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Threshold Dynamics for Compartmental Epidemic Models in Periodic Environments

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Abstract The basic reproduction ratio and its computation formulae are established for a large class of compartmental epidemic models in periodic environments. It is proved that a disease cannot invade the disease-free state if the ratio is less than unity and can invade if it is greater than unity. It is also shown that the basic reproduction number of the time-averaged autonomous system is applicable in the case where both the matrix of new infection rate and the matrix of transition and dissipation within infectious compartments are diagonal, but it may underestimate and overestimate infection risks in other cases. The global dynamics of a periodic epidemic model with patch structure is analyzed in order to study the impact of periodic contacts or periodic migrations on the disease transmission.

Keywords Compartmental models · Reproduction ratio · Periodicity · Threshold dynamics

Mathematics Subject Classification (2000) 34D20 · 37B55 · 92D30

1 Introduction

The basic reproduction number of an infectious disease is a fundamental concept in the study of disease transmissions. It is the expected number of secondary cases produced, in a completely susceptible population, by a typical infective individual. Usually, the basic reproduction number defines the threshold behavior for classical epidemic models. It is a common case that a disease dies out if the basic reproduction number is less than unity and the disease is established in the population if it is greater than unity. For autonomous epidemic models,

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Diekmann et al. [7], van den Driessche and Watmough [31] presented general approaches for the calculations of basic reproduction numbers. Computations of basic reproduction numbers for specific infectious diseases are carried out in [20] for sexual diseases, in [13] for tuberculosis in possums, in [12] for dengue fever, in [15,24,33,40] for SARS. Furthermore, basic reproduction numbers were studied in [1,2] for the epidemic models with population traveling among cities where the residences of individuals are maintained, and in [32,34–36] for the patchy models without the record of residence of individuals.

It is well-known that periodic fluctuations are common in the evolution of disease transmissions. Contact rates vary seasonally for childhood diseases because of opening and closing of schools [5,9,26–28]. Periodic changes in birth rates of populations are evidenced in many biological works, see, e.g., [6,23,39]. Vaccination program is also a source of periodicity [10]. We refer to [3,4,8,14,17,22,25,30,37,38] and references therein for other types of periodic epidemic models. A natural and important problem associated with periodic epidemic models is to define and compute their basic reproduction numbers. Intuitively, one may expect to use the basic reproduction number of the time-averaged autonomous system of a periodic epidemic model over a time period. Unfortunately, this average basic reproduction number is applicable only in certain circumstances, but overestimates or underestimates infection risks in many other cases (see examples in Sect. 3). The effective reproduction number is also used in the literature, which is defined as the average number of secondary cases arising from a single typical infective introduced at time t into the population [11]. Its magnitude is a useful indicator of both the risk of an epidemic and the effort required to control an infection. However, this number is not a threshold parameter to determine whether the disease can invade the susceptible population successfully. Recently, Bacaër and Guernaoui [4] presented a general definition of the basic reproduction number in a periodic environment. The purpose of our current paper is to establish the basic reproduction ratio for a large class of periodic compartmental epidemic models and show that it is a threshold parameter for the local stability of the disease-free periodic solution, and even for the global dynamics under certain circumstances.

The remaining parts of this paper are organized as follows. In the next section, we present the theory of the basic reproduction ratio for periodic compartmental models. Section 3 provides three examples to illustrate the applicability of the basic reproduction number of the time-averaged systems. In Sect. 4, we obtain a threshold condition for the global persistence and extinction of diseases. Based on this result, we analyze an epidemic model with periodic population dispersal and periodic contact rates.

2 The Basic Reproduction Ratio

We consider a heterogeneous population whose individuals can be grouped into n homogeneous compartments. Let $x = (x_1, \ldots, x_n)^T$, with each $x_i \geq 0$, be the state of individuals in each compartment. We assume that the compartments can be divided into two types: infected compartments, labeled by $i = 1, \ldots, m$, and uninfected compartments, labeled by $i = m+1, \ldots, n$. Define X_s to be the set of all disease-free states:

$$X_s := \{x \geq 0 : x_i = 0, \forall i = 1, \ldots, m\}.$$

Let $\mathcal{F}_i(t, x)$ be the input rate of newly infected individuals in the ith compartment, $\mathcal{V}_i^+(t, x)$ be the input rate of individuals by other means (for example, births, immigrations), and $\mathcal{V}_i^-(t, x)$ be the rate of transfer of individuals out of compartment i (for example, deaths, recovery and emigrations). Thus, the disease transmission model is governed by a nonautonomous ordinary differential system:

$$\frac{dx_i}{dt} = \mathcal{F}_i(t,x) - \mathcal{V}_i(t,x) \triangleq f_i(t,x), \quad i = 1,\dots,n, \tag{2.1}$$

where $\mathcal{V}_i = \mathcal{V}_i^- - \mathcal{V}_i^+$. Following the setting of [31] for autonomous compartmental epidemic models, we make the following assumptions:

(A1) For each $1 \le i \le n$, the functions $\mathcal{F}_i(t,x)$, $\mathcal{V}_i^+(t,x)$ and $\mathcal{V}_i^-(t,x)$ are nonnegative and continuous on $\mathbb{R}\times\mathbb{R}_+^n$ and continuously differential with respect to x.
(A2) There is a real number $\omega > 0$ such that for each $1 \le i \le n$, the functions $\mathcal{F}_i(t,x)$, $\mathcal{V}_i^+(t,x)$ and $\mathcal{V}_i^-(t,x)$ are ω-periodic in t.
(A3) If $x_i = 0$, then $\mathcal{V}_i^- = 0$. In particular, if $x \in X_s$, then $\mathcal{V}_i^- = 0$ for $i = 1,\dots,m$.
(A4) $\mathcal{F}_i = 0$ for $i > m$.
(A5) If $x \in X_s$, then $\mathcal{F}_i(x) = \mathcal{V}_i^+(x) = 0$ for $i = 1,\dots,m$.

Note that (A1) arises from the simple fact that each function denotes a directed non-negative transfer of individuals. Biologically, (A2) describes a periodic environment (e.g., due to seasonality); (A3) represents that if a compartment is empty, then there is no transfer of individuals out of the compartment; (A4) means that the incidence of infection for uninfected compartments is zero; and (A5) implies that the population will remain free of disease if it is free of disease at the beginning.

We assume that the model (2.1) has a disease-free periodic solution $x^0(t) = (0,\dots,0,x_{m+1}^0(t),\dots,x_n^0(t))^T$ with $x_i^0(t) > 0, m+1 \le i \le n$ for all t. Let $f = (f_1,\dots,f_n)^T$, and define an $(n-m)\times(n-m)$ matrix

$$M(t) := \left(\frac{\partial f_i(t,x^0(t))}{\partial x_j}\right)_{m+1\le i,j\le n}.$$

Let $\Phi_M(t)$ be the monodromy matrix of the linear ω-periodic system $\frac{dz}{dt} = M(t)z$. We further assume that $x^0(t)$ is linearly asymptotically stable in the disease-free subspace X_s, that is,

(A6) $\rho(\Phi_M(\omega)) < 1$, where $\rho(\Phi_M(\omega))$ is the spectral radius of $\Phi_M(\omega)$.

