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A Model Analysis for the Transmission Dynamics of Avian Influenza

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Abstract

This paper examines the transmission dynamics of avian influenza. A nonlinear mathematical model for the problem is formulated and analysed. For the prevalence of the disease and the ease of analysis, we considered the model in proportions of susceptible, infectious, isolated and recovered compartments. The basic reproduction number was computed and used to prove the stability of the disease free equilibrium states. It is proved that the basic reproduction number is a decreasing function of the culling rate of infected birds. It is further proved that the disease free equilibrium state is locally asymptotically stable whenever the basic reproduction number is less than unity.

Key words: Avian influenza, Mathematical model, Basic reproduction number. Disease free equilibrium

1.0: Introduction

Avian influenza or "bird-flu" (also called influenza A virus) is a virus that infects wild birds (such as ducks, gulls and shore birds) and domestic poultry (such as chickens, turkey, ducks and geese). In recent times the term bird-flu has been used to describe the H5N1 avian influenza virus that occurs mainly in birds, and can be deadly to them (Alexander, 2000; Arora and Arora, 2008)

Infected birds shed influenza virus in their saliva, nasal secretions and faeces. Susceptible birds become infected when they have contact with contaminated secretions or excretions with surfaces that are contaminated by infected birds (De Jong and Hien, 2006). Fecal – to - oral transmission is the most common mode of spread between birds.

Highly pathogenic avian influenza can be spread from birds to people as a result of extensive direct contact with nasal discharge or fecal droppings in infected birds (The Writing Committee of the World Health Org. (WHO) Consultation on Human Influenza A/H5, 2006). Highly pathogenic avian influenza virus subtype H5N1 has severely affected poultry populations in Southeast Asia since 2003. Initial outbreaks were confined to Indonesia, Vietnam, Thailand, Cambodia and China. However from late July 2005, the highly pathogenic virus spread in a north east direction, causing out breaks in wild birds and poultry in eastern, central and western Russia, Mongolia and Central Kazakhstan (The Writing Committee of the World Health Org. (WHO) Consultation on Human Influenza A/H5, 2006).

In February, 2006, highly pathogenic avian influenza virus of the H5N1 subtype was detected in chickens in Kaduna State in Northern Nigeria, the first African country reporting a confirmed highly pathogenic avian influenza (H5N1) outbreak (Monne *et al*, 2008; Fusaro *et al*, 2009; WHO, 2006). According to Monne *et al* (2008), by the end of February, 2006, local laboratory tests had detected the virus in seven contiguous states in the north and central parts of the country (Kaduna, Kano, Plateau, Katsina, Bauchi, Yobe and Nasarawa) and the Federal Capital Territory of Abuja.

Since avian influenza virus is highly contagious and easily spread, the most common method of control is the culling of the infected flocks. Another method is the quarantine of affected areas until the disease is no longer present. While vaccination is possible and has been tested on a small scale, it is not widely considered a viable control method. The virus can also be killed by common disinfectants or heat (WHO, 2004; Le Menach *et al*, 2006). Persons recovering from natural infection according to Todar (2008) acquire some resistance to re infection with the particular antigenic strain. Bodewes *et al* (2010) also asserted that the induction of antibodies

of proper specificity will afford strain – specific protection and this strain specific immunity can be very long lasting.

A number of mathematical models both deterministic and stochastic have been used to predict the world wide spread of pandemic influenza and for comparing interventions aimed at preventing and controlling avian influenza. See for example, Ferguson *et al*, (2005); Derouich and Boutayeb (2008) and Srinivasa (2008).

Okosun and Yusuf (2007); Iwani *et al*(2007); Derouich and Bontayeb (2008) presented various mathematical models for avian influenza (H5N1). These models does not explicitly take into account any control measures.

Using the data from the avian influenza epidemic in the Netherlands, LeMenach *et al* (2006) analysed a spartial farm-based model, which treats poultry farms as units, and found that an immediate depopulation of infected flock following an accurate and quick diagnosis would have a greater impact than simply depopulating surrounding flocks.

Ferguson *et al* (2005) used a simulation model of influenza transmission in Southeast Asia to evaluate the potential effectiveness of targeted mass prophylactic use of antiviral drugs as a containment strategy.

On the same note, Longini *et al* (2005) used a stochastic influenza simulation model for rural Southeast Asia to investigate the effectiveness of targeted antiviral prophylaxis, quarantine and pre – vaccination in containing an emerging influenza strain at the source.

Although many of mathematical modelling studies tend to emphasize the use of pharmaceutical interventions, it could be useful to carry out modelling studies that focus on non – pharmaceutical intervention such as culling of infected birds and isolation of humans with symptoms.

The main aim of this study is to build on the model by Okosun and Yusuf (2007), by incorporating the dynamics of wild and domestic birds, culling of infected birds and the isolation of infected individuals with avian influenza strain. The paper is organized as follows, in Section 2, we derive a model consisting of ordinary differential equations (ODE) that describes the interaction between birds and human population and the underlying assumptions. In Section 3, we compute the basic reproduction number and use it to establish the local stability of the disease free equilibrium states. Our conclusions are discussed in Section 4.

