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A MATHEMATICAL SIR MODEL ON THE SPREAD OF INFECTIOUS DISEASES CONSIDERING HUMAN IMMUNITY

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Abstract. In this paper, we propose a mathematical model of infection by infectious diseases, taking into account the division of the population according to the criteria of immunity. Our objective is to demonstrate the positive effect of this idea against the different epidemics. We have proposed two strategies to reduce the great human and material losses caused by these diseases, respectively awareness programs on the importance of the exercise of sport and a healthy food to increase human immunity, treatment and health care for people with low immunity. The Pontryagin maximum principle is applied to characterize the optimal controls, and the optimality system is solved using an iterative approach. Finally, numerical simulations are performed to verify the theoretical analysis using MATLAB.

Keywords: mathematical model; human immunity; optimal control; Covid-19; virus; Pontryagin maximum; discrete Time.

2010 AMS Subject Classification: 92C60, 92D30.

1. INTRODUCTION

Each human body requires a protective system that supports its genetic constancy and assures its protection from the penetration of viruses, bacteria, protozoa, poisons and allergens.

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Immunity destroys cells that have mutated due to diseases and other pathological processes [1, 2]. However, the immune system consists of a set of lymphoid organs and tissues that perform crucial functions as production of immune cells, and provision of necessary conditions for cell maturation. The immune system contains central and peripheral organs. The central ones includes the spleen, lymph nodes and lymphoid tissue, liver, blood, and lymph [3, 4].

There are a huge number of different immune cells located through the body including: T-lymphocytes (t-from the name of the organ in which they mature, the thymus): these include T-killers (destroying infected cells of the body and blocking the further spread of infection), T-suppressors (responsible for the duration and strength of immune responses), and T-helpers (stimulate the immune response, transmit information to other immune cells) [5].

B-lymphocytes-synthesize immunoglobulins (antibodies), special proteins that envelop and destroy foreign microorganisms and weaken the danger of toxic substances [6]. Neutrophils and macrophages (provide phagocytosis by capturing and "devouring" foreign agents, and macrophages also transmit information about the microorganisms they destroy to other immune cells [7]. Natural killers (their function is to destroy cells that have mutated under the influence of viruses or a malignant process [8]. Basophils (produce cytokines, special substances that attract the attention of other cells of the immune system during the development of inflammation. Eosinophils-fight allergens and helminths [9].

The state of immunity is provided by inherited and individually formed mechanisms. Innate or non-specific immunity considered as the first line of defense. A genetically fixed ability to resist infection, inherent in every organism from birth, or the first part of the body that creates barriers to the penetration of multiple invaders (viruses, bacteria, parasites and toxins), during the detection of these agents, inherited immunity starts cells activation in order to attack invaders or initiates repairing. Innate immunity's own capabilities are often not enough to clear the body of invading pathogens and contain the development of infection [10, 11]. For this reason, adaptive (acquired immunity or specific immunity) was added to innate immunity; a second line of defense. However, in contrast with the innate, an immune response that is triggered instantly or within the first few hours after contact with a pathogen, an adaptive immune response develops only in response to contact with an invading object. Thus, two branches interact with

each other to eliminate the antigen. It is determined by the involvement of mainly B cells and circulating antibodies, the other direction is determined by the involvement of T cells which do not synthesize and release various cytokines that act on other cells. Hence, Induction protective response to a pathogen includes a complex interaction between cells and molecules of the innate and adaptive immune systems included in the general complex mechanism of immunity [12, 13].

Bacterial infection that are intracellular cause chronic persistent infections, diseases, emerging imitations proceed both in a latent form with reactivation, and in the form bacteriocarrier; their parasitism inside the cell limits access to them. When macrophages deal with bacteria, a different pieces are brought to their surface in order to introduce T-helper cells and B-lymphocytes to the structure of these bacteria. These cells study the pieces of digested bacteria and select the appropriate antibody structure. After that, the B-lymphocyte that successfully coped with the task turns into a plasma cell and begins to synthesize antibodies in large quantities. They enter the bloodstream, spread throughout the body and bind to all the invaded bacteria. In addition, bacteria with stuck antibodies are absorbed by macrophages much faster, which also helps to destroy the infection [14, 15].