By the arguments similar to those in [31, Lemma 1], it then follows that

$$D_x\mathcal{F}(t,x^0(t)) = \begin{pmatrix} F(t) & 0 \\ 0 & 0 \end{pmatrix}, \quad D_x\mathcal{V}(t,x^0(t)) = \begin{pmatrix} V(t) & 0 \\ J(t) & -M(t) \end{pmatrix},$$

where $F(t)$ and $V(t)$ are two $m\times m$ matrices defined by

$$F(t) = \left(\frac{\partial \mathcal{F}_i(t,x^0(t))}{\partial x_j}\right)_{1\le i,j\le m}, \quad V(t) = \left(\frac{\partial \mathcal{V}_i(t,x^0(t))}{\partial x_j}\right)_{1\le i,j\le m}, \tag{2.2}$$

respectively, and $J(t)$ is an $(n-m)\times n$ matrix. Furthermore, $F(t)$ is non-negative, and $-V(t)$ is cooperative in the sense that the off-diagonal elements of $-V(t)$ are non-negative.

Let $Y(t,s)$, $t \ge s$, be the evolution operator of the linear ω-periodic system

$$\frac{dy}{dt} = -V(t)y. \tag{2.3}$$

That is, for each $s \in \mathbb{R}$, the $m\times m$ matrix $Y(t,s)$ satisfies

$$\frac{d}{dt}Y(t,s) = -V(t)Y(t,s), \ \forall t \ge s, \quad Y(s,s) = I,$$

where I is the $m\times m$ identity matrix. Thus, the monodromy matrix $\Phi_{-V}(t)$ of (2.3) equals $Y(t,0)$, $t \ge 0$. Note that the internal evolution of individuals in the infectious compartments

due to deaths and movements among the compartments is dissipative, and exponentially decays in many cases because of the loss of infective members from natural mortalities and disease-induced mortalities. Thus, we assume that

(A7) $\rho(\Phi_{-V}(\omega)) < 1$.

Based on the assumptions above, we are now able to analyze the reproduction ratios for the epidemic model (2.1). For this purpose, we always assume that the population is near the disease-free periodic state $x^0(t)$.

By the standard theory of linear periodic systems (see, e.g., [16, Sect. III.7]), there exist $K > 0$ and $\alpha > 0$ such that

$$\|Y(t,s)\| \le Ke^{-\alpha(t-s)}, \quad \forall t \ge s, \quad s \in \mathbb{R}. \tag{2.4}$$

It follows that

$$\|Y(t,t-a)F(t-a)\| \le K\|F(t-a)\|e^{-\alpha a}, \quad \forall t \in \mathbb{R}, \quad a \in [0,\infty). \tag{2.5}$$

In view of the periodic environment, we suppose that $\phi(s)$, ω-periodic in s, is 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 $t \ge s$, then $Y(t,s)F(s)\phi(s)$ gives the distribution of those infected individuals who were newly infected at time s and remain in the infected compartments at time t. It follows that

$$\psi(t) := \int_{-\infty}^{t} Y(t,s)F(s)\phi(s)ds = \int_{0}^{\infty} Y(t,t-a)F(t-a)\phi(t-a)da$$

is the distribution of accumulative new infections at time t produced by all those infected individuals $\phi(s)$ introduced at previous time to t.

Let C_ω be the ordered Banach space of all ω-periodic functions from $\mathbb{R}$ to $\mathbb{R}^m$, which is equipped with the maximum norm $\|\cdot\|$ and the positive cone $C_\omega^+ := \{\phi \in C_\omega : \phi(t) \ge 0, \ \forall t \in \mathbb{R}\}$. Then we can define a linear operator $L : C_\omega \to C_\omega$ by

$$(L\phi)(t) = \int_{0}^{\infty} Y(t,t-a)F(t-a)\phi(t-a)da, \quad \forall t \in \mathbb{R}, \ \phi \in C_\omega. \tag{2.6}$$

Motivated by the concept of next generation matrices introduced in [7,31], we call L the next infection operator, and define the spectral radius of L as the basic reproduction ratio

$$R_0 := \rho(L) \tag{2.7}$$

for the periodic epidemic model (2.1).

By using the approach in [4, Sect.5], we can obtain another linear operator on C_ω:

$$(\bar{L}\phi)(t) = \int_{0}^{\infty} F(t)Y(t,t-a)\phi(t-a)da = F(t)\int_{0}^{\infty} Y(t,t-a)\phi(t-a)da.$$

The spectral radius of $\bar{L}$, $\rho(\bar{L})$, was defined in [4] as the basic reproduction number. Let A and B be two bounded linear operators on C_ω defined by

$$A(\phi)(t) = \int_{0}^{\infty} Y(t,t-a)\phi(t-a)da, \quad B(\phi)(t) = F(t)\phi(t).$$

Since $L = AB$ and $\bar{L} = BA$, it then follows that $\rho(L) = \rho(\bar{L})$. Thus, the basic reproduction ratio defined in (2.7) coincides with the basic reproduction number defined in [4]. However, two kernels

$$A(t,a) := Y(t,t-a)F(t-a) \quad \text{and} \quad \bar{A}(t,a) := F(t)Y(t,t-a)$$

have different biological interpretations. Indeed, let $\phi \in \mathbb{R}^m$ be the distribution of the infected individuals introduced at time $t-a$. Then $A(t,a)\phi$ gives the distribution of the individuals who were newly infected at time $t-a$ and remain in the infected compartments at time t, while $\bar{A}(t,a)\phi$ represents the distribution of the individuals newly infected at time t by those infected individuals who were introduced at time $t-a$ and remain in the infected compartments at time t.

As in the autonomous case, we wonder whether the basic reproduction ratio (or number) R_0 characterizes the threshold of disease invasion, in the sense that the disease-free periodic solution is stable if $R_0 < 1$ and unstable if $R_0 > 1$. In order to provide an affirmative answer, we choose to use the linear operator L and elementary arguments.

To consider the case where $V(t)$ is reducible, we define

$$V_\epsilon(t) := V(t) - \epsilon E, \quad \forall \epsilon \in [0,\infty),$$

where E is the $m \times m$ matrix with each element being 1. Then $-V_\epsilon(t)$ is cooperative and irreducible for each $t \in \mathbb{R}$. Let $Y_\epsilon(t,s)$ be the evolution operator of the linear system (2.3) with $V(t)$ replaced by $V_\epsilon(t)$. By the theory of perturbed linear systems (see, e.g., [16, Sect. III.2]), it follows that there exists an $\epsilon_0 > 0$ such that for any $\epsilon \in [0,\epsilon_0]$, $Y_\epsilon(t,s)$ admits a similar property as in (2.4). Accordingly, we define the linear operator L_ϵ by replacing $Y(t,s)$ in (2.6) with $Y_\epsilon(t,s)$, and set $R_0^\epsilon := \rho(L_\epsilon)$ for $\epsilon \in [0,\epsilon_0]$.

Lemma 2.1 *Let* (A1)–(A7) *hold. Then the following statements are valid*:

(i) *The operator L is positive, continuous and compact on C_ω.*
(ii) $\lim_{\epsilon\to 0^+} \rho(\Phi_{F-V_\epsilon}(\omega)) = \rho(\Phi_{F-V}(\omega))$, *and* $\lim_{\epsilon\to 0^+} R_0^\epsilon = R_0$.

Proof Clearly, the linear operator L is positive in the sense that $L(C_\omega^+) \subset C_\omega^+$. It is easy to see from (2.5) that L is bounded, and hence, continuous on C_ω. Since

$$(L\phi)(t) = \int_{-\infty}^{t} Y(t,s)F(s)\phi(s)ds, \quad \forall t \in \mathbb{R}, \quad \phi \in C_\omega,$$

we have

$$\frac{d}{dt}(L\phi)(t) = F(t)\phi(t) - V(t)(L\phi)(t), \quad \forall t \in \mathbb{R}, \quad \phi \in C_\omega.$$

It then follows that for any $b > 0$, there exists $H = H(b) > 0$ such that $|\frac{d}{dt}(L\phi)(t)| \le H$ for all $t \in [0,\omega]$ and $\phi \in C_\omega$ with $\|\phi\| \le b$. Thus, the Ascoli–Arzela theorem implies that L is compact on C_ω.

