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A SCENARIO TO FIGHT MONKEYPOX USING A MATHEMATICAL MODEL

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Abstract. In this paper, we consider a scenario to attack the infectious disease monkeypox taking advantage of the experience we have with the Corona epidemic to reduce its negative effects on humanity and the world economies. This by proposing three control strategies. Pontryagin's maximum principle is applied in order to characterize the optimal controls, and the optimality system is resolved using an iterative approach. At last, numerical simulations are executed to verify the theoretical analysis using MATLAB.

Keywords: mathematical model; optimal control; monkeypox; contagious virus; Pontryagin maximum.

2010 AMS Subject Classification: 92C60.

1. INTRODUCTION

The recent outbreak of monkey pox virus has now occurred in various countries and almost on continents [1]; humanity froze in anticipation of the next lock-down; the (MPXV) infectious disease can be transmitted by airborne droplets, began to spread around the world, and considered as a rare viral zoonotic disease that is transmitted to humans from animals [2]. The monkey pox virus is a relative of natural black smallpox, belongs to the same genus orthopoxvirus,

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hence, is characterized by a lighter course of the disease and relatively low mortality. The probability of a severe course is higher in children, pregnant women, and people suppressed immune systems [3]. The virus was first detected in laboratory monkeys in Denmark, where Singapore's crab-eating macaques were kept together with rhesus monkeys-hence the name "monkey pox". The first human case was reported in 1970 in the Republic of the Congo. The first outbreak outside of Africa occurred in the United States in 2003. Approximately 800 exotic animals from Ghana were brought to the country, including hamster rats infected with this virus, and prairie dogs were infected from them, which were sold as pets in Texas [4, 5].

Europe faced the disease in 2018, when the UK recorded 7 cases of monkeypox in people who returned from exotic countries [6]. The latest reports of new infections in various countries of the world with monkey pox appeared in May 2022. And although there are just over a hundred confirmed cases and a slightly smaller number of people with suspected monkeypox, WHO has started talking about a new pandemic and the need to vaccinate the population [7]. Nowadays, the disease is spreading in an unusual way for many cases of monkey pox are recorded in different parts of the world, although most of them are in Europe. Portugal, Spain, and the United Kingdom are now the leaders in the number of cases. other records concern Australia, Belgium, Canada, France, Germany, Italy, the Netherlands, Sweden, and the United States. There were also reports of cases in Switzerland and Greece. At the end of May, the number of countries outside Africa where cases were detected rose 17 [8, 9].

Monkey pox is transmitted to humans from small rodents (squirrels, rats, mice) and primates through bites and direct contact with the animal. The causative agent of monkey pox virus is believed to have a moderate transmissibility (ability to infect) among humans. Human to human transmission of the virus requires very close contact with someone who is already sick [10]. In most cases, infection occurs by airborne droplets, through biological fluids (semen, saliva, blood), as well as contact with contaminated materials (bed linen, clothing), if the integrity of the skin is violated. The risk of transmission during sexual intercourse is considered high, and low in all other cases [11]. However, the monkeypox is not very contagious: the R_0 index (the number of people who can be infected by one carrier) for it ranges from 1 to 2. For comparison, in the omicron variant of coronavirus, the R_0 index could reach 12. The virus is also poorly

transmitted by airborne droplets [12]. Most cases are related to sexual contact and contact with body fluids [13]. Unlike coronavirus, monkey does not form aerosols (small droplets that create an air suspension and can travel long distances). In addition, monkeypox is not a disease that is transmitted during the asymptomatic phase, and even if it is mutated, there is already a proven vaccine that can be modified if necessary. And new variants of the monkeypox virus may not appear as quickly as in COVID-19, due to the biology of the virus itself. Thus, it should be borne in mind that the virus may at some point mutate in such a way that it begins to be effectively transmitted from person to person. This probability is what causes the greatest concern [14, 2].

According to the WHO, there are two main branches of strains: the West African which is considered as milder, with a mortality rate of 3.6% of cases, and the Central African (Congolese), considered more severe, with a fatality rate of 10.6%. The pathogenesis of monkeypox is slightly understood. It is known that after entering the human body, the virus stays for a long time in the regional lymph nodes, where it multiplies, and then spreads hematogenically and lymphogenically through the organs. The virus has tropicity to the epithelium, increases the production of pro-inflammatory interleukin-10; Pathohistological changes are characterized by epidermal necrosis with progressive epithelial hyperplasia, expansion of the boundaries of necrotic changes; by the time the sebaceous glands and hair follicles are destroyed [15, 16].

The incubation period is 7-21 days, usually about two weeks [17]. The disease begins acutely with headache, weakness, chills, fever up to 39.5-40°C. The lymph nodes located near the site of the pathogen's penetration become inflamed, increase in size, and become painful when touched. Due to severe intoxication, there may be a decrease in appetite, nausea, vomiting. On day 3-4, the fever subsides to 38.5°C or less, a rash appears on the face, feet, palms, and then the trunk. Monkey pox rashes go through several stages sequentially [18]. First, a spot with a diameter of up to 1 cm is formed, which turns into a bump. The next stage is the appearance of a bubble with transparent, and then cloudy contents, then a crust is formed with the outcome on the scar. During the last three stages, the temperature rises again to 39°C, chills are observed, and the state of health worsens. Due to the presence of rashes on the pharyngeal mucosa, there is a cough, a feeling of rawness, and dryness in the throat [19, 20]. Up to 70% of patients have a rash on the oral mucosa, which is manifested by pronounced discomfort when chewing food,

increased salivation. In 30% of cases, the genitals are affected, in 20% the eyelids, which is accompanied by sharp pain in the genitals and eyes, respectively. With the addition of pyogenic flora, an increase in fever, a decrease in blood pressure, an increased heart rate, and impaired consciousness are detected-harbingers of infectious and toxic shock [20, 21].

The main complications of monkeypox are purulent-necrotic lesions. These include bronchopneumonia, which is characterized by the formation of necrotic foci, respiratory distress syndrome and high mortality. Rare complications include damage to the eyes and gastrointestinal tract, sepsis. Post-infection corneal scarring can lead to blindness. In the second week of the disease, profuse diarrhea, vomiting and progressive dehydration may occur. Septic conditions were recorded with a large number of rash elements (over 4500) [22, 2].

Most monkeypox infections recover with no medical treatment, especially gastrointestinal symptoms (e.g., diarrhea, vomiting) will require oral/intravenous rehydration to reduce gastrointestinal fluid losses [23]. Etiotropic antiviral drugs may be effective in treating monkeypox patients, yet these drugs were approved for the management of smallpox implicated on animal models. Studies concerning these drugs have been conducted in humans, however the efficiency of these agents has not been thoroughly developed [22]. The main measures in the treatment of monkeypox are infusion and oral detoxification, anesthesia and other means of symptomatic therapy. The use of antibiotics is indicated in the presence of purulent complications. It is recommended to irrigate the mucous membranes after each meal and toilet visit with antiseptic properties. Wet bandages should be used to prevent scarring, especially on the face. However, the use of cidofovir, originally licensed for the treatment of cytomegalovirus retinitis in HIV infection, is currently in clinical trials. The development of oral modifications worked by inhibiting the viral DNA polymerase for the treatment of this infection is underway in New York and North Carolina. These drugs have completed the second phase of human clinical trials, proving their safety [24, 25].

