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Research article

Dynamic analysis of a discrete SIVS epidemic model with vaccination

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Abstract: In this paper, we consider a discrete-time Susceptible-Infected-Vaccinated-Susceptible (SIVS) epidemic model with vaccinations and a variable population size. The model includes the recruitment of susceptible and vaccinated individuals, vaccination of susceptible individuals, loss of vaccine-induced immunity, disease-induced mortality, and two possible outcomes after infection. We prove that the solutions remain nonnegative and that the total population is ultimately bounded. The disease-free and endemic equilibria are obtained, together with the basic reproduction number R_0. By applying the Jury criterion, we show that the disease-free equilibrium is locally asymptotically stable when $R_0 < 1$. The endemic equilibrium exists uniquely when $R_0 > 1$, and is locally asymptotically stable whenever the corresponding Jury conditions are satisfied. A Lyapunov function is used to obtain a sufficient condition for the global asymptotic stability of the disease-free equilibrium. Taking the transmission rate as the bifurcation parameter, we verify the transversality and quadratic nondegeneracy conditions and establish a nondegenerate forward transcritical bifurcation at $R_0 = 1$. A numerical example within the biologically admissible parameter region confirms disease extinction for $R_0 < 1$, convergence to a positive endemic equilibrium for $R_0 > 1$, and the exchange of stability at the threshold. Additional simulations illustrate oscillatory and irregular dynamics after the admissibility restrictions are relaxed; these behaviors are interpreted as mathematical extensions of the discrete map.

Keywords: SIVS epidemic model; vaccination; variable population size; stability; bifurcation
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1. Introduction

Epidemics have long affected human health and daily life, and their transmission dynamics have therefore been extensively studied. Mathematical epidemic models provide a useful framework to identify conditions for disease extinction or persistence. Classical examples include the Susceptible-Infected-Susceptible (SIS) and Susceptible-Infected-Recovered (SIR) models initiated by Kermack

and McKendrick. The references [1, 2] analyzed the stability and the bifurcations at the equilibria of SIS and SIR models. Lu et al. [3] mainly studied the stability of a Susceptible-Infected-Recovered-Susceptible (SIRS) epidemic model with a generalized non-monotonic saturation incidence rate.

Discrete-time epidemic models are appropriate when infection, vaccination, recovery, and reporting are recorded at fixed time intervals. Additionally, their use is mathematically motivated by the distinct dynamical structure of discrete maps. In particular, discrete systems may undergo flip and Neimark-Sacker bifurcations, which do not arise in the same form in continuous-time models. Allowing the total population to vary further changes the positively invariant region, equilibrium structure, stability conditions, and bifurcation thresholds. These features motivate the discrete Susceptible-Infected-Vaccinated-Susceptible (SIVS) framework with variable population size considered in this paper.

Several discrete epidemic models have been studied in relation to their continuous counterparts. For example, discrete Susceptible-Infected (SI) and SIS models were compared with the corresponding continuous models in [4], and a discrete SIS model with rich dynamical behavior was analyzed in [5]. Most of these studies, as well as related works, were developed within the SIS framework [6]. In parallel, a number of discrete models based on the SIR structure have also been proposed and investigated [7, 8]. Vaccinations have often been incorporated into SIS type models by introducing an additional vaccinated class. For instance, an SIS model with vaccinations was considered in [9], where the vaccine was assumed to be partially effective and vaccinations were applied to susceptible individuals. In that model, the total population size was fixed, and disease transmission was described by the standard incidence rate.

With the development of medicine, vaccination plays an important role in preventing some epidemics, such as smallpox, measles, and influenza. Vaccinations have been widely incorporated into epidemic models; representative applications can be found in [10, 11]. Parsamanesh et al. [12] studied the stability of an SIVS epidemic model at the equilibria. Parsamanesh [13] discretized the continuous model by applying the forward Euler method and analyzed the dynamic behavior of an SIS epidemic model with vaccinations. Besides, the transmission of the COVID-19 virus in the past few years around the world has been shown to be random; Ullah et al [14] used stochastic differential equations to study the relationship between vaccinations and the dynamics of the spread of COVID-19 and obtained the conditions for the persistence and extinction of diseases.

The population size is generally variable due to the influence of parameters. Wei et al. [15] studied an SIR model with a variable total population size and obtained the uniqueness of the solution as well as the conditions for disease persistence and extinction. The references [13,16,17] studied the dynamic behavior of different epidemic models with a variable population size.

Compared with the related discrete-time epidemic models, the present model introduces several mechanisms. Parsamanesh et al. [12] studied a discrete epidemic model with vaccinations and vital dynamics, while Parsamanesh and Erfanian [13] considered a discrete SIVS model with saturated incidence. In the present model, the constant recruitment term A is divided between the susceptible and vaccinated classes, with a fraction q of newly recruited individuals directly entering the vaccinated class. Additionally, the model distinguishes two post-infection pathways: Infected individuals may either return to the susceptible class after cure at a rate of ω, or enter the vaccinated class after recovery with temporary immunity at a rate of α. Together with vaccinations of susceptible individuals, loss of vaccine-induced immunity, and disease-induced mortality, these assumptions make the total population variable and lead to different expressions for the equilibria, the basic reproduction number

R_0, boundedness, stability conditions, and local bifurcation conditions. Then, we derive the equilibria and R_0, study the local and global stability of the equilibria, discuss local bifurcations, and use numerical simulations to illustrate the theoretical results and the effect of vaccination on disease control.

The rest of the paper is organized as follows: In Section 2, the model is formulated and the equilibria and R_0 are obtained; Section 3 studies the stability of the disease-free and endemic equilibria; Section 4 discusses possible local bifurcations; Section 5 presents numerical simulations; and Section 6 gives the conclusion and some further remarks.

2. Modeling

In this section, we introduce a discrete SIVS model with vaccinations and formulate it with a set of difference equations. Moreover, the equilibria and the basic reproduction number R_0 of the model are solved.

2.1. Analysis of the model

The population is divided into three compartments: Susceptible, infected, and vaccinated, with S_n, I_n, *and* V_n denoting the number of individuals at the time of n, respectively, N_n is the total population size at the time of n, and $N_n = S_n + I_n + V_n$. All the parameters in the model are positive, the number of individuals introduced population per unit time are denoted by A, the fraction of new members and susceptible individuals who vaccinated are denoted by q and δ respectively, θ is the immune loss rate, μ is natural mortality, d is mortality due to disease, ω is the cure rate, and the recovery rate is α. Assume that the disease spreads with the saturated incidence rate $\frac{\beta S_n I_n}{1+\gamma I_n}$. The transmission diagram of diseases between compartments is shown in Figure 1.

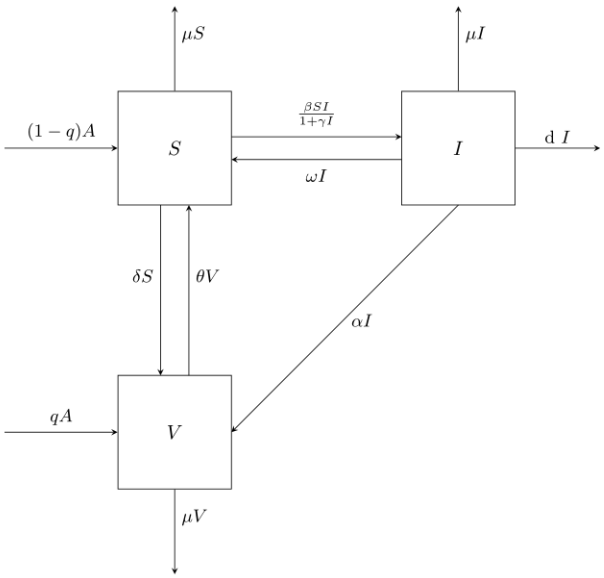


Figure 1. Flow diagram of disease transmission.