By the continuity of solutions with respect to parameter ϵ, we see that

$$\lim_{\epsilon\to 0^+} \Phi_{F-V_\epsilon}(\omega) = \Phi_{F-V}(\omega).$$

Thus, the continuity of the spectrum for matrices ([21, Section II.5.8]) implies

$$\lim_{\epsilon\to 0^+} \rho(\Phi_{F-V_\epsilon}(\omega)) = \rho(\Phi_{F-V}(\omega)).$$

Since both L and L_ϵ are compact on C_ω, it follows that their spectrums consist of zero and countably many eigenvalues, and zero is the only possible point of accumulation of these eigenvalues. In the case where $R_0 > 0$, the Krein–Rutman theorem for positive and compact linear operators (see, e.g., [18, Theorem 7.1]) implies that R_0 is an eigenvalue of L with an eigenvector $w > 0$ in C_ω. By the upper semicontinuity of the spectrum ([21, Sect. IV.3.1]) and the continuity of a finite system of eigenvalues ([21, Sect. IV.3.5]), it then follows that $\lim_{\epsilon\to 0^+} R_0^\epsilon = R_0$. □

In order to characterize R_0, we consider the following linear ω-periodic equation

$$\frac{dw}{dt} = \left[-V(t) + \frac{F(t)}{\lambda}\right] w, \quad t \in \mathbb{R} \tag{2.8}$$

with parameter $\lambda \in (0, \infty)$. Let $W(t, s, \lambda)$, $t \geq s$, $s \in \mathbb{R}$, be the evolution operator of the system (2.8) on $\mathbb{R}^m$. Clearly, $\Phi_{F-V}(t) = W(t, 0, 1)$, $\forall t \geq 0$. Note that for each $\lambda \in (0, \infty)$, the matrix $-V(t) + \frac{F(t)}{\lambda}$ is cooperative. It then follows that the linear operator $W(t, s, \lambda)$ is positive in $\mathbb{R}^m$ for each $t \geq s$, $s \in \mathbb{R}$. Thus, the Perron–Frobenius theorem (see, e.g., [29, Theorem A.3]) implies that $\rho(W(\omega, 0, \lambda))$ is an eigenvalue of $W(\omega, 0, \lambda)$ with a nonnegative eigenvector. It is easy to verify that the matrix $W(s + \omega, s, \lambda)$ is similar to the matrix $W(\omega, 0, \lambda)$, and hence, $\sigma(W(s + \omega, s, \lambda)) = \sigma(W(\omega, 0, \lambda))$ for any $s \in \mathbb{R}$, where $\sigma(D)$ denotes the spectrum of the matrix D.

Theorem 2.1 *Let* (A1)–(A7) *hold. Then the following statements are valid:*

(i) *If* $\rho(W(\omega, 0, \lambda)) = 1$ *has a positive solution* λ_0*, then* λ_0 *is an eigenvalue of* L*, and hence* $R_0 > 0$.
(ii) *If* $R_0 > 0$*, then* $\lambda = R_0$ *is the unique solution of* $\rho(W(\omega, 0, \lambda)) = 1$.
(iii) $R_0 = 0$ *if and only if* $\rho(W(\omega, 0, \lambda)) < 1$ *for all* $\lambda > 0$.

Proof (i) Assume that $\rho(W(\omega, 0, \lambda_0)) = 1$ for some $\lambda_0 > 0$. Then 1 is an eigenvalue of $W(\omega, 0, \lambda_0)$ with a nonnegative eigenvector ϕ_0. Since $W(\omega, 0, \lambda_0)\phi_0 = \phi_0$, it follows that $\phi(t) := W(t, 0, \lambda_0)\phi_0$ is an ω-periodic solution of the ω-periodic system (2.8) with $\lambda = \lambda_0$. By the constant-variation formula, we obtain

$$\phi(t) = Y(t, \tau)\phi(\tau) + \int_\tau^t Y(t, s)\frac{F(s)}{\lambda_0}\phi(s)ds, \quad \forall t \geq \tau, \quad \tau \in \mathbb{R}. \tag{2.9}$$

In view of (2.4) and the boundedness of $\phi(t)$ on $\mathbb{R}$, letting $\tau \to -\infty$ in (2.9), we further have

$$\phi(t) = \int_{-\infty}^t Y(t, s)\frac{F(s)}{\lambda_0}\phi(s)ds, \quad \forall t \in \mathbb{R},$$

that is, $L\phi = \lambda_0\phi$. Then $\lambda_0 \in \sigma(L) \setminus \{0\}$, which implies that $R_0 := \rho(L) > 0$.

(ii) Assume that $R_0 := \rho(L) > 0$. By Lemma 2.1 (ii), there exists $\epsilon_1 \in (0, \epsilon_0]$ such that $R_0^\epsilon := \rho(L_\epsilon) > 0$ for all $\epsilon \in [0, \epsilon_1]$. Since L_ϵ is positive, bounded and compact, the Krein–Rutman theorem (see, e.g., [18, Theorem 7.1]) implies that R_0^ϵ is an eigenvalue of L_ϵ with an eigenvector $w > 0$ in C_ω, i.e., $w \in C_\omega^+ \setminus 0$. Thus, there is $s_0 \geq 0$ such that $w(s_0) > 0$ in $\mathbb{R}^m$. Let $W_\epsilon(t, s, \lambda)$, $t \geq s$, $s \in \mathbb{R}$, be the evolution operator of the linear periodic system

$$\frac{dw}{dt} = \left[-V_\epsilon(t) + \frac{F(t)}{\lambda}\right] w, \quad t \in \mathbb{R} \tag{2.10}$$

with parameter $\lambda \in (0, \infty)$. Since $L_\epsilon w = R_0^\epsilon w$, it follows that $w(t)$ satisfies the linear Eq. 2.10 with $\lambda = R_0^\epsilon$, and hence

$$w(t) = W_\epsilon(t, s_0, R_0^\epsilon)w(s_0), \quad \forall t \geq s_0.$$

In particular, $w(s_0) = w(s_0 + \omega) = W_\epsilon(s_0 + \omega, s_0, R_0^\epsilon)w(s_0)$, which implies that 1 is an eigenvalue of $W_\epsilon(s_0 + \omega, s_0, R_0^\epsilon)$ with eigenvector $w(s_0) > 0$. Note that $W_\epsilon(s_0 + \omega, s_0, R_0^\epsilon)$ is compact and strongly positive on $\mathbb{R}^m$. Then the Krein–Rutman theorem (see, e.g., [18, Theorem 7.2]) implies that $\rho(W_\epsilon(s_0 + \omega, s_0, R_0^\epsilon)) = 1$. Since $\sigma(W_\epsilon(s_0 + \omega, s_0, R_0^\epsilon)) = \sigma(W_\epsilon(\omega, 0, R_0^\epsilon))$, it follows that $\rho(W_\epsilon(\omega, 0, R_0^\epsilon)) = 1$. Letting $\epsilon \to 0^+$, we obtain $\rho(W(\omega, 0, R_0)) = 1$. It remains to prove that $\rho(W(\omega, 0, \lambda)) = 1$ has at most one positive solutions for λ. Since $F(t)$ is nonnegative and $-V(t)$ is cooperative, the standard comparison theorem implies that $\rho(W(\omega, 0, \lambda))$ is nonincreasing in $\lambda \in (0, \infty)$. Suppose, by contradiction, that $\rho(W(\omega, 0, \lambda)) = 1$ has two positive solutions $\lambda_1 < \lambda_2$. Then $\rho(W(\omega, 0, \lambda)) = 1$ for all $\lambda \in [\lambda_1, \lambda_2]$. By conclusion (i), it follows that any $\lambda \in [\lambda_1, \lambda_2]$ is an eigenvalue of L, which is impossible since the compact linear operator L has countably many eigenvalues.

