Avian-human influenza epidemic model with diffusion

Kwang Ik Kim and Zhigui Lin

1) Department of Mathematics, POSTECH, Pohang 790-784, KOREA
2) School of Mathematical Science, Yangzhou University, Yangzhou 225002, P. R. China

Corresponding Author: Kwang Ik Kim, kimki@postech.ac.kr

ABSTRACT

A diffusive epidemic model is investigated. This model describes the transmission of avian influenza among birds and humans. The behavior of positive solutions to a reaction-diffusion system with homogeneous Neumann boundary conditions are investigated. Sufficient conditions for the local and global asymptotical stability are given by spectral analysis and by using Lyapunov functional. Our result shows that the disease-free equilibrium is globally asymptotically stable if the contact rate for the susceptible birds and the contact rate for the susceptible humans are small. It suggests that the best policy to prevent the occurrence of pandemic is not only to exterminate the infected birds with avian influenza and but also to reduce the contact rate for susceptible humans with the individuals infected with mutant avian influenza. Numerical simulations are presented to illustrate the main results.

1 INTRODUCTION

In the 20th century, three influenza pandemics have occurred (1918, 1957, 1968). The 1918 influenza pandemic is now estimated to have killed 50-100 million people [6,15,21]. What would be the next pandemic caused by? The most possible answer is Avian influenza.

Avian influenza has the high virulence for birds and the highly pathogenic avian influenza can cause almost 100 percent death for birds. As outbreaks of highly pathogenic avian influenza across Eurasia and Africa continue to occur, it seems that we do not have much control on those outbreaks. Avian influenza threatens to be more widespread than the SARS outbreaks that occurred in 2003. It was formerly believed that the avian influenza virus cannot infect humans, but recent reports showed that the avian influenza has now caused 385 human infections (as of June 19, 2008), with an approximate 50 percent mortality rate. Fortunately, there is still no evidence that the avian influenza can be transmitted among humans [19]. When the avian influenza virus mutates to be able to transmit among humans, we will have to worry whether this mutant avian influenza virus will cause a high death toll among humans. In this paper, we propose a model to study whether this will occur and how we can protect it to occur.

Over the last fifty years, a great attention has been paid to the mathematical description of the spread of an epidemic, see for example the earlier work [1], the book [14,17] and the references therein. The standard SIR (Kermack-McKendrik) epidemic model assumes that the disease incubation period is negligible so that each susceptible individual becomes infectious and later recovers with a permanent or temporary acquired immunity. The reader can also see some related works in [3,5,10,12,24,25]. Unlike SIR models, SEI models suppose that a susceptible individual first goes through a latent (exposed) period before becoming infectious. SEI
models have been studied intensively [2,7,20] and a typical example of SEI epidemic model is
the transmission of SARS [22] which is one of the serious problems that humans face at present.

Recently Iwami, Takeuchi and Liu proposed in [9] an avian-human influenza epidemic model
to interpret the mutation process of avian influenza. They showed that in order to prevent
spread of avian influenza in the human world, we must take the measures not only for the birds
infected with avian influenza to exterminate but also for the humans infected with mutant avian
influenza to quarantine when mutant avian influenza has already occurred.

But it must be pointed out that the system in [9] neglects any spatial structure of disease
spreading and is definitely inapplicable for moving individuals such as birds and humans. When
the distribution of the individuals is in different spatial locations, the standard method of includ-
ing the spatial effects consists in the introduction of diffusion terms and thus an extended version
of the SI-SIR avian-human epidemic model in [9] can be described by

\[
\begin{align*}
X_t - D_1 \Delta X &= c - bX - \omega XY, \\
Y_t - D_1 \Delta Y &= \omega XY - (b + m)Y, \\
S_t - D_2 \Delta S &= \lambda - \mu S - S \int_\Omega \int_{-\infty}^t K(x, y, t - s)(\beta_1 Y + \beta_2 H)(s, y)dsdy, \\
B_t - D_2 \Delta B &= \beta_1 S \int_\Omega \int_{-\infty}^t K(x, y, t - s)Y(s, y)dsdy - (\mu + d + \varepsilon)B, \\
H_t - D_2 \Delta H &= \beta_2 S \int_\Omega \int_{-\infty}^t K(x, s, y, t - s)H(s, y)dsdy + \varepsilon B - (\mu + \alpha + \gamma)H, \\
R_t - D_2 \Delta R &= \gamma H - \mu R,
\end{align*}
\]

