

## DYNAMICS OF A SIRC EPIDEMIOLOGICAL MODEL

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**ABSTRACT.** This article concerns the SIRC epidemiological model for influenza A, which efficiently describes the mechanism of disease spreading, including the susceptible (S), the infected (I) and the recovered (R), along with a cross-immune class (C) that recovers after being infected by different strains of the same viral subtype. The dynamics of the model is completely determined by the basic reproduction number  $R_0$ . If  $R_0 \leq 1$ , the disease-free equilibrium of the SIRC model is globally asymptotically stable, which means influenza A will die out. Otherwise, the SIRC model may have exactly one endemic equilibrium which is globally asymptotically stable under certain parametric conditions. Also, numerical simulations are given to support our analytical results.

### 1. INTRODUCTION

Influenza [7], a member of the family Orthomyxoviridae, is a RNA virus which can give rise to epidemic disease between mankind and animals. In general, influenza is primarily divided into A, B and C types and every type contains a wide variety of subtypes according to hemagglutinin (HA) and neuraminidase (NA) differences. From an epidemiologic standpoint, influenza A is the most common and the most terrible virus among three types, and can result in the highest pathogenicity because of the easiest way to generate variation. What's worse, the virus has brought about more than century pandemic influenza in the past years. The pathogenicity of influenza B virus is the same as type A, but performed studies are shown that type B virus does not contribute to century pandemic. Last, influenza C virus just leads to unobtrusive or feeble respiratory infection and almost doesn't incur a pandemic either.

In this article, we pay attention to influenza A virus whose surface often has a tiny variation, which is referred to as drift. That is, the virus camouflages itself by a subtle change, and it thereby can elude identification by the human immune system. As a result of the drift, all kinds of flu strains appear every year. Therefore, human annually need to take a vaccine against influenza for prevention. On the other hand, shift means influenza A takes place genetic mutations causing a new "subtype". In this case, it can lead to outbreaking a global pandemic influenza. For example,

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Spanish outbreak H1N1 influenza virus in 1918-1919 about the death toll up to 20-40 million [24]. In 1957-1958, Asia influenza erupted due to the result of H2N2 virus. Furthermore, influenza A virus which threatens the lives of even the healthiest individuals brings serious economic loss for many countries. Some research data indicate the direct economic loss up to 10-30 billion dollars in America, and the potential economic losses at 10-15 billion dollars [23]. Consequently, devoting to studying the spread of influenza A is extremely indispensable.

Mathematically modelling, as a bridge, plays a crucial role in understanding of the spread of the epidemic. Researchers transform their focus from considering threshold value of pathophoresis to prevent epidemic propagation by taking some strategies for control [2]. The model of influenza is parallel to the traditional standard SIR model in which the crowd are segmented into three compartments: susceptible (S), infectious (I), recovered (R). A number of compartmental models have been established based on this idea [26]. Nonetheless, some scholars indicate that the traditional SIR model is not adequate to describe actual situation of influenza spreading because every type can evolve different subtypes. Hence, Andreasen et al. [1] initially considered the dynamics of system with multiple virus strains consisting of partial cross-immunity [22], concluding that the system exists stable equilibrium when the number of strains are less than or equal to three. However, in the work of Lin et al. [13], they demonstrated that oscillations can be sustained under a linear chain of three cocirculating influenza A strains. These researchers only consider a special case where cross-protection is symmetric to analyze complex system because the analysis and computation of multiple viral strains are much intractable. Recently, Minayev and Ferguson [18] proposed a deterministic model of multi-strain pathogens with symmetric equilibrium, self-organized strain structures, regular periodic and chaotic regimes, which is determined by cross-immune response function. In addition to these properties, Kooi et al. [11] revealed bifurcation analysis, Lyapunov exponent calculation as well as quantitative and qualitative results by numerical simulations in three multi-strain compartment models. Nuño et al. [21] developed a general multiple strains pathogens model with all kinds of cross-immunity structures. They illustrated the weaker cross-immunity structures are more likely to appear instability in the strain coexistence mode. For seasonal influenza, there is another case where strong and weak cross-immunity can result in coexistence with the following pandemic. On the contrary, the intermediate level one may take the place of the seasonal subtype. Chung and Liu [6] extended the result of Nuño et al. [20] and elucidated the local asymptotic stability of a two-strain influenza model. Also, the stability may be lost when two strains are far apart. All the above published works didn't analyze global asymptotical stability of models with cross-immune class. In this paper, we investigate a SIRC model with cross-immune class proposed by Casagrandi et al. [5], which is represented by

the following system of four ordinary differential equations,

$$\begin{aligned}\frac{dS(t)}{dt} &= \gamma(1 - S) - \beta SI + \eta C, \\ \frac{dI(t)}{dt} &= \beta SI + \mu\beta CI - (\gamma + \alpha)I, \\ \frac{dR(t)}{dt} &= (1 - \mu)\beta CI + \alpha I - (\gamma + \delta)R, \\ \frac{dC(t)}{dt} &= \delta R - \beta CI - (\gamma + \eta)C,\end{aligned}\tag{1.1}$$

with initial conditions:

$$(S(0), I(0), R(0), C(0))^T \in \mathbb{R}_+^4 := \{(S, I, R, C)^T \in \mathbb{R}^4 : S \geq 0, I \geq 0, R \geq 0, C \geq 0\}$$

and positive real parameters  $\gamma$ ,  $\beta$ ,  $\eta$ ,  $\mu$ ,  $\alpha$ , and  $\delta$ . The parameters  $\alpha$ ,  $\delta$  and  $\eta$  are the inverses of the average time spent by the individuals in each of the three compartments  $I$ ,  $R$ , and  $C$ , respectively. The parameter  $\gamma$  denotes the mortality rate in every compartment and is assumed to equal to the rate of newborn in the population. The parameter  $\mu$  is interpreted as the average reinfection probability of a cross-immune subject, whereas the parameter  $\beta$  is the contact rate. The state space of model (1.1) is  $\mathbb{R}_+^4$ . The novelty of this model is that it takes into account the presence of cross-immune (C) subjects, i.e., subjects that are temporarily immune. It efficiently describes the mechanism for influenza A virus spreading. In this paper, we shall not only consider existence, number, and local asymptotical stability of equilibrium for the model, but also verify the globally asymptotical stability. By analyzing the SIRC model, we can gain the law of development of influenza A. In some sense, this paper puts forward an important theoretical approach and decision-making basis in order to prevent influenza A spreading.

Let

$$R_0 = \frac{\beta}{\gamma + \alpha}$$

which is called the basic reproduction number [3, 10] or the contact number that represents the average number of secondary infections from a single infections host. We can prove that the dynamics of model (1.1) is completely determined by the threshold value  $R_0$ . If  $R_0 \leq 1$  then the disease-free equilibrium  $E_0$  is globally asymptotically stable and hence the disease will die out (see Theorem 4.2). If  $R_0 > 1$  then the unique endemic equilibrium  $E^*$  is globally asymptotically stable so that the disease always persists at the unique endemic level (see Theorem 6.3). Thus, we can conclude that the spread of the disease should be controlled by way of suitable protection measures of the society to reduce the value of  $\beta$  (transmission rate of disease) when susceptible individuals contact with infected individuals. This can be done by adopting some strategies to detect early cases among the passengers coming from the infected countries and individuals should follow simple steps like cough etiquettes, stay away from persons coughing or sneezing, avoid gathering and so on. Disease spreading can also be kept under control by increasing  $\alpha$  (recovery rate of the infected population). It is recommended that if one feels any respiratory distress, one should report to a nearby hospital immediately. The global stability of  $E_0$  when  $R_0 \leq 1$  can be routinely proved by using a well-known Lyapunov function, but the global stability of  $E^*$  when  $R_0 > 1$  has been an open problem in

the literature due to the high dimensionality for the model. Our proof is based on a theoretical approach developed in [14, 15].

