SUPER-SPREADING MODEL FOR RESPIRATORY DISEASES

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Abstract - The respiratory diseases are found around the world. The super-spreading of the virus can occur when there is the gathering of the people, and then the virus can spread easily between people. In this paper, we formulate and analyze the mathematical model of respiratory disease considering the super-spreading of the virus. The analytical results of our model are shown by using standard dynamical modeling method. The numerical simulations are presented in this study.

Index Terms - Mathematical model, respiratory disease, standard dynamic modeling method, transmission, virus

I. INTRODUCTION

Respiratory disease refers to respiratory infections. This is caused by many viruses such as influenza virus, parainfluenza virus, respiratory syncytial virus (RVS). SARS and MERS are two respiratory diseases that can have the super-spreading of virus. Severe Acute Respiratory Syndrome or SARS was first occurred in Guangdong Province, China in November 2002. The spreading of virus was rapid and occurred 29 countries around the world. The spreading of virus was occurred from 1 November 2002 to the end of the epidemic on 31 July, 2003. There were 8,096 patients and 774 deaths, 9.6% of patients were death. 21% of all patients were infected from take care the patients who infected with the virus [1]. Middle East respiratory syndrome coronavirus (MERS-CoV) is a new species of coronavirus that caused Middle Eastern respiratory disease. The report of this disease was first reported in 2012.

After the genome sequence of the virus isolated from the sputum samples of patients who are sick in the new influenza outbreak in 2012, the original name of the virus is New Coronal Virus. In June 2014, MERS patients were reported in 22 countries, including Saudi Arabia, Malaysia, Jordan, Qatar, Egypt, United Arab Emirates, Kuwait, Turkey, Oman, Algeria, Bangladesh, Indonesia (not yet confirmed), Austria, United Kingdom kingdom, South Korea and the United States [2]. The latest outbreak occurred in South Korea. According to a report on June 12, 2015, there were 126 confirmed cases of death, 13 of whom were under surveillance. This is the largest outbreak of this disease, except the outbreak in Saudi Arabia [3]. The spreading of MERS in South Korea was called as super-spreading. The spreading of MERS virus was rapid and many people were infected with MERS. The outbreak was happened from the Korean who travelled from the Middle-east countries and can transmit the MERS virus to the other 30 persons in 2 weeks. Thailand has the risk of infection with MERS and the other emerging diseases [4,5]. Coronal virus has many strains. Some strains are infected viruses. There are some species that are infectious in pets and wildlife such as camels and bats, etc. Later, the virus has developed to spread the infection to humans. The first infected patient in Saudi Arabia in 2012. In case of chills, coughing is uncomfortable. Eventually, it may die. Symptoms of the disease vary from mild to severe. About 20 percent do not show symptoms. In 2014, Chowell et al.[6] studied and constructed the transmission model of MERS by considering the MERS data during April to October in 2013. They also considered the basic reproductive rate and the severe of the disease. After that, Gardner et al.[7] studied the spreading of MERS found in the Arabian Peninsula, Europe, North America, Southeast Asia, the Middle East and the United States. They considered the nature of the outbreak and the source of the disease. In this study, we consider the super-spreading of the virus. We formulate and analyze the mathematical model for describing the spreading of respiratory virus. The dynamical modeling method is used in this study. .

II. TRANSMISSION MODEL

In this model, we consider the super-spreading and regular spreading of virus. The human is separated into 5 classes such as susceptible, exposed, regular infected, super-spreading infected and recovered human.

We define the variables and parameters as follows:

S = Number of susceptible human population,
E = Number of exposed human population,
I_r = Number of regular infected human,
I_s = Number of super-spreading infected human,
R = Number of recovered human population
I = Total infected human population,
$I = I_r + I_s$,
R = Number of recovered human population,
b = Birth rate of human population,
N = Total human population,
μ = Death rate of human population,
β = Transmission rate of virus between human,
IIP = Incubation time of virus in human,
p = Probability that a new case will be a regulated infected human,

(1-p) = Probability that a new case will be a super-spreading infected human,

r_1 = Recovery rate of a regular infected human,

r_2 = Recovery rate of a super-spreading infected human.

