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Dynamical Network Transmission of H1N1 Virus at the Local Level Transmission Model

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***Abstract*—**A new strain of Type A influenza virus can cause the transmission of H1N1 virus. This virus can spread between the people by coughing and sneezing. Because the people are always movement, so this virus can be easily spread. In this study, we construct the dynamical network model of H1N1 virus by separating the human into five groups; susceptible, exposed, infectious, quarantine and recovered groups. The movement of people between houses (local level) is considered. The behaviors of solutions to our dynamical model are shown for the different parameters.

***Keywords*—**Dynamical network, H1N1virus, local level, simulation.

I. Introduction

H1N1 virus is an orthomyxovirus. This virus has three groups, called as type A, type B or type C. Only type A viruses can infect pigs. This type of virus is also can be subdivided into subtypes based on their hemaglutinin (H) and neuraminidases (N). In humans, there are 15 hemaglutinins (H) and 9 neuramindases (N) that have been identified in humans, animals and birds. Because it is an RNA virus, antigenic drift can occur as genetic material is exchanged between viruses. The pig populations can be infected with three main subtypes of classical swine influenza virus; H1N1, H3N2, and H1N2 [1]. "Pandemic Influenza", "Novel Influenza" or "Swine Flu" is occurred from the subtype identified H1N1 (2009). This type of influenza virus can cause respiratory disease in human. The spread of a swine flu virus from pigs to humans is occasionally possible. However, sporadic human infections with swine flu have occurred. Generally, these cases occur in persons with direct exposure to pigs [2]. Factors that may influence to the transmission of swine flu between human include smoking and, especially, not wearing gloves when working with infected animals. However, food is not the factor of H1N1 virus transmission. Eating cooked pork products are not risk to the infection of this disease. Cooking pork to an internal temperature of 160 ^{o}F can kill the swine flu virus as it can kill other bacteria and viruses. The swine flu virus can spread between the human by coughing and sneezing of the infected people. The virus transmits when droplets from a cough or sneeze of an infected person are propelled through the air and deposited on the mouth or nose of people nearby (within approximately one metre). The virus also is on surfaces in the surrounding environment and can live on a hard surface for up to 24 hours and a soft surface for about 20 minutes.

When the person touches droplets of the virus on surfaces and then touches their own mouth or nose before washing their hand, it usually takes 1 to 4 days before each person is sick. Infected adults may transmit the disease to the others people for one day before symptoms appear and up to seven or more days after becoming sick. The younger children might be contagious for longer periods [3]. The swine flu virus has symptoms similarly to the general influenza-like illness. Fever, cough, sore throat, muscle pain, headache, runny nose, chills and fatigue are symptoms of this disease. Some people with swine flu also have reported vomiting and diarrhea associated with swine flu. Swine flu may take chronic medical conditions worse [4-8]. This influenza virus was first proposed to be a disease related to human influenza during the 1918 flu pandemic, when pigs become sick at the same time as human. Ten years later, the first identification of an influenza virus as a cause of disease in pigs occurred in the United States in 1930 [9]. Since that time, they have become an economically important cause of respiratory disease in pigs throughout the world and a human public health risk. For the next 60 years, swine influenza strains were almost exclusively H1N1. Then, between 1997 and 2002, new strains of three different subtypes and five different genotypes appeared as causes of influenza among pigs in North America. In 2009, the first case of the current H1N1 (2009) virus reported to WHO on 24 April 2009 on the American continent. The virus has spread in 160 countries and territories. By mid-year of 2009, there were 135,000 cases and 816 deaths recorded. The H1N1 (2009) virus has spread from the American continent to their world regions, including Europe, the Middle East, Asia, the Pacific and Africa. In 2003, D. Klinkenberg, A.Everts-van der Wind, et al. [10] assessed the effect of control strategies with and without vaccination by presenting a mathematical model of CSFV transmission between pig herds. In 2011 [11], we analyzed the mathematical model of H1N1 virus by classifying the patients into symptomatic and asymptomatic cases. Recently [12], we modified the transmission model of H1N1 virus by including the effect of re-infection and presented the way for reducing the outbreak of this disease. In this paper, we formulate the dynamical model for the transmission of H1N1 virus when there is the movement of people between houses.

