International Journal of Pure and Applied Mathematics

Volume 52 No. 2 2009, 247-265

A *SEIQR* MODEL FOR PANDEMIC INFLUENZA AND ITS PARAMETER IDENTIFICATION

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Abstract: In this paper, we first propose a pandemic influenza susceptib-le-exposed-infected-quarantined-recovered ($SEIQR$) model and analyze the model properties. We then introduce a differential evolution (DE) algorithm for determining the numerical values of the parameters in the model. For a given set of measured data, e.g. from the first outbreak, all the values of the model parameters can be determined by the algorithm. We have also shown from numerical simulations that the DE algorithm yields the same parameter values for different sets of initial guesses. With the values of the parameters determined, the model can then be used to capture the behavior of the next outbreaks of the disease. The work provides an effective tool for predicting the spread of the disease.

AMS Subject Classification: 03C98
Key Words: $SEIQR$ model, influenza pandemic, stability, differential evolution algorithm

Received: March 10, 2009

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1. Introduction

Historically, flu pandemics have occurred around the world several times. The most notable of which was the 1918 Spanish Flu. The spread of this flu resulted in the death of over 50 million humans. Recently, the possibility of a new flu pandemic has risen. This new pandemic will be due to the highly pathogenic avian influenza A (HPAI) subtype H5N1 virus in Southeast Asia, the Middle East of Africa and other countries around the world [3]. It has been reported that clusters of H5N1 infections have appeared in families, mostly in Southeast Asia [17]. Today it is still in question whether there is human-to-human transmission of H5N1. If it really occurs and there is no preparedness against the pandemic, H5N1 would spread worldwide through the global transportation network. Due to the fear that a mutant avian influenza may occur, interest in the study of pandemic H5N1 flu has increased significantly. Thus in many countries, preparedness against the pandemic H5N1 flu has become a high priority public health issue. To have the pandemic preparedness plans that maximize realism, generality and precision, the design of such plans and the effects of different intervention strategies, such as quarantine and vaccination, need to be investigated. For the first outbreak of H5N1, a simple intervention procedure to control the spread of H5N1 is to isolate humans infected with mutant avian influenza, in order to reduce transmission to susceptible group. Once the disease has occurred, vaccine can be developed. To control the spread of H5N1 successfully, understanding the behavior of the spread of the disease is important. Recently, epidemic model plays an essential role for the pandemic preparedness plans because it allows one to predict and compare the effects of different intervention strategies. However, it has been recognized that the uncertainty in predictions of the epidemic model cause the uncertainty in prediction of disease spread and public health responses.

Over the last two decades, a number of epidemic models for predicting the spread of influenza through human population have been proposed based on either the classical susceptible-infected-removed (SIR) model [1, 8, 26] developed by Kermack and McKendrick [12] or the classical susceptible-exposed-infected-removed ($SEIR$) model [2, 4, 6, 10, 14] developed by Rvachev and Longini [21]. It has long been recognized that using any epidemic model having inaccurate values of the parameters in the model will lead to wrong predictions of the spread of the disease and therefore to inappropriate public health responses. However, only a few attempts have been carried out on identification of the model parameters used in the epidemic model. In 2007, Yan et al [27] presented the application of the optimal and sub-optimal control for SARS

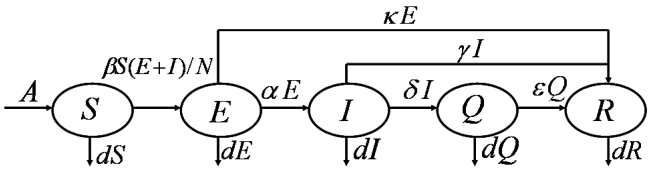

Figure 1: The general transfer diagram for the $SEIQR$ model

outbreak control via Pontryagin's Maximum Principle and genetic algorithms, respectively. In order to be better prepared against future catastrophic epidemics, they studied quarantine and isolation strategies and found that these were the most effective measures in SARS outbreak control. Recently, the Differential Evolution (DE) method has been developed to find optimum solutions for engineering optimization problems in different areas [11, 25, 23, 24, 13, 20]. DE is a fast and efficient algorithm for model optimization [18].

In this study, we propose an epidemic $SEIQR$ model, analyze its mathematical property and establish a DE algorithm for the parameter identification of the model. The effect of the parameter uncertainty on the prediction of the spread of the disease is investigated by a sensitivity analysis.

2. Mathematical Model for the Spread of Epidemic Diseases

In this study, the epidemic $SEIR$ model implemented by Massad et al [14] is extended by adding a new class of quarantine individuals and a new rate at which infected individuals move to the quarantine class. Thus by taking into account the effect of infected-quarantined class, we propose the $SEIQR$ model that monitors the dynamics of five sub-populations (classes) including susceptible ($S(t)$), exposed ($E(t)$), infectious ($I(t)$), quarantined ($Q(t)$), and recovered ($R(t)$) individuals. We then apply it to study the spread of the infectious diseases in a region having a population of 50 million people. Figure 1 shows a typical pathway of the disease transmission, i.e., from S to E then to I, then Q, and finally to R. The system of differential equations for the $SEIQR$ model is:

$$\begin{aligned} \frac{dS}{dt} &= A - \frac{\beta S(E+I)}{N} - dS, \\ \frac{dE}{dt} &= \frac{\beta S(E+I)}{N} - (d+\alpha+\kappa)E, \end{aligned}$$

$$\begin{aligned} \frac{dI}{dt} &= \alpha E - (d+\gamma+\delta)I, \\ \frac{dQ}{dt} &= \delta I - (d+\varepsilon)Q, \\ \frac{dR}{dt} &= \kappa E + \gamma I + \varepsilon Q - dR, \end{aligned} \tag{1}$$