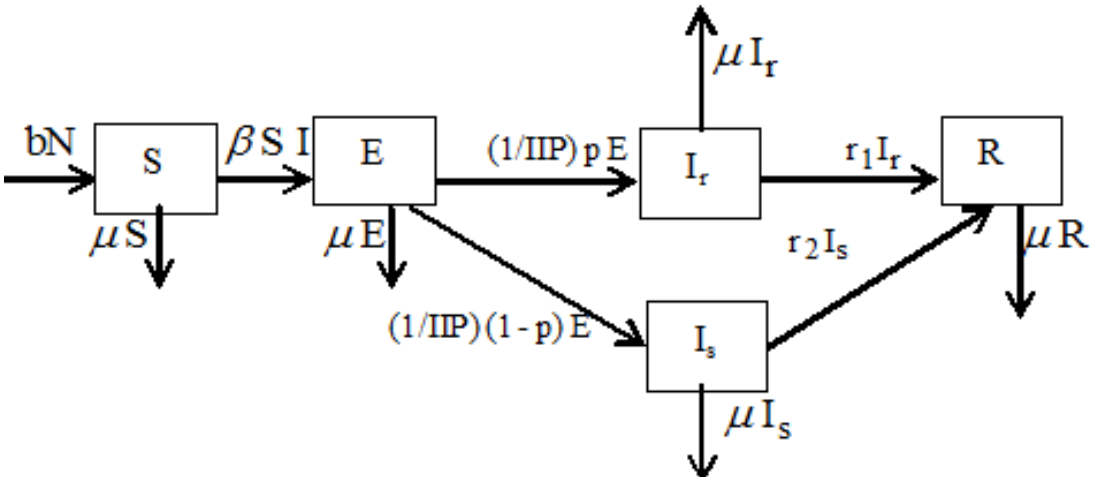


Figure 1. Diagram of our model

From figure1, we can write the dynamical equations as follows:

$$\frac{d}{dt}S = bN - \mu S - \beta SI \quad (1)$$

$$\frac{d}{dt}E = \beta SI - (1/IIP)\,p\,E - \mu E - (1/IIP)(1-p)E \quad (2)$$

$$\frac{d}{dt}I_r = (1/IIP)\,p\,E - \mu I_r - r_1 I_r \quad (3)$$

$$\frac{d}{dt}I_s = (1/IIP)(1-p)\,E - \mu I_s - r_2 I_s \quad (4)$$

$$\frac{d}{dt}R = r_1 I_r + r_2 I_s - \mu R \quad (5)$$

where

$$N = S + E + I_r + I_s + R.$$

We suppose that the total population is constant, then the rate of change for total population equals to zero. Thus we can have

$$\frac{d}{dt}N = \frac{d}{dt}(S + E + I_r + I_s + R) = 0 \text{ or } b = \mu.$$

The reduced equations are found by setting the following normalized variables:

$$s = \frac{S}{N}, e = \frac{E}{N}, i_r = \frac{I_r}{N}, i_s = \frac{I_s}{N}, r = \frac{R}{N}.$$

Thus, we can have

$$\frac{d}{dt}s = b - (\mu + \beta(i_r + i_s)N)s, \quad (6)$$

$$\frac{d}{dt}e = -e\ ((1/IIP) + \mu) + \beta(i_r + i_s)Ns \quad (7)$$

$$\frac{d}{dt}i_r = e\,p\,(1/IIP) - i_r(\mu + r_1) \quad (8)$$

$$\frac{d}{dt}i_s = e\,(1-p)\,(1/IIP) - i_s(\mu + r_2) \quad (9)$$

With $r = 1 - (s + e + i_r + i_s)$.

A. Analytical Results

i) Steady states:

Steady states are found from setting the rate of change for each variable to zero. The two steady states are

$E_0 = (1,0,0,0)$ and $E_1 = (s^*, e^*, i_r^*, i_s^*)$ where

$$s^* = \frac{(1 + IIP\mu)(\mu + r_1)(\mu + r_2)}{\beta N(\mu + r_1 + p(r_2 - r_1))}, \quad (10)$$

$$e^* = -\frac{(IIP(IIP\mu^4 + b\beta N((p-1)r_1 - pr_2) + \mu(-b\beta N + r_1 r_2) + \mu^2(r_1 + r_2 + IIP r_1 r_2) + \mu^3(1 + IIP(r_1 + r_2))))}{\beta(1 + IIP\mu)N(\mu + r_1 + p(r_2 - r_1))}, \quad (11)$$

$$i_r^* = -\frac{(p(IIP\mu^4 + b\beta N((p-1)r_1 - pr_2) + \mu(-b\beta N + r_1 r_2) + \mu^2(r_1 + r_2 + IIP r_1 r_2) + \mu^3(1 + IIP(r_1 + r_2))))}{\beta(1 + IIP\mu)N(\mu + r_1)(\mu + r_1 + p(r_2 - r_1))}, \quad (12)$$

$$i_s^* = (p-1)\left(-\frac{b}{(1 + IIP\mu)(\mu + r_2)} + \frac{\mu(\mu + r_1)}{\beta N(\mu + r_1 + p(r_2 - r_1))}\right). \quad (13)$$

ii) Local stability of steady states:

The characteristic of each steady state can be determined from the sign of all eigenvalues for each steady state. If all eigenvalues have negative real parts, then the steady state will be local stable.

