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Research Article

Mathematical Modeling of the Transmission Dynamics of Gumboro Disease

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Gumboro disease is a viral poultry disease that causes immune suppression on the infected birds leading to poor production, mortality, and exposure to secondary infections, hence a major threat in the poultry industry worldwide. A mathematical model of the transmission dynamics of Gumboro disease is developed in this paper having four compartments of chicken population and one compartment of Gumboro pathogen population. The basic reproduction number R_{og} is derived, and the dynamical behaviors of both the disease-free equilibrium (DFE) and endemic equilibrium are analyzed using the ordinary differential equation theory. From the analysis, we found that the system exhibits an asymptotic stable DFE whenever $R_{og} < 1$ and an asymptotic stable EE whenever $R_{og} > 1$. The numerical simulation to verify the theoretical results was carried out using MATLAB ode45 solver, and the results were found to be consistent with the theoretical findings.

Keywords: basic reproduction number; Gumboro; mathematical model; stability

1. Introduction

Infectious bursal disease (IBD) popularly known as Gumboro is a viral poultry diseases that cause high morbidity and mortality, hence a major threat to the poultry industry due to high economic losses associated with it worldwide. Gumboro disease is associated with clinical disease symptoms such as depression, watery diarrhea, ruffled feathers, and dehydration [1].

Gumboro disease affects mostly young chickens around 3–6 weeks of age. Gumboro virus is extremely difficult to eradicate as it is hardy and can live in a great range of environmental conditions, and it is transmitted from one bird to another through feacal–oral route [2].

Generally, the poultry sector plays an important role in the growth of the economy as well as in poverty reduction. According to the Agricultural Sector Development Strategy 2010, in Kenya, each year, about 20 tonnes of poultry meat worth 3.5 billion Kenyan shillings and 1.3 billion eggs worth 9.7 billion Kenyan shillings are produced. This increased production of poultry is necessitated by the increased demand for quality protein especially in developing countries [3].

The IBD virus was observed 40 years ago with Kenya's first case reported in 1991 in commercial birds on the Kenyan coast; the disease has remained to be a great threat to the commercial poultry industry not only in Kenya but also in the whole world [4].

Mathematical modeling over the years has become a very important tool that is used in the prediction, assessment, and control of various outbreaks. A number of these models that describe the impacts of preventive and control strategies on the transmission dynamics of various poultry infectious diseases have been developed. A study to investigate the impacts of quarantine and vaccination in controlling avian

influenza disease was done by [5], and the results established that combining quarantine with vaccination is an effective strategy for control of the disease.

A mathematical model of Newcastle disease with optimal control having five compartments was formulated by [6]. The findings of the study showed that in the absence of control measures, the number of infected bird increased significantly and reduced significantly in the presence of control measure implying that the control measures were effective methods of controlling the disease.

Several mathematical models have been done to describe the dynamics of Gumboro disease; for instance, [7] formulated a model to investigate the impact of the environment in the spread of Gumboro infections while [8] described a model to describe the effects of vaccination and biosecurity measures in controlling Gumboro infection.

Although several Gumboro models have been developed, to the best of our knowledge, a Gumboro model with pathogen compartment has not been developed. In this paper, we formulate a Gumboro model capturing the pathogen compartment to study the dynamics of Gumboro disease.

2. Model Formulation

A model with Gumboro pathogen population N_g and the chicken population N_c is developed in this research. Thus, the total population at a given time (t) is $N(t) = N_c(t) + N_g(t)$. Gumboro pathogen population N_g has one compartment consisting of concentration of Gumboro virus in the environment C_V. The chicken population N_c is grouped into four compartments which consist of susceptible chicken S, birds that are at early stages of infection with Gumboro E_g, birds that are in acute stages of infection with Gumboro I_g, and those that will recover from Gumboro disease in both early and acute stages of infections simultaneously R. The model assumes that the bird population recruitment rate to the susceptible compartment will be Λ. The susceptible birds are infected with Gumboro at the rate of $\omega C_V/\tau_2 + C_V$. Where ω is the contact rate of susceptible birds with an IBD virus-contaminated environment, τ_2 is the IBD virus concentration in the environment with a 50% probability of Gumboro infections. All bird populations experience natural death at the rate η. Additionally, they die from Gumboro at the rate of μ. The Gumboro-infected birds in both stages of infection shed the virus to the environment at the rates ω_{1-2} which die at the rate α_2, while σ_g and δ_g are recovery rates for birds at both early and acute stages of infection. Birds at the early stages of Gumboro infections move to the acute stages of infection at the rates ϕ_g.

2.1. Model Assumptions. The model assumptions are as follows:

1. The bird species that are infected with Gumboro is chicken.
2. Chickens are recruited into the system by birth or immigration.

2.2. Model Flow Chart and Equations. From Figure 1, the following equations are developed:

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \left(\frac{\omega C_v}{\tau_2 + C_v} + \eta\right) S \\
\frac{dE_g}{dt} &= \frac{\omega C_v}{\tau_2 + C_v} S - \left(\sigma_g + \phi_g + \eta + \mu\right) E_g \\
\frac{dI_g}{dt} &= \phi_g E_g - (\delta_g + \eta + \mu) I_g \\
\frac{dR}{dt} &= \sigma_g E_g + \delta_g I_g - \eta R \\
\frac{dC_V}{dt} &= \omega_1 E_g + \omega_2 I_g - \alpha_2 C_V
\end{aligned} \tag{1}$$

3. Basic Properties of the Model

In this section, we discuss the positivity and boundedness of the solutions of the model.

3.1. Positivity of the Solutions of the Model

Theorem 1. *There exists a nonnegative solution set* $\{S, E_g, I_g, R, C_V\}(t)$ *of model (1) for all* $t > 0$ *given that the initial conditions* $S(0) > 0, E_g(0) \geq 0, I_g(0) \geq 0 \quad R(0) \geq 0, C_V(0) \geq 0$ *in* $\mathbb{R}^5_+$ *have nonnegative values.*

Proof 1. From the first equation of system (1), we have

$$\frac{ds}{dt} = \Lambda - \left(\gamma_g + \eta\right) S \quad \text{where} \quad \gamma_g = \frac{\omega C_v}{\tau_2 + C_v} \tag{2}$$

Equation (2) can be expressed as

$$\frac{ds}{dt} > -\left(\gamma_g + \eta\right) S \tag{3}$$

Separating the variables, we obtain

$$\frac{ds}{s} > -\left(\gamma_g + \eta\right) dt \tag{4}$$

Integrating on both sides of Equation (4), we have $\ln S > -(\gamma_g + \eta)t + c_1$ or

$$S(t) > ce^{-\left(\gamma_g + \eta\right)t} \tag{5}$$

From Equation (5), it is clear that $S(0) = c$ for $t = 0$.

Therefore, $S(t) > S(0)e^{-(\gamma_g + \eta)t}$ and as $t \longrightarrow \infty$, we have $S(t) > 0 \forall t > 0$.

