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On the stability analysis of solutions of an integral equation with an application in epidemiology

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Abstract

This paper concerns a nonlinear integral equation modelling the spread of epidemics in which immunity does not occur after recovery. The model is mainly based on the return of some of the individuals who have been exposed to the pathogen and who have completed the incubation period, into the susceptible class. We firstly prove the uniqueness of the global solution of the model with the given initial conditions. After determining the positively invariant region for the model, using LaSalle invariance principle [J. P. LaSalle, IRE Trans. CT, **7** (1960), 520–527] and the concept of persistence we present some results about the stability analysis of the solutions according to the case of the reproduction number $\mathcal{R}_0$ which is a vital threshold in spread of diseases.

Keywords: Global stability analysis, Lyapunov function, LaSalle invariance principle, mathematical epidemiology, persistence.

2010 MSC: 34D05, 34D08, 34D20, 92B05, 92D25, 92D30.

1. Introduction

As is known, mathematical modelling frequently used to describe many nonlinear problems arising from the most areas of natural sciences such as physics, engineering, economics, biology, epidemiology and so on [2, 9, 13, 16–18]. In particular, nonlinear integral and differential equations are often used in characterization of some problems of real world such as theory of radioactive transfer [16], the modelling of the behaviour of viscoelastic materials in mechanics [29], the modelling of soft tissues like mitral valves or the aorta in the human heart [12], the modelling of tumor growth [8] and so on.

Especially, modelling of epidemic diseases as mathematically is quite important in terms of understanding, controlling and reducing the effects of the outbreaks. There are many mathematical models that determine the basic principles for the spread of a disease in a population using integral and differential equations. For example, Kermack and McKendrick with their study [17] have pioneered these mathematical models which are used extensively. By using the following system of differential equations, they have tried to explain the spread of infectious disease in the course of time for a closed population

$$\frac{dS}{dt} = -\beta S(t) I(t),$$

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$$\frac{dI}{dt} = \beta S(t) I(t) - \gamma I(t),$$
$$\frac{dR}{dt} = \gamma I(t).$$

In the model, known as SIR model, the population has been divided into non-intersecting three groups; Susceptibles (S), Infectious (I) and Recovered (R). β is the effective contact rate between S and I. γ represents the recovery rate of infectious. Then many authors studied on this model, [1, 4, 5, 22–25].

Also, for many important diseases, there is a significant incubation period during which individuals have been infected but are not yet infectious themselves. In general, the individuals in the incubation period are evaluated in a separate compartment E (Exposed). In the literature, there are many results about such epidemic models [11, 19, 26, 27].

In [26] the authors analyzed a SEIR epidemic model in which prophylactic for the exposed individuals is included. They considered that there were transitions from class E to I and R without any delay terms.

Korobeinikov, in [19], presented global properties of a SEIR model with horizontal and vertical transmissions.

Also, as it is known, in some epidemic diseases, any long-lasting immunity may not be formed against the pathogen after recovery and such diseases could be modeled via SIRS or SEIRS form, [3]. Moreover in some diseases especially transmitted by bacteria such as tuberculosis, meningitis and gonorrhea even short-term immunity do not occur. That is, the infectious return to the susceptible class on recovery because the disease confers no immunity against reinfection, [6]. Since no need for recovery class, such diseases could be modeled via SIS or SEIS form. In the literature, there are many mathematical models given via these forms, [7, 19, 30]. In [19] the author considers a SEIS model without any delay terms. According to this model an infective either returns to the susceptibles compartment immediately after recovery, or dies.

On the other hand, in addition to differential equations, integral equations are also used in epidemiology. In 1980, Hethcote and Tudor [15] used a system of nonlinear Volterra integral equations of convolution type to describe endemic infectious diseases for which infection confers permanent immunity. Then the system used in this paper was reduced to the system of ordinary differential equations known as the SIR model.

