Hindawi Publishing Corporation
The Scientific World Journal
Volume 2013, Article ID 470646, 10 pages
http://dx.doi.org/10.1155/2013/470646

Research Article

Positive Periodic Solutions of an Epidemic Model with Seasonality

Gui-Quan Sun,[1,2,3] Zhenguo Bai,[4] Zi-Ke Zhang,[5,6,7] Tao Zhou,[6] and Zhen Jin[1,2]

[1] *Complex Sciences Center, Shanxi University, Taiyuan 030006, China*
[2] *School of Mathematical Sciences, Shanxi University, Taiyuan, Shanxi 030006, China*
[3] *Department of Mathematics, North University of China, Taiyuan, Shanxi 030051, China*
[4] *Department of Applied Mathematics, Xidian University, Xi'an, Shaanxi 710071, China*
[5] *Institute of Information Economy, Hangzhou Normal University, Hangzhou 310036, China*
[6] *Web Sciences Center, University of Electronic Science and Technology of China, Chengdu, Sichuan 610054, China*
[7] *Department of Physics, University of Fribourg, Chemin du Musée 3, 1700 Fribourg, Switzerland*

Correspondence should be addressed to Gui-Quan Sun; gquansun@126.com

Received 17 August 2013; Accepted 12 September 2013

Academic Editors: P. Leach, O. D. Makinde, and Y. Xia

An SEI autonomous model with logistic growth rate and its corresponding nonautonomous model are investigated. For the autonomous case, we give the attractive regions of equilibria and perform some numerical simulations. Basic demographic reproduction number R_d is obtained. Moreover, only the basic reproduction number R_0 cannot ensure the existence of the positive equilibrium, which needs additional condition $R_d > R_1$. For the nonautonomous case, by introducing the basic reproduction number defined by the spectral radius, we study the uniform persistence and extinction of the disease. The results show that for the periodic system the basic reproduction number is more accurate than the average reproduction number.

1. Introduction

Bernoulli was the first person to use mathematical method to evaluate the effectiveness of inoculation for smallpox [1–6]. Then in 1906, Hawer studied the regular occurrence of measles by a discrete-time model. Moreover, Ross [3, 4] adopted the continuous model to study the dynamics of malaria between mosquitoes and humans in 1916 and 1917. In 1927, Kermack and McKendrick [5, 6] extended the above works and established the threshold theory. So far, mathematical models have gotten great development and have been used to study population dynamics, ecology, and epidemic, which can be classified in terms of different aspects. From the aspect of the incidence of infectious diseases, there are bilinear incidence, standard incidence, saturating incidence, and so on. According to the type of demographic import, the constant import, the exponential import, and the logistic growth import are the most common forms. The simple exponential growth models can provide an adequate approximation to population growth for the initial period. If no predation or intraspecific competition for populations is included, the population can continue to increase. However, it is impossible to grow immoderately due to the intraspecific competition for environmental resources such as food and habitat. So, for this case, logistic model is more reasonable and realistic which has been adopted and studied [7–18]. Moreover, due to its rich dynamics, the logistic models have been applied to many fields. Fujikawa et al. [9] applied the logistic model to show *Escherichia coli* growth. Invernizzi and Terpin [14] used a generalized logistic model to describe photosynthetic growth and predict biomass production. Min et al. [15] used logistic dynamics model to describe coalmining cities' economic growth mechanism and sustainable development. There is a good fit in simulating the coalmining cities' growth and development track based on resource development cycle. Banaszak et al. [17] investigated logistic models in flexible manufacturing, and Brianzoni et al. [18] studied a business-cycle model with logistic population

growth. Muroya [13] investigated discrete models of non-autonomous logistic equations. As a result, this paper builds an SEI ordinary differential model with the logistic growth rate and the standard incidence.

For general epidemic models, we mainly study their threshold dynamics, that is, the basic reproduction number which determines whether the disease can invade the susceptible population successfully. However, for the system with logistic growth rate, besides the basic reproduction number, the qualitative dynamics are controlled by a demographic threshold R_d which has a similar meaning and is called as the basic demographic reproduction number. If $R_d > 1$, the population grows; that is, a critical mass of individuals for the disease to spread may be supported. If $R_d < 1$, the population will not survive; that is, not enough mass of individuals may be supported for the disease to spread. For this case, the dynamical behavior of disease will be decided by two thresholds R_0 and R_d.

It is well known that many diseases exhibit seasonal fluctuations, such as whooping cough, measles, influenza, polio, chickenpox, mumps, and rabies [19–22]. Seasonally effective contact rate [22–26], periodic changing in the birth rate [27], and vaccination program [28] are often regarded as sources of periodicity. Seasonally effective contact rate is related to the behavior of people and animals, the temperature, and the economy. Due to the existence of different seasons, people have different activities which may lead to a different contact rate. Because of various factors, the economy in a different season has a very big difference. Therefore, this paper studies the corresponding non-autonomous system which is obtained by changing the constant transmission rate into the periodic transmission rate. Seasonal transmission is often assumed to be sinusoidal (cosine function has the same meaning), such that $\lambda(t) = \lambda(1 + \eta \sin(\pi t/b))$ where η is the amplitude of seasonal variation in transmission (typically referred to as the "strength of seasonal forcing") and $2b$ is the period, which is a crude assumption for many infectious diseases [29–31]. When $\eta = 0$, there is no nonseasonal infections. Motivated by biological realism, some recent papers take the contact rate as $\lambda(t) = \lambda(1 + \eta\,\mathrm{term}(t))$, where term is a periodic function which is +1 during a period of time and −1 during other time. More natural term can be written as $\lambda(t) = \lambda(1 + \eta)^{\mathrm{term}(t)}$ [29]. Here, we take the form $\beta(t) = a[1 + b\sin(\pi t/10)]$.

The paper is organized as follows. In Section 2, we introduce an autonomous model and analyze the equilibria and their respective attractive region. In Section 3, we study the non-autonomous system in terms of global asymptotic stability of the disease-free equilibrium and the existence of positive periodic solutions. Moreover, numerical simulations are also performed. In Section 4, we give a brief discussion.

2. Autonomous Model and Analysis

2.1. Model Formulation. The model is a system of SEI ordinary differential equations, where S is the susceptible, E is the exposed, I is the infected, and $N = S + E + I$. This system considers the logistic growth rate and the standard incidence which is fit for the long-term growth of many large populations. The incubation period is considered for many diseases which do not develop symptoms immediately and need a period of time to accumulate a pathogen quantity for clinical outbreak, such as rabies, hand-foot-mouth disease, tuberculosis, and AIDS [22, 32]. The model we employ is as follows:

$$\begin{aligned}\frac{dS}{dt} &= rN\left(1 - \frac{N}{k}\right) - \frac{\beta SI}{N} - mS,\\ \frac{dE}{dt} &= \frac{\beta SI}{N} - \sigma E - mE,\\ \frac{dI}{dt} &= \sigma E - mI - \mu I,\end{aligned} \tag{1}$$

where all parameters are positive whose interpretations can be seen in Table 1.

Table 1: Descriptions and values of parameters in model (1).

