SABA Publishing

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

J Math Anal & Model
(2024)5(3) : 100-120
doi:10.48185/jmam.v5i3.1214

Modeling the Transmission Dynamics of Avian Influenza in Cattle

FADHILI M. MROPE[a,*]

[a] Department of Mathematics and Statistics, Sokoine University of Agriculture, P.O. BOX 3038, Morogoro, Tanzania.

• *Received: 23 June 2024* • *Accepted: 12 October 2024* • *Published Online: 21 December 2024*

Abstract

Avian Influenza (AI) poses a critical threat to cattle production worldwide, resulting in significant yield losses and economic damages. Despite the severity of AI, comprehensive modeling studies on its transmission dynamics within cattle populations remain limited. In this study, we present a mathematical model to describe the spread of AI among cattle. The model is based on the Susceptible-Infectious-Recovered (SIR) framework, adapted to capture the unique characteristics of AI transmission. The disease-free equilibrium of the model was computed, and the basic reproduction number for AI was calculated using the next-generation matrix method. Sensitivity analysis was conducted using the normalized forward sensitivity method to determine the impact of various parameters on the basic reproduction number ($\mathcal{R}_0$). Analytical and numerical analyses indicate that increased contact rates between susceptible cattle and infected viruses significantly raise the transmission rate of AI, impacting cattle health and productivity. Sensitivity analysis highlights that the recruitment rates of cattle and infection rates are the most influential parameters affecting $\mathcal{R}_0$. Control measures such as introducing AI-resistant cattle breeds and improving farm management practices to reduce infection rates may be used to mitigate the disease spread. This study enhances the understanding of AI transmission dynamics, providing valuable insights for developing targeted control strategies to protect cattle health and improve production.

Keywords: Avian Influenza, Cattle, Transmission dynamics, Basic reproduction number ($\mathcal{R}_0$), Sensitivity analysis.
2010 MSC: .

1. Introduction

Avian influenza is a highly contagious viral disease affecting both domestic and wild birds [10, 12]. It can also infect mammals, including cattle [12, 15, 14, 13]. The disease is caused by various subtypes of influenza viruses a member of the genus Influenzavirus A and family *Orthomyxoviridae* such as H5N1, H5N3, and H5N8, with evolving genetic characteristics [16, 17, 5]. Avian influenza viruses are classified into low and high

*Corresponding author: fadhili.mrope@sua.ac.tz

pathogenicity forms, with HPAI A(H5N1) known to infect mammals, particularly those in environments with high virus concentrations [23, 18, 19, 20], instances of dairy cattle (or goats) contracting the H5N1 virus are relatively uncommon [44]. The disease, has been a global concern since its identification in 1878 in Italy [9, 6].Avian influenza virus type A (H5N1) has been confirmed in dairy cattle in 12 states: 25 herds in Michigan, 22 in Idaho, 18 in Texas, eight in New Mexico, five each in South Dakota and Colorado, four in Kansas, three in Minnesota, two in Iowa, and one each in North Carolina, Ohio, and Wyoming [42, 45].

Common clinical signs in affected cows include low appetite, reduced milk production, and abnormal appearance of milk (thickened, discolored) [42, 43, 45]. The spread of the H5N1 virus within and among herds indicates that bovine-to-bovine spread occurs, likely through mechanical means. Evidence also indicates that the virus can spread from dairy cattle premises to nearby poultry facilities [45].

Controlling avian influenza in cattle involves the implementation of vaccination programs to enhance immunity, strict biosecurity measures to prevent the introduction and spread of the virus, and isolation or quarantine of new or sick animals [54, 55]. Regular surveillance and monitoring are essential for early detection and response to outbreaks [56]. Improving farm management practices, such as maintaining clean and sanitary conditions and reducing stress in cattle, can enhance their overall health and resistance to diseases [57]. Limiting contact between cattle and wild birds through barriers and other measures is also crucial, as wild birds can be carriers of the virus [58]. Educating farmers and farm workers about avian influenza and ensuring compliance with control measures through public awareness campaigns can further mitigate the disease's spread [59]. Additionally, breeding cattle strains that are genetically resistant to avian influenza offers a long-term solution [60]. Effective environmental controls, proper waste management, and having emergency response plans in place for rapid action during outbreaks are also vital components of a successful control strategy [61].

Despite these comprehensive efforts, the problem of avian influenza in cattle persists, necessitating the exploration of other approaches to understand its dynamics better. Mathematical models can offer valuable insights into spread of avian influenza in cattle [48]. By applying these models, we can effectively address real-life challenges in managing and mitigating the transmission dynamics of avian influenza within cattle populations [49, 46]. Several models have been developed to study the transmission dynamics of Avian Influenza [50, 51, 52, 53].

In a recent study, Pinotti et al. [8] explored the transmission dynamics of H9N2 avian influenza viruses in a live bird market, providing significant insights into how the virus spreads within this environment. Their study found that, H9N2 avian influenza virus (AIV) spreads rapidly among chickens in Chattogram's live bird market due to a short latent period, especially in broilers, high transmission rates, and a constant influx of susceptible chickens. Poor cleaning and frequent introduction of infectious chickens further support virus persistence. Malek and Hoque [46] conducted a study using mathematical modeling to investigate the infectious spread and outbreak dynamics of avian influenza with seasonal transmission on chicken farms. Their study noted that the extent of seasonal variation influenced the timing of disease transmission patterns and influenced the efficacy of control measures in managing outbreaks.

Li et al. [11] provided insights into the global stability of a degenerate diffusion avian influenza model incorporating seasonality and spatial heterogeneity. Their study found that, spatial heterogeneity and environmental factors influence the transmission dynamics of avian influenza viruses. Lou et al. [25] conducted predictive evolutionary modeling for influenza virus, focusing on site-based dynamics of mutations to forecast virus evolution. Zheng et al. [7] explored the role of seasonality and spatial heterogeneity in the transmission dynamics of avian influenza, focusing on nonlinear analysis in real-world applications. Their study found that, birds migrate in response to seasonal and habitat changes, thereby facilitating the spread of the avian influenza virus.

Alhassan and Achema [50] conducted a qualitative and quantitative analysis to model the transmission dynamics of avian influenza. Their study found that, the disease can be eliminated with highly effective vaccination of 80% to 90% of susceptible individuals and diligent treatment of infected cases. Khan et al. [21] conducted a study on the transmission dynamics of avian influenza, focusing on half-saturated incidence models using modeling and scientific computing techniques. Their study found that, influenza could potentially be eliminated from the community if the threshold does not exceed unity. Ali et al. [26] explored the dynamics of a stochastic avian influenza model incorporating asymptomatic carriers, utilizing spectral methods. The findings suggest that the influenza will spread rapidly if all infected individuals show symptoms and receive inadequate treatment.

While mathematical modeling has extensively studied avian influenza in poultry and humans, there remains a significant gap in understanding its implications for cattle, as existing models primarily focus on poultry and human populations, neglecting distinctive transmission dynamics and control challenges in cattle; this study aims to address this gap.

The paper is organized as follows: The immediate section presents materials and methods where the proposed model is extensively explained and formulated. Section 3 presents results and discussions followed by simulations of the model in section 4. Section 5 is the conclusion part of the paper.

2. Avian Influenza in Cattle Model Formulation

In this section, we develop a mathematical model for the transmission dynamics of Avian Influenza (AI) in cattle, extending previous models in the field. Cattle form the host population, divided into three sub-populations: Susceptible $S(t)$, Infected $I(t)$, and Recovered $R(t)$, with the total cattle population given by $N_1 = S(t) + I(t) + R(t)$. Birds, which act as disease vectors, are categorized into Susceptible $B(t)$ and Infected $J(t)$ subpopulations, with the total bird population given by $N_2 = B(t) + J(t)$. The model incorporates the recruitment rate of susceptible birds $B(t)$ at the rate of Λ_2 and their transition to infected birds $J(t)$ at the rate of β_2 following contact with infected cattle. Susceptible cattle $S(t)$ are replenished at the rate of Λ_1 and transition to infected cattle $I(t)$ at the rate of β_1 after exposure to infected birds. Infected cattle may recover at the rate of α, potentially returning to the susceptible state at the rate of ϕ post-exposure to vectors. Additionally, the model accounts for natural death rates: μ_1 for cattle and μ_2 for birds. Moreover, it is assumed that the cattle population undergoes constant natural mortality,

there is a constant replenishment rate of susceptible cattle, infected cattle can die due to disease at the rate of δ, bird populations increase proportionally with the number of infected cattle, susceptible cattle get infected upon contact with infected birds, and there is a constant natural mortality rate within the bird population. The parameters used in this model are respectively summarized in Table 1.

