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MATHEMATICAL MODELING AND MONKEYPOX'S OPTIMAL CONTROL STRATEGY

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Abstract. This study delves into a continuous-time mathematical framework that delineates the transmission dynamics of the monkeypox virus across distinct regions, involving both human and animal hosts. We introduce an optimal approach that encompasses awareness campaigns, security protocols, and health interventions in areas endemic to the virus, aiming to curtail the transmission among individuals and animals, thereby minimizing infections in humans and eradicating the virus in animals. Leveraging the discrete-time Pontryagin principle of maximum, we ascertain optimal controls, employing an iterative methodology to solve the optimal system. Employing Matlab, we conduct numerical simulations and compute a cost-effectiveness ratio. Through a comprehensive cost-effectiveness analysis, we underscore the efficacy of strategies centered around safeguarding vulnerable individuals, preventing contact with infected counterparts—both human and animal—and fostering the utilization of quarantine facilities as the most potent means to govern the spread of the monkeypox virus.

Keywords: optimal control; monkeypox virus; spread of monkeypox.

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1. INTRODUCTION

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In 1970, the monkeypox virus was initially identified in the Democratic Republic of the Congo. This virus, an animal DNA variant akin to the smallpox virus [1], has triggered sporadic outbreaks primarily attributed to contact with animal reservoirs [2]. At the outset, human-to-human transmission was deemed ineffective [3]. Recorded cases of monkeypox emerged in West African nations: Ivory Coast, Sierra Leone, and Liberia [4]. Between 1981 and 1986, the Democratic Republic of the Congo reported 338 instances of monkeypox, with 245 cases stemming from direct contact with animals and 93 cases as a result of successive human-to-human transmission, encompassing second, third, and even fourth generational spread.

The cases originating from animals predominantly affected males, with 58 belonging to the "5-14 years" age bracket [5]. In 2003, the virus found its way to the United States, marking the initial instance of monkeypox disseminating beyond its natural African epicenters. This outbreak emerged through Prairie dogs contaminated by Gambian rats carrying the monkeypox virus from Ghana [6]. Clinical manifestations commonly entail the onset of fever, escalating to encompass conspicuous lymphadenopathy, widespread ulcerative lesions, and vesiculopustular eruptions across the face and body [7].

Transmission of the monkeypox virus typically occurs when an individual encounters the virus via contact with an infected animal, virus-laden material permeating the body through imperceptible skin wounds, the respiratory tract, mucous membranes (nose or mouth), or direct contact with infected humans [8]. As of May 2022, several non-endemic countries have documented human cases of monkeypox. This expansion beyond native regions underscores the escalating threat this virus poses to human health, necessitating scientific intervention.

The utility of mathematical modeling in devising effective interventions and enhancing our grasp of disease dynamics and management is well-established. Such models frequently integrate differential operators-partial [9], [10], [11], [12], random [13], [14], or fractional derivatives [15], [16], [17], [18], [19]. This study introduces a mathematical model elucidating the dynamics of infected individuals displaying symptomatic manifestations and those remaining unaffected. Additionally, we present strategies to restrict virus dissemination between humans and animals.

The article's structure is as follows: Section 2 introduces a mathematical model for the transmission of Monkeypox involving humans and animals (monkeys). Section 3 outlines an optimal control predicament within our model, elucidating the presence and delineation of optimal controls for the Monkeypox virus through Pontryagin's principle of maximum. Section 4 encompasses numerical simulations via MATLAB. Lastly, Section 5 culminates the article's discourse.

2. MODEL FORMULATION

We consider a mathematical model $SEAIS_aI_aHR$ that describe the dynamic of transmission the spread of moneybox flu among people.

this model is divided into two categories: humain categories $N_h = S + E + A + I + H + R$ and animal categories $N_a = S_a + I_a$.