2.0 Model Formulation

In describing the new model we subdivide the total avian (birds) population at time *t*, denoted by $N_B(t)$ into susceptible wild birds, $S_W(t)$, susceptible domestic birds, $S_D(t)$, infected wild birds, $I_W(t)$, and infected domestic birds, $I_D(t)$, so that

$$N_B(t) = S_W(t) + S_D(t) + I_W(t) + I_D(t).$$

In the human population, we assume that humans infected with avian influenza cannot infect susceptible humans. Thus the total human population at time *t*, denoted by $N_H(t)$ is sub-divided into susceptible humans, $S_H(t)$, infected humans, $I_H(t)$, isolated infected humans, $Q_H(t)$, and recovered humans, $R_H(t)$, so that

$$N_H(t) = S_H(t) + I_H(t) + Q_H(t) + R_H(t)$$

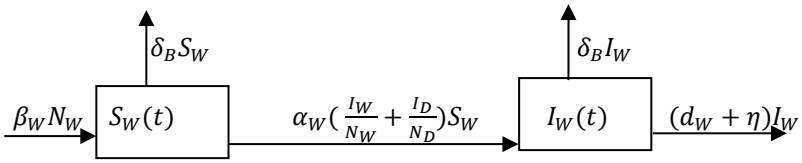
The variables and parameters used in the model are defined in Table 1.

Table 1: Variables and Parameters used in the model and their description

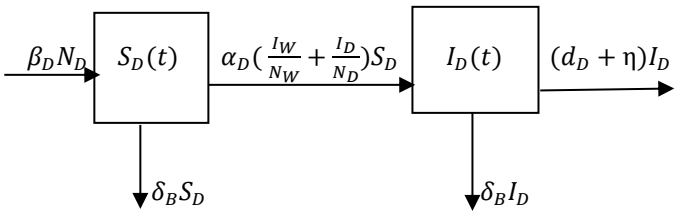
Variable/Parameter	Description
$N_W(t)$	Total number of wild birds at time t
$N_D(t)$	Total number of domestic birds at time t
$N_H(t)$	Total number of humans at time t
$S_W(t)$	Total number of Susceptible wild birds at time t
$I_W(t)$	Total number of Infected wild birds at time t
$S_D(t)$	Total number of Susceptible domestic birds at time t
$I_D(t)$	Total number of Infected domestic birds at time t
$S_H(t)$	Total number of Susceptible humans at time t
$I_D(t)$	Total number of Infected humans at time t
$Q_H(t)$	Total number of Isolated humans with avian strain at time t
$R_H(t)$	Total number of Recovered humans at time t
β_W	Average birth rate in wild birds
β_D	Average birth rate in domestic birds
$\alpha_W, \alpha_D, \alpha_A$	Infection transmission rates for birds
η	Destruction (culling) rate for infected birds
δ_B	Natural death rate in birds
d_w	Flu induced death rate in wild birds
d_D	Flu induced death rate in domestic birds
β_H	Average birth rate in humans
δ_H	Natural death rate in humans
d_H	Flu induced death rate in humans
ε_H	Isolation rate for humans with avian stain
ϑ_H	Flu induced death rate in Isolated humans ($\vartheta_H < d_H$)
v	Recovery rate without immunity
γ	Recovery rate with substantial immunity
σ	Loose of immunity rate in recovered humans

A schematic flow diagram of the extended model for the birds' population and human population is shown in Figure 1 below.

Wild Birds



Domestic Birds



Humans

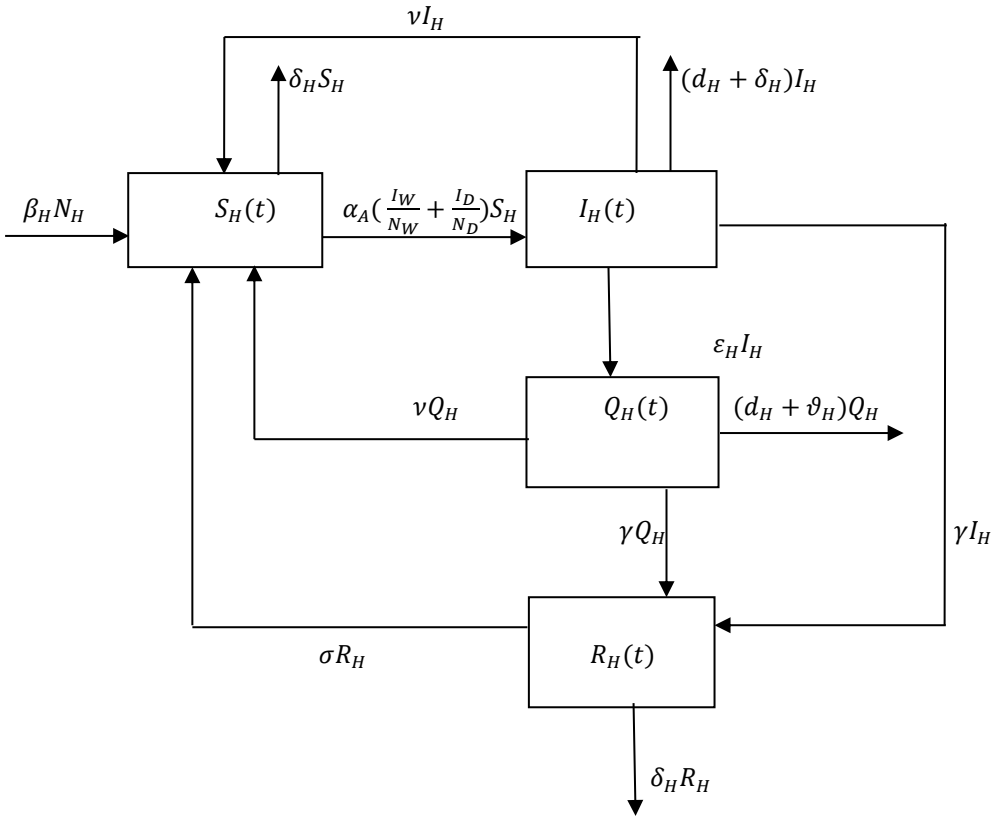


Figure 1: Schematic diagram of the transmission dynamics of avian influenza (H5N1) in birds and human population.