Table 1: Model parameters and their description

Parameter	Description	Value	Source
Λ_1	Recruitment rate in Susceptible cattle population	100	[36]
Λ_2	Recruitment rate of avian influenza virus	0.03	Assumed
α	Recovery rate of infected cattle	0.56	[39]
β_1	Infection rate of susceptible cattle by infected virus	0.01	Assumed
β_2	Infection rate of susceptible virus by infected cattle	0.02	Assumed
μ_1	Natural death rate of cattle	0.02	[35]
μ_2	Natural death rate of avian influenza virus	0.00137	[37]
ϕ	Rate of returning to susceptible state by cattle	0.05	[34]
δ	Disease induced death rate of cattle	0.8	[35]

2.1. Compartmental Flow Diagram of Avian Influenza Transmission Dynamics

This is a visual representation of the the spread of the virus among susceptible, infected, and recovered cattle and susceptible and infected birds. Figure 1 shows the interactions between Susceptible, Infected, and Recovered individuals.

2.2. Model Equations for the Avian Influenza Dynamics in Cattle

These equations explain the intricate dynamics of Avian Influenza in cattle, capturing how the infection propagates and evolves within the cattle population, influencing disease transmission, progression, and potential control strategies. The differential equations describing the model are as follows:

$$\begin{cases} \dfrac{dS}{dt} &= \Lambda_1 - (\beta_1 J + \mu_1)\, S \\ \dfrac{dI}{dt} &= \beta_1 JS - (\alpha + \delta + \mu_1) I \\ \dfrac{dR}{dt} &= \alpha I - (\phi + \mu_1) R \\ \dfrac{dB}{dt} &= \Lambda_2 - \beta_2 IB - \mu_2 B \\ \dfrac{dJ}{dt} &= \beta_2 IB - \mu_2 J \end{cases} \tag{2.1}$$

subject to the non-negative initial conditions: $S(0) > 0$, $I(0) \geqslant 0$, $R(0) \geqslant 0$, $B(0) > 0$, and $J(0) \geqslant 0$.

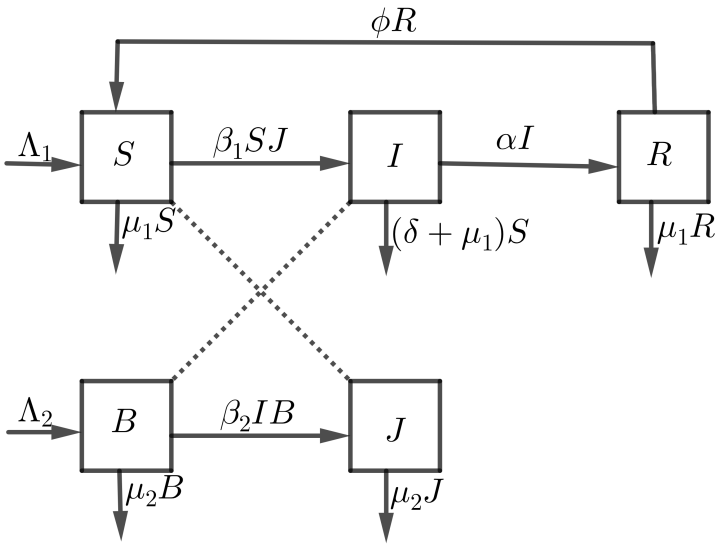

Figure 1: Dynamics of Avian Influenza transmission in cattle. The sold line indicates the rate of transfer into and out of the populations. The dotted lines indicates the usual interactions among populations

3. Model Properties and Analysis

3.1. Positivity of solution

To ensure our model is ecologically and epidemiologically meaningful and well-posed, it is crucial to establish that all state variables in the system, starting with positive initial values, remain positive for all $t \geqslant 0$. The following theorem is employed to confirm the positivity of the system.

Theorem 1.

$$\text{Let } \Omega = \left\{(S, I, R, B, J) \in \mathbb{R}^5_+ \mid S(0) > 0, I(0) \geqslant 0, R(0) \geqslant 0, B(0) > 0, J(0) \geqslant 0\right\}.$$

Then the solution set $S(t), I(t), R(t), B(t), J(t)$ *of model system (2.1) is positive for all* $t \geqslant 0$.

Proof. From the first equation of model system (2.1) we have:

$$\frac{dS}{dt} \geqslant \Lambda_1 - (\beta_1 J + \mu_1) S$$

By separating the variables, we have:

$$\int_{S(0)}^{S(t)} \frac{dS}{S} \geqslant -\int (\beta_1 J + \mu_1) dt$$

$$\ln S(t) - \ln S(0) \geqslant -\int (\beta_1 J + \mu_1) dt$$

$$\ln S(t) \geqslant -(\beta_1 J + \mu_1)\, t$$

Upon simplifying further, we get:

$$S(t) \geqslant S(0)e^{-(\beta_1 J+\mu_1)t} > 0$$

Following the same procedures, we have:

$$\begin{aligned} I(t) &\geqslant I(0)e^{-(\alpha+\delta+\mu_1)t} > 0 \\ R(t) &\geqslant R(0)e^{-(\phi+\mu_1)t} > 0 \\ B(t) &\geqslant B(0)e^{-(\beta_2 I+\mu_2)t} > 0 \\ J(t) &\geqslant J(0)e^{-(\mu_2)t} > 0 \end{aligned}$$

Therefore, it can be deduced that $S(t)$ will remain positive for all t if the initial susceptible population $S(0)$ is positive, and the exponential term $e^{-(\beta_1 J+\mu_1)t}$ remains non-negative. Using the same procedures, it can be proven that $I(0) \geqslant 0$, $R(0) \geqslant 0$, $B(0) \geqslant 0$, and $J(0) \geqslant 0$. □

3.2. Invariant region

This section evaluates the biological relevance of model variables and the validity of a bounded solution over time, using the integration factor method on the model system (2.1).

$$\frac{dN_1}{dt} = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = \Lambda_1 - \mu_1 N_1 - \delta I. \tag{3.1}$$

$$\frac{dN}{dt} \leqslant \Lambda_1 - \mu_1 N$$

Solving this, we obtain

$$0 \leqslant N_1(t) \leqslant \frac{\Lambda_1}{\mu_1} + N_1(0)\exp^{-\mu_1 t}. \tag{3.2}$$

As $t \to \infty$, we have $0 < N_1(t) \leqslant \frac{\Lambda_1}{\mu_1}$.
Also considering the pathogen population,$N_2 = B + J$ we have;

$$\frac{dN_2}{dt} = \frac{dB}{dt} + \frac{dJ}{dt} = \Lambda_2 - \mu_2 N_2. \tag{3.3}$$

$$\frac{dN}{dt} \leqslant \Lambda_2 - \mu_2 N$$

Solving this, we obtain

$$0 \leqslant N_2(t) \leqslant \frac{\Lambda_2}{\mu_2} + N_2(0)\exp^{-\mu_2 t}. \tag{3.4}$$

As $t \to \infty$, we have $0 < N_2(t) \leqslant \frac{\Lambda_2}{\mu_2}$. where N_1 = total cattle population and N_2 = total bird population. Therefore, the set of feasible solutions for the Avian Influenza model is defined as:

$$\Omega = \Omega_1 \times \Omega_2 = \left\{ (S, I, R, B, J) \in \mathbb{R}^5_+ \leqslant \frac{\Lambda_1}{\mu_1} \leqslant \frac{\Lambda_2}{\mu_2} \right\} \tag{3.5}$$

A feasible solution that remains positively invariant in $\mathbb{R}^5_+$ indicates the model's epidemiological and mathematical validity, enabling further analysis.