Parameter	Interpretation	Value
r	The intrinsic growth rate	
k	The carrying capacity	100000
β	The transmission rate	
m	The natural mortality rate	0.1
σ	Clinical outcome rate	0.2
μ	The disease-induced mortality rate	0.1
a	The baseline contact rate	
b	The magnitude of forcing	

Noticing the equations in model (1), we have

$$\frac{dN}{dt} = rN\left(1 - \frac{N}{k}\right) - mN - \mu I. \tag{2}$$

When there exists no disease, we have

$$\frac{dN}{dt} = rN\left(1 - \frac{N}{k}\right) - mN = \left[r\left(1 - \frac{N}{k}\right) - m\right]N. \tag{3}$$

Let $R_d = r/m$, if $R_d > 1$, $N \to N^0 = (1 - m/r)k$ for $N(0) > 0$, as $t \to +\infty$; that is, the population will grow and tend to a steady state N^0. If $R_d < 1$, then $dN/dt < 0$ which will cause the population to disappear. Thus, R_d is the basic demographic reproduction number. From the above equation, the feasible region can be obtained: $X = \{(S, E, I) \mid S, E, I \geq 0,\ 0 \leq S + E + I \leq (1 - m/r)k\}$, where $r > m$.

Theorem 1. *The region X is positively invariant with respect to system* (1).

2.2. Dynamical Analysis. Let the right hand of system (1) to be zero; it is easy to see that system (1) has three equilibria:

$$\begin{aligned}O &= (0, 0, 0),\\ E_0 &= \left(\left(1 - \frac{m}{r}\right)k, 0, 0\right),\\ E_* &= (S^*, E^*, I^*),\end{aligned} \tag{4}$$

where O is the origin, E_0 is the disease-free equilibrium, and E_* is the endemic equilibrium. Concretely, one can have

$$S^* = \frac{[m\beta(m+\sigma+\mu)+\mu(m+\mu)(m+\sigma)(R_0-1)]N^*}{\beta[(m+\mu)(m+\sigma)(R_0-1)+m(m+\sigma+\mu)]}, \tag{5}$$

$$E^* = \frac{[r(1-N^*/k)-m](m+\mu)N^*}{\sigma\mu}, \tag{6}$$

$$I^* = \frac{(m+\mu)(m+\sigma)(R_0-1)N^*}{\beta(m+\sigma+\mu)}, \tag{7}$$

$$N^* = (k[\beta m(m+\sigma+\mu)(R_d-1) + \mu(m+\sigma)(m+\mu)(1-R_0)]) \times (r\beta(m+\sigma+\mu))^{-1}, \tag{8}$$

where $R_d = r/m$ is the basic demographic reproduction number and $R_0 = \beta\sigma/(m+\sigma)(m+\mu)$ is the basic reproduction number which can be obtained by the next-generation matrix method [33–35]. The introduction of the basic demographic reproduction number can be found in [36].

Moreover, from (6) and (7), the conditions of the endemic equilibrium to exist are $R_0 > 1$ and $R_d > R_1$, where $R_1 = 1+\mu(m+\mu)(m+\sigma)(R_0-1)/m\beta(m+\sigma+\mu)$. So, we can obtain the following theorems.

Theorem 2. *The system* (1) *has three equilibria: origin O, disease-free equilibrium E_0, and the endemic equilibrium E_*. O always exists; if $R_d > 1$, E_0 exists; if $R_0 > 1$ and $R_d > R_1$, E_* exists.*

Theorem 3. *When $R_d > 1$ and $R_0 < 1$, E_0 is globally asymptotically stable.*

Proof. By [33–35], we know that E_0 is locally asymptotically stable. Now we define a Lyapunov function

$$V = E + \frac{m+\sigma}{\sigma}I \geq 0. \tag{9}$$

When $R_d > 1$ and $R_0 < 1$, the Lyapunov function satisfies

$$\begin{aligned}\dot{V} &= \dot{E} + \frac{m+\sigma}{\sigma}\dot{I} \\ &= \frac{\beta SI}{N} - \frac{(m+\sigma)(m+\mu)}{\sigma}I \\ &\leq \left[\beta - \frac{(m+\sigma)(m+\mu)}{\sigma}\right]I \\ &= \frac{(m+\sigma)(m+\mu)}{\sigma}[R_0-1]I \\ &\leq 0.\end{aligned} \tag{10}$$

Moreover, $\dot{V} = 0$ only hold when $I = 0$. It is easy to verify that the disease-free equilibrium point E_0 is the only fixed point of the system. Hence, applying the Lyapunov-LaSalle asymptotic stability theorem in [37, 38], the disease-free equilibrium point E_0 is globally asymptotically stable. □

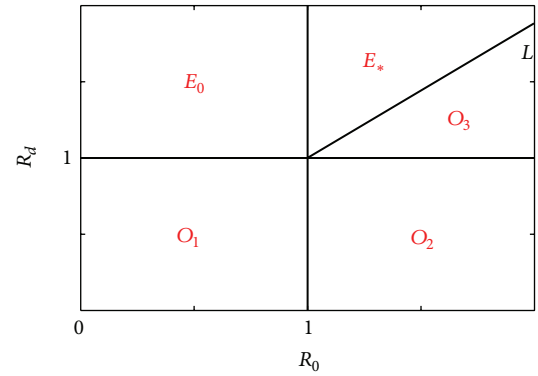

FIGURE 1: R_d in terms of R_0. The region of O_1, O_2, and O_3 is the basin of attraction of equilibrium O; the region of E_0 is the basin of attraction of equilibrium E_0; the region of E_* is the basin of attraction of equilibrium E_*; the line L is $R_d = R_1 = 1+\mu(m+\mu)(m+\sigma)(R_0-1)/m\beta(m+\sigma+\mu)$.

Since the proof of the stability of equilibria O and E_* is more difficult, we only give some numerical results.

In sum, we can show the respective basins of attraction of the three equilibria which can be seen in Figure 1 and confirmed in Figure 2.

(1) When $R_d < 1$, O is stable; see Figures 2(b) and 2(c).

(2) When $R_d > 1$ and $R_0 < 1$, E_0 is stable; see Figure 2(d).

(3) When $R_d > 1$, $R_0 > 1$, and $R_d < R_1$, O is stable; see Figure 2(a).

(4) When $R_d > 1$, $R_0 > 1$, and $R_d > R_1$, E_* is stable; see Figure 2(e).

3. Nonautonomous Model and Analysis

3.1. The Basic Reproduction Number. Now, we consider the non-autonomous case of the model (1) when the transmission rate is periodic, which is given as follows:

$$\begin{aligned}\frac{dS}{dt} &= rN\left(1-\frac{N}{k}\right) - \frac{\beta(t)SI}{N} - mS, \\ \frac{dE}{dt} &= \frac{\beta(t)SI}{N} - \sigma E - mE, \\ \frac{dI}{dt} &= \sigma E - mI - \mu I,\end{aligned} \tag{11}$$

where $\beta(t)$ is a periodic function which is proposed by [39]. In the subsequent section, we will discuss the dynamical behavior of the system (11).

