THE BASIC REPRODUCTION NUMBER OF DISCRETE SIR AND SEIS MODELS WITH PERIODIC PARAMETERS

HUI CAO
School of Science
Shaanxi University of Science & Technology
Xi’an, 710021, China

YICANG ZHOU
Department of Applied Mathematics
Xi’an Jiaotong University
Xi’an, 710049, China

Abstract. Seasonal fluctuations have been observed in many infectious diseases. Discrete epidemic models with periodic epidemiological parameters are formulated and studied to take into account seasonal variations of infectious diseases. The definition and the formula of the basic reproduction number $R_0$ are given by following the framework in [1, 2, 3, 4, 5]. Threshold results for a general model are obtained which show that the magnitude of $R_0$ determines whether the disease will go extinct (when $R_0 < 1$) or not (when $R_0 > 1$) in the population. Applications of these general results to discrete periodic SIR and SEIS models are demonstrated. The disease persistence and the existence of the positive periodic solution are established. Numerical explorations of the model properties are also presented via several examples including the calculations of the basic reproduction number, conditions for the disease extinction or persistence, and the existence of periodic solutions as well as its stability.

1. Introduction. Periodic fluctuations are known to be a common phenomenon in outbreaks of many infectious diseases. For diseases such as malaria, diarrhea, and tuberculosis, seasonality plays an important role in the disease dynamics [6, 7]. For childhood diseases, contact rates vary seasonally due to the opening and closing of schools [8, 9, 10]. Periodic birth rates in some populations are also evidenced [11, 12, 13]. It is natural to introduce periodic parameter functions in epidemiological models for disease dynamics that can be influenced by seasonally fluctuating factors.

Periodic epidemic models have been considered by many researchers [14, 15, 16, 17, 18, 19]. There has been a growing interest in the study of discrete time epidemiological models. Allen et al [20, 21, 22] and Castillo-Chavez et al [23, 24, 25] studied deterministic and stochastic discrete SI, SIR, and SIS models. Other studies involving discrete epidemiological models include Zhou and Paolo [26](age-structured SIS model); Zhou and Ma [27](age-structured SIS models with immigration); Li and Wang [28](stage-structured model); and Franke and Yakubu [29](seasonal variation in environment). One of the reasons for this upsurge of interest in discrete time
models is that these types of models have advantages in modeling infectious disease due to the fact that epidemic data are usually collected in discrete time units, which would make it more convenient to use discrete-time models [30].

One of the most important concepts in epidemiological modeling is the basic reproduction number, denoted by $R_0$, which is defined as the expected number of secondary cases produced by a typical infective individual in a completely susceptible population during his/her entire period of infection [1]. The quantity of $R_0$ is often used to assess how likely an infectious disease can spread in a population [2]. Diekmann et al [1], van den Driessche and Watmough [2] have considered continuous models and presented general approaches for computing $R_0$. Allen and van den Driessche [4] have investigated a large class of discrete-time epidemic models and illustrated the method for computing $R_0$ using the next generation matrix approach.

For a class of continuous epidemic or population models with periodic coefficients, the definition and threshold property of the basic reproduction number have been investigated [3, 14, 16, 31]. The biological interpretation and the threshold property of $R_0$ can be found in these studies. As in the case of continuous-time models, it will be very helpful to define a parallel quantity, $R_0$, for discrete epidemic models with periodic parameter functions and provide a method for computing $R_0$. Bacaër [5] has defined the basic reproduction number for periodic matrix population models, and presented an explicit formula for $R_0$. Particularly, some inequalities that connect the growth rate $\lambda$ and the basic reproduction number $R_0$ is established in [5].

The central ideal in defining the basic reproduction number for an epidemic model is to find an expression for new infections generated by a typical infectious individual. The general framework and procedures for doing this include: (1) rearranging the order of model equations and separating new infection terms from stage transition terms; (2) linearizing the model at the disease-free state; (3) defining a linear operator and finding its spectral radius of the linear operator. It is often the case that the spectral radius of the linear operator is the basic reproduction number for the epidemic model. For autonomous continuous ordinary differential equation models or autonomous difference equation models, the linear operator is an $n \times n$ matrix, and it is relatively easier to derive an explicit formula for the basic reproduction number $R_0$, or at least, to give an explicit expression for the linear operator or matrix. However, for continuous epidemic models with periodic parameters, the operator is usually a linear integral operator defined on a space of continuous and periodic functions, in which case, it is not easy to obtain an explicit formula for the basic reproduction number $R_0$. The common approach for these periodic models is to calculate $R_0$ numerically.

In this paper, we define the basic reproduction number $R_0$ for the discrete periodic epidemic models in a general setting. We consider a linear operator matrix $L$ (the spectral radius of $L$ is $R_0$), present the explicit formula for $R_0$, and establish the threshold result. In our applications, we also consider the persistence of the disease when $R_0 > 1$ and the existence of positive periodic solution. Particularly, it is shown that $R_0 > 1$ is the condition for persistence as well as the existence of the periodic solution.

The paper is organized as follows. Some properties of a linear periodic difference system are discussed at the end of the introduction section. In section 2, we present a general discrete periodic model and outline the process in which the basic reproduction number $R_0$ is defined. An explicit formula of $R_0$ is presented and
the stability of the disease free periodic state is studied. In section 3, the periodic
discrete SIR and SEIS models are discussed to demonstrate the application of our
general results. The sufficient conditions for the persistence and the existence of
the periodic solution of those periodic discrete SIR and SEIS models are given.
The global stability of the periodic solution for each of the models is investigated
numerically. Discussions and concluding remarks are included in section 4.

In the remainder of this section, we present some results for linear periodic dif-
fERENCE systems. Consider the following \( \omega \)-periodic difference system (\( \omega \) is a natural
number):

\[
u(t + 1) = A(t)u(t), \quad t = 0, 1, \cdots,
\]

where \( u(t) = (u_1(t), u_2(t), \cdots, u_n(t))^T \) \((n \in \mathbb{Z}^+)\), and \( A(t) \) is an \( n \times n \) matrix
whose entries are function of \( t \) satisfying \( A(t) = A(t + \omega) \). Using the same notation as
in [32], we call \( U(t, s) \) \((t \geq s, s = 0, 1, \cdots)\) the evolution operator of system (1),
that is, \( U(t, s) = A(t - 1)A(t - 2) \cdots A(s) \), and \( U(s, s) = I \), where \( I \) is the identity
matrix. We have two lemmas on the spectral radius of \( U(t, s) \).

**Lemma 1.1.** The spectral radius of \( U(i + \omega, i) \) \((i = 0, 1, \cdots, \omega - 1)\) is the same as
that of \( U(\omega, 0) \) [5].

For any given \( s \), the periodicity of \( A(t) \) implies that there exists an \( i \), \((i =
0, 1, \cdots, \omega - 1)\), such that \( U(i + \omega, i) = U(s + \omega, s) \), and \( \rho(U(s + \omega, s)) = \rho(U(i + \omega, i)) \).
So we have \( \rho(U(\omega, 0)) = \rho(U(s + \omega, s)) \), \( s = 0, 1, \cdots \).

**Lemma 1.2.** The solution of system (1) is globally asymptotically stable if and only
if \( \rho(U(\omega, 0)) < 1 \). The solution is unstable if \( \rho(U(\omega, 0)) > 1 \).

The solution of system (1) is \( u(t) = U(t, 0)u(0) \). For any \( t = 0, 1, \cdots \), the
periodicity of \( A(t) \) implies that there are positive numbers \( k \) and \( i \) such that

\[
U(t, 0) = A(t - 1)A(t - 2) \cdots A(0) = A(i)A(i - 1) \cdots A(0)U^k(\omega, 0),
\]

where \( 0 \leq i \leq \omega - 1 \), \( k = \text{mod}(t, \omega) \). Since the conclusion of Lemma 1.2 can be
obtained from [32, 33], the detailed proof is omitted.

From those two lemmas, we know that the spectral radius of \( U(\omega, 0) \) determines
the stability of the solution of system (1). The solution of the system is globally
asymptotically stable if \( \rho(U(\omega, 0)) < 1 \), and unstable if \( \rho(U(\omega, 0)) > 1 \).

2. The basic reproduction number and the stability of the disease free
periodic state. Consider an infectious disease with seasonal fluctuation spreading
in a population. We group individuals into \( n \) epidemiological compartments. Let \( x = (x_1, x_2, \cdots, x_n) \), with \( x_i \geq 0 \) \((i = 1, 2, \cdots, n)\) denoting the number of individuals in the \( ith \) compartment. According to their epidemiological status we sort these compartments so that the first \( m \) \((m < n)\) compartments correspond to
infected individuals, and the remaining \( n - m \) compartments correspond to uninfected
individuals. Let \( F_i(t, x) \) be the number of newly infected individuals of the
\( ith \) compartment at time \( t \), \( V_i^+(t, x) \) be the number of individuals transferring into
compartment \( i \) by all other means (e.g., death, recovery, etc.) at time \( t \), and \( V_i^-(t, x) \) be the number of individuals transferring out of compartment \( i \)
(e.g., death, recovery, etc.) at time \( t \). The disease transmission model is represented
by the following non-autonomous difference system:

\[
x_i(t + 1) = F_i(t, x(t)) + V_i(t, x(t)) \triangleq f_i(t, x(t)), i = 1, 2, \cdots, n, t = 0, 1, \cdots,
\]
where $V_i(t, x(t)) = V_i^+(t, x(t)) - V_i^-(t, x(t))$.

