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# Time scale theory on stability of explicit and implicit discrete epidemic models: applications to Swine flu outbreak

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**Abstract**
Time scales theory has been in use since the 1980s with many applications. Only very recently, it has been used to describe within-host and between-hosts dynamics of infectious diseases. In this study, we present explicit and implicit discrete epidemic models motivated by the time scales modeling approach. We use these models to formulate the basic reproduction number, which determines whether an outbreak occurs or the disease dies out. We discuss the stability of the disease-free and endemic equilibrium points using the linearization method and Lyapunov function. Furthermore, we apply our models to swine flu outbreak data to demonstrate that the discrete models can accurately describe the epidemic dynamics. Our comparison analysis shows that the implicit discrete model can best describe the data regardless of the data frequency. In addition, we perform the sensitivity analysis on the key parameters of the models to study how these parameters impact the basic reproduction number.

**Keywords** SIR epidemic models · Difference equations · Positive equilibrium points · Local stability · Global stability · Lyapunov · Influenza outbreak in rural campus

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## 1 Introduction

Mathematical models in the form of differential equations have a long history of providing important contributions to the study of infectious diseases. One of the most frequently used model classes is based on SIR (susceptible-infected-recovered) type formulation, which describes the transmission dynamics of diseases. In the basic SIR model, the population is divided into three compartments as the susceptible, $S$, the infected, $I$, and the removed/recovered, $R$. A basic continuous SIR (C-SIR) model with susceptible recruitment and natural death rate similar to the one introduced by Kermack and McKendrick (1927) is:

$$\text{C-SIR Model: } \begin{cases} S^{'} = -\beta SI - \gamma S + \alpha, \\ I^{'} = \beta SI - (\gamma + \lambda)I, \\ R^{'} = \lambda I - \gamma R. \end{cases} \tag{1}$$

In this model, the susceptible individuals are infected upon successful contact with infected individuals at the transmission rate $\beta$ per individual per unit time. The infected individuals are recovered from the disease at the per capita recovery rate $\lambda$ per unit time. The parameters $\alpha$ and $\gamma$ represent the recruitment rate of susceptible individuals per unit time and the per capita death rate of the population per unit time, respectively. Many variants of the incidence rate $\beta SI$ have been considered, including a nonlinear form $\frac{\beta SI}{1+\alpha I}$ as in Capasso and Serio (1978) and the form $\frac{\beta S(t)I(t-\tau)}{1+\alpha I(t-\tau)}$ as in Cooke (1979); McCluskey (2010a); McCluskey (2010b). Here, $\tau$ indicates the time delay required by the infected individuals from the time they contract pathogens to the time they become infectious. The global stability analysis of these models have been extensively studied, including the one by McCluskey (2010b), who considered the system with $\frac{\beta S(t)I(t-\tau)}{1+\alpha I(t-\tau)}$ incidence rate and established the global stability analysis of the equilibrium by constructing Lyapunov functions.

The continuous SIR models are often validated using the data-fitting process, which provides reasonable estimates to the model parameters. However, the data are discrete in general, quite often collected with varying time intervals. In this regard, the discrete models may be suitable to recover parameters from the discrete data sets. While some other continuous and discrete SIR models have been investigated (Allen 1994; Jang and Elaydi 2003; Saito 2016; Kermack and McKendrick 1927; Enatsu et al. 2010, 2012), including some for the transmission dynamics of H1N1 (Kim et al. 2017, 2020; Lee et al. 2021; Tan et al. 2012; Vaidya et al. 2015), the application and analysis of time-scale based discrete models in the context of infectious diseases are not well-advanced yet.

In Akın and Yeni (2020), the unification of continuous and discrete models of SIS (susceptible-infected-susceptible) is formulated on an arbitrary closed subset of real numbers, so-called a time scale. The discrete SIS model in Akın and Yeni (2020) is given as a system of two nonlinear dynamic equations, and the exact solution is derived

by the approach of the Bernoulli equation on time scales (Akın-Bohner and Bohner 2003). Motivated by Akın and Yeni (2020), we consider two variants of the discrete SIR model, one with the explicit formulation and another with the implicit formulation. In the explicit formulation, we assume that the incidence rates are calculated based on the population interaction in the previous time. The explicit discrete SIR (ED-SIR) model we introduce takes the following form:

$$\text{ED-SIR Model:} \begin{cases} \Delta S_n = -\dfrac{\beta(1-\gamma)}{1+\beta I_n} S_n I_n - \gamma S_n + \alpha, \\ \Delta I_n = \dfrac{\beta(1-\gamma)}{1+\beta I_n} S_n I_n - (\gamma+\lambda) I_n, \\ \Delta R_n = \lambda I_n - \gamma R_n, \end{cases} \tag{2}$$

with initial conditions $S_0 > 0$, $I_0 > 0$ and $R_0 \geq 0$. The numbers of susceptible, infected, and recovered individuals for $n \geq 0$ are denoted by $S_n$, $I_n$, and $R_n$, respectively. We assume that all parameters are positive and $\gamma < 1$.