For system (11), firstly we can give the basic reproduction number R_0. According to the basic reproduction number under non-autonomous system, we can refer to the method of [40, 41]. From the last section, we know that system (11)

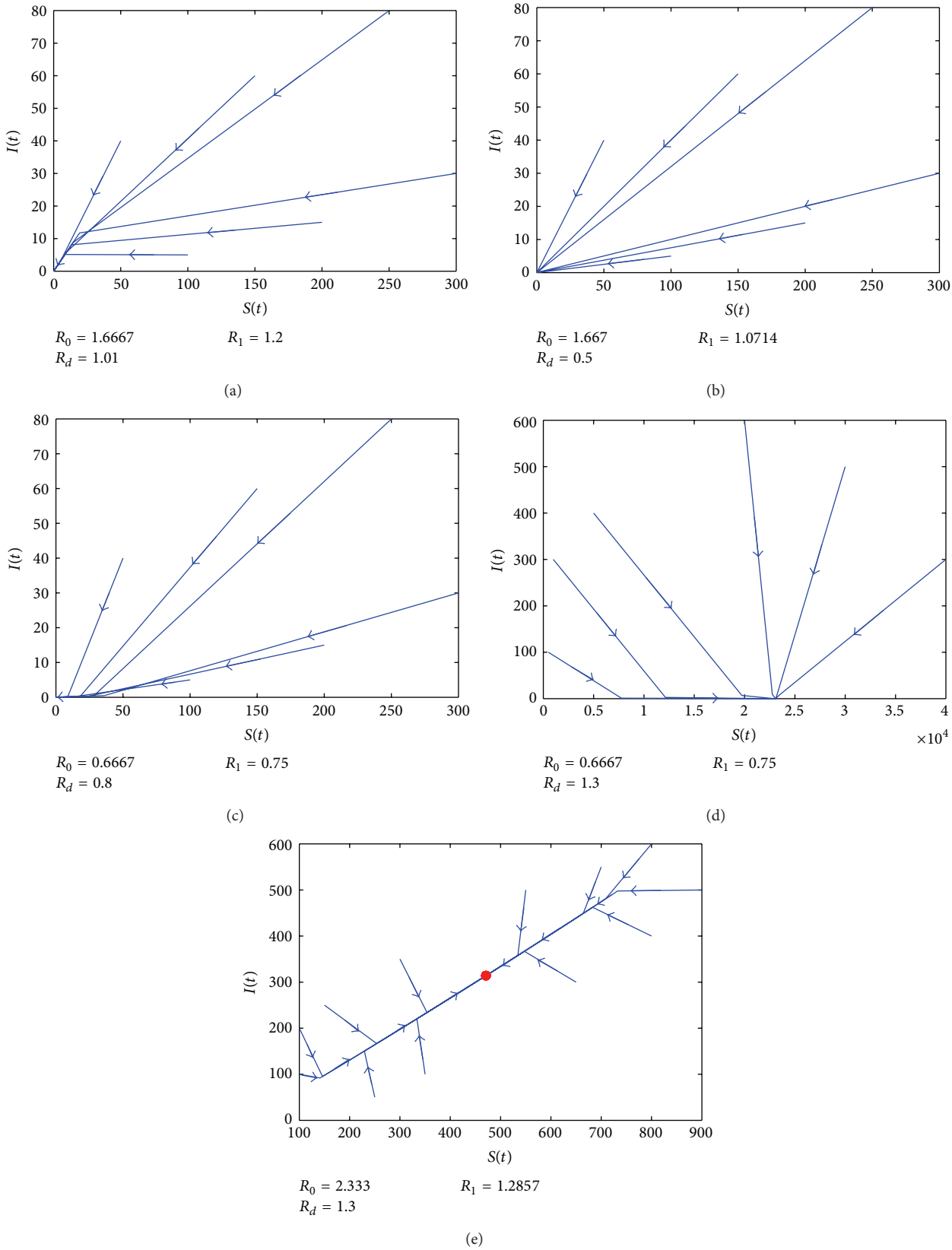

FIGURE 2: The phase curves of the system under different initial conditions. (a) In the region O_3 with $r = 0.101$ and $\beta = 0.5$; (b) in the region O_2 with $r = 0.05$ and $\beta = 0.35$; (c) in the region O_1 with $r = 0.08$ and $\beta = 0.2$; (d) in the region E_0 with $r = 0.13$ and $\beta = 0.2$; (e) in the region E_* with $r = 0.13$ and $\beta = 0.7$. The value of other parameters can be seen in Table 1.

has one disease-free equilibrium $E_0 = (N^0, 0, 0)$, where $N^0 = (1 - m/r)k$. By giving a new vector $x = (E, I)$, we have

$$F = \begin{pmatrix} \frac{\beta(t)SI}{N} \\ 0 \end{pmatrix}, \qquad V = \begin{pmatrix} mE + \sigma E \\ mI + \mu I - \sigma E \end{pmatrix}, \tag{12}$$
$$V^- = \begin{pmatrix} mE + \sigma E \\ mI + \mu I \end{pmatrix}, \qquad V^+ = \begin{pmatrix} 0 \\ \sigma E \end{pmatrix}.$$

Taking the partial derivative of the above vectors about variables E, I and substituting the disease-free equilibrium, we have

$$F(t) = \begin{pmatrix} 0 & \beta(t) \\ 0 & 0 \end{pmatrix}, \tag{13}$$
$$V(t) = \begin{pmatrix} m + \sigma & 0 \\ -\sigma & m + \mu \end{pmatrix}.$$

According to [41], denote C_ω to be the ordered Banach space of all ω-periodic functions from $\mathbb{R}$ to $\mathbb{R}^4$ which is equipped with the maximum norm $\|\cdot\|$ and the positive cone $C_\omega^+ := \{\phi \in C_\omega : \phi(t) \geq 0, \forall t \in \mathbb{R}_+\}$. Over the Banach space, we define a linear operator $L : C_\omega \to C_\omega$ by

$$(L\phi)(t) = \int_0^\infty Y(t, t-a) F(t-a) \phi(t-a)\, da, \quad \forall t \in \mathbb{R}_+, \phi \in C_\omega, \tag{14}$$

where L is called the next infection operator and the interpretation of $Y(t, t-a)$, $\phi(t-a)$ can be seen in [41]. Then the spectral radius of L is defined as the basic reproduction number

$$R_0 := \rho(L). \tag{15}$$

In order to give the expression of the basic reproduction number, we need to introduce the linear ω-periodic system

$$\frac{dw}{dt} = \left[-V(t) + \frac{F(t)}{\lambda}\right] w, \quad t \in \mathbb{R}_+, \tag{16}$$

with parameter $\lambda \in \mathbb{R}$. Let $W(t, s, \lambda)$, $t \geq s$, be the evolution operator of system (16) on $\mathbb{R}^2$. In fact, $W(t, s, \lambda) = \Phi_{(F/\lambda)-V}(t)$, and $\Phi_{F-V}(t) = W(t, 0, 1)$, for all $t \geq 0$. By Theorems 2.1 and 2.2 in [41], the basic reproduction number also can be defined as λ_0 such that $\rho(\Phi_{(F/\lambda_0)-V}(\omega)) = 1$, which can be straightforward to calculate.

