^* &= \max\left\{0, \min\left\{1, \frac{C_B p_9}{w_6}\right\}\right\} \\
u_7(t)^* &= \max\left\{0, \min\left\{1, \frac{C_V p_{10}}{w_7}\right\}\right\}
\end{aligned}
\tag{5.3}
$$

□

6. Numerical Simulation Results

Finding the solutions of the optimality system analytically is sometimes impossible which leads to the employment of the numerical methods in the approximation of the solutions and the displaying of the results. The optimality system contains the state system (2.1), the adjoint system (5.2), control characterization (5.3) and corresponding initial conditions and it is solved using the fourth-order runge kutta iteration method to so as to produce the simulation results shown in this section. The state and adjoint equations are solved using the fourth-order Runge Kutta algorithms. The parameter values in Table 1 are used.

Table 1: Parameter description

Parameter	**Value**	**Source**
Λ	10/day	[17]
ω	0.000143/day	[17]
τ_1	0.009/day	Assumed
ρ	0.01/day	[20]
τ_2	0.009/day	Assumed
η	0.000143/day	[17]
μ	0.032143/day	[17]
κ	0.091/day	Assumed
ω_1	0.008/day	Assumed
ω_2	0.009/day	Assumed
ω_3	0.0091/day	Assumed
ω_4	0.0092/day	Assumed
α_2	0.0900982/day	Assumed
ρ_1	0.03/day	[20]
ρ_2	0.1/day	[20]
ρ_3	0.00811/day	Assumed
ρ_4	0.1/day	Assumed
α_1	0.1/day	[20]
ξ	0.009/day	Assumed
π_s	0.009/day	Assumed
π_g	0.009/day	Assumed
θ_s	0.0039/day	Assumed
θ_g	0.0039/day	Assumed
σ_s	0.01/day	Assumed
σ_g	0.0165 /day	[18]
σ_{gs}	0.01/day	Assumed
δ_s	0.0048/day	[20]
δ_g	0.021429/day	[17]
δ_{gs}	0.0052/day	Assumed
ϕ_s	0.5 /day	[20]
ϕ_g	0.033/day	[18]
ϕ_{gs}	0.1/day	Assumed

In the simulation process, the following control strategies were employed to determine the effective control strategy for Gumboro-Salmonellosis co-infection.

i) Strategy I: Vaccination only
Here, chickens are vaccinated against Salmonella and Gumboro diseases (u_1 and $u_2 \neq 0$, $u_3 = u_4 = u_5 = u_6 = u_7 = 0$).

ii) Strategy II: Supportive measures
Here the supportive measures that help in the recovery of the birds at both early and later stages of infections are used (u_3, u_4 and $u_5 \neq 0, u_1 = u_2 = u_6 = u_7 = 0$).

iii) Strategy III: Environmental sanitation only
Here the environmental strategies to eliminate the Salmonella bacteria and Gumboro virus in the environment are employed ($u_6 = u_7 \neq 0, u_1 = u_2 = u_3 = u_4 = u_5 = 0$).

iv) Strategy IV: Vaccination and Supportive Measures.
Vaccination of the birds and the Supportive measures are employed in this stage (u_1, u_2, u_3, u_4 and $u_5 \neq 0, u_6 = u_7 = 0$).

v) Strategy V: Vaccination and Environmental Sanitation
Here vaccination of chicken and environmental sanitation control measures are employed (u_1, u_2, u_6 and $u_7 \neq 0, u_3 = u_4 = u_5 = 0$.

vi) Strategy VI: Supportive measures and Environmental sanitation
Here supportive measures and environmental sanitation strategies are employed.($u_1 = u_2 = 0, u_3, u_4, u_5, u_6$ and $u_7 \neq 0$.

vii) Strategy VII: Vaccination, Supportive Measures and Environmental Sanitation
Here all the control strategies are employed ($u_1 = u_2 = u_3 = u_4 = u_5 = u_6 = u_7 \neq 0$).

6.1. Numerical results and discussion

6.1.1. Control Strategy I-vaccination of birds against Salmonella and Gumboro (u_1,u_2)

In this strategy vaccination of birds against salmonellosis u_1 and vaccination of birds against Gumboro u_2 are used to optimize the objective function J while the other control strategies , that is (u_3, u_4, u_5, u_6, u_7) are set at zero. The simulation results of the infected, co-infected, and pathogen compartments when the vaccination strategy is implemented are illustrated in Figure 2 below.

From Figures 2(a), 2(b), 2(c) and 2(d), it's shown that whenever birds are vaccinated against salmonella and gumboro diseases the solution curves for birds at early of Salmonella infection E_s, birds at early Gumboro infection E_g, birds at early stages of Salmonella-Gumboro co-infection E_{gs} and birds at acute stage of salmonella infection I_s, converges to zero implying that vaccination has a positive impact in controlling bird infections in E_s, E_g and E_{gs}. Figure 2(f) shows no significant impact of the control strategy at acute stages of Gumboro-Salmonella co-infections as the solution curves with and without control converge to zero an observation that can be related to the increased death rates at this stage. From 2(e), 2(g), and 2(h) it is observed that birds at acute stages of Gumboro infection I_g, salmonella pathogens C_B, and Gumboro pathogens C_V increase with control

