MATHEMATICAL MODELING AND COMPUTING, Vol. 10, No. 4, pp. 1071–1083 (2023)

# A discrete mathematical model SIRS with the evolution of regions to attack infectious diseases

Benfatah Y., Khaloufi I., Boutayeb H., Laarabi H., Rachik M.

*Laboratory of Analysis, Modeling and Simulation, Casablanca, Morocco, 20670*

(Received 21 November 2022; Revised 21 November 2023; Accepted 23 November 2023)

This paper presents a new SIRS mathematical model describing the evolution of an infectious disease, assuming that the spatial supports of this infection are also evolutionary and obey a compartmental model. We propose four control strategies to manage the spread of the disease among individuals and regions. The Pontryagin maximum principle is employed to characterize the optimal controls, and the optimality system is solved using an iterative approach. Finally, numerical simulations are conducted to validate the theoretical analysis using MATLAB.

**Keywords:** *mathematical model; discrete-time systems; SIRS model; optimal control; contagious virus; Pontryagin maximum; zones evolution.*

**2010 MSC:** 26B10, 39A05, 39A10, 39A12, 49J10 **DOI:** 10.23939/mmc2023.04.1071

## 1. Introduction

Epidemics have wreaked widespread destruction around the world throughout human history, constantly changing the course of events in the global order [1]. Infectious diseases such as malaria, tuberculosis, smallpox, influenza, SARS, Ebola, HIV, and others have become part of history and claimed millions of lives. These pandemics have crossed borders and affected many countries or regions [2]. From 1346 to 1353, an outbreak of plague swept Europe, Africa, and Asia, and killed about a third of the world's population at that time. The history of plague epidemics shows that this pandemic (black death) has always spread much faster in the most populated areas, as in Europe in the 14th century [3]. The fast spread of an epidemic among certain populations, significantly exceeding the usual level of morbidity for a given area, it progresses over time and can become a source of emergency not only in a certain locality but also in the territory of several countries [4]. In the 19th century lasting from 1852 to 1860, the cholera pandemic originated in India, spreading from the Ganges Delta to Asia, Europe, North America, and Africa, killing more than 12 million people [5]. In the 20th century, HIV the virus that leads to AIDS (acquired immunodeficiency syndrome) became a global pandemic started by the Democratic Republic of the Congo and arrived in the United States in the 1970s precisely from Europe, Japan, Thailand, and Australia. Haitians working in Africa brought the virus to their homeland in 1964, and from there, it spread to the US [6]. The progressive development of infectious diseases reached the 21st century, the COVID-19 coronavirus pandemic from 2019 is one example that has emerged in the Wuhan region of China and spread incredibly fast among humans and around the world within a few months [7].

The fight against epidemics as a natural disaster was and remains a difficult task. Despite all existing measures to prevent the spread of diseases, the number of victims of infection can be estimated at millions of moralities. Thus, epidemiology came with the aid of mathematics to study and describe the occurrence's patterns and epidemic spread, as well as measures to combat and prevent their occurrence [8]. Hence, an epidemic process is the continuous spread of an infectious disease that occurs when three conditions are met: first, the presence of the source of infection; second, the transmission mechanism and people who are susceptible to infection. Thus, the absence of at least one of these conditions leads to disruption of the chain of the epidemic process and stops the transmission of the disease [9, 10].

As a result, spatial epidemiology issues are extremely essential, with viral propagation in populated areas being one of the most serious challenges. In order to construct dynamic models that provide a worldwide perspective into outbreak behavior, the incidence and prevalence of infectious illnesses in a specific population with various geographic and demographic parameters must be examined over the spatial and temporal domain [11, 12].

As a result, throughout the last century, a lot of studies have been conducted on the mathematical modeling of epidemics. The structure of these basic epidemic models allows for the simulation of occurrences that would be difficult to replicate in a laboratory setting. This type of model's basic assumption is that the population in which a pathogenic agent is active is made up of distinct subgroups of people, and they only look at the temporal dynamics of the infection cycle [13,14]. When it comes to the influence of epidemics' spatiotemporal spread, mathematical modeling should take into account the geographical characteristic in order to depict the spatial spread of an infectious illness across distinct geographical zones [15, 16].

However, an infectious disease can be spread by a variety of pathways, including direct contact with contaminated people, water, aerosol inhalation, and vector-borne spread. Direct contact of susceptible persons with infected individuals will be regarded as the main transmission medium of infectious diseases for the purposes of this study. As a result, infectious diseases are thought to spread from person to person through a network of contact. This paper's main goal is to investigate the global dynamics of a SIRS model with zone evolution. Consistently, the model simulation is based on the center idea that the population could be categorized into compartments discerned by a specific state taking into consideration the ongoing epidemic. The model classifies the population into Susceptible (persons who have the potential to get the disease), Infected (those individuals who are capable of spreading the disease), and Recovered (individuals who received immunity and cannot contract the infection again), from which the SIR acronym is utilized. A Susceptible could become Infected (S→I) by contacting an infected person or can get immunity to the disease and Recover (I→R) or die. If the assumption of the epidemic is based on a permanent immunity after the first infection to the disease. However, in case of the absence of this assumption and the immunity is not immutable then recovered individuals could be susceptible again (R→S), so it called the SIRS model [17, 18].

The combination of various scientifically based methods of epidemic control and prevention policies (anti-epidemic) measures helps to prevent the development of pandemics among the most vulnerable groups of the population, reduce the overall incidence in the country, and even completely eliminate individual diseases. This control strategy aimed at limiting the disease's source for restrictive measures there are two options quarantine aimed at isolating infected people; whereas, observation focuses on the isolation of healthy individuals who have been in contact with patients or carriers of infection in order to prevent the outbreak of an epidemic or its spread. Another way to prevent infectious diseases and their active spread among the population, (preventive vaccinations) is carried out [19, 20]. In [21], three approaches are provided in order to control the propagation of the pandemic including raising awareness by organizing vaccination campaigns, travel-blocking movements coming from infected areas, and treatment.

Travel restrictions are one part of a range of epidemic measures that could be used to control a global pandemic. In [22] have exposed supporting evidence, during the Influenza infectious disease in the United States illustrating that the grounding of airplanes in the US after September 11, 2001, slowed the dynamics of influenza during 2001/2002 by approximately two weeks. While air travel restrictions in the United States could have a slight effect on a contagious disease changing aspects due to ground transportation, the global spread, as a transfer of pandemic flu from Asia or Europe to the United States, might be delayed more remarkably by international travel restrictions [23]. For the same target, restriction policies were also introduced during the COVID-19 pandemic in China, showing positive results in decreasing the propagation of the disease in China which automatically affects the international control of the pandemic, for it slows the speed of infection transmission. Besides this, studies have shown the effect of travel restrictions to and from China on case importation

that were decreased approximately to 80% [24, 25] along with other models that emphasize the power of flight bans and border closures on the arrival of people with COVID-19 to many countries during the first phase of the pandemic [26, 27]. In [15], a discrete-time model was organized to show the spatial-temporal spread of epidemics, illustrating the transmission process from one region to another through air travel. The model provides an accurate survey of the propagation of the pandemic in different geographical areas.

The rest of the paper is organized as follows: in section 2, we present the discrete-time mathematical model $SIRS$ and $Z^S Z^I Z^R$ describing the evolution of infectious diseases or viruses between individuals. In section 3, the optimal control problem of the considered model is studied, and results and a discussion are provided to ensure the effectiveness of our control strategies in section 4. To conclude our paper, a conclusion is provided in section 5.