3.2. Global Stability of the Disease-Free Equilibrium

Theorem 4. *The disease-free equilibrium E_0 is globally asymptotically stable when $R_0 < 1$ and $R_d > 1$.*

Proof. Theorem 2.2 in [41] implies that E_0 is locally asymptotically stable when $R_0 < 1$ and $R_d > 1$. So we only need to prove its global attractability. It is easy to know that $S(t) \leq N^0 = (1 - (m/r))k$. Thus,

$$\frac{dE}{dt} \leq \beta(t) I - (m + \sigma) E, \qquad \frac{dI}{dt} = \sigma E - mI - \mu I. \tag{17}$$

The right comparison system can be written as

$$\frac{dE}{dt} = \beta(t) I - (m + \sigma) E, \qquad \frac{dI}{dt} = \sigma E - mI - \mu I; \tag{18}$$

that is,

$$\frac{dh}{dt} = (F(t) - V(t)) h(t), \quad h(t) = (E(t), I(t)). \tag{19}$$

For (19), Lemma 2.1 in [42] shows that there is a positive ω-periodic function $\widehat{h}(t) = (E(t), I(t))^T$ such that $h(t) = e^{pt}\widehat{h}(t)$ is a solution of system (18), where $p = (1/\omega) \ln \rho(\Phi_{F-V}(\omega))$. By Theorem 2.2 in [41], we know that when $R_0 < 1$ and $R_d > 1$, $\rho(\Phi_{F-V}(\omega)) < 1$ and $p < 0$, which implies $h(t) \to 0$ as $t \to \infty$. Therefore, the zero solution of system (18) is globally asymptotically stable. By the comparison principle [43] and the theory of asymptotic autonomous systems [44], when $R_0 < 1$ and $R_d > 1$, E_0 is globally attractive. Therefore, the proposition that E_0 is globally asymptotically stable holds. □

3.3. Existence of Positive Periodic Solutions. Before the proof of the existence of positive periodic solutions, we firstly introduce some denotations. Let $u(t, x_0)$ be the solution of system (11) with the initial value $x_0 = (S(0), E(0), I(0))$. By the fundamental existence-uniqueness theorem [45], $u(t, x_0)$ is the unique solution of system (11) with $u(0, x_0) = x_0$.

Next, we need to introduce the Poincaré map $P : X \to X$ associated with system (11); that is,

$$P(x_0) = u(\omega, x_0), \quad \forall x_0 \in X, \tag{20}$$

where ω is the period. Theorem 1 implies that X is positively invariant and P is a dissipative point.

Now, we introduce two subsets of X, $X_0 := \{(S, E, I) \in X : E > 0, I > 0\}$ and $\partial X_0 = X \setminus X_0$.

Lemma 5. *(a) When $R_0 > 1$ and $r > m + \mu$, there exists a $\delta > 0$ such that when*

$$\|(S(0), E(0), I(0)) - E_0\| \leq \delta \tag{21}$$

for any $(S(0), E(0), I(0)) \in X_0$, one has

$$\limsup_{m \to \infty} d\left[P^m(S(0), E(0), I(0)), E_0\right] \geq \delta, \tag{22}$$

where $E_0 = (N^0, 0, 0)$.

(b) When $R_0 > 1$ and $r > m + \mu$, there exists a $\delta > 0$ such that when

$$\|(S(0), E(0), I(0)) - O\| \le \delta \tag{23}$$

for any $(S(0), E(0), I(0)) \in X_0$, one has

$$\limsup_{m\to\infty} d\left[P^m(S(0), E(0), I(0)), O\right] \ge \delta, \tag{24}$$

where $O = (0, 0, 0)$.

Proof. (a) By Theorem 2.2 in [41], we know that when $R_0 > 1$, $\rho(\Phi_{F-V}(\omega)) > 1$. So there is a small enough positive number ϵ such that $\rho(\Phi_{F-V-M_\epsilon}(\omega)) > 1$, where

$$M_\epsilon = \begin{pmatrix} 0 & \dfrac{\beta(t)\epsilon I}{N^0} \\ 0 & 0 \end{pmatrix}. \tag{25}$$

If proposition (a) does not hold, there is some $(S(0), E(0), I(0)) \in X_0$ such that

$$\limsup_{m\to\infty} d\left(P^m(S(0), E(0), I(0)), E_0\right) < \delta. \tag{26}$$

We can assume that for all $m \ge 0$, $d(P^m(S(0), E(0), I(0)), E_0) < \delta$. Applying the continuity of the solutions with respect to the initial values,

$$\begin{aligned} \left\|u\left(t, P^m(S(0), E(0), I(0))\right) - u\left(t, E_0\right)\right\| &\le \epsilon, \\ \forall m \ge 0,\ \forall t_1 &\in [0, \omega]. \end{aligned} \tag{27}$$

Let $t = m\omega + t_1$, where $t_1 \in [0, \omega]$ and $m = [t/\omega]$. $m = [t/\omega]$ is the greatest integer which is not more than t/ω. Then, for any $t \ge 0$,

$$\begin{aligned} &\left\|u(t, (S(0), E(0), I(0))) - u\left(t, E_0\right)\right\| \\ &= \left\|u\left(t_1, P^m(S(0), E(0), I(0))\right) - u\left(t_1, E_0\right)\right\| \le \epsilon. \end{aligned} \tag{28}$$

So $N^0 - \epsilon \le S(t) \le N^0 + \epsilon$. Then, when $\limsup_{m\to\infty} d(P^m(S(0), E(0), I(0)), E_0) < \delta$,

$$\begin{aligned} \frac{dE}{dt} &\ge \beta(t) I - \frac{\beta(t)\epsilon I}{N^0} - (m + \sigma) E, \\ \frac{dI}{dt} &= \sigma E - mI - \mu I. \end{aligned} \tag{29}$$

Thus, we can study the right linear system

$$\begin{aligned} \frac{dE}{dt} &= \beta(t) I - \frac{\beta(t)\epsilon I}{N^0} - (m + \sigma) E, \\ \frac{dI}{dt} &= \sigma E - mI - \mu I. \end{aligned} \tag{30}$$

For the system (30), there exists a positive ω-periodic function $\widehat{g}(t) = (E(t), I(t))^T$ such that $g(t) = e^{pt}\widehat{g}(t)$ is a solution of system (11), where $p = (1/\omega)\ln\rho(\Phi_{F-V-M\epsilon}(\omega))$. When $R_0 > 1$, $\rho(\Phi_{F-V-M\epsilon}(\omega)) > 1$, which means that when $g(0) > 0$, $g(t) \to \infty$ as $t \to \infty$. By the comparison principle [43], when $E(0) > 0$, $I(0) > 0$, $E(t) \to \infty$, $I(t) \to \infty$ as $t \to \infty$. There appears a contradiction. Thus, the proposition (a) holds.

(b) When $R_0 > 1$ and $r > m + \mu$, we have

$$\begin{aligned} \frac{dN}{dt} &= rN\left(1 - \frac{N}{k}\right) - mN - \mu I \\ &\ge \left[r\left(1 - \frac{N}{k}\right) - m - \mu\right] N > 0. \end{aligned} \tag{31}$$

So if $R_d > 1$, $N \to N^0$ for $N(0) > 0$ as $t \to +\infty$; that is, $W^S(O) \cap X_0 = \emptyset$. □

Theorem 6. *When $R_0 > 1$ and $r > m + \mu$, there exists a $\delta > 0$ such that any solution $(S(t), E(t), I(t))$ of system (11) with initial value $(S(0), E(0), I(0)) \in \{(S, E, I) \in X : E > 0,\ I > 0\}$ satisfies*

$$\liminf_{t\to\infty} E(t) \ge \delta, \qquad \liminf_{t\to\infty} I(t) \ge \delta, \tag{32}$$

and system (11) has at least one positive periodic solution.

