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STRATEGIC APPROACHES TO SOCIAL DISTANCING AND TESTING FOR CONTROLLING THE MPOX OUTBREAK

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Abstract. Thousands of people have contracted Mpox, and it has resulted in thousands of deaths globally. Several nations have enacted testing laws that include contact tracing and social distancing to slow the spread of the continuing outbreak. Considering the significant social and financial consequences, achieving the best possible control over infection and therapies is critical. We created a mathematical model of Mpox transmission. We used optimal control theory to establish the best way to minimize the cost of reducing the epidemiological burden of Mpox to select the optimum social distancing and testing procedures. The findings show that, while testing alone would be an effective method, it would not significantly affect the eventual size of an epidemic. Instead, it would cause the pandemic to peak later. If social distancing is the only control method, the best action would be to progressively raise the degree of social distancing as the Mpox incidence curve rises and then lighten up on the measures once the curve has peaked. It has been shown that combining social distancing and testing tactics is more effective than using either tactic alone to lower the disease burden and delay the disease's peak. To maximize the effectiveness of these strategies, social separation should be increased when the disease's prevalence exceeds 15%. At the same time, testing should be kept at its highest level during the epidemic's early stages and after its peak. As a result, public health organizations ought to start testing early and transition to social distancing as soon as the incidence rises. It would be best to progressively ease social separation and return to testing after the pandemic's peak.

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1. Introduction

Globally, the economy and public health have been significantly impacted by the current Mpox outbreak, though the effects have been less severe than in other recent global health crises. Thousands of Mpox cases and fatalities had been documented as of July 23, 2022 [13, 18]. With an estimated basic reproduction number ranging from 1.0 to 1.5, the Mpox virus, which causes the ongoing outbreak, is not

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as highly contagious as some respiratory viruses but spreads through close physical contact, including skin lesions and bodily fluids [1,2,9,14]. Furthermore, pre-symptomatic or asymptomatic individuals have been suggested to be responsible for between 40% and 80% of Mpox infections. This indicates that a significant number of infectious individuals who are unknown to others may continue to expose them without realizing it [12,19] Although the Mpox vaccine availability is still restricted in many nations, there have been urgent attempts undertaken to prevent and minimize transmission worldwide as Mpox illnesses have surged dramatically. However, non-pharmaceutical measures have been taken to slow down the Mpox pandemic or at least contain it long enough to allow for the development and application of vaccinations and treatments. Social distancing [8,26,28], testing [4,23,27], donning face masks [10,22], and travel restrictions [20,21] are some examples of these non-pharmaceutical public health interventions.

Due to its ease of implementation and lack of reliance on microbiological data, social distancing has been chosen by many countries as the primary mitigation strategy for Mpox transmission [8,12,19,26]. Many previously unheard-of social distancing measures, such as lockdowns and prohibitions on large gatherings, have been implemented as the number of confirmed Mpox cases was significantly increasing [1]. Implementing social distancing treatments was linked to an overall decrease in the risk of infection, according to recent studies [3,5,8,16,28].

Contact, tracing and testing is a key non-pharmaceutical technique for reducing the transmission of Mpox. Community infections can be avoided by screening and isolating infected persons before they spread the virus. United States, for instance, has implemented extensive testing programs that include drive-through testing stations, an investigation of infection-related clusters, nursing homes, and social welfare establishments [6,10,15,17,23]. Approximately 1500 people in 2022 were tested daily in the United States when the daily confirmed case count was roughly 100. When social separation was subsequently relaxed, the number of new cases increased again. Still, this technique successfully reduced the daily new case count to less than 10 between November and December 2022 [4]. Furthermore, a few studies have shown that identifying and isolating infected individuals before they come into contact with others can help reduce the number of infections [2,19,23,27].

Even though these decisive actions should successfully stop the spread of Mpox, they may also significantly disrupt society and cause economic loss [21]. Furthermore, non-pharmaceutical therapies have the potential to decelerate the spread of the disease and seem to be effective in decreasing the number of confirmed cases; but, if these interventions are discontinued, the infection may rebound quickly [11,25,28]. It is crucial to identify the best mitigation and containment techniques to limit the possible Mpox pandemic transmission risk and strike an acceptable balance between economic and public health goals.

