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Research article

Dynamics analysis of an SVEIR epidemic model in a patchy environment

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Abstract: In this paper, we propose a multi-patch SVEIR epidemic model that incorporates vaccination of both newborns and susceptible populations. We determine the basic reproduction number R_0 and prove that the disease-free equilibrium P_0 is locally and globally asymptotically stable if $R_0 < 1$, and it is unstable if $R_0 > 1$. Moreover, we show that the disease is uniformly persistent in the population when $R_0 > 1$. Numerical simulations indicate that vaccination strategies can effectively control disease spread in all patches while population migration can either intensify or prevent disease transmission within a patch.

Keywords: patchy environment; vaccination; migration; asymptotically stable; basic reproduction number

1. Introduction

Infectious diseases are a significant global public health threat that greatly impact people's lives and economies. Since the beginning of the 20-th century, convenient transportation has made traveling more frequent and common, leading to an increased risk of infectious disease transmission among regions. As a result, it is essential to investigate how population mobility affects disease transmission and whether it can increase disease persistence or not. In most classical deterministic epidemic models, the spatial structure is assumed to be homogeneous, but this does not take into account the spatial heterogeneity of population and disease spread [1, 2]. To address this limitation, patch epidemic models have been recently proposed to investigate disease transmission in spatially heterogeneous populations [3–11].

Hethcote in [12] formulated an epidemic model with population migration among two patches. The patches can be towns, cities or relatively isolated regions. Ruan et al. [13] considered a multiregional model to analyze the effect of global travel on the geographical SARS. Wang and

Zhao [14] developed an epidemic model of muti-patches and determined the diseases' threshold for persistence and extinction. Under the assumption that the migration rates of populations are the same, they proved that population migration does not alter the global attractivity of the disease-free equilibrium. Salmani et al. [15] introduced an SEIRS model with population migration among p patches. The authors determined the range of the basic reproduction number and proved that the disease-free equilibrium is globally asymptotically stable if $R_0 < 1$. However, the global stability of the endemic equilibrium is not considered because of the high dimension of the model. Michael and Shuai [16] studied an SIR epidemic model in a patchy environment. Using a graph-theoretic approach to the method of Lyapunov functions, they discussed the uniqueness and global stability of the endemic equilibrium. Gao [17] presented a multi-patch epidemic model in order to consider travel frequency variations among residents, and he illustrated the influence of heterogeneity in human movement on the geographic spread of diseases. Wang et al. [18] proposed an n-patch SEIQR epidemic model to investigate the effect of entry-exit screening on the spread and control of diseases.

As is known to us, vaccination strategies prove to be the most effective control measure against many infectious diseases. Over the past few decades, many researchers have formulated various epidemic models with vaccination and analyzed their dynamical behavior, see [19–24]. However, most of these studies have only examined the impact of vaccination on control of disease spread in an isolated patch, ignoring spatial variation. Taking this into account, Cui et al. [25] established the following SIR epidemic model with vaccination in a patchy environment

$$\begin{cases} \frac{\mathrm{d}S_i}{\mathrm{d}t} = (1-p_i)\mu_i N_i - \beta_i \frac{I_i}{N_i} S_i - \mu_i S_i + \sum\limits_{j\neq i}^{n} \left(m_{ij} S_j - m_{ji} S_i \right), \\ \frac{dI_i}{dt} = \beta_i \frac{I_i}{N_i} S_i - (\mu_i + \gamma_i) I_i, \\ \frac{dR_i}{dt} = p_i \mu_i N_i + \gamma_i I_i - \mu_i R_i + \sum\limits_{j\neq i}^{n} \left(m_{ij} R_j - m_{ji} R_i \right), \quad i = 1, 2, \ldots, n. \end{cases} \tag{1.1}$$

where N_i is the total population in the i-th patch; S_i, I_i, and R_i denote the sizes of the susceptible, infected, and recovered individuals in patch i, respectively. They investigated the effect of the increasing mobility of populations on the vaccination strategy. Manuel et al. [26] considered an n-patch SIR epidemic model with constant and proportional vaccination controls. The authors illustrated the effect of vaccination control on disease eradication in a patchy environment.

In reality, many infectious diseases have an incubation period, such as influenza, tuberculosis, malaria and hepatitis B [27]. During the incubation period, the individuals may travel from one patch to another, which has a significant impact on disease spread. There are several that include both latency and mobility of individuals, see [28–31]. Zhang et al. [28] and Lou and Zhao [29] proposed a reaction-diffusion model with incubation periods. San and Wang [30] presented a two-group SIR epidemic model with incubation periods over a patchy environment. Li and Zou [31] synthesized latency, demographic structure and spatial heterogeneity into the SIR model and investigated the dynamics of the derived model. Motivated by the above works, we extend the model in paper [25] by including the vaccinated class and the exposed class, and formulate a multi-patch SVEIR model that incorporates vaccination for newborns and susceptible individuals. The model formulated in this paper can contribute to studying the impacts of migration and vaccination on disease transmission.

The paper is organized as follows. In Section 2, we introduce an n-patch epidemic model with vaccination. In Section 3, we derive the disease-free equilibrium and the basic reproduction number.

In Section 4, we discuss the local and global stability of the disease-free equilibrium and establish uniform persistence of the disease. In Section 5, we perform some numerical simulations to confirm the theoretical results and investigate the impacts of migration and vaccination. Finally, the paper ends with a summary of our conclusions.

2. Mathematical model

In this section, an n-patch SVEIR epidemic model that incorporates vaccination for newborns and susceptible individuals is formulated. Assume that the total population in patch i is divided into five compartments: susceptible, vaccinated, exposed (infected but non-infectious), infectious, and recovered. The number of individuals in each compartment at time t is denoted by $S_i(t), V_i(t)$, $E_i(t), I_i(t)$ and $R_i(t)$, respectively. The flow diagram of the disease transmission in each patch is depicted in Figure 1.