In the implicit formulation, the incidence rate is computed according to the population interaction at the present time. This assumption allows us to develop the following implicit discrete SIR (ID-SIR) model:

$$\text{ID-SIR Model:} \begin{cases} \Delta S_n = -\dfrac{\beta(1-\gamma)}{1+\beta I_{n+1}} S_{n+1} I_{n+1} - \gamma S_{n+1} + \alpha, \\ \Delta I_n = \dfrac{\beta(1-\gamma)}{1+\beta I_{n+1}} S_{n+1} I_{n+1} - (\gamma+\lambda) I_{n+1}, \\ \Delta R_n = \lambda I_{n+1} - \gamma R_{n+1}. \end{cases} \tag{3}$$

We begin by introducing the preliminary results for the system of difference equations and discrete stability analysis and refer readers to two books introducing the theory of time scales by Bohner and Peterson (2001, 2003). In Sect. 3, we derive disease-free equilibrium (DFE) and endemic equilibrium (EE) points and compute basic reproduction number ($\mathscr{R}_0$ ) for the ED-SIR model (2). We note that ED-SIR model (2) and ID-SIR model (3) have the same equilibrium points and the same $\mathscr{R}_0$ formulation. In Sects. 4 and 5, we discuss the necessary conditions for their local and global stability analysis for the ED-SIR model (2) and ID-SIR model (3). In addition, we consider the swine flu (H1N1 influenza) data in a rural university campus and study how these discrete models (2) and (3) are different from continuous model (1) to describe this data set (see Sect. 6). In the last section, we conclude our results, discuss the importance of the theory of time scales, and share some open problems.

## 2 Preliminaries

In this paper, we discuss the stability analysis of ED-SIR and ID-SIR models, (2) and (3), respectively. Therefore, we first present some necessary definitions and results related to stability theory from the books written by Elaydi (2005) and Kelley and Peterson (2001).

The following system of $m$ linear equations:

$$\begin{aligned} x_1(n+1) &= a_{11}x_1(n) + a_{12}x_2(n) + \cdots + a_{1m}x_m(n) \\ x_2(n+1) &= a_{21}x_1(n) + a_{22}x_2(n) + \cdots + a_{2m}x_m(n) \\ &\vdots \qquad\quad \vdots \qquad\quad \vdots \qquad\quad \vdots \\ x_m(n+1) &= a_{m1}x_1(n) + a_{m2}x_2(n) + \cdots + a_{mm}x_m(n) \end{aligned}$$

may be written in the vector form

$$x(n+1) = Ax(n), \tag{4}$$

where $x(n) = (x_1(n), x_2(n), \cdots, x_m(n))^T \in \mathbb{R}^m$, and $A = (a_{ij})$ is an $m \times m$ real nonsingular matrix. System (4) is considered autonomous or time-invariant, since the values of $A$ are all constants. The spectral radius of $A$ is defined as

$$r(A) = \max\{|\xi| : \xi \text{is an eigenvalue of A}\}.$$

We consider the vector difference equation

$$x(n+1) = f(x(n)), \tag{5}$$

with $x(n_0) = x_0$, where $x(n) \in \mathbb{R}^k$, $f : \mathbb{R}^k \to \mathbb{R}^k$ is continuous. A point $x^*$ in $\mathbb{R}^k$ is called an equilibrium point of (4) if $f(x^*) = x^*$ for all $n \geq n_0$ and is classified as follows:

**Definition 1** ((Elaydi 2005), Definition 4.2) The equilibrium point $x^*$ of (5) is said to be:

(i) Stable if given $\epsilon > 0$ and $n_0 \geq 0$, there exists $\delta = \delta(\epsilon, n_0)$ such that $||x_0 - x^*|| < \delta$ implies $||x(n, n_0, x_0) - x^*|| < \epsilon$ for all $n \geq n_0$.
(ii) Attracting if there exists some constant $\mu = \mu(n_0)$ such that $||x(n, n_0, x_0) - x^*|| < \mu$ implies $\lim_{n\to\infty} x(n, n_0, x_0) = x^*$.
(iii) Asymptotically stable if it is stable and attracting.
(iv) Globally asymptotically stable if $\mu = \infty$ in parts (ii) and (iii).

The next theorem summarizes the main stability results for the linear autonomous system (4).

**Theorem 1** *((Elaydi 2005), Theorem 4.13) The following statements hold:*

*(i) The zero solution of (4) is stable if and only if $r(A) \leq 1$ and the eigenvalues of unit modulus are semisimple, i.e., if the corresponding Jordan block is diagonal.*

*(ii) The zero solution of (4) is asymptotically stable if and only if* $r(A) < 1$.

For two dimensional systems, if

$$|\mathrm{tr}A| < 1 + \det A < 2 \tag{6}$$

holds, then the zero solution of (4) is asymptotically stable, see Elaydi (2005).

Let $x^*$ be an equilibrium point of $f$ in (4). A real-valued continuous function $V$ on some ball $B$ about $x^*$ is called a "Lyapunov function" for $f$ at $x^*$ provided $V(x^*) = 0$, $V(x) > 0$ for $x \neq x^*$ in $B$, and

$$\Delta_n V(x) \equiv V(f(x)) - V(x) \leq 0 \tag{7}$$

for all $x$ in $B$. If the inequality (7) is strict for $x \neq x^*$, then $V$ is a "strict Lyapunov function". We have the following theorem, which plays an important role in showing the global stability of an equilibrium of the system of autonomous difference equations.

