Ilorin Journal of Science
FAPSCON 2024 PROCEEDINGS, 2024, pp. 157 – 166 (Printed in Nigeria)
ISSN: 2408 – 4840 © 2024 Faculty of Physical Sciences, University of Ilorin
https://doi.org/10.54908/iljs.fapscon.2024.11.03.1021

ILORIN
JOURNAL OF SCEINCE

ILJS-24-071 (SPECIAL EDITION)

Dynamical Analysis of a Deterministic Bird-Human Avian Influenza Model

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Abstract

This article presents a transmission dynamics model and management of avian influenza. In order to analyze the effectiveness of this viral disease, that do occur from animal to human, vaccine, treatment and quarantine were used as intervention strategies. The qualitative properties of the model were examined with appropriate techniques using the Jacobian determinant, and the effective and basic reproductive ratios were computed using the next generation matrix. The bird population was classified into four groups, whereas there were seven divisions for people. To comprehend pandemic phenomena and how to regulate them, the dynamics of the model were examined. When effectively $R_c^{b,h} < 1$, the local stability analysis findings revealed that the DFE is locally asymptotically stable. The model, when simulated on different sets of parameter values, demonstrates that using the three controls together reduces the infection better than using either one or a pair of the controls.
Keywords: Avian influenza, deterministic model, basic reproductive number, equilibrium, asymptotic stability

1. Introduction

A highly infectious viral infection that mostly affects the respiratory system is avian influenza, generally known as the flu. Thousands of people perish worldwide because of this seasonal sickness, which has annual outbreaks. There are four different varieties of avian influenza viruses: P, Q, R, and S. P and Q are the ones that cause the seasonal flu epidemics in humans; R is extremely uncommon, and produces a mild respiratory disease, and is not known to generate epidemics; and S mostly affects cattle. As a result of its extreme severity, avian influenza infection has a mortality rate of around 60%, caused a great deal of hospitalizations, fatalities, and economic damage. Depending on the kind, the flu in birds might start showing signs and symptoms anywhere between two and seven days after infection.
In humans, influenza virus can cause mild to severe diseases and, in some cases, can result to death, according to 2012 report from the CDCP. All or part of the following symptoms are frequently experienced by those who have the flu, body pains, headaches, exhaustion (tiredness), and shortness of breath. The isolation and quarantine of infected individuals, as well as pharmacological treatments such as the injection of antivirals and vaccinations, can be used to prevent or suppress infection [1, 2]. Avoiding the causes of flu exposure is the greatest form of defense. Treatments may change depending on the type of avian flu present and the symptoms it produces. Avian influenza is a contagious illness that has caused severe problems for both bird and human life all over the world. Through the use of a mathematical model, [1, 2] investigate the dynamics of influenza transmission on the impacts of receiving vaccination effectively.

A key element in shaping the dynamics of influenza transmission is the recruitment of affected people. Their study revealed the situation in which the population does not accept additional infected people and the case in

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which the population does accept the inflow of infective individuals. The model takes into account immunizations that have been proven to be effective in preventing influenza from spreading across the population.
[1,2,5,13] considered Susceptible - Vaccinated - Infected - Treatment - Recovered ($S-V-\boldsymbol{I}-T-R$) model. Using six classes for the human population and three classes for the bird population, [8] developed a model of avian influenza on both people and birds. The R_h and R_b reproduction ratios and control measures for both humans and birds were calculated. In particular, the more the infectious population is confined, the better the recovery, according to their study's findings, which also noted that the impacts of vaccination and quarantine played critical roles in the disease's early recovery. The history, transmission, and methods for controlling the avian influenza epidemic must thus be further researched. In order to have a more reliable categorization, a larger range of classes is taken into account for avian influenza infection in this research. Additionally, by expanding the class of the current S I R in an open population to $S_b - V_b - E_b - I_b$ and $S_h - V_h - E_h - I_h - Q - T - R$, analytical and numerical techniques are used to address transmission dynamics and control of the flu transferred from bird populations to human populations with a deterministic model.

2. Mathematical Formulation of the Model

The dynamics and transmission of the avian influenza infection from birds to the human population are described in this section using a mathematical model. Incorporating the epidemiological characteristics that take into account both birds and humans, i.e., ($S_b - V_b - E_b - I_b$) and ($S_h - V_h - E_h - I_h - Q - T - R$).
The core S, I, and R model from Kermack (1927) is maintained, but the open population is taken into account by introducing recruitment into both human and avian species with extra classes like immunized, exposed, quarantined, and treated subclasses. Then, the model includes three controls (vaccination, isolation, and therapy). $N_h(t) = S_h - V_h - E_h - I_h - Q - T - R$ and $N_b(t) = S_b - V_b - E_b - I_b$ are used to signify the populations of both humans and birds at any given period (t). Keeping in mind that when a person comes into touch with an infected bird or consumes undercooked bird, the virus starts to grow within the cells of the affected area of the bird.
Figure 1 depicts the suggested avian influenza model's schematic flow diagram while taking the aforementioned factors into account.

