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On a Discretized Fractional-Order SIR Model for Influenza A Viruses

*Sanaaa Moussa Salman**

Faculty of Education, Alexandria University, Alexandria, Egypt

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Abstract: In the present work, a discretized fractional-order SIR model for an Influenza A viruses is derived. The basic reproductive number $\Re_0$ is defined and the dynamic behavior of the discretized model is investigated. Local stability of both the disease free equilibrium and the endemic equilibrium is investigated. Equations and inequalities of critical bifurcation surfaces at the disease free equilibrium are given. Numerical simulations are performed to assure the analytical results obtained and to reveal the complex dynamics of the discretized model.

Keywords: SIR models, fractional calculus, influenza A viruses, local stability, bifurcation, chaos.

1 Introduction

Infectious diseases have always been an important part of our life. Many of them, such as Influenza, have small symptoms and are purely an annoyance; but others, such as AIDS, fill us with dread. Over the past decades, the spread of diseases has been understood with the help of mathematics by relating basic public-health questions to some infection parameters. Mathematical modeling is central to infectious disease epidemiology. They describe the dynamical evolution of infectious diseases which improve our understanding and predictive ability. Simplest models classify individuals as one of susceptible, infectious or recovered; this is known as the SIR model. An infectious disease transmission dynamics can be described by modeling the individuals. Epidemic models defined in continuous-time have been widely investigated by many researchers ([1]-[5]). Meanwhile, many researchers used some discretization methods to the continuous-time systems to study the consistency, convergence, permanence, stability of the discrete system ([6]-[9]).

In recent years, many authors have suggested that discrete-time models are more suitable than their continuous counterparts. This actually because They do not only have the basic properties of their continuous counterparts but also they provide a reduction of computer time [10]. In fact, discrete models exhibit more complex dynamics than observed in the continuous-time models and their dynamics can not be predicted biologically ([11]-[15]).

Influenza, or “flu”, is a birds and mammals infectious disease. In virus classification, influenza viruses are RNA viruses that make up three of the five genera of the family Orthomyxoviridae: Influenza virus A, Influenza virus B, and Influenza virus C. The the most virulent human pathogens among the three influenza types are type A ([16]-[20]).

Fractional calculus is a branch of mathematics which concerns the possibility of taking arbitrary orders of the differential and integral operators. Actually, this branch attracted the attention of many researchers and engineers since long time ago (see [21]-[31]). Many systems in different fields may be described via fractional derivatives in which both integration and differentiation can be applied to any arbitrary order. For those who are interested in existence of solutions for fractional differential equations, they can see [32]-[35]. In this paper we adopt the basic definitions (Caputo) of fractional-order calculus:

Definition 1.*The fractional integral of order $\beta \in \mathbb{R}^+$ of the function $f(t)$, $t > 0$ is defined by*

$$I^{\beta} f(t) = \int_0^t \frac{(t-s)^{\beta-1}}{\Gamma(\beta)} f(s) ds,$$

* Corresponding author e-mail: samastars9@gmail.com

and the fractional derivative of order $\alpha \in (0,1)$ *of* $f(t)$, $t > 0$ *is defined by*

$$D^{\alpha} f(t) = I^{1-\alpha} \frac{df(t)}{dt}.$$

The following results are also of the main importance in fractional calculus. Let $\beta, \gamma \in \mathbb{R}^{+}$, $\alpha \in (0,1)$,
• $I_a^{\beta} : L^1 \to L^1$, and if $f(x) \in L^1$, then$I_a^{\gamma} I_a^{\beta} f(x) = I_a^{\gamma+\beta} f(x)$.
• $\lim_{\beta \to n} I_a^{\beta} f(x) = I_a^n f(x)$ uniformly on $[a,b]$, $n = 1,2,3,...$, where
$I_a^1 f(x) = \int_a^x f(s)ds$.
• $\lim_{\beta \to 0} I_a^{\beta} f(x) = f(x)$ weakly.
• If $f(x)$ is absolutely continuous on $[a,b]$, then $\lim_{\alpha \to 1} D_a^{\alpha} f(x) = \frac{df(x)}{dx}$.
The rest of the paper is organized as follows. Section 2 represents the formulation of the model for Influenza. In Section 3, we discuss the equilibria of the model and their local stability analysis. Section 4 investigates the analytical representation of bifurcations of the discretized model. Numerical simulations are carried out in Section 5. Finally, we conclude in Section 6.

2 Formulation of the Model

Kermack and McKendrick set a SIR model [36] to explain increasing and decreasing number of infective individuals observed in epidemics and is given by

$$\begin{aligned} \frac{dS}{dt} &= -\beta SI, \\ \frac{dI}{dt} &= \beta SI - \gamma I, \\ \frac{dR}{dt} &= \gamma I, \end{aligned} \tag{1}$$

where S denotes the susceptible number, I denotes the infectious number and R is the recovered individuals number, β is the rate of transmission , and γ is the rate of recovery. From a biological point of view, all parameters to be non-negative. Suppose that N denote the size of population. Obiviously,

$$\begin{aligned} &N = S + I + R, \quad and \\ &N^{\cdot} = S^{\cdot} + I^{\cdot} + R^{\cdot} = 0, \end{aligned}$$

