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Global analysis for a general epidemiological model with vaccination and varying population

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ABSTRACT

An *SIR* model with vaccination and varying population is formulated. The global dynamics of this model and its corresponding proportionate system are investigated. The correlations between the two systems in terms of disease eradication and persistence are presented. Three critical vaccination rates ϕ_{1c}, ϕ_{2c} and ϕ_{3c} are obtained. It is found that when $\phi > \phi_{1c}$ the disease can be eradicated by increasing the vaccination rate until it exceeds ϕ_{3c}. When $\phi < \phi_{1c}$, the disease can be controlled to an endemic level by taking the appropriate vaccination rate ϕ_{2c}.

1. Introduction

The spread of communicable diseases is often described mathematically by compartmental models. In 1927, Kermack and McKendrick proposed, as a particular case of a more general model presented in their seminal work [14], a classical *SIR* model for the number of people infected with a disease in a closed population, which well explained the rapid rise and fall in the number of infected patients during the plague (London, 1665–1666) and cholera (London, 1865). In their model, S, I and R denote respectively the number of individuals susceptible to the disease, the number of infectives and the number of individuals who are removed from being infectious, through recovery or death. Following an infecting contact, susceptible individuals (S) become infectious (I) and later recover or die (R). One of the distinctive characteristics of the model of [14] is that it assumes a constant population not subject to any demographic processes. By extending the ordinary differential equations model in [14] to a fluctuating population (with births and deaths), Anderson and May (1979) [1] brought the Kermack–McKendrick model back to prominence after decades of neglect.

More complicated *SIR* models were then formulated from different perspectives of epidemiology and demography. Capasso and Serio [7], Xiao and Ruan [19] focused on the vital effects of specific non-linear incidence rates on the *SIR* model. Cooke [9], Beretta and Takeuchi [3] incorporated a time delay to *SIR* models to account for disease incubation, and carried out the stability analysis.

The inclusion of practical control strategies in models is important in order to assess the intervention of public health authorities. There are two major types of control strategies available to curtail the spread of infectious diseases: pharmaceutical interventions (drugs, vaccines) and non-pharmaceutical interventions (social distancing, quarantine). Vaccination, when it is available, is an effective preventive strategy. Arino et al. [2] introduced vaccination of susceptible individuals into an *SIRS* model and also considered vaccinating a fraction of newborns. Buonomo et al. [6] studied the traditional *SIR* model

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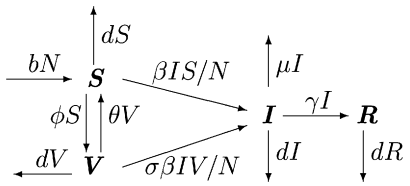

Fig. 1. Flow diagram of the *SIRV* model.

with 100% efficacious vaccine. Pulse vaccination was highly successful in the control of poliomyelitis and measles throughout central and south America (see [10,17]), and therefore has gained in prominence. Shulgin et al. [18] incorporated it to an *SIR* epidemic model. In fact, the large majority of vaccines are imperfect and their protections from catching the disease wane as time evolves.

Most disease modeling literature assumes constant or asymptotically constant total population. This seems relatively plausible for diseases with short duration (influenza, SARS, ...) and negligible mortality rate (West Nile virus in human or livestock). However, for endemic diseases (malaria) or diseases with high mortality rate (HIV/AIDS in poor countries), the changes in population size are far from negligible. The total population changes through natural birth and death as well as disease-induced death, which may imbalance the inflow and outflow of a given population and thus cause the total population vary with time. For models with varying population size, we refer readers to [5,13,8,12,4,15]. Most of these works deal with a specific disease, or are general models that do not consider a disease control strategy.

In this paper, we revisit the general *SIR* model [1]; by adding non-constant population to the vaccination model in [2], we formulate an *SIRV* epidemiological model with varying total population by considering the imperfect vaccination towards a portion of susceptible individuals. In the case of varying total population $N(t)$, one typically considers first the transformed proportionate system describing the evolution of the fractions of individuals in each of the compartments [5,15], which in turn determines the dynamical behaviors of the population sizes. Although this technique reduces the dimensionality of the system and simplifies the mathematical analysis, the dynamics of two systems are not always identical, sometimes very different in determining the epidemiological properties of communicable diseases [5,15]. Therefore, for models with varying total population, the notions of endemicity and eradication of diseases can be somewhat confusing. Li et al. [15], for example, define endemicity using the fraction of infected individuals in the population. To be in accordance with the epidemiological definition of the term, in this paper, we call disease prevalence the fraction $i = I/N$ and say that the disease is endemic when the infected population size I persists above a certain positive level. We then add quotation marks (" ") when studying the proportionate system, referring in that case to disease-"free" and "endemic" situations. This is important, as it may be possible for the disease to be "endemic" in the proportionate system while the total population is going extinct, leading in effect to a disease-free (and population-free) situation.

The existence and uniqueness of the "endemic" equilibrium is established by studying the intersection of two polynomial functions. A modified vaccination rate ϕ_{1c} is then derived. We prove the global asymptotic stability of the disease-"free" equilibrium e_0 by using a real analysis method, quite different from the traditional ways of constructing suitable Lyapunov functions or applying the LaSalle's Invariance Principle. The sufficient conditions are obtained for the "endemic" equilibrium e_* by the autonomous convergence theorem [15]. With the help of global dynamic behaviors in the proportionate system, we present the global properties of the population model and derive the two critical vaccination rates ϕ_{2c} and ϕ_{3c}. The main mathematical results are summarized in Table 1 and Table 2. This paper ends up with a discussion, in which the epidemiological implications are compared and explained on the global dynamics of both the proportionate system and the population system. Numerical simulations are carried out to illustrate the obtained results and also to find out the dynamic behaviors of the two systems unobtainable by analytical analysis.

2. Model formulation

The total population $N(t)$ is partitioned into four compartments depending on the epidemiological status of individuals: fully susceptible $S(t)$, vaccinated susceptible $V(t)$, infectious $I(t)$ and recovered with full immunity $R(t)$. The model transfer diagram indicating the possible transitions between these compartments is shown in Fig. 1.

The total population increases by birth at the rate b and decreases at the rate constant d because of natural death. Also, some disease-induced death contributes to an additional population decrease at the constant rate μ. The population is assumed to undergo homogeneous mixing. We assume that each infective individual contacts an average number ς other individuals per unit time. Hence, the total number of contact by infectives per unit time is ςI, among which a proportion S/N (V/N) is with susceptible (vaccinated) individuals. We assume that a proportion ν of the contacts between an infective and a susceptible (vaccinated) individual are effective in transmitting the disease. The incidence rate at which the susceptible (vaccinated) become infected is $\beta IS/N$ ($\beta IV/N$), where the transmission coefficient $\beta = \nu\varsigma$. Susceptible individuals are vaccinated at rate ϕ. Since the vaccine only provides partial protection to the infection, vaccinated individuals may still become infected, but at the lower infection rate $\sigma\beta$ than fully susceptible individuals, where $1-\sigma \in [0,1]$ describes vaccine efficacy: when $\sigma = 0$, the vaccine is perfectly effective and when $\sigma = 1$, the vaccine has no effect at all on the immunity of vaccinated individuals. The effect of vaccination is assumed to wane at the rate constant θ, i.e., vaccinated individuals

are protected by the vaccine for an average $1/\theta$ time units. Parameter γ is the disease recovery rate from the infective compartment. We assume that the disease transmits only horizontally in the population; there is no transmission between parents and their offspring.

The model differential equations are derived based on the aforementioned basic assumptions and Fig. 1.

$$\begin{aligned}
\frac{dS}{dt} &= bN - \beta IS/N - (d+\phi)S + \theta V,\\
\frac{dI}{dt} &= \beta IS/N + \sigma\beta IV/N - (d+\mu+\gamma)I,\\
\frac{dR}{dt} &= \gamma I - dR,\\
\frac{dV}{dt} &= \phi S - \sigma\beta IV/N - (d+\theta)V.
\end{aligned} \tag{1}$$

The total population $N = S + I + R + V$ is governed by

$$\frac{dN}{dt} = (b-d)N - \mu I, \tag{2}$$

which is derived by adding the four equations in (1).

Remark 1. The formulation of (1) is very similar to that of [8]. The main differences are as follows. Model (1) is *SIR* while [8] is *SIRS*. In (1), all newborns are susceptible, while in [8] a certain fraction are vaccinated at birth. Model (1) incorporates disease induced death and different birth and death rates. As a consequence, the total population $N(t)$ is variable in (1), whereas it is a constant in [8].

3. Dimensionless transformation

Denote $s = S/N$, $i = I/N$, $r = R/N$ and $v = V/N$ the fractions of the number of individuals in compartments S, I, R and V in the total population N, respectively. It is easy to verify that s, i, r and v satisfy the system

$$\begin{aligned}
\frac{ds}{dt} &= b - \beta si - (b+\phi)s + \theta v + \mu si,\\
\frac{di}{dt} &= \beta si + \sigma\beta iv - (b+\mu+\gamma)i + \mu i^2,\\
\frac{dr}{dt} &= \gamma i - br + \mu ir,\\
\frac{dv}{dt} &= \phi s - \sigma\beta iv - (b+\theta)v + \mu iv,
\end{aligned} \tag{3}$$

where solutions are restricted to the hyperplane $s + i + r + v = 1$. Also observe that the variable r does not appear in the other three equations of (3). We can then attack (3) by studying the subsystem

$$\begin{aligned}
\frac{ds}{dt} &= b - \beta si - (b+\phi)s + \theta v + \mu si,\\
\frac{di}{dt} &= \beta si + \sigma\beta iv - (b+\mu+\gamma)i + \mu i^2,\\
\frac{dv}{dt} &= \phi s - \sigma\beta iv - (b+\theta)v + \mu iv,
\end{aligned} \tag{4}$$

and determining r from

$$\frac{dr}{dt} = \gamma i - br + \mu ir \tag{5}$$

or from $r = 1 - s - i - v$.