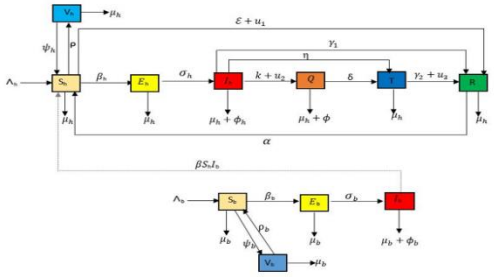


Figure 1: Flowchart of Avian Influenza transmission model with intervention

The shattered line between the infected bird class, I_b and the susceptible human population, S_h, shows that the avian viral infection was transferred from the sick bird population to susceptible people. The dynamics of an avian influenza infection with a variety of treatments, under the assumptions and descriptions shown in the flowchart in Figure 1 and is the new deterministic model:

$$\left.\begin{aligned}
\frac{dS_h}{dt} &= \Lambda_h - \beta S_h I_b - \beta_h S_h I_h - (\mu_h + \rho + \varepsilon + u_1)S_h + \alpha R + \psi_h V_h,\\
\frac{dV_h}{dt} &= \rho S_h - (\psi h + \mu_h)V_h,\\
\frac{dE_h}{dt} &= \beta S_h I_b + \beta_h S_h I_h - (\mu_h + \sigma_h)E_h,\\
\frac{dI_h}{dt} &= \sigma_h E_h - (k + u_2 + \gamma_1 + \eta + \mu_h + \phi_h)I_h\\
\frac{dQ}{dt} &= (k + u_2)I_h - (\delta + \mu_h + \phi)Q,\\
\frac{dT}{dt} &= \eta I_h + \delta Q - (\mu_h + \gamma_2 + u_3)T,\\
\frac{dR}{dt} &= (\gamma_2 + u_3)T + \gamma_1 I_h + (\varepsilon + u_1)Sh - (\mu_h + \alpha)R\\
\frac{dS_b}{dt} &= \wedge_b - \beta_b S_b I_b - (\mu_b + \rho_b)S_b + \psi_b V_b,\\
\frac{dV_b}{dt} &= \rho_b S_b - (\psi_b + \mu_b)V_b,\\
\frac{dE_b}{dt} &= \beta_b S_b I_b - (\sigma_b + \mu_b)E_b,\\
\frac{dI_b}{dt} &= \sigma_b E_b - (\mu_b + \phi_b)I_b.
\end{aligned}\right\} \quad (2.1)$$

3. Qualitative Analysis of the Model

The autonomous model approach is given below:

3.1 Dynamics of populations N(t)

In order to define the human population (N_h),

$$N_h(t) = S_h - V_h - E_h - I_h - Q - T - R, \quad (3.1)$$

Additionally, the definition of the bird population $N_b(t)$ is

$$N_b(t) = S_b - V_b - E_b - I_b \quad (3.2)$$

where the letters S_b, V_b, $E_b$and I_b stand for susceptible, immunized, exposed and infected birds, respectively.

Derivatives of (3.1) and (3.2) result in

$$\frac{dN_h}{dt} = \frac{dS_h}{dt} + \frac{dV_h}{dt} + \frac{dE_h}{dt} + \frac{dI_h}{dt} + \frac{dQ}{dt} + \frac{dT}{dt} + \frac{dR}{dt}, \quad (3.3)$$

and

$$\frac{dN_b}{dt} = \frac{dS_b}{dt} + \frac{dV_b}{dt} + \frac{dE_b}{dt} + \frac{dI_b}{dt}. \quad (3.4)$$

Substituting (2.1) into (3.3) after simplification, we have

$$\frac{dN_h}{dt} = \Lambda_h - \mu_h(S_h - V_h - E_h - I_h - Q - T - R) - \phi_h I_h - \phi Q \quad (3.5)$$

and on substituting (2.1) into (3.4), yields

$$\frac{dN_b}{dt} = \Lambda_b - \mu_b(S_b - V_b - E_b - I_b) - \phi_b I_b. \quad (3.6)$$

Finally, (3.5) and (3.6) can be expressed respectively in the form of population dynamics:

$$\frac{dN_h}{dt} = \Lambda_h - \mu_h N_h - \phi_h I_h - \phi Q, \quad (3.7)$$

and

$$\frac{dN_b}{dt} = \Lambda_b - \mu_b N_b - \phi_b I_b. \quad (3.8)$$

The Model's Solutions' Boundedness and Positivity

To generate the standard for the positivity of solutions and the well-posedness of the system, the fundamental characteristics of the model system (2.1) are used in this section.

3.1.1 **Boundedness of the model**

The model's viability, which represents the area where the model's solution (2.1) is examined due to its biological significance.

Theorem 3.1 Let the solution **Ω** of the model (2.1) with starting conditions in $\Re_+^{11}$ (set of vectors with eleven non-negative components), for which (3.7) and (3.8) hold, approach and remain in the solution's domain as $t \to \infty$. A positively invariant set provided is thus the model's workable solution.

$$\Omega = \Omega_h \times \Omega_b = \left\{(S_h, V_h, E_h, I_h, Q, T, R, S_b, V_b, E_b, I_b) \in \Re_+^{11} : N_h(t) \leq \frac{\Lambda_h}{\mu_h}, N_b(t) \leq \frac{\Lambda_b}{\mu_b}\right\}.$$

Proof.