where the "dots" denotes derivative w.r.t. time.
In model (1), both rate of birth and death are not considered while the rate of transmission β is considered as a constant. In this paper, the following assumptions are taken into account:
(1) Introduced a new susceptible at a constant rate of birth μ,
(2) The same constant birth rate μ is introduced to both the infectious and recovered individuals ,
(3) Constant death rate is introduced to the three classes of individuals and is equal to the birth rate μ.
From assumption (3), it is pretty clear that the population size is fixed. To make it simple, we set $N = 1$. The new SIR model reads:

$$\begin{aligned} \frac{dS}{dt} &= \mu - \mu S - \beta SI, \\ \frac{dI}{dt} &= \beta SI - (\mu + \gamma) I, \\ \frac{dR}{dt} &= \gamma I - \mu R. \end{aligned} \tag{2}$$

A significant difference between fractional-order models and their integer-order counterparts is that the first possess memory. In the the current paper, we will introduce the fractional-order derivative to model (2) with piecewise constant

arguments as follows

$$\begin{aligned}
\frac{d^\alpha S}{dt^\alpha} &= \mu - \mu S(r[\frac{t}{r}]) - \beta S(r[\frac{t}{r}])I(r[\frac{t}{r}]), \quad t \in (nr,(n+1)r], n = 0,1,2,..., \\
\frac{d^\alpha I}{dt^\alpha} &= \beta S(r[\frac{t}{r}])I(r[\frac{t}{r}]) - (\mu+\gamma)I(r[\frac{t}{r}]), \\
\frac{d^\alpha R}{dt^\alpha} &= \gamma I(r[\frac{t}{r}]) - \mu R(r[\frac{t}{r}]),
\end{aligned} \tag{3}$$

where $[.]$ denotes the greatest integer function, $0 < r < 1$ is the discretization step size, and $\alpha \in (0,1)$ is the fractional-order parameter. Most of biological processes may be described more suitably by discrete-time models rather than their continuous counterparts. Biological dynamics described via discrete systems should be meaningful: Influenza outbreaks occur during winter while the disease is somewhere else during summer. Now, we apply the discretization method represented in [37]-[40] and letting $t \to (n+1)r$, we end up with the discretized model

$$\begin{aligned}
S_{n+1} &= S_n + \frac{r^\alpha}{\Gamma(1+\alpha)}(\mu - \mu S_n - \beta S_n I_n), \\
I_{n+1} &= I_n + \frac{r^\alpha}{\Gamma(1+\alpha)}(\beta S_n I_n - (\mu+\gamma)I_n), \\
R_{n+1} &= R_n + \frac{r^\alpha}{\Gamma(1+\alpha)}(\gamma I_n - \mu R_n).
\end{aligned} \tag{4}$$

To analyze model (4), suppose that

$$S(0), I(0), R(0) \geq 0, \quad S(0) + I(0) + R(0) = 1. \tag{5}$$

Biologically, the solution of model (4)should be non-negative with initial values satisfying (5). This can be guaranteed if the two following inequalities hold:

$$\frac{r^\alpha}{\Gamma(1+\alpha)}(\beta+\mu) < 1, \quad \text{and} \quad \frac{r^\alpha}{\Gamma(1+\alpha)}(\mu+\gamma) < 1. \tag{6}$$

The two inequalities in (6) are essential demands for model (4). The first, $\frac{r^\alpha}{\Gamma(1+\alpha)}(\beta+\mu) < 1$, illustrates that the susceptible in percentage who get infected or die is less than one within a unit time. The second, $\frac{r^\alpha}{\Gamma(1+\alpha)}(\mu+\gamma) < 1$, shows that the infected individuals in percentage who get recovered or die is less than one within a unit time. Obliviously, those two inequalities ensure that $S(n), I(n), R(n) \geq 0$ for all $n \geq 0$ if (5) is satisfied.

Since the basic reproductive number $\mathfrak{R}_0$ is one of the essential concepts in mathematical biology. It is known as the average number of secondary infected individuals caused by a single infectious individual during their whole infectious lifetime. We would like to pay attention that $\mathfrak{R}_0$ is a dimensionless number and not a rate which would have units of $time^{-1}$. As it is mentioned in [41] about the importance of $\mathfrak{R}_0$ "*One of the foremost and most valuable ideas that mathematical thinking has brought to epidemic theory*". $\mathfrak{R}_0$ often used as a threshold that can predict whether a certain disease dies out or persists in a population. We calculate the basic reproductive number for model (4) following instructions in [42]-[44] which is given by $\mathfrak{R}_0 = \frac{\beta}{\mu+\gamma}$. In the next section, we analyze the local stability of equilibria of model (4) depending on $\mathfrak{R}_0$.

