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Stability Analysis of a Mathematical Model SI_uI_aQR for Covid-19 with the Effect of Contamination Control (Filiation) Strategy

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Abstract

In this study, using a system of delay nonlinear ordinary differential equations, we introduce a new compartmental epidemic model considered effect of filiation (contamination) control strategy to the spread of Covid-19. Firstly, the formulation of this new SI_uI_aQR epidemic model with delay process and the parameters arised from isolation and filiation is formed. Then the disease-free and endemic equilibrium points of the model is obtained. Also, the basic reproduction number $\mathscr{R}_0$ is found by using the next generation matrix method and the results on stabilities of the disease-free and endemic equilibrium points are investigated. Finally some examples are presented to show the effect of filiation control strategy.

1. Introduction

In December 2019, Hubei province in Wuhan, China, became the centre of an outbreak of Covid-19. Then the disease caused by the new type coronavirus has affected hundreds of countries by spreading rapidly all around the world. The World Health Organization (WHO) declared the epidemic as a pandemic (global epidemic) on March 11, 2020 due to the fact that it caused the loss of thousands of lives. Since it could not be found exactly being effective drugs or vaccine in today's stages for Covid-19, to control of spreading of the disease, the governments with the support of its health authorities have immediately tried to made effective control measures including procedures such as isolating of people suspected to have the disease, closely tracking of contacts, collecting of epidemiological and clinical data from patients, boosting of diagnostic and providing treatment services.

Mathematical modelling has an important role in understanding of problems and phenomenons in some areas such as medicine, biology and epidemiology, [1]-[3]. Especially, epidemic diseases such as Covid-19, the global agenda in 2020, are some of the main threats that are seriously affecting humanity. Various studies have been introduced to overcome the problems caused by such diseases for a long time, [4]-[6]. Modelling of epidemic diseases as mathematically is quite important in terms of controlling and reducing effects of the outbreaks, [7]-[10]. Looking at the literature, there are many compartmental models determined the basic principles for the spread of a disease in a population. Kermack and Mc Kendrick with their study [7] have pioneered these compartmental mathematical models which are used extensively. They have tried to explain the spreading of an infectious disease in the course of time for a closed population. In the model, the population exposed to an infectious disease has been divided into three groups. First group (S : Susceptibles) consists from individuals who are not yet infected and have not immune to the pathogen. In the other group (I : Infectious) consisting of infectious individuals, the members can be transmitted the disease to the susceptible individuals via effective contacts. The last group (R : Recovered) is formed from individuals who recovered and have immune against the pathogen. This model is called as "SIR" model with the initials of the group names.

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After, many authors have studied intensively on this model and in detail to carry further forward this model, [11]-[15].

In recent years, various complemental models have been considered in order to explain the more complex phenomena in diseases. In this sense, by giving some additional circumstances which will have more reality to the basic epidemic models, many studies have been obtained. For instance, a lot of *SEIR* epidemic models constructed by taking into account a latent compartment (E : Exposed) have been considered. This compartment consists of individuals who are infected but are not yet infectious although an effective contact has occurred between the infectious. Many *SEIR* models have been studied with various meaningful details, [16]-[22].

On the other hand, "quarantine" is another factor affecting to control of the spreading of diseases. This transaction is one of commonly used method for preventing and controlling spread of diseases. In today's society, with increasing the effectiveness of the quarantine process, spreading of some diseases has significantly decreased. This fact entails to consider the quarantine process in the models by giving an additional compartment Q represented the quarantined individuals. Recently, the epidemic models with quarantine has been investigated by some authors, [23]-[26].

During the early stages of the Covid-19 outbreak, a lot of studies, such as [27]-[33],[35], on its transmission dynamics has been revealed. In this study, using a system of delay nonlinear ordinary differential equations, we aim to introduce a time delay compartmental epidemic model considered the effect of filiation control strategy via quarantine in spreading of Covid-19 and other diseases. In the literature, there exist some compartmental models including the quarantine class consisting of some of the exposed or infectious, [33, 34]. However, in the model presented in this study depends on the fact that individuals who contacted with the infectious but whose exposure status is not yet known are quarantined. The model differs from many studies in the literature with this feature. Considering that the latent period for Covid-19 can be completed before the incubation period and the rate of asymptomatic infectious is quite high, it can be seen that the model is competent in modeling diseases such as Covid-19.

In the next section, firstly, the formulation of this new SI_uI_aQR epidemic model with delay process and the parameters arised from isolation and filiation is formed. Then the feasible region which is being positive invariant set for the model and guaranteeing the boundedness of the functions is determined.

In the third section, disease-free and endemic equilibrium points of the model are obtained. After the basic reproduction number $\mathscr{R}_0$ is found by using the next generation matrix method, the local stabilities of the disease-free and endemic equilibrium points are proved using the corresponding characteristic equation. Then the global stability of disease-free equilibrium point is handled via LaSalle's Invariance Principle associated with the Lyapunov function. In the last section, some examples are presented to show the effect of filiation control strategy.

2. Description of the model

In this part instruction of the model, defining the parameters and the transitions between the compartments are introduced.

As it is known that some individuals may have no symptoms throughout their infectiousness and these individuals are called as asymptomatic infectious. We use the notations I_u (who are unaware of their infectious) and I_a (who are aware of their infectious) to denote the compartment of asymptomatic and symptomatic infectious, respectively. Indeed I_a consists of symptomatics, and some of asymptomatic individuals whose positivity is known via test (i.e. confirmed cases). Further, asymptomatic individuals are unaware of the fact that own being infectious, and what is worse they may not avoid contact with susceptibles.

Also we assume that the all members of I_a have been isolated during the treatment in a hospital or home, and any members of I_a have not contacted with susceptibles. So the class I_a can be seen as the *treatment compartment.* Thus it is assumed that the disease spreads only via I_u. As a result of this fact, it is very important detecting of asymptomatics (members of I_u) for the course of the disease.

On the other hand we should mention the latent and incubation period. Incubation period is the time elapsed between exposure to a pathogen and when symptoms and signs are first apparent. Depending on the disease, the person may or may not be infectious during the incubation period. The latent period is the time interval between when an individual or host is infected by a pathogen and when he or she becomes infectious, i.e. capable of transmitting pathogens to other susceptible individuals.