Multiple studies believed that those who were vaccinated against smallpox in childhood can be protected from monkey pox, since the vaccine gave a sterile immunity, that is, the vaccinated person ceased to be a source of new viruses and vaccination gave lifelong immunity. According to WHO, vaccination against smallpox is 85% effective against smallpox infection in monkeys.

The two viruses are similar, so the immune response formed as a result of using the smallpox vaccine is effective against monkeypox [26]. Nevertheless, it is impossible to talk about significant collective immunity, since the vast majority of people under the age of 40 are not vaccinated with the smallpox vaccine. However, there are two smallpox vaccine available in the US: ACAM2000 and Jynneos, the first one was approved by the US Food and Drug Administration (FDA) in 2007; this vaccine is based on a live smallpox virus that can be transmitted to people who are in close contact with the vaccinated person, and the common side effects include lymphadenitis, malaise, fever, myalgia, and headache. These effects are less common in re-vaccinated individuals than in those who received the vaccine for the first time. The Jynneos vaccine was approved by the FDA in 2019, and made on the basis of a weakened virus. It is intended for the prevention of smallpox and monkeypox in adults aged 18 years and older. The latter is suitable for those who cannot be vaccinated with ACAM2000 due to contraindications, for example, atopic dermatitis, weak immunity, as well as during breastfeeding or pregnancy. This is the only FDA-approved monkey pox vaccine that is intended not only for military personnel, but also for civilians [27, 28, 29]. The manuscript is arranged as follows. In Section 2, we briefly describe our proposed model. In section 3, we provide some basic properties of the model. In section 4 we introduce the problem with control, and we give some results on the existence and characterization of the optimal control using Pontryagin's maximum principle. The numerical simulation on MATLAB confirms the theoretical results in section 5. Finally we conclude our work in section 6.

2. MATHEMATICAL MODEL

We consider a mathematical model $SEIQHI^cR$ that describe the dynamic of transmission of Monkeypox virus in a given population. We divide the population into seven compartments.

2.1. Model description. The graphical representation of the proposed model is shown in Figure 1.

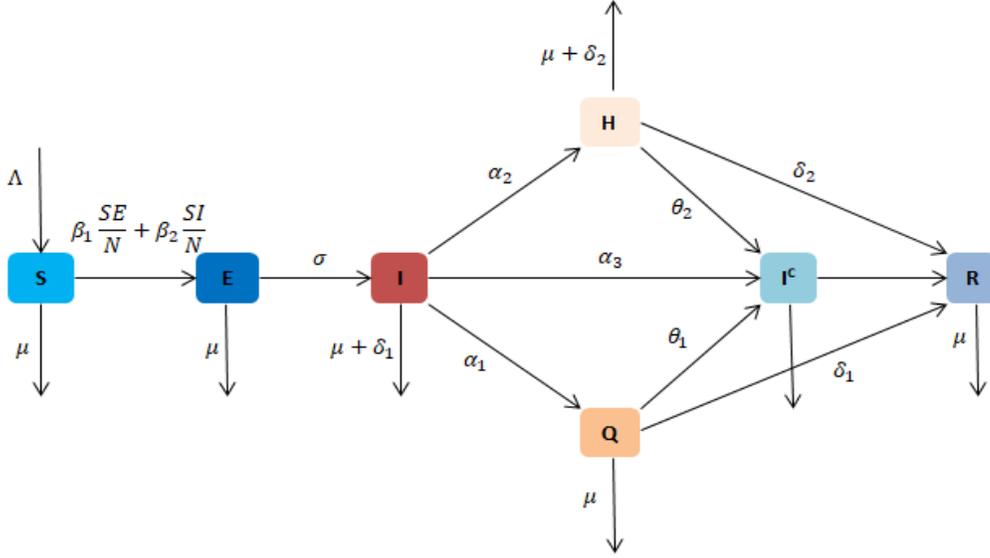


FIGURE 1. Model description.

Compartment S is representing the number of individuals who may be infected with the Monkeypox. The compartment S is increasing by Λ and decreasing by the amount μ (natural mortality), $\beta_1 \frac{SI}{N}$ (The number of people who were infected with the virus by contact with the infected patients) and $\beta_2 \frac{SE}{N}$ (The number of people who were infected with the virus by contact with the exposed patient)

$$(1) \quad \frac{dS(t)}{dt} = \Lambda - \beta_1 \frac{S(t)I(t)}{N} - \beta_2 \frac{S(t)E(t)}{N} - \mu S(t)$$

Compartment E is representing the number of individuals infected by the virus but not yet symptomatic (according [11] to the disease necessarily passes through this phase). The compartment E is increasing by $\beta_1 \frac{SI}{N}$ and $\beta_2 \frac{SE}{N}$. This Compartment decreasing by the amount μ (natural mortality) and the the amount σE (the rate of normally infected people with symptoms)

$$(2) \quad \frac{dE(t)}{dt} = \beta_1 \frac{S(t)I(t)}{N} + \beta_2 \frac{S(t)E(t)}{N} - (\mu + \sigma) E(t)$$

Compartment I is representing the number of individuals infected with symptoms, increasing by the amount σE and decreasing by the amount μ , δ_1 (the induced death rate of Monkeypox), α_1 (The rate of infected people is usually with symptoms and they need quarantined at home),

α_2 (The rate of people who developed the virus rapidly) and α_3 (The rate of people suffering serious complications after the virus has destroyed a large part of their lungs)

$$(3) \quad \frac{dI(t)}{dt} = \sigma E(t) - (\alpha_1 + \alpha_2 + \alpha_3 + \delta_1 + \mu)I(t)$$

Compartment Q the number of people who have been quarantined in their homes, increasing by $\alpha_1 I$ and decreasing by the amount μ , γ_1 and θ_1 .

$$(4) \quad \frac{dQ(t)}{dt} = \alpha_1 I(t) - (\theta_1 + \gamma_1 + \mu)Q(t)$$

Compartment H the number of people who have been quarantined in the hospital. This compartment increase by $\alpha_2 I(t)$ and $\theta_1 Q$ and decrease by the amount μ , δ_2 (The rate of people dying under quarantine in hospitals), γ_2 (The rate of people with a quarantine in hospitals recovered from the virus) and θ_2 (The rate of hospitalized people suffering serious complications after the virus has destroyed a large part of their lungs)

$$(5) \quad \frac{dH(t)}{dt} = \alpha_2 I(t) + \theta_1 Q(t) - (\theta_2 + \gamma_2 + \delta_2 + \mu)H(t)$$

Compartment I^c : the number of patients with severe complications after the virus destroyed a large part of their lung, decreasing by the amount μ , δ_3 and γ_3 . In addition, this compartment is increasing by $\alpha_3 I$ and $\theta_2 H$

$$(6) \quad \frac{dI^c(t)}{dt} = \alpha_3 I(t) + \theta_2 H(t) - (\gamma_3 + \delta_3 + \mu)I^c(t)$$