The model can be presented by the following system of difference equations:

$$\begin{cases} S_{n+1} = (1-q)A + (1-\mu-\delta-\frac{\beta I_n}{1+\gamma I_n})S_n + \omega I_n + \theta V_n, \\ I_{n+1} = (1-\alpha-\omega-\mu-d)I_n + \frac{\beta S_n I_n}{1+\gamma I_n}, \\ V_{n+1} = (1-\mu-\theta)V_n + \alpha I_n + \delta S_n + qA. \end{cases} \tag{2.1}$$

Lemma 2.1. Assume that all parameters of System (2.1) are nonnegative, $0 \le q \le 1$, and

$$0 < \mu + \delta + \frac{\beta}{\gamma} < 1, \tag{2a}$$

$$0 < \alpha + \omega + \mu + d < 1, \tag{2b}$$

$$0 < \mu + \theta < 1. \tag{2c}$$

Then, for any nonnegative initial value

$$(S_0, I_0, V_0) \in \mathbb{R}^3_+,$$

the solution of System (2.1) remains nonnegative, that is,

$$S_n \ge 0, \qquad I_n \ge 0, \qquad V_n \ge 0, \qquad n \ge 0.$$

Proof. Suppose that

$$S_n \ge 0, \qquad I_n \ge 0, \qquad V_n \ge 0.$$

Since $I_n \ge 0$, we have the following:

$$0 \le \frac{\beta I_n}{1+\gamma I_n} < \frac{\beta}{\gamma}.$$

It follows from (2a) that

$$1-\mu-\delta-\frac{\beta I_n}{1+\gamma I_n} > 1-\mu-\delta-\frac{\beta}{\gamma} > 0.$$

Moreover, (2b) and (2c) give the following:

$$1-\alpha-\omega-\mu-d > 0, \qquad 1-\mu-\theta > 0.$$

Together with $0 \le q \le 1$, all terms in System (2.1) are nonnegative whenever $(S_n, I_n, V_n) \in \mathbb{R}^3_+$. Hence,

$$S_{n+1} \ge 0, \qquad I_{n+1} \ge 0, \qquad V_{n+1} \ge 0.$$

By induction, the solution remains nonnegative for all $n \ge 0$.

Lemma 2.2. Assume that Conditions (2a)–(2c) hold. Then, the solutions of System (2.1) with nonnegative initial values remain nonnegative. Moreover, the set

$$\Omega = \left\{(S, I, V) \in \mathbb{R}^3_+ : S + I + V \le \frac{A}{\mu}\right\}$$

is positively invariant. In addition, every nonnegative solution of System (2.1) is ultimately bounded and satisfies the following:

$$\limsup_{n\to\infty} N_n \le \frac{A}{\mu},$$

where $N_n = S_n + I_n + V_n$.

Proof. Under Conditions (2*a*)–(2*c*), all transition coefficients in System (2.1) are nonnegative. Hence, for any nonnegative initial value $(S_0, I_0, V_0) \in \mathbb{R}^3_+$, the corresponding solution satisfies the following:

$$S_n \ge 0, \qquad I_n \ge 0, \qquad V_n \ge 0, \qquad n \ge 0.$$

Adding the three equations of System (2.1), we obtain he following:

$$N_{n+1} = A + (1-\mu)N_n - dI_n.$$

Since $I_n \ge 0$, it follows that

$$N_{n+1} \le A + (1-\mu)N_n.$$

If $(S_n, I_n, V_n) \in \Omega$, then $N_n \le A/\mu$; therefore,

$$N_{n+1} \le A + (1-\mu)\frac{A}{\mu} = \frac{A}{\mu}.$$

Thus, $(S_{n+1}, I_{n+1}, V_{n+1}) \in \Omega$, which proves that Ω is positively invariant.

For general nonnegative initial values, repeated use of the above inequality gives the following:

$$\begin{aligned} N_{n+1} &\le A + (1-\mu)N_n \\ &\le A[1 + (1-\mu)] + (1-\mu)^2 N_{n-1} \\ &\le A[1 + (1-\mu) + (1-\mu)^2] + (1-\mu)^3 N_{n-2} \\ &\le A\frac{1-(1-\mu)^{n+1}}{\mu} + (1-\mu)^{n+1} N_0 \\ &\le \frac{A}{\mu} + N_0. \end{aligned}$$

Since $0 < \mu < 1$, we have $(1-\mu)^{n+1} \to 0$ as $n \to \infty$. Hence

$$\limsup_{n\to\infty} N_n \le \frac{A}{\mu}.$$

Therefore, the total population size is variable but ultimately bounded.

2.2. *Solving for equilibria*

At the equilibria, System (2.1) is equivalent to the following system:

$$\begin{cases} (1-q)A + \omega I_n + \theta V_n - (\mu + \delta + \frac{\beta I_n}{1+\gamma I_n})S_n = 0, \\ (\alpha + \omega + \mu + d)I_n - \frac{\beta S_n I_n}{1+\gamma I_n} = 0, \\ \alpha I_n + \delta S_n + qA - (\mu + \theta)V_n = 0. \end{cases} \tag{2.2}$$

From the second equation of System (2.2), we have $I = 0$ or $\frac{\beta S}{1+\gamma I} = \alpha + \omega + \mu + d$.
When $I = 0$, then $S = \frac{A[(1-q)\mu+\theta]}{\mu(\mu+\theta+\delta)}$, *and* $V = \frac{A(\delta+q\mu)}{\mu(\mu+\theta+\delta)}$, which is the disease-free equilibrium E^0 of System (2.1):

$$E^0 = (S^0, I^0, V^0) = (\frac{A[(1-q)\mu+\theta]}{\mu(\mu+\theta+\delta)}, 0, \frac{A(\delta+q\mu)}{\mu(\mu+\theta+\delta)}).$$

Lemma 2.3. The endemic equilibrium $E^* = (S^*, I^*, V^*)$ of System (2.1) exists uniquely if and only if $R_0 > 1$.

Proof. Let

$$\eta = \alpha + \omega + \mu + d.$$

At an endemic equilibrium, $I^* > 0$, and the second equilibrium equation gives

$$S^* = \frac{\eta(1+\gamma I^*)}{\beta}.$$

Substituting this expression into the remaining equilibrium equations yields

$$I^* = \frac{\beta A[(1-q)\mu+\theta] - \mu(\mu+\theta+\delta)\eta}{D},$$

where

$$D = \beta\left[(\mu+\theta)(\alpha+\mu+d) - \alpha\theta\right] + \mu\eta\gamma(\mu+\delta+\theta).$$

Since

$$(\mu+\theta)(\alpha+\mu+d) - \alpha\theta = \mu(\alpha+\mu+d) + \theta(\mu+d) > 0,$$

we have $D > 0$. Moreover,

$$\beta A[(1-q)\mu+\theta] - \mu(\mu+\theta+\delta)\eta = \mu(\mu+\theta+\delta)\eta(R_0 - 1).$$

Therefore, $I^* > 0$ if and only if $R_0 > 1$. Once I^* is determined, S^* and V^* are uniquely determined by the equilibrium equations. Hence, the endemic equilibrium exists uniquely if and only if $R_0 > 1$.

The basic reproduction number R_0 is obtained using the next-generation matrix method. Let the second equation of System (2.2) is $\hat{I} = \mathcal{F} - \mathcal{V}$, where $\mathcal{F} = \frac{\beta S I}{1+\gamma I}$, *and* $\mathcal{V} = (\alpha+\omega+\mu+d)I$; then, take the partial derivatives of $\mathcal{F}$, *and* $\mathcal{V}$ with respect to I at E^0, as follows:

$$F = \frac{\partial \mathcal{F}}{\partial I}\Big|_{E^0} = \beta S^0 = \frac{\beta A[(1-q)\mu+\theta]}{\mu(\mu+\delta+\theta)},$$
$$V = \frac{\partial \mathcal{V}}{\partial I}\Big|_{E^0} = \alpha+\omega+\mu+d.$$

Therefore,

$$R_0 = \rho(FV^{-1}) = \frac{\beta S^0}{\alpha+\omega+\mu+d} = \frac{\beta A[(1-q)\mu+\theta]}{\mu(\mu+\delta+\theta)(\alpha+\omega+\mu+d)},$$
$$S^0 = \frac{(\alpha+\omega+\mu+d)}{\beta}R_0,$$
$$I^* = \frac{\mu(\mu+\delta+\theta)(\alpha+\omega+\mu+d)(R_0-1)}{\beta[(\mu+\theta)(\alpha+\mu+d) - \alpha\theta] + \mu\gamma(\alpha+\omega+\mu+d)(\mu+\delta+\theta)}.$$

Thus, $R_0 = 1$ is the threshold for the existence of the endemic equilibrium. The stability properties of the equilibria are studied in the next section.

3. Stability

In this section, we obtain the stability of System (2.1) at the equilibria through the properties of Jacobian matrices, Jury conditions, and Lyapunov functions.