Also G. Gripenberg in the paper [14] studied the following integral equation which arises in the study of the spread of an infectious disease that does not induce permanent immunity

$$x(t) = k\left[q(t) - \int_0^t A(t-s)\,x(s)\,ds\right]\left[h(t) - \int_0^t B(t-s)\,x(s)\,ds\right],\ t \in [0,\infty).$$

Where, it is assumed that, in a population with constant size K, the average of infectivity of an individual infected at time s is proportional to $B(t-s)$ at time t. If the rate at which individuals susceptible to the disease have become infected up to time t is $x(s)$, $s < t$, then the integral $\int_{-\infty}^{t} B(t-s)\,x(s)\,ds$ will be approximately proportional to the total infectivity. If the cumulative probability function for the loss of immunity of an individual infected at time s is $1 - A(t-s)$, $t \geqslant s$, then $K - \int_{-\infty}^{t} A(t-s)\,x(s)\,ds$ will approximate the number of susceptibles.

In this paper we deal with the following equation modeling the spread of epidemics in which immunity does not occur after recovery.

$$\begin{aligned} x(t) &= g(0) + \int_0^t u(s, x^s)\,ds,\ t \geqslant 0, \\ x(t) &= g(t),\ -\tau \leqslant t \leqslant 0. \end{aligned} \tag{1.1}$$

Where $g, x^s \in C\left([-\tau,0],[0,1]^2\right)$, $x^s(\theta) = x(s+\theta)$ and $x \in C\left([-\tau,\infty),[0,1]^2\right)$. Also $u : [0,\infty) \times \Omega \to [0,1]^2$ such that $\Omega \subset C\left([-\tau,0],[0,1]^2\right)$.

We should immediately note that the equation (1.1) will turn to a SEIS model after appropriate choosing of mentioned functions. Unlike the assumption that all exposed individuals transfer to the infectious class, which is valid in classical SEIS models, in the model presented in this study, after the incubation period, exposed individuals either transfer to the infectious class or return to the susceptible class.

As known $C([-\tau,0],\mathbb{R}^n)$ is a Banach space of continuous functions, and $\|\cdot\|_C$ denotes the norm on $C([-\tau,0],\mathbb{R}^n)$ and is defined by

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

On the other hand we can say that the equation (1.1) has a unique solution if u is Lipschitz continuous according to the second variable in every compact subset $M \subset \Omega$. Indeed this result depends on Schauder fixed point theorem, [20].

If we choose $u(s,x^s) = f(x^s)$, $g = (g_1,g_2)$ then finding the solution of the above system is equivalent to solving the following equation

$$\begin{aligned} x'(t) &= f\left(x^t\right),\ t \geqslant 0 \\ x_0 &= g, \end{aligned} \tag{1.2}$$

where $f : \Omega \to [0,1]^2$.

We construct a mathematical model, (which consists of system of delay ordinary differential equations), for represent spreading of a disease in a closed population. We define this model by choosing $x = (x_1,x_2)$, $x_1 = S$, $x_2 = I$ and $f = (f_1,f_2)$ in (1.2) such that

$$\begin{aligned} f_1(x) &= \mu(1-x_1(0)) - \beta x_1(0)x_2(0) + (1-p)\beta x_1(-\tau)x_2(-\tau)e^{-\mu\tau} + rx_2(0), \\ f_2(x) &= p\beta x_1(-\tau)x_2(-\tau)e^{-\mu\tau} - (\mu+r)x_2(0), \end{aligned}$$

with initial function $x_1(t) = g_1(t)$, $x_2(t) = g_2(t)$ for $-\tau \leqslant t \leqslant 0$.

Theorem 1.1. *There exists a unique solution of the equation* (1.1) *such that* $f = (f_1,f_2)$ *with initial function* $x_1(t) = g_1(t)$, $x_2(t) = g_2(t)$ *for* $-\tau \leqslant t \leqslant 0$.