In this work, we created a mathematical model of Mpox transmission that takes testing and social distancing into account over time. The model was then subjected to the theory of optimal control in order to determine the best course of action for different epidemiological scenarios. In particular, we determined the best practices that reduce the expenses of infection and intervention in situations where either a single control or a combination of controls is available.

2. Model setup

As intervention techniques, we built a mathematical model of Mpox transmission that took social distancing and testing into account. We assumed all the people who tested positive were either hospitalized or in isolation. According to Table 1, the model divides people into four categories: susceptible (S), exposed (E), infectious $(P_1, P_2, I_1, I_2, I_3, I_s, I_h)$, and recovered (R). Infectious people are further separated into seven categories depending on how isolated they are and how they feel. In particular, if pre-symptomatic and tested people acquire none-to-mild or severe symptoms, respectively, they move into the class I_2 or I_h and are isolated (P_2). Similarly, pre-symptomatic persons in the non-isolated class I_1 or I_s if they develop none to-mild or severe symptoms, respectively, are included in this group if they are not (yet) isolated (P_1). It's also assumed for simplicity's sake that those with severe symptoms who haven't been tested will wind up in a hospital. In other words, those in I_s will eventually check into a hospital and become members of I_h. Likewise, persons in P_1 who are pre-symptomatic and have not been tested will be moved to I_1 if they experience none-to-mild symptoms.

Table 1. Description of the model variables

Variable	Description
S	Individuals who are susceptible
E	Individuals who are exposed
P_1	Pre-symptomatic individuals who haven't been tested yet
P_2	Isolated pre-symptomatic and tested individuals
I_1	Mild infectious disease individuals not tested
I_2	Isolated and tested individuals with mild infectious diseases
I_3	Isolated individuals with mild infectious diseases
I_s	Extremely infectious individuals not yet been hospitalized
I_h	Hospitalized infectious individuals due to severe symptoms
R	Recovered individuals

According to recent reports, none-to-mild symptoms are present in about 80% of confirmed Mpox individuals [7, 18]. It is highlighted that the classes I_1, I_2, and I_3 in the proposed model comprise infectious individuals with none-to-mild symptoms. Additionally, we presume that pre-symptomatic people and infected people with no, mild, or severe symptoms are equally contagious [4, 23]. It follows that vulnerable people are thought to contract the infection at a specific rate.

$$\frac{\beta_0(1-\alpha)\left(P_1+I_1+I_s+(1-\theta)\left(P_2+I_2+I_3+I_h\right)\right)}{N-\theta\left(P_2+I_2+I_3+I_h\right)}$$

where $N(t) = S(t)+ E(t) + P_1(t) + P_2(t) + I_1(t) + I_2(t) + I_3(\mathrm{t}) + I_s(t) + I_h(t) + R(\mathrm{t})$ represents the total population size. Given the short time scale of this study, births, and deaths are disregarded, resulting in an asymptotically constant population (i.e., $\lim_{t\to\infty} N(t) = K$). The model's θ represents how well hospitalization and isolation work to stop the spread of infection ($0 \leq \theta \leq 1$). In other words, when $\theta = 0$, individuals hospitalized or secluded due to infection can interact with the general community. Conversely, $\theta = 1$ denotes the complete effectiveness of hospitalization and isolation. Consequently, (1-θ) quantifies the amount that hospitalized and isolated infected individuals contribute to the overall infection force [4]. In this case, the transmission rate is represented by β_0. To account for social distancing, we assume that susceptible people lower their rate of contact by a fraction $\alpha(t)$ $(0 \leq \alpha(t) \leq \alpha_{\max} < 1)$.

The average time between the beginning of symptoms and diagnosis for an infected individual with none-to-mild symptoms is $1/\psi_m$; for a severe infection, it is $1/\psi_s$. In addition, the latent period is specified as $1/\delta$, and the infectious period before symptom manifestation is indicated by $1/k$. v represents the percentage of highly contagious people who will require hospitalization. Furthermore, it is assumed that the recovery rate for persons with moderate infection is μ_M. The values of $1/\mu_{\text{iso}}$ and $1/\mu_{\text{hosp}}$ represent the average length of hospitalization and isolation, respectively.