We study (4) in the closed, positively invariant set

$$\Sigma = \left\{(s, i, v) \in \mathbb{R}^3_+ \;\middle|\; 0 \leqslant s + i + v \leqslant 1\right\},$$

where $\mathbb{R}^3_+$ denotes the non-negative cone of $\mathbb{R}^3$ with its lower dimensional faces. We denote by $\partial\Sigma$ and $\mathring{\Sigma}$ the boundary and the interior of Σ in $\mathbb{R}^3_+$, respectively.

In the next few sections, the reduced proportionate system (4) will be analyzed first.

4. Existence of equilibria

System (4) always has a disease-"free" equilibrium (DFE) e_0, where

$$e_0 := (s_0, i_0, v_0) = \left(\frac{b+\theta}{b+\theta+\phi}, 0, \frac{\phi}{b+\theta+\phi}\right).$$

Suppose that $e_* := (s_*, i_*, v_*)$ is an "endemic" equilibrium (EE) of (4). From the right-hand side of (5) we have $\gamma i_* = r(b-\mu i_*) > 0$, which implies that

$$0 < i_* < \min\{1, b/\mu\}. \tag{6}$$

Let the right side of (4) equal zero. A straightforward calculation leads to

$$\begin{aligned} s_* &= \frac{b[(\sigma\beta-\mu)i_* + b + \theta]}{[(\sigma\beta-\mu)i_* + b + \theta][(\beta-\mu)i_* + b + \phi] - \phi\theta}, \\ v_* &= \frac{b\phi}{[(\sigma\beta-\mu)i_* + b + \theta][(\beta-\mu)i_* + b + \phi] - \phi\theta}, \end{aligned} \tag{7}$$

and the component i_* is a positive solution of equation

$$f(i) = g(i) \tag{8}$$

satisfying (6), where

$$\begin{aligned} f(i) &\triangleq \big[(\sigma\beta-\mu)i + b + \theta\big]\big[(\beta-\mu)i + b + \phi\big]\big[\mu i - (b+\gamma+\mu)\big], \\ g(i) &\triangleq \big[\phi\theta\mu - b\beta(\sigma\beta-\mu)\big]i - \phi\theta(b+\gamma+\mu) - b\beta(b+\theta+\sigma\phi). \end{aligned} \tag{9}$$

We have

$$\begin{aligned} &f(0) = -(b+\theta)(b+\phi)(b+\gamma+\mu) < 0, \\ &g(0) = -\phi\theta(b+\gamma+\mu) - b\beta(b+\theta+\sigma\phi) < 0, \\ &f(1) = -(b+\gamma)(\sigma\beta-\mu+b+\theta)(\beta-\mu+b+\phi), \\ &g(1) = -\big[b\beta(\sigma\beta-\mu) + \theta\phi(b+\gamma) + b\beta(b+\theta+\sigma\phi)\big], \\ &f(b/\mu) = -(\mu+\gamma)(\sigma\beta b/\mu + \theta)(\beta b/\mu + \phi) < 0, \\ &g(b/\mu) = -\big[\sigma b^2\beta^2/\mu + \phi\theta(\gamma+\mu) + b\beta(\theta+\sigma\phi)\big] < 0. \end{aligned} \tag{10}$$

Denote the threshold parameter as

$$\mathcal{R}_{\text{vac}} = \frac{\beta s_0 + \sigma\beta v_0}{b+\gamma+\mu} = \frac{\beta(b+\theta+\sigma\phi)}{(b+\gamma+\mu)(b+\theta+\phi)}. \tag{11}$$

We derive a critical vaccination rate ϕ_{1c} by solving $\mathcal{R}_{\text{vac}} = 1$ in terms of ϕ, giving

$$\phi_{1c} = \frac{(b+\theta)[\beta - (b+\mu+\gamma)]}{(b+\mu+\gamma) - \beta\sigma}.$$

It follows that $\mathcal{R}_{\text{vac}} < 1 \Leftrightarrow \phi > \phi_{1c}$, $\mathcal{R}_{\text{vac}} > 1 \Leftrightarrow \phi < \phi_{1c}$.

Besides the threshold parameter $\mathcal{R}_{\text{vac}}$, we derive the critical vaccination rate ϕ_{1c} here in order to investigate the impact of different vaccination strategies on disease control.

From (10), we have

$$\begin{aligned} &\big|f(0)\big| - \big|g(0)\big| = b(b+\gamma+\mu)(b+\theta+\phi)(1-R_{\text{vac}}), \\ &\big|f(b/\mu)\big| - \big|g(b/\mu)\big| = \epsilon b\beta(\sigma b\beta + \sigma\phi\mu + \theta\mu)/\mu^2. \end{aligned}$$

Obviously, $|f(b/\mu)| > |g(b/\mu)|$. Also $|f(0)| < |g(0)|$ is guaranteed by $R_{\text{vac}} > 1$ (i.e., $\phi < \phi_{1c}$), from which $\beta > \mu$ can be deduced. Moreover, if $b/\mu \geqslant 1$, then $|f(1)| - |g(1)| = b(\sigma\beta-\mu+b+\theta+\phi)(b-\mu) + \gamma(\sigma\beta-\mu+b+\theta)(\beta-\mu+b) + \gamma\phi(\sigma\beta-\mu+b) > 0$.

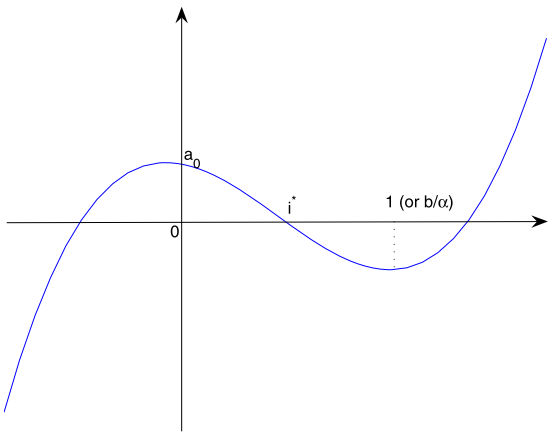

Fig. 2.1. $\sigma\beta > \mu$.

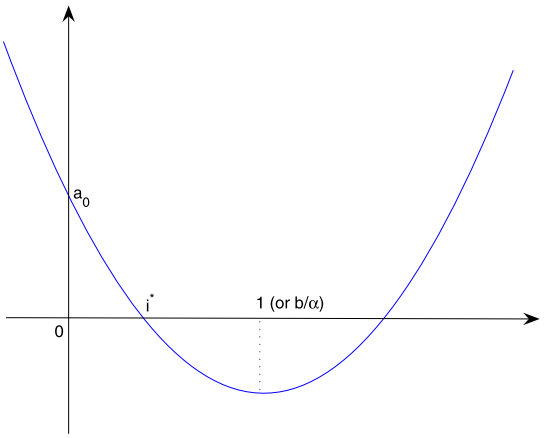

Fig. 2.2. $\sigma\beta = \mu$.

Denote $\varphi(i) = f(i) - g(i) = a_3 i^3 + a_2 i^2 + a_1 i + a_0 = 0$, where

$$
\begin{aligned}
a_3 &= \mu(\sigma\beta - \mu)(\beta - \mu),\\
a_2 &= \mu(\beta - \mu)(b + \theta) + (\sigma\beta - \mu)\mu(b + \phi) - (\sigma\beta - \mu)(\beta - \mu)(b + \mu + \gamma),\\
a_1 &= \mu(b + \theta)(b + \phi) - (\sigma\beta - \mu)(b + \phi)(b + \gamma + \mu) - \big[\phi\theta\mu - b\beta(\sigma\beta - \mu)\big] - (\beta - \mu)(b + \theta)(b + \gamma + \mu),\\
a_0 &= b(b + \theta + \phi)(b + \epsilon + \mu)(R_{\text{vac}} - 1).
\end{aligned}
$$

(i) Assume $\mathcal{R}_{\text{vac}} > 1$.

(1) If $\sigma\beta > \mu$, then $a_3 > 0$. We have $\varphi(-\infty) < 0$, $\varphi(+\infty) > 0$, and $\varphi(0) = a_0 > 0$. Moreover, $\varphi(1) = f(1) - g(1) < 0$ (if $b/\mu \geqslant 1$) and $\varphi(b/\mu) = f(b/\mu) - g(b/\mu) < 0$. Therefore, we obtain a unique i_* such that $\varphi(i_*) = 0$ (see Fig. 2.1).
(2) If $\sigma\beta = \mu$, then $a_3 = 0$ and $\varphi(i) = a_2 i^2 + a_1 i + a_0$, where $a_2 = \mu(\beta - \mu)(b + \theta) > 0$. $\varphi(-\infty) > 0$, $\varphi(+\infty) > 0$, $\varphi(0) = a_0 > 0$, $\varphi(1) < 0$ (if $b/\mu \geqslant 1$) and $\varphi(b/\mu) < 0$. Therefore, there is a unique i_* such that $\varphi(i_*) = 0$ (see Fig. 2.2).