Assume there exists a positive invariant set $\mathcal{X} \subseteq \mathbb{R}_n^+ := \{(x_1, x_2, \ldots, x_n) \in \mathbb{R}^n \mid x_i \geq 0, i = 1, 2, \ldots, m\}$, such that $f(t, x(0)) \geq 0$ and $f(t, x(0)) \in \mathcal{X}$ for any $x(0) \in \mathcal{X}$, with $f(t, x(t)) = (f_1(t, x(t)), f_2(t, x(t)), \ldots, f_n(t, x(t)))^T$. This assumption is reasonable since the total population is usually bounded due to the resource limitation, and we study the solution dynamics of model (2) with the initial values located in the state space $\mathcal{X}$. Further, we assume that model (2) has a unique disease free periodic state

$$x^0(t) = (0, \ldots, 0, x_{m+1}^0(t), \ldots, x_n^0(t)),$$

$x^0(t) \in \mathcal{X}$, with $x_i^0(t) \geq 0$, $m + 1 \leq i \leq n$ for all $t = 0, 1, \ldots$, and there exists at least a $j$, $m + 1 \leq j \leq n$, such that $x_j^0(t) > 0$. The disease free periodic state $x^0(t)$ may be an equilibrium or a period solution, depending on the parameters in the model.

Following the framework and notations used in [2, 3, 4], we make following assumptions:

(A1) For each $1 \leq i \leq n$, the functions $F_i(t, x)$, $V_i^+(t, x)$ and $V_i^-(t, x)$ are all non-negative and continuous on $\mathbb{R}_n^+$ and continuously differentiable with respect to $x$;

(A2) There is a natural number $\omega > 0$ such that for each $1 \leq i \leq n$, the functions $F_i(t, x)$, $V_i^+(t, x)$ and $V_i^-(t, x)$ are $\omega$-periodic in $t$;

(A3) If $x_i = 0$, then $V_i^- = 0$;

(A4) $F_i = 0$ for $i > m$;

(A5) $F_i(t, x^0(t)) = V_i^+(t, x^0(t)) = 0$ for $i = 1, 2, \ldots, m$.

These assumptions are based on epidemiological interpretations as well as mathematical requirements. (A1) is the natural assumption for the uniqueness and existence of solutions. (A2) describes a periodic environment (due to seasonal variations in climate and social activities). (A3) comes from the fact that there is no transfer of individuals out of the compartment if a compartment is empty. (A4) represents that the incidence of infection for the uninfected compartment is zero. (A5) implies that the population will remain free of disease if it is free of disease at the beginning.

The Jacobian matrix $D_x f(t, x^0(t))$ of $f(t, x)$ at the disease free periodic state $x^0(t)$ is $D_x F(t, x^0(t)) + D_x V(t, x^0(t))$. $D_x F(t, x^0(t))$ and $D_x V(t, x^0(t))$ can be partitioned as

$$D_x F(t, x^0(t)) = \begin{pmatrix} F(t) & 0 \\ 0 & 0 \end{pmatrix}, \quad D_x V(t, x^0(t)) = \begin{pmatrix} V(t) & 0 \\ T(t) & C(t) \end{pmatrix},$$

where $F(t)$ and $V(t)$ are the $m \times m$ matrices, $C(t)$ is an $(n-m) \times (n-m)$ matrix, and $T(t)$ is a $(n-m) \times m$ matrix defined by

$$F(t) = \left( \frac{\partial F_i(t, x^0(t))}{\partial x_j} \right)_{1 \leq i, j \leq m}, \quad V(t) = \left( \frac{\partial V_i(t, x^0(t))}{\partial x_j} \right)_{1 \leq i, j \leq m},$$

$$C(t) = \left( \frac{\partial V_i(t, x^0(t))}{\partial x_j} \right)_{m+1 \leq i, j \leq n}, \quad T(t) = \left( \frac{\partial V_i(t, x^0(t))}{\partial x_j} \right)_{m+1 \leq i \leq n, 1 \leq j \leq m}.$$
Let $U_C(t, s)$ and $U_V(t, s)$ $(t \geq s)$ be the evolution operators of the linear $\omega$-periodic systems
\begin{equation}
y(t + 1) = C(t)y(t), \quad \text{and} \quad u(t + 1) = V(t)u(t),
\end{equation}
respectively. The internal evolution of individuals in the infectious compartments is dissipative due to deaths and movements. The loss of infective members from natural mortalities and disease-induced mortalities may lead exponential decays [3]. So we can assume
\begin{equation}
(A6) \quad \rho(U_C(\omega, 0)) < 1, \quad \text{and} \quad \rho(U_V(\omega, 0)) < 1.
\end{equation}
It follows from Lemma 1.2 that the zero solutions of $y(t + 1) = C(t)y(t)$ and $u(t + 1) = V(t)u(t)$ are asymptotically stable if assumption (A6) holds.

Under those assumptions, we can define the basic reproduction number for the discrete periodic epidemic model. Let the population be near the disease-free periodic state $x^0(t)$ of (2), and let $\phi(s)$, $\omega$-periodic in $s$, be the initial distribution of infectious individuals. Then $F(s)\phi(s)$ is the distribution of new infections produced by the infected individuals who were introduced at time $s$. Given $t \geq s$, then $U_V(t, s + 1)F(s)\phi(s)$ gives the distribution of these infected individuals who were newly infected at time $s$ and remain in the infected compartments at time $t$. It follows that
\begin{equation}
\varphi(t) = \sum_{s=-\infty}^{t-1} U_V(t, s + 1)F(s)\phi(s) = \sum_{a=1}^{\infty} U_V(t, t - a + 1)F(t - a)\phi(t - a)
\end{equation}
is the distribution of accumulative new infectious at time $t$ produced by all those infected individuals $\phi(s)$ introduced at previous time to $t$.

Let $C_\omega$ be the order Banach space of all $\omega$-periodic discrete functions from $N$ to $R^n$, equipped with the maximum norm $\| \cdot \|$. Let $C_\omega^+ = \{ \phi \in C_\omega \mid \phi(t) \geq 0, \forall t = 0, 1, \ldots \}$ be the positive cone in $C_\omega$. Then we can define a linear operator $L : C_\omega \rightarrow C_\omega$ by
\begin{equation}
(L\phi)(t) = \sum_{a=1}^{\infty} U_V(t, t - a + 1)F(t - a)\phi(t - a), \phi \in C_\omega.
\end{equation}
Obviously, the operator $L$ is continuous and compact on $C_\omega$, and $L$ is positive in the sense that $L(C_\omega^+) \subset C_\omega^+$. We call $L$ the next infection operator. The basic reproduction number of model (2) is defined to be the spectral radius of $L$,
\begin{equation}
R_0 = \rho(L).
\end{equation}
The similar linear operator $(\hat{L}\phi)(t) = \sum_{a=1}^{\infty} F(t)U_V(t, t - a + 1)\phi(t - a)$ was introduced in [5]. If we define $A(\phi)(t) = \sum_{a=1}^{\infty} U_V(t, t - a + 1)\phi(t - a)$ and $B(\phi)(t) = F(t)\phi(t)$, we can have $L = AB$, $\hat{L} = BA$. Therefore, $\rho(L) = \rho(\hat{L})$.

When $V(t)$ is reducible, we can define $V_\epsilon(t) = V(t) + \epsilon E$, where $\epsilon$ is a non-negative positive number, and $E$ is the $m \times m$ matrix with each element being 1. Then $V_\epsilon(t)$ is non-negative and irreducible for each $t \in N$. Let $U_{V_\epsilon}(t, s)$ be the evolution operator of the linear system $u(t + 1) = V_\epsilon(t)u(t)$. The linear operator $L_\epsilon$ can be defined by replacing $U_V(t, s + 1)$ in (3) with $U_{V_\epsilon}(t, s + 1)$. The corresponding basic reproduction number is $R_\epsilon^0 = \rho(L_\epsilon)$. Following the idea in [3], we can have the lemma.
Lemma 2.1. Let (A1)-(A6) hold. Then, \( \lim_{t \to 0^+} \rho(U_{F+V}^t(\omega, 0)) = \rho(U_{F+V}^t(\omega, 0)) \) and \( \lim_{t \to 0^+} R_0^t = R_0 \).