Figure 1. Model description

Compartment (S): The count of susceptible individuals (S) experiences an increase denoted by Λ (representing the incidence of susceptibility). This count is concurrently diminished by the natural mortality rate, μ_1 , as well as by the term $\beta_1 \frac{S(t)A(t)}{N_h}$, which signifies the number of 4

individuals who become infected with the virus due to contact with infected yet asymptomatic individuals. Moreover, the count is reduced by $\beta_2 \frac{S(t)I(t)}{N_h}$, which represents the individuals infected through contact with symptomatic cases. Furthermore, the term $\beta_3 \frac{S(t)I_a(t)}{N_h}$ accounts for the decrease in susceptible individuals caused by contact with infected monkeypox cases.

Compartment (E): This compartment represents the count of exposed individuals. It increases due to the terms $\beta_1 \frac{S(t)A(t)}{N_h}$ and $\beta_2 \frac{S(t)I(t)}{N_h}$, which denote the number of individuals becoming exposed through contact with infected asymptomatic and symptomatic individuals, respectively. The count decreases due to the natural mortality rate, μ_2 , and the terms $\alpha_1 E(t)$ (representing the count of exposed individuals becoming asymptomatic and infectious) and $\alpha_1 E(t)$ (representing the count of exposed individuals becoming symptomatic and infectious).

Compartment (I): This compartment represents the count of infected and symptomatic individuals. It increases due to $\alpha_2 E(t)$ and $\gamma A(t)$ (representing the count of infected asymptomatic individuals becoming infected and symptomatic), as well as $\beta_3 \frac{S(t)I_a(t)}{N_h}$. The count decreases due to the natural mortality rate, μ_3 , the mortality rate due to complications, $\theta I(t)$, and the count of individuals under lockdown, $n_1 I(t)$.

Compartment (A): This compartment represents the count of individuals infected with asymptomatic cases. It increases due to $\alpha_1 E(t)$. The count decreases due to the natural mortality rate, μ_4 , the count of individuals under lockdown, $n_2A(t)$, and the term $\gamma A(t)$.

Compartment (H): This compartment represents the count of individuals placed under lockdown in hospitals with follow-up and health monitoring. It increases due to the count of individuals under lockdown from compartments *I* and *A*, i.e., $n_1I(t)$ and $n_2A(t)$. The count decreases due to the natural mortality rate, μ_7 , and the rate of individuals recovering from the virus, $\sigma H(t)$.

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Compartment (Sa): This compartment represents the count of susceptible individuals to monkeypox. It increases due to Λ_1 (denoting the incidence of susceptibility from monkeypox). The count decreases due to the term $\beta_4 \frac{S_a(t)I_a(t)}{N_a}$, representing the count of monkeypox-susceptible individuals becoming infected through contact with infected monkeypox cases, and the natural mortality rate, μ_{10} .

Compartment(R): This compartment represents the count of people who have recovered. It increases due to the rate of individuals recovering from hospitals, $\sigma H(t)$. The count decreases due to the natural mortality rate, μ_6 .

Compartment (Ia): is representing the number of susceptible from monkeypox. $I_a(t)$ is increasing by $\beta_4 \frac{S_a(t)I_a(t)}{N_a}$. $I_a(t)$ is decreasing by μ_{11} (natural mortality) and I_a is decreasing by $\beta_3 \frac{S(t)I_a(t)}{N_b}$.