Theorem 3.1. *The disease-free equilibrium E^0 of Model* (2.1) *is locally asymptotically stable, when $R_0 < 1$.*

Proof. Let the saturation incidence rate $\frac{\beta S_n I_n}{1+\gamma I_n} = \Phi$; then, $\Phi_I = \frac{\partial \Phi}{\partial I} = \frac{\beta S}{(1+\gamma I)^2}$ *and* $\Phi_S = \frac{\partial \Phi}{\partial S} = \frac{\beta I}{1+\gamma I}$. The Jacobian matrices of the System (2.1) at $E(S, I, V)$ are as follows:

$$J(S, I, V) = \begin{pmatrix} 1-\mu-\delta-\Phi_S & \omega-\Phi_I & \theta \\ \Phi_S & 1-\eta+\Phi_I & 0 \\ \delta & \alpha & 1-\mu-\theta \end{pmatrix}, \tag{3.1}$$

so the Jacobian matrice at E^0 is

$$J^0 = \begin{pmatrix} 1-\mu-\delta & \omega-\beta S^0 & \theta \\ 0 & 1-\eta+\beta S^0 & 0 \\ \delta & \alpha & 1-\mu-\theta \end{pmatrix}. \tag{3.2}$$

It can be found that the eigenvalues of J^0 are $\lambda_1 = \beta S^0 + (1-\alpha-\omega-\mu-d)$, $\lambda_2 = 1-(\mu+\delta+\theta)$, $\lambda_3 = 1-\mu$. According to Conditions (2a)-(2c), we have $|\lambda_2| < 1$, $|\lambda_3| < 1$.

If $\frac{\beta S^0}{\mu+\omega+\alpha+d} < 1$ (i.e., $R_0 < 1$), then $|\lambda_1| < 1$. Therefore, the modulus of all eigenvalues of J^0 are less than one, the system is locally asymptotically stable when $R_0 < 1$.

Theorem 3.2. *Assume Conditions* (2a)–(2c) *hold and that $R_0 > 1$. Then, System* (2.1) *admits a unique endemic equilibrium as follows:*

$$E^* = (S^*, I^*, V^*), \qquad I^* > 0.$$

Let

$$f(\lambda) = \det(\lambda I - J^*) = \lambda^3 + p_1\lambda^2 + p_2\lambda + p_3$$

be the characteristic polynomial of the Jacobian matrix $J^* = J(E^*)$. The first two Jury conditions,

$$f(1) > 0 \tag{C1}$$

and

$$-f(-1) > 0, \tag{C2}$$

are satisfied under Conditions (2a)–(2c) *and $R_0 > 1$. Additionally, if*

$$1 - p_3^2 > |p_2 - p_1 p_3|, \tag{C3}$$

then the endemic equilibrium E^* is locally asymptotically stable.

Proof. Let

$$\eta = \alpha + \omega + \mu + d,$$

and define

$$x = \frac{\beta I^*}{1+\gamma I^*}, \qquad y = \frac{\beta S^*}{(1+\gamma I^*)^2},$$

together with

$$a = \eta - y, \qquad b = \mu + \delta + x, \qquad c = \mu + \theta.$$

Using the state-vector ordering

$$X_n = (S_n, I_n, V_n)^{\mathrm{T}},$$

the Jacobian matrix at E^* is

$$J^* = \begin{pmatrix} 1-b & \omega - y & \theta \\ x & 1-a & 0 \\ \delta & \alpha & 1-c \end{pmatrix}.$$

A direct expansion of $f(\lambda) = \det(\lambda I - J^*)$ gives

$$p_1 = a + b + c - 3,$$

$$p_2 = 3 - 2(a+b+c) + ab + ac + bc + x(y-\omega) - \theta\delta,$$

and

$$\begin{aligned} p_3 = & -1 + a + b + c - ab - ac - bc - x(y-\omega) + \theta\delta \\ & + abc + xc(y-\omega) - a\theta\delta - \alpha\theta x. \end{aligned}$$

At the endemic equilibrium,

$$\frac{\beta S^*}{1+\gamma I^*} = \eta.$$

Consequently,

$$y = \frac{\eta}{1+\gamma I^*},$$

and hence

$$a = \eta - y = \frac{\eta\gamma I^*}{1+\gamma I^*}.$$

Since $I^* > 0$ and Condition *(2b)* gives $0 < \eta < 1$, we have

$$0 < a < 1.$$

Moreover,

$$0 < x = \frac{\beta I^*}{1+\gamma I^*} < \frac{\beta}{\gamma}.$$

Therefore, Condition *(2a)* yields the following:

$$0 < b = \mu + \delta + x < \mu + \delta + \frac{\beta}{\gamma} < 1.$$

Condition *(2c)* gives the following:

$$0 < c = \mu + \theta < 1.$$

First, we verify Condition *(C1)*. Direct calculation gives the following:

$$f(1) = a(bc - \theta\delta) + x\left[c(y-\omega) - \alpha\theta\right].$$

Substituting

$$a = \frac{\eta\gamma I^*}{1+\gamma I^*}, \qquad x = \frac{\beta I^*}{1+\gamma I^*}, \qquad y = \frac{\eta}{1+\gamma I^*},$$

and using $c = \mu + \theta$, we obtain

$$f(1) = \frac{I^*}{1+\gamma I^*}\Big\{\beta\left[(\mu+\theta)(\alpha+\mu+d) - \alpha\theta\right] + \mu\eta\gamma(\mu+\delta+\theta)\Big\}.$$

With

$$D = \beta\left[(\mu+\theta)(\alpha+\mu+d) - \alpha\theta\right] + \mu\eta\gamma(\mu+\delta+\theta),$$

this becomes

$$f(1) = \frac{I^* D}{1+\gamma I^*}.$$

Since

$$I^* D = \mu\eta(\mu+\delta+\theta)(R_0 - 1),$$

we have

$$f(1) = \frac{\mu\eta(\mu+\delta+\theta)(R_0-1)}{1+\gamma I^*} > 0.$$

Thus, Condition *(C1)* holds.

Next, we verify Condition *(C2)*. A direct determinant calculation gives the following:

$$-f(-1) = (2-a)\left[(2-b)(2-c) - \theta\delta\right] + x\left[(2-c)(y-\omega) + \alpha\theta\right].$$

Set

$$P = (2-b)(2-c) - \theta\delta.$$

Since

$$x < \frac{\beta}{\gamma} < 1 - \mu - \delta,$$

we obtain

$$\begin{aligned} P - x(2-c) &= (2-c)(2-b-x) - \theta\delta \\ &= (2-c)(2-\mu-\delta-2x) - \theta\delta \\ &> (2-c)(\mu+\delta) - \theta\delta \\ &= \mu(2-\mu-\theta) + \delta(2-\mu-2\theta). \end{aligned}$$

Condition $\mu + \theta < 1$ implies

$$2 - \mu - \theta > 0$$

and

$$2 - \mu - 2\theta = (1-\mu-\theta) + (1-\theta) > 0.$$

Therefore,

$$P > x(2-c) > 0.$$

Furthermore, since $a = \eta - y$,

$$y - \omega = \eta - a - \omega = \alpha + \mu + d - a > -a.$$

It follows that

$$\begin{aligned} -f(-1) &> (2-a)P - ax(2-c) \\ &> (2-a)x(2-c) - ax(2-c) \\ &= 2(1-a)x(2-c) > 0. \end{aligned}$$

Thus, Condition *(C2)* also holds.

For a real monic cubic polynomial, the Jury criterion states that all roots lie strictly inside the unit disk when *(C1)*–*(C3)* hold. Notice that *(C3)* also implies $1 - p_3^2 > 0$, and hence $|p_3| < 1$.

Conditions *(C1)* and *(C2)* have been analytically established, whereas Condition *(C3)* is explicitly imposed as an additional hypothesis of the theorem. Therefore, all eigenvalues of J^* lie strictly inside the unit disk, and the endemic equilibrium E^* is locally asymptotically stable.