Proof. From system (11),

$$\begin{aligned} S(t) &= e^{-\int_0^t (\beta(s)I(s)/N + m)ds} \\ &\quad \times \left[S(0) + \int_0^t rN(s)\left(1 - \frac{N(s)}{k}\right) \times e^{\int_0^s (\beta(c)I(c)/N(c) + m)dc} ds\right] \\ &> 0, \quad \forall t > 0, \end{aligned} \tag{33}$$

$$\begin{aligned} E(t) &= e^{-(m+\sigma)t}\left[E(0) + \int_0^t \frac{\beta(s) S(s) I(s)}{N(s)} e^{(m+\sigma)s} ds\right] \\ &> 0, \quad \forall t > 0, \end{aligned} \tag{34}$$

$$\begin{aligned} I(t) &= e^{-(m+\mu)t}\left[I(0) + \int_0^t \sigma E(s) e^{(m+\mu)s} ds\right] \\ &> 0, \quad \forall t > 0, \end{aligned} \tag{35}$$

for any $(S(0), E(0), I(0)) \in X_0$, which shows that X_0 is positively invariant. Moreover, it is obvious to see that ∂X_0 is relatively closed in X. Denote

$$\begin{aligned} M_\partial = \{&(S(0), E(0), I(0)) \in \partial X_0 : \\ &P^m(S(0), E(0), I(0)) \in \partial X_0, \forall m \ge 0\}. \end{aligned} \tag{36}$$

Next, we prove that

$$M_\partial = \{(S, 0, 0) \in X : S \ge 0\}. \tag{37}$$

We only need to show that $M_\partial \subseteq \{(S, 0, 0) \in X : S \ge 0\}$, which means that for any $(S(0), E(0), I(0)) \in \partial X_0$, $E(m\omega) = I(m\omega) = 0$, for all $m \ge 0$. If it does not hold, there exists a $m_1 \ge 0$ such that $(E(m_1\omega), I(m_1\omega))^T > 0$. Taking $m_1\omega$ as

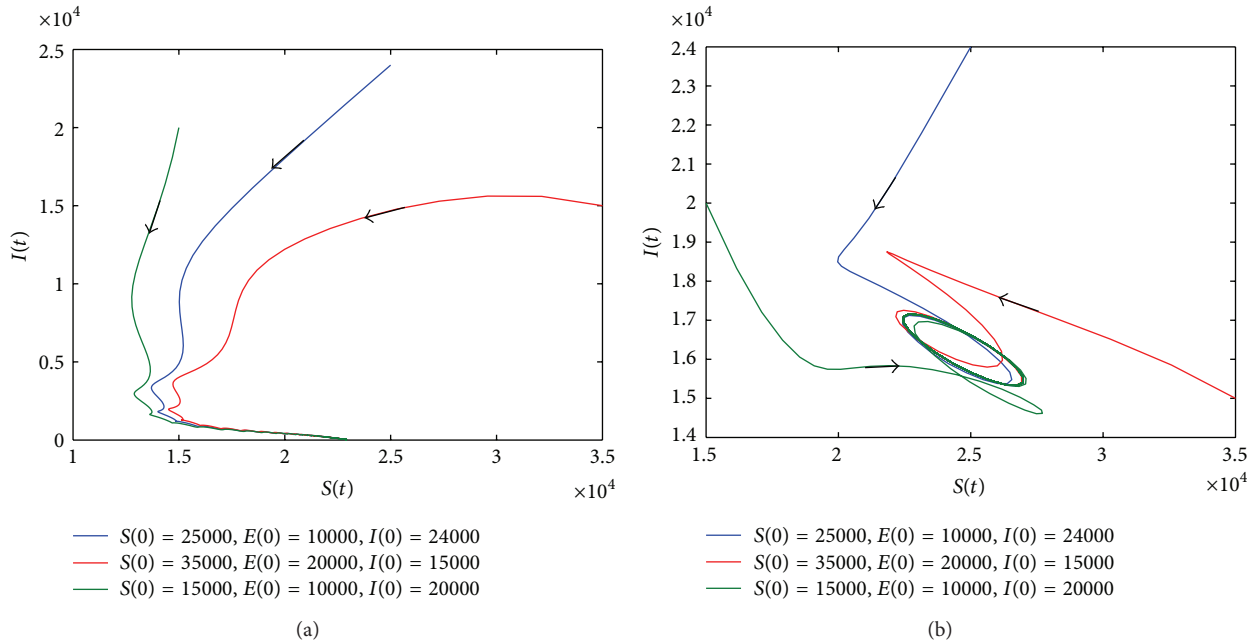

FIGURE 3: Phase plane of $S(t)$ and $I(t)$. (a) When the parameter values are $r = 0.13$, $a = 0.3$, and $b = 0.2$, $R_0 = 0.9029 < 1$, $R_d = 1.3 > 1$. (b) When the parameter values are $r = 0.3$, $a = 0.7$, and $b = 0.2$, $R_0 = 2.1067$ and $r > m + \mu$. The value of other parameters can be seen in Table 1.

the initial time and repeating the processes as in (33)–(35), we can have that $(S(t), E(t), I(t))^T > 0$, for all $t > m_1\omega$. Thus, $(S(t), E(t), I(t)) \in X_0$, for all $t > m_1\omega$. There appears a contradiction, which means that the equality (37) holds. Therefore, E_0 is acyclic in ∂X_0. Obviously, when $R_0 > 1$ and $r > m + \mu$, O is acyclic in ∂X_0.

Furthermore, by Lemma 5, $E_0 = (N^0, 0, 0)$ and $O = (0, 0, 0)$ are isolated invariant sets in X, $W^S(E_0) \cap X_0 = \emptyset$, and $W^S(O) \cap X_0 = \emptyset$. By Theorem 1.3.1 and Remark 1.3.1 in [46], it can be obtained that P is uniformly persistent with respect to $(X_0, \partial X_0)$; that is, there exists a $\delta > 0$ such that any solution $(S(t), E(t), I(t))$ of system (11) with the initial value $(S(0), E(0), I(0)) \in \{(S, E, I) \in X : E > 0,\ I > 0\}$ satisfies

$$\liminf_{t\to\infty} E(t) \geq \delta, \qquad \liminf_{t\to\infty} I(t) \geq \delta. \tag{38}$$

Applying Theorem 1.3.6 in [46], P has a fixed point

$$(S^*(0), E^*(0), I^*(0)) \in X_0. \tag{39}$$

From (33), we know $S^* > 0$, for all $t \in [0, \omega]$. $S^*(t)$ is also more than zero for all $t > 0$ due to the periodicity. Similarly, for all $t \geq 0$, $E^*(t) > 0$, $I^*(t) > 0$. Therefore, it can be obtained that one of the positive ω-periodic solutions of system (11) is $(S^*(t), E^*(t), I^*(t))$. □

3.4. Numerical Simulations. Firstly, we give some notations. If $g(t)$ is a periodic function with period ω, we define $\overline{g} = (1/\omega)\int_0^\omega g(t)dt$, $g^l = \min_{t\in[0,\omega]} g(t)$, $g^u = \max_{t\in[0,\omega]} g(t)$. As described in the previous section,

$$R_1(t) = 1 + \frac{\mu(m+\mu)(m+\sigma)(R_0 - 1)}{m\beta(t)(m+\sigma+\mu)}. \tag{40}$$

So $R_1^l = 1 + \mu(m+\mu)(m+\sigma)(R_0^l - 1)/m\beta^u(m+\sigma+\mu)$, $R_1^u = 1 + \mu(m+\mu)(m+\sigma)(R_0^u - 1)/m\beta^l(m+\sigma+\mu)$.