## 2. Presentation of the model

Infectious diseases are modeled by different types of mathematical models with compartments. Inspired by the following research [28, 29], we consider a SIRS model that considers that the spatial patterns of the infection by an infectious disease also change over time and adhere to a compartmental model. The model considered is divided into two parts. The first SIRS describes the evolution of individuals during an infectious disease, where $S$ represents the number of susceptible, $I$ the infected, and $R$ the recovered. The second part $Z$ represents the various types of regions $Z^S$ represents the number of susceptible regions, where there are only susceptible individuals, after visiting an infected person, a susceptible region is likely to be infected, which we will note as $Z^I$, the last compartment $Z^R$ designates the infected areas which are recovered.

Therefore, we obtain the following two models: (1) for the evolution of individuals, and (2) for the evolution of regions.

$$\begin{cases} S_{i+1} = \Lambda + S_i - \alpha\, S_i I_i - \mu\, S_i + \delta R_i, \\ I_{i+1} = I_i + \alpha\, S_i I_i - (\mu + d)\, I_i - r I_i, \\ R_{i+1} = -\mu\, R_i + r I_i + R_i - \delta R_i, \end{cases} \tag{1}$$

$$\begin{cases} Z^S_{i+1} = Z^S_i - \beta Z^S_i I^I_i + \theta Z^R_i, \\ Z^I_{i+1} = Z^I_i + \beta Z^S_i I_i - \gamma Z^I_i, \\ Z^R_{i+1} = Z^R_i + \gamma Z^I_i - \theta Z^R_i, \end{cases} \tag{2}$$

with initial conditions $S_0 \geqslant 0$, $I_0 \geqslant 0$, $R_0 \geqslant 0$, $Z^S_0 \geqslant 0$, $Z^I_0 \geqslant 0$, and $Z^R_0 \geqslant 0$ and where $i \in \{0, 1, \ldots, N-1\}$.

The description of the parameters is given in the following Table 1.

**Table 1.** List of all parameters of system (1).

| Parameter | Physical interpretation |
|---|---|
| $\Lambda$ | The incidence of susceptible |
| $\alpha$ | The rate of people who were infected by contact with infected. |
| $\beta$ | The rate of regions that become infected since infected people. |
| $\theta$ | The rate of conversion from recovered regions to susceptible regions. |
| $\mu$ | The natural death rate. |
| $\delta$ | The rate at which the recoveries become susceptible. |
| $d$ | Mortality since the virus. |
| $r$ | The rate of conversion from infected people to recovered people. |
| $\gamma$ | The rate of conversion from infected regions to recovered regions. |

## 3. The optimal control problem

### 3.1. Presentation of the controls

In this section, we consider four control strategies. The first strategy (control $u$) is based on educating people about the necessity of avoiding contact with infected persons and taking precautionary measures. The second strategy (control $v$) gives medical care to patients and makes recovery rooms available in case of serious health problems. The last two strategies ($w$ and $p$ control) are applied to the regions. This is done by isolating infected areas and striving to reduce the number of infected people in these areas.

Then, two discrete-time controlled systems associated with (1) and (2) are given as follows

$$\begin{cases} S_{i+1} = \Lambda + S_i + -\alpha(1-u_i)S_iI_i - \mu S_i + \delta R_i, \\ I_{i+1} = I_i + \alpha(1-u_i)S_iI_i - (\mu+d)\,I_i - (r+v_i)J_i, \\ R_{i+1} = -\mu\,R_i + (r+v_i)I_i - \delta R_i, \end{cases} \tag{3}$$

$$\begin{cases} Z^S_{i+1} = Z^S_i - \beta w_i Z^S_i I_i + \theta Z^R_i, \\ Z^I_{i+1} = Z^I_i + \beta w_i Z^S_i I_i - \gamma Z^I_i - p_i Z^I_i, \\ Z^R_{i+1} = Z^R_i + \gamma Z^I_i + p_i Z^I_i - \theta Z^R_i, \end{cases} \tag{4}$$

with initial conditions $S_0 \geqslant 0$, $I_0 \geqslant 0$, $R_0 \geqslant 0$, $Z^S_0 \geqslant 0$, $Z^I_0 \geqslant 0$, and $Z^R_0 \geqslant 0$, and where $i \in \{0, 1, \ldots, N-1\}$.

The strategy of control $v$ represents the vaccination of susceptible individuals and thus protects them from the virus, while the strategies of controls $u$ and $w$ represent travel-blocking, where we block the meeting between susceptible and infected people and the visiting of infected people to the susceptible regions.

### 3.2. Objective functional

The problem is to minimize the objective functional $J(u,v,w,p)$ given by

$$J(u,v,w,p) = \alpha_I\,J_N - \alpha_R\,R_N + \alpha_{Z^I}{Z^I}_N + \sum_{i=0}^{N-1}\left(\alpha_I I_i - \alpha_R R_i + \alpha_{Z^I} Z^I_i + \frac{A}{2}u_i^2 + \frac{B}{2}v_i^2 + \frac{C}{2}w_i^2 + \frac{Dp_i^2}{2}\right),$$

where $A>0$, $B>0$, $C>0$, $D>0$, $\alpha_I>0$, $\alpha_R>0$, $\alpha_{Z^I}>0$ are the weight constants of controls, $u=(u_0,\ldots,u_{N-1})$, $v=(v_0,\ldots,v_{N-1})$, $w=(w_0,\ldots,w_{N-1})$, $p=(p_0,\ldots,p_{N-1})$, and $N$ is the final time of our strategy of control. Our goal is to minimize the infected individuals and the infected regions, minimize the cost of applying controls, and increase the number of removed. In other words, we are seeking optimal controls $u^*$, $v^*$, $w^*$, and $p^*$ such that

$$J(u^*,v^*,w^*,p^*) = \min\left\{J(u,v,w,p)/u\in\mathcal{U}, v\in\mathcal{V}, w\in\mathcal{W}, p\in\mathcal{P}\right\}, \tag{5}$$

where $\mathcal{U}$, $\mathcal{V}$, $\mathcal{W}$, and $\mathcal{P}$ are the control sets defined by

$$\mathcal{U} = \{u/u_{\min}\leqslant u_i\leqslant u_{\max}, i=0,\ldots,N-1\}, \tag{6}$$

$$\mathcal{V} = \{v/v_{\min}\leqslant v_i\leqslant v_{\max}, i=0,\ldots,N-1\}, \tag{7}$$

$$\mathcal{W} = \{w/w_{\min}\leqslant w_i\leqslant w_{\max}, i=0,\ldots,N-1\}, \tag{8}$$

$$\mathcal{P} = \{p/p_{\min}\leqslant p_i\leqslant p_{\max}, i=0,\ldots,N-1\}, \tag{9}$$

such that

$$0<u_{\min}<u_{\max}<1, \quad 0<v_{\min}<v_{\max}<1, \quad 0<w_{\min}<w_{\max}<1, \quad 0<p_{\min}<p_{\max}<1.$$

### 3.3. Sufficient conditions

**Theorem 1.** *There exists an optimal control $(u^*,v^*,w^*,p^*)\in\mathcal{U}\times\mathcal{V}\times\mathcal{W}\times\mathcal{P}$ such that*

$$J(u^*,v^*,w^*,p^*) = \min\{J(u,v,w,p)/u\in\mathcal{U}, v\in\mathcal{V}, w\in\mathcal{W}, p\in\mathcal{P}\}$$

*associated with the controlled systems (3), (4) and the initial conditions.*

**Proof.** Since the parameters of the system are bounded and there are a finite number of time steps, that is $I$, $S$, $R$, $Z^S$, $Z^I$, and $Z^R$ are uniformly bounded for all $(u, v, w, p)$ in the control set $\mathcal{U}\times\mathcal{V}\times\mathcal{W}\times\mathcal{P}$, thus $J(u, v, w, p)$ is also bounded for all $(u, v, w, p) \in \mathcal{U} \times \mathcal{V} \times \mathcal{W} \times \mathcal{P}$.