The relationship between immunity and infection determines the development of many diseases. For instance, in the dynamic confrontation of living systems-viral infection and immunity, the T cells detect small fragments of viral proteins embedded on the surface of the infected cell. The task of T killer is to identify all infected cells and kill them by apoptosis, yet not to harm neighboring uninfected cells [16]. However, when killer T cells do not detect signs of infection on the surface of infected cells and turn into useless weapons of immunity, NK cells come to the rescue, which have the same apparatus for killing other cells as killer T cells [17]. The expression of genes of DNA-containing viruses occurs in accordance with the central dogma of molecular biology: “DNA-mRNA-protein”. Viral and cellular enzymes, usually non-structural proteins, are involved in the transcription process [18]. By localization, DNA viruses are divided into nuclear (herpesvirus, adenovirus, papovavirus) and cytoplasmic (smallpox virus). In some of the nuclear DNA viruses (herpesvirus, papovavirus), it is possible to integrate the genome into cell chromosomes [19]. In large DNA viruses, polycistronic RNA is

first synthesized, which is then sliced and processed. In cytoplasmic DNA viruses, transcription is performed by viral RNA polymerases. RNA-containing retroviruses first reverse transcribe the genome into DNA, then integrate it into cell chromosomes, and only then transcribe genes [20]. Cytopathic effects in viral infections are diverse, they are determined by both the virus and the cell and are reduced to the destruction of the cell (cytolytic effect), the coexistence of the virus and the cell without death of the latter (latent and persistent infection) and cell transformation. However, the involvement of the body in the infectious process depends on a number of circumstances: the number of dead cells, the toxicity of viruses and cell breakdown products, and the body's reactions, ranging from reflex to immune. The number of dead cells affects the severity of the infection process. For instance, whether the flu affects only nose's cells and trachea, or whether the virus affects the cells of the alveolar epithelium, depends on the severity and outcome of the disease [21, 22].

Although viruses do not form typical toxins, both virions and viral components that accumulate in the affected tissues, leaving the bloodstream, have a toxic effect. The products of cell decay also have an equally toxic effect. In this case, the effect of a viral infection is as non-specific as that of pathogenic organisms that kill cells and cause their autolysis. The entry of toxins into the blood causes a response: fever, inflammation. Fever is mainly a reflex response to the entry of toxic substances into the blood and exposure to the central nervous system [23]. When inflammation occurs, infiltration of affected tissues by macrophages, utilization of decay products, repair and regeneration occur. At the same time, reactions of cellular and humoral immunity develop [24]. When the organism encounters a viral infection, the production of interferon (a soluble factor produced by virus-infected cells that can induce antiviral status in uninfected cells) becomes the fastest response to infection, forming a protective barrier to viruses much earlier than specific immune responses, stimulating cellular resistance, making cells unsuitable for virus reproduction [25].

On the other hand, human immunity fights not only pathogenic bacteria, viruses and fungi; hence, the crucial role of immune system is to recognize each unfamiliar substance as foreign and destroy transformed cells, which can degenerate into malignant tumors. However, failures in the immune system (for genetic or other reasons) lead to the fact that after cell division in

our body, daughter cells have an incorrect structure and can become cancerous. An overgrown tumor becomes insensitive to the body's attacks and not only successfully avoids destruction, but also actively "reprograms" protective cells to meet its own needs. Cancer cells are insidious, they can acquire different types of protection: if the cancer cells does not produce enough foreign substances, the immune system does not notice it. Some tumors can produce substances that inhibit the work of immune cells, and sometimes the leading role belongs to the tumor microenvironment the cells and molecules that surround cancer cells. In order to deal with the tumor, the immune system must be activated, or provided with necessary components [26, 27]. The cancer treatment is called immunotherapy including a several varieties of it: Monoclonal antibodies considered as artificial analogues of the immune system. Each of them has a specific target a specific substance produced by cancer cells [28]; Checkpoint inhibitors is another type of immunotherapy, a control points and substances that suppress the immune system, they are needed in order to prevent the immune system from attacking healthy tissues, because cancer cells often use checkpoints for masking purposes, and these inhibitors remove this block after which the tumor is attacked [29]; Modulators of the immune system or medications that usually include interferons, interleukins, and growth factors, and their mission is to improve the functioning of the immune system in general not against any specific components of cancer cells [30]; the last type is cellular immunotherapy a direction that has shown success in some studies. In this technique, immune cells are taken from the patient's peripheral blood and treated with cytokines to increase their functional activity. Then the cells are returned to the patient, which leads to activation of various parts of the immune system, strengthening the immune response against a malignant tumor, overcoming existing tumor immunosuppression, reducing the severity of pain syndrome, and improving the quality of life of the cancer patient [31, 32].