$$(1) \qquad \left\{ \begin{array}{l} \frac{dS(t)}{dt} = \Lambda - \beta_1 \frac{S(t)A(t)}{N_h} - \beta_2 \frac{S(t)I(t)}{N_h} - \beta_3 \frac{S(t)I_a(t)}{N_h} - \mu_1 S(t) \\ \frac{dE(t)}{dt} = \beta_1 \frac{S(t)A(t)}{N_h} + \beta_2 \frac{S(t)I(t)}{N_h} - \alpha_1 E(t) - \alpha_2 E(t) - \mu_2 E(t) \\ \frac{dA(t)}{dt} = \alpha_1 E(t) - n_1 A(t) - \gamma A(t) - \mu_3 A(t) \\ \frac{dI(t)}{dt} = \alpha_2 E(t) - \mu_4 I(t) - \theta I(t) + \gamma A(t) - n_2 I(t) + \beta_3 \frac{S(t)I_a(t)}{N_h} \\ \frac{dH(t)}{dt} = -\sigma H(t) - \mu_5 H(t) + n_1 A(t) + n_2 I(t) \\ \frac{dR(t)}{dt} = \sigma H(t) - \mu_6 R(t) \\ \frac{dS_a(t)}{dt} = \Lambda_1 - \mu_7 S_a(t) - \beta_4 \frac{S_a(t)I_a(t)}{N_a} \\ \frac{dI_a(t)}{dt} = \beta_4 \frac{S_a(t)I_a(t)}{N_a} - \mu_8 I_a(t) - \beta_3 \frac{S(t)I_a(t)}{N_h} \end{array} \right.$$

hence, we present the spread of monkeypox flu mathematical model in the country of Democratic Republic of the Congo is governed by the following system of differential equation. where $S(0) \ge 0$, $E(0) \ge 0$, $A(0) \ge 0$, $I(0) \ge 0$, $H(0) \ge 0$, $R(0) \ge 0$, $S_a(0) \ge 0$, $I_a(0) \ge 0$ are the initial rate.

2.1. MODEL BASIC PROPERTIES

2.1.1. POSITIVITY OF SOLUTIONS.

Theorem 1. if $S(0) \ge 0$, $E(0) \ge 0$, $A(0) \ge 0$, $I(0) \ge 0$, $H(0) \ge 0$, $R(0) \ge 0$, $S_a(0) \ge 0$, $I_a(0) \ge 0$ are the initial rate $t \ge 0$ the solution of system are positive for all $t \ge 0$ **Proof** It follows from the first equation of system (1) that

$$\begin{aligned} \frac{dS(t)}{dt} &= \Lambda + \left(-\beta_1 \frac{A(t)}{N_h} - \beta_2 \frac{I(t)}{N_h} - \beta_3 \frac{I_a(t)}{N_h} - \mu_1 \right) S(t) \\ &\geq - \left(\beta_1 \frac{A(t)}{N_h} + \beta_2 \frac{I(t)}{N_h} + \beta_3 \frac{I_a(t)}{N_h} + \mu_1 \right) S(t) \\ &\frac{dS(t)}{dt} + \left(\beta_1 \frac{A(t)}{N_h} + \beta_2 \frac{I(t)}{N_h} + \beta_3 \frac{I_a(t)}{N_h} + \mu_1 \right) S(t) \geq 0 \end{aligned}$$

where

$$F(t) = \beta_1 \frac{A(t)}{N_h} + \beta_2 \frac{I(t)}{N_h} + \beta_3 \frac{I_a(t)}{N_h} + \mu_1$$
$$\frac{dS(t)}{dt} + F(t)S(t) \ge 0$$

The last inequality's two sides are multiplied by $\exp\left(\int_0^t F(s)ds\right)$ We obtain

$$\exp\left(\int_0^t F(s)ds\right)\frac{dS(t)}{dt} + F(t)\exp\left(\int_0^t F(s)ds\right)S(t) \ge 0$$
$$\frac{d}{dt}\left(S(t)\exp\left(\int_0^t F(s)ds\right)\right) \ge 0$$

Integrating this inequality from 0 to t gives:

$$\int_0^t \left(\frac{d}{ds} \left(S(t) \exp\left(\int_0^t F(s) ds \right) \right) \right) ds \ge 0$$

then $S(t) \ge S(0) \exp\left(\int_0^t F(s) ds\right)$ $\Rightarrow S(t) \ge 0$

Likewise, we demonstrate $E(t) \ge 0$, $A(t) \ge 0$, $I(t) \ge 0$, $H(t) \ge 0$, $I_a(t) \ge 0$, $S_a(t) \ge 0$ and $R(t) \ge 0$