(ii) Assume $\mathcal{R}_{\text{vac}} < 1$.

(1) If $\mu \geqslant \beta$, we deduce from the equation for i in (4) that

$$
\begin{aligned}
\frac{\mathrm{d}i}{\mathrm{d}t} &= i\big(\beta s + \sigma\beta v - (b + \mu + \gamma) + \mu i\big)\\
&\leqslant i\big(\mu(s + v + i) - (b + \mu + \gamma)\big)\\
&\leqslant -i(b + \epsilon),
\end{aligned}
$$

which implies $\lim_{t\to\infty} i(t) = 0$. Then there is no positive root for $\varphi(i) = 0$.
(2) If $\mu < \beta$, we can obtain from the equation for s in (4) that

$$
\begin{aligned}
\frac{\mathrm{d}s}{\mathrm{d}t} &= b - \beta s i - (b + \phi)s + \theta(1 - s - i - r) + \mu s i\\
&\leqslant (b + \theta) - (b + \theta + \phi)s,
\end{aligned}
$$

from which, we know that

$$
s(t) \leqslant \left(s(0) - \frac{b + \theta}{b + \theta + \phi}\right)e^{-(b+\theta+\phi)t} + \frac{b + \theta}{b + \theta + \phi} \to \frac{b + \theta}{b + \theta + \phi}.
$$

Then $\forall \delta_1 > 0$, $\exists T > 0$, for all $t > T$,

$$
s(t) \leqslant \frac{b + \theta}{b + \theta + \phi} + \delta_1 \triangleq \bar{s}.
$$

Denote $A(t) = \beta s(t) + \sigma\beta v(t) + \mu i(t) - (b + \mu + \gamma)$. Then

$$
\frac{\mathrm{d}i(t)}{\mathrm{d}t} = A(t)i(t).
$$

If $\sigma\beta \geqslant \mu$, then

$$\begin{aligned}
A(t) &\leqslant \beta s(t) + \sigma\beta\big(v(t)+i(t)\big) - (b+\mu+\gamma)\\
&\leqslant \beta s(t) + \sigma\beta\big(1-s(t)\big) - (b+\mu+\gamma)\\
&= \beta\big(s(t)-\bar{s}\big) + \beta\bar{s} + \sigma\beta\big(1-s(t)\big) + \sigma\beta(1-\bar{s}) - \sigma\beta(1-\bar{s}) - (b+\mu+\gamma)\\
&= \beta\bar{s} + \sigma\beta(1-\bar{s}) + \beta(1-\sigma)\big(s(t)-\bar{s}\big) - (b+\mu+\gamma)\\
&\leqslant \beta\left(\frac{b+\theta}{b+\theta+\phi}+\delta_1\right) + \sigma\beta\left(\frac{\phi}{b+\theta+\phi}-\delta_1\right) - (b+\mu+\gamma)\\
&= (b+\mu+\gamma)(R_{\mathrm{vac}}-1) + \beta\delta_1(1-\sigma).
\end{aligned}$$

If $\mathcal{R}_{\mathrm{vac}} < 1$, then $\mathcal{R}_{\mathrm{vac}} - 1 < 0$. Choose $\delta_1 = \delta^*$ such that $\beta\delta^*(1-\sigma) = (b+\mu+\gamma)(1-\mathcal{R}_{\mathrm{vac}})/2$, then $A(t) < -(b+\mu+\gamma)(1-\mathcal{R}_{\mathrm{vac}})/2$. Therefore, $\lim_{t\to+\infty} i(t) = 0$. Then there is no positive root for $\varphi(i) = 0$.

Based on the discussions above, we obtain the following theorem.

Theorem 4.1. *Assume that $\sigma\beta \geqslant \mu$. If $\mathcal{R}_{\mathrm{vac}} < 1$, then there is only the disease-"free" equilibrium e_0 for system* (4)*; if $\mathcal{R}_{\mathrm{vac}} > 1$, then there is a unique "endemic" equilibrium besides the disease-"free" equilibrium.*

When $\sigma\beta < \mu$, the dynamics are quite complicated, and will be presented in Appendix A. Therefore, in Sections 5, 6 and 7, the analyses are carried out under the condition that $\sigma\beta \geqslant \mu$.

5. Global dynamics

5.1. Global stability of the disease-"free" equilibrium

The Jacobian matrix at the DFE e_0 is

$$J(e_0) = \begin{pmatrix} -(b+\phi) & -\beta s_0 + \mu s_0 & \theta \\ 0 & \beta s_0 + \sigma\beta v_0 - (b+\epsilon+\mu) & 0 \\ \phi & -\sigma\beta v_0 + \mu v_0 & -(b+\theta) \end{pmatrix}.$$

If $\mathcal{R}_{\mathrm{vac}} > 1$, there exists one positive eigenvalue ($\mathcal{R}_{\mathrm{vac}} - 1$) and e_0 is unstable; if $\phi > \phi_{1c}$, all eigenvalues of $J(e_0)$ are negative, which implies that e_0 is locally asymptotically stable.

We next prove that all solutions in Σ are attracted to e_0 when $\mathcal{R}_{\mathrm{vac}} < 1$.

In the previous section, we have proved that $i(t)$ converges to zero globally if $\beta \leqslant \mu$ or $\sigma\beta \geqslant \mu$. Therefore, the asymptotical system for $s(t)$ and $v(t)$ is as follows.

$$\begin{aligned}
\frac{\mathrm{d}s}{\mathrm{d}t} &= b - (b+\phi)s + \theta v \triangleq h_1(s,v),\\
\frac{\mathrm{d}v}{\mathrm{d}t} &= \phi s - (b+\theta)v \triangleq h_2(s,v).
\end{aligned}$$

It is easy to show that

$$\lim_{t\to\infty} s(t) = \frac{b+\theta}{b+\theta+\phi}, \qquad \lim_{t\to\infty} v(t) = \frac{\phi}{b+\theta+\phi}.$$

To sum up, all the solutions in Σ are attracted to e_0. We then obtain the following results.

Theorem 5.1. *If $\mathcal{R}_{\mathrm{vac}} < 1$, then the disease-"free" equilibrium* (*DFE*) *e_0 is locally asymptotically stable in Σ. Moreover, if $\beta \leqslant \mu$ or $\sigma\beta \geqslant \mu$, then e_0 is globally asymptotically stable in Σ.*

5.2. Global stability of the "endemic" equilibrium

In this section, we apply the autonomous convergence theorems in finite dimension, a geometric approach developed by Li and Muldowney [16], to investigate the global asymptotic stability of the unique "endemic" equilibrium when $e_* > 1$. Here we omit the detailed introduction of this approach and refer interested readers to [16].

The Jacobian matrix of system (4) at any point is

$$J=\begin{pmatrix} -(\beta-\mu)i-(b+\phi) & -(\beta-\mu)s & \theta \\ \beta i & \beta s+\sigma\beta v+2\mu i-(b+\mu+\gamma) & \sigma\beta i \\ \phi & -(\sigma\beta-\mu)v & -\sigma\beta i-(b+\theta)+\mu i \end{pmatrix}$$

and its corresponding second compound matrix of J takes the form

$$J^{[2]}=\begin{pmatrix} \beta s+\sigma\beta v-\beta i-(2b+\mu+\epsilon+\phi)+3\mu i & \sigma\beta i & -\theta \\ -(\sigma\beta-\mu)v & -\beta i-\sigma\beta i+2\mu i-(2b+\theta+\phi) & -(\beta-\mu)s \\ -\phi & \beta i & \beta s+\sigma\beta v-\sigma\beta i-(2b+\mu+\gamma+\theta)+3\mu i \end{pmatrix}.$$

Choose $P=\frac{1}{i}I_3$, then $P_fP^{-1}=[(b+\mu+\gamma)-\beta s-\sigma\beta v-\mu i]I_3$, where I_3 is the 3 by 3 identity matrix and P_f is the directional derivative of P in the direction of the vector field of system (4). Moreover,

$$\begin{aligned} Q&=P_fP^{-1}+PJ^{[2]}P^{-1}\\ &=\begin{pmatrix} -\beta i-(b+\phi)+2\mu i & \sigma\beta i & -\theta \\ -\sigma\beta v+\mu v & -\beta i-\sigma\beta i+\mu i-\beta s-\sigma\beta v-(b+\theta+\phi)+\mu+\gamma & -\beta s+\mu s \\ -\phi & \beta i & -\sigma\beta i-(b+\theta)+2\mu i \end{pmatrix}. \end{aligned}$$

Let $z=(z_1,z_2,z_3)^T$ be the solution of the linear homogeneous system $\mathrm{d}z/\mathrm{d}t=Qz$, where

$$\begin{aligned} \frac{\mathrm{d}z_1}{\mathrm{d}t}&=\big[-\beta i+2\mu i-(b+\phi)\big]z_1+\sigma\beta i z_2-\theta z_3,\\ \frac{\mathrm{d}z_2}{\mathrm{d}t}&=\big[-\beta i-\sigma\beta i+\mu i-\beta s-\sigma\beta v+\mu+\gamma-(b+\theta+\phi)\big]z_2+(-\sigma\beta v+\mu v)z_1+(-\beta s+\mu s)z_3,\\ \frac{\mathrm{d}z_3}{\mathrm{d}t}&=-\phi z_1+\beta i z_2+\big[-\sigma\beta i+2\mu i-(b+\theta)\big]z_3. \end{aligned}$$

We have from (3) that

$$\begin{aligned} s'/s&=b/s-\beta i-(b+\phi)+\theta v/s+\mu i,\\ i'/i&=\beta s+\sigma\beta v-(b+\gamma+\mu)+\mu i,\\ v'/v&=\phi s/v-\sigma\beta i-(b+\theta)+\mu i,\\ r'/r&=\gamma i/r-b+\mu i, \end{aligned} \tag{12}$$

where the prime ($'$) stands for the derivative with respect to time t.