Another example of immune diseases is HIV (human immunodeficiency virus) that attacks the body's immune system and could lead to AIDS (acquired immunodeficiency syndrome). After the penetration of the inherited HIV into the cell, the process of reverse transcription occurs (the process of providing double-standard DNA based on information in single-standarded RNA). This is because the the viral genome is written as RNA, and the human genome is written as DNA, the virus aims to write itself into the genome of the cell, because it controls the latter.

In addition, if the immune system has the means to recognize viral proteins and RNA, then the immune system cannot recognize the DNA integrated into the cell genome. Reverse transcription is performed by the viral protein, RNA is very unstable contrasted to DNA [33, 34].

Therefore, HIV is characterized by a huge mutation rate-tens of thousands of times faster than in humans. After reverse transcription, the viral genome is inserted into the human genome by the viral protein integrase. Having penetrated the genome, the virus can sit in it for several years without showing itself. Basically, the virus begins to multiply in activated (dividing) T-lymphocytes, although it can also work a little in a non-dividing cell. The is the main reason why HIV is incurable taking into consideration that HIV infects mainly T-helper cells, once the cell is activated the virus attacks it and destroy it. Thus, after the copy of the virus in the cell genome begins to act, the familiar gp41 and gp120 proteins appear on its surface, and the remaining viral proteins and viral RNA appear in the cytoplasm. And after a while, more and more copies of HIV begin to bud off from the infected cell [35, 36]. Since the human body loses its ability to defend itself in the case of HIV infection, the only treatment is highly active antiretroviral therapy, which should be given for life. Only the development of effective vaccines and HIV prevention methods can solve this problem. Unlike traditional antiviral vaccines, which prevent the development of the disease but allow the infection to start, the HIV vaccine should completely prevent it. In other words, the prevention of HIV infection requires the presence of neutralizing antibodies that block the virus from entering the cells [37, 38].

In the next section, we will give some necessary mathematical preliminaries that will be used in this paper.

2. Presentation of the Model

The world has experienced several infectious epidemics throughout history that have left behind many deaths. Despite scientific and medical development, these disease outbreaks still pose a serious threat to human societies and global economies, especially for people with low immunity. We propose a simple SIR model in which we divide the first two compartments into two subcompartments.

The susceptible compartment is decomposed into two compartments.

(S) The compartment of susceptible people with low immunity.

(T) The susceptible compartment with normal immunity.

The compartment of the infected is decomposed into two compartments.

(J) The compartment of the infected with low immunity.

(K) The compartment of the infected with normal immunity.

(R) The compartment of recovered individuals.

We obtain the following model

$$
(1)\qquad \begin{cases} S_{i+1} = \Lambda_1 + (1-\mu-\theta_1)\,S_i - (\alpha_1 J_i + \alpha_2 K_i)\,S_i + \theta_2 T_i \\ T_{i+1} = \Lambda_2 + (1-\mu)\,T_i - (\beta_1 J_i + \beta_2 K_i)\,T_i + \theta_1 S_i - \theta_2 T_i \\ J_{i+1} = (1-\mu-\delta_1-r_1)\,J_i + (\alpha_1 J_i + \alpha_2 K_i)\,S_i \\ K_{i+1} = (1-\mu-\delta_2-r_2)\,K_i + (\beta_1 J_i + \beta_2 K_i)\,T_i \\ M_{i+1} = \delta_1 J_i + \delta_2 K_i + \mu(S_i + T_i + J_i + K_i + R_i) \\ R_{i+1} = (1-\mu)\,R_i + r_1 J_i + r_2 K_i \end{cases}
$$

where $S_0 > 0, T_0 > 0, J_0 > 0, K_0 > 0$, $M_0 > 0$ and $R_0 > 0$.