In this section, we adopt $\beta(t) = a[1 + b\sin(\pi t/10)]$. Then, applying the numerical simulation to verify the above solution, we give the following conclusion:

(1) when $R_d < 1$, O is stable;

(2) when $R_d > 1$ and $R_0 < 1$, E_0 is stable; see Figure 3(a);

(3) when $R_0 > 1$ and $r > m + \mu$, system (11) has at least one positive periodic solution; see Figure 3(b).

We can give more results about the conditions of existence of the positive periodic solution.

(1′) When $R_d > 1$, $R_0 > 1$, and $R_d < R_1^u$, O is stable; see Figures 4(a) and 4(b).

(2′) When $R_d > 1$, $R_0 > 1$, and $R_d > R_1^u$, system (11) has at least one positive periodic solution, see Figure 5.

By numerical simulations, we can give that the conditions which ensure the existence of positive periodic solution are $R_0 > 1$ and $r > m + \mu$ or $R_d > 1$, $R_0 > 1$, and $R_d > R_1^u$. In fact, $R_d > 1$, $R_0 > 1$, and $R_d > R_1^u$ are the sufficient conditions for $R_0 > 1$ and $r > m + \mu$. As a result, the conditions $R_0 > 1$ and $r > m + \mu$ are broader.

4. Discussion

This paper considers a logistic growth system whose birth process incorporates density-dependent effects. This type of model has a rich dynamical behavior and practical significance. By analyzing its equilibria and respective attractive region, we find that the dynamical behavior of a disease will

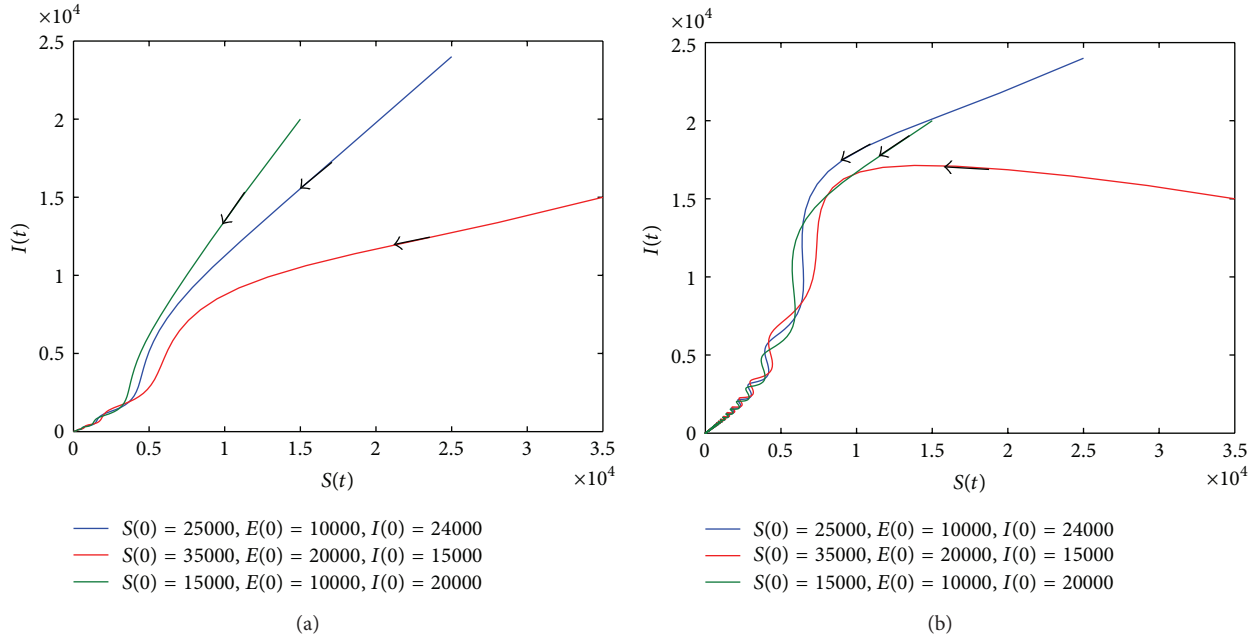

FIGURE 4: Phase plane of $S(t)$ and $I(t)$. (a) When the parameter values are $r = 0.11$, $a = 0.9$, $b = 0.2$ and $\mu = 0.3$, $R_0 = 1.4991$, $R_d = 1.1 > 1$, $R_d < R_1^u = 1.4159$, and $R_d < R_1^l = 1.2773$. (b) When the parameter values are $r = 0.126$, $a = 0.7$, $b = 0.2$, and $\mu = 0.3$, $R_0 = 2.1067$, $R_d = 1.26 > 1$, $R_d < R_1^u = 1.2964$, and $R_d > R_1^l = 1.1976$. The value of other parameters can be seen in Table 1.

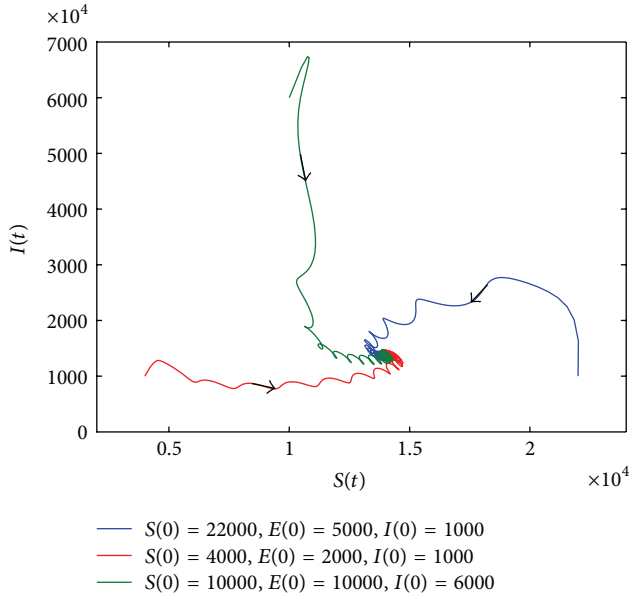

FIGURE 5: Phase plane of $S(t)$ and $I(t)$. When the parameter values are $r = 0.13$, $a = 0.36$, and $b = 0.2$, $R_0 = 1.0834$, $R_d = 1.3 > 1$, $R_d > R_1^u = 1.0435$, and $R_d > R_1^l = 1.029$. The value of other parameters can be seen in Table 1.

be determined by two thresholds R_0 and R_d. Only $R_0 > 1$ cannot promise the existence of the endemic equilibrium which also needs $R_d > R_1$. When $R_0 > 1$ and $R_d < R_1$, the solutions of the system (1) will tend to the origin O. It is caused by the phenomenon that the death number due to disease cannot be supplemented by the birth number promptly. Finally, all people are infected and die out. The fact interpreted by this model is more reasonable. Theoretically, we prove the global asymptotic stability of the disease-free equilibrium and give respective attractive regions of equilibria.