Which implies that $\inf\limits_{(u,v,w,p)\in\mathcal{U}\times\mathcal{V}\times\mathcal{W}\times\mathcal{P}} J(u, v, w, p)$ is finite, and there exists a sequence $(u^n, v^n, w^n, p^n) \in \mathcal{U} \times \mathcal{V} \times \mathcal{W} \times \mathcal{P}$ such that

$$\lim_{n\to+\infty} J(u^n, v^n, w^n, p^n) = \inf_{(u,v,w,p)\in\mathcal{U}\times\mathcal{V}\times\mathcal{W}\times\mathcal{P}} J(u, v, w, p)$$

and corresponding sequences of states $I^n$, $S^n$, $R^n$, and $Z^{S^n}$, $Z^{I^n}$, $Z^{R^n}$. Since there exists a finite number of uniformly bounded sequences such that $(u^*, v^*, w^*, p^*) \in \mathcal{U} \times \mathcal{V} \times \mathcal{W} \times \mathcal{P}$ and $I^*$, $S^*$, $R^*$, and $Z^{S^*}$, $Z^{I^*}$, $Z^{R^*}$ such as, in a sequence,

$$\begin{aligned}(u^n, v^n, w^n, p^n) &\to (u^*, v^*, w^*, p^*),\\ S^n &\to S^*,\\ I^n &\to I^*,\\ R^n &\to R^*,\\ Z^{S^n} &\to Z^{S^*},\\ Z^{I^n} &\to Z^{I^*},\\ Z^{R^n} &\to Z^{R^*}.\end{aligned}$$

Finally, due to the finite-dimensional structure of the system (3), (4) and the objective function $J(u, v, w, p)$, the control $(u^*, v^*, w^*, p^*)$ is an optimal control with corresponding states $I^*$, $S^*$, $R^*$, $Z^{S^*}$, $Z^{I^*}$, and $Z^{R^*}$. Which completes the proof. ∎

### 3.4. Necessary conditions

By using a discrete version of Pontryagin's maximum principle [30–32], we derive the necessary conditions for our optimal controls. For this purpose, we define the Hamiltonian as

$$\begin{aligned}\mathcal{H}_i = {}& \alpha_I I_i - \alpha_R R_i + \alpha_{Z^I} Z_i^I + \frac{Au_i^2}{2} + \frac{Bv_i^2}{2} + \frac{Cw_i^2}{2} + \frac{Dp_i^2}{2}\\ &+ \zeta_{1,i}\big(\Lambda + S_i - \alpha(1-u_i)S_iI_i - \mu S_i + \delta R_i\big) + \zeta_{2,i}\big(I_i + \alpha(1-u_i)S_iI_i - (\mu+d)I_i - rI_i - v_iI_i\big)\\ &+ \zeta_{3,i}\big(-\delta R_i - \mu R_i + rI_i + v_iI_i + R_i\big) + \zeta_{4,i}\big(-\beta w_i Z_i^S I_i + \theta Z K_i + Z_i^S\big)\\ &+ \zeta_{5,i}\big(\beta w_i Z_i^S I_i - \gamma Z_i^I - p_i Z_i^I + Z_i^I\big) + \zeta_{6,i}\big(\gamma Z_i^I - \theta Z K_i + p_i Z_i^I + Z K_i\big).\end{aligned}$$

**Theorem 2.** *Given optimal controls $u^*$, $v^*$, $w^*$, $p^*$, and solutions $I^*$, $S^*$, $R^*$, $Z^{S_i^*}$, $Z^{I_i^*}$, and $Z^{R_i^*}$, there exists $\zeta_{k,i}$, $i = 1, \dots, N$, $k = 1, 2, \dots, 6$, the adjoint variables satisfying the following equations*

$$\begin{aligned}\Delta\zeta_{1,i} &= -\big[\zeta_{1,i}\big(1 - \alpha(1-u_i)I_i - \mu\big) + \zeta_{2,i}\alpha(1-u_i)I_i\big],\\ \Delta\zeta_{2,i} &= -\big[\alpha_I - \zeta_{1,i}\alpha(1-u_i)S_i + \zeta_{3,i}(r+v_i) - \beta Z_i^S\zeta_{4,i}w_i + \beta Z_i^S\zeta_{5,i}w_i\\ &\qquad + \zeta_{2,i}(1 + \alpha(1-u_i)S_i - \mu - d - r - v_i)\big],\\ \Delta\zeta_{3,i} &= -\big[-\alpha_R + a_{i+1}\delta + \zeta_{3,i}(-\mu - \delta + 1)\big],\\ \Delta\zeta_{4,i} &= -\big[\zeta_{4,i}(-\beta I_i w_i + 1) + \zeta_{5,i}\beta I_i w_i\big],\\ \Delta\zeta_{5,i} &= -\big[\alpha_{Z^I} + \zeta_{5,i}(-\gamma - p_i + 1) + g_{i+1}(\gamma + p_i)\big],\\ \Delta\zeta_{6,i} &= -\big[\zeta_{4,i}\theta + \zeta_{6,i}(-\theta + 1)\big],\end{aligned} \tag{10}$$

*where $\zeta_{1,N} = 0$, $\zeta_{2,N} = \alpha_I$, $\zeta_{3,N} = -\alpha_R$, $\zeta_{4,N} = 0$, $\zeta_{5,N} = \alpha_{Z^I}$, $\zeta_{6,N} = 0$ are the transversality conditions. In addition $u^* = (u_0^*, \dots, u_{N-1}^*)$, $v^* = (v_0^*, \dots, v_{N-1}^*)$, $w^* = (w_0^*, \dots, w_{N-1}^*)$, and $p^* = (p_0^*, \dots, p_{N-1}^*)$ are given by*

$$\begin{aligned}u_i^* &= \min\left\{\max\left\{u_{\min}, \frac{(-\zeta_{1,i}-\zeta_{2,i})\alpha S_iI_i}{A}\right\}, u_{\max}\right\}, \quad i = 0, \dots, N-1,\\ v_i^* &= \min\left\{\max\left\{v_{\min}, \frac{-(\zeta_{3,i}-\zeta_{2,i})\alpha I_i}{B}\right\}, v_{\max}\right\}, \quad i = 0, \dots, N-1,\end{aligned}$$

$$w_i^* = \min\left\{\max\left\{w_{\min}, \frac{-(\zeta_{5,i}-\zeta_{4,i})\beta Z_i^S I_i}{C}\right\}, w_{\max}\right\}, \quad i = 0, \dots, N-1,$$
$$p_i^* = \min\left\{\max\left\{p_{\min}, \frac{-(\zeta_{6,i}-\zeta_{5,i})Z_i^I}{D}\right\}, p_{\max}\right\}, \quad i = 0, \dots, N-1.$$