II. Transmission Model

Transmission of H1N1 virus is studied through dynamical modeling with the movement of people. The human

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population is divided into five classes; susceptible, exposed, infectious, quarantine and recovered classes. The transmission of H1N1 virus at the local level (between houses) is studied. The movement of human between houses is satisfied the following assumptions:

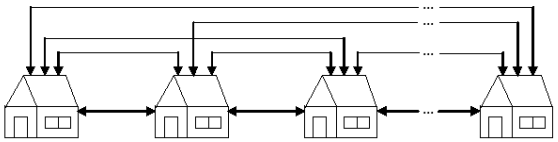

Fig. 1 The diagram shows the movement of human between houses

We assume that in each village has N populations. The movement of people is considered by random process. This process is done by random the 1st person to the N th person (with uniformly distribution) travel between houses everyday. This village, all people can go only one time in one house per day. Everybody has the same probability of visiting to each house. There is no person come from the outside of this village. Each person who stays in any house at the beginning time will come back to his/her house at the ending time. At the first day, there is only one infected human in one house and there is no infected human for the other houses in the village. Our dynamical model considers the transmission of H1N1 virus between the people. The people are divided into 5 classes; susceptible, exposed, infectious, quarantine and recovered humans.

The variables and parameters in our model are defined as follows:

$S_{t,i}$ is number of susceptible persons in the i^{th} house after visited at day t,

$E_{t,i}$ is number of exposed persons in the i^{th} house after visited at day t,

$I_{t,i}$ is number of infectious persons in the i^{th} house after visited at day t,

$Q_{t,i}$ is number of quarantine persons in the i^{th} house after visited at day t,

$R_{t,i}$ is number of recovered persons in the i^{th} house after visited at day t,

M is the total number of persons,

n is the total number of houses,

γ is the transmission rate of H1N1 virus between people,

IIP is the incubation period of H1N1 virus,

a is rate at which the infectious human change to be the quarantine human,

b is rate at which the quarantine human change to be the recovered human,

f is rate at which the infectious human change to be the recovered human,

c is rate at which the exposed human change to be the recovered human,

We assume that the total human is constant. The dynamical changes for human classes are described as follows:

$$\Delta S_{t,i} = -\gamma S_{t,i}(E_{t,i} + I_{t,i}) \quad (1)$$

$$\Delta E_{t,i} = \gamma S_{t,i}(E_{t,i} + I_{t,i}) - \frac{1}{IIP} E_{t,i} - cE_{t,i} \quad (2)$$

$$\Delta I_{t,i} = \frac{1}{IIP} E_{t,i} - fI_{t,i} - aI_{t,i} \quad (3)$$

$$\Delta Q_{t,i} = aI_{t,i} - bQ_{t,i} \quad (4)$$

$$\Delta R_{t,i} = fI_{t,i} + bQ_{t,i} + cE_{t,i} \quad (5)$$

III. Numerical Solutions

We simulate the results by using numerical analysis. Simulations of the model are calculated to see the time distributions of this disease. The time distributions of exposed ed and dormant humans for the different situations are shown in the following figures.

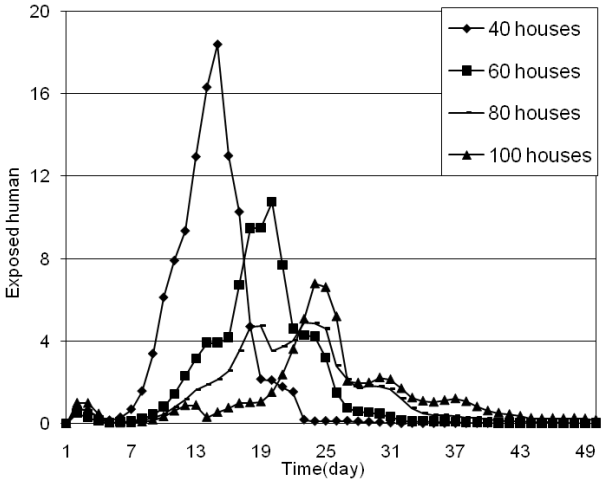

Fig. 2 Model outputs display the time distribution of exposed human for the different number of house in each village. The parameters used in this study are N = 100, γ=0.25, IIP = 5, a = 1/8, b = 1/6, c = 1/7, f = 1/10