TABLE 1. compartments meaning

Compartment	Meaning
S_i	susceptible individuals without immunity
T_i	susceptible individuals with immunity
J_i	infected individuals without immunity .
K_i	Infected individuals with immunity .
M_i	Dead individuals.
R_i	recovered individuals.

3. OPTIMAL CONTROL

3.1. Presentation of the controls. In order to avoid the dangers of epidemics that affect societies, and considering that people with normal immunity are more protected from the complications of epidemics than individuals with low immunity, we suggest two control strategies.

The first strategy (control u) is to motivate people with low immunity to do things that increase their immunity, such as practicing sports, eating healthy, and not smoking.

The second strategy (control v) is to provide additional health care to patients with low immunity and to prepare recovery rooms for those with severe complications.

$$
\begin{cases}
S_{i+1} = \Lambda_1 + (1-\mu-\theta_1-u_i)\,S_i - (\alpha_1 J_i + \alpha_2 K_i)\,S_i + \theta_2 T_i \\
T_{i+1} = \Lambda_2 + (1-\mu)\,T_i - (\beta_1 J_i + \beta_2 K_i)\,T_i + (\theta_1+u_i)\,S_i - \theta_2 T_i \\
J_{i+1} = (1-\mu-\delta_1-r_1-v_i)\,J_i + (\alpha_1 J_i + \alpha_2 K_i)\,S_i \\
K_{i+1} = (1-\mu-\delta_2-r_2)\,K_i + (\beta_1 J_i + \beta_2 K_i)\,T_i \\
M_{i+1} = \delta_1 J_i + \delta_2 K_i + \mu(S_i + T_i + J_i + K_i + R_i) \\
R_{i+1} = (1-\mu)\,R_i + r_1 J_i + r_2 K_i + v_i J_i
\end{cases} \tag{2}
$$

3.2. Objective functional. The objective function J is described as follows

$$
J(u,v) = \alpha J_N + \beta M_N + \sum_{i=1}^{N-1} \left(\alpha J_i + \beta M_i + \frac{1}{2} A {u_i}^2 + \frac{1}{2} B {v_i}^2 \right) \tag{3}
$$

3.3. Sufficient conditions. The sufficient condition of existence of an optimal control (u^*, v^*) for the problem 2 is derived from the following theorem.

Theorem 3.1. *There exists an optimal control $(u^*, v^*) \in \mathscr{U} \times \mathscr{V}$ such that*

$$
J(u^*, v^*) = min\{J(u,v)/u \in \mathscr{U}, v \in \mathscr{V}\}
$$

subjected to the control system 2 with initial conditions.

Proof. Since the system parameters are bounded and there is a finite number of time steps, that is S, T, J, K, M and R are uniformly bounded for all $(u,v,)$ in the control set $\mathscr{U} \times \mathscr{V}$. Thus $J(u,v)$ is also bounded for all $(u,v) \in \mathscr{U} \times \mathscr{V}$.

Which implies that $\inf_{(u,v)\in \mathscr{U}\times\mathscr{V}} J(u,v)$ is finite, and there exists a sequence $(u^n, v^n) \in \mathscr{U} \times \mathscr{V}$ such as that

$$
\lim_{n\to+\infty} J(u^n, v^n) = \inf_{(u,v)\in\mathscr{U}\times\mathscr{V}} J(u,v)
$$

and corresponding sequences of states S^n, T^n, J^n, K^n,M^n and R^n. Since there exists a finite number of uniformly bounded sequences such that $(u^*, v^*) \in \mathscr{U} \times \mathscr{V}$ and S^*, T^*, J^*, K^*,M^* and