Seasonally effective contact rate is the most common form which may be related to various factors, and thus this paper studies the corresponding non-autonomous system which is obtained by changing the constant transmission rate of the above system into the periodic transmission rate. For the periodic systems, their dynamical behaviors, especially the basic reproduction number, have been investigated in depth by [41, 47–55] which provide many methods that we can utilize. For the obtained periodic model, by analyzing the global asymptotic stability of the disease-free equilibrium and the existence of positive periodic solution, we have the similar results as the autonomous system. The dynamic behavior of disease will be decided by two conditions $R_0 > 1$ and $r > m + \mu$ that show that when the disease is prevalent, the birth rate should be larger than the death rate to guarantee the sustainable growth of population. Otherwise, the population will disappear. In addition, we will evaluate and compare the basic reproduction number R_0 and the average basic reproduction number $\overline{R}_0$ which has been adopted by [27, 56–59]. In this paper, we can calculate the average reproduction number

$$\overline{R}_0 = \frac{\overline{\beta}\sigma}{(m+\sigma)(m+\mu)}, \tag{41}$$

where $\overline{\beta} = (1/20)\int_0^{20}\beta(t)dt$. When $r = 0.13$, $a = 0.3$, and $b = 0.2$, we know that $R_0 = 0.9029$ and $\overline{R}_0 = 1$. When $r = 0.13$, $k = 100000$, $a = 0.36$, $b = 0.2$, and $m = 0.1$, $\sigma = 0.2$, $\mu = 0.1$, then $R_0 = 1.0834$ and $\overline{R}_0 = 1.2$. In that sense, it is confirmed that the basic reproduction number R_0 defined by [40] is more accurate than the average reproduction number $\overline{R}_0$ which overestimates the risk of disease.

It should be noted that we live in a spatial world and it is a natural phenomenon that a substance goes from high density regions to low density regions. As a result, epidemic models should include spatial effects. In a further study, we need to investigate spatial epidemic models with seasonal factors.

Acknowledgments

The research was partially supported by the National Natural Science Foundation of China under Grant nos. 11301490, 11301491, 11331009, 11147015, 11171314, 11101251, and 11105024, Natural Science Foundation of ShanXi Province Grant no. 2012021002-1, and the Opening Foundation of Institute of Information Economy, Hangzhou Normal University, Grant no. PD12001003002003.

References

[1] K. Dietz and J. A. P. Heesterbeek, "Bernoulli was ahead of modern epidemiology," *Nature*, vol. 408, no. 6812, pp. 513–514, 2000.

[2] K. Dietz and J. A. P. Heesterbeek, "Daniel Bernoulli's epidemiological model revisited," *Mathematical Biosciences*, vol. 180, pp. 1–21, 2002.

[3] R. Ross, "An application of the theory of probabilities to the study of a priori pathometry (part I)," *Proceedings of the Royal Society A*, vol. 92, no. 638, pp. 204–230, 1916.

[4] R. Ross and H. P. Hudson, "An application of the theory of probabilities to the study of a priori pathometry (part II)," *Proceedings of the Royal Society A*, vol. 93, no. 650, pp. 212–225, 1917.

[5] W. O. Kermack and A. G. McKendrick, "A contribution to the mathematical theory of epidemics (part I)," *Proceedings of the Royal Society A*, vol. 115, no. 772, pp. 700–721, 1927.

[6] W. O. Kermack and A. G. McKendrick, "A contribution to the mathematical theory of epidemics (part II)," *Proceedings of the Royal Society A*, vol. 138, no. 834, pp. 55–83, 1932.

[7] A. Tsoularis and J. Wallace, "Analysis of logistic growth models," *Mathematical Biosciences*, vol. 179, no. 1, pp. 21–55, 2002.

[8] I. Nåsell, "On the quasi-stationary distribution of the stochastic logistic epidemic," *Mathematical Biosciences*, vol. 156, no. 1-2, pp. 21–40, 1999.

[9] H. Fujikawa, A. Kai, and S. Morozumi, "A new logistic model for *Escherichia coli* growth at constant and dynamic temperatures," *Food Microbiology*, vol. 21, no. 5, pp. 501–509, 2004.

[10] L. Berezansky and E. Braverman, "Oscillation properties of a logistic equation with several delays," *Journal of Mathematical Analysis and Applications*, vol. 247, no. 1, pp. 110–125, 2000.

[11] M. Tabata, N. Eshima, and I. Takagi, "The nonlinear integro-partial differential equation describing the logistic growth of human population with migration," *Applied Mathematics and Computation*, vol. 98, pp. 169–183, 1999.

[12] L. Korobenko and E. Braverman, "On logistic models with a carrying capacity dependent diffusion: stability of equilibria and coexistence with a regularly diffusing population," *Nonlinear Analysis: Real World Applications*, vol. 13, no. 6, pp. 2648–2658, 2012.

[13] Y. Muroya, "Global attractivity for discrete models of nonautonomous logistic equations," *Computers and Mathematics with Applications*, vol. 53, no. 7, pp. 1059–1073, 2007.

[14] S. Invernizzi and K. Terpin, "A generalized logistic model for photosynthetic growth," *Ecological Modelling*, vol. 94, no. 2-3, pp. 231–242, 1997.

[15] Z. Min, W. Bang-Jun, and J. Feng, "Coalmining cities' economic growth mechanism and sustainable development analysis based on logistic dynamics model," *Procedia Earth and Planetary Science*, vol. 1, no. 1, pp. 1737–1743, 2009.

[16] L. I. Aniţa, S. Aniţa, and V. Arnăutu, "Global behavior for an age-dependent population model with logistic term and periodic vital rates," *Applied Mathematics and Computation*, vol. 206, no. 1, pp. 368–379, 2008.

[17] Z. A. Banaszak, X. Q. Tang, S. C. Wang, and M. B. Zaremba, "Logistics models in flexible manufacturing," *Computers in Industry*, vol. 43, no. 3, pp. 237–248, 2000.

[18] S. Brianzoni, C. Mammana, and E. Michetti, "Nonlinear dynamics in a business-cycle model with logistic population growth," *Chaos, Solitons and Fractals*, vol. 40, no. 2, pp. 717–730, 2009.

[19] W. P. London and J. A. Yorke, "Recurrent outbreaks of measles, chickenpox and mumps. I. Seasonal variation in contact rates," *The American Journal of Epidemiology*, vol. 98, no. 6, pp. 453–468, 1973.

[20] S. F. Dowell, "Seasonal variation in host susceptibility and cycles of certain infectious diseases," *Emerging Infectious Diseases*, vol. 7, no. 3, pp. 369–374, 2001.

[21] O. N. Bjørnstad, B. F. Finkenstädt, and B. T. Grenfell, "Dynamics of measles epidemics: estimating scaling of transmission rates using a Time series SIR model," *Ecological Monographs*, vol. 72, no. 2, pp. 169–184, 2002.

[22] J. Zhang, Z. Jin, G. Q. Sun, X. Sun, and S. Ruan, "Modeling seasonal rabies epidemics in China," *Bulletin of Mathematical Biology*, vol. 74, no. 5, pp. 1226–1251, 2012.

[23] I. B. Schwartz, "Small amplitude, long period outbreaks in seasonally driven epidemics," *Journal of Mathematical Biology*, vol. 30, no. 5, pp. 473–491, 1992.

[24] I. B. Schwartz and H. L. Smith, "Infinite subharmonic bifurcation in an SEIR epidemic model," *Journal of Mathematical Biology*, vol. 18, no. 3, pp. 233–253, 1983.