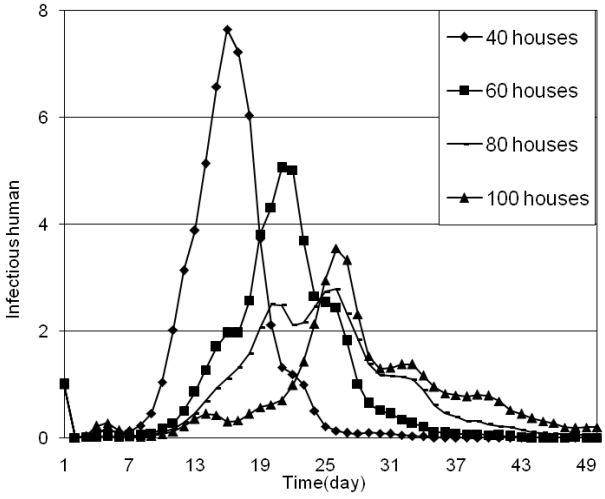

Fig. 3 Model outputs display the time distribution of infectious human for the different number of house in each village. The parameters are same as in Fig. 2

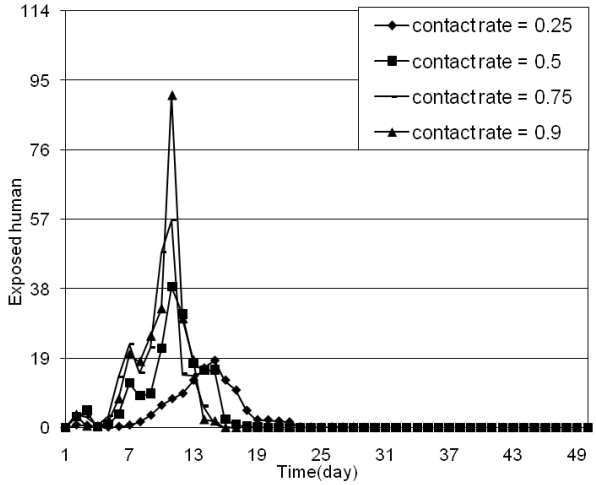

Fig. 4 Model outputs display the time distribution of exposed human for the different contact rate in each village. The parameters used in this study are N = 100, n = 40, IIP = 5, a = 1/8, b = 1/6, c = 1/7, f = 1/10

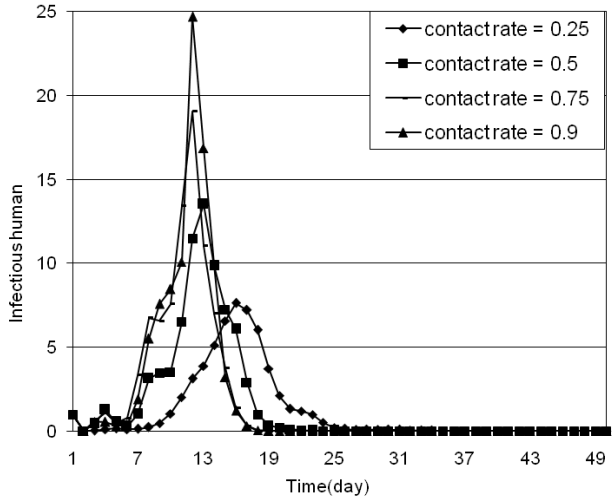

Fig. 5 Model outputs display the time distribution of infectious human for the different contact rate in each village. The parameters are same as in Fig. 4