From a mathematical viewpoint, it is interesting to notice that, in the absence of cross-immunity ( $\mu = 1$ ), the two classes  $S$  and  $C$  are immunologically indistinguishable, since

$$\frac{d(S+C)}{dt} = \gamma[1 - (S+C)] - \beta(S+C)I + \delta R.$$

Consequently, in the limit of  $\mu \rightarrow 1$ , the SIRC model reduces to the following classical SIRS model

$$\begin{aligned} \frac{dS(t)}{dt} &= \gamma(1-S) - \beta SI + \delta R, \\ \frac{dI(t)}{dt} &= \beta SI - (\gamma + \alpha)I, \\ \frac{dR(t)}{dt} &= \alpha I - (\gamma + \delta)R. \end{aligned} \tag{1.2}$$

Thus, our results generalize some earlier results on the SIRS model. In this paper, we shall prove the global stability of a unique endemic equilibrium of (1.2) when the basic reproduction number  $R_0$  is greater than 1.

This article is organized as follows. The positively invariant set is shown in section 2. Section 3 is devoted to the existence of equilibrium including a disease-free equilibrium and a unique endemic equilibrium. The stability of the disease-free equilibrium and the endemic equilibrium is presented in sections 4 and 6, respectively. Section 5 is devoted to the persistence of the model and the work of numerical simulation reveals in section 7. Finally, this paper ends up with a brief conclusion in section 8.

## 2. POSITIVELY INVARIANT SET

This section is devoted to proving the positivity and boundedness of solutions of model (1.1) with initial conditions  $(S(0), I(0), R(0), C(0))^T \in \mathbb{R}_+^4$ . We first introduce the following lemma.

**Lemma 2.1** ([25]). *Suppose  $\Omega \subset \mathbb{R} \times \mathbb{C}^n$  is open,  $f_i \in C(\Omega, \mathbb{R})$ ,  $i = 1, 2, 3, \dots, n$ . If  $f_i|_{x_i(t)=0, X_t \in \mathbb{C}_{+0}^n} \geq 0$ ,  $X_t = (x_{1t}, x_{2t}, \dots, x_{nt})^T$ ,  $i = 1, 2, 3, \dots, n$ , then  $\mathbb{C}_{+0}^n$  is the invariant domain of the following equations*

$$\frac{dx_i(t)}{dt} = f_i(t, X_t), \quad t \geq \sigma, \quad i = 1, 2, \dots, n.$$

**Theorem 2.2.** *Each solution  $(S(t), I(t), R(t), C(t))$  of model (1.1) with the non-negative initial conditions is non-negative for all  $t > 0$ .*

*Proof.* Let  $X = (S, I, R, C)^T$  and  $f(X) = (f_1(X), f_2(X), f_3(X), f_4(X))^T$  then we can rewrite model (1.1) as

$$\dot{X} = f(X)$$

where

$$f(X) = \begin{pmatrix} f_1(X) \\ f_2(X) \\ f_3(X) \\ f_4(X) \end{pmatrix} = \begin{pmatrix} \gamma(1-S) - \beta SI + \eta C \\ \beta SI + \mu \beta CI - (\gamma + \alpha)I \\ (1-\mu)\beta CI + \alpha I - (\gamma + \delta)R \\ \delta R - \beta CI - (\gamma + \eta)C \end{pmatrix}.$$

Note that

$$\begin{aligned} \frac{dS(t)}{dt}|_{S=0} &= r + \eta C > 0, & \frac{dI(t)}{dt}|_{I=0} &= 0, \\ \frac{dR(t)}{dt}|_{R=0} &= (1 - \mu)\beta CI + \alpha I \geq 0, & \frac{dC(t)}{dt}|_{C=0} &= \delta R \geq 0. \end{aligned}$$

Then it follows from Lemma 2.1 that  $\mathbb{R}_+^4$  is invariant set.  $\square$

**Theorem 2.3.**  $\mathbb{D} = \{(S, I, R, C)^T \in \mathbb{R}_+^4 : 0 \leq S + I + R + C \leq 1\}$  is a positively invariant set and also a globally attractive set of model (1.1).

*Proof.* Consider the total population  $N(t) = S(t) + I(t) + R(t) + C(t)$ . Direct calculation leads to

$$\frac{dN}{dt} = \gamma - \gamma N. \quad (2.1)$$

Solving this equation, we obtain

$$N(t) = 1 - (1 - N(0))e^{-\gamma t}.$$

It is straight forward to show that  $N(t) \leq 1$  if  $N(0) \leq 1$ . This means that the set  $\mathbb{D}$  is a positively invariant set for model (1.1). If  $N(0) > 1$  then it turns out that  $\lim_{t \rightarrow \infty} N(t) = 1$ . Thereby the set  $\mathbb{D}$  is the globally attractive set for model (1.1).  $\square$

**Remark 2.4.** In view of the above two theorems, we see that the positively invariant set  $\mathbb{D}$  can attract every solution with initial conditions starting in its state space  $\mathbb{R}_+^4$ . Namely, every trajectory of model (1.1) with the initial conditions in  $\mathbb{R}_+^4$  eventually stays in  $\mathbb{D}$ .

### 3. EXISTENCE OF EQUILIBRIA

In this section, we consider the existence, type, and number of the equilibria. Obviously, model (1.1) has two equilibria: one is disease-free equilibrium  $E_0 = (1, 0, 0, 0)$  which exists for all parameter values; and the other is the endemic equilibrium  $E^* = (S^*, I^*, R^*, C^*)$ , which is a positive solution of the following equation

$$\begin{aligned} \gamma(1 - S^*) - \beta S^* I^* + \eta C^* &= 0, \\ \beta S^* I^* + \mu\beta C^* I^* - (\gamma + \alpha)I^* &= 0, \\ (1 - \mu)\beta C^* I^* + \alpha I^* - (\gamma + \delta)R^* &= 0, \\ \delta R^* - \beta C^* I^* - (\gamma + \eta)C^* &= 0. \end{aligned} \quad (3.1)$$

It follows from the third and fourth equations of (3.1) that

$$C^* = \frac{\delta\alpha I^*}{(\delta\mu + \gamma)\beta I^* + (\gamma + \delta)(\gamma + \eta)}. \quad (3.2)$$

Next, substituting (3.2) in the second equation of (3.1), we obtain

$$S^* = \frac{\gamma + \alpha}{\beta} - \frac{\mu\delta\alpha}{(\delta\mu + \gamma)\beta I^* + (\gamma + \delta)(\gamma + \eta)} \cdot I^*. \quad (3.3)$$

Combining (3.2) with (3.3) and then substituting them in the first equation of (3.1), we have

$$aI^{*2} + bI^* + d = 0, \quad (3.4)$$

where

$$\begin{aligned} a &= \beta^2(\gamma + \alpha)(\delta\mu + \gamma) - \beta^2\mu\delta\alpha = \beta^2(\gamma\delta\mu + \gamma^2 + \alpha\gamma) > 0, \\ b &= \beta\gamma(\gamma + \alpha)(\delta\mu + \gamma) + \beta(\gamma + \alpha)(\gamma + \delta)(\gamma + \eta) \\ &\quad - \gamma\beta\mu\delta\alpha - \gamma\beta^2(\delta\mu + \delta) - \beta\eta\delta\alpha, \\ d &= \gamma(\gamma + \alpha)(\gamma + \delta)(\gamma + \eta) - \gamma\beta(\gamma + \delta)(\gamma + \eta). \end{aligned}$$

Therefore, we have the following result.