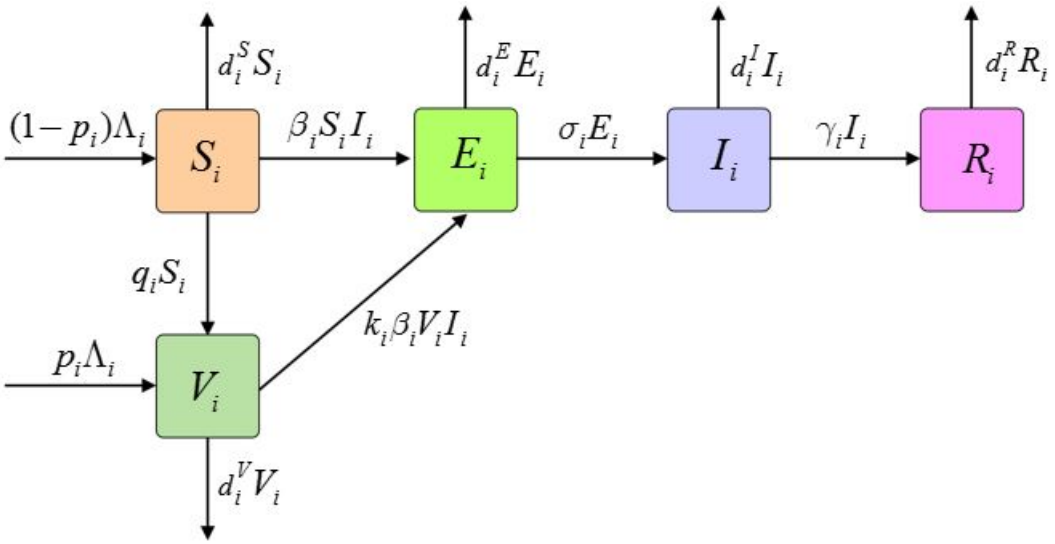

Figure 1. Diagram of the disease transmission in the i-th patch.

All individuals are assumed to be born susceptible, and a fraction p_i of newborns in patch i are vaccinated, where $p_i \in (0, 1]$. Susceptible individuals in patch i receive vaccination at rate q_i. The parameter Λ_i denotes the influx of newborns into patch i, and β_i denotes the transmission coefficient in patch i. Since the vaccination fails to confer complete immunity to all vaccinated recipients, vaccinated individuals may become infected due to contacting with infected individuals. Thus, we use a scaling factor k_i to reflect the vaccine efficacy, where $k_i \in [0, 1]$. For $k_i = 0$, the vaccine can provide complete protection against infection, that is, susceptible individuals will not be infected after vaccination. For $k_i = 1$, the vaccine will have no effect. The parameter σ_i is the rate that exposed individuals become infectious in patch i and γ_i is the recovery rate of the infectious individuals in patch i. The parameters $d_i^S, d_i^V, d_i^E, d_i^I$ and d_i^R are mortality rates of the susceptible, vaccinated, exposed, infected and recovered individuals, respectively, in the i-th patch. The migration rates of susceptible, vaccinated, exposed, infected and recovered populations from patch j to patch i are given by $a_{ij}, b_{ij}, c_{ij}, d_{ij}$ and e_{ij}, respectively. All the parameters are assumed to be non-negative.

Based on the above assumptions and Figure 1, an SVEIR epidemic model is presented as follows,

$$\begin{cases} \frac{\mathrm{d}S_i}{\mathrm{d}t} = (1-p_i)\Lambda_i - \beta_i S_i I_i - \left(d_i^S + q_i\right) S_i + \sum\limits_{j=1}^{n}\left(a_{ij}S_j - a_{ji}S_i\right), \\ \frac{dV_i}{dt} = p_i\Lambda_i - k_i\beta_i V_i I_i - d_i^V V_i + q_i S_i + \sum\limits_{j=1}^{n}\left(b_{ij}V_j - b_{ji}V_i\right), \\ \frac{dE_i}{dt} = \beta_i S_i I_i + k_i\beta_i V_i I_i - \left(d_i^E + \sigma_i\right) E_i + \sum\limits_{j=1}^{n}\left(c_{ij}E_j - c_{ji}E_i\right), \\ \frac{dI_i}{dt} = \sigma_i E_i - \left(d_i^I + \gamma_i\right) I_i + \sum\limits_{j=1}^{n}\left(d_{ij}I_j - d_{ji}I_i\right), \\ \frac{dR_i}{dt} = \gamma_i I_i - d_i^R R_i + \sum\limits_{j=1}^{n}\left(e_{ij}R_j - e_{ji}R_i\right), \quad i = 1,2,\ldots,n. \end{cases} \tag{2.1}$$

Notice that the equations for R_i are independent of the first four equations of system (2.1) and hence, the dynamics is governed by the following system,

$$\begin{cases} \frac{\mathrm{d}S_i}{\mathrm{d}t} = (1-p_i)\Lambda_i - \beta_i S_i I_i - \left(d_i^S + q_i\right) S_i + \sum\limits_{j=1}^{n}\left(a_{ij}S_j - a_{ji}S_i\right), \\ \frac{dV_i}{dt} = p_i\Lambda_i - k_i\beta_i V_i I_i - d_i^V V_i + q_i S_i + \sum\limits_{j=1}^{n}\left(b_{ij}V_j - b_{ji}V_i\right), \\ \frac{dE_i}{dt} = \beta_i S_i I_i + k_i\beta_i V_i I_i - \left(d_i^E + \sigma_i\right) E_i + \sum\limits_{j=1}^{n}\left(c_{ij}E_j - c_{ji}E_i\right), \\ \frac{dI_i}{dt} = \sigma_i E_i - \left(d_i^I + \gamma_i\right) I_i + \sum\limits_{j=1}^{n}\left(d_{ij}I_j - d_{ji}I_i\right), \quad i = 1,2,\ldots,n. \end{cases} \tag{2.2}$$

3. Equilibria and basic reproduction number

In this section, we show that the system (2.2) has a unique disease-free equilibrium, and then, using the next generation matrix method, we derive the basic reproduction number R_0.