Theorem 5.2. *The unique "endemic" equilibrium e_* of (4) is globally asymptotically stable in $\mathring{\Sigma}$ if $\mathcal{R}_{\mathrm{vac}}>1$ and the following inequality is satisfied,*

$$\mu+\gamma<b+\theta. \tag{13}$$

Moreover, e_* attracts all trajectories in Σ except those on the invariant $s-v$ plane which converge to e_0.

Proof. If $\mathcal{R}_{\mathrm{vac}}>1$, then $\beta>b+\gamma+\mu$. We choose the following norm for z,

$$\|z\|=\begin{cases} |z_1|+|z_2|+|z_3|, & \text{if } z_1z_2\geqslant 0 \text{ and } z_2z_3\geqslant 0,\\ \max\{|z_1|+|z_3|,|z_2|+|z_3|\}, & \text{if } z_1z_2<0 \text{ and } z_2z_3\geqslant 0,\\ \max\{\frac{v}{i}|z_1|,|z_2|,\frac{s}{i}|z_3|\}, & \text{if } z_1z_2<0 \text{ and } z_2z_3<0,\\ \max\{|z_1|+|z_3|,|z_2|\}, & \text{if } z_1z_2\geqslant 0 \text{ and } z_2z_3<0. \end{cases}$$

(a) If $z_1z_2 \geqslant 0$ and $z_2z_3 \geqslant 0$,

$$\|z\| = |z_1| + |z_2| + |z_3|,$$

then we have

$$\begin{aligned}
D_+|z_1| &= \big[-\beta i + 2\mu i - (b+\phi)\big]|z_1| + \sigma\beta i|z_2| - \theta|z_3|,\\
D_+|z_2| &= \big[-\beta i - \sigma\beta i + \mu i - \beta s - \sigma\beta v + \mu + \gamma - (b+\theta+\phi)\big]|z_2| + (-\sigma\beta v + \mu v)|z_1| + (-\beta s + \mu s)|z_3|\\
&\leqslant \big[-\beta i - \sigma\beta i + \mu i - \beta s - \sigma\beta v + \mu + \gamma - (b+\theta+\phi)\big]|z_2| - (b+\gamma)s|z_3|,\\
D_+|z_3| &= -\phi|z_1| + \beta i|z_2| + \big[-\sigma\beta i + 2\mu i - (b+\theta)\big]|z_3|.
\end{aligned}$$

Then

$$\begin{aligned}
D_+\|z\| &= D_+|z_1| + D_+|z_2| + D_+|z_3|\\
&\leqslant (-\beta i + 2\mu i - b)|z_1| + (-\sigma\beta i + 2\mu i - b)|z_3| + \big[\mu i - \beta s - \sigma\beta v + \mu + \gamma - (b+\theta+\phi)\big]|z_2|.
\end{aligned}$$

From (12), the following relations are deduced

$$\begin{aligned}
&\mu i - b = r'/r - \gamma i/r,\\
&-\beta s - \sigma\beta v = -i'/i + \mu i - (b+\gamma+\mu).
\end{aligned}$$

Combining with $\sigma\beta \geqslant \mu$ and $\beta > b+\mu+\gamma$, we can obtain

$$\begin{aligned}
D_+\|z\| &\leqslant \big[-(b+\gamma)i + r'/r - \gamma i/r\big]|z_1| + \big(r'/r - \gamma i/r\big)|z_3| + \big[-i'/i - 2\big(r'/r - \gamma i/r\big) - (\theta+\phi)\big]|z_2|\\
&\leqslant \max\big\{-(b+\gamma)i + r'/r - \gamma i/r, r'/r - \gamma i/r - i'/i + 2\big(r'/r - \gamma i/r\big) - (\theta+\phi)\big\}\|z\|.
\end{aligned}$$

Suppose that ψ is a simple closed orbit in Σ. We have

$$\begin{aligned}
&\int_\psi \big[-(b+\gamma)i + r'/r - \gamma i/r\big]\,dl \leqslant -C,\\
&\int_\psi \big(r'/r - \gamma i/r\big)\,dl \leqslant -C,\\
&\int_\psi \big[-i'/i + 2\big(r'/r - \gamma i/r\big) - (\theta+\phi)\big]\,dl \leqslant -2C - A,
\end{aligned}$$

where $C = \int_\psi \gamma i/r\,dl > 0$ and $A = \int_\psi (\theta+\phi)\,dl > 0$. Thus

$$\int_\psi D_+\|z\|\,dl \leqslant -\gamma C < 0.$$

The last relation precludes the existence of any closed curves in Σ as the solution of system (3), including periodic orbits, homoclinic orbits, and heteroclinic cycles.

(b) If $z_1z_2 < 0$ and $z_2z_3 \geqslant 0$,

$$\|z\| = \max\big\{|z_1| + |z_3|, |z_2| + |z_3|\big\},$$

then we have

$$\begin{aligned}
D_+\big(|z_1| + |z_3|\big) &= (-\beta i + 2\mu i - b)|z_1| + (-\sigma\beta i + 2\mu i - b)|z_3| + (\beta i - \sigma\beta i)|z_2|\\
&\leqslant (\mu i - b)\big(|z_1| + |z_3|\big)\\
&\leqslant \big(r'/r - \gamma i/r\big)\big(|z_1| + |z_3|\big),\\
D_+\big(|z_2| + |z_3|\big) &= \big(-\sigma\beta i + \mu i - \beta s + \mu + \gamma - (b+\theta) - \mu v\big)|z_2| + \big[-\sigma\beta i + 2\mu i - (b+\theta) - \beta s + \mu s\big]|z_3|,
\end{aligned}$$

if $\mu + \gamma < b + \theta$, then

$$D_+\big(|z_2| + |z_3|\big) \leqslant \max\big\{-\beta s - \mu v, -\theta + r'/r - \gamma i/r - \beta s + \mu s\big\}\big(|z_2| + |z_3|\big).$$

Therefore,

$$D_+\|z\| \leqslant \max\{r'/r - \gamma i/r, -\beta s - \mu v, -\theta + r'/r - \gamma i/r - \beta s + \mu s\}\|z\|,$$

using the same method as in (a), we have the conclusion.

(c) If $z_1 z_2 < 0$ and $z_2 z_3 < 0$,

$$\|z\| = \max\{v|z_1|/i, s|z_3|/i, |z_2|\},$$

then we have

$$\begin{aligned}
D_+\left(\frac{v}{i}|z_1|\right) &\leqslant \frac{v}{i}\left(\frac{v'}{v} - \frac{i'}{i}\right)|z_1| + \frac{v}{i}\left[-\beta i + 2\mu i - (b+\phi)\right]|z_1| \\
&\leqslant \left(\frac{v'}{v} - \frac{i'}{i} + \frac{r'}{r} - \frac{\gamma i}{r} - \phi\right)\frac{v}{i}|z_1|, \\
D_+\left(\frac{s}{i}|z_3|\right) &\leqslant \frac{s}{i}\left(\frac{s'}{s} - \frac{i'}{i}\right)|z_3| + \frac{s}{i}\left[-\sigma\beta i + 2\mu i - (b+\theta)\right]|z_3| \\
&\leqslant \left(\frac{s'}{s} - \frac{i'}{i} + \frac{r'}{r} - \frac{\gamma i}{r} - \theta\right)\frac{s}{i}|z_3|, \\
D_+\big(|z_2|\big) &\leqslant (-\sigma\beta i - \mu i)\frac{v}{i}|z_1| + (\beta i - \mu i)\frac{s}{i}|z_3| + \left[-\beta i - \sigma\beta i + \mu i - \beta s - \sigma\beta v + \mu + \gamma - (b+\theta+\phi)\right]|z_2| \\
&\leqslant \left[-\frac{i'}{i} - (2b + \theta + \phi)\right]|z_2|.
\end{aligned}$$

Therefore,

$$D_+\|z\| \leqslant \max\left\{\frac{v'}{v} - \frac{i'}{i} + \frac{r'}{r} - \frac{\gamma i}{r} - \phi, \frac{s'}{s} - \frac{i'}{i} + \frac{r'}{r} - \frac{\gamma i}{r} - \theta, -\frac{i'}{i} - (2b + \theta + \phi)\right\}\|z\|,$$

using the same method as in (a), we have the conclusion.