2.1 Susceptible and Infected Wild Birds

The population of susceptible wild birds is generated by birth of wild birds (at the rate β_W). It is reduced by infection, following contact with infected wild birds and infected domestic birds (at the rate α_W), where α_W is the infection transmission rate for wild birds and further reduced by natural death (at the rate δ_B). Hence

$$\frac{dS_W}{dt} = \beta_W N_W - \alpha_W \frac{I_W}{N_W} S_W - \alpha_W \frac{I_D}{N_D} S_W - \delta_B S_W,$$

$$= \beta_W N_W - \alpha_W \left(\frac{I_W}{N_W} + \frac{I_D}{N_D}\right) S_W - \delta_B S_W,$$

The population of infected wild birds is increased through the infection of susceptible wild birds following contact with infected wild birds and infected domestic birds. It decreased either by natural death (at the rate δ_B) and avian induced mortality (at the rate d_W) and by culling of infected wild birds (at the rate η). So that

$$\frac{dI_W}{dt} = \alpha_W \left(\frac{I_W}{N_W} + \frac{I_D}{N_D}\right) S_W - (d_W + \delta_B + \eta) I_W,$$

2.2 Susceptible and Infected Domestic Birds

The population of susceptible domestic birds is generated by birth of domestic birds (at the rate β_D). It is reduced by infection, following contact with infected wild birds and infected domestic birds (at the rate α_D), where α_D is the infection transmission rate for domestic birds and further reduced by natural death (at the rate δ_B). Thus

$$\frac{dS_D}{dt} = \beta_D N_D - \alpha_D \frac{I_W}{N_W} S_D - \alpha_D \frac{I_D}{N_D} S_D - \delta_B S_D,$$

$$= \beta_D N_D - \alpha_D \left(\frac{I_W}{N_W} + \frac{I_D}{N_D}\right) S_D - \delta_B S_D,$$

The population of infected domestic birds is increased through the infection of susceptible domestic birds following contact with infected wild birds and infected domestic birds. It decreased either by natural death (at the rate δ_B) and avian induced mortality (at the rate d_D) and by culling of infected wild birds (at the rate η). This yield

$$\frac{dI_D}{dt} = \alpha_D \left(\frac{I_W}{N_W} + \frac{I_D}{N_D}\right) S_D - (d_D + \delta_D + \eta) I_D,$$

2.3 Susceptible, Infected, Isolated and Recovered Humans

The population of susceptible humans are increased by birth (at the rate β_H), recovered humans who lost immunity to return to susceptible humans (at the rate σ), recovered infected and isolated humans without immunity (at the rate ν). It decreased by infection of susceptible humans following contact with infected wild birds and infected domestic birds (at the rate α_B), where α_B is the infection transmission rate for humans and further reduced by natural death (at the rate δ_H). This gives

$$\frac{dS_H}{dt} = \beta_H N_H - \alpha_B \left(\frac{I_W}{N_W} + \frac{I_D}{N_D}\right) S_H - \delta_H S_H + \nu I_H + \nu Q_H + \sigma R_H,$$

Infected humans are generated through infection of susceptible humans following contact with infected wild birds and infected domestic birds (at the rate α_B) and reduced by natural death (at the rate δ_H) and avian induced mortality (at the rate d_D). It is further reduced by isolation of infected humans (at the rate ε_H) recovered infected humans without immunity and with substantial immunity (at the rate ν and γ respectively). Thus,

$$\frac{dI_H}{dt} = \alpha_B \left(\frac{I_W}{N_W} + \frac{I_D}{N_D}\right) S_H - (\varepsilon_H + d_H + \delta_H + \nu + \gamma) I_H,$$

Isolated humans are generated by isolation of infected humans (at the rate ε_H) and reduced by natural death (at the rate δ_H) and avian induced mortality (at the rate ϑ_H where, $\vartheta_H < d_H$; it is assumed that isolated individuals are given some treatment such as Tamiflu). It is further reduced by recovered isolated humans without immunity and those with substantial immunity (at the rate ν and γ respectively). Hence,

$$\frac{dQ_H}{dt} = \varepsilon_H I_H - (\nu + \vartheta_H + \gamma + \delta_H) Q_H,$$

