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Swine Flu Transmission Model in Risk and Non-Risk Human Population

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Abstract**—**The Swine flu outbreak in humans is due to a new strain of influenza A virus subtype H1N1 that derives in part from human influenza, avian influenza, and two separated strains of swine influenza. It can be transmitted from human to human. A mathematical model for the transmission of Swine flu is developed in which the human populations are divided into two classes, the risk and non-risk human classes. Each class is separated into susceptible, exposed, infectious, quarantine and recovered sub-classes. In this paper, we formulate the dynamical model of Swine flu transmission and the repetitive contacts between the people are also considered. We analyze the behavior for the transmission of this disease. The Threshold condition of this disease is found and numerical results are shown to confirm our theoretical predictions.

Keywords**—**Mathematical model, Steady state, Swine flu, threshold condition.

I. Introduction

SWINE Influenza Virus (SIV) is a viral infection in swine that is common throughout the world. It is a common cause of respiratory disease in pigs [1]. This virus is an orthomyxovirus. This type of virus is divided into three groups; type A, type B or type C. Only type A viruses infect pigs. Type A virus is further divided into subtypes based on their hemaglutinin (H) and neuraminidases (N). 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. Three main subtypes currently circulating in the pig population are classical swine influenza virus and reassortant viruses of H1N1, H3N2, and H1N2 [2]. The subtype identified H1N1 (2009) influenza virus (also referred to as "Pandemic Influenza", "Novel Influenza" or "Swine Flu") is a type of influenza virus that causes respiratory disease that can spread between people. Swine influenza 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. The first identification of an influenza virus as a cause of disease in pigs occurred about ten years later, in the United States in 1930 [3]. 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 following 60 years, swine influenza strains were almost exclusively H1N1. Then, between 1997 and 2002, new strains of three different subtypes and five different genotypes emerged 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-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 Thailand, H1N1 (2009) was the first subtype isolated from pigs with an influenza-like symptom in 1990 [4]. Subsequently, in 2005 a new subtype H1N1 was isolated from pigs in Saraburi province [5]. H1N1 (2009) is thought to spread from person to person through coughing or sneezing of people with the virus. Persons may also become infected by touching something, contaminated with flu viruses and then touching their eyes, nose or mouth. The most common clinical findings of the 2009 H1N1 influenza A pandemic have been fever, cough, sore throat, malaise, headache, vomiting and diarrhea have also been common, both of which are unusual features of seasonal influenza. People with H1N1 should be considered contagious as long as they have symptoms and up to 7 days following the onset of illness. Other frequent findings have included chills, myalgias and arthralgias [6-7]. The Centers for Disease Control and Prevention (CDC) recommends the antiviral drugs Tamiflu (oseltamivir) or Relenza (zanamivir) for treatment and prevention of infection with the swine flu virus. Antiviral drugs work best if started within 2 days of symptoms.

In this study, the mathematical model is used for describing the transmission of swine flu in human. We separate the human into the risk and non-risk human populations. A person vulnerable to the disease depends on the environment around him/her and the occupation.

II. Transmission Model

The human population is separated into risk and non-risk human populations;

$S_r(t)$ denotes the number of susceptible risk human at time t,

$E_r(t)$ denotes the number of exposed risk human at time t,

$I_r(t)$ denotes the number of infectious risk human at time t,

$Q_r(t)$ denotes the number of quarantine risk human at time t,

$R_r(t)$ denotes the number of recovered risk human at time t,

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$S_n(t)$ denotes the number of susceptible non-risk human at time t,
$E_n(t)$ denotes the number of exposed non-risk human at time t,
$I_n(t)$ denotes the number of infectious non-risk human at time t,
$Q_n(t)$ denotes the number of quarantine non-risk human at time t,
$R_n(t)$ denotes the number of recovered non-risk human at time t.
The transmission of Swine flu is described by the following equations:

$$S_r'(t) = P_r\lambda S_0 - (\gamma_h+\gamma_s+\gamma_p+\gamma_o)S_r(t)\frac{(E_r(t)+I_r(t))}{N}\beta_r - \mu S_r(t),$$