3.3. Avian Influenza-Free Equilibrium Point

The Avian Influenza-free equilibrium in the model occurs when the disease is absent in the cattle population, achieved by setting all equations in the transmission dynamics model (2.1) to zero. Thus,

$$E^0 = (S^0, I^0, R^0, B^0, J^0) = \left(\frac{\Lambda_1}{\mu_1}, 0, 0, \frac{\Lambda_2}{\mu_2}, 0\right) \tag{3.6}$$

3.4. Basic Reproduction Number ($\mathcal{R}_0$)

The basic reproduction number R_0 quantifies the average secondary infections caused by a single infectious host in a fully susceptible population [30, 1, 31], calculated using the next-generation operator approach by Diekmann et al. [1, 2]. This study follows Mfinanga et al.'s [32] method, focusing on the infective classes to determine $\mathcal{R}_0$.

$$\frac{dI}{dt} = \beta_1 JS - (\alpha + \delta + \mu_1)I$$
$$\frac{dJ}{dt} = \beta_2 IB - \mu_2 J$$

Applying the next-generation matrix approach, we obtain:

$$\mathcal{F}_i = \begin{bmatrix} \beta_1 JS \\ \beta_2 IB \end{bmatrix}$$

and

$$\mathcal{V}_i = \begin{bmatrix} (\alpha + \delta + \mu_1) \\ \mu_2 \end{bmatrix}$$

Taking partial derivatives of $\mathcal{F}_i$ and $\mathcal{V}_i$ with respect to I and J at the disease-free equilibrium point yields:

$$F = \begin{bmatrix} \beta_1 \frac{\Lambda_1}{\mu_1} & 0 \\ 0 & \beta_2 \frac{\Lambda_2}{\mu_2} \end{bmatrix}$$

and

$$V = \begin{bmatrix} 0 & (\alpha + \delta + \mu_1) \\ \mu_2 & 0 \end{bmatrix}$$

Given that the basic reproduction number $\mathcal{R}_0$ corresponds to the maximum eigenvalue of FV^{-1}. Hence,

$$FV^{-1} = \begin{bmatrix} 0 & \frac{\beta_1 \Lambda_1}{\mu_1 \mu_2} \\ \frac{\beta_2 \Lambda_2}{\mu_2(\alpha+\delta+\mu_1)} & 0 \end{bmatrix}$$

The characteristic equation of the above equation is:

$$\lambda^2 - \frac{\beta_1 \beta_2 \Lambda_1 \Lambda_2}{\mu_1 \mu_2^2 (\alpha + \delta + \mu_1)} = 0$$

Solving for λ, we get:

$$\lambda = \sqrt{\frac{\beta_1 \beta_2 \Lambda_1 \Lambda_2}{\mu_1 \mu_2^2 (\alpha + \delta + \mu_1)}}$$

Thus, The value of the basic reproduction number $\mathcal{R}_0$ is determined as:

$$\mathcal{R}_0 = \sqrt{\frac{\beta_1\beta_2\Lambda_1\Lambda_2}{\mu_1\mu_2^2(\alpha+\delta+\mu_1)}} = \mathcal{R}_{01} \times \mathcal{R}_{02} \tag{3.7}$$

Here,

- $\mathcal{R}_{01} = \sqrt{\frac{\beta_1\Lambda_1}{\mu_1}}$ represents the number of infected birds caused by a typical infectious cattle.
- $\mathcal{R}_{02} = \sqrt{\frac{\beta_2\Lambda_2}{\mu_2^2(\alpha+\delta+\mu_1)}}$ represents the number of infected cattle caused by a typical infectious bird.

3.5. Local Stability Analysis of Avian Influenza-free equilbrium in Cattle

In this subsection, we utilize the eigenvalue method to assess the local stability of the disease-free equilibrium point for the model system describing Avian Influenza transmission dynamics in cattle.

Theorem 2. *The disease-free equilibrium point for the Avian Influenza model in cattle is locally asymptotically stable if the basic reproduction number $\mathcal{R}_0 < 1$, and unstable if $\mathcal{R}_0 > 1$.*

Proof. To establish the local stability of the disease-free equilibrium point, we examine the eigenvalues of the Jacobian matrix of the model system. The Jacobian matrix for the Avian Influenza model in cattle is given by:

$$J(E_0) = \begin{bmatrix} -\mu_1 & 0 & 0 & 0 & -\beta_1 S^0 \\ 0 & -(\alpha+\delta+\mu_1) & 0 & 0 & \beta_1 S^0 \\ 0 & \alpha & -(\phi+\mu_1) & 0 & 0 \\ 0 & -\beta_2 B^0 & 0 & -\mu_2 & 0 \\ 0 & \beta_2 B^0 & 0 & 0 & -\mu_2 \end{bmatrix}$$

We see that the Jacobian matrix $J(E_0)$ have $-\mu_1$, $-\mu_2$, and $-(\phi+\mu_1)$ as its eigenvalues. Eliminating the rows and columns containing μ_1 and $-(\phi+\mu_1)$, the matrix $J(E_0)$ is reduced to:

$$J_1(E_0) = \begin{bmatrix} -(\alpha+\delta+\mu_1) & \beta_1 S^0 \\ \beta_2 B^0 & -\mu_2 \end{bmatrix}$$

From the Jacobian matrix, we obtain a characteristic polynomial as

$$\begin{aligned} (\lambda + (\alpha+\delta+\mu_1))(\lambda+\mu_2) - \beta_2 B^0 \beta_1 S^0 &= 0 \\ \lambda^2 + \lambda\mu_2 + \lambda(\alpha+\delta+\mu_1) + \mu_2(\alpha+\delta+\mu_1) - \frac{\beta_1\beta_2\Lambda_1\Lambda_2}{\mu_1\mu_2} &= 0 \\ \lambda^2 + \lambda(\alpha+\delta+\mu_1+\mu_2) + \mu_2(\alpha+\delta+\mu_1)[1 - \frac{\beta_1\beta_2\Lambda_1\Lambda_2}{\mu_1\mu_2^2(\alpha+\delta+\mu_1)}] &= 0 \\ \lambda^2 + \lambda(\alpha+\delta+\mu_1+\mu_2) + \mu_2(\alpha+\delta+\mu_1)[1-\mathcal{R}_0^2] &= 0 \\ \lambda^2 + a_1\lambda + a_2 &= 0 \end{aligned} \tag{3.8}$$

We applied Routh-Hurwitz criteria, and by the principle, equation (3.8) has strictly negative real root if $a_1 > 0$ and $a_2 > 0$. Clearly we see that $a_1 > 0$ because it is the sum of positive parameters and $a_2 > 0$ as $1 - \mathcal{R}_0^2 > 0$ at DFE. Hence the DFE is locally asymptotically stable if $\mathcal{R}_0 < 1$. □

3.6. Global stability of Avian Influenza-free Equilibrium

In this subsection, we perform a global stability analysis of the Avian Influenza-free equilibrium point using the approach explained by Castillo-Chavez et al. [3]. The formulation of the model system can be expressed as follows:

$$\frac{dX_s}{dt} = A(X_s - X_{DFE,S}) + A_1 X_i$$
$$\frac{dX}{dt} = A_2 X_i$$

Here, X_s represents the vector of non-transmitting compartments, and X_i represents the transmitting components. The Disease-Free Equilibrium (DFE) achieves global asymptotic stability if the matrix A possesses real negative eigenvalues, and A_2 constitutes a Metzler matrix, where the off-diagonal elements of A_2 are non-negative. It is essential to verify whether the matrix A associated with the non-transmitting compartments possesses real negative eigenvalues and that A_2 forms a Metzler matrix. From the model system, we can define $X_s = (S, I, R)^T$ and $X_i = (B, J)^T$. Referring to the equation for non-transmitting compartments, we obtain:

$$A = \begin{bmatrix} -(\beta J + \mu_1) & 0 & 0 \\ 0 & -(\phi + \mu_1) & 0 \\ 0 & 0 & -\mu_2 \end{bmatrix}$$

with eigenvalues $\lambda_1 = -(\beta J + \mu_1)$, $\lambda_2 = -(\phi + \mu_1)$, and $\lambda_3 = -\mu_2$. Additionally,

$$A_2 = \begin{bmatrix} -(\alpha + \delta + \mu_1) & \beta_1 S^0 \\ \beta_2 B^0 & -\mu_2 \end{bmatrix}$$

It can be seen that A_2 is a Metzler matrix, and both A and A_2 have real negative eigenvalues. This implies that the disease-free equilibrium for the model system (2.1) is globally asymptotically stable.