3 Equilibria and Local Stability

Rewrite model (4) in terms of $\mathfrak{R}_0$ we have

$$\begin{aligned}
S_{n+1} &= S_n + \frac{r^\alpha}{\Gamma(1+\alpha)}(\mu - \mu S_n - \beta S_n I_n), \\
I_{n+1} &= I_n + \frac{r^\alpha}{\Gamma(1+\alpha)}(\mu+\gamma)(\mathfrak{R}_0 S_n I_n - I_n), \\
R_{n+1} &= R_n + \frac{r^\alpha}{\Gamma(1+\alpha)}(\gamma I_n - \mu R_n).
\end{aligned} \tag{7}$$

Equilibria of the model (7) should satisfy the following set of algebraic equations

$$\begin{aligned}\mu-\mu S-\beta SI&=0,\\ \mathfrak{R}_0SI-I&=0,\\ \gamma I-\mu R&=0,\end{aligned}$$

where $\frac{r^\alpha}{\Gamma(1+\alpha)}>0$. It is clear that model (7) has two equilibria, the disease free equilibrium $E^0=(1,0,0)$ for all parameters values and a unique endemic equilibrium when $\mathfrak{R}_0>1$ given by $E^*=(\frac{1}{\mathfrak{R}_0},\frac{\mu(\mathfrak{R}_0-1)}{\beta},\frac{\gamma(\mathfrak{R}_0-1)}{\beta})$. The local asymptotic stability of the disease free equilibrium E^0 is discussed in the next theorem

Theorem 1.*The disease free equilibrium E^0 of model (4) is locally asymptotically stable if $0<\mathfrak{R}_0<1$, and E^0 is unstable if $\mathfrak{R}_0>1$.*

*Proof.*The local asymptotic stability of E^0 can be investigated by linearization. The Jacobian Matrix for model (4) is given by

$$J=\begin{pmatrix}1-K(\mu+\beta I) & -K\beta S & 0\\ K(\mu+\gamma)\mathfrak{R}_0I & 1+K(\mu+\gamma)(\mathfrak{R}_0S-1) & 0\\ 0 & K\gamma & 1-K\mu\end{pmatrix},$$

where $K=\frac{r^\alpha}{\Gamma(1+\alpha)}$. The jacobian matrix evaluated at E^0 is given by

$$J=\begin{pmatrix}1-K\mu & -K\beta & 0\\ 0 & 1+K(\mu+\gamma)(\mathfrak{R}_0-1) & 0\\ 0 & K\gamma & 1-K\mu\end{pmatrix}.$$

We look for the necessary and sufficient conditions for E^0 to have all the eigenvalues ; roots of the characteristic polynomial; less than one in modulus

$$F(\lambda)=\lambda^3-(A+B+C)\lambda^2+(AB+AC+BC+K\beta SD)\lambda-ABC-K\beta SDC,$$

where $A=1-K(\mu+\beta I)$, $B=1+K(\mu+\gamma)(\mathfrak{R}_0S-1)$, $C=1-K\mu$, and $D=K(\mu+\gamma)\mathfrak{R}_0I$. Following [45] the eigenvalues of the polynomial given above have to satisfy the following conditions:

1. $1-(A+B+C)+AB+AC+BC+K\beta SD-ABC-K\beta SDC>0$,

2. $1+A+B+C+AB+AC+BC+K\beta SD+ABC+K\beta SDC>0$,

3. $1-(AB+AC+BC+K\beta SD)+(A+B+C)(ABC+K\beta SDC)+(ABC+K\beta SDC)^2>0$,

4. $AB+AC+BC+K\beta SD<3$.

The eigenvalues associated to J evaluated at E^0 are $\lambda_{1,2}=1-K\mu$, and $\lambda_3=1+K(\mu+\gamma)(\mathfrak{R}_0-1)$. Now we have $|\lambda_{1,2}|<1$ if $0<K\mu<2$, while $|\lambda_3|<1$ if $0<K(\mu+\gamma)(\mathfrak{R}_0-1)<2$. The condition $\mu+\gamma<1$ together with $\mathfrak{R}_0<1$ guarantees that $|\lambda_i|<1$, $i=1,2,3$ and hence E^0 is locally asymptotic stable. If $\mathfrak{R}_0>1$, we will have $|\lambda_3|>1$ and hence E^0 is unstable.

Next, we discuss the stability of E^*.

Theorem 2.*E^* is locally asymptotically stable if $\mathfrak{R}_0>1$.*

*Proof.*Evaluating J at E^* gives

$$J=\begin{pmatrix}1-K\mu\mathfrak{R}_0 & \frac{-K\beta}{\mathfrak{R}_0} & 0\\ 1+\frac{K\mu}{\beta}(\mu+\gamma)(\mathfrak{R}_0-1) & 1 & 0\\ 0 & K\gamma & 1-K\mu\end{pmatrix}.$$