The eigenvalues are found from solving the characteristic equation: $\det(J - \lambda I) = 0$

where J is the Jacobian matrix evaluated at each steady state and I is the identity matrix.

First steady state: E_0 = (1,0,0,0).

Jacobian matrix is given as

$$\begin{pmatrix} -\mu & 0 & -\beta N & -\beta N \\ 0 & -\frac{1+IIP\mu}{IIP} & \beta N & \beta N \\ 0 & \frac{p}{IIP} & 0 & -\mu - r_1 \\ 0 & \frac{1-p}{IIP} & -\beta N & \beta N \end{pmatrix}.$$

The characteristic equation is given as

$$(\lambda+\mu)(\lambda^3 + a_2\lambda^2 + a_1\lambda + a_0) = 0 \qquad (14)$$

where

$$a_2 = \frac{1}{IIP} + \mu + \beta N, \qquad (15)$$

$$a_1 = -\beta N r_1, \quad a_0 = \frac{\beta N(IIP\,\mu + p)(\mu + r_1)}{IIP}. \qquad (16)$$

From Routh Hurwitz, the eigenvalues have negative real parts when it satisfies the following conditions [8].

i) $a_2 > 0$ (17)

ii) $a_0 > 0$ (18)

iii) $a_2 a_1 > a_0$. (19)

From our evaluations, (17)-(19) are satisfied for $X_0 < 1$,

where $X_0 = r_1 \frac{(1-p) + \beta\, IIP\, N}{\mu\,(IIP\,\mu + p)}$. (20)

Second steady state: E_1 = (s^*, e^*, i_r^*, i_s^*) where s^*, e^*, i_r^*, i_s^* are defined in (10)-(13).

Jacobian matrix is defined by

$$\begin{pmatrix} -\mu - \beta(i_r^* + i_s^*)N - \lambda & 0 & -\beta N & -\beta N \\ \beta(i_r^* + i_s^*)N & -\frac{1+IIP\,\mu}{IIP} & \beta N & \beta N \\ 0 & \frac{p}{IIP} & 0 & -\mu - r_1 \\ 0 & \frac{1-p}{IIP} & -\beta N & \beta N \end{pmatrix}.$$

The characteristic equation is given as

$$(\lambda^4 + c_3\lambda^3 + c_2\lambda^2 + c_1\lambda + c_0) = 0$$

(21)

where

$$c_3 = \frac{1}{1+IIP\mu}\left(\frac{\frac{(1+IIP\mu)^2(p+IIP\mu)}{IIP} + (1+IIP\mu)^2 r_1 + \frac{b\beta N p^2}{(\mu+r_1)}}{p} + \frac{b\beta N(1-p)}{(\mu+r_2)} + \frac{(1+IIP\mu)^2(-1+p)(\mu+r_1)^2}{p(\mu+r_1-pr_1+pr_2)} \right),$$

(22)

$$c_2 = \frac{-IIP(1+IIP\mu)r_1(\mu+r_1)^2(\mu+r_2)^2 + b\beta N(\mu+r_1-pr_1+pr_2)}{IIP(\mu+r_1)(\mu+r_2)(\mu+r_1-pr_1+pr_2)} \cdot \frac{(\mu+IIP\mu^2+r_1+IIP\mu r_1-pr_1+(p+IIP(\mu+r_1))r_2)}{},$$

(23)

$$c_1 = -\frac{1}{IIP(\mu + r_1(1-p) + pr_2)}$$

$$\left(\begin{array}{l} IIP^2\mu^5 + pr_1^2 r_2 + IIP\mu^4(2 + p + 2IIPr_1 \\ + IIP^2 r_1^2 + IIP(2 + p + 2IIPr_1)r_2) \\ + \mu^2(r_2 + pr_2 + IIPr_1^2(1 + p + IIPr_2) + r_1 \\ (1 + 2p + IIP(3 + p)r_2)) + \mu(b\beta N(-1 + IIPr_1) \\ + r_1(r_2 + IIPr_1 r_2 + p(r_1 + 2r_2 + IIPr_1 r_2))) \end{array} \right),$$