Theorem 3.3. Assume that Conditions (2*a*)–(2*c*) hold, and define the following:

$$\widehat{R_0} = \frac{\beta A}{\mu(\alpha + \omega + \mu + d)}.$$

If $\widehat{R_0} \leq 1$, then the disease-free equilibrium E^0 of System (2.1) is globally asymptotically stable in the feasible region

$$\Omega = \left\{(S, I, V) \in \mathbb{R}^3_+ : S + I + V \leq \frac{A}{\mu}\right\}.$$

Proof. By Lemma 2.2, the set

$$\Omega = \left\{(S, I, V) \in \mathbb{R}^3_+ : S + I + V \leq \frac{A}{\mu}\right\}$$

is compact and positively invariant. Hence, for every solution starting from Ω, we have

$$0 \leq S_n \leq N_n \leq \frac{A}{\mu}, \qquad n \geq 0.$$

Let the Lyapunov function hold:

$$L(S_n, I_n, V_n) = I_n.$$

From the second equation of System (2.1), we obtain

$$\begin{aligned} \Delta L &= L(S_{n+1}, I_{n+1}, V_{n+1}) - L(S_n, I_n, V_n) \\ &= I_{n+1} - I_n \\ &= \left(\frac{\beta S_n}{1 + \gamma I_n} - (\alpha + \omega + \mu + d)\right) I_n. \end{aligned}$$

Since $S_n \leq N_n \leq A/\mu$, we have the following:

$$\frac{\beta S_n}{1 + \gamma I_n} \leq \beta S_n \leq \frac{\beta A}{\mu}.$$

Therefore,

$$\Delta L \leq \left(\frac{\beta A}{\mu} - (\alpha + \omega + \mu + d)\right) I_n = (\alpha + \omega + \mu + d)(\widehat{R}_0 - 1) I_n.$$

If $\widehat{R}_0 \leq 1$, then

$$\Delta L \leq 0.$$

By the discrete LaSalle invariance principle, every solution starting in Ω approaches the largest invariant set contained in

$$\mathcal{M} = \{(S, I, V) \in \Omega : \Delta L = 0\}.$$

Now, we characterize this set. If $\widehat{R}_0 < 1$, then $\Delta L = 0$ implies $I_n = 0$. If $\widehat{R}_0 = 1$, then equality in

$$\frac{\beta S_n}{1 + \gamma I_n} \leq \frac{\beta A}{\mu}$$

with $I_n > 0$ would require

$$\frac{S_n}{1 + \gamma I_n} = \frac{A}{\mu}.$$

This is impossible because $S_n \leq A/\mu$ and $1 + \gamma I_n > 1$. Hence, as is in the case when $\widehat{R}_0 = 1$, we must have $I_n = 0$. Therefore, the largest invariant set contained in $\mathcal{M}$ lies in

$$\{(S, I, V) \in \Omega : I = 0\}.$$

Consequently,

$$I_n \to 0, \qquad n \to \infty.$$

It remains to show that $S_n \to S^0$ and $V_n \to V^0$. Let

$$z_n = \begin{pmatrix} S_n - S^0 \\ V_n - V^0 \end{pmatrix}.$$

Using the equilibrium equations for S^0 and V^0, we obtain the following:

$$z_{n+1} = B z_n + r_n,$$

where

$$B = \begin{pmatrix} 1 - \mu - \delta & \theta \\ \delta & 1 - \mu - \theta \end{pmatrix}$$

and

$$r_n = \begin{pmatrix} \left(\omega - \dfrac{\beta S_n}{1 + \gamma I_n}\right) I_n \\ \alpha I_n \end{pmatrix}.$$

Since the solution remains bounded in Ω and $I_n \to 0$, we have the following:

$$r_n \to 0.$$

The eigenvalues of B are as follows:

$$\lambda_1 = 1 - \mu, \qquad \lambda_2 = 1 - \mu - \delta - \theta.$$

Under Conditions (2a)–(2c), both eigenvalues have modulus less than one. Hence, $\rho(B) < 1$. It follows from the stability of the linear system with the vanishing perturbation r_n that

$$z_n \to 0.$$

Therefore,

$$S_n \to S^0, \qquad V_n \to V^0.$$

Combining these limits with $I_n \to 0$, we obtain the following:

$$(S_n, I_n, V_n) \to (S^0, 0, V^0) = E^0.$$

Moreover,

$$R_0 = \widehat{R}_0 \frac{(1-q)\mu + \theta}{\mu + \delta + \theta} < \widehat{R}_0 \leq 1.$$

Hence, $R_0 < 1$, and Theorem 3.1 implies that E^0 is locally asymptotically stable. Combining local stability with global attractivity, we conclude that E^0 is globally asymptotically stable in Ω.

Remark 3.1. The quantity $\widehat{R}_0$ is only used as a sufficient condition for global stability. It does not replace the basic reproduction number R_0. The basic reproduction number is as follows:

$$R_0 = \frac{\beta S^0}{\alpha + \omega + \mu + d},$$

where

$$S^0 = \frac{A[(1-q)\mu + \theta]}{\mu(\mu + \theta + \delta)}.$$

Thus

$$R_0 = \widehat{R}_0 \cdot \frac{(1-q)\mu + \theta}{\mu + \theta + \delta}.$$

Since

$$0 < \frac{(1-q)\mu + \theta}{\mu + \theta + \delta} < 1,$$

we have

$$R_0 < \widehat{R}_0.$$

Therefore, $\widehat{R}_0 \leq 1$ implies $R_0 \leq 1$. The reason for using $\widehat{R}_0$ in Theorem 3.3 is that the global feasible region gives the uniform estimate $S_n \leq A/\mu$, rather than $S_n \leq S^0$. Hence, $R_0 \leq 1$ is sufficient for local stability of E^0, while $\widehat{R}_0 \leq 1$ provides a rigorous sufficient condition for global stability.

4. Bifurcations

A local bifurcation of a discrete map may occur when one or more multipliers of the corresponding fixed point lie on the unit circle. However, this spectral condition alone is not sufficient to determine the type of bifurcation. A simple multiplier equal to 1 may be associated with a saddle-node, transcritical, or pitchfork bifurcation, depending on the corresponding transversality and nonlinear nondegeneracy conditions. A multiplier equal to -1 indicates a possible period-doubling bifurcation, whereas a

pair of nonreal complex-conjugate multipliers crossing the unit circle may lead to a Neimark-Sacker bifurcation; for the construction of such bifurcations in discrete-time nonlinear systems, see [18].

Theorem 4.1. *Let*

$$\eta = \alpha + \omega + \mu + d, \qquad S^0 = \frac{A[(1-q)\mu + \theta]}{\mu(\mu + \delta + \theta)},$$

and take the transmission rate β as the bifurcation parameter. Define he following:

$$\beta_c = \frac{\eta}{S^0} = \frac{\mu(\mu + \delta + \theta)\eta}{A[(1-q)\mu + \theta]}.$$

Assume that

$$0 < \eta < 1, \qquad 0 < \mu + \theta < 1, \qquad 0 < \mu + \delta + \frac{\beta_c}{\gamma} < 1.$$

Then the disease-free equilibrium

$$E^0 = (S^0, 0, V^0)$$

undergoes a nondegenerate forward transcritical bifurcation at

$$\beta = \beta_c,$$

or equivalently at $R_0 = 1$.

More precisely, for β sufficiently close to β_c, the disease-free equilibrium is locally asymptotically stable for $\beta < \beta_c$ and unstable for $\beta > \beta_c$. For $\beta > \beta_c$ sufficiently close to β_c, a unique positive endemic equilibrium emerges and is locally asymptotically stable.

Moreover, as β varies in the biologically admissible region, neither a period-doubling nor a Neimark-Sacker bifurcation occurs at the disease-free equilibrium.

Proof. Let

$$\mathcal{F}(X, \beta) = \begin{pmatrix} \mathcal{F}_1(X, \beta) \\ \mathcal{F}_2(X, \beta) \\ \mathcal{F}_3(X, \beta) \end{pmatrix}$$

denote the map defined by System *(2.1)*, where $X = (S, I, V)^{\mathrm{T}}$ and

$$\mathcal{F}_2(X, \beta) = (1 - \eta)I + \frac{\beta S I}{1 + \gamma I}.$$

The disease-free equilibrium

$$E^0 = (S^0, 0, V^0)$$

exists for every β and is independent of β. Since

$$R_0 = \frac{\beta S^0}{\eta},$$

the condition $R_0 = 1$ is equivalent to

$$\beta = \beta_c = \frac{\eta}{S^0}.$$

The Jacobian matrix at E^0 is as follows:

$$J^0(\beta) = D_X\mathcal{F}(E^0,\beta) = \begin{pmatrix} 1-\mu-\delta & \omega-\beta S^0 & \theta \\ 0 & 1-\eta+\beta S^0 & 0 \\ \delta & \alpha & 1-\mu-\theta \end{pmatrix}.$$

Its multipliers are as follows:

$$\lambda_c(\beta) = 1-\eta+\beta S^0,$$
$$\lambda_s^{(1)} = 1-\mu, \qquad \lambda_s^{(2)} = 1-\mu-\delta-\theta.$$

At $\beta = \beta_c$,

$$\lambda_c(\beta_c) = 1-\eta+\beta_c S^0 = 1.$$

Moreover,

$$\left.\frac{d\lambda_c}{d\beta}\right|_{\beta=\beta_c} = S^0 > 0.$$

Thus, the critical multiplier crosses 1 transversely.