$$E_r'(t) = (\gamma_h+\gamma_s+\gamma_p+\gamma_o)\frac{S_r(t)(E_r(t)+I_r(t))}{N}\beta_r - (\theta+a+\mu)E_r(t),$$

$$I_r'(t) = aE_r(t) - (\alpha+b+\mu)I_r(t),$$

$$Q_r'(t) = bI_r(t) - (c+\mu)Q_r(t),$$

$$R_r'(t) = cQ_r(t) + \alpha I_r(t) + \theta E_r(t) - \mu R_r(t), \quad (1)$$

$$S_n'(t) = (1-P_r)\lambda S_0 - (\gamma_h+\gamma_s+\gamma_p+\gamma_o)\frac{S_n(E_n(t)+I_n(t))}{N}\beta_n - \mu S_n(t)$$

$$E_n'(t) = (\gamma_h+\gamma_s+\gamma_p+\gamma_o)\frac{S_n(t)(E_n(t)+I_n(t))}{N}\beta_n - (\theta+a+\mu)E_n(t),$$

$$I_n'(t) = aE_n(t) - (\alpha+b+\mu)I_n(t),$$

$$Q_n'(t) = bI_n(t) - (c+\mu)Q_n(t),$$

$$R_n'(t) = cQ_n(t) + \alpha I_n(t) + \theta E_n(t) - \mu R_n(t),$$

where

$$N = S_r + E_r + I_r + Q_r + R_r + S_n + E_n + I_n + Q_n + R_n,$$

$$N_r = P_r N = S_r + E_r + I_r + Q_r + R_r, \quad (2)$$

and $N_n = (1-P_r)N = S_n + E_n + I_n + Q_n + R_n$.

The parameters are defined as follows:
N is the total human population,
N_r is the total risk human population,
N_n is the total non-risk human population,
P_r is the fraction of the people who are risk of infection,
γ_h is the probability of the repetitive contact between people in the house,
γ_s is the probability of the repetitive contact between people in the school,
γ_p is the probability of the repetitive contact between people in the airplane,
γ_o is the probability of the repetitive contact between people in the other places,
λ is the birth rate of the human,
μ is the death rate of the human,
S_0 is the initial value of the susceptible human population,
β_r is the contact rate between the risk people,
β_n is the contact rate between the non-risk people,
$a = \frac{1}{IIP}$ where IIP is the intrinsic incubation period of the swine flu in human,
α is the recovery rate,
θ is the rate at which the exposed human change to be the recovered human,
b is the rate at which the infectious human change to be the quarantine,
c is the rate at which the quarantine change to be the recovered human.

The total human, the numbers of risk and non-risk humans are assumed to be constant, then the rates of change for all human population equal to zero. From setting the rate of change for each human population equals to zero, we obtained

$N_r = \frac{P_r\lambda S_0}{\mu}$, $N_n = \frac{(1-P_r)\lambda S_0}{\mu}$ and

$N = \frac{P_r\lambda S_0}{\mu} + \frac{(1-P_r)\lambda S_0}{\mu} = \frac{\lambda S_0}{\mu}$.

After we normalized (1), the reduced equations are

$$s_r'(t) = \lambda P_r - (\gamma_h+\gamma_s+\gamma_p+\gamma_o)s_r(t)(e_r(t)+i_r(t))\beta_r - \lambda s_r(t),$$

$$e_r'(t) = (\gamma_h+\gamma_s+\gamma_p+\gamma_o)s_r(t)(e_r+i_r)\beta_r - (\theta+a+\lambda)e_r(t),$$

$$i_r'(t) = ae_r(t) - (\alpha+b+\lambda)i_r(t),$$

$$q_r'(t) = bi_r(t) - (c+\lambda)q_r(t),$$

$$s_n'(t) = \lambda(1-P_r) - (\gamma_h+\gamma_s+\gamma_p+\gamma_o)s_n(t)(e_n+i_n)\beta_n - \lambda s_n(t), \quad (3)$$

$$e_n'(t) = (\gamma_h+\gamma_s+\gamma_p+\gamma_o)s_n(t)(e_n+i_n)\beta_n - (\theta+a+\lambda)e_n(t),$$

$$i_n'(t) = ae_n(t) - (\alpha+b+\lambda)i_n(t),$$

$$q_n'(t) = bi_n(t) - (c+\lambda)q_n(t),$$

where $s_r(t)+e_r(t)+i_r(t)+q_r(t)+r_r(t) = 1$

and $s_n(t)+e_n(t)+i_n(t)+q_n(t)+r_n(t) = 1$. (4)