where the parameters b, d, β α, κ, γ, δ and ε are non-negative constants; $A = bS_0$ is a constant recruitment in which the constant b is the natural birth rate and S_0 is the initial value of susceptible individual; d is the natural mortality rate; $\beta = \beta_1 c_n$ in which β_1 is the probability of catching the disease per contact to the infected/exposed person; c_n is the average number of people contacted by each person per day; α is the rate at which the exposed individuals E becomes the infected individuals I; δ is the rate at which individuals leave the infective individuals I for the quarantined individual Q; and κ, γ and ε are the rates at which individuals in the E, I, Q classes recover from the disease or die. Here we consider model (1) with a recruitment-death demographic structure such that

$$\frac{dN}{dt} = A - dN(t),$$

where A may correspond to a constant recruitment, bS_0, or a transient recruitment, $bS(t)$. The model with constant recruitment is used to predict disease transmission in the short period (1-2 months) in a rural area whereas the model with transient recruitment is suitable for the study of the disease transmission in the long period (over 3 months) in the urban area in which immigration rate depends on time [5, 9].

3. Stability Analysis

The differential equation for the total population implies that solutions of the system (1) starting in the region $\Re^5_+ \cup \{0\}$ either approach, enter, or remain in the subset of $\Re^5_+ \cup \{0\}$ defined by

$\mathcal{D} = \{(S, E, I, Q, R) | S \geq 0, E \geq 0, I \geq 0, Q \geq 0, R \geq 0, S+E+I+Q+R \leq A/d\}$.

It can be verified that $\mathcal{D}$ is positively invariant with respect to (1). To find endemic equilibrium, let the right hand side of each equation in system (1) equal zero which gives

$$\begin{aligned} A - \frac{\beta S^*(I^* + E^*)}{N^*} - dS^* &= 0, \\ \frac{\beta S^*(I^* + E^*)}{N^*} - D_1 E^* &= 0, \end{aligned}$$

$$\begin{aligned} \alpha E^* - D_2 I^* &= 0, \\ \delta I^* - D_3 Q^* &= 0, \\ \kappa E^* + \gamma I^* + \varepsilon Q^* - d R^* &= 0, \end{aligned} \tag{2}$$

where $D_1 = d + \alpha + \kappa$, $D_2 = d + \gamma + \delta$ and $D_3 = d + \varepsilon$.

System (2) has two non-negative solutions: $P_0 = (A/d, 0, 0, 0, 0)$ corresponding to the disease-free equilibrium, and $P^* = (S^*, E^*, I^*, Q^*, R^*)$ representing the endemic equilibrium.

Let R_0 denote the basic reproductive number

$$R_0 = \frac{\beta(\alpha + D_2)}{D_1 D_2} = \frac{\beta}{d + \alpha + \kappa} + \frac{\beta\alpha}{(d + \alpha + \kappa)(d + \gamma + \delta)},$$

we obtain from (2) that

$$\begin{aligned} S^* &= \frac{A}{dR_0}, \\ E^* &= \frac{A}{D_1 R_0}\left[R_0 - 1\right], \\ I^* &= \frac{A\alpha}{D_1 D_2 R_0}\left[R_0 - 1\right], \\ Q^* &= \frac{A\alpha\delta}{D_1 D_2 D_3 R_0}\left[R_0 - 1\right], \\ R^* &= \frac{A\left[R_0 - 1\right]}{dD_1 R_0}\left[\kappa + \frac{\gamma\alpha}{D_2} + \frac{\varepsilon\delta\alpha}{D_2 D_3}\right]. \end{aligned}$$

Obviously, P^* is positive only when $R_0 > 1$. Thus it suffices to consider solutions in the region $\mathcal{D}$. It can be shown that the initial value problem is well posed both mathematically and epidemiologically in $\mathcal{D}$.

3.1. Stability of Disease-Free Equilibrium

Theorem 1. *The disease-free equilibrium P_0 of system (1) is asymptotically stable in $\mathcal{D}$ if $R_0 \leq 1$, and is unstable if $R_0 > 1$.*

Proof. To determine the local stability of P_0, we determine the Jacobian

matrix $J(P_0)$ of system (1) at the equilibrium P_0 as follows

$$J(P_0) = \begin{bmatrix} -d & -\beta & -\beta & 0 & 0 \\ 0 & \beta - D_1 & \beta & 0 & 0 \\ 0 & \alpha & -D_2 & 0 & 0 \\ 0 & 0 & \delta & -D_3 & 0 \\ 0 & \kappa & \gamma & \varepsilon & -d \end{bmatrix}.$$

The eigenvalues of the above matrix are $\lambda_1 = -D_2$, $\lambda_2 = \lambda_3 = -d$, $\lambda_4, \lambda_5 = \frac{1}{2}\left[\beta - D_1 - D_2 \pm \sqrt{\Delta}\right]$ where $\Delta = \beta^2 - 2\beta D_1 + 2\beta D_2 + D_1^2 - 2D_1D_2 + D_2^2 + 4\alpha\beta$.