In the absence of the disease (i.e., $\phi_h = \phi = 0$), (3.7) reduces to

$$\frac{dN_h}{dt} = \Lambda_h - \mu_h N_h. \tag{3.9}$$

Applying Lemma 2 in Birkhoff and Rota (1989; pg. 27) on (3.9), leads to

$$\frac{dN_h}{dt} = \Lambda_h - \mu_h N_h(t) - \phi_h I_h - \phi Q \leq \Lambda_h - \mu_h N_h(t) \tag{3.10}$$

Thus,

$$\frac{dN_h}{dt} \leq \Lambda_h - \mu_h N_h(t), \tag{3.11}$$

Solving inequality (3.11) by integrating factor (IF) approach, yields

$$N_h(t) \leq N_h(0)e^{-\mu_h t} + \frac{\Lambda_h}{\mu_h}(1 - e^{-\mu_h t}). \tag{3.12}$$

As $t\ approaches\ infinity$, approach $\frac{\Lambda_h}{\mu_h}$

$$N_h(t) \leq \frac{\Lambda_h}{\mu_h}. \tag{3.13}$$

This means that $0 \leq N_h \leq \frac{\Lambda_h}{\mu_h}$, which denotes that the model (2.1) trajectories are limited.

As a result, the region receives all practical solutions for the human population of the model system (2.1).

$$\Omega_h = \left\{(S_h, V_h, E_h, I_h, Q, T, R) \in \Re_+^7 : N_h(t) \leq \frac{\Lambda_h}{\mu_h}\right\}.$$

Similarly, the case of the bird population in (2.1) is bounded as $0 \leq N_b \leq \frac{\Lambda_b}{\mu_b}$.

Thus, the feasible solutions of the bird population only enters the region

$$\Omega_b = \left\{(S_b, V_b, E_b, I_b) \in \Re_+^4 : N_b(t) \leq \frac{\Lambda_b}{\mu_b}\right\},$$

Hence, the feasible solution set for the system (2.1) is given by

$$\Omega = \left\{(S_h, V_h, E_h, I_h, Q, T, R, S_b, V_b, E_b, I_b) \in \Re_+^{11} : N_h(t) \leq \frac{\Lambda_h}{\mu_h}, N_b(t) \leq \frac{\Lambda_b}{\mu_b}\right\}.$$

Furthermore, whenever $N_h > \frac{\Lambda_h}{\mu_h}$, then by (3.9), $\frac{dN_h}{dt} < 0$. Similarly, whenever $N_b > \frac{\Lambda_b}{\mu_b}$, then $\frac{dN_b}{dt} < 0$. The host population is asymptotically stable. Thus, $\Re_+^{11}$ in the region Ω for $t > 0$, and its positively invariant. Hence, the system of equation (2.1) is epidemiologically meaningful and mathematically well-posed in the domain, Ω.

3.1.2 **Positivity of solutions**

Definition 3.1: If all of the state variables in the model assume non-negative values, the solution to model (2.1) is said to be positive. The model in (2.1) must be shown to have non-negative state variables for all time t in order for it to have epidemiological significance. It must be demonstrated that model (2.1) solutions with positive beginning data continue to be positive for all times t > 0. The outcome is displayed below.

Theorem 3.2 : Let the initial value of the system in (2.1) be $\{(S_h(0), V_h(0), E_h(0), I_h(0), Q(0), T(0), R(0), S_b(0), V_b(0), E_b(0), I_b(0))\} \in \Omega$.

Then, the solution set $\{S_h(t), V_h(t), E_h(t), I_h(t), Q(t), T(t), R(t), S_b(t), V_b(t), E_b(t), I_b(t)\}$ of (2.1) is positive for all $t > 0$.

Proof. The first equation in (2.1), that is,

$$\frac{dS_h}{dt} = \Lambda_h - \beta S_h I_b - \beta_h S_h I_h - (\mu_h + \rho + \varepsilon + u_1)S_h + \alpha R + \psi_h V_h.$$

$$\text{for } \beta \in [0,1] \text{ and } \beta_h \leq \frac{\Lambda_h}{S_h I_h}$$

Thus,

$$\int \frac{1}{S_h} dS_h \geq -(\mu_h + \rho + \varepsilon + u_1)dt,$$

$$S_h(t) \geq Ae^{-(\mu_h + \rho + \varepsilon + u_1)t}, \text{ where A is a constant}$$

Setting t = 0 and applying the initial conditions, yield

$$S_h(t) \geq S_h(0)e^{-(\mu_h + \rho + \varepsilon + u_1)t} \geq 0, \text{since } (\mu_h + \rho + \varepsilon + u_1) > 0. \quad (3.14)$$

Hence, S_h is always positive for $t > 0$.

Other state variables in (2.1) are obtained as above

This shows that all the state variables are positive for all $t > 0$. Hence, the proof of Theorem 3.2 is completed.