III. Analysis of the Model

A. Analytical Solutions

The two steady states are obtained when we set the right hand side of (3) equal to zero. The disease free steady state: $E_0 = (P_r,0,0,0,(1-P_r),0,0,0)$ and the endemic steady state: $E_1 = (s_r^*, e_r^*, i_r^*, q_r^*, s_n^*, e_n^*, i_n^*, q_n^*)$.

where

$$s_r^* = \frac{P_r}{D_{0_r}},$$

$$e_r^* = P_r\frac{\mu}{L_1 D_{0_r}}[D_{0_r}-1],$$

$$i_r^* = aP_r\frac{\mu}{L_1 L_2 D_{0_r}}[D_{0_r}-1],$$

$$q_r^* = abP_r\frac{\mu}{L_1 L_2 L_3 D_{0_r}}[D_{0_r}-1],$$

$$s_n^* = \frac{(1-P_r)}{D_{0_n}} \quad ,$$

$$e_n^* = (1-P_r)\frac{\mu}{L_1 D_{0_n}}[D_{0_n}-1],$$

$$i_n^* = a(1-P_r)\frac{\mu}{L_1 L_2 D_{0_n}}[D_{0_n}-1],$$

$$q_n^* = ab(1-P_r)\frac{\mu}{L_1 L_2 L_3 D_{0_n}}[D_{0_n}-1],$$

where

$L_1 = (\theta + a + \mu)$, $L_2 = (\alpha + b + \mu)$, $L_3 = (c + \mu)$,

$D_{0_r} = \frac{P_r \beta_r (\gamma_h + \gamma_s + \gamma_p + \gamma_o)(a + L_2)}{L_1 L_2}$ and

$D_{0_n} = \frac{(1 - P_r)\beta_n (\gamma_h + \gamma_s + \gamma_p + \gamma_o)(a + L_2)}{L_1 L_2}$.

The characteristic equation of (3) for the disease free steady state is

$$(\eta + L_3)^2 (\eta + \mu)^4 (\eta^2 + A_1 \lambda + A_0)(\eta^2 + B_1 \eta + B_0) = 0$$

where

$A_1 = L_1 + L_2 - (\gamma_h + \gamma_s + \gamma_p + \gamma_o)(1 - p_r)\beta_n$,

$A_0 = L_1 L_2 - (\gamma_h + \gamma_s + \gamma_p + \gamma_o)(a + L_2)(1 - p_r)\beta_n$,

$B_1 = L_1 + L_2 - (\gamma_h + \gamma_s + \gamma_p + \gamma_o)p_r \beta_r$ and

$B_0 = L_1 L_2 - (\gamma_h + \gamma_s + \gamma_p + \gamma_o)(a + L_2)p_r \beta_r$.

The eigenvalues are

$\eta_1 = \eta_2 = -L_3$,

$\eta_3 = \eta_4 = \eta_5 = \eta_6 = -\mu$,

η_7, η_8, η_9 and λ_{10} are the solutions of

$(\eta^2 + A_1 \eta + A_0)(\eta^2 + B_1 \eta + B_0) = 0$ or

$\eta_7 = \frac{-A_1 + \sqrt{A_1^2 - 4A_0}}{2}$, $\eta_8 = \frac{-A_1 - \sqrt{A_1^2 - 4A_0}}{2}$,

$\eta_9 = \frac{-B_1 + \sqrt{B_1^2 - 4B_0}}{2}$ and $\eta_{10} = \frac{-B_1 - \sqrt{B_1^2 - 4B_0}}{2}$.

The local stability of a steady state is determined from the Jacobian matrix of the right hand side of (3) evaluated at the steady state. If all the eigenvalues have negative real parts then that steady state is local asymptotically stable. From our calculations, we found that the disease free state is local asymptotically stability for $D_{0_r} < 1$ and $D_{0_n} < 1$.