[25] H. L. Smith, "Multiple stable subharmonics for a periodic epidemic model," *Journal of Mathematical Biology*, vol. 17, no. 2, pp. 179–190, 1983.

[26] J. Dushoff, J. B. Plotkin, S. A. Levin, and D. J. D. Earn, "Dynamical resonance can account for seasonality of influenza epidemics," *Proceedings of the National Academy of Sciences of the United States of America*, vol. 101, no. 48, pp. 16915–16916, 2004.

[27] J. L. Ma and Z. Ma, "Epidemic threshold conditions for seasonally forced SEIR models," *Mathematical Biosciences and Engineering*, vol. 3, no. 1, pp. 161–172, 2006.

[28] D. J. D. Earn, P. Rohani, B. M. Bolker, and B. T. Grenfell, "A simple model for complex dynamical transitions in epidemics," *Science*, vol. 287, no. 5453, pp. 667–670, 2000.

[29] M. J. Keeling, P. Rohani, and B. T. Grenfell, "Seasonally forced disease dynamics explored as switching between attractors," *Physica D*, vol. 148, no. 3-4, pp. 317–335, 2001.

[30] J. L. Aron and I. B. Schwartz, "Seasonality and period-doubling bifurcations in an epidemic model," *Journal of Theoretical Biology*, vol. 110, no. 4, pp. 665–679, 1984.

[31] N. C. Grassly and C. Fraser, "Seasonal infectious disease epidemiology," *Proceedings of the Royal Society B*, vol. 273, no. 1600, pp. 2541–2550, 2006.

[32] J. Zhang, Z. Jin, G. Q. Sun, T. Zhou, and S. Ruan, "Analysis of rabies in China: transmission dynamics and control," *PLoS ONE*, vol. 6, no. 7, Article ID e20891, 2011.

[33] O. Diekmann, J. A. P. Heesterbeek, and J. A. Metz, "On the definition and the computation of the basic reproduction ratio R_0 in models for infectious diseases in heterogeneous populations," *Journal of mathematical biology*, vol. 28, no. 4, pp. 365–382, 1990.

[34] O. Diekmann, J. A. P. Heesterbeek, and M. G. Roberts, "The construction of next-generation matrices for compartmental epidemic models," *Journal of the Royal Society Interface*, vol. 7, no. 47, pp. 873–885, 2010.

[35] P. van den Driessche and J. Watmough, "Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission," *Mathematical Biosciences*, vol. 180, pp. 29–48, 2002.

[36] F. Berezovsky, G. Karev, B. J. Song, and C. C. Chavez, "A simple epidemic model with surprising dynamics," *Mathematical Biosciences and Engineering*, vol. 2, pp. 133–152, 2005.

[37] J. LaSalle and S. Lefschetz, *Stability by Liapunov's Direct Method*, Academic Press, New York, NY, USA, 1961.

[38] E. A. Barbashin, *Introduction to the Theory of Stability*, Wolters-Noordhoff, Groningen, The Netherlands, 1970.

[39] D. Schenzle, "An age-structured model of pre- and post-vaccination measles transmission," *Mathematical Medicine and Biology*, vol. 1, no. 2, pp. 169–191, 1984.

[40] N. Bacaër and S. Guernaoui, "The epidemic threshold of vector-borne diseases with seasonality: the case of cutaneous leishmaniasis in Chichaoua, Morocco," *Journal of Mathematical Biology*, vol. 53, no. 3, pp. 421–436, 2006.

[41] W. D. Wang and X. Q. Zhao, "Threshold dynamics for compartmental epidemic models in periodic environments," *Journal of Dynamics and Differential Equations*, vol. 20, no. 3, pp. 699–717, 2008.

[42] F. Zhang and X. Q. Zhao, "A periodic epidemic model in a patchy environment," *Journal of Mathematical Analysis and Applications*, vol. 325, no. 1, pp. 496–516, 2007.

[43] H. L. Smith and P. Waltman, *The Theory of the Chemostat*, Cambridge University Press, New York, NY, USA, 1995.

[44] H. R. Thieme, "Convergence results and a Poincaré-Bendixson trichotomy for asymptotically automous differential equations," *Journal of Mathematical Biology*, vol. 30, pp. 755–763, 1992.

[45] L. Perko, *Differential Equations and Dynamical Systems*, Springer, New York, NY, USA, 2000.

[46] X. Q. Zhao, *Dynamical Systems in Population Biology*, Springer, New York, NY, USA, 2003.

[47] Z. G. Bai and Y. C. Zhou, "Threshold dynamics of a bacillary dysentery model with seasonal fluctuation," *Discrete and Continuous Dynamical Systems B*, vol. 15, no. 1, pp. 1–14, 2011.

[48] Z. G. Bai, Y. C. Zhou, and T. L. Zhang, "Existence of multiple periodic solutions for an SIR model with seasonality," *Nonlinear Analysis: Theory, Methods and Applications*, vol. 74, no. 11, pp. 3548–3555, 2011.

[49] N. Bacaër, "Approximation of the basic reproduction number R_0 for vector-borne diseases with a periodic vector population," *Bulletin of Mathematical Biology*, vol. 69, no. 3, pp. 1067–1091, 2007.

[50] N. Bacaër and X. Abdurahman, "Resonance of the epidemic threshold in a periodic environment," *Journal of Mathematical Biology*, vol. 57, no. 5, pp. 649–673, 2008.

[51] N. Bacaër, "Periodic matrix population models: growth rate, basic reproduction number, and entropy," *Bulletin of Mathematical Biology*, vol. 71, no. 7, pp. 1781–1792, 2009.

[52] N. Bacaër and E. H. A. Dads, "Genealogy with seasonality, the basic reproduction number, and the influenza pandemic," *Journal of Mathematical Biology*, vol. 62, no. 5, pp. 741–762, 2011.

[53] L. J. Liu, X. Q. Zhao, and Y. C. Zhou, "A tuberculosis model with seasonality," *Bulletin of Mathematical Biology*, vol. 72, no. 4, pp. 931–952, 2010.

[54] J. L. Liu, "Threshold dynamics for a HFMD epidemic model with periodic transmission rate," *Nonlinear Dynamics*, vol. 64, no. 1-2, pp. 89–95, 2011.

[55] Y. Nakata and T. Kuniya, "Global dynamics of a class of SEIRS epidemic models in a periodic environment," *Journal of Mathematical Analysis and Applications*, vol. 363, no. 1, pp. 230–237, 2010.

[56] B. G. Williams and C. Dye, "Infectious disease persistence when transmission varies seasonally," *Mathematical Biosciences*, vol. 145, no. 1, pp. 77–88, 1997.

[57] I. A. Moneim, "The effect of using different types of periodic contact rate on the behaviour of infectious diseases: a simulation study," *Computers in Biology and Medicine*, vol. 37, no. 11, pp. 1582–1590, 2007.

[58] C. L. Wesley and L. J. S. Allen, "The basic reproduction number in epidemic models with periodic demographics," *Journal of Biological Dynamics*, vol. 3, no. 2-3, pp. 116–129, 2009.

[59] D. Greenhalgh and I. A. Moneim, "SIRS epidemic model and simulations using different types of seasonal contact rate," *Systems Analysis Modelling Simulation*, vol. 43, no. 5, pp. 573–600, 2003.