(24)

$$c_0 = \frac{1}{IIP(\mu + r_1(1-p) + pr_2)}(1+IIP\mu)(\mu+r_1)(b\beta\mu N + \mu^3(-1+p) + \mu(-1+p)r_1 r_2 + \mu^2(-1+p)(r_1+r_2) + b\beta N(r_1(1-p) + pr_2)).$$

(25)

By Routh Hurwitz theorem, All four eigenvalues have negative real parts when the following conditions are satisfied:

i) $c_3 > 0, c_1 > 0, c_0 > 0$ (26)

ii) $c_1 c_2 c_3 > c_1^2 + c_3^2 c_0$. (27)

After calculations, the endemic steady state will be local stable for $X_0 > 1$, where X_0 is defined in (20). Next section, we will show the numerical results of our model.

B. Numerical Results

From our analytical results, The threshold condition is defined as

$$X_0 = r_1 \frac{(1-p) + \beta\, IIP\, N}{\mu\,(IIP\,\mu + p)}.$$

If X_0 > 1, the endemic steady state will be local stable and the disease free state will be local stable when X_0 < 1.

The disease free state means the state which has no individual infectious. We consider the case when there

is the outbreak of the respiratory disease. Then, we consider the numerical solutions of the endemic areas. The parameters in our simulations are as follows: b = 1/(365 x 65) corresponding to the 65 years of life cycle for human population, N = 100, μ = 1/(365 x 65) corresponding the equivalence between birth and death rate, β = 0.25, p = 0.8 are arbitrary chosen, IIP = 7 satisfy the 7 days of incubation period for the virus,

r_1 = 1/14 , r_2 = 1/25 corresponding to the 14 days of recovery for regular infected and 25 days for super-spreading infected human and X_0 = 370,990.

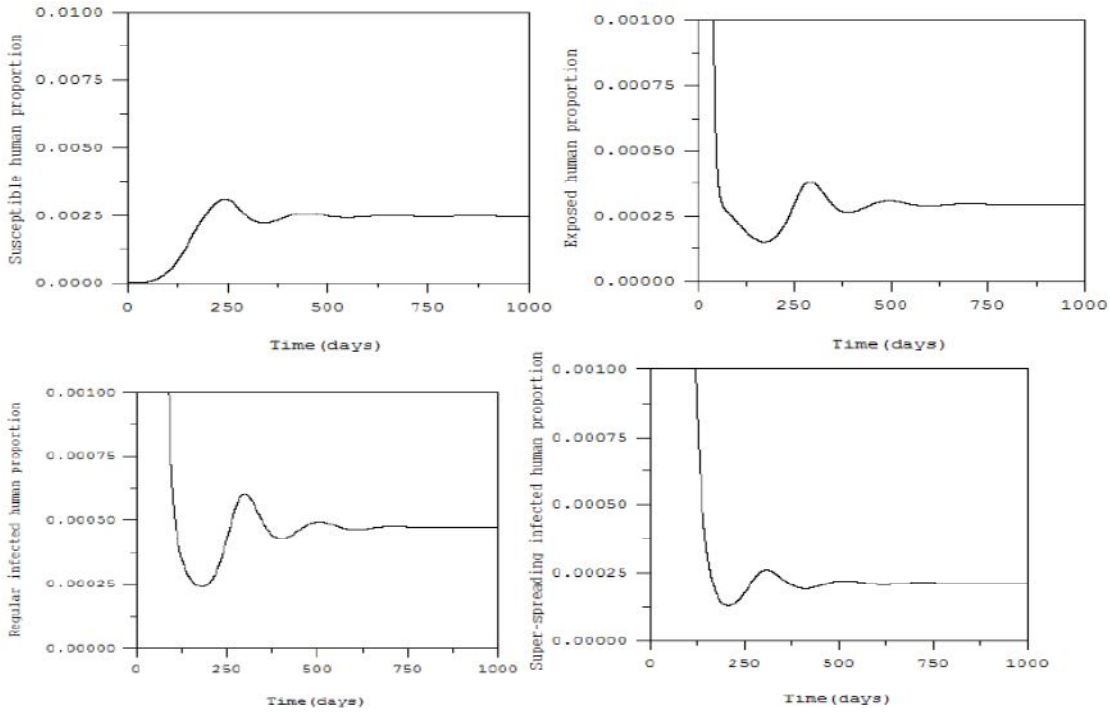


Figure2. Time series of our model for X_0 = 370,990.