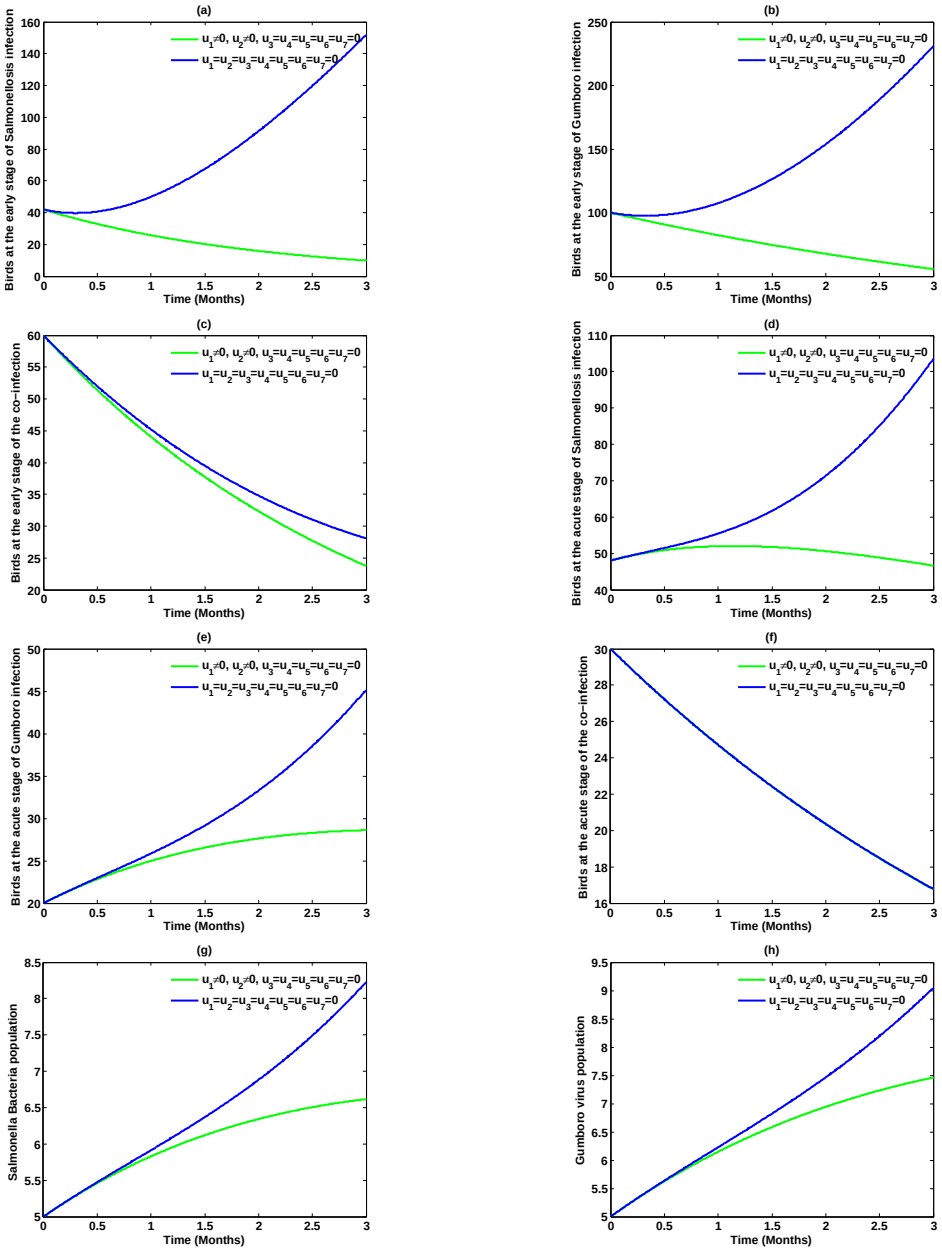

Figure 2: Effects of Strategy I on the dynamics of the co-infection optimal control model

though at a slower rate than without control probably because no other strategies are employed to curb shedding of pathogens to the environment and thus vaccination strategy alone is not an effective strategy in controlling the salmonella -Gumboro co-infection

6.2. Control Strategy II

Here the simulation results are illustrated to show the impact of implementing supportive measures control strategy only on infection, co-infection, and pathogen classes