By assuming that exposed people are segregated with a probability $\sigma(t)$, where $0 \leq \sigma(t) \leq \sigma_{\max} < 1$, based on their test results, we integrate testing with contact tracing into the model. Testing intensity determines the likelihood of finding exposed individuals. Those who have recovered are believed to be immune to contracting the disease again for the duration of the outbreak. Table 2 lists the baseline values for the epidemiological parameters.

The following ordinary differential equations can be used to define a transmission dynamic model that includes the time-dependent control parameters $\alpha(t)$ and $\sigma(t)$, given the definitions and presumptions covered above:

$$\begin{cases} \frac{dS}{dt} &= -(1-\alpha(t))\lambda(t)S(t) \\ \frac{dE}{dt} &= (1-\alpha(t))\lambda(t)S(t) - \delta E(t) \\ \frac{dP_1}{dt} &= \delta(1-\sigma(t))E(t) - kP_1(t) \\ \frac{dP_2}{dt} &= \delta\sigma(t)E(t) - kP_2(t) \\ \frac{dI_1}{dt} &= k(1-v)P_1(t) - (\psi_m + \mu_M)\, I_1(t) \\ \frac{dI_2}{dt} &= k(1-v)P_2(t) - \mu_{iso}I_2(t) \\ \frac{dI_3}{dt} &= \psi_m I_1(t) - \mu_{iso}I_3(t) \\ \frac{dI_s}{dt} &= kvP_1(t) - \psi_s I_s(t) \\ \frac{dI_h}{dt} &= kvP_2(t) + \psi_s I_s(t) - \mu_{\text{hosp}}\, I_h(t) \\ \frac{dR}{dt} &= \mu_M I_1(t) + \mu_{\text{iso}}\, I_2(t) + \mu_{\text{iso}}\, I_3(t) + \mu_{\text{hosp}}\, I_h(t) \end{cases}$$

where the force of infection $\lambda(t)$ is defined as

$$\lambda(t) = \frac{\beta_0\left(P_1 + I_1 + I_s + (1-\theta)\left(P_2 + I_2 + I_3 + I_h\right)\right)}{N - \theta\left(P_2 + I_2 + I_3 + I_h\right)}$$

.

TABLE 2. Definitions of parameters and their baseline values used in numerical simulations

Parameter	Description	Value
$\mathscr{R}_0$	Basic reproduction number	2.8
β_0	Transmission rate	0.50
α	Social distancing level	control
σ	Probability of testing and isolating pre-symptomatic individuals	control
$1/\delta$	Latent period duration (days)	3.1
$1/k$	Pre-symptomatic infectious period (days)	2
v	Percentage of severely infectious individuals hospitalized	0.2
$1/\mu_M$	Mild infection infectious period (days)	7
$1/\mu_{iso}$	Average isolation duration for mild infections (days)	10
$1/\mu_{hosp}$	Average hospital stay for severe infections (days)	14.5
$1/\psi_s$	Average time from symptom onset to diagnosis in severe cases (days)	4
$1/\psi_m$	..... to diagnosis in mild or asymptomatic cases (days)	4
θ	Efficacy of hospitalization and isolation in preventing further transmission	0.9
χ_T	Average tests needed to identify one infected person	10
π_S	Social distancing's cost (USD)	178
π_T	Cost of Mpox diagnostic testing (USD)	142
π_I	Treatment costs for a hospitalized Mpox sufferer (USD)	25,617
π_M	Treatment costs for people with mild cases of Mpox (USD)	3,045
$\alpha_{\max}$	The maximum level of social distancing	0.5
$\sigma_{\max}$	Testing intensity upper bound	0.2