**Theorem 2** *(Lyapunov Stability Theorem) Let* $x^*$ *be a equilibrium point of* $f$*, and assume* $f$ *is continuous on some ball about* $x^*$*. If there is a Lyapunov function for* $f$ *at* $x^*$*, then* $x^*$ *is stable. If there is a strict Lyapunov function for* $f$ *at* $x^*$*, then* $x^*$ *is asymptotically stable. Moreover, if* $V(x) \to \infty$ *as* $||x|| \to \infty$*, then* $x^*$ *is globally asymptotically stable.*

## 3 Equilibrium points and the basic reproduction number

In this section, we derive equilibrium points and $\mathscr{R}_0$ of ED-SIR and ID-SIR models (2) and (3), respectively. The first two equations of ED-SIR epidemic model (2) can be rewritten as

$$\begin{cases} S_{n+1} = -\dfrac{\beta(1-\gamma)}{1+\beta I_n} S_n I_n + (1-\gamma) S_n + \alpha \\ I_{n+1} = \dfrac{\beta(1-\gamma)}{1+\beta I_n} S_n I_n + (1-\gamma-\lambda) I_n. \end{cases} \tag{8}$$

It is clear that the equilibrium solution of (2) has

$$S^* = \frac{\alpha + \alpha\beta I^*}{\gamma + \beta I^*}. \tag{9}$$

From the second equation of (2), one can get $\beta(1-\gamma)S^*I^* + (1-\gamma-\lambda)I^*(1+\beta I^*) = (1+\beta I^*)I^*$. Simplification yields $I^*(\beta I^*(\gamma+\lambda) + \gamma + \lambda - \beta S^* + \beta\gamma S^*) = 0$. Therefore,

$$I^* = 0 \quad \text{or} \quad \beta I^*(\gamma+\lambda) + \gamma + \lambda - \beta S^* + \beta\gamma S^* = 0. \tag{10}$$

Now we want to solve (9) and (10). If $I^* = 0$, then $S^* = \frac{\alpha}{\gamma}$. If $\beta I^*(\gamma + \lambda) + \gamma + \lambda - \beta S^* + \beta\gamma S^* = 0$, then

$$I^{*2} + \left(\frac{\gamma + 1}{\beta} + \frac{\alpha(\gamma - 1)}{\gamma + \lambda}\right) I^* + \left(\frac{\gamma}{\beta^2} + \frac{\alpha(\gamma - 1)}{\beta(\gamma + \lambda)}\right) = 0. \tag{11}$$

Solving the algebraic equation (11) gives

$$I^*_{1,2} = \frac{-\left(\frac{\gamma+1}{\beta} + \frac{\alpha(\gamma-1)}{\gamma+\lambda}\right) \pm \sqrt{\left(\frac{\gamma+1}{\beta} + \frac{\alpha(\gamma-1)}{\gamma+\lambda}\right)^2 - 4\left(\frac{\gamma}{\beta^2} + \frac{\alpha(\gamma-1)}{\beta(\gamma+\lambda)}\right)}}{2}.$$

To find $I^*_1$ and $I^*_2$ values, the expression in the square root needs to be simplified as follows

$$\begin{aligned}\left(\frac{\gamma + 1}{\beta} + \frac{\alpha(\gamma - 1)}{\gamma + \lambda}\right)^2 - 4\left(\frac{\gamma}{\beta^2} + \frac{\alpha(\gamma - 1)}{\beta(\gamma + \lambda)}\right) &= (\gamma - 1)^2\left[\frac{1}{\beta^2} + \frac{2\alpha}{\beta(\gamma + \lambda)} + \frac{\alpha^2}{(\gamma + \lambda)^2}\right] \\ &= (\gamma - 1)^2\left[\frac{1}{\beta} + \frac{\alpha}{\gamma + \lambda}\right]^2.\end{aligned}$$

Using the fact that $\gamma < 1$, we get

$$I^*_{1,2} = \frac{-\left(\frac{\gamma+1}{\beta} + \frac{\alpha(\gamma-1)}{\gamma+\lambda}\right) \pm (1 - \gamma)\left[\frac{1}{\beta} + \frac{\alpha}{\gamma+\lambda}\right]}{2}.$$

Hence,

$$I^*_1 = -\frac{\gamma}{\beta} + (1 - \gamma)\frac{\alpha}{\gamma + \lambda} \quad \text{and} \quad I^*_2 = -\frac{1}{\beta}.$$

If $I^* = I^*_1$, then substituting $I^*$ into (9) gives $S^* = \frac{\alpha+\alpha\beta I^*}{\gamma+\beta I^*} = \frac{\gamma+\lambda}{\beta} + \alpha$ immediately. Therefore, ED-SIR model (2) with initial conditions has a disease free equilibrium $E_0 = (S^*_0, I^*_0, R^*_0)$, where

$$S^*_0 = \frac{\alpha}{\gamma}, \quad I^*_0 = 0, \quad \text{and} \quad R^*_0 = 0 \tag{12}$$

and a positive endemic equilibrium $E^+ = (S^*, I^*, R^*)$, where

$$\begin{aligned} S^* &= \frac{\gamma + \lambda}{\beta} + \alpha, \quad I^* = -\frac{\gamma}{\beta} + (1 - \gamma)\frac{\alpha}{\gamma + \lambda}, \\ R^* &= \frac{\lambda}{\gamma}\left(-\frac{\gamma}{\beta} + (1 - \gamma)\frac{\alpha}{\gamma + \lambda}\right). \end{aligned} \tag{13}$$

Note that DFE and EE points of ID-SIR model (3) are equivalent to DFE and EE points of ED-SIR model (2) and are given as in (12) and (13), respectively. Now we define the threshold index, called the basic reproduction number, which helps us establish the stability of discrete models. For our models, we define the basic reproduction number as

$$\mathscr{R}_0 = \frac{(1-\gamma)\alpha\beta}{\gamma} + 1 - (\gamma + \lambda). \tag{14}$$

Note that $I^* = \frac{\gamma}{\beta(\gamma+\lambda)}(\mathscr{R}_0 - 1)$ and $R^* = \frac{\lambda}{\beta(\gamma+\lambda)}(\mathscr{R}_0 - 1)$. Therefore, the endemic equilibrium exists if and only if $\mathscr{R}_0 > 1$. We note that for our time-scale-based discrete model formulation, the basic reproduction number, $\mathscr{R}_0$, may differ from the general basic reproduction number defined in an epidemiological context and/or continuous model. The basic reproduction number derived here should be interpreted as a critical threshold for stability.