(d) If $z_1 z_2 \geqslant 0$ and $z_2 z_3 < 0$,

$$\|z\| = \max\{|z_1| + |z_3|, |z_2|\},$$

then we have

$$\begin{aligned}
D_+\big(|z_1| + |z_3|\big) &\leqslant (-\beta i + 2\mu i - b)|z_1| + (-\sigma\beta i + 2\mu i - b)|z_3| \\
&\leqslant \big(r'/r - \gamma i/r\big)\big(|z_1| + |z_3|\big), \\
D_+\big(|z_2|\big) &\leqslant \left[-\beta i - \sigma\beta i + \mu i - \sigma\beta v + \mu + \gamma - (b+\theta+\phi) - \mu s\right]|z_2| \\
&\leqslant (-\beta i - \sigma\beta v - \mu s)|z_2|.
\end{aligned}$$

Therefore,

$$D_+\|z\| \leqslant \max\{r'/r - \gamma i/r, -\beta i - \sigma\beta v - \mu s\}\|z\|,$$

using the same method as in (a), we have the conclusion. The proof is complete. □

6. Correlations of dynamics in fraction and population size

We now return to a discussion of the dynamic behavior of $S(t)$, $I(t)$, $R(t)$, $V(t)$ and $N(t)$ in system (1). It is obvious that if $b < d$ and $\mu \geqslant 0$ or $b \leqslant d$ and $\mu > 0$, (2) implies that $N(t)$ converges to zero monotonically as t goes to infinity for all solutions with the disease initially present. If $b = d$ and $\mu = 0$, then $N(t)$ remains constant, and (1) reduces to an *SIVR* model with constant population, whose dynamics are the same as the proportionate system (4). In the rest of this section, we suppose $b > d$ and $\mu > 0$.

From system (1), it is easy to get a trivial equilibrium $E_0 := (0, 0, 0, 0, 0)$. Assume $E_* := (S_*, I_*, R_*, V_*, N_*)$, the endemic equilibrium of system (1) with I_*, where $N_* = S_* + I_* + R_* + V_*$. It exists if and only if the following relations are satisfied

$$\begin{aligned}
&\frac{I_*}{N_*} = \frac{b-d}{\mu} =: m, \qquad \frac{R_*}{N_*} = \frac{\gamma}{d}m, \\
&\frac{V_*}{N_*} = \frac{\phi(d+\mu+\gamma)}{\beta(\sigma\beta m + d + \theta + \sigma\phi)}, \qquad \frac{S_*}{N_*} = \frac{(\sigma\beta m + d + \theta)(d+\mu+\gamma)}{\beta(\sigma\beta m + d + \theta + \sigma\phi)}.
\end{aligned} \tag{14}$$

Eliminating S_*, I_*, R_*, V_*, N_* in (14) leads to the equation below:

$$\frac{b\beta(\sigma\beta m+d+\theta+\sigma\phi)}{d+\gamma+\mu}=(\sigma\beta m+d+\theta)(\beta m+d)+\phi(\sigma\beta m+d), \tag{15}$$

which is equivalent to

$$f(m)=g(m).$$

Here functions f and g are shown in (9) of Section 4.

From Eq. (15) we derive the second critical vaccination rate ϕ_{2c},

$$\phi_{2c}=-\frac{(d+\theta+\beta\sigma m)(k_1+k_2)}{\sigma k_1+k_2}, \tag{16}$$

where

$$k_1=\beta\big[m(d+\gamma)-d\big],\qquad k_2=d(d+\mu+\gamma).$$

The necessary condition for $\mu_{2c}>0$ is $b<d\mu/(d+\gamma)+d$. We deduce from above that $f(m)=g(m)\Leftrightarrow\phi=\phi_{2c}$, $f(m)>g(m)\Leftrightarrow\phi<\phi_{2c}$ and $f(m)<g(m)\Leftrightarrow\phi>\phi_{2c}$.

Theorem 6.1. *System* (1) *always has the trivial equilibrium* $(0,0,0,0,0)$; *it has an endemic equilibrium line* $L(S_*(N_*), I_*(N_*), R_*(N_*), V_*(N_*), N_*)$ *if* (15) *is satisfied, where* N_* *is an arbitrary positive number.*

In Theorem 6.1, we know from (14) that S_*, I_*, R_*, V_* are linear functions of N_*. It makes sense that there may exist an equilibrium line L since the total population varies. If $\mathcal{R}_{\text{vac}}<1$, based on the results in Sections 4 and 5, $(s(t), i(t), r(t), v(t))$ converges to $(s_0, 0, 0, v_0)$, as $t\to\infty$. Equation $\mathrm{d}N(t)/\mathrm{d}t=[(b-d)-\mu i(t)]N(t)$ from (2) implies that $N(t)\to\infty$ exponentially as $t\to\infty$ (see the autonomous asymptotical theory in [11]). From the facts $s(t)=S(t)/N(t)\to s_0$, $v(t)=V(t)/N(t)\to v_0$ $(t\to\infty)$, we have $(S(t),V(t))\to(\infty,\infty)$ exponentially as $t\to\infty$. Moreover, the equations for $I(t)$ and $R(t)$ can be written in the following form

$$\frac{\mathrm{d}}{\mathrm{d}t}\begin{pmatrix}I(t)\\R(t)\end{pmatrix}=\left[\begin{pmatrix}-(d+\mu+\gamma)+\beta s_0+\sigma\beta v_0 & 0\\ \gamma & -d\end{pmatrix}+\begin{pmatrix}\beta(s(t)-s_0)+\sigma\beta(v(t)-v_0) & 0\\ 0 & 0\end{pmatrix}\right]\begin{pmatrix}I(t)\\R(t)\end{pmatrix}. \tag{17}$$

System (17) can be considered as a perturbation of a linear system. Denote

$$\mathcal{R}_c=\frac{\beta s_0+\sigma\beta v_0}{d+\gamma+\mu}=\frac{\beta(b+\theta+\sigma\phi)}{(d+\gamma+\mu)(b+\theta+\phi)}. \tag{18}$$

If $\mathcal{R}_c<1$, the solution $(I(t),R(t))$ to the principle part of (17) converges to $(0,0)$; if $\mathcal{R}_c>1$, the solution $(I(t),R(t))$ to the principle part of (17) diverges to (∞,∞). Likewise, the solution of the perturbed system (17) behaves the same way since the perturbation term decays exponentially as $t\to\infty$ (see [11, Chapter 3, Theorem 2.3]).

It is noted from (18) that $\mathcal{R}_c=1$ is equivalent to the existence of the third critical vaccination rate μ_{3c} such that

$$\phi=\phi_{3c}=\frac{(b+\theta)[\beta-(d+\mu+\gamma)]}{(d+\mu+\gamma)-\beta\sigma} \tag{19}$$

where $\sigma\beta<d+\mu+\gamma<\beta$. $\mathcal{R}_c<1$ and $\mathcal{R}_c>1$ imply $\phi>\phi_{3c}$ and $\phi<\phi_{3c}$, respectively.

Theorem 6.2. *Suppose* $\phi>\phi_{1c}$. *Then* $(N(t),S(t),V(t))\to(\infty,\infty,\infty)$ *exponentially as* $t\to\infty$. *Moreover,* $(I(t),R(t))\to(0,0)$ *if* $\phi>\phi_{3c}$ *and* $(I(t),R(t))\to(\infty,\infty)$ *if* $\phi<\phi_{3c}$.

If $\phi<\phi_{1c}$, the disease becomes "endemic". Suppose that (15) is not satisfied. Specifically, if $f(m)>g(m)$, using the graphs of f and g in Section 4, we can obtain that $m<i_*$, since $0<m,i_*<\min\{1,b/\mu\}$; similarly, if $f(m)<g(m)$, then $m>i_*$. From the global stability of e_* in Theorem 5.2 and the equation

$$\frac{\mathrm{d}N(t)}{\mathrm{d}t}=(b-d)N(t)-\mu I(t)=\mu\big[(m-i_*)-\big(i(t)-i_*\big)\big]N(t),$$

we obtain that $(S(t),I(t),R(t),V(t),N(t))$ goes to $(0,0,0,0,0)$ or $(\infty,\infty,\infty,\infty,\infty)$ if $m<i_*$ or $m>i_*$, respectively. If $f(m)=g(m)$, then $m=i_*$. From the global stability of i_*, we have $N(t)$ converges to some N_* as t goes to infinity. Since $s=S/N$, $i=I/N$, $r=R/N$, $v=V/N$, in this case, we have $S_*=s_*N_*$, $I_*=i_*N_*$, $R_*=r_*N_*$, $V_*=v_*N_*$. We summarize these results in the following theorem.

Theorem 6.3. *Suppose* $\phi<\phi_{1c}$ *and inequality* (13) *being satisfied. As* $t\to\infty$, *then* $(S(t),I(t),R(t),V(t),N(t))$ *converges to the trivial equilibrium* $(0,0,0,0,0)$ *or diverges to* $(\infty,\infty,\infty,\infty,\infty)$ *if* $\phi<\phi_{2c}$ *or* $\phi>\phi_{2c}$, *respectively*; $(S(t),I(t),R(t),V(t),N(t))$ *converges to the endemic equilibrium* (S_*,I_*,R_*,V_*,N_*) *if* $\phi=\phi_{2c}$.

Finally, we summarize the main results in two tables (see Tables 1 and 2).

Table 1
Existence and stability of equilibria for system (1) and system (4).