(iii) From (i) and (ii) above, we see that $R_0 > 0$ if and only if $\rho(W(\omega, 0, \lambda)) = 1$ has a positive solution for some λ. Thus, $R_0 = 0$ if and only if $\rho(W(\omega, 0, \lambda)) \neq 1$ 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

$$\lim_{\lambda \to \infty} \rho(W(\omega, 0, \lambda)) = \rho(\Phi_{-V}(\omega)) < 1.$$

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

For a continuous periodic function $g(t)$ with the period ω, we define its average as

$$[g] := \frac{1}{\omega} \int_0^\omega g(t)dt.$$

The following result gives explicit formulae for R_0 in two special cases of the periodic model (2.1).

Lemma 2.2 *Let* (A1)–(A7) *hold. Then the following statements are valid*:

(i) *If* $V(t) = diag\,(V_1(t), \ldots, V_m(t))$ *and* $F(t) = diag\,(F_1(t), \ldots, F_m(t))$, *then* $R_0 = \max_{1 \leq i \leq m} \left\{ \frac{[F_i]}{[V_i]} \right\}$.

(ii) *If* $V(t) = V$ *and* $F(t) = F$ *are constant matrices, then* $R_0 = \rho(V^{-1}F) = \rho(FV^{-1})$.

Proof In the case (i), we have

$$W(\omega, 0, \lambda) = diag \left(e^{\int_0^\omega \left(-V_1(t) + \frac{1}{\lambda} F_1(t)\right)dt}, \ldots, e^{\int_0^\omega \left(-V_m(t) + \frac{1}{\lambda} F_m(t)\right)dt} \right), \quad \forall \lambda > 0,$$

and hence

$$\rho(W(\omega, 0, \lambda)) = \max_{1 \leq i \leq m} \left\{ e^{\int_0^\omega \left(-V_i(t) + \frac{1}{\lambda} F_i(t)\right)dt} \right\}, \quad \forall \lambda > 0.$$

Clearly, (A7) implies that $[V_i] > 0$ for all $1 \leq i \leq m$. By Theorem 2.1, it then follows that $R_0 = \max_{1 \leq i \leq m} \left\{ \frac{[F_i]}{[V_i]} \right\}$.

In the case (ii), we have

$$W(\omega, 0, \lambda) = e^{\left(-V+\frac{1}{\lambda}F\right)\omega}, \quad \forall \lambda > 0.$$

Without loss of generality, we assume that V is irreducible. Otherwise, we replace V with V_ϵ and then use the limiting argument (see the proof of Theorem 2.1 (ii)). Thus, the matrix $-V + \frac{1}{\lambda}F$ is cooperative and irreducible for each $\lambda > 0$. By [29, Theorem A.5], it follows that the stability modulus of $-V + \frac{1}{\lambda}F$,

$$s(\lambda) := \max\left\{Re\,\mu : \ \mu \in \sigma\left(-V + \frac{1}{\lambda}F\right)\right\},$$

is a simple eigenvalue of $-V + \frac{1}{\lambda}F$ with an eigenvector $v^* \in Int(\mathbb{R}^m_+)$, and any nonnegative eigenvalue of $-V + \frac{1}{\lambda}F$ is a positive multiple of v^*. Thus, we have

$$\rho(W(\omega, 0, \lambda)) = e^{s(\lambda)\omega}, \quad \forall \lambda > 0.$$

It is easy to verify that $V^{-1} = \int_0^\infty e^{-Va}da$. By the Perron–Frobenius theorem (see, e.g., [29, Theorem A.3]), $\rho(V^{-1}F)$ is an eigenvalue of $V^{-1}F$ with a nonnegative eigenvector w^*. Note that if $s(\lambda_0) = 0$ for some $\lambda_0 > 0$, then $\lambda_0 \in \sigma(V^{-1}F)$ and hence $\rho(V^{-1}F) > 0$. If $\rho(V^{-1}F) = 0$, then Theorem 2.1 implies that $R_0 = 0$. If $\rho(V^{-1}F) > 0$, then $\left(-V + \frac{1}{\rho(V^{-1}F)}F\right)w^* = 0$. It follows that $s(\rho(V^{-1}F)) = 0$, and hence $\rho(W(\omega, 0, \rho(V^{-1}F))) = 1$. Thus, Theorem 2.1 implies that $R_0 = \rho(V^{-1}F))$. Since $V(V^{-1}F)V^{-1} = FV^{-1}$, we have $\sigma(V^{-1}F) = \sigma(FV^{-1})$. Consequently, we have $R_0 = \rho(V^{-1}F) = \rho(FV^{-1})$. □

In view of Lemma 2.2 (ii), our definition of R_0 is consistent with that given in [31] where R_0 is defined as $\rho(FV^{-1})$ for autonomous compartmental epidemic models. The following result shows that R_0, as in the autonomous case, is a threshold parameter for the local stability of the disease-free periodic solution $x^0(t)$ for the model (2.1).

Theorem 2.2 *Assume that* (A1)-(A7) *hold. Then the following statements are valid*:

(i) $R_0 = 1$ *if and only if* $\rho(\Phi_{F-V}(\omega)) = 1$.
(ii) $R_0 > 1$ *if and only if* $\rho(\Phi_{F-V}(\omega)) > 1$.
(iii) $R_0 < 1$ *if and only if* $\rho(\Phi_{F-V}(\omega)) < 1$.

Thus, $x^0(t)$ *is asymptotically stable if* $R_0 < 1$, *and unstable if* $R_0 > 1$.

Proof (i) Note that $\rho(W(\omega, 0, 1)) = \rho(\Phi_{F-V}(\omega))$. If $R_0 = 1$, then Theorem 2.1 (ii) implies that $\rho(W(\omega, 0, 1)) = 1$. If $\rho(\Phi_{F-V}(\omega)) = 1$, then Theorem 2.1 (i) and (ii) imply that $R_0 = 1$.

(ii) (a) Assume that $R_0 > 1$. Since $R_0 > 0$, the Krein–Rutman theorem (see, e.g., [18, Theorem 7.1]) implies that there exist $w > 0$ in C_ω such that $Lw = R_0 w$. It then follows that $w(t_0) > 0$ in $\mathbb{R}^m$ for some $t_0 \in [0, \omega]$ and $w(t)$ satisfies

$$\frac{dw(t)}{dt} = (F(t) - V(t))w(t) + \left(\frac{1}{R_0} - 1\right)F(t)w(t), \quad \forall t \in \mathbb{R}. \tag{2.11}$$

