MATHEMATICAL MODELLING OF THE STABILITY OF HIGHLY PATHOGENIC AVIAN INFLUENZA WITH SATURATED CONTACT RATE

[1]Akpan Collins Emmanuel & [1]Ibrahim Mohammed Olanrewaju

[1]*Department of Mathematics*
University of Ilorin, Ilorin, Nigeria
Email: akpan_collins@yahoo.com, moibraheem@yahoo.com

Abstract

In this paper, we present a Highly Pathogenic Avian Influenza model with Saturated Contact rate. We assume that human can only contact HPAI by direct contact with infected birds. A mathematical model consisting of ordinary differential equations has been proposed in order to analyze / determine time evolution of susceptible birds and infected birds. Analysis based on the model shows that the population of domestic birds can be made secured against infection by proper vaccination and proper removal of infected birds. During the study the basic reproduction number of the model was calculated and the following conclusion was made. If $R_0 < 1$, disease - free equilibrium is stable, the disease will die out; if however, $R_0 > 1$, there exists an endemic equilibrium which is stable, Avian Influenza will spread.

Keywords*: mathematical modelling, avian influenza, ordinary differential equation, reproductive number*.

Introduction

Avian Influenza (AI) the Bird Flu is a virus that infects wild birds (such as ducks, gulls and shore birds) and domestic poultry (such as chickens, turkeys, ducks and geese). There is flu for birds just as there is for humans as with people. Avian Influenza is caused by influenza virus A, which is called H5N1.

Avian Influenza is classified by a combination of two groups based upon the ability of the virus to produce disease in poultry. Low Pathogenic Avian Influenza (LPAI) and Highly Pathogenic Avian Influenza (HPAI).

Highly pathogenic virus result in high death rate up to 100% mortality within 48 hours for some poultry species. Low pathogenicity virus also cause outbreak in poultry but they are not generally associated with severe disease (World Health Organization [12]). Avian Influenza viruses are classified by a combination of two groups of proteins namely the hemagglutinin or H proteins of which there are 16 (H1-H16) and neuraminidase or N proteins, of which there are 9 (N1-N9). Until date, we found that avian influenza virus subtypes which can directly infect human are H5N1, H7N1, H7N2, H7N2, H7N3, H7N7, H9N2, H7N9, subtype. Among them, the new subtype, which was first discovered in 2013 March, and the high

pathogenic Avian Influenza H5N1 subtype are particularly noteworthy. The Avian Influenza virus not only caused human casualties, but also hit the poultry industry. A first infected human was reported in 1997 during a poultry outbreak in Hong Kong SAR (World Health Organization [13]). To control the transmission of avian influenza, some control strategies such as pharmaceutical protections have to be considered (Bowman et al.. [1], Yang et al. [14]). For non-pharmaceutical protection, we implement personal protection and isolation, whereas we adopt vaccination for pharmaceutical protection.

Avian influenza refers to the disease caused by infection with avian (bird) influenza (flu) Type A viruses. These viruses occur naturally among wild aquatic birds worldwide and can infect domestic poultry and other bird and animal species. Avian flu viruses do not normally infect humans. However, sporadic human infections with avian flu viruses have occurred

Although avian influenza A viruses usually do not infect humans, rare cases of human infection with these viruses have been reported. Infected birds shed avian influenza virus in their saliva, mucous and feces. Human infections with bird flu viruses can happen when enough virus gets into a person's eyes, nose or mouth, or is inhaled. This can happen when virus is in the air (in droplets or possibly dust) and a person breathes it in, or when a person touches something that has virus on it then touches their mouth, eyes or nose. Rare human infections with some avian viruses have occurred most often after unprotected contact with infected birds or surfaces contaminated with avian influenza viruses. However, some infections have been identified where direct contact was not known to have occurred. Illness in humans has ranged from mild to severe.

The spread of avian influenza A viruses from one ill person to another has been reported very rarely, and has been limited, inefficient and not sustained. However, because of the possibility that avian influenza A viruses could change and gain the ability to spread easily between people, monitoring for human infection and person-to-person transmission is extremely important for public health.

The reported signs and symptoms of low pathogenic avian influenza (LPAI) A virus infections in humans have ranged from conjunctivitis to influenza-like illness (e.g., fever, cough, sore throat, muscle aches) to lower respiratory disease (pneumonia) requiring hospitalization. Highly pathogenic avian influenza (HPAI) A virus infections in people have been associated with a wide range of illness from conjunctivitis only, to influenza-like illness, to severe respiratory illness (e.g. shortness of breath, difficulty breathing, pneumonia, acute respiratory distress, viral pneumonia, respiratory failure) with multi-organ disease, sometimes accompanied by nausea, abdominal pain, diarrhea, vomiting and sometimes neurologic changes (altered mental status, seizures). LPAI H7N9 and HPAI Asian H5N1 have been responsible for most human illness worldwide to date, including the most serious illnesses and deaths. The best way to prevent infection with avian influenza A viruses is to avoid sources of exposure. Most human infections with avian influenza A viruses have occurred following direct or close contact with infected poultry.

People who have had contact with infected birds may be given influenza antiviral drugs preventatives. While antiviral drugs are most often used to treat flu, they also can be used to prevent infection in someone who has been exposed to influenza viruses. When used to prevent seasonal influenza, antiviral drugs are 70% to 90% effective.

Seasonal influenza vaccination will not prevent infection with avian influenza A viruses, but can reduce the risk of co-infection with human and avian influenza A viruses.