In order to find the disease-free equilibrium with all $I_i = 0$ of system (2.2), we consider the following linear system:

$$\begin{cases} (1-p_i)\Lambda_i - \left(d_i^S + q_i\right) S_i + \sum\limits_{j=1}^{n}\left(a_{ij}S_j - a_{ji}S_i\right) = 0, \\ p_i\Lambda_i - d_i^V V_i + q_i S_i + \sum\limits_{j=1}^{n}\left(b_{ij}V_j - b_{ji}V_i\right) = 0, \\ -\left(d_i^E + \sigma_i\right) E_i + \sum\limits_{j=1}^{n}\left(c_{ij}E_j - c_{ji}E_i\right) = 0, \\ \sigma_i E_i = 0, \quad i = 1,2,\ldots,n. \end{cases} \tag{3.1}$$

Then $E_i = 0$ $(i = 1,2,\ldots,n)$ and

$$\begin{cases} (1-p_i)\Lambda_i - \left(d_i^S + q_i\right) S_i + \sum\limits_{j=1}^{n}\left(a_{ij}S_j - a_{ji}S_i\right) = 0, \\ p_i\Lambda_i - d_i^V V_i + q_i S_i + \sum\limits_{j=1}^{n}\left(b_{ij}V_j - b_{ji}V_i\right) = 0, \quad i = 1,2,\cdots,n. \end{cases} \tag{3.2}$$

Converted into the form of matrix system, we can get

$$\begin{cases} H_1 S = B, \\ H_2 V = C, \end{cases}$$

where

$$S = (S_1, S_2, \ldots, S_n)^T, B = ((1-p_1)\Lambda_1, (1-p_2)\Lambda_2, \ldots, (1-p_n)\Lambda_n)^T,$$
$$V = (V_1, V_2, \ldots, V_n)^T, C = (p_1\Lambda_1 + q_1 S_1, p_2\Lambda_2 + q_2 S_2, \ldots, p_n\Lambda_n + q_n S_n)^T,$$

$$H_1 = \begin{bmatrix} d_1^S + q_1 + \sum\limits_{j\neq 1}^{n} a_{j1} & -a_{12} & \cdots & -a_{1n} \\ -a_{21} & d_2^S + q_2 + \sum\limits_{j\neq 2}^{n} a_{j2} & \cdots & -a_{2n} \\ \vdots & \vdots & & \vdots \\ -a_{n1} & -a_{n2} & \cdots & d_n^S + q_n + \sum\limits_{j\neq n}^{n} a_{jn} \end{bmatrix},$$

$$H_2 = \begin{bmatrix} d_1^V + \sum\limits_{j\neq 1}^{n} b_{j1} & -b_{12} & \cdots & -b_{1n} \\ -b_{21} & d_2^V + \sum\limits_{j\neq 2}^{n} b_{j2} & \cdots & -b_{2n} \\ \vdots & \vdots & & \vdots \\ -b_{n1} & -b_{n2} & \cdots & d_n^V + \sum\limits_{j\neq n}^{n} b_{jn} \end{bmatrix}.$$

It is clear that all off-diagonal entries of H_1 are negative, and the sum of the entries in each column is positive. Hence, it follows from [32] that H_1 is a nonsingular M-matrix and $H_1^{-1} > 0$. Similarly, H_2 is also a nonsingular matrix and $H_2^{-1} > 0$. Therefore, linear system (3.2) has a unique positive solution $S^0 = (S_1^0, S_2^0, \ldots, S_n^0)^T = H_1^{-1}B$, $V^0 = (V_1^0, V_2^0, \ldots, V_n^0)^T = H_2^{-1}C$, where S_i^0, V_i^0 satisfies

$$\begin{aligned} 0 = \frac{\mathrm{d}S_i^0}{\mathrm{d}t} &= (1-p_i)\Lambda_i - \left(d_i^S + q_i\right) S_i^0 + \sum_{j=1}^{n} \left(a_{ij} S_j^0 - a_{ji} S_i^0\right), \\ 0 = \frac{\mathrm{d}V_i^0}{\mathrm{d}t} &= p_i\Lambda_i - d_i^V V_i^0 + q_i S_i^0 + \sum_{j=1}^{n} \left(b_{ij} V_j^0 - b_{ji} V_i^0\right). \end{aligned} \tag{3.3}$$

Thus, system (2.2) always has a unique disease-free equilibrium $P_0 = (S_1^0, V_1^0, 0, 0, \ldots, S_n^0, V_n^0, 0, 0)$.

Adding all the equations of (2.2), we have

$$\begin{aligned} \frac{\mathrm{d}N(t)}{\mathrm{d}t} &= \bar{\Lambda} - \sum_{j=1}^{n} \left[d_i^S S_i + d_i^V V_i + d_i^E E_i + (d_i^I + \gamma_i) I_i\right] \\ &\quad + \sum_{i=1}^{n} \left[\sum_{j=1}^{n} \left(a_{ij} S_j - a_{ji} S_i\right) + \sum_{j=1}^{n} \left(b_{ij} V_j - b_{ji} V_i\right) + \sum_{j=1}^{n} \left(c_{ij} E_j - c_{ji} E_i\right) + \sum_{j=1}^{n} \left(d_{ij} I_j - d_{ji} I_i\right)\right] \\ &= \bar{\Lambda} - \sum_{j=1}^{n} \left[d_i^S S_i + d_i^V V_i + d_i^E E_i + (d_i^I + \gamma_i) I_i\right] \\ &\leq \bar{\Lambda} - d^* N, \end{aligned} \tag{3.4}$$

where $\bar{\Lambda} = \sum\limits_{i=1}^{n} \Lambda_i$, $d^* = \min\left\{d_i^S, d_i^V, d_i^E, d_i^I + \gamma_i\right\}$ and $N(t) = \sum\limits_{i=1}^{n} (S_i + V_i + E_i + I_i)$. By the comparison principle, it is easy to see that $\limsup_{t\to\infty} N(t) \leq \frac{\bar{\Lambda}}{d^*}$.

From the first equation of system (2.2), we obtain

$$\frac{\mathrm{d}S_i}{\mathrm{d}t} \le (1-p_i)\Lambda_i - \left(d_i^S + q_i\right)S_i + \sum_{j=1}^{n}\left(a_{ij}S_j - a_{ji}S_i\right) = (H_1S^0 - H_1S)_i, \tag{3.5}$$

where $(H_1S^0 - H_1S)_i$ is the i-th element of $H_1S^0 - H_1S$. Thus, $\frac{\mathrm{d}S_i}{\mathrm{d}t} \le 0$ if $S_i = S_i^0, S_j \le S_j^0, i, j = 1, 2, \ldots, n$, and $j \ne i$. Similarly, $\frac{\mathrm{d}V_i}{\mathrm{d}t} \le 0$ if $V_i = V_i^0, V_j \le V_j^0$.