Proof. The proof depends on the result in [3, 20]. So it is sufficient to show that f 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

$$\begin{aligned} &\|f(x) - f(y)\| \\ &= |f_1(x) - f_1(y)| + |f_2(x) - f_2(y)| \\ &= |\mu[y_1(0) - x_1(0)] + \beta[y_1(0)y_2(0) - x_1(0)x_2(0)] \\ &+ (1-p)\beta e^{-\mu\tau}[x_1(-\tau)x_2(-\tau) - y_1(-\tau)y_2(-\tau)] + r[x_2(0) - y_2(0)]| \\ &+ |p\beta e^{-\mu\tau}[x_1(-\tau)x_2(-\tau) - y_1(-\tau)y_2(-\tau)] + (\mu+r)[y_2(0) - x_2(0)]| \\ &\leqslant \mu|y_1(0) - x_1(0)| + \beta|y_1(0)y_2(0) - x_1(0)x_2(0)| \\ &+ \beta e^{-\mu\tau}|x_1(-\tau)x_2(-\tau) - y_1(-\tau)y_2(-\tau)| + r|x_2(0) - y_2(0)| \\ &+ (\mu+r)|y_2(0) - x_2(0)| \\ &\leqslant 2(\mu+r)\|x-y\|_C + \beta|y_1(0)y_2(0) - y_1(0)x_2(0)| \\ &+ \beta|y_1(0)x_2(0) - x_1(0)x_2(0)| + \beta e^{-\mu\tau}|x_1(-\tau)x_2(-\tau) - y_1(-\tau)x_2(-\tau)| \\ &+ \beta e^{-\mu\tau}|y_1(-\tau)x_2(-\tau) - y_1(-\tau)y_2(-\tau)| \\ &\leqslant 2(\mu+r)\|x-y\|_C + \beta(|y_1(0)| + |x_2(0)|)\|x-y\|_C \\ &+ \beta e^{-\mu\tau}(|x_2(-\tau)| + |y_1(-\tau)|)\|x-y\|_C. \end{aligned} \tag{1.3}$$

Taking into account the facts $e^{-\mu\tau} \leqslant 1$ and $|x_i(\theta)| \leqslant 1$ for $-\tau \leqslant \theta \leqslant 0$, $i = 1, 2$ then we conlude

$$\|f(x) - f(y)\| \leqslant 2(\mu + r + 2\beta)\|x - y\|_C,$$

from (1.3). So if we take $k \geqslant 2(\mu + r + 2\beta)$, the inequality

$$\|f(x) - f(y)\| \leqslant k\|x - y\|_C,$$

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

2. Description of the model

In this paper we form a compartmental model by dividing into three groups (S, E, I) of the population exposed to an infectious disease. In the model, we firstly assume that all new members of the population get involved in the susceptible class S at a constant rate μ. Also the constant μ represents the natural death rate of all compartments and we neglect disease induced deaths. So the total population $N(t)$ is constant and equal to 1 for all $t \geqslant 0$. Incubation period is the time elapsed between exposure to the pathogen and when symptoms and signs are first apparent. Depending on the disease, the individual may or may not be infectious during the incubation period. We assume that τ denotes the incubation period for the model. Also, after the incubation period some of exposed (at rate p) become infectious and the others (at rate $1 - p$) turn to class S. On the other hand, β is the transmission rate from susceptibles to E. Finally r denotes rate of recovery in which immunity does not occur.

Let we write $\widetilde{E}(t, \theta)$ to represent the number of individuals exposed and entered in the incubation period with exposure age θ (i.e, time elapsed since exposure to the pathogen) at time t.

Taking into account that the natural death rate μ we obtain the description

$$\widetilde{E}(t, \tau) = \beta S(t-\tau) I(t-\tau) e^{-\mu\tau},$$

from the following initial value problem

$$\left(\frac{\partial}{\partial t} + \frac{\partial}{\partial \tau}\right)\widetilde{E}(t, \tau) = -\mu\widetilde{E}(t, \tau), \quad \widetilde{E}(t, 0) = \beta S(t) I(t).$$

So, depending all the above descriptions and equation (1.2), we present a SEIS model given by the following system

$$\begin{aligned} S'(t) &= \mu(1 - S(t)) - \beta S(t) I(t) + (1-p)\beta S(t-\tau) I(t-\tau) e^{-\mu\tau} + rI(t), \\ E'(t) &= \beta S(t) I(t) - \beta S(t-\tau) I(t-\tau) e^{-\mu\tau} - \mu E(t), \\ I'(t) &= p\beta S(t-\tau) I(t-\tau) e^{-\mu\tau} - (\mu + r) I(t). \end{aligned} \tag{2.1}$$

Where $S(t)$, $E(t)$ and $I(t)$ denote the size of the subclasses: susceptibles, exposed to the pathogen and infectious individuals at time t, respectively. Only susceptible individuals that have contact with infectious at time $t - \tau$, become infectious at time t provided that they have survived the incubation period of τ. Thus taking into account that the number of new individuals exposed to the pathogen at time $t - \tau$ is $\beta S(t-\tau) I(t-\tau)$, and only p-ratio of exposed get infectious then the incidence of newly infectious is given by the mass action term $p\beta S(t-\tau) I(t-\tau) e^{-\mu\tau}$.