In order to investigate the stability of the disease-free periodic state, we consider the following linear \( \omega \)-periodic equation:

\[
w(t + 1) = \left( V(t) + \frac{F(t)}{\lambda} \right) w(t), \quad t = 0, 1, \ldots, \tag{6}
\]

with parameter \( \lambda \in (0, \infty) \). Let \( W(t, s, \lambda) \), \( t \geq s, \ s = 0, 1, \ldots \), be the evolution operator of the system (6). For each \( \lambda \in (0, \infty) \), the matrix \( V(t) + \frac{F(t)}{\lambda} \) is cooperative. The linear operator \( W(t, s, \lambda) \) is positive for each \( t \geq s, \ s = 0, 1, \ldots \). The Perron-Frobenius theorem (Theorem A.3 in [34]) implies that \( \rho(W(\omega, 0, \lambda)) \) is an eigenvalue of \( W(\omega, 0, \lambda) \) with a nonnegative eigenvector. Lemma 1.1 implies, for any \( s = 0, 1, \ldots \), that the spectral radius of \( W(s + \omega, s, \lambda) \) is the same as that of \( W(\omega, 0, \lambda) \), that is, \( \rho(W(s + \omega, s, \lambda)) = \rho(W(\omega, 0, \lambda)) \).

Lemma 2.2. Let (A1)-(A6) hold. Then

(i) \( R_0 > 0 \) if and only if \( \rho(W(\omega, 0, \lambda)) = 1 \) has a unique positive solution for some \( \lambda \).

(ii) \( R_0 = 0 \) if only if \( \rho(W(\omega, 0, \lambda)) < 1 \) for all \( \lambda > 0 \).

Proof. (i) From [5], we know \( R_0 > 0 \) is the unique positive root of \( \rho(W(\omega, 0, \lambda)) = 1 \). It implies that (i) holds.

(ii) The conclusion in (i) says that \( R_0 > 0 \) if and only if \( \rho(W(\omega, 0, \lambda)) = 1 \) has a positive solution for some \( \lambda \). Thus, \( R_0 = 0 \) if and only if \( \rho(W(\omega, 0, \lambda)) < 1 \) for all \( \lambda \in (0, \infty) \). By the continuity of the spectrum for matrices, it follows that \( \rho(W(\omega, 0, \lambda)) \) is continuous in \( \lambda \in (0, \infty) \) and

\[
\lim_{\lambda \to \infty} \rho(W(\omega, 0, \lambda)) = \rho(U_V^t(\omega, 0)) < 1.
\]

This implies that \( R_0 = 0 \) if and only if \( \rho(W(\omega, 0, \lambda)) < 1 \) for all \( \lambda \in (0, \infty) \).

Since \( \rho(L) = \rho(\tilde{L}) \), we use the expression in [5] directly to obtain \( R_0 = \rho(M) \), where \( M \) is the product of matrices \( M_F \) and \( M_{V^{-1}} \) defined by

\[
M_F = \begin{pmatrix} F(0) & 0 & \cdots & 0 \\ 0 & F(1) & \cdots & \vdots \\ \vdots & \ddots & \ddots & 0 \\ 0 & \cdots & 0 & F(\omega - 1) \end{pmatrix}, \quad \text{and}
\]

\[
M_{V^{-1}} = \begin{pmatrix} -V(0) & I & 0 & \cdots & 0 \\ 0 & -V(1) & I & \ddots & \vdots \\ \vdots & \ddots & \ddots & \ddots & 0 \\ 0 & \cdots & \cdots & I & \vdots \\ I & 0 & \cdots & 0 & -V(\omega - 1) \end{pmatrix}^{-1}.
\]

The equivalent definition of \( R_0 = \rho(M) \) and the expression of matrix \( M \) given in [5] has the advantage that reduces the computation of the spectral radius of the linear operator \( L \) (or \( \tilde{L} \)) to the computation of an eigenvalue of the matrix \( M \). The
inverse matrix $M_V^{-1}$ can also be written as a product of the two matrices, e.g., in the case $\omega = 2$,

$$M_V^{-1} = \begin{pmatrix} (I - V(1)V(0))^{-1} & 0 \\ 0 & (I - V(0)V(1))^{-1} \end{pmatrix} \begin{pmatrix} V(1) & I \\ I & V(0) \end{pmatrix}.$$ 

For the constant environment, [4] shows that $R_0 = \rho(F(I - V)^{-1})$, that is, for $\forall \bar{x} \neq 0$, $\bar{x} \in X$, we have $F(I - V)^{-1}\bar{x} = R_0\bar{x}$. In order to explain our result in the constant environment is consistent with [4], we take $F(0) = F(1) = \cdots = F(\omega - 1) = F$, $V(0) = V(1) = \cdots = V(\omega - 1) = V$, and chose matrix $M_F$ and $M_V$ are both $2 \times 2$ matrix. Namely,

$$M_F = \begin{pmatrix} F & 0 \\ 0 & F \end{pmatrix}, \quad M_V^{-1} = \begin{pmatrix} (I - V^2)^{-1} & 0 \\ 0 & (I - V^2)^{-1} \end{pmatrix} \begin{pmatrix} V & I \\ I & V \end{pmatrix},$$

and

$$M_F M_V^{-1} = \begin{pmatrix} F(I - V^2)^{-1}V & F(I - V^2)^{-1}V \\ F(I - V^2)^{-1}V & F(I - V^2)^{-1}V \end{pmatrix}.$$ 

Furthermore, we have

$$M_F M_V^{-1} \begin{pmatrix} I \\ I \end{pmatrix} \bar{x} = \begin{pmatrix} F(I - V^2)^{-1}V & F(I - V^2)^{-1}V \\ F(I - V^2)^{-1}V & F(I - V^2)^{-1}V \end{pmatrix} \begin{pmatrix} I \\ I \end{pmatrix} \bar{x}$$

$$= \begin{pmatrix} F(I - V)^{-1} \\ F(I - V)^{-1} \end{pmatrix} \bar{x} = \begin{pmatrix} F(I - V)^{-1}\bar{x} \\ F(I - V)^{-1}\bar{x} \end{pmatrix}, \quad (7)$$

Namely, $R_0$ is the eigenvalue of $M_F M_V^{-1}$ with non-negative eigenvector. Since $M_F M_V^{-1}$ is an non-negative matrix, Perron-Frobenius theorem [34] implies that $R_0$ is the eigenvalue with maximum modular. That is, $R_0$ is the spectral radius of $M_F M_V^{-1}$. Therefore, $\rho(M_F M_V^{-1}) = R_0 = \rho(F(I - V)^{-1})$, which is consistent with [4].

It is natural to expect that the basic reproduction number $R_0$ can characterize the dynamics of epidemiological models. The similar threshold result (see [2, 3, 4]) is given in our next theorem.

**Theorem 2.3.** Assume that (A1)-(A6) hold. Then $R_0 = 1$ ($R_0 > 1$, or $R_0 < 1$) if and only if $\rho(U_{F+V}(\omega, 0)) = 1$ ($\rho(U_{F+V}(\omega, 0)) > 1$, or $\rho(U_{F+V}(\omega, 0)) < 1$). Furthermore, $x^0(t)$ is asymptotically stable if $R_0 < 1$, and unstable if $R_0 > 1$.

**Proof.** The conclusion that $R_0 = 1$ is equivalent to $\rho(U_{F+V}(\omega, 0)) = 1$ can be obtained directly from (i) of Lemma 2.2.

Assume that $R_0 > 1$. The Krein-Rutman ([35], Theorem 7.1) theorem implies that there exists $u(t) > 0$ in $\mathcal{C}_\omega$ such that $Lw(t) = R_0w(t)$. It then follows that $w(t_0) > 0$ for some $0 \leq t_0 \leq \omega - 1$ and $w(t)$ satisfies

$$w(t + 1) = (F(t) + V(t))w(t) + \left(\frac{1}{R_0} - 1\right)F(t)w(t), \forall t \in \mathbb{N}. \quad (8)$$

We first claim that $F(t)w(t) \neq 0$. Otherwise, $F(t)w(t) = 0$, and (8) reduces to

$$w(t + 1) = V(t)w(t), \forall t \in \mathbb{N}. \quad (9)$$

Let $U_V(t, s), t \geq s, s \in \mathbb{N}$ be the evolution operator of the linear system (9). It then follows that

$$w(t_0) = w(t_0 + \omega) = U_V(t_0 + \omega, t_0)w(t_0).$$
This equality implies that \( 1 \in \sigma(U_V(t_0 + \omega, t_0)) \). However, \( \rho(U_V(t_0 + \omega, t_0)) = \rho(U_V(\omega, 0)) < 1 \). It is contradiction.

The constant-variation formula of difference equation [32] yields \( w(t_0) = W(t_0 + \omega, t_0, 1)w(t_0) + h \), with \( h = \sum_{k=t_0}^{t-1} (\frac{1}{R_0} - 1)W(t_0 + \omega, k + 1, 1)F(k)w(k) \). In the case where \( V(t) \) is irreducible for \( 0 \leq t \leq \omega - 1 \), \( W(t, s, 1) \) is strongly positive for each \( t \geq s, s = 0, 1, \cdots \). Since \( F(t)w(t) \neq 0 \), we have

\[
\sum_{k=t_0}^{t-1} (\frac{1}{R_0} - 1)W(t_0 + \omega, k + 1, 1)F(k)w(k) < 0.
\]

It then follows that \((-w(t_0)) - W(t_0 + \omega, t_0, 1)(-w(t_0)) = -h > 0\). The inequality \(-w(t_0) < 0\) and Theorem 7.3 in [35] imply that \( 1 < \rho(W(t_0 + \omega, t_0, 1)) = \rho(U_{F+V}(\omega, 0)) \). In the general case of \( V(t) \), replacing \( V(t) \) with \( V_r(t) \) and using the limiting argument can lead to \( \rho(U_{F+V}(\omega, 0)) \geq 1 \). The equivalency of \( R_0 = 1 \) and \( \rho(U_{F+V}(\omega, 0)) = 1 \) implies that \( \rho(U_{F+V}(\omega, 0)) \neq 1 \), and we have \( \rho(U_{F+V}(\omega, 0)) > 1 \).