From the aforementioned analysis, the compact feasible region

$$\Gamma = \left\{(S_1, V_1, E_1, I_1, \ldots, S_n, V_n, E_n, I_n) \in R_+^{4n} : N(t) \le \frac{\bar{\Lambda}}{d^*}, 0 \le S_i \le S_i^0, 0 \le V_i \le V_i^0, i = 1, 2, \ldots, n\right\}$$

is positively invariant with respect to system (2.2).

The basic reproduction number R_0 is known as the threshold of disease outbreak, which has important implications for disease control. In the following, the next-generation matrix method in van den Driessche and Watmough [33] is used to calculate the reproduction number of system (2.2).

Define

$$F = \begin{bmatrix} 0 & F_1 \\ 0 & 0 \end{bmatrix}, V = \begin{bmatrix} V_{11} & 0 \\ V_{21} & V_{22} \end{bmatrix}, where$$

$$F_1 = diag(\beta_1 S_1^0 + k_1\beta_1 V_1^0, \beta_2 S_2^0 + k_2\beta_2 V_2^0, \ldots, \beta_n S_n^0 + k_n\beta_n V_n^0),$$

$$V_{11} = \begin{bmatrix} d_1^E + \sigma_1 + \sum\limits_{j\ne 1}^{n} c_{j1} & -c_{12} & \cdots & -c_{1n} \\ -c_{21} & d_2^E + \sigma_2 + \sum\limits_{j\ne 2}^{n} c_{j2} & \cdots & -c_{2n} \\ \vdots & \vdots & & \vdots \\ -c_{n1} & -c_{n2} & \cdots & d_n^E + \sigma_n + \sum\limits_{j\ne n}^{n} c_{jn} \end{bmatrix}.$$

$$V_{21} = diag(-\sigma_1, -\sigma_2, \ldots, -\sigma_n),$$

$$V_{22} = \begin{bmatrix} d_1^I + \gamma_1 + \sum\limits_{j\ne 1}^{n} d_{j1} & -d_{12} & \cdots & -d_{1n} \\ -d_{21} & d_2^I + \gamma_2 + \sum\limits_{j\ne 2}^{n} d_{j2} & \cdots & -d_{2n} \\ \vdots & \vdots & & \vdots \\ -d_{n1} & -d_{n2} & \cdots & d_n^I + \gamma_n + \sum\limits_{j\ne n}^{n} d_{jn} \end{bmatrix}.$$

It is clear that both V_{11} and V_{22} are nonsingular M-matrixes [32], which means that their inverses are nonnegative. Therefore, the matrix V is a nonsingular matrix. The next generation matrix is given by

$$FV^{-1} = \begin{bmatrix} -F_1V_{22}^{-1}V_{21}V_{11}^{-1} & F_1V_{22}^{-1} \\ 0 & 0 \end{bmatrix},$$

Thus, the basic reproduction number R_0 is defined as the spectral radius of the matrix FV^{-1}, that is,

$$R_0 = \rho(FV^{-1}) = \rho(-F_1V_{22}^{-1}V_{21}V_{11}^{-1}).$$

4. Stability analysis

In this section, the local and global stability of the disease-free equilibrium P_0 are discussed, and the uniform persistence of system (2.2) is established. In order to discuss the stability of the disease-free equilibrium, we introduce the following lemma.

Lemma 4.1. *If F is a non-negative matrix and V is a nonsingular matrix, then*

$$s(F-V)<0 \Longleftrightarrow \rho(FV^{-1})<1, \quad s(F-V)>0 \Longleftrightarrow \rho(FV^{-1})>1, \tag{4.1}$$

where $s(A) := \max\{Re(\lambda) : \lambda \text{ is an eigenvalue of matrix } A\}$.

Theorem 4.1. *The disease-free equilibrium P_0 is locally asymptotically stable if $R_0 < 1$ and it is unstable if $R_0 > 1$.*

Proof. From the third and the fourth equations of system (2.2), we obtain

$$\dot{X} = (F-V)X, \tag{4.2}$$

where $X = (E_1, E_2, \ldots, E_n, I_1, I_2, \ldots, I_n)^T$.

It follows from Lemma 4.1 that $s(F-V) < 0 (> 0)$ if and only if $R_0 < 1 (> 1)$. Thus, if $R_0 < 1$, all the eigenvalues of the matrix $F-V$ lie in the left half plane, and therefore, the disease-free equilibrium P_0 is locally asymptotically stable. Similarly, if $R_0 > 1$, at least one eigenvalue of matrix $F-V$ lies in the right half plane, and the disease-free equilibrium P_0 is unstable. □

Theorem 4.2. *Suppose that $R_0 < 1$, then the disease-free equilibrium P_0 is globally asymptotically stable.*

Proof. Since $V^{-1}F = V^{-1}FV^{-1}V$, according to the properties of similar matrixs, we have $\rho(V^{-1}F) = \rho(FV^{-1}) = R_0$. Then from the Perron-Frobenius theorem [32], we can obtain that the non-negative irreducible matrix $V^{-1}F$ has a positive left eigenvector $(w_1, w_2, \ldots, w_{2n})$, which corresponds to the spectral radius $\rho(V^{-1}F)$, that is,

$$(w_1, w_2, \ldots, w_{2n})V^{-1}F = (w_1, w_2, \ldots, w_{2n})\rho(V^{-1}F).$$

Let us consider a Lyapunov function

$$L = \sum_{i=1}^{n} u_i E_i + \sum_{i=1}^{n} v_i I_i, \tag{4.3}$$

where $(u_1, u_2, \ldots, u_n, v_1, v_2, \ldots, v_n) = (w_1, w_2, \ldots, w_{2n})V^{-1}$.
Calculating the differentiation of L along the solutions of system (2.2), we obtain

$$\begin{aligned}
\frac{\mathrm{d}L}{\mathrm{d}t} &= \sum_{i=1}^{n} u_i \frac{\mathrm{d}E_i}{\mathrm{d}t} + \sum_{i=1}^{n} v_i \frac{\mathrm{d}I_i}{\mathrm{d}t} \\
&= \sum_{i=1}^{n} u_i \left[\beta_i S_i I_i + k_i \beta_i V_i I_i - \left(d_i^E + \sigma_i\right) E_i + \sum_{j=1}^{n} \left(c_{ij}E_j - c_{ji}E_i\right) \right] \\
&\quad + \sum_{i=1}^{n} v_i \left[\sigma_i E_i - \left(d_i^I + \gamma_i\right) I_i + \sum_{j=1}^{n} \left(d_{ij}I_j - d_{ji}I_i\right) \right].
\end{aligned}$$