Next, we consider the numerical simulations of each variable when we decrease the value of X_0.

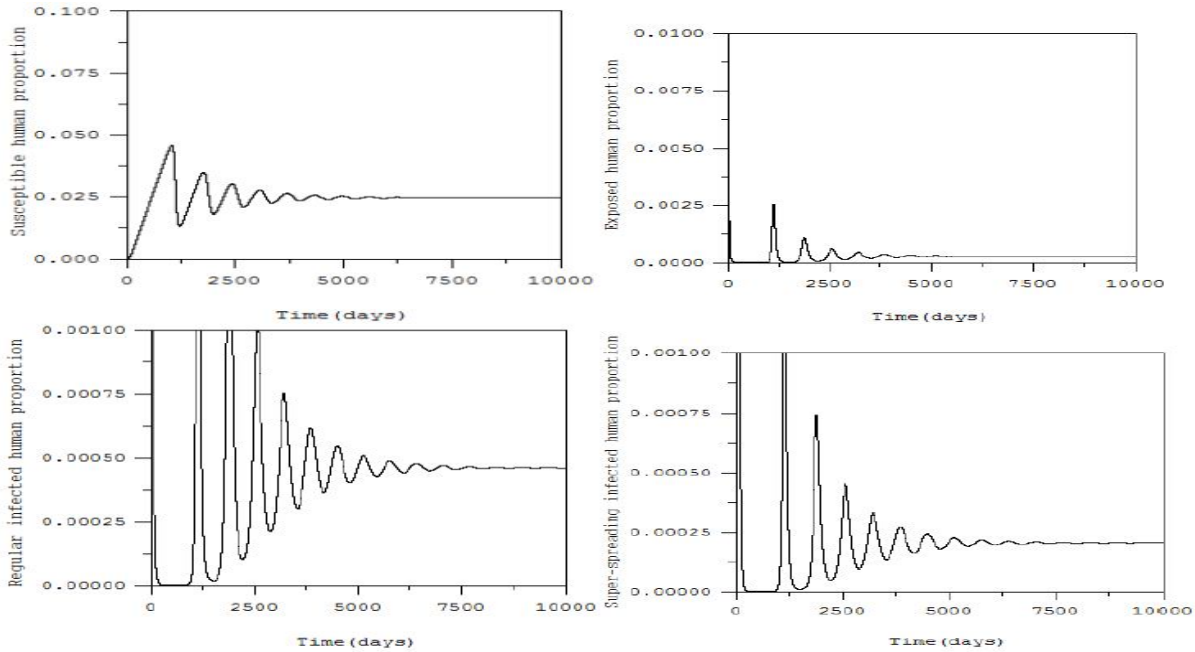


Figure3. Time series of our model for X_0 = 1758.

From figure2 and figure3, we can see that the time series of individual variable converges to the endemic steady state for both two cases. We note that the time of convergences for the less value of X_0 is longer than the great value of X_0.

III. DISCUSSION AND CONCLUSION

In this study, we consider the super-spreading and the regular spreading of the virus for the transmission of respiratory diseases. The threshold parameter is defined as

$$X_0 = r_1 \frac{(1-p) + \beta \, IIP \, N}{\mu \, (IIP \, \mu + p)}.$$

If $X_0 < 1$, the disease free steady state will be local stable and the endemic steady state will be local stable for $X_0 > 1$. The geometric mean of X_0 is described as the basic reproductive of the disease. This indicates the number of secondary cases produced from primary infection. Furthermore, we compare the numerical solutions when there are the different values of the transmission rate and the total human population. The results are given in the following figures.

