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## Mathematical Analysis of a Vaccination–Culling Model for the Control of Fowl Pox in Birds

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### Abstract

Fowl pox is a highly contagious viral disease that poses significant economic challenges to poultry production through reduced productivity, morbidity, and mortality. This study extends an existing fowl pox transmission model by incorporating vaccination of susceptible and newly recruited birds, culling of symptomatic infectious birds, and separate symptomatic and asymptomatic infectious classes to better capture disease progression and control dynamics. A deterministic compartmental model consisting of susceptible, exposed, symptomatic infectious, asymptomatic infectious, and recovered bird populations is formulated and analyzed. Fundamental properties of the model, including positivity and boundedness of solutions, are established. The disease-free equilibrium and the basic reproduction number are derived using the next-generation matrix approach. Using biologically relevant parameter values, the basic reproduction number was evaluated as ( $R_0 = 0.1420$ ), indicating that each infectious bird generates less than one secondary infection on average. Theoretical analysis shows that the disease-free equilibrium is both locally and globally asymptotically stable whenever  $R_0 < 1$ . Sensitivity analysis reveals that the culling rate  $\tau$  and vaccination rate  $\nu$  exert the strongest negative influence on disease transmission, with sensitivity indices of  $(-1.7196)$  and  $(-0.9434)$ , respectively, while the symptomatic transmission coefficient  $\alpha_1$  and recruitment rate  $\beta$  contribute most positively to disease spread. Numerical simulations demonstrate that increasing vaccination coverage and culling rates substantially reduce the exposed, symptomatic, and asymptomatic infectious bird populations while increasing the recovered population. The results indicate that the combined implementation of vaccination and culling is an effective strategy for suppressing fowl pox transmission and preventing disease persistence in poultry populations. These findings provide quantitative support for the adoption of integrated vaccination and culling programmes in poultry farms and contribute to the mathematical understanding of fowl pox control.

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## 1.0 Introduction

Fowl pox is a contagious viral disease of domestic and wild birds caused by members of the genus *Avipoxvirus* in the family *Poxviridae* (Tripathy et al., 2000; Afonso, et al., 2000; Thomas et al., 2024; Udofia et al., 2024). The disease is widely distributed throughout the world and remains one of the most economically significant viral infections affecting poultry production. Chickens, turkeys, pigeons, ducks and several other avian species are susceptible to infection, making the disease a major threat to both commercial and backyard poultry systems (Udofia & Sampson, 2014). Outbreaks of fowl pox are associated with reduced productivity, poor growth performance, decreased egg production, increased management costs, and mortality, thereby causing substantial economic losses to poultry farmers (Udofia et al., (2016). Transmission of the fowl pox virus occurs through direct contact with infected birds, contaminated environments, skin abrasions, and mechanical vectors such as mosquitoes (Buller & Palumbo, 1991; Francis, 1956). Following infection, the virus replicates in epithelial tissues before disseminating through the bloodstream to other parts of the body (Singh et al., 2005). Clinically, the disease manifests in two principal forms: the cutaneous (dry) form and the diphtheritic (wet) form. The cutaneous form is characterized by proliferative lesions on unfeathered parts of the body, while the diphtheritic form affects the mucous membranes of the respiratory and digestive tracts, often resulting in breathing difficulties, feeding impairment, and increased mortality (Weli & Tryland, 2011; Udofia & Sampson, 2014; Yeo et al., 2019). Although mortality in the dry form is usually low, the wet form can result in mortality rates ranging from 5% to 50%, particularly in severe outbreaks (Yeo et al., 2019).

Control of fowl pox relies primarily on vaccination, biosecurity measures, vector control, and proper farm management practices (Udofia & Inyama 2011; Alehegn et al., 2014;

James et al., 2021). Vaccination has been recognized as one of the most effective preventive measures, particularly in regions where mosquito populations are abundant and disease prevalence is high (Udofia & Inyama, 2011; Weli & Tryland, 2011; Ariyoshi et al., 2003). Other control strategies include environmental sanitation, elimination of mosquito breeding sites, insecticide application, culling of infected birds, and surveillance programs aimed at reducing transmission within poultry farms (Gulbudak & Martcheva, 2014; Chiganga et al., 2025). Despite the availability of these interventions, outbreaks continue to occur in many poultry-producing regions, highlighting the need for a better understanding of the disease transmission dynamics and the effectiveness of available control measures.

Mathematical modelling has become an indispensable tool for studying the transmission dynamics of infectious diseases and evaluating intervention strategies (Onuoha et al., 2014, 2015; Ekere, 2023; Gulbudak & Martcheva, 2014). Mathematical models provide quantitative and qualitative insights into disease progression, identify critical epidemiological thresholds, and assist policymakers in designing effective control programs (Thomas et al., 2025; Ekere et al., 2024; Agwu et al., 2018). Several mathematical models have been developed to investigate avian diseases and related epidemiological processes. In particular, Udofia and Sampson (2014) formulated a mathematical model for fowl pox transmission incorporating a discrete time delay to account for the incubation period of the disease. Their study provided important insights into the role of delayed infection processes in the spread of fowl pox and established a foundation for subsequent modelling efforts. However, the model of Udofia, and Sampson (2014) possesses several limitations from both epidemiological and control perspectives. First, the model did not distinguish between symptomatic and asymptomatic infectious birds, despite evidence

that asymptomatic infected birds may contribute significantly to disease transmission. Second, vaccination was not explicitly incorporated as a dynamic control mechanism. Third, the model did not account for the culling of infected birds, a common practice employed during disease outbreaks to reduce transmission. Finally, a comprehensive investigation of the effectiveness of these intervention measures through sensitivity analysis and stability analysis was not undertaken.

Motivated by these limitations, the present study extends the work of Udofia and Sampson (2014) by developing a more comprehensive compartmental model for the transmission dynamics and control of fowl pox in bird populations. The proposed model subdivides infected birds into exposed, symptomatic infectious, and asymptomatic infectious classes, thereby capturing the heterogeneous nature of disease progression. Furthermore, the model incorporates vaccination of susceptible birds and vaccinated recruitment as preventive strategies, while introducing culling of symptomatic infectious birds as a control mechanism aimed at reducing disease transmission. These additions enhance the biological realism of the model and provide a framework for assessing the effectiveness of practical intervention measures used in poultry farms. Vaccination and culling are considered together because they act on different stages of disease transmission and complement one another in controlling fowl pox outbreaks.

### 1.1 Epidemiological Justification

Vaccination is a preventive measure. It reduces the number of susceptible birds available for infection by increasing immunity within the flock. However, vaccination alone may not immediately stop an ongoing outbreak because some birds may already be infected before vaccination takes effect.

Culling, on the other hand, is a reactive measure. It removes infected birds from the population, thereby reducing the number of infectious

individuals capable of transmitting the virus to healthy birds. Consequently:

- vaccination reduces the future pool of susceptible birds.
- culling reduces the current pool of infectious birds.
- together, vaccination and culling reduce transmission more effectively than either strategy alone.

### 1.2 Knowledge Gap and Novel Contributions of the Present Study

Although several studies have investigated the epidemiology and control of fowl pox infection, important gaps remain in the existing literature. In particular, the pioneering model of Udofia and Sampson (2014) focused on the transmission dynamics of fowl pox with discrete delay effects but did not explicitly incorporate key intervention strategies commonly employed in poultry farms, such as vaccination and culling of infected birds. Furthermore, the model did not distinguish between symptomatic and asymptomatic infectious birds, despite evidence that asymptomatic infections can significantly contribute to disease transmission within poultry populations.

Most existing mathematical models of fowl pox have concentrated on understanding disease spread rather than evaluating the combined effects of practical control measures. Consequently, there remains limited theoretical understanding of how vaccination of susceptible birds, vaccination of newly recruited birds, and culling of infectious birds interact to influence disease dynamics and disease eradication. In addition, comprehensive analyses involving disease-free equilibrium stability, parameter sensitivity, and the relative effectiveness of these interventions are scarce in the fowl pox modelling literature.

To address these gaps, the present study extends the model of Udofia and Sampson (2014) by introducing a more detailed compartmental structure consisting of susceptible, exposed,

symptomatic infectious, asymptomatic infectious, and recovered bird populations. The model explicitly incorporates vaccination of susceptible birds, vaccination of recruited birds, and culling of symptomatic infectious birds as control mechanisms. Unlike previous studies, the proposed model derives the basic reproduction number, establishes both local and global stability conditions for the disease-free equilibrium, and identifies the most influential epidemiological parameters through sensitivity analysis.

Therefore, the novelty of this work lies not only in extending the existing fowl pox transmission framework but also in providing a comprehensive mathematical and epidemiological assessment of practical disease-control interventions applicable to poultry production systems.