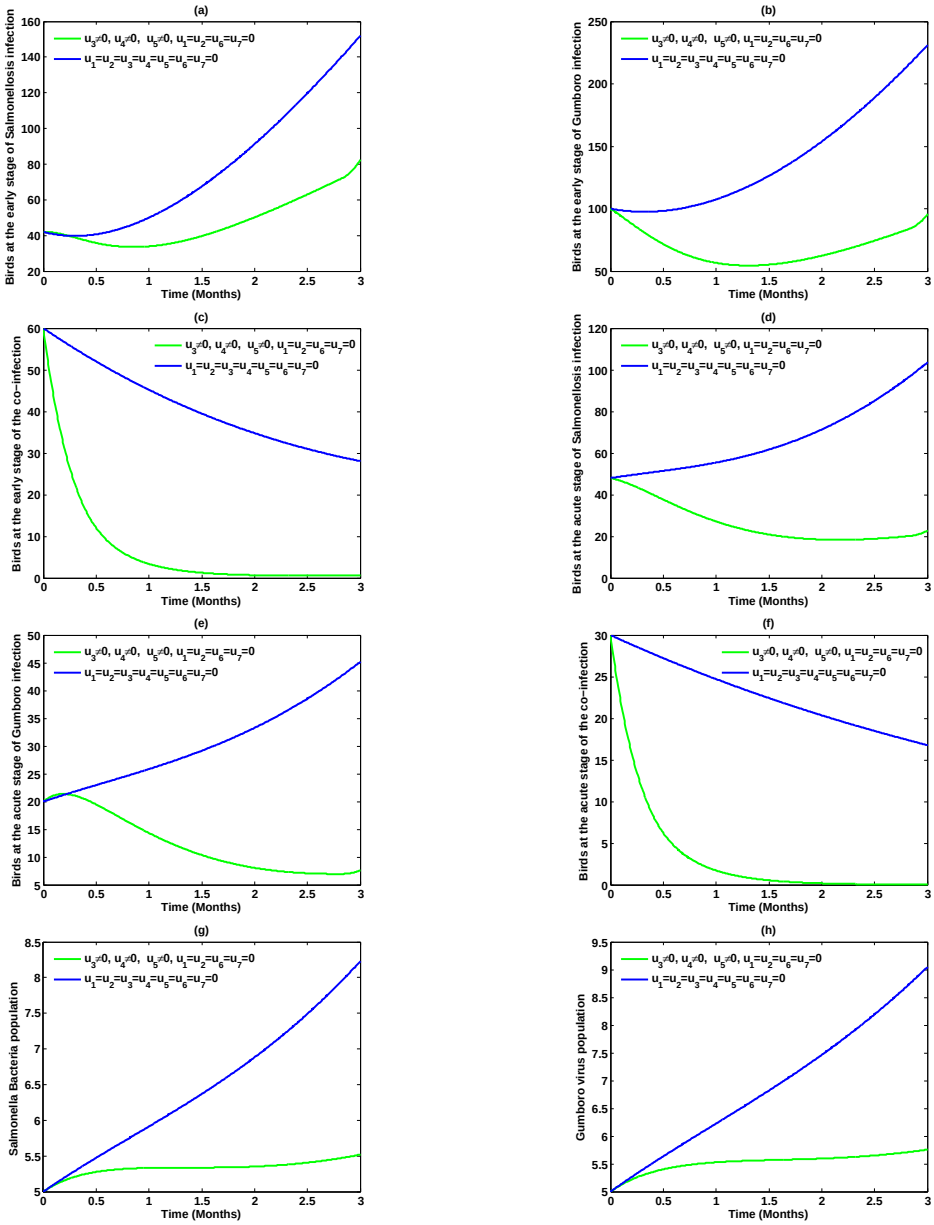

Figure 3: Effects of Strategy II on the dynamics of the co-infection optimal control model

(See Figure 3) From figures 3(a), 3(b), and 3(d), it is observed that birds in E_s, E_g and I_s decreases then increases with control probably because birds aren't immune and their environment is contaminated. Figure 3(e), birds at I_g initially increase then decrease, and then increase. From Figure 3(c) and Figure 3(f) birds at early stage of Gumboro-Salmonella co-infection E_{gs} and birds at acute stages Gumboro-Salmonlla co-infection I_{gs} decreases sharply and then converges to zero with control hence the control strategy is effective for birds at E_{gs} and I_{gs}. From Figure 3(g) and Figure 3(h) the solution curves for C_B and C_V with control are seen to increase though at a lower rate than without control

probably because birds atE_s,E_g,$I_s$and I_g still sheds pathogens Which renders this strategy ineffective.

6.3. Control Strategy III

Figure 4 illustrates the simulation results whenever the environmental sanitation-only control strategy is implemented on the infection, co-infection and pathogen classes. From Figure 4(a), 4(b), 4(c),4(d),4(e) and 4(f) it is observed that the control strategy has no impact on birds at E_s,E_g,$E_g s$,I_s,$I_g$and $I_g s$ as the solution curves with and without control are same however in figures 4(c) and 4(f) the solution curves with and without control for birds at $E_g s$ and $I_g s$ assumes a down tragectory probably due increased death rates again, From Figure 4(g) and Figure 4(h) it observed that the salmonella pathogen and Gumboro pathogen decrease initially and then rise. This can be associated to the fact that chickens at E_s,E_g,I_s, and I_g keep on shedding pathogens to the environment hence the control is ineffective in preventing and controlling the Gumboro-Salmonella co-infection.

6.4. Control Strategy IV

Figure 5 illustrates the simulation results whenever vaccination and supportive measures control strategies are implemented on the infected,co-infection, and the pathogen concentration in the environment. From figure 5(a), 5(b), 5(c), 5(d), and 5(f) it is shown that birds in E_s, E_g, $E_g s$, I_s, I_{gs} converges to zero with controls. From Figure 5(g) and 5(h), it is observed that the C_B and C_V increases initially and then decreases.Probably because of the reduced shedding from ineffective birds. however, 5(e) shows an increase followed by a decrease then an increase of birds at I_g hence the inefficiency of the control strategy.

6.5. Control Strategy V

Figure 6 illustrates the simulation results whenever vaccination and environment sanitation control strategies are implemented on the infection, co-infection, and pathogen concentration in the environment. From the Figures 6(a), 6(b), 6(c) it is observed that the solution curves for birds at E_s, E_g and E_{gs} converges to zero,while from figure 6(d), the birds at I_s are seen to increase and decrease with controls. From figure 6(f) it is observed that vaccination combined with environmental sanitation has no impact on birds at I_{gs} and therefore, the decrease of chicken at this stage with and without control might be as a result of mortality cases experienced at this stage. Figure 6(e), shows that the solution curves for birds at I_g increase though at a lower rate with controls than without controls and also from figures 6(g) and 6(h) a decrease followed by an increase is observed in Salmonella $C_B$and Gumboro pathogenC_V with controls probably because of shedding by birds at infective classes declaring this control ineffective.