Since the variable $R(t)$ only appears in dR/dt in the model, we find it after solving for the other classes. In addition, we use dimensionless variables $\left(s = \frac{S}{\mathrm{K}}, e = \frac{E}{\mathrm{K}}, p_1 = \frac{P_1}{K}, p_2 = \frac{P_2}{K}, m_1 = \frac{I_1}{K}, m_2 = \frac{I_2}{K}, g = \frac{\pi}{K}, i_1 = \frac{l_1}{K}, i_2 = \frac{l_2}{K}\right)$ and present the following simplified model:

$$\begin{cases} \frac{ds}{dt} = -(1-\alpha(t))\lambda(t)s(t) \\ \frac{de}{dt} = (1-\alpha(t))\lambda(t)s(t) - \delta e(t) \\ \frac{dp_1}{dt} = \delta(1-\sigma(t))e(t) - kp_1(t) \\ \frac{dp_2}{dt} = \delta\sigma(t)e(t) - kp_2(t) \\ \frac{dm_1}{dt} = k(1-v)p_1(t) - (\psi_m + \mu_M)\, m_1(t) \\ \frac{dm_2}{dt} = k(1-v)p_2(t) - \mu_{iso}m_2(t) \\ \frac{dg}{dt} = \psi_m m_1(t) - \mu_{iso}g(t) \\ \frac{di_1}{dt} = kvp_1(t) - \psi_s i_1(t) \\ \frac{di_2}{dt} = kvp_2(t) + \psi_s i_1(t) - \mu_{\text{hosp}}\, i_2(t) \end{cases}$$

where

$$\lambda(t) = \frac{\beta_0\left(p_1 + m_1 + i_1 + (1-\theta)\left(p_2 + m_2 + g + i_2\right)\right)}{1 - \theta\left(p_2 + m_2 + g + +_i\right)}$$

2.1. **Basic reproduction number.** The disease-free equilibrium (DFE) of the model is

$$\text{DFE} = (s^*, e^*, p_1^*, p_2^*, m_1^*, m_2^*, g, i_1^*, i_2^*) = (1, 0, 0, 0, 0, 0, 0, 0)$$

The average number of secondary infections caused by an infected person when control measures are in place and constant over time ($\alpha(t) = \alpha$ and $\sigma(t) = \sigma$) is measured by the control reproduction number $\mathscr{R}_c(\alpha, \sigma)$. To calculate $\mathscr{R}_c(\alpha, \sigma)$, we employ the next-generation operator approach. In particular, we use the new infection and transition terms to define the matrices $\mathscr{F}$ and $\mathscr{V}$, respectively:

$$\mathscr{F} = \begin{bmatrix} (1-\alpha)\lambda(t)s(t) \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}$$

$$\mathscr{V} = \begin{bmatrix} \delta e(t) \\ -\delta(1-\sigma)e(t) + kp_1(t) \\ -\delta\sigma e(t) + kp_2(t) \\ -k(1-v)p_1(t) + (\psi_m + \mu_M)\, m_1(t) \\ -k(1-v)p_2(t) + \mu_{is} m_2(t) \\ -\psi_m m_1(t) + \mu_{isg}(t) \\ -kvp_1(t) + \psi_s i_1(t) \\ -kvp_2(t) - \psi_s i_1(t) + \mu_{hosp} i_2(t) \end{bmatrix}.$$

We linearize the matrices $\mathscr{F}$ and $\mathscr{V}$ at the DFE as follows:

$$F = \beta_0(1-\alpha) \begin{bmatrix} 0 & 1 & (1-\theta) & 1 & (1-\theta) & (1-\theta) & 1 & (1-\theta) \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

and

$$V = \begin{bmatrix} \delta & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ -\delta(1-\sigma) & k & 0 & 0 & 0 & 0 & 0 & 0 \\ -\delta\sigma & 0 & k & 0 & 0 & 0 & 0 & 0 \\ 0 & -k(1-v) & 0 & \psi_m + \mu_M & 0 & 0 & 0 & 0 \\ 0 & 0 & -k(1-v) & 0 & \mu_{\text{iso}} & 0 & 0 & 0 \\ 0 & 0 & 0 & -\psi_m & 0 & \mu_{\text{iso}} & 0 & 0 \\ 0 & -kv & 0 & 0 & 0 & 0 & \psi_s & 0 \\ 0 & 0 & -kv & 0 & 0 & 0 & -\psi_s & \mu_{\text{hosp}} \end{bmatrix}.$$