Since $S_i \le S_i^0$ and $V_i \le V_i^0$, when $R_0 < 1$, we have

$$\begin{aligned}\frac{\mathrm{d}L}{\mathrm{d}t} \le & \sum_{i=1}^{n} u_i \left[\beta_i S_i^0 I_i + k_i \beta_i V_i^0 I_i - \left(d_i^E + \sigma_i\right) E_i + \sum_{j=1}^{n} \left(c_{ij} E_j - c_{ji} E_i\right)\right] \\ & + \sum_{i=1}^{n} v_i \left[\sigma_i E_i - \left(d_i^I + \gamma_i\right) I_i + \sum_{j=1}^{n} \left(d_{ij} I_j - d_{ji} I_i\right)\right] \\ = & (u_1, u_2, \ldots, u_n)(F_1 I - V_{11} E) - (v_1, v_2, \ldots, v_n)(V_{21} E + V_{22} I) \\ = & (u_1, u_2, \ldots, u_n, v_1, v_2, \ldots, v_n)(F - V) X \\ = & (w_1, w_2, \ldots, w_{2n}) V^{-1} (F - V) X \\ = & (w_1, w_2, \ldots, w_{2n})(V^{-1} F - E) X \\ = & (w_1, w_2, \ldots, w_{2n})(R_0 - 1) X \le 0,\end{aligned} \tag{4.4}$$

where $E = (E_1, E_2, \ldots, E_n)^T, I = (I_1, I_2, \ldots, I_n)^T$.

Hence, $\frac{\mathrm{d}L}{\mathrm{d}t} = 0$ if and only if $S_i = S_i^0, V_i = V_i^0$ and $E_i = 0, I_i = 0$. When $S_i = S_i^0$, from the first equation of system (2.2), we have

$$0 = \frac{\mathrm{d}S_i^0}{\mathrm{d}t} = (1 - p_i)\Lambda_i - \beta_i S_i^0 I_i - \left(d_i^S + q_i\right) S_i^0 + \sum_{j=1}^{n} \left(a_{ij} S_j^0 - a_{ji} S_i^0\right).$$

It can be obtained that $I_i = 0$ for $i = 1, 2, \ldots, n$. This implies that the largest compact invariant set in $\{(S_1, V_1, E_1, I_1, \ldots, S_n, V_n, E_n, I_n) \in \Gamma : S_i = S_i^0, V_i = V_i^0, E_i = I_i = 0, i = 1, 2, \ldots n\}$ is the singleton $\{P_0\}$. From LaSalle's Invariance Principle [34], we obtain that P_0 is globally attractive. Therefore, P_0 is globally asymptotically stable in Γ once $R_0 < 1$.

□

Theorem 4.3. *Suppose that $c_{ij} > 0$, $d_{ij} > 0$ $(i, j = 1, 2, \ldots, n, j \ne i)$. If $R_0 > 1$, then system (2.2) is uniformly persistent, i.e., there exists a constant $c > 0$ such that every solution $\varphi_t(x_0) \equiv (S_1, V_1, E_1, I_1, \ldots, S_n, V_n, E_n, I_n)$ of system (2.2) satisfies*

$$\liminf_{t\to\infty} E_i \ge c, \ \liminf_{t\to\infty} I_i \ge c, \ i = 1, 2, \ldots, n,$$

where $x_0 = (S_1(0), V_1(0), E_1(0), I_1(0), \ldots, S_n(0), V_n(0), E_n(0), I_n(0)) \in R_+^n \times R_+^n \times (R_+^n \setminus \{0\}) \times (R_+^n \setminus \{0\})$, and system (2.2) admits at least one endemic equilibrium.

Proof. According to the uniform persistence theorem formulated in Wang and Zhao [14, 35], we prove our result as follows. For convenience, we denote the solution $(S_1, V_1, E_1, I_1, \ldots, S_n, V_n, E_n, I_n)$ of system (2.2) by (S, V, E, I). Define

$$\begin{aligned} X &= \{(S, V, E, I) : S_i \ge 0, V_i \ge 0, E_i \ge 0, I_i \ge 0, i = 1, 2, \ldots, n\}, \\ X_0 &= \{(S, V, E, I) \in X : E_i > 0, I_i > 0, i = 1, 2, \ldots, n\}, \\ \partial X_0 &= X \setminus X_0. \end{aligned}$$

It suffices to prove that system (2.2) is uniformly persistent with respect to $(X_0, \partial X_0)$. By the form of (2.2), it is clear that both X and X_0 are positively invariant. ∂X_0 is relatively closed in X. Moreover, system (2.2) is point dissipative in Section 3.