Compartment R the number of individuals who have recovered, decreasing by the amount μ and increasing by $\gamma_1 Q$, $\gamma_2 H$ and $\gamma_3 I^c$

$$(7) \quad \frac{dR(t)}{dt} = \gamma_1 Q(t) + \gamma_2 H(t) + \gamma_3 I^c(t) - \mu R(t)$$

we obtain the following model

$$(8) \quad \left\{ \begin{array}{l} \frac{dS(t)}{dt} = \Lambda - \beta_1 \frac{S(t)I(t)}{N} - \beta_2 \frac{S(t)E(t)}{N} - \mu S(t) \\ \frac{dE(t)}{dt} = \beta_1 \frac{S(t)I(t)}{N} + \beta_2 \frac{S(t)E(t)}{N} - (\mu + \sigma) E(t) \\ \frac{dI(t)}{dt} = \sigma E(t) - (\alpha_1 + \alpha_2 + \alpha_3 + \delta_1 + \mu) I(t) \\ \frac{dQ(t)}{dt} = \alpha_1 I(t) - (\theta_1 + \gamma_1 + \mu) Q(t) \\ \frac{dH(t)}{dt} = \alpha_2 I(t) + \theta_1 Q(t) - (\theta_2 + \gamma_2 + \delta_2 + \mu) H(t) \\ \frac{dI^C(t)}{dt} = \alpha_3 I(t) + \theta_2 H(t) - (\gamma_3 + \delta_3 + \mu) I^C(t) \\ \frac{dR(t)}{dt} = \gamma_1 Q(t) + \gamma_2 H(t) + \gamma_3 I^C(t) - \mu R(t) \end{array} \right.$$

where $S(0) \geq 0, E(0) \geq 0, I(0) \geq 0, Q(0) \geq 0, H(0) \geq 0, I^C(0) \geq 0$ and $R(0) \geq 0$, are the initial states.

Parameter	Physical interpretation
Λ	The incidence of susceptible
μ	Mortality natural
β_1	The rate of people who were infected by contact with exposed
β_2	The rate of people who were infected by contact with infected.
σ	The rate of normally infected people with symptoms.
α_1	The rate of infected people is usually with symptoms and they need quarantined at home .
α_2	The rate of people who developed the virus rapidly.
α_3	The rate of people suffering serious complications after the virus has destroyed a large part of their lungs.
γ_1	The rate of people having a quarantine at home recovered from the virus
γ_2	The rate of people with a quarantine in hospitals recovered from the virus
γ_3	The rate of infected people with complications recovered from the virus
θ_1	The rate of quarantined people suffering serious complications after the virus has destroyed a large part of their lungs.
θ_2	The rate of hospitalized people suffering serious complications after the virus has destroyed a large part of their lungs.
δ_1	The death rate of people in quarantine at home
δ_2	The rate of people dying under quarantine in hospitals
δ_3	The death rate of people infected with health problems

TABLE 1. List of all parameters of system (9)

3. RESULTS

In this section we will give some preliminary results

Theorem 3.1. *If $S(0) \geq 0, E(0) \geq 0, I(0) \geq 0, Q(0) \geq 0, H(0) \geq 0, I^C(0) \geq 0$ and $R(0) \geq 0$, the solutions $S(t), E(t), I(t), Q(t), H(t), I^C(t)$ and $R(t)$ of the proposed system (8) are positive for all $t \geq 0$.*

Proof. We have from the first equation of the system that

$$\frac{dS(t)}{dt} = \Lambda - \beta_1 \frac{S(t)I(t)}{N} - \beta_2 \frac{S(t)E(t)}{N} - \mu S(t) \geq -S(t) \left(\beta_1 \frac{I(t)}{N} + \beta_2 \frac{E(t)}{N} + \mu \right)$$

Then

$$\frac{dS(t)}{dt} + S(t) \left(\beta_1 \frac{I(t)}{N} + \beta_2 \frac{E(t)}{N} + \mu \right) \geq 0$$

We multiply this inequation by $\exp \left(\int_0^t \left(\beta_1 \frac{I(s)}{N} + \beta_2 \frac{E(s)}{N} + \mu \right) ds \right)$, we obtain that

$$\begin{aligned} & \exp \left(\int_0^t \left(\beta_1 \frac{I(s)}{N} + \beta_2 \frac{E(s)}{N} + \mu \right) ds \right) \cdot \frac{dS(t)}{dt} \\ & + \left(\beta_1 \frac{I(t)}{N} + \beta_2 \frac{E(t)}{N} + \mu \right) \exp \left(\int_0^t \left(\beta_1 \frac{I(s)}{N} + \beta_2 \frac{E(s)}{N} + \mu \right) ds \right) \cdot S(t) \geq 0 \end{aligned}$$

hence

$$\frac{d}{dt} \left(S(t) \exp \left(\int_0^t \left(\beta_1 \frac{I(s)}{N} + \beta_2 \frac{E(s)}{N} + \mu \right) ds \right) \right) \geq 0, \quad \forall t \geq 0.$$

By integrating this inequality from 0 to t we obtain

$$\int_0^t \frac{d}{ds} \left(S(s) \exp \left(\int_0^t \left(\beta_1 \frac{I(s)}{N} + \beta_2 \frac{E(s)}{N} + \mu \right) ds \right) \right) ds \geq 0$$

hence

$$S(t) \geq S(0) \exp \left(\int_0^t \left(\beta_1 \frac{I(s)}{N} + \beta_2 \frac{E(s)}{N} + \mu \right) ds \right) \geq 0.$$

Then $S(t) \geq 0$ for all $t \geq 0$. Similary, we can also prove that $E(t), I(t), Q(t), H(t), I^C(t)$ and $R(t)$ are positive for all $t \geq 0$. \square

Theorem 3.2. *The set $\Omega = \left\{ (S, E, I, Q, H, I^C, R) \in \mathbb{R}_+^7 : S + E + I + Q + Q + H + I^C + R \leq \frac{\Lambda}{\mu} \right\}$ is a positively invariant set for the proposed system (8).*

Proof. Let $N = S + E + I + Q + Q + H + I^C + R$, then

$$\frac{dN}{dt} = \Lambda - \mu N - \delta_1 I - \delta_2 H - \delta_3 I^C \leq \Lambda - \mu N.$$

Hence

$$N(t) \leq \frac{\Lambda}{\mu} + N(0)e^{-\mu t}$$

This proves that the set Ω is positively invariant for the proposed system (8). \square

Theorem 3.3. *For any given $(S(0), E(0), I(0), Q(0), H(0), I^C(0), R(0))$, there exists a unique solution of the proposed system (8).*

Proof. Let $X = \begin{pmatrix} S(t) \\ E(t) \\ I(t) \\ Q(t) \\ H(t) \\ I^C \\ R(t) \end{pmatrix}$, then

$$\frac{dX(t)}{dt} = \Psi(X(t)) = AX(t) + F(X(t))$$

where

$$A = \begin{pmatrix} -\mu & 0 & -0 & 0 & 0 & 0 & 0 \\ 0 & -(\mu + \sigma) & 0 & 0 & 0 & 0 & 0 \\ 0 & \sigma & -(\alpha + \delta_1 + \mu) & 0 & 0 & 0 & 0 \\ 0 & 0 & \alpha_1 & -(\theta_1 + \mu + \gamma_1) & 0 & 0 & 0 \\ 0 & 0 & \alpha_2 & \theta_1 & -(\mu + \gamma_2 + \delta_2 + \theta_2) & 0 & 0 \\ 0 & 0 & \alpha_3 & 0 & \theta_2 & -(\gamma_3 + \delta_3 + \mu) & 0 \\ 0 & 0 & 0 & \gamma_1 & \gamma_2 & \gamma_3 & -\mu \end{pmatrix}$$

and

$$F(X) = \begin{pmatrix} \Lambda - \beta_1 \frac{S(t)E(t)}{N} - \beta_2 \frac{S(t)I(t)}{N} \\ \beta_1 \frac{S(t)E(t)}{N} + \beta_2 \frac{S(t)I(t)}{N} \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}$$

and $\alpha = \alpha_1 + \alpha_2 + \alpha_3$;