6.6. Control Strategy VI

Figure 7 illustrates the simulation results whenever supportive measures and environmental sanitation control strategies are implemented on the infection, co-infection, and pathogen environment concentration. Figures 7(a), 7(b), 7(d), 7 (g) and 7 (h) show that the birds at E_s, E_g, I_s, and C_B and C_V initially decrease then rise with control and also

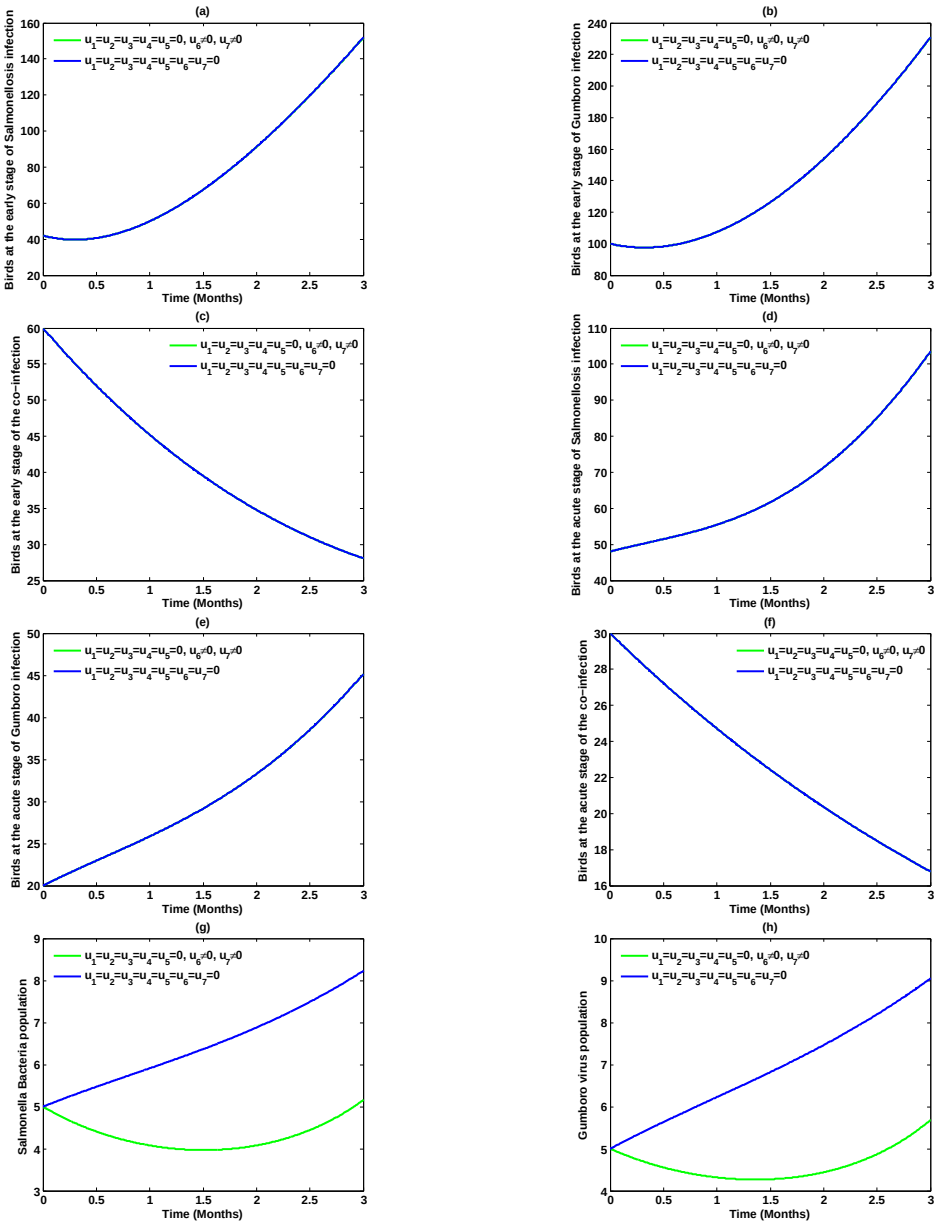

Figure 4: Effects of Strategy III on the dynamics of the co-infection optimal control model