Then, the control reproduction number $\mathscr{R}_c(\alpha, \sigma)$ is defined by the spectral radius of the next generation matrix FV^{-1} as

$\mathscr{R}_c(\alpha, \sigma) = \mathscr{R}_{Pc} + \mathscr{R}_{Mc} + \mathscr{R}_{Ic}$,

where

$\mathscr{R}_{Pc} = \beta_0(1-\alpha)\left[\frac{1-\sigma}{k} + \frac{(1-\theta)\sigma}{k}\right]$,

$\mathscr{R}_{Mc} = \beta_0(1-\alpha)\left[\frac{(1-\sigma)(1-\nu)}{\psi_m+\mu_M} + \frac{(1-\theta)\sigma(1-\mu)}{\mu_{\text{so}}} + \frac{(1-\theta)(1-\sigma)(1-v)\psi_m}{\mu_{\text{io}}\,(\psi_m+\mu_M)}\right]$, and

$\mathscr{R}_{I\pi} = \beta_0(1-\alpha)\left[\frac{(1-\sigma)v}{\psi_s} + \frac{(1-\theta)v}{\mu_{\text{hosp}}}\right]$.

The total number of reproduction numbers linked to the number of new Mpox cases caused by presymptomatic people ($\mathscr{R}_{\text{Pc}}$), mildly infectious people ($\mathscr{R}_{M\pi}$), and severely infectious people ($\mathscr{R}_{1\pi}$) is equal to $\mathscr{R}_c$. Fig. 2 displays the level curve for the control reproduction number ($\mathscr{R}_c$). In the absence of control measures, the basic reproduction number $mathscrR_0$ represents the average number of secondary infections that result from introducing one infected individual into the whole vulnerable population. It is defined as

$$\mathscr{R}_0 = \mathscr{R}_c(0,0) = \beta_0 \left[\frac{1}{k} + \frac{1-v}{\psi_m + \mu_M} + \frac{(1-\theta)(1-v)\psi_m}{\mu_{\text{iso}}\ (\psi_m + \mu_M)} + \frac{v}{\psi_s} + \frac{(1-\theta)v}{\mu_{\text{hosp}}}\right]$$

2.2. **Sensitivity analysis.** Now, we examine the sensitivity of the model results to changes in the following parameters: transmission rate (β_0), rate of progression to symptomatic stage (k), percentage of hospitalized severely infectious individuals (v), rate of recovery for mild cases (μ_M), and rate of diagnosis (ψ_m and ψ_s). Now, we explore the sensitivity of the model outcomes to changes in these parameters. We examined variations in the epidemic curve of infectious people and altered the transmission rate β_0 for sensitivity analysis. It has been demonstrated that the attack rate rises with increased transmissibility. To ascertain the lowest and maximum values of $\mathscr{R}_0$, we additionally varied each parameter separately (apart from β_0) within the 95% confidence interval of its uncertainty distribution (Fig.1). According to the simulation, the proportion of hospitalized extremely infectious persons (v) is the least influential parameter, whereas the rate of progression to symptomatic stage (k) is the most influential.

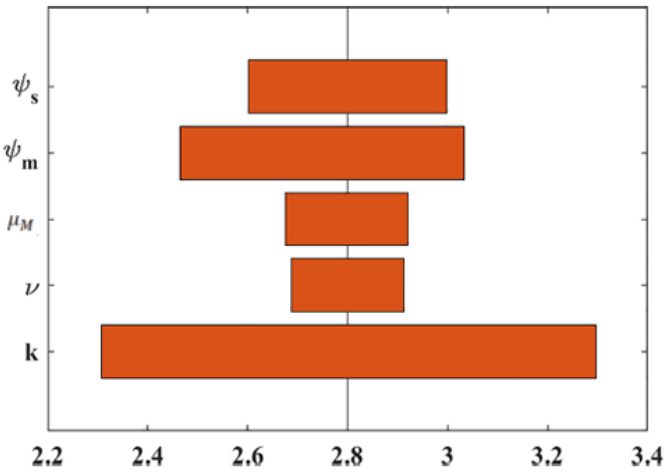

FIGURE 1. *Local sensitivity analysis of parameter contributions to the fundamental reproduction number uncertainty in a single direction. The fundamental reproduction number under baseline conditions is shown by the vertical line.*

2.3. **Dynamics with optimal control.** To reduce the epidemiological burden and control costs associated with Mpox transmission, we employ optimal control theory. In this study, two time-dependent control variables are introduced: $u_1(t)$, representing social distancing, and $u_2(t)$, representing testing. The control $u_1(t)$ reduces the effective contact rate, while $u_2(t)$ enhances the detection and isolation of infected individuals.