Recently some authors have researched some Avian Influenza model; they had constructed a mathematical model which interprets the spread of avian influenza (Bird flu) from the bird world to the human world. Literature has introduced a piecewise treatment function. The conclusion had indicated that in the prevention and treatment of avian flu, drugs under the condition are limited; culling of infected birds was the most effective way to control the spread of avian flu in humans.

MATHEMATICAL FORMULATION OF THE PROBLEM

The main aim of this research is to model the Stability of Highly Pathogenic Avian influenza with Saturated Contact Rate.

The objectives of the study are:

1. to modify XYSIR model by Che et al [2]
2. to extend model *XYSIR* to X_b Y_b E_b S_h I_h R_h V_h
3. to combat the outbreak by proposing both pharmaceutical (vaccination) and non-pharmaceutical (personal protection and isolation) control methods to reduce the transmission of Avian Influenza.
4. to calculate the basic production number R_0 of the modify model.
5. to carry out simulations with respect to data collected.
6. to interpret the results to policy makers, stakeholders and general public.

VARIABLE DESCRIPTION

VARIABLE	DESCRIPTION
X_b	number of susceptible birds
E_b	number of exposed birds at time t
Y_b	number of HPAI infected birds
S_h	number of susceptible human at time t
I_h	number of infected human

R_h	number of recovered human
V_b	rate of vaccination birds

PARAMETER DESCRIPTION

PARAMETER	DESCRIPTION
Λ	natural birth rate of avian/ bird
π	Human recruitment rate
μ_b	natural mortality of bird
μ_h	natural mortality of human
ν	incubation rate of avian influenza
β_b	rate at which susceptible birds change to be exposed birds
$\in$	rate of vaccination
ò	Mortality of human due illness
γ	recovery rate that infects individuals through treatment
ϕ	mortality rate of birds due to illness
m	infectious rate of susceptible birds to infect birds
β	infected birds of the infection rate of susceptible individual
δ	waning period of the vaccine

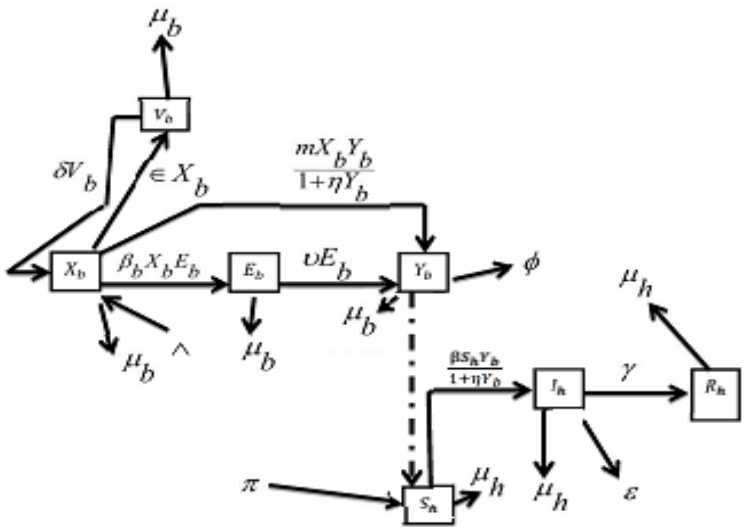
The $X_bY_bE_bS_hI_hR_hV_b$ model

$$\left.\begin{aligned}
\frac{dX_b}{dt} &= \Lambda - \beta_b X_b E_b - \frac{mX_bY_b}{1+\eta Y_b} - Q_1 X_b + \delta V_b \\
\frac{dE_b}{dt} &= \beta_b X_b E_b - Q_2 E_b \\
\frac{dY_b}{dt} &= \frac{mX_bY_b}{1+\eta Y_b} + \nu E_b - Q_3 Y_b \\
\frac{dV_b}{dt} &= \in X_b - Q_4 Y_b \\
\frac{dS_h}{dt} &= \pi - \frac{\beta S_h Y_b}{1+\eta Y_b} - \mu_h S_h \\
\frac{dI_h}{dt} &= \frac{\beta S_h Y_b}{1+\eta Y_b} - Q_5 I_h \\
\frac{dR_h}{dt} &= \gamma I_h - \mu_h R_h
\end{aligned}\right\} \quad (1)$$

Where

$$\left.\begin{aligned}
Q_1 &= \in + \mu_b \\
Q_2 &= \nu + \mu_b \\
Q_3 &= \phi + \mu_b \\
Q_4 &= \delta + \mu_b \\
Q_5 &= \mu_h + \grave{o} + \gamma
\end{aligned}\right\} \quad (2)$$

FLOW CHART OF MODEL



METHOD OF SOLUTION

1.3 Existence and Uniqueness of Solution

The validity and stability of any mathematical model depend on whether the given equation has a solution and whether the solution is unique or not. Using the Lipchitz condition to verify the existence and uniqueness, we simply state the following theorem:

Theorem 1: Derrick and Grossman [4]

Let D denote the region $0 \le \alpha \le R$ then the below equation have a unique solution we show that $\frac{\partial f_i}{\partial x_i}$ for $i = 1,2,3,4,5,6,7$ are continuous and bounded in D'.