(1.1)

for \( t > 0, x \in \Omega \) with homogeneous Neumann boundary conditions
\[
\frac{\partial X}{\partial \eta} = \frac{\partial Y}{\partial \eta} = \frac{\partial S}{\partial \eta} = \frac{\partial B}{\partial \eta} = \frac{\partial H}{\partial \eta} = \frac{\partial R}{\partial \eta} = 0, \quad t > 0, \quad x \in \partial \Omega
\]

(1.2)

and initial conditions
\[
\begin{align*}
X(0, x) &= \phi_1(x) \geq 0, \quad Y(0, x) = \phi_2(x) \geq 0, \quad S(0, x) = \phi_3(x) \geq 0, \quad x \in \overline{\Omega}, \\
B(0, x) &= \phi_4(x) \geq 0, \quad H(0, x) = \phi_5(x) \geq 0, \quad R(0, x) = \phi_6(x) \geq 0, \quad x \in \overline{\Omega}, \\
Y(t, x) &\geq 0, \quad H(t, x) \geq 0, \quad (t, x) \in (-\infty, 0) \times \overline{\Omega},
\end{align*}
\]

(1.3)

where \( \Omega \) is a bounded domain in \( \mathbb{R}^n \) with smooth boundary \( \partial \Omega, \eta \) is the outward unit normal
vector on the boundary. The positive constants \( D_1 \) and \( D_2 \) are the diffusion coefficients
for birds and humans, respectively. The homogeneous Neumann boundary condition implies that
the above system is self-contained and there is no infection across the boundary. The initial
function \( \phi_i \) is nonnegative, Hölder continuous and satisfies \( \partial \phi_i / \partial \eta = 0 \) on the boundary. The terms
\[
\int_\Omega \int_{-\infty}^t K(x, y, t - s)Y(s, y)dsdy, \quad \int_\Omega \int_{-\infty}^t K(x, y, t - s)H(s, y)dsdy
\]
account for the infection of individuals to their present position at time \( t \) caused by the infected
individuals from all possible positions at all previous times [23]. The kernel \( K(x, y, t) \) depends
on both the spatial and the temporal variables. Here we further assume that
\[
K(x, y, t) = G(x, y, t)k(t) \geq 0, \quad x, y \in \Omega, \quad t > 0,
\]
\[
\int_\Omega G(x, y, t)dx = \int_\Omega G(x, y, t)dy = 1, \quad \int_0^{+\infty} k(t)dt = 1, \quad tk(t) \in L^1((0, +\infty); R).
\]
For example, \( G(x, y, t) \) is Green’s function of the operator \( \frac{\partial}{\partial t} - D_2 \Delta \) subject to homogeneous Neumann boundary condition, and \( k(t) = \frac{1}{\tau} e^{-t/\tau} \) with a constant \( \tau \).

In this paper, we treat an avian-human influenza epidemic model with diffusion terms in a bounded domain \( \Omega \) and investigate the asymptotic behaviors of the reaction diffusion system. In the next section, uniform bounds for the solutions of (1.1)-(1.3) are given. In Section 3, local and global stabilities of the equilibria for the bird system are discussed and Section 4 deals with asymptotical behaviors of the solutions to the full system. Numerical simulations are given in Section 5 and we end the paper with a discussion section.

Acknowledgements. This work is supported by the POSTECH BSRI Research Fund-2008, by PRC grant NSFC 10671172 and also by “Blue Project” of Jiangsu Province.

REFERENCES