3.7. Disease Endemic Equilibrium Point of the Model

The endemic equilibrium point, E^*, of the model is the steady state solution where *avian flu* persist in the population of cattle. The endemic equilbrium point can be obtained by equating each equation of the model system (2.1) equal to zero; that is,

$$\begin{aligned} \Lambda_1 - (\beta_1 J^* + \mu_1) S^* &= 0 \\ \beta_1 J^* S^* - (\alpha + \delta + \mu_1) I^* &= 0 \\ \alpha I^* - (\phi + \mu_1) R^* &= 0 \\ \Lambda_2 - (\beta_2 I^* + \mu_2) B^* &= 0 \\ \beta_2 I^* B^* - \mu_2 J^* &= 0 \end{aligned} \tag{3.9}$$

From the equation above, we get

$$S^* = \frac{\Lambda_1}{\beta_1 J^* + \mu_1} \tag{3.10}$$

$$I^* = \frac{\beta_1 J^* S^*}{(\alpha + \delta + \mu_1)} \tag{3.11}$$

$$R^* = \frac{\alpha I^*}{(\phi + \mu_1)} \tag{3.12}$$

$$B^* = \frac{\Lambda_2}{\beta_2 I^* + \mu_2} \tag{3.13}$$

$$J^* = \frac{\beta_2 I^* B^*}{\mu_2} \tag{3.14}$$

Upon solving and making correct substitutions we get;

$$\begin{aligned}
S^* &= \frac{\Lambda_1[(\beta_2\mu_2(\mathcal{R}_0^2-1)+(\beta_1\Lambda_2+\mu_1\mu_2)]}{\beta_1\beta_2\Lambda_2(\mathcal{R}_0^2-1)+\mu_1[(\beta_2\mu_2(\mathcal{R}_0^2-1)+(\beta_1\Lambda_2+\mu_1\mu_2)]} \\
I^* &= \frac{(\mathcal{R}_0^2-1)\mu_2^2}{\beta_1\Lambda_2+\mu_1\mu_2} \\
R^* &= \frac{\alpha(\mathcal{R}_0^2-1)\mu_2^2}{(\phi+\mu_1)(\beta_1\Lambda_2+\mu_1\mu_2)} \\
B^* &= \frac{\Lambda_2(\beta_1\Lambda_2+\mu_1\mu_2)}{\beta_2(\mathcal{R}_0^2-1)\mu_2^2+\mu_2(\beta_1\Lambda_2+\mu_1\mu_2)} \\
J^* &= \frac{(\mathcal{R}_0^2-1)\beta_2\Lambda_2}{\beta_2\mu_2(\mathcal{R}_0^2-1)+(\beta_1\Lambda_2+\mu_1\mu_2)}
\end{aligned} \tag{3.15}$$

Thus, the endemic equilibrium point exists if $\mathcal{R}_0 > 1$.

3.8. Local stability of Endemic Equilbrium points

In this subsection, we employ the Jacobian stability method to examine the local stability of the endemic equilibrium point of the disease.

Theorem 3. *The endemic equilibrium* E^* *of system(2.1) is locally asymptotically stable in* Ω *when* $R_0 > 1$.

Proof. The local stability of the endemic equilibrium E^* is assessed by analyzing the eigenvalues of the Jacobian matrix computed at E^*, with the matrix expressed as:

$$J(E^*) = \begin{bmatrix} -\mu_1 & 0 & 0 & 0 & -\beta_1 S^* \\ 0 & -(\alpha+\delta+\mu_1) & 0 & 0 & \beta_1 S^* \\ 0 & \alpha & -(\phi+\mu_1) & 0 & 0 \\ 0 & -\beta_2 Y^* & 0 & -\mu_2 & 0 \\ 0 & \beta_2 Y^* & 0 & 0 & -\mu_2 \end{bmatrix}$$

The characteristic equation of the Jacobian matrix of equation (3.8) at the endemic equilibrium point, E^*, is $|J(E_0) - \lambda I_5| = 0$. That is,

$$|J(E^*) - \lambda I_5| = \begin{vmatrix} -\mu_1 - \lambda & 0 & 0 & 0 & -\beta_1 S^* \\ 0 & -(\alpha + \delta + \mu_1) - \lambda & 0 & 0 & \beta_1 S^* \\ 0 & \alpha & -(\phi + \mu_1) - \lambda & 0 & 0 \\ 0 & -\beta_2 Y^* & 0 & -\mu_2 - \lambda & 0 \\ 0 & \beta_2 Y^* & 0 & 0 & -\mu_2 - \lambda \end{vmatrix} = 0$$

The matrix can be partitioned into smaller matrices that can be dealt with separately. Starting with the top-left 3×3 block: Let the matrix be A, then;

$$|A| = \begin{vmatrix} -\mu_1 - \lambda & 0 & 0 \\ 0 & -(\alpha + \delta + \mu_1) - \lambda & 0 \\ 0 & \alpha & -(\phi + \mu_1) - \lambda \end{vmatrix}$$

$$|A| = (-\mu_1 - \lambda) \cdot [-(\alpha + \delta + \mu_1) - \lambda] \cdot [-(\phi + \mu_1) - \lambda]$$

Then dealing with the bottom-right 2×2 block: Let the matrix be B, then;

$$|B| = \begin{vmatrix} -\mu_2 - \lambda & 0 \\ \beta_2 Y^* & -\mu_2 - \lambda \end{vmatrix}$$

$$|B| = (-\mu_2 - \lambda)^2$$

Combining these results, we get the characteristic equation:

$$(-\mu_1 - \lambda) \cdot [-(\alpha + \delta + \mu_1) - \lambda] \cdot [-(\phi + \mu_1) - \lambda] \cdot (-\mu_2 - \lambda)^2 = 0$$

Thus, the characteristic equation confirms that the eigenvalues (solutions to this equation) determine the stability of the equilibrium point. If all eigenvalues have negative real parts, the equilibrium is locally asymptotically stable. The eigenvalues are:

$$\begin{aligned} \lambda_1 &= -\mu_1, \\ \lambda_2 &= -(\alpha + \delta + \mu_1), \\ \lambda_3 &= -(\phi + \mu_1), \\ \lambda_4 &= -\mu_2 \text{ (with multiplicity 2).} \end{aligned}$$

Since μ_1, α, δ, ϕ, and μ_2 are all positive parameters, all the eigenvalues are negative. Therefore, the endemic equilibrium point is locally asymptotically stable. □

3.9. Global Stability of Endemic Equilbrium Points

The Lyapunov function was used to confirm the global stability of the endemic equilibrium E^* with a negative derivative.