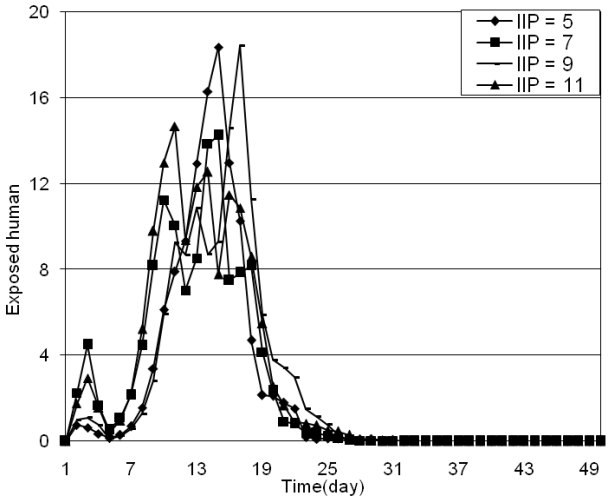

Fig. 6 Model outputs display the time distribution of exposed human for the different incubation period of H1N1 virus in each village. The parameters used in this study are N = 100, n = 40, γ=0.25, a = 1/8, b = 1/6, c = 1/7, f = 1/10

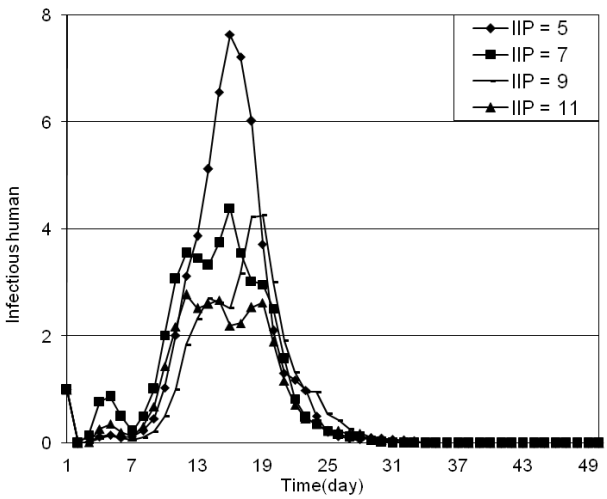

Fig. 7 Model outputs display the time distribution of infectious human for the different incubation period of H1N1 virus in each village. The parameters are same as in Fig. 6

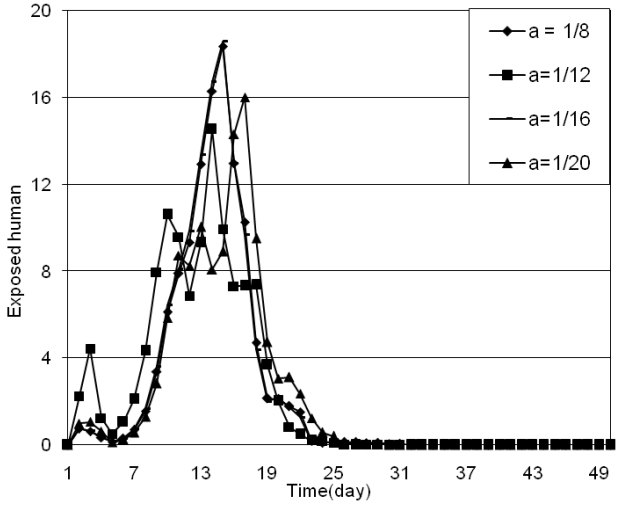

Fig. 8 Model outputs display the time distribution of exposed human for the different rate at which the infectious human change to be the quarantine human in each village. The parameters used in this study are N = 100, n = 40, γ=0.25, IIP = 5, b = 1/6, c = 1/7, f = 1/10