The recovered humans are generated by the recovery of infected humans and isolated humans (at the rate γ). Decreased by natural death (at the rate δ_H) and losing immunity (at the rate σ). So that

$$\frac{dR_H}{dt} = \gamma I_H + \gamma Q_H - (\sigma + \delta_H) R_H,$$

The above assumptions and derivations leads to the following system of ordinary differential equations

$$\frac{dS_W}{dt} = \beta_W N_W - \alpha_W \left(\frac{I_W}{N_W} + \frac{I_D}{N_D}\right) S_W - \delta_B S_W, \quad (1)$$

$$\frac{dI_W}{dt} = \alpha_W \left(\frac{I_W}{N_W} + \frac{I_D}{N_D}\right) S_W - (d_W + \delta_B + \eta) I_W, \quad (2)$$

$$\frac{dS_D}{dt} = \beta_D N_D - \alpha_D \left(\frac{I_W}{N_W} + \frac{I_D}{N_D}\right) S_D - \delta_B S_D, \quad (3)$$

$$\frac{dI_D}{dt} = \alpha_D\left(\frac{I_W}{N_W}+\frac{I_D}{N_D}\right)S_D - (d_D+\delta_D+\eta)I_D, \quad (4)$$

$$\frac{dS_H}{dt} = \beta_H N_H - \alpha_B\left(\frac{I_W}{N_W}+\frac{I_D}{N_D}\right)S_H - \delta_H S_H + \nu I_H + \nu Q_H + \sigma R_H, \quad (5)$$

$$\frac{dI_H}{dt} = \alpha_B\left(\frac{I_W}{N_W}+\frac{I_D}{N_D}\right)S_H - (\varepsilon_H + d_H + \delta_H + \nu + \gamma)I_H, \quad (6)$$

$$\frac{dQ_H}{dt} = \varepsilon_H I_H - (\nu + \vartheta_H + \gamma + \delta_H)Q_H, \quad (7)$$

$$\frac{dR_H}{dt} = \gamma I_H + \gamma Q_H - (\sigma + \delta_H)R_H, \quad (8)$$

For prevalence of the disease, it is necessary to consider the model in proportions of susceptible, infectious, isolated and recovered compartments.

Adding equations (1) - (2) and equations (3) – (4) gives

$$\frac{dN_W}{dt} = \beta_W N_W - \delta_B N_W - (d_w+\eta)I_W \quad (9)$$

and

$$\frac{dN_D}{dt} = \beta_D N_D - \delta_B N_D - (d_D+\eta)I_D \quad (10)$$

Similarly, adding equations (5) – (8) gives the rate of change of the total human population:

$$\frac{dN_H}{dt} = \beta_H N_H - \delta_H N_H - d_H I_H - \vartheta_H Q_H \quad (11)$$

We now define the proportion for each class as follows:

$$s_w = \frac{S_W}{N_W},\ i_W = \frac{I_W}{N_W}, s_D = \frac{S_D}{N_D},\ i_D = \frac{I_D}{N_D}, s_H = \frac{S_H}{N_H}, i_H = \frac{I_H}{N_H}, q_H = \frac{Q_H}{N_H}, r_H = \frac{R_H}{N_H},$$

So that

$$s_W + i_W = 1 \Rightarrow s_W = 1 - i_W, s_D + i_D = 1 \Rightarrow s_D = 1 - i_D$$

and

$$s_H + i_H + q_H + r_H = 1 \Rightarrow s_H = 1 - i_H - q_H - r_H$$

Thus, the system (1) – (8) expressed in proportion is given below:

$$\frac{ds_W}{dt} = \beta_W - \alpha_W(i_W+i_D)s_W - \beta_W s_W + (d_w+\eta)i_W s_W \quad (12)$$

$$\frac{di_W}{dt} = \alpha_W(i_W+i_D)s_w - (d_W+\beta_W+\eta)i_W + (d_w+\eta)i_W^2 \quad (13)$$

$$\frac{ds_D}{dt} = \beta_D - \alpha_D(i_W+i_D)s_D - \beta_D s_D + (d_D+\eta)i_D s_D \quad (13)$$

$$\frac{di_D}{dt} = \alpha_D(i_W+i_D)s_D - (d_D+\beta_D+\eta)i_D + (d_D+\eta)i_D^2 \quad (14)$$

$$\frac{ds_H}{dt} = \beta_H - \alpha_B(i_W+i_D)s_H + v(i_H+q_H) + \sigma r_H - \beta_H s_H + d_H s_H i_H + \vartheta_H s_H q_H \quad (15)$$

$$\frac{di_H}{dt} = \alpha_B(i_W+i_D)s_H - (\varepsilon + d_H + \beta_H + v + \gamma)i_H + \vartheta_H i_H q_H + d_H i_H^2 \quad (16)$$

$$\frac{dq_H}{dt} = \varepsilon_H i_H - (v + \vartheta_H + \gamma + \beta_H)q_H + d_H i_H q_H + \vartheta_H q_H^2 \quad (17)$$

$$\frac{dr_H}{dt} = \gamma i_H + \gamma q_H - (\sigma + \beta_H)r_H + d_H i_H r_H + \vartheta_H q_H r_H \quad (18)`$$

The system (12) – (18) can be reduced further by setting

$$s_W = 1 - i_W, s_D = 1 - i_D \text{ and } s_H = 1 - i_H - q_H - r_H$$

$$\frac{di_W}{dt} = \alpha_W(i_W + i_D)(1 - i_W) - (d_W + \beta_W + \eta)i_W + (d_w + \eta)i_W^2 \quad (19)$$

$$\frac{di_D}{dt} = \alpha_D(i_W + i_D)(1 - i_D) - (d_D + \beta_D + \eta)i_D + (d_D + \eta)i_D^2 \quad (20)$$

$$\frac{di_H}{dt} = \alpha_B(i_W + i_D)(1 - i_H - q_H - r_H) - (\varepsilon + d_H + \beta_H + v + \gamma)i_H + \vartheta_H i_H q_H + d_H i_H^2 \quad (21)$$

$$\frac{dq_H}{dt} = \varepsilon_H i_H - (v + \vartheta_H + \gamma + \beta_H)q_H + d_H i_H q_H + \vartheta_H q_H^2 \quad (22)$$

$$\frac{dr_H}{dt} = \gamma i_H + \gamma q_H - (\sigma + \beta_H)r_H + d_H i_H r_H + \vartheta_H q_H r_H \quad (23)`$$

These are the governing equations of the model.