**Theorem 3.1.** *If  $R_0 \leq 1$ , model (1.1) always has a disease-free equilibrium  $E_0 = (1, 0, 0, 0)$ . If  $R_0 > 1$ , the model has exactly one endemic equilibrium  $E^* = (S^*, I^*, R^*, C^*)$ .*

*Proof.* It is not difficult to observe that model (1.1) has a disease-free equilibrium  $E_0(1, 0, 0, 0)$ . Now, we consider the existence of the endemic equilibrium  $E^*(S^*, I^*, R^*, C^*)$  when  $R_0 > 1$ . It follows from  $R_0 > 1$  that  $d < 0$  and hence that (3.4) has exactly one positive solution  $I^*$ , which, together with (3.2), implies that  $C^*$  is positive. Similarly, from the first and fourth equations of (3.1), we see that both  $S^*$  and  $R^*$  are positive. Therefore, model (1.1) has exactly one endemic equilibrium  $E^*$  when  $R_0 > 1$ .  $\square$

#### 4. STABILITY OF DISEASE-FREE EQUILIBRIUM

This section is devoted to the local and global stability of the disease-free equilibrium.

**Theorem 4.1.** *The disease-free equilibrium  $E_0(1, 0, 0, 0)$  is locally asymptotically stable if  $R_0 < 1$ . If  $R_0 > 1$  then  $E_0$  is unstable and all solutions starting from sufficiently close to  $E_0$  in  $\mathbb{D}$  ultimately leave away from  $E_0$ , except those starting on  $S$ -axis close to  $E_0$  along this.*

*Proof.* The Jacobian matrix of (1.1) at  $E_0$  is

$$\begin{pmatrix} -\gamma & -\beta & 0 & \eta \\ 0 & \beta - (\gamma + \alpha) & 0 & 0 \\ 0 & \alpha & -(\gamma + \delta) & 0 \\ 0 & 0 & \delta & -(\gamma + \eta) \end{pmatrix}.$$

Its characteristic equation is

$$(\lambda + \gamma)[\lambda - (\beta - (\gamma + \alpha))](\lambda + \gamma + \delta)(\lambda + \gamma + \eta) = 0.$$

If  $R_0 < 1$  then all the characteristic roots are less than 0. That is,  $E_0$  is locally asymptotically stable. If  $R_0 > 1$  then there exists a characteristic value  $\beta - (\gamma + \alpha) > 0$ . Therefore,  $E_0$  is unstable. Moreover, the trajectory starting from sufficiently close to  $E_0$  will be away from a neighborhood of  $E_0$  except that those are on the  $S$ -axis, where model (1.1) can translate into  $\frac{dS(t)}{dt} = \gamma(1 - S(t))$  and hence  $\lim_{t \rightarrow \infty} S(t) = 1$ . This completes the proof.  $\square$

Next we will prove that  $E_0$  is globally asymptotically stable by means of Lyapunov function.

**Theorem 4.2.** *The disease-free equilibrium  $E_0$  for (1.1) is globally asymptotically stable when  $R_0 \leq 1$ .*

*Proof.* Consider a Lyapunov function  $V = V(S, I, R, C)$  defined by

$$V = \frac{1}{2}(S - 1 + I + R + C)^2.$$

The time derivative of  $V$  along a solution of (1.1) is

$$\frac{dV}{dt} = -\gamma(S - 1 + I + R + C)^2 \leq 0.$$

We see that  $\frac{dV}{dt} = 0$  if and only if  $S + I + R + C = 1$ . Consequently, the maximal invariant set in  $\{(S, I, R, C)^T \in \mathbb{R}_+^4 : \frac{dV}{dt} = 0\}$  is in  $\{(S, I, R, C)^T \in \mathbb{R}_+^4 : S + I + R + C = 1\}$ . Therefore, to prove that the disease-free equilibrium  $E_0$  of (1.1) is globally asymptotically stable, it suffices to show that the equilibrium  $(0, 0, 0)$  of the following model is globally asymptotically stable:

$$\begin{aligned} \frac{dI(t)}{dt} &= \beta I(1 - I - R - C) + \mu\beta CI - (\gamma + \alpha)I, \\ \frac{dR(t)}{dt} &= (1 - \mu)\beta CI + \alpha I - (\gamma + \delta)R, \\ \frac{dC(t)}{dt} &= \delta R - \beta CI - (\gamma + \eta)C. \end{aligned} \quad (4.1)$$

Again define a Lyapunov function by

$$V_1 = \frac{1}{2}(R + C)^2 + \frac{k_1}{2}R^2 + k_2I + \frac{k_3}{2}C^2,$$

where

$$k_1 = \frac{\mu}{1 - \mu}, \quad k_2 = \frac{\alpha}{(1 - \mu)\beta}, \quad k_3 = \frac{2\gamma + \eta}{\delta}.$$

Using a similar argument as above, we have

$$\begin{aligned} \frac{dV_1}{dt} &= (R + C)(\dot{R} + \dot{C}) + k_1R\dot{R} + k_2\dot{I} + k_3C\dot{C} \\ &= -k_2\beta I^2 - (\gamma + k_1(\gamma + \delta))R^2 - (\gamma + \eta + k_3(\gamma + \eta))C^2 + k_2(\beta - (\gamma + \alpha))I \\ &\quad + (-\mu + k_1(1 - \mu))\beta CIR + (\alpha + k_1\alpha - k_2\beta)IR + (-(2\gamma + \eta) + k_3\delta)RC \\ &\quad - (\mu + k_3)\beta C^2I + (\alpha + k_2(\mu - 1)\beta)CI. \end{aligned}$$

Therefore,

$$\begin{aligned} \frac{dV_1}{dt} &= -\frac{\alpha}{1 - \mu}I^2 - (\gamma + \frac{\mu(\gamma + \delta)}{1 - \mu})R^2 - (\gamma + \eta)(1 + \frac{2\gamma + \eta}{\delta})C^2 \\ &\quad + \frac{\alpha}{(1 - \mu)\beta}(\beta - (\gamma + \alpha))I - (\mu + \frac{2\gamma + \eta}{\delta})\beta C^2I. \end{aligned}$$

Since  $R_0 \leq 1$ , we obtain

$$\frac{dV_1}{dt} \leq 0.$$

Obviously, (4.1) implies that the largest invariant set in the set of  $\frac{dV_1}{dt} = 0$  is  $(0, 0, 0)$ . By LaSalle's invariant principle [12, 16], we conclude that model (4.1) is global asymptotically stable at  $(0, 0, 0)$ .  $\square$

**Remark 4.3.** From the perspective of epidemiological significance, Theorem 4.2 demonstrates that the disease ultimately dies out regardless of the initial values of model (1.1) when  $R_0 \leq 1$ .

Take  $\mu = 1$ , then (1.1) can reduce to the classical SIRS model of the form (1.2). Hence, we can deduce that the disease-free equilibrium  $G_0 = (1, 0, 0)$  of (1.2) is globally asymptotically stable when  $R_0 \leq 1$ . Namely, we have the following result.