We first claim that $F(t)w(t) \not\equiv 0$. Assume, by contradiction, that $F(t)w(t) = 0, \ \forall t \in \mathbb{R}$. Then (2.11) reduces to

$$\frac{dw(t)}{dt} = -V(t)w(t), \quad \forall t \in \mathbb{R}. \tag{2.12}$$

Let $\Phi_{-V}(t,s), t \geq s, s \in \mathbb{R}$ be the evolution operator of the linear system (2.12). It then follows that $\Phi_{-V}(t) = \Phi_{-V}(t,0)$, $\forall t \geq 0$, and

$$w(t_0) = w(t_0+\omega) = \Phi_{-V}(t_0+\omega, t_0)w(t_0).$$

This implies that $1 \in \sigma(\Phi_{-V}(t_0+\omega,t_0)) = \sigma(\Phi_{-V}(\omega))$, which contradicts the assumption (A7). By the constant-variation formula, as applied to Eq. 2.11, we obtain

$$w(t_0) = w(t_0+\omega) = W(t_0+\omega, t_0, 1)w(t_0) + h$$

with

$$h := \left(\frac{1}{R_0} - 1\right) \int_{t_0}^{t_0+\omega} W(t_0+\omega, s, 1)F(s)w(s)ds,$$

and hence

$$w(t_0) - W(t_0+\omega, t_0, 1)w(t_0) = h. \tag{2.13}$$

In the case where $V(t)$ is irreducible for each $t \in [0,\omega]$, $W(t,s,1)$ is strongly positive for each $t > s$, $s \in \mathbb{R}$. Since $F(t)w(t) \not\equiv 0$, we have

$$\int_{t_0}^{t_0+\omega} W(t_0+\omega, s, 1)F(s)w(s)ds \gg 0 \quad \text{in} \quad \mathbb{R}^m.$$

It then follows that

$$(-w(t_0)) - W(t_0+\omega, t_0, 1)(-w(t_0)) = -h \gg 0 \quad \text{in} \quad \mathbb{R}^m.$$

Since $-w(t_0) < 0$ in $\mathbb{R}^m$, [18, Theorem 7.3] implies that $1 < \rho(W(t_0+\omega,t_0,1)) = \rho(\Phi_{F-V}(\omega))$. In the general case of $V(t)$, replacing $V(t)$ with $V_\epsilon(t)$ and using the limiting argument (see the proof of Theorem 2.1 (ii)), we obtain $\rho(\Phi_{F-V}(\omega)) \geq 1$. Since the conclusion (i) implies that $\rho(\Phi_{F-V}(\omega)) \neq 1$, we have $\rho(\Phi_{F-V}(\omega)) > 1$.

(b) Assume that $\rho(\Phi_{F-V}(\omega)) > 1$. By the conclusion (i), we have $R_0 \neq 1$. Since $\rho(W(\omega,0,1)) = \rho(\Phi_{F-V}(\omega)) > 1$, Theorem 2.1 (iii) implies that $R_0 > 0$. It then follows that (2.13) is still valid. We need 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 $t \in [0,\omega]$, we see that Eq. 2.13 holds with $h \gg 0$ in $\mathbb{R}^m$. By [18, Theorem 7.3]), it follows that $1 > \rho(W(t_0+\omega,t_0,1)) = \rho(\Phi_{F-V}(\omega))$. In the general case of $V(t)$, replacing $V(t)$ with $V_\epsilon(t)$ and using the limiting argument (see the proof of Theorem 2.1 (ii)), we obtain $1 \geq \rho(\Phi_{F-V}(\omega))$, a contradiction. Thus, we have $R_0 > 1$.

(iii) is a straightforward consequence of the conclusions (i) and (ii) above.

Finally, we observe that

$$D_x f(t, x^0(t)) = \begin{pmatrix} F(t) - V(t) & 0 \\ -J(t) & M(t) \end{pmatrix}$$

and $\rho(\Phi_M(\omega)) < 1$. It then follows that $x^0(t)$ is asymptotically stable if $\rho(\Phi_{F-V}(\omega)) < 1$ (equivalently, $R_0 < 1$), and unstable if $\rho(\Phi_{F-V}(\omega)) > 1$ (equivalently, $R_0 > 1$). □

3 Three Examples

In this section, we present three examples to show that the basic reproduction number of the time-averaged autonomous system may coincide with the basic reproduction ratio of the periodic epidemic model, underestimate infection risk, or overestimate infection risk.

Example 1 By Lemma 2.2 (i), we see that if a periodic compartmental model has the property that both the matrix of new infection rate and the matrix of transition and dissipation within infectious compartments are diagonal, then the basic reproduction ratio is the same as the basic reproduction number of its time-averaged autonomous system. To be more specific, let us consider the following two strain model

$$\begin{aligned} \dot{I}_1 &= \beta_1(t)SI_1 - (b+\gamma_1)I_1 + \nu I_1 I_2, \\ \dot{I}_2 &= \beta_2(t)SI_2 - (b+\gamma_2)I_1 - \nu I_1 I_2, \\ \dot{S} &= b - bS + \gamma_1 I_1 + \gamma_2 I_2 - (\beta_1 I_1 + \beta_2 I_2)S, \end{aligned} \tag{3.1}$$

where S is the number of susceptible members, I_1 is the number of strain 1 of infectious agents, and I_2 is the number of strain 2 of infectious agents. Here, strain one may 'super-infect' an individual infected with strain two. This model was proposed in [12] for Dengue fever where the contact coefficients β_1 and β_2 are constants. Here, we assume that β_1 and β_2 are continuous nonnegative periodic functions with a common period ω, and all the other parameters are positive constants.

It is easy to see that the disease-free steady state is $x^0 = (0, 0, 1)^T$. According to [31], we have

$$F(t) = \begin{pmatrix} \beta_1(t) & 0 \\ 0 & \beta_2(t) \end{pmatrix}, \quad V(t) = \begin{pmatrix} b+\gamma_1 & 0 \\ 0 & b+\gamma_2 \end{pmatrix}.$$

It follows from Lemma 2.2 (i) that the basic production ratio is

$$R_0 = \max\left\{ \frac{[\beta_i]}{b+\gamma_i} : i = 1, 2 \right\}.$$

Thus, Theorem 2.2 implies that the disease-free steady state x^0 is stable if $R_0 < 1$, and is unstable if $R_0 > 1$.

Example 2 We consider a vector-host model for Dengue fever, which was proposed in [12]:

$$\begin{aligned} \dot{I} &= \beta_s SV - (b+\gamma)I, \\ \dot{V} &= \beta_m MI - cV, \\ \dot{S} &= b - bS + \gamma I - \beta_s SV, \\ \dot{M} &= c - cM - \beta_m MI, \end{aligned} \tag{3.2}$$

where I is the number of infected hosts, V is the number of infected vectors, S is the number of susceptible hosts, M is the number of susceptible vectors, β_s and β_m are disease transmission coefficients. The birth rates have been scaled to $b > 0$ for the host and $c > 0$ for the vector. In the autonomous case, the basic reproduction number of the disease has been shown to be

$$R_0 = \sqrt{\frac{\beta_s \beta_m}{c(b+\gamma)}}.$$

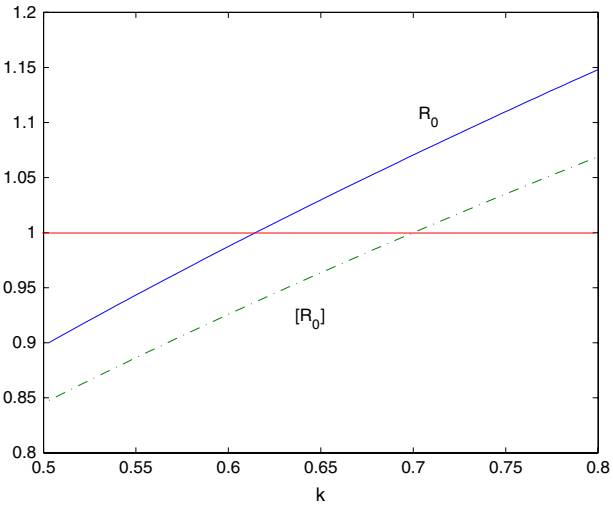

Fig. 1 The graph of the basic reproduction ratio and the average basic reproduction number when k varies