3.0 Model Analysis

The nonlinrar system (12) – (18) will be analysed so as to find the conditions for the existence and stability of the disease free equilibrium states (DFEs). To achieve this, we will compute the Basic Reproduction number and use it to determine if the disease can be eliminated from the population or not.

3.1 Invariant Region

The avian influenza model (12) – (18) in proportions will be analyzed to establish the biological feasible region as follows. The system (12) – (18) is split into two parts, namely the avian population where $n_B(t) = s_w(t) + i_W(t) + s_D(t) + i_D(t)$and the human population where $n_H(t) = S_H(t) + i_H(t) + q_H(t) + r_H(t)$.

Consider the feasible region

$$\Omega = \Omega_B \cup \Omega_H \subset \mathbb{R}_+^4 \times \mathbb{R}_+^4$$

with

$$\Omega_B = \{(s_w, i_W, s_D, i_D) \in \mathbb{R}_+^4 : s_w + i_W + s_D + i_D \leq 1\}$$

and

$$\Omega_H = \{(s_H, i_H, q_H, r_H) \in \mathbb{R}_+^4 : s_H + i_H + q_H + r_H \leq 1\}$$

The following steps are followed to establish the positive invariance of Ω (i.e., the solution in Ω remain inΩ for all $t > 0$). The rate of change of the avian and human population (by adding the first four equations and the last four equations of the model (12) – (18)) is given

$$\frac{dn_B}{dt} = \beta_B - \beta_B n_B + (d_w + \eta)i_W s_W - (d_W + \eta)i_W + (d_w + \eta)i_W^2 +$$

$$(d_D +)i_D s_D - (d_D + \eta)i_D + (d_D + \eta)i_D^2 \quad (24)$$

and

$$\frac{dn_H}{dt} = \beta_H - \beta_H n_H + d_H s_H i_H + \vartheta_H s_H q_H - \beta_H i_H + \vartheta_H i_H q_H + d_H i_H^2 - \beta_H q_H + d_H i_H q_H + \vartheta_H q_H^2 - \beta_H r_H +$$

$$d_H i_H r_H + \vartheta_H q_H r_H \quad (25)$$

It follows from (24) and (25) that

$\frac{dn_B}{dt} \leq \beta_B - \beta_B N_B$ (26)

and

$\frac{dn_H}{dt} \leq \beta_H - \beta_H n_H$ (27)

Integrating (26) with respect to t where the integrating factor, $IF = e^{\int \beta_B dt} = e^{\beta_B t}$ we have

$e^{\beta_B t} n_B \leq \int \beta_B e^{\beta_B t} dt + C$

$\Rightarrow e^{\beta_B t} n_B \leq e^{\beta_B t} + C$

$\therefore n_B(t) \leq 1 + Ce^{-\beta_B t}$

At $t = 0$,

$C = n_B(0) - 1$

$\therefore n_B(t) \leq 1 + (n_B(0) - 1)e^{-\beta_B t}$

$n_B(t) \leq n_B(0)e^{-\beta_B t} + 1 - e^{-\beta_B t}$ (28)

Also integrating (27) with respect to t where the integrating factor, $IF = e^{\int \beta_H dt} = e^{\beta_H t}$ we have

$e^{\beta_H t} n_H \leq \int \beta_H e^{\beta_H t} dt + C$

$\Rightarrow e^{\beta_H t} n_H \leq e^{\beta_H t} + C$

$n_H(t) \leq 1 + Ce^{-\beta_H t}$

At $t = 0$,

$C = n_H(0) - 1$

$\therefore n_H(t) \leq 1 + (n_H(0) - 1)e^{-\beta_H t}$

$n_H(t) \leq n_H(0)e^{-\beta_H t} + 1 - e^{-\beta_H t}$ (29)

Applying the theorem of differential inequality (Birkhof and Rota, 1982) on equations (28) and (29) we obtain

$0 \leq n_B(t) \leq 1$ and $0 \leq n_H(t) \leq 1$ as $t \to \infty$

Thus, the region Ω is positively invariant. Hence it is sufficient to consider the dynamics of the flow generated by (12) – (18) in Ω. In this region, the model can be considered as being epidemiologically and mathematically well posed. Thus every solution of the model (12) – (18) with initial conditions in Ω remains in Ω for all $t > 0$. This result is summarized below.

Lemma1: The region $\Omega = \Omega_B \cup \Omega_H \subset \mathbb{R}_+^4 \times \mathbb{R}_+^4$ is positively invariant for the basic model (12) – (20) with non-negative initial conditions in $\mathbb{R}_+^8$.

3.2 Computation of The Basic Reproduction Number, R_0

The model in proportion given by equations (19) – (23) has a unique disease – free equilibrium state $\mathcal{E}_0 = (i_W, i_D, i_H, q_H, r_H) = (0,0,0,0,0)$ obtained by setting $i_W = 0, i_D = 0, i_H = 0, q_H = 0, r_H = 0$.