Also from the second equation of system (1)

$$\frac{dE_g}{dt} = \gamma_g S + -\left(\sigma_g + \phi_g + \eta + \mu\right) E_g$$

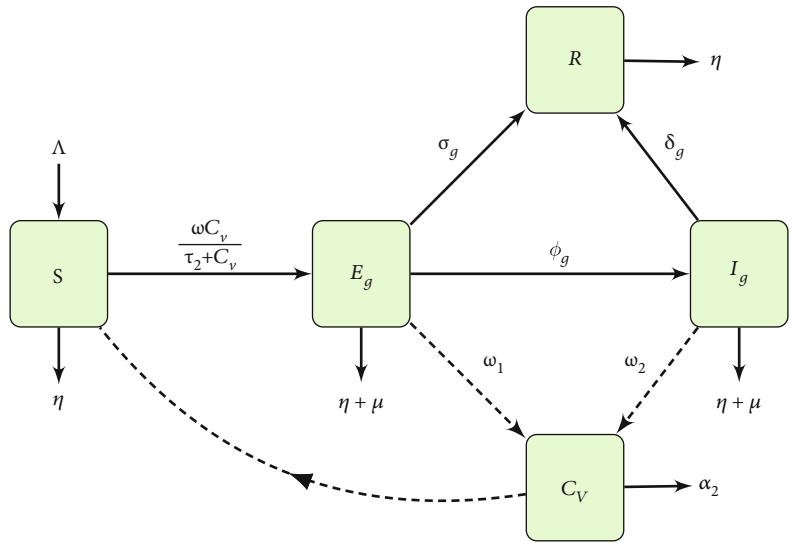

Figure 1: Flow chart.

we have

$$\frac{dE_g}{dt} \geq -\left(\sigma_g + \phi_g + \eta + \mu\right)E_g$$

Solving the above equation by separation of variables, we have

$$\frac{dE_g}{E_g} \geq -\left(\sigma_g + \phi_g + \eta + \mu\right)dt$$

Upon integrating on both sides, we have $\ln E_g(t) \geq -(\sigma_g + \phi_g + \eta + \mu)t + c$:

$$\Rightarrow \ln E_g(t) > ce^{-\left(\sigma_g+\phi_g+\eta+\mu\right)t}. \tag{6}$$

Clearly for $t = 0$, $c = E_g(0)$.

Thus, Equation (6) becomes

$$E(t) \geq E_g(0)e^{-\left(\sigma_g+\phi_g+\eta+\mu\right)t}$$

and as $t \longrightarrow \infty$, we have

Applying the same method to other equations of the system (1), we get

$$\frac{dI_g}{dt} \geq -\left(\delta_g + \eta + \mu\right)I_g \Rightarrow I_g(t) \geq I_g(0)e^{-\left(\delta_g+\eta+\mu\right)t} \geq 0$$

$$\frac{dR}{dt} \geq -\eta R \Rightarrow R(t) \geq R(0)e^{-\eta t} \geq 0$$

$(dC_V/dt) \geq -\alpha_2 C_V \Rightarrow C_V(t) > C_V(0)e^{-\alpha_2 t} \geq 0$. Thus $(S(0) > 0, E_g(0) \geq 0, I_g(0) \geq 0, R(0) \geq 0, C_V(0) \geq 0)$ for all $t > 0$. □

3.2. Boundedness of the Solutions of the Model. Let $\Omega = (\Omega_C \cup \Omega_{C_V}) \subset \mathbb{R}^5_+$ 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 the bird population and that of the Gumboro pathogen population is given by Ω_{C_V}. We show that the solutions of the system (1) are bounded in the feasible region.

The total bird population is N_C given by

$$N_C = S(t) + E_g(t) + I_g(t) + R(t)$$

$$\frac{dN_C}{dt} = \frac{dS}{dt} + \frac{dE_g}{dt} + \frac{dI_g}{dt} + \frac{dR}{dt}$$

Thus, from system (1), we have

$$\frac{dN_C}{dt} = \Lambda - \eta\left(S(t) + E_g(t) + I_g(t) + R(t)\right) - \left(\mu E_g + \mu I_g\right) \tag{7}$$

When the birds are not infected, Equation (7) reduces to

$$\frac{dN_C}{dt} \leq \Lambda - \eta N_c \tag{8}$$

Upon solving Equation (8), we get

$$N_c(t) \leq \frac{\Lambda}{\eta} + \left(N_c(0) - \frac{\Lambda}{\eta}\right)e^{-\eta t} \tag{9}$$

And taking limits as $t \longrightarrow \infty$, we have

$$N_c \leq \frac{\Lambda}{\eta} \tag{10}$$

Thus, the bird population is bounded in

$$\Omega_C = \left\{\left(S(t), E_g(t), I_g(t), R(t)\right) \in \mathbb{R}^4_+ : N_c \leq \frac{\Lambda}{\eta}\right\}$$

Considering the last equation of system (1), that is Equation (10), we have

$$\frac{dC_V}{dt} = \omega_1 E_g + \omega_2 I_g - \alpha_2 C_V \tag{11}$$

Upon reduction of Equation (11), we have

$$\frac{dN_g}{dt} \leq \frac{\Lambda(\omega_1 + \omega_2)}{\eta} - \alpha_2 N_g \tag{12}$$

By using the integrating factor, we solve Equation (12) to get

$$N_g \leq \frac{\Lambda(\omega_1 + \omega_2}{\eta\alpha_2} + \left(N_g(0) - \frac{\Lambda(\omega_1 + \omega_2)}{\eta\alpha_2}\right)e^{-\alpha_2 t} \tag{13}$$

Taking the limit of Equation (13), as t tends to infinity, gives $N_g \leq \Lambda(\omega_1 + \omega_2)/\eta\alpha_2$. Thus, the Gumboro population is bounded in the region

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

Since the bird population and Gumboro pathogen population are bounded, then the model will be analyzed in a suitable feasible region

$$\Omega = \left\{ \left(S, E_g, I_g, R\right) \in \mathbb{R}_+^4 ; C_V \in \mathbb{R}_+ ; S > 0 \right.$$

$$\left. E_g, I_g, R, C_V \geq 0 ; N_c \leq \frac{\Lambda}{\eta} \right.$$

$$N_g \leq \frac{\Lambda(\omega_1 + \omega_2)}{\eta\alpha_2}$$

4. Analysis of the Model

4.1. Disease-Free Equilibrium (DFE) Point. The DFE of the system (1) is computed by letting $S = S^*$, $E_g = E_g^* = 0$, $I_g = I_g^* = 0$, $R = R^* = 0$, and $C_V = C_V^* = 0$ and setting the right-hand side of the equations of the system (1) equal to zero, then solving the resulting system of equations. Hence, we get

$$\text{DFE} = \mathscr{E}^* = \left(S^*, E_g^*, I_g^*, R^*, C_V^*\right) = \left(\frac{\Lambda}{\eta}, 0, 0, 0, 0\right)$$

4.2. Reproduction Number for Gumboro Model. The reproduction number R_{0g} is described as the average number of secondary cases that result from an average initial case in a completely susceptible population in [9]. In our situation, the reproduction number R_{0g} is the average number of secondary cases resulting from a typical Gumboro infection case in a completely uninfected population. R_{0g} is found using the Next Generation Matrix approach by [10]. Let the rates of new infections in class j be denoted by f_j, while the rates of chicken transfers into and out of class j are represented by v_j. The Next Generation Matrix is given by FV^{-1}, where F and V are the Jacobian matrices of the vectors f_j and v_j, respectively, at $\mathscr{E}_g^*$. The following equations capture the infected population.

$$\begin{aligned}
\frac{dE_g}{dt} &= \frac{\omega C_v}{\tau_2 + C_v} S - \left(\sigma_g + \phi_g + \eta + \mu\right) E_g \\
\frac{dI_g}{dt} &= \phi_g E_g - \left(\delta_g + \eta + \mu\right) I_g \\
\frac{dC_V}{dt} &= \omega_1 E_g + \omega_2 I_g - \alpha_2 C_V
\end{aligned} \tag{14}$$