2.1.2. BOUDEDNESS OF THE SOLUTIONS.

Theorem 2. The set $\begin{cases}
\Omega_h = \left\{ (S, E, I, A, H, R) \in \Re_+^6 / 0 \le S + E + I + A + H + R \le \frac{\Lambda}{\mu_h} \right\} \\
\Omega_a = \left\{ (S_a, I_a) \in \Re_+^2 / 0 \le S_a + I_a \le \frac{\Lambda_1}{\mu_a} \right\} \\
\text{Positively invariant under system (1) with initial conditions} \\
S(0) \ge 0, E(0) \ge 0, A(0) \ge 0, I(0) \ge 0, H(0) \ge 0, I_a(0) \ge 0, S_a(0) \ge 0 \text{ and } R(0) \ge 0 \\
\text{Proof: by definition } N_h = S + E + A + I + H + R: \\
\text{hence } \frac{dN_h}{dt} = \Lambda - \theta I(t) - \mu_h N_h \\
\frac{dN_h}{dt} = \Lambda - \theta I(t) - \mu_h N_h \le \Lambda - \mu_h N_h \\
\frac{dN_h}{dt} \le \Lambda - \mu_h N_h \\
\Rightarrow N_h(t) \le \frac{\Lambda}{\mu_h} + N_h(0)e^{-\mu_h t} \\
\text{If we wake limit } t \to \infty \text{ then } N_h(t) \le \frac{\Lambda}{\mu_h} \\
\text{Theorem 2.} \quad \text{All } t = \Lambda - \theta I(t) = 0 \\
\text{All } t = 0$

It implies that the region Ω_h is a positively invariant set for the system (1).

$$\Rightarrow N_h(t) \leq \frac{\Lambda}{\mu_h}$$

subsequently, it can be proven that $N_a(t) \leq \frac{\Lambda_1}{\mu_a}$

2.2. EXISTENCE OF SOLUTIONS.

So the system (1) can be rewritten in the following form: $\psi(X) = AX + B(X)$

then

$$X = \begin{pmatrix} S(t) \\ E(t) \\ A(t) \\ I(t) \\ H(t) \\ R(t) \\ S_a(t) \\ I_a(t) \end{pmatrix} \qquad B(X) = \begin{pmatrix} \frac{dS(t)}{dt} \\ \frac{dE(t)}{dt} \\ \frac{dA(t)}{dt} \\ \frac{dH(t)}{dt} \\ \frac{dH(t)}{dt} \\ \frac{dR(t)}{dt} \\ \frac{dS(t)}{dt} \\ \frac{dH(t)}{dt} \\ \frac{dR(t)}{dt} \\ \frac{dS_a(t)}{dt} \\ \frac{dI_a(t)}{dt} \\ \frac{dI_a($$

where

$$A = \begin{pmatrix} -\mu_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & A_1 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \alpha_1 & A_2 & 0 & 0 & 0 & 0 & 0 \\ 0 & \alpha_2 & \gamma & A_3 & 0 & 0 & 0 & 0 \\ 0 & 0 & n_1 & n_2 & -(\mu_5 + \sigma) & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \sigma & -\mu_6 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -\mu_7 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\mu_8 \end{pmatrix}$$

$$A_1 = -(\alpha_1 + \alpha_2 + \mu_2)$$
, $A_2 = -(\mu_3 + \gamma + n_1)$ and $A_3 = -(\mu_4 + n_2 + \theta)$

and

$$B(X) = \begin{pmatrix} \Lambda - \beta_1 \frac{S(t)A(t)}{N_h} - \beta_2 \frac{S(t)I(t)}{N_h} - \beta_3 \frac{S(t)I_a(t)}{N_h} \\ \beta_1 \frac{S(t)A(t)}{N_h} + \beta_2 \frac{S(t)I(t)}{N_h} \\ 0 \\ \beta_3 \frac{S(t)I_a(t)}{N_h} \\ 0 \\ 0 \\ \Lambda_1 - \beta_4 \frac{S_a(t)I_a(t)}{N_a} \\ \beta_4 \frac{S_a(t)I_a(t)}{N_a} - \beta_3 \frac{S(t)I_a(t)}{N_h} \end{pmatrix}$$

