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Numerical Simulation of Bird-Flu Epidemics

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Abstract: This study presents a numerical simulation of Bird flu epidemic model. Appropriate systems of ordinary differential equations formulated with respect to the birds and humans' model population were solved numerically and analyzed. The disease free equilibrium of the system was found to be locally asymptotically stable.

Key words: Stability, disease free equilibrium, jacobian matrix

INTRODUCTION

Avian influenza (Bird-Flu) is a respiratory infection in mammals and birds. An RNA virus in the family Orthomyxoviridae causes it. Surprisingly, little is known about the transmission of Bird-Flu disease is some part of the world like Africa. Bird flu is an underrated disease; perhaps because, it is a recurrent disease with which we are all familiar and from which man usually recovers naturally, it is not as dangerous as AIDS, tuberculosis or malaria and yet it is a major contributor to mortality and morbidity throughout the world[1]. The World Health Organization (WHO) estimated that respiratory infections killed more than four million people in 1999, making them the most dangerous category of infectious disease. Flu contributes to many of these deaths, but calculating how much mortality is caused directly and indirectly by flu has proven to be difficult[2].

There are several reasons for this, including:

- flu predisposes individuals to potentially fatal secondary infection with bacterial pathogens;
- flu or bacterial super infections kill in conjunction with other diseases, such as chronic cardiopulmonary conditions. However, flu poses a very real threat to people of all ages with various chronic medical conditions and flu pandemics can cause n heavy mortality in all age groups[3].

The world has been experiencing a relentless spread of bird flu due to importation of chicken birds, as they move around the world to seasonal breeding and feeding grounds infecting domestic flocks around the world. More than 150 million birds, mostly chickens, have died or been culled. Sixty-three out of 124 infected humans have died since December 2003.

It is only a matter of time before an avian flu virus-most likely H5N1-acquires the ability to be transmitted from human to human, sparking the outbreak of human pandemic influenza. We don't know when this will happen. But we do know that it will happen. (WHO).

The widespread persistence of H5N1 in poultry populations poses two main risks for human health.

The first is the risk of direct infection when the virus passes from poultry to humans, resulting in very severe disease. Of the few avian influenza viruses that have crossed the species barrier to infect humans, H5N1 has caused the largest number of cases of severe disease and death in humans. Unlike normal seasonal influenza, where infection causes only mild respiratory symptoms in most people, the disease caused by H5N1 follows an unusually aggressive clinical course, with rapid deterioration and high fatality. Primary viral pneumonia and multi-organ failure are common. In the present outbreak, more than half of those infected with the virus have died. Most cases have occurred in previously healthy children and young adults.

A second risk, of even greater concern, is that the virus-if given enough opportunities-will change into a form that is highly infectious for humans and spreads easily from person to person. Such a change could mark the start of a global outbreak (a pandemic). (WHO)

There is, therefore, the need to have a mathematical description of the disease dynamics, for better understanding of Flu spreads while this will also enable them to take steps that will help curtail its spread to the barest minimum.

MODEL FORMULATION

The proposed model describes the dynamics in the birds (chickens) and humans population subject to Avian Influenza (high pathogenic type). However, it is obvious that in humans the infection by this disease causes no permanent immunity and there is no effective vaccination

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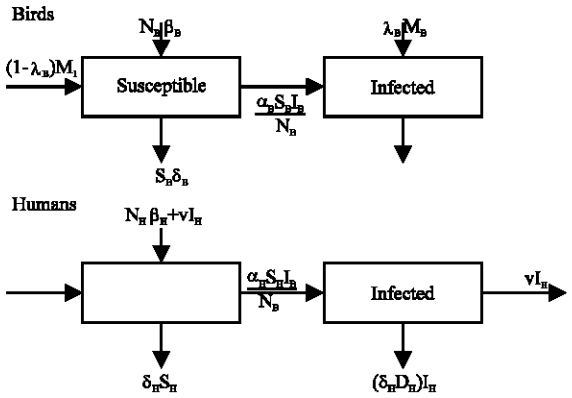

Fig. 1: Schematic descriplia of the madle

against its infection at present. Consequently, we will adopt the SIRS model, which is denoted with "susceptible (S), infectious (I), or recovered but susceptible (RS)"[3], each of the sub-population is compartmentalised into two categories-susceptible or infectious. Here, the recovered category for birds is ignored since birds (chickens) hardly recover from highly pathogenic avian influenza[4] while in humans the recovered category is factored back into its susceptible category due to the possibility of infection after recovery from Flu, though there exist some kind of temporary immunity[1].