From the system (14), we have

$$f_i = \begin{bmatrix} \dfrac{\omega C_V}{\tau_2 + C_V} S \\ 0 \\ 0 \end{bmatrix}$$

Also, from the system (14), we have

$$v_i = \begin{bmatrix} \left(\sigma_g + \phi_g + \eta + \mu\right) E_g \\ -\phi_g E_g + \left(\delta_g + \eta + \mu\right) I_g \\ -\omega_1 E_g - \omega_2 I_g + \alpha_2 C_V \end{bmatrix}$$

By definition of F and V, we have

$$F = \begin{bmatrix} 0 & 0 & \dfrac{\omega\Lambda}{\tau_2\eta} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

and $V = \begin{bmatrix} \left(\sigma_g + \phi_g + \eta + \mu\right) & 0 & 0 \\ -\phi_g & \left(\delta_g + \eta + \mu\right) & 0 \\ -\omega_1 & -\omega_2 & \alpha_2 \end{bmatrix}$

where

$c_2 = \sigma_g + \phi_g + \eta + \mu c_5 = \delta_g + \eta + \mu$

Using Mathematica software, the inverse of V is given by

$$V^{-1} = \begin{bmatrix} \dfrac{1}{\left(\sigma_g + \phi_g + \eta + \mu\right)} & 0 & 0 \\ \dfrac{\phi_g}{\left(\sigma_g + \phi_g + \eta + \mu\right)\left(\delta_g + \eta + \mu\right)} & \dfrac{1}{\left(\delta_g + \eta + \mu\right.} & 0 \\ \dfrac{\left(\delta_g + \eta + \mu\right)\omega_1 + \phi_g\omega_2}{\left(\sigma_g + \phi_g + \eta + \mu\right)c_5\alpha_2} & \dfrac{\omega_2}{\left(\delta_g + \eta + \mu\right)\alpha_2} & \dfrac{1}{\alpha_2} \end{bmatrix}$$

Thus

$$FV^{-1} = \begin{bmatrix} \dfrac{\omega\Lambda\left((\delta_g+\eta+\mu)\omega_1+\phi_g\omega_2\right)}{\tau_2\eta\left(\sigma_g+\phi_g+\eta+\mu\right)(\delta_g+\eta+\mu)\alpha_2} & \dfrac{\omega\Lambda\omega_2}{\tau_2\eta(\delta_g+\eta+\mu)\alpha_2} & \dfrac{\omega\Lambda}{\tau_2\eta\alpha_2} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

which implies that the basic reproduction number, R_{0g}, for the Gumboro model is given by

$$R_{0g} = \frac{\omega\Lambda\left(c_5\omega_1+\phi_g\omega_2\right)}{\tau_2\eta c_2 c_5\alpha_2} = \frac{\omega\Lambda\left((\delta_g+\eta+\mu)\omega_1+\phi_g\omega_2\right)}{\tau_2\eta\left(\sigma_g+\phi_g+\eta+\mu\right)(\delta_g+\eta+\mu)\alpha_2}$$

4.3. Local Stability of DFE. The DFE $\mathscr{E}_g^*$ is locally asymptotically stable if all the real parts of the eigenvalues of the Jacobian matrix of the system (1) at the DFE $\mathscr{E}_g^*$ are all negative.

The Jacobian matrix of the system (1) at the DFE $\mathscr{E}_g^*$ is given by

$$J\left(\mathscr{E}_g^*\right) = \begin{bmatrix} -\eta & 0 & 0 & 0 & -\dfrac{\omega\Lambda}{\tau_2\eta} \\ 0 & -\left(\sigma_g+\phi_g+\eta+\mu\right) & 0 & 0 & \dfrac{\omega\Lambda}{\tau_2\eta} \\ 0 & \phi_g & -(\delta_g+\eta+\mu) & 0 & 0 \\ 0 & \sigma_g & \delta_g & -\eta & 0 \\ 0 & \omega_1 & \omega_2 & 0 & -\alpha_2 \end{bmatrix} \tag{15}$$

From the Jacobian matrix (15), we have the following characteristic equation:

$$(\lambda+\eta)^2\left\{\lambda^3+\left[\left(\sigma_g+\phi_g+\eta+\mu\right)+(\delta_g+\eta+\mu)+\alpha_2\right]\lambda^2 + \left[\left(\sigma_g+\phi_g+\eta+\mu\right)(\delta_g+\eta+\mu)+\alpha_2\left(\left(\sigma_g+\phi_g+\eta+\mu\right)+(\delta_g+\eta+\mu)\right)-\frac{\omega\Lambda\omega_1}{\tau_2\eta}\right]\lambda+\left(\sigma_g+\phi_g+\eta+\mu\right)(\delta_g+\eta+\mu)\alpha_2 - \frac{\omega\Lambda\left((\delta_g+\eta+\mu)\omega_1+\phi_g\omega_2\right)}{\tau_2\eta}\right\}=0 \tag{16}$$

In view of Equation (16), $\lambda_1 = -\eta$ and $\lambda_2 = -\eta$ are the eigenvalues of the Jacobian matrix (15). The other three eigenvalues can be obtained from the following reduced characteristic equation:

$$a_0\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0 \tag{17}$$

where

$$a_0 = 1$$

$$a_1 = \left(\sigma_g+\phi_g+\eta+\mu\right)+(\delta_g+\eta+\mu)+\alpha_2$$

$$a_2 = \left(\sigma_g+\phi_g+\eta+\mu\right)(\delta_g+\eta+\mu)+\alpha_2\left(\left(\sigma_g+\phi_g+\eta+\mu\right)+(\delta_g+\eta+\mu)\right)-\frac{\omega\Lambda\omega_1}{\tau_2\eta} = (\delta_g+\eta+\mu)\left(\left(\sigma_g+\phi_g+\eta+\mu\right)+\alpha_2\right)+\frac{\omega\Lambda\phi_g\omega_2}{(\delta_g+\eta+\mu)\tau_2\eta}+\alpha_2\left(\sigma_g+\phi_g+\eta+\mu\right)(1-R_{0g})$$

$$a_3 = \left(\sigma_g+\phi_g+\eta+\mu\right)(\delta_g+\eta+\mu)\alpha_2-\frac{\omega\Lambda\left((\delta_g+\eta+\mu)\omega_1+\phi_g\omega_2\right)}{\tau_2\eta}$$

It is clear that

$$a_0, a_1 > 0$$

and $a_2, a_3 > 0$ if $R_{0g} < 1$

According to Routh–Hurwitz criteria, Equation (17) has roots with negative real parts if $a_0 > 0, a_1 > 0, a_2 > 0, a_3 > 0$ and $a_1a_2 - a_0a_3 > 0$. Thus, the DFE of the system of Equation (1) is locally asymptotically stable if the following theorem holds.