Also we write $N(t)$ to show the total number of the population at time t such that $S(t) + E(t) + I(t) = N(t) \equiv 1$ for all $t \geqslant 0$. Naturally, the functions S, E, I, N are nonnegative and all parameters are positive. We should immediately that since $E(t) = 1 - S(t) - I(t)$, function E does not affect the solution of (2.1) and so it can be omitted. Hence we mean the following system by the model

$$S'(t) = \mu(1 - S(t)) - \beta S(t) I(t) + (1-p)\beta S(t-\tau) I(t-\tau) e^{-\mu\tau} + rI(t),$$

$$I'(t) = p\beta S(t-\tau) I(t-\tau) e^{-\mu\tau} - (\mu + r) I(t), \tag{2.2}$$

with initial function $S(t) = g_1(t)$, $I(t) = g_2(t)$ for $-\tau \leqslant t \leqslant 0$. It can be easily seen that Theorem 1.1 guarantees the uniqueness of the global solution of the model. Now we present the positively invariant set for the model by using a similar method in [3].

Theorem 2.1. *The set*

$$\Psi = \left\{ (S, I) \in C\left([-\tau, 0], [0,1]^2\right) : \beta \int_{-\tau}^{0} S(\theta) I(\theta) e^{\mu\theta} d\theta \leqslant 1 - S(0) - I(0) \right\},$$

is positively invariant for the model (2.2).

Proof. Let

$$\beta \int_{-\tau}^{0} S(\theta) I(\theta) e^{\mu\theta} d\theta \leqslant 1 - S(0) - I(0).$$

If we define

$$F(t) = 1 - S(t) - I(t) - \beta \int_{t-\tau}^{t} S(\theta) I(\theta) e^{\mu(\theta - t)} d\theta,$$

then $F(0) \geqslant 0$. On the other hand

$$\begin{aligned}
F'(t) &= -S'(t) - I'(t) - \beta \left[S(t) I(t) - S(t-\tau) I(t-\tau) e^{-\mu\tau} \right] \\
&\quad - \mu\beta \int_{t-\tau}^{t} S(\theta) I(\theta) e^{\mu(\theta - t)} d\theta \\
&= -\mu + \mu S(t) + \beta S(t) I(t) - (1-p) \beta S(t-\tau) I(t-\tau) e^{-\mu\tau} \\
&\quad - rI(t) - p\beta S(t-\tau) I(t-\tau) e^{-\mu\tau} + (\mu + r) I(t) \\
&\quad - \beta S(t) I(t) + \beta S(t-\tau) I(t-\tau) e^{-\mu\tau} - \mu\beta \int_{t-\tau}^{t} S(\theta) I(\theta) e^{\mu(\theta - t)} d\theta \\
&= -\mu + \mu S(t) + \mu I(t) - \mu\beta \int_{t-\tau}^{t} S(\theta) I(\theta) e^{\mu(\theta - t)} d\theta \\
&= -\mu \left(1 - S(t) - I(t) - \beta \int_{t-\tau}^{t} S(\theta) I(\theta) e^{\mu(\theta - t)} d\theta \right) \\
&= -\mu F(t).
\end{aligned}$$

This result shows that $F(0) \geqslant 0$ implies $F(t) \geqslant 0$ for all $t \geqslant 0$. Also it means that if $S(0) + I(0) \leqslant 1$ then $S(t) + I(t) \leqslant 1$ for all $t \geqslant 0$. These results complete the proof. □

Corollary 2.2. *Individuals who complete the incubation period at time* t *are those exposed to the pathogen at time* $t - \tau$. *Therefore, for* $t = 0$, *the total number of exposed and survived individuals in* $[-\tau, 0]$ *(represented by* $\beta \int_{-\tau}^{0} S(\theta) I(\theta) e^{\mu\theta} d\theta$*) should not exceed the size of class* E *at time* $t = 0$ *(*$E(0) = 1 - S(0) - I(0)$*). This is needed, so that the model can be epidemiologically meaningful, and this condition is satisfied in* Ψ.