The objective functional to be minimized is given by

$$\begin{aligned}\mathscr{F}(u_1(t), u_2(t)) = \int_0^T \Big\{ & \pi_M \left(k(1-v)p_2(t) + \psi_m m_1(t)\right) \\ & + \pi_I \left(kvp_2(t) + \psi_s i_1(t)\right) \\ & + \pi_S u_1^2(t)s(t) + \pi_T \chi_T \delta e(t) u_2^2(t) \Big\} dt.\end{aligned}$$

The quadratic terms in $u_1(t)$ and $u_2(t)$ represent the implementation costs of social distancing and testing, respectively, where π_S and π_T denote the associated cost weights. The parameters π_M and π_I represent the costs associated with isolation of mild cases and hospitalization of severe cases, respectively. The parameter χ_T denotes the average number of tests required to identify one infected individual.

The optimal control functions $u_1^*(t)$ and $u_2^*(t)$ are obtained by solving

$$\mathscr{F}(u_1^*, u_2^*) = \min_{\Theta} \mathscr{F}(u_1, u_2),$$

where the admissible control set is defined as

$$\Theta = \left\{ (u_1, u_2) \in L^1(0,T) \mid 0 \le u_1(t) \le u_{1\max},\ 0 \le u_2(t) \le u_{2\max} \right\}.$$

To solve this optimal control problem, we apply Pontryagin's Maximum Principle. The associated Hamiltonian is defined as

$$\begin{aligned}H = & \pi_M \left(k(1-v)p_2(t) + \psi_m m_1(t)\right) + \pi_I \left(kvp_2(t) + \psi_s i_1(t)\right) \\ & + \pi_S u_1^2(t)s(t) + \pi_T \chi_T \delta e(t) u_2^2(t) \\ & + \gamma_s\{-(1-u_1)\lambda(t)s(t)\} + \gamma_e\{(1-u_1)\lambda(t)s(t) - \delta e(t)\} \\ & + \gamma_{p_1}\{\delta(1-u_2)e(t) - kp_1(t)\} + \gamma_{p_2}\{\delta u_2 e(t) - kp_2(t)\} \\ & + \gamma_{m_1}\{k(1-v)p_1(t) - (\psi_m + \mu_M)m_1(t)\} \\ & + \gamma_{m_2}\{k(1-v)p_2(t) - \mu_{iso} m_2(t)\} \\ & + \gamma_g\{\psi_m m_1(t) - \mu_{iso} g(t)\} \\ & + \gamma_{i_1}\{kvp_1(t) - \psi_s i_1(t)\} \\ & + \gamma_{i_2}\{kvp_2(t) + \psi_s i_1(t) - \mu_{hosp} i_2(t)\}.\end{aligned}$$

The adjoint system is given by

$$\frac{d\gamma_x}{dt} = -\frac{\partial H}{\partial x}, \quad x \in \{s, e, p_1, p_2, m_1, m_2, g, i_1, i_2\}.$$

The transversality conditions are

$$\gamma_s(T) = \gamma_e(T) = \gamma_{p_1}(T) = \gamma_{p_2}(T) = \gamma_{m_1}(T) = \gamma_{m_2}(T) = \gamma_g(T) = \gamma_{i_1}(T) = \gamma_{i_2}(T) = 0.$$

The optimal controls are obtained from the conditions

$$\frac{\partial H}{\partial u_1} = 0, \qquad \frac{\partial H}{\partial u_2} = 0.$$