## 2.0 Model Formulation

The model is made up of five compartments comprising the susceptible birds ( $S$ ), Exposed birds ( $E$ ), Symptomatic birds (i.e. those birds that are showing symptoms of the disease), ( $I_S$ ), Asymptomatic birds (i.e. those birds that are infected but not showing symptoms of the disease), ( $I_A$ ), and Recovered birds ( $R$ ). The model incorporates two control measures; vaccination and culling of sick birds. The model assumes that some proportions,  $\rho$  of recruited birds are vaccinated. The birds are recruited into the susceptible population at the rate  $\beta$ . It is assumed that the susceptible birds are vaccinated at the rate  $v$ , while the parameters  $\lambda$  and  $d$  are the force of infection and natural death rate, respectively. It is also assumed that a proportion,  $1 - \omega$  of infectious birds are symptomatic while  $\omega$  represents the proportion of infectious birds which are asymptomatic. Both the symptomatic and asymptomatic birds recover at the rates,  $r_1$  and  $r_2$ , respectively. Figure 1 describes the disease transmission dynamics while Table 1 describes the state variables and parameters used

in the model. The disease transmission dynamics is given by:

$$\begin{aligned}\frac{dS}{dt} &= (1 - \rho)\beta - (\lambda + d + v)S \\ \frac{dE}{dt} &= \lambda S - (\delta + d)E \\ \frac{dI_S}{dt} &= (1 - \omega)\delta E - (r_1 + d + d_1)I_S \\ \frac{dI_A}{dt} &= \omega\delta E - (r_2 + d + d_2)I_A \\ \frac{dR}{dt} &= r_1 I_S + r_2 I_A + \rho\beta + vS - dR\end{aligned}\quad (1)$$

where  $\lambda = \frac{(1-\tau)\alpha_1 I_S}{N} + \frac{\alpha_2 I_A}{N}$  with the initial condition to system (1) given as  $S(0) = S^0, E(0) = E^0, I_S(0) = I_S^0, I_A(0) = I_A^0, R(0) = R^0$  at  $t = 0$ .

## 2.2 Positivity of solutions and invariant region

**Lemma 2.2.1:** Given the initial conditions of the system (1)  $(S^0, E^0, I_S^0, I_A^0, R^0) > 0$  at  $t = 0$  lies in the interval  $\Omega$ , then the solution set  $(S(t), E(t), I_S(t), I_A(t), R(t))$  to the system remains positive, for all  $t > 0$ .

### Proof

Consider the susceptible population equation

$$\frac{dS}{dt} = (1 - \rho)\beta - (\lambda + d + v)S, \text{ where,}$$

$$\lambda = \frac{(1-\tau)\alpha_1 I_S}{N} + \frac{\alpha_2 I_A}{N}.$$

Since  $(1 - \rho)\beta > 0$ , it follows that:

$$\frac{dS}{dt} \geq -(\lambda + d + v)S \Rightarrow \frac{dS}{S} \geq -(\lambda + d + v)dt.$$

Integrating from 0 to  $t$ , gives

$$\ln S(t) - \ln S^0 \geq - \int (\lambda(u) + d + v) du.$$

Hence,

$$S(t) \geq S^0 \exp\left(- \int \left[\frac{(1-\tau)\alpha_1 I_S(u) + \alpha_2 I_A(u)}{N(u)} + d + v\right] du\right) > 0, \text{ for all } t > 0$$

Similarly,  $E(t) \geq E^0 e^{-(\delta+d)t}$ , for all  $t > 0$ ,  $I_S(t) \geq I_S^0 e^{-(r_1+d+d_1)t}$ , for all  $t > 0$ ,  $I_A(t) \geq I_A^0 e^{-(r_2+d+d_2)t}$ , for all  $t > 0$ ,  $R(t) \geq R^0 e^{-dt}$ , for all  $t > 0$ . Therefore,

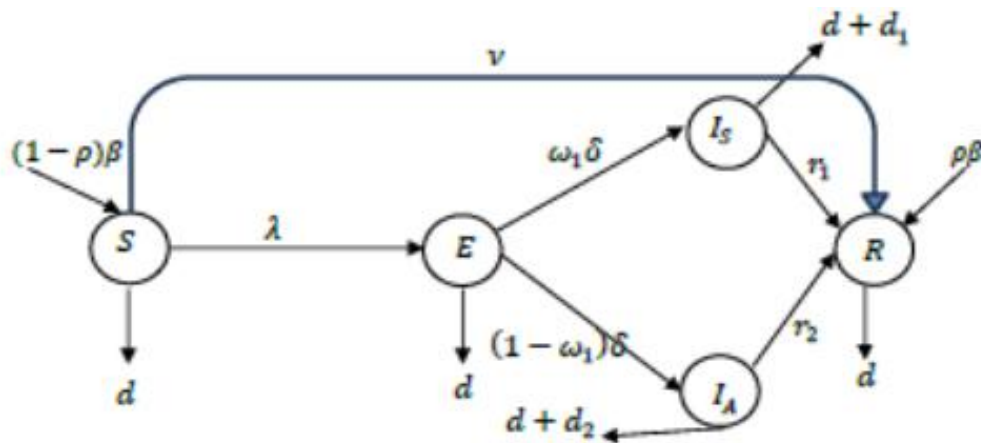
$$S(t) > 0, \quad E(t) > 0, \quad I_S(t) > 0, \quad I_A(t) > 0, \quad R(t) > 0$$

For all  $t > 0$ . Thus, all solutions of system (1) that start in the positive orthant remain positive for all time.  $\therefore \mathbb{R}_+^5 = \{(S, E, I_S, I_A, R) \in$

$\mathbb{R}^5: S, E, I_S, I_A, R > 0 \}$ , is positively invariant under system (1).

**Table 1:** Variables and Parameters with their description

Nomenclature	Description
$S$	Susceptible birds
$E$	Exposed birds
$I_S$	Symptomatic birds
$I_A$	Asymptomatic birds
$R$	Recovered birds
$\lambda$	Force of infection
$d$	Natural death rate
$d_1$	Mortality rate due to disease
$v$	Rate at which susceptible birds are vaccinated
$\rho$	Proportion of recruited birds that are vaccinated
$\beta$	Rate of recruitment
$\omega$	Proportion of exposed birds, $E$ that are asymptomatic
$\delta$	Rate of development of infectiousness
$r_1$	Rate of recovery of symptomatic birds, $I_S$
$r_2$	Rate of recovery of asymptomatic birds, $I_A$
$\tau$	Culling rate of sick birds
$\alpha_1$	Probability of transmission from $I_S$ to $S$
$\alpha_2$	Probability of transmission from $I_A$ to $S$
$N$	Total population of birds



**Figure 1:** Flow diagram for the disease

**Lemma 2.2.2.** Every solution to the system (1) lies within the region  $\Omega$ . That is, the region  $\Omega$  is bounded.

**Proof.** In this section, the region,  $\Omega$  is shown to be positively invariant by showing that all the solutions to the model system will enter and remain in the region  $\Omega$ . The total bird population is given by:

$$N = S + E + I_S + I_A + R,$$

and satisfies the differential equation:

$$\begin{aligned} \frac{dN}{dt} &= \frac{dS}{dt} + \frac{dI_S}{dt} + \frac{dI_A}{dt} + \frac{dR}{dt} \\ &= (1 - \rho)\beta - (\lambda + d + v)S + \lambda S - (\delta + d)E + (1 - \omega)\delta E - (r_1 + d + d_1)I_S + \omega\delta E \\ &\quad - (r_2 + d + d_2)I_A + r_1I_S + r_2I_A + \rho\beta + vS - dR \\ &= (1 - \rho)\beta - \left(\frac{(1-\tau)\alpha_1 I_S}{N} + \frac{\alpha_2 I_A}{N} + d + v\right)S + \left(\frac{(1-\tau)\alpha_1 I_S}{N} + \frac{\alpha_2 I_A}{N}\right)S - (\delta + d)E + (1 - \omega)\delta E - \\ &\quad (r_1 + d + d_1)I_S + \omega\delta E - (r_2 + d + d_2)I_A + r_1I_S + r_2I_A + \rho\beta + vS - dR \\ &= \beta - d_1(I_S + I_A) - d(S + E + I_S + I_A + R) = \beta - dN - d_1(I_S + I_A). \end{aligned}$$

$$\text{But, } \frac{dN}{dt} \leq \beta - dN. \quad \Rightarrow \quad \frac{dN}{dt} + dN \leq \beta. \text{ By the use of integrating factor, } IF = e^{\int dt} = e^{dt},$$

$$N(t)e^{dt} \leq \beta \int e^{dt} dt + c, \quad \Rightarrow \quad N(t)e^{dt} \leq \beta \frac{e^{dt}}{d} + c, \quad \therefore N(t) \leq \frac{\beta}{d} + ce^{-dt}.$$

As  $t \rightarrow \infty$ , we have  $N(t) \leq \frac{\beta}{d}$ , showing that the total population is bounded and the set,  $\Omega$  is positively invariant and is an attractor of all positive solutions to the system. The proofs of Lemmas 2.2.1 and 2.2.2 show that the system is well - posed mathematically and epidemiologically. This is a sufficient condition for the system to be studied and analysed (Castilo-Chavez & Song, 2004; Udofia & Sampson, 2014).