Theorem 4. *The model system exhibits a distinct endemic equilibrium point E^*, which is globally asymptotically stable when $\mathcal{R}_0 > 1$, and unstable otherwise.*

Proof. A Lyapunov function for the model system (2.1) was utilized in this study, following the approach of Vargas-De-León [28], Korobeinikov et al. [24], and Korobeinikov [27]. The Lyapunov function V is defined as

$$V(\mathbf{x}) = \sum_{i=1}^{n} \frac{1}{2}(\mathbf{x}_i - \mathbf{x}_i^*)^2,$$

where $\mathbf{x}_i$ represents the population in the i-th compartment and $\mathbf{x}_i^*$ denotes the endemic equilibrium point. The model system (2.1) demonstrates the following positive definite function.

$$F(S, I, R, B, J) = \sum_{i=1}^{5} \frac{1}{2}(\mathbf{x}_i - \mathbf{x}_i^*)^2$$

Then, the Lyapunov function for the AI model system is expressed as:

$$V = \frac{1}{2}[(S - S^*) + (I - I^*) + (R - R^*) + (B - B^*) + (J - J^*)]^2.$$

Differentiating $V(t)$ with respect to time yields:

$$\frac{dV}{dt} = [(S - S^*) + (I - I^*) + (R - R^*) + (B - B^*) + (J - J^*)]\frac{d}{dt}[S + I + R + B + J].$$

$$\frac{dV}{dt} = [S + I + R + B + J - (S^* + I^* + R^* + B^* + J^*)]\frac{d}{dt}[S + I + R + B + J].$$

Next, we evaluate:

$$\frac{d}{dt}(S + I + R + B + J) = \Lambda_1 + \Lambda_2 - \delta I - (\mu_1 N_1 + \mu_2 N_2).$$

We find:

$$\begin{aligned} \Lambda_1 - \delta I^* - \mu_1 N_1^* + \Lambda_2 - \mu_2 N_2^* &= 0, \\ \Rightarrow (\Lambda_1 + \Lambda_2) - \delta I^* - (\mu_1 + \mu_2)(S^* + I^* + R^* + B^* + J^*) &= 0, \\ (S^* + I^* + R^* + B^* + J^*) &= \frac{(\Lambda_1 + \Lambda_2) - \delta I^*}{(\mu_1 + \mu_2)}. \end{aligned}$$

Inserting into $\frac{dV}{dt}$ yields:

$$\begin{aligned} \frac{dV}{dt} &= \left[N(t) - \frac{(\Lambda_1 + \Lambda_2) - \delta I^*}{\mu_1 + \mu_2}\right][(\Lambda_1 + \Lambda_2) - \delta I - (\mu_1 + \mu_2)N(t)] \\ \frac{dV}{dt} &= \left[N(t) - \frac{(\Lambda_1 + \Lambda_2) - \delta I^*}{\mu_1 + \mu_2}\right]\left[-(\mu_1 + \mu_2)\left(N(t) - \frac{(\Lambda_1 + \Lambda_2) - \delta I}{\mu_1 + \mu_2}\right)\right] \\ \frac{dV}{dt} &= -(\mu_1 + \mu_2)\left[N(t) - \frac{\Lambda_1 + \Lambda_2}{\mu_1 + \mu_2} + \frac{\delta I^*}{\mu_1 + \mu_2}\right]\left[N(t) - \frac{\Lambda_1 + \Lambda_2}{\mu_1 + \mu_2} + \frac{\delta I}{\mu_1 + \mu_2}\right] \\ \frac{dV}{dt} &\leqslant -(\mu_1 + \mu_2)\left[N(t) - \frac{\Lambda_1 + \Lambda_2}{\mu_1 + \mu_2}\right]^2 < 0. \end{aligned}$$

$$\frac{dV}{dt} \leqslant -(\mu_1+\mu_2)\left[N(t)-\left(\frac{(\Lambda_1+\Lambda_2)}{(\mu_1+\mu_2)}\right)\right]^2 < 0.$$

Hence, it is evident that, $\frac{dV}{dt} < 0$.
Therefore, the endemic equilibrium point (E^*) for avian influenza disease in cattle is globally asymptotically stable, suggesting that the disease prevalence will ultimately stabilize around this equilibrium point after sufficient time, regardless of the initial conditions. □

4. Findings and Discussion

4.1. Sensitivity Analysis of the Avian Influenza model

Sensitivity analysis examines how variations in model parameters affect outcomes, crucial for optimizing disease transmission dynamics by reducing $\mathcal{R}_0$. Applying the normalized forward sensitivity index method developed by Chitnis et al. [4], we computed sensitivity indices for parameters affecting the basic reproduction number ($\mathcal{R}_0$), indicating their impact on whether $\mathcal{R}_0$ increases or decreases. Table 2 displays these sensitivity indices for the model parameters.

Table 2: Sensitivity Indices

Parameter	**Index**
Λ_1	+0.5000
Λ_2	+0.5000
β_1	+0.5000
β_2	+0.5000
μ_1	-0.5072
μ_2	-1.0000
α	-0.2029
δ	-0.2899

4.2. Interpretation of the sensitivity indices

Higher recruitment rates in susceptible cattle (Λ_1 and Λ_2) and infection rates (β_1 and β_2) lead to increased avian influenza transmission dynamics among cattle. For instance, a higher Λ_1 leads to a higher incidence of avian influenza cases, potentially by up to 50%, while higher β_1 and β_2 increase disease spread significantly, potentially by up to 50%. Therefore, interventions targeting these parameters, like controlling cattle population size or lowering transmission rates, are crucial in mitigating avian influenza spread.

Negative sensitivity indices indicate that increases in certain parameters, such as natural death rates of cattle and the avian influenza virus, recovery rate of infected cattle, and disease-induced death rate of cattle, lead to decreased transmission dynamics of avian influenza. For instance, a higher μ_2 decreases virus prevalence by up to 100%, α speeds recovery, reducing transmission by 20%, and δ accelerates infected individuals' removal, potentially lowering transmission by 28.99%.

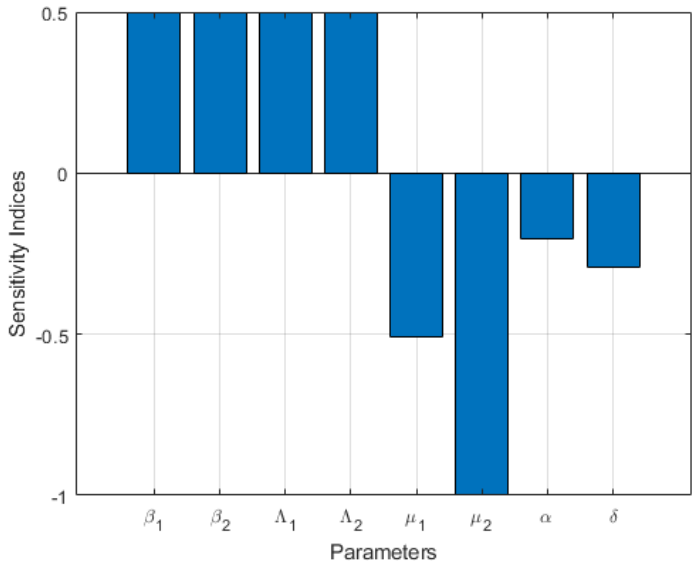

Figure 2: Graph of sensitivity indices of $\mathcal{R}_0$ with respect to the model parameters

This highlights the importance of strategies aimed at increasing the recovery rate of infected cattle and the natural death rate of the virus, such as effective veterinary care and antiviral treatments, to control the spread of avian influenza among cattle. Additionally, managing the cattle population to ensure a balanced natural death rate can help mitigate the transmission dynamics of avian influenza.

4.3. Numerical Simulation

Numerical algorithms in MATLAB are employed to simulate avian influenza transmission in cattle, utilizing parameter values from Table 1 and initial conditions ($S = 1000$, $I = 100$, $R = 0$, $B = 500$, $J = 50$) for real-world accuracy, facilitating analysis of the model's behavior under various scenarios.

4.3.1. Avian Influenza Prevalence among the Entire Cattle Population

This section discusses the rate of new avian influenza cases within the entire cattle population, as modeled in the study. Figure 3 shows the disease trend over time.

Figure 3 illustrates the changing dynamics of cattle and bird populations in response to disease progression. Initially, the susceptible cattle population is high, decreasing over time as individuals become infected. Similarly, the number of infected cattle decreases as recovery occurs, though with temporary immunity. Recovered cattle populations initially rise, peak, then stabilize due to temporary immunity loss. Meanwhile, susceptible bird populations decline rapidly as infected bird numbers rise swiftly.