Thus, applying the above theorem to equation 1. We have the proof as follows:

Let,

$$\begin{aligned} F_1 &= \Lambda - \beta_b X_b E_b - \frac{m X_b Y_b}{1+\eta Y_b} - Q_1 X_b + \delta V_b \\ F_2 &= \beta_b X_b E_b - Q_2 E_b \\ F_3 &= \frac{m X_b Y_b}{1+\eta Y_b} + \nu E_b - Q_3 Y_b \\ F_4 &= \in X_b - Q_4 Y_b \\ F_5 &= \pi - \frac{\beta S_h Y_b}{1+\eta Y_b} - \mu_h S_h \\ F_6 &= \frac{\beta S_h Y_b}{1+\eta Y_b} - Q_5 I_h \\ F_7 &= \gamma I_h - \mu_h R_h \end{aligned} \tag{3}$$

The system of equation above has a unique solution if

$$\left|\frac{\partial F_i}{\partial x_i}\right| < \infty \text{ for } i = 1,2,3,4,5,6,7$$

Hence, for F_1

$$\left|\frac{\partial F_1}{\partial X_b}\right| = \left|-\left(\beta_b E_b + \frac{m Y_b}{1+\eta Y_b} + Q_1\right)\right| < \infty, \left|\frac{\partial F_1}{\partial E_b}\right| = \left|-\beta_b X_b\right| < \infty,$$

$$\left|\frac{\partial F_1}{\partial Y_b}\right| = \left|\frac{-[(1+\eta Y_b)mY_b - m\eta X_b Y_b]}{(1+\eta Y_b)^2}\right| < \infty, \quad \left|\frac{\partial F_1}{\partial V_b}\right| = |\delta| < \infty, \quad \left|\frac{\partial F_1}{\partial S_h}\right| = 0 < \infty, \backslash\backslash \quad \left|\frac{\partial F_1}{\partial I_h}\right| = 0 < \infty,$$

$$\left|\frac{\partial F_1}{\partial R_h}\right| = 0 < \infty$$

For F_2

$$\left|\frac{\partial F_2}{\partial X_b}\right| = |\beta_b E_b| < \infty, \quad \left|\frac{\partial F_2}{\partial E_b}\right| = |\beta_b X_b - Q_2| < \infty, \quad \left|\frac{\partial F_2}{\partial Y_b}\right| = 0 < \infty, \quad \left|\frac{\partial F_2}{\partial V_b}\right| = 0 < \infty, \quad \left|\frac{\partial F_2}{\partial S_h}\right| = 0 < \infty,$$

$$\left|\frac{\partial F_2}{\partial I_h}\right| = 0 < \infty, \left|\frac{\partial F_2}{\partial R_h}\right| = 0 < \infty$$

For F_3

$$\left|\frac{\partial F_3}{\partial X_b}\right| = \left|\frac{mY_b}{1+\eta Y_b}\right| < \infty, \quad \left|\frac{\partial F_3}{\partial E_b}\right| = |\nu| < \infty, \quad \left|\frac{\partial F_3}{\partial Y_b}\right| = \left|\frac{mX_b - Q_3(1+\eta Y_b)^2}{(1+\eta Y_b)^2}\right| < \infty, \quad \left|\frac{\partial F_3}{\partial V_b}\right| = 0 < \infty,$$

$$\left|\frac{\partial F_3}{\partial S_h}\right| = 0 < \infty, \left|\frac{\partial F_3}{\partial I_h}\right| = 0 < \infty, \left|\frac{\partial F_3}{\partial R_h}\right| = 0 < \infty$$

These partial derivatives exist, continuous and are bounded, similarly for F_4 through F_7, hence existence and uniqueness of the model is established.

STABILITY ANALYSIS OF THE MODEL

In this section, we study and analyze the stability of the equilibrium solutions.

The first six (6) equations of system (1) do not contain R (at steady state), we need to study the following systems.

$$\begin{aligned}
0 &= \Lambda - \beta_b X_b E_b - \frac{mX_b Y_b}{1+\eta Y_b} - Q_1 X_b + \delta V_b \\
0 &= \beta_b X_b E_b - Q_2 E_b \\
0 &= \frac{mX_b Y_b}{1+\eta Y_b} + \nu E_b - Q_3 Y_b \\
0 &= \in X_b - Q_4 Y_b \\
0 &= \pi - \frac{\beta S_h Y_b}{1+\eta Y_b} - \mu_h S_h \\
0 &= \frac{\beta S_h Y_b}{1+\eta Y_b} - Q_5 I_h
\end{aligned} \tag{4}$$

DISEASE-FREE EQUILIBRIUM

At disease free, we assume the absence of influenza A virus (H5N1), hence

$$Y_b = 0 \;\Rightarrow\; I_h = 0 \;\Rightarrow\; E_h = 0$$

Setting $Y_b = 0$ in the fifth equation of (4)

$$\Rightarrow \quad S_h = \frac{\pi}{\mu_h} \tag{5}$$

Setting $Y_b = 0 = E_b = 0$ in the first equation of (4) and solving the first equation of (4) and the fourth equation of (4) simultaneously, we have

$$X_b = \frac{\Lambda Q_4}{Q_2 Q_4 - \delta \in} \tag{6}$$

and

$$V_b = \frac{\in X_b}{Q_4} \tag{7}$$

Substituting equation (6) into (7), we have

$$V_b = \frac{\Lambda \in}{Q_1 Q_4 - \delta \in} \tag{8}$$

Where

$$Q_1 = \in + \mu_b \qquad Q_4 = \delta + \mu_b$$

Hence Disease free equilibrium of system (4) becomes

$$E_0\left(X_b^{\circ}, E_b^{\circ}, Y_b^{\circ}, V_b^{\circ}, S_h^{\circ}, I_h^{\circ}\right) = \left(\frac{\Lambda Q_4}{Q_1 Q_4 - \delta \in}, 0, 0, \frac{\Lambda \in}{Q_1 Q_4 - \delta \in}, \frac{\pi}{\mu_h}, 0\right)$$