If $R_0 < 1$, we have

$$\beta D_2 - D_1 D_2 + \alpha\beta \ < \ 0$$

and thus

$$\triangle = (D_1 + D_2 - \beta)^2 + 4(\beta D_2 - D_1 D_2 + \alpha\beta) \ < \ (D_1 + D_2 - \beta)^2.$$

That is

$$\sqrt{\triangle} \ < \ D_1 + D_2 - \beta \quad \text{if} \ \ D_1 + D_2 - \beta \geq 0.$$

Consequently

$$\begin{aligned} \lambda_4 &< \frac{1}{2}[-(D_1 + D_2 - \beta) + D_1 + D_2 - \beta] = 0, \\ \lambda_5 &< \frac{1}{2}[-(D_1 + D_2 - \beta) + D_1 - D_2 - \beta] \leq 0. \end{aligned}$$

Thus all the roots of the characteristic equations have negative real parts if $R_0 < 1$. Therefore, the disease-free equilibrium P_0 is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

In order to prove the global stability of the system (1) in the form of $\dot{x} = f(t, x)$ where the function $f(t, x)$ is continuous, we choose the Liapunov function [7]

$$L = \frac{\beta(D_2 + \alpha)}{D_1} E + \beta I,$$

which is continuous in $\mathcal{D}$ and its derivative along the solution of the system

$$\begin{aligned} L^{'}|_{(1)} &= \frac{\partial L}{\partial S} S'(t) + \frac{\partial L}{\partial E} E'(t) + \frac{\partial L}{\partial I} I'(t) + \frac{\partial L}{\partial Q} Q'(t) + \frac{\partial L}{\partial R} R'(t) \\ &= D_2\beta(E + I)\left(\frac{S}{N} R_0 - 1\right) \leq D_2\beta(E + I)(R_0 - 1) \leq 0 \ \text{ for } \ R_0 \leq 0. \end{aligned}$$

Obviously $L^{'} = 0$ only if $I = E = 0$ or $R_0 = 0$. The maximum invariant set in $\{(S, E, I, Q, R) : L^{'} = 0\}$ is the singleton $\{P_0\}$. By *Lasalle*'s invariance principle, P_0 is globally asymptotically stable in $\mathcal{D}$ when $R_0 \leq 1$. □

3.2. Stability of Endemic Equilibrium

Theorem 2. *If $R_0 > 1$, system (1) has a unique endemic equilibrium P^* in $\mathcal{D}$, which is locally asymptotically stable.*

Proof. The Jacobian matrix at P^* is given by

$$J(P^*) = \begin{bmatrix} -\frac{\beta}{N^*}(I^*+E^*) - d & -\frac{\beta}{N^*}S^* & -\frac{\beta}{N^*}S^* & 0 & 0 \\ \frac{\beta}{N^*}(I^*+E^*) & \frac{\beta}{N^*}S^* - D_1 & \frac{\beta}{N^*}S^* & 0 & 0 \\ 0 & \alpha & -D_2 & 0 & 0 \\ 0 & 0 & \delta & -D_3 & 0 \\ 0 & \kappa & \gamma & \varepsilon & -d \end{bmatrix}.$$

The characteristic equation of the above matrix is

$$f(\lambda) = (\lambda + d)(\lambda + D_3)(\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3),$$

where: $S^* = \frac{N^* D_1 E^*}{\beta(I^*+E^*)}$, $I^* = \frac{\alpha E^*}{D_2}$,

$$\begin{aligned} a_1 &= d + D_2 + D_1 I^* + \frac{\beta}{N^*}(I^* + E^*) > 0, \\ a_2 &= dD_2 + \frac{\beta}{N^*}(I^* + E^*)D_1 + \frac{\beta}{N^*}(I^* + E^*)D_2 + \frac{dD_1 I^*}{I^* + E^*} > 0, \\ a_3 &= D_1 D_2 \frac{\beta}{N^*}(I^* + E^*) > 0. \end{aligned}$$

It can be easily seen that $a_1 a_2 - a_3 > 0$ as the term of $-a_3$ can be cleared with the product of the second term of a_1 and the second term of a_2. Hence the Routh-Hurwitz conditions are satisfied. Thus it follows that the endemic equilibrium P^* of (1), which exists if $R_0 > 1$, is always locally asymptotically stable. □

4. DE Algorithm for Parameter Identification

Differential evolution (DE) algorithm which was introduced by Price and Storn [19] can be used to determine the values of the system parameters in many problems. DE algorithm is a new heuristic approach with three major advantages: capable of finding the true global minimum regardless the initial parameter values, fast convergence, and using fewer control parameters [22]. It is a population-based direct-search algorithm like genetic algorithms using mutation, crossover and selection operators. Here, we use the DE algorithm to find β, α, κ, γ, δ, ε that minimize the objective function J:

$$J(\beta,\alpha,\kappa,\gamma,\delta,\varepsilon) = \left[\frac{\sum_{m=1}^{M_s}\left|S_m-\widetilde{S}_m\right|^2}{\sum_{m=1}^{M_s}|S_m|^2}\right]^{1/2} + \left[\frac{\sum_{m=1}^{M_e}\left|E_m-\widetilde{E}_m\right|^2}{\sum_{m=1}^{M_e}|E_m|^2}\right]^{1/2}$$
$$+ \left[\frac{\sum_{m=1}^{M_i}\left|I_m-\widetilde{I}_m\right|^2}{\sum_{m=1}^{M_i}|I_m|^2}\right]^{1/2} + \left[\frac{\sum_{m=1}^{M_q}\left|Q_m-\widetilde{Q}_m\right|^2}{\sum_{m=1}^{M_q}|Q_m|^2}\right]^{1/2} + \left[\frac{\sum_{m=1}^{M_r}\left|R_m-\widetilde{R}_m\right|^2}{\sum_{m=1}^{M_r}|R_m|^2}\right]^{1/2},$$

where S, E, I ,Q and R are the measured data as shown in Figure 2, $\widetilde{S}$, $\widetilde{E}$, $\widetilde{I}$, $\widetilde{Q}$ and $\widetilde{R}$ are the computed data with parameters $\{\beta,\alpha,\kappa,\gamma,\delta,\varepsilon\}$, and M_s, M_e, M_i, M_q and M_r denote respectively the the final time step of each classes.