The set $M_\partial = \{x_0 \in \partial X_0 : \varphi_t(x_0) \in \partial X_0, \forall t > 0\}$ and $M = \{(S, V, 0, 0) : S \geq 0, V \geq 0\}$. We next claim that $M_\partial = M$. Clearly, $M \subset M_\partial$. it suffices to show that $M_\partial \setminus M = \emptyset$. Assume that $x_0 \in M_\partial \setminus M$, then there is an i_0, $1 \leq i_0 \leq n$ and a $t_0 \geq 0$ such that $E_{i_0}(t_0) > 0$, $I_{i_0}(t_0) > 0$. The set $\{1, 2, \ldots, n\}$ can be decomposed into two sets L_1 and L_2, where

$$\begin{aligned} &E_i(t_0) = I_i(t_0) = 0, \quad \forall i \in L_1, \\ &E_i(t_0) > 0, I_i(t_0) > 0, \quad \forall i \in L_2. \end{aligned}$$

Clearly, L_1 and L_2 are both nonempty. For any $j \in L_1$, $i_0 \in L_2$, we have

$$\begin{aligned} \frac{dE_j(t_0)}{dt} &\geq \sum_{m=1}^{n} c_{jm} E_m(t_0) \geq c_{ji_0} E_{i_0}(t_0) > 0, \\ \frac{dI_j(t_0)}{dt} &\geq \sum_{m=1}^{n} d_{jm} I_m(t_0) \geq d_{ji_0} I_{i_0}(t_0) > 0. \end{aligned} \tag{4.5}$$

Hence there exist an ϵ_0 such that $E_j(t) > 0, I_j(t) > 0$, $j \in L_1$ for $t_0 < t < t_0 + \epsilon_0$. Obviously, we can restrict ϵ_0 small enough such that $E_i(t) > 0, I_i(t) > 0$, $i \in L_2$ for $t_0 < t < t_0 + \epsilon_0$. Therefore, we know that $\varphi_t(x_0) \notin \partial X_0$ for $t_0 < t < t_0 + \epsilon_0$, which contradicts the assumption that $x_0 \in M_\partial$.

It is easy to verify that P_0 is the unique equilibrium in M_∂. Next, we will show that $W^S(P_0) \cap X_0 = \emptyset$, where $W^S(P_0)$ is the stable manifold of P_0. Choose δ small enough such that $\limsup_{t\to\infty} |\varphi_t(x_0) - P_0| > \delta$ for $x_0 \in X_0$. Suppose that this does not hold. Then we have $|\varphi_t(x_0) - P_0| \leq \delta$ for all $t \geq 0$. This implies that, for any sufficiently small positive constant ϵ, there exists a $T > 0$ such that

$$S_i^0 - \epsilon \leq S_i(t), \quad V_i^0 - \epsilon \leq V_i(t), \quad \text{for } \forall\, t > T.$$

From system (2.2), we obtain

$$\begin{aligned} \tfrac{\mathrm{d}E_i}{\mathrm{d}t} &\geq \beta_i I_i (S_i^0 - \varepsilon) + k_i \beta_i I_i (V_i^0 - \varepsilon) - \left(d_i^E + \sigma_i\right) + \sum_{j=1}^{n} \left(c_{ij} E_j - c_{ji} E_i\right), \\ \tfrac{\mathrm{d}I_i}{\mathrm{d}t} &= \sigma_i E_i - \left(d_i^I + \gamma_i\right) + \sum_{j=1}^{n} \left(d_{ij} I_j - d_{ji} I_i\right), \quad \text{for } \forall\, t > T. \end{aligned} \tag{4.6}$$

Let

$$G = \begin{bmatrix} -V_{11} & F_1 - \varepsilon G_1 \\ -V_{21} & -V_{22} \end{bmatrix} = F - V - \varepsilon \begin{bmatrix} 0 & G_1 \\ 0 & 0 \end{bmatrix},$$

where $G_1 = diag\{\beta_1(k_1 + 1), \ldots, \beta_n(k_n + 1)\}$. Then it follows from (4.2) and (4.6) that $\frac{\mathrm{d}X}{\mathrm{d}t} \geq GX$. Applying Lemma 4.1 yields that $s(F - V) > 0$ if and only if $R_0 > 1$. Hence there exists a small enough ε_1 such that $s(G) > 0$ for $\varepsilon_1 \in [0, \varepsilon]$. Therefore, matrix G has a positive eigenvalue $s(G)$, which has a positive eigenvector. By the comparison principle, it is clear that $(E_i, I_i) \to (\infty, \infty)$ as $t \to \infty, i = 1, 2, \ldots, n$. This contradicts the previous assumption. Consequently, $\limsup_{t\to\infty} |\varphi_t(x_0) - P_0| > \delta$, that is, $W^S(P_0) \cap X_0 = \emptyset$.

Obviously, every orbit in M_∂ converges to $\{P_0\}$ and $\{P_0\}$ is an isolated invariant set and acyclic. Thus, Theorem 4.6 of Thieme [36] implies that system (2.2) is uniformly persistent with respect to $(X_0, \partial X_0)$. The proof is completed. By Theorem 2.4 in Zhao [35], system (2.2) has an equilibrium $E^* = (S_1^*, V_1^*, E_1^*, I_1^*, \ldots, S_n^*, V_n^*, E_n^*, I_n^*)$. The equations governing S_i and V_i in system (2.2) ensure that $S_i^* > 0$ and $V_i^* > 0$ for $i = 1, 2, \ldots, n$. This indicates that E^* is an endemic equilibrium of system (2.2). □

Table 1. Values of migration rates.

Para.	a_{12}	a_{13}	a_{21}	a_{23}	a_{31}	a_{32}	b_{12}	b_{13}	b_{21}	b_{23}	b_{31}	b_{32}
Value	0.025	0.027	0.022	0.039	0.034	0.032	0.06	0.1	0.08	0.15	0.12	0.16
Para.	c_{12}	c_{13}	c_{21}	c_{23}	c_{31}	c_{32}	d_{12}	d_{13}	d_{21}	d_{23}	d_{31}	d_{32}
Value	0.05	0.06	0.04	0.1	0.08	0.12	0.03	0.05	0.05	0.09	0.06	0.1

Table 2. Values of parameters in system (2.2).

Para.	p_i	Λ_i	β_i	q_i	k_i	σ_i	γ_i	d_i^S	d_i^V	d_i^E	d_i^I
$i = 1$	0.6	30	0.004	0.01	0.03	0.14	0.15	0.013	0.06	0.02	0.12
$i = 2$	0.7	20	0.005	0.018	0.04	0.2	0.18	0.012	0.1	0.01	0.15
$i = 3$	0.8	40	0.008	0.04	0.05	0.28	0.2	0.005	0.13	0.03	0.1

5. Numerical simulations

In this section, we give some numerical simulations to show the feasibility of our theoretical results and illustrate the impacts of vaccination and migration on disease prevalence.