$$J=\begin{pmatrix} -Q_1-\lambda & \frac{-\wedge\beta_b Q_4}{Q_1Q_4-\delta\in} & \frac{-m\wedge Q_4}{Q_1Q_4-\delta\in} & \delta & 0 & 0 \\ 0 & \frac{\wedge\beta_b Q_4}{Q_1Q_4-\delta\in}-Q_2-\lambda & 0 & 0 & 0 & 0 \\ 0 & \nu & \frac{m\wedge Q_4}{Q_1Q_4-\delta\in}-Q_3-\lambda & 0 & 0 & 0 \\ \in & 0 & 0 & -Q_4-\lambda & 0 & 0 \\ 0 & 0 & -\frac{\pi\beta}{\mu_h} & 0 & -\mu_h-\lambda & 0 \\ 0 & 0 & \frac{\pi\beta}{\mu_h} & 0 & 0 & -Q_5-\lambda \end{pmatrix} \quad (9)$$

The Jacobian matrix for the model in system (1) R_h is given by

which in the disease-free equilibrium E_0 is

$$J=\begin{pmatrix} -Q & \frac{-\wedge\beta_b Q_4}{Q_1Q_4-\delta\in} & \frac{-m\wedge Q_4}{Q_1Q_4-\delta\in} & \delta & 0 & 0 \\ 0 & \frac{\wedge\beta_b Q_4}{Q_1Q_4-\delta\in}-Q_2-\lambda & 0 & 0 & 0 & 0 \\ 0 & \nu & \frac{m\wedge Q_4}{Q_1Q_4-\delta\in}-Q_3-\lambda & 0 & 0 & 0 \\ \in & 0 & 0 & -Q_4-\lambda & 0 & 0 \\ 0 & 0 & -\frac{\pi\beta}{\mu_h} & 0 & -\mu_h-\lambda & 0 \\ 0 & 0 & \frac{\pi\beta}{\mu_h} & 0 & 0 & -Q_5-\lambda \end{pmatrix} \quad (10)$$

Calculating the Eigen values

$$= \begin{pmatrix} -Q_1-\lambda & \frac{-\wedge\beta_b Q_4}{Q_1Q_4-\delta\in} & \frac{-m\wedge Q_4}{Q_1Q_4-\delta\in} & \delta & 0 & 0 \\ 0 & \frac{\wedge\beta_b Q_4}{Q_1Q_4-\delta\in}-Q_2-\lambda & 0 & 0 & 0 & 0 \\ 0 & \nu & \frac{m\wedge Q_4}{Q_1Q_4-\delta\in}-Q_3-\lambda & 0 & 0 & 0 \\ \in & 0 & 0 & -Q_4-\lambda & 0 & 0 \\ 0 & 0 & -\frac{\pi\beta}{\mu_h} & 0 & -\mu_h-\lambda & 0 \\ 0 & 0 & \frac{\pi\beta}{\mu_h} & 0 & 0 & -Q_5-\lambda \end{pmatrix} \quad (10)$$

The characteristics equation of the Jacobian matrix

$$J_{E_0} = (\lambda+Q_1)[(\lambda+Q_2)-\wedge\beta_b h][(\lambda+Q_3)-\wedge mh](\lambda+Q_4)(\lambda+\mu_h)(\lambda+Q_5)$$

Here,

$$h=\frac{Q_4}{Q_1Q_4-\delta\in},\ Q_1=\in+\mu_b,\ Q_2=\nu+\mu_b,\ Q_3=\phi+\mu_b,$$

$$Q_4=\delta+\mu_b, Q_5=\mu_h+\grave{o}+\gamma$$

The eigen values are $\lambda_1=-Q_1$, $\lambda_2=-Q_4$, $\lambda_3=-\mu_h$, $\lambda_4=-Q_5, \lambda_5=\wedge\beta_b h-Q_2, \lambda_6=\wedge mh-Q_3$ where λ denotes the indeterminate of the polynomial. If and only if $R_0<1$, all roots of this characteristics equation have negative real part. It implies that E_0 is locally asymptotically stable. Then we can get the following theorem.

Theorem 2:

If $R_0\le 1$, the disease- free equilibrium E_0 is locally asymptotically stable, if $R_0>1$, the disease free equilibrium E_0 is unstable.

So by theorem 2, the proof is as follows:

From eigenvalue λ_4 and λ_5, the value $Q_2>\wedge\beta_b h$ and $Q_3>\wedge mh$

Rearranging in order to get the largest eigen value we have,

$$\wedge\beta_b h - Q_2 < 0 \Rightarrow \wedge\beta_b h < Q_2 \text{ dividing through by } Q_2$$

$$\Rightarrow \frac{\wedge\beta_b h}{Q_2} < 1 \Rightarrow R_0 < 1$$

Thus

$$R_0^{X_b E_b Y_b V_b S_h I_h df_e} = \frac{\beta_b h \wedge}{Q_2} \text{ where } h = \frac{Q_4}{Q_1 Q_4 - \delta \in}$$

and substituting the values for Q_1 and Q_4 we have:

$$\Rightarrow R_0 = \frac{\beta_b(\delta + \mu_b)\wedge}{(v + \mu_b)[(\in + \mu_b)(\delta + \mu_b) - \delta \in]} < 1$$

Hence the proof.