Theorem 2. *The DFE of the system of Equation (1) is locally asymptotically stable when $R_{0g} < 1$ and unstable otherwise.*

4.4. Global Stability of DFE. In this section, we use the Castillo–Chavez theorem in [11] to analyse the disease-free state's global asymptotic stability. To begin, the system (1) must be expressed in the following format:

$$\frac{d\mathscr{X}_g}{dt} = F\left(\mathscr{X}_g, \mathscr{Z}_g\right)$$
$$\frac{d\mathscr{Z}_g}{dt} = H\left(\mathscr{X}_g, \mathscr{Z}_g\right), H\left(\mathscr{X}_g, 0\right) = 0 \tag{18}$$

where $\mathscr{X}_g = (S, R)$ and $\mathscr{Z}_g = (E_g, I_g, C_V)$. Uninfected individuals are represented by the components of $\mathscr{X}_g \in \mathbb{R}$, while infected ones are represented by the components of $\mathscr{Z}_g \in \mathbb{R}$. The system's DFE now becomes $\mathscr{E}_g^* = (\mathscr{X}^*, 0)$, $\mathscr{X}_g^* = ((\Lambda/\eta), 0)$. The following two conditions must be met to provide global asymptotic stability.

1. $d\mathscr{X}_g/dt = F(\mathscr{X}_g, 0), \mathscr{X}_g$ is globally asymptotically stable (GAS)

$$2.\ H\left(\mathscr{X}_g, \mathscr{Z}_g\right) = G\mathscr{Z}_g - \tilde{H}\left(\mathscr{X}_g, \mathscr{Z}_g\right), \tilde{H}\left(\mathscr{X}_g, \mathscr{Z}_g\right) \geq 0 \forall \left(\mathscr{X}_g, \mathscr{Z}_g\right) \in \Omega \tag{19}$$

where $G = D_{\mathscr{Z}_g} H(\mathscr{X}_g^*, 0)$ is an M-matrix (the off-diagonal components of G are nonnegative) and Ω represents the region where the model makes biological sense. The following theorem holds if the system (18) meets the aforementioned two conditions.

Theorem 3. *The DFE $\mathscr{E}_g^* = (\mathscr{X}_g^*, 0)$ is a GAS equilibrium of system (18) provided that $R_{0g} < 1$ and the assumptions in Equation (19) are met.*

Proof 2. From Theorem 2, the DFE ($\mathscr{E}_g^*$) is locally asymptotically stable when $R_{0g} < 1$. Consider

$$\frac{d\mathscr{X}_g}{dt} = F\left(\mathscr{X}_g, \mathscr{Z}_g\right) = \begin{bmatrix} \Lambda - \left(\dfrac{\omega C_v}{\tau_2 + C_v} + \eta\right) S \\ \sigma_g E_g + \delta_g I_g + -\eta R \end{bmatrix} \Rightarrow F\left(\mathscr{X}_g, 0\right) = \begin{bmatrix} \Lambda - \eta S \\ 0 \end{bmatrix}$$

$$\frac{d\mathscr{Z}_g}{dt} = H\left(\mathscr{X}_g, \mathscr{Z}_g\right) = \begin{bmatrix} \dfrac{\omega C_v}{\tau_2 + C_v} S - \left(\sigma_g + \phi_g + \eta + \mu\right) E_g \\ \phi_g E_g - \left(\delta_g + \eta + \mu\right) I_g \\ \omega_1 E_g + \omega_2 I_g - \alpha_2 C_V \end{bmatrix} \tag{20}$$

From Equation (20), we have

$$G = D_{\mathscr{Z}_g} H\left(\mathscr{X}_g^*, 0\right) = \begin{bmatrix} 0 & -\left(\sigma_g + \phi_g + \eta + \mu\right) & 0 & 0 & \dfrac{\omega\Lambda}{\tau_2 \eta} \\ 0 & \phi_g & -\left(\delta_g + \eta + \mu\right) & 0 & 0 \\ 0 & \omega_1 & \omega_2 & 0 & -\alpha_2 \end{bmatrix} \tag{21}$$

and it follows that

$$G\mathscr{Z}_g = \begin{bmatrix} \dfrac{\omega C_V \Lambda}{\tau_2 \eta} - \left(\sigma_g + \phi_g + \eta + \mu\right) E_g \\ \phi_g E_g - \left(\delta_g + \eta + \mu\right) I_g \\ \omega_1 E_g + \omega_2 I_g - \alpha_2 C_V \end{bmatrix} \tag{22}$$

Thus

$$\tilde{H}\left(\mathscr{X}_g, \mathscr{Z}_g\right) = G\mathscr{Z}_g - H\left(\mathscr{X}_g, \mathscr{Z}_g\right) = \begin{bmatrix} \dfrac{\omega C_V \Lambda}{\tau_2 \eta} - \left(\sigma_g + \phi_g + \eta + \mu\right) E_g \\ \phi_g E_g - \left(\delta_g + \eta + \mu\right) I_g \\ \omega_1 E_g + \omega_2 I_g - \alpha_2 C_V \end{bmatrix} - \begin{bmatrix} \dfrac{\omega C_v}{\tau_2 + C_v} S - \left(\sigma_g + \phi_g + \eta + \mu\right) E_g \\ \phi_g E_g - \left(\delta_g + \eta + \mu\right) I_g \\ \omega_1 E_g + \omega_2 I_g - \alpha_2 C_V \end{bmatrix} = \begin{bmatrix} \dfrac{\omega C_V \Lambda}{\tau_2 \eta} - \dfrac{\omega C_v}{\tau_2 + C_v} S \\ 0 \\ 0 \end{bmatrix}$$

Thus, the first and the second conditions in Equation (19) are satisfied since

$$\frac{d\mathscr{X}_g}{dt} = F\left(\mathscr{X}_g, 0\right)$$

and $\tilde{H}(\mathscr{X}_g, \mathscr{Z}_g) \geq 0$, respectively. Therefore, $\mathscr{E}_g^*$ has a global asymptotic stability □

4.5. Existence of the Endemic Equilibrium Point ($\mathscr{E}_g^{**}$)

Theorem 4. *There exists a positive endemic equilibrium point ($\mathscr{E}_g^{**}$) for the system of Equation (1) provided that $R_{0g} > 1$.*

Proof 3. Letting $S = S^{**}$, $E_g = E_g^{**}$, $I_g = I_g^{**}$, $R = R^{**}$, and $C_V = C_V^{**}$ and setting the right-hand side of the equations of the system (1) equal to zero, we get

$$\begin{aligned} 0 &= \Lambda - \left(\frac{\omega C_v^{**}}{\tau_2 + C_v^{**}} + \eta\right) S^{**} \\ 0 &= \frac{\omega C_v^{**}}{\tau_2 + C_v^{**}} S^{**} - \left(\sigma_g + \phi_g + \eta + \mu\right) E_g^{**} \\ 0 &= \phi_g E_g^{**} - \left(\delta_g + \eta + \mu\right) I_g^{**} \\ 0 &= \sigma_g E_g^{**} + \delta_g I_g^{**} - \eta R^{**} \\ 0 &= \omega_1 E_g^{**} + \omega_2 I_g^{**} - \alpha_2 C_V^{**} \end{aligned} \tag{23}$$