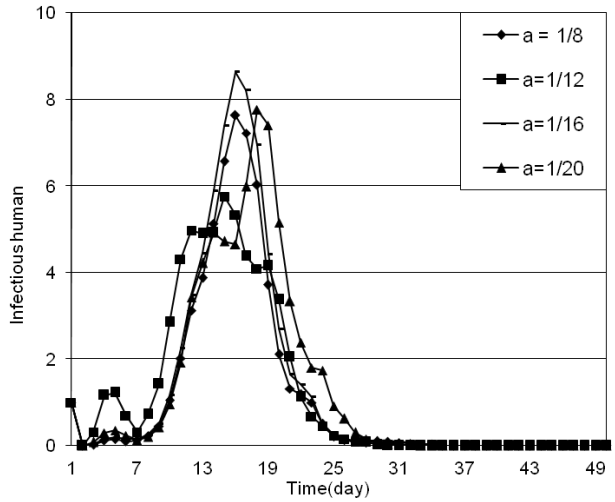

Fig. 9 Model outputs display the time distribution of infectious human for the different rate at which the infectious human change to be the quarantine human in each village. The parameters are same as in Fig. 8

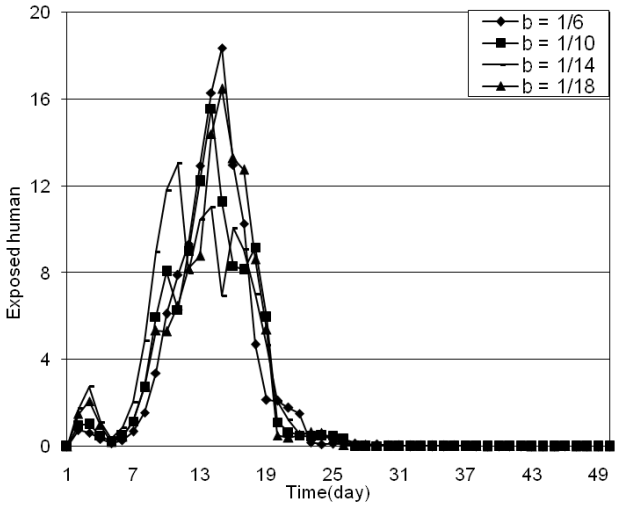

Fig. 10 Model outputs display the time distribution of exposed human for the different rate at which the quarantine human change to be the recovered human in each village. The parameters used in this study are N = 100, n = 40, γ=0.25, IIP = 5, a = 1/8, c = 1/7, f = 1/10

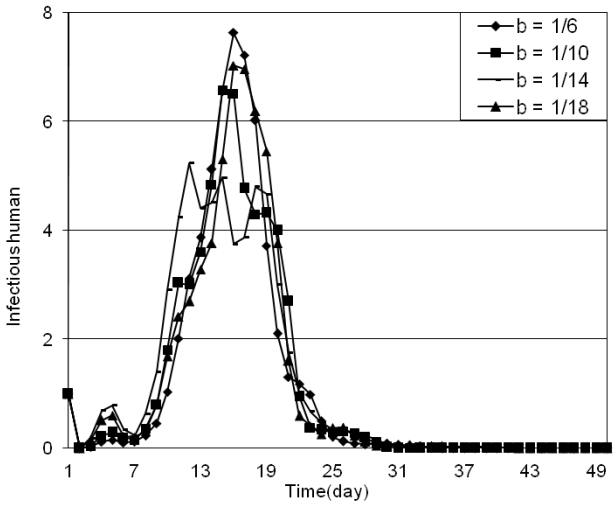

Fig. 11 Model outputs display the time distribution of infectious human for the different rate at which the quarantine human change to be the recovered human in each village. The parameters are same as in Fig. 10