**Corollary 4.4.** *The disease-free equilibrium for model (1.2) is globally asymptotically stable when  $R_0 \leq 1$ .*

In fact, it is straightforward to show that the disease-free equilibrium  $G_0 = (1, 0, 0)$  is locally asymptotically stable. We can also illustrate that  $G_0$  is globally asymptotically stable by considering the following Lyapunov function

$$V_2 = \frac{1}{2}(S - 1 + I + R)^2 + \frac{2\gamma}{\beta}I + \frac{\gamma}{\alpha}R^2.$$

## 5. PERSISTENCE

In the section, we will establish the persistence theorem of disease when  $R_0 > 1$ . Persistence implies that the infected individuals will persist in the future. In this paper, we first use the definition given by Butler and Waltman [4]. That is, model (1.1) is said to be uniformly persistent if there exists a positive number  $b$  such that

$$\min \left\{ \liminf_{t \rightarrow \infty} (S(t)), \liminf_{t \rightarrow \infty} (I(t)), \liminf_{t \rightarrow \infty} (R(t)), \liminf_{t \rightarrow \infty} (C(t)) \right\} = b \quad (5.1)$$

for every trajectory with positive initial conditions.

For a region  $E$ , denote by  $\partial E$  and  $\overset{\circ}{E}$  the boundary and the interior of  $E$ , respectively. Denote by  $\partial \mathcal{F}$  the restriction of the flow  $\mathcal{F}$  to  $\partial E$  and note that  $\partial E$  is, in general, not positively invariant. Let  $N$  be the maximal invariant set of  $\partial E$ . Suppose  $N$  is a closed invariant set and there exists a cover  $\{N_\alpha\}_{\alpha \in A}$  of  $N$ , where  $A$  is a nonempty index set.  $N_\alpha \subset \partial E$ ,  $N \subset \cup_{\alpha \in A} N_\alpha$  and  $N_\alpha (\alpha \in A)$  are pairwise disjoint closed invariant sets. Furthermore, we propose the following hypotheses:

- (H1) All  $N_\alpha$  are isolated invariant sets of the flow  $\mathcal{F}$ ;
- (H2)  $N_\alpha (\alpha \in A)$  is acyclic, that is, any finite subset of  $N_\alpha (\alpha \in A)$  does not form a cycle;
- (H3) Any compact subset of  $\partial E$  contains, at most, finitely many sets of  $N_\alpha (\alpha \in A)$ .

The following lemma plays an important role in analyzing the uniform persistence.

**Lemma 5.1** ([9]). *Let  $E$  be a closed positively invariant subset of  $X$  on which a continuous flow  $\mathcal{F}$  is defined. Suppose there is a constant  $\alpha > 0$  such that  $\mathcal{F}$  is point dissipative on  $S[\partial E, \alpha] \cap \overset{\circ}{E}$  and the assumptions (H1)–(H3) hold. Then the flow  $\mathcal{F}$  is uniformly persistent if and only if  $W^+(N_\alpha) \cap S[\partial E, \alpha] \cap \overset{\circ}{E} = \emptyset$  for all  $\alpha \in A$ , where  $W^+(N_\alpha) = \{y \in X \mid \Lambda^+(y) \subset N_\alpha\}$ .*

**Theorem 5.2.** *Model (1.1) is uniformly persistent in  $\mathbb{D}$  if and only if  $R_0 > 1$ .*

*Proof.* It is easy to prove the necessity by means of Theorems 4.1 and 4.2 because the asymptotical stability of  $E_0$  excludes all kinds of persistence. Now, we prove the sufficiency of this theorem by using Lemma 5.1. Choose  $X = \mathbb{R}^4$  and  $E = \mathbb{D}$ . We only need to prove that model (1.1) satisfies all the conditions of Lemma 5.1. Note that the maximal invariant set on the boundary  $\partial \mathbb{D}$  only contains a point  $E_0$  which is isolated. Then the assumptions (H1)–(H3) are satisfied. By Lemma 5.1, we observe that the uniform persistence of model (1.1) is equivalent to the instability of the disease-free equilibrium. Thereby, the proof is complete.  $\square$

**Remark 5.3.** It follows from Theorem 5.2 that the uniform persistence of model (1.1) in the bounded set  $\mathring{\mathbb{D}}$  is equivalent to the existence of a compact attractor  $K \subset \mathring{\mathbb{D}}$ .

## 6. STABILITY OF THE ENDEMIC EQUILIBRIUM

This section concerns the stability of the endemic equilibrium  $E^*(S^*, I^*, R^*, C^*)$  when  $R_0 > 1$ .

**Theorem 6.1.**  $E^*(S^*, I^*, R^*, C^*)$  is locally asymptotically stable when  $R_0 > 1$ .

*Proof.* Let  $N(t) = S(t) + I(t) + R(t) + C(t)$ . It is easy to see that  $\dot{N}(t) = \gamma - \gamma N(t)$ . By a change of variables, we see that model (1.1) is equivalent to the model

$$\begin{aligned} \dot{N} &= \gamma - \gamma N, \\ \dot{S} &= \gamma(1 - S) - \beta SI + \eta C, \\ \dot{I} &= \beta SI + \mu\beta CI - (\gamma + \alpha)I, \\ \dot{C} &= \delta(N - S - I - C) - \beta CI - (\gamma + \eta)C. \end{aligned} \tag{6.1}$$

By Theorem 3.1 model (6.1) has a unique endemic equilibrium  $(N^*, S^*, I^*, C^*)$ , where  $N^* = S^* + I^* + R^* + C^*$ . Now, it suffices to verify that  $(N^*, S^*, I^*, C^*)$  is locally asymptotically stable to show that  $E^*(S^*, I^*, R^*, C^*)$  of model (1.1) is locally asymptotically stable.

The Jacobian matrix of model (6.1) at  $(N^*, S^*, I^*, C^*)$  is

$$\begin{pmatrix} -\gamma & 0 & 0 & 0 \\ 0 & -\gamma - \beta I^* & -\beta S^* & \eta \\ 0 & \beta I^* & \beta S^* + \mu\beta C^* - (\gamma + \alpha) & \mu\beta I^* \\ \delta & -\delta & -\delta - \beta C^* & -\delta - \beta I^* - (\gamma + \eta) \end{pmatrix}.$$

Since  $\beta S^* + \mu\beta C^* - (\gamma + \alpha) = 0$ , its characteristic equation is

$$(\lambda + \gamma)(d_0\lambda^3 + d_1\lambda^2 + d_2\lambda + d_3) = 0,$$

where

$$\begin{aligned} d_0 &= 1, & d_1 &= \delta + \beta I^* + \gamma + \eta + \gamma + \beta I^*, \\ d_2 &= \mu\beta I^*(\delta + \beta C^*) + (\gamma + \beta I^*)(\delta + \beta I^* + \gamma + \eta) + \beta I^*\beta S^* + \delta\eta, \\ d_3 &= (\gamma + \beta I^*)\mu\beta I^*(\delta + \beta C^*) + \beta I^*\beta S^*(\beta I^* + \gamma + \eta) + \beta I^*\eta(\delta + \beta C^*) \\ &\quad + \delta(1 - \mu)\beta I^*\beta S^*. \end{aligned}$$