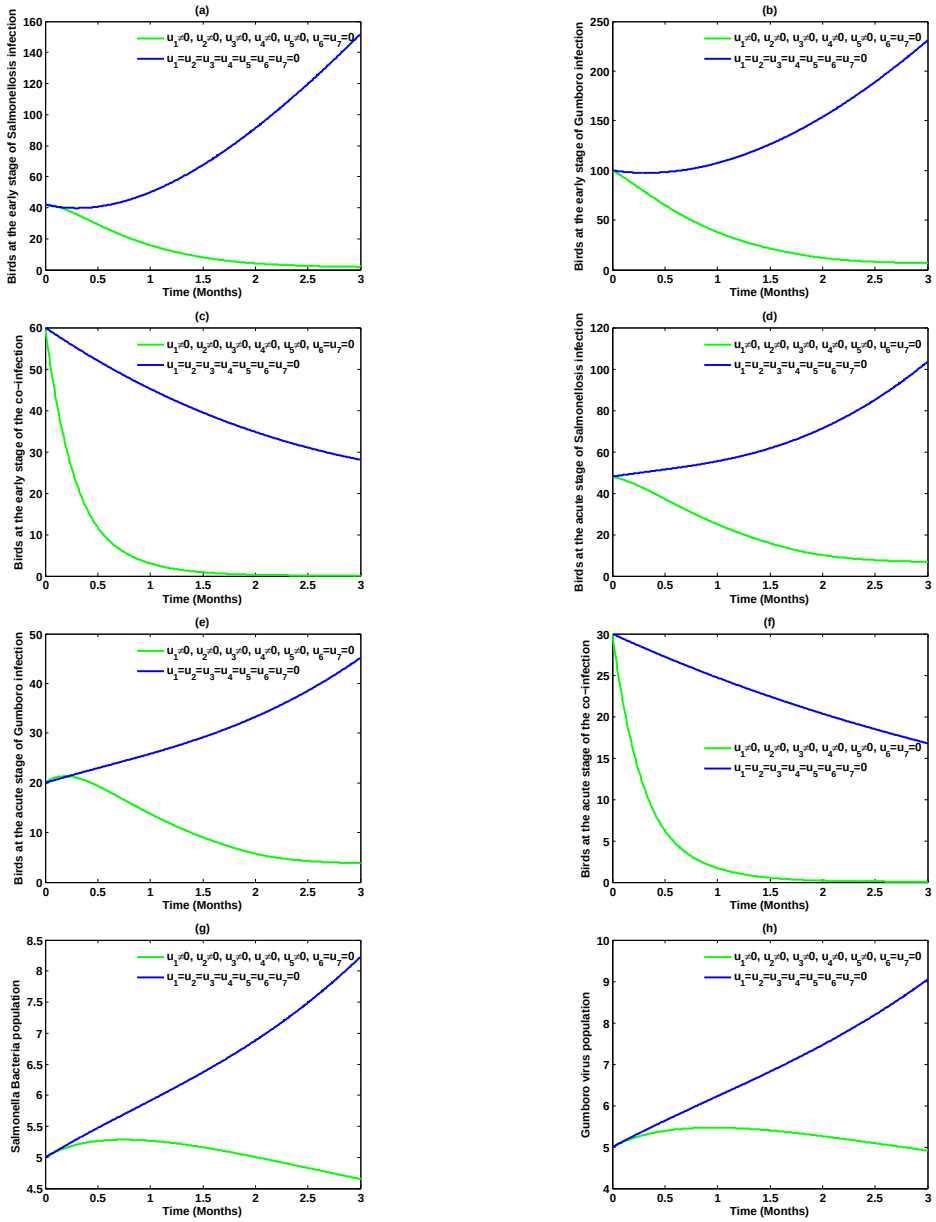

Figure 5: Effects of Strategy IV on the dynamics of the co-infection optimal control model

from Figure7(e) shows an initial increase followed by a decrease and then an increase for I_g.Results that can be associated to the fact that the birds are not immuned to diseases. Hence, rendering the strategy ineffective although from 7(c) and 7(f) the strategy seems effective as E_{gs} and I_{gs} converges to zero.

6.7. Control Strategy VII

Figure 8 illustrates the simulation results whenever all control strategies that is vaccination, supportive measures, and environmental sanitation control strategies are im-