Endemic Equilibrium

For the endemic equilibrium, it means disease exist, hence $Y_b \neq 0$, $I_h \neq 0$. The systems of equation should satisfy the following conditions.

$$(X_b^* > 0, E_b^* > 0, Y_b^* > 0, V_b^* > 0, S_h^* > 0, I_h^* > 0)$$

The epidemic equilibrium state is the state in which there is persistence of the disease in the population.

By the positive of the endemic equilibrium point, we can get that if $R_0 > 1$, there is a unique endemic

$E_+(X_b^*, E_b^*, Y_b^*, V_b^*, S_h^*, I_h^*)$ which satisfied from the system in equation (4).

Using equation the second equation of (4) we have

$$0 = \beta_b X_b E_b - Q_2 E_b \Rightarrow Q_2 E_b = \beta_b X_b E_b \Rightarrow X_b^* = \frac{Q_2}{\beta_b} \qquad (12)$$

from equation the third equation of (4) $0 = \in X_b - Q_4 V_b$ putting equation (12) we have

$$V_b^* = \frac{\in Q_2}{\beta_b Q_4} \qquad (13)$$

from equation the fifth equation of (4)

$$0=\pi-\frac{\beta S_h Y_b}{1+\eta Y_b}-\mu_h S_h S_h\left(\frac{\beta Y_b}{1+\eta Y_b}+\mu_h\right)\Rightarrow S_h\left(\frac{\beta Y_b+\mu_h(1+\eta Y_b)}{1+\eta Y_b}\right)=\pi\Rightarrow S_h=\frac{\pi(1+\eta Y_b)}{\beta Y_b+\mu_h(1+\eta Y_b)}$$

hence,

$$S_h^*=\frac{\pi(1+\eta Y_b^*)}{\beta Y_b^*+\mu_h(1+\eta Y_b^*)} \tag{14}$$

Also,

from equation the sixth equation of (4)

$$0=\frac{\beta S_h Y_b}{1+\eta Y_b}-Q_5\Rightarrow I_h=\frac{\beta S_h^* Y_b^*}{(1+\eta Y_b^*)Q_5} \tag{15}$$

substituting equation (14) into equation (15) we have

$$I_h^*=\frac{\beta Y_b^*\pi}{Q_5(\beta Y_b^*+\mu_h(1+\eta Y_b^*))} \tag{16}$$

using equation the third equation of (4)

$$0=\frac{mX_b Y_b}{1+\eta Y_b}+vE_b-Q_3 Y_b\Rightarrow$$

$$E_b^*=\frac{Q_3 Y_b^*(1+\eta Y_b^*)-mX_b^* Y_b^*}{v(1+\eta Y_b^*)} \tag{17}$$

substituting equation (12) into equation (17) we have

$$E_b^*=\frac{\beta_b Q_3 Y_b^*(1+\eta Y_b^*)-mQ_2 Y_b^*}{\beta_b v(1+\eta Y_b^*)} \tag{18}$$

using equation (4)

$$0=\wedge-\beta_b X_b E_b-\frac{mX_b Y_b}{1+\eta Y_b}-Q_1 X_b+\delta V_b$$

substituting equation (17) and (23) into the above equation, we have

$$0=\wedge-Q_2\left(\frac{\beta_b Q_3 Y_b^*(1+\eta Y_b^*)-nQ_2 Y_b^*}{\beta_b v(1+\eta Y_b*)}\right)-\frac{mQ_2 Y_b^*}{\beta_b(1+\eta Y_b^*)}-Q_1\left(\frac{Q_2}{\beta_b}\right)+v\left(\frac{\in Q_2}{\beta_b Q_4}\right)$$

$$0 = \frac{\wedge\beta_b v Q_4(1+\eta Y_b^*) - Q_2 Q_4(\beta_b Q_3 Y_b^* + \beta_b Q_3 \eta Y_b^{*2} - mQ_2 Y_b^*) - vQ_4 mQ_2 Y_b^* - vQ_4 Q_1 Q_2(1+\eta Y_b^*) + v\delta \in Q_2(1+\eta Y_b^*)}{\beta_b v Q_4(1+\eta Y_b^*)}$$

$$0 = \wedge\beta_b v Q_4 + \wedge\beta_b v Q_4 \eta Y_b^* - Q_2 Q_3 Q_4 \beta_b Y_b^* - Q_2 Q_3 Q_4 \beta_b Y_b^{*2} + mQ_2^2 Q_4 Y_b^*$$
$$-vmQ_2 Q_4 Y_b^* - vQ_4 Q_1 Q_2 - vQ_1 Q_2 Q_4 \eta Y_b^* + v\delta \in Q_2 + v\delta \in Q_2 \eta Y_b^*$$

re-arranging to obtain the quadratic form we have

$$\eta Q_2 Q_3 Q_4 \beta_b Y_b^{*2} + (Q_2 Q_3 Q_4 \beta_b - \wedge\beta_b v Q_4 \eta + vmQ_2 Q_4 - mQ_2^2 Q_4 + vQ_1 Q_2 Q_4 \eta - v\delta \in Q_2 \eta) Y_b^*$$
$$+vQ_1 Q_2 Q_4 - \wedge\beta_b v Q_4 - v\delta \in Q_2 = 0 \tag{19}$$