Explicitly solving for values of $\{S^{**}, E_g^{**}, I_g^{**}, R^{**}, C_V^{**}\}$, we obtain

$$
\begin{aligned}
S^{**} &= \frac{R_{0g}\tau_2\eta\left(\sigma_g+\phi_g+\eta+\mu\right)\left(\delta_g+\eta+\mu\right)\alpha_2+\tau_2\omega\left(\sigma_g+\phi_g+\eta+\mu\right)\left(\delta_g+\eta+\mu\right)\alpha_2}{\left(\omega_1\left(\delta_g+\eta+\mu\right)+\omega_2\phi_g\right)\omega(\omega+\eta)}\\
E_g^{**} &= \frac{\eta\tau_2\alpha_2\left(\delta_g+\eta+\mu\right)\left(R_{0g}-1\right)}{\left(\omega_1\left(\delta_g+\eta+\mu\right)+\omega_2\phi_g\right)(\omega+\eta)}\\
I_g^{**} &= \frac{\eta\tau_2\alpha_2\phi_g\left(R_{0g}-1\right)}{\left(\omega_1\left(\delta_g+\eta+\mu\right)+\omega_2\phi_g\right)(\omega+\eta)}\\
R^{**} &= \frac{\tau_2\alpha_2\left(\sigma_g\left(\delta_g+\eta+\mu\right)+\delta_g\phi_g\right)\left(R_{0g}-1\right)}{\left(\omega_1\left(\delta_g+\eta+\mu\right)+\omega_2\phi_g\right)(\omega+\eta)}\\
C_V^{**} &= \frac{\left(\omega_1\left(\delta_g+\eta+\mu\right)+\omega_2\phi_g\right)\eta\tau_2\left(R_{0g}-1\right)}{\left(\omega_1\left(\delta_g+\eta+\mu\right)+\omega_2\phi_g\right)(\omega+\eta)}
\end{aligned}
\tag{24}
$$

Thus, a positive $\mathscr{E}_g^{**}=\{S^{**},E_g^{**},I_g^{**},R^{**},C_V^{**}\}$ exists if $R_{0g}>1$. □

4.6. Local Stability of Endemic Equilibrium. We use the center manifold theory as described in [12] to assess the stability of the endemic equilibrium, $\mathscr{E}_g^{**}$, because evaluating the eigenvalues of the Jacobian matrix of system (1) at the endemic equilibrium is complicated. Theorem 4.1 in [12] outlines the procedure of analyzing the local stability of endemic equilibrium in a nutshell. The coefficients, a and b of the normal form, are two key quantities in expressing the system's dynamics on the center manifold theory as described in Theorem 4.1 in [12]. According to part (iv) of Theorem 4.1 in [12], if $a<0$ and $b>0$, the endemic equilibrium $\mathscr{E}_g^{**}$ is locally asymptotically stable for $R_{0g}>1$ but close to 1.

Theorem 5. *The endemic equilibrium $\mathscr{E}_g^{**}$ is locally asymptotically stable if $R_{0g}>1$*

Proof 4. By using the center manifold theory, Theorem 4.1 in [12], we rename the variables in the system (1) as $S=x_1$, $E_g=x_2$, $I_g=x_3$, $R=x_4$, and $C_V=x_5$ such that $X=(x_1,x_2,x_3,x_4,x_5)^T$. Further, by using $X=(x_1,x_2,x_3,x_4,x_5)^T$, the system (1) can be written in the form $dX/dt=F(X)$, with $(f_1,f_2,f_3,f_4,f_5)^T$, as follows:

$$
\begin{aligned}
\frac{dx_1}{dt} &= f_1=\Lambda-\left(\frac{\omega x_5}{\tau_2+x_5}+\eta\right)x_1\\
\frac{dx_2}{dt} &= f_2=\frac{\omega x_5}{\tau_2+x_5}x_1-\left(\sigma_g+\phi_g+\eta+\mu\right)x_2\\
\frac{dx_3}{dt} &= f_3=\phi_g x_2-\left(\delta_g+\eta+\mu\right)x_3\\
\frac{dx_4}{dt} &= f_4=\sigma_g x_2+\delta_g x_3-\eta x_4\\
\frac{dx_5}{dt} &= f_5=\omega_1 x_2+\omega_2 x_3-\alpha_2 x_5
\end{aligned}
\tag{25}
$$

Suppose that $\omega=\omega^*$ is a bifurcation parameter when $R_{0g}=1$, solving for ω^* for $R_{0g}=1$ from

$$
R_{0g}=\frac{\omega\Lambda\left(\left(\delta_g+\eta+\kappa\right)\omega_1+\phi_g\omega_2\right)}{\tau_2\eta\left(\sigma_g+\phi_g+\eta+\mu\right)\left(\delta_g+\eta+\kappa\right)\alpha_2}=1
$$

we have

$$
\omega^*=\frac{\tau_2\eta\left(\sigma_g+\phi_g+\eta+\mu\right)\left(\delta_g+\eta+\mu\right)\alpha_2}{\Lambda\left(\left(\delta_g+\eta+\mu\right)\omega_1+\phi_g\omega_2\right)} \tag{26}
$$

The Jacobian matrix of the system (25) at $\mathscr{E}_g^*$ with $\omega=\omega^*$ is given as

$$
J^*\left(\mathscr{E}_g^*\right)=\begin{bmatrix}
-\eta & 0 & 0 & 0 & -\dfrac{\omega^*\Lambda}{\tau_2\eta}\\
0 & -\left(\sigma_g+\phi_g+\eta+\mu\right) & 0 & 0 & \dfrac{\omega^*\Lambda}{\tau_2\eta}\\
0 & \phi_g & -\left(\delta_g+\eta+\mu\right) & 0 & 0\\
0 & \sigma_g & \delta_g & -\eta & 0\\
0 & \omega_1 & \omega_2 & 0 & -\alpha_2
\end{bmatrix}
\tag{27}
$$

The Jacobian matrix (27) has zero eigenvalues close to $\omega=\omega^*$; thus, the center manifold theory is utilized to examine the dynamics of the system. Let $w=(w_1,w_2,w_3,w_4,w_5)^T$, a right eigenvector associated with the Jacobian matrix (Equation (27)), be close to $\omega=\omega^*$, then

$$
\begin{bmatrix}
-\eta & 0 & 0 & 0 & -\dfrac{\omega^*\Lambda}{\tau_2\eta}\\
0 & -\left(\sigma_g+\phi_g+\eta+\mu\right) & 0 & 0 & \dfrac{\omega^*\Lambda}{\tau_2\eta}\\
0 & \phi_g & -\left(\delta_g+\eta+\mu\right) & 0 & 0\\
0 & \sigma_g & \delta_g & -\eta & 0\\
0 & \omega_1 & \omega_2 & 0 & -\alpha_2
\end{bmatrix}
\cdot\begin{pmatrix} w_1\\ w_2\\ w_3\\ w_4\\ w_5\end{pmatrix}=\begin{pmatrix}0\\0\\0\\0\\0\end{pmatrix}\Rightarrow\begin{cases}
-\eta w_1-\dfrac{\omega^*\Lambda}{\tau_2\eta}w_5=0\\
-\left(\sigma_g+\phi_g+\eta+\mu\right)w_2+\dfrac{\omega^*\Lambda}{\tau_2\eta}w_5=0\\
\phi_g w_2-\left(\delta_g+\eta+\mu\right)w_3=0\\
\sigma_g w_2+\delta_g w_3-\eta w_4=0\\
\omega_1 w_2+\omega_2 w_3-\alpha_2 w_5=0
\end{cases}
\tag{28}
$$