3.3. Disease-free equilibrium state

When no one is infected or the illness has been completely eradicated, the situation is known as the disease-free stability. After solving system (2.1) simultaneously, we obtained

$$\mathbb{E}^+_{b,h}(S_h^+, V_h^+, E_h^+, I_h^+, Q^+, T^+, R^+, S_b^+, V_b^+, E_b^+, I_b^+) = \left\{ \begin{matrix} \frac{(\psi h + \mu_h)(\mu_h + \alpha)\Lambda_h}{\mu_h[(\mu_h + \alpha + \varepsilon + u_1)(\mu_h + \psi_h) + \rho(\mu + \alpha)]}, \frac{\Lambda_h(\mu_h + \alpha)}{\mu_h[(\mu_h + \alpha + \varepsilon + u_1)(\mu_h + \psi_h) + \rho(\mu + \alpha)]}, 0,0,0, \\ , 0, \frac{\Lambda_h(\varepsilon + u_1)(\psi_h + \mu_h)}{\mu_h[(\mu_h + \alpha + \varepsilon + u_1)(\mu_h + \psi_h) + \rho(\mu + \alpha)]}, \frac{\Lambda_h(\psi_b + \mu_b)}{\mu_b(\psi_b + \mu_b + \rho_b)}, \frac{\Lambda_b \rho_b}{\mu_b(\psi_b + \mu_b + \rho_b)}, 0,0 \end{matrix} \right\}. \quad (3.15)$$

3.4. Basic Reproductive Number

This study employs the next generation approach, and the (R_0) which is basic reproductive number, that is, the average number of secondary infections lifespan. The differential equations related to the compartments E_h, I_h, Q, T, E_b and I_b given below are used to derive the effective reproduction number ${R_c}^{b,h}$. Figure 1 shows the rate of new infection function $(\mathcal{F}_i)$, and the transfer rate (V_i). The system (2.1) starting with the infected compartments E_h, I_h, Q, T, E_b and I_b, then the uninfected classes S_h, V_h, R, S_b and V_b. The next generation matrix method is employed to determine the rate of emergence of a new infection in compartments E_h and E_b.

$$\mathcal{F}_i = \begin{pmatrix} \beta S_h I_b + \beta_h S_h I_h \\ 0 \\ 0 \\ 0 \\ \beta_b S_b I_b \\ 0 \end{pmatrix}, \qquad F = \begin{pmatrix} 0 & \beta_h S_h & 0 & 0 & 0 & \beta S_b \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \beta_b S_b \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}. \quad (3.16)$$

$$V_i = \begin{pmatrix} (\mu_h + \sigma_h)E_h \\ -\sigma_h E_h + (k + u_2 + \gamma_1 + \eta + \mu_h + \phi_h)I_h \\ -(k + u_2)I_h + (\delta + \mu_h + \phi)Q \\ -\eta I_h - \delta Q + (\mu_h + \gamma_2 + u_3)T \\ (\sigma_b + \mu_b)E_b \\ -\sigma_b E_b + (\mu_b + \phi_b)I_b \end{pmatrix},$$

$$V = \begin{pmatrix} (\mu_h + \sigma_h) & 0 & 0 & 0 & 0 & 0 \\ -\sigma_h & (k + u_2 + \gamma_1 + \eta + \mu_h + \phi_h) & 0 & 0 & 0 & 0 \\ 0 & -(k + u_2) & (\delta + \mu_h + \phi) & 0 & 0 & 0 \\ 0 & -\eta & -\delta & (\mu_h + \gamma_2 + u_3) & 0 & 0 \\ 0 & 0 & 0 & 0 & (\sigma_b + \mu_b) & 0 \\ 0 & 0 & 0 & 0 & -\sigma_b & (\mu_b + \phi_b) \end{pmatrix}$$

Solving FV^{-1}, the effective reproductive number for bird and human population are obtained as

$R_c^b = \frac{\beta_b \Lambda_b \sigma_b (\psi_b + \mu_b)}{\mu_b (\psi_b + \mu_b + \rho_b)(\mu_b + \phi_b)(\sigma_b + \mu_b)}$ and $R_c^h = \frac{\beta_h \Lambda_h \sigma_h (\psi h + \mu_h)(\mu_h + \alpha)}{\mu_h [(\mu_h + \alpha + \varepsilon + u_1)(\mu_h + \psi_h) + \rho(\mu + \alpha)] \mu_h (\mu_h + \sigma_h)(k + u_2 + \gamma_1 + \eta + \mu_h + \phi_h)}$

Hence, the interface of effective reproductive number for both bird and human population is obtained as

$$R_c^{b,h} = R_c^b \, . R_c^h,$$

where, $$R_c^{b,h} = \frac{\beta_b \Lambda_b \sigma_b \beta_h \Lambda_h \sigma_h (\psi_b + \mu_b)(\psi h + \mu_h)(\mu_h + \alpha)}{\mu_b (\psi_b + \mu_b + \rho_b)(\mu_b + \phi_b)(\sigma_b + \mu_b) D} \quad (3.17)$$

and

$$D = \mu_h [(\mu_h + \alpha + \varepsilon + u_1)(\mu_h + \psi_h) + \rho(\mu + \alpha)] \mu_h (\mu_h + \sigma_h)(k + u_2 + \gamma_1 + \eta + \mu_h + \phi_h).$$