Now, let us find the disease-free equilibrium point of the system (2.2). Let $(S(t), I(t)) = (\bar{S}, \bar{I})$ be an equilibrium of the model. Then, solving the system of algebraic equations

$$\begin{aligned}
0 &= \mu(1 - \bar{S}) - \beta \bar{S} \bar{I} + (1-p) \beta \bar{S} \bar{I} e^{-\mu\tau} + r\bar{I}, \\
0 &= p\beta \bar{S} \bar{I} e^{-\mu\tau} - (\mu + r) \bar{I},
\end{aligned} \tag{2.3}$$

we find the disease-free equilibrium point of the model as $P_0 = (S_0, I_0) = (1, 0)$.

3. Stability analysis of the model

By using the next generation matrix method [10] we can calculate basic reproduction number $\mathcal{R}_0$. Let $X = (I, S)^T$, so model (2.2) can be written as

$$\frac{dX}{dt} = \mathcal{P}(X) - \mathcal{F}(X),$$

such that

$$\mathcal{P}(X) = \begin{bmatrix} p\beta S(t-\tau) I(t-\tau) e^{-\mu\tau} \\ 0 \end{bmatrix},$$

and

$$\mathcal{F}(X) = \begin{bmatrix} (\mu + r) I(t) \\ \beta S(t) I(t) - \mu(1 - S(t)) - (1-p)\beta S(t-\tau) I(t-\tau) e^{-\mu\tau} - rI(t) \end{bmatrix}.$$

Differentiating $\mathcal{P}(X)$ and $\mathcal{F}(X)$ with respect to I, S and computing respectively them at the disease-free equilibrium P_0, we get

$$P = d\mathcal{P}_{1x1}(P_0) = \left[p\beta e^{-\mu\tau}\right],$$
$$F = d\mathcal{F}_{1x1}(P_0) = [\mu + r],$$

and

$$PF^{-1} = \left[\frac{p\beta e^{-\mu\tau}}{\mu + r}\right].$$

Therefore, the basic reproduction number of the model (2.2) can be found as

$$\mathcal{R}_0 = \rho\left(PF^{-1}\right) = \frac{p\beta e^{-\mu\tau}}{\mu + r}.$$

On the other hand, to determine whether another equilibrium point $(\bar{S}, \bar{I})$ of the system (2.2) exists or not, we must solve the following system of algebraic equations with $\bar{I} \neq 0$.

$$\begin{aligned} 0 &= \mu\left(1 - \bar{S}\right) - \beta\bar{S}\bar{I} + (1-p)\beta\bar{S}\bar{I}e^{-\mu\tau} + r\bar{I}, \\ 0 &= p\beta\bar{S}\bar{I}e^{-\mu\tau} - (\mu + r)\bar{I}. \end{aligned} \tag{3.1}$$

From the system (3.1), we can obtain the endemic equilibrium point $P_* = (S_*, I_*)$ of the model such that

$$S_* = \frac{1}{\mathcal{R}_0}, \; I_* = \frac{\mu(\mathcal{R}_0 - 1)}{\beta\left(1 - (1-p)e^{-\mu\tau}\right)}. \tag{3.2}$$

Corollary 3.1. *Model (2.2) always has a disease-free equilibrium P_0. If $\mathcal{R}_0 > 1$ then there are two equilibrias; disease-free equilibrium P_0 and endemic equilibrium P_*.*

Now we deal with stabilities of the disease-free equilibrium point P_0 and the endemic equilibrium point P_* of the system (2.2).