Solving system (28), we get

$$\begin{cases} w_1 = -\dfrac{\omega^* \Lambda\left(\omega_1\left(\delta_g+\eta+\mu\right)+\omega_2\phi_g\right)}{\tau_2\eta^2\alpha_2\left(\delta_g+\eta+\mu\right)} w_2 < 0 \\ w_2 = w_2 > 0 \\ w_3 = \dfrac{\phi_g}{\left(\delta_g+\eta+\mu\right)} w_2 > 0 \\ w_4 = \dfrac{\sigma_g\left(\delta_g+\eta+\mu\right)+\delta_g\phi_g}{\eta\left(\delta_g+\eta+\mu\right)} > 0 \\ w_5 = \dfrac{\omega_1\left(\delta_g+\eta+\mu\right)+\omega_2\phi_g}{\alpha_2\left(\delta_g+\eta+\kappa\right)} w_2 > 0 \end{cases} \tag{29}$$

Also, let $v = (v_1, v_2, v_3, v_4, v_5)^T$, a left eigenvector associated with the Jacobian matrix (Equation (27)), be close to $\omega = \omega^*$, such that

$$\begin{bmatrix} -\eta & 0 & 0 & 0 & 0 \\ 0 & -\left(\sigma_g+\phi_g+\eta+\mu\right) & \phi_g & \sigma_g & \omega_1 \\ 0 & 0 & -\left(\delta_g+\eta+\mu\right) & \delta_g & \omega_2 \\ 0 & 0 & 0 & -\eta & 0 \\ -\dfrac{\omega\Lambda}{\tau_2\eta} & \dfrac{\omega\Lambda}{\tau_2\eta} & 0 & 0 & -\alpha_2 \end{bmatrix} \cdot \begin{pmatrix} v_1 \\ v_2 \\ v_3 \\ v_4 \\ v_5 \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}$$

$$\Rightarrow \begin{cases} -\eta v_1 = 0 \\ -\left(\sigma_g+\phi_g+\eta+\mu\right)v_2 + \phi_g v_3 + \sigma_g v_4 + \omega_1 v_5 = 0 \\ -\left(\delta_g+\eta+\mu\right)v_3 + \delta_g v_4 + \omega_2 v_5 = 0 \\ -\eta v_4 = 0 \\ -\dfrac{\omega^*\Lambda}{\tau_2\eta} v_1 + \dfrac{\omega^*\Lambda}{\tau_2\eta} v_2 - \alpha_2 v_5 = 0 \end{cases} \tag{30}$$

Solving system (30), we obtain

$$\begin{cases} v_1 = 0 \\ v_2 = v_2 > 0 \\ v_3 = \dfrac{\omega_2\omega^*\Lambda}{\alpha_2\tau_2\eta\left(\delta_g+\eta+\mu\right)} v_2 > 0 \\ v_4 = 0 \\ v_5 = \dfrac{\omega^*\Lambda}{\alpha_2\tau_2\eta} v_2 > 0 \end{cases} \tag{31}$$

Table 1: Parameter description.

Parameter	Value	Source
Λ	10	[7]
ω	0.000143 (0.000143–0.0143)/day	[7]
τ_2	0.009/day	Assumed
η	0.0001543/day	[7]
μ	0.032143/day	[7]
ω_1	0.008 (0.008–0.08)/day	Assumed
ω_2	0.009 (0.009–0.09)/day	Assumed
α_2	0.0900982/day	Assumed
θ_g	0.0039 (0.0039–0.39)/day	Assumed
σ_g	0.0165/day	[8]
δ_g	0.021429/day	[7]
ϕ_g	0.033/day	[8]

Using the formula described in [12], we compute a and b:

$$a = \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}\left(\mathscr{E}_g^*\right)$$

$$b = \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \rho}\left(\mathscr{E}_g^*\right)$$

To get the bifurcation coefficient a, we first obtain the nonzero partial derivatives of the model system (25) evaluated at $(\mathscr{E}_g^*, \omega^*)$. Thus, it is evident that

$$\begin{aligned} \frac{\partial^2 f_1}{\partial x_1 \partial x_5} &= -\frac{\omega^*}{\tau_2} \\ \frac{\partial^2 f_2}{\partial x_1 \partial x_5} &= \frac{\omega^*}{\tau_2} \\ \frac{\partial^2 f_1}{\partial x_5^2} &= \frac{2\omega^*\Lambda}{\tau_2^2\eta} \\ \frac{\partial^2 f_2}{\partial x_5^2} &= -\frac{2\omega^*\Lambda}{\tau_2^2\eta} \end{aligned} \tag{32}$$

so that

$$a = -v_1 w_1 w_5 \frac{\omega^*}{\tau_2} + v_1 w_5^2 \frac{2\omega^*\Lambda}{\tau_2^2\eta} + v_2 w_1 w_5 \frac{\omega^*}{\tau_2} - v_2 w_5^2 \frac{2\omega^*\Lambda}{\tau_2^2\eta} \tag{33}$$