The initial high susceptibility among cattle indicates a potential vulnerability to the disease, highlighting the importance of preventive measures such as vaccination and biosecurity protocols to mitigate transmission. The decline in susceptible cattle over time suggests successful infection control measures or natural immunity development, but the

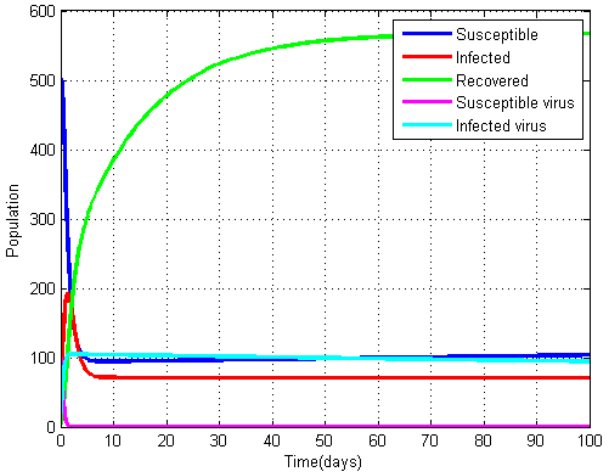

Figure 3: Dynamics of Avian Influenza transmission in cattle

subsequent rise in infected bird populations underscores the ongoing risk of disease transmission between species. This underscores the need for ongoing monitoring, surveillance, and targeted interventions to manage avian influenza dynamics effectively and minimize its impact on both cattle and bird populations.

4.3.2. Avian influenza infection rates in cattle populations with and without implemented interventions

This simulation compares those infection rates with and without implemented interventions for disease control. Figure 4, shows the infection rates trend over time. From

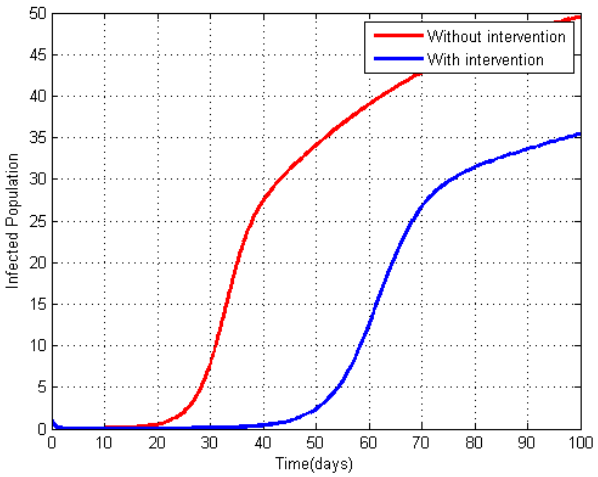

Figure 4: Dynamics of Avian Influenza in cattle with and without interventions

Figure 4, it shows that, the intervention reduces the infection rate, resulting in a significantly lower peak and overall number of infected cattle compared to the scenario without intervention. This implies that, that implementing the intervention effectively controls the spread of the disease, reducing the burden on veterinary resources and improving cattle health. This leads to fewer cattle deaths, lower treatment costs, and a reduced risk of transmission to other animals and potentially humans.

4.4. Effect of varying parameter values

This section involves performing simulation exercises where parameter values are adjusted to observe how state variables respond within the model framework. The focus is on identifying the most influential parameters.

4.4.1. Impact of varying natural death rate of avian influenza virus on infected population

This simulates the prevalence of infected cattle changes in response to varying levels of virus mortality. Figure 5 shows the trend. Figure 5, shows that, with higher μ_2 leads

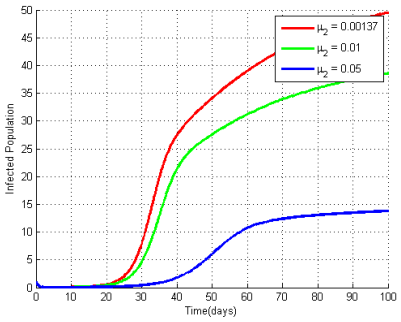

Figure 5: Impact of varying natural death rate of avian influenza virus on infected population

to a reduced infections of cattle and vice versa. Thus, increasing the natural death rate of the avian influenza virus through measures like environmental conservations, UV radiation, chemical disinfectants, competition among viral strains, host immune responses, and antiviral treatments can increase the natural death rate of avian influenza viruses which can effectively control disease transmission among cattle.

4.4.2. Effect of varying effective contact rate between susceptible cattle and infected virus

This simulates how the prevalence of infected cattle responds to changes in the effective contact rate (β_1) between susceptible cattle and infected virus. Figure 6 illustrates this relationship. Figure 6 illustrates that as the effective contact rate (β_1) between susceptible cattle and infected virus increases, the prevalence of infected cattle rises accordingly. This highlights the critical role of β_1 in influencing disease transmission dynamics among cattle populations. Adjusting β_1 through measures such as controlling animal interactions, implementing biosecurity practices, or modifying farming protocols can effectively mitigate disease spread and enhance livestock health management.

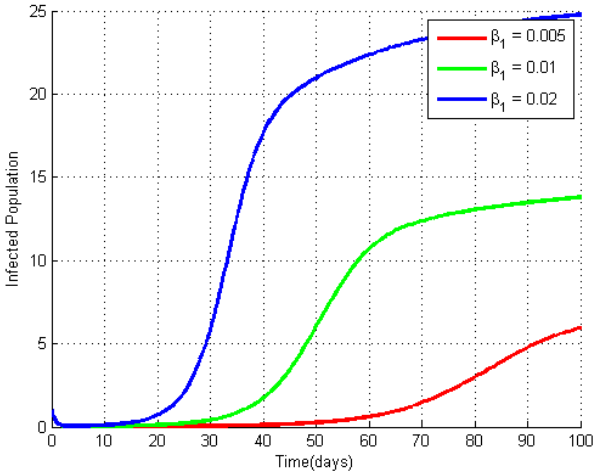

Figure 6: Effect of varying effective contact rate between susceptible cattle and infected virus

5. Conclusion

This paper aims to formulate and analyze a mathematical model for the transmission dynamics of avian influenza in cattle. Given the significant threat that avian influenza poses to cattle health and agricultural productivity, this study underscores the necessity of understanding the specific impacts of this disease on cattle populations, particularly given their economic importance. The disease-free equilibrium of the model was identified, and the basic reproduction number ($\mathcal{R}_0$) for avian influenza was calculated using the next-generation matrix method.

The findings from both analytical and numerical analyses suggest that increases in the effective contact rate between infected cattle and susceptible individuals (β_1) and the effective contact rate between infected birds and susceptible cattle (β_2) lead to an increased transmission rate of avian influenza in cattle, significantly impacting the population dynamics. Control measures such as vaccination, biosecurity protocols, and managing cattle population size can be implemented to mitigate disease spread, offering avenues for disease management and improving cattle health. Furthermore, sensitivity analysis shows that parameters such as the natural death rate of the virus (μ_2) and the recovery rate of infected cattle (α) play critical roles in decreasing the transmission rate of the disease in the population. The model can provide invaluable insights for agricultural authorities to forecast the implications of avian influenza transmission, examine its underlying determinants, and guide new control strategies.

Despite several important findings, our study can be enhanced by incorporating control strategies such as antiviral treatments, improved veterinary care, and the use of resistant cattle breeds in managing the transmission dynamics of avian influenza. Additionally, the study could benefit from integrating the influence of climatic conditions on the dynamics of avian influenza transmission in cattle. We also believe that considering spatial hetero-

geneity within cattle populations could yield more detailed insights.

Future studies can focus on analyzing the impact of control strategies such as antiviral treatments, biosecurity protocols, and the use of resistant cattle breeds on the transmission dynamics of avian influenza. Additionally, extending the model to incorporate the influence of climatic conditions, spatial heterogeneity within cattle populations, and seasonality on the dynamics of avian influenza transmission would be beneficial. Assessing the optimal control and cost-effectiveness of these strategies as future work would also provide valuable insights.

Acknowledgements

I extend my heartfelt appreciation to all those who made significant contributions to the successful completion of this project.

Data Availability

All data generated in this manuscript are included in the list of references.

Declaration of competing interest

The author declare that no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Funding

Authors did not receive any fund for this study.