Theorem 3:

If $R_0 \leq 1$, the system equation (4-9) only exists the disease- free equilibrium $E_0\left(\frac{\Lambda Q_4}{Q_1 Q_4 - \delta \in}, 0, 0, \frac{\Lambda \in}{Q_1 Q_4 - \delta \in}, \frac{\pi}{\mu_h}, 0\right)$;

$R_0 > 1$, there exists only one endemic equilibrium

$$E_+^*\left(\frac{Q_2}{\beta_b}, \frac{Q_3 Y_b^*(1+\eta Y_b^*) - mX_b^* Y_b^*}{v(1+\eta Y_b^*)}, Y_b^*, \frac{\pi(1+\eta Y_b^*)}{\beta Y_b^* + \mu_h(1+\eta Y_b^*)}, \frac{\beta Y_b^* \pi}{Q_5(\beta Y_b^* + \mu_h(1+\eta Y_b^*))}\right)$$

Y_b^* is the positive solution of the following equation and hence the proof is given below: We simply let

$$a_0 Y_b^{*2} + a_1 Y_b^* + a_2 = 0 \tag{20}$$

where

$$\left.\begin{aligned} a_0 &= \beta_b \eta Q_2 Q_3 Q_4 \\ a_1 &= Q_2 Q_3 Q_4 \beta_b - \wedge\beta_b v Q_4 \eta + vmQ_2 Q_4 - mQ_2^2 Q_4 + vQ_1 Q_2 Q_4 \eta - v\delta \in Q_2 \eta \\ a_2 &= vQ_1 Q_2 Q_4 - \wedge\beta_b v Q_4 - v\delta \in Q_2 \end{aligned}\right\} \tag{21}$$

for the equilibrium to exist, the solution (21) must be real and positive, we note

$$a_0 \geq 0;\ a_2 < 0 \Leftrightarrow R_0 > 1;\ a_2 \geq 0 \Leftrightarrow R_0 \leq 1$$

from

$a_2 < 0$ we have

$$vQ_1Q_2Q_4 - \wedge\beta_b vQ_4 - v\delta \in Q_2 < 0 \Rightarrow$$
$$vQ_1Q_2Q_4 < \wedge\beta_b vQ_4 + v\delta \in Q_2 \text{ (Divide through by } vQ_1Q_2Q_4)$$

$$1 < \frac{\wedge\beta_b vQ_4 + v\delta \in Q_2}{vQ_1Q_2Q_4} \Rightarrow 1 < \frac{\wedge\beta_b Q_4 + \delta \in Q_2}{Q_1Q_2Q_4}$$

substituting the values for Q_1Q_2 and Q_4 we have

$$1 < \frac{\wedge\beta_b(\delta + \mu_b) + \delta \in (v + \mu_b)}{(\in + \mu_b)(v + \mu_b)(\delta + \mu_b)} \Rightarrow$$

$1 < R_0 \Rightarrow R_0 > 1$. Hence the proof. This implies that the avian influenza will spread.

Basic Reproduction Number

Using the Next Generation Matrix

The next generation matrix has a number of desirable properties from a mathematical stand point. In particular, if is non-negative matrix and as such, it is guaranteed that there will be a single, unique eigen value which is positive, real and strictly greater than all the other. This is R_0.

The basic reproduction number is given by the spectral radius of G. The spectral radius is also known as the dominant eigen value of G.

Consider the next generation matrix G. It is comprised of two parts. F and V^{-1}, where

$$F = \left[\frac{\partial F_i(x_0)}{\partial x_j}\right] \quad (22)$$

and

$$V = \left[\frac{\partial V_i(x_0)}{\partial x_j}\right] \quad (23)$$

The F_i are the new infections, while the V_i transfers of infections from one compartment to another. x_0 is the disease-free equilibrium state. R_0 is the dominant eigen value of the matrix.

$$G = FV^{-1} \quad \text{(Hefferman et al. [6])} \quad (24)$$

Rearranging the equation in system (1) starting with the infective classes.

$$\left.\begin{aligned}
\frac{dI_h}{dt} &= \frac{\beta S_h Y_b}{1+\eta Y_b} - Q_5 I_h \\
\frac{dY_b}{dt} &= \frac{mX_b Y_b}{1+\eta Y_b} + vE_b - Q_3 Y_b \\
\frac{dE_b}{dt} &= \beta_b X_b E_b - Q_2 E_b \\
\frac{dX_b}{dt} &= \wedge - \beta_b X_b E_b - \frac{mX_b Y_b}{1+\eta Y_b} - Q_1 X_b + \delta V_b \\
\frac{dS_h}{dt} &= \pi - \frac{\beta S_h Y_b}{1+\eta Y_b} - \mu_h S_h \\
\frac{dV_b}{dt} &= \in X_b - Q_4 V_b \\
\frac{dR_h}{dt} &= \gamma I_h - \mu_h R_h
\end{aligned}\right\} \quad (25)$$

we have three infected classes in this model which includes exposed birds E_b, number of HPAI infected poultry Y_b and number of infected human I_h, hence our $m=3$

$$F = \begin{pmatrix} \frac{\beta S_h Y_b}{1+\eta Y_b} \\ 0 \\ 0 \end{pmatrix}, V = \begin{pmatrix} Q_5 I_h \\ Q_3 Y_b - vE_b - \frac{mX_b Y_b}{1+\eta Y_b} \\ Q_2 E_b - \beta_b X_b E_b \end{pmatrix} \quad (26)$$