We now analyze the stability of the equilibria of ED-SIR model (2) and ID-SIR model (3) in the next sections based on the basic reproduction number (14).

## 4 Local stability of equilibrium points of ED-SIR model (2)

In this section, we show that if $\mathscr{R}_0 < 1$, then all solutions of ED-SIR model (2) approach DFE as in (12). For the proof, it is sufficient to consider system (8).

**Theorem 3** *If $\mathscr{R}_0 < 1$, then the disease free equilibrium $E_0$ of ED-SIR model (2) is locally asymptotically stable, and if $\mathscr{R}_0 > 1$, then $E_0$ is unstable.*

***Proof*** The Jacobian matrix for the variables of system (8) is

$$J(S, I) = \begin{bmatrix} \frac{1-\gamma}{1+\beta I} & \frac{\beta(\gamma-1)S}{(1+\beta I)^2} \\ \frac{\beta(1-\gamma)I}{1+\beta I} & \frac{\beta(1-\gamma)S}{(1+\beta I)^2} + 1 - \gamma - \lambda \end{bmatrix}. \tag{15}$$

For the disease free equilibrium $(S_0^*, 0)$ of system (8), the Jacobian matrix is given by

$$J(S_0^*, 0) = \begin{bmatrix} 1-\gamma & \frac{(\gamma-1)\alpha\beta}{\gamma} \\ 0 & \frac{(1-\gamma)\alpha\beta}{\gamma} + 1 - \gamma - \lambda \end{bmatrix}$$

whose eigenvalues are

$$\xi_1 = 1 - \gamma \quad \text{and} \quad \xi_2 = \frac{(1-\gamma)\alpha\beta}{\gamma} + 1 - \gamma - \lambda. \tag{16}$$

It follows that if $\mathscr{R}_0 < 1$, then $|\xi_1| < 1$ and $|\xi_2| < 1$. Therefore, $(S_0^*, 0)$ is locally asymptotically stable. If $\mathscr{R}_0 > 1$, then $|\xi_2| > 1$ and thus $(S_0^*, 0)$ is unstable. Now one

can consider ED-SIR model (2), where $R_{n+1} = \lambda I_n + (1-\gamma)R_n$. In this case, the Jacobian matrix for $E_0$ is given by

$$J(E_0 = (S_0^*, 0, 0)) = \begin{bmatrix} 1-\gamma & \frac{(\gamma-1)\alpha\beta}{\gamma} & 0 \\ 0 & \frac{(1-\gamma)\alpha\beta}{\gamma} + 1 - \gamma - \lambda & 0 \\ 0 & \lambda & 1-\gamma \end{bmatrix}$$

whose eigenvalues are $\xi_1, \xi_2$ given as in (16), and $\xi_3 = 1-\gamma$. Since $\gamma < 1$, we have $|\xi_3| < 1$. Therefore, if $\mathscr{R}_0 < 1$, then $E_0$ is locally asymptotically stable, and unstable if $\mathscr{R}_0 > 1$ by Theorem 1. Hence, the proof is completed. □

Following similar steps as in Theorem 3, one can also show that if $\mathscr{R}_0 > 1$, then all solutions of ED-SIR model (2) approach $E^+ = (S^*, I^*, R^*)$ as in (13). Note that one can get the DFE, i.e., $S_0^* = \frac{\alpha}{\gamma}$, $I_0^* = 0$, and $R_0^* = 0$ if $\mathscr{R}_0 = 1$.

**Theorem 4** *If $\mathscr{R}_0 > 1$, then the endemic equilibrium point $E^+$ of ED-SIR model (2) is locally asymptotically stable.*

***Proof*** Assume $\mathscr{R}_0 > 1$. In the proof of Theorem 3, the Jacobian matrix for the variables of system (8) is computed as in (15). Hence, for the endemic equilibrium $(S^*, I^*)$, the Jacobian matrix is

$$\begin{aligned} J(S^*, I^*) &= \begin{bmatrix} \frac{1-\gamma}{1+\beta I^*} & \frac{\beta(\gamma-1)S^*}{(1+\beta I^*)^2} \\ \frac{\beta(1-\gamma)I^*}{1+\beta I^*} & \frac{\beta(1-\gamma)S^*}{(1+\beta I^*)^2} + 1 - \gamma - \lambda \end{bmatrix} \\ &= \begin{bmatrix} \frac{\gamma+\lambda}{\gamma+\lambda+\alpha\beta} & -\frac{(\gamma+\lambda)^2}{(1-\gamma)(\gamma+\lambda+\alpha\beta)} \\ -\gamma + \frac{\alpha\beta}{\gamma+\lambda+\alpha\beta} & \frac{(\gamma+\lambda)^2}{(1-\gamma)(\gamma+\lambda+\alpha\beta)} + 1 - \gamma - \lambda \end{bmatrix}. \end{aligned}$$