The characteristic equation of (3) for the endemic steady state is

$$(\eta + L_3)^2 (\eta + \mu)^2 (\eta^3 + C_2 \eta^2 + C_1 \eta + C_0)$$

$$(\eta^3 + D_2 \eta^2 + D_1 \eta + D_0) = 0$$

where

$$C_2 = L_1 + L_2 + \mu + \frac{(\gamma_h + \gamma_s + \gamma_p + \gamma_o)(1 - P_r)\beta_n (L_1 L_2 + (D_{0_n} - 1)(a + L_2)\mu}{D_{0_n} L_1 L_2}$$

$$C_1 = \mu(L_1 + L_2) + L_1 L_2 + \frac{\mu(\gamma_h + \gamma_s + \gamma_p + \gamma_o)(1 - P_r)\beta_n}{D_{0_n}} \left(\frac{(D_{0_n} - 1)(a + L_2)(L_1 + L_2)}{L_1 L_2} + \frac{a + L_2 + \mu}{\mu} \right)$$

$$C_0 = \mu \left(L_1 L_2 + \frac{(\gamma_h + \gamma_s + \gamma_p + \gamma_o)(1 - P_r)\beta_n (D_{0_n} - 1)(a + L_2)}{D_{0_n}} \right)$$

$$D_2 = L_1 + L_2 + \mu + \frac{(\gamma_h + \gamma_s + \gamma_p + \gamma_o)P_r \beta_r (L_1 L_2 + (D_{0_r} - 1)(a + L_2)\mu)}{D_{0_r} L_1 L_2}$$

$$D_1 = \mu(L_1 + L_2) + L_1 L_2 + \frac{\mu(\gamma_h + \gamma_s + \gamma_p + \gamma_o)P_r \beta_r}{D_{0_r}} \left(\frac{(D_{0_r} - 1)(a + L_2)(L_1 + L_2)}{L_1 L_2} + \frac{a + L_2 + \mu}{\mu} \right)$$

$$D_0 = \mu \left(L_1 L_2 + \frac{(\gamma_h + \gamma_s + \gamma_p + \gamma_o)P_r \beta_r (D_{0_r} - 1)(a + L_2)}{D_{0_r}} \right).$$

Thus, the eigenvalues are given by

$\eta_1 = \eta_2 = -L_3, \eta_3 = \eta_4 = -\mu$, η_5, η_6 and η_7 are the solutions of $(\eta^3 + C_2 \eta^2 + C_1 \eta + C_0) = 0$,

η_8, η_9 and η_{10} are the solutions of

$(\eta^3 + D_2 \eta^2 + D_1 \eta + D_0) = 0$.

From the Routh-Hurwitz condition, the eigenvalues $\eta_5, \eta_6, \eta_7, \eta_8, \eta_9$ and η_{10} are negative when

1) $C_2 C_1 - C_0 > 0$, $C_2 > 0$ and $C_0 > 0$,

2) $D_2 D_1 - D_0 > 0$, $D_2 > 0$ and $D_0 > 0$.

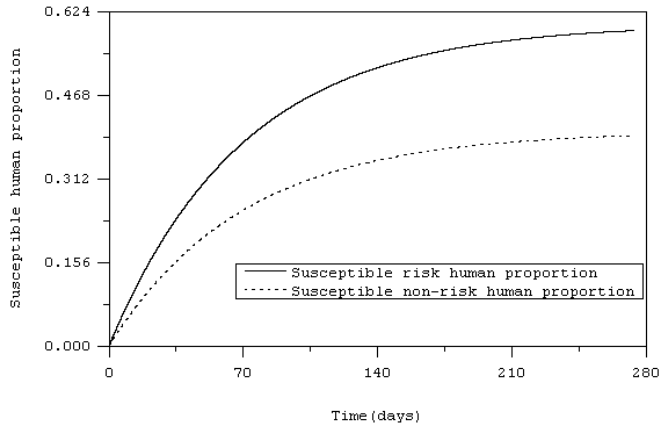
From the calculation, we found that 1) and 2) are satisfied when