$\phi ? \phi_{1c}$	Condition	$\phi ? \phi_{2c}$	# of equilibria		Type of equilibria		Stability type	
		(4)	(1)	(4)	(1)	(4)	(1)	(4)
$>$	–	NA**	1	1	DFE	Trivial	GAS*	US**
$<$	(13)	$>$	2	1	DFE/EE	Trivial	US**/GAS*	GAS*
$<$	(13)	$<$	–	1	–	Trivial	–	US**
$<$	(13)	$=$	–	∞	–	Trivial/EE line	–	US/GAS*

* "GAS" is the abbreviation of "globally asymptotically stable".
** "NA" is the abbreviation of "not applicable".
** "US" is the abbreviation of "unstable".

Table 2
Limit values of variables in proportion and in population size for systems (1) and (4).

$\phi ? \phi_{1c}$	$\phi ? \phi_{3c}$	$\phi ? \phi_{2c}$	(s, i, v)	(S, I, R, V, N)
$>$	$>^*$	NA**	$(s_0, 0, v_0)$	$(\infty, 0, 0, \infty, \infty)$
$>$	$<^*$	NA	$(s_0, 0, v_0)$	$(\infty, \infty, \infty, \infty, \infty)$
$<$	NA	$<^\star$	(s_*, i_*, v_*)	$(0, 0, 0, 0, 0)$
$<$	NA	$>^\star$	(s_*, i_*, v_*)	$(\infty, \infty, \infty, \infty, \infty)$
$<$	NA	$=^\star$	(s_*, i_*, v_*)	$(S_*, I_*, R_*, V_*, N_*)$

Note: When $\phi < \phi_{1c}$, all the conclusions are obtained under the conditions (13).
* Sufficient condition for system (1) but not for system (4).
* Necessary condition for system (4) but not for system (1).
** "NA" is the abbreviation of "not applicable".

Table 3
Dimension of parameters and variables used in simulations.

Variable or parameter	Dimension	Implication
s, i, v, r	Dimensionless	Proportional population
S, I, V, R, N	Thousand·Day^{-1}	Population
b	Unity $\cdot$ (100 Years)$^{-1}$	Birth rate
d	Unity $\cdot$ (100 Years)$^{-1}$	Natural death rate
μ	Unity $\cdot$ Year^{-1}	Disease-induced death rate
γ	Unity $\cdot$ Year^{-1}	Recover rate
β	Unity $\cdot$ Day^{-1}	Disease transmission coefficient
ϕ	Unity $\cdot$ Day^{-1}	Vaccination rate
θ	Unity $\cdot$ Year^{-1}	Vaccine waning rate
σ	Dimensionless	Describing the vaccine effectiveness

7. Discussions and simulations

This paper deals with an *SIR* model with vaccination and varying total population. It concerns diseases with long duration and substantial mortality rate (for example, the three notorious and most devastating diseases: HIV/AIDS, malaria and tuberculosis). Our main results present the global dynamics of an *SIRV* population model and its transformed proportionate system, the epidemiological correlations between the two systems in disease eradication and persistence, and the effects of different vaccination strategies on the disease control.

Numerical simulations (parameters and variables used in simulations are summarized in Table 3) carried out for system (4) show that the disease "dies out" when the modified vaccination rate $\phi > \phi_{1c}$ and the disease persists at an "endemic" level when $\phi < \phi_{1c}$ under condition (13) (Figs. 3.1, 3.2). We here question that if the inequality (13) is violated, i.e., $\mu > \min\{\gamma, b+\phi, b+\theta-\gamma\}$ when $\phi < \phi_{1c}$, what are the dynamics of the proportionate system (4) and the population models (1) and (4)? With different initial values, Fig. 3.3 shows numerically that the disease (with different initial values) still can be "endemic" even if the parameters do not satisfy (13), which implies that there are weaker conditions for the global asymptotic stability of the "endemic" equilibrium e^*. Taking the same parameter values as in Fig. 3.3, there is no difference in the dynamics of the population model (1) and (4) with conditions (13) satisfaction or violation (see Section 6).

When the modified vaccination rate $\phi > \phi_{1c}$ the proportionate system (4) and the population model (1) produce quite different dynamics. The global asymptotic stability of disease-"free" equilibrium in (4) cannot guarantee disease eradication in (1) and instead, it may have the communicable disease explode. Furthermore, the disease may counter-intuitively die out when the unique "endemic" equilibrium of (4) is globally stable if $\phi < \phi_{1c}$. All these phenomena are quite different from the epidemiological models with constant population.

Both the modified vaccination rate ϕ_{1c} and the critical vaccination rate ϕ_{3c} are increasing functions of the effective disease infection rate β, the vaccination waning rate θ and the vaccine efficacy-related parameter σ, the less perfect vaccine (greater than θ or the smaller than $(1-\sigma)$) and the higher infection rate, the greater critical vaccination coverage. $\phi_{1c} < \phi_{3c}$

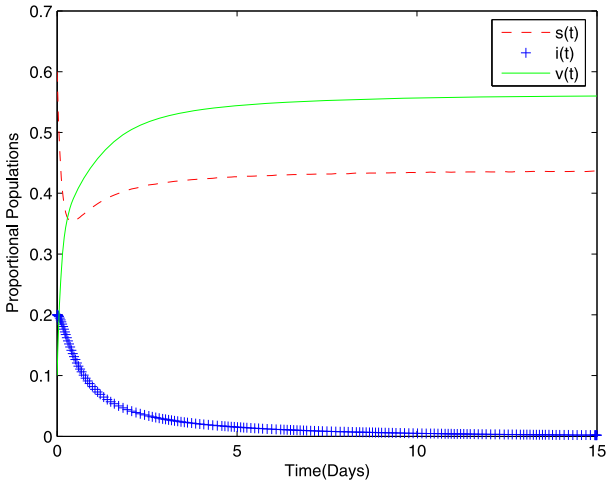

Fig. 3.1. $b = 2.000$; $\beta = 0.008$; $\phi = 0.150$; $\theta = 0.300$; $\gamma = 1.000$; $\mu = 1.500$; $\sigma = 0.500$; $\phi > \phi_{1c} = 0.129$.

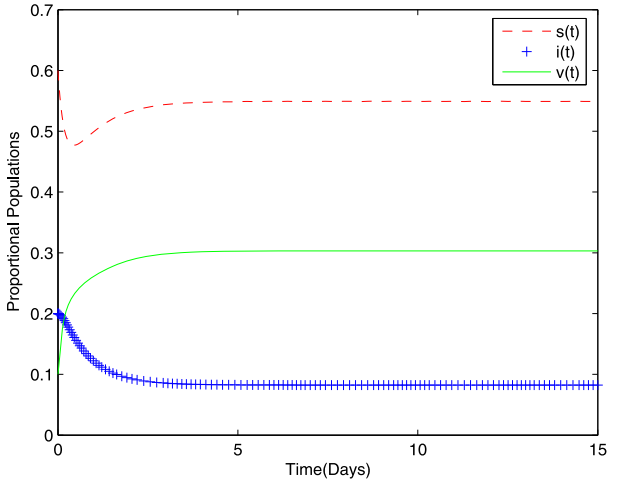

Fig. 3.2. $b = 2.000$; $\beta = 0.004$; $\phi = 0.300$; $\theta = 1.000$; $\gamma = 0.400$; $\mu = 0.500$; $\sigma = 0.100$; $\phi < \phi_{1c} = 0.714$.

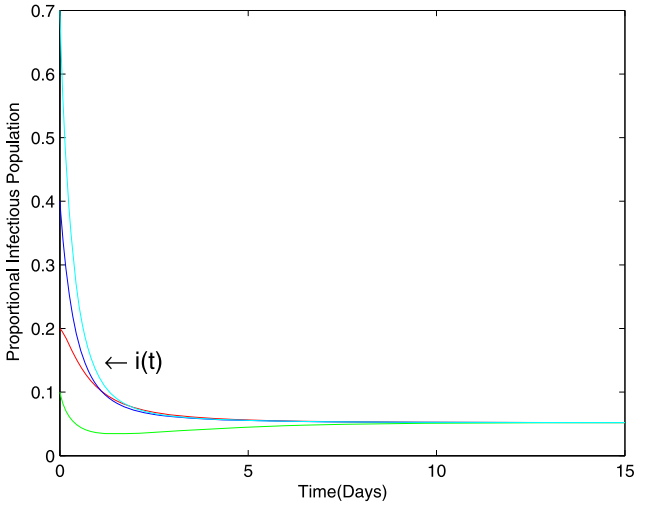

Fig. 3.3. $b = 2.000$; $\beta = 0.008$; $\phi = 0.100$; $\theta = 0.300$; $\gamma = 1.000$; $\sigma = 0.500$; $\mu = 1.500$; $\phi < \phi_{1c} = 0.129$.

