British Journal of Mathematics & Computer Science 14(5): 1-8, 2016, Article no.BJMCS.23142

ISSN: 2231-0851

SCIENCEDOMAIN *international* www.sciencedomain.org

Mathematical Analysis of a Swine Flu Model with Mixed Transmission

Nidhi Nirwani1* and V. H. Badshah¹

1 School of Studies in Mathematics, Vikram University, Ujjain (M.P.), India.

Authors' contributions

This work was carried out in supervision of author VHB. Both the authors designed the study and wrote the first draft of the manuscript. They also performed the mathematical analysis of the study and the simulations, managed literature searches. Both authors read and approved the final manuscript.

Article Information

DOI: 10.9734/BJMCS/2016/23142 *Editor(s):* (1) Dijana Mosic, Department of Mathematics, University of Nis, Serbia. *Reviewers:* (1) J. Prakash, University of Botswana, Botswana. (2) Rachana Pathak, University of Lucknow, Uttar Pradesh, India. (3) Ette Harrison Etuk, Rivers State University of Science and Technology, Nigeria. (4) Anonymous, Karnatak University, Dharwad, India. (5) Gbolahan Bolarin, Federal University of Technology, Minna, Nigeria. Complete Peer review History: http://sciencedomain.org/review-history/13267

Original Research Article

Received: 16th November 2015 Accepted: 19th January 2016 Published: 11th February 2016

Abstract

In this paper, we proposed and analyzed an SEIR compartment model of Swine flu with mixing transmission. The stability of the disease-free equilibrium and the endemic equilibrium is obtained by Routh-Hurwitz criteria. The Basic Reproduction number R_0 has also been discussed, when $R_0 < 1$, the disease free equilibrium point is stable. In case $R_0 > 1$, there exists endemic equilibrium. Numerical simulations are carried out for different values of contact rate to understand the transmission behavior of the disease.

Keywords: Epidemic model; swine flu; compartment model; stability.

**Corresponding author: E-mail: nd.mathematics2009@gmail.com;*

1 Introduction

Swine flu is a respiratory virus of pigs which was first identified in 1918 and although historic diffusion to human beings has been sporadic, the infection rate in humans is intensifying at present. Chills, dyspnea, headache, vomiting, diarrhea, myalgia, and fatigue are most common symptoms of swine flu. The virus has not previously circulated in human the virus is entirely new [1].

Many mathematical models have been analyzed to understand the spread of swine flu within human and also in pig populations like in [2,3,4]. Kermack and McKendrick [5] were the first person that's describe an influenza epidemic early in the $20th$ century. Their model is known as the SIR which has been used as a basis for all subsequent influenza models. By modifying the basic SIR model in a variety of ways by including seasonality influenza epidemics can be shown to have sustained cycles [6,7]. The SIR model has also been extended so that it can be used to represent and/or predict the spatial dynamics of an influenza epidemic.

Most recently several investigation have concern themselves with modeling of dynamics of influenza virus [8, 9,2,10,11].

In this paper we have modified the model of Das et al. [12] with recovery class. In the first section we present the model in which c is the contact rate at which the susceptible population is converted into the exposed population. $S(t)$, $E(t)$, $I(t)$ and $R(t)$ represents the number of susceptible, exposed, infectious, and recovered Population at the time t respectively, A is the requirement rate of the population, μ is the natural death rate of the population, γ is the natural recovery rate of the infective individuals. In the next section we obtained the disease free and the endemic equilibrium and analyzed the stability conditions for both. In the last section numerical results are also provided.

2 The Mathematical Model

$$
\frac{dS}{dt} = A - \frac{cSI}{S+I} + rI - \mu S
$$
\n
$$
\frac{dE}{dt} = \frac{cSI}{S+I} - (\lambda + \mu)E
$$
\n
$$
\frac{dI}{dt} = \lambda E - (r + \gamma + \mu)I
$$
\n
$$
\frac{dR}{dt} = \gamma I - \mu R
$$
\n(2.1)

Where S, E, I and R stands for susceptible, exposed infective and recovered individuals, respectively. The parameters in the model are

- A= The recruitment rate of the population
- μ = The natural death rate of the population
- $c =$ The contact rate at which the susceptible population is converted into exposed population.