The model monitors the temporary dynamics of the populations of susceptible birds $S_B(t)$, infectious birds $I_B(t)$, susceptible humans $S_H(t)$ and infectious humans $I_H(t)$ as captured in model equations. It is important to note that $S_B = S_B(t)+I_B(t)$ represents the total population of birds in the location of interest while $S_H = S_H(t)+I_H(t)$ represents the total human population in that same location. A schematic description of the model is in Fig.1.

As can be derived from the diagrams above, the time rate of change for birds and humans population is modelled by the following ODE dynamical system:

$$\left.\begin{aligned}
\frac{dS_B}{dt} &= N_B\beta_B + (1-\lambda_B)M_B - \alpha_B S_B\frac{I_B}{N_B} - \delta_B S_B,\\
\frac{dI_B}{dt} &= \alpha_B S_B\frac{I_B}{N_B} - (\delta_B + d_B)I_B + \lambda_B M_B,\\
\frac{dS_H}{dt} &= N_H\beta_H - \frac{\alpha_H S_H I_B}{N_B} - \delta_H S_H + \nu I_H,\\
\frac{dI_H}{dt} &= \frac{\alpha_H S_H I_B}{N_B} - (\delta_H + d_H + \nu)I_H
\end{aligned}\right\} \quad (1)$$

The interpretations of the parameters in the above system of equations and their assigned values are given in the Table that follows[5,1]:

Also, we assumed that each of the total subpopulations N_B, N_H> at t = 0 to avoid indeterminate situations in the model equations while non-negative initial conditions were used to ensure that variables in (1) remain non-negative.

RESULTS AND DISCUSSION

The model equations were solved numerically using Maple software and results were plotted graphically.

In the first instance, numerical simulations were obtained for three different cases depending on whether the infection starts from migrated birds, birds on ground, or both sources at a time. The results from these simulations were as shown in Fig. 2 and 3.

As we can see from the graph in Fig. 2, the Flu-disease spread in birds is much rapid when the infection starts from both migrated birds and birds on ground than when it starts from either of the sources. However, the difference in the spread in the latter and the former situations diminish over time while the latter spreads more rapidly afterwards. Realistically, the situation where the