Assume that \( \rho(U_{F+V}(\omega, 0)) > 1 \). Then, the beginning of the proof implies that \( R_0 \neq 1 \). Since \( \rho(W(\omega, 0, 1)) = \rho(U_{F+V}(\omega, 0)) > 1 \), Lemma 2.2 (ii) implies that \( R_0 > 0 \). It is sufficient to prove that \( R_0 > 1 \). Suppose, by contradiction, that \( R_0 \in (0, 1) \). In the case where \( V(t) \) is irreducible for each \( 0 \leq t \leq \omega \), we see that \( w(t_0) - W(t_0 + \omega, t_0, 1)w(t_0) = h \) holds with \( h > 0 \). By Theorem 7.3 in [35], it follows that \( 1 > \rho(W(t_0 + \omega, t_0, 1)) = \rho(U_{F+V}(\omega, 0)) \). In the general case of \( V(t) \), replacing \( V(t) \) with \( V_r(t) \) and using the limiting argument, we can obtain \( 1 \geq \rho(U_{F+V}(\omega, 0)) \). This contradiction shows that \( R_0 > 1 \).

The last equivalent conclusion can be obtained directly from those first two.

Finally, from the fact \( \rho(C(t)) < 1 \) and the linearized matrix at \( x^0(t) \)

\[
D_x f(t, x^0(t)) = \begin{pmatrix}
F(t) + V(t) & 0 \\
T(t) & C(t)
\end{pmatrix},
\]

we obtain that \( x^0(t) \) is asymptotically stable (unstable) if \( \rho(U_{F+V}(\omega, 0)) < 1 \) \( (\rho(U_{F+V}(\omega, 0)) > 1 \). The condition \( \rho(U_{F+V}(\omega, 0)) < 1 \) \( (\rho(U_{F+V}(\omega, 0)) > 1 \) is equivalent to \( R_0 < 1 \) \( (R_0 > 1 \).

Theorem 2.3 shows that the magnitude of \( R_0 \) determines the stability of the disease-free periodic state \( x^0(t) \). Furthermore, \( R_0 = 1 \) may be the threshold value for the global stability of the disease-free periodic state, the persistence of the disease, and the existence of the periodic solution of model (2). This conjecture is verified for the discrete periodic SIR and SEIS models in next section.

3. Application examples. In this section, the discrete periodic SIR and SEIS models are studied to illustrate the application of our results. Similar to the discrete epidemic model without the effect of seasonal fluctuation [21, 23, 24, 25], we assume that susceptible individuals become infected with nonlinear probability

\[
1 - G \left( \beta(t) \frac{I(t)}{N(t)} \right)
\]

per unit time, where \( G : [0, \infty) \to [0, 1] \) is a monotone concave probability function with \( G(0) = 1 \), \( G'(x) < 0 \), and \( G''(x) < 0 \) for all \( x \in [0, \infty) \), and \( \beta(t) \) is the transmission function, satisfies \( \beta(t) = \beta(t + \omega) \), and \( 0 \leq \beta(t) \leq 1 \) for \( 0 \leq t \leq \omega - 1 \). For the SIR and SEIS models discussed in this section, \( G \left( \beta(t) \frac{I(t)}{N(t)} \right) \)

is taken to be

\[
1 - \frac{\beta(t)I(t)}{N(t)}.
\]
The main focus in this section is on the global stability of the disease-free periodic state, the persistence, and the existence of the positive periodic solution of those models. The implicit expression of the basic reproduction number $R_0$ is given in the simple case $\omega = 2$ for the SIR model. The calculation of $R_0$ for large $\omega$ can be done numerically.

3.1. SIR model. Let $S(t)$, $I(t)$, and $R(t)$ denote the numbers of individuals in the susceptible, infectious and recovery compartments at time $t$, respectively. The discrete periodic SIR model is

$$
\begin{align*}
I(t+1) &= pS(t) \frac{\beta(t)I(t)}{N(t)} + p(1 - \gamma)I(t), \\
S(t+1) &= \Lambda + pS(t) \left(1 - \frac{\beta(t)I(t)}{N(t)}\right), \\
R(t+1) &= p\gamma I(t) + pR(t),
\end{align*}
$$

(10)

where $\Lambda$ is the constant recruitment of the population, $p$ is the probability of survival after a time unit, $\gamma$ is the probability that an infectious individual gets recovered. $N(t)$ is the total number of population at time $t$, that is, $N(t) = S(t) + I(t) + R(t)$, and $N(t)$ satisfies

$$
N(t+1) = \Lambda + pN(t), \quad \text{or} \quad N(t) = \Lambda \frac{1 - p^t}{1 - p} + p^t N(0).
$$

(11)

The fact $0 < p < 1$ and the last equation in (11) implies that $N^* = \frac{\Lambda}{1 - p}$ is the unique equilibrium of (11), and $N^*$ is globally asymptotically stable, i.e., for any solution $N(t)$ of (11) with positive initial value, $\lim_{t \to \infty} N(t) = N^*$ holds.

We study model (10) in the following compact, positively invariant set

$$
\Omega_1 = \left\{(I, S, R) \in \mathbb{R}_+^3 \mid I \geq 0, \ S \geq 0, \ R \geq 0, \ I + S + R \leq \frac{\Lambda}{1 - p}\right\}.
$$

Let $x = (I, S, R)^\gamma$, and

$$
\mathcal{F}(t, x) = \left( \begin{array}{c}
  pS(t) \frac{\beta(t)I(t)}{N(t)} \\
  0 \\
  0
\end{array} \right), \quad \mathcal{V}(t, x) = \left( \begin{array}{c}
  p(1 - \gamma)I(t) \\
  \Lambda + pS(t) \left(1 - \frac{\beta(t)I(t)}{N(t)}\right) \\
  p\gamma I(t) + pR(t)
\end{array} \right).
$$

The SIR model (10) has a unique disease free state $P^0_1 = \begin{pmatrix} 0 & \frac{\Lambda}{1 - p} & 0 \end{pmatrix}$. The linearization at the disease free state yields $F(t) = p\beta(t)$, $V(t) = p(1 - \gamma)$, $T(t) = \begin{pmatrix} -p\beta(t) \\ p\gamma \end{pmatrix}$, and $C(t) = \begin{pmatrix} p & 0 \\ 0 & p \end{pmatrix}$. It is easy to verify that (10) satisfies (A1)-(A6).

The basic reproduction number $R_0$ of (10) is given by the spectral radius of the matrix $M_F M_V^{-1}$, i.e., $R_0 = \rho(M_F M_V^{-1})$, where $M_F = \text{diag}(p\beta(0), p\beta(1), \ldots, p\beta(\omega - 1))$, and

$$
M_V = \begin{pmatrix}
  -p(1 - \gamma) & 1 & 0 & \cdots & 0 \\
  0 & -p(1 - \gamma) & 1 & \cdots & \vdots \\
  \vdots & \ddots & \ddots & \ddots & \vdots \\
  0 & 0 & \ddots & \ddots & 0 \\
  1 & 0 & \cdots & 0 & -p(1 - \gamma)
\end{pmatrix}.
$$
When $\omega = 2$, the direct calculation gives the explicit formula
\[
R_0 = p^2(1-\gamma)(\beta(0)+\beta(1)) + p\sqrt{p^2(1-\gamma)^2(\beta(0)-\beta(1))^2 + 4\beta(0)\beta(1)}}{2(1-p^2(1-\gamma)^2)}.
\]

The threshold result of model (10) is given in the following theorem.

**Theorem 3.1.** The disease free equilibrium state of model (10) is globally asymptotically stable if $R_0 < 1$. $P_0^0$ is unstable and the disease persists if $R_0 > 1$. Furthermore, there exists at least one positive periodic solution of model (10) if $R_0 > 1$.

**Proof.** By Theorem 2.3, we know that if $R_0 < 1$, the disease-free state $P_0^0$ of (10) is asymptotically stable, while if $R_0 > 1$, $P_0^0$ is unstable.