According to our model it is assumed that all new cases born from the contact between susceptibles and asymptomatics infectious (I_u). The individuals who are infected at time t are *asymptomatic at the rate r.* So, the number of new individuals who are symptomatic (i.e, known to be positive) is $(1-r)\beta S(t) I_u(t)$ at each time t. Where β is the effective contact rate between susceptibles and asymptomatics infectious (I_u). Also, when an individual who became infectious is detected, the persons who are contacted to him in the past several days should be taken to quarantine from S and controlled during one incubation period.

In serious cases, quarantine of individuals suspected of being exposed to an epidemic disease is one of the most important and effective public health measures used in struggle against the disease. We assume that the number of individuals who contact with an individual identified as positive yet is m and the part of them at the rate q are taken to quarantine. We are called q as "*filiation (chain of contamination) control rate*" such as $0 \leq q \leq 1$. So $(1-r)\,mq\beta S(t)\,I_u(t)$ is the number of new individuals transferred to Q from S. Also some of individuals, who are not quarantined although they contact with newly positive cases, may be asymptomatic (at the rate p). We represent this transmission with $(1-r)\,m\,(1-q)\,p\beta S(t)\,I_u(t)$.

On the other hand it is assumed that all individuals in the quarantine (Q) do not contact with each other and susceptibles. In addition, according to our model, the individuals who have completed the quarantine process (this is one incubation period) are tested at the rate y and it is assumed that there exist positive cases at the rate p of them.

We should immediately note that, of course there will the individuals who have not been tested but have the risk in the sense of becoming positive. (Even if they may think that no there is any positivity risk). So these individuals will take part in I_u. Thus it is assumed that the individuals whose quarantine process has been completed but who have not been tested have join to I_u at the rate $(1-y)p$ (the rate of positivity of not tested individuals) and to S at the rate $(1-y)\,(1-p)$ (the rate of negativity of not tested individuals). In addition, taking into account that rate of individuals who have been tested and negative is $y\,(1-p)$, the individuals whose quarantine process has been completed turn to S at the rate $(1-p)$.

Also, taking into account that the time taking in quarantine is τ and some individuals will death at the rate d with natural causes (not caused by disease), it is obtained that the total number of individuals who leave from Q at time t is $(1-r)\,mq\beta S(t-\tau)\,I_u(t-\tau)\,e^{-d\tau}$. This number is obtained by the solution of following initial value problem

$$\left(\frac{\partial}{\partial t}+\frac{\partial}{\partial \tau}\right)\overline{Q}(t,\tau)=-d\overline{Q}(t,\tau),\quad \overline{Q}(t,0)=(1-r)\,mq\beta S(t)I_u(t).$$

Where $\overline{Q}(t,\tau)$ describes the number of individuals who are joined to quarantine at time $t-\tau$ and still surviving at the time t.

Under these assumptions, the system of ordinary differential equations which reflects the model is as follows. In order to better understand, in the first stage, the transitions between compartments, simplifications have not been made intentionally in the equations.

$$\begin{aligned}
\frac{dS(t)}{dt} &= \Lambda-(1-r)\,\beta S(t)\,I_u(t)-[r+(1-r)\,m\,(1-q)\,p]\,\beta S(t)\,I_u(t)-(1-r)\,mq\beta S(t)\,I_u(t)\\
&\quad -dS(t)+(1-p)\,(1-r)\,mq\beta S(t-\tau)\,I_u(t-\tau)\,e^{-d\tau},\\
\frac{dI_u(t)}{dt} &= [r+(1-r)\,m\,(1-q)\,p]\,\beta S(t)\,I_u(t)+(1-y)\,p\,(1-r)\,mq\beta S(t-\tau)\,I_u(t-\tau)\,e^{-d\tau}\\
&\quad -(d+\theta)\,I_u(t),\\
\frac{dI_a(t)}{dt} &= (1-r)\,\beta S(t)\,I_u(t)+yp\,(1-r)\,mq\beta S(t-\tau)\,I_u(t-\tau)\,e^{-d\tau}\\
&\quad -(d+\mu+\gamma)\,I_a(t),\\
\frac{dQ(t)}{dt} &= (1-r)\,mq\beta S(t)\,I_u(t)-(1-y)\,p\,(1-r)\,mq\beta S(t-\tau)\,I_u(t-\tau)\,e^{-d\tau}\\
&\quad -(1-p)\,(1-r)\,mq\beta S(t-\tau)\,I_u(t-\tau)\,e^{-d\tau}\\
&\quad -yp\,(1-r)\,mq\beta S(t-\tau)\,I_u(t-\tau)\,e^{-d\tau}-dQ(t),\\
\frac{dR(t)}{dt} &= \theta I_u(t)+\gamma I_a(t)-dR(t),
\end{aligned}\tag{2.1}$$

Where $S(t)$, $I_u(t)$, $I_a(t)$, $Q(t)$ and $R(t)$ denote the number of the susceptible, unaware infectious, aware infectious, quarantined and recovered individuals at time t, respectively. The total population size at time t is $N(t)$ and $N(t)=S(t)+I_a(t)+I_u(t)+Q(t)+R(t)$ for all $t>0$, such that all these functions are nonnegative. Also, all newborn individuals is be included the population by entering with the rate Λ to the compartment S. μ represents the death rate derived from the disease. θ denotes the transition rate to R from I_u. On the other hand, all parameters in the model are nonnegative constants. After necessary simplifications and abbreviations, the transition diagram between compartments of the model is as follows.