To solve the optimization problem, we represent 6 parameters by a 6-dimensional vector. The DE algorithm randomly creates a population of possible solution vectors $\{x_i^G : i = 1,2,...,N_p\}$ at each generation G. Each element of the solution vector composed of 6-parameters is defined by $x_i^G := \{x_{i,j}^G : j = 1,2,3,4,5,6\}$. The DE algorithm starts with randomly creating a population of Np solution vectors, followed by performing the following three main steps (mutation, crossover, and selection) until the termination criteria are met:

Step 1. Initialization: The initial population is generated as

$$x_{i,j}^G = x_j^L + \psi_i(x_j^U - x_j^L) \quad (j = 1,2,...,6)\,,$$

where x_j^L and x_j^U are the lower and upper bounds of the j-th parameter, respectively, ψ_i is a random number which is uniformly distributed between 0 and 1.

Step 2. Mutation: For each vector x_i^G, a mutant vector v_i^G is generated according to

$$v_i^G = x_{\text{best}}^G + P(x_m^G - x_n^G) \quad (i = 1,2,...,N_p)$$

where x_{best}^G is the best solution and $\{x_m^G, x_n^G\}$ are two distinct arbitrary vectors at generation G, $P \in [0,2]$ is a user-supplied mutation factor.

Step 3. Crossover: A set of vectors $\{u_i^G\}$ are formed where

$$u_i^G = \begin{cases} v_{i,j}^G & \text{for } \text{rand}_j(0,1) \le C_R\,, \\ x_{i,j}^G & \text{otherwise}\,, \end{cases}$$

where $C_R \in [0,1]$ is a crossover constant.

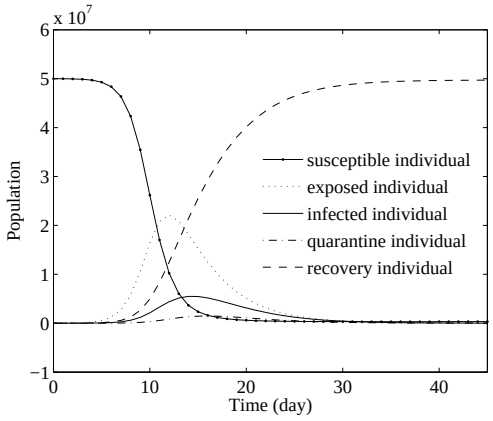

Figure 2: The measured data of the epidemic $SEIQR$ model

Step 4. Selection: The next generation vectors (offspring) are selected as

$$x_i^{G+1} = \begin{cases} u_i^G & \text{for } J(u_i^G) < J(x_i^G)\,, \\ x_i^G & \text{otherwise.} \end{cases}$$

Step 5. Repeat steps 2-4 until the termination criteria are met.

5. Validation and Sensitivity Analysis

5.1. Validation Analysis of the DE Algorithm

To obtain optimal solutions for the β, α, κ, γ, δ and ε of the $SEIQR$ model, we run the DE algorithm twice to make sure that with different initial guess, we obtain the same parameter values. The measured input data S, E, I, Q and R are given in Figure 2. Also, $b = 3.526301 \times 10^{-5}$, $d = 1.73180 \times 10^{-5}$ (demographics of Thailand are obtained from department of provincial administration). The DE algorithm estimates the model parameter: $\beta = 1$, $\alpha = 1/9$ (the mean value of the incubation period is 9 days [16]), $\kappa = 1/7$, $\gamma = 1/7$ (the mean value of infectious or death period is 7 days [15]), $\delta = 1/5$ and $\varepsilon = 0.7$ by minimizing the objective function J.

Figure 3 shows convergence of J, β, α, κ, γ, δ and ε. The objective function $J = 8.045998 \times 10^{-6}$ and the simulated parameters $\beta = 1.0$, $\alpha = 1.111119 \times 10^{-1}$, $\kappa = 1.428566 \times 10^{-1}$, $\gamma = 1.428621 \times 10^{-1}$, $\delta = 1.999976 \times 10^{-1}$, $\varepsilon = 6.999882 \times 10^{-1}$ from the first run; while for the second round the objective