The characteristic equation of eigenvalues associated to J evaluated at E^* reads:

$$P(\lambda) = (1-K\mu-\lambda)((1-K\mu\Re_0-\lambda)(1-\lambda)+K^2\mu(\mu+\gamma)(\Re_0-1)) = 0, \tag{8}$$

with $\lambda_1 = 1-K\mu$, where $|\lambda_1| < 1$ if $0 < K\mu < 2$. As a matter of fact, to discuss the stability of E^*, we should study the roots of

$$\begin{aligned} F(\lambda) &= (1-K\mu\Re_0-\lambda)(1-\lambda)+K^2\mu(\mu+\gamma)(\Re_0-1), \\ &= \lambda^2+\lambda(K\mu\Re_0-2)+1-K\mu\Re_0+K^2\mu(\mu+\gamma)(\Re_0-1), \end{aligned} \tag{9}$$

as follows:
When $\Re_0 > 1$, we have

$$\begin{aligned} F(1) &= K^2\mu(\Re_0\mu+\Re_0\gamma-\mu-\gamma) \\ &= K^2(\Re_0\mu^2+\mu\Re_0\gamma-\mu^2-\mu\gamma) \\ &= K^2(\Re_0(\mu^2+\mu\gamma)-(\mu^2+\mu\gamma)) \\ &= K^2(\mu^2+\mu\gamma)(\Re_0-1) > 0. \end{aligned}$$

$$\begin{aligned} F(-1) &= 4-2K\mu\Re_0+K^2\mu(\mu+\gamma)\Re_0-K^2\mu(\mu+\gamma) \\ &= 4-K\mu\Re_0+K^2\mu^2\Re_0+K^2\mu\gamma\Re_0-K^2\mu^2-K^2\mu\gamma < 0. \end{aligned}$$

Now we are left with the constant term in $F(\lambda)$ which is given by
$C = 1-K\mu\Re_0+K^2\mu(\mu+\gamma)(\Re_0-1) < 1$. Thus, the Jury criteria is satisfied for the characteristic equation (9) which implies that $|\lambda_2| < 1$ and $|\lambda_3| < 1$. Hence, E^* is locally asymptotically stable.

Depending on λ_1, λ_2 and λ_3, the three roots of the jacobian matrix J, we have the following topological properties of the equilibria of model (7) as follows:
(1) If $|\lambda_1| < 1, |\lambda_2| < 1$ and $|\lambda_3| < 1$, then E(S, I, R) is a sink which is locally asymptotic stable;
(2) If $|\lambda_1| > 1, |\lambda_2| > 1$ and $|\lambda_3| > 1$, then E(S, I, R) is a source which is unstable;
(3) If $|\lambda_1| > 1, |\lambda_2| > 1$ and $|\lambda_3| < 1$ (or $|\lambda_1| < 1, |\lambda_2| > 1$ and $|\lambda_1 3| > 1$), then E(S, I, R) is a saddle which is unstable;
(4) If $|\lambda_1| = 1$ or $|\lambda_2| = 1$ or $|\lambda_3| = 1$, then E(S, I, R) is non-hyperbolic.

According to these definitions, we can classify equilibria of the model (7) topologically in the next propositions

Proposition 1. *E^0 has the following properties:*
E^0 is a sink if:
(i) $0 < K\mu < 2$, *and (ii)* $0 < K(\mu+\gamma)(\Re_0-1) < 2$.
E^0 is a source if:
(i) $K\mu > 2$ *and (ii)* $K(\mu+\gamma)(\Re_0-1) \in (-\infty,-2]\cup[0,\infty)$, *provided* $\Re_0 < 1$.
E^0 is a saddle if:
(i) $0 < K\mu < 2$, *(ii)* $K(\mu+\gamma)(\Re_0-1) > 0$, *and (iii)* $K(\mu+\gamma)(\Re_0-1) < -2$.
Or
(i) $K\mu > 2$, *and (ii)* $0 < K(\mu+\gamma)(\Re_0-1) < 2$.
E^0 is a non-hyperbolic if:
(i) $K\mu = 2$, *or (ii)* $\Re_0 = 1$.

Proposition 2. *The endemic equilibria E^* has the following properties:*
E^* is a sink if:
(i) $\Re_0(K\mu+K\gamma-1) < K(\mu+\gamma)$, *(ii)* $\Re_0(K\mu+K\gamma-2) < K+\gamma$, *and (iii)* $0 < K\mu < 2$.
E^* is a source if:
(i) $\Re_0(K\mu+K\gamma-2) < K+\gamma$, *(ii)* $\Re_0(K\mu+K\gamma-1) > K(\mu+\gamma)$, *and (iii)* $K\mu > 2$.
E^* is a saddle if and only if:
(i) $\Re_0(K\mu+K\gamma-1)K\mu < K^2(\mu+\gamma)-4$, *and (ii)* $0 < K\mu < 2$.
E^* is non-hyperbolic if:
(i) $\Re_0(K\mu+K\gamma-1) = K(\mu+\gamma)$, *(ii)* $4(\mu+\gamma) < \Re_0(4\gamma+3\mu)$, *and (iii)* $K\mu = 2$.

4 Analytical Representation of Bifurcations of the Discretized Model

This section investigates the analytical representation of bifurcations of the model (7) relaying on the bifurcation parameters. If λ_1, λ_2, and λ_3 are the roots of the characteristic equation

$$\lambda^3 + c_1\lambda^2 + c_2\lambda + c_3 = 0,$$

then as well known:

$$c_1 = -(\lambda_1+\lambda_2+\lambda_3), c_2 = \lambda_1\lambda_2+\lambda_1\lambda_3+\lambda_2\lambda_3, c_3 = -\lambda_1\lambda_2\lambda_3. \quad (10)$$

By the Von Neumann theorem of equilibria, $E(S,I,R)$ is asymptotically stable iff for all eigenvalues of J we have $|\lambda_i| < 1$, $i = 1,2,3$. The latter condition defines the domain of attraction of equilibria.