To compute the basic reproduction number, we rewrite the model equation which contribute to the transmission of infection, in this case the i_W, i_D and i_H classes. Thereafter write down matrix of infection rates F_i and the transition rate matrix V_i which represents rates of appearance of new infections into infective class and the transfer of individuals into and out of this class by all other means respectively.
The rate of appearance of new infection in compartments i_W, i_D and i_H are given by

$$F(x) = \begin{pmatrix} \alpha_W(i_W - i_W^2 + i_D - i_W i_D) \\ \alpha_D(i_W - i_W i_D + i_D - i_D^2) \\ \alpha_B(i_W + i_D)(1 - i_H - q_H - r_H) \end{pmatrix}.$$

While the remaining transfer terms in compartments i_W, i_D and i_H are given by

$$V(x) = \begin{pmatrix} (d_W + \beta_W + \eta)i_W - (d_w + \eta)i_W^2 \\ (d_D + \beta_D + \eta)i_D - (d_D + \eta)i_D^2 \\ (\varepsilon + d_H + \beta_H + v + \gamma)i_H - \vartheta_H i_H q_H + d_H i_H^2 \end{pmatrix}.$$

Taking partial derivatives of $F(x)$ with respect to i_W, i_D and i_H at the disease – free equilibrium state $\mathcal{E}_0 = (i_W, i_D, i_H, q_H, r_H) = (0,0,0,0,0)$, to obtain

$$F_x(\mathcal{E}_0) = \begin{pmatrix} \alpha_w & \alpha_w & 0 \\ \alpha_D & \alpha_D & 0 \\ \alpha_B & \alpha_B & 0 \end{pmatrix}.$$

Similarly the matrix of partial derivatives of $V(x)$ at the disease – free equilibrium state $\mathcal{E}_0 = (i_W, i_D, i_H, q_H, r_H) = (0,0,0,0,0)$ is given by

$$V_x(\mathcal{E}_0) = \begin{pmatrix} d_W + \beta_W + \eta & 0 & 0 \\ 0 & d_D + \beta_D + \eta & 0 \\ 0 & 0 & \varepsilon + d_H + \beta_H + v + \gamma \end{pmatrix}.$$

and

$$V_x^{-1}(\mathcal{E}_0) = \begin{pmatrix} \frac{1}{d_W + \beta_W + \eta} & 0 & 0 \\ 0 & \frac{1}{d_D + \beta_D + \eta} & 0 \\ 0 & 0 & \frac{1}{\varepsilon + d_H + \beta_H + v + \gamma} \end{pmatrix}.$$

Then

$$F_x(\mathcal{E}_0)V_x^{-1}(\mathcal{E}_0) = \begin{pmatrix} \frac{\alpha_w}{d_W + \beta_W + \eta} & \frac{\alpha_w}{d_D + \beta_D + \eta} & 0 \\ \frac{\alpha_D}{d_W + \beta_W + \eta} & \frac{\alpha_D}{d_D + \beta_D + \eta} & 0 \\ \frac{\alpha_B}{d_W + \beta_W + \eta} & \frac{\alpha_B}{d_D + \beta_D + \eta} & 0 \end{pmatrix}.$$

The eigenvalues are determined by solving the characteristic equation

$$\det(F_x(\mathcal{E}_0)V_x^{-1}(\mathcal{E}_0) - \lambda) = 0$$

$$det\begin{pmatrix} \frac{\alpha_w}{d_W+\beta_W+\eta}-\lambda & \frac{\alpha_w}{d_D+\beta_D+\eta} & 0 \\ \frac{\alpha_D}{d_W+\beta_W+\eta} & \frac{\alpha_D}{d_D+\beta_D+\eta}-\lambda & 0 \\ \frac{\alpha_B}{d_W+\beta_W+\eta} & \frac{\alpha_B}{d_D+\beta_D+\eta} & 0-\lambda \end{pmatrix}=0$$

That is

$$(0-\lambda)\left[\left(\frac{\alpha_w}{d_W+\beta_W+\eta}-\lambda\right)\left(\frac{\alpha_D}{d_D+\beta_D+\eta}-\lambda\right)-\frac{\alpha_D}{d_W+\beta_W+\eta}\frac{\alpha_w}{d_D+\beta_D+\eta}\right]=0$$

$$(0-\lambda)\left[\lambda^2-\left(\frac{\alpha_w}{d_W+\beta_W+\eta}+\frac{\alpha_D}{d_D+\beta_D+\eta}\right)\lambda\right]=0$$

$$(0-\lambda)\lambda\left[\lambda-\left(\frac{\alpha_w}{d_W+\beta_W+\eta}+\frac{\alpha_D}{d_D+\beta_D+\eta}\right)\right]=0$$

$\therefore\ \lambda=0$ or $\lambda=\frac{\alpha_w}{d_W+\beta_W+\eta}+\frac{\alpha_D}{d_D+\beta_D+\eta}$

The maximum eigenvalue of $F_x(\mathcal{E}_0){V_x}^{-1}(\mathcal{E}_0)$ is given as:

$$\lambda=\frac{\alpha_w}{d_W+\beta_W+\eta}+\frac{\alpha_D}{d_D+\beta_D+\eta}$$

Thus, the basic reproduction number is given as:

$$R_0=\frac{\alpha_w}{d_W+\beta_W+\eta}+\frac{\alpha_D}{d_D+\beta_D+\eta} \qquad (30)$$

This leads us to the following result.