### 2.3 Disease-free equilibrium point (DFE) of the system

The disease-free equilibrium point (DFE) is the steady state solution of (2.1) where there is no disease in the system. It is obtained by setting the infectious class  $X = \{E, I_S, I_A\}$  to zero. To obtain the DFE point, the system (1) is equated to zero and the values of the state variables are obtained (Ahman et al., 2025; Udofia & Amos, 2018; Zarin et al., 2025). Denoting the disease-free equilibrium point as  $E^0$ , then, we will have:

$$E^0 = \left( \left( \frac{(1-\rho)\beta}{d+v} \right), 0, 0, 0, \frac{\beta}{d} \left( \frac{\rho d + v}{d+v} \right) \right) \quad (2)$$

### 2.4 Basic Reproduction number

The basic reproduction number,  $R_0$  is the average number of new cases of the disease that can be caused by one new infectious case introduced into an entire susceptible population (Zuhur et al., 2025; Pathak & Kota 2025). This important threshold number is calculated using the Next-generation matrix approach  $FV^{-1}$ , where  $F$  and  $V$  are the Jacobian matrices

obtained from the disease classes in the system (Duru et al., 2025a, b; Udofia et al., 2024). First, the system is separated into two column matrices denoted by  $\mathcal{F}$  and  $\mathcal{V}$ , where  $\mathcal{F}$  is the rate of appearance of fowl pox in the system and  $\mathcal{V}$  represents inflow and outflow from the system by other means. Thus,

$$\mathcal{F} = \begin{bmatrix} \left( \frac{(1-\tau)\alpha_1 I_S}{N} + \frac{\alpha_2 I_A}{N} \right) S \\ 0 \\ 0 \end{bmatrix} \quad \text{and}$$

$$\mathcal{V} = \begin{bmatrix} (\delta + d)E \\ -(1 - \omega)\delta E + (r_1 + d + d_1)I_S \\ -\omega\delta E + (r_2 + d + d_2)I_A \end{bmatrix}$$

The matrices  $F = \frac{\partial \mathcal{F}}{\partial X}$  and  $V = \frac{\partial \mathcal{V}}{\partial X}$  and they are given by:

$$F = \begin{bmatrix} \frac{\partial \mathcal{F}_1}{\partial E} & \frac{\partial \mathcal{F}_1}{\partial I_S} & \frac{\partial \mathcal{F}_1}{\partial I_A} \\ \frac{\partial \mathcal{F}_2}{\partial E} & \frac{\partial \mathcal{F}_2}{\partial I_S} & \frac{\partial \mathcal{F}_2}{\partial I_A} \\ \frac{\partial \mathcal{F}_3}{\partial E} & \frac{\partial \mathcal{F}_3}{\partial I_S} & \frac{\partial \mathcal{F}_3}{\partial I_A} \end{bmatrix} = \begin{bmatrix} 0 & A_3 & A_4 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \quad \text{and}$$

$$V = \begin{bmatrix} \frac{\partial \mathcal{V}_1}{\partial E} & \frac{\partial \mathcal{V}_1}{\partial I_S} & \frac{\partial \mathcal{V}_1}{\partial I_A} \\ \frac{\partial \mathcal{V}_2}{\partial E} & \frac{\partial \mathcal{V}_2}{\partial I_S} & \frac{\partial \mathcal{V}_2}{\partial I_A} \\ \frac{\partial \mathcal{V}_3}{\partial E} & \frac{\partial \mathcal{V}_3}{\partial I_S} & \frac{\partial \mathcal{V}_3}{\partial I_A} \end{bmatrix} = \begin{bmatrix} A_5 & 0 & 0 \\ -A_6 & A_1 & 0 \\ -\omega\delta & 0 & A_2 \end{bmatrix},$$

where  $A_1 = r_1 + d + d_1$  and  $A_2 = r_2 + d + d_2$ ,

$$A_3 = \frac{(1-\tau)\alpha_1}{N^0} \left( \frac{(1-\rho)\beta}{d+v} \right), A_4 = \frac{\alpha_2}{N^0} \left( \frac{(1-\rho)\beta}{d+v} \right),$$

$A_5 = \delta + d$  and  $A_6 = (1 - \omega)\delta$ . The total population at the DFE point is given by:

$$N^0 = S^0 + R^0. \text{ Then,}$$

$$V^{-1} = \begin{bmatrix} \frac{1}{A_5} & 0 & 0 \\ \frac{A_6}{A_1 A_5} & \frac{1}{A_1} & 0 \\ \frac{\omega \delta}{A_5 A_2} & 0 & \frac{1}{A_2} \end{bmatrix} \text{ and}$$

$$FV^{-1} = \begin{bmatrix} \frac{A_3 A_6}{A_5 A_1} + \frac{A_4 \omega \delta}{A_5 A_2} & \frac{A_3}{A_1} & \frac{A_4}{A_5} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

The eigenvalues of the Next-generation matrix,  $FV^{-1}$  is given by:

$$\Gamma = \begin{bmatrix} 0 \\ 0 \\ \frac{A_3 A_6}{A_5 A_1} + \frac{A_4 \omega \delta}{A_5 A_2} \end{bmatrix}$$

The basic reproduction number which corresponds to the dominant eigenvalue of  $FV^{-1}$  is denoted by  $R_0$  and given by:

$$R_0 = \frac{A_3 A_6}{A_1 A_5} + \frac{A_4 \omega \delta}{A_2 A_5} = \frac{A_2 A_3 A_6 + A_1 A_4 \omega \delta}{A_1 A_2 A_5} \quad (3)$$

$R_0$  can be written in full as:

$$J = \begin{bmatrix} \frac{\partial f_1}{\partial S} & \frac{\partial f_1}{\partial E} & \frac{\partial f_1}{\partial I_S} & \frac{\partial f_1}{\partial I_A} & \frac{\partial f_1}{\partial R} \\ \frac{\partial f_2}{\partial S} & \frac{\partial f_2}{\partial E} & \frac{\partial f_2}{\partial I_S} & \frac{\partial f_2}{\partial I_A} & \frac{\partial f_2}{\partial R} \\ \frac{\partial f_3}{\partial S} & \frac{\partial f_3}{\partial E} & \frac{\partial f_3}{\partial I_S} & \frac{\partial f_3}{\partial I_A} & \frac{\partial f_3}{\partial R} \\ \frac{\partial f_4}{\partial S} & \frac{\partial f_4}{\partial E} & \frac{\partial f_4}{\partial I_S} & \frac{\partial f_4}{\partial I_A} & \frac{\partial f_4}{\partial R} \\ \frac{\partial f_5}{\partial S} & \frac{\partial f_5}{\partial E} & \frac{\partial f_5}{\partial I_S} & \frac{\partial f_5}{\partial I_A} & \frac{\partial f_5}{\partial R} \end{bmatrix} = \begin{bmatrix} -\left(\frac{(1-\tau)}{N^0} \alpha_1 I_S^0 + \frac{\alpha_2 I_A^0}{N^0} + d + v\right) & 0 & -\frac{(1-\tau)}{N^0} \alpha_1 S^0 & \frac{-\alpha_2 S^0}{N^0} & 0 \\ \left(\frac{(1-\tau)}{N^0} \alpha_1 I_S^0 + \frac{\alpha_2 I_A^0}{N^0}\right) & -(\delta + d) & \frac{(1-\tau)}{N^0} \alpha_1 S^0 & \frac{\alpha_2 S^0}{N^0} & 0 \\ 0 & A_6 & -(r_1 + d + d_1) & 0 & 0 \\ 0 & \omega \delta & 0 & -(r_2 + d + d_2) & 0 \\ v & 0 & r_1 & r_2 & -d \end{bmatrix}$$

At the DFE, the Jacobian,  $J\left(\left(\frac{(1-\rho)\beta}{d+v}, 0, 0, 0, \frac{\beta}{d}\left(\frac{\rho d+v}{d+v}\right)\right)\right)$  is given by:

$$\begin{bmatrix} -(d+v) & 0 & -\frac{(1-\tau)}{N^0} \alpha_1 \left(\frac{(1-\rho)\beta}{d+v}\right) & \frac{-\alpha_2}{N^0} \left(\frac{(1-\rho)\beta}{d+v}\right) & 0 \\ 0 & -(\delta+d) & \frac{(1-\tau)}{N^0} \alpha_1 \left(\frac{(1-\rho)\beta}{d+v}\right) & \frac{\alpha_2}{N^0} \left(\frac{(1-\rho)\beta}{d+v}\right) & 0 \\ 0 & A_6 & -(r_1 + d + d_1) & 0 & 0 \\ 0 & \omega \delta & 0 & -(r_2 + d + d_2) & 0 \\ v & 0 & r_1 & r_2 & -d \end{bmatrix}$$

The matrix  $J$  can be reduced to a system of  $3 \times 3$  matrix by deleting the last and first columns with their corresponding rows since they have only one element. The eigenvalues of  $J(E^0)$  are  $-d$ ,  $-(d+v)$  and the roots of the characteristic polynomial:

$$\rho(X) = X^3 + (A_1 + A_2 + A_5)X^2 + (A_1 A_2 + A_2 A_5 + A_1 A_5 - A_4 A_6 - A_3 \omega_1 \delta)X + A_1 A_2 A_5 - A_1 A_4 A_6 - A_2 A_3 \omega_1 \delta$$

$$R_0 = \frac{(1-\tau)\alpha_1(1-\rho)\beta}{N^0(d+v)(r_1+d+d_1)(\delta+d)} + \frac{\alpha_2(1-\rho)\beta\omega\delta}{N^0(d+v)(r_2+d+d_2)(\delta+d)} \equiv R_{I_S} + R_{I_A} \quad (4)$$

where the first term,  $R_{I_S}$  is the average number of new cases caused by one symptomatic infectious bird introduced into the system before treatment while the second term,  $R_{I_A}$  is the number of new infections which the asymptomatic birds cause in the system.

### 3.0 Stability Analysis

#### 3.1 Local asymptotic stability of the disease-free equilibrium point

**Theorem 3.1.1:** The disease-free equilibrium point is locally asymptotically stable (LAS) if the reproduction number,  $R_0 < 1$ , and unstable if  $R_0 > 1$ .