From (3.17), the basic reproduction number when there is no control, implies that $c = 0$, is obtained below

$$R_0^{b,h} = \frac{\beta_b \Lambda_b \sigma_b \beta_h \Lambda_h \sigma_h (\psi_b + \mu_b)(\psi h + \mu_h)(\mu_h + \alpha)}{\mu_b (\psi_b + \mu_b + \rho_b)(\mu_b + \phi_b)(\sigma_b + \mu_b) \mu_h [(\mu_h + \alpha + \varepsilon)(\mu_h + \psi_h) + \rho(\mu + \alpha)] \mu_h (\mu_h + \sigma_h)(k + \gamma_1 + \eta + \mu_h + \phi_h)} \quad (3.18)$$

Remarks 3.3.1: Epidemiologically,

(i) if $R_0 < 1$, the disease's prevalence will decline and ultimately vanish;

(ii) if $R_0 = 1$, there will always be cases of the sickness;

(iii) if $R_0 > 1$, the sickness will continue to spread frequently.

3.4.2 Endemic-equilibrium state

When a disease cannot be completely eliminated but still exists in the population, it is said to be in an endemic equilibrium. Then, none of the state variables in (2.1) may disappear, i.e.

$\mathbb{E}_{b,h}^* = (S_h^*, V_h^*, E_h^*, I_h^*, Q^*, T^*, R^*, S_b^*, V_b^*, E_b^*, I_b^*) \Omega_{b,h}^* \neq (0,0,0,0,0,0,0,0,0,0,0)$.

The solution to (2.1) at **EE** is obtained in terms of reproductive ratio.

With, $S_b^* = \frac{S_b^+}{R_c^b}$ and $S_h^* = \frac{S_h^+}{R_c^h}$, where, $S_b^+ = \frac{\Lambda_b (\psi_b + \mu_b)}{\mu_b (\psi_b + \mu_b + \rho_b)}$, $R_c^b = \frac{\beta_b \Lambda_b \sigma_b (\psi_b + \mu_b)}{\mu_b (\psi_b + \mu_b + \rho_b)(\mu_b + \phi_b)(\sigma_b + \mu_b)}$, $S_h^+ = \frac{(\psi h + \mu_h)(\mu_h + \alpha)\Lambda_h}{\mu_h [(\mu_h + \alpha + \varepsilon + u_1)(\mu_h + \psi_h) + \rho(\mu + \alpha)]}$, and $R_c^h = \frac{\beta_h \Lambda_h \sigma_h (\psi h + \mu_h)(\mu_h + \alpha)}{\mu_h [(\mu_h + \alpha + \varepsilon + u_1)(\mu_h + \psi_h) + \rho(\mu + \alpha)] \mu_h (\mu_h + \sigma_h)(k + u_2 + \gamma_1 + \eta + \mu_h + \phi_h)}$

After solving the system (3.34) simultaneously, the result obtained is given as:

$$
\begin{cases}
S_h^* = \frac{\Lambda_h(\psi_h+\mu_h)(\mu_h+\alpha)}{\mu_h[(\mu_h+\alpha+\varepsilon+u_1)(\mu_h+\psi_h)+\rho_b(\mu_h+\alpha)]R_c^h}, \\
V_h^* = \frac{\Lambda_h\rho(\mu_h+\alpha)}{\mu_h[(\mu_h+\alpha+\varepsilon+u_1)(\mu_h+\psi_h)+\rho_b(\mu_h+\alpha)]R_c^h}, \\
E_h^* = \frac{\beta\Lambda_h\mu_b(\psi_h+\mu_h)(\mu_h+\alpha)(\psi_b+\mu_b+\rho_b)[\![R]\!]_c^b-1][A+\beta_h\sigma_h\Lambda_h(\psi_h+\mu_h)(\mu_h+\alpha)]}{\beta_b(\psi_b+\mu_b)(\mu_h+\sigma_h)BA} \\
I_h^* = \frac{\sigma_h\beta\Lambda_h\mu_b(\psi_h+\mu_h)(\mu_h+\alpha)(\psi_b+\mu_b+\rho_b)[\![R]\!]_c^b-1]}{\beta_b(\psi_b+\mu_b)[AR_c^h-\Lambda_h\sigma_h\beta_h(\psi_h+\mu_h)(\mu_h+\alpha)]} \\
T^* = \frac{\sigma_h\beta\Lambda_h\mu_b(\psi_h+\mu_h)(\mu_h+\alpha)(\psi_b+\mu_b+\rho_b)[\![R]\!]_c^b-1][\eta(\delta+\mu_h+\phi)+\delta(k+u_2)]}{\beta_b(\mu_h+\gamma_2+u_3)(\delta+\mu_h+\phi)(\psi_b+\mu_b)[AR_c^h-\Lambda_h\sigma_h\beta_h(\psi_h+\mu_h)(\mu_h+\alpha)]}, \\
Q^* = \frac{\sigma_h\beta\Lambda_h\mu_b(k+u_2)(\psi_h+\mu_h)(\mu_h+\alpha)(\psi_b+\mu_b+\rho_b)[\![R]\!]_c^b-1]}{\beta_b(\delta+\mu_h+\phi)(\psi_b+\mu_b)[AR_c^h-\Lambda_h\sigma_h\beta_h(\psi_h+\mu_h)(\mu_h+\alpha)]}, \\
R^* = \frac{B(\gamma_2+u_3)\sigma_h\beta\Lambda_h\mu_b(\psi_h+\mu_h)(\psi_b+\mu_b+\rho_b)[\![R]\!]_c^b-1]C+(\mu_h+\gamma_2+u_3)(\delta+\mu_h+\phi)\,A[B+(\psi_b+\mu_b)]}{\beta_b(\mu_h+\gamma_2+u_3)(\delta+\mu_h+\phi)(\psi_b+\mu_b)A\Lambda_h(\varepsilon+u_1)(\psi_h+\mu_h)}, \\
S_b^* = \frac{\Lambda_b(\psi_b+\mu_b)}{\mu_b(\psi_b+\mu_b+\rho_b)R_c^b}, \\
V_b^* = \frac{\rho_b\Lambda_b}{\mu_b(\psi_b+\mu_b+\rho_b)R_c^b}, \\
E_b^* = \frac{\mu_b(\mu_b+\phi_b)(\psi_b+\mu_b+\rho_b)[R_c^b-1]}{\beta_b\sigma_b(\psi_b+\mu_b)}, \\
I_b^* = \frac{\mu_b(\psi_b+\mu_b+\rho_b)[R_c^b-1]}{\beta_b(\psi_b+\mu_b)},
\end{cases}
\tag{3.19}
$$