4.1 Critical bifurcation surfaces at E^0

For $E^0 = (1,0,0)$, we find that

$c_1 = 3K\mu + K\gamma - 3 - K\Re_0(\mu+\gamma)$,
$c_2 = 2 - K\gamma + K\Re_0(\mu+\gamma) + (1-K\mu)^2 + (1-K\mu)(1+K(\mu+\gamma)(\Re_0-1))$,
$-c_3 = (1-K\mu)^2(1+K(\mu+\gamma)(\Re_0-1))$.
Introduce the new parameters b_0, b_1, b_2, b_3 such that:
$b_0 = 1 + c_1 + c_2 + c_3$, $b_1 = 3 + c_1 - c_2 + 3c_3$, $b_2 = 3c_1 - c_2 + 3c_3$, $b_3 = 1 - c_1 + c_2 - c_3$. Thus we have
$b_0 = 3K\mu + (1-K\mu)(1+K\mu(1+K\mu(\mu+\gamma)(\Re_0-1))$,
$b_1 = 3K\mu + 2K\gamma - 2K\Re_0(\mu+\gamma) - 2 + (1-K\mu)(-1+K\mu+(2-3K\mu)1+K(\mu+\gamma)(\Re_0-1))$,
$b_2 = (4-3K\mu)(1-(1-K\mu)(1+K(\mu+\gamma)(\Re_0-1))) - (1-K\mu)^2$,
$b_3 = 6 - 3K\mu - 2K\gamma + 2K\Re_0(\mu+\gamma) + (1-K\mu)^2 + (1-K\mu)(2-K\mu)(1+K(\mu+\gamma)(\Re_0-1))$.

By constructing the Routh-Hurwitz matrix

$$\begin{pmatrix} b_1 & b_3 & 0 \\ b_0 & b_2 & 0 \\ 0 & b_1 & b_3 \end{pmatrix},$$

and its main minors
$\triangle_1 = b_1$, $\triangle_2 = b_1b_2 - b_0b_3$, $\triangle_3 = b_3\triangle_2$. The classical condition of asymptotic stability are
$b_0 > 0; b_1 > 0; b_2 > 0; b_3 > 0; b_1b_2 > b_0b_3$. The domain of attraction of E^0 is defined by three inequalities:
(1)$3K\mu + (1-K\mu)(1+K\mu(1+K\mu(\mu+\gamma)(\Re_0-1)) > 0$,

(2)$6 + 2K\Re_0(\mu+\gamma) + (1-K\mu)^2 + (1-K\mu)(2-K\mu)(1+K(\mu+\gamma)(\Re_0-1)) > 3K\mu + 2K\gamma$,

(3)$(3K\mu + 2K\gamma - 2K\Re_0(\mu+\gamma) - 2 + (1-K\mu)(-1+K\mu+(2-3K\mu)1+K(\mu+\gamma)(\Re_0-1)))((4-3K\mu)(1-(1-K\mu)(1+K(\mu+\gamma)(\Re_0-1))) - (1-K\mu)^2) > (3K\mu + (1-K\mu)(1+K\mu(1+K\mu(\mu+\gamma)(\Re_0-1)))(6 - 3K\mu - 2K\gamma + 2K\Re_0(\mu+\gamma) + (1-K\mu)^2 + (1-K\mu)(2-K\mu)(1+K(\mu+\gamma)(\Re_0-1)))$.

The domain of attraction has boundaries which are given by the two planes,

(i) $b_0 = 0 \Rightarrow (\mu+\gamma)(\Re_0-1) = \frac{2K\mu+1}{K^2\mu(K\mu-1)}$,

(ii) $b_3 = 0 \Rightarrow 6 + 2K\Re_0(\mu+\gamma) + (1-K\mu)^2 + (1-K\mu)(2-K\mu)(1+K(\mu+\gamma)(\Re_0-1)) = 3K\mu + 2K\gamma$,

and a saddle $(3K\mu + 2K\gamma - 2K\Re_0(\mu+\gamma) - 2 + (1-K\mu)(-1+K\mu+(2-3K\mu)1+K(\mu+\gamma)(\Re_0-1)))((4-3K\mu)(1-(1-K\mu)(1+K(\mu+\gamma)(\Re_0-1))) - (1-K\mu)^2) = (3K\mu + (1-K\mu)(1+K\mu(1+K\mu(\mu+\gamma)(\Re_0-1)))(6 - 3K\mu - 2K\gamma + 2K\Re_0(\mu+\gamma) + (1-K\mu)^2 + (1-K\mu)(2-K\mu)(1+K(\mu+\gamma)(\Re_0-1)))$.

In Figure (1)[46],the first plane intersects the saddle by the sides AD and BD; and the second plane intersects the saddle by the sides AC and BC; these two planes and the saddle will be called critical bifurcation surfaces.