Thus, the optimal control functions are

$$u_1^*(t) = \min\left\{\max\left\{0, \frac{\beta_0(\gamma_e - \gamma_s)\left[p_1 + m_1 + i_1 + (1-\theta)(p_2 + m_2 + g + i_2)\right]}{2\pi_S\left[1 - \theta(p_2 + m_2 + g + i_2)\right]}\right\}, u_{1\max}\right\},$$

and

$$u_2^*(t) = \min\left\{\max\left\{0, \frac{\gamma_{p_1} - \gamma_{p_2}}{2\pi_T \chi_T}\right\}, u_{2\max}\right\}.$$

2.3.1. *Single-strategy model with social distancing.* We consider the case where social distancing is the only control strategy, i.e., $u_2(t) = 0$. The model reduces to

$$\begin{cases} \frac{ds}{dt} = -(1-u_1(t))\lambda(t)s(t) \\ \frac{de}{dt} = (1-u_1(t))\lambda(t)s(t) - \delta e(t) \\ \frac{dp_1}{dt} = \delta e(t) - k p_1(t) \\ \frac{dm_1}{dt} = k(1-v)p_1(t) - (\psi_m + \mu_M)m_1(t) \\ \frac{dg}{dt} = \psi_m m_1(t) - \mu_{iso} g(t) \\ \frac{di_1}{dt} = kvp_1(t) - \psi_s i_1(t) \\ \frac{di_2}{dt} = \psi_s i_1(t) - \mu_{hosp} i_2(t) \end{cases}$$

with

$$\lambda(t) = \frac{\beta_0\left(p_1 + m_1 + i_1 + (1-\theta)(g + i_2)\right)}{1 - \theta(g + i_2)}.$$

The control reproduction number becomes

$$\mathscr{R}_c(u_1, 0) = (1 - u_1)\mathscr{R}_0.$$

The objective functional simplifies to

$$\mathscr{F}(u_1) = \int_0^T \left\{\pi_M \psi_m m_1(t) + \pi_I \psi_s i_1(t) + \pi_S u_1^2(t) s(t)\right\} dt.$$

2.3.2. *Single-strategy model with testing.*

2.3.3. *Single-strategy model with testing.* We now consider the case where testing is the only control strategy, i.e., $u_1(t) = 0$. The model reduces to

$$\begin{cases} \frac{ds}{dt} = -\lambda(t)s(t) \\ \frac{de}{dt} = \lambda(t)s(t) - \delta e(t) \\ \frac{dp_1}{dt} = \delta(1-u_2(t))e(t) - kp_1(t) \\ \frac{dp_2}{dt} = \delta u_2(t)e(t) - kp_2(t) \\ \frac{dm_1}{dt} = k(1-v)p_1(t) - (\psi_m + \mu_M)m_1(t) \\ \frac{dm_2}{dt} = k(1-v)p_2(t) - \mu_{iso}m_2(t) \\ \frac{dg}{dt} = \psi_m m_1(t) - \mu_{iso}g(t) \\ \frac{di_1}{dt} = kvp_1(t) - \psi_s i_1(t) \\ \frac{di_2}{dt} = kvp_2(t) + \psi_s i_1(t) - \mu_{hosp}i_2(t) \end{cases}$$

The control reproduction number is

$$\begin{aligned} \mathscr{R}_{u_2} = \beta_0 \Bigg[& \frac{1-u_2}{k} + \frac{(1-\theta)u_2}{k} + \frac{(1-u_2)(1-v)}{\psi_m + \mu_M} \\ & + \frac{(1-\theta)u_2(1-v)}{\mu_{iso}} + \frac{(1-\theta)(1-u_2)(1-v)\psi_m}{\mu_{iso}(\psi_m + \mu_M)} \\ & + \frac{(1-u_2)v}{\psi_s} + \frac{(1-\theta)v}{\mu_{hosp}} \Bigg]. \end{aligned}$$

The objective functional becomes

$$\begin{aligned} \mathscr{F}(u_2) = \int_0^T \Big\{ & \pi_M \left(k(1-v)p_2(t) + \psi_m m_1(t)\right) \\ & + \pi_I \left(kvp_2(t) + \psi_s i_1(t)\right) + \pi_T \chi_T \delta e(t) u_2^2(t) \Big\} dt. \end{aligned}$$