Next, we discuss the global stability of the disease-free state $P_0^0$ of (10) as $R_0 < 1$. We first consider the following system:
\[
\begin{pmatrix}
I_1(t+1) \\
R_1(t+1)
\end{pmatrix} = A_1 \begin{pmatrix}
I_1(t) \\
R_1(t)
\end{pmatrix}, \quad \text{and} \quad \begin{pmatrix}
I_1(0) \\
R_1(0)
\end{pmatrix} = \begin{pmatrix}
I(0) \\
R(0)
\end{pmatrix},
\]
with $A_1 = \begin{pmatrix} p\beta(t) + p(1-\gamma) & 0 \\
p\gamma & p\end{pmatrix}$. Since $0 \leq p \leq 1$, and Theorem 2.3 shows that $R_0 < 1$ if and only if $\rho(U_{F+V}(\omega,0)) < 1$, which implies that $\lim_{t \to \infty} I_1(t) = \lim_{t \to \infty} R_1(t) = 0$ as $R_0 < 1$. That is, the zero solution of system (12) is globally asymptotically stable when $R_0 < 1$. The fact that $\frac{S(t)}{N(t)} \leq 1$ and the comparison theorem imply that $\lim_{t \to \infty} I(t) = \lim_{t \to \infty} R(t) = 0$ when $R_0 < 1$. Furthermore, since $N(t) = I(t) + S(t) + R(t)$ and $\lim_{t \to \infty} N(t) = N^*$, we obtain $\lim_{t \to \infty} S(t) = \lim_{t \to \infty} (N(t) - I(t) - R(t)) = \frac{\Lambda}{1-p}$. Therefore, we see that the disease free equilibrium state $P_0^0$ of (10) is globally asymptotically stable when $R_0 < 1$.

In the case where $R_0 > 1$, we assume that the total population is $N^*$, and consider the limiting system of model (10):
\[
\begin{align*}
I(t+1) &= pS(t)\frac{\beta(t)I(t)}{N^*} + p(1-\gamma)I(t), \\
S(t+1) &= \Lambda + pS(t)\left(1 - \frac{\beta(t)I(t)}{N^*}\right), \\
R(t+1) &= p\gamma I(t) + pR(t).
\end{align*}
\]

The limiting system (13) possesses the same dynamical property as that for the original system (10) [36, 37].

We denote $\mathcal{X} = \Omega_1$, $\mathcal{X}_0 = \{(I, S, R) \in \mathcal{X} \mid I > 0\}$, and $\partial\mathcal{X}_0 = \mathcal{X}\setminus\mathcal{X}_0$. Let $P : \mathcal{X} \to \mathcal{X}$ be the Poincare map associated with (13), that is,
\[
P(x^0) = \phi(\omega, x^0), \quad \forall \ x^0 \in \mathcal{X},
\]
where $\phi(t, x^0)$ is the unique solution of (13) with $\phi(0, x^0) = x^0$.

Let $\mathcal{M} = \{P_1^0\}$ and
\[
M_0 = \{(I, S, R) \in \partial\mathcal{X}_0 \mid P^q(I, S, R) \in \partial\mathcal{X}_0, \forall \ q \geq 0\}.
\]
It is clear that $M_0 = \{(0, S, 0) \in \partial\mathcal{X}_0 \mid S \geq 0\}$. Furthermore, there is exactly one fixed point $P_1^0 = \left(0, \frac{\Lambda}{1-p}, 0\right)$ of $P$ in $M_0$, since the following equation
\[
S_1(t+1) = \Lambda + pS_1(t)
\]
has a positive equilibrium point $S_*$, which is globally attractive. We obtain that $P_0^\sigma$ is asymptotically stable in $\partial X_0$. By use of Lemma 5.9 in [38], we know that no subset of $\mathcal{M}$ forms a cycle in $\partial X_0$. The definition of $M_\sigma$ implies that $M_\sigma$ is the maximum positive invariant set in $\partial X_0$, that is $P(M_\sigma) \subset M_\sigma$. Therefore, $\Omega(M_\sigma) \in \{P_0^\sigma\}$.

Theorem 2.3 implies that $\rho(U_{F+V}(\omega, 0)) > 1$ if $R_0 > 1$. There exists a small $\eta > 0$ such that $\rho(U_{F+V-M}(\omega, 0)) > 1$ with $M_\eta(t) = \frac{p\beta(t)\eta}{N^*}$. For any given positive constant $\sigma$, the perturbed equation

$$
\tilde{S}(t + 1) = \Lambda + p(1 - \frac{\beta(t)\sigma}{N^*})\tilde{S}(t)
$$

admits a periodic solution

$$
\tilde{S}^*(t, \sigma) = \Lambda + \Lambda \sum_{k=1}^{t-1} p^k \prod_{j=t-k}^{t-1} (1 - \frac{\beta(j)\sigma}{N^*}) + p^t \prod_{i=0}^{t-1} (1 - \frac{\beta(i)\sigma}{N^*})\tilde{S}^*(0, \sigma),
$$

with $\tilde{S}^*(0, \sigma)$ being given by $\frac{\Lambda + \Lambda \sum_{k=1}^{\omega-1} p^k \prod_{j=\omega-k}^{\omega-1} (1 - \frac{\beta(j)\sigma}{N^*})}{1 - p^\omega \prod_{i=0}^{\omega-1} (1 - \frac{\beta(i)\sigma}{N^*})}$. The periodic solution $\tilde{S}^*(t, \sigma)$ of (15) is globally attractive. $\tilde{S}^*(0, \sigma)$ is continuous in $\sigma$. We can choose $\sigma > 0$ small enough such that

$$
\tilde{S}^*(t, \sigma) > S_* - \eta, \forall \ t > 0.
$$

By the continuity of the solutions with respect to initial values, there exists $\sigma^* > 0$ ($\sigma^* < \sigma$) such that $\|I(t^*, S^0(t^*), R^0(t^*)) - P_0^\sigma\| \leq \sigma^*$ leads to $\|\phi(t, (I^0, S^0, R^0)) - \phi(t, P_1^0)\| < \sigma$ for $0 \leq t \leq \omega - 1$. We further claim that

$$
\lim\sup_{q \to \infty} d(P^q(I^0, S^0, R^0), P_1^0) \geq \sigma^*.
$$

If it is not true, then $\lim\sup_{q \to \infty} d(P^q(I^0, S^0, R^0), P_1^0) < \sigma^*$ for some $(I^0, S^0, R^0) \in X_0$. Without loss of generality, we can assume that $d(P^q(I^0, S^0, R^0), P_1^0) < \sigma^*$ for all $q \geq 0$. Then, we have

$$
\|\phi(t, (P^q(I^0, S^0, R^0)) - \phi(t, P_1^0)\| < \sigma, \forall \ 0 \leq t \leq \omega - 1.
$$

For any $t = 0, 1, \cdots$, let $t = q\omega + t'$, where $0 \leq t' \leq \omega - 1$ and $q$ is the greatest integer less than or equal to $\frac{t}{\omega}$. We have

$$
\|\phi(t, (I^0, S^0, R^0)) - \phi(t, P_1^0)\| = \|\phi(t', (P^q(I^0, S^0, R^0)) - \phi(t, P_1^0)\| < \sigma, \forall \ t = 0, 1, \cdots.
$$

From $I(t), S(t), R(t)) = \phi(t, (I^0, S^0, R^0))$, we have $0 < I(t) < \sigma$, and

$$
S(t + 1) \geq \Lambda + pS(t)(1 - \frac{\beta(t)\sigma}{N^*}).
$$

The global attraction of the periodic solution $\tilde{S}^*(t, \sigma)$ of (15) implies that $S(t) \geq S_* - \eta$ if $t$ is large enough. If $t$ is large enough, then we have

$$
I(t + 1) \geq p(1 - \gamma)I(t) + p(S_* - \eta)\frac{\beta(t)}{N^*}I(t).
$$
The above inequality and $\rho(U_{F+V-(\omega,0)}) > 1$ as well as Lemma 1.2 imply that the solution of the system

$$
\begin{align*}
\dot{I}(t+1) & = p(1-\gamma)\dot{I}(t) + p(S_0 - \eta)N^*\dot{I}(t) \\
& = (p(1-\gamma) + p\beta(t) - \frac{p\beta(t)\eta}{N^*})\dot{I}(t)
\end{align*}
$$

(18)

with initial value $\dot{I}(0) = I(0) > 0$ satisfies $\lim_{t \to \infty} \dot{I}(t) = +\infty$. By the comparison theorem, it follows that $\lim_{t \to \infty} I(t) = +\infty$, which is a contradiction. Therefore, (16) holds, $P^0$ is isolated in $\mathcal{X}_0$ and $W^s(P^0) \cap \mathcal{X}_0 = \emptyset$. In fact, $P^0$ is also isolated in $\partial \mathcal{X}_0$ because $N^*$ is globally attractive in $\partial \mathcal{X}_0$. Thus, $P^0$ is isolated in $\mathcal{X}$. From Theorem 1.3.1 and Remark 1.3.1 in [11], it follows that $P$ is uniformly persistent with respect to $(\mathcal{X}_0, \partial \mathcal{X}_0)$. Furthermore, Theorem 3.1.1 in [11] implies the solutions of system (13) are uniformly persistent with respect to $(\mathcal{X}_0, \partial \mathcal{X}_0)$.

By use of Theorem 1.3.6 and Remark 1.3.3 in [11], there exists $x^* \in \mathcal{X}_0$, such that $P(t, x^*) = x^*$. Namely, system (13) has a periodic solution $\phi(t, x^*)$ with $\phi(0, x^*) = x^*$.

The limiting system theory implies that model (10) is persistent when $R_0 > 1$ and has a positive periodic solution.