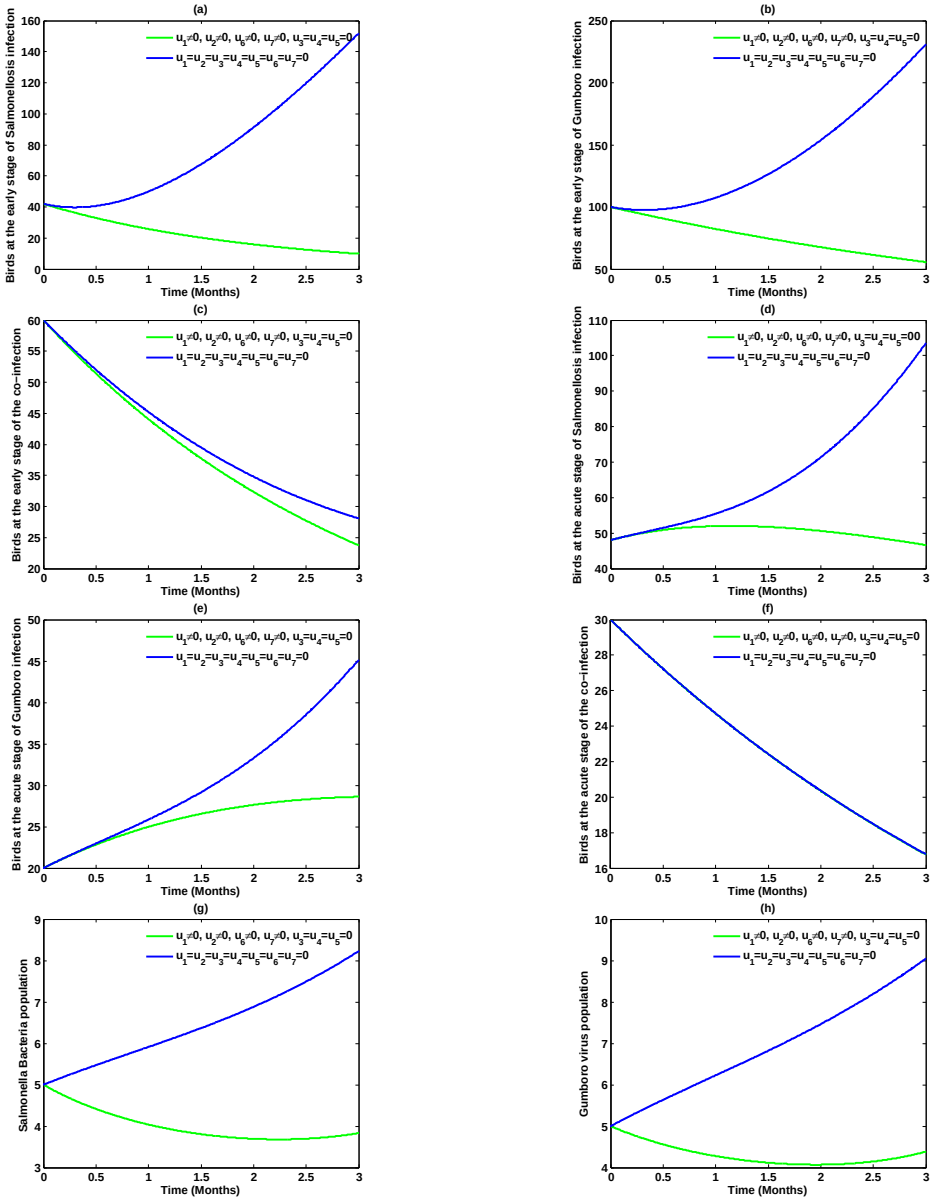

Figure 6: Effects of Strategy V on the dynamics of the co-infection optimal control model

plemented on the infection,co-infection, and pathogen environment concentration. From figure 8(a), 8(b), 8(c), 8(d), 8(f), 8(g), 8(h) it is observed that birds at E_s, E_g, E_{gs}, I_s, I_g, I_{gs} together with C_B, C_V converges to zero with controls, also figure 8(e) indicates a initial increase then decrease of birds at I_g with controls which qualifies this strategy as the most effective in prevention and control of Gumboro-Salmonella co-infection.

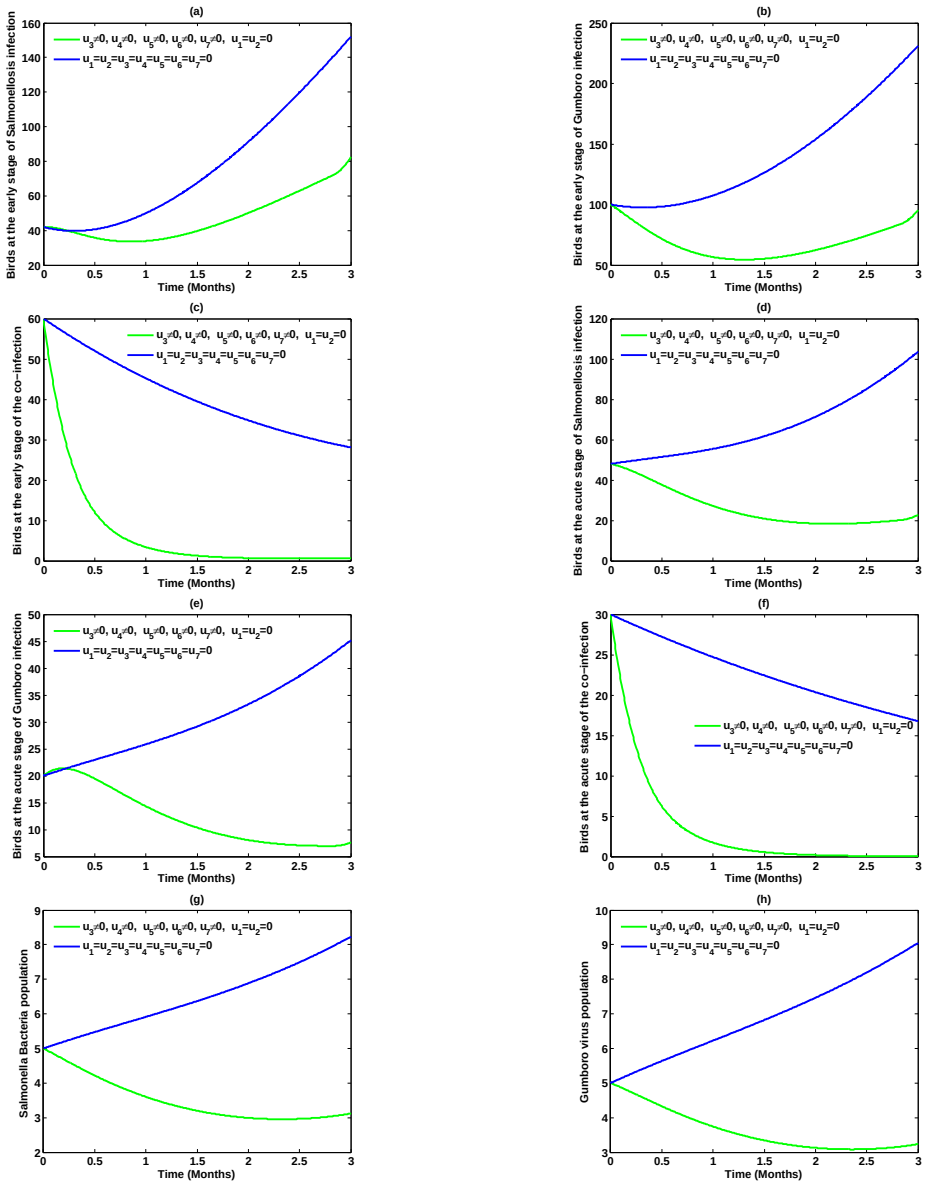

Figure 7: Effects of Strategy VI on the dynamics of the co-infection optimal control model