From (10) we can write
$b_0 = (1-\lambda_1)(1-\lambda_2)(1-\lambda_3)$,

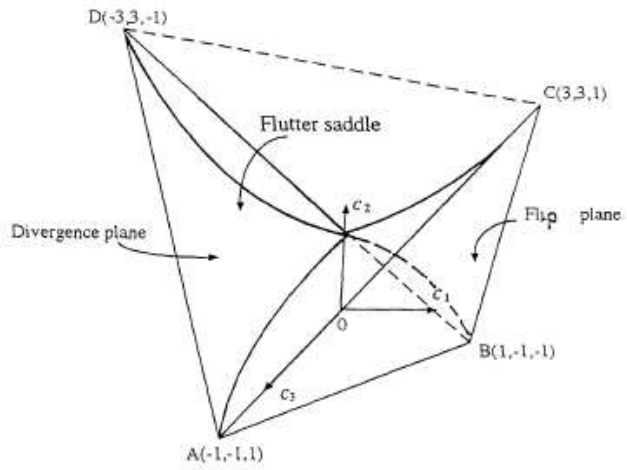

Fig. 1: Domain of attraction of model (7).

$b_3 = (1+\lambda_1)(1+\lambda_2)(1+\lambda_3)$.
Thus $b_0 = 0$ is a divergence plane because at least one of the eigenvalues is equal to 1. That is, the dynamics are divergent. On $b_3 = 0$ at least one of the eigenvalues is equal to 1, that is, the dynamics are oscillatory. Every point on the flip triangle ABC matches two-periodic cycle. The periodic doubling sequence is generated by the movements of the equilibria through it leading to chaos.

5 Numerical simulations

This section is mainly devoted to perform some numerical simulations to illustrate our analytical results and to reveal the complex dynamics of the discretized model (7) such as bifurcation, chaos, phase portrait. In all numerical simulations we take $r = 0.01, \mu = 1, \gamma = 1$, and $\beta = 3$.
With these parameters values, direct calculations give $\Re_0 = 1.5$. Figure (2) shows the trajectories of the discretized SIR model (7) for $\alpha = 0.80, 0.85, 0.90, 0.95$. When $\alpha \to 1$, the number of infected individuals increases compared with the case when $\alpha = 0.80$, while the number of recovered individuals decreases when $\alpha \to 1$. Figure (3) insures that taking α smaller than 1, the number of infected individuals is being decreased while the number of recovered individuals is being increased at the same time. Figure (4) shows the phase portraits of model (7) for different α. Theorem 1 and Theorem 2 imply that $E^0 = (1,0,0)$ is locally asymptotically stable when $\Re_0 < 1$ and unstable when $\Re_0 > 1$. On the contrary, $E^* = (0.6667, 0.1667, 0.1667)$ is asymptotically stable when $\Re_0 > 1$. This means that when $\Re_0 < 1$, every person contracts to the disease will infect less than one person before dying or recovering while when $\Re_0 > 1$, there will be disease outbreak. This is clearly shown in Figure (5) and Figure (6).

From the epidemiological point of view, we assume that the inequalities $\frac{r^\alpha}{\Gamma(1+\alpha)}(\beta+\mu) < 1$ and $\frac{r^\alpha}{\Gamma(1+\alpha)}(\mu+\gamma) < 1$ hold. If they are hold, then all solutions of model (7) with positive initial conditions are non-negative, then the numerical simulations show that model (7) does not show any complex dynamics at all. On the contrary, if they are not satisfied, then model (7) may exhibit more complex dynamics. The numerical simulations illustrate that there exists a period-doubling bifurcation sequence leads to chaos as shown in Figures (5) and (6). For small $\Re_0$ the endemic equilibrium is unique and locally asymptotically stable (see Figure (5)(a)). When $\Re_0 \simeq 99$, the endemic equilibrium losses its stability, and a stable periodic solution of period 2 appears (see Figure (5)d). When $\Re_0 \simeq 112$, the periodic solution of period 2 losses its stability, and a stable periodic solution of period 4 appears. If $\Re_0$ is increased further, the periodic solution of period 4 becomes unstable, and a periodic solution of period 8 appears. The numerical simulations illustrate that the period-doubling bifurcation may continue and go to chaos (see Figure (5)d)). From Figures (5) and (6), we can see that the chaotic behavior is being delayed when $\alpha = 0.95$ if compared with $\alpha = 0.85$.