Using Equations (29) and (31), Equation (33) can be written as

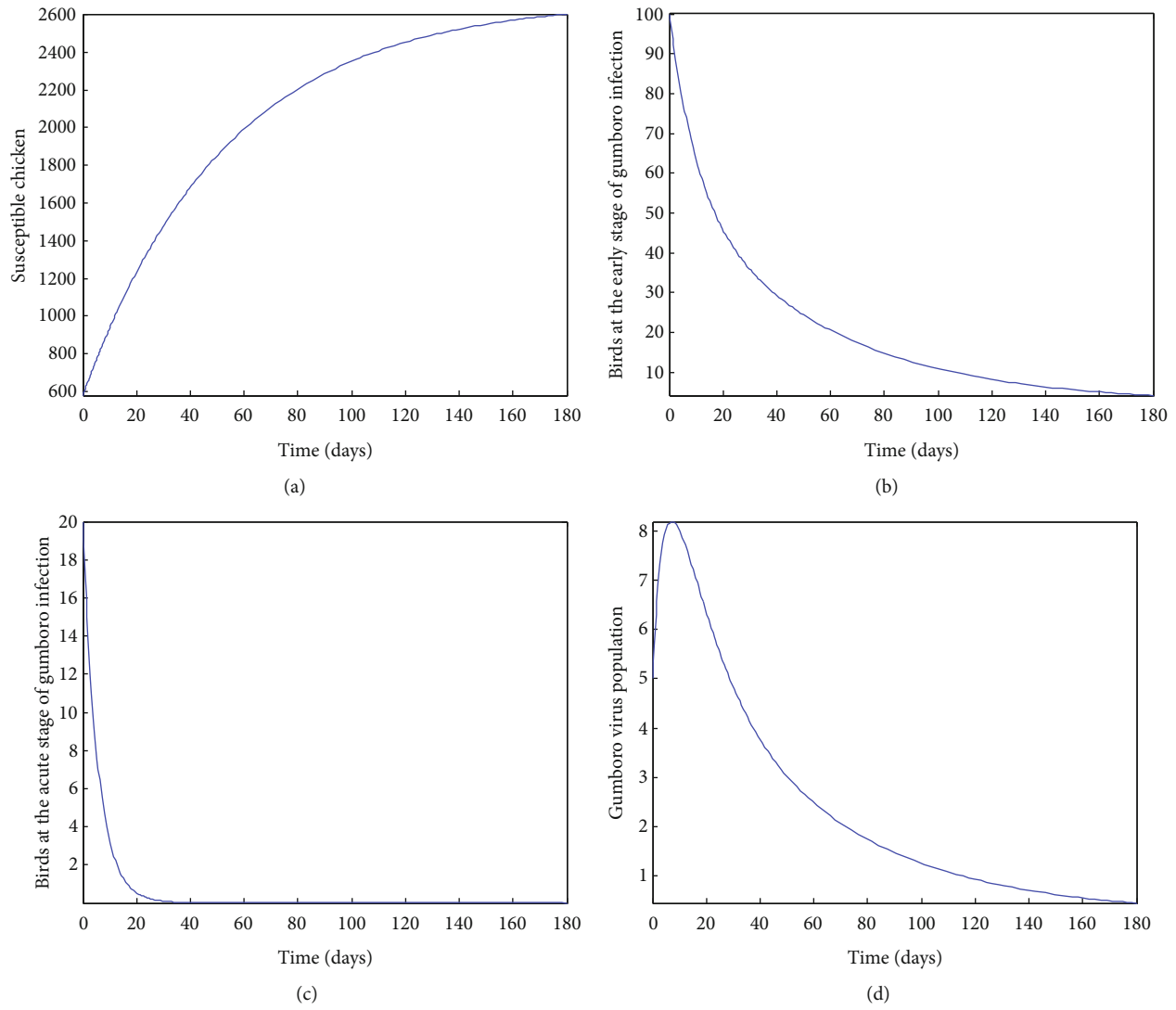

FIGURE 2: Graphs showing the dynamics of the model when $R_{0g} = 0.0737$ (a) for susceptible chicken, (b) birds at the early stage of Gumboro infection, (c) birds at the acute stage of Gumboro infection, and (d) Gumboro virus population with parameter values $\Lambda = 10$, $\omega = 0.000143$, $\tau_2 = 0.009$, $\eta = 0.0001543$, $\mu = 0.032143$, $\omega_1 = 0.008$, $\omega_2 = 0.009$, $\alpha_2 = 0.0900982$, $\theta_g = 0.0039$, $\sigma_g = 0.0165$, $\delta_g = 0.021429$, and $\phi_g = 0.033$.

$$a = -\nu_2 w_2^2 \left[\frac{\omega^{*2} \Lambda \left(\omega_1 \left(\delta_g + \eta + \mu \right) + \omega_2 \phi_g \right)^2}{\tau_2^2 \eta \alpha_2^2 \left(\delta_g + \eta + \mu \right)^2} + \frac{2\omega^* \Lambda}{\tau_2^2 \eta} \left(\frac{\omega_1 \left(\delta_g + \eta + \mu \right) + \omega_2 \phi_g}{\alpha_2 \left(\delta_g + \eta + \mu \right)} \right)^2 \right] < 0$$

For the coefficient b, we have the following nonzero partial derivatives of the model system (25) evaluated at $(\mathscr{E}_g^*, \omega^*)$:

$$\frac{\partial^2 f_1}{\partial x_5 \partial \omega^*} = -\frac{\Lambda}{\tau_2 \eta}$$
$$\frac{\partial^2 f_2}{\partial x_5 \partial \omega^*} = \frac{\Lambda}{\tau_2 \eta} \quad (34)$$

Hence,

$$b = -\nu_1 w_5 \frac{\Lambda}{\tau_2 \eta} + \nu_2 w_5 \frac{\Lambda}{\tau_2 \eta} \quad (35)$$

Using Equations (29) and (31), Equation (35) can be written as

$$b = \nu_2 w_2 \frac{\Lambda \left(\omega_1 \left(\delta_g + \eta + \mu \right) + \omega_2 \phi_g \right)}{\alpha_2 \left(\delta_g + \eta + \mu \right) \tau_2 \eta} > 0$$

Since $a < 0$ and $b > 0$, it follows that the endemic equilibrium $\mathscr{E}_g^{**}$ is locally asymptotically stable if $R_{0g} > 1$. □

4.7. Global Stability of the Endemic Equilibrium Point

Theorem 6. *The endemic equilibrium point E_g^{**} of the system (1) is GAS if $R_{0g} > 1$.*

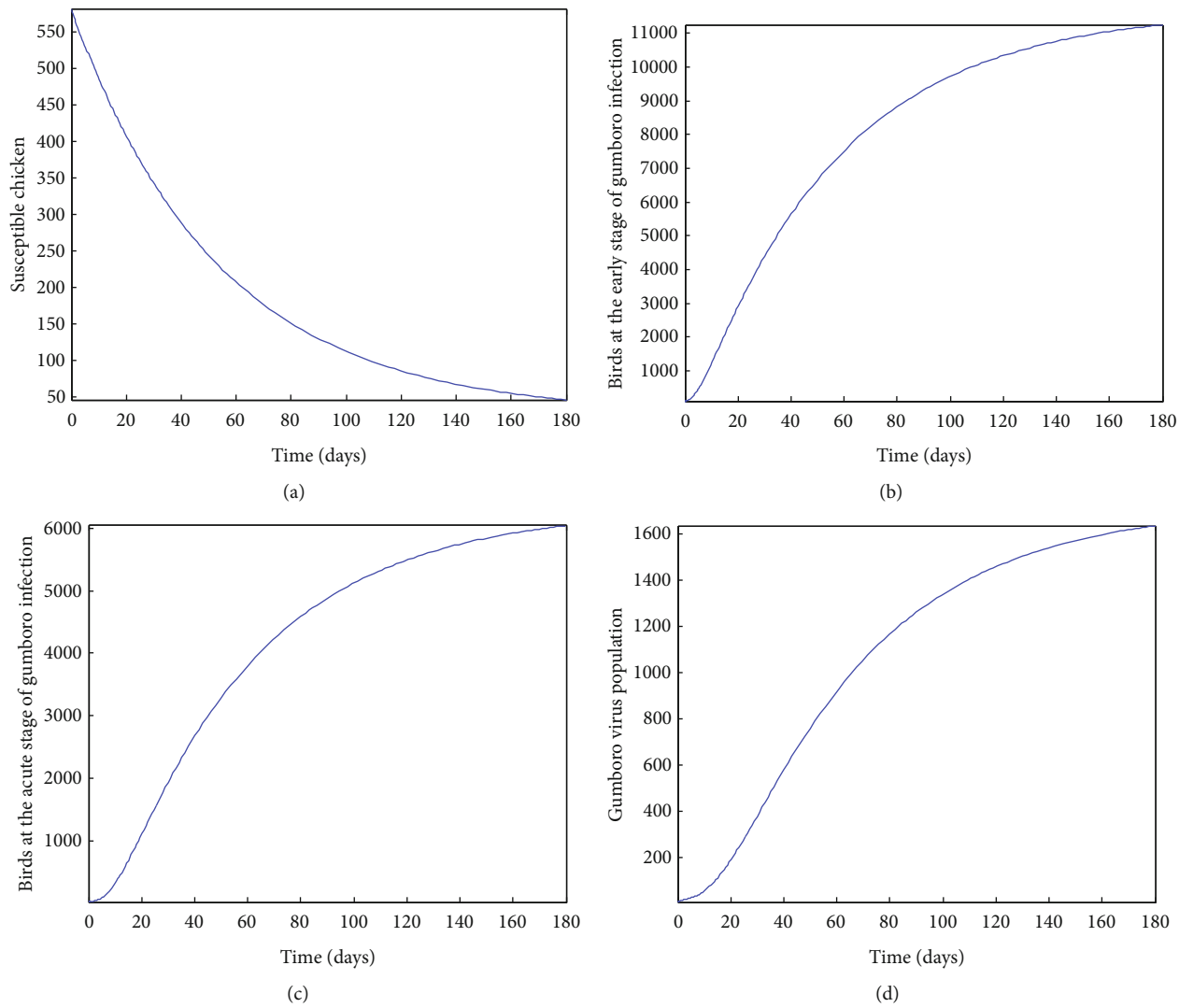

Figure 3: Graphs showing the dynamics of the model (a) for susceptible chicken, (b) birds at the early stage of Gumboro infection, (c) birds at the acute stage of Gumboro infection, and (d) Gumboro virus population when $R_{0g} = 1.2528$ with parameter values $\Lambda = 10$, $\omega = 0.0143$, $\tau_2 = 0.009$, $\eta = 0.0001543$, $\mu = 0.032143$, $\omega_1 = 0.08$, $\omega_2 = 0.09$, $\alpha_2 = 0.0900982$, $\theta_g = 0.39$, $\sigma_g = 0.0165$, $\delta_g = 0.021429$, and $\phi_g = 0.033$.