**Proof.** Using the discrete version of Pontryagin's maximum principle [33–37] we obtain the following adjoint equations:

$$\begin{aligned}
\Delta\zeta_{1,i} &= -\frac{\partial \mathcal{H}}{\partial S_i} \\
&= -\big[\zeta_{1,i}(1-\alpha(1-u_i)I_i - \mu) + \zeta_{2,i}\alpha(1-u_i)I_i\big], \\
\Delta\zeta_{2,i} &= -\frac{\partial \mathcal{H}}{\partial I_i} \\
&= -\big[\alpha_I - \zeta_{1,i}\alpha(1-u_i)S_i + \zeta_{3,i}(r+v_i) - \beta Z_i^S\zeta_{4,i}w_i + \beta Z_i^S\zeta_{5,i}w_i \\
&\qquad + \zeta_{2,i}(1+\alpha(1-u_i)S_i - \mu - d - r - v_i)\big], \\
\Delta\zeta_{3,i} &= -\frac{\partial \mathcal{H}}{\partial R_i} \\
&= -\big[-\alpha_R + \zeta_{1,i}\delta + \zeta_{3,i}(-\mu-\delta+1)\big], \\
\Delta\zeta_{4,i} &= -\frac{\partial \mathcal{H}}{\partial Z_i^I} \\
&= -\big[\zeta_{4,i}(-\beta I_i w_i + 1) + \zeta_{5,i}\beta I_i w_i\big] \\
\Delta\zeta_{5,i} &= -\frac{\partial \mathcal{H}}{\partial Z_i^I} \\
&= -\big[\alpha_{Z^I} + \zeta_{5,i}(-\gamma - p_i + 1) + g_{i+1}(\gamma + p_i)\big] \\
\Delta\zeta_{6,i} &= -\frac{\partial \mathcal{H}}{\partial Z_i^R} = \\
&= -\big[\zeta_{4,i}\theta + \zeta_{6,i}(-\theta+1)\big]
\end{aligned}$$

with $\zeta_{1,N} = 0$, $\zeta_{2,N} = \alpha_I$, $\zeta_{3,N} = -\alpha_R$, $\zeta_{4,N} = 0$, $\zeta_{5,N} = \alpha_{Z^I}$, $\zeta_{6,N} = 0$. To obtain the optimality conditions we take the variation with respect to controls $(u_i,\, v_i,\, w_i,\, p_i)$ and set it equal to zero

$$\begin{aligned}
\frac{\partial \mathcal{H}_i}{\partial u_i} &= Au_i + (\zeta_{1,i} - \zeta_{2,i})\alpha S_i I_i = 0, \\
\frac{\partial \mathcal{H}_i}{\partial v_i} &= Bv_i + (\zeta_{3,i} - \zeta_{2,i})\alpha I_i = 0, \\
\frac{\partial \mathcal{H}_i}{\partial w_i} &= Cw_i + (\zeta_{5,i} - \zeta_{4,i})\beta Z_i^S I_i = 0, \\
\frac{\partial \mathcal{H}_i}{\partial p_i} &= Dp_i + (\zeta_{6,i} - \zeta_{5,i})Z_i^I = 0.
\end{aligned}$$

Then we obtain the optimal control

$$\begin{aligned}
u_i &= \frac{-(\zeta_{1,i}-\zeta_{2,i})\alpha S_i I_i}{A}, \\
v_i &= \frac{-(\zeta_{3,i}-\zeta_{2,i})\alpha I_i}{B}, \\
w_i &= \frac{-(\zeta_{5,i}-\zeta_{4,i})\beta Z_i^S I_i}{C}, \\
p_i &= \frac{-(\zeta_{6,i}-\zeta_{5,i})Z_i^I}{D}.
\end{aligned}$$

By the bounds in $\mathcal{U}$, $\mathcal{V}$, $\mathcal{W}$, and $\mathcal{P}$ of the controls in the definitions (6), (7) and (8), it is easy to obtain $u_i^*$, $v_i^*$, $w_i^*$, and $p_i^*$ in the following form

$$u_i^* = \min\left\{\max\left\{u_{\min}, \frac{(-\zeta_{1,i}-\zeta_{2,i})\alpha S_i I_i}{A}\right\}, u_{\max}\right\}, \quad i = 0, \dots, N-1,$$
$$v_i^* = \min\left\{\max\left\{v_{\min}, \frac{-(\zeta_{3,i}-\zeta_{2,i})\alpha I_i}{B}\right\}, v_{\max}\right\}, \quad i = 0, \dots, N-1,$$
$$w_i^* = \min\left\{\max\left\{w_{\min}, \frac{-(\zeta_{5,i}-\zeta_{4,i})\beta Z_i^S I_i}{C}\right\}, w_{\max}\right\}, \quad i = 0, \dots, N-1,$$
$$p_i^* = \min\left\{\max\left\{p_{\min}, \frac{-(\zeta_{6,i}-\zeta_{5,i})Z_i^I}{D}\right\}, p_{\max}\right\}, \quad i = 0, \dots, N-1.$$

■

## 4. Results and discussion

### 4.1. Numerical simulation

In this section, we present the numerical simulations related to the previously mentioned optimization problem (5). More specifically, the program code is described in MATLAB (see Algorithm 1) and we perform simulations of our results. A discrete iterative method is applied to solve the optimality systems, and it converges after an adequate test such as the one for FBSM. The state system (3), (4) is then solved forward in time under the initial hypothesis, followed by the adjoint system (10) which is solved backward in time due to the transversality conditions. Next, we will make sure to update the optimal control values through state and co-state values acquired in the previous steps. Finally, the previous steps are carried out when the required standard of tolerance is reached.

### 4.2. Algorithm

**Algorithm 1** Determination of states of the controlled system and controls $u$, $v$, $w$, and $p$.

**Require:** $S_0$, $I_0$, $R_0$, $Z_0^S$, $Z_0^I$, $Z_0^R$, $N$, $u(0) = v(0) = w(0) = p(0) = 0$, $\zeta_{1,N} = 0$, $\zeta_{2,N} = \alpha_I$, $\zeta_{3,N} = -\alpha_R$, $\zeta_{4,N} = 0$, $\zeta_{5,N} = \alpha_{Z^I}$, $\zeta_{6,N} = 0$.

**for** $i = 1, \ldots, N-1$

$$\begin{cases} S_{i+1} = \Lambda + S_i - \alpha(1-u_i)S_iI_i - \mu S_i + \delta R_i, \\ I_{i+1} = I_i + \alpha(1-u_i)S_iI_i - (\mu+d)I_i - (r+v_i)J_i, \\ R_{i+1} = -\mu R_i + (r+v_i)I_i - \delta R_i, \end{cases} \qquad \begin{cases} Z_{i+1}^S = Z_i^S - \beta w_i Z_i^S I_i + \theta Z_i^R, \\ Z_{i+1}^I = Z_i^I + \beta w_i Z_i^S I_i - \gamma Z_i^I - p_i Z_i^I, \\ Z_{i+1}^R = Z_i^R + \gamma Z_i^I + p_i Z_i^I - \theta Z_i^R. \end{cases}$$