Therefore, the conclusion of this theorem is verified if the real parts of all solutions of  $d_0\lambda^3 + d_1\lambda^2 + d_2\lambda + d_3 = 0$  are negative. From above, we have  $d_0 > 0$ ,  $d_1 > 0$ ,  $d_2 > 0$ ,  $d_3 > 0$ , and

$$\begin{aligned} d_1d_2 - d_0d_3 &= (\gamma + \beta I^*)(\delta + \eta + \gamma + \beta I^*)(\delta + \eta + \gamma + \beta I^* + \gamma + \beta I^*) \\ &\quad + \mu\beta I^*(\delta + \beta C^*)(\delta + \eta + \gamma + \beta I^*) + \delta\eta(\delta + \gamma + \eta + \gamma + \beta I^*) \\ &\quad + \delta\mu\beta I^*\beta S^* + \beta I^*\beta S^*(\gamma + \beta I^*) - \beta I^*\beta C^*\eta. \end{aligned}$$

It follows from the second of model (6.1) that

$$\eta\beta C^* = -\gamma\beta + \gamma\beta S^* + \beta S^*\beta I^*.$$

We get

$$\beta I^*\beta S^*(\gamma + \beta I^*) - \beta I^*\beta C^*\eta = \gamma\beta\beta I^*,$$

namely,  $d_1d_2 - d_0d_3 > 0$ . By applying the Routh-Hurwitz criterion, we can verify that  $(N^*, S^*, I^*, C^*)$  is locally asymptotically stable.  $\square$

Next, we consider the global asymptotical stability of the endemic equilibrium  $E^*$ . The routine technique of the global asymptotical stability of endemic equilibrium is based on Lyapunov function and the Poincaré-Bendixson trichotomy. Here we will utilize another method, which is developed by Li and Muldowney [14, 15].

Let  $x \mapsto f(x) \in \mathbb{R}^n$  be a  $C^1$  function for  $x$  in an open set  $D \subset \mathbb{R}^n$ . Consider the differential equation

$$\dot{x} = f(x). \quad (6.2)$$

Denote by  $x(t, x_0)$  the solution to (6.2) such that  $x(t, x_0) = x_0$ , and introduce the following two assumptions:

- (H3) There exists a compact absorbing set  $K \subset D$ ;
- (H4) Equation (6.2) has a unique equilibrium  $\bar{x}$  in  $D$ .

Let  $A$  be an  $n \times n$  matrix,  $A^{[2]}$  is called the second additive compound matrix of  $A$ , which is an  $\binom{n}{2} \times \binom{n}{2}$  matrix. For instance, when  $n = 3$ ,

$$f(x) = \begin{pmatrix} a_{11} + a_{22} & a_{23} & -a_{13} \\ a_{32} & a_{11} + a_{33} & a_{12} \\ -a_{31} & a_{21} & a_{22} + a_{33} \end{pmatrix}.$$

For the detailed discussions of compound matrix and their properties we refer the reader to [8, 19]. Let  $x \mapsto P(x)$  be a  $\binom{n}{2} \times \binom{n}{2}$  matrix-value function that is  $C^1$  for  $x \in D$ . Assume that  $P^{-1}(x)$  exists and is continuous in  $x \in K$ , where  $K$  is the compact absorbing set. A quantity  $\bar{q}_2$  is defined as

$$\bar{q}_2 = \limsup_{t \rightarrow \infty} \sup_{x_0 \in K} \frac{1}{t} \int_0^t \mu(B(x(s, x_0))) ds,$$

where

$$B = P_f P^{-1} + P \frac{\partial f^{[2]}}{\partial x} P^{-1},$$

and the matrix  $P_f$  is obtained by replacing each entry  $p_{ij}$  of  $P$  by its derivative in the direction of  $f$ ,  $p_{ijf}$ . The quantity  $\mu(B)$  is Lozinskii measure of  $B$  with respect to a vector norm  $|\cdot|$  in  $\mathbb{R}^N$ ,  $N = \binom{n}{2} \times \binom{n}{2}$ , defined by

$$\mu(B) = \lim_{h \rightarrow 0^+} \frac{|I + hB| - 1}{h},$$

see [17]. The following global stability result is [14, Theorem 3.5].

**Lemma 6.2.** *Assume that  $\mathbb{D}$  is simple connected and that assumptions (H3) and (H4) hold. Then the unique equilibrium  $\bar{x}$  of (6.2) is global stable in  $\mathbb{D}$  if  $\bar{q}_2 < 0$ .*

**Theorem 6.3.** *If  $R_0 > 1$  then the endemic equilibrium  $E^*$  of (1.1) is globally asymptotically stable when  $\eta < \gamma$  and  $\delta - \eta + \alpha + \beta < \gamma$ .*

*Proof.* From the discussions of Theorem 3.1 and Remark 5.3, we conclude that model (1.1) satisfies the assumptions (H3) and (H4) in  $\mathbb{D}$ . From the proof of Theorem 2.3, we have  $\lim_{t \rightarrow \infty} N(t) = 1$ . Thus, we just need to consider the following

limiting equation of model (1.1):

$$\begin{aligned}\dot{S} &= \gamma(1 - S) - \beta SI + \eta C, \\ \dot{I} &= \beta SI + \mu\beta CI - (\gamma + \alpha)I, \\ \dot{C} &= \delta(1 - S - I - C) - \beta CI - (\gamma + \eta)C.\end{aligned}\tag{6.3}$$

Let  $f = (f_1, f_2, f_3)^T$ , where  $f_1, f_2$  and  $f_3$  represent the right-hand sides of model (6.3), respectively. Furthermore, let  $x = (S, I, C)^T$ , then the Jacobian matrix associated with a general solution  $x(t)$  of model (6.3) is

$$\frac{\partial f}{\partial x} = \begin{pmatrix} -\gamma - \beta I & -\beta S & \eta \\ \beta I & \beta S + \mu\beta C - (\gamma + \alpha) & \mu\beta I \\ -\delta & -\delta - \beta C & -\delta - \beta I - (\gamma + \eta) \end{pmatrix}.$$

The second additive compound matrix of  $\frac{\partial f}{\partial x}$  is

$$\frac{\partial f^{[2]}}{\partial x} = \begin{pmatrix} g_{11} & \mu\beta I & -\eta \\ -\delta - \beta C & g_{22} & -\beta S \\ \delta & \beta I & g_{33} \end{pmatrix},$$

where

$$\begin{aligned}g_{11} &= -\gamma - \beta I + \beta S + \mu\beta C - (\gamma + \alpha), \\ g_{22} &= -\gamma - \beta I - \delta - \beta I - (\gamma + \eta), \\ g_{33} &= \beta S + \mu\beta C - (\gamma + \alpha) - \delta - \beta I - (\gamma + \eta).\end{aligned}$$

Set the function  $P(x) = P(S, I, C) = \text{diag}(\frac{S}{I}, \frac{S}{I}, \frac{S}{I})$ , then we have

$$P_f P^{-1} = \text{diag}\left(\frac{\dot{S}}{S} - \frac{\dot{I}}{I}, \frac{\dot{S}}{S} - \frac{\dot{I}}{I}, \frac{\dot{S}}{S} - \frac{\dot{I}}{I}\right).$$

Therefore,

$$B = P_f P^{-1} + P \frac{\partial f^{[2]}}{\partial x} P^{-1} = \begin{pmatrix} B_{11} & B_{12} \\ B_{21} & B_{22} \end{pmatrix},$$

where

$$\begin{aligned}B_{11} &= g_{11} + \frac{\dot{S}}{S} - \frac{\dot{I}}{I}, & B_{12} &= (\mu\beta I, -\eta), \\ B_{21} &= (-\delta - \beta C, \delta)^T, & B_{22} &= \begin{pmatrix} g_{22} + \frac{\dot{S}}{S} - \frac{\dot{I}}{I} & -\beta S \\ \beta I & g_{33} + \frac{\dot{S}}{S} - \frac{\dot{I}}{I} \end{pmatrix}.\end{aligned}$$