 $r =$ Recovery rate

- λ = The effective transmission coefficient
- ν = The natural recovery rate of the infective individuals

The transfer diagram is depicted in the following Fig. 1:

Fig. 1. A compartment model of swine flu

3 Stability Analysis

For the equilibrium points the above differential equation should be equated to zero.

i.e.
$$
\frac{dS}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0
$$

We have two equilibrium points are given by $P_0 = (A/\mu, 0.0, 0)$ is the disease free equilibrium points of the system (2.1) and the unique endemic equilibrium point $P^* = (S^*, E^*, I^*, R^*)$, where

$$
S^* = \frac{p}{c - p} I^*,
$$

\n
$$
E^* = \frac{(r + \gamma + \mu)}{\lambda} I^*,
$$

\n
$$
I^* = \frac{A}{\left[\frac{\mu p}{c - p} + p - r\right]}
$$

\n
$$
R^* = \frac{\gamma}{\mu} I^*
$$

\nwhere $p = \frac{(\lambda + \mu)(r + \gamma + \mu)}{\lambda}$

The basic reproduction number defined as

$$
R_0 = \frac{c\lambda}{(\lambda + \mu)(r + \gamma + \mu)}
$$

3.1 Theorem. The disease free equilibrium of the system is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof: We consider equations

$$
F_1 = A - \frac{cSI}{S+I} + rI - \mu S
$$

\n
$$
F_2 = \frac{cSI}{S+I} - (\lambda + \mu)E
$$

\n
$$
F_3 = \lambda E - (r + \gamma + \mu)I
$$

\n
$$
F_4 = \gamma I - \mu R
$$

The Jacobian matrix

$$
J_0 = \begin{bmatrix} \frac{-cI^2}{(S+I)^2} - \mu & 0 & \frac{-cS^2}{(S+I)^2} + r & 0\\ \frac{cI^2}{(S+I)} & -(\lambda + \mu) & \frac{cS^2}{(S+I)^2} & 0\\ 0 & \lambda & -(r + \gamma + \mu) & 0\\ 0 & 0 & \gamma & -\mu \end{bmatrix}
$$

At equilibrium point $P_0 = (A/\mu, 0, 0, 0)$ the Jacobian matrix becomes

$$
J_0 = \begin{bmatrix} -\mu & 0 & -c+r & 0 \\ 0 & -(\lambda + \mu) & c & 0 \\ 0 & \lambda & -(r+\gamma + \mu) & 0 \\ 0 & 0 & \gamma & -\mu \end{bmatrix}
$$

The characteristics equation $|J_0 - \varphi I| = 0$ is given as

$$
\begin{vmatrix} -(\mu + \varphi) & 0 & -c + r & 0 \\ 0 & -(\lambda + \mu + \varphi) & c & 0 \\ 0 & \lambda & -(r + \gamma + \mu + \varphi) & 0 \\ 0 & 0 & \gamma & -(\mu + \varphi) \end{vmatrix} = 0
$$

$$
\Rightarrow (\mu + \varphi)^2 [(\lambda + \mu + \varphi)(r + \gamma + \mu + \varphi) - c\lambda] = 0
$$

Clearly two Eigen values $\varphi = -\mu$, $-\mu$ are negative, other Eigen values are given by the quadratic equation.

$$
\varphi^2 + a_1 \varphi + a_2 = 0,
$$

Where

$$
a_1 = \lambda + r + \gamma + 2\mu
$$

$$
a_2 = (\lambda + \mu)(r + \gamma + \mu) - c\lambda
$$

Therefore, by Routh-Hurwitz criteria the disease-free equilibrium stable if $a_1 > 0$ and $a_2 > 0$, which is possible if $(\lambda + \mu)(r + \gamma + \mu) > c\lambda$, i.e $R_0 < 1$.