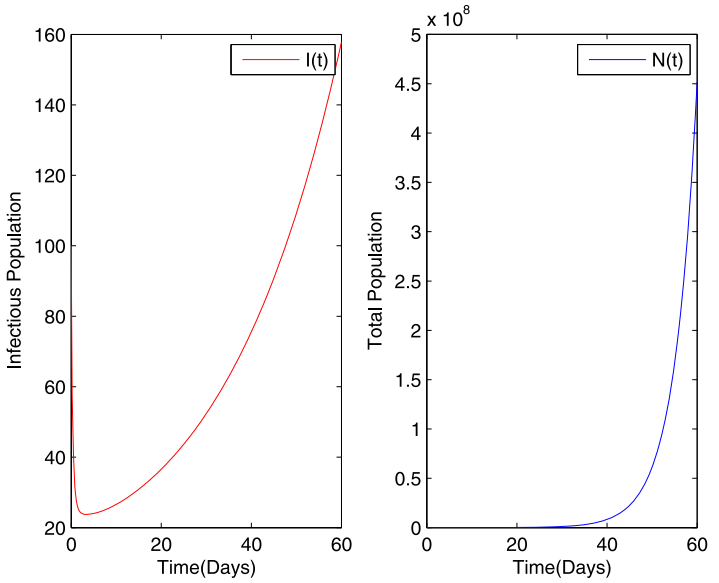

Fig. 3.4. $b = 2.000$; $\beta = 0.008$; $d = 1.800$; $\phi = 0.130$; $\theta = 0.300$; $\gamma = 1.000$; $\mu = 1.500$; $\sigma = 0.500$; $0.129 = \phi_{1c} < \phi < \phi_{3c} = 0.131$.

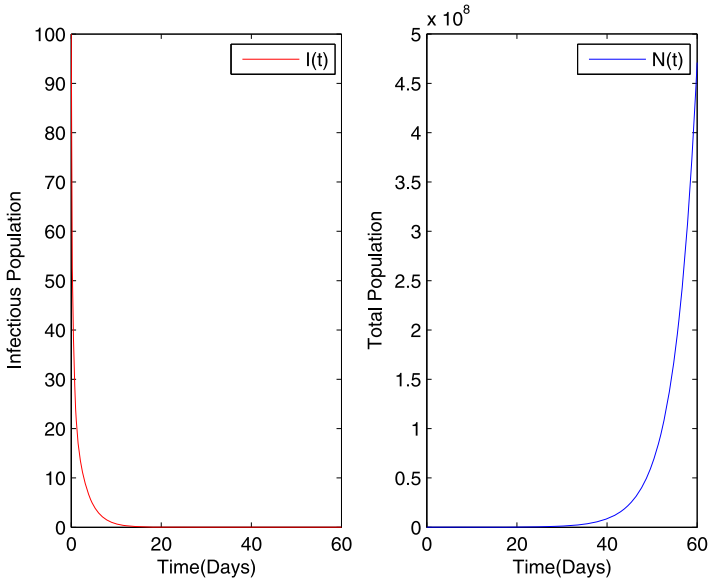

Fig. 3.5. $b = 2.000$; $\beta = 0.008$; $d = 1.800$; $\phi = 0.160$; $\theta = 0.300$; $\gamma = 1.000$; $\mu = 1.500$; $\sigma = 0.500$; $\phi > \max\{\phi_{1c} = 0.129, \phi_{3c} = 0.131\}$.

is always true due to the assumption $b > d$ in Section 6. The communicable disease "dies out" for the reduced proportionate system (4) when the vaccination rate exceeds ϕ_{1c}, and however, for the original population model (1), eradicating the disease needs greater magnitude of vaccination, i.e., $\phi > \phi_{3c}$ (Fig. 3.5). Otherwise, the disease will spread rampantly and is out of control in the end (Fig. 3.4). When $\phi < \phi_{1c}$, the unique "endemic" equilibrium E_* of system (4) is globally asymptotically stable under conditions (13), which implies that the disease "persists" in the end. As to the epidemiology in models (1), unfortunately, the disease is always endemic when $\phi_{2c} \leqslant \phi < \phi_{1c}$ (Figs. 3.7 and 3.8). Although the disease disappears, the whole population would die out due to the natural and disease-caused death when $\phi < \min\{\phi_{1c}, \phi_{2c}\}$, the less vaccination coverage (see Fig. 3.6). The disease eradication or persistence in the reduced proportionate system do not imply the same happening in the original population model.

Since some diseases are seasonal, the public health needs to vaccinate the susceptible individuals regularly. We then can assume that the vaccination rate is periodic over time, which may take the form $\phi \sin(t + \varpi)$ (or $\phi \cos(t + \varpi)$) instead of the constant type. The corresponding dynamics may undergo oscillations. We leave this for the future work.

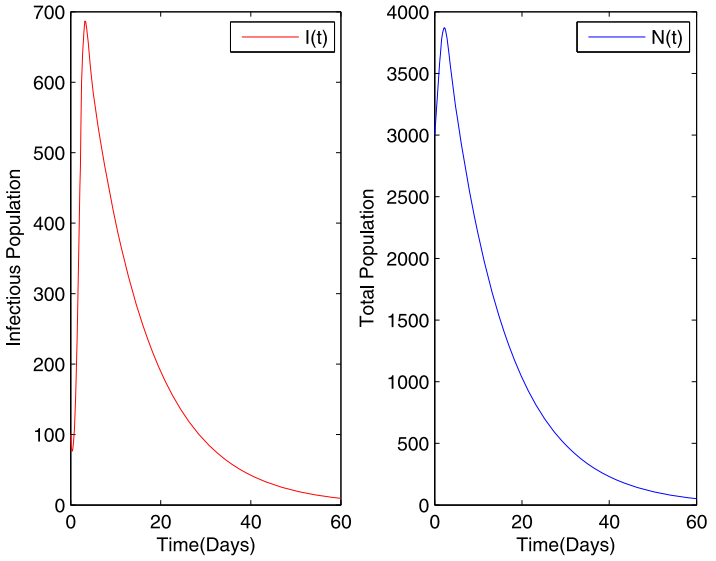

Fig. 3.6. $b = 2.000$; $\beta = 0.004$; $d = 1.800$; $\phi = 0.500$; $\theta = 0.900$; $\gamma = 0.400$; $\mu = 0.500$; $\sigma = 0.100$; $\phi < \max\{\phi_{1c} = 0.714, \phi_{2c} = 0.910\}$.

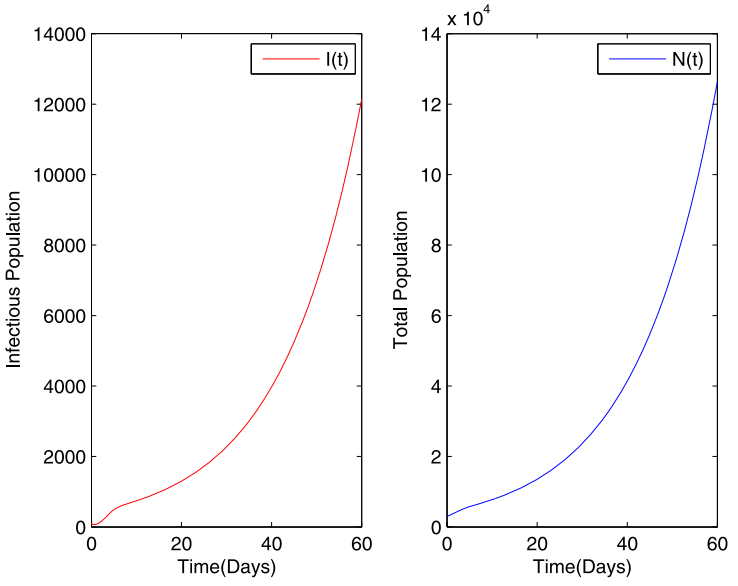

Fig. 3.7. $b = 2.000$; $\beta = 0.004$; $d = 1.800$; $\phi = 1.500$; $\theta = 0.900$; $\gamma = 0.400$; $\mu = 0.500$; $\sigma = 0.500$; $0.910 = \phi_{2c} < \phi < \phi_{1c} = 2.701$.

Acknowledgments

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Appendix A

We give the discussion of the existence of the equilibrium here if $\sigma\beta < \mu$.

(i) Assume $\mathcal{R}_{\text{vac}} > 1$ and $\sigma\beta < \mu$. Then $a_3 < 0$. We have $\varphi(-\infty) > 0$, $\varphi(+\infty) < 0$ and still $\varphi(0) > 0$, $\varphi(1) < 0$ (if $b/\mu \geqslant 1$), $\varphi(b/\mu) < 0$. In this case, we can say that there is at least one root, specifically, only one root or three roots (identical ones included) such that $\varphi(i) = 0$ in the interval $(0, 1)$ if $b/\mu \geqslant 1$ or $(0, b/\mu)$ if $b/\mu < 1$.