**Proof:** To check if the DFE point is LAS, we compute the Jacobian of the system and obtain the nature of the eigenvalues. If the eigenvalues are all negative, then the DFE point is LAS, otherwise, it is not. The Jacobian of the system (2.1) is given by:

associated with the Jacobian submatrix:

$$J_1(E^0) = \begin{bmatrix} -A_5 & A_3 & A_4 \\ -A_6 & -A_1 & 0 \\ -\omega\delta & 0 & -A_2 \end{bmatrix}$$

where all the  $A_i$ 's  $i = 1,2,3,4,5$  are as described in section 2.4.

Let

$$\rho(X) = Q_3X^3 + Q_2X^2 + Q_1X + Q_0$$

with

$$\begin{aligned} Q_3 &= 1 \\ Q_2 &= A_1 + A_2 + A_5 \\ Q_1 &= A_1A_2 + A_2A_5 + A_1A_5 - A_4\omega\delta - A_3A_6 \\ Q_0 &= A_1A_2A_5 - A_1A_4\omega\delta - A_2A_3A_6 \end{aligned}$$

This  $Q_i$ 's can be written in terms of  $R_0$  as:

$$\begin{aligned} Q_0 &= A_1A_2A_5 - (A_1A_4\omega\delta + A_3A_6) = A_1A_2A_5 - A_1A_2A_5R_0 = A_1A_2A_5(1 - R_0) \\ Q_1 &= A_1A_2 + A_2A_5 + A_1A_5 - \left( A_2A_5R_0 - \frac{A_2A_3\omega_1\delta}{A_1} \right) - \left( A_1A_5R_0 - \frac{A_1A_4\omega\delta}{A_2} \right) \\ &= A_1A_2 + A_2A_5 + A_1A_5 - A_2A_5R_0 + \frac{A_2A_3A_6}{A_1} - A_1A_5R_0 + \frac{A_1A_4\omega\delta}{A_2} \\ &= A_1A_2 + \frac{A_2A_3A_6}{A_1} + \frac{A_1A_4\omega\delta}{A_2} + A_2A_5(1 - R_0) + A_1A_5(1 - R_0) \end{aligned}$$

By Routh-Hurwitz criterion, all the eigenvalues will be negative if  $Q_0 > 0, Q_1 > 0, Q_2 > 0, Q_3 > 0$  and  $Q_1Q_2 > Q_0$  (Herdicho et al., 2025; Castillo-Chavez & Song, 2004). This will only happen if  $R_0 < 1$ . Hence, the DFE points LAS if  $R_0 < 1$ . The local asymptotical stability of the DFE point shows that an influx of a small number of infectious birds into the system will not lead to an outbreak if  $R_0 < 1$  depending on the initial sizes of the infected population.

### 3.2 Global asymptotic stability of the disease-free equilibrium point

The method of Castillo-Chaves and Song (2004), Nana-Kyere et al., (2024), Naandam et al., (2025) and Akinyemi et al., (2023) summarized in Lemma 3 is used to check if the disease-free equilibrium point is globally asymptotically stable or not.

**Lemma 3.2.1:** Consider the system of differential equation:

$$\frac{dX}{dt} = F_1(X, 0), \quad (5)$$

$$\frac{dX}{dt} = F_2(X, Y), F_2(X, 0) = 0, \quad (6)$$

where,  $X = (S, R)$  corresponds to the system of equation (5) satisfied by non-diseased classes and  $Y = (E, I_S, I_A)$  corresponds to the system of equation (3) satisfied by the diseased classes. The disease-free equilibrium point is globally asymptotically stable if (5) is globally asymptotically stable, and if  $BY - F_2(X, Y) \geq 0$ , where  $B$  is the Jacobian matrix of  $F_2(X, Y)$ , evaluated at the disease-free equilibrium point.

**Theorem 3.2.2:** The disease-free equilibrium point of the model (1) is globally asymptotically stable if  $R_0 < 1$  and unstable if  $R_0 > 1$ .

**Proof.** Lemma 3 is employed to show that the conditions on (3.2.1) and (3) holds if  $R_0 < 1$ . At the disease-free equilibrium point, the corresponding differential equations for  $X = (S, R)$ ,

taking note that  $E = I_S = I_A = 0$ .

$$F_1(X, 0) = \left\{ \begin{array}{l} ((1-\rho)\beta - (\lambda + d + v)S) \\ r_1 I_S + r_2 I_A + \rho\beta + vS - dR \end{array} \right\} = \left\{ \begin{array}{l} (1-\rho)\beta - (d+v)S \\ vS - dR + \rho\beta \end{array} \right\}$$

Therefore,  $\frac{dS}{dt} = (1-\rho)\beta - (d+v)S \Rightarrow \frac{dS}{dt} + (d+v)S = (1-\rho)\beta$ , and  $\frac{dR}{dt} = \rho\beta + vS - dR \Rightarrow \frac{dR}{dt} + dR = \rho\beta + vS$ .

By integration,

$$S(t) = \frac{(1-\rho)\beta}{d+v} + \left[ S_0 - \frac{(1-\rho)\beta}{d+v} \right] e^{-(d+v)t},$$

$$R(t) = \frac{\rho\beta + vS^0}{d} + \left[ R - \frac{\rho\beta + vS^0}{d} \right] e^{-dt}$$

As  $t \rightarrow \infty$ ,  $S(t) \rightarrow \frac{(1-\rho)\beta}{d+v}$  and  $R(t) \rightarrow \frac{\rho\beta + vS^0}{d}$  which is the value of  $S(t)$  and  $R(t)$  respectively at the disease-free equilibrium. Hence,  $X = (S, R)$  is globally asymptotically stable. Also,

$$F_2(X, Y) = \begin{bmatrix} \lambda S - (\delta + d)E \\ (1-\omega)\delta E - (r_1 + d + d_1)I_S \\ \omega\delta E - (r_2 + d + d_2)I_A \end{bmatrix}$$

$$BY - F_2(X, Y) = \begin{bmatrix} -A_5 & A_3 & A_4 \\ (1-\omega)\delta & -A_1 & 0 \\ \omega\delta & 0 & -A_2 \end{bmatrix} \begin{bmatrix} E \\ I_S \\ I_A \end{bmatrix} - \begin{bmatrix} \lambda S - (\delta + d)E \\ (1-\omega)\delta E - (r_1 + d + d_1)I_S \\ \omega\delta E - (r_2 + d + d_2)I_A \end{bmatrix}$$

$$= \begin{bmatrix} -A_5 E + A_3 I_S + A_4 I_A - \lambda S + A_5 E \\ (1-\omega)\delta E - A_1 I_S - (1-\omega)\delta E + A_1 I_S \\ \omega\delta E - A_2 I_A - \omega\delta E + A_2 I_A \end{bmatrix} = \begin{bmatrix} A_3 I_S + A_4 I_A - \lambda S \\ 0 \\ 0 \end{bmatrix}$$

$$= \begin{bmatrix} [(1-\tau)\alpha_1 I_S + \alpha_2 I_A] \frac{S^0}{N^0} \left[ 1 - \frac{SN^0}{S^0 N} \right] \\ 0 \\ 0 \end{bmatrix}$$

Since  $S(t) \leq S^0$  and letting  $N = N^0$ , then  $1 - \frac{SN^0}{S^0 N} \geq 0$ . Hence, condition (3.2.1) is satisfied since  $BY - F_2(X, Y) \geq 0$ . Thus, the DFE is globally asymptotically stable. The global asymptotical stability of the disease-free equilibrium shows that no matter the initial sizes of the infected population, fowl pox will be controlled in the system when the  $R_0 < 1$ .

Note that since the total bird population satisfies  $\frac{dN}{dt} \leq \beta - dN$ , it follows that  $N(t) \leq \frac{\beta}{d} = N^0$  for all  $t \geq 0$ . Hence, the region  $\Omega = \{N \leq N^0\}$  is positively invariant. At the disease-free equilibrium,  $E = I_S = I_A = R = 0$ , so that  $N = S = \frac{\beta}{d} = N^0$ . Therefore,  $N = N^0$  can be used in the global stability analysis of the disease-free equilibrium.

### 3.3 Endemic Equilibrium Point

The endemic equilibrium point corresponds to the steady-state solution at which the disease persists in the population. Let

$$J^* = (S^*, E^*, I_S^*, I_A^*, R^*). \quad (7)$$

Donate the endemic equilibrium. Setting the right-hand side of system (2.1) equal to zero to get

$$S^* = \frac{(1-\rho)\beta}{\lambda^* + d + v}, E^* = \frac{\lambda^* S^*}{A_5}, I_S^* = \frac{(1-\omega)\delta}{A_1} E^*, I_A^* = \frac{\omega\delta}{A_2} E^*, R^* = \frac{r_1 I_S^* + r_2 I_A^* + \rho\beta + vS^*}{d}$$

Substituting into the force of infection, gives:

$$\lambda^* = \frac{\delta E^*}{N^*} \left[ \frac{(1-\tau)\alpha_1(1-\omega)}{A_1} + \frac{\alpha_2\omega}{A_2} \right] \quad (8)$$

and

$$E^* = \frac{\lambda^*(1-\rho)\beta}{A_5(\lambda^*+d+v)} \quad (9)$$

Therefore,

$$J^* = \left( \frac{(1-\rho)\beta}{\lambda^*+d+v}, \frac{\lambda^*(1-\rho)\beta}{A_5(\lambda^*+d+v)}, \frac{(1-\omega)\delta\lambda^*(1-\rho)\beta}{A_1A_5(\lambda^*+d+v)}, \frac{\omega\delta\lambda^*(1-\rho)\beta}{A_2A_5(\lambda^*+d+v)}, R^* \right) \quad (10)$$

where

$$R^* = \frac{\beta}{d} \left[ \rho + \frac{v(1-\rho)}{\lambda^*+d+v} + \frac{\delta\lambda^*(1-\rho)}{A_5(\lambda^*+d+v)} \left( \frac{r_1(1-\omega)}{A_1} + \frac{r_2\omega}{A_2} \right) \right]$$

### 3.4 Existence of the Endemic Equilibrium

Theorem 3.4.1: The endemic equilibrium  $J^*$  exists if and only if  $R_0 > 1$ .