Where, $A = [\mu_h(\mu_h + \sigma_h)(k + u_2 + \gamma_1 + \eta + \mu_h + \phi_h)[(\mu_h + \alpha + \varepsilon + u_1)(\mu_h + \psi_h) + \rho(\mu_h + \alpha)]R_c^h - \Lambda_h\sigma_h\beta_h(\psi_h + \mu_h)(\mu_h + \alpha)]$,

$B = \mu_h[(\mu_h + \alpha + \varepsilon + u_1)(\mu_h + \psi_h) + \rho(\mu_h + \alpha)]R_c^h, \quad C = [\eta(\delta + \mu_h + \phi) + \delta(k + u_2)].$

Thus, if $R_c^b > 1$ and $R_c^h > 1$, then $I_h^* > 0$ and $I_b^* > 0$, then the model (2.1) has a unique endemic-equilibrium point given by $\mathbb{E}^* = (S_h^*, V_h^*, E_h^*, I_h^*, Q^*, T^*, R^*, S_b^*, V_b^*, E_b^*, I_b^*)$, where in the presence of infection ($I_h \neq 0$). Therefore, to ensure the existence of a positive endemic-equilibrium, it suffice that $R_c^{b,h} > 1$. Since $S_h^*, V_h^*, E_h^*, I_h^*, Q^*, T^*, R^*, S_b^*, V_b^*, E_b^*, I_b^* > 0$ (when $R_c^{b,h} > 1$), the endemic-equilibrium $\mathbb{E}^*$ is positive and $I_{b,h}^* > 0$. This complete the proof of equation (2.1).

3.5 Local Stability of DFE State

The behaviour of the model population close to the equilibrium point is examined to ascertain whether or not the conditions necessary for the disease-free equilibrium state to be stable and, the disease to be completely eradicated from the entire population.

To determine the stability or otherwise of the system (2.1) at the disease-free equilibrium point $\mathbb{E}^+$, the behaviour of the model population near the equilibrium point is examined to determine the satisfaction of the conditions that must be met for the disease-free equilibrium state to be stable and at the same time for the disease to be totally eradicated from the population.

Theorem 3.3 : The disease-free equilibrium point $\mathbb{E}^+$ is locally asymptotically stable if $R_c^{b,h} < 1$ for $tr(J_{\mathbb{E}^+}) < 0$ and $det(J_{\mathbb{E}^+}) > 0$, and unstable if $R_c^{b,h} > 1$ for $tr(J_{\mathbb{E}^+}) \geq 0$ and $det(J_{\mathbb{E}^+}) < 0$.

Proof. Following equation (2.1) and assessing the model at the DFE point $\mathbb{E}^+$. At the disease-free equilibrium point $\mathbb{E}^+$, the Jacobian matrix

$$J_{\mathbb{E}^+} = \begin{pmatrix} -\mathrm{A}_1 & \psi_h & 0 & -L & 0 & 0 & \alpha & 0 & 0 & 0 & -P \\ \rho & -\mathrm{A}_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -\mathrm{A}_3 & L & 0 & 0 & 0 & 0 & 0 & 0 & P \\ 0 & 0 & \sigma_h & -\mathrm{A}_4 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \mathrm{A}_5 & -\mathrm{A}_6 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \eta & \delta & -\mathrm{A}_7 & 0 & 0 & 0 & 0 & 0 \\ \mathrm{A}_8 & 0 & 0 & \gamma_1 & 0 & \mathrm{A}_9 & -\mathrm{A}_{10} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\mathrm{B}_1 & \psi_b & 0 & -M \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \rho_b & -\mathrm{B}_2 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\mathrm{B}_3 & M \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \sigma_b & -\mathrm{B}_4 \end{pmatrix}, \quad (3.20)$$