Let  $(u, v, w)$  denote the vector in  $\mathbb{R}^3$ , we consider the following norm in  $\mathbb{R}^3$ ,

$$|(u, v, w)| = \max\{|u|, |v| + |w|\},$$

and let  $\mu_1$  denote the Lozinskii measure with respect to this norm. Using the method of estimating  $\mu_1$  in [17], we have

$$\mu_1(B) \leq \sup\{g_1, g_2\},$$

where  $g_1 = \mu_1(B_{11}) + |B_{12}|$ ,  $g_2 = \mu_1(B_{22}) + |B_{21}|$ , and  $|B_{12}|, |B_{21}|$  are matrix norms with respect to the  $L^1$  vector norm and  $\mu_1$  denote the Lozinskii measure with respect to the  $L^1$  norm. From (6.1), it implies that

$$\frac{\dot{I}}{I} = \beta S + \mu\beta C - (\gamma + \alpha).$$

Therefore,

$$\begin{aligned}\mu_1(B_{11}) &= \frac{\dot{S}}{S} - \gamma - \beta I, \quad |B_{12}| = \max\{\mu\beta I, \eta\}, \quad |B_{21}| = 2\delta + \beta C, \\ \mu_1(B_{22}) &= \frac{\dot{S}}{S} - \gamma + \max\{-\delta - \beta I - \eta - \beta S - \mu\beta C + \alpha, \beta S - \delta - \beta I - \eta\}.\end{aligned}$$

Thus, we have

$$\begin{aligned}g_1 &= \frac{\dot{S}}{S} - \gamma + \max\{(\mu - 1)\beta I, -\beta I + \eta\} < \frac{\dot{S}}{S} - \gamma + \eta, \\ g_2 &= \frac{\dot{S}}{S} - \gamma + \max\{\delta - \beta I - \eta - \beta S + (1 - \mu)\beta C + \alpha, \beta S + \delta - \beta I - \eta + \beta C\} \\ &< \frac{\dot{S}}{S} - \gamma + \delta - \eta + \max\{\alpha + \beta C, \beta S + \beta C\} \\ &< \frac{\dot{S}}{S} - \gamma + \delta - \eta + \alpha + \beta.\end{aligned}$$

This leads to

$$\mu_1(B) \leq \frac{\dot{S}}{S} - \gamma + \max\{\eta, \delta - \eta + \alpha + \beta\},$$

where  $\omega = \max\{\eta, \delta - \eta + \alpha + \beta\} < \gamma$ . Consequently,

$$\frac{1}{t} \int_0^t \mu_1(B) ds \leq \frac{1}{t} \log \frac{S(t)}{S(0)} - (\gamma - \omega),$$

which yields  $\bar{q}_2 < 0$ . The proof is complete.  $\square$

**Remark 6.4.** Theorems 5.2 and 6.3 describe that the disease always persists and becomes endemic at an endemic level, no matter how small size the initial value of infections has. To eradicate the disease, what we need to do is to reduce the key of threshold value  $R_0$  to below 1.

In view of Theorem 6.3, we can obtain the global asymptotical stability of endemic equilibrium  $E^*$  of model (1.1) when  $R_0 > 1$ ,  $\eta < \gamma$  and  $\delta - \eta + \alpha + \beta < \gamma$ . In the following theorem, we shall see that the constraint that  $\eta < \gamma$  and  $\delta - \eta + \alpha + \beta < \gamma$  is not necessary if we consider the global asymptotical stability of endemic equilibrium of model (1.2), which can be regarded as a special case of model (1.1).

**Theorem 6.5.** *If  $R_0 > 1$  then the unique endemic equilibrium of (1.2) is globally asymptotically stable.*

*Proof.* It is easy to see that if  $R_0 > 1$  then model (1.2) has a unique endemic equilibrium  $G^* = (S_1^*, I_1^*, R_1^*)$ , where

$$S_1^* = \frac{\gamma + \alpha}{\beta}, \quad I_1^* = \frac{(\gamma + \delta)(\beta - \gamma - \alpha)}{(\gamma + \delta + \alpha)\beta}, \quad R_1^* = \frac{\alpha(\beta - \gamma - \alpha)}{(\gamma + \delta + \alpha)\beta}.$$

Meanwhile, we further see that  $G^*$  is locally asymptotically stable. Next, we show that  $G^*$  is globally asymptotically stable by considering the following Lyapunov function

$$V_3 = \frac{1}{2}(S - S_1^* + I - I_1^* + R - R_1^*)^2 + k_4(I - I_1^* + I_1^* \log \frac{I}{I_1^*}) + \frac{k_5}{2}(S - S_1^* + I - I_1^*)^2,$$

where  $k_4$  and  $k_5$  are positive constants to be determined later. The time derivative of  $V_3$  along the solutions of (1.2) is

$$\frac{dV_3}{dt} = (S - S_1^* + I - I_1^* + R - R_1^*)(\dot{S} + \dot{I} + \dot{R}) + k_4 \frac{I - I_1^*}{I} \dot{I} + k_5 (S - S_1^* + I - I_1^*)(\dot{S} + \dot{I}).$$

Note that

$$\begin{aligned} \gamma(1 - S_1^*) - \beta S_1^* I_1^* + \delta R_1^* &= 0, \\ \beta S_1^* I_1^* - (\gamma + \alpha) I_1^* &= 0, \\ \alpha I_1^* - (\gamma + \delta) R_1^* &= 0. \end{aligned}$$

Then, we have

$$\begin{aligned} \frac{dV_3}{dt} &= (S - S_1^* + I - I_1^* + R - R_1^*)(-\gamma)(S - S_1^* + I - I_1^* + R - R_1^*) \\ &\quad + k_4 (I - I_1^*) \beta (S - S_1^*) + k_5 (S - S_1^* + I - I_1^*)(-\gamma(S - S_1^*) \\ &\quad + \delta(R - R_1^*) - (\gamma + \alpha)(I - I_1^*)) \\ &= -\gamma(1 + k_5)(S - S_1^*)^2 - (\gamma + k_5(\gamma + \alpha))(I - I_1^*)^2 - \gamma(R - R_1^*)^2 \\ &\quad + (-2\gamma + k_4\beta - k_5(2\gamma + \alpha))(S - S_1^*)(I - I_1^*) \\ &\quad + (-2\gamma + k_5\delta)(S - S_1^*)(R - R_1^*) + (-2\gamma + k_5\delta)(I - I_1^*)(R - R_1^*). \end{aligned}$$

Take  $k_4 = 2\gamma(\delta + 2\gamma + \alpha)/(\delta\beta)$  and  $k_5 = 2\gamma/\delta$ , then we have

$$\frac{dV_3}{dt} = -\gamma\left(1 + \frac{2\gamma}{\delta}\right)(S - S_1^*)^2 - \left(\gamma + \frac{2\gamma}{\delta}(\gamma + \alpha)\right)(I - I_1^*)^2 - \gamma(R - R_1^*)^2 \leq 0.$$

Therefore, the LaSalle’s invariant principle [12, 16] implies that  $G^*$  is globally asymptotically stable. □

### 7. NUMERICAL SIMULATION

In this section, we aim to provide a numerical simulation to substantiate the theoretical results established in the previous sections by using the Runge-Kutta fourth order iterative method. Consider model (1.1) with the parameters given as follows:  $\gamma = 0.3, \eta = 0.2, \mu = 0.05, \alpha = 0.5, \delta = 0.5, \beta = 0.4$ .