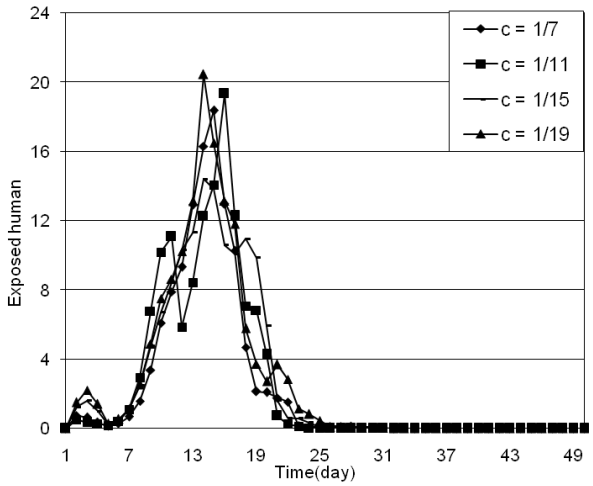

Fig. 12 Model outputs display the time distribution of exposed human for the different rate at which the exposed human change to be the recovered human in each village. The parameters used in this study are N = 100, n = 40, γ=0.25, IIP = 5, a = 1/8, b = 1/6, f = 1/10

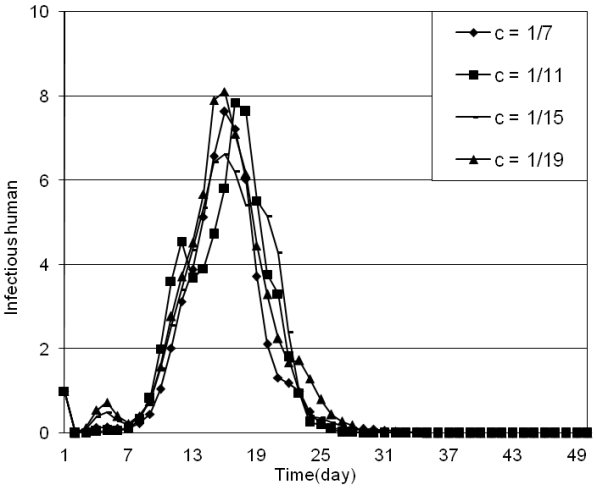

Fig. 13 Model outputs display the time distribution of infectious human for the different rate at which the exposed human change to be the recovered human in each village. The parameters are same as in Fig. 12

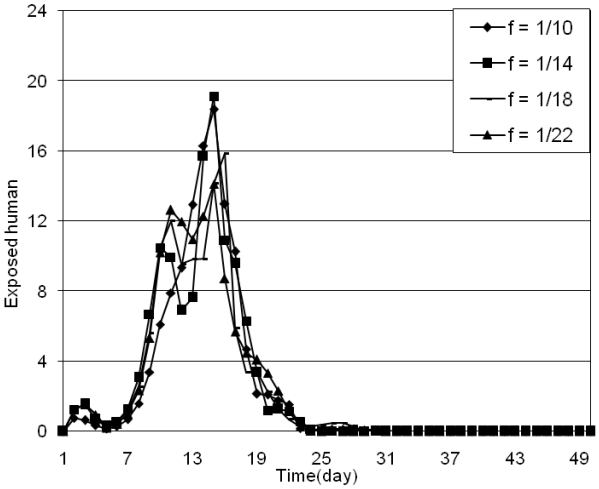

Fig. 14 Model outputs display the time distribution of exposed human for the different rate at which the infectious human change to be the recovered human in each village. The parameters used in this study are N = 100, n = 40, γ=0.25, IIP = 5, a = 1/8, b = 1/6, c = 1/7, f = 1/10

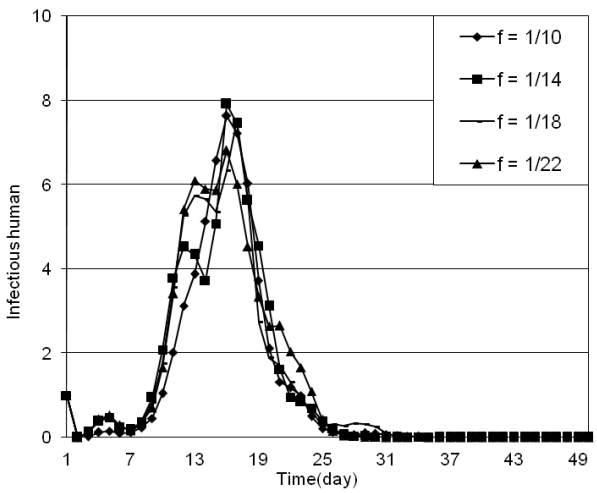

Fig. 15 Model outputs display the time distribution of infectious human for the different rate at which the infectious human change to be the recovered human in each village. The parameters are same as in Fig. 14