To show that $(S^*, I^*)$ is locally asymptotically stable, condition (6) needs to be held for $J(S^*, I^*)$, i.e.,

$$\left|\mathrm{tr}J(S^*, I^*)\right| < 1 + \det J(S^*, I^*) < 2. \tag{17}$$

First, note that

$$\begin{aligned} \det J(S^*, I^*) &= \frac{(\gamma+\lambda)^2}{(1-\gamma)(\gamma+\lambda+\alpha\beta)} \left( \frac{\gamma+\lambda}{\gamma+\lambda+\alpha\beta} + \frac{\alpha\beta}{\gamma+\lambda+\alpha\beta} - \gamma \right) \\ &\quad + \frac{\gamma+\lambda}{\gamma+\lambda+\alpha\beta}(1-\gamma-\lambda) \\ &= \frac{(\gamma+\lambda)^2}{\gamma+\lambda+\alpha\beta} + \frac{\gamma+\lambda}{\gamma+\lambda+\alpha\beta}(1-\gamma-\lambda) \end{aligned}$$

$$= \frac{\gamma + \lambda}{\gamma + \lambda + \alpha\beta}. \tag{18}$$

The assumption $\mathscr{R}_0 > 1$ implies that

$$\frac{\gamma + \lambda}{(1-\gamma)(\gamma + \lambda + \alpha\beta)} - 1 < 0. \tag{19}$$

Therefore,

$$\begin{aligned} \left|\mathrm{tr}J(S^*, I^*)\right| &= \left| \frac{\gamma + \lambda}{\gamma + \lambda + \alpha\beta} + \frac{(\gamma + \lambda)^2}{(1-\gamma)(\gamma + \lambda + \alpha\beta)} + 1 - (\gamma + \lambda) \right| \\ &= 1 + \frac{\gamma + \lambda}{\gamma + \lambda + \alpha\beta} + (\gamma + \lambda)\left( \frac{\gamma + \lambda}{(1-\gamma)(\gamma + \lambda + \alpha\beta)} - 1 \right) \\ &< 1 + \det J(S^*, I^*) \end{aligned} \tag{20}$$

by (18) and (19). Furthermore,

$$1 + \det J(S^*, I^*) = 1 + \frac{\gamma + \lambda}{\gamma + \lambda + \alpha\beta} = 1 + \frac{1}{1 + \frac{\alpha\beta}{\gamma + \lambda}} < 2. \tag{21}$$

Hence, (17) holds from (20) and (21). For system (2), the characteristic equation is

$$\begin{aligned} &\det(J(E^+ = (S^*, I^*, R^*)) - rI_{3\times 3}) \\ &= \begin{vmatrix} \frac{\gamma+\lambda}{\gamma+\lambda+\alpha\beta} & -\frac{(\gamma+\lambda)^2}{(1-\gamma)(\gamma+\lambda+\alpha\beta)} & 0 \\ -\gamma + \frac{\alpha\beta}{\gamma+\lambda+\alpha\beta} & \frac{(\gamma+\lambda)^2}{(1-\gamma)(\gamma+\lambda+\alpha\beta)} + 1 - \gamma - \lambda & 0 \\ 0 & -\lambda & 1 - \gamma - r \end{vmatrix} \\ &= \det(J((S^*, I^*)) - rI_{2\times 2}) \times (1 - \gamma - r) = 0, \end{aligned}$$

where $I_{i\times i}$ is the $i \times i$ unit matrix for $i = 2, 3$. We already have the conditions to be held for $r_1$ and $r_2$ of $\det(J((S^*, I^*)) - rI_{2\times 2})$. Since $r_3 = 1 - \gamma$ follows from the characteristic equation, we finally get $|r_1| < 1$, $|r_2| < 1$, and $|r_3| < 1$ by (17). Following from Theorem 1, the endemic equilibrium $E^+$ is locally asymptotically stable if $\mathscr{R}_0 > 1$. □

## 5 Global stability of the endemic equilibrium for ID-SIR model (3)

In this section, we show the global stability of the endemic equilibrium $E^+$ of ID-SIR model (3) by using a suitable Lyapunov function.

**Theorem 5** *If $\mathscr{R}_0 > 1$, then the endemic equilibrium $E^+$ of ID-SIR model (3) is globally asymptotically stable.*

***Proof*** Let $f(x) = \frac{x}{1+\beta x}$ and $g(x) = x - 1 - \ln x$, $x > 0$. It is clear that $g$ has minimum at $x = 1$ such that $g(1) = 0$ and $g(x) \geq 0$ for $x > 0$. We define the following Lyapunov function

$$V_n = V(S_n, I_n, R_n) = \frac{1}{\beta(1-\gamma)f(I_0^*)} V_{S_n} + \frac{I^*}{\beta(1-\gamma)S^* f(I^*)} V_{I_n}, \tag{22}$$

where

$$V_{S_n} = g\left(\frac{S_n}{S^*}\right), \quad V_{I_n} = g\left(\frac{I_n}{I^*}\right). \tag{23}$$

Now we calculate $\Delta V_{S_n}$ and $\Delta V_{I_n}$ in order to show $\Delta V_n < 0$.

$$\begin{aligned}
\Delta V_{S_n} &= g\left(\frac{S_{n+1}}{S^*}\right) - g\left(\frac{S_n}{S^*}\right) \\
&= \frac{S_{n+1} - S_n}{S^*} + \ln\frac{S_n}{S_{n+1}} \\
&\leq (S_{n+1} - S_n)\left(\frac{S_{n+1} - S^*}{S^* S_{n+1}}\right) \\
&= -\gamma\frac{(S_{n+1} - S^*)^2}{S^* S_{n+1}} - \beta(1-\gamma)f(I^*)\left(\frac{S_{n+1} f(I_{n+1})}{S^* f(I^*)} - 1\right)\left(1 - \frac{S^*}{S_{n+1}}\right),
\end{aligned} \tag{24}$$

where we use $\ln(1-x) \leq -x$ for $x < 1$ and replace $\alpha$ by $\beta(1-\gamma)S^* f(I^*) + \gamma S^*$. Similarly,