On the other hand, let $Y = \begin{pmatrix} S_Y \\ E_Y \\ I_Y \\ Q_Y \\ H_Y \\ I_Y^C \\ R_Y \end{pmatrix}$ and $Z = \begin{pmatrix} S_Z \\ E_Z \\ I_Z \\ Q_Z \\ H_Z \\ I_Z^C \\ R_Z \end{pmatrix}$ then

$$\begin{aligned} \|F(Y) - F(Z)\| &\leq \left| \beta_1 \frac{S_Y E_Y}{N} + \beta_2 \frac{S_Y I_Y}{N} - \beta_1 \frac{S_Z E_Z}{N} - \beta_2 \frac{S_Z I_Z}{N} \right| \\ &= \left| \beta_1 \frac{S_Y E_Y}{N} - \beta_1 \frac{S_Y E_Z}{N} + \beta_2 \frac{S_Y I_Y}{N} - \beta_2 \frac{S_Y I_Z}{N} - \beta_1 \frac{S_Z E_Z}{N} + \beta_1 \frac{S_Y E_Z}{N} - \beta_2 \frac{S_Z I_Z}{N} + \beta_2 \frac{S_Y I_Z}{N} \right| \\ &= \left| \beta_1 \frac{S_Y}{N} (E_Y - E_Z) + \beta_2 \frac{S_Y}{N} (I_Y - I_Z) - \beta_1 \frac{E_Z}{N} (S_Z - S_Y) - \beta_2 \frac{I_Z}{N} (S_Z - S_Y) \right| \\ &\leq \left| \beta_1 \frac{S_Y}{N} \right| |E_Y - E_Z| + \left| \beta_2 \frac{S_Y}{N} \right| |I_Y - I_Z| + \left| \beta_1 \frac{E_Z}{N} \right| |S_Y - S_Z| + \left| \beta_2 \frac{I_Z}{N} \right| |S_Y - S_Z| \\ &\leq |B(X_1) - B(X_2)| \\ &\leq \frac{\Lambda}{\mu} \left(\left| \frac{\beta_1}{N} \right| |E_Y - E_Z| + \left| \frac{\beta_2}{N} \right| |I_Y - I_Z| + \left| \frac{\beta_1}{N} \right| |S_Y - S_Z| + \left| \frac{\beta_2}{N} \right| |S_Y - S_Z| \right) \\ &\leq C \|Y - Z\| \end{aligned}$$

with

$$C = \frac{\Lambda}{\mu} \cdot \max \left(\frac{\beta_1}{N}, \frac{\beta_2}{N} \right)$$

hence

$$\|\Psi(Y) - \Psi(Z)\| \leq K \cdot \|Y - Z\|$$

where

$$K = \max(C, \|A\|).$$

□

4. MODEL WITH CONTROL

Considering the waste left by the Corona epidemic and its impact on the world's economies and on the health structures of various countries, and to avoid similar results of monkeypox we propose four control strategies:

The first control strategy (control u_2) can be interpreted as the proportion using smallpox vaccine because the monkeypox virus is closely related to the virus that causes smallpox, so the smallpox vaccine can protect people from smallpox [30]. The second control strategy (control u_1) can be interpreted as the proportion to be submitted to the awareness and prevention of individuals. the third control strategy (control u_3) can be interpreted as hospitalization and medical management of patients to spare them from the health problems of the disease. the fourth control strategy (control u_4) can be interpreted as hospitalization, intensive care and recovery room for those infected with serious health problems

$$(9) \quad \left\{ \begin{array}{l} \frac{dS(t)}{dt} = \Lambda - \beta_1 u_1(t) \frac{S(t)I(t)}{N} - \beta_2 u_1(t) \frac{S(t)E(t)}{N} - \mu S(t) - u_2(t)S(t) \\ \frac{dE(t)}{dt} = \beta_1 u_1(t) \frac{S(t)I(t)}{N} + \beta_2 u_1(t) \frac{S(t)E(t)}{N} - (\mu + \sigma) E(t) \\ \frac{dI(t)}{dt} = \sigma E(t) - (\alpha_1 + \alpha_2 + \alpha_3 u_3(t) + \delta_1 + \mu) I(t) \\ \frac{dQ(t)}{dt} = \alpha_1 I(t) - (\theta_1 + \gamma_1 + \mu) Q(t) \\ \frac{dH(t)}{dt} = \alpha_2 I(t) + \theta_1 Q(t) - (\theta_2 u_4(t) + \gamma_2 + \delta_2 + \mu) H(t) \\ \frac{dI^C(t)}{dt} = \alpha_3 u_3(t) I(t) + \theta_2 u_4(t) H(t) - (\gamma_3 + \delta_3 + \mu) I^C(t) \\ \frac{dR(t)}{dt} = \gamma_1 Q(t) + \gamma_2 H(t) + \gamma_3 I^C(t) - \mu R(t) + u_2(t)S(t) \end{array} \right.$$

If we take $S = \frac{S}{N}$, $E = \frac{E}{N}$, $I = \frac{I}{N}$, $Q = \frac{Q}{N}$, $H = \frac{H}{N}$, $I^C = \frac{I^C}{N}$ and $R = \frac{R}{N}$ then we obtain the normalized system

$$(10) \quad \left\{ \begin{array}{l} \frac{dS(t)}{dt} = \Lambda - \beta_1 u_1(t) S(t) I(t) - \beta_2 u_1(t) S(t) E(t) - \mu S(t) - u_2(t) S(t) \\ \frac{dE(t)}{dt} = \beta_1 u_1(t) S(t) I(t) + \beta_2 u_1(t) S(t) E(t) - (\mu + \sigma) E(t) \\ \frac{dI(t)}{dt} = \sigma E(t) - (\alpha_1 + \alpha_2 + \alpha_3 u_3(t) + \delta_1 + \mu) I(t) \\ \frac{dQ(t)}{dt} = \alpha_1 I(t) - (\theta_1 + \gamma_1 + \mu) Q(t) \\ \frac{dH(t)}{dt} = \alpha_2 I(t) + \theta_1 Q(t) - (\theta_2 u_4(t) + \gamma_2 + \delta_2 + \mu) H(t) \\ \frac{dI^C(t)}{dt} = \alpha_3 u_3(t) I(t) + \theta_2 u_4(t) H(t) - (\gamma_3 + \delta_3 + \mu) I^C(t) \\ \frac{dR(t)}{dt} = \gamma_1 Q(t) + \gamma_2 H(t) + \gamma_3 I^C(t) - \mu R(t) + u_2(t) S(t) \end{array} \right.$$

this system could be rewritten as follows

$$(11) \quad \dot{x}(t) = f(S(t), E(t), I(t), Q(t), H(t), I^C(t), R(t))$$

were $x = [S(t), E(t), I(t), Q(t), H(t), I^C(t), R(t)]^\top$.