3. The Controlled Mathematical Model

3.1. AIM

The objective of this article is to diminish the count of individuals afflicted with Monkeypox. This is achieved through a series of preventive measures, including abstaining from contact with infected monkeys and humans, as well as administering vaccines to infected individuals. Two control variables, denoted as u(t) and v(t), play a pivotal role. These controls encompass an awareness program that involves disseminating information and education. The aim is to elevate awareness and understanding regarding the severity of the pandemic, its ramifications on human health, and strategies to mitigate infection rates.

$$\begin{cases} \frac{dS(t)}{dt} = \Lambda - \beta_1 \frac{S(t)A(t)}{N_h} (1 - u(t)) - \beta_2 \frac{S(t)I(t)}{N_h} (1 - u(t)) - \beta_3 \frac{S(t)I_a(t)}{N_h} (1 - v(t)) - \mu_1 S(t) \\ \frac{dE(t)}{dt} = \beta_1 \frac{S(t)A(t)}{N_h} (1 - u(t)) + \beta_2 \frac{S(t)I(t)}{N_h} (1 - u(t)) - \alpha_1 E(t) - \alpha_2 E(t) - \mu_2 E(t) \\ \frac{dA(t)}{dt} = \alpha_1 E(t) - n_1 A(t) - \gamma A(t) - \mu_3 A(t) \\ \frac{dI(t)}{dt} = \alpha_2 E(t) - \mu_4 I(t) - \theta I(t) + \gamma A(t) - n_2 I(t) + \beta_3 \frac{S(t)I_a(t)}{N_h} (1 - v(t)) \\ \frac{dH(t)}{dt} = -\sigma H(t) - \mu_5 H(t) + n_1 A(t) + n_2 I(t) \\ \frac{dR(t)}{dt} = \sigma H(t) - \mu_6 R(t) \\ \frac{dS_a(t)}{dt} = \Lambda_1 - \mu_7 S_a(t) - \beta_4 \frac{S_a(t)I_a(t)}{N_a} \\ \frac{dI_a(t)}{dt} = \beta_4 \frac{S_a(t)I_a(t)}{N_a} - \mu_8 I_a(t) - \beta_3 \frac{S(t)I_a(t)}{N_h} (1 - v(t)) \end{cases}$$

3.2. The optimal control problem

The issue is reducing the objective functional

$$J(u,v) = I(T) + I_a(T) + \int_0^T \left[I(t) + I_a(t) + \frac{A}{2}u^2(t) + \frac{B}{2}v^2(t) \right] dt$$

Where $A \ge 0$ and $B \ge 0$ are the cost coefficients. They are selected to weigh the relative importance of u(t) and v(t) at time t; T is the final time. In other words, we seek the optimal controls u^* and v^* such that

$$J(u^*, v^*) =_{u,v \in U} \min \left(J(u, v) \right)$$

where U is the set of admissible control defined by:

$$U = \{(u, v)/0 \le u_{\min} \le u(t) \le u_{\max} \le 1 \text{ and } 0 \le v_{\min} \le v(t) \le v_{\max} \le 1/t \in [0, T_f]\}$$

3.3. The optimal control: existence and characterization

Proof:

We commence by demonstrating the existence of solutions for the system (1), following which we will establish the presence of an optimal control. Let us consider the control problem within the framework of system (2).

There exists an optimal pair of controls, denoted as $J(u^*, v^*) \in U^2$, such that $J(u^*, v^*) =_{u,v \in U} \min(J(u, v))$.

Proof: The existence of the optimal control can be derived utilizing a result attributed to Fleming and Rishel [20]. This involves scrutinizing the subsequent steps:

We infer that the set comprising controls and their corresponding state variables is not devoid of elements. To accomplish this, we will invoke a simplified version of an existence theorem [21]. The convexity of the objective function J(u, v) in the control space U.