$$\begin{aligned}
\Delta V_{I_n} &= g\left(\frac{I_{n+1}}{I^*}\right) - g\left(\frac{I_n}{I^*}\right) \\
&= \frac{I_{n+1} - I_n}{I^*} + \ln\frac{I_n}{I_{n+1}} \\
&\leq (I_{n+1} - I_n)\left(\frac{I_{n+1} - I^*}{I^* I_{n+1}}\right) \\
&= \frac{\beta(1-\gamma)S^* f(I^*)}{I^*}\left(\frac{S_{n+1} f(I_{n+1})}{S^* f(I^*)} - \frac{I_{n+1}}{I^*}\right)\left(1 - \frac{I^*}{I_{n+1}}\right),
\end{aligned} \tag{25}$$

since $(\gamma + \lambda)I^* = \beta S^* f(I^*)$. Therefore, from (24) and (25)

$$\begin{aligned}
\Delta V_n &= V_{n+1} - V_n \\
&\leq -\gamma\frac{(S_{n+1} - S^*)^2}{\beta(1-\gamma)f(I^*)S^* S_{n+1}} - \left(\frac{S_{n+1} f(I_{n+1})}{S^* f(I^*)} - 1\right)\left(1 - \frac{S^*}{S_{n+1}}\right)
\end{aligned}$$

$$+\left(\frac{S_{n+1}f(I_{n+1})}{S^*f(I^*)}-\frac{I_{n+1}}{I^*}\right)\left(1-\frac{I^*}{I_{n+1}}\right). \tag{26}$$

For simplicity, let $x_{n+1}=\frac{S_{n+1}}{S^*}$, $y_{n+1}=\frac{I_{n+1}}{I^*}$ and $F(y_{n+1})=\frac{f(I_{n+1})}{f(I^*)}$. Then, (26) becomes

$$\Delta V_n \le -\gamma\frac{(S_{n+1}-S^*)^2}{\beta(1-\gamma)f(I^*)S^*S_{n+1}}+F(y_{n+1})-\frac{x_{n+1}}{y_{n+1}}F(y_{n+1})-\frac{1}{x_{n+1}}-y_{n+1}+2. \tag{27}$$

Adding and subtracting $\ln\frac{x_{n+1}}{y_{n+1}}F(y_{n+1})$ in (27) yield

$$\begin{aligned}\Delta V_n \le -\gamma\frac{(S_{n+1}-S^*)^2}{\beta(1-\gamma)f(I^*)S^*S_{n+1}}-g\left(\frac{1}{x_{n+1}}\right)-g\left(\frac{x_{n+1}}{y_{n+1}}F(y_{n+1})\right)\\ +F(y_{n+1})-y_{n+1}+\ln y_{n+1}-\ln F(y_{n+1}).\end{aligned} \tag{28}$$

Let $h(z)=F(z)-z+\ln z-\ln F(z)$, where $z=y_{n+1}$. Then, $h(1)=0$ and $h'(z)=(1-z)\left(\frac{2\beta I^*+(\beta I^*)^2 z}{(1+\beta I^* z)^2}\right)$. Hence, $h'(z)>0$ if $z<1$ and $h'(z)<0$ if $z>1$.

From the above discussion and the fact that $g$ is nonnegative, if $(S_{n+1},I_{n+1})=(S^*,I^*)$, then $h=0$ and $\Delta V_n=0$. If $(S_{n+1},I_{n+1})\neq(S^*,I^*)$, then $h<0$ and hence $\Delta V_n<0$ for any $n\ge 0$ from (28). Since $V$ is a monotone decreasing sequence, $\lim_{n\to\infty}V_n\ge 0$ and $\lim_{n\to\infty}(V_{n+1}-V_n)=0$. Therefore, (27) implies that

$$\lim_{n\to\infty}S_{n+1}=S^*. \tag{29}$$

By solving the first equation of system (3) for $I_{n+1}$ and using (29), we have

$$\lim_{n\to\infty}I_{n+1}=\lim_{n\to\infty}\frac{\alpha-(\gamma+1)S_{n+1}+S_n}{\beta(2S_{n+1}-S_n-\alpha)}=\frac{\alpha-\gamma S^*}{\beta(S^*-\alpha)}=I^*. \tag{30}$$

From the third equation of system (3) and (30), $\lim_{n\to\infty}R_n=R^*$ can be shown similarly. Hence, we obtain $\lim_{n\to\infty}(S_n,I_n,R_n)=(S^*,I^*,R^*)$. Therefore, the endemic equilibrium $E^+$ of ID-SIR model (3) is globally asymptotically stable by LaSalle Invariance principle. □

## 6 Application of models to Swine flu data

In this section, we apply our discrete models (2) and (3) to the epidemic spread of the H1N1 influenza (swine flu) outbreak in a rural university town. It is well known that H1N1 in humans is an airborne disease, so transmission from infected individuals to susceptible individuals occurs via direct interaction between susceptible and infected individuals. Therefore, our models is suitable to describe H1N1 transmission