In Figure 1, we set up two sets of initial values. One case is that  $S(0) = 0.3, I(0) = 0.5, R(0) = 0, C(0) = 0$ . Another case is that  $S(0) = 20, I(0) = 20, R(0) = 0, C(0) = 0$ . It is observed in Figure 1 that all trajectories of model (1.1) eventually stay in the positively invariant set  $\mathbb{D}$  regardless of whether or not the initial values are in  $\mathbb{D}$  and that we can obtain the pivotal threshold value  $R_0 = 0.5$  for the choice of parameters. In this case, it follows from Theorem 3.1 that (1.1) has a unique equilibrium  $E_0 = (1, 0, 0, 0)$ . Theorem 4.2 means that this disease-free equilibrium is globally asymptotically stable.

Figure 1 shows that the infected individuals are eventually eradicated from the crowd, while the susceptible individuals will ultimately approach the maximum value. The epidemiological implication of Figure 2(a) is that the infected population vanish over time. In other words, the disease will die out in the long time. If we change the value of  $\beta$  into 0.98, then  $R_0 = 1.225$  and hence  $E_0$  is unstable (see Figure 2(b)).

If  $(\gamma, \eta, \mu, \alpha, \delta, \beta) = (0.3, 0.2, 0.05, 0.5, 0.5, 0.9)$ , then there exists a unique endemic equilibrium  $E^*$  which is locally asymptotically stable (see Theorem 6.1). Figure 3 implies that the number of infected individuals persist and gradually tend

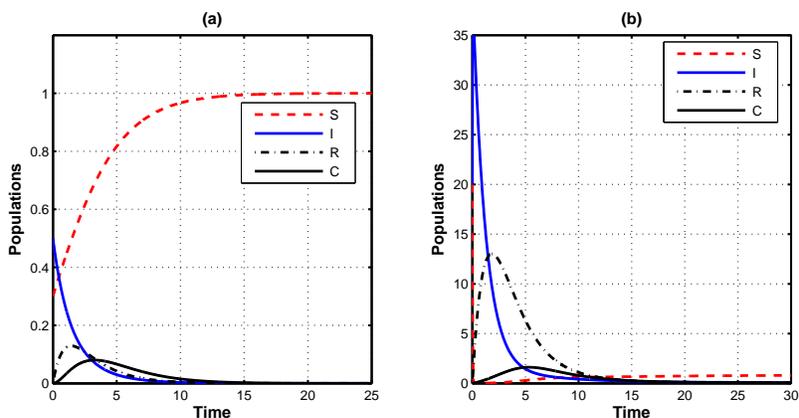


FIGURE 1. Solution of (1.1) with  $\gamma = 0.3$ ,  $\eta = 0.2$ ,  $\mu = 0.05$ ,  $\alpha = 0.5$ ,  $\delta = 0.5$ ,  $\beta = 0.4$ , where the initial value is (a):  $S(0) = 0.3$ ,  $I(0) = 0.5$ ,  $R(0) = 0$ ,  $C(0) = 0$ , and (b):  $S(0) = 20$ ,  $I(0) = 20$ ,  $R(0) = 0$ .

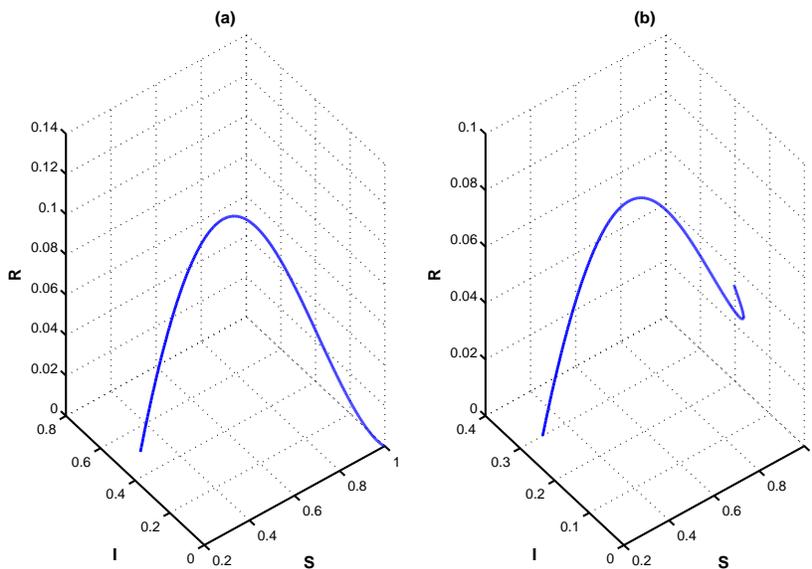


FIGURE 2. Solution of (1.1) with  $S(0) = 0.3$ ,  $I(0) = 0.5$ ,  $R(0) = 0$ ,  $C(0) = 0$  and  $\gamma = 0.3$ ,  $\eta = 0.2$ ,  $\mu = 0.05$ ,  $\alpha = 0.5$ ,  $\delta = 0.5$ , where (a)  $\beta = 0.4$  and (b)  $\beta = 0.98$ .

to a positive constant when  $R_0 = 1.125 > 1$ . If we take the parameters  $\gamma = 0.4$ ,  $\eta = 0.35$ ,  $\mu = 0.05$ ,  $\alpha = 0.05$ ,  $\delta = 0.05$ ,  $\beta = 0.5$ , then all the conditions of Theorem

6.3 are satisfied, and hence that  $E^*$  is globally asymptotically stable. The epidemiological implication of Figure 4 is that the infected individuals always exist if its initial value is non-negative.

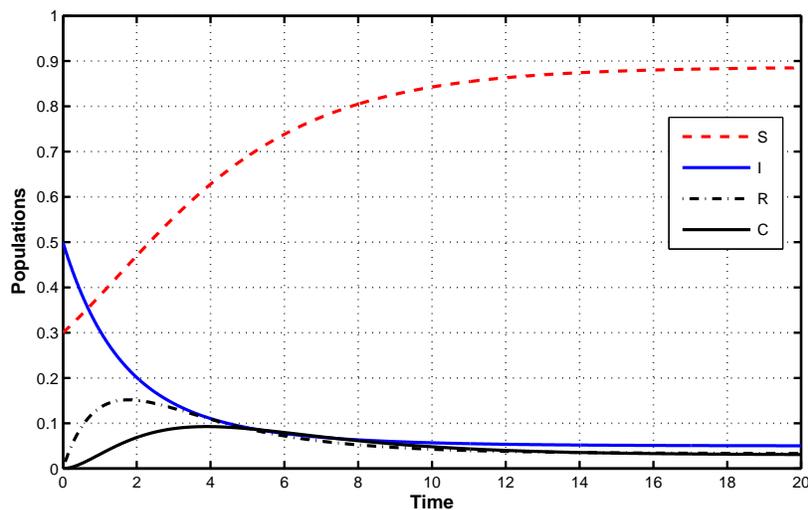


FIGURE 3. Solution of (1.1) with  $S(0) = 0.3$ ,  $I(0) = 0.5$ ,  $R(0) = 0$ ,  $C(0) = 0$  and  $\gamma = 0.3$ ,  $\eta = 0.2$ ,  $\mu = 0.05$ ,  $\alpha = 0.5$ ,  $\delta = 0.5$ ,  $\beta = 0.9$ .