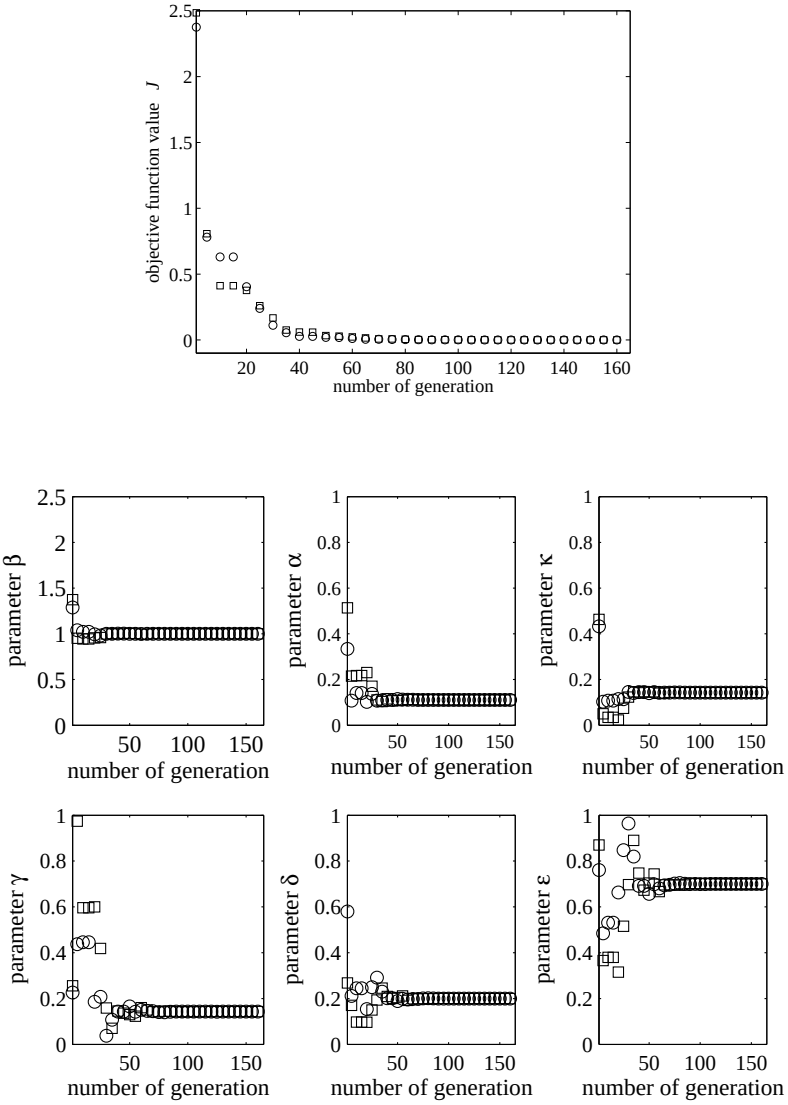

Figure 3: Validation of the DE for predicting model parameters: Objective function J and parameter β, α, κ, γ, δ and ε. The circles and squares represent results obtained from the first run and the second run respectively.

function $J = 8.913791 \times 10^{-6}$ and the simulated parameters $\beta = 9.999998 \times 10^{-1}$, $\alpha = 1.111121 \times 10^{-1}$, $\kappa = 1.428562 \times 10^{-1}$, $\gamma = 1.428612 \times 10^{-1}$, $\delta = 1.999970 \times 10^{-1}$, $\varepsilon = 6.999933 \times 10^{-1}$. Both can fit well to the measured data.

5.2. Sensitivity Analysis

Common method for quantifying the effects of uncertainties in the values of the parameters in the model on the predictions of the epidemic model is sensitivity analysis In this study, a few parameters are varied, whereas the other are held constant. Let the natural birth rate $b = 3.526301 \times 10^{-5}$, the natural mortality rate $d = 1.7318 \times 10^{-5}$, and other parameters be constant. Influence of transmission rate on the spread of the disease is investigated using three values of transmission rate: $\beta = \beta_0$ and $(1 \pm 20\%)\beta_0$, where $\beta_0 = 1$. The results as shown in Figure 4 indicate that transmission rate has significant effect on the spread of the disease on each group of population. By increasing transmission rate, the number of susceptible individuals decreases and the number of other individuals increases significantly. It is also apparent that a higher transmission rate gives a faster spread of the disease as shown in Figure 4(b-d). Influence of incubation rate on the disease transmission is shown in Figure 5. Three different incubation rates used in this investigation are set to $\alpha = \alpha_0$ and $(1 \pm 80\%)\alpha_0$, where $\alpha_0 = 1/9$. The incubation rate has very little effect on the susceptible and recovered groups as shown in Figure 5(a) and 5(e), but it has significant effect on the exposed, infected and quarantined groups as shown in Figure 5(b), 5(c) and 5(d), respectively.

To investigate the effect of quarantine on the spread of the disease, we plot the solution sets of the *SEIQR* model with three different quarantine rates, $\delta = \delta_0$ and $(1 \pm 80\%)\delta_0$, where $\delta_0 = 1/5$, for individuals leaving the infected class to the quarantine class. The results as shown in Figure 6(c-d) indicate that increasing the quarantine rate leads to decrease in the number of infected individuals and increases the number of quarantined individuals.

To analyze sensitivity between model parameters, we plot E and I with respect to investigated model parameters. Figure 7 shows profiles of the exposed individuals and the infected individuals obtained from the models with 40% and 80% increments of investigated parameters (β,α and δ) from β_0, α_0, and δ_0 when other parameters are fixed. The results as shown in Figure 7(a) and 7(b) indicate that transmission rate β gives higher effect on disease transmission than other two rates, α and δ. Increasing the transmission rate β from 1.0 to 1.4 (40% increment) and 1.8 (80%increment) gives earlier critical period of