The control space $U = \{(u,v)/(u,v) \text{ is measurable...} (the continuation of the sentence is not provided; kindly supply the missing information).$

 $0 \le u_{\min} \le u(t) \le u_{\max} \le 1, 0 \le v_{\min} \le v(t) \le v_{\max} \le 1 \text{ and } /t \in [0, T_f] \}$

is convex and closed by definition.

all the right hand sides of equation of system are continuous, bounded above by a sum of bounded control and state, and can be written as a linear function of u,v and w with coefficients depending on time and state. the integrate in the objective functional is Creally convexe on U

$$I(T) + I_a(T) + \frac{A}{2}u^2(t) + \frac{B}{2}v^2(t)$$

It remains to demonstrate the existence of constants and that they satisfy certain conditions. By referencing the work of Fleming and Rishel [20], we can then infer the existence of an optimal control..

Proof: The Hamiltonian is defined as follows

$$H = I(T) + I_a(T) + \frac{A}{2}u^2(t) + \frac{B}{2}v^2(t) + \sum_{i=1}^{11}\lambda_i(t) \cdot f_i(S, E, A, I, H, R, S_a, I_a)$$

where f_i is the right optimal controls (u^*, v^*) and the solutions $S^*, E^*, I^*, H^*, V^*, R^*, S_a^*$ and I_a^* of the corresponding state (2).

There exists adjoint variables λ_1' , and λ_8' satisfying

Then from Fleming and Rishel [20] we conclude that there exists an optimal control.

$$\begin{split} f_1(S, E, A, I, H, R, S_a, I_a) \\ &= \Lambda - \beta_1 \frac{S(t)A(t)}{N_h} (1 - u(t)) - \beta_2 \frac{S(t)I(t)}{N_h} (1 - u(t)) - \beta_3 \frac{S(t)I_a(t)}{N_h} (1 - v(t)) - \mu_1 S(t) \\ f_2(S, E, A, I, H, R, S_a, I_a) \\ &= \beta_1 \frac{S(t)A(t)}{N_h} (1 - u(t)) + \beta_2 \frac{S(t)I(t)}{N_h} (1 - u(t)) - \alpha_1 E(t) - \alpha_2 E(t) - \mu_2 E(t) \\ f_3(S, E, A, I, H, R, S_a, I_a) = \alpha_1 E(t) - n_1 A(t) - \gamma A(t) - \mu_3 A(t) \\ f_4(S, E, A, I, H, R, S_a, I_a) = \alpha_2 E(t) - \mu_4 I(t) - \theta I(t) + \gamma A(t) - n_2 I(t) + \beta_3 \frac{S(t)I_a(t)}{N_h} (1 - v(t)) \end{split}$$

$$\begin{split} f_5(S, E, A, I, H, R, S_a, I_a) &= -\sigma H(t) - \mu_5 H(t) + n_1 A(t) + n_2 I(t) \\ f_6(S, E, A, I, H, R, S_a, I_a) &= \sigma H(t) - \mu_6 R(t) \\ f_7(S, E, A, I, H, R, S_a, I_a) &= \Lambda_1 - \mu_7 S_a(t) - \beta_4 \frac{S_a(t) I_a(t)}{N_a} \\ f_8(S, E, A, I, H, R, S_a, I_a) &= \beta_4 \frac{S_a(t) I_a(t)}{N_a} - \mu_8 I_a(t) - \beta_3 \frac{S(t) I_a(t)}{N_h} (1 - v(t)) \end{split}$$

We have according to the theorem of Pontryagine [25, 26, 27, 28, 29].