We consider a special case of system (2.2) with $n = 3$, where the values of migration rates are given in Table 1 and the remaining parameter values are shown in Table 2. The initial condition is considered as $(S_1(0), V_1(0), E_1(0), I_1(0), S_2(0), V_2(0), E_2(0), I_2(0), S_3(0), V_3(0), E_3(0), I_3(0)) = (800, 120, 100, 80, 1000, 400, 248, 250, 1200, 300, 240, 200)$. Based on these parameter values, we can calculate that $P_0 = (317, 259, 0, 0, 258, 265, 0, 0, 244, 304, 0, 0)$ and $R_0 = 0.5 < 1$. Figure 2 shows that the trajectories of system (2.2) ultimately converge to P_0, which is globally asymptotically stable. So the disease will eventually die out. Now, we assume that the transmission coefficients β_i, are relatively high, e.g., $\beta_1 = 0.0012, \beta_2 = 0.0015, \beta_3 = 0.0025$, and keep the remaining parameter values unchanged. Then we can calculate that $R_0 = 1.53 > 1$. As seen in Figure 3, system (2.2) has a positive equilibrium, which confirms that the disease is uniformly persistent.

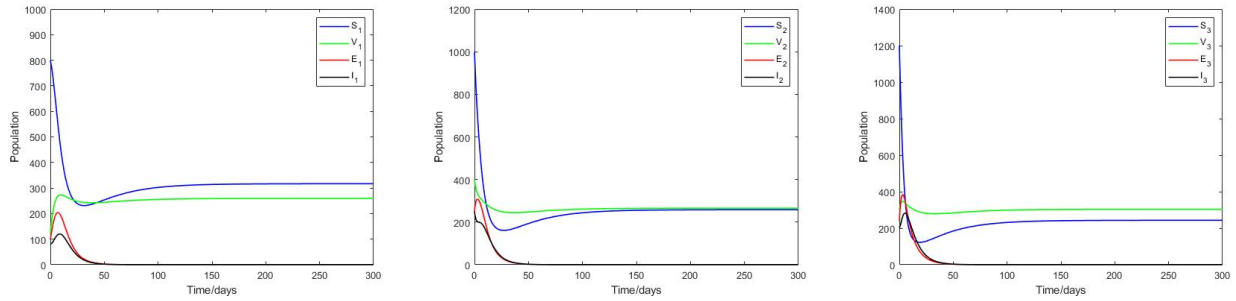

Figure 2. Time evolution of the population for each patch when $R_0 < 1$.

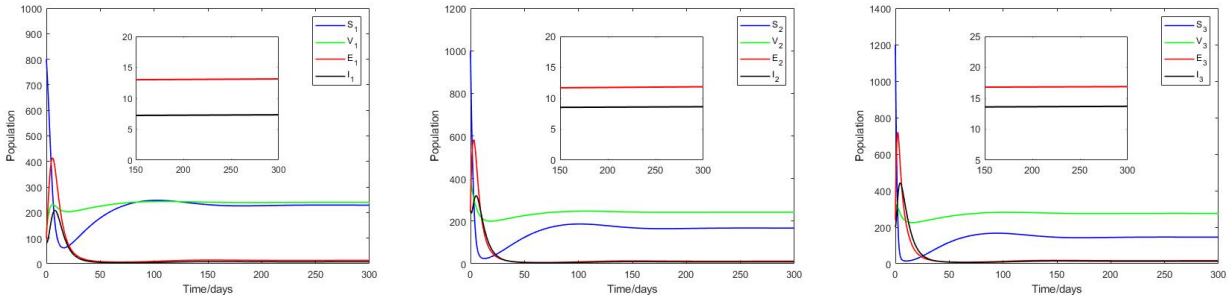

Figure 3. Time evolution of the population for each patch when $R_0 > 1$.

5.1. The effects of vaccination

Next, the effects of vaccination on infectious populations are shown in Figures 4 and 5. Assume $p_1 = p_2 = p_3$ and keep the other parameters in Tables 1 and 2 unchanged. From Figure 4, it can be observed that the numbers of infectious population decrease as the vaccination rate increases. The results imply that vaccination strategy plays an important role in preventing disease spread. Then, assume $q_1 = q_2 = q_3$. The impact of different vaccination strategies is shown in Figure 5. The blue solid represents that no vaccination strategy is implemented; the green solid represents vaccination of only susceptible populations; the red solid represents vaccination of only newborns; and the black solid represents that both vaccination strategies are implemented simultaneously. It can be seen from Figure 5 that the simultaneous execution of both vaccination strategies is the most effective way to reduce the number of infectious populations. Therefore, it should be suggested that both kinds of vaccination strategies should be implemented simultaneously.

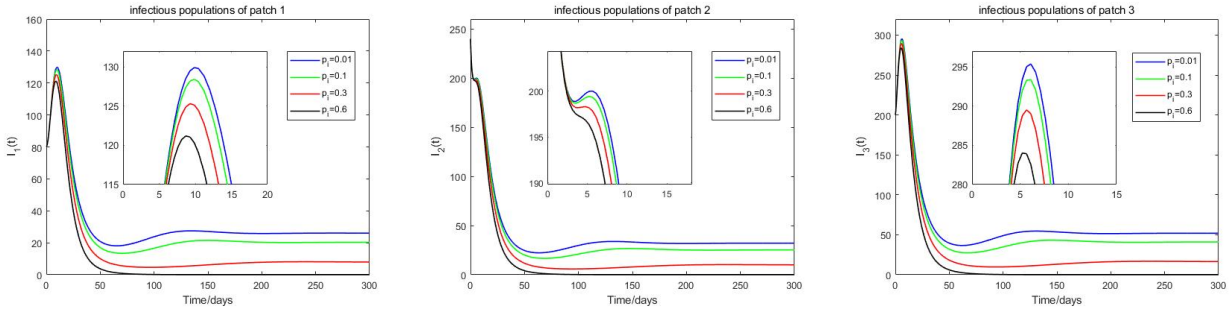

Figure 4. The effect of vaccination rate on infectious populations.