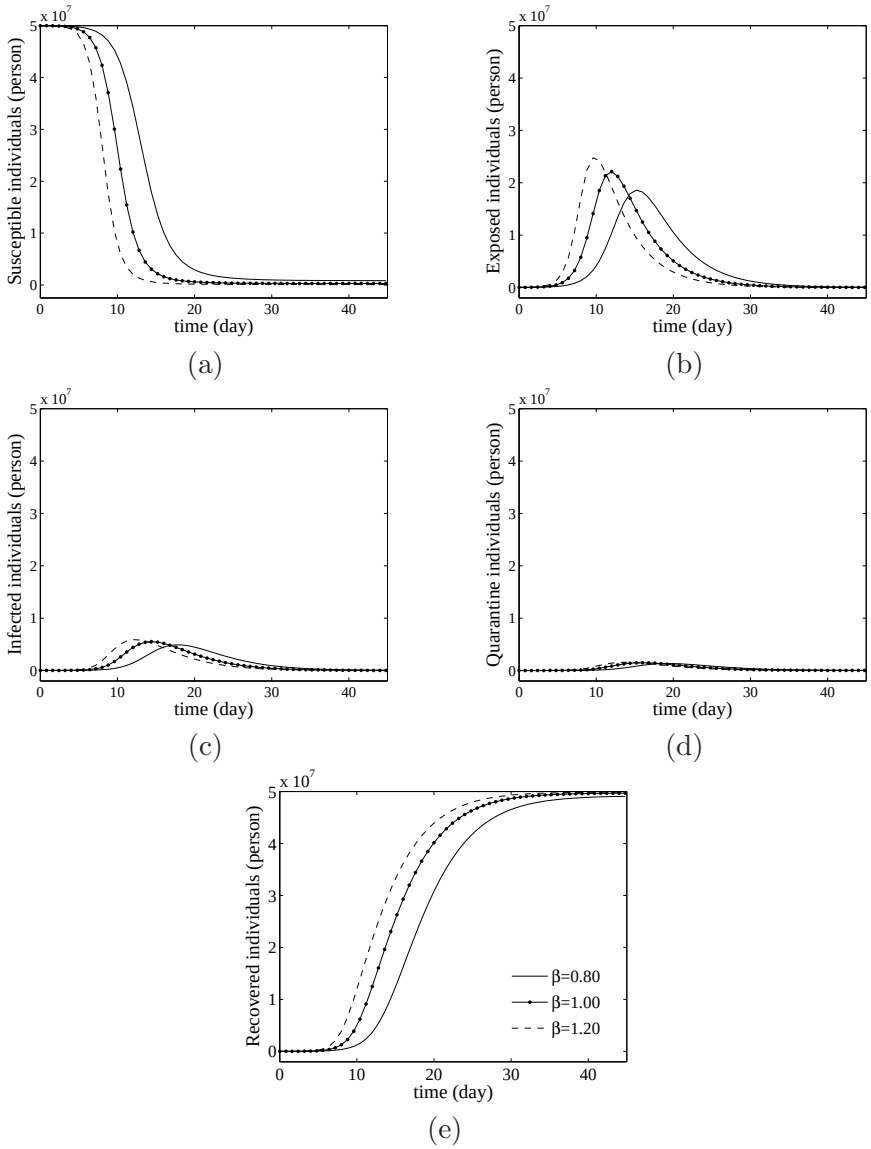

Figure 4: Influence of transmission rate on each group of population: (a) Susceptible individuals; (b) Exposed individuals; (c) Infected individuals; (d) Quarantined individuals; (e) Recovered individuals

disease transmission from 11-13 days to 8-11 days and 6-9 days after outbreaks, respectively. The number of exposed individuals increases from 22×10^6 persons

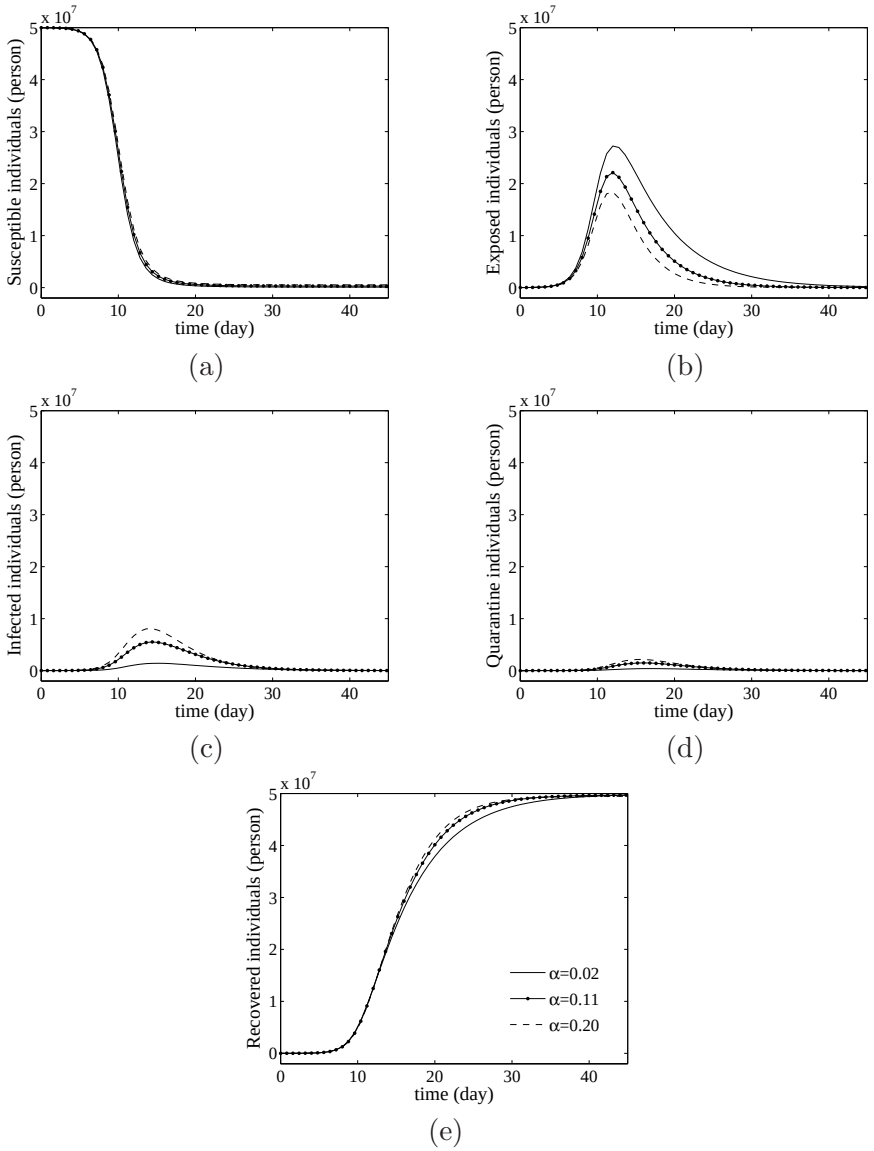

Figure 5: Influence of incubation rate on each group of population: (a) Susceptible individuals; (b) Exposed individuals; (c) Infected individuals; (d) Quarantined individuals; (e) Recovered individuals

to 26×10^6 persons and to 30×10^6 persons and the number of infected individuals increases from 5.5×10^6 persons to 6.1×10^6 persons and to 6.4×10^6 persons,