References

[1] Diekmann, O., Heesterbeek, J. A. P., & Metz, J. A. J. (1990). On the definition and the computation of the basic reproduction ratio R0 in models for infectious diseases in heterogeneous populations. *Journal of Mathematical Biology*, 28, 365–382. Springer.

[2] Diekmann, O., Heesterbeek, J. A. P., & Roberts, M. G. (2010). The construction of next-generation matrices for compartmental epidemic models. *Journal of the Royal Society Interface*, 7(47), 873–885. The Royal Society.

[3] Castillo-Chavez, C., Blower, S., van den Driessche, P., Kirschner, D., & Yakubu, A.-A. (2002). *Mathematical Approaches for Emerging and Reemerging Infectious Diseases: Models, Methods, and Theory* (Vol. 126). Springer Science & Business Media.

[4] Chitnis, N., Hyman, J. M., & Cushing, J. M. (2008). Determining important parameters in the spread of malaria through the sensitivity analysis of a mathematical model. *Bulletin of Mathematical Biology*, 70, 1272–1296. Springer.

[5] World Organisation for Animal Health. (2024). AI (Avian Influenza). Retrieved from https://www.woah.org/fileadmin/Home/eng/Animal_Health_in_the_World/docs/pdf/2.03.04_AI.pdf. Accessed: 2024-06-06.

[6] Lupiani, B., & Reddy, S. M. (2009). The history of avian influenza. *Comparative Immunology, Microbiology and Infectious Diseases*, 32(4), 311–323. Elsevier.

[7] Zheng, T., Nie, L., Zhu, H., Luo, Y., & Teng, Z. (2022). Role of seasonality and spatial heterogeneous in the transmission dynamics of avian influenza. *Nonlinear Analysis: Real World Applications*, 67, 103567. Elsevier.

[8] Pinotti, F., Kohnle, L., Lourenço, J., Gupta, S., Hoque, M. A., Mahmud, R., Biswas, P., Pfeiffer, D., & Fournié, G. (2024). Modelling the transmission dynamics of H9N2 avian influenza viruses in a live bird market. *Nature Communications*, 15(1), 3494. Nature Publishing Group UK London.

[9] Subedi, D., Farhan, M. H. R., Niraula, A., Shrestha, P., Chandran, D., Acharya, K. P., & Ahmad, M. (2024). Avian Influenza in Low and Middle-Income Countries (LMICs): Outbreaks, Vaccination Challenges and Economic Impact. *Pakistan Veterinary Journal*, 44(1).

[10] Graziosi, G., Lupini, C., Catelli, E., & Carnaccini, S. (2024). Highly Pathogenic Avian Influenza (HPAI) H5 Clade 2.3.4.4 b Virus Infection in Birds and Mammals. *Animals*, 14(9), 1372. MDPI.

[11] Li, W., Guan, Y., Cao, J., & Xu, F. (2024). A note on global stability of a degenerate diffusion avian influenza model with seasonality and spatial Heterogeneity. *Applied Mathematics Letters*, 148, 108884. Elsevier.

[12] World Organisation for Animal Health. (2024). Avian Influenza. Retrieved from https://www.woah.org/en/disease/avian-influenza/. Accessed: 2024-06-06.

[13] Reperant, L. A., Rimmelzwaan, G. F., Kuiken, T., et al. (2009). Avian influenza viruses in mammals. *Revue scientifique et technique*, 28(1), 137.

[14] Root, J. J., & Shriner, S. A. (2020). Avian influenza A virus associations in wild, terrestrial mammals: A review of potential synanthropic vectors to poultry facilities. *Viruses*, 12(12), 1352. MDPI.

[15] ENETWILD Consortium, Flavia, O., Sascha, K., Carola, S.-L., Christoph, S., Valerie, A., Alina, A., Sophia, B., Hannes, B., Caroline, B., et al. (2024). The role of mammals in avian influenza: A review. *EFSA Supporting Publications*, 21(3), 8692E. Wiley Online Library.

[16] Zeng, J., Du, F., Xiao, L., Sun, H., Lu, L., Lei, W., Zheng, J., Wang, L., Shu, S., Li, Y., et al. (2024). Spatiotemporal genotype replacement of H5N8 avian influenza viruses contributed to H5N1 emergence in 2021/2022 panzootic. *Journal of Virology*, e01401–23. Am Soc Microbiol.

[17] Huang, M.-L., Wu, H.-D. I., & Chao, D.-Y. (2023). Approaches for Spatial and Temporal-Spatial Clustering Analysis in Avian Influenza Outbreaks. In *Earth Data Analytics for Planetary Health* (pp. 169–184). Springer.

[18] Tseng, I., Pan, B.-Y., Feng, Y.-C., & Fang, C.-T. (2024). Re-evaluating efficacy of vaccines against highly pathogenic avian influenza virus in poultry: a systematic review and meta-analysis. *One Health*, 100714. Elsevier.

[19] Lee, D.-H., Torchetti, M. K., Killian, M. L., Brown, I., & Swayne, D. E. (2024). Genome sequences of hemagglutinin cleavage site predict the pathogenicity phenotype of avian influenza virus: statistically validated data for facilitating rapid declarations and reducing reliance on in vivo testing. *Avian Pathology*, just-accepted, 1–42. Taylor & Francis.

[20] Lee, D.-H., Criado, M. F., & Swayne, D. E. (2021). Pathobiological origins and evolutionary history of highly pathogenic avian influenza viruses. *Cold Spring Harbor perspectives in medicine*, 11(2), a038679. Cold Spring Harbor Laboratory Press.

[21] Khan, Muhammad Altaf, Ullah, Saif, Khan, Yasir, & Farhan, Muhammad. (2020). Modeling and scientific computing for the transmission dynamics of avian influenza with half-saturated incidence. *International Journal of Modeling, Simulation, and Scientific Computing*, 11(04), 2050035. World Scientific.

[22] Centers for Disease Control and Prevention. (2023). Highly Pathogenic Avian Influenza A(H5N1) Virus: Technical Report December 2022. Retrieved from https://www.cdc.gov/flu/avianflu/spotlights/2022-2023/h5n1-technical-report_december.html. Accessed: 2024-06-06.

[23] Centers for Disease Control and Prevention. (2024). Avian Influenza A Viruses in Mammals. Retrieved from https://www.cdc.gov/flu/avianflu/mammals.htm. Accessed: 2024-06-06.

[24] Korobeinikov, A., & Wake, G. C. (2002). Lyapunov functions and global stability for SIR, SIRS, and SIS epidemiological models. *Applied Mathematics Letters*, 15(8), 955–960. Elsevier.

[25] Lou, J., Liang, W., Cao, L., Hu, I., Zhao, S., Chen, Z., Chan, R. W. Y., Cheung, P. P. H., Zheng, H., Liu, C., et al. (2024). Predictive evolutionary modelling for influenza virus by site-based dynamics of mutations. *Nature Communications*, 15(1), 2546. Nature Publishing Group UK London.

[26] Ali, A., Khan, S. U., Ali, I., & Khan, F. U. (2022). On dynamics of stochastic avian influenza model with asymptomatic carrier using spectral method. *Mathematical Methods in the Applied Sciences*, 45(13), 8230–8246. Wiley Online Library.

[27] Korobeinikov, A. (2004). Lyapunov functions and global properties for SEIR and SEIS epidemic models. *Mathematical medicine and biology: a journal of the IMA*, 21(2), 75–83. OUP.