3.2 Theorem. If $R_0 > 1$ the endemic equilibrium P^* is locally asymptotically stable.

Proof: The variation matrix at the endemic point $P^*(S^*, E^*, I^*, R^*)$

$$
J_1 = \begin{bmatrix} \frac{-cI^{*2}}{(S^* + I^*)^2} - \mu & 0 & \frac{-cS^{*2}}{(S^* + I^*)^2} + r & 0\\ \frac{cI^{*2}}{(S^* + I^*)} & -(\lambda + \mu) & \frac{cS^{*2}}{(S^* + I^*)^2} & 0\\ 0 & \lambda & -(r + \gamma + \mu) & 0\\ 0 & 0 & \gamma & -\mu \end{bmatrix}
$$

Consider that

$$
w_1 = \frac{cI^{*2}}{(S^* + I^*)^2} \text{ and } w_2 = \frac{cS^{*2}}{(S^* + I^*)^2}
$$

Then J_1 becomes

$$
J_1 = \begin{bmatrix} -w_1 - \mu & 0 & -w_2 + r & 0\\ w_1 & -(\lambda + \mu) & w_2 & 0\\ 0 & \lambda & -(r + \gamma + \mu) & 0\\ 0 & 0 & \gamma & -\mu \end{bmatrix}
$$

The characteristics equation $|J_1 - \varphi I| = 0$ is given as

$$
\begin{vmatrix}-(w_1 + \mu + \varphi) & 0 & -w_2 + r & 0 \\ w_1 & -(\lambda + \mu + \varphi) & w_2 & 0 \\ 0 & \lambda & -(r + \gamma + \mu + \varphi) & 0 \\ 0 & 0 & \gamma & -(\mu + \varphi)\end{vmatrix} = 0
$$

$$
\Rightarrow (\mu + \varphi)[(w_1 + \mu + \varphi)(\lambda + \mu + \varphi)(r + \gamma + \mu + \varphi) - (w_1 + \mu + \varphi)w_2\lambda + (w_2 - r)w_1\lambda] = 0
$$

Clearly one eigen value is negative $\varphi = -\mu$ and other eigen values are given by the cubic equation.

$$
\varphi^3 + a_1 \varphi^2 + a_2 \varphi + a_3 = 0
$$

Where

$$
a_1 = 3\mu + \lambda + w_1 + r + \gamma
$$

\n
$$
a_2 = [(\lambda + \mu)(w_1 + \mu) + (w_1 + 2\mu + \lambda)(r + \gamma + \mu) - w_2\lambda]
$$

\n
$$
a_3 = (\lambda + \mu)(w_1 + \mu)(r + \gamma + \mu) - (\mu w_2 + r w_1)\lambda
$$

By Routh-Hurwitz criteria, the system (2.1) is locally asymptotically stable if $a_1 > 0$, $a_3 > 0$ and $a_1 a_2 >$ a_3 . Thus, P^* is locally asymptotically stable.

4 Discussion and Numerical Simulation

From practical point of view, numerical solutions are very important beside analytical study. In our study, we propose and analyze a swine flu model. We also performed the numerical solutions by using hypothetical set of parameter values with Excel. Since A is the recruitment rate of the population, we choose a suitable value as unit. From the study we observe that the disease free equilibrium is locally stable for basic reproduction number R_0 <1 and is unstable otherwise, which is directly proportional to contact rate 'c'. For existence of endemic equilibrium point we have increased value the contact rate. Further local stability of the endemic equilibrium of the system is also contact rate dependent. We performed numerical simulation for different values of c. Numerical solutions are presented graphically by taking the population in hundreds and time is in per day.

4.1 Numerical simulation for disease free equilibrium

From the numerical values of the parameters as $A = 1$, $c = 0.003$, $r = 0.1$, $\mu = 0.02$, $\lambda = 0.1$ and $\gamma = 0.01$ Then the calculated disease free equilibrium point and basic reproductive number are: $P_0(S, 0, 0, 0) =$ $(50,0,0,0)$ and $R_0 = 0.192307 < 1$. Fig. 2 shows that $S(t)$ goes to its steady state, while $E(t)$, $I(t)$ and $R(t)$ goes to zero with respect to time. Hence the disease dies out.

Fig. 2. The figure shows that the disease free equilibrium is locally stable for the choice of parameter values

4.2 Numerical simulation for endemic equilibrium

We change the value of $c = 0.3$ and all other parameters are as above. Then, we obtain $P^*(S^*, E^*, I^*, R^*) =$ $(13.9496, 16.7395, 12.8766, 6.4383)$ and $R_0 = 1.92307 > 1$. Therefore, the endemic equilibrium P^* is locally asymptotically stable. Fig. 3 shows that S, E, I and R goes to their steady state values. Hence the disease becomes endemic.