where, $\mathrm{A}_1 = (\mu_h + \rho + \varepsilon + u_1)$, $\mathrm{A}_2 = (\psi_h + \mu_h)$, $\mathrm{A}_3 = (\mu_h + \sigma_h)$, $\mathrm{A}_4 = (k + u_2 + \gamma_1 + \eta + \mu_h + \phi_h)$, $\mathrm{A}_5 = (k + u_2)$, $\mathrm{A}_6 = (\delta + \mu_h + \phi)$, $\mathrm{A}_7 = (\mu_h + \gamma_2 + u_3)$, $\mathrm{A}_8 = (\varepsilon + u_1)$, $\mathrm{A}_9 = (\gamma_2 + u_3)$, $\mathrm{A}_{10} = (\mu_h + \alpha)$, $\mathrm{B}_1 = (\mu_b + \rho_b)$, $\mathrm{B}_2 = (\psi_b + \mu_b)$, $\mathrm{B}_3 = (\sigma_b + \mu_b)$, $B_4 = (\mu_b + \phi_b)$, $L = \frac{C_1}{C_2}$, $P = \frac{C_3}{C_2}$, $\mathrm{M} = \frac{d_1}{d_2}$, $C_1 = \beta_h \Lambda_h (\psi h + \mu_h)(\mu_h + \alpha)$, $C_2 = \mu_h[(\mu_h + \alpha + \varepsilon + u_1)(\mu_h + \psi_h) + \rho(\mu + \alpha)]$, $C_3 = \beta \Lambda_h (\psi h + \mu_h)(\mu_h + \alpha)$, $d_1 = \beta_b \Lambda_b (\psi_b + \mu_b)$, $d_2 = \mu_b (\psi_b + \mu_b + \rho_b)$.

Matrix $J_{\mathbb{E}^+}$ in (3.20) is stable if $tr(J_{\mathbb{E}^+}) < 0$ and $det(J_{\mathbb{E}^+}) \geq 0$. The trace of matrix $J_{\mathbb{E}^+}$ is obtained as:

$$tr(J_{\mathbb{E}^+}) = -(\mathrm{A}_1 + \mathrm{A}_2 + \mathrm{A}_3 + \mathrm{A}_4 + \mathrm{A}_6 + \mathrm{A}_7 + \mathrm{A}_{10} + \mathrm{B}_1 + \mathrm{B}_2 + \mathrm{B}_3 + \mathrm{B}_4)$$

$$\therefore tr(J_{\mathbb{E}^+}) < 0$$

Also, the determinant of matrix $J_{\mathbb{E}^+}$ is generated as

$$detJ_{\mathbb{E}^+} = A_6 A_7 A_3 A_4 B_2 B_1 (B_2 B_1 - \rho_b \psi_b)(A_{10} \rho \psi_h + A_2 A_8 \alpha - A_{10} A_2 A_1)(1 + R_c^h)(1 - R_c^b)$$

Thus, $detJ_{\mathbb{E}^+} > 0$ provided $B_2 B_1 > \rho_b \psi_b$, $A_{10} A_2 A_1 < (A_{10} \rho \psi_h + A_2 A_8 \alpha)$, and $R_c^{b.h} < 1$.

Consequently, the outcome implies that the sickness will eventually disappear biologically.

4. Numerical Simulation and Analysis

Maple, Matlab, and Julia software of a fourth-order Runge − Kutta scheme were employed for the analysis of the results. The objective of the work is to numerically validate the analytical findings previously made for the model (2.1).

The validation were carried out in Table 1. The value of $R_c^{b.h}$ is calculated using the values of the vaccination control (u_1), quarantine control (u_2), and treatment control (u_3). The simulation graphs, which are shown for different values of the three critical parameters u_1, u_2, and u_3, are shown in Figures 2 to 6.

Table 1: Parameter values of new avian influenza model

Symbol	Description	Estimated Value
Λ_h	Rate of recruiting individuals	1000
Λ_b	Rate of bird recruitment	30
β_h	Efficacy of human-to-human interaction	0.1025
β_b	Effective contact rate between birds	0.012
β	Effective human-bird contact rate	0.2
ε	gain in immunity	0.7
σ_h	Rate of transformation from exposed human compartment	0.05
σ_b	Rate of transition from the exposed bird compartment	0.85
γ_1	Natural recovery rate of infected people	0.3
γ_2	Human recovery rate as a result of therapy	0.7