4.1. Description of Optimal Control. In this section, we minimize the total number of exposed and infected humans to Monkeypox in the population using the control variables $u_1(t)$, $u_2(t)$ and $u_3(t)$ in the model. We define the objective function as

$$(12) \quad J(u_1, u_2, u_3, u_4) = \int_0^T a_1 E(t) + a_2 I(t) + a_3 I^C(t) + \frac{1}{2} (b_1 u_1^2(t) + b_2 u_2^2(t) + b_3 u_3^2(t) + b_4 u_4^2(t)) dt$$

subject to model (19), where a_1, a_2 and a_3 are positive weight constants of exposed, infected humans and infected with complication respectively. b_1, b_2, b_3 and b_4 are positive weight constants of control variables $u_1(t), u_2(t), u_3(t)$ and $u_4(t)$ respectively.

The purpose is to find an optimal control of u_1^*, u_2^*, u_3^* and u_4^* such that

$$(13) \quad J(u_1^*, u_2^*, u_3^*, u_4^*) = \min_{u_1, u_2, u_3, u_4 \in \Gamma} J(u_1, u_2, u_3, u_4)$$

where

$$(14) \quad \Gamma = \{u_i / u_i \text{ Lebesgue measurable, } 0 \leq u_i(t) \leq 1, \forall t \in [0, T], i = 1, 2, 3, 4\}$$

is the control set subject to the model (19) with initial conditions.

4.2. Existence of the optimal control. In this section we present the theorem that proves the existence of an optimal control $u^* = (u_1^*, u_2^*, u_3^*, u_4^*)$ that minimizes the cost function J .

Theorem 4.1. *There exists an optimal control $(u_1^*, u_2^*, u_3^*, u_4^*) \in \Gamma$ such that $J(u_1^*, u_2^*, u_3^*, u_4^*) = \min_{(u_1, u_2, u_3, u_4) \in \Gamma} J(u_1, u_2, u_3, u_4)$*

Proof. The existence of optimal control may be obtained by utilizing a result of Fleming and Rishel [31, 32]. \square

4.3. Necessary conditions for optimal control. In order to derive the necessary conditions for optimal control, we applied Pontryagin's maximum principle [32, 33] to the Hamiltonian H at time t associated with system (19) and the cost function J defined by

$$(15) \quad H(x(t), u(t), \lambda(t)) = L(x(t), u(t)) + \lambda^\top(t) f(x(t), u(t))$$

i.e.

$$\begin{aligned} H(x(t), u(t), \lambda(t)) &= a_1 E(t) + a_2 I(t) + a_3 I^c(t) + \frac{1}{2} [b_1 u_1^2(t) + b_2 u_2^2(t) + b_3 u_3^2(t) + b_4 u_4^2(t)] \\ &+ \lambda_1(t) [\Lambda - \beta_1 u_1(t) S(t) I(t) - \beta_2 u_1(t) S(t) E(t) - \mu S(t) - u_2(t) S(t)] \\ &+ \lambda_2(t) [\beta_1 u_1(t) S(t) I(t) + \beta_2 u_1(t) S(t) E(t) - (\mu + \sigma) E(t)] \\ &+ \lambda_3(t) [\sigma E(t) - (\alpha_1 + \alpha_2 + \alpha_3 u_3(t) + \delta_1 + \mu) I(t)] \\ &+ \lambda_4(t) [\alpha_1 I(t) - (\theta_1 + \gamma_1 + \mu) Q(t)] \\ &+ \lambda_5(t) [\alpha_2 I(t) + \theta_1 Q(t) - (\theta_2 u_4(t) + \gamma_2 + \delta_2 + \mu) H(t)] \\ &+ \lambda_6(t) [\alpha_3 u_3(t) I(t) + \theta_2 u_4(t) H(t) - (\gamma_3 + \delta_3 + \mu) I^c(t)] \\ &+ \lambda_7(t) [\gamma_1 Q(t) + \gamma_2 H(t) + \gamma_3 I^c(t) - \mu R(t) + u_2(t) S(t)] \end{aligned}$$

where $\lambda(t) = [\lambda_1(t), \lambda_2(t), \lambda_3(t), \lambda_4(t), \lambda_5(t), \lambda_6(t), \lambda_7(t)]^\top$ is the vector of adjoint variables.

The existence of optimal control of the model (19) would be considered by applying the theorem.

Therefore, we can now apply the necessary conditions to the Hamiltonian, H , in eq.(15).

Theorem 4.2. *Let $S^*, E^*, I^*, Q^*, H^*, I^{c*}$ and R^* be optimal state solutions associated with optimal control $(u_1^*, u_2^*, u_3^*, u_4^*)$ for the optimal control problem in model (19) and eq.(12). There*

exist the adjoint variables λ_i that verify the equation (16) with the transversality conditions $\lambda_i(T) = 0$ in eq.(17) for $i = 1, \dots, 7$ and in eq. (18) the control variables $(u_1^*, u_2^*, u_3^*, u_4^*)$.

$$(16) \quad \left\{ \begin{array}{l} \dot{\lambda}_1 = -\lambda_1(t) [-\beta_1 u_1(t)I(t) - \beta_2 u_1(t)E(t) - \mu - u_2(t)] - \lambda_2(t) [\beta_1 u_1(t)I(t) + \beta_2 u_1(t)E(t)] \\ \quad + \lambda_7(t)u_2(t) \\ \dot{\lambda}_2 = -a - \lambda_1(t) [-\beta_2 u_1(t)S(t)] - \lambda_2(t) [\beta_2 u_1(t)S(t) - (\mu + \sigma)] - \lambda_3(t)\sigma \\ \dot{\lambda}_3 = -a_2 - \lambda_1(t) [-\beta_1 u_1(t)S(t)] - \lambda_2(t) [\beta_1 u_1(t)S(t)] - \lambda_3(t) [-(\alpha_1 + \alpha_2 + \alpha_3 u_3(t) + \delta_1 + \mu)] - \\ \quad - \lambda_4(t)\alpha_1 - \lambda_5(t)\alpha_2 - \lambda_6(t)\alpha_3 u_3(t) \\ \dot{\lambda}_4 = -\lambda_4(t) [-(\theta_1 + \gamma_1 + \mu)] - \lambda_5(t)\theta_1 - \lambda_7(t)\gamma_1 \\ \dot{\lambda}_5 = -\lambda_5(t) [-(\theta_2 u_4(t) + \gamma_2 + \delta_2 + \mu)] - \lambda_6(t)\theta_2 u_4(t) - \lambda_7(t)\gamma_2 \\ \dot{\lambda}_6 = -a_3 - \lambda_6(t) [-(\gamma_3 + \delta_3 + \mu)] - \lambda_7(t)\gamma_3 \\ \dot{\lambda}_7 = \mu \lambda_7(t) \end{array} \right.$$

with the transversality conditions

$$(17) \quad \lambda_1(T) = 0; \lambda_2(T) = a_1; \lambda_3(T) = a_2; \lambda_4(T) = 0; \lambda_5(T) = 0; \lambda_6(T) = a_3; \lambda_7(T) = 0$$

$$(18) \quad \left\{ \begin{array}{l} u_1^*(t) = \max \left(0, \min \left(\frac{(\lambda_1(t) - \lambda_2(t))(\beta_1 I^*(t) + \beta_2 E^*(t))S^*(t)}{b_1}, 1 \right) \right) \\ u_2^*(t) = \max \left(0, \min \left(\frac{(\lambda_1 - \lambda_7)S^*(t)}{b_2}, 1 \right) \right) \\ u_3^*(t) = \max \left(0, \min \left(\frac{(\lambda_3 - \lambda_6)\alpha_3 I^*(t)}{b_3}, 1 \right) \right) \\ u_4^*(t) = \max \left(0, \min \left(\frac{(\lambda_5 - \lambda_6)\theta_2 H^*(t)}{b_4}, 1 \right) \right) \end{array} \right.$$