Here, we take

$$\begin{aligned} \beta_s &= k(1+\delta\cos(2\pi t)),\\ \beta_m &= \beta_0(1+\delta\cos(2\pi t)). \end{aligned} \tag{3.3}$$

The disease-free steady state of (3.2) is $(0, 0, 1, 1)^T$. For (3.2), we have

$$F(t) = \begin{pmatrix} 0 & \beta_s(t) \\ \beta_m(t) & 0 \end{pmatrix}, \quad V(t) = \begin{pmatrix} b+\gamma & 0 \\ 0 & c \end{pmatrix}. \tag{3.4}$$

Let $[R_0]$ be the basic reproduction number of the time-averaged autonomous system of (3.2). Now we need to use Theorem 2.1 (ii) to compute R_0. If we fix $\beta_0 = 0.3, b = 2, \gamma = 0.1, c = 0.1$ and $\delta = 1$, by numerical calculations, we see that $[R_0] = 1$ when $k = 0.70$ and $R_0 = 1$ when $k = 0.614$. Further, by numerical calculations we obtain the curve of the average basic reproduction number of the disease with respect to k and the curve of R_0 with respect to k in Fig. 1. This shows that the average basic reproduction number underestimates the disease transmission risk. Now, if we fix $k = 0.65$ and vary δ in $[0, 1]$ in (3.3), with other parameters unchanged as above, numerical calculations indicate again that the average basic reproduction number underestimates the disease transmission risk (see Fig. 2), where the average basic reproduction number $[R_0]$ is always 0.9636, and the basic reproduction ratio R_0 is greater than 1 when $0.75 < \delta < 1$.

Example 3 We consider the staged progression model, which was proposed in [20]:

$$\begin{aligned} \dot{I}_1 &= \sum_{k=1}^{m-1} \beta_k(t) S\frac{I_k}{N} - (\nu_1 + d_1)I_1,\\ \dot{I}_i &= \nu_{i-1}I_{i-1} - (\nu_i + d_i)I_i,\\ \dot{I}_m &= \nu_{m-1}I_{m-1} - d_m I_m,\\ \dot{S} &= b - bS - \sum_{k=1}^{m-1} \beta_k S\frac{I_k}{N}. \end{aligned} \tag{3.5}$$

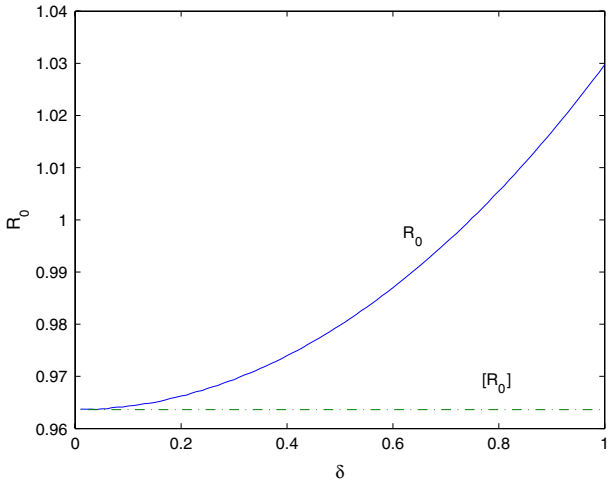

Fig. 2 The graph of the basic reproduction ratio and the average basic reproduction number when δ varies

The basic reproduction number of (3.5) in the autonomous case was obtained in [31]. Here, we consider the case that $m = 3$ and assume that the valid contact rates between susceptible and infective individuals are given by

$$\begin{aligned} &\beta_1(t) = \beta, \quad \beta_2(t) = \alpha, \qquad \text{for } 0 \le t \le \zeta, \\ &\beta_1(t) = \beta_2(t) = 0, \qquad\quad \text{for } \zeta \le t \le 1. \end{aligned} \tag{3.6}$$

Further, we assume that other parameters remain positive constants.

The unique disease-free steady state has $I_i = 0, i = 1, 2, 3$ and $S = 1$. If we define $\nu_3 = 0$, then the matrices $F(t)$ and $V(t)$ are defined by

$$F_{ij}(t) = \begin{cases} \beta_j(t), & i = 1, j \le 2, \\ 0 & \text{otherwise}, \end{cases} \tag{3.7}$$

$$V_{ij}(t) = \begin{cases} \nu_i + d_i, & j = i, \\ -\nu_j & i = 1 + j, \\ 0 & \text{otherwise}. \end{cases} \tag{3.8}$$

Since the elements of the last row and the last column of F and V are zeroes, from the point of view of threshold of disease spread, we can confine ourselves to the matrices consisting of the first two rows and two columns of F and V, which are again denoted by F and V, respectively. Let $[R_0]$ be the basic reproduction number of the time-averaged autonomous system of (3.5). It then follows that

$$[R_0] = \zeta \left(\frac{\beta}{\nu_1 + d_1} + \frac{\alpha\, \nu_1}{(\nu_1 + d_1)\,(\nu_2 + d_2)} \right). \tag{3.9}$$

Thus, $[R_0] = 1$ if

$$\beta = -\frac{\zeta\, \alpha\, \nu_1 - \nu_1\, \nu_2 - \nu_1\, d_2 - d_1\, \nu_2 - d_1\, d_2}{\zeta\ (\nu_2 + d_2)}. \tag{3.10}$$

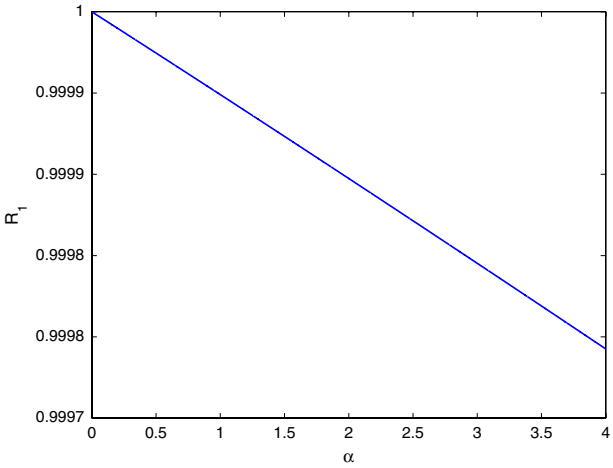

Fig. 3 The graph of $R_1 := \rho(\Phi_{F-V}(1))$ versus α

If we fix $\nu_2 = 0.1, d_2 = 0.5, d_1 = 0.05, \nu_1 = 0.05, \zeta = 0.3$, and let β satisfy (3.10), then $[R_0] = 1$ when α varies in $[0, 4]$. On the other hand, numerical calculations show that $\rho(\Phi_{F-V}(1))$ decreases from 1 as α increases from 0 (see Fig. 3). This, together with Theorem 2.2 (iii), suggests that the heterogeneity of staged progression of infectives induce overestimates of infection risks if the average basic reproduction number is used.

We should mention that some other cases of underestimate and overestimate for the average basic reproduction number can also be found in [3], where an approximate formula of the basic reproduction number was obtained for a class of periodic vector-borne disease models with a small perturbation parameter.

4 Threshold Dynamics in a Patchy Model

In this section, we investigate the global dynamics of a patchy model and the impact of periodic migrations and periodic contacts on propagation of epidemic diseases.