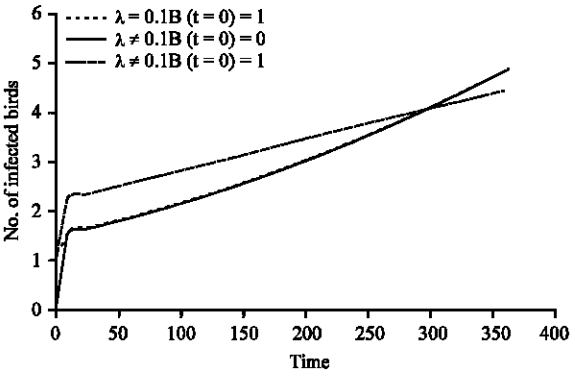

Fig. 2: Graph of infected birds against time using different infection sources

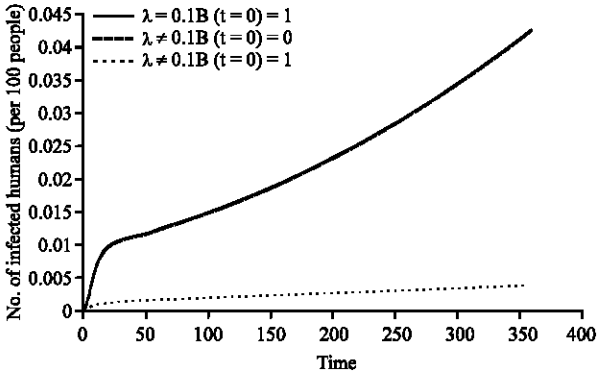

Fig. 3: Graph of infected human against time using different infection sources

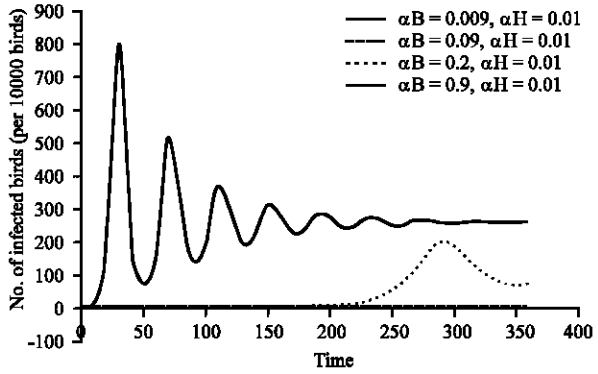

Fig. 4: Graph of infected birds against time using different flu-transmission sources

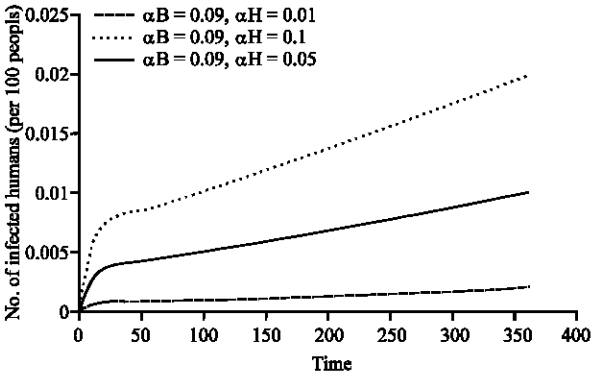

Fig. 5: Graph of infected humans against time using different Flu-transmission sources.

latter then spreads more rapidly may not actually arise, since solution to the spread could have been reached before then.

In Fig. 3, we observed that the spread Flu in humans is independent of whether the infection starts from birds on ground or migrated birds because the cause approximately the same number of people to be infected. Amazingly, when the infection starts from both sources, it causes relatively fewer-number of people to infect. This may be due to the fact that large numbers of birds get infected when infection starts from both sources leading mass death of majority of infected birds leaving just relative few infected birds to infect man. The implication of this result is that the only way to stop bird-Flu is to avoid its emergence completely, whenever it occurred the infected birds should be totally destroyed to forestall rapid spread of the disease and eventual infection of humans. Consequently, the results can help inform policies and measures that will help check outbreak of bird-Flu and curtail its spread where it is already prevalent. Also, another series of simulations were carried out using different Flu-disease transmission rates from birds-birds ($á_B$) or birds-humans ($á_H$) while its effect on the spread of the disease is examined. The results are as shown the Fig. 4 and 5.

The results in Fig. 4 and 5 showed that as the Flu-transmission rates from birds-birds and birds-humans increases, the number of the infected birds and humans' increases. This implies that if humans and birds can be nourished or genetically built to have resistance to Flu infection; then the disease transmission rate will invariably be low. Moreover, the results gives an indication that if effective vaccination can be produced for the treatment of flu either in birds or humans; the administration of such vaccines will help reduce the spread of Flu even when susceptible groups are continuously expose to the infection.

EXISTENCE AND STABILITY OF EQUILIBRUM

The disease-free equilibrium of model Eq. (1) is obtained by setting the right hand side of (1) to zero and taking all the infected terms in (1) to be zero. This gives

$$E_0 : (S_B^*, I_B^*, S_H^*, I_H^*) = \left(\frac{1}{\delta_B}(N_B\beta_B + (1-\lambda_B)M_B), 0, \frac{1}{\delta_H}N_H\beta_H, 0\right)$$