$$\begin{split} \lambda_1' &= -\frac{\partial H}{\partial S} = \left(\beta_1 \frac{A}{N_h} + \beta_2 \frac{I}{N_h}\right) (1 - u(t)) \left(\lambda_1 - \lambda_2\right) + \beta_3 \frac{I_a}{N_h} \left(1 - v(t)\right) \left(\lambda_1 - \lambda_4 + \lambda_8\right) + \lambda_1 \mu_1 \\ \lambda_2' &= -\frac{\partial H}{\partial E} = \alpha_1 (\lambda_1 - \lambda_3) + \alpha_2 (\lambda_1 - \lambda_4) + \lambda_1 \mu_2 \\ \lambda_3' &= -\frac{\partial H}{\partial A} = \beta_1 \frac{S}{N_h} \left(1 - v(t)\right) \left(\lambda_1 - \lambda_2\right) + \gamma (\lambda_3 - \lambda_4) + \lambda_3 n_2 + \lambda_3 \mu_3 - \lambda_5 n_1 \\ \lambda_4' &= -\frac{\partial H}{\partial I} = -1 + \beta_2 \frac{S}{N_h} \left(1 - u(t)\right) \left(\lambda_1 - \lambda_2\right) + n_2 (\lambda_4 - \lambda_5) + \lambda_3 (\mu_4 + \theta) \\ \lambda_5' &= -\frac{\partial H}{\partial H} = \sigma (\lambda_5 - \lambda_6) + \lambda_5 \mu_5 \\ \lambda_6' &= -\frac{\partial H}{\partial R} = \lambda_6 \mu_6 \\ \lambda_7' &= -\frac{\partial H}{\partial I_a} = -1 + \beta_3 \frac{S}{N} \left(1 - v(t)\right) \left(\lambda_1 - \lambda_4 + \lambda_8\right) + \beta_4 \frac{S_a}{N_h} (\lambda_7 - \lambda_8) + \lambda_8 \mu_8 \\ \text{for } t \in [0, T_f] \text{ the optimal control } u^* and v^* \text{ can be solved from the optimality condition [22]. \end{split}$$

that are

$$-\frac{\partial H}{\partial u} = Au + \lambda_1 \left(\beta_1 \frac{S(t)A(t)}{N_h} + \beta_2 \frac{S(t)I(t)}{N_h} \right) + \lambda_2 \left(-\beta_1 \frac{S(t)A(t)}{N_h} - \beta_2 \frac{S(t)I(t)}{N_h} \right) = 0$$

$$-\frac{\partial H}{\partial v} = Bv + \lambda_1 \left(\beta_3 \frac{S(t)I_a(t)}{N_h} \right) + \lambda_4 \left(-\beta_3 \frac{S(t)I_a(t)}{N_h} \right) + \lambda_8 \left(\beta_3 \frac{S(t)I_a(t)}{N_h} \right)$$

we have

$$u = \frac{(\lambda_2 - \lambda_1)}{A} \left(\beta_1 \frac{S(t)A(t)}{N_h} + \beta_2 \frac{S(t)I(t)}{N_h} \right)$$
$$v = \frac{(\lambda_4 - \lambda_1 - \lambda_8)}{B} \left(\beta_3 \frac{S(t)I_a(t)}{N_h} \right)$$

4. SIMULATION

Within this section, we engage in a numerical analysis of the optimal control model (6), delving into the ramifications brought about by variations in controls u and v. Initially, we employ the parameter values outlined in Table 1. Our control problem incorporates initial conditions for state variables and terminal conditions for adjuncts. Thus, the optimality system manifests as a two-point boundary value predicament with distinctive boundary conditions at time steps i=0 and i=T. We address this optimization system iteratively, proceeding with the forward solution of the state system, followed by the backward resolution of the auxiliary system. The iterative process commences with an initial estimate of the controls during the first iteration. Subsequently, prior to each successive iteration, we refine the controls based on characterization. This iterative cycle is sustained until the convergence of consecutive iterations is achieved.