[28] Vargas-De-León, C. (2009). Constructions of Lyapunov functions for classic SIS, SIR and SIRS epidemic models with variable population size. *Foro-Red-Mat: Revista electrónica de contenido matemático*, 26(5),

1–12.
[29] Malek, A., & Hoque, A. (2024). Mathematical modeling of the infectious spread and outbreak dynamics of avian influenza with seasonality transmission for chicken farms. *Comparative Immunology, Microbiology and Infectious Diseases*, 104, 102108. Elsevier.
[30] Delamater, P. L., Street, E. J., Leslie, T. F., Yang, Y. T., & Jacobsen, K. H. (2019). Complexity of the basic reproduction number (R0). *Emerging infectious diseases*, 25(1), 1. Centers for Disease Control and Prevention.
[31] Van den Driessche, P. (2017). Reproduction numbers of infectious disease models. *Infectious disease modelling*, 2(3), 288–303. Elsevier.
[32] Mfinanga, I. S., Shaban, N., & Marijani, T. (2024). Modelling Foot and Mouth Disease in the Context of Active Immigrants. *Journal of Applied Mathematics and Physics*, 12(1), 147–167. Scientific Research Publishing.
[33] Alhassan, C. J., & Achema, K. O. (2020). Modeling the transmission dynamics of an avian influenza: qualitative and quantitative analysis. *IOSR Journal of Mathematics (IOSR-JM)*, 16, 44–55.
[34] Tessema, K. M. (2019). Epidemiological modelling of foot and mouth disease control in cattle: incorporating time and spatial spread of disease dynamics. (Doctoral dissertation).
[35] Ndendya, J. Z., Leandry, L., & Kipingu, A. M. (2023). A next-generation matrix approach using Routh–Hurwitz criterion and quadratic Lyapunov function for modeling animal rabies with infective immigrants. *Healthcare Analytics*, 4, 100260. Elsevier.
[36] Gashirai, T. B., Musekwa-Hove, S. D., Lolika, P. O., & Mushayabasa, S. (2020). Global stability and optimal control analysis of a foot-and-mouth disease model with vaccine failure and environmental transmission. *Chaos, Solitons & Fractals*, 132, 109568. Elsevier.
[37] Kharis, M., et al. (2018). Mathematical modeling of Avian Influenza epidemic with bird vaccination in constant population. *Journal of Physics: Conference Series*, 983(1), 012116. IOP Publishing.
[38] Sofiane, K., Oubouskour, K., & Omar, B. (2024). Mathematical modeling and optimal control strategies to limit the spread of fowl pox in poultry. *Results in Control and Optimization*, 100428. Elsevier.
[39] Mushayabasa, S., Bhunu, C. P., & Dhlamini, M. (2011). Impact of vaccination and culling on controlling foot and mouth disease: a mathematical modelling approach. Scientific Research.
[40] Alexander, D. J. (2007). An overview of the epidemiology of avian influenza. *Vaccine*, 25(30), 5637–5644. Elsevier.
[41] Liu, J., Liang, Z., Sun, W., Hua, W., Huang, S., & Wen, F. (2024). The H4 subtype of avian influenza virus: a review of its historical evolution, global distribution, adaptive mutations and receptor binding properties. *Poultry Science*, 103913. Elsevier.
[42] Ly, H. (2024). Highly pathogenic avian influenza H5N1 virus infections of dairy cattle and livestock handlers in the United States of America. *Virulence*, 15(1), 2343931. Taylor & Francis.
[43] Garg, S. (2024). Outbreak of highly pathogenic avian influenza A (H5N1) viruses in US dairy cattle and detection of two human cases—United States, 2024. *MMWR. Morbidity and Mortality Weekly Report*, 73.
[44] Liang, Y. (2023). Pathogenicity and virulence of influenza. *Virulence*, 14(1), 2223057. Taylor & Francis.
[45] American Veterinary Medical Association. (2024). Avian Influenza Virus Type H5N1 in US Dairy Cattle. https://www.avma.org/resources-tools/animal-health-and-welfare/animal-health/avian-influenza/avian-influenza-virus-type-h5n1-us-dairy-cattle#:~:text=Common%20clinical%20signs%20in%20affected,the%20case%20for%20dairy%20cattle. Accessed: 2024-06-14.
[46] Malek, A., & Hoque, A. (2024). Mathematical modeling of the infectious spread and outbreak dynamics of avian influenza with seasonality transmission for chicken farms. *Comparative Immunology, Microbiology and Infectious Diseases*, 104, 102108. Elsevier.
[47] Li, J., & Nie, L. (2024). Global Dynamics Analysis of Non-Local Delayed Reaction-Diffusion Avian Influenza Model with Vaccination and Multiple Transmission Routes in the Spatial Heterogeneous Environment. *Qualitative Theory of Dynamical Systems*, 23(5), 1–42. Springer.
[48] Modnak, C. (2017). Mathematical modelling of an avian influenza: optimal control study for intervention strategies. *Appl. Math. Inf. Sci*, 11(4), 1049–1057.
[49] Gourram, H., Baroudi, M., Labzai, A., & Belam, M. (2023). Mathematical modeling and optimal control strategy for the influenza (H5N1). *Commun. Math. Biol. Neurosci.*, 2023, Article-ID.
[50] Alhassan, C. J., & Achema, K. O. (2020). Modeling the transmission dynamics of an avian influenza: qualitative and quantitative analysis. *IOSR Journal of Mathematics (IOSR-JM)*, 16, 44–55.
[51] Khan, M. A., Farhan, M., Islam, S., & Bonyah, E. (2019). Modeling the transmission dynamics of avian

influenza with saturation and psychological effect. *Discrete and Continuous Dynamical Systems-S*, 12(3), 455–474. Discrete and Continuous Dynamical Systems-S.

[52] Yang, S., Shen, F., & Zhang, Q. (2024). Sliding dynamics and optimal control of avian influenza model with saturated incidence rate. *Asian Journal of Control*, 26(3), 1178–1203. Wiley Online Library.

[53] Vizzotto Osowski, G., Castilho Duarte, S., Soares Ferreira Neto, J., Agnes Cardoso Côrtes, V., Schmidt, A. C., Acosta, A., Belleza Maciel, P., Silvestrini Machado, J., & Ferreira, F. (n.d.). Mathematical Model to Simulate the Spread of Avian Influenza in Santa Catarina, Brazil.

[54] Swayne, D. E., & Kapczynski, D. (2008). Avian influenza control strategies. *Avian influenza*, 444, 287–297. Blackwell Publishing, Ames, Iowa, USA.

[55] Yadav, M. P., Singh, R. K., & Malik, Y. S. (2020). Emerging and transboundary animal viral diseases: Perspectives and preparedness. *Emerging and transboundary animal viruses*, 1–25. Springer.

[56] Branda, F., Giovanetti, M., Scarpa, F., & Ciccozzi, M. (2024). Monitoring avian influenza in mammals with real-time data. *Pathogens and Global Health*, 1–5. Taylor & Francis.

[57] Kumbhar, S. M. (2024). Cow Health and Disease Management: Preventive Care and Treatment Approaches. *Revista Electronica de Veterinaria*, 25(1), 325–339.

[58] Sánchez-Cano, A., Camacho, M.-C., Ramiro, Y., & Cardona-Cabrera, T., & Höfle, U. (2024). Seasonal changes in bird communities on poultry farms and house sparrow—wild bird contacts revealed by camera trapping. *Frontiers in Veterinary Science*, 11, 1369779. Frontiers Media SA.

[59] European Food Safety Authority (EFSA), European Centre for Disease Prevention and Control (ECDC), Melidou, A., Enkirch, T., Willgert, K., Adlhoch, C., Alm, E., Lamb, F., Marangon, S., Monne, I., et al. (2024). Drivers for a pandemic due to avian influenza and options for One Health mitigation measures. *EFSA Journal*, 22(4), e8735. Wiley Online Library.

[60] Warren, C. J., Brookes, S. M., Arnold, M. E., Irvine, R. M., Hansen, R. D. E., Brown, I. H., Banyard, A. C., & Slomka, M. J. (2024). Assessment of Survival Kinetics for Emergent Highly Pathogenic Clade 2.3. 4.4 H5Nx Avian Influenza Viruses. *Viruses*, 16(6), 889. MDPI.

[61] Yadav, R. K. B., Chandra, A., Meena, T., Divkar, J. K., Vikas, Mishra, A., Pujari, R. N., Narsaiah, M. V. R., & Prabath, R. (2024). Radiation Emergency Preparedness and Response. In *Handbook on Radiation Environment, Volume 1: Sources, Applications and Policies* (pp. 531–579). Springer.