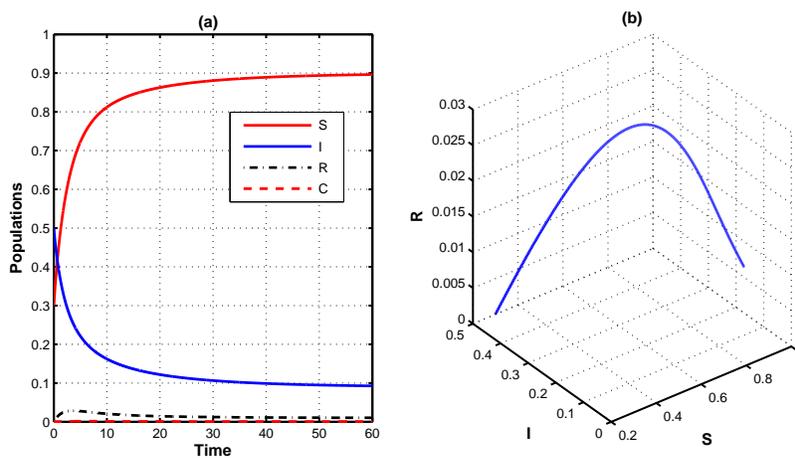


FIGURE 4. Solution of (1.1) with  $S(0) = 0.3$ ,  $I(0) = 0.5$ ,  $R(0) = 0$ ,  $C(0) = 0$  and  $\gamma = 0.4$ ,  $\eta = 0.35$ ,  $\mu = 0.05$ ,  $\alpha = 0.05$ ,  $\delta = 0.05$ ,  $\beta = 0.5$ .

In what follows, we carry out sensitivity analyses for (1.1) by the change of the recovery rate  $\alpha$  as well as the contact rate  $\beta$ . In Figure 5, we study the effect

of parameter  $\alpha$  on model (1.1). We see that parameter  $\alpha$  is directly proportional with the number of susceptible, recovered, cross-immune individuals. However, it is inversely proportional with the number of infectious individuals, and infectious individuals finally eradicate. It follows from Figure 5 that the threshold value  $R_0$  reduces to be less than 1 by increasing  $\alpha$  so that the endemic equilibrium vanishes. Therefore, model (1.1) just has a disease-free equilibrium. This means that the larger the parameter  $\alpha$  is, the smaller the basic reproduction number  $R_0$  is, and hence the faster the disease die out. From a biological perspective, we should reduce the value of  $R_0$  as possible as we can in order that the disease dies out quickly.

Finally, we examine the influence of the contact rate  $\beta$ . In Figure 6, we observe that the number of infectious, recovered, cross-immune individuals are directly proportional with the parameter  $\beta$ , but the number of susceptible individuals are inversely proportional with the parameter  $\beta$ , and finally approach to 1. Why these phenomena happened is that the disease-free equilibrium becomes unstable and an endemic equilibrium appears in (1.1) when the threshold value  $R_0$  increases and passes through 1. This implies that the larger the parameter  $\beta$  is, the larger the value of  $R_0$  is, and then the higher the endemic level will be. As a result, more and more population contacted with infected individuals will make the disease persist at an endemic level.

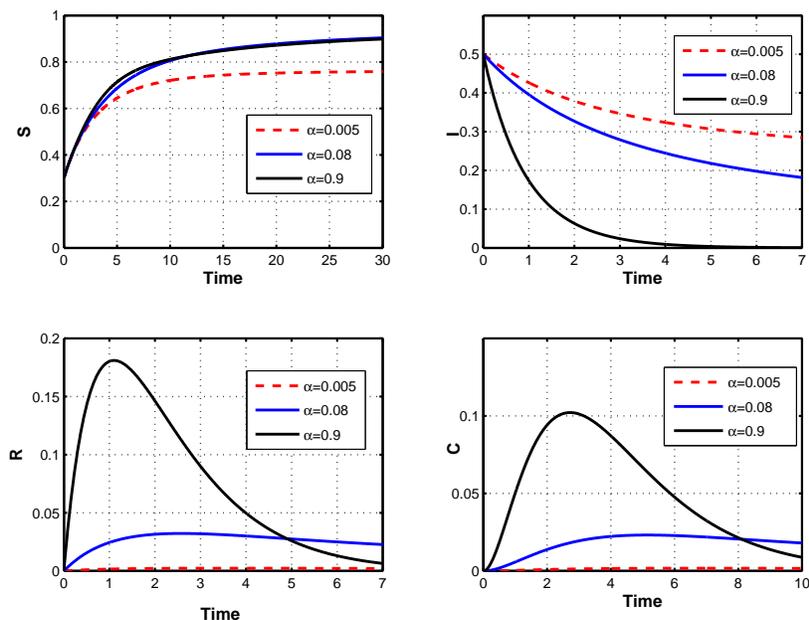


FIGURE 5. Sensitivity of model (1.1) for different values of  $\alpha$ .

**Conclusions.** This paper presents a mathematical study on the dynamics of an SIRC epidemiological model established by Casagrandi et al. [5]. The basic reproduction number  $R_0$  plays a vital role in determining the global dynamics of (1.1).

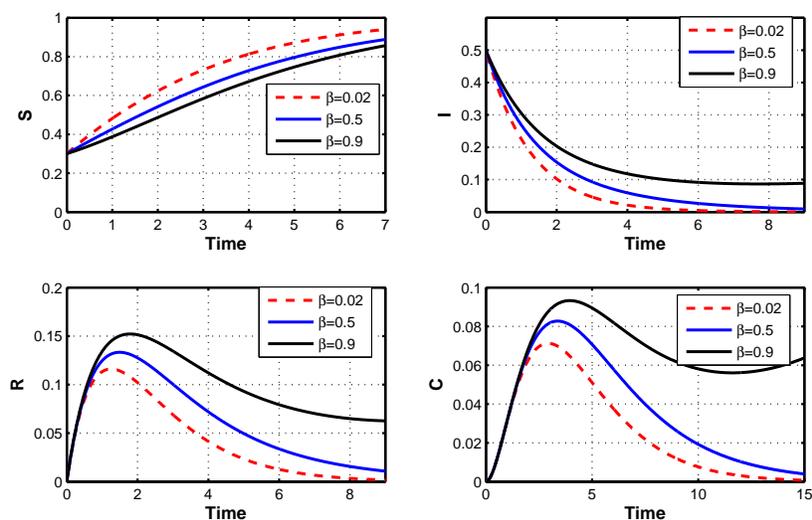


FIGURE 6. Sensitivity of model (1.1) for different values of  $\beta$ .

It is noted that the model always has a disease-free equilibrium, which is globally asymptotically stable when  $R_0 \leq 1$ . When  $R_0 > 1$ , we apply the Routh-Hurwitz criterion to prove that the model has a unique endemic equilibrium, which is locally asymptotically stable. In this case, the disease-free equilibrium become unstable. Based on Li-Muldowney's global-stability criterion [14], we show that the unique endemic equilibrium can be globally asymptotically stable in a feasible region, i.e., influenza A becomes endemic. Although we have established the global stability of the unique endemic equilibrium  $E^*$  when  $R_0 > 1$ , our results are obtained under the assumptions that  $\eta < \gamma$  and  $\delta - \eta + \alpha + \beta < \gamma$ . From Theorem 6.5, we conjecture that the condition that  $\eta < \gamma$  and  $\delta - \eta + \alpha + \beta < \gamma$  is not necessary. Therefore, the perspective of our work is to show the assumption that  $R_0 > 1$  is a sufficient and necessary condition ensuring the globally asymptotical stability of the unique endemic equilibrium  $E^*$ .

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