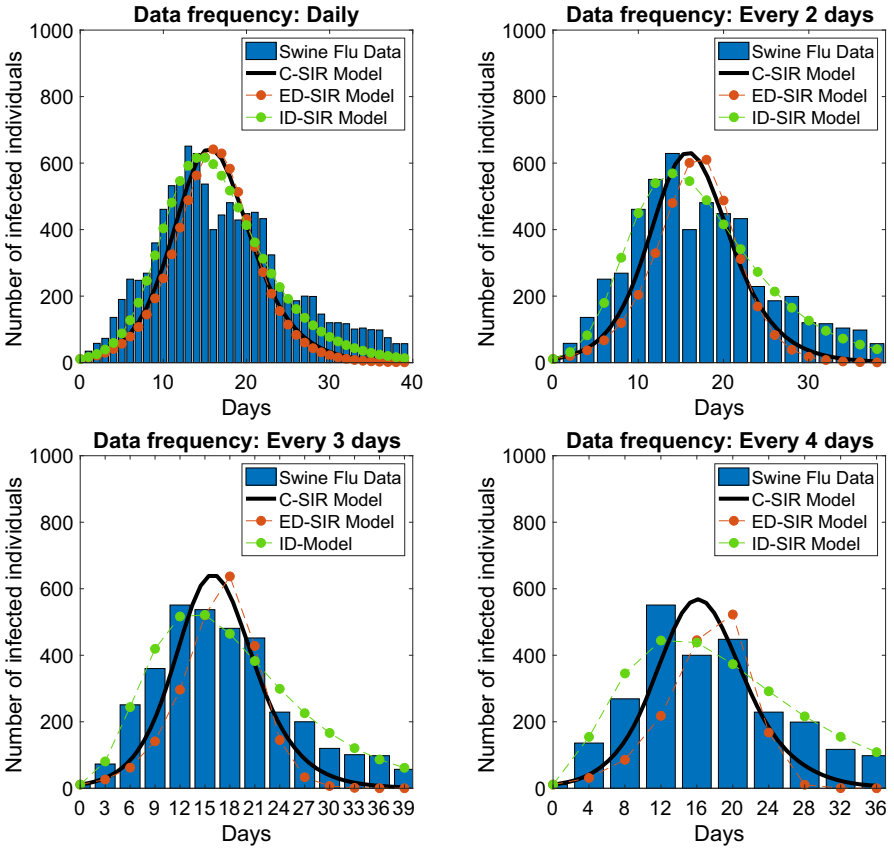


**Fig. 1** Fitting of C-SIR, ED-SIR, and ID-SIR models to Swine Flu Data from WSU (Vaidya et al. 2015) for the data frequency of daily, every 2 days, every 3 days, and every 4 days. The fixed parameters are $S_0 = 18223$ and $\alpha = \gamma = 0$ corresponding to the H1N1 outbreak situations in WSU (Vaidya et al. 2015). Since most infection occurs early during the semester, we have considered only 40-day data to capture critical transmission events. Also, the prevalence given in Vaidya et al. (2015) has been converted to the actual number of infected individuals for more clarity

dynamics. We used the published epidemiological data (Vaidya et al. 2015) of H1N1 influenza cases among students of Washington State University (WSU). Note that our models do not include the vaccination group because the vaccination data were unavailable, and vaccination against seasonal flu may not be effective against H1N1 strains. Since in the rural university town of WSU, with students as the majority of the population, birth-death is zero during a single semester of the data collection period, we took $\alpha = \gamma = 0$ to make it consistent with the data.

We fit all of C-SIR (1), ED-SIR (2), and ID-SIR (3) models to the data for 40 days and compare the models using the Akaike information criterion (AIC) described by the following formula (Akaike 1974; Burnham et al. 2011):

**Table 1** Model comparison based on AIC values

| | AIC Values | | |
|---|---|---|---|
| Data frequency | C-SIR Model | ED-SIR model | ID-SIR model |
| Daily | 381 | 390 | 341 |
| Every 2 days | 196 | 204 | 165 |
| Every 3 days | 136 | 145 | 109 |
| Every 4 days | 104 | 110 | 90 |

$$AIC = n_d \log\left(\frac{SSR}{n_d}\right) + \frac{2n_d(n_p+1)}{n_d - n_p - 2}, \tag{31}$$

where $n_d$ represents the number of data points, $n_p$ represents the number of parameter estimated, and $SSR$ represents the sum of squared residuals given by $SSR = \sum_{n_d}(I_{model} - I_{data})^2$. Here $I_{model}$ and $I_{data}$ represent the infected populations predicted by the model and given by the data, respectively. The lower the AIC value, the better the model to fit the data.

The data-fitting process indicates that each model can capture the epidemic trend of the H1N1 Influenza outbreak (Fig. 1). However, the computed AIC values (Table 1) indicate that the ID-SIR model fits the data best, followed by the C-SIR model and then by the ED-SIR model (AIC = 341, 381, 390 for ID-SIR, C-SIR, and ED-SIR models, respectively). To represent the situations, in which the frequency of the data is limited, we considered three data subsets by selecting the influenza cases every 2, 3, and 4 days from the original daily data set. For each data subset, we performed model fitting and comparison. We found that the order {ID-SIR, C-SIR, ED-SIR} of the goodness of the models remains the same for each data subset (Fig. 1, Table 1), indicating that implicit discrete formulation (ID-SIR model (3)) may be the suitable strategy to describe discrete epidemiological data best.

The best model, ID-SIR model (3), and the corresponding $\mathscr{R}_0$ formulation provide the basic reproduction number of H1N1 outbreak in WSU to be 1.35. Note that in the case of $\alpha = \gamma = 0$ in WSU, $\alpha/\gamma$ (an indeterminate form) needs to be replaced by the initial susceptible population, $S_0$, (i.e., entire susceptibility, $S_0 \approx S_0^*$, the disease-free equilibrium) in the formulation of $\mathscr{R}_0$.

We also considered the wider parameter space of two important parameters, $\beta$ (transmission rate), and $\lambda$ (recovery rate), and observed how $\mathscr{R}_0$ changes across these parameter spaces (Fig. 2). We identified the combinations of $\beta$ and $\lambda$ corresponding to $\mathscr{R}_0 = 1$ (line in Fig. 2), below which $\mathscr{R}_0 < 1$ (DFE is stable, implying the outbreak is avoided) and above which $\mathscr{R}_0 > 1$ (DFE is unstable, and the outbreak occurs). The decrease in $\beta$ and increase in $\lambda$ cause a decrease in $\mathscr{R}_0$, eventually bringing it below unity. In practice, the decrease of $\beta$ can be linked to the contact reducing preventions, such as wearing masks and self-protection through awareness, and the increase in $\lambda$ can be linked to the preventions such as detection and isolation of infected individuals. In Fig. 2 (right), we present the bifurcation diagram showing the stability of equilibriums (disease-free and endemic) using $\mathscr{R}_0$ as bifurcation parameters.