$$F = \begin{pmatrix} 0 & \frac{\beta S_h Y_b}{(1+\eta Y_b)^2} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}, V = \begin{pmatrix} Q_5 & 0 & 0 \\ 0 & Q_3 - \frac{mX_b}{(1+\eta Y_b)^2} & -v \\ 0 & 0 & Q_2 - \beta_b X_b \end{pmatrix} \quad (27)$$

for the disease free- equilibrium point of the system of equation

$$E_0 = (X_b^0, 0, 0, V_b^0, S_h^0, 0)$$

where

$$X_b^0 = \frac{\wedge Q_4}{Q_1 Q_4 - \delta \in}, V_b^0 = \frac{\in \wedge}{Q_1 Q_4 - \delta \in}, S_h^0 = \frac{\pi}{\mu_h}$$

Thus equation (27) becomes

$$F=\begin{pmatrix}0 & \beta S_h^0 & 0\\ 0 & 0 & 0\\ 0 & 0 & 0\end{pmatrix}, V=\begin{pmatrix}Q_5 & 0 & 0\\ 0 & Q_3-mX_b^0 & -v\\ 0 & 0 & Q_2-\beta_b X_b^0\end{pmatrix}\Bigg\} \tag{28}$$

The matrices in equation (28) can be re written as

$$F=\begin{pmatrix}0 & \beta S_h^0 & 0\\ 0 & 0 & 0\\ 0 & 0 & 0\end{pmatrix}, V=\begin{pmatrix}Q_5 & 0 & 0\\ 0 & k_1 & -v\\ 0 & 0 & k_2\end{pmatrix}\Bigg\} \tag{29}$$

where $k_1=Q_3-mX_b^0$, $k_2=Q_2-\beta_b X_b^0$

To find V^{-1} we know that

$$V^{-1}=\frac{1}{|V|}\cdot \mathrm{Adj}V$$

$$|V|=\begin{vmatrix}Q_5 & 0 & 0\\ 0 & k_1 & -v\\ 0 & 0 & k_2\end{vmatrix}=Q_5k_1k_2$$

let

$$C=\begin{pmatrix}k_1k_2 & 0 & 0\\ 0 & Q_5k_2 & 0\\ 0 & vQ_5 & Q_5k_1\end{pmatrix}$$

so that

$$C^T = \mathrm{Adj}V=\begin{pmatrix}k_1k_2 & 0 & 0\\ 0 & Q_5k_2 & vQ_5\\ 0 & 0 & Q_5k_1\end{pmatrix}$$

$$\Rightarrow V^{-1} = \frac{1}{Q_5k_1k_2}\begin{pmatrix}k_1k_2 & 0 & 0\\ 0 & Q_5k_2 & vQ_5\\ 0 & 0 & Q_5k_1\end{pmatrix}$$

$$\Rightarrow V^{-1} = \begin{pmatrix} \frac{1}{Q_5} & 0 & 0 \\ 0 & \frac{1}{k_1} & \frac{v}{k_1 k_2} \\ 0 & 0 & \frac{1}{k_2} \end{pmatrix} \tag{30}$$

where $k_1 = Q_3 - mX_b^0$, $k_2 = Q_2 - \beta_b X_b^0$

$$FV^{-1} = \begin{pmatrix} 0 & \beta S_h^0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \begin{pmatrix} \frac{1}{Q_5} & 0 & 0 \\ 0 & \frac{1}{k_1} & \frac{v}{k_1 k_2} \\ 0 & 0 & \frac{1}{k_2} \end{pmatrix} \tag{31}$$

$$= \begin{pmatrix} 0 & \frac{\beta S_h^0}{k_1} & \frac{\beta S_h^0 v}{k_1 k_2} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \tag{32}$$

Thus,the spectral radius is the largest eigenvalues of the matrix, hence we look for the eigenvalues of the above matrix

$$R_0 = \frac{\beta S_h^0 v}{k_1 k_2}$$

Substituting the value for $S_h^0 = \frac{\pi}{\mu_h}$, $k_1 = Q_3 - mX_b^0$, $k_2 = Q_2 - \beta_b X_b^0$, $X_b = \frac{\wedge Q_4}{Q_1 Q_4 - \delta \in}$ we have,

$$R_0 = \frac{\beta\left(\frac{\pi}{\mu_h}\right)v}{\left[Q_3 - m\left(\frac{\wedge Q_4}{Q_1 Q_4 - \delta \in}\right)\right]\left[Q_2 - \beta_b\left(\frac{\wedge Q_4}{Q_1 Q_4 - \delta \in}\right)\right]}$$

Simplifying the above, we have

$$R_0 = \frac{\beta \pi v (Q_1 Q_4 - \delta \in)^2}{\mu_h (Q_1 Q_3 Q_4 - Q_3 \delta \in -m \wedge Q_4)(Q_1 Q_2 Q_4 - Q_2 \delta \in -\beta_b \wedge Q_4)}$$

where

$$Q_1 = \in + \mu_b,\ Q_2 = \nu + \mu_b,\ Q_3 = \phi + \mu_b$$

$$Q_4 = \delta + \mu_b$$

Numerical Simulation

In this section, the model has been analysed numerically using Maple 18, to confirm the result we have obtained, we made use of figures from reference material [Che et al. [2]] and others are hypothetical data. For this purpose we have used the following set of parameters values.