Since

$$0 < \mu < 1,$$

we have

$$|\lambda_s^{(1)}| = |1-\mu| < 1.$$

Furthermore, the assumptions

$$\mu+\delta+\frac{\beta_c}{\gamma} < 1 \qquad \text{and} \qquad \mu+\theta < 1$$

imply

$$\delta < 1-\mu, \qquad \theta < 1-\mu.$$

Consequently,

$$0 < \mu+\delta+\theta < 2,$$

and hence

$$|\lambda_s^{(2)}| = |1-\mu-\delta-\theta| < 1.$$

Therefore, the multiplier 1 is simple, while the remaining two multipliers lie strictly inside the unit circle.

Set

$$\varepsilon = \beta-\beta_c$$

and introduce the translated variables

$$s = S-S^0, \qquad i = I, \qquad v = V-V^0.$$

At $\varepsilon = 0$, a right eigenvector corresponding to the multiplier 1 can be normalized as follows:

$$r = \begin{pmatrix} r_S \\ 1 \\ r_V \end{pmatrix},$$

where

$$r_S = -\frac{\mu(\alpha+\mu+d)+\theta(\mu+d)}{\mu(\mu+\delta+\theta)}$$

and

$$r_V = \frac{\alpha\mu-\delta(\mu+d)}{\mu(\mu+\delta+\theta)}.$$

Indeed, direct substitution gives the following:

$$(J^0(\beta_c)-I)r = 0.$$

In particular,

$$r_S < 0.$$

The map $\mathcal{F}$ is smooth in a neighborhood of (E^0,β_c) because $1+\gamma I$ is nonzero there. By the parameter-dependent center manifold theorem for discrete maps [19], the local center manifold can be written as follows:

$$s = h_1(i,\varepsilon), \qquad v = h_2(i,\varepsilon),$$

where

$$h_1(0,\varepsilon) = h_2(0,\varepsilon) = 0$$

and

$$h_1(i,\varepsilon) = r_S\, i + O(i^2+|\varepsilon i|),$$
$$h_2(i,\varepsilon) = r_V i + O(i^2+|\varepsilon i|).$$

Restricting the infected equation to the center manifold gives the following:

$$i_{n+1} = (1-\eta)i_n + \frac{(\beta_c+\varepsilon)\left[S^0+h_1(i_n,\varepsilon)\right]i_n}{1+\gamma i_n}.$$

Using

$$\beta_c S^0 = \eta,$$

together with

$$\frac{1}{1+\gamma i} = 1-\gamma i + O(i^2),$$

we obtain

$$i_{n+1} = i_n + a\varepsilon i_n + bi_n^2 + O\left(|i_n|(|i_n|+|\varepsilon|)^2\right),$$

where

$$a = S^0 > 0$$

and

$$b = \beta_c r_S - \eta\gamma.$$

Since $r_S < 0$, we have the following:

$$b = -\left[\frac{\beta_c\{\mu(\alpha+\mu+d)+\theta(\mu+d)\}}{\mu(\mu+\delta+\theta)}+\eta\gamma\right] < 0.$$

Let the reduced map be denoted by the following:

$$i_{n+1} = g(i_n, \varepsilon).$$

It satisfies the following:

$$g(0, \varepsilon) = 0, \qquad g_i(0, 0) = 1,$$
$$g_{i\varepsilon}(0, 0) = a = S^0 > 0, \qquad g_{ii}(0, 0) = 2b < 0.$$

Therefore, the parameter transversality and quadratic nondegeneracy conditions for a transcritical bifurcation of a one-dimensional map are satisfied. Hence, the reduced map, and therefore the full system, undergoes a nondegenerate transcritical bifurcation at $\varepsilon = 0$.

For completeness, the two local fixed-point branches can be explicitly identified. The fixed-point equation of the reduced map is

$$i\left[a\varepsilon + bi + O\left((|i| + |\varepsilon|)^2\right)\right] = 0.$$

One branch is

$$i_0(\varepsilon) = 0,$$

which corresponds to the disease-free equilibrium. Since $b \neq 0$, the implicit function theorem gives a unique second branch

$$i_*(\varepsilon) = -\frac{a}{b}\varepsilon + O(\varepsilon^2).$$

Because $a > 0$ and $b < 0$,

$$-\frac{a}{b} > 0.$$

Thus,

$$i_*(\varepsilon) > 0 \quad \text{for} \quad \varepsilon > 0,$$

whereas

$$i_*(\varepsilon) < 0 \quad \text{for} \quad \varepsilon < 0.$$

Therefore, the second branch is only biologically admissible for $\beta > \beta_c$, which shows that the transcritical bifurcation is forward.

This branch agrees with the explicitly calculated endemic equilibrium. Indeed, the infected component of the endemic equilibrium can be written as follows:

$$I^*(\beta) = \frac{A[(1-q)\mu + \theta](\beta - \beta_c)}{\beta K + \mu\eta\gamma(\mu + \delta + \theta)},$$

where

$$K = \mu(\alpha + \mu + d) + \theta(\mu + d) > 0.$$

Let

$$D_c = \beta_c K + \mu\eta\gamma(\mu + \delta + \theta) > 0.$$

Then,

$$I^*(\beta) = \frac{A[(1-q)\mu + \theta]}{D_c}(\beta - \beta_c) + O\left((\beta - \beta_c)^2\right).$$

On the other hand,

$$b = -\frac{D_c}{\mu(\mu + \delta + \theta)}$$

and

$$a = S^0 = \frac{A[(1-q)\mu + \theta]}{\mu(\mu + \delta + \theta)}.$$

Therefore,

$$-\frac{a}{b} = \frac{A[(1-q)\mu + \theta]}{D_c},$$

which confirms that the center-manifold branch is exactly the endemic equilibrium branch.

Finally, we verify the exchange of stability. Along the disease-free branch, the critical multiplier satisfies the following:

$$m_0(\varepsilon) = 1 + a\varepsilon.$$

Thus, the disease-free equilibrium is locally asymptotically stable for $\varepsilon < 0$ sufficiently close to zero and unstable for $\varepsilon > 0$.

Along the nontrivial branch,

$$m_*(\varepsilon) = 1 + a\varepsilon + 2bi_*(\varepsilon) + O(\varepsilon^2).$$

Using

$$i_*(\varepsilon) = -\frac{a}{b}\varepsilon + O(\varepsilon^2),$$

we obtain

$$m_*(\varepsilon) = 1 - a\varepsilon + O(\varepsilon^2).$$

Hence, for $\varepsilon > 0$ sufficiently small,

$$|m_*(\varepsilon)| < 1.$$

The two transverse multipliers remain inside the unit circle by continuity. Therefore, the positive endemic equilibrium is locally asymptotically stable for $\beta > \beta_c$ sufficiently close to β_c.

The disease-free and endemic equilibrium branches consequently intersect and exchange stability at $\beta = \beta_c$. This proves that the bifurcation is a nondegenerate forward transcritical bifurcation.

It remains to exclude the other codimension-one bifurcations at E^0. For every biologically admissible $\beta \geq 0$,

$$\lambda_c(\beta) = 1 - \eta + \beta S^0 > 0,$$

because $0 < \eta < 1$. Therefore, the critical multiplier cannot cross -1. The other two multipliers are independent of β and remain strictly inside the unit circle. Thus, no period-doubling bifurcation occurs at E^0 as β varies.

Moreover, all three multipliers of $J^0(\beta)$ are real for every β. Hence, no complex-conjugate pair can cross the unit circle, and a Neimark-Sacker bifurcation cannot occur at E^0.

Theorem 4.2. Assume that $R_0 > 1$, so that the endemic equilibrium E^* exists. Let

$$f(\lambda) = \lambda^3 + p_1\lambda^2 + p_2\lambda + p_3$$

be the characteristic equation of the Jacobian matrix $J^* = J(E^*)$, where p_1, p_2, p_3 are given in Section 3. Then the following statements hold:

(i) No transcritical bifurcation occurs at E^* in the interior region $R_0 > 1$;
(ii) A period-doubling bifurcation at E^* can occur only if

$$f(-1) = 0,$$

together with the corresponding nondegeneracy and transversality conditions;
(iii) A Neimark-Sacker bifurcation at E^* can occur only if

$$f(1) > 0, \qquad -f(-1) > 0, \qquad |p_3| < 1,$$

and

$$p_2 - p_1 p_3 = 1 - p_3^2,$$

together with the nonresonance and transversality conditions.