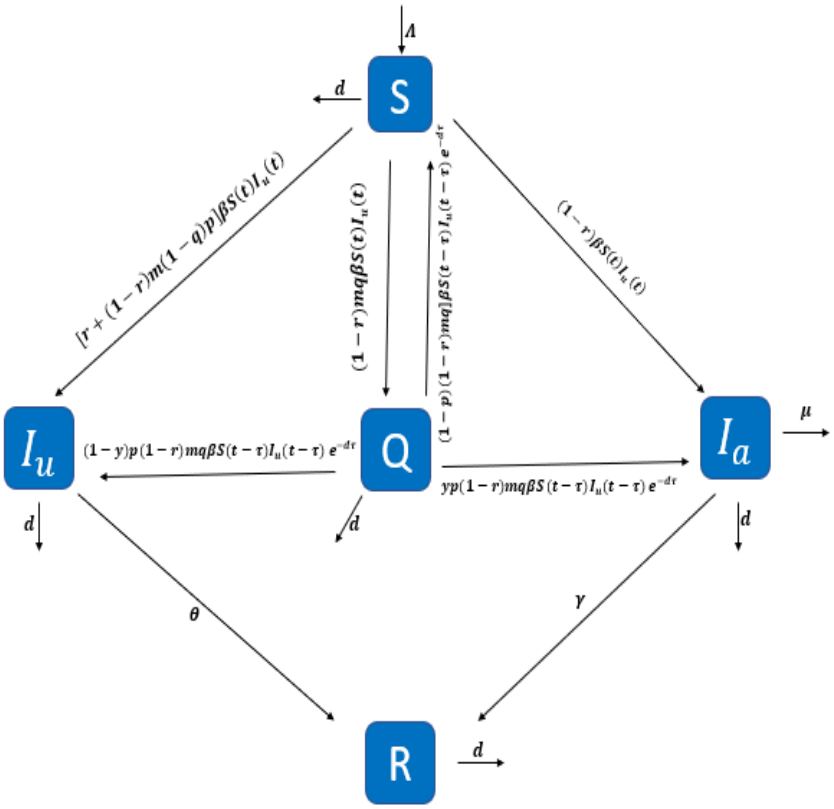

Figure 2.1: Transition diagram of the SI_uI_aQR model for spreading of the disease

Now, we determine the feasible region which is being positive invariant set for system (2.1).
Summing equations in (2.1), we obtain

$$\begin{aligned} \frac{dS}{dt}+\frac{dI_u}{dt}+\frac{dI_a}{dt}+\frac{dQ}{dt}+\frac{dR}{dt} &= \frac{dN}{dt} \\ &= \Lambda - dN(t) - \mu I_a(t) \\ &\leq \Lambda - dN(t). \end{aligned} \tag{2.2}$$

If we use the fact that

$$x(t) = \frac{\Lambda}{d}\left(1-e^{-dt}\right)+x(0)e^{-dt}$$

is the solution of the equation

$$x'(t) = \Lambda - dx(t)$$

then we get the maximal solution of (2.2) as

$$N(t) = \frac{\Lambda}{d}\left(1-e^{-dt}\right)+N(0)e^{-dt}$$

for all $t \geq 0$. Then we can say $N(0) \leq \frac{\Lambda}{d}$ implies $N(t) \leq \frac{\Lambda}{d}$, for all $t \geq 0$. This means that all solutions of system (2.1) are eventually confined in this region bounded with Λ/d. So

$$\Gamma = \left\{(S,I_u,I_a,Q,R) \in C\left([-\tau,\infty), \left[0,\frac{\Lambda}{d}\right]^5\right) : N(t) \leq \frac{\Lambda}{d}\right\}$$

is positively invariant set for the model (2.1) and to concentrate on this restricted area will be enough for analysing of the model.
It can be seen that functions I_a, Q and R do not appear in the other equations of the system (2.1). Also there is no nonlinear relationship between these functions. So the dynamics of (2.1) are the same as the following reduced system (2.3), and it is sufficient to study on the system (2.3). Dynamics and behaviour of functions S and I_u determine the state of the others. Also

taking into account that the disease spreads only contacts between S and I_u, this reducing is meaningful epidemiologically as well.

$$\begin{cases} \frac{dS}{dt} = \Lambda - [1+(1-r)m(q+p(1-q))]\beta S(t) I_u(t) - dS(t) \\ \quad +(1-p)(1-r)mq\beta S(t-\tau) I_u(t-\tau) e^{-d\tau} \\ \frac{dI_u}{dt} = [r+(1-r)m(1-q)p]\beta S(t) I_u(t) - (d+\theta) I_u(t) \\ \quad +(1-y)p(1-r)mq\beta S(t-\tau) I_u(t-\tau) e^{-d\tau} \\ S(t) = g_1(t) \\ I_u(t) = g_2(t) \end{cases} \begin{array}{l} t \geq 0 \\ \\ \\ -\tau \leq t \leq 0 \end{array} \tag{2.3}$$

Where $g_i \in C\left([-\tau,0], \left[0, \frac{\Lambda}{d}\right]\right)$, $i=1,2$ and $(S,I_u) \in C\left([-\tau,\infty), \left[0,\frac{\Lambda}{d}\right]^2\right)$. If we choose $x=(x_1,x_2)$, $x^t(\theta) = x(t+\theta)$, $g=(g_1,g_2)$ and $f: \Omega \to \left[0,\frac{\Lambda}{d}\right]^2$ such that $\Omega \subset C\left([-\tau,0], \left[0,\frac{\Lambda}{d}\right]^2\right)$ then finding the solution of the system (2.3) is equivalent to solving the following equation

$$\begin{aligned} x'(t) &= f\left(x^t\right), t \geq 0 \\ x_0 &= g. \end{aligned} \tag{2.4}$$

Where f is defined by

$$\begin{aligned} f_1(x) &= \Lambda - [1+(1-r)m(q+p(1-q))]\beta x_1(0) x_2(0) - dx_1(0) + (1-p)(1-r)mq\beta x_1(-\tau) x_2(-\tau) e^{-d\tau} \\ f_2(x) &= [r+(1-r)m(1-q)p]\beta x_1(0) x_2(0) - (d+\theta) x_2(0) + (1-y)p(1-r)mq\beta x_1(-\tau) x_2(-\tau) e^{-d\tau} \end{aligned}$$

for $x=(x_1,x_2)=(S,I_u) \in \Omega$. Also, as known $C\left([-\tau,0], \left[0,\frac{\Lambda}{d}\right]^n\right)$ is a Banach space of continuous functions, and $\|\cdot\|_C$ denotes the norm on $C\left([-\tau,0], \left[0,\frac{\Lambda}{d}\right]^n\right)$ and is defined by

$$\|x\|_C = \sup\left\{\sum_{i=1}^{n} |x_i(t)| : -\tau \leq t \leq 0\right\}.$$

3. Analysis of the model

In this section, we interest with qualitative analysis of the model (2.3). We firstly show the uniqueness of the solution of the model (2.3).