Proof. We have according to the theorem of Pontryagin [33, 34]

$$\begin{aligned} \dot{\lambda}_1 &= -\frac{\partial H}{\partial S} = -\lambda_1(t) [-\beta_1 u_1(t)I(t) - \beta_2 u_1(t)E(t) - \mu - u_2(t)] - \lambda_2(t) [\beta_1 u_1(t)I(t) + \beta_2 u_1(t)E(t)] \\ &\quad + \lambda_7(t)u_2(t) \\ \dot{\lambda}_2 &= -\frac{\partial H}{\partial E} = -a - \lambda_1(t) [-\beta_2 u_1(t)S(t)] - \lambda_2(t) [\beta_2 u_1(t)S(t) - (\mu + \sigma)] - \lambda_3(t)\sigma \\ \dot{\lambda}_3 &= -\frac{\partial H}{\partial I} = -a_2 - \lambda_1(t) [-\beta_1 u_1(t)S(t)] - \lambda_2(t) [\beta_1 u_1(t)S(t)] - \lambda_3(t) [-(\alpha_1 + \alpha_2 + \alpha_3 u_3(t) + \delta_1 + \mu)] \\ &\quad - \lambda_4(t)\alpha_1 - \lambda_5(t)\alpha_2 - \lambda_6(t)\alpha_3 u_3(t) \end{aligned}$$

$$\begin{aligned}
\dot{\lambda}_4 &= -\frac{\partial H}{\partial Q} = -\lambda_4(t)[-(\theta_1 + \gamma_1 + \mu)] - \lambda_5(t)\theta_1 - \lambda_7(t)\gamma_1 \\
\dot{\lambda}_5 &= -\frac{\partial H}{\partial H} = -\lambda_5(t)[-(\theta_2 u_4(t) + \gamma_2 + \delta_2 + \mu)] - \lambda_6(t)\theta_2 u_4(t) - \lambda_7(t)\gamma_2 \\
\dot{\lambda}_6 &= -\frac{\partial H}{\partial I^c} = -a_3 - \lambda_6(t)[-(\gamma_3 + \delta_3 + \mu)] - \lambda_7(t)\gamma_3 \\
\dot{\lambda}_7 &= -\frac{\partial H}{\partial R} = \mu\lambda_7(t).
\end{aligned}$$

In other words we have

$$\begin{aligned}
\frac{\partial H(x^*, u^*, \lambda)}{\partial u_1} &= 0 \\
\frac{\partial H(x^*, u^*, \lambda)}{\partial u_2} &= 0 \\
\frac{\partial H(x^*, u^*, \lambda)}{\partial u_3} &= 0 \\
\frac{\partial H(x^*, u^*, \lambda)}{\partial u_4} &= 0
\end{aligned}$$

i.e.,

$$\begin{aligned}
b_1 u_1^*(t) + \lambda_1(t)[-\beta_1 S^*(t)I^*(t) - \beta_2 S^*(t)E^*(t)] + \lambda_2(t)[\beta_1 S^*(t)I^*(t) + \beta_2 S^*(t)E^*(t)] &= 0 \\
b_2 u_2^*(t) - \lambda_1(t)S^*(t) + \lambda_7 S^*(t) &= 0 \\
b_3 u_3^*(t) - \lambda_3 \alpha_3 I^*(t) + \lambda_6 \alpha_3 I^*(t) &= 0 \\
b_4 u_4^*(t) - \lambda_5 \theta_2 H^*(t) + \lambda_6 \theta_2 H^*(t) &= 0
\end{aligned}$$

thus

$$\begin{cases}
u_1^*(t) = \frac{(\lambda_1(t) - \lambda_2(t))(\beta_1 I^*(t) + \beta_2 E^*(t))S^*(t)}{b_1} \\
u_2^*(t) = \frac{(\lambda_1 - \lambda_7)S^*(t)}{b_2} \\
u_3^*(t) = \frac{(\lambda_3 - \lambda_6)\alpha_3 I^*(t)}{b_3} \\
u_4^*(t) = \frac{(\lambda_5 - \lambda_6)\theta_2 H^*(t)}{b_4}
\end{cases}$$

therefore, we have

$$\begin{cases} u_1^*(t) = \max \left(0, \min \left(\frac{(\lambda_1(t) - \lambda_2(t))(\beta_1 I^*(t) + \beta_2 E^*(t))S^*(t)}{b_1}, 1 \right) \right) \\ u_2^*(t) = \max \left(0, \min \left(\frac{(\lambda_1 - \lambda_7)S^*(t)}{b_2}, 1 \right) \right) \\ u_3^*(t) = \max \left(0, \min \left(\frac{(\lambda_3 - \lambda_6)\alpha_3 I^*(t)}{b_3}, 1 \right) \right) \\ u_4^*(t) = \max \left(0, \min \left(\frac{(\lambda_5 - \lambda_6)\theta_2 H^*(t)}{b_4}, 1 \right) \right) \end{cases}$$

this completes the demonstration. \square

5. NUMERICAL SOLUTION OF THE OPTIMAL CONTROL

We present numerical simulations for the aforementioned optimization problem in this section. We make the program in MATLAB (5.1), and we simulate our work with diverse data. The optimization systems are solved using a discretized iterative process that converges after a proper test analogous to the FBSM. First, the state system is solved with the initial hypothesis forward in time, and then the adjoint system is solved backward in time due to the transversality conditions. Then, we update our optimal control values with the state and co-state resources derived in the above steps. Finally, we run the preceding steps until the standard tolerance is reached.

5.1. Algorithm. Consider our control system

$$(19) \quad \begin{cases} \frac{dS(t)}{dt} = \Lambda - \beta_1 S(t)I(t) - \beta_2(1 - u_1^*(t))S(t)E(t) - \mu S(t) \\ \frac{dE(t)}{dt} = \beta_1 S(t)I(t) + \beta_2(1 - u_1^*(t))S(t)E(t) - (\mu + \sigma)E(t) \\ \frac{dI(t)}{dt} = \sigma E(t) - (\alpha_1 + \alpha_2 + \alpha_3 + \delta_1 + \mu)I(t) - u_2(t)I(t) \\ \frac{dQ(t)}{dt} = \alpha_1 I(t) - (\theta_1 + \gamma_1 + \mu)Q(t) \\ \frac{dH(t)}{dt} = \alpha_2 I(t) + \theta_1 Q(t) - (\theta_2 + \gamma_2 + \delta_2 + \mu)H(t) - u_3(t)H(t) \\ \frac{dI^C(t)}{dt} = \alpha_3 I(t) + \theta_2 H(t) - (\gamma_3 + \delta_3 + \mu)I^C(t) - u_4(t)I^C \\ \frac{dR(t)}{dt} = \gamma_1 Q(t) + \gamma_2 H(t) + \gamma_3 I^C(t) - \mu R(t) + u_2^*(t)I(t) + u_3^*(t)H(t) + u_4^*(t)I^C(t) \end{cases}$$

We discretize the interval $[0, T]$ at the points $t_i = ih, i = 0, 1, \dots, N$ with $h = \frac{T}{N}$.