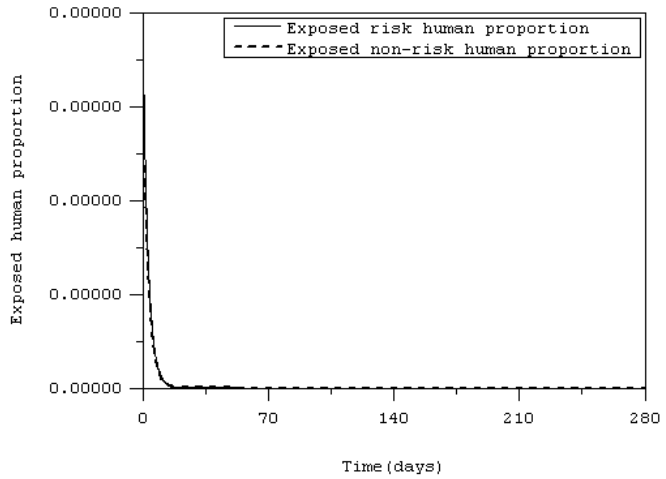
$$D_{0_r} > 1 \text{ and } D_{0_n} > 1.$$

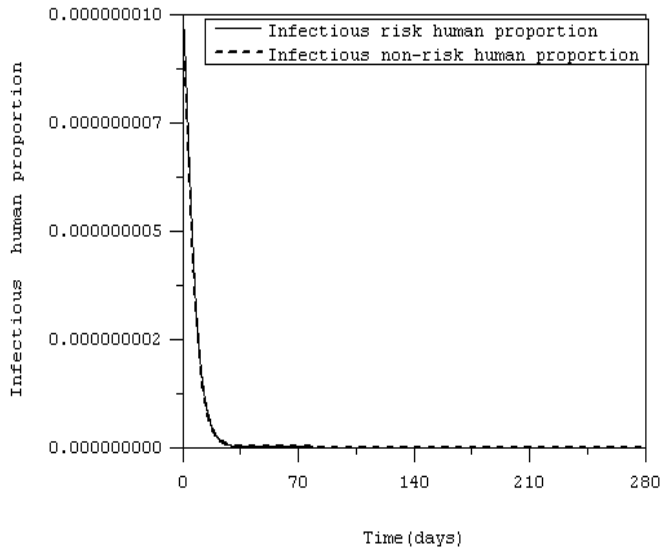
So the endemic disease state is local asymptotically stability for $D_{0_r} > 1$ and $D_{0_n} > 1$.

B. Numerical Simulation

We consider the transmission of Swine flu by separating the human into risk and non-risk human populations. The parameters in the simulation are: $\lambda = 1/(365 * 70)$ corresponds to the life expectancy of 70 years for human. $P_r, \gamma_h, \gamma_s, \gamma_p, \gamma_o, \beta_r, \beta_n$ are arbitrary chosen. $a = 1/5$ satisfies to the intrinsic incubation period of 5 days, $\theta = 1/7$ correspond to the 7 days which the exposed human change to be the recovered human, $\alpha = 1/10$ correspond to the recovery of 10 days, $b = 1/8$ correspond to the 8 days which the infectious human change to be the quarantine, $c = 1/6$ corresponds to the 6 days at which the quarantine change to be the recovered human.