Theorem 3.1. *There exists a unique solution of the eqution (2.3) with initial function $x_1(t) = g_1(t)$, $x_2(t) = g_2(t)$ for $-\tau \leq t \leq 0$.*

Proof. It sufficient to show that f, given in (2.4), is Lipschitz continuous in every compact subset $M \subset \Omega$. Let $x=(x_1,x_2)$, $y=(y_1,y_2) \in M$, then we can write from the description of f

$$\begin{aligned} & \|f(x) - f(y)\| \\ = \; & |f_1(x) - f_1(y)| + |f_2(x) - f_2(y)| \\ = \; & [1+(1-r)m(q+p(1-q))]\beta |x_1(0)x_2(0) - y_1(0)y_2(0)| \\ & +d|y_1(0) - x_1(0)| \\ & +(1-p)(1-r)mq\beta e^{-d\tau} |x_1(-\tau)x_2(-\tau) - y_1(-\tau)y_2(-\tau)| \\ & +[r+(1-r)m(1-q)p]\beta |x_1(0)x_2(0) - y_1(0)y_2(0)| \\ & +(d+\theta)|y_2(0) - x_2(0)| \\ & +(1-y)p(1-r)mq\beta e^{-d\tau} |x_1(-\tau)x_2(-\tau) - y_1(-\tau)y_2(-\tau)| \\ \leq \; & [1+(1-r)m(q+p(1-q))]\beta (|x_2(0)| + |y_1(0)|) \|x-y\|_C \\ & +(d+\theta)\|x-y\|_C \\ & +(1-p)(1-r)mq\beta e^{-d\tau} (|x_2(-\tau)| + |y_1(-\tau)|) \|x-y\|_C \\ & +[r+(1-r)m(1-q)p]\beta (|x_2(0)| + |y_1(0)|) \|x-y\|_C \\ & +(1-y)p(1-r)mq\beta e^{-d\tau} (|x_2(-\tau)| + |y_1(-\tau)|) \|x-y\|_C \\ = \; & [1+r+(1-r)m(q+2p(1-q))]\beta (|x_2(0)| + |y_1(0)|) \|x-y\|_C \\ & +(d+\theta)\|x-y\| \\ & +[(1-r)mq(1-p+p(1-y))]\beta e^{-d\tau} (|x_2(-\tau)| + |y_1(-\tau)|) \|x-y\|_C \end{aligned} \tag{3.1}$$

Taking into account the fact $|x_i(t)| \leq \frac{\Lambda}{d}$ for $-\tau \leq t \leq 0$, $i = 1,2$ then we conlude

$$\|f(x) - f(y)\| \leq \left(\frac{2\Lambda}{d}(A+B) + d + \theta \right) \|x - y\|_C$$

from (3.1), where $A = [1 + r + (1-r)m(q + 2p(1-q))]\beta$ and $B = [(1-r)mq(1-p+p(1-y))]\beta e^{-d\tau}$.
So if we take

$$l \geq \frac{2\Lambda}{d}(A+B) + d + \theta,$$

the inequality

$$\|f(x) - f(y)\| \leq l \|x - y\|_C$$

hold in every compact subset $M \subset \Omega$. This completes the proof. □

3.1. Disease-free equilibrium point and basic reproduction number

The disease-free equilibrium point of the model (2.3) is easily found as

$$\varepsilon_0 = (S^0, I_u^0) = \left(\frac{\Lambda}{d}, 0 \right).$$

The number of secondary infections produced by a single infected individual introduced into a population is a threshold value and very significant for providing information about the course of the disease in the population. This number represented by $\mathscr{R}_0$ is also known as the basic reproduction number. Now, let us get the basic reproduction number $\mathscr{R}_0$ of system (2.3) by means of the next generation matrix method.
Let $X = (I_u, S)^T$. Then the system (2.3) can be written in the form

$$\frac{dX}{dt} = \mathscr{F}(X) - \mathscr{V}(X).$$

such that

$$\mathscr{F}(X) = \begin{bmatrix} [r + (1-r)m(1-q)p]\beta S(t) I_u(t) + (1-y)p(1-r)mq\beta S(t-\tau) I_u(t-\tau) e^{-d\tau} \\ \\ 0 \end{bmatrix}$$

and

$$\mathscr{V}(X) = \begin{bmatrix} (d+\theta) I_u(t) \\ \\ [1 + (1-r)m(q + p(1-q))]\beta S(t) I_u(t) + dS(t) - \Lambda - (1-p)(1-r)mq\beta S(t-\tau) I_u(t-\tau) e^{-d\tau} \end{bmatrix}.$$

The basic reproduction number belonging to the model (2.3) is based on the linearization of the system about disease-free equilibrium. The jacobian matrices of $\mathscr{F}(X)$ and $\mathscr{V}(X)$ at the disease-free equilibrium $\varepsilon_0 = \left(\frac{\Lambda}{d}, 0\right)$ are respectively found as

$$d\mathscr{F}(\varepsilon_0) = \begin{bmatrix} \mathscr{F}_{11} & \mathscr{F}_{12} \\ \mathscr{F}_{21} & \mathscr{F}_{22} \end{bmatrix},$$

$$d\mathscr{V}(\varepsilon_0) = \begin{bmatrix} \mathscr{V}_{11} & \mathscr{V}_{12} \\ \mathscr{V}_{21} & \mathscr{V}_{22} \end{bmatrix}.$$

Where

$$\begin{aligned} \mathscr{F}_{11} &= [r + (1-r)m(1-q)p]\beta S^0 + (1-y)p(1-r)mq\beta S^0 e^{-d\tau} \\ \mathscr{F}_{12} &= [r + (1-r)m(1-q)p]\beta I_u^0 + (1-y)p(1-r)mq\beta I_u^0 e^{-d\tau} \\ \mathscr{F}_{21} &= 0 \\ \mathscr{F}_{22} &= 0 \end{aligned}$$

and

$$\begin{aligned} \mathscr{V}_{11} &= d + \theta, \\ \mathscr{V}_{12} &= 0, \\ \mathscr{V}_{21} &= [1 + (1-r)m(q + p(1-q))]\beta S^0 - (1-p)(1-r)mq\beta S^0 e^{-d\tau}, \\ \mathscr{V}_{22} &= [1 + (1-r)m(q + p(1-q))]\beta I_u^0 + d - (1-p)(1-r)mq\beta I_u^0 e^{-d\tau}. \end{aligned}$$

Then

$$F=\mathscr{F}_{1\times 1}=\left[(r+(1-r)m(1-q)p)\beta S^0+(1-y)p(1-r)mq\beta S^0e^{-d\tau}\right],$$

$$V=\mathscr{V}_{1\times 1}=[d+\theta]$$

and

$$FV^{-1}=\left[\frac{\left[r+(1-r)m(1-q)p+(1-y)p(1-r)mqe^{-d\tau}\right]\beta S^0}{d+\theta}\right].$$