Next, we define the state variables $S(t), E(t), I(t), Q(t), H(t), I^C(t), R(t)$ and adjoint variables

$\lambda_1(t), \lambda_2(t), \lambda_3(t), \lambda_4(t), \lambda_5(t), \lambda_6(t), \lambda_7(t)$ and the controls $u_1(t), u_2(t), u_3(t)$ and $u_4(t)$ in terms of nodal points $S_i, E_i, I_i, Q_i, H_i, I_i^C, R_i, \lambda_i^1, \lambda_i^2, \lambda_i^3, \lambda_i^4, \lambda_i^5, \lambda_i^6, \lambda_i^7, u_i^1, u_i^2, u_i^3$ and u_i^4 . Now a combination of forward and backward difference approximation is used as follows :

$$(20) \quad \left\{ \begin{array}{l} \frac{S_{i+1} - S_i}{h} = \Lambda - \beta_1 S_{i+1} I^i - \beta_2 (1 - u_1^i) S_{i+1} E_i - \mu S_{i+1} \\ \frac{E_{i+1} - E_i}{h} = \beta_1 S_{i+1} I_i + \beta_2 (1 - u_1^i) S_{i+1} E_{i+1} - (\mu + \sigma) E_{i+1} \\ \frac{I_{i+1} - I_i}{h} = \sigma E_{i+1} - (\alpha_1 + \alpha_2 + \alpha_3 + \delta_1 + \mu) I_{i+1} - u_2^i I_{i+1} \\ \frac{Q_{i+1} - Q_i}{h} = \alpha_1 I_{i+1} - (\theta_1 + \gamma_1 + \mu) Q_{i+1} \\ \frac{H_{i+1} - H_i}{h} = \alpha_2 I_{i+1} + \theta_1 Q_{i+1} - (\theta_2 + \gamma_2 + \delta_2 + \mu) H_{i+1} - u_3^i H_{i+1} \\ \frac{I_{i+1}^C - I_i^C}{h} = \alpha_3 I_{i+1} + \theta_2 H_{i+1} - (\gamma_3 + \delta_3 + \mu) I_{i+1}^C - u_4^i I_{i+1}^C \\ \frac{R_{i+1} - R_i}{h} = \gamma_1 Q_{i+1} + \gamma_2 H_{i+1} + \gamma_3 I_{i+1}^C - \mu R_{i+1} + u_2^i I_{i+1} + u_3^i H_{i+1} + u_4^i I_{i+1}^C \end{array} \right.$$

$$\left\{ \begin{array}{l} \frac{\lambda_1^{n-i} - \lambda_1^{n-i-1}}{h} = -\lambda_1^{n-i-1} [-\beta_1 I_{i+1} - \beta_2 (1 - u_1^i) E_{i+1} - \mu] - \lambda_2^{n-i} [\beta_1 I^{i+1} + \beta_2 (1 - u_1^i) E^{i+1}] \\ \frac{\lambda_2^{n-i} - \lambda_2^{n-i-1}}{h} = -a_1 - \lambda_1^{n-i-1} [-\beta_2 (1 - u_1^i) S_{i+1}] - \lambda_2^{n-i-1} [\beta_2 (1 - u_1^i) S_{i+1} - (\mu + \sigma)] - \lambda_3^{n-i} \sigma \\ \frac{\lambda_3^{n-i} - \lambda_3^{n-i-1}}{h} = -a_2 - \lambda_1^{n-i-1} [-\beta_1 S_{i+1}] - \lambda_2^{n-i-1} [\beta_1 S_{i+1}] - \lambda_3^{n-i-1} [-(\alpha_1 + \alpha_2 + \alpha_3 + \delta_1 + \mu) - u_2^i] \\ -\lambda_4^{n-i} \alpha_1 - \lambda_5^{n-i} \alpha_2 - \lambda_6^{n-i} \alpha_3 - \lambda_7^{n-i} u_2^i \\ \frac{\lambda_4^{n-i} - \lambda_4^{n-i-1}}{h} = -\lambda_4^{n-i-1} [-(\theta_1 + \gamma_1 + \mu)] - \lambda_5^{n-i} \theta_1 - \lambda_7^{n-i} \gamma_1 \\ \frac{\lambda_5^{n-i} - \lambda_5^{n-i-1}}{h} = -\lambda_5^{n-i-1} [-(\theta_2 + \gamma_2 + \delta_2 + \mu) - u_3^i] - \lambda_6^{n-i} \theta_2 u_4^i - \lambda_7^{n-i} (\gamma_2 + u_3^i) \\ \frac{\lambda_6^{n-i} - \lambda_6^{n-i-1}}{h} = -a_3 - \lambda_6^{n-i-1} [-(\gamma_3 + \delta_3 + \mu) - u_4^i] - \lambda_7^{n-i} (\gamma_3 + u_4^i) \\ \frac{\lambda_7^{n-i} - \lambda_7^{n-i-1}}{h} = \mu \lambda_7^{n-i-1} \end{array} \right.$$

Algorithm. Step 1 : $S_0, E_0, I_0, Q_0, H_0, I_0^C, R_0$ given and $\lambda_j^n = 0, j = 1, \dots, 7, u_j^0 = 0, j = 1, \dots, 4$

Step 2 : for $i = 0, \dots, n-1$ do

$$S_{i+1} = \frac{h\Lambda + S_i}{1 + h(\beta_1 I^i - \beta_2 (1 - u_1^i) E_i - \mu)}$$

$$E_{i+1} = \frac{h\beta_1 S_{i+1} I_i + E_i}{1 + h(\mu + \sigma - \beta_2(1 - u_1^i) S_{i+1})}$$

$$I_{i+1} = \frac{h\sigma E_{i+1} + I_i}{1 + h(\alpha_1 + \alpha_2 + \alpha_3 + \delta_1 + \mu + u_2^i)}$$

$$Q_{i+1} = \frac{h\alpha_1 I_{i+1} + Q_i}{1 + h(\theta_1 + \gamma_1 + \mu)}$$

$$H_{i+1} = \frac{h\alpha_2 I_{i+1} + h\theta_1 Q_{i+1} + H_i}{1 + h(\theta_2 + \gamma_2 + \delta_2 + \mu + u_3^i)}$$

$$I_{i+1}^c = \frac{h\alpha_3 I_{i+1} + h\theta_2 H_{i+1} + I_i^c}{1 + h(\gamma_3 + \delta_3 + \mu + u_4^i)}$$

$$R_{i+1} = \frac{h\gamma_1 Q_{i+1} + h\gamma_2 H_{i+1} + h\gamma_3 I_{i+1}^c + hu_2^i I_{i+1} + hu_3^i H_{i+1} + hu_4^i I_{i+1}^c + R_i}{1 + h\mu}$$

$$\lambda_1^{n-i-1} = \frac{\lambda_1^{n-i} + h\lambda_2^{n-i} [\beta_1 I^{i+1} + \beta_2(1 - u_1^i) E^{i+1}]}{1 - h[-\beta_1 I_{i+1} - \beta_2(1 - u_1^i) E_{i+1} - \mu]}$$

$$\lambda_2^{n-i-1} = \frac{\lambda_2^{n-i} + ha_1 + h\lambda_1^{n-i-1} [-\beta_2(1 - u_1^i) S_{i+1}] + h\lambda_3^{n-i} \sigma}{1 - h[\beta_2(1 - u_1^i) S_{i+1} - (\mu + \sigma)]}$$