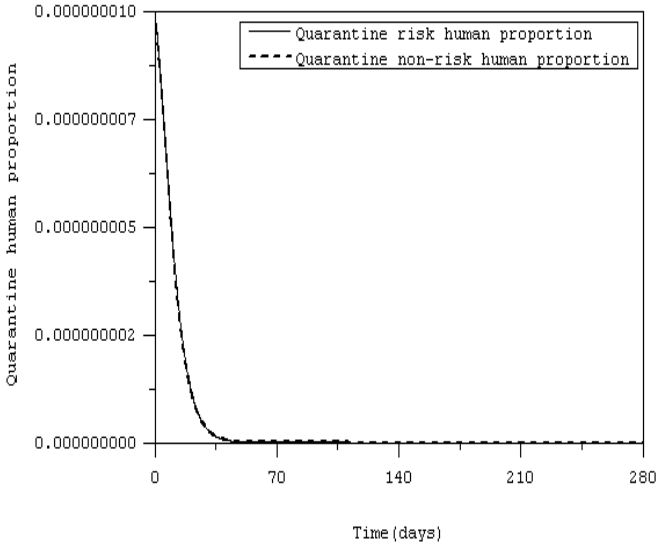
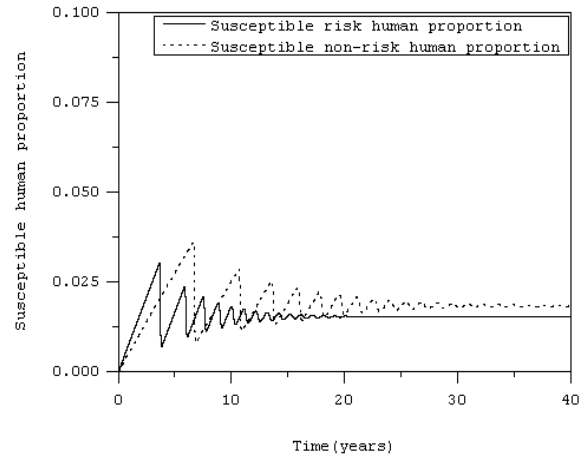
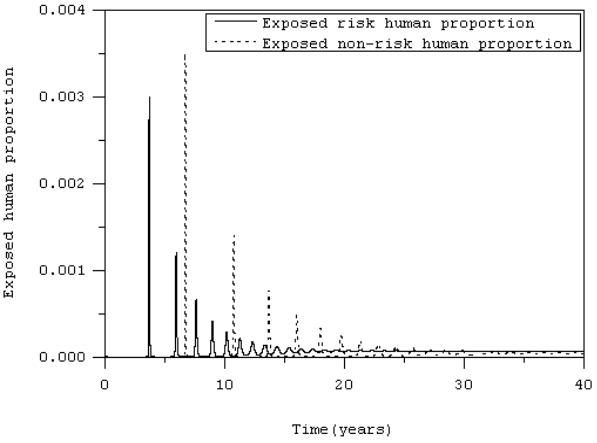


Fig. 1 Numerical solutions of (3) , demonstrate the times series of each human population, for $D_{0_r} < 1$ and $D_{0_n} < 1$, $P_r = 0.6, \gamma_h = 0.2, \gamma_s = 0.25, \gamma_p = 0.3, \gamma_o = 0.25,$ $\beta_r = 0.28, \beta_n = 0.2, a = 1/5, \theta = 1/7, \alpha = 1/10, b = 1/8, c = 1/6,$ $D_{0_n} = 0.44, D_{0_r} = 0.93$ .The fractions of populations converge to the disease free state $E_0 = (0.6,0,0,0,0.4,0,0,0,)$.