**Proof**

At equilibrium,

$$E^* > 0, \quad I_S^* > 0, \quad I_A^* > 0.$$

$E^*$ ,  $I_S^*$  and  $I_A^*$ , positivity of these variables requires,  $\lambda^* > 0$ .

The infected subsystem linearized around the disease-free equilibrium yield the basic reproduction number:

$$R_0 = \frac{(1-\tau)\alpha_1(1-\rho)\beta(1-\omega)\delta}{N^0(d+v)A_1A_5} + \frac{\alpha_2(1-\rho)\beta\omega\delta}{N^0(d+v)A_2A_5}$$

When  $R_0 < 1$ , the disease-free equilibrium is globally asymptotically stable, implying that all infected class tend to zero. Consequently, no positive endemic equilibrium can exist. When  $R_0 > 1$ , the disease invasion threshold is exceeded and a positive solution  $\lambda^* > 0$  exist. Hence,

$S^* > 0$ ,  $E^* > 0$ ,  $I_S^* > 0$ ,  $I_A^* > 0$ ,  $R^* > 0$ , which establishes the existence of a unique biologically feasible endemic equilibrium. Therefore, the endemic equilibrium exists if and only if  $R_0 > 1$ .

### 3.5 Local Stability of the Endemic Equilibrium

#### Theorem 3.5.1

The endemic equilibrium  $J^*$  is locally asymptotically stable whenever  $R_0 > 1$ .

**Proof**

The Jacobian matrix of system (2.1) evaluated at  $J^*$  is:

$$J(J^*) = \begin{pmatrix} -(\lambda^*+d+v) & 0 & -B_1 & -B_2 & 0 \\ \lambda^* & -A_5 & B_1 & B_2 & 0 \\ 0 & (1-\omega)\delta & -A_1 & 0 & 0 \\ 0 & \omega\delta & 0 & -A_2 & 0 \\ v & 0 & r_1 & r_2 & -d \end{pmatrix} \quad (11)$$

where

$$B_1 = \frac{(1-\tau)\alpha_1 S^*}{N^*}, \quad B_2 = \frac{\alpha_2 S^*}{N^*}.$$

The characteristic polynomial of  $J(J^*)$  is:

$$P(\mu) = \mu^5 + c_1\mu^4 + c_2\mu^3 + c_3\mu^2 + c_4\mu + c_5 \quad (12)$$

For  $R_0 > 1$ , all coefficients satisfy:

$$c_i > 0, \quad i = 1, \dots, 5$$

And the Routh-Hurwitz conditions:  $c_1c_2 > c_3$ ,

$$c_1c_2c_3 > c_3^2 + c_1^2c_4,$$

are satisfied.

Hence, all eigenvalues of  $J(J^*)$  have negative real parts. Therefore, the endemic equilibrium is locally asymptotically stable whenever  $R_0 > 1$ .

### 4.0 Sensitivity Analysis

The sensitivity analysis carried out using the normalized forward sensitivity index of  $R_0$  for each parameter,  $q$  is given by:

$$S_q^{R_0} = \frac{\partial R_0}{\partial q} \times \frac{q}{R_0}.$$

The method has been used in many works such as Anjam et al. (2024), Rasheed et al. (2020), Sharma et al. (2024), Udofia et al. (2024). The analysis helps to verify which parameters affect the basic reproduction number,  $R_0$  more for effective control of the disease (Umar et al., 2021). The sensitivity indices are presented in Table 2 and Figures 2 – 3. In the sensitivity analysis, parameters with negative sensitivity indices contribute to reducing disease transmission when increased and also reduce the

basic reproduction number when increased while parameters with positive index affect the endemicity of the disease and need to be reduced to control the spread of the disease.

**Table 2: Sensitivity indices of parameters of  $R_0$**

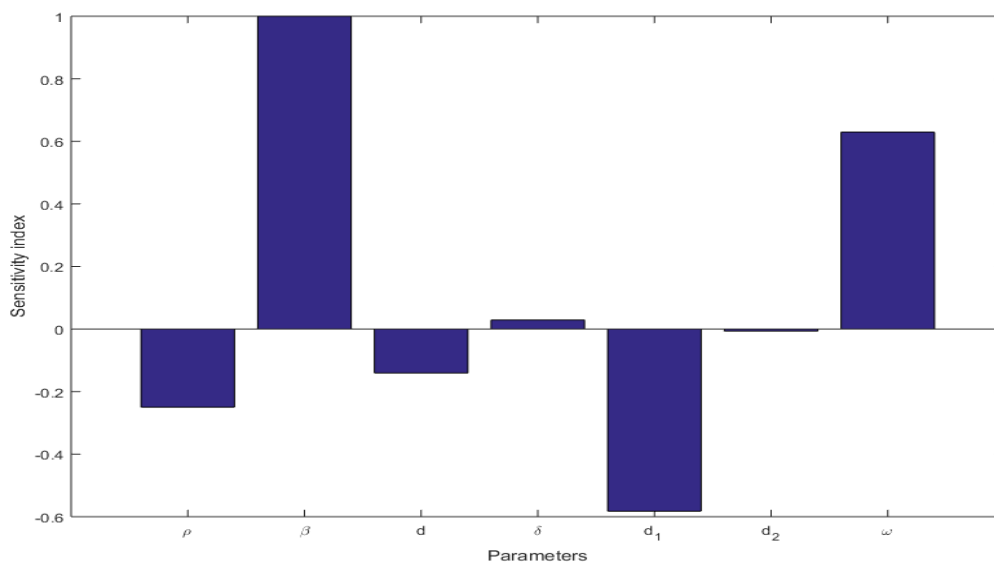
Parameters	Sensitivity index	Parameters	Sensitivity index
$\beta$	1	$\delta$	0.0291
$\rho$	-0.25	$r_1$	-0.2912
$d$	-0.1405	$r_2$	-0.0652
$d_1$	-0.5824	$\tau$	-1.7196
$d_2$	-0.0065	$\alpha_1$	0.9259
$\nu$	-0.9434	$\alpha_2$	0.0741
$\omega$	0.6297		

The sensitivity analysis shows that rate of recruitment,  $\beta$ , probabilities of transmission,  $\alpha_1$  and  $\alpha_2$ , rate of development of infectiousness,  $\delta$  and proportion of birds which are symptomatic increase the endemicity of the disease. To control the disease, measures that will help reduce the values of these parameters need to be implemented. This is the essence of the control measures proposed in this work. As more birds are vaccinated and efforts are made to see that the recruited birds are mostly vaccinated, there will be a smaller number of infections in the system. Also, if the sick birds are effectively culled as proposed in this work, the infectious will not interact with others and the probabilities of infection will be drastically reduced. The

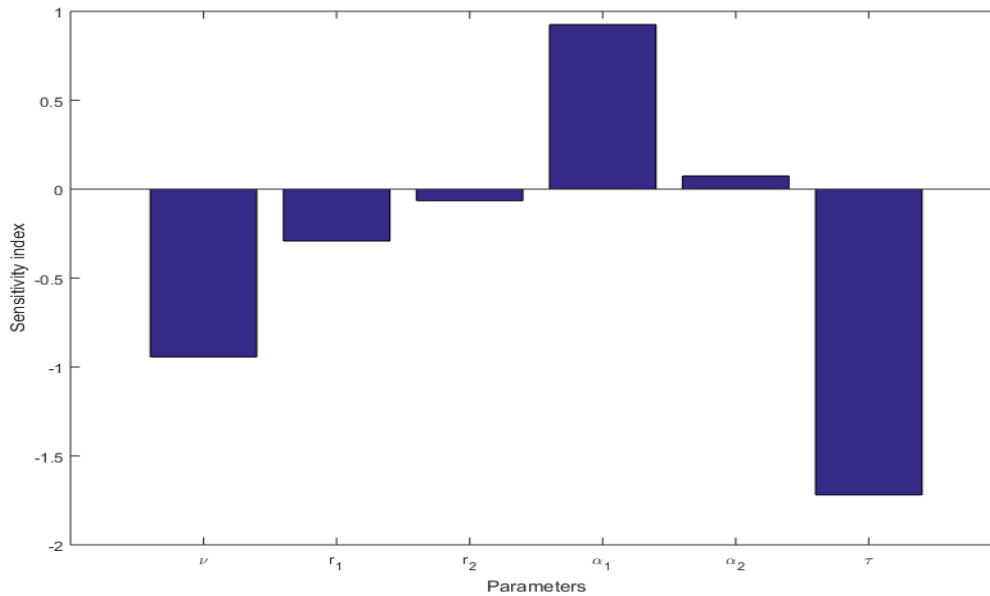
other parameters which are negative need to be improved for effective control of the disease. For example, if the rate of culling,  $\tau$  and rate of vaccination,  $\nu$  is increased, the endemicity of the disease is reduced, highlighting the role each parameter plays in the dynamics of the disease.