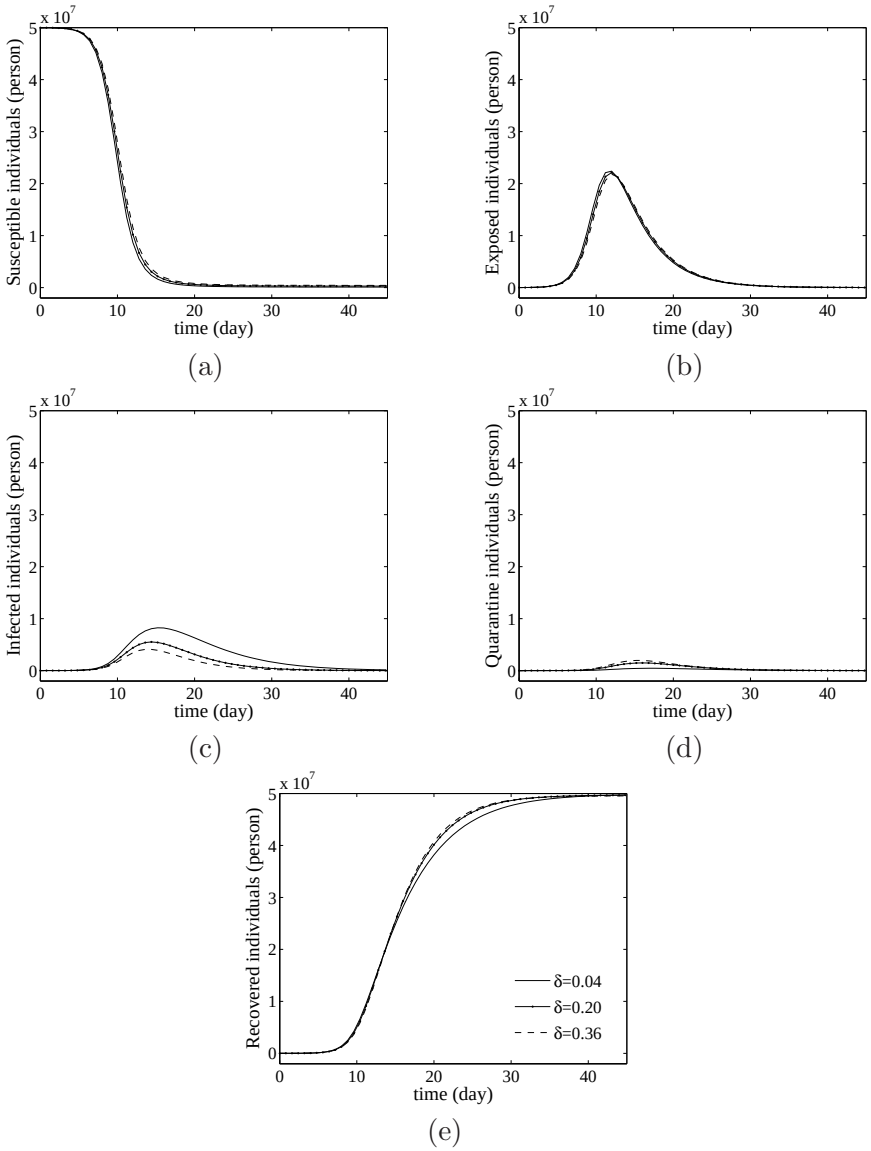

Figure 6: Influence of the rate for individuals leaving the infective compartment to quarantine compartment on each group of population: (a) Susceptible individuals; (b) Exposed individuals; (c) Infected individuals; (d) Quarantined individuals; (e) Recovered individuals