ϕ_h	Avian influenza mortality rate in humans	0.05
ϕ_b	A bird's chance of dying from avian influenza	0.5
ϕ	Death rate as a result of quarantining sick people	0.01
μ_h	Humans from each class's rate of mortality due to natural causes	0.1
μ_b	Ratio of birds from each class that die naturally	0.033
α	Loss of immunity frequency	0.08333
ρ	Percentage of vulnerable people who must get immunized	0.8
ρ_b	Proportion of birds to be vaccinated	0.95
ψ_h	Rate of decline of human vaccine-based immunity	0.002
ψ_b	Progression rate from the infectious class to the quarantine	0.003
k	Vaccination effectiveness rate	0.6
u_1	Vaccination effectiveness rate	[0 - 1.0]
u_2	Quarantine effectiveness	[0 - 1.0]

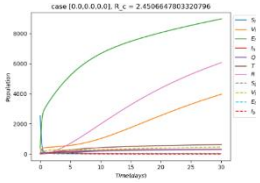
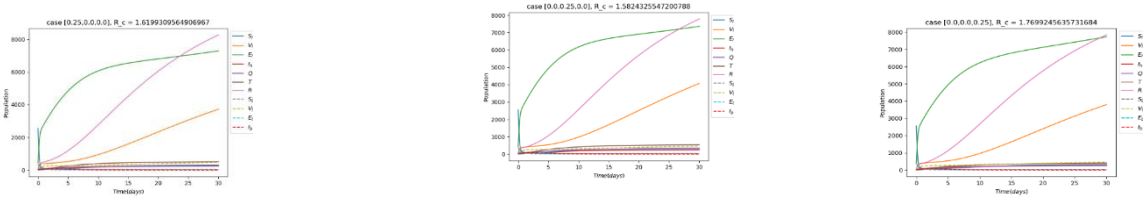


Figure 2: Graph of the state variables with time using controls $u_1 = 0.0$, $u_2 = 0.0$ and $u_3 = 0.0$; $R_0 = 2.4507 > 1$. Figure 2 shows that if none of the controls is applied against the spread of avian influenza, the disease will persists.



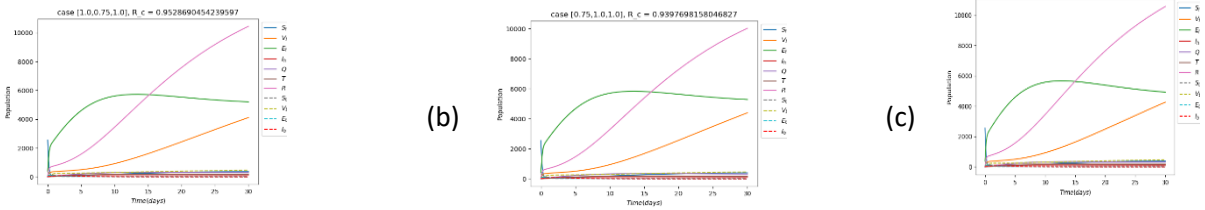
(a) (b)

(c)

Figure 3: Graph of state variables with time using controls: (a) $u_1 = 0.75, u_2 = 0.0$ and $u_3 = 0.0$; $R_0 = 1.3852 > 1$, (b) $u_1 = 0.0, u_2 = 0.75$ and $u_3 = 0.0$; $R_0 = 1.3058 > 1$ and (c) $u_1 = 0.0, u_2 = 0.0$ and $u_3 = 0.75$; $R_0 = 1.7699 > 1$.

Figure 3 (a), (b) and (c) show that if the rate of applying only one of the controls is at 75%, the disease will still continue to spread.

(a)



(b) (c)

Figure 4: Plot showing variation in the state variables with time using controls: (a) $u_1 = 0.75$,u_2= 1.0 and $u_3 = 1.0$;$R_0 = 0.9398 < 1$, (b) $u_1 = 1.0, u_2 = 0.75$ and $u_3 = 1.0$;$R_0 = 0.9529 < 1$ and (c) $u_1 = 1.0, u_2 = 1.0$ and $u_3 = 1.0$; $R_0 = 0.8763 < 1$

Figure 4 (a), (b) and (c) show that the disease will be eradicated totally from the population if one of the three controls is applied for a minimum of 75% rate and the

remaining at 100%.

5. conclusion

In the present study, a deterministic human-bird avian influenza model in an open population is developed and evaluated to understand the dynamics of the disease's transmission from birds to people while taking infection controls like vaccination, isolation, and treatment into account. The model took into account by adding new members to the vulnerable class through birth and immigration, and those newcomers who are exposed to the disease can be added to the exposed class. In order to evaluate the possible effects of these techniques on the dynamics of the disease's transmission, the preventative strategies (such as immunization, quarantine, and therapy) are incorporated in the model.

It was determined that if all the controls are put in place, the infection will decline and the disease will disappear. To help with comprehension of the scenarios, the numerical outcomes are graphically depicted. According to the findings, combining vaccination and quarantine measures is the most efficient way to cut down on disease-related issues. Therefore, a lot of focus needs to be placed on the previously outlined ways to totally eradicate illness, and then therapy should come next to take care of the affected individuals.

Acknowledgement

The authors of this paper gratefully acknowledge the contributions Dr. B.A. Peter of the Department of Mathematics and Statistics, Kampala International University, Kampala, Uganda, made to enrich the paper, while he was on research visits to the University of Ilorin, under the sponsorship of the Third World Academy of Science (TWAS).

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