Theorem 3.2. *The disease-free equilibrium P_0 is locally asymptotically stable in the positively invariant region Ψ if $\mathcal{R}_0 < 1$, and unstable if $\mathcal{R}_0 > 1$.*

Proof. The Jacobian matrix at the disease-free equilibrium P_0 of the system (2.2) is

$$J(P_0) = \begin{bmatrix} -\mu & -\beta + (1-p)\,\beta e^{-\mu\tau} + r \\ 0 & p\beta e^{-\mu\tau} - (\mu + r) \end{bmatrix}.$$

It can be easily seen that $J(P_0)$ always has the eigenvalues $\lambda_1 = -\mu < 0$ and $\lambda_2 = p\beta e^{-\mu\tau} - (\mu + r)$. Also $p\beta e^{-\mu\tau} - (\mu + r) < 0$ for $\mathcal{R}_0 < 1$. So the characteristic equation of $J(P_0)$ has two negative roots. On the other hand, in the case $\mathcal{R}_0 > 1$, one of roots of the characteristic equation has positive real part. Therefore, if $\mathcal{R}_0 < 1$ then the disease-free equilibrium P_0 is locally asymptotically stable; if $\mathcal{R}_0 > 1$, is unstable. □

Theorem 3.3. *The disease-free equilibrium point of system* (2.2) P_0 *is globally asymptotically stable if* $\mathcal{R}_0 < 1$.

Proof. Let us consider the following nonnegative function:

$$L(t) = I(t) + p\beta e^{-\mu\tau} \int_{t-\tau}^{t} S(x)\, I(x)\, dx.$$

It is easy see that $L(t) = 0$ if $I(t) = I_0$, and $S(t) = S_0$. Differentiating with respect to time yields and considering the fact $S(t) \leqslant 1$ then we obtain

$$\begin{aligned} \frac{dL}{dt} &= I'(t) + p\beta e^{-\mu\tau} \left(S(t)\, I(t) - S(t-\tau)\, I(t-\tau)\right) \\ &= p\beta S(t-\tau)\, I(t-\tau)\, e^{-\mu\tau} - (\mu + r)\, I(t) \\ &+ p\beta e^{-\mu\tau} \left[S(t)\, I(t) - S(t-\tau)\, I(t-\tau)\right] \\ &= I(t) \left[p\beta e^{-\mu\tau} S(t) - (\mu + r)\right] \\ &\leqslant I(t) \left[p\beta e^{-\mu\tau} - (\mu + r)\right] \\ &= I(t)(\mu + r)\left(\mathcal{R}_0 - 1\right). \end{aligned}$$

So we obtain

$$\frac{dL}{dt} \leqslant 0,$$

for $\mathcal{R}_0 < 1$. If the fact $\frac{dL}{dt} = 0$ at the point P_0 is used, this shows that the function L is a Lyapunov function in Ψ for system (2.2). According to LaSalle's Invariance Principle the limit set of each solution is contained in the largest invariant subset of

$$\left\{(S, I) : \frac{dL}{dt} = 0\right\}.$$

Also the largest invariant subset consists only singleton P_0 for $\mathcal{R}_0 < 1$. Hence P_0 is globally asymptotically stable. □

Theorem 3.4. *The endemic equilibrium* P_* *is locally asymptotically stable in the positively invariant region* Ψ *if* $\mathcal{R}_0 > 1$.

Proof. The Jacobian matrix at the endemic equilibrium P_* of the system (2.2) is

$$\begin{aligned} J(P_*) &= \begin{bmatrix} -\mu - \beta I_* + (1-p)\,\beta e^{-\mu\tau} I_* & -\beta S_* + (1-p)\,\beta e^{-\mu\tau} S_* + r \\ p\beta e^{-\mu\tau} I_* & p\beta e^{-\mu\tau} S_* - (\mu + r) \end{bmatrix} \\ &= \begin{bmatrix} -\mu - \mu\left(\mathcal{R}_0 - 1\right) & \frac{\beta}{\mathcal{R}_0}\left[(1-p)\, e^{-\mu\tau} - 1\right] + r \\ \frac{\mu(\mathcal{R}_0 - 1) p e^{-\mu\tau}}{1-(1-p)e^{-\mu\tau}} & 0 \end{bmatrix}. \end{aligned}$$