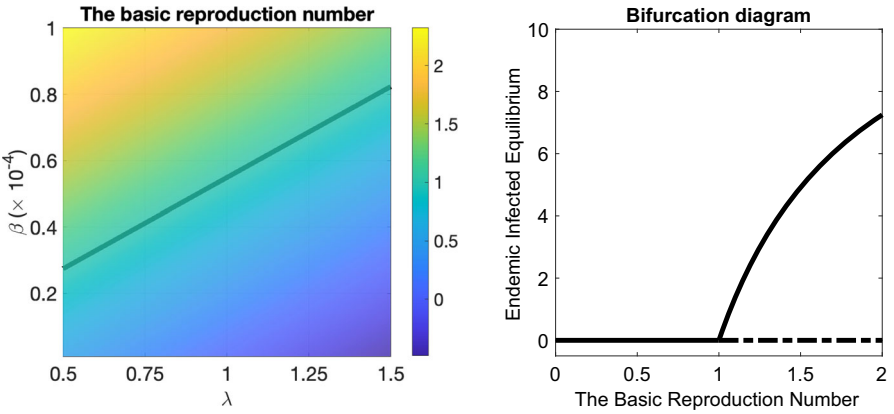


**Fig. 2** [Left] The basic reproduction number, $\mathscr{R}_0$, corresponding to the best-fit ID-SIR model for different values of $\beta$ and $\lambda$. The line represents the combinations of $\beta$ and $\lambda$ corresponding to $\mathscr{R}_0 = 1$. Other parameters used are $S_0 = 18223$ and $\alpha = \gamma = 0$ corresponding to the H1N1 outbreak situations in WSU (Vaidya et al. 2015). [Right] Bifurcation diagram showing infected class at equilibriums with $\mathscr{R}_0$ as a bifurcation parameter. To observe endemic equilibrium, the parameters used are $\gamma = 6.85 \times 10^{-4}$ (corresponding to the average university period of 4 years) and $\alpha = \gamma S_0$ (assuming that the system was in equilibrium before the outbreak)

## 7 Conclusion

In this paper, we propose two discrete epidemic models, ED-SIR (2) and ID-SIR (3), and develop the basic reproduction number, $\mathscr{R}_0$, for the models in order to obtain the stability of disease-free and endemic equilibria. We show the local stability of disease-free and endemic equilibria of ED-SIR model (2) by the linearization method, yet the global stability is left as an open problem. On the other hand, we successfully show the global stability of the endemic equilibrium of ID-SIR model (3) by constructing a suitable Lyapunov function, but the global stability of the disease free equilibrium is still not known. We also apply our discrete models, namely ED-SIR (2) and ID-SIR (3) together with continuous model C-SIR (1) to data from Swine Flu outbreak in a university community, and find that ID-SIR model (3) can describe the outbreak best among these models based on AIC value (31). We also compute the value of $\mathscr{R}_0$ for the best model ID-SIR (3) and discuss the stability with $\mathscr{R}_0$ as a bifurcation parameter. Note that our discrete models, ED-SIR (2) and ID-SIR (3), motivated from the time-scale modeling approach developed by Akın and Yeni (2020) for infectious disease models, have the same equilibria (12) and (13) and the basic reproduction number (14), respectively.

Now we would like to mention our observations and the importance of studying the theory of time scales. Note that the incidence rates of discrete models ED-SIR (2) and ID-SIR (3) depend on the death rate $\gamma$ and the transmission rate $\beta$ while continuous model C-SIR (1) has the incidence rate independent of $\gamma$. For the theory of time scales, we define the forward jump operator $\sigma$, giving the next point in a time scale. In our paper, our time scale only consists of integers $\mathbb{Z}$ for the discrete models and $\sigma(n) = n + 1$, $n \in \mathbb{Z}$. Note that the incidence rate of ID-SIR model (3) involves the forward

jump operator but not the incidence rate of ED-SIR model (2). Therefore, taking $\sigma$ into account provides an opportunity for studying discrete analysis theory. Importantly, the forward jump operator allows us to consider variants of a discrete model with the required time scale, while the continuous models lack this advantage since $\sigma(t) = t$ for $t \in \mathbb{R}$. In the theory of time scales, we can define the step-size function $\mu$ giving the distance between two consecutive points in a time scale, i.e., $\mu(t) = \sigma(t) - t$. Note that $\mu = 0$ allows us to recover the continuous models. We observe that AIC values for each model are smaller for a larger data frequency, indicating that a choice of suitable time-scale may be important for modeling. Therefore, it is worthwhile to consider other variants of discrete models (Akın and Yeni 2020) with the step-size function $\mu$ other than 1. For example, one can investigate the stability of equilibrium points of explicit and implicit discrete models on $h\mathbb{Z}$, $h > 0$ and estimate $h$ to determine the suitable time scale for the best model describing infectious diseases. Considering models on $h\mathbb{Z}$ might result in $\mathscr{R}_0$ that depends on $h$.

In summary, we provide time-scale-based discrete models, which may describe the epidemiological data better, presumably because of the discrete nature of the data. Our results underscore the importance of time-scale modeling for applications of infectious disease models for their control and prevention.

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