$$\lambda_3^{n-i-1} = \frac{\lambda_3^{n-i} + ha_2 + h\lambda_1^{n-i-1} [-\beta_1 S_{i+1}] + h\lambda_2^{n-i-1} [\beta_1 S_{i+1}] + h\lambda_4^{n-i} \alpha_1 + h\lambda_5^{n-i} \alpha_2 + h\lambda_6^{n-i} \alpha_3 + \lambda_7^{n-i} u_2^i}{1 - h[-(\alpha_1 + \alpha_2 + \alpha_3 + \delta_1 + \mu + u_2^i)]}$$

$$\lambda_4^{n-i-1} = \frac{\lambda_4^{n-i} + h\lambda_5^{n-i}\theta_1 + h\lambda_7^{n-i}\gamma_1}{1 - h[-(\theta_1 + \gamma_1 + \mu)]}$$

$$\lambda_5^{n-i-1} = \frac{\lambda_5^{n-i} + h\lambda_6^{n-i}\theta_2 + h\lambda_7^{n-i}(\gamma_2 + u_3^i)}{1 - h[-(\theta_2 u_4^i + \gamma_2 + \delta_2 + \mu + u_3^i)]}$$

$$\lambda_6^{n-i-1} = \frac{\lambda_6^{n-i} + ha_3 + \lambda_7^{n-i}(\gamma_3 + u_4^i)}{1 - h[-(\gamma_3 + \delta_3 + \mu + u_4^i)]}$$

$$\lambda_7^{n-i-1} = \frac{\lambda_7^{n-i}}{1 + h\mu}$$

$$u_1^{i+1} = \max \left(0, \min \left(\frac{(\lambda_2^{n-i-1} - \lambda_1^{n-i-1}) \beta_2 S_{i+1} E_{i+1}}{b_1}, 1 \right) \right)$$

$$u_2^{i+1} = \max \left(0, \min \left(\frac{(\lambda_3^{n-i-1} - \lambda_7^{n-i-1}) I_{i+1}}{b_2}, 1 \right) \right)$$

$$u_3^{i+1} = \max \left(0, \min \left(\frac{(\lambda_5^{n-i-1} - \lambda_7^{n-i-1}) H_{i+1}}{b_3}, 1 \right) \right)$$

$$u_4^{i+1} = \max \left(0, \min \left(\frac{(\lambda_6^{n-i-1} - \lambda_7^{n-i-1}) I_{i+1}^C}{b_4}, 1 \right) \right).$$

5.2. Discussion. Due to the impact that the Corona epidemic has had on global health systems [35] and economies [36], any major spread of monkeypox will have catastrophic consequences. Therefore, we have developed a vision for confronting this disease and avoiding its impact on humanity and economies through the four control strategies above (section (4)).

These strategies proved to be effective in reducing the number of infected people and individuals

who have serious health problems (See fig. (2) and (3)), which will reduce the strain on health systems and avoid the scenario of the Corona epidemic.

Our control plans have also proven to increase the number of people recovering (See fig. (5)), thereby reducing any potential impact of this disease on individuals and the economy.

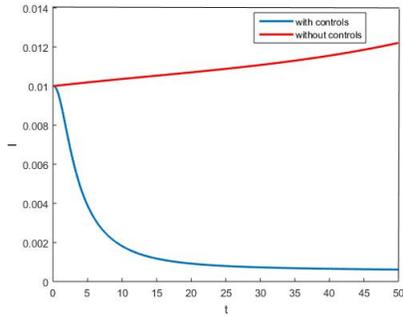


FIGURE 2. Infected Individual with symptoms

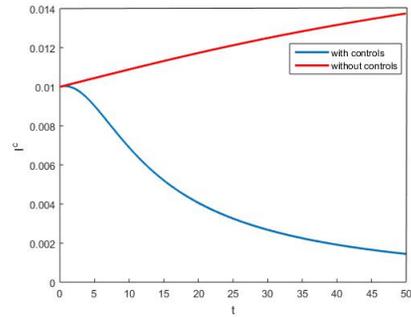


FIGURE 3. Infected Individual with complications

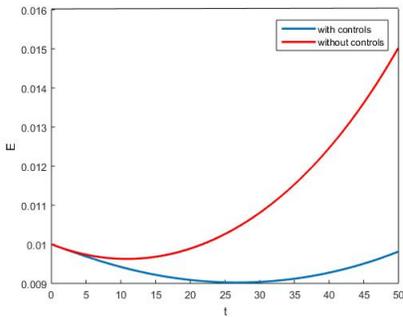


FIGURE 4. Exposed Individual

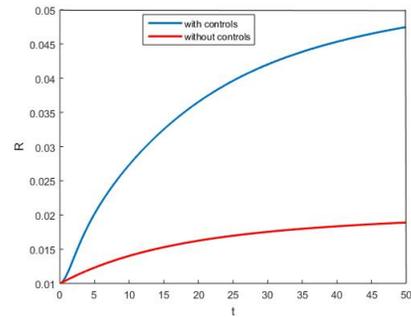
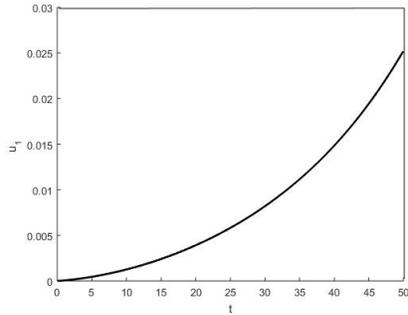
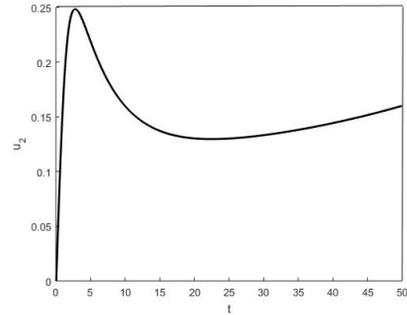
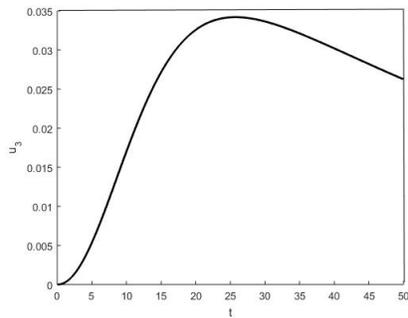
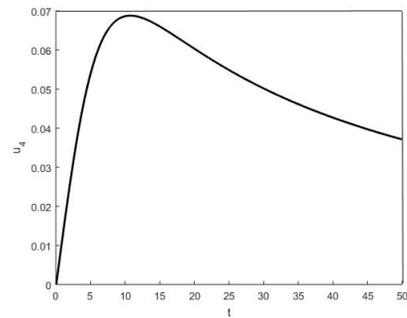


FIGURE 5. Recovered Individual

FIGURE 6. Control u_1 FIGURE 7. Control u_2 FIGURE 8. Control u_3 FIGURE 9. Control u_4

6. CONCLUSION

In this study, we propose a scenario for combating the infectious disease monkeypox, drawing on lessons learned from the Corona outbreak to minimize the disease's serious effects on humanity and global economies. This is accomplished through the use of three different control strategies. The optimal controls are characterized using Pontryagin's maximum principle, and the optimality system is resolved using an iterative approach. Finally, using MATLAB, numerical simulations are run to verify the theoretical analysis.

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DATA AVAILABILITY

The disciplinary data used to support the findings of this study have been deposited in the Network Repository (<http://www.networkrepository.com>).

CONFLICT OF INTERESTS

The authors declare that there is no conflict of interests.

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