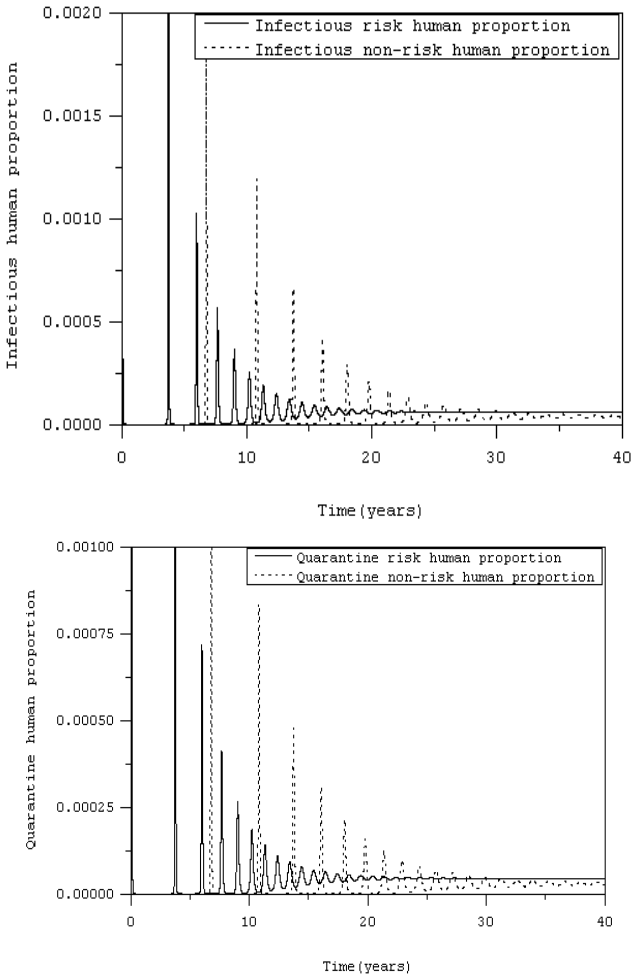


Fig. 2 Numerical solutions of (3) , demonstrate the times series of each human population, for $D_{0_r} > 1$ and $D_{0_n} > 1$, $P_r = 0.6, \gamma_h = 0.2, \gamma_s = 0.25, \gamma_p = 0.3, \gamma_o = 0.25, \beta_r = 12, \beta_n = 10, a = 1/5, \theta = 1/7, \alpha = 1/10, b = 1/8, c = 1/6, D_{0_n} = 22, D_{0_r} = 39$.

The fractions of populations oscillate to the endemic state E_1= (0.015129,0.000067,0.00006,0.000044, 0.0181548,0.000043, 0.000038, 0.000029).

IV. Discussion and Conclusion

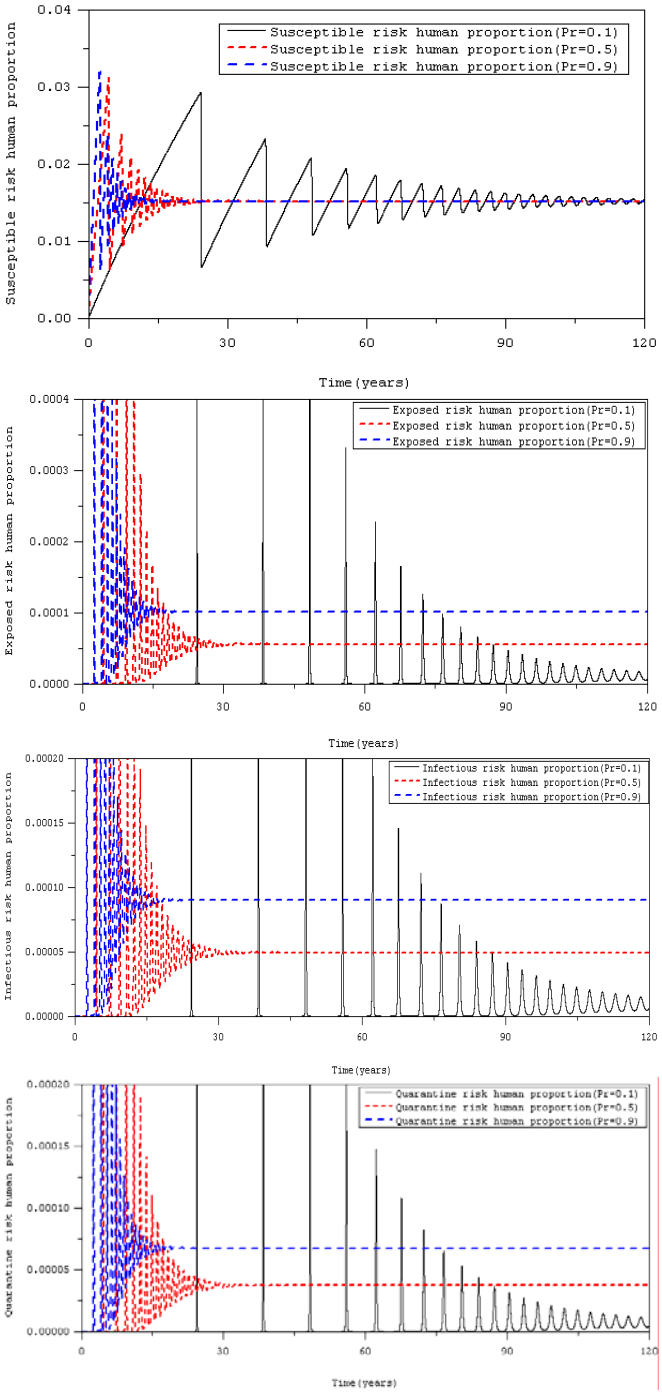
From the analysis of the model, the threshold number is defined as

$$D_{0_r} = \frac{P_r \beta_r (\gamma_h + \gamma_s + \gamma_p + \gamma_o)(a + L_2)}{L_1 L_2} \quad (5)$$