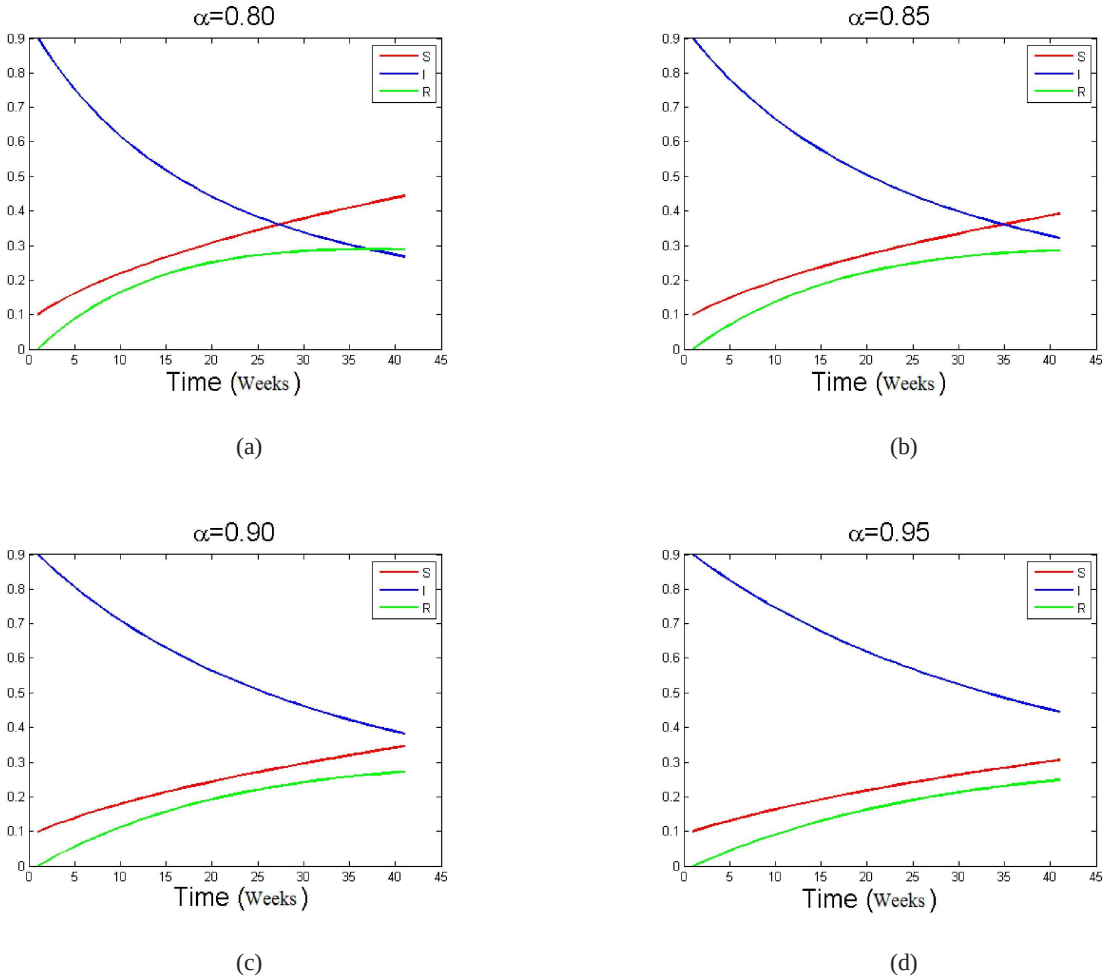

Fig. 2: Trajectories of model (7) for different α with initial conditions $(S_0, I_0, R_0) = (0.1, 0.9, 0)$

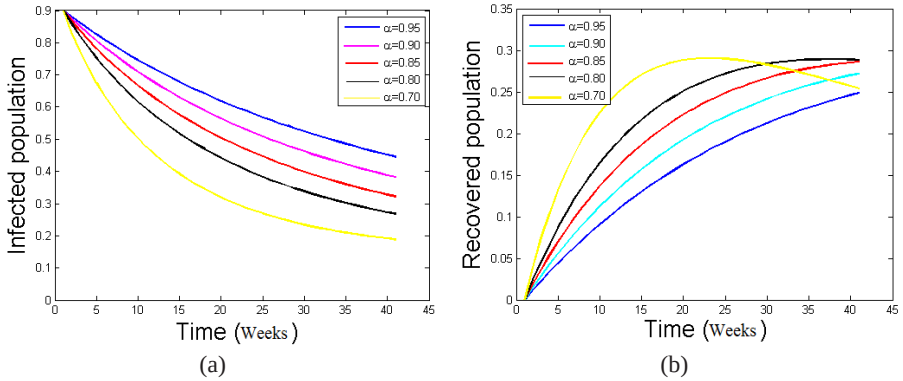

Fig. 3: (a)Infected individuals and (b) recovered individuals of (7) for different α