We use following equivalent system to investigate the stability of the periodic solution of model (10) with $\omega = 2$.

$$
\begin{align*}
\begin{cases}
I(t+1) = p(N^* - I(t) - R(t))\frac{\beta I(t)}{N^*} + p(1-\gamma)I(t), \\
R(t+1) = p\gamma I(t) + pR(t).
\end{cases}
\end{align*}
$$

(19)

In the case $\omega = 2$, we have $\beta(t+1) = \beta(t)$. Let $\beta(2k) = \beta_0$, $\beta(2k+1) = \beta_1$, $k = 0, 1, \cdots$, and we take $\beta_0 = 0$, $\beta_1 > 0$. When we start from $t = 0$, then we have

$$
\begin{align*}
I(1) & = p(1-\gamma)I(0), \\
R(1) & = p\gamma I(0) + pR(0),
\end{align*}
$$

where

$$
\begin{align*}
I(0) & = \frac{N^*(1-p^2)(p^2\beta_1(1-\gamma) + p^2(1-\gamma)^2 - 1)}{p^3\beta_1(1-\gamma)(1-p^2 + p^2\gamma(2-\gamma))}, \\
R(0) & = \frac{p^2\gamma(2-\gamma)I(0)}{1-p^2}.
\end{align*}
$$

When we start from $t = 1$, then we have

$$
\begin{align*}
I(2) & = p(N^* - I(1) - R(1))\frac{\beta I(1)}{N^*} + p(1-\gamma)I(1), \\
R(2) & = p\gamma I(1) + pR(1),
\end{align*}
$$

where

$$
\begin{align*}
I(1) & = \frac{N^*(1-p^2)(1-\gamma)(p^2\beta_1(1-\gamma) + p^2(1-\gamma)^2 - 1)}{p^2\beta_1(1-\gamma)((1-\gamma)(1-p^2) + \gamma(1+p^2(1-\gamma)))}, \\
R(1) & = \frac{\gamma(1+p^2(1-\gamma)I(1)}{(1-\gamma)(1-p^2)}.
\end{align*}
$$

Since $R_0 > 1$ implies that $p^2(1-\gamma)\beta_1 > 1 - p^2(1-\gamma)^2$, we obtain the positive periodic state of (19), $P^*(I^*(t), R^*(t))$ where

$$
(I^*(t), R^*(t)) = \begin{cases} (I(0), R(0)), & t = 2k, \\
(I(1), R(1)), & t = 2k + 1, k = 0, 1, \cdots.
\end{cases}
$$
The linearization at the positive periodic state $P^*$ of model (19) yields
\[
J(t) = \left( p\beta(t)\left(1 - \frac{2I^*(t)}{N^*} - \frac{R^*(t)}{N^*}\right) + p(1 - \gamma) - \frac{p\beta(t)I^*(t)}{\rho}\right).
\]

The straight forward calculation of the eigenvalues of matrix $U_J(\omega,0)$ for $\omega = 2$ leads to
\[
\lambda^2 + b_1 \lambda + b_2 = 0,
\]
where
\[
b_1 = p^2(\beta(1-\gamma)(1 - \frac{2I^*(1)+R^*(1)}{N^*}) + (1-\gamma)^2 - \frac{\gamma\beta I^*(1)}{N^*} + 1),
\]
\[
b_2 = p^4(\beta(1-\gamma)(1 - \frac{2I^*(1)+R^*(1)}{N^*}) + (1-\gamma)^2 - \frac{\beta\gamma I^*(1)}{N^*})
\]
\[
+ p^4 \beta_1\gamma(2-\gamma)I^*(1) N^*.
\]

If $\lambda_1$ and $\lambda_2$ are two roots of the equation $\lambda^2 + b_1 \lambda + b_2 = 0$, then $\rho(U_J(\omega,0)) = \max\{||\lambda_1|| < 1, ||\lambda_2|| < 1\}$. Lemma 1.2 implies that the periodic solution $P^*(I^*(t), R^*(t))$ is asymptotically stable if $||\lambda_1|| < 1$ and $||\lambda_2|| < 1$.

As an application example we take $\Lambda = 1000$, $p = 0.994$, $\gamma = 1/8$, and $\beta = 0.3$ obtaining $R_0 = 1.06498 > 1$. The positive periodic solution is
\[
(I^*(t), R^*(t)) = \begin{cases} 
(502.58064, 9727.77257), & t = 2k, \\
(437.11952, 9731.85158), & t = 2k + 1, \ k = 0,1, \ldots
\end{cases}
\]

The matrix $U_J(\omega,0)$ is
\[
\begin{pmatrix}
0.99922 & -0.00078 \\
0.23157 & 0.98804
\end{pmatrix}
\]
with $\rho(U_J(\omega,0)) = 0.9937 < 1$.

The positive periodic state, therefore, is asymptotically stable.

Next, we illustrate our theoretical results on disease extinction or persistence by numerical simulation. The same parameter values are taken in the simulation, except for $\omega = 12$ and $\beta(t) = \beta_c(0.01 + 0.0005 \cos \frac{\pi}{2} t)$, with $\beta_c = 5$ or $\beta_c = 60$.

In the case where $\beta_c = 5$, we have $R_0 = 0.38154 < 1$, and the disease-free state, $P^F_0(0,166666.67,0)$ is globally asymptotically stable. The solution curves of the infectious individuals $I(t)$ with the initial values $(60,200000,120000)$ and $(100,150000,120000)$ are given in the top plot of Fig 1. We observe that solutions with positive initial values will converge to the disease-free state quickly.

In the case where $\beta_c = 60$, we obtain that $R_0 = 4.57851 > 1$, and the disease-free equilibrium state $P^F_0(0,166666.67,0)$ is unstable. Theorem 3.1 implies that the disease will persist in the population, and model (10) has a positive periodic solution. The numerical simulations demonstrate that the positive periodic solution may be globally asymptotically stable. The positive periodic solution and other two solutions with initial values $(6300,36397.32,124246.27)$ and $(5700,36397.32,124246.2682)$ are shown in the the bottom plot of Fig 1. The plot show that solutions starting from a positive initial value will converge to the positive periodic solution.

The childhood disease which transmits from one child to the other is quasi-instantaneous, and it is governed by periods of school terms and holidays. Therefore, we can use epidemic model with periodic parameters to describe the transmission of childhood disease, such as measles, mumps, chickenpox, rubella, or whooping cough. Measles is an infectious disease suitable for SIR model with periodic parameters. The periodic SIR models has been formulated to describe the transmission of measles [6]. So we can use model (10) to describe the transmission of measles.
3.2. SEIS model. Let us consider the transmission of infectious diseases with an exposed/latent stage. We assume that individuals can’t obtain the lifelong immunity when they get recovered. The population is divided into the susceptibles, the exposed/latent, and the infectious. Let $S(t), E(t)$, and $I(t)$ denote the numbers of individuals in the susceptible, exposed/latent, and infectious compartments at time $t$, respectively. The discrete SEIS model in the constant environment was formulated and studied by [39]. We study the dynamical behavior of the discrete SEIS model in the seasonal environment by introducing periodic parameters. The model is

$$\begin{align*}
E(t+1) &= pS(t) \frac{\beta(t)I(t)}{N(t)} + p(1 - \alpha(t))E(t), \\
I(t+1) &= p\alpha(t)E(t) + p(1 - \gamma)I(t), \\
S(t+1) &= \Lambda + pS(t) \left(1 - \frac{\beta(t)I(t)}{N(t)}\right) + p\gamma I(t),
\end{align*}$$

where $\Lambda, p, \gamma$ and $\beta(t)$ have the same interpretation as that in SIR model (10). $\alpha(t)$ is the progression rate of the exposed/latent individuals becoming infectious, and $\alpha(t) > 0$ is a continuous and periodic function with period $\omega$.

From epidemiological interpretation and mathematical requirement, we assume that

$$(E(0), I(0), S(0)) \in \Omega_2 = \left\{(E, I, S) \in \mathbb{R}_+^3 \mid E + I + S \leq \frac{\Lambda}{1 - p}\right\}.$$ 

The equation for the total population, $N(t) = S(t) + E(t) + I(t)$, implies that $N(t) = \Lambda \frac{1 - p^t}{1 - p} + p^t N(0)$. The domain $\Omega_2$ is a compact, positively invariant set of (20).

Let $x = (E, I, S)^\tau$, and

$$F(t, x) = \begin{pmatrix}
pS(t) \frac{\beta(t)I(t)}{N(t)} \\
0 \\
0
\end{pmatrix}, \quad V(t, x) = \begin{pmatrix}
p(1 - \alpha(t))E(t) \\
p\alpha(t)E(t) + p(1 - \gamma)I(t) \\
\Lambda + pS(t) \left(1 - \frac{\beta(t)I(t)}{N(t)}\right) + p\gamma I(t)
\end{pmatrix}.$$
The SEIS model (20) has a unique disease free equilibrium state \( P^0_2 = \left(0, 0, \frac{\Lambda}{1 - p}\right) \).