$$\begin{aligned} \zeta_{1,i} &= \zeta_{1,i+1} + \left[\zeta_{1,i}(1-\alpha(1-u_i)I_i - \mu) + \zeta_{2,i}\alpha(1-u_i)I_i\right], \\ \zeta_{2,i} &= \zeta_{2,i+1} + \big[\alpha_I - \zeta_{1,i}\alpha(1-u_i)S_i + \zeta_{3,i}(r+v_i) + \beta Z_i^S(\zeta_{5,i} - \zeta_{4,i})w_i \\ &\qquad + \zeta_{2,i}(1+\alpha(1-u_i)S_i - \mu - d - r - v_i)\big], \\ \zeta_{3,i} &= \zeta_{3,i} + \left[-\alpha_R + a_{i+1}\delta + \zeta_{3,i}(-\mu - \delta + 1)\right], \\ \zeta_{4,i} &= \zeta_{4,i} + \left[\zeta_{4,i}(-\beta I_i w_i + 1) + \zeta_{5,i}\beta I_i w_i\right], \\ \zeta_{5,i} &= \zeta_{5,i} + \left[\alpha_{Z^I} + \zeta_{5,i}(-\gamma - p_i + 1) + g_{i+1}(\gamma + p_i)\right], \\ \zeta_{6,i} &= \zeta_{6,i} + \left[\zeta_{4,i}\theta + \zeta_{6,i}(-\theta + 1)\right], \end{aligned}$$

$$u_i^* = \min\left\{\max\left\{u_{\min}, \frac{(-\zeta_{1,i} - \zeta_{2,i})\alpha S_i I_i}{A}\right\}, u_{\max}\right\}, \quad i = 0, \ldots, N-1,$$

$$v_i^* = \min\left\{\max\left\{v_{\min}, \frac{-(\zeta_{3,i} - \zeta_{2,i})\alpha I_i}{B}\right\}, v_{\max}\right\}, \quad i = 0, \ldots, N-1,$$

$$w_i^* = \min\left\{\max\left\{w_{\min}, \frac{-(\zeta_{5,i} - \zeta_{4,i})\beta Z_i^S I_i}{C}\right\}, w_{\max}\right\}, \quad i = 0, \ldots, N-1,$$

$$p_i^* = \min\left\{\max\left\{p_{\min}, \frac{-(\zeta_{6,i} - \zeta_{5,i})Z_i^I}{D}\right\}, p_{\max}\right\}, \quad i = 0, \ldots, N-1.$$

### 4.3. Simulation with and without the control $u$ and discussions on the results

In this particular approach, we exclusively apply the initial control parameter, denoted by "$u$", which consists of conducting awareness programs to emphasize the importance of adhering to preventive measures. It is worth noting that this strategy resulted in a marginal decrease in the number of infected individuals, as illustrated in Figure 1. However, it is essential to recognize that this strategy, when employed in isolation, proves less effective for the other compartments, as illustrated in Figures 2 and 3.

### 4.4. Simulation with and without the control $v$ and discussions on the results

In this second strategy, we focus on the exclusive application of the second control strategy, "$v$". This strategic intervention involves the implementation of comprehensive healthcare programs specifically

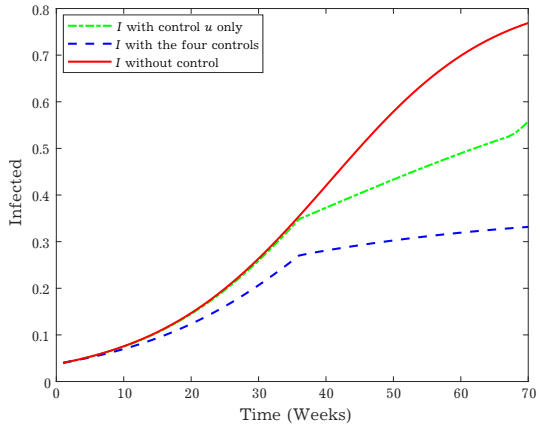


**Fig. 1.** Infected individuals without and with the control $u$.

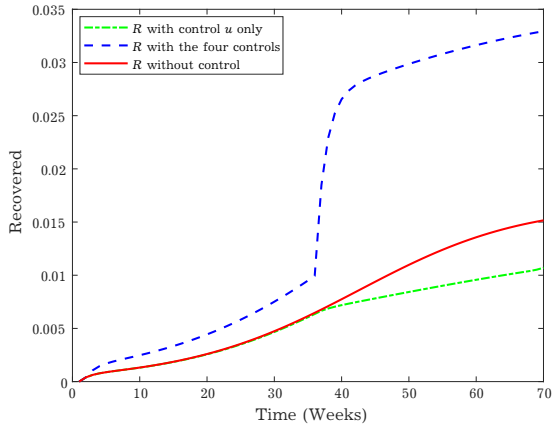


**Fig. 2.** Recovered individuals without and with the control $u$.

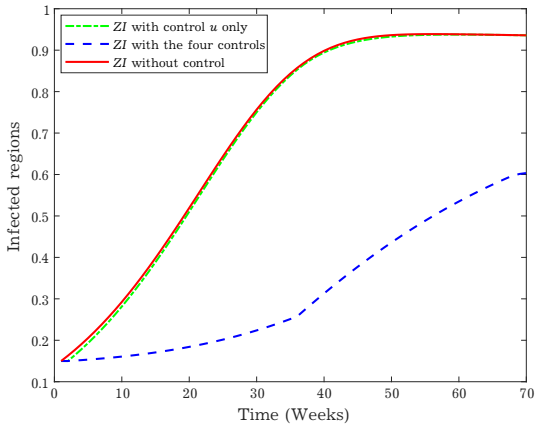


**Fig. 3.** Infected regions without and with the control $u$.

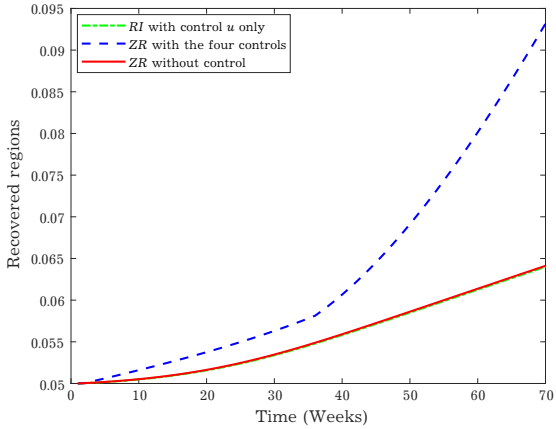


**Fig. 4.** Recovered regions without and with the control $u$.

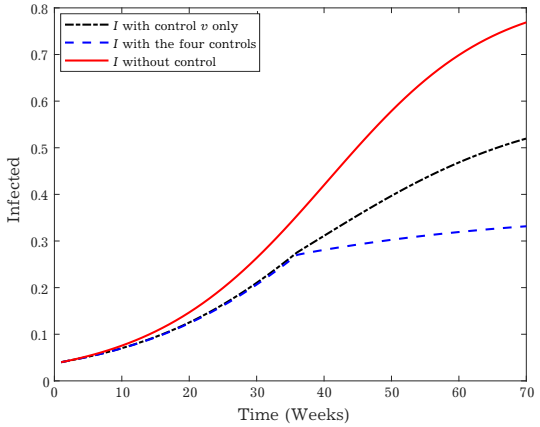


**Fig. 5.** Infected individuals without and with the control $v$.

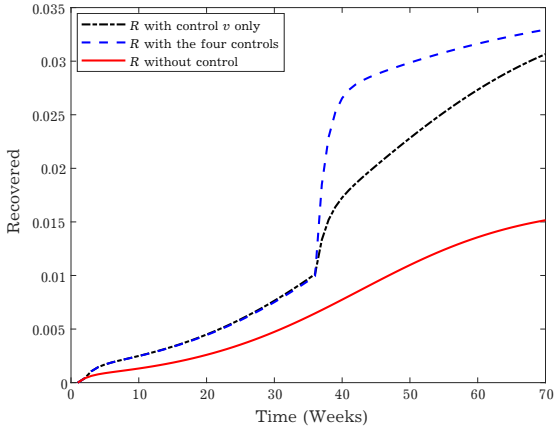


**Fig. 6.** Recovered individuals without and with the control $v$.

designed to meet the needs of patients affected by the disease. It also involves setting up specialized recovery rooms for people facing serious health problems due to the progression of the disease.