Numerical Solutions of Our Model for $R_0 < 1$

The parameters are:

Parameters	**Figures**	**Source**
Λ	2	Che et al. [2]
π	0.97	Che et al. [2]
η	2	Che et al. [2]
μ_b	0.03	Che et al. [2]
μ_h	0.069	Che et al. [2]
ν	0.5	Hypothetical
β_b	0.01	Che et al. [2]
$\in$	0.2	Hypothetical
ò	0.63	Che et al. [2]
γ	0.301	Che et al. [2]
ϕ	0.97	Che et al. [2]
m	0.14	Hypothetical
β	0.02	Che et al. [2]
δ	0.2	Hypothetical

$R_0 = 0.4837929$

These parameters values satisfy the condition $R_0 < 1$ which implies that the avian influenza will die out.

Numerical Solutions of Our Model for $R_0 > 1$

The parameters are

Parameters	**Figures**	**Source**
Λ	2	Che et al. [2]
π	0.97	Che et al. [2]
η	2	Che et al. [2]
μ_b	0.03	Che et al. [2]
μ_h	0.069	Che et al. [2]
ν	0.5	Hypothetical
β_b	0.01	Che et al. [2]
$\in$	0.2	Hypothetical
ò	0.63	Che et al. [2]
γ	0.301	Che et al. [2]
ϕ	0.97	Che et al. [2]
m	0.14	Hypothetical
β	0.02	Che et al. [2]
δ	0.2	Hypothetical

$R_0 = 1.077044$

These parameters values satisfy the condition $R_0 > 1$, the unique and endemic equilibrium E^* is global asymptotically stable hereby causing the avian influenza will spread.

Conclusion

In this study, we constructed the mathematical model for the stability of highly pathogenic avian influenza with saturated contact Rate. Standard dynamical modeling method is used for analyzing the behaviour of solution.

The basic reproductive number is defined by R_0

where

$$R_0 = \frac{\beta\pi v(Q_1Q_4 - \delta \in)^2}{\mu_h(Q_1Q_3Q_4 - Q_3\delta \in -m \wedge Q_4)(Q_1Q_2Q_4 - Q_2\delta \in -\beta_b \wedge Q_4)}$$

for $R_0 < 1$ the disease-free equilibrium state is stable and endemic equilibrium is stable when $R_0 > 1$.

The solution approach to the disease equilibrium state

$$E^0(19.04762, 0, 0, 47.619048, 14.492754, 0)$$

where $R_0 < 1$. E_+^* is globally asymptotically stable, that implies that the disease will sustain and lead to epidemics, through the analysis of the model some control strategies such as pharmaceutical protections have to be considered. For non-pharmaceutical protection, we implement personal protection and quarantine (isolation) whereas we adopt vaccination for pharmaceutical protection. Optimal Control analysis and cost effectiveness are recommended for further study in order to help decision makers understand best strategy and cost implication to manage Avian Influenza. Finally the government must strictly manage meat sources.

REFERENCES:

[1] Bowman C, Gumel AB, van den Driessche P, Wu J, Zhu H (2005). A mathematical model for assessing control strategies against West Nile virus. Bull Math Biol 67:1107-1133

[2] Che, S.Q, Xue, Y.k and Ma, L.K (2014). The stability of highly stability of highly pathogenic Avian influenza Epidemic Model with Saturated Contact Rate. Applied Mathematics 5, 3365-3371.

[3] Chong, N.S., Tehuenche, J. M., Smith R. J (2014). The mathematical model of Avian Influenza with half saturated incidence. Theory Bio Sci. 133:23-38.

[4] Derrick, N. R. and Grossman, S. L. (1976) Differential equations with applications. Addison Wesley Publishing Company.

[5] Gumel AB (2009). Global dynamics of a two-strain avian influenza model. Int J Comput Math 86: 85-108.

[6] Hefferman, J. R. Smith and Whahl, L. (2005) Perspectives on the basic reproduction ratio. Journal of the Royal Society interface 2, 281-293.

[7] Iwami, S., Takeuchi, Y., Liu, X., (2007). Avian-human influenza epidemic model. Math. Biosci, 207, 1-25.

[8] Iwami, S., Takeuchi,Y., Korobeinikov, A. and Liu, X. (2008). Prevention of Avian Influenza Epidemic. What Policy Should We Choose. Journal of Theoretical Biology, 252,732-741.

[9] Martcheva, M (2014). Modeling and implications for control. J . Bio Syst 22,151

[10] Nu\tilde{\text{n}}o M, Chowell, Gumel AB (2006). Assessing the role of basic control measures, antivirals and vaccine in curtailing pandemic influenza: scenarios for the US, Uk and the Netherlands. J R Soc interface 4:505-521.

[11] Webster, R.G., Chen, H., S.,Guan, Y., (2006). H5N1 outbreaks and enzootic influenza. Emerging Infect. Dis. 12, 3-8.

[12] World Health Organization (2011) New: WHO comment on the importance of monitoring of variant influenza viruses. http://www.who.int/influenza/ human _animal _interface/ avian_influenza/ h5n1-2011_12_19/ en/ index. html (Accessed 30 Jan 2013).

[13] World Health Organization (2012) H5N1 avian influenza: timeline of major events. http://www.who.int/influenza/human_animal_interface/avian_influenza/ H5N1_avian_influenza_update. Pdf (Accessed 30 J2013).

[14] Yang J, Sugigimoto J.D., Halloran M.E., (2009). The transmissibility and control of Pandemic influenza A (H1N1) virus. Sci Agric 326:729-733.