Proof 5. We have shown in Section (4.5) that the endemic equilibrium point E_g^{**} exists when $R_{0g} > 1$. Using the Poincaré–Bendixson theorem, the global stability of the endemic equilibrium point E_g^{**} is investigated [13]. It follows from Dulac's multiplier, $1/SE_gI_gRC_V$, that

$$\frac{\partial}{\partial S}\left(\frac{\Lambda - ((\omega C_v/(\tau_2 + C_v)) + \eta)S}{SE_gI_gRC_V}\right) = -\frac{\Lambda}{S^2E_gI_gRC_V} < 0$$

$$\frac{\partial}{\partial E_g}\left(\frac{(\omega C_v/\tau_2 + C_v)S - \left(\sigma_g + \phi_g + \eta + \mu\right)E_g}{SE_gI_gRC_V}\right) = -\frac{(\omega C_v/(\tau_2 + C_v))S}{SE_g^2I_gRC_V} < 0$$

$$\frac{\partial}{\partial I_g}\left(\frac{\phi_gE_g - (\delta_g + \eta + \mu)I_g}{SE_gI_gRC_V}\right) = -\frac{\phi_gE_g}{SE_gI_g^2RC_V} < 0$$

$$\frac{\partial}{\partial R}\left(\frac{\sigma_gE_g + \delta_gI_g - \eta R}{SE_gI_gRC_V}\right) = -\frac{\sigma_gE_g + \delta_gI_g}{SE_gI_gR^2C_V} < 0$$

$$\frac{\partial}{\partial C_V}\left(\frac{\omega_1E_g + \omega_2I_g - \alpha_2C_V}{SE_gI_gRC_V}\right) = -\frac{\omega_1E_g + \omega_2I_g}{SE_gI_gRC_V^2} < 0$$

$$\begin{aligned}
&\frac{\partial}{\partial S}\left(\frac{\Lambda - ((\omega C_v/(\tau_2 + C_v)) + \eta)S}{SE_gI_gRC_V}\right) \\
&\quad + \frac{\partial}{\partial E_g}\left(\frac{(\omega C_v/(\tau_2 + C_v))S - \left(\sigma_g + \phi_g + \eta + \mu\right)E_g}{SE_gI_gRC_V}\right) \\
&\quad + \frac{\partial}{\partial I_g}\left(\frac{\phi_gE_g - (\delta_g + \eta + \mu)I_g}{SE_gI_gRC_V}\right) + \frac{\partial}{\partial R}\left(\frac{\sigma_gE_g + \delta_gI_g - \eta R}{SE_gI_gRC_V}\right) \\
&\quad + \frac{\partial}{\partial C_V}\left(\frac{\omega_1E_g + \omega_2I_g - \alpha_2C_V}{SE_gI_gRC_V}\right) \\
&= -\frac{\Lambda}{S^2E_gI_gRC_V} - \frac{(\omega C_v/(\tau_2 + C_v))S}{SE_g^2I_gRC_V} - \frac{\phi_gE_g}{SE_gI_g^2RC_V} \\
&\quad - \frac{\sigma_gE_g + \delta_gI_g}{SE_gI_gR^2C_V} - \frac{\omega_1E_g + \omega_2I_g}{SE_gI_gRC_V^2} < 0
\end{aligned} \tag{36}$$

Due to the fact that Ω is positively invariant and the endemic equilibrium exists whenever $R_{0g} > 1$, there are no

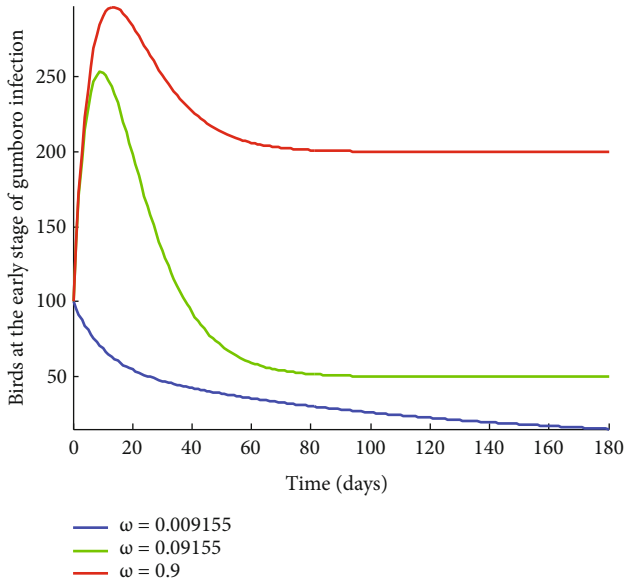

FIGURE 4: A graph showing the dynamics of birds at the early stage of Gumboro infection with different values of ω.

periodic orbits in Ω according to Dulac's criterion. From the Poincaré–Bendixson theorem, it follows that all solutions of the limiting system originating in Ω remain in Ω for all time t. The absence of periodic orbits suggests that whenever $R_{0g} > 1$, the special endemic equilibrium of the Gumboro model is GAS. □

5. Numerical Simulation

Numerical simulations to prove the theoretical results for the Gumboro disease mathematical model were carried out using MATLAB ode 45 solver. This was made possible by the use of some parameters in the literature and others that were estimated or assumed as shown in Table 1.

From Figure 2, it was shown that the number of birds at early stages of infection, acute stages of infection, and the pathogen population converges to zero while the susceptible birds' population tends to a constant λ/η when $R_{0g} < 1$ which implies that whenever Gumboro disease dies out, only the susceptible chicken would remain. Figure 3 also shows that birds at early and acute stages of infection together with the pathogen population tend towards the endemic equilibrium point when $R_{0g} > 1$ while the susceptible birds converge to zero indicating that Gumboro disease remains endemic. From Figure 4, it was shown that birds at early stages of infection increase with the increase in the contact rate of susceptible to Gumboro disease-contaminated environment.

6. Conclusion

A mathematical model of Gumboro disease transmission dynamics was formulated in this paper. The disease-free and endemic equilibrium points were determined, and the reproduction number was derived. The findings showed that Gumboro disease dies out whenever $R_{0g} < 1$ and persists in the chicken population whenever $R_{0g} > 1$. Also, it was realized that minimizing the contact rate of chicken to a contaminated environment lowers cases of infections in a population. These numerical simulation findings were found to be in harmony with the theoretical stability analysis results.

Data Availability Statement

All data is provided in this paper.

Conflicts of Interest

The authors declare no conflicts of interest.

Author Contributions

All the authors collaborated in carrying out this study. Author I.C. designed the study, author J.S.M. wrote the first draft of the manuscript and performed the mathematical analysis of the study and the literature review, and author W.N.M. performed the numerical simulation. All authors read and approved the final manuscript.

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