Fig. 3. The figure shows that the endemic equilibrium is locally asymptotically stable for the choice of parameter values

Now if we change c value as $c = 0.162$ then the endemic equilibrium point changes as $P^*(S^*, E^*, I^*, R^*) =$ 45.1388,2.2569,1.73611,0.86805 .

Fig. 4. The figure shows that the exposed, infective and recovered classes are decreasing in number for the choice of parameter values

Keeping other parameters fixed, if we change the values of $c = 0.1560312$ we get endemic equilibrium point becomes $P^*(S^*, E^*, I^*, R^*) = (49.978, 0.01299428, 0.0099956, 0.0049978)$, which shows the exposed, infective and recovery classes are going extinct for choice of parameter value.

Fig. 5. Exposed, infective and recovery classes are going extinct for choice of parameter value

5 Conclusion

In this paper, we analyzed an SEIR compartment model of Swine flu, the results are helpful to predict the developing tendency of disease and recovery. We analyzed the Steady state and stability of the equilibrium points. The model equations were solved analytically. We can conclude that the basic reproduction number R_0 < 1 then the disease free equilibrium P_0 is locally asymptotically stable and if $R_0 > 1$ the endemic equilibrium P^* is locally asymptotically stable.

Numerical simulations were presented graphically. We have also observed that contact rate c plays an important role in stability; the basic reproduction number R_0 will be decrease if the contact rate c decreases when disease is endemic.

Competing Interests

Authors have declared that no competing interests exist.

References

- [1] WHO, World now at the started of 2009 influenza pandemic. Available: http://www.who.int/mediacentre/news/tatements/2009/h1n1_pandemic_phase6_20090611
- [2] Changpuek T, Pongsumpun P. The age structural transmission model for swine flu. Proceeding of the 3rd Biomedical Engineering International conference (BMEiCON 2010), August 27-28, Kyoto, Japan. 2010;1-6.
- [3] Pongsumpun P. Swine flu transmission in risk and non-risk human population. World Academy of Science, Enggineering and Technology. 2010;68:166-1171.
- [4] Pongsumpun P, Tang IM. Mathematical model of the symptomatic and asymptomatic infections of swine flu. International Journal of Mathematical Models and Methods in Applied Sciences. 2011;5(2):257-254.
- [5] Kermack WO, McKendrick AG. A contribution to the mathematical theory of epidemics. Proc Roy Soc Lond. 1927;115:700-721.
- [6] Dushoff J, Plotkin JB, Levin SA, Earn DJD. Dynamical resonance can account for seasonal influenza epidemics**.** Proc Natl Acad Sci USA. 2004;101:16915-16916.
- [7] Stone L, Olinky R, Huppert A. Seasonal dynamics of recurrent epidemics**.** Nature. 2007;446**:**533-536.
- [8] Casagrandi R, Bolzoni L, Levin SA, Andreasen V. The SIRC model and influenza A. Math Biosci. 2006;200:152–169.
- [9] Coburn BJ, Wagner BG, Blower S. Modeling influenza epidemics and pandemics: Insights into the future of swine flu (H1N1). BMC Med. 2009;7:30-7015-7-30.
- [10] Karim SAA, Razali R. A proposed mathematical model of influenza A, H1N1 for Malaysia. J Applied Sci. 2011;1-3. DOI: 10.3923/jas2011
- [11] Reynolds JJH, Torremorell M, Craft ME. Mathematical modeling of influenza A virus dynamics within swine farms and the effects of vaccination. PLoS ONE. 2014;9(8):e106177. DOI: 10.1371/journal.pone.0106177
- [12] Das P, Gazi NH, Das K, Mukherjee D. Stability analysis of swine flu transmission. A Mathematical Approach Computational and Mathematical Biology. 2014;3(1):1-5. ___

© 2016 Nirwani and Badshah; This is an Open Access article distributed under the terms of the Creative Commons Attribution License (http://creativecommons.org/licenses/by/4.0), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.