Following Diekmann and Heesterbeek [36], the matrix FV^{-1} is referred to as the next generation matrix for the system at the disease-free equilibrium and the basic reproduction number is defined as the spectral radius of the matrix FV^{-1}. Now let us find maximum of the eigenvalues of this matrix. The characteristic polynomial of FV^{-1} is

$$\det\left(\lambda I_1-FV^{-1}\right)=\lambda-\frac{\left[r+(1-r)m(1-q)p+(1-y)p(1-r)mqe^{-d\tau}\right]\beta S^0}{d+\theta}.$$

Then, the spectral radius of the next generation matrix is

$$\rho\left(FV^{-1}\right)=\frac{\left[r+(1-r)m(1-q)p+(1-y)p(1-r)mqe^{-d\tau}\right]\beta S^0}{d+\theta}.$$

Taking into $S^0=\Lambda/d$ account that, the basic reproduction number of the model (2.3) is found as

$$\mathscr{R}_0=\frac{\beta\Lambda\left\{r+(1-r)m\left[(1-q)p+(1-y)pqe^{-d\tau}\right]\right\}}{d(d+\theta)}.$$

Now, let us open another matter and consider its results. It is clear that

$$\frac{\partial\mathscr{R}_0}{\partial y}=-p(1-r)mqe^{-d\tau}\leq 0$$

and

$$\frac{\partial\mathscr{R}_0}{\partial q}=m(1-r)p\left[(1-y)e^{-d\tau}-1\right]\leq 0,$$

taking into account that $(1-y)e^{-d\tau}<1$. So we can say that the test rate y and the filiation (chain of contamination) control rate q have opposite effects on $\mathscr{R}_0$.
On the other hand

$$\frac{\partial\mathscr{R}_0}{\partial r}=\frac{\beta\Lambda\left[1-mp\left(1-q+(1-y)qe^{-d\tau}\right)\right]}{d(d+\theta)}$$

and so, if $1>mp\left[1-q\left(1+(1-y)e^{-d\tau}\right)\right]$ then increasing of asymptomatic individuals increases the value $\mathscr{R}_0$. As a result of this fact, it should be aimed that the following inequality is hold

$$mp\left[1-q\left(1+(1-y)e^{-d\tau}\right)\right]\geq 1. \tag{3.2}$$

Hence, it can be concluded what is relation of the test rate y and the filiation control rate q according to other parameters from (3.2). In other words, this relation can give an answer to the question: "What should filiation control rate q is required with the test rate y so that the disease brought under control and does not turn into an epidemic?"

3.2. Existence of endemic equilibrium point

It can be seen that, from in subsection 3.1, the system (2.3) always has a disease-free equilibrium point. Now, we investigate the existence of endemic equilibrium point of the system (2.3).
If we take $S(t)=S^*$ and $I_u(t)=I_u^*\neq 0$, the endemic equilibrium point of the system (2.3) can be calculated via following system of algebraic equations

$$\begin{aligned}0&=\Lambda-[1+(1-r)m(q+p(1-q))]\beta S^*I_u^*-dS^*+(1-p)(1-r)mq\beta S^*I_u^*e^{-d\tau},\\0&=[r+(1-r)m(1-q)p]\beta S^*I_u^*+(1-y)p(1-r)mq\beta S^*I_u^*e^{-d\tau}-(d+\theta)I_u^*.\end{aligned} \tag{3.3}$$

From the second equation of the system (3.3), we write

$$I_u^*\left\{[r+(1-r)m(1-q)p]\beta S^*+(1-y)p(1-r)mq\beta S^*e^{-d\tau}-(d+\theta)\right\}=0.$$

Since $I_u^*\neq 0$ for endemic equilibrium point, we can say

$$[r+(1-r)m(1-q)p]\beta S^*+(1-y)p(1-r)mq\beta S^*e^{-d\tau}-(d+\theta)=0$$

and obtain

$$S^*=\frac{(d+\theta)}{\beta\{r+(1-r)m[(1-q)p+(1-y)pqe^{-d\tau}]\}}=\frac{\Lambda}{d\mathscr{R}_0}. \tag{3.4}$$

Substituting expression in (3.4) into first equation of (3.3) and arranging, we get

$$\begin{aligned} I_u^* &= \frac{\Lambda-dS^*}{\beta S^*[1+(1-r)m(q+p(1-q))-(1-p)(1-r)mqe^{-d\tau}]} \\ &= \frac{d(\mathscr{R}_0-1)}{\beta\{1+(1-r)m[q(1-e^{-d\tau})+p(1-q)+pqe^{-d\tau}]\}}. \end{aligned}$$

Then taking into account that $q,r<1$ and $e^{-d\tau}<1$, it can be obtain $I_u^*>0$ for $\mathscr{R}_0>1$.
Therefore, we say the system (2.3) has a unique endemic equilibrium point $\varepsilon_*=(S^*,I_u^*)$ when $\mathscr{R}_0>1$.
$\varepsilon_*=(S^*,I_u^*)$ can be written as

$$(S^*,I_u^*)=\left(\frac{\Lambda}{d\mathscr{R}_0},\frac{d(\mathscr{R}_0-1)}{\beta\{1+(1-r)m[q(1-e^{-d\tau})+p(1-q)+pqe^{-d\tau}]\}}\right)$$

according to $\mathscr{R}_0$.

3.3. Stabilities of the equilibrium points

In this part it is examined the stability behaviour of system (2.3). Firstly, for local stabilities of disease-free and endemic equilibrium points, the characteristic equations which correspond to Jacobian matrices at the equilibrium points are analysed. Next by using the Lyapunov functional technique, global stability of disease-free equilibrium point is proved.

Theorem 3.2. *The disease-free equilibrium ε_0 of the system (2.3) is locally asymptotically stable in the positively invariant region Γ for $\mathscr{R}_0<1$, and unstable for $\mathscr{R}_0>1$.*

Proof. The Jacobian matrix at the disease-free equilibrium point $\varepsilon_0=\left(S^0,I_u^0\right)$ of the system (2.3) is

$$J(\varepsilon_0)=\begin{bmatrix} J_{11} & J_{12} \\ J_{21} & J_{22} \end{bmatrix},$$

where

$$\begin{aligned} J_{11} &= -[1+(1-r)m(q+p(1-q))]\beta I_u^0-d+(1-p)(1-r)mq\beta I_u^0e^{-d\tau}, \\ J_{12} &= -[1+(1-r)m(q+p(1-q))]\beta S^0+(1-p)(1-r)mq\beta S^0e^{-d\tau}, \\ J_{21} &= [r+(1-r)m(1-q)p]\beta I_u^0+(1-y)p(1-r)mq\beta I_u^0e^{-d\tau}, \\ J_{22} &= [r+(1-r)m(1-q)p]\beta S^0+(1-y)p(1-r)mq\beta S^0e^{-d\tau}-(d+\theta). \end{aligned}$$