The results of this strategy reveal some interesting signs. Firstly, there has been a marked reduction in the total number of people contracting the disease, as illustrated in Figure 5. In addition, the geographical impact of the disease was also reduced, with a smaller number of regions affected, as illustrated in Figure 7.

In addition, this strategy led to a commendable increase in the number of people who managed to recover from the disease, as illustrated in Figure 6. These positive results underline the effectiveness of targeted healthcare programs and convalescent wards in mitigating the impact of the disease.

However, it is essential to stress that while this strategy has shown promise and produced several positive results, it should not be used in isolation.

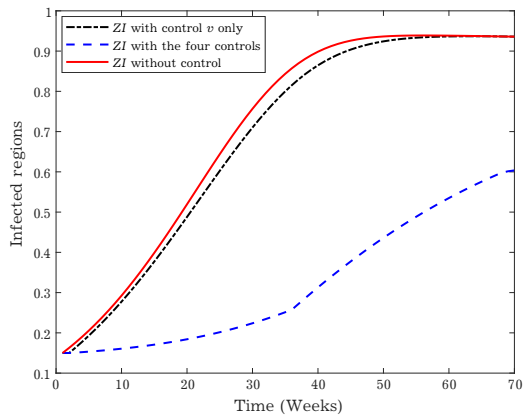


**Fig. 7.** Infected regions without and with the control $v$.

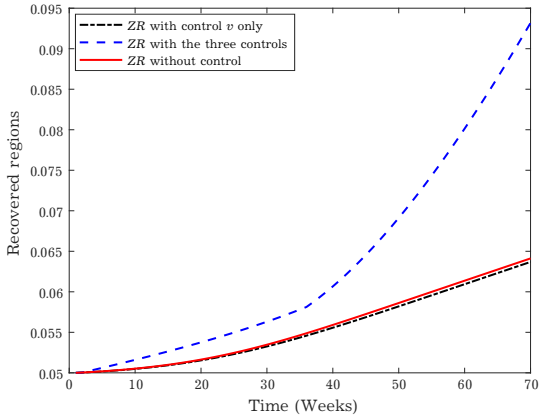


**Fig. 8.** Recovered regions without and with the control $v$.

### 4.5. Simulation with and without the control $w$ and discussions on the results

In the third strategy, we focus exclusively on implementing the third control, "$w$". This particular strategy involves the strategic deployment of measures such as erecting barriers to prevent the movement of individuals already infected with the disease to areas with susceptible populations. It also involves the application of travel restrictions, which limit the mobility of individuals likely to be carriers of the infection.

Figure 9, which illustrates the number of infected geographical zones, highlights a notable result of this strategy. We can see that this strategy has considerably reduced the number of areas affected by the disease. The visual representation of these reduced areas of infection highlights the effectiveness of these control measures in containing the spread of the disease within the regions.

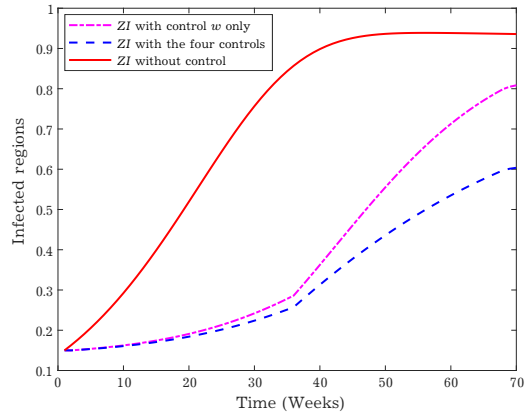


**Fig. 9.** Infected regions without and with the control $w$.

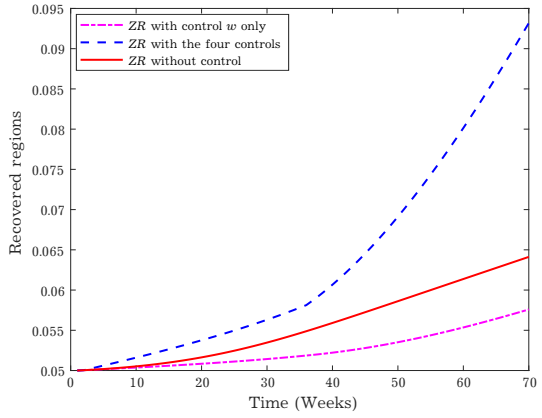


**Fig. 10.** Recovered regions without and with the control $w$.

### 4.6. Simulation with and without the control $p$ and discussions on the results

In this strategy, we focus exclusively on implementing the fourth control, designated "$p$". This strategy involves a comprehensive approach to increasing support for regions heavily affected by the disease. This support includes increasing the allocation of medical personnel to these regions and building large-scale medical facilities, often referred to as "mega-hospitals", in these affected areas.

The results of this strategic intervention are remarkable. By channeling more resources and medical expertise to affected regions, we see a marked reduction in the number of areas still struggling with the disease, as Figure 11 clearly illustrates. At the same time, this approach is helping to increase the number of regions that have managed to recover from the disease, as illustrated in Figure 12.

The construction of mega-hospitals, combined with the deployment of additional medical cadres, plays a key role in reducing disease prevalence and promoting recovery in these regions. This strategy underlines the importance of targeted investment and resource allocation as effective means of combating the impact of the disease on a regional scale.

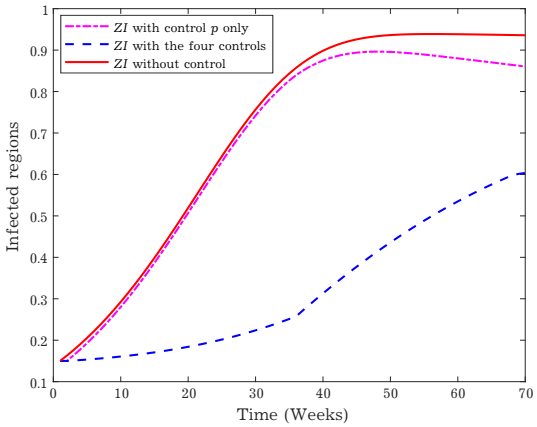


**Fig. 11.** Infected regions without and with the control $p$.

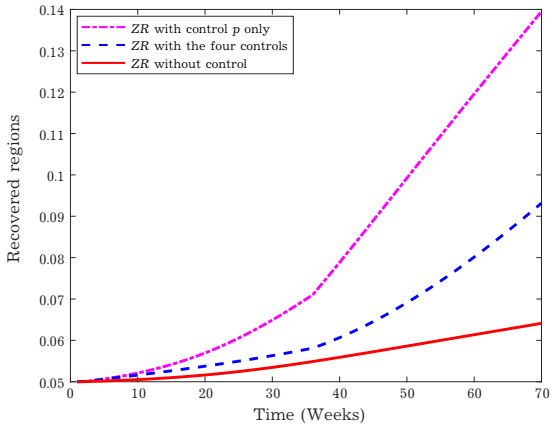


**Fig. 12.** Recovered regions without and with the control $p$.

### 4.7. Simulation with and without four controls and discussions on the results

In this last strategy, we take a holistic approach, combining four strategies mentioned above. This holistic approach is designed to maximize the impact on disease containment and recovery efforts.