As we know, $\varphi(i) = 0$ has three real roots if and only if

$$\frac{q^2}{4} + \frac{p^3}{27} \leqslant 0,$$

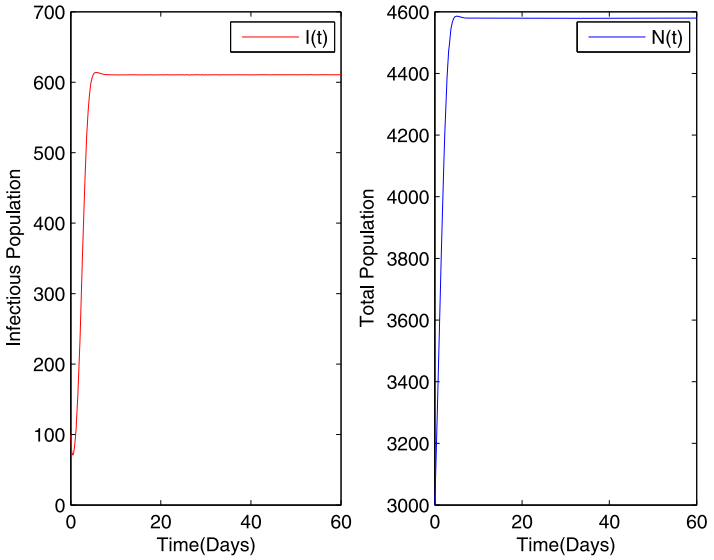

Fig. 3.8. $b = 2.000$; $\beta = 0.004$; $d = 1.800$; $\phi = 0.910$; $\theta = 0.900$; $\gamma = 0.400$; $\mu = 0.500$; $\sigma = 0.500$; $0.910 = \phi_{2c} = \phi < \phi_{1c} = 2.701$.

where

$$p = \frac{a_1}{a_3} - \frac{a_2^2}{3a_3^2}, \qquad q = \frac{a_0}{a_3} - \frac{a_1 a_2}{3a_3^2} + \frac{2a_2^3}{27a_3^3},$$

or, equivalently,

$$\widetilde{\mathcal{R}_1} \triangleq \frac{18a_0a_1a_2a_3 - 4a_0a_2^3 - 4a_1^3a_3 + a_1^2a_2^2}{27a_0^2a_3^2} \geqslant 1.$$

If $\widetilde{\mathcal{R}_1} < 1$, there is a unique i_* such that $\varphi(i^*) = 0$ in the feasible interval.

If $\widetilde{\mathcal{R}_1} > 1$, there are three different real roots for $\varphi(i) = 0$, say i_{1*}, i_{2*}, i_{3*} ($i_{1*} < i_{2*} < i_{3*}$). $\varphi'(i) = 3a_3i^2 + 2a_2i + a_1$. The three different real roots for $\varphi(i) = 0$ are in the feasible interval if and only if the following inequalities are satisfied

$$\begin{aligned} &0 < -\frac{a_2}{3a_3} < 1, \qquad \varphi'(0) = a_1 < 0, \qquad \varphi'(1) = 3a_3 + 2a_2 + a_1 < 0 \quad (\text{if } b/\mu \geqslant 1), \\ &\varphi'(b/\mu) = 3a_3(b/\mu)^2 + 2a_2b/\mu + a_1 < 0 \quad (\text{if } b/\mu < 1). \end{aligned} \tag{A.1}$$

If $\widetilde{\mathcal{R}_1} = 1$, there are three real roots for $\varphi(i) = 0$, in which at least two are identical. Similarly, if inequalities (A.1) are satisfied, then there are three real roots for $\varphi(i) = 0$ in the feasible interval, say i_{1*}, i_{2*}, i_{3*} ($i_{2*} = i_{3*}$).

(ii) Assume $\mathcal{R}_{\text{vac}} < 1$ and $\sigma\beta < \mu$. In this case, we have $a_3 < 0$, $\varphi(-\infty) > 0$, $\varphi(+\infty) < 0$, $\varphi(0) < 0$, $\varphi(1) < 0$ if $b/\mu \geqslant 1$, and $\varphi(b/\mu) < 0$. Then there is at least one negative root.

If $\widetilde{R_1} < 1$, then there is no positive root for $\varphi(i) = 0$.

If $\widetilde{R_1} > 1$, then there are three different real roots for $\varphi(i) = 0$. If $\varphi'(i) = 0$ has a root in the feasible interval $(0, 1)$ (if $b/\mu \geqslant 1$) or $(0, b/\mu)$ (if $b/\mu < 1$) and $\varphi'(i) < 0$ is always satisfied when $i > 1$ (if $b/\mu \geqslant 1$) or $i > b/\mu$ (if $b/\mu < 1$), then there are two different real roots for $\varphi(i) = 0$ in the feasible interval, say i_{1*}, i_{2*} ($i_{1*} < i_{2*}$). Namely, the following inequalities are satisfied

$$\begin{aligned} &0 < -\frac{a_2}{3a_3} < 1, \qquad \varphi'(1) = 3a_3 + 2a_2 + a_1 < 0 \quad (\text{if } b/\mu \geqslant 1), \\ &\varphi'(b/\mu) = 3a_3(b/\mu)^2 + 2a_2(b/\mu) + a_1 < 0 \quad (\text{if } b/\mu < 1). \end{aligned} \tag{A.2}$$

If $\widetilde{\mathcal{R}_1} = 1$, then there is one negative real root and two identical real roots for $\varphi(i) = 0$. Similarly, if inequalities (A.2) are satisfied, then there are two identical positive roots for $\varphi(i) = 0$ in the feasible interval, say i_{1*}, i_{2*} ($i_{1*} = i_{2*}$). Based on the discussions above, we summarize the results in the following theorem.

Theorem A.1. *The disease-"free" equilibria e_0 always exists. Moreover,*

(i) *Assume $\mathcal{R}_{\text{vac}} > 1$, then*
  (1) *if $\sigma\beta \geqslant \mu$, there is a unique "endemic" equilibrium*;
  (2) *if $\sigma\beta < \mu$, and*
    (a) *$\widetilde{\mathcal{R}_1} > 1$ and* (A.1) *are satisfied, there are three "endemic" equilibria, $i_{1*} < i_{2*} < i_{3*}$,*
    (b) *$\widetilde{\mathcal{R}_1} = 1$ and* (A.1) *are satisfied, there are three "endemic" equilibria, $i_{1*}, i_{2*} = i_{3*}$,*
    (c) *in other cases, there is a unique "endemic" equilibrium.*

(ii) *Assume $\mathcal{R}_{\text{vac}} < 1$, then*
  (1) *if $\beta \leqslant \mu$, or $\sigma\beta \geqslant \mu$, there is no "endemic" equilibrium*;
  (2) *if $\sigma\beta < \mu$ and $\beta > \mu$,*
    (a) *$\widetilde{\mathcal{R}_1} > 1$ and* (A.2) *are satisfied, there are two "endemic" equilibria, $i_{1*} < i_{2*}$,*
    (b) *$\widetilde{\mathcal{R}_1} = 1$ and* (A.2) *are satisfied, there are two identical "endemic" equilibria, $i_{1*} = i_{2*}$,*
    (c) *in other cases, there is no "endemic" equilibrium.*

References

[1] R.M. Anderson, R.M. May, Population biology of infectious diseases I, Nature 180 (1979) 361–367.
[2] J. Arino, C.C. McCluskey, P. van den Driessche, Global results for an epidemic model with vaccination that exhibits backward bifurcation, SIAM J. Appl. Math. 64 (2003) 260–276.
[3] E. Beretta, Y. Takeuchi, Global stability of an SIR epidemic model with time delays, J. Math. Biol. 33 (1995) 250–260.
[4] F. Brauer, Models for the spread of universally fatal diseases, J. Math. Biol. 28 (1990) 451–462.
[5] S. Busenberg, P. van den Driessche, Analysis of a disease transmission model in a population with varying size, J. Math. Biol. 28 (1990) 257–270.
[6] B. Buonomo, A. d'Onofrio, D. Lacitignola, Global stability of an SIR epidemic model with information dependent vaccination, Math. Biosci. 216 (2008) 9–16.
[7] V. Capasso, G. Serio, A generalization of the Kermack–McKendrick deterministic epidemic model, Math. Biosci. 42 (1978) 43.
[8] C. Castillo-Chavez, K.L. Cooke, L. Huang, S.A. Levin, On the role of long periods of infectiousness in the dynamics of AIDS. Part 1. Single population models, J. Math. Biol. 27 (1989) 373–398.
[9] K.L. Cooke, Stability analysis for a vector disease model, Rocky Mountain J. Math. 7 (1979) 253–263.
[10] C.A. De Quadros, J.K. Andrus, J.M. Olivé, C.M. Da Silveira, R.M. Eikhof, P. Carrasco, J.W. Fitzsimmons, F.P. Pinheiro, Eradication of poliomyelitis: progress in Americas, Pediatr. Infect. Dis. J. 10 (3) (1991) 222–229.
[11] J.K. Hale, Ordinary Differential Equations, Wiley, New York, 1969.
[12] J.M. Hyman, E.A. Stanley, Using mathematical models to understand the AIDS epidemic, Math. Biosci. 90 (1988) 415–473.
[13] J.A. Jacquez, C.P. Simon, J. Koopman, L. Sattenspiel, T. Perry, Modelling and analyzing HIV transmission: The effect of contact patterns, Math. Biosci. 92 (1988) 119–199.
[14] W.O. Kermack, A.G. McKendrick, A contribution to the mathematical theory of epidemics, Proc. Roy. Soc. A 115 (1927) 700–721.
[15] M.Y. Li, J.R. Graef, L. Wang, J. Karsai, Global dynamics of a SEIR model with varying total population size, Math. Biosci. 160 (1999) 191–213.
[16] M.Y. Li, J.S. Muldowney, A geometric approach to global stability problem, SIAM J. Math. Anal. 27 (1996) 1070–1083.
[17] A.B. Sabin, Measles, killer of millions in developing countries: strategies of elimination and continuing control, EUK J. Epidemiol. 7 (1991) 1–22.
[18] B. Shulgin, L. Stone, Z. Agur, Pulse vaccination strategy in the SIR epidemic model, Bull. Math. Biol. 60 (1998) 1123–1148.
[19] D. Xiao, S. Ruan, Global analysis of an epidemic model with nonmonotone incidence rate, Math. Biosci. 208 (2007) 419–429.