In particular, if the equations in (ii) or (iii) have no solution in the biologically admissible parameter region, then the corresponding bifurcation does not occur in that region. If they can only be satisfied after relaxing the admissible parameter restrictions, the resulting bifurcations should be regarded as mathematical extensions of the discrete map.

Proof. At the endemic equilibrium E^*, the characteristic equation of J^* is

$$f(\lambda) = \lambda^3 + p_1\lambda^2 + p_2\lambda + p_3.$$

First, we consider the possibility of a transcritical bifurcation. Such a bifurcation requires that 1 be an eigenvalue of J^*, namely

$$f(1) = 0.$$

By direct calculation, one obtains the following:

$$f(1) = 1 + p_1 + p_2 + p_3 = \frac{\mu(\alpha + \omega + \mu + d)(\mu + \theta + \delta)(R_0 - 1)}{1 + \gamma I^*}.$$

Since $R_0 > 1$, $\mu > 0$, $\alpha + \omega + \mu + d > 0$, $\mu + \theta + \delta > 0$, and $1 + \gamma I^* > 0$, it follows that

$$f(1) > 0.$$

Therefore, $\lambda = 1$ cannot be an eigenvalue of J^* when E^* lies in the interior region $R_0 > 1$. Hence, no transcritical bifurcation occurs at E^* in this region.

Next, a period-doubling bifurcation can only occur when a real eigenvalue of J^* crosses -1. This requires

$$f(-1) = 0.$$

Thus, $f(-1) = 0$, together with the standard nondegeneracy and transversality conditions, gives the possible period-doubling bifurcation condition.

Finally, a Neimark-Sacker bifurcation can only occur when a pair of complex conjugate eigenvalues of J^* crosses the unit circle. For the cubic polynomial

$$f(\lambda) = \lambda^3 + p_1\lambda^2 + p_2\lambda + p_3,$$

this boundary is characterized by

$$f(1) > 0, \qquad -f(-1) > 0, \qquad |p_3| < 1,$$

and

$$p_2 - p_1 p_3 = 1 - p_3^2,$$

together with the corresponding nonresonance and transversality conditions. Hence, the stated Neimark-Sacker condition follows.

Consequently, the occurrence of period-doubling or Neimark-Sacker bifurcations at E^* depends on whether the above algebraic conditions can be satisfied in the admissible parameter region.

5. Numerical simulations

In this section, we first provide a numerical illustration entirely within the biologically admissible parameter region specified by Conditions *(2a)–(2c)*. This example is used to verify the threshold dynamics and the forward transcritical bifurcation established in Theorem 4.1. Then, we examine the more complicated dynamics of the discrete map after Condition *(2a)* is relaxed. The latter simulations are only included as mathematical extensions and are not interpreted as direct epidemiological predictions.

5.1. Numerical illustration in the biologically admissible region

To support the biological interpretation of Theorem 4.1, we take the following:

$$A = 0.25, \quad q = 0.30, \quad \mu = 0.10, \quad d = 0.05,$$

$$\alpha = 0.15, \quad \theta = 0.25, \quad \gamma = 0.50, \quad \delta = 0.15, \quad \omega = 0.20.$$

The initial condition is chosen as follows:

$$(S_0, I_0, V_0) = (1, 0.2, 0.3).$$

Since

$$S_0 + I_0 + V_0 = 1.5 < \frac{A}{\mu} = 2.5,$$

the initial condition belongs to the positively invariant region Ω.

For this parameter set,

$$\eta = \alpha + \omega + \mu + d = 0.50,$$

and the disease-free equilibrium is

$$E^0 = (S^0, 0, V^0) = (1.6, 0, 0.9).$$

The critical transmission rate is as follows:

$$\beta_c = \frac{\eta}{S^0} = 0.3125.$$

We restrict the transmission parameter to the following:

$$\beta \in [0.25, 0.35].$$

Throughout this interval,

$$\mu + \delta + \frac{\beta}{\gamma} \leq 0.10 + 0.15 + \frac{0.35}{0.50} = 0.95 < 1.$$

Moreover,

$$\alpha + \omega + \mu + d = 0.50 < 1, \qquad \mu + \theta = 0.35 < 1.$$

Thus, Conditions *(2a)–(2c)* are satisfied throughout the entire numerical interval.

Figure 2 presents the local equilibrium branches and two representative time-series solutions on opposite sides of the threshold β_c.

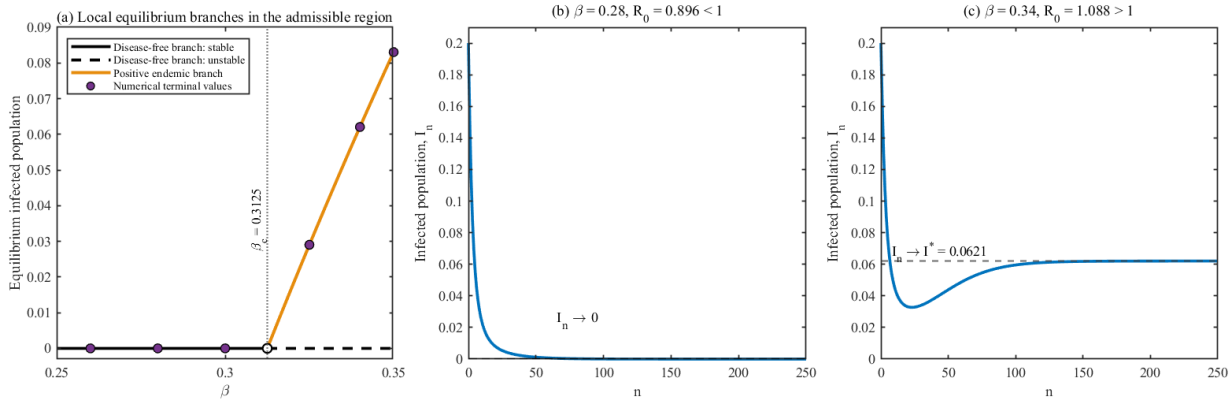


Figure 2. Numerical illustration of the forward transcritical bifurcation within the biologically admissible parameter region. The parameter values are $A = 0.25$, $q = 0.30$, $\mu = 0.10$, $d = 0.05$, $\alpha = 0.15$, $\theta = 0.25$, $\gamma = 0.50$, $\delta = 0.15$, and $\omega = 0.20$. Throughout $\beta \in [0.25, 0.35]$, Conditions *(2a)–(2c)* are satisfied. (a) Local equilibrium branches and numerical terminal values. The vertical dotted line denotes $\beta_c = 0.3125$. The solid and dashed black curves denote the locally stable and unstable disease-free branches, respectively, while the positive branch denotes the endemic equilibrium. (b) Time series of I_n for $\beta = 0.28$, where $R_0 = 0.896 < 1$ and $I_n \to 0$. (c) Time series of I_n for $\beta = 0.34$, where $R_0 = 1.088 > 1$ and $I_n \to I^* \approx 0.062059$.

For $\beta = 0.28$, we have the following:

$$R_0 = \frac{\beta S^0}{\eta} = 0.896 < 1.$$

As shown in Figure 2(b), the infected population decreases to zero and the solution approaches the disease-free equilibrium

$$E^0 = (1.6, 0, 0.9).$$

For $\beta = 0.34$, we obtain the following:

$$R_0 = \frac{\beta S^0}{\eta} = 1.088 > 1.$$

The corresponding endemic equilibrium is as follows:

$$E^* = (S^*, I^*, V^*) \approx (1.516220, 0.062059, 0.890691).$$

Its total population satisfies the following:

$$S^* + I^* + V^* \approx 2.468970 < \frac{A}{\mu} = 2.5.$$

The eigenvalues of the Jacobian matrix at E^* are approximately

$$0.500825, \qquad 0.912534, \qquad 0.951128,$$

and therefore all lie strictly inside the unit disk. Hence, E^* is locally asymptotically stable for this admissible parameter set.

Figure 2(a) shows that the disease-free equilibrium is stable for $\beta < \beta_c$ and loses stability when β passes through β_c. A positive endemic branch emerges for $\beta > \beta_c$. The numerical terminal values agree with the analytically calculated equilibrium branches. Therefore, the three panels confirm disease extinction for $R_0 < 1$, disease persistence for $R_0 > 1$, and the exchange of stability at $R_0 = 1$ within the biologically admissible parameter region.