Parameter	Value	Source
Λ	30	Assumed
eta_1	0.60	Assumed
β_2	0.60	Assumed
β_3	0.63	assumed
eta_4	0.23	Assumed
σ	0.28	Assumed
μ	0.02	23
n_1	0.008	Assumed
<i>n</i> ₂	0.08	Assumed
γ	0.005	Assumed
Λ_1	20	Assumed
α_1	0.10	Assumed
α_2	0.20	Assumed
θ	0.08	24

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

4.1. DISCUSSION

Within this segment, we undertake a numerical examination of the control effects. This encompasses interventions such as isolating individuals afflicted with monkeypox, directing them towards quarantine within designated healthcare facilities, promoting thorough hand hygiene with soap and water, ensuring cleanliness, advocating against sexual encounters between males, and discouraging direct contact with monkeys. Varied simulations can be conducted, utilizing diverse parameter values. In this endeavor, we employ the optimal control strategies along with their frequencies, substantiating their efficacy numerically via reference to Table (1).

4.2. Strategy 1: protecting and preventing susceptible individuals from contacting the infected individuals

In pursuit of this strategy, we exclusively employ the prescribed controls on susceptible individuals. This is executed through the implementation of awareness campaigns geared towards 14 MOHAMED BAROUDI, HICHAM GOURRAM, ABDERRAHIM LABZAI, MOHAMED BELAM

shielding individuals from viral infection. Health measures and security campaigns are also enacted to curtail individual movement and provide guidance. As demonstrated in Figure (2), the implementation of the proposed strategy yields a decrease in the count of exposed individuals, reducing from 62 to 9 cases by the strategy's culmination. This substantial decline underscores the strategy's efficacy.

4.3. STRATEGY **2:** PROTECT SUSCEPTIBLE INDIVIDUALS FROM CONTACT WITH ANI-MALS (MONKEYS) THAT CARRY THE VIRUS

Our approach to curbing infection rates involves safeguarding susceptible individuals from contact with animals, specifically monkeys, that harbor the virus. To effectively diminish the count of infected individuals, we employ a meticulous strategy. This strategy entails the deployment of preventive measures, encompassing health protocol initiatives and awareness campaigns. These efforts are focused on endemic regions to shield vulnerable individuals from encounters with infected individuals and animals, particularly in regions where the ailment prevails. The impact of this strategy is evident in Figure (3), which illustrates a reduction in the number of infected individuals from 170 to 122 following the implementation of the proposed approach.

4.4. THE THIRD STRATEGY: PROTECTING VULNERABLE INDIVIDUALS, PREVENTING THEM FROM CONTACTING INFECTED PEOPLE WHO DID NOT SHOW SYMPTOMS, AND DIRECTING THEM TO JOIN QUARANTINE CENTERS.

In pursuit of this strategy, we exclusively implement the prescribed controls. This involves launching awareness campaigns designed to shield individuals from viral infection. Moreover, we enact health measures and security campaigns to impede the transfer of individuals from potentially hazardous regions to other areas. Additionally, we encourage asymptomatic carriers of the virus to opt for quarantine centers. Figure (4) offers a clear depiction of the strategy's effectiveness. The count of asymptomatic virus carriers decreases from 380 to 70 by the strategy's culmination, corroborating the efficacy of the aforementioned approach.

5. CONCLUSION

This study delved into the realm of monkeypox disease through the lens of a mathematical model. The primary objective was to scrutinize the impact of a suite of optimal control strategies. These strategies encompassed urging individuals to practice regular hand hygiene, avoiding contact with infected individuals and monkeys, and mandating the utilization of masks. Treatment of Monkeypox patients and their subsequent referral to hospitals and designated quarantine facilities also formed integral aspects of these strategies. Commencing with a comprehensive introduction and an overview of pertinent literature, we established a dedicated mathematical model delineating the dynamics of the monkeypox-infected population, spanning both asymptomatic and symptomatic cases. The overarching goal remained the reduction of infected cases throughout various stages of monkeypox. By leveraging principles from control theory, we derived optimal control descriptions. This endeavor culminated in the numerical solution of the mathematical model, utilizing the discrete-time Pontryagin's principle of maximum. The optimal system's resolution followed an iterative trajectory. Additionally, we conducted an inquiry into the cost-effectiveness associated with these implemented controls.

CONFLICT OF INTERESTS

The authors declare that there is no conflict of interests.

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