and

$$D_{0_n} = \frac{(1 - P_r)\beta_n (\gamma_h + \gamma_s + \gamma_p + \gamma_o)(a + L_2)}{L_1 L_2}, \quad (6)$$

where D_{0_r} is the basic reproductive number of the risk human and D_{0_n} is the basic reproductive number of the non-risk human population. From Fig. 1 and Fig. 2, we will see that the proportions of populations converge to the disease free steady state for D_{0_r} and D_{0_n} less than 1. For D_{0_r} and D_{0_n} greater than 1, the proportions of populations oscillate to the endemic disease equilibrium point. Moreover, we compare the behavior of the solutions when there is the different fraction of the risk people.



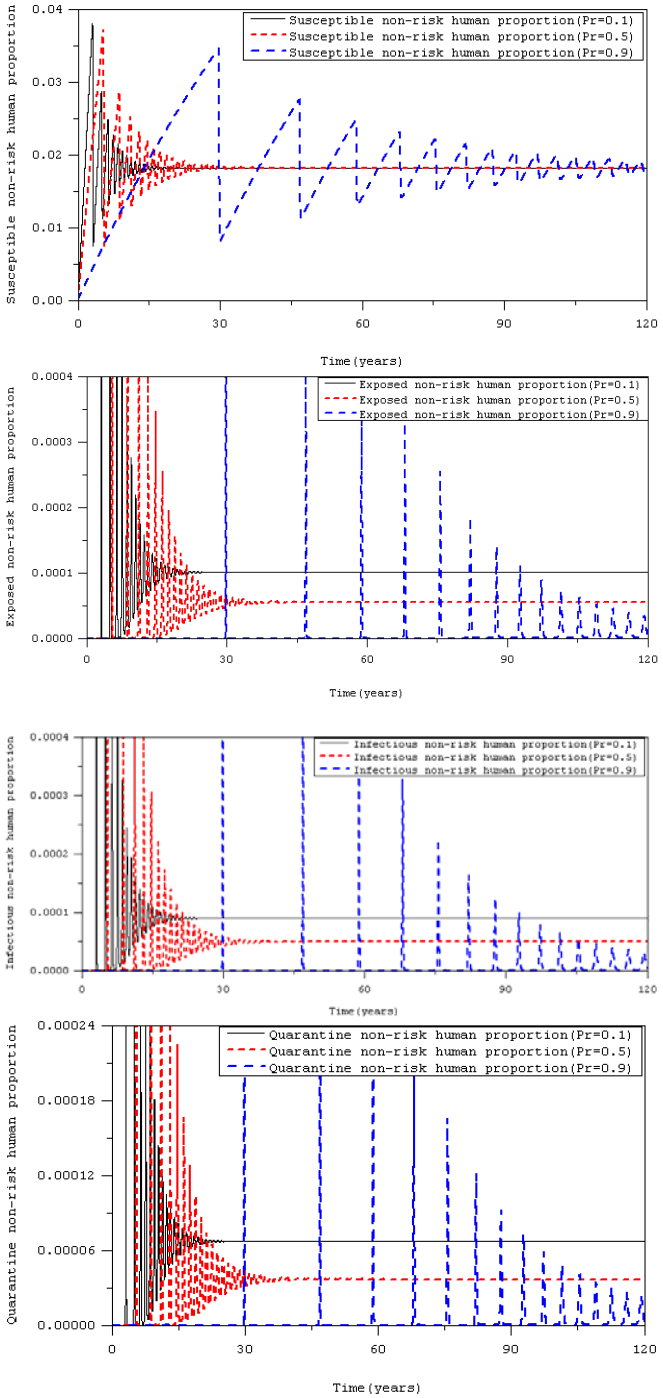


Fig. 3 The time series of the proportion of each population for P_r = 0.1, P_r = 0.5 and P_r = 0.9. The other parameters are same as in figure 2.

We can see that when the fraction of the people who are risk of infection is higher then the steady state proportions of the risk human are higher but the steady state proportions of the non-risk human are smaller. The threshold numbers are used for reducing the outbreak of the several diseases [8-11]. The results of this study should point the way for reducing the outbreak of this disease.

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