R^* such as, in a sequence,$(u^n, v^n) \to (u^*, v^*)$, $S^n \to S^*$, $T^n \to T^*$, $J^n \to J^*$, $K^n \to K^*$, $M^n \to M^*$, and $R^n \to R^*$. Finally, due to the finite dimensional structure of the system (2)and the objective function $J(u,v)$, the control (u^*, v^*) is an optimal control with corresponding states S^*, T^*, J^*, K^*, M^* and R^*. Which complete the proof. □

3.4. Necessary conditions. We now have the Hamiltonian $\mathscr{H}$ in time step i, given by :

(4)

$$\mathscr{H}_i = \alpha J_i + \beta M_i + \frac{1}{2}Au_i{}^2 + \frac{1}{2}Bv_i{}^2 + \xi^1_{i+1}(\Lambda_1 + (1-\mu-\theta_1-u_i)S_i - (\alpha_1 J_i + \alpha_2 K_i)S_i + \theta_2 T_i)$$
$$+ \xi^2_{i+1}(\Lambda_2 + (1-\mu)T_i - (\beta_1 J_i + \beta_2 K_i)T_i + (\theta_1+u_i)S_i - \theta_2 T_i) + \xi^3_{i+1}((1-\mu-\delta_1-r_1-v_i)J_i +$$
$$+ (\alpha_1 J_i + \alpha_2 K_i)S_i) + \xi^4_{i+1}((1-\mu-\delta_2-r_2)K_i + (\beta_1 J_i + \beta_2 K_i)T_i)$$
$$+ \xi^5_{i+1}(\delta_1 J_i + \delta_2 K_i + \mu\,(S_i + T_i + J_i + K_i + R_i)) + \xi^6_{i+1}((1-\mu)R_i + r_1 J_i + r_2 K_i + v_i J_i)$$

Theorem 3.2. *Given optimal controls u^*, v^* and solutions S^*, T^*, J^*, M^* and R^* of corresponding state system 2, there exists ξ_i^j, $i = 1...N$, $j = 1,2,\cdots,6$, the adjoint variables that satisfy the following equations*

$$\Delta\xi^1_{i+1} = -[\xi^1_{i+1}(-\alpha_1 J_i - \alpha_2 K_i - \mu - \theta_1 - u_i + 1) + \xi^2_{i+1}(u_i + \theta_1) + \xi^3_{i+1}(\alpha_1 J_i + \alpha_2 K_i) + \xi^5_{i+1}\mu]$$
$$\Delta\xi^2_{i+1} = -[\theta_2\,\xi^1_{i+1} + \xi^2_{i+1}(-\beta_1 J_i - \beta_2 K_i - \mu - \theta_2 + 1) + \xi^4_{i+1}(\beta_1 J_i + \beta_2 K_i) + \xi^5_{i+1}\mu]$$
$$\Delta\xi^3_{i+1} = -[\alpha - \xi^1_{i+1}\alpha_1 S_i - \xi^2_{i+1}\beta_1 T_i + \xi^3_{i+1}(\alpha_1 S_i - \mu - r_1 - \delta_1 - v_i + 1 + \xi^4_{i+1}\beta_1 T_i)$$
$$+ \xi^5_{i+1}(\mu + \delta_1) + \xi^6_{i+1}(r_1 + v_i)]$$
$$\Delta\xi^4_{i+1} = -[-S_i\alpha_2\,\xi^1_{i+1} - \xi^2_{i+1}\beta_2 T_i + \xi^3_{i+1}\alpha_2 S_i + \xi^4_{i+1}(\beta_2 T_i - \mu - r_2 - \delta_2 + 1) + \xi^5_{i+1}(\mu + \delta_2) + \xi^6_{i+1}r_2]$$
$$\Delta\xi^5_{i+1} = -[\beta + \xi^5_{i+1}]$$
$$\Delta\xi^6_{i+1} = -[\xi^5_{i+1}\mu + \xi^6_{i+1}(1-\mu)]$$

with the conditions of transversality at time N

$$\xi^1_N = 0, \xi^2_N = 0, \xi^3_N = \alpha, \xi^4_N = 0, \xi^5_N = \beta, \xi^6_N = 0, \xi^6_N = 0$$