Since $\mathcal{R}_0 > 1$, it can be easily seen that $\mathrm{tr}\left(J(P_*)\right) = -\mu - \mu\left(\mathcal{R}_0 - 1\right) < 0$. Also taking into account that $(1-p)\, e^{-\mu\tau} < 1$ then we obtain

$$\begin{aligned}\det(J(P_*)) &= \frac{\beta\mu(\mathcal{R}_0-1)pe^{-\mu\tau}}{\mathcal{R}_0} + \frac{r\mu(\mathcal{R}_0-1)pe^{-\mu\tau}}{(1-p)e^{-\mu\tau}-1}\\ &= pe^{-\mu\tau}\mu(\mathcal{R}_0-1)\left(\frac{\beta}{\mathcal{R}_0}+\frac{r}{(1-p)e^{-\mu\tau}-1}\right)\\ &= pe^{-\mu\tau}\mu(\mathcal{R}_0-1)\left(\frac{\mu+r}{pe^{-\mu\tau}}+\frac{r}{(1-p)e^{-\mu\tau}-1}\right)\\ &= \mu(\mathcal{R}_0-1)\left(\frac{(\mu+r)(e^{-\mu\tau}-1)-\mu pe^{-\mu\tau}}{(1-p)e^{-\mu\tau}-1}\right) > 0.\end{aligned}$$

So the characteristic equation of $J(P_*)$ has two negative roots. Therefore, if $\mathcal{R}_0 > 1$ then the disease-free equilibrium P_* is locally asymptotically stable. □

On the other hand we focus the persistence of the model (2.2) for $\mathcal{R}_0 > 1$. Before starting to do this, it may be necessary to remember some concepts about the persistence which is a property of dynamical systems that describe the long-term behavior of the solutions.

Definition 3.5. [28] Let X be nonempty set, $\rho : X \to \mathbb{R}_+$ and $\Phi : \mathbb{R}_+ \times X \to X$ be a semiflow. Then

1. Φ is called uniformly weakly $\rho-$persistent, if there exists some $\varepsilon > 0$ such that
$$\limsup_{t\to\infty} \rho(\Phi(t,x)) > \varepsilon,$$
for all $x \in X$ and $\rho(x) > 0$.
2. Φ is called uniformly strongly $\rho-$persistent, if there exists some $\varepsilon > 0$ such that
$$\liminf_{t\to\infty} \rho(\Phi(t,x)) > \varepsilon,$$
for all $x \in X$ and $\rho(x) > 0$.
3. A set $M \subset X$ is called weakly $\rho-$repelling if there is no $x \in X$ such that $\rho(x) > 0$ and $\Phi(t,x) \to M$ as $t \to \infty$.

Theorem 3.6. *Let Φ be the semiflow on Ψ, defined by unique global solutions. Also let the persistence function $\rho : \Psi \to \mathbb{R}_+$ be defined as $\rho(x) = x_2(0)$. Then Φ is uniformly strongly $\rho-$persistent for $\mathcal{R}_0 > 1$.*

Proof. Firstly let us divide the invaryant set Ψ to nonintersecting two parts as

$$\Psi_0 = \{x \in \Psi : \rho(x) = 0\} \text{ and } \Psi_+ = \Psi \setminus \Psi_0.$$

Assume that there exists $x \in \Psi$ such that $\rho(x) > 0$ and $\lim_{t\to\infty} \Phi(t,x) = P_0$, that is $x_2(0) = I(0) > 0$ and $I(t) \to 0$ as $t \to \infty$. This means that for any $\varepsilon > 0$ there exists a $T_1 > 0$ such that $\beta I(t) < \varepsilon$ for all $t > T_1$. Also, in view of $\mathcal{R}_0 > 1$, there exists a $T_2 > 0$ such that

$$\sup_{t>T_2} \frac{I(t+\tau)}{I(t)} < \mathcal{R}_0.$$

On the other hand, since S and I are nonnegative and smaller than 1 we can write

$$\begin{aligned}S'(t) &= \mu(1-S(t)) - \beta S(t) I(t) + (1-p)\beta S(t-\tau) I(t-\tau) e^{-\mu\tau} + rI(t)\\ &\geqslant \mu(1-S(t)) - \beta S(t) I(t)\\ &\geqslant \mu(1-S(t)) - \beta I(t),\end{aligned}$$

from (2.2). Then we get $S'(t) \geqslant \mu(1-S(t)) - \varepsilon$ for $t > T_1$ and so