Proposition 1: R_0 is a strictly decreasing function of $\eta\ \epsilon\ (0,1)$.

Proof

Taking the partial derivative of R_0 with respect to η (0,1) to obtain

$$\frac{\partial R_o}{\partial \eta}=-\left(\frac{\alpha_D}{(d_D+\beta_D+\eta)^2}+\frac{\alpha_W}{(d_W+\beta_W+\eta)^2}\right)$$

Therefore R_0 is a strictly decreasing function of $\eta\ \epsilon\ (0,1)$.

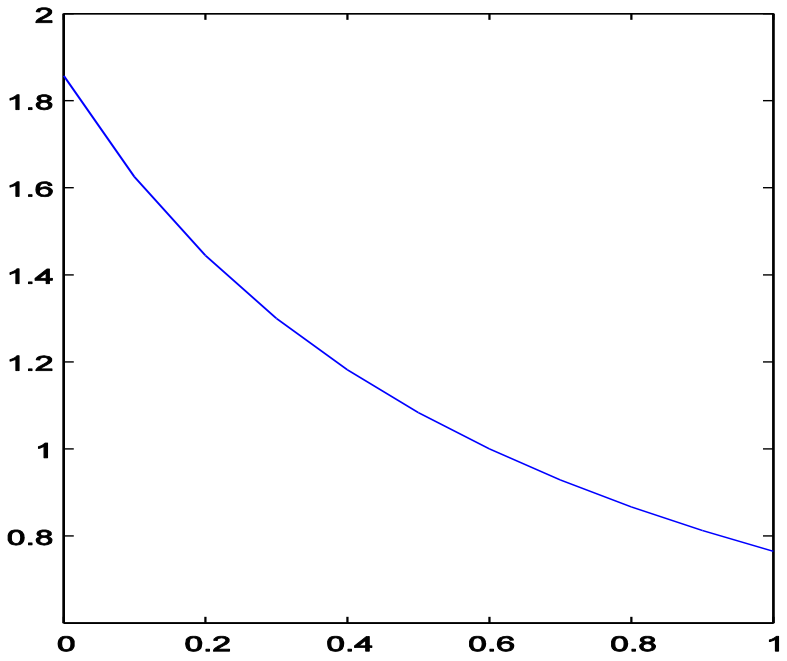


Figure 2: Graph of The Basic Reproduction Number, R_0 as a function of the culling rate, ($\eta \in [0,1]$).

The simulation in Figure 2 shows that by increasing the culling rate η, the value of the basic reproduction number R_0 decreases. At threshold, $R_0 = 1$, corresponding to $\eta = 0.6$. Any control programme with culling of infected birds ($\eta > 0.6$) will be effective.

3.3 Existence And Local Stability of The Disease Free Equilibrium (DFE) State

As stated in Section 3.2, the model given by equations (19) – (23) has a unique disease – free equilibrium state $\mathcal{E}_0 = (i_W, i_D, i_H, q_H, r_H) = (0,0,0,0,0)$ obtained by setting $i_W = 0, i_D = 0, i_H = 0, q_H = 0, r_H = 0$.

To establish the local stability of the disease – free equilibrium (DFE) state, the associated Jacobian of (19) – (23) is evaluated at the DFE state.
The Jacobian matrix of the system (3.4.12) – (3.4.16) evaluated at the disease – free equilibrium state at $J(\mathcal{E}_0)$ is given by

$$J(\mathcal{E}_0) = \begin{pmatrix} \alpha_W - (d_W + \beta_W + \eta) & \alpha_W & 0 & 0 & 0 \\ \alpha_D & \alpha_D - (d_D + \beta_D + \eta) & 0 & 0 & 0 \\ \alpha_B & \alpha_B & -(\varepsilon_H + d_H + \beta_H + v + \gamma) & 0 & 0 \\ 0 & 0 & \varepsilon_H & -(v + \vartheta_H + \gamma + \beta_H) & 0 \\ 0 & 0 & \gamma & \gamma & -(\sigma + \beta_H) \end{pmatrix}$$

The disease – free equilibrium state is locally and asymptotically stable if and only if all of the eigenvalues of the Jacobian matrix $J(\mathcal{E}_0)$ have negative real part (Benjah, 2007). The eigenvalues are determined by solving the characteristic equation $\det(J - \lambda I) = 0$.

$$\begin{vmatrix} \alpha_W - (d_W + \beta_W + \eta) - \lambda & \alpha_W & 0 & 0 & 0 \\ \alpha_D & \alpha_D - (d_D + \beta_D + \eta) - \lambda & 0 & 0 & 0 \\ \alpha_B & \alpha_B & -(\varepsilon_H + d_H + \beta_H + v + \gamma) - \lambda & 0 & 0 \\ 0 & 0 & \varepsilon_H & -(v + \vartheta_H + \gamma + \beta_H) - \lambda & 0 \\ 0 & 0 & \gamma & \gamma & -(\sigma + \beta_H) - \lambda \end{vmatrix} = 0$$