The results of this integrated strategy are particularly encouraging, as illustrated in Figures 13 and 15. These figures clearly demonstrate the effectiveness of this multi-faceted approach in achieving several crucial objectives. Firstly, the number of people contracting the disease has fallen considerably, as shown in Figure 13. At the same time, the geographical spread of the disease is considerably reduced, with fewer areas affected, as illustrated in Figure 15.

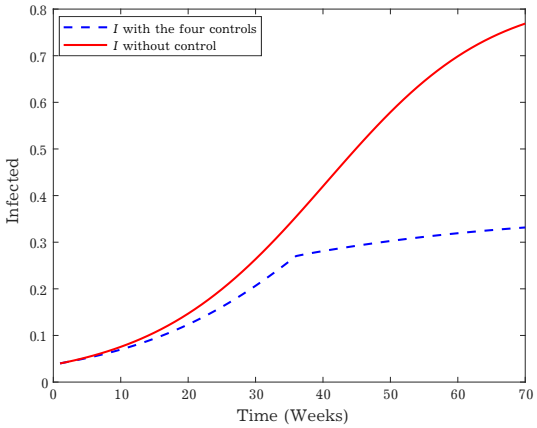


**Fig. 13.** Infected individuals without and with four controls.

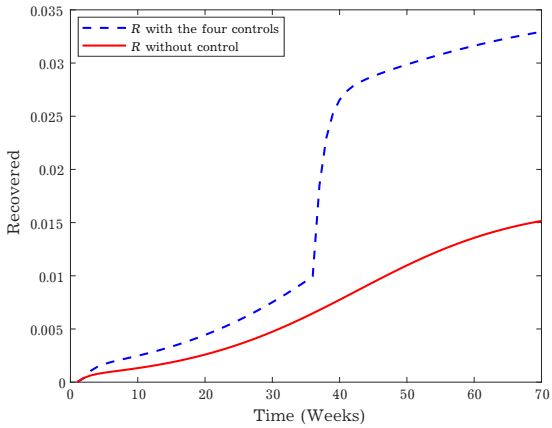


**Fig. 14.** Recovered individuals without and with four controls.

In addition, this global strategy leads to a significant increase in the number of people successfully cured of the disease, as illustrated in Figure 14. In addition, the number of regions that moved from disease to cure also increased substantially, as illustrated in Figure 16.

The integration of these four strategies underlines the synergy achieved by simultaneously tackling the disease from multiple angles. This approach relies on a combination of awareness-raising programs, travel restrictions, healthcare initiatives, resource allocation, and medical infrastructure development to comprehensively combat the impact of the disease. It is a convincing example of the effectiveness of a multi-faceted, coordinated response to complex challenges such as those posed by infectious diseases.

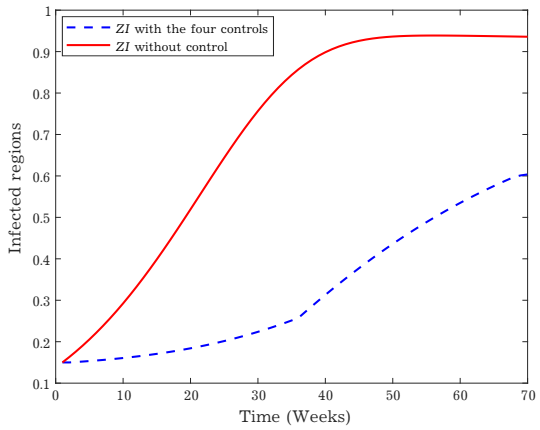


**Fig. 15.** Infected regions without and with four controls.

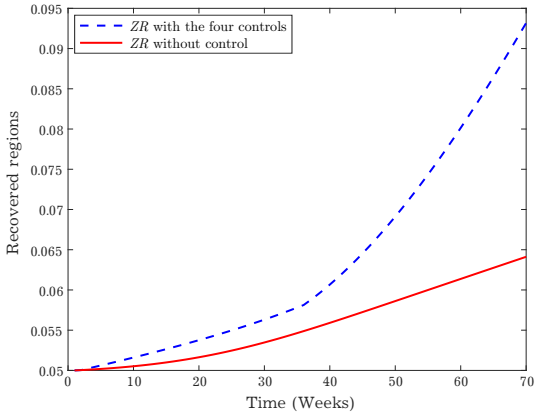


**Fig. 16.** Recovered regions without and with four controls.

## 5. Conclusion

In this article, we presented a discrete mathematical SIRS model designed to capture the dynamics of infectious diseases with regional evolution. We introduced and examined in detail four distinct disease control strategies, addressing both individual and regional aspects of disease management. To determine the most effective control measures, we used Pontryagin's maximum principle.

Our analysis, based on Pontryagin's maximum principle, not only characterized the optimal controls but also provided solid empirical support for the effectiveness of the proposed strategies. Thanks to this approach, we have demonstrated the potential of these strategies to mitigate the spread of infectious diseases, reduce the number of people and regions affected, and increase the number of successful cures.

In conclusion, our research provides valuable insights into the field of disease control by combining mathematical modeling and optimization techniques. The results presented in this paper underline the importance of employing multi-faceted strategies to combat infectious diseases. As we continue to face complex global health challenges, the integration of mathematical models and optimization principles offers a promising avenue for developing effective disease control strategies and improving our ability to respond to public health crises.

[1] Watts S. Epidemics and History. Disease, Power and Imperialism. Yale University Press (1997).

[2] Hays J. N. Epidemics and Pandemics: Their Impacts on Human History. ABC-CLIO (2005).

[3] Kiple K. F. Epidemics and History: Disease, Power, and Imperialism by Sheldon Watts. Journal of Interdisciplinary History. **30** (1), 104–105 (1999).

[4] Sahneh F. D., Scoglio C. Epidemic spread in human networks. 2011 50th IEEE Conference on Decision and Control and European Control Conference. 3008–3013 (2011).

[5] Huber V. Pandemics and the politics of difference: Rewriting the history of internationalism through nineteenth-century cholera. Journal of Global History. **15** (3), 394–407 (2020).

[6] De Sousa J. D., Müller V., Lemey P., Vandamme A. M. High GUD incidence in the early 20th century created a particularly permissive time window for the origin and initial spread of epidemic HIV strains. PloS One. **5** (4), (2010).

[7] Ndaïrou F., Area I., Nieto J. J., Torres D. F. M. Mathematical modeling of COVID-19 transmission dynamics with a case study of Wuhan. Chaos, Solitons & Fractals. **135**, 109846 (2020).

[8] Brauer F., Driessche P., Wu J. Mathematical Epidemiology. Vol. 1945, Springer (2008).

[9] Goffman W. An epidemic process in an open population. Nature. **205** (4973), 831–832 (1965).

[10] Grassberger P. On the critical behavior of the general epidemic process and dynamical percolation. Mathematical Biosciences. **63** (2), 157–172 (1983).

[11] Elliot P., Wakefield J. C., Best N. G., Briggs D. J., et al. Spatial Epidemiology: Methods and Applications. Oxford University Press (2000).