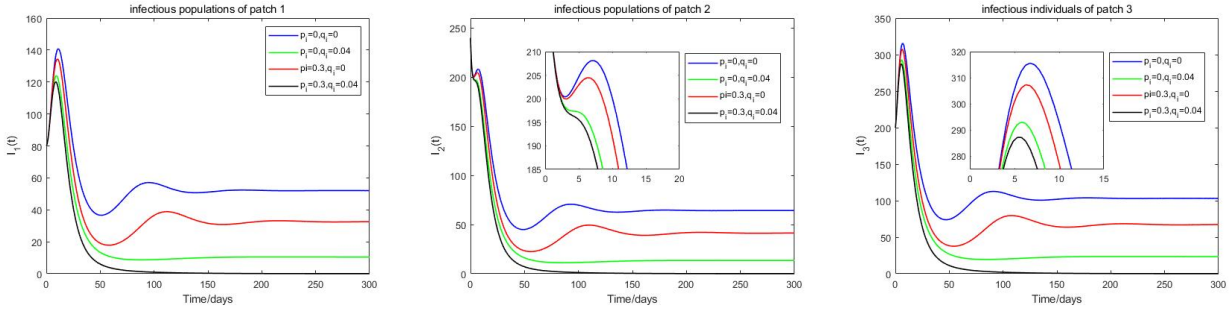

Figure 5. The effect of different vaccination strategies on infectious populations.

5.2. *The effects of migration*

The effects of migration on disease transmission are presented in Figure 6. Figure 6(a)–(c) show that as the migration rate increases, the numbers of infectious populations in patch 1 and patch 2 both decrease. However, with the increase in migration rate, the number of infectious populations in patch 3 increases. Hence, population migration can inhibit disease spread in patch 1 and patch 2, while spread in patch 3. Then, assume $\beta_1 = 0.0008, \beta_2 = 0.001, \beta_3 = 0.0015$. It is shown that R_0 increases with the increase of migration rate in Figure 6d. Thus, in order to prevent the outbreak of disease, we should limit or prohibit the migration of infectious populations from patch 1 to patch 3.

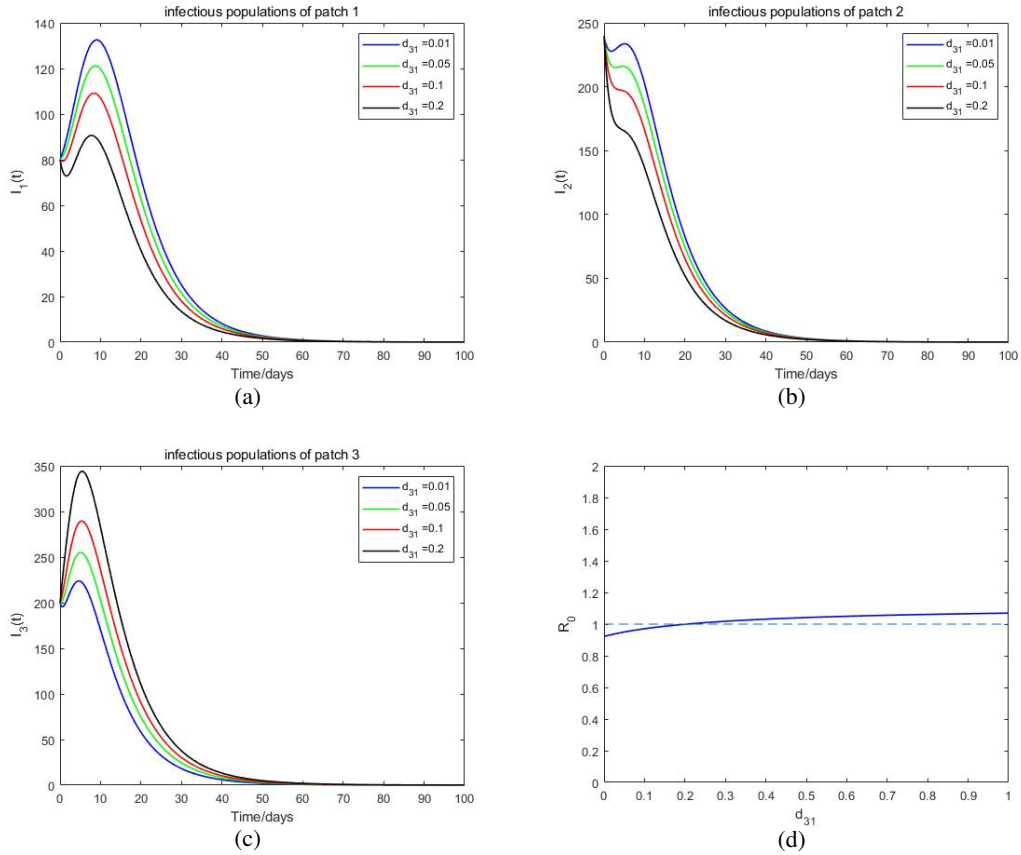

Figure 6. The effect of migration rate on disease transmission.

6. Conclusions

In this paper, we present an SVEIR epidemic model with vaccination in a patchy environment to investigate the impacts of vaccination strategy and population migration on disease dynamics. Based on the basic reproduction number R_0, we prove that the disease-free equilibrium P_0 is locally as well as globally asymptotically stable if $R_0 < 1$. In the case of $R_0 > 1$, we show that the system (2.2) is uniformly persistent. The numerical simulation results validate our stability analysis. In addition, numerical simulations indicate that vaccination is helpful for disease control in all patches and simultaneous execution of two vaccination strategies can be more effective in controlling the disease.

However, population migration does not always have a positive impact on disease spread. An increase in migration rate can either promote or inhibit disease transmission within a patch.

The impacts of vaccination strategy and population migration have been investigated in our paper. It can be concluded that vaccination can effectively control the spread of diseases. However, in the early stage of some emerging infectious diseases, vaccines have not been developed, so it is necessary to consider more control measures to prevent the outbreaks of diseases. In future work, we will extend the model by taking into account other control measures such as treatment and isolation.

Use of AI tools declaration

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

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Conflict of interest

The authors declare there is no conflict of interest.

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