That is

$$[-(\sigma + \beta_H) - \lambda][-(v + \vartheta_H + \gamma + \beta_H) - \lambda][-(\varepsilon_H + d_H + \beta_H + v + \gamma) - \lambda]$$
$$\left[[\alpha_W - (d_W + \beta_W + \eta) - \lambda][\alpha_D - (d_D + \beta_D + \eta) - \lambda] - \alpha_W \alpha_D\right] = 0$$

or

$$[-(\sigma + \beta_H) - \lambda][-(v + \vartheta_H + \gamma + \beta_H) - \lambda][-(\varepsilon_H + d_H + \beta_H + v + \gamma) - \lambda]$$
$$[\lambda^2 - [\alpha_W - (d_W + \beta_W + \eta) + \alpha_D - (d_D + \beta_D + \eta)]\lambda + \left(\alpha_W - (d_W + \beta_W + \eta)\right)$$
$$\left(\alpha_D - (d_D + \beta_D + \eta)\right) - \alpha_W \alpha_D] = 0$$

Thus $\lambda = -(\sigma + \beta_H),\ -(v + \vartheta_H + \gamma + \beta_H), -(\varepsilon_H + d_H + \beta_H + v + \gamma)$

and

$$[\lambda^2 - [\alpha_W - (d_W + \beta_W + \eta) + \alpha_D - (d_D + \beta_D + \eta)]\lambda + \left(\alpha_W - (d_W + \beta_W + \eta)\right)$$
$$\left(\alpha_D - (d_D + \beta_D + \eta)\right) - \alpha_W \alpha_D] = 0 \qquad (31)$$

Clearly, three eigenvalues are negative. We further need to show that equation (31) has negative eigenvalues. Now equation (31) is the characteristic equation of sub matrix J_1,
where

$$J_1 = \begin{pmatrix} \alpha_W - (d_W + \beta_W + \eta) & \alpha_W \\ \alpha_D & \alpha_D - (d_D + \beta_D + \eta) \end{pmatrix}$$

We shall use the trace and determinant method to show that sub matrix (J_1), has negative eigenvalues. The sub matrix (J_1) satisfy $Re(\lambda_j) < 0\ i = 1,2$, if and only if only trace of $(J_1) < 0$ and $detJ_1 > 0$ (Benjah, 2007).

The trace of $(J_1) = \alpha_W - (d_W + \beta_W + \eta) + \alpha_D - (d_D + \beta_D + \eta)$

$$= (d_W + \beta_W + \eta)\left[\frac{\alpha_W}{d_W + \beta_W + \eta} - 1\right] + (d_D + \beta_D + \eta)\left[\frac{\alpha_D}{d_D + \beta_D + \eta} - 1\right]$$

$$= (d_W + \beta_W + \eta)\left[\left(R_0 - \frac{\alpha_D}{d_D + \beta_D + \eta}\right) - 1\right] + (d_D + \beta_D + \eta)\left[\left(R_0 - \frac{\alpha_W}{d_W + \beta_W + \eta}\right) - 1\right]$$

$$= (d_W + \beta_W + \eta)\left[(R_0 - 1) - \frac{\alpha_D}{d_D + \beta_D + \eta}\right] + (d_D + \beta_D + \eta)\left[(R_0 - 1) - \frac{\alpha_W}{d_W + \beta_W + \eta}\right]$$

< 0 if $R_0 < 1$.

and

The $detJ_1 = \left(\alpha_W - (d_W + \beta_W + \eta)\right)\left(\alpha_D - (d_D + \beta_D + \eta)\right) - \alpha_D \alpha_W$

$$= (d_W + \beta_W + \eta)(d_D + \beta_D + \eta) - \alpha_W(d_D + \beta_D + \eta) - \alpha_D(d_W + \beta_W + \eta)$$
$$= (d_W + \beta_W + \eta)(d_D + \beta_D + \eta) - [\alpha_W(d_D + \beta_D + \eta) + \alpha_D(d_W + \beta_W + \eta)]$$

$$= 1 - \left[\frac{\alpha_W}{d_W + \beta_W + \eta} + \frac{\alpha_D}{d_D + \beta_D + \eta}\right]$$

$= 1 - R_0 > 0$ if $R_0 < 1$.

Thus we proved the following lemma.

Lemma 2: The DFEs of the model (3.4.12) – (3.4.16), given by $\mathcal{E}_0$, is locally asymptotically stable (LAS) if $R_0 < 1$ and $\mathcal{E}_0$ is unstable if $R_0 > 1$.

4.0: Conclusion.

The stability analysis of the model showed that the existing domain Ω is positively – invariant and attracting. In this region, the model can be considered as being epidemiologically meaningful and mathematically well – defined. Thus every solution of the basic model with initial conditions in Ω will remains in Ω for all $t > 0$.

Crucial to the stability analysis is the basic reproduction number, R_0. R_0 is an important threshold parameter used to determine the threshold between disease eradication and outbreak. We computed the basic reproduction number, R_0 using the next generation method. Further analysis shows that the basic reproduction number, R_0 is affected by the culling rate $(\eta)$for infected birds as shown in proposition 1 and figure 2. The result shows that increasing culling of infected birds can reduce the basic reproduction number below unity. From the computation for R_0it was obvious that R_0 is not affected by isolation rate for infected humans.

We further ascertain the stability for the disease free equilibrium states (DFEs) using linearization method, taking R_0 as the threshold parameter. The result in Lemma 2 shows that if $R_0 < 1$, the DFEs is locally asymptotically stable. Lemma 2 implies that a small influx of new infective will not generate large outbreaks and avian flu can be eliminated from the avian-human population (when $R_o < 1$) if the initial sizes of the populations of the avian-human model are in the basin of attraction of the DFE, $\mathcal{E}_o$.

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