$$\liminf_{t\to\infty} S(t) \geqslant 1 - \frac{\varepsilon}{\mu}.$$

From the last relation we conclude that there exists at least one $T_3 > T_2$ such that

$$S(t)\mathcal{R}_0 - \frac{I(t+\tau)}{I(t)} > 0,$$

holds for $t > T_3$. So, if we choose T as $T = \max\{T_1, T_3\}$ then we have

$$\begin{aligned} I'(t+\tau) &= p\beta S(t) I(t) e^{-\mu\tau} - (\mu+r) I(t+\tau) \\ &= (\mu+r) I(t) \left(\frac{p\beta S(t) e^{-\mu\tau}}{\mu+r} - \frac{I(t+\tau)}{I(t)} \right) \\ &= (\mu+r) I(t) \left(S(t) \mathcal{R}_0 - \frac{I(t+\tau)}{I(t)} \right) > 0. \end{aligned}$$

for $t > T$. It can be easily seen that this result contradicts with the assumption $I(0) > 0$ and $I(t) \to 0$ as $t \to \infty$. This means that the set $\{P_0\} \subset \Psi$ is weakly $\rho-$repelling. Taking into account that the sets Ψ_0 and Ψ_+ are forward invariant under Φ, it can be conclude that Φ is uniformly weakly $\rho-$persistent by using the method in [3]. Finally we can state that Φ is uniformly strongly $\rho-$persistent from the similar case given in [3]. □

4. Conclusion

As is known, transitions between integral equations and differential equations are possible. Thus, in the mathematical modeling of real life problems, besides differential equations, integral equations can be widely used.

In this study, in which the spread of epidemic diseases in which immunity does not occur after recovery with the incubation period has been mathematically analyzed, a compartmental model has been introduced with the appropriate choosing of known functions in an integral equation.

Unlike the assumption that all exposed individuals transfer to the infectious class, which is valid in classical SEIS models, in the model presented in this study, after the incubation period, exposed individuals either transfer to the infectious class (at rate p) or return to the susceptible class (at rate $1-p$).

After the equilibrium points and the basic reproduction number have been obtained, stability of the presented model has been analyzed. It has been proven that the disease-free equilibrium point is locally and globally stable. It has also been shown that the endemic equilibrium point is uniformly strongly $\rho-$persistent, as well as its local stability. In the model, it is assumed that individuals exposed to the pathogen become infectious at a certain rate (p) at the end of the incubation period (τ) and the remaining individuals transfer to the susceptible class again.

Let we consider a population with the assumptions; $N(t) = 7 \times 10^7$, $\mu = 0.000015$, $\beta = 5 \times 10^{-6}$, $r = 0.2$ and $\tau = 14$. Then the course of the number of infectious for different values of the p with the estimated choosing of parameters has been presented in the simulation below. The figures have been obtained by using the Wolfram Mathematica 12.1 with "NDSolve" code.

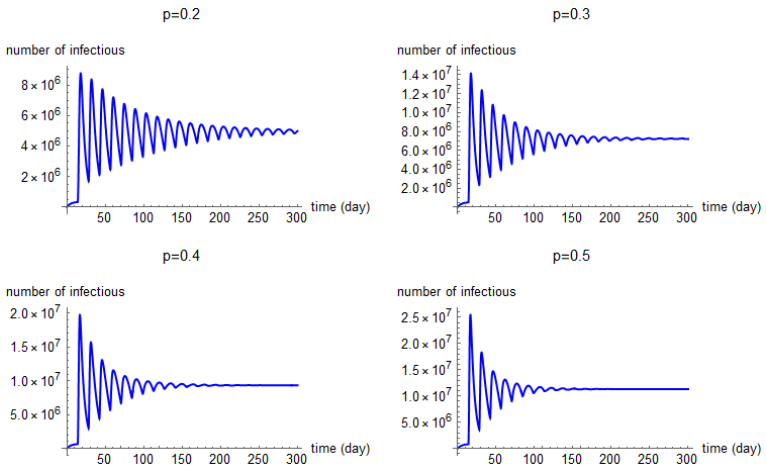

Figure 1: Effect of different values of p.

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