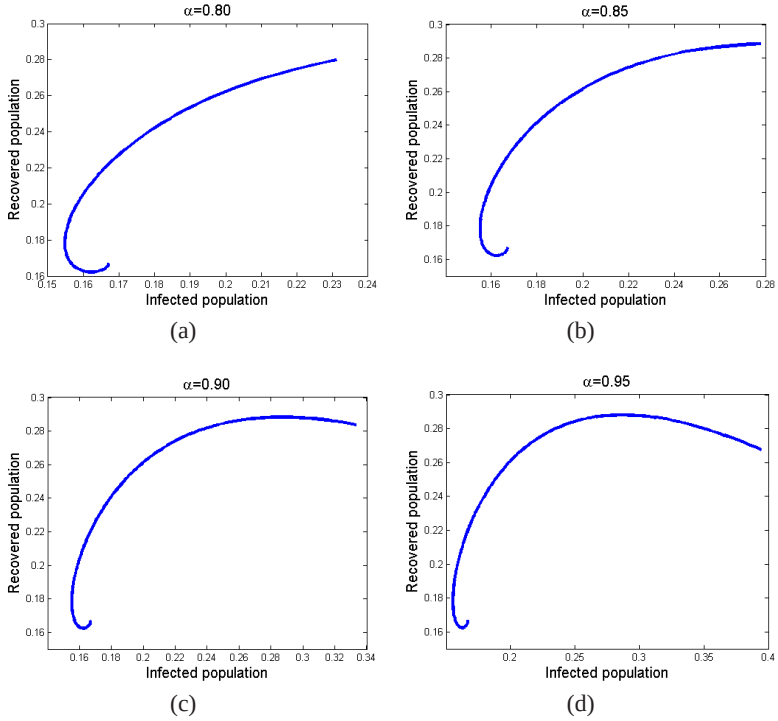

Fig. 4: Phase portraits of model (7) for different α

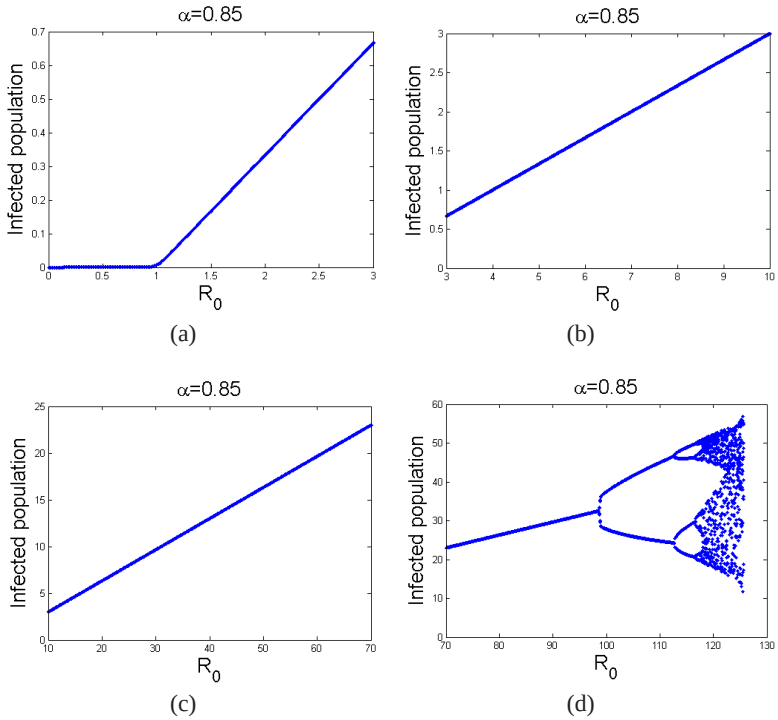

Fig. 5: Bifurcation diagram of model (7) as a function of $\Re_0$ with $\alpha = 0.85$

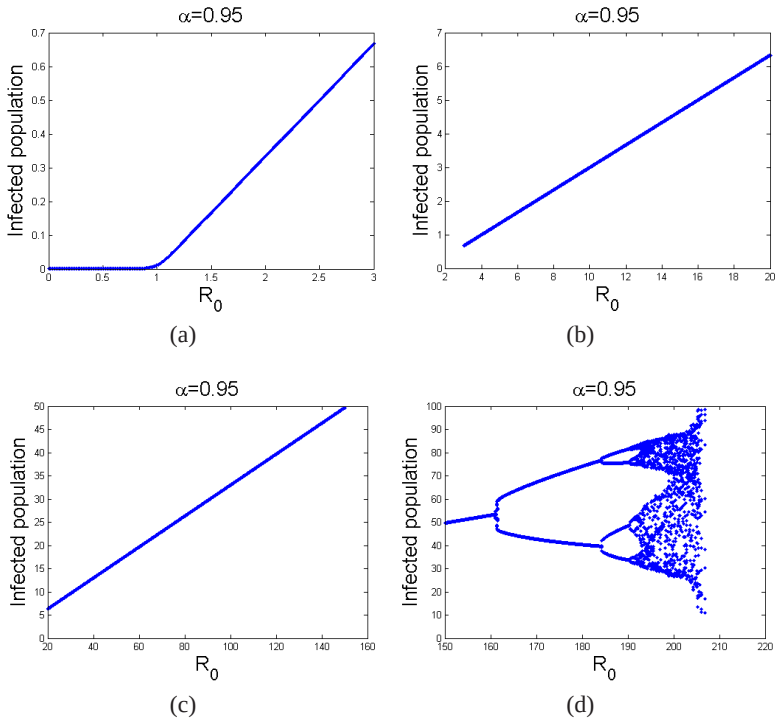

Fig. 6: Bifurcation diagram of model (7) as a function of $\mathfrak{R}_0$ with $\alpha = 0.95$

6 Conclusion

The asymptotic behavior of a discrete SIR epidemic model for Influenza A viruses have been considered. The discrete model is obtained by applying a discretization method for fractional-order differential equations with piecewise constant arguments. Explicit conditions for local asymptotic stability of the disease free equilibrium and endemic equilibrium are given. Equations and inequalities of critical bifurcation surfaces at the disease free equilibrium are given. Indeed, taking the fractional-order parameter $\alpha \to 1$, the number of infected individuals will be increased while reducing the value of α below 1, such as $\alpha = 0.95, 090, 85, 80$, gives better results. That is choosing $0 < \alpha < 1$ decreases the number of infected individuals and increases the number of recovered ones. In reality, α should be chosen to be not less than 0.80 to better simulate biological processes.

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