We consider two population centers. One is central and dominant, and the other one is smaller. Two centers are connected by population dispersal. This is the case studied in [26] for childhood diseases, where the central city is like New York and the smaller city is like Baltimore. We assume a standard incidence for the large population center and a bilinear incidence for the smaller center. This is because the standard incidence is more suitable for higher population density and the mass action incidence is more suitable when the population density is low [8,19]. If the periodic population dispersal is introduced into the model for the disease transmission of SIR type, we can obtain

$$\begin{aligned}
\dot{I}_1 &= \beta_1(t)\frac{I_1}{N_1}S_1 - (\mu_1 + \gamma_1 + b_1(t))I_1 + b_2(t)I_2,\\
\dot{I}_2 &= \beta_2(t)S_2I_2 - (\mu_2 + \gamma_2 + b_2(t))I_2 + b_1(t)I_1,\\
\dot{S}_1 &= \mu_1 - (\mu_1 + a_1(t))S_1 - \beta_1(t)\frac{I_1}{N_1}S_1 + a_2(t)S_2,\\
\dot{S}_2 &= \mu_2 - (\mu_2 + a_2(t))S_2 - \beta_2(t)S_2I_2 + a_1(t)S_1,\\
\dot{R}_1 &= \gamma_1 I_1 - (\mu_1 + c_1(t))R_1 + c_2(t)R_2(t),\\
\dot{R}_2 &= \gamma_2 I_2 - (\mu_2 + c_2(t))R_2 + c_1(t)R_1(t),
\end{aligned} \tag{4.1}$$

where $N_1 = S_1 + I_1 + R_1$, S_i is the density of susceptible individuals in patch i, I_i is the density of infectious individuals in patch i, R_i is the density of recovered individuals in patch i, $\mu_i > 0$ is the birth rate and death rate of the population in the ith patch, β_i is the disease transmission coefficient of the disease in the ith patch, γ_i is the recovery rate of infective individuals in the ith patch, a_i represents the emigration rate of susceptible individuals in the ith patch, b_i represents the emigration rate of infective individuals in the ith patch, and $c_i(t)$ is the emigration rate of recovered individuals in the ith patch. We assume that the dispersal coefficients and the disease transmission coefficients are ω-periodic in time t and the other parameters are constants.

It is easy to see that $\mathbb{R}^2_+$ is positively invariant for the periodic cooperative system:

$$\begin{aligned} \dot{S}_1 &= \mu_1 - (\mu_1 + a_1(t))S_1 + a_2(t)S_2 := F_1(t, S_1, S_2), \\ \dot{S}_2 &= \mu_2 - (\mu_2 + a_2(t))S_2 + a_1(t)S_1 := F_2(t, S_1, S_2), \end{aligned} \tag{4.2}$$

and that $F(t, S) := (F_1(t, S_1, S_2), F_2(t, S_1, S_2))$ is strongly subhomogeneous in $S \in \mathbb{R}^2_+$ in the sense that $F(t, \alpha S) \gg \alpha F(t, S)$ for any $t \geq 0$, $S \in \mathbb{R}^2_+$ and $\alpha \in (0, 1)$. Note that every nonnegative solution $S(t) = (S_1(t), S_2(t))$ of (4.2) satisfies

$$\frac{d}{dt}(S_1(t) + S_2(t)) \leq (\mu_1 + \mu_2) - \min(\mu_1, \mu_2)(S_1(t) + S_2(t)), \quad \forall t \geq 0.$$

Thus, solutions of (4.2) are ultimately bounded in $\mathbb{R}^2_+$. By [39, Theorem 2.3.2] as applied to the Poincaré map associated with system (4.2), it follows that system (4.2) has a unique positive periodic solution $(S_{10}(t), S_{20}(t))$, which is globally attractive in $\mathbb{R}^2_+$.

Now we consider the disease-free periodic state $E_0(t) = (0, 0, S_{10}(t), S_{20}(t), 0, 0)$ of (4.1). For model (4.1), we have

$$F(t) = \begin{pmatrix} \beta_1(t) & 0 \\ 0 & \beta_2(t)S_{20}(t) \end{pmatrix}, \quad V(t) = \begin{pmatrix} \mu_1 + \gamma_1 + b_1(t) & -b_2(t) \\ -b_1(t) & \mu_1 + \gamma_2 + b_2(t) \end{pmatrix}.$$

Let the basic reproduction ratio R_0 be as defined in Sect. 2. Then we have the following threshold type result on the global dynamics of (4.1).

Theorem 4.1 *The following two statements are valid*:

(i) *If $R_0 < 1$, then the disease-free periodic state $E_0(t)$ of (4.1) is globally stable.*
(ii) *If $R_0 > 1$, then (4.1) admits at least one positive periodic solution and there is $\delta > 0$ such that any positive solution of (4.1) satisfies $\liminf_{t\to\infty} I_i(t) \geq \delta$ for each $i = 1, 2$.*

Proof In the case where $R_0 < 1$, Theorem 2.2 implies that the disease-free periodic state $E_0(t)$ is locally stable. We now show that it attracts all nonnegative solutions of (4.1). If $(I_1(t), I_2(t), S_1(t), S_2(t), R_1(t), R_2(t))$ is a nonnegative solution of (4.1), then we have

$$\begin{aligned} \dot{S}_1 &\leq \mu_1 - (\mu_1 + a_1(t))S_1 + a_2(t)S_2, \\ \dot{S}_2 &\leq \mu_2 - (\mu_2 + a_2(t))S_2 + a_1(t)S_1. \end{aligned} \tag{4.3}$$

Note that any nonnegative solution $(\bar{S}_1(t), \bar{S}_2(t))$ of (4.2) approaches $(S_{10}(t), S_{20}(t))$ as t approaches infinity. It then follows from the standard comparison theorem (see, e.g., [29, Theorem A.4]) that for any $\epsilon > 0$, there is a $T > 0$ such that

$$S_i(t) < S_{i0}(t) + \epsilon, \quad i = 1, 2, \quad \text{for } t > T. \tag{4.4}$$

Thus, the first equation and the second equation of (4.1) imply that

$$\begin{aligned} \dot{I}_1 &\le \beta_1(t)I_1 - (\mu_1 + \gamma_1 + b_1(t))I_1 + b_2(t)I_2, \\ \dot{I}_2 &\le \beta_2(t)(S_{20}(t) + \epsilon)I_2 - (\mu_2 + \gamma_2 + b_2(t))I_2 + b_1(t)I_1. \end{aligned} \tag{4.5}$$

Define

$$F_\epsilon(t) = \begin{pmatrix} \beta_1(t) & 0 \\ 0 & \beta_2(t)(S_{20}(t) + \epsilon) \end{pmatrix}.$$

By Theorem 2.2, we have $\rho(\Phi_{F-V}(\omega)) < 1$. Now we restrict ϵ sufficiently small such that $\rho(\Phi_{F_\epsilon - V}(\omega)) < 1$. As a consequence, the trivial solution $(0, 0)$ of the following linear periodic system

$$\begin{aligned} \dot{I}_1 &= \beta_1(t)I_1 - (\mu_1 + \gamma_1 + b_1(t))I_1 + b_2(t)I_2, \\ \dot{I}_2 &= \beta_2(t)(S_{20}(t) + \epsilon)I_2 - (\mu_2 + \gamma_2 + b_2(t))I_2 + b_1(t)I_1 \end{aligned} \tag{4.6}$$

is globally stable. Again by the comparison theorem, we see that $I_i(t) \to 0$ as $t \to \infty$. By the last two equations of (4.1), it then follows that $R_i(t) \to 0$ as $t \to \infty$. Finally, the third equation and the fourth equation of (4.1) imply that $S_i(t) \to S_{i0}(t)$ as $t \to \infty$. This proves the conclusion (i).