5.2. Extended dynamics under relaxed admissibility restrictions

Next, we return to the parameter set

$$A = 0.25, \quad q = 0.30, \quad \mu = 0.10, \quad d = 0.05,$$

$$\alpha = 0.15, \quad \theta = 0.25, \quad \gamma = 0.30, \quad \delta = 0.15, \quad \omega = 0.20.$$

For this parameter set, Conditions *(2b)* and *(2c)* remain satisfied, whereas Condition *(2a)* requires

$$\mu + \delta + \frac{\beta}{\gamma} < 1,$$

or equivalently,

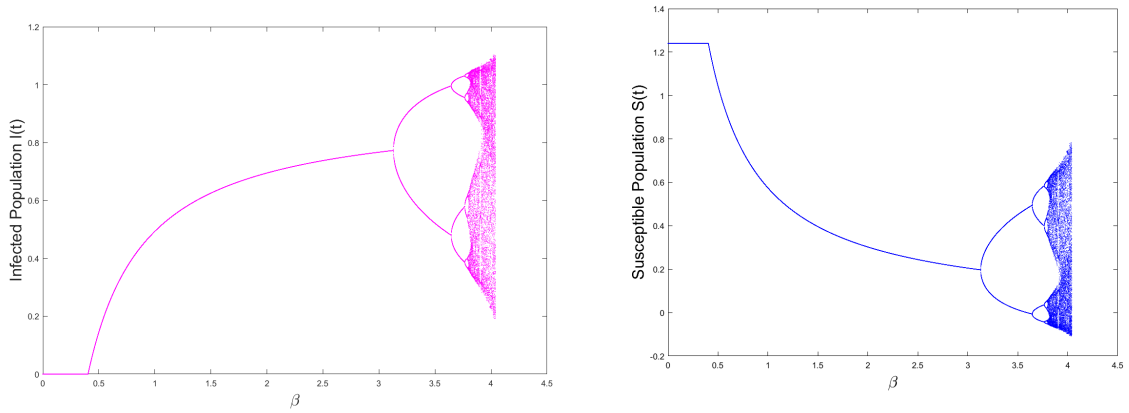
$$\beta < 0.225.$$

Consequently, the simulations in this subsection involving $\beta > 0.225$ lie outside the positivity-preserving parameter region specified in Lemma 2.1. They are only retained to illustrate the possible period-doubling and irregular dynamics of the extended discrete map and should not be interpreted as direct epidemiological predictions.

Figures 3 and 4 illustrate the dynamics of the extended discrete map as the transmission parameter β increases beyond the biologically admissible interval. The numerical solutions exhibit fixed-point, oscillatory, and irregular behaviors. Since Condition *(2a)* is not satisfied in this parameter range, these results are only presented as mathematical properties of the extended map.

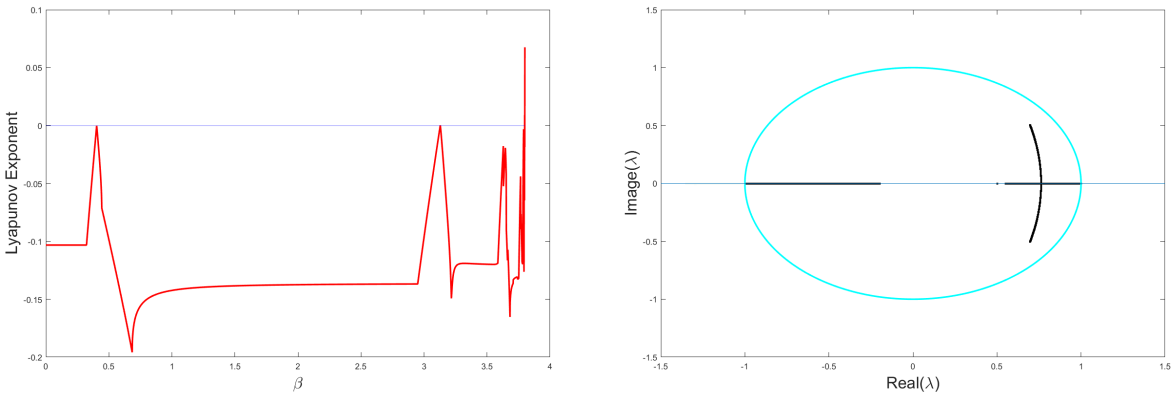
Figure 4(a) shows the variation of the numerical Lyapunov indicator with β, while Figure 4(b) presents the corresponding multipliers in the complex plane. These results are consistent with the transition from a fixed-point behavior to oscillatory and irregular dynamics shown in Figure 3. A

multiplier on the unit circle indicates a possible bifurcation boundary, but the bifurcation type must also satisfy the corresponding transversality and nondegeneracy conditions. Figure 5 presents representative time-series solutions for different values of β. For $\beta = 0.25$, the infected population decreases to zero, while for $\beta = 2.1$, the solution approaches a fixed point. An oscillatory behavior is observed for $\beta = 3.3$, and the orbit for $\beta = 3.9$ displays irregular fluctuations. These solutions are consistent with the qualitative changes shown in Figure 3. Since the corresponding parameter values lie outside the admissible region, no direct epidemiological interpretation is made.



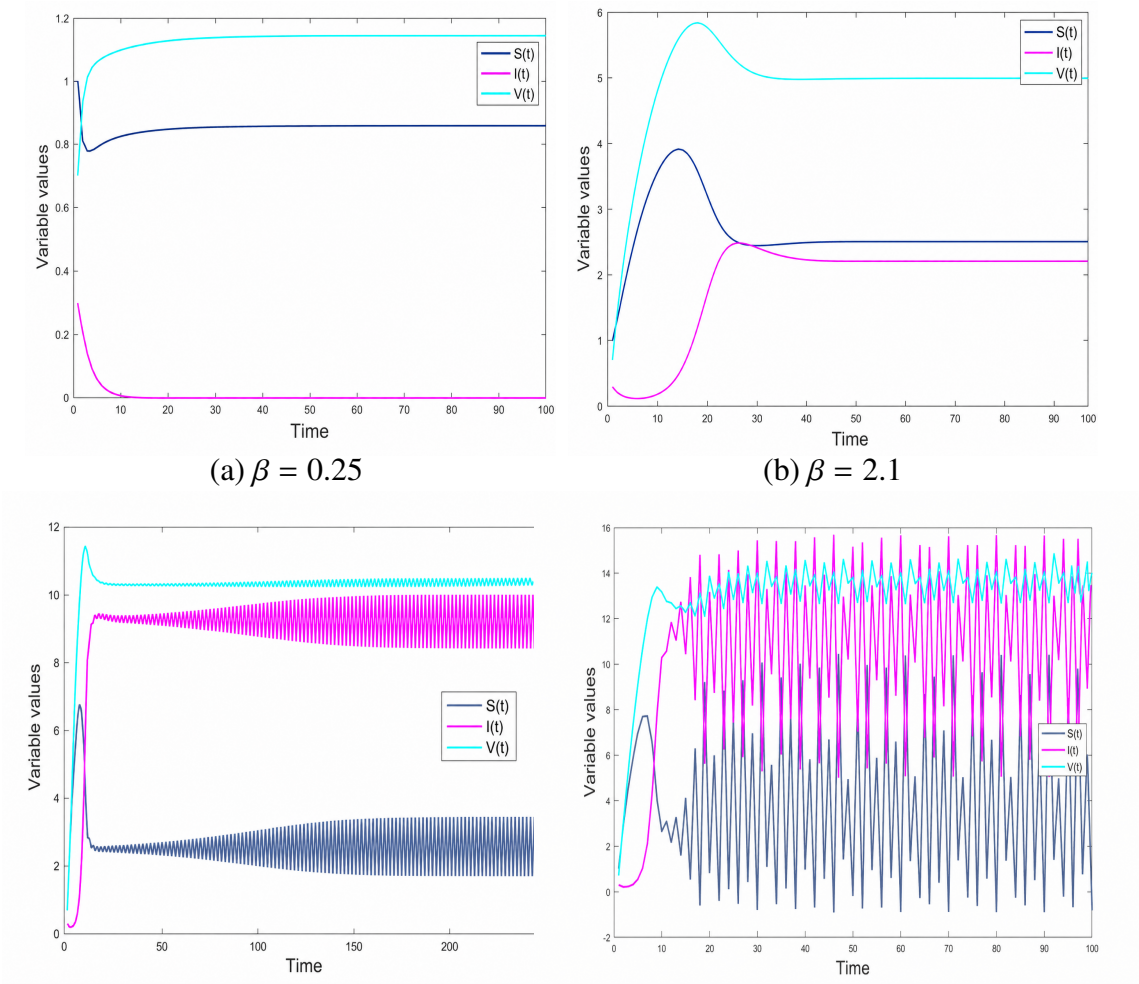
(a) (b)

Figure 3. Bifurcation diagrams of System (2.1) with respect to the transmission parameter β: (a) infected population I_n; (b) susceptible population S_n. Other parameters are fixed at $A = 0.25$, $q = 0.3$, $\mu = 0.1$, $d = 0.05$, $\alpha = 0.15$, $\theta = 0.25$, $\gamma = 0.3$, $\delta = 0.15$, and $\omega = 0.2$.