**5.0 Numerical Simulations**

Numerical simulations on the model are performed here using MATLAB version 7.5.0.342 (R2007b), simulation time interval of (0:0.05:0.5), (ode45) and the following assumed initial conditions of the state variables;  $S = 300, E = 80, I_S = 42, I_A = 28, R = 6$  and values of the parameters in Table 3.



**Figure 2: Sensitivity indices**



**Figure 3:** Sensitivity indices

**Table 3:** Parameters, values and sources

Parameters	Values	Sources
$\beta$	50	James et al. (2021)
$\rho$	0.2	James et al. (2021)
$d$	0.018	Udofia and Inyama (2011)
$d_1$	0.2	Agwu et al. (2018)
$d_2$	0.05	Agwu et al. (2018)
$\nu$	0.3	Udofia and Inyama (2011)
$\omega$	0.8	Udofia and Inyama (2011)
$\delta$	0.6	Agwu et al. (2018)
$r_1$	0.1	Okwor et al. (2018)]
$r_2$	0.5	Agwu et al. (2018)
$\tau$	0.65	Assumed
$\alpha_1$	0.25	Assumed
$\alpha_2$	0.05	Assumed

Since reliable field estimates for the transmission coefficients of symptomatic and asymptomatic fowlpox-infected birds are scarce, parameter values were adopted from ranges reported in avian epidemic and fowlpox transmission studies. The symptomatic transmission rate was assumed to exceed the asymptomatic transmission rate ( $\alpha_1 > \alpha_2$ ) due to increased viral shedding by clinically infected birds. Vaccination efficacy/coverage was

assumed to be 70%, consistent with reports of successful fowl pox vaccination programs in poultry populations Okwor et al., 2018).

### 5.1 Numerical Evaluation of the Basic Reproduction number, $R_0$

Using the parameter values in Table 3 and initial total bird population:

$$N^0 = 300 + 80 + 42 + 28 + 6 = 456$$

The Basic Reproduction number is:

$$R_0 = \frac{(1-\tau)\alpha_1(1-\rho)\beta}{N^0(d+\nu)(r_1+d+d_1)(\delta+d)} + \frac{\alpha_2(1-\rho)\beta\omega\delta}{N^0(d+\nu)(r_2+d+d_2)(\delta+d)}. \text{ Substituting the values gives:}$$

$$R_0 = \frac{(1-0.65)(0.25)(1-0.2)50}{456(0.018+0.3)(0.1+0.018+0.2)(0.6+0.018)} + \frac{(0.05)(1-0.2)(50)(0.8)(0.6)}{456(0.028+0.3)(0.5+0.018+0.05)(0.6+0.018)} = 0.141954$$

$$R_0 \approx 0.1420 \quad (13)$$

### Interpretation

The Basic Reproduction is  $R_0 = 0.1420 < 1$ . This means that an infectious bird generates, on average, only about 0.1420 secondary infections during its infectious period since each infected bird produces less than one new infection, the disease cannot establish itself in the population and will eventually die out. Therefore, the disease-free equilibrium is locally asymptotically stable. Biologically, this indicates that the combined effects of vaccination, culling, recovery, and mortality are sufficient to suppress the spread of fowl pox in the bird population. Under the assumed parameter values, no sustained outbreak is expected.

### 5.2 Effects of vaccination on the population

Here, the effects of vaccinating birds are shown by varying the rate of vaccination,  $\nu$  and the proportion of recruited birds,  $\rho$  which are

vaccinated. The control measure employed in this case is to see how increasing vaccination rate and proportion of recruited birds which are vaccinated will affect the spread of fowl pox. The results of the simulation are shown in Figures 4 – 8.

Figure 4 illustrates the dynamics of the susceptible bird population for different vaccination rates. As the vaccination rate increases, the number of susceptible birds decreases more rapidly over time. This reduction occurs because vaccination transfers birds from the susceptible class to the protected or recovered class, thereby reducing the pool of birds that can become infected. The result demonstrates that vaccination is an effective preventive strategy for limiting disease transmission within the flock.

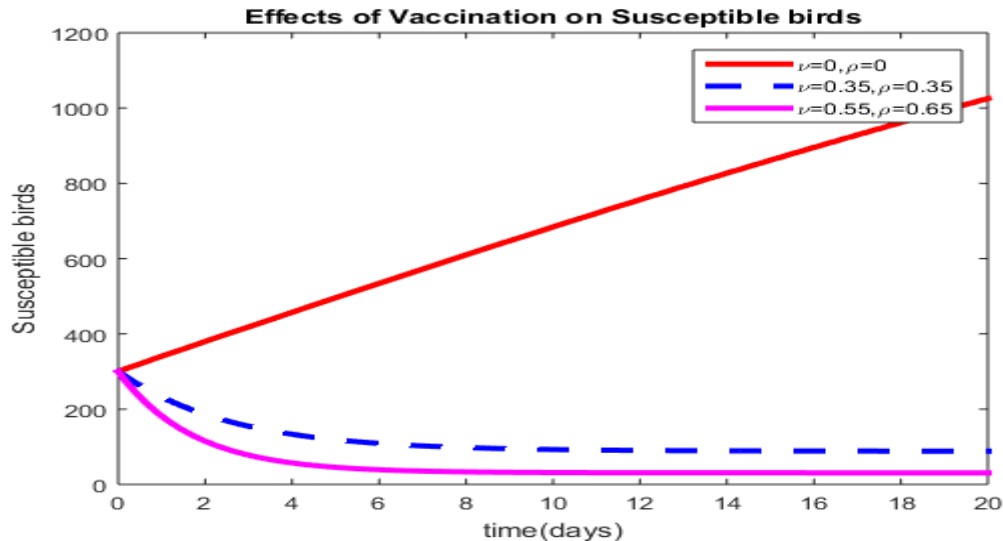


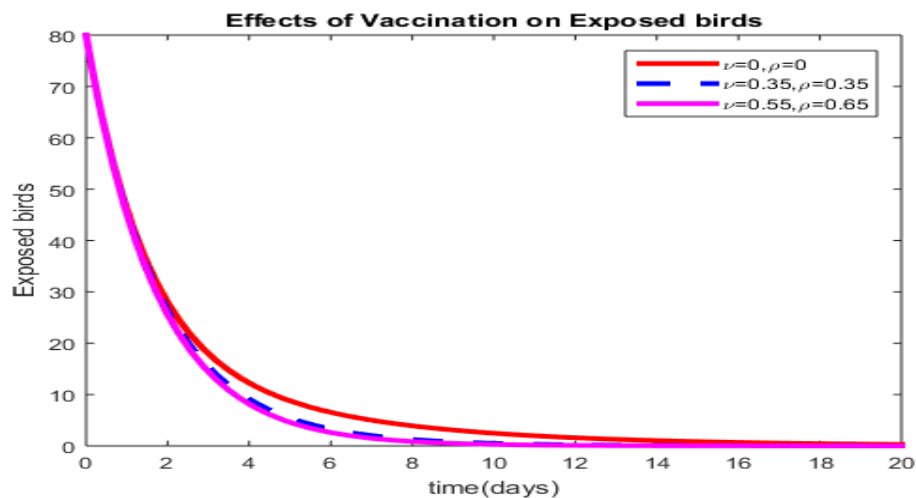
Figure 4: Susceptible birds under vaccination

Figure 5 shows the variation in the exposed bird population under different vaccination rates. It is observed that increasing the vaccination rate leads to a significant decline in the number of exposed birds. Since fewer susceptible birds are available to contract the infection, the number of newly infected birds entering the exposed class decreases. This finding indicates that

vaccination effectively interrupts disease transmission at an early stage. Figure 6 depicts the effect of vaccination on the symptomatic infectious bird population. The results reveal that higher vaccination rates substantially reduce the number of symptomatic birds over time. This reduction is attributable to the lower number of exposed birds progressing to the symptomatic

infectious stage. Consequently, vaccination helps to reduce clinical manifestations of fowl

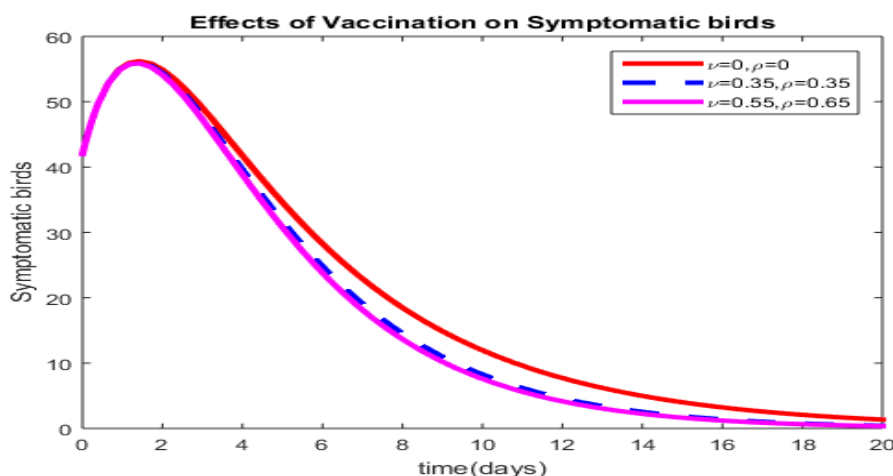
pox and minimizes disease-related losses in poultry populations.