In addition, for $i = 0,1,\cdots,N-1$ we obtain the optimal control (u^*, v^*) as

$$u_i = min\left\{max\left\{u_{min}, \frac{\xi^1_{i+1}S_i - \xi^2_{i+1}S_i}{A}\right\}, u_{max}\right\} \tag{5}$$

$$v_i = min\left\{max\left\{v_{min}, \frac{\xi_{i+1}^3 J_i - \xi_{i+1}^6 J_i}{B}\right\}, v_{max}\right\} \tag{6}$$

Proof. The Hamiltonian $\mathscr{H}_i$ k at time step i is obtained by 4. For $i = 1, \cdots, N-1$, the adjoint equations and transversality conditions can be derived by using the discrete-time Pontryagin maximum principle given in [39, 40], as follows

$$\begin{aligned}
\Delta\xi_i^1 &= -\frac{\partial \mathscr{H}_i}{\partial S_i}, \qquad \xi_N^1 = \frac{\partial\left(\alpha J_N + \beta M_N\right)}{\partial S_N} = 0 \\
\Delta\xi_i^2 &= -\frac{\partial \mathscr{H}_i}{\partial T_i}, \qquad \xi_N^2 = \frac{\partial\left(\alpha J_N + \beta M_N\right)}{\partial T_N} = 0 \\
\Delta\xi_i^3 &= -\frac{\partial \mathscr{H}_i}{\partial J_i}, \qquad \xi_N^3 = \frac{\partial\left(\alpha J_N + \beta M_N\right)}{\partial J_N} = \alpha \\
\Delta\xi_i^4 &= -\frac{\partial \mathscr{H}_i}{\partial K_i}, \qquad \xi_N^4 = \frac{\partial\left(\alpha J_N + \beta M_N\right)}{\partial K_N} = 0 \\
\Delta\xi_i^5 &= -\frac{\partial \mathscr{H}_i}{\partial M_i}, \qquad \xi_N^5 = \frac{\partial\left(\alpha J_N + \beta M_N\right)}{\partial M_N} = \beta \\
\Delta\xi_i^6 &= -\frac{\partial \mathscr{H}_i}{\partial R_i}, \qquad \xi_N^6 = \frac{\partial\left(\alpha J_N + \beta M_N\right)}{\partial R_N} = 0
\end{aligned}$$

For $i = 1, \cdots, N-1$, the optimal controls (u^*, v^*) can be determined from the optimality conditions

$$\begin{aligned}
\frac{\partial \mathscr{H}}{\partial u_i} &= Au_i - \xi_{i+1}^1 S_i + \xi_{i+1}^2 S_i = 0 \\
\frac{\partial \mathscr{H}}{\partial v_i} &= Bv_i - \xi_{i+1}^3 J_i + \xi_{i+1}^6 J_i = 0
\end{aligned} \tag{7}$$

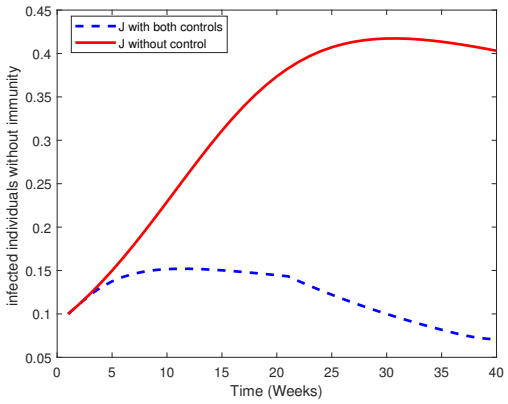
Thus, we obtain

$$\begin{aligned}
u_i &= = \frac{\xi_{i+1}^1 S_i - \xi_{i+1}^2 S_i}{A} \\
v_i &= \frac{\xi_{i+1}^3 J_i - \xi_{i+1}^6 J_i}{B}
\end{aligned} \tag{8}$$