Taking into account $\left(S^0,I_u^0\right)=\left(\frac{\Lambda}{d},0\right)$, the characteristic equation which is correspond to this matrix is

$$(-d-\lambda)\left(\beta\frac{\Lambda}{d}\left[(r+(1-r)m(1-q)p)+(1-y)p(1-r)mqe^{-d\tau}\right]-(d+\theta)-\lambda\right)=0. \tag{3.5}$$

This equation always have negative eigenvalue $-d$. The other eigenvalue of characteristic equation (3.5) is determined by

$$\begin{aligned} \lambda_2 &= (d+\theta)\left(\frac{\beta\Lambda\left\{r+(1-r)m\left[(1-q)p+(1-y)pqe^{-d\tau}\right]\right\}}{d(d+\theta)}-1\right) \\ &= (d+\theta)(\mathscr{R}_0-1). \end{aligned}$$

If $\mathscr{R}_0<1$, then two roots of Eq. (3.5) are negative. If $\mathscr{R}_0=1$, then we say that one of roots of Eq. (3.5) is zero. In the case $\mathscr{R}_0>1$, one of roots of Eq. (3.5) has positive real parts. Therefore, the disease-free equilibrium point ε_0 is locally asymptotically stable for $\mathscr{R}_0<1$, is stable for $\mathscr{R}_0=1$, and is unstable for $\mathscr{R}_0>1$. □

Theorem 3.3. *The endemic equilibrium ε_* is locally asymptotically stable in the positively invariant region Γ for $\mathscr{R}_0 > 1$.*

Proof. The Jacobian matrix at the endemic equilibrium point ε_* of the system (2.3) is

$$J(\varepsilon_*) = \begin{bmatrix} J_{11} & J_{12} \\ J_{21} & J_{22} \end{bmatrix},$$

where

$$\begin{aligned}
J_{11} &= -[1+(1-r)m(q+p(1-q))]\beta I_u^* - d + (1-p)(1-r)mq\beta I_u^* e^{-d\tau}, \\
J_{12} &= -[1+(1-r)m(q+p(1-q))]\beta S^* + (1-p)(1-r)mq\beta S^* e^{-d\tau}, \\
J_{21} &= [r+(1-r)m(1-q)p]\beta I_u^* + (1-y)p(1-r)mq\beta I_u^* e^{-d\tau}, \\
J_{22} &= [r+(1-r)m(1-q)p]\beta S^* + (1-y)p(1-r)mq\beta S^* e^{-d\tau} - (d+\theta).
\end{aligned}$$

If we take into account that

$$(S^*, I_u^*) = \left(\frac{\Lambda}{d\mathscr{R}_0}, \frac{d(\mathscr{R}_0 - 1)}{\beta\{1+(1-r)m[q(1-e^{-d\tau}) + p(1-q) + pqe^{-d\tau}]\}} \right),$$

and make necessary arrangements, we obtain the followings:

$$\begin{aligned}
J_{11} &= -[1+(1-r)m(q+p(1-q))]\beta I_u^* - d + (1-p)(1-r)mq\beta I_u^* e^{-d\tau} \\
&= -\beta I_u^* \left\{ 1 + (1-r)m\left[q\left(1-e^{-d\tau}\right) + p(1-q) + pqe^{-d\tau} \right] \right\} - d \\
&= d(1-\mathscr{R}_0) - d \\
&= -d\mathscr{R}_0,
\end{aligned}$$

$$\begin{aligned}
J_{12} &= -[1+(1-r)m(q+p(1-q))]\beta S^* + (1-p)(1-r)mq\beta S^* e^{-d\tau} \\
&= -\beta S^* \left\{ 1 + (1-r)m\left[q\left(1-e^{-d\tau}\right) + p(1-q) + pqe^{-d\tau} \right] \right\} \\
&= -\frac{\Lambda(\mathscr{R}_0 - 1)}{I_u^* \mathscr{R}_0},
\end{aligned}$$

$$\begin{aligned}
J_{21} &= [r+(1-r)m(1-q)p]\beta I_u^* + (1-y)p(1-r)mq\beta I_u^* e^{-d\tau} \\
&= \beta I_u^* \left\{ r + (1-r)m\left[(1-q)p + (1-y)pqe^{-d\tau} \right] \right\} \\
&= I_u^* \frac{d(d+\theta)\mathscr{R}_0}{\Lambda}
\end{aligned}$$

and

$$\begin{aligned}
J_{22} &= [r+(1-r)m(1-q)p]\beta S^* + (1-y)p(1-r)mq\beta S^* e^{-d\tau} - (d+\theta) \\
&= \beta S^* \left\{ r + (1-r)m\left[(1-q)p + (1-y)pqe^{-d\tau} \right] \right\} - (d+\theta) \\
&= S^* \frac{d(d+\theta)\mathscr{R}_0}{\Lambda} - (d+\theta) \\
&= 0.
\end{aligned}$$

After from the simplification, the corresponding characteristic equation for $J(\varepsilon_*)$ is found as

$$\lambda^2 + C_1\lambda + C_2 = 0, \tag{3.6}$$

where

$$C_1 = d\mathscr{R}_0$$

and

$$C_2 = d(d+\theta)(\mathscr{R}_0 - 1).$$

Then we can say

$$C_1 = d\mathscr{R}_0 > 0, \text{ for } \mathscr{R}_0 > 1$$

and

$$C_2 = d(d+\theta)(\mathscr{R}_0 - 1) > 0, \text{ for } \mathscr{R}_0 > 1.$$

Therefore, we obtain $tr(J(\varepsilon_*)) = -C_1 < 0$ and $\det(J(\varepsilon_*)) = C_2 > 0$. So, each of the eigenvalues of $J(\varepsilon_*)$ (i.e. two roots of the equation (3.6) have negative real parts. Consequently, if $\mathscr{R}_0 > 1$ then endemic equilibrium $\varepsilon_* = (S^*, I_u^*)$ is locally asymptotically stable. □

Theorem 3.4. *The disease-free equilibrium ε_0 of the system (2.3) is globally asymptotically stable in the positively invariant region Γ for $\mathscr{R}_0 < 1$.*