The linear stability of E_0 is established using the Jacobian. This is done by obtaining the Jacobian matrix to the model equations. The associated Jacobian is given as

$$J = \begin{bmatrix} -(\delta_B + \frac{\alpha_B I_B}{N_B}) & -\frac{\alpha_B S_B}{N_B} & 0 & 0 \\ \frac{\alpha_B I_B}{N_B} & \frac{\alpha_B S_B}{N_B} - (\delta_B + d_B) & 0 & 0 \\ 0 & -\frac{\alpha_H S_H}{N_B} & -(\delta_H + \frac{\alpha_H I_B}{N_B}) & \nu \\ 0 & \frac{\alpha_H S_H}{N_B} & \frac{\alpha_H I_B}{N_B} & -(\delta_H + d_H + \nu) \end{bmatrix}$$

The Jacobian was evaluated at E_0 and it yields

$$J^* = \begin{bmatrix} -\delta_B & -\frac{\alpha_B(N_B\beta_B + (1-\lambda)M)}{N_B\delta_B} & 0 & 0 \\ 0 & \frac{\alpha_B(N_B\beta_B + (1-\lambda)M)}{N_B\delta_B} - (\delta_B + d_B) & 0 & 0 \\ 0 & -\frac{\alpha_H N_H\beta_H}{N_B\delta_H} & -\delta_H & \nu \\ 0 & \frac{\alpha_H N_H\beta_H}{N_B\delta_H} & 0 & -(\delta_H + d_H + \nu) \end{bmatrix}$$

Table 1: Model parameters and their interpretations

Parameter	Description	Estimated value
N_B	Total number of birds in the location of interest	10000
N_H	Total number of humans in the location of interest	Variable
β_B	Average birth rate in birds	0.03
β_H	Average birth rate in humans	0.001
λ_B	Probability of infection in migrated birds	0.01
M_B	Total number of migrated birds (per day)	10
δ_H	Natural death rate in humans	1/(365×75)
δ_B	Natural death rate in birds	1/(365×2)
α_H	Infection transmission rate from birds to humans	0.1
α_B	Infection transmission rate from bird to bird	0.9
d_B	Flu-induced death rate for birds	0.99
d_H	Flu-induced death rate for humans	0.009
1/	Recovery rate for humans (per days)	1/7

After substituting the values of the parameters as given in Table 1, we obtained the eigenvalues of J^* and it was found that all the eigenvalues were strictly negative. This implies that E_0 is an attractor (i.e. sink), hence it is locally asymptotically stable. Therefore, the Bird-Flu disease can be eradicated from the birds-humans' population whenever the initial sizes of the subpopulations of the model are in the basin of the attractor (i.e. $N_B \le \frac{1}{\delta_B}(N_B\beta_B + (1-\lambda_B)M_B), N_H \le \frac{N_H\beta_H}{\delta_H}$).

CONCLUSION

In this study, we developed a mathematical model to depict the birds and humans population dynamics subject to Avian Influenza. The resulting model equations were solved numerically while situations with different infection sources and different Flu-disease transmission rates were simulated. The graphical profiles of the infected subpopulations with time were presented based on the results from our simulations. Also, the disease-free equilibrium of the system was established and analyzed for stability. It was found to be locally asymptotically stable.

REFERENCES

1. David, J.D. *et al*., 2002. Ecology and evolution of the Flu. Trends in Ecology and Evolution, 17: 7.
2. Andreasen, V. *et al*., 1997. The dynamics of co circulating influenza strains conferring partial cross-immunity. J. Math. Biology, 35: 825-842.
3. Andrei, K., 2006. Lyapunov functions and global stability for SIR and SIRS epidemiological models with non-linear transmission.
4. World Health Organization, 2005. Epidemic and Pandemic Alert and Response.
5. Bowan, C. *et al*., 2005. A Mathematical model for Assessing control strategies against West Nile virus. Bulletin of Mathematical Biology 67: 1107-1133.
6. Garnett, G.P., 2002. An introduction to Mathematical models in sexually transmitted disease epidemiology. Sex Transmission Inf. 78: 7-12.
7. Hethcote, H.W., 2000. The Mathematics of infectious disease. SIAM Rev. 42: 599 -653.
8. Mark Lewis, *et al*., 2006. Travelling waves and spread rates for a West Nile Virus Model. Bulletin of Mathematical Biology.
9. World Health Organization, 2005. Report of the meeting on avian influenza and pandemic human influenza.