(a) (b)

Figure 4. The multipliers $\lambda = 1$ and $\lambda = -1$ indicate possible boundaries for transcritical and period-doubling bifurcations, respectively. Moreover, the bifurcation type must satisfy the corresponding transversality and nondegeneracy conditions.

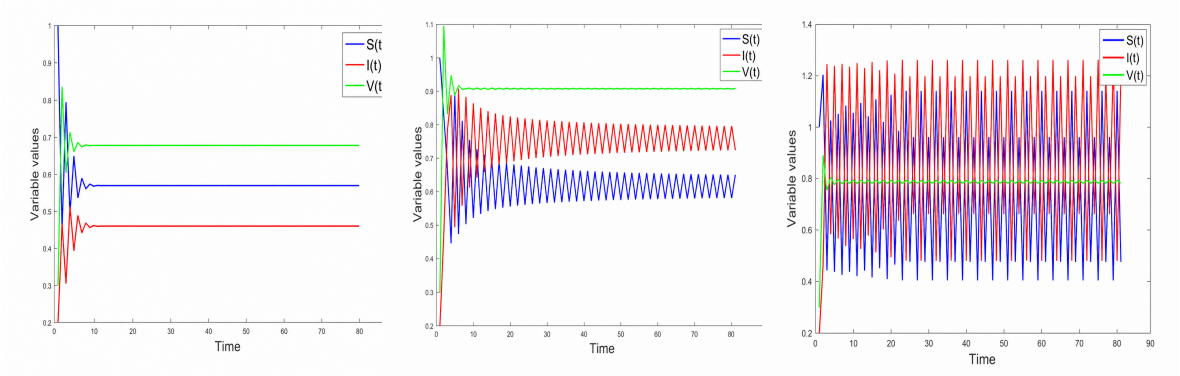


(a) $\beta = 0.25$ (b) $\beta = 2.1$

(c) $\beta = 3.3$ (d) $\beta = 3.9$

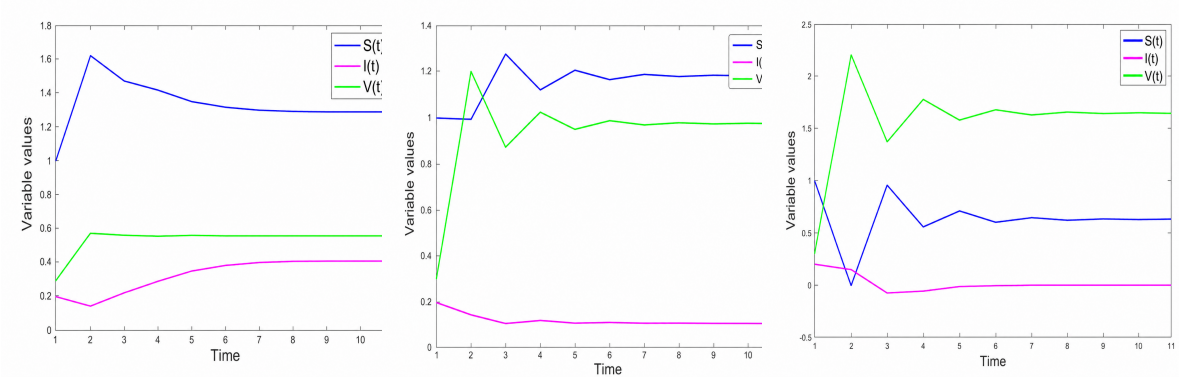
Figure 5. Time-series solutions of System (2.1) for different values of β: (a) $\beta = 0.25$; (b) $\beta = 2.1$; (c) $\beta = 3.3$; and (d) $\beta = 3.9$. These simulations show the transition from disease extinction to persistence and complicated oscillatory behavior.

Figure 6 illustrates the sensitivity of the extended map to the recruitment rate A. Changes in A affect both the equilibrium levels and the long-term numerical behavior. The solutions change from convergence to oscillatory and irregular behaviors as A increases. These simulations are only included to illustrate the parameter dependence of the extended discrete system. Figure 7 illustrates how changes in the vaccination fraction q affect the numerical trajectories of the extended map. Since these simulations are outside the admissible parameter region, no direct epidemiological conclusion is drawn from them.



(a) $A = 1.9$ (b) $A = 2.55$ (c) $A = 2.72$

Figure 6. Time-series solutions of System (2.1) for different recruitment rates A with $\beta = 3.0$: (a) $A = 1.9$; (b) $A = 2.55$; and (c) $A = 2.72$. The results illustrate the influence of population input on the stability of the endemic state.



(a) $q = 0.2$ (b) $q = 0.5$ (c) $q = 0.75$

Figure 7. Time-series solutions of System (2.1) for different vaccination fractions q: (a) $q = 0.2$; (b) $q = 0.5$; and (c) $q = 0.75$. The simulations illustrate the influence of the vaccination fraction q on the numerical solutions of the extended map.

6. Conclusions

In this paper, we studied a discrete-time SIVS epidemic model with vaccinations and a variable population size. The model incorporates constant recruitment, natural and disease-induced mortality, vaccinations of newly recruited and susceptible individuals, loss of vaccine-induced immunity, and two post-infection pathways. Under the stated parameter restrictions, we proved that the solutions remain nonnegative. Additionally, we established a positively invariant region and showed that the total population is ultimately bounded.

We obtained the disease-free equilibrium, the endemic equilibrium, and the basic reproduction number R_0. The disease-free equilibrium is locally asymptotically stable when $R_0 < 1$. The endemic equilibrium exists uniquely when $R_0 > 1$, and its local stability is determined by the Jury conditions.

For global stability, the boundedness of the total population gives the following sufficient condition:

$$\widehat{R}_0 = \frac{\beta A}{\mu(\alpha + \omega + \mu + d)} \leq 1.$$

This auxiliary threshold is only used for the global stability estimate and does not replace the basic reproduction number R_0.

Taking the transmission rate β as the bifurcation parameter, we proved that the disease-free and endemic equilibrium branches undergo a nondegenerate forward transcritical bifurcation at $R_0 = 1$. At the critical point, the critical multiplier is simple, the remaining multipliers lie strictly inside the unit circle, and the transversality and quadratic nondegeneracy conditions are satisfied on the local center manifold. The two equilibrium branches intersect and exchange stability at the threshold. Additionally, we derived the spectral conditions associated with possible period-doubling and Neimark-Sacker bifurcations at the endemic equilibrium.

A numerical example was entirely carried out within the biologically admissible parameter region. The simulations show that the infected population converges to zero when $R_0 < 1$ and approaches a positive endemic equilibrium when $R_0 > 1$. The numerical equilibrium branches agree with the analytical results and confirm the exchange of stability at $R_0 = 1$. Additional oscillatory and irregular dynamics were illustrated after the admissibility restrictions were relaxed. These behaviors should be regarded as mathematical properties of the extended discrete map rather than direct epidemiological predictions.

The model still has several limitations. All parameters are assumed to be constant, and random perturbations, spatial movement, age structure, and time-dependent vaccination are not included. Future work may consider stochastic effects, seasonal transmission, age-structured populations, and vaccination strategies that vary with time. Additionally, it would be useful to study discretization methods that preserve positivity under less restrictive parameter conditions.

Author contributions

All authors contributed to the study conception and design. The theoretical derivation and formal analysis were performed by Jia Xu. The numerical simulations and code implementation were carried out by Ke Wang. Xiaoling Han supervised the project and provided critical revisions. The first draft of the manuscript was written by Author Jia Xu, and all authors commented on previous versions. All authors read and approved the final manuscript.

Use of Generative-AI tools declaration

The authors declare they have not used Artificial Intelligence (AI) tools in the creation of this article.

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Conflict of interest

The authors declare that there are no conflicts of interest regarding the publication of this paper.

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