Proof. Let us define the following function as a candidate for Lyapunov function.

$$W(t) = I_u(t) + (1-y)p(1-r)mqe^{-d\tau}\int_{t-\tau}^{t}\beta S(x)I_u(x)\,dx.$$

Differentiating $W(t)$ according to time t, we get

$$\begin{aligned}
&\dot{W}(t)\\
=\;& [r+(1-r)m(1-q)p]\beta S(t)I_u(t) + (1-y)p(1-r)mq\beta S(t-\tau)I_u(t-\tau)e^{-d\tau} - (d+\theta)I_u(t)\\
&+(1-y)p(1-r)mqe^{-d\tau}\beta S(t)I_u(t) - (1-y)p(1-r)mqe^{-d\tau}\beta S(t-\tau)I_u(t-\tau)\\
=\;& I_u(t)\left\{[r+(1-r)m(1-q)p]\beta S(t) + (1-y)p(1-r)mqe^{-d\tau}\beta S(t) - (d+\theta)\right\}\\
=\;& I_u(t)\left\{\beta S(t)\left[r+(1-r)m\left((1-q)p+(1-y)pqe^{-d\tau}\right)\right] - (d+\theta)\right\}\\
\leq\;& I_u(t)\left[\frac{\beta\Lambda\left\{r+(1-r)m\left[(1-q)p+(1-y)pqe^{-d\tau}\right]\right\}}{d} - (d+\theta)\right]\\
=\;& I_u(t)\left[(d+\theta)\left(\frac{\beta\Lambda\left\{r+(1-r)m\left[(1-q)p+(1-y)pqe^{-d\tau}\right]\right\}}{d(d+\theta)} - 1\right)\right]\\
=\;& I_u(t)(d+\theta)(\mathscr{R}_0 - 1).
\end{aligned}$$

Hence, it can be concluded that $W(t) > 0$ and $\dot{W}(t) < 0$ when $\mathscr{R}_0 < 1$ and for all points which is different from equilibrium points. So W is a Lyapunov function for the system (2.3) on the set Γ. Now, let us define the set $\Phi = \left\{(S,I) : \dot{W}(t) = 0\right\}$ and let ϕ be largest invariant subset of Φ. It can be easily seen that $\phi = \{\varepsilon_0\}$ and ϕ is invariant. We say that ε_0 is globally asymptotically stable in Γ by aid of LaSalle's Invariance Principle [37] well-known from global stability thecniques. □

4. Conclusion

This study describes and analyses an SI_uI_aQR mathematical model that investigates the effect of quarantine on spreading of the Covid-19. To avoid second major or interim sub-waves of Covid-19 pandemic, one of the most effective methods that will minimize the harm and spread of the outbreak is to quarantine the exposed people and monitor the individuals they are in contact with. With this study, which aims to evaluate the effect of quarantine on the transmission of the Covid-19, it is thought that a different perspective and contribution will be provided to the literature.
Let us start to present the examples with the course of Q, I_u and I_a such that the estimated parameters are as follows

Parameters	Value (Estimated)
Λ	4000 (per day)
β	1.1×10^{-9}
r	0.5
m	5 (individual)
q	0.3
p	0.2
d	0.000015
y	0.7
μ	0.02
τ	12 (day)
θ	0.04
γ	0.2

with the initial conditions $S(0) = 7 \times 10^7$, $I_u(0) = 5000$, $I_a(0) = 500$, $Q(0) = 0$ and $R(0) = 0$.

According to the above parameters, the dynamical behaviours of the model has been described in Figure 4.1.

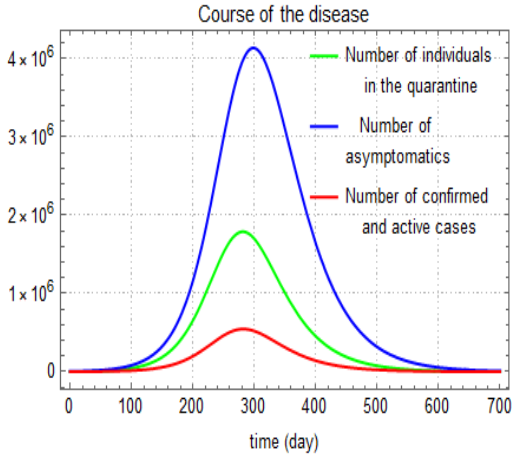

Figure 4.1: The dynamical behaviours of the compartments I_u, I_a and Q.

The other figures respectively reflect that the effect of filiation control rate q, of the rate of tests y for finding the infected individuals, and of the average number contacted persons m with the new cases, to spread of Covid-19. The figures, prepared to visualize the effect of these parameters in (2.3) has been generated using the Wolfram Mathematica 12.1 with NDSolve code. The control of filiation is a method of tracking from whom the virus is transmitted to the positive case and who the case has infected. In other words, in every positive case, it is the method of tracking the infection of the virus and determining the chain of spread. In the monitoring method of chain of contamination, the followings of contacted cases are provided. The individuals those who come in contact with positive cases are reached in a short time and asked to isolate themselves, and their evaluations and tests are made by visiting their locations. So, it is provided to prevent their infection potentials.
With the help of filiation studies, the cases are detected early and the risk of transmitting the disease to others is minimized. Again, the early detection of the patients by means of the filiation provides an early start of the treatment process and increases the recovery rate, [38]. Therefore the case detection and control works, and filiation activities belong to these play a very important role in struggle with Covid-19. Hence, all countries that are trying to control the disease should particular importance to the filiation activities for the strict monitoring and isolation of people having contact with cases within the community in addition to put communal limitations to keep the disease limited. With the filiation works, it is clear that great advances will be recorded. In our model, q corresponds to filiation control rate and the reader can see the effect of q (in a short period) on the spread of the Covid-19, in Figure 4.2.