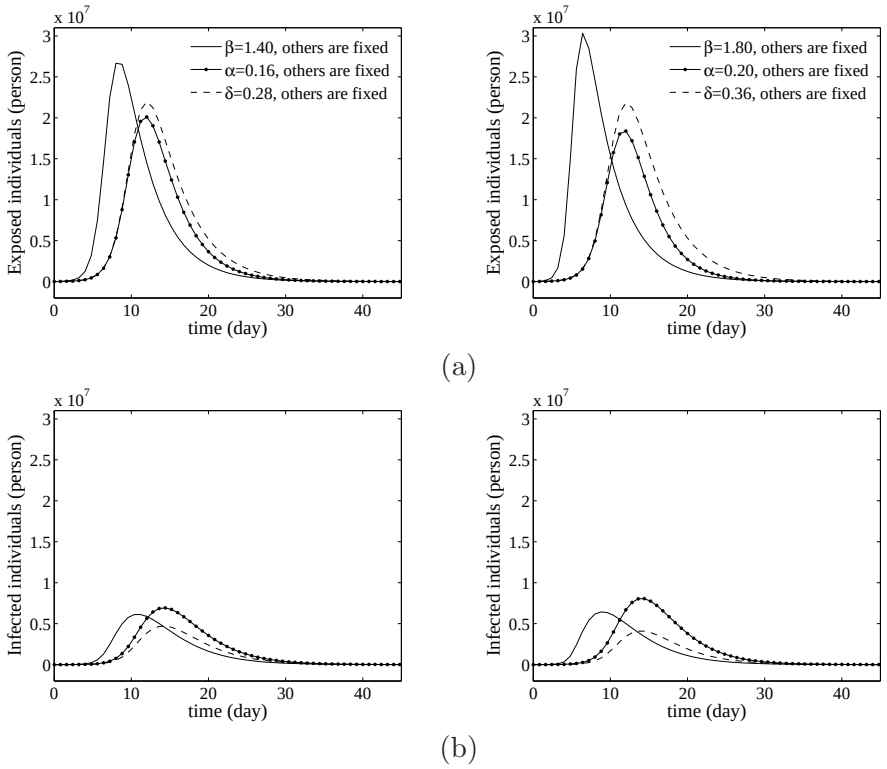

Figure 7: Influence of model parameters β, α and δ on disease transmission: (a) Exposed individuals 40% (left column) and 80% (right column) increments of investigated parameters; (b) Infected individuals 40% and 80% increments of investigated parameters

respectively.

Figure 8 shows profiles of the exposed individuals and the infected individuals obtained from the models with 40% and 80% increments of investigated parameters (κ, γ and ε) from κ_0, γ_0, and ε_0 when other parameters are fixed. The results as shown in Figure 8(a) and 8(b) indicate that the transmission rate κ has higher effect on disease transmission than other two rates, γ and ε. Increasing the transmission rate κ from 0.14 to 0.20 (40% increment) and 0.26 (80%increment) gives later critical period of disease transmission from 11-13 days to 12-14 days and 13-15 days after outbreaks, respectively. The number of exposed individuals decreases from 22×10^6 persons to 19×10^6 persons and to 16×10^6 persons and the number of infected individuals decreases from 5.5×10^6

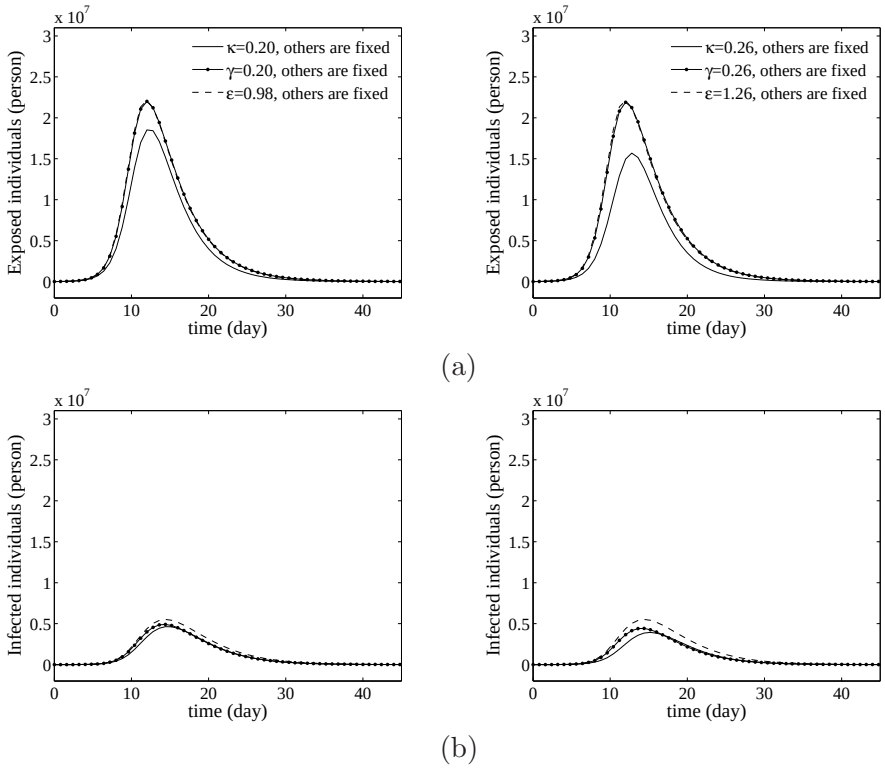

Figure 8: Influence of recovery rates κ, γ and ε on disease transmission: (a) Exposed individuals 40% (left column) and 80% (right column) increments of investigated parameters; (b) Infected individuals 40% and 80% increments of investigated parameters

persons to 4.6×10^6 persons and to 3.9×10^6 persons, respectively.

6. Conclusions

In order to be prepared against future catastrophic epidemics, each country should have an epidemic model which is appropriate for the specific conditions of the country. This means to determine the values of the parameters in the model which can capture the behavior of the next outbreak. In this paper, an epidemic $SEIQR$ model has been proposed and analyzed mathematically. The local and global stability of the disease-free equilibrium, and the existence and uniqueness of the endemic equilibrium and its local stabil-

ity have been established. A differential evolution based algorithm has also been established for identifying model parameters of the susceptible-exposed-infected-quarantined-recovered ($SEIQR$) model. Using a set of measured data from the first outbreak, model parameters of the epidemic $SEIQR$ model can be identified successfully by the algorithm. To quantify the effects of the parameter uncertainty on the predictions of the epidemic $SEIQR$ model, a sensitivity analysis is carried out and the results have been presented in the paper. The proposed $SEIQR$ model enables to capture the spread dynamic of severe disease for the first outbreak. Once the disease occurs, vaccine can be developed. The model should be extended by adding a new vaccination class in order to predict the behavior of the spread of the disease correctly and to control the spread of the disease successfully.

Acknowledgments

The research was supported by the Commission on Higher Education Staff Development Project of the University of Mahidol University for the support of this research.

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