The linearization at the disease free equilibrium state yields \( F(t) = \left(0, \rho \beta(t), 0\right) \),

\[
V(t) = \begin{pmatrix}
-\rho(1 - \alpha(t)) & 0 & 0 & \cdots & 0 \\
0 & -\rho(1 - \gamma) & I & \cdots & \cdots \\
\vdots & \ddots & \ddots & \ddots & \vdots \\
0 & 0 & \cdots & \cdots & -\rho(\omega - 1) \\
I & 0 & \cdots & 0 & -\rho(\omega - 1)
\end{pmatrix},
\]

where \( M_V \) is given by the spectral radius of the matrix \( M = \frac{\rho(1 - \alpha(t))}{\rho(1 - \gamma)} \).

It is easy to verify that (20) satisfies (A1)-(A6). The basic reproduction number, \( R_0 \), of (20) is given by the spectral radius of the matrix \( M = \frac{\rho(1 - \alpha(t))}{\rho(1 - \gamma)} \), where \( M = \text{diag}(F(0), F(1), \ldots, F(\omega - 1)) \), and

\[
R_0 = \frac{p(\beta(0)a_1 + \beta(1)a_4) + p\sqrt{[\beta(0)a_1 - \beta(1)a_4]^2 + 4\beta(0)\beta(1)a_2a_3}}{2(1 - p^2 (1 - \gamma)^2)(1 - p^2(1 - \alpha(0))(1 - \alpha(1)))}
\]

where

\[
a_1 = p^3\alpha(0)(1 - \alpha(1)(1 - \gamma) + p\alpha(1), \quad a_2 = p^2(\alpha(0)(1 - \gamma) + \alpha(1)(1 - \alpha(0)),
\]

\[
a_3 = p^2(\alpha(0)(1 - \alpha(1)) + \alpha(1)(1 - \gamma), \quad a_4 = p^3\alpha(1)(1 - \gamma)(1 - \alpha(0)) + p\alpha(0).
\]

The stability of disease free equilibrium state, the persistence of disease, and the existence of the positive periodic solution of model (20) is given in Theorem 3.2.

**Theorem 3.2.** The disease free equilibrium state \( P^0_2 \) of (20) is globally asymptotically stable if \( R_0 < 1 \). The disease-free periodic state \( P^0_2 \) is unstable if \( R_0 > 1 \).

**Proof.** By Theorem 2.3, we know that if \( R_0 < 1 \), the disease free equilibrium state \( P^0_2 \) of (20) is asymptotically stable, while if \( R_0 > 1 \), \( P^0_2 \) is unstable.

We now prove the global stability of the disease free equilibrium state \( P^0_2 \) of (20) when \( R_0 < 1 \). Let us first consider the system

\[
\begin{pmatrix} E_1(t + 1) \\ I_1(t + 1) \end{pmatrix} = A_2 \begin{pmatrix} E_1(t) \\ I_1(t) \end{pmatrix}, \quad \text{and} \quad \begin{pmatrix} E_1(0) \\ I_1(0) \end{pmatrix} = \begin{pmatrix} E(0) \\ I(0) \end{pmatrix},
\]

with \( A_2 = \begin{pmatrix} p(1 - \alpha(t)) & \rho \beta(t) \\ \rho \alpha(t) & p(1 - \gamma) \end{pmatrix} \). Theorem 2.3 shows that \( R_0 < 1 \) if and only if \( \rho(U_{F+V}(\omega, 0)) < 1 \), which implies that \( \lim_{t \to \infty} E_1(t) = \lim_{t \to \infty} I_1(t) = 0 \) if \( R_0 < 1 \). That is, the zero solution of system (21) is globally asymptotically stable when \( R_0 < 1 \). The fact that \( \frac{S(t)}{N(t)} \leq 1 \) and the comparison theorem imply that \( \lim_{t \to \infty} E(t) = \lim_{t \to \infty} I(t) = 0 \) when \( R_0 < 1 \). Furthermore, since \( N(t) = E(t) + I(t) + S(t) \) and \( \lim_{t \to \infty} N(t) = N^* \), we obtain \( \lim_{t \to \infty} S(t) = \frac{\Lambda}{\rho} \). Therefore, we obtain that the disease-free state \( P^0_2 \) of (20) is globally asymptotically stable if \( R_0 < 1 \).
In the case where $R_0 > 1$, we assume that the total population is $N^*$, and consider the limiting system of model (20):

$$
\begin{align*}
E(t+1) &= pS(t)\frac{\beta(t)I(t)}{N^*} + p(1 - \alpha(t))E(t), \\
I(t+1) &= p\alpha(t)E(t) + p(1 - \gamma)I(t), \\
S(t+1) &= \Lambda + pS(t)\left(1 - \frac{\beta(t)I(t)}{N^*}\right) + p\gamma I(t).
\end{align*}
$$

The limiting system (22) possesses the same dynamical property as that of the original system (20) [36, 37].

We denote $\mathcal{X} = \Omega_2$, $\mathcal{X}_0 = \{(E, I, S) \in \mathcal{X} \mid I > 0, E > 0\}$, and $\partial \mathcal{X}_0 = \mathcal{X}\\setminus\mathcal{X}_0$. Let $P : \mathcal{X} \to \mathcal{X}$ be the Poincare map associated with (22), that is,

$$
P(x^0) = \phi(\omega, x^0), \quad \forall x^0 \in \mathcal{X},$$

where $\phi(t, x^0)$ is the unique solution of (22) with $\phi(0, x^0) = x^0$.

Define $\mathcal{M} = \{P_2^0\}$, then $\mathcal{M}$ is a compact and isolated invariant set in $\partial \mathcal{X}_0$. Let

$$
M_\beta = \{(E, I, S) \in \partial \mathcal{X}_0 : P^q(E, I, S) \in \partial \mathcal{X}_0, \forall q \geq 0\}.
$$

If $(E(0), I(0), S(0)) \in \partial \mathcal{X}_0$, then $E(0) = 0$ or $I(0) = 0$. In the case where $E(0) > 0$ and $I(0) = 0$, the first two equations of (22) implies that $E(t) > 0$ and $I(t) > 0$ for $t \geq 0$ if $S(0) > 0$. If $S(0) = 0$, then the third equation of (22) implies that $S(1) > 0$, and we can also have $E(t) > 0$ and $I(t) > 0$ for $t > 1$. Therefore, $(E(0), I(0), S(0)) \notin M_\beta$. In the case where $E(0) = 0$ and $I(0) > 0$, the similar argument yields that $(E(0), I(0), S(0)) \notin M_\beta$. Those conclusions imply that $M_\beta = \{(0, 0, S) \in \partial \mathcal{X}_0 : S \geq 0\}$. Furthermore, the Poincare map $P$ has exactly one fixed point $P_2^0 = \left(0, 0, \frac{\Lambda}{1 - p}\right)$ in $M_\beta$. Since the following equation

$$
S_1(t+1) = \Lambda + pS_1(t)
$$

has a positive equilibrium point $S^* = \frac{\Lambda}{1 - p}$, which is globally attractive, we obtain that $P_2^0$ is asymptotically stable in $\partial \mathcal{X}_0$. From Lemma 5.9 in [38], we know that no subset of $\mathcal{M}$ forms a cycle in $\partial \mathcal{X}_0$. The definition of $M_\beta$ implies that $M_\beta$ is the maximum positive invariant set in $\partial \mathcal{X}_0$, that is $P(M_\beta) \subset M_\beta$. Therefore, $\Omega(M_\beta) \subset \{P_2^0\}$.

The rest part of the proof of Theorem 3.2 is similar to that of Theorem 3.1, and the detailed process is omitted. \qed

We use numerical simulation to demonstrate our theoretical results on disease extinction or persistence. The following parameter values are taken in the simulation: $\omega = 12$, $\Lambda = 1000$, $p = 0.994$, $\gamma = 1/3$, $\alpha(t) = 40 \left(0.008 + 0.002\sin \frac{\pi t}{6}\right)$, and $\beta(t) = \beta_c \left(0.01 + 0.005\cos \frac{\pi t}{6}\right)$, with $\beta_c = 20$ or $\beta_c = 50$.

In the case of $\beta_c = 20$, we obtain that $R_0 = 0.55031 < 1$, and the disease free equilibrium state, $P_2^0(0, 0, 166666.67)$ is globally asymptotically stable. Two solutions of (20) with initial values $(50000, 80000, 100000)$ and $(19600, 26375, 120720)$ are shown in Fig 2. The curves in the top plot represent the numbers of exposed individuals $E(t)$, and the curves in the bottom plot show the numbers of infectious individuals $I(t)$. We observe that solutions with positive initial values will converge to the disease free equilibrium state quickly.
The number of the exposed individuals

The number of the infectious individuals

Figure 2. The stability of the disease free equilibrium state of (20)

In the case of $\beta_c = 50$, we obtain that $R_0 = 1.37578 > 1$, and the disease free equilibrium state $P_0 = (0, 0, 166666.67)$ is unstable. Theorem 3.2 implies that the disease will persist in the population, and model (20) has a positive periodic solution. The numerical simulations demonstrate that the positive periodic solution may be globally asymptotically stable (see Fig 3). The positive periodic solution and other two solutions with the initial values $(100, 10000, 80000)$ and $(50000, 80000, 100000)$ are shown in Fig 3. From these curves in Fig 3, we find that solutions starting from a positive initial value will converge to the positive periodic solution.