**Figure 5:** Exposed birds under vaccination

Figure 7 presents the behavior of the asymptomatic infectious population under varying vaccination rates. The graph indicates that as vaccination coverage increases, the number of asymptomatic birds decreases. Since asymptomatic birds can unknowingly contribute to disease spread, their reduction highlights the importance of vaccination in controlling both visible and hidden sources of infection. Figure 8 illustrates the recovered bird population under

different vaccination rates. The graph shows that the number of recovered birds increases as the vaccination rate increases. This outcome is expected because vaccination provides immunity and increases the proportion of birds protected from infection. The growth of the recovered class demonstrates the ability of vaccination to enhance herd immunity and contribute to long-term disease control.



**Figure 6:** Symptomatic birds under vaccination

**5.2 Effects of culling sick birds:** In this section, the effects of culling infected birds are investigated by varying the culling rate. The results are presented in Figures 9 – 11. Figure 9 shows that increasing the culling rate leads to a reduction in the exposed bird population. The

prompt removal of infected birds decreases the likelihood of disease transmission to susceptible birds, thereby reducing the number of new infections. This finding suggests that culling can effectively limit the spread of fowl pox within the flock.

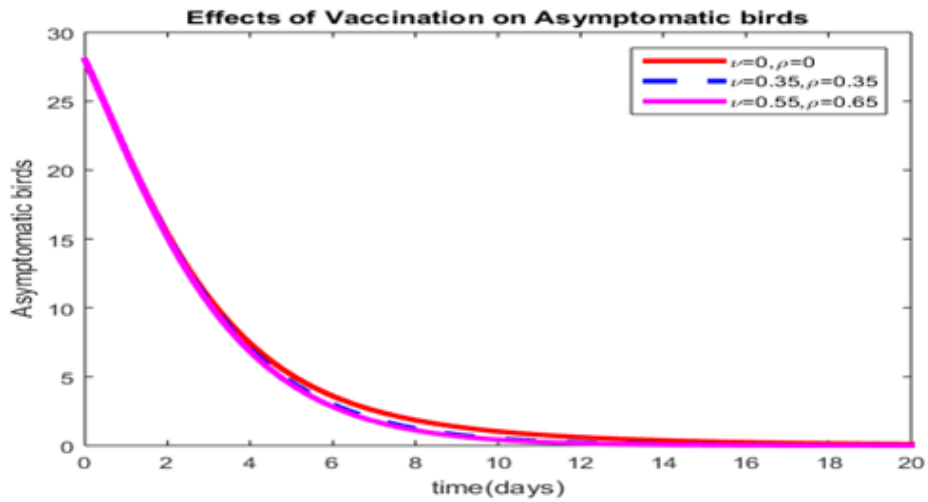


Figure 7: Asymptomatic birds under vaccination

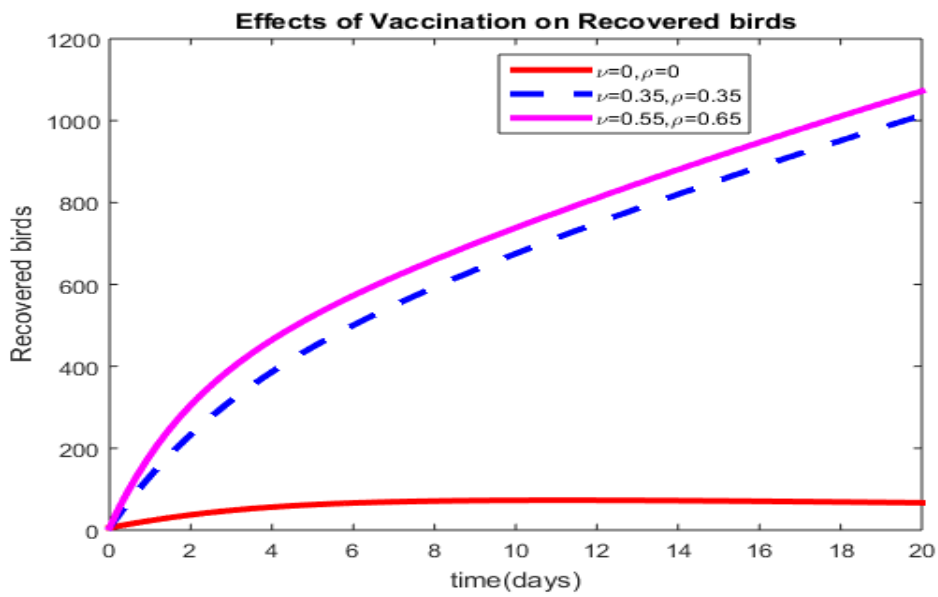


Figure 8: Recovered birds under vaccination

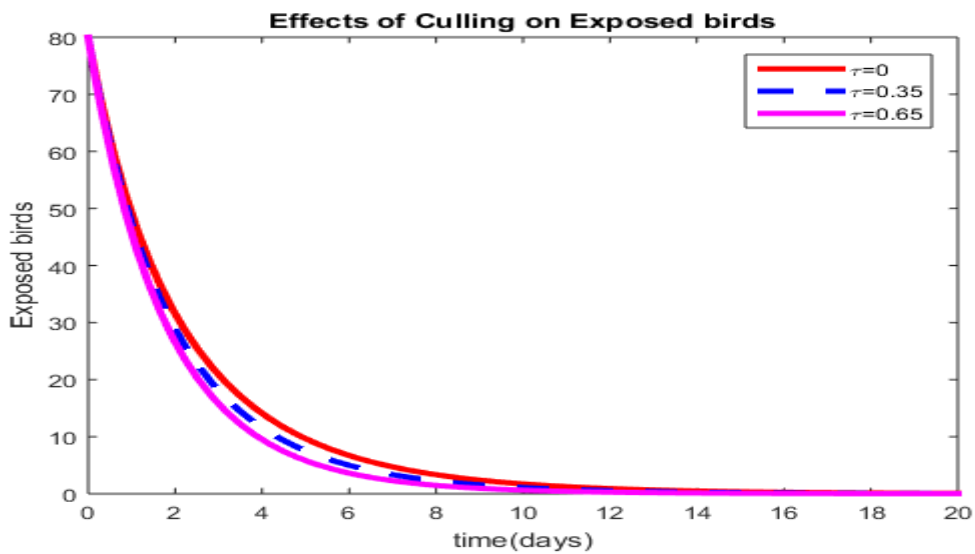
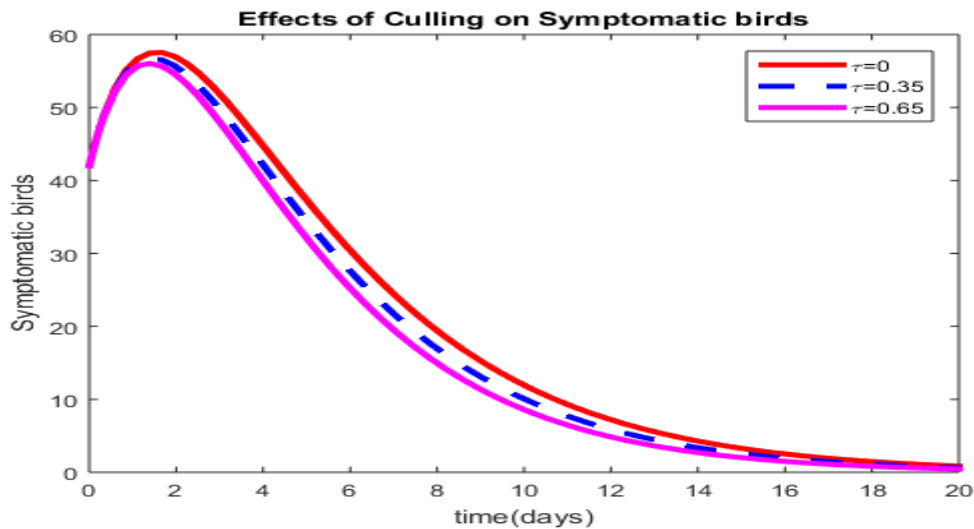


Figure 9: Exposed birds under culling



**Figure 10:** Symptomatic birds under culling

Figure 10 demonstrates that the population of symptomatic infectious birds decreases as the culling rate increases. Since infected birds are removed from the system more rapidly, the duration of infection and the opportunity for further disease transmission are significantly reduced. The result confirms that culling is an effective intervention for lowering the prevalence of clinically infected birds. Figure 11 illustrates the effect of culling on the asymptomatic infectious population. The graph indicates that higher culling rates result in fewer asymptomatic birds over time. By reducing the overall level of infection in the flock, culling indirectly decreases the number of birds that progress into or remain within the asymptomatic infectious class. This outcome contributes to the overall reduction of disease transmission.

## 6.0 Results and Discussion

### 6.1 Basic Reproduction Number and Stability Analysis

The basic reproduction number obtained for the model was  $R_0 = 0.1420$ . Since  $R_0 < 1$ , the disease-free equilibrium is both locally and globally asymptotically stable. This result implies that, under the assumed parameter values and intervention strategies, an infectious bird generates less than one secondary infection on average before recovery, death, or removal from the population. Consequently, fowl pox

cannot establish sustained transmission within the bird population and will eventually die out. The decomposition further shows that both symptomatic and asymptomatic infectious birds contribute to disease transmission. However, the symptomatic class contributes more significantly because the transmission coefficient associated with symptomatic birds  $\alpha_1$  is considerably larger than that of asymptomatic birds  $\alpha_2$ . This observation is biologically realistic since birds exhibiting clinical signs of fowl pox often shed larger quantities of virus and may therefore contribute more to disease spread.