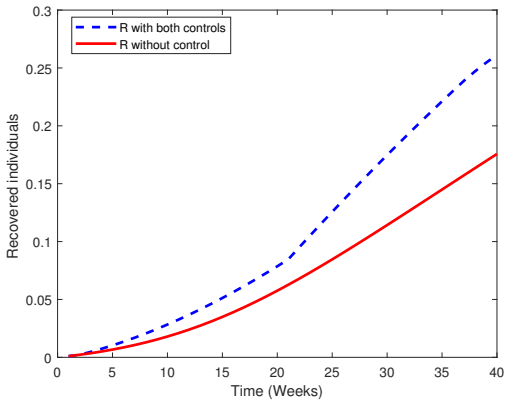
By the bounds in $\mathscr{U}$ *and* $\mathscr{V}$ of the controls, it is simple to obtain u and v in the form of 5 and 6. □

4. Numerical Simulation and Discussion

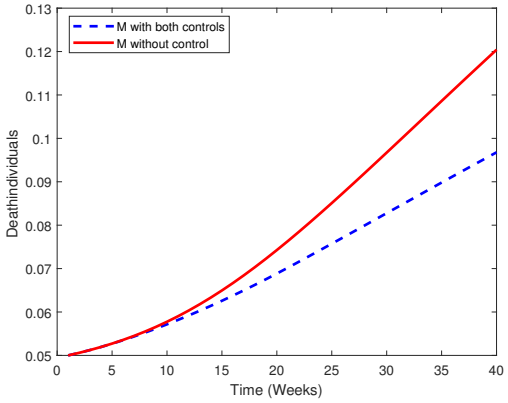
In this section, we present numerical simulations for the above given optimization problem. By writing the program in MATLAB , we simulate the work using various data. The optimization systems are solved using a discrete iterative process that converges after a proper test similar to FBSM. First, the state system is solved with the initial assumption forward in time, and then the adjoint system is solved backward in time due to transversality conditions. Next, we update our optimal control values with the state and co-state resources derived in the previous steps. Finally, we run the above steps until the standard tolerance is reached.



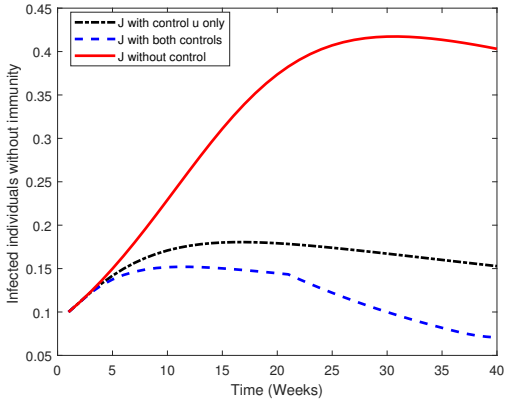
(A) The number of infected individuals without immunity



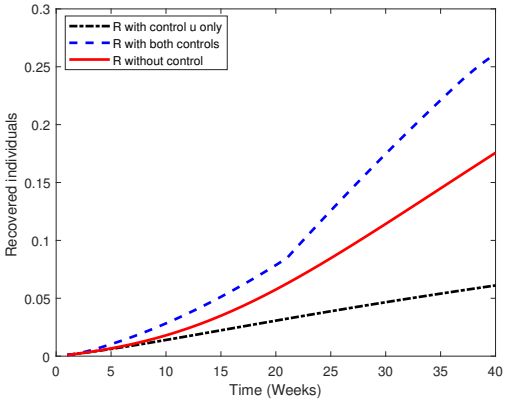
(B) The number of recovered individuals



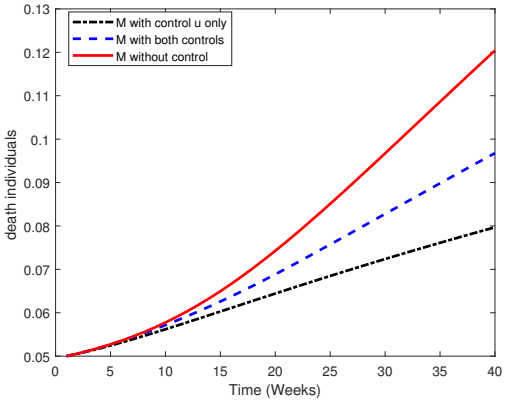
(C) The number death individuals



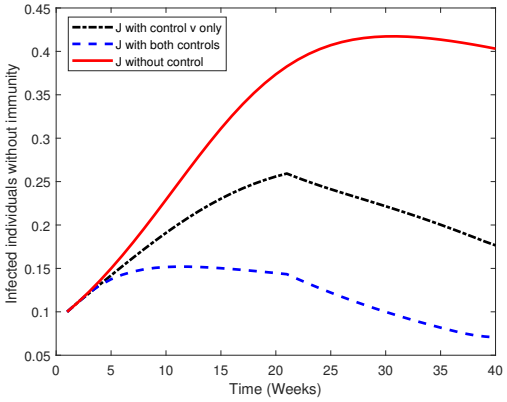
(D) The number of infected individuals without immunity