In the case where $R_0 > 1$, Theorem 2.2 implies that $\rho(\Phi_{F-V}(\omega)) > 1$. By the theory of uniform persistence and coexistence states for periodic semiflows developed in [39], we can prove the conclusion (ii). Since the arguments are essentially the same as in [38, Theorem 2.3], we omit the details here. □

In order to study the impact of periodic migrations and periodic contacts on the basic reproduction ratio, we use the implicit formula for R_0 in Theorem 2.1 (ii) and the computer simulations by fixing parameters in (4.1) similar to those in [26] for childhood diseases. We take $\mu_1 = 0.04/365$, $\mu_2 = 0.02/365$, $\gamma_1 = \gamma_2 = 100/365$. This means that the first patch has higher birth rate than the second patch, and the two patches have the same recovery rate.

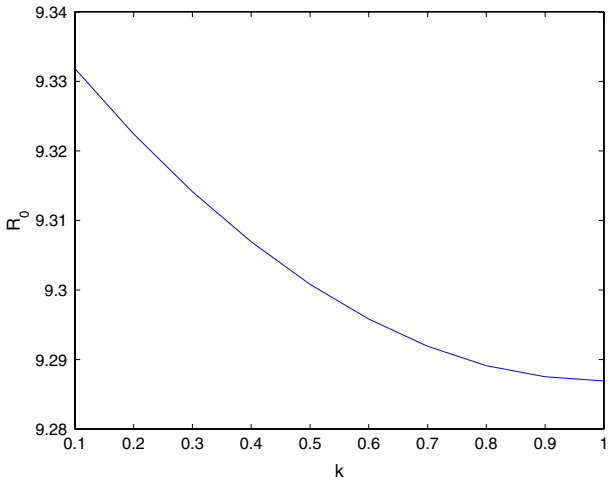

Fig. 4 The graph of the basic reproduction ratio versus k

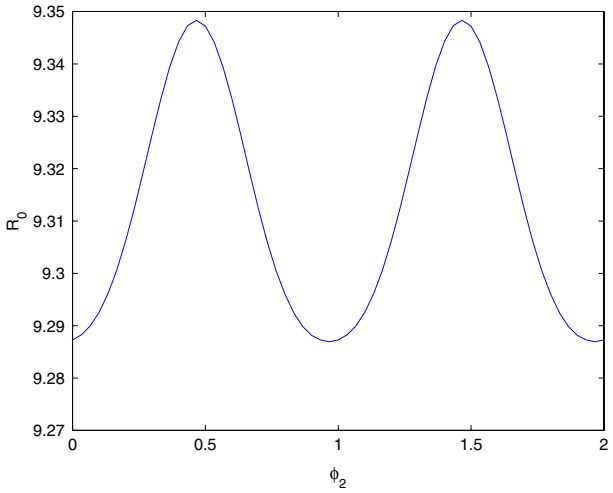

Fig. 5 The graph of the basic reproduction ratio versus ϕ_2

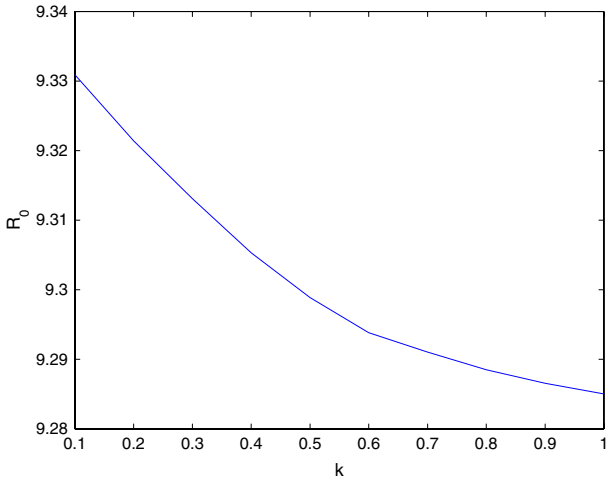

Fig. 6 The graph of the basic reproduction ratio versus k

Following common lines for periodic contacts [26,28], we suppose that $\beta_i(t)$, $a_i(t)$ and $b_i(t)$ take the form:

$$\begin{aligned} \beta_i(t) &= \beta_{i0}(1+\delta_i\cos(2\pi(t+\phi_i)), \quad i=1,2,\\ a_i(t) &= a_{i0}(1+\delta_{ia}\cos(2\pi(t+\phi_{ia})), \quad i=1,2,\\ b_i(t) &= b_{i0}(1+\delta_{ib}\cos(2\pi(t+\phi_{ib})), \quad i=1,2. \end{aligned} \tag{4.7}$$

Let us fix $\beta_{10} = 1202/365, \beta_{20} = 600/365, a_{10} = 60/365, a_{20} = 30/365, b_{10} = 45, b_{20} = 30$. If $\phi_i = \phi_{ia} = \phi_{ib} = 0, \delta_{ia} = \delta_{ib} = 1, \delta_i = k$ for $i = 1, 2$, as k varies in [0, 1], we obtain the graph for the relation of the basic reproduction ratio to k (Fig. 4). This graph shows that the basic reproduction ratio is lowered as the amplitude δ is larger. Next, we choose $\phi_1 = \phi_{ia} = \phi_{ib} = 0, \delta_i = \delta_{ia} = \delta_{ib} = 1$ for $i = 1, 2$. As ϕ_2 varies in [0, 1],

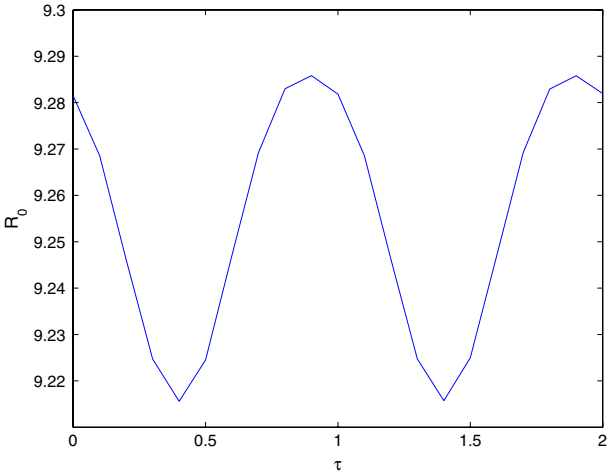

Fig. 7 The graph of the basic reproduction ratio versus τ

numerical simulations provide the relation of the basic reproduction ratio to ϕ_2 (Fig. 5). This figure shows that the phase difference can reduce or increase the risk of epidemic disease outbreak in two patches.

We now turn to the impact of periodic migrations. Again, we concentrate on the amplitudes and the phase differences. Fixing $\beta_{10} = 1202/365$, $\beta_{20} = 600/365$, $a_{10} = 60/365$, $a_{20} = 30/365$, $b_{10} = 45$, $b_{20} = 30$ and taking $\phi_i = \phi_{ia} = \phi_{ib} = 0$, $\delta_i = 1$, $\delta_{ia} = \delta_{ib} = k$ for $i = 1, 2$, we compute, as k varies in $[0, 1]$, the relation between the basic reproduction ratio R_0 and k to obtain Fig. 6. This figure shows that the increase of the amplitude of periodic migrations reduces the risk of epidemic prevalence. One interpretation for this is that the second patch is a better patch and the diffusion of population relieves the disease spread. On the other line, if we delay the phase of periodic migration from the second patch to the first patch by τ and let $\delta_i = \delta_{ia} = \delta_{ib} = 1$, with other parameters unchanged, numerical calculations give the relation between the basic reproduction ratio and τ in Fig. 7. Compared with Fig. 5, the reproduction ratio is decreased until $\tau = 0.5$.

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