### 6.2 Effects of Vaccination on Disease Dynamics

Numerical simulations showed that increasing the vaccination rate  $v$  and the proportion of recruited birds vaccinated  $\rho$  substantially reduced the susceptible, exposed, symptomatic infectious, and asymptomatic infectious populations while increasing the recovered class. Vaccination affects disease dynamics by reducing the number of susceptible birds available for infection. As more birds acquire immunity through vaccination, the effective contact between susceptible and infectious birds decreases, thereby lowering the force of infection. Consequently, fewer birds progress into the exposed and infectious classes, leading to a reduction in disease prevalence. The

sensitivity analysis supports this observation; with the vaccination parameter  $\nu$  having a large negative sensitivity index of  $(-0.9434)$ . This indicates that increasing vaccination coverage produces a significant reduction in the basic

reproduction number. Therefore, vaccination remains one of the most effective preventive strategies for controlling fowl pox outbreaks in poultry populations.

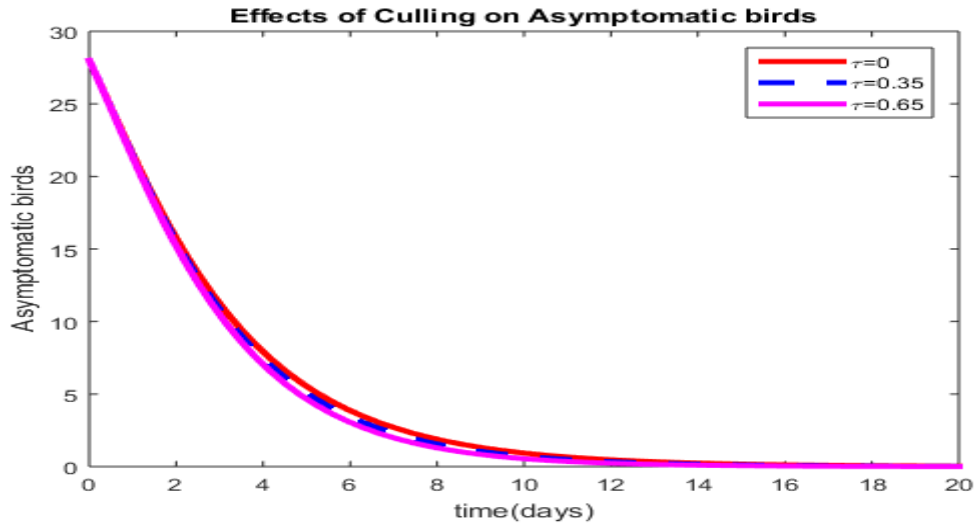


Figure 11: Asymptomatic birds under culling

### 6.3 Effects of Culling on Disease Dynamics

The simulations also demonstrated that increasing the culling rate  $\tau$  significantly reduced the exposed, symptomatic, and asymptomatic infectious bird populations. This reduction occurs because culling removes infectious birds from the population before they can infect additional susceptible birds. Among all parameters investigated, the culling rate exhibited the largest negative sensitivity index  $(-1.7196)$ , indicating that it exerts the strongest suppressive effect on disease transmission. Epidemiologically, this suggests that rapid identification and removal of infected birds can substantially reduce the spread of fowl pox during outbreaks. The combined implementation of vaccination and culling therefore provides a dual mechanism of disease control: vaccination reduces susceptibility, while culling reduces infectiousness. Together, these strategies lower transmission more effectively than either intervention alone.

### 6.4 Comparison with Previous Studies

The present study extends the work of Udofia and Sampson (2014), who developed a

mathematical model for fowl pox transmission incorporating discrete delay effects associated with the incubation period of the disease. While their model provided valuable insights into disease progression, it did not explicitly include vaccination, culling, or separate symptomatic and asymptomatic infectious classes. The current findings are consistent with those of Udofia and Sampson (2014) in demonstrating the existence of a threshold reproduction number that determines disease persistence or eradication. However, the present study provides additional epidemiological insight by quantifying the impact of intervention measures. The obtained value ( $R_0 = 0.1420$ ) indicates that vaccination and culling can reduce disease transmission to a level well below the epidemic threshold. The results also agree with findings reported in avian influenza and poultry disease models, where vaccination reduces the susceptible population and lowers disease prevalence, while removal of infected individuals reduces the effective infectious pool. Similar conclusions were reported by Gulbudak and Martcheva (2014), who showed that

vaccination plays a critical role in reducing disease transmission in avian populations. The strong negative sensitivity indices associated with vaccination and culling observed in this study further support the widely accepted role of these interventions in controlling infectious diseases among birds. Unlike previous fowl pox models, the present work explicitly evaluates the relative contributions of symptomatic and asymptomatic infections and demonstrates the effectiveness of combining preventive and reactive control measures within a unified modelling framework.

### 6.5 Epidemiological Implications

The findings of this study have important implications for poultry disease management and policy formulation. First, the reproduction number below unity indicates that maintaining adequate vaccination coverage and prompt culling practices can prevent large-scale outbreaks of fowl pox. Second, the sensitivity analysis suggests that control programmes should prioritize increasing vaccination coverage and strengthening surveillance systems capable of rapidly identifying infected birds. Since the transmission coefficient of symptomatic birds was found to contribute strongly to disease spread, early detection and isolation of clinically infected birds are essential. Third, the inclusion of asymptomatic infectious birds highlights the possibility of hidden transmission within poultry flocks. Consequently, disease-control programmes should not rely solely on visible clinical signs but should also incorporate routine monitoring and biosecurity measures. Finally, the results emphasize the importance of integrated disease management strategies. Vaccination alone reduces susceptibility, whereas culling directly reduces the infectious population. The combined application of both interventions provides the greatest reduction in transmission and offers a practical framework for controlling fowl pox in commercial and backyard poultry systems. Overall, the study demonstrates that sustained

vaccination programmes, coupled with rapid removal of infected birds, can effectively reduce disease transmission, maintain ( $R_0 = 0.1420 < 1$ ) and ultimately eliminate fowl pox from affected bird populations.

In general, the numerical simulations demonstrate that both vaccination and culling are effective strategies for controlling the transmission of fowl pox among birds. Vaccination reduces the susceptible population, lowers the number of exposed and infectious birds, and increases the number of immune (recovered) birds. These effects collectively reduce disease transmission and promote flock immunity. On the other hand, culling infected birds reduces the number of exposed, symptomatic, and asymptomatic birds by removing sources of infection from the population. The results are consistent with epidemiological theory, which predicts that reducing the susceptible population through vaccination and eliminating infectious individuals through culling will decrease disease prevalence. The findings also agree with previous studies that have reported vaccination and culling as effective disease-control measures in poultry populations. The combined implementation of these interventions can significantly reduce the basic reproduction number and suppress disease outbreaks. From an epidemiological perspective, the study suggests that increasing vaccination coverage while maintaining appropriate culling of infected birds can substantially reduce the burden of fowl pox and improve poultry health and productivity. Therefore, vaccination and culling should be considered important complementary control measures for the effective management of fowl pox outbreaks in bird populations.

### 7.0 Conclusion

This study developed and analyzed a deterministic mathematical model for the transmission dynamics of fowl pox in a bird population. The model extends the work of

Udofia and Sampson (2014) by incorporating vaccination of susceptible and newly recruited birds, culling of symptomatic infectious birds, and separate symptomatic and asymptomatic infectious classes. These modifications provide a more realistic representation of disease transmission and control in poultry populations. The fundamental properties of the model, including positivity and boundedness of solutions, were established, ensuring that the model is mathematically and epidemiologically well posed. The disease-free equilibrium was derived and analyzed, and the basic reproduction number was obtained using the next-generation matrix approach. Numerical evaluation of the reproduction number yielded ( $R_0 = 0.1420$ ), which is less than unity. This result indicates that fowl pox cannot persist in the population under the assumed parameter values and control measures. Furthermore, the disease-free equilibrium was shown to be both locally and globally asymptotically stable whenever ( $R_0 < 1$ ). Sensitivity analysis revealed that the culling rate and vaccination rate are the most influential parameters in reducing disease transmission, whereas the recruitment rate and symptomatic transmission coefficient contribute positively to the spread of infection. Numerical simulations further demonstrated that increasing vaccination coverage and culling intensity significantly reduces the exposed, symptomatic infectious, and asymptomatic infectious bird populations while increasing the recovered population. From an epidemiological perspective, the study highlights the effectiveness of integrating vaccination and culling strategies in controlling fowl pox outbreaks. Vaccination reduces the pool of susceptible birds and limits disease transmission, while culling removes infectious birds from the population before they can generate additional infections. The combined implementation of these interventions was shown to be more effective than relying on either strategy alone. Overall, the findings of this study provide quantitative evidence that sustained

vaccination programmes, coupled with timely culling of infected birds, can successfully suppress the transmission of fowl pox and prevent disease persistence in poultry populations. The model contributes to the understanding of fowl pox epidemiology and provides a useful framework for evaluating control strategies aimed at reducing the economic burden of the disease on the poultry industry.

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