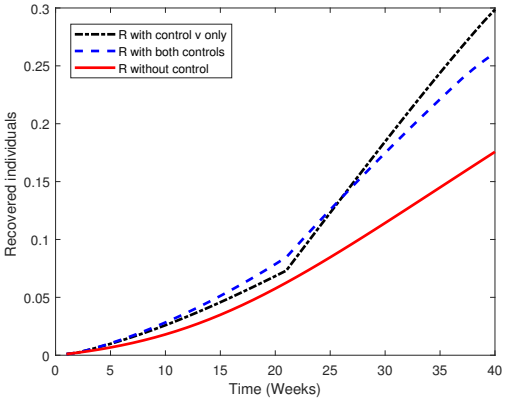
(E) The number of recovered individuals



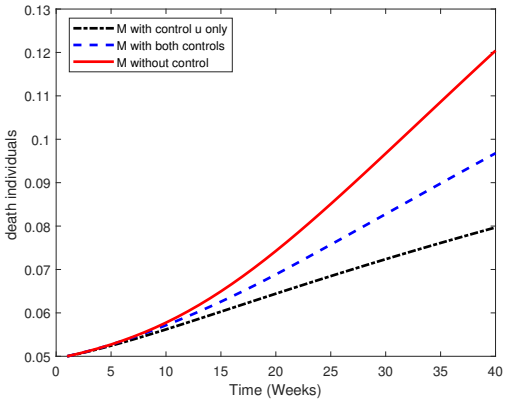
(F) The number death individuals



(G) The number of infected individuals without immunity



(H) The number of recovered individuals



(I) The number death individuals

4.1. Strategy one:Raising awareness of the importance of sports and a healthy food for people with low immunity. We use only optimum control u.

In this strategy, we conduct awareness programs on the importance of sports and healthy eating to increase people's immunity, through television and online advertisements, as well as through awareness campaigns in hospitals and schools.Figures (1d) and (1f) show the effectiveness of this plan in controlling the decrease in the number of infected with low immunity and significantly reducing the number of deaths, but it does not work well in increasing the number of recovered patients (see Figure (1e)).

4.2. Strategy two: Treatment. We use only optimum control v.

As immunocompromised people are considered the most vulnerable to the health problems of any viral disease, we implement the strategy based on the medical care of this category and the increase in the number of medical beds allocated to them. From the figure (1h) we notice that this plan has improved the previous strategy, in increasing the number of recovered.

4.3. Strategy three:Awareness and treatment. We combine the optimal controls u and v.

In this new strategy, the two optimal commands u(t) and v(t) are applied at the same time to improve the statistical performance of both proposed strategies. Based on Figures (1a) , (1b)and (1c), after applying both strategies, we obtain the suggested results, with a decrease in the number of infected people with low immunity, and an increase in the number of people recovering from the disease. Thus, the decrease in the number of people dying from the disease.

In the following section, we will present a conclusion of our work.

5. CONCLUSION

People with low immunity are the groups most affected by various epidemics and infectious diseases, so we proposed a mathematical model SIR that takes into account the division of the compartment of susceptible and infected into two parts with immunity and without immunity. We have proposed two control strategies, the first is an awareness program on the importance of sport and a healthy diet to increase human immunity, the second treatment and health care for people with low immunity. We applied the control theory results and successfully obtained

the characterizations of the optimum controls. The numerical simulation of the obtained results demonstrated the effectiveness of the proposed control strategies.

CONFLICT OF INTERESTS

The author(s) declare that there is no conflict of interests.

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