[12] Pybus O. G., Suchard M. A., Lemey P., Bernardin F. J., Rambaut A., Crawford F. W., Gray R. R., Arinaminpathy N., Stramer S. L., Busch M. P., Delwart E. L. Unifying the spatial epidemiology and molecular evolution of emerging epidemics. Proceedings of the national academy of sciences. **109** (37), 15066–15071 (2012).

[13] Okabe Y., Shudo A. A mathematical model of epidemics — A tutorial for students. Mathematics. **8** (7), 1174 (2020).

[14] Chowell G., Sattenspiel L., Bansal S., Viboud C. Mathematical models to characterize early epidemic growth: A review. Physics of Life Reviews. **18**, 66–97 (2016).

[15] Zakary O., Rachik M., Elmouki I. On the analysis of a multi-regions discrete SIR epidemic model: an optimal control approach. International Journal of Dynamics and Control. **5** (3), 917–930 (2017).

[16] Marouane K., Ben Rhila S., Kouidere A., Rachik M. Tikhonov regularization for a spatiotemporal model of the human monkeypox outbreak. Mathematical Modeling and Computing. **10** (3), 37–72 (2023).

[17] Cai Y., Kang Y., Banerjee M., Wang W. A stochastic SIRS epidemic model with infectious force under intervention strategies. Journal of Differential Equations. **259** (12), 7463–7502 (2015).

[18] Vargas-De-León C. On the global stability of *SIS*, *SIR* and *SIRS* epidemic models with standard incidence. Chaos, Solitons & Fractals. **44** (12), 1106–1110 (2011).

[19] Kutyrev V., Popova A. Y., Smolensky V. Y., Ezhlova E., Demina Yu V., Safronov V., Karnaukhov I., Ivanova A., Shcherbakova S. Epidemiological features of new coronavirus infection (COVID-19). Communication 1: Modes of implementation of preventive and anti-epidemic measures. Problems of Particularly Dangerous Infections. **1**, 6–13 (2020).

[20] Wilder-Smith A., Gubler D. J., Weaver S. C., Monath T. P., Heymann D. L., Scott T. W. Epidemic arboviral diseases: priorities for research and public health. The Lancet Infectious Diseases. **17** (3), e101–e106 (2017).

[21] Zakary O., Rachik M., Elmouki I. A multi-regional epidemic model for controlling the spread of Ebola: awareness, treatment, and travel-blocking optimal control approaches. Mathematical Methods in the Applied Sciences. **40** (4), 1265–1279 (2017).

[22] Brownstein J. S., Wolfe C. J., Mandl K. D. Empirical evidence for the effect of airline travel on interregional influenza spread in the United States. PLoS Medicine. **3** (10), e401 (2006).

[23] Viboud C., Miller M. A., Grenfell B. T., Bjørnstad O. N., Simonsen L. Air travel and the spread of influenza: important caveats. PLoS Medicine. **3** (11), e503 (2006).

[24] Liu Z., Li R., Wang X., Shang P. Effects of vehicle restriction policies: Analysis using license plate recognition data in Langfang, China. Transportation Research Part A: Policy and Practice. **118**, 89–103 (2018).

[25] Ji H., Tong H., Wang J., Yan D., Liao Z., Kong Y. The effectiveness of travel restriction measures in alleviating the COVID-19 epidemic: evidence from Shenzhen, China. Environmental Geochemistry and Health. **44**, 3115–3132 (2022).

[26] Chinazzi M., Davis J. T., Ajelli M., Gioannini C., Litvinova M., Merler S., Piontti A. P. Y., Mu K., Rossi L., Sun K., Viboud C., Xiong X., Yu H., Halloran M. E., Longini I. M. Jr., Vespignani A. The effect of travel restrictions on the spread of the 2019 novel coronavirus (COVID-19) outbreak. Science. **368** (6489), 395–400 (2020).

[27] Kupferschmidt K., Wadman M. Delta variant triggers new phase in the pandemic. Science. **372** (6549), 1375–1376 (2021).

[28] Benfatah Y., Khaloufi I., Boutayeb H., Rachik M., Laarabi H. Optimal control for a discrete time epidemic model with zones evolution. Communications in Mathematical Biology and Neuroscience. **2022**, 51 (2022).

[29] Khaloufi I., Benfatah Y., Moutamanni H., Boutayeb H., Rachik M. A discrete mathematical model SEIR with the evolution of the regions. Communications in Mathematical Biology and Neuroscience. **2022**, 120 (2022).

[30] Khaloufi I., Benfatah Y., Lafif M., Ghazaoui A., Rachik M. Optimal control strategy for the administration of the third vaccine dose in the treatment of pandemic COVID-19. Mathematical Modeling and Computing. **10** (3), 841–853 (2023).

[31] Elyoussoufi L., Kouidere A., Kada D., Balatif O., Daouia A., Rachik M. On stability analysis study and strategies for optimal control of a mathematical model of hepatitis HCV with the latent state. Mathematical Modeling and Computing. **10** (1), 101–118 (2023).

[32] Moumine E., Balatif O., Rachik M. Modeling and mathematical analysis of drug addiction with the study of the effect of psychological and biological treatment, Mathematical Modeling and Computing. **10** (3), 935–943 (2023).

[33] Khaloufi I., Benfatah Y., Laarabi H., Rachik M. A scenario to fight monkeypox using a mathematical model. Communications in Mathematical Biology and Neuroscience. **2022**, 99 (2022).

[34] El Bhih A., Benfatah Y., Ben Rhila S., Rachik M., El Alami Laaroussi A. A spatiotemporal prey-predator discrete model and optimal controls for environmental sustainability in the multifishing areas of Morocco. Discrete Dynamics in Nature and Society. **2020**, 2780651 (2020).

[35] Sadki M., Harroudi S., Allali K. Dynamical analysis of an HCV model with cell-to-cell transmission and cure rate in the presence of adaptive immunity. Mathematical Modeling and Computing. **9** (3), 579–593 (2022).

[36] Kada D., Kouidere A., Balatif O., Rachik M. Mathematical modeling of the gaming disorder model with media coverage: optimal control approach. Mathematical Modeling and Computing. **10** (1), 245–260 (2023).

[37] Khaloufi I., Karim M., Rhila S. B., Laarabi H., Rachik M. A mathematical model describing the correlation between smokers and tuberculosis patients. Mathematics in Engineering, Science and Aerospace. **14** (2), 347–361 (2023).

## Дискретна математична модель SIRS з еволюцією регіонів для боротьби з інфекційними захворюваннями

Бенфатах Ю., Халуфі І., Бутаєб Х., Лаарабі Х., Рачік М.

*Лабораторія аналізу, моделювання та симуляції, Касабланка, Марокко, 20670*

У цій статті подано нову математичну модель SIRS, яка описує еволюцію інфекційного захворювання, припускаючи, що просторові основи цієї інфекції також є еволюційними та описуються компартментною моделлю. Запропоновано чотири стратегії боротьби з поширенням хвороби серед окремих людей і регіонів. Для характеристики оптимальних керувань використовується принцип максимуму Понтрягіна, а система оптимальності розв'язується за допомогою ітераційного підходу. Накінець, здійснено чисельне моделювання для підтвердження теоретичного аналізу за допомогою MATLAB.

**Ключові слова:** *математична модель; системи з дискретним часом; модель SIRS; оптимальне керування; заразний вірус; максимум Понтрягіна; еволюція зон.*