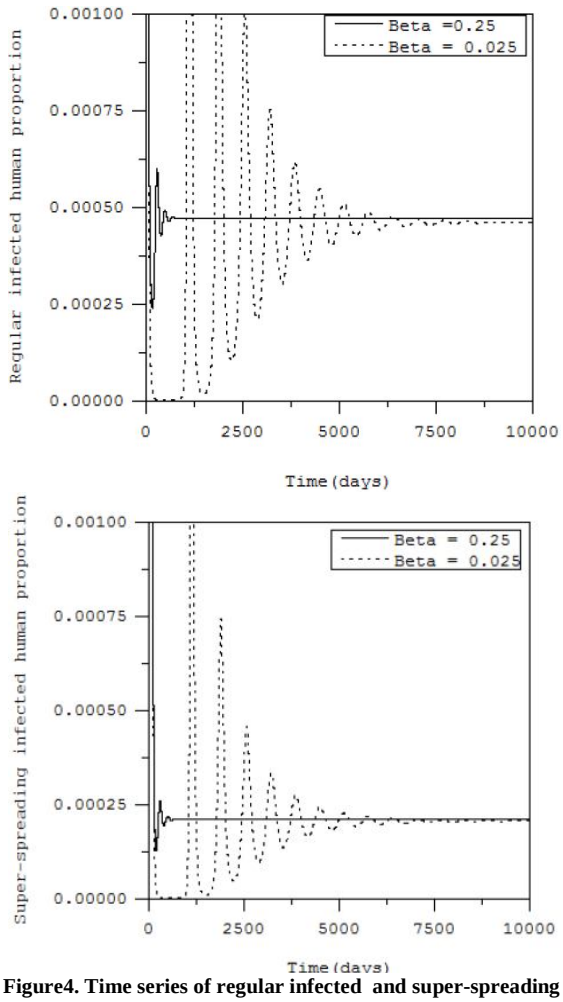


Figure4. Time series of regular infected and super-spreading infected human when there is the different transmission rate.

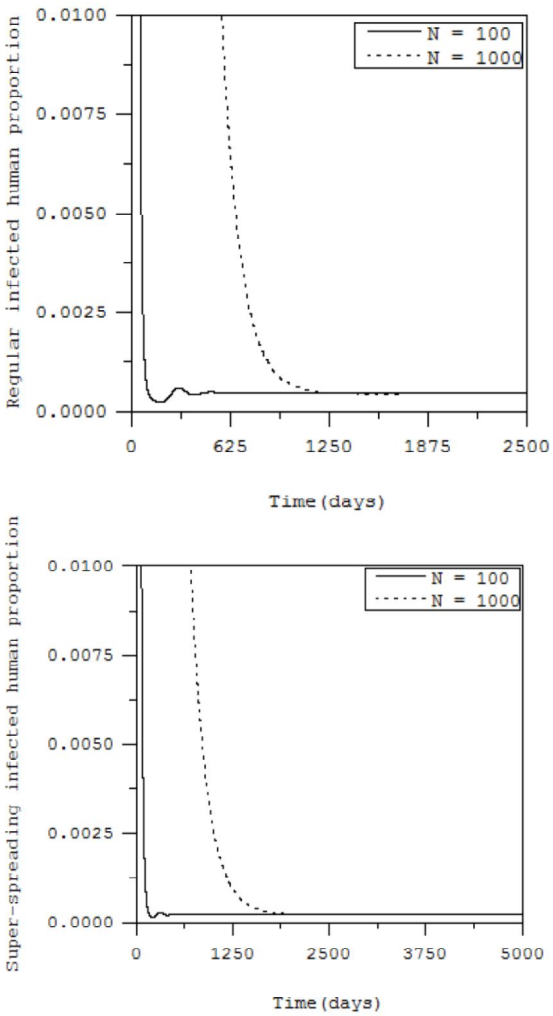


Figure5. Time series of regular infected and super-spreading infected human when there is the different total human population.

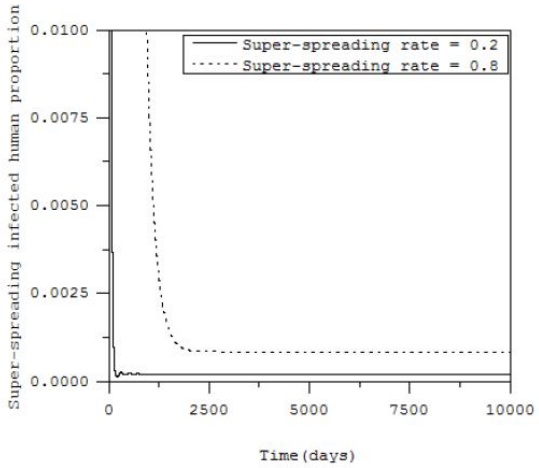


Figure6. Time series of regular infected and super-spreading infected human when there is the different super-spreading rate.

From figure 4, 5 and figure 6, we can conclude that when there is the less transmission rate of virus, the infected human will be less but the time of outburst is longer. When there is the total human population is higher, the time of outburst is longer. When there is

the higher super-spreading rate, the number of super-spreading infected human will be greater and the longer time of outburst transmission.

ACKNOWLEDGMENT

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