7. Conclusion

A Gumboro-Salmonella co-infection mathematical model with vaccination of birds against gumboro diseases, vaccination of birds against salmonella disease, treatment of birds with Gumboro infections, Salmonella infections, and Gumboro-salmonella co-infection by use of supportive measures, elimination of Gumboro virus from the environment and elimination of salmonella bacteria from the environment was developed in this paper. The model's solution set is positive and bounded as revealed by the model's qualitative examination. Pontryagin's maximum principle was used to formulate the optimal control problem and the optimal control condition was analyzed. The optimality system is de-

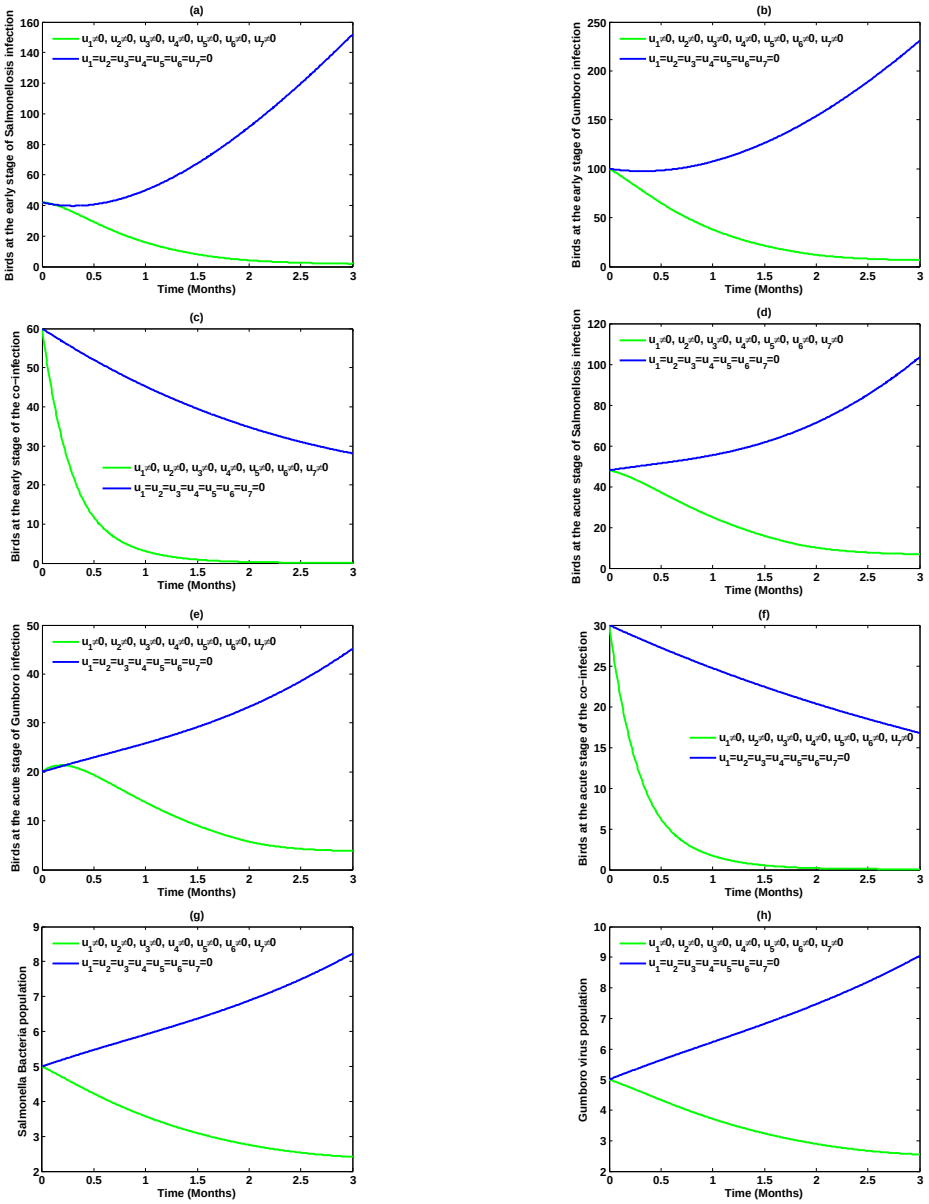

Figure 8: Effects of Strategy VII on the dynamics of the co-infection optimal control model

veloped and the optimal control requirement existence identified seven (7) strategies for elimination of Gumboro-Salmonella co-infection are recommended and the effectiveness of each strategy is carefully examined. A numerical examination of the recommended strategies was used and the results graphically presented. From the results, it was realized prevention and control of Gumboro-salmonella co-infection is possible only by combining all the strategies.

Acknowledgement

The authors appreciate all support accorded to them by their colleagues and the university in the writing of this manuscript.

References

[1] Zootecnica intenational 2016. Poultry sector in Kenya. Retrieved from https://zootecnicainternational.com/field-reports/poultry-sector-kenya/On8/8/2021

[2] Mengesha, M., and Tsega, W. (2011). Phenotypic and genotypic characteristics of indigenous chicken in Ethiopia. African Journal of Agricultural Research Vol. 6(24), pp. 5398-5404, 26 October, 2011. DOI:10.5897/AJAR11.434I.