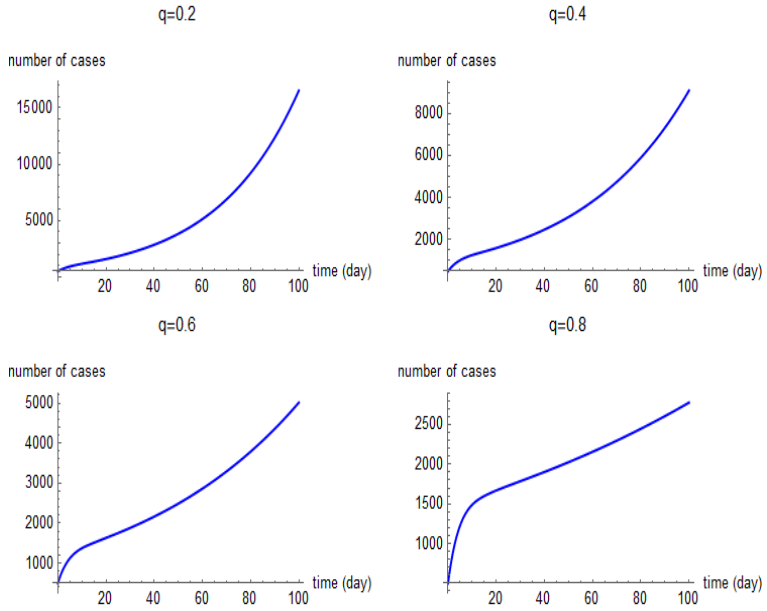

Figure 4.2: The effect of filiation control rate q in a short period for $y = 1$.

It can be seen that the increasing tendency of the disease decreases as q increases. Also the following figure shows the course of number of infectious (confirmed cases) according to q.

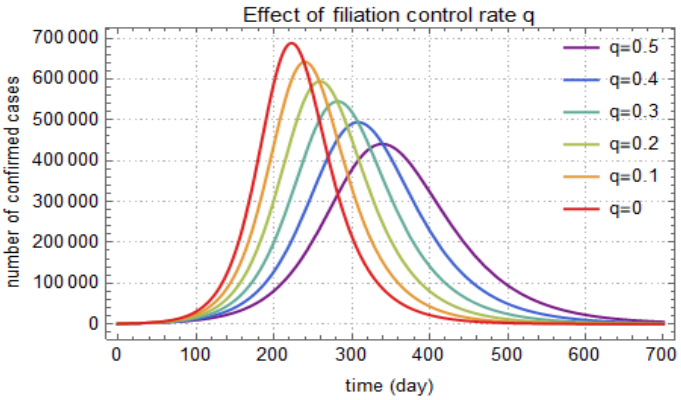

Figure 4.3: The effect of filiation control rate q in a long period.

Another big problem in the spread of disease is the number of contacts within the population. Unfortunately, no one can be sure that the people with whom they are in contact are not positive. So it should be avoided from dispensable contact. The following figure shows the result of this fact.

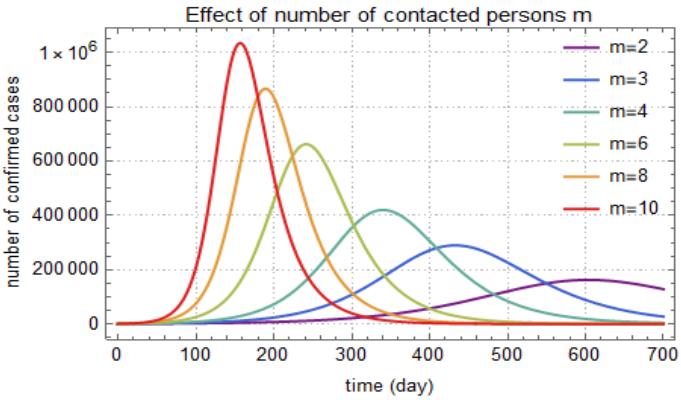

Figure 4.4: The effect of the average number of contacted individuals for $m = 2, m = 3, m = 4, m = 6, m = 8, m = 10$.

Let us come to the rate of test y. The rate of tests for determining the infected individuals is crucial in reducing the size of I_u. Let us note immediately that, as known, the potential danger group here is the asymptomatic individuals in the circulation in the community. Since it is assumed that the members of I_a will be isolated during the treatment in a hospital or home, they don't transmit the disease to susceptible. The rate of test at the end of the quarantine process and the rate of positive test within total tests determine the percentage of individuals that moved to the classes I_a, I_u and S from Q. In this regard, the rate of tests to be performed is reasonably significant. The following figure shows how the compartments can be influenced from evolution in y, for $q = 0.5$ and different values of the parameter y.

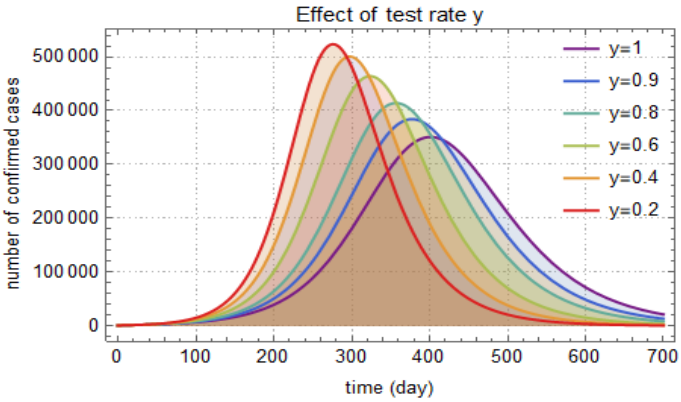

Figure 4.5: The effect of test rates for $y = 0.2, y = 0.4, y = 0.4, y = 0.6, y = 0.8, y = 0.9, y = 1$.

Finally we want to present, according to q, the bound of ratio of asymptomatic to confirmed cases.

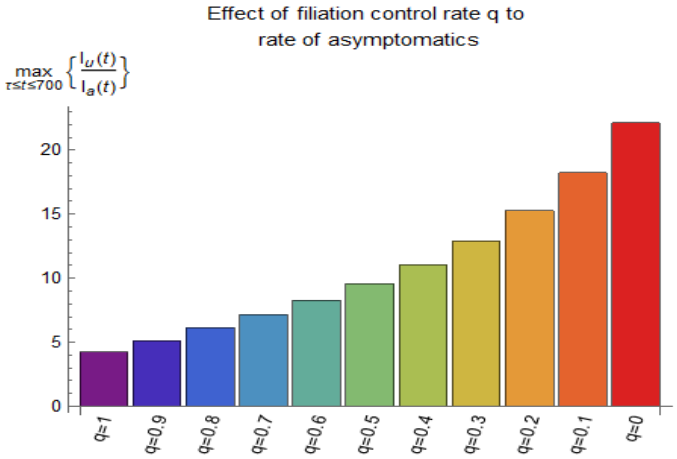

Figure 4.6: Different scenario of maximum rate between I_u and I_a after one incubation period from the initial of the disease.

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Author's contributions

All authors contributed equally to the writing of this paper. All authors read and approved the final manuscript.

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