IV. Discussion and Conclusion

The results of the simulations are shown when different values of several parameters are used. Fig. 2 to 3 show the time distributions of exposed and infectious human when the number of houses in each village is difference. Fig. 4 to 5 show the time distributions of exposed and infectious human when the contact rate in each village is difference. Fig. 6 to 7 show the time distributions of exposed and infectious human when the different incubation period of H1N1 virus in each village is difference. Fig. 8 to 9 show the time distributions of exposed and infectious human when the different rate at which the infectious human change to be the quarantine

human in each village is difference. Fig. 10 to 11 show the time distributions of exposed and infectious human when the different rate at which the quarantine human change to be the recovered human in each village is difference. Fig. 12 to 13 show the time distributions of exposed and infectious human when the different rate at which the exposed human change to be the recovered human in each village is difference. Fig. 14 to 15 show the time distributions of exposed and infectious human when the different rate at which the infectious human change to be the recovered human in each village is difference. We will see that the epidemic sizes are higher when the smaller number of households, the higher contact rates, the smaller incubation period of H1N1 virus, the smaller day at which the infectious human change to be the quarantine human, the smaller day at which the quarantine human change to be the recovered human. But when the higher day at which the exposed human change to be the recovered human and the higher day at which the infectious human change to be the recovered human, the outburst of epidemic are longer. The next step in this study is to formulate a network of villages in a province and perform similar simulations of dynamics of the transmission of H1N1 at the next level. The results of this study will be the way for finding the parameters which effects to the epidemic of H1N1 virus when there is the movement of people.

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REFERENCES

[1] S. A. Dee, "Respiratory Disease of Pigs," In The Merck Veterinary Manuath 9th edition. Pensylvania: National Publishing Inc, 2005,pp. 1228.
[2] CDC, Influenza (Flu): Key Facts about Swine Influenza (Swine Flu), December 6, 2006.Available: http://www.cdc.gov/H1N1flu/qa.htm.
[3] Massachusetts Department of Public Health, "H1N1 Flu (Swine Flu)," Public Health Fact Sheet, 2009.
[4] Massachusetts Department of Public Health, "H1N1 Flu (Swine Flu)," Public Health Fact Sheet, 2009.
[5] Centers for Disease Control and Prevention (April 26, 2009), CDC Health Update: Swine Influenza A (H1N1) Update: New Interim Recommendations and Guidance for Health Directors about Strategic National Stockpile Materiel, Health Alert Network. Available: http://www.cdc.gov/swineflu/HAN/042609.htm.
[6] WHO, What is the pandemic (H1N1) 2009 virus?. Available: http://www.who.int/csr/disease/swineflu/ frequently_ asked_questions /about_disease/en/index.html
[7] WHO, Antiviral drugs for pandemic (H1N1) 2009: definitions and use. Available : http://www.who.int/csr/disease/swineflu/frequently_asked_questions/antivirals/definitions_use/en/index.html
[8] BMJ Group, Swine flu. Available: http://besttreatments.bmj.com/btuk/about/12.html.]
[9] WHO. Pandemic (H1N1) 2009-update 70. (Online) 2009.Available:http//www.who.int/csr/disease/swinflu/en/.
[10] D. Klinkenberg, A . Everts-van der Wind, and et al., "Quantification of the effect of control strategies on classical swine fever epidemics," Math BioSci, vol. 2, pp. 145-173, 2003.
[11] P.Pongsumpun, "Mathematical model of the symptomatic and asymptomatic infections of Swine flu," Int. J. Math. Model and methods in applied Sciences, vol. 2, pp. 247-254, 2011.
[12] P.Pongsumpun, "The Reinfectious Model of H1N1 Virus Transmission," Proceedings of World Academy of Science, Engineering and Technology, Issue 66, pp.240-246, 2012.

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