[3] Kingori, A.M., Wachira, A.M., Tuitoek, J.K. (2010). Indigenous chicken production in kenya:Areview.international journal of poultry science v.9 pg309-316. Doi:10.3923/ijps.2010.309-316.

[4] Nyaga, P. (2007) Strategies for prevention and control of infectious disease.Food and agricultural organization of the united nations.

[5] George, T. and Jessica, B.W. (2018). Infectious bursal disease(GUMBORO)in backyard chicken. Mississippi state university publication number P3211.

[6] Mutinda, W.U., Nyaga, P.N., Njagi, L.W., Bebora L.C., Mbuthia, P.G. (2018). Gumboro disease outbreaks cause high mortality rates in indigenous chickens in kenya.Bulletin health and production Africa 61.571-578.

[7] Marketing Kepro (2017) Salmonellosis in poultry. Retrieved from https://www.viv.net/articles/blog/salmonellosis-in-poultryon23/6/2021

[8] Liljebjelke, K.A, Hofacre, C.L., Liu, T., White, D.G., Ayers, S., Young, S., Maurer J.J. (2005). Vertical and horizontal transmission of salmonella within integrated broiler production system. Foodborne Pathog Dis. 2005 Spring;2(1):90-102. doi:10.1089/fpd.2005.2.90.PMID:15992303.

[9] Chong, S.N., Michel, T.J., Robert, J.S. (2013). A mathematical model of Avian influenza with half saturated incidence. Theory Biosci. (2014) 133:23–38. DOI10.1007/s12064-013-0183-6

[10] Mishra, B.K., Sinha, D.N. (2016). A Mathematical Model on Avian Infuenza with Quarantine and Vaccination. J Immunol Tech Infect Dis 5:4. doi:10.4172/2329-9541.1000152

[11] Zhang, J., Yun, L., Zhen, J.,and Zhu H.,(2019).Dynamics analysis of avian influenza a(H7N9) Epidemic m,odel with vaccination and seasonality. Volume 2019, Article ID 4161287, 15 pages https://doi.org/10.1155/2019/4161287

[12] Sunday, E. and Sampson, M.I. (2014). Mathematical model for the epidemiological of the fowl pox infection transmission that incorporates discrete delay. IOSR Journal of Mathematics (IOSR-JM);Vol 10, Issue 4, PP 08-16

[13] Sunda, E.U. and Inyama, S.C. (2011a). Mathematical model of the impact of Vaccination on transmission dynamics of fowl pox in poultry. Journal of Modern Mathematics and Statistics. DOI:10.3923/jmmstat.2011.102.105

[14] Sunday,E. and Inyama S.C.(2012).Application of optimal control to the epidemiology of fowl pox transmission dynamics in poultry. Journal of Mathematics and Statistics; Vol 8, pages 248-252.

[15] Uwakwe, J.I., Inyama, S.C., Omame, A. (2020) Mathematical Model and Optimal Control of New-Castle Disease (ND); Applied and Computational Mathematics; Vol. 9, No. 3, 2020, pp. 70-84. doi:10.11648/j.acm.20200903.14 (June 4th)

[16] Alfred, H, Makinde, O.D., Kumar, S. and Chibwana, F. (2017). Optimal control and cost effectiveness analysis for Newcastle disease eco-epidemiological model in Tanzania. Journal of biological Dynamics 11(1):190-209. Doi10.1080/17513758.2016.1258093.

[17] Omollo, A.E., and George, K.,(2020).Formulation of a Mathematical Model for the Transmission Dynamics of Infectious Bursal Disease (IBD), Incorporating Eects of Environmental Factors. Asian Research Journal of Mathematics, Page 20-35. DOI:10.9734/arjom/2020/v16i930217 Published: 21 August 2020

[18] Uwakwe, J.,Inyama, S.C.,Emerenini B.O.,Celestine A.N(2019) .A mathematical and control model of Bursal disease (ibd). IOSR Journal of Mathematics (IOSR-JM) e-ISSN: 2278-5728, p-ISSN: 2319-765X. Volume 15, Issue 4 Ser. III (Jul – Aug 2019), PP 15-29 www.iosrjournals.org

[19] Musaili, J. S., Chepkwony, I., & Mutuku, W. N. (2024). Mathematical Modeling of the Transmission Dynamics of Gumboro Disease. Journal of Applied Mathematics, 2024(1), 2514740.
[20] Prevost, K., Magal, P., & Beaumont, C. (2006). A model of Salmonella infection within industrial house hens. Journal of Theoretical Biology, 242(3).
[21] Daozhou G,Shigui R and Travis P (2016).Coinfection dynamics of two diseases in a single host population. Journal of Mathematical Analysis and Applications 442(1). DOI:10.1016/j.jmaa.2016.04.039
[22] Shaibu, O., and Daniel, O.M.,(2018). A mathematical model for coinfection of listeriosis and antrax diseases. International journal of mathematics and mathematics sciences.Vol. 2018, 1725671. https://doi.org/10.1155/2018/1725671
[23] Lusiana, V., Nuraini, N. and Soewono, E. (2017) Mathematical modelling of transmission coinfection dynamics of tuberculosis in HIV community. Indonesia
[24] Pontryagin, L. S., Boltyanskii, V. G., Gamrelidze, R. V., and Mishchenko, E. F. (1962). The Mathematical Theory of Optimal Processes. *John Wiley, New York*