Figure 3. The existence and stability of the positive periodic state of (20)

Tuberculosis (TB) is an infectious disease spreading worldwide. TB has a very long latent period, which may last for several months, even decades. The SEIS or SEIR models are often used to describe the transmission of TB. In addition, according to the monthly reporting data about new case of TB from the Chinese Center for Disease Control and Prevention (China CDC), we found an obvious
seasonal variation of TB incidence in China. The periodic SEIS and SEIT models have formulated to study the TB infection in China [7]. Therefore, model (20) can be used to describe the transmission of TB with seasonal fluctuation.

4. Concluding remarks. The discrete epidemic models with periodic parameters or parameter functions are more realistic in describing disease dynamics under the influence of seasonal fluctuation. We define the basic reproduction number for a large class of discrete periodic epidemic model. Although $R_0$ is defined by the radius of a linear operator, it is also the eigenvalue of a specific matrix. It is easy to calculate $R_0$ when the values of the model’s parameters or parameter functions are provided. The possibility of deriving an explicit formula of $R_0$ is one of the advantages over continuous periodic models. Under natural and general assumptions, the threshold theorems are established: the disease free state is asymptotically stable if $R_0 < 1$, and it is unstable if $R_0 > 1$.

After the general framework and theory are established, discrete periodic SIR and SEIS models are discussed as application examples to demonstrate our method and results. The specific expression of $R_0$ for SIR and SEIS models with period $\omega = 2$ are presented. The global stability conditions of the disease free state, the persistence, and the existence of the positive periodic solution are established for those two models. Numerical simulations are done to demonstrate the calculation of $R_0$, the disease extinction, or the persistence, the existence and the stability of the periodic solutions. The numerical simulation provide us with possibility of investigating the dynamical behavior of any model after parameter values are determined.

The limitation of the paper is that we can not give the specific expression $R_0$ as a function of the model parameters though the explicit formula for the matrix $M_F$ and $M_V$ is given. The inverse $M_V^{-1}$, the product $M_F M_V^{-1}$, and the spectral radius $M_F M_V^{-1}$ (i.e., the eigenvalue of $M_F M_V^{-1}$) will be very complicated for the model with more infectious compartments and long period $\omega$. The expression of the positive periodic solution is also very difficult, though the expression of a positive periodic solution can be obtained by the iteration, theoretically. The iteration might be too complicated to manage even with computer software.

Acknowledgments. The authors thank Professor Zhilan Feng of the Purdue University for her valuable suggestions and kind help to prepare the manuscript.

REFERENCES

[1] O. Diekmann, J. Heesterbeek and J. Metz, On the definition and the computation of the basic reproduction ratio $R_0$ in models for infectious disease in heterogeneous populations, J. Math. Biol., 28 (1990), 365–382.

[2] P. van den Driessche and J. Watmough, Reproduction numbers and sub-threshold endemic equilibrium for compartmental models of disease transmission, Math. Biosci., 180 (2002), 29–48.

[3] W. Wang and X. Zhao, Threshold dynamics for compartmental epidemic models in periodic environments, J. Dyn. Diff. Equat., 20 (2008), 699–717.

[4] L. Allen and P. van den Driessche, The basic reproduction number in some discrete-time epidemic models, J. Difference Equations and Applications, 14 (2008), 1127–1147.

[5] N. Bacaër, Periodic matrix population models: growth rate, basic reproduction number, and entropy, Bull. Math. Biol., 71 (2009), 1781-1792.

[6] M. Keeling and P. Rohani, “Modeling Infectious Diseases in Humans and Animals,” Princeton University Press, 2008.
[7] Y. Zhou and H. Cao, Discrete tuberculosis transmission models and their application, in “Fields Communications Series: New Perspectives in Mathematical Biology,” (ed.S. Sivaloganathan), A co-publication of the AMS and Fields Institute, Canada, 57 (2010), 83–112.

[8] I. Schwartz and H. Smith, Infinite subharmonic bifurcation in an SIER epidemic model, J. Math. Biol., 18 (1983), 233–253.

[9] I. Schwartz, Small amplitude, long periodic outbreaks in seasonally driven epidemics, J. Math. Biol., 30 (1992), 473–491.

[10] H. Smith, Multiple stable subharmonics for a periodic epidemic model, J. Math. Biol., 17 (1983), 179–190.

[11] X. Zhao, “Dynamical Sytems in Population Biology,” Springer-Verlag, New York, 2003.

[12] J. M. Cushing, A juvenile-adult model with periodic vital rates, J. Math. Biol., 53 (2006), 520–539.

[13] J. Ma and Z. Ma, Epidemic threshold conditions for seasonally forced SEIR models, Math. Biosci. Eng., 3 (2006), 161–172.

[14] N. Bacaër, Approximation of the basic reproduction number $R_0$ for vector-borne diseases with a periodic vector population, Bull. Math. Biol., 69 (2007), 1067–1091.

[15] N. Bacaër and M. G. M. Gomes, On the final size of epidemics with seasonality, Bull. Math. Biol., 71 (2009), 1954–1966.

[16] N. Bacaër and S. Guernaoui, The epidemic threshold of vector-borne diseases with seasonality, J. Math. Biol., 53 (2006), 421–436.

[17] F. Zhang and X. Zhao, A periodic epidemic model in a patchy environment, J. Math. Anal. Appl., 325 (2007), 496–516.

[18] B. G. Williams and C. Dye, Infectious disease persistence when transmission varies seasonally, Math. Biosci., 145 (1997), 77–88.

[19] H. R. Thieme, Renewal theorems for linear periodic Volterra integral equations, J. Integral Equations, 7 (1984), 253–277.

[20] L. Allen, Some discrete-time SI, SIR, and SIS epidemic models, Math. Biosci., 124 (1994), 83–105.

[21] L. Allen and A. Burgin, Comparison of deterministic and stochastic SIS and SIR models in discrete time, Math. Biosci., 183 (2000), 1–33.

[22] L. Allen, D. Flores, R. Ratnayake and J. Herbold, Discrete-time deterministic and stochastic models for the spread of rabies, Appl. Math. Comput., 132 (2002), 271–292.

[23] C. Castillo-Chavez and A. A. Yakubu, Discrete-time SIS models with complex dynamics, Nonlinear Anal., 47 (2001), 4753–4762.

[24] C. Castillo-Chavez and A. A. Yakubu, Dispersal, disease and life-history evolution, Math. Biosci., 173 (2001), 35–53.

[25] C. Castillo-Chavez and A. A. Yakubu, Discrete-time SIS models with simple and complex population dynamics, in “Mathematical Approaches for Emerging and Reemerging Infectious Diseases: A introduction,” (ed. C. Castillo-Chavez with S. Blower, P. van den Driessche, D. Kirschner, and A. A. Yakubu), Springer-Verlag, New York, (2002), 153–163.

[26] Y. Zhou and P. Fergola, Dynamic of a discrete age-structured SIS models, Discrete Contin. Dyn. Syst. Ser. B., 4 (2004), 843–852.

[27] Y. Zhou and Z. Ma, Global stability of a class of discrete age-structured SIS models with immigration, Math. Biosci. Eng., 6 (2009), 409–425.

[28] X. Li and W. Wang, A discrete epidemic model with stage structure, Chaos, Solitons and Fractals, 26 (2005), 947–958.

[29] J. E. Franke and A. A. Yakubu, Discrete-time SIS epidemic model in a seasonal environment, SIAM J. Appl. Math., 66 (2006), 1563–1587.

[30] Ira M. Longini, Jr., The generalized discrete-time epidemic model with immunity: Asynthesis, Math. Biosci., 82 (1986), 19–41.

[31] N. Bacaër and R. Ouifki, Growth rate and basic reproduction number for population models with a simple periodic factor, Math. Biosci., 210 (2007), 647–58.

[32] M. I. Gil, “Difference Equations in Normed Spaces Stability and Oscillations,” Elsevier Science, 2007.

[33] R. A. Horn and C. A. Johnson, “Matrix Analysis,” Cambridge University press, Cambridge, 1985.

[34] H. Smith and P. Waltman, “Theory of the Chemostat,” Cambridge University Press, Cambridge, 1995.
[35] P. Hess, “Periodic-Parabolic Boundary Value Problems and Positivity,” Pitman Research Notes in Mathematics, Series 247, Longman Scientific and Technical, 1991.

[36] H. R. Thieme, Convergence results and a Poincare-Bendixson trichotomy for asymptotically autonomous differential equations, J. Math. Biol., 30 (1992), 755–763.

[37] X. Q. Zhao, Asymptotic behavior for asymptotically periodic semiflows with applications, Commun. Appl. Nonlinear Anal., 3 (1996), 43–66.

[38] P. Salceanu and H. Smith, Persistence in a discrete-time, stage-structured epidemic model, J. Difference Equa. Appl., 16 (2010), 73–103.

[39] P. A. Gonzalez, R. A. Saenz, B. N. Sanchez, C. Castillo-Chavez and A. A. Yakubu, “Dispersal Between Two Patches in a Discrete Time SEIS Model,” MTBI technical Report, 2000.

Received April 2011; revised May 2012.

E-mail address: caohui0103@163.com
E-mail address: zhouyc@mail.xjtu.edu.cn