3. Numerical simulation

Based on the suggested mathematical model, we offer numerical simulations of the best intervention tactics to stop the spread of Mpox. We examined both single and combination control situations to examine the financial limitations and accessibility of intervention strategies. Unless otherwise noted, we assumed that the control intervention started 50 days following the first Mpox case and utilized the values from Table 1 as baseline parameters for the simulations [14]. With these baseline parameter values and no control measure, i.e., $\alpha(t) = \sigma(t) = 0$, the cumulative proportion of highly infectious individuals reached 19% under the assumption that $\mathscr{R}_0 = 2.8$. For sensitivity analysis, important model parameters including cost, upper bound of controls, and basic reproduction number are changed.

3.1. **Single strategy.**

3.1.1. *Optimal social distancing strategy.* We investigated the impact of optimal social distancing strategies on the transmission dynamics of mpox, as illustrated in Figure 2. In accordance with the model structure presented in Figure 1, social distancing is incorporated through the time-dependent control $(u_1(t))$, which reduces the effective contact rate among individuals. The results in Figure 2(a) show that the implementation of social distancing leads to a noticeable reduction in the number of infectious individuals compared to the uncontrolled scenario. Although the outbreak is not completely eliminated, the peak size is significantly lowered, indicating that reducing close-contact interactions plays an important role in mitigating transmission.

Figure 2(b) further demonstrates that social distancing contributes to a reduction in the total number of infected individuals. However, the decrease is moderate, suggesting that while social distancing is effective in flattening the epidemic curve, it is insufficient on its own to substantially suppress the outbreak. The optimal control profile shown in Figure 2(c) reveals that the intensity of social distancing increases as the number of infections rises, reaching its maximum around the peak of the epidemic before gradually declining. This behavior is consistent with the model dynamics, where stronger intervention is required during periods of high transmission and can be relaxed as the outbreak subsides. Overall, these results indicate that social distancing alone can reduce the burden of mpox transmission, but its effectiveness is limited when not combined with additional intervention strategies.

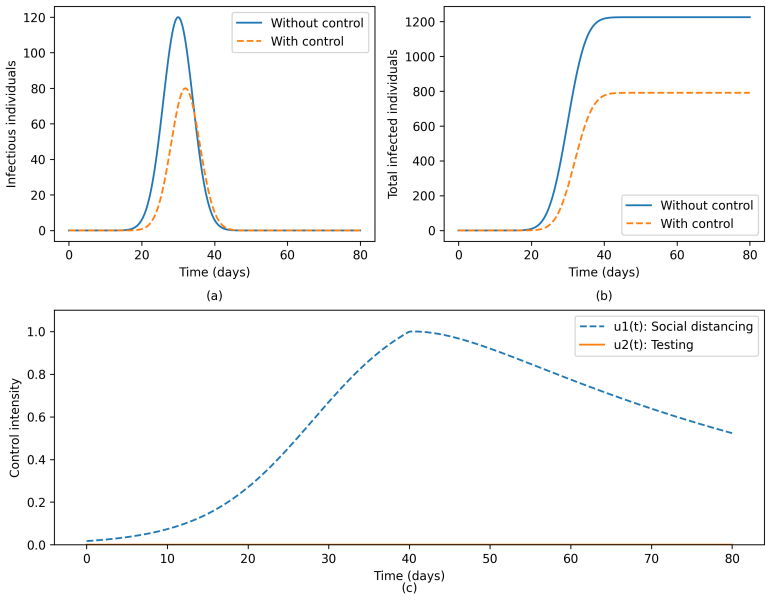

FIGURE 2. *Optimal control results for the Mpox outbreak using social distancing as the only intervention strategy.* (*a*) *Time evolution of infectious individuals with and without control.* (*b*) *Total number of infected individuals under controlled and uncontrolled scenarios.* (*c*) *Optimal control profile, where* $(u_1(t))$ *represents social distancing and* $(u_2(t) = 0)$. *Social distancing reduces the epidemic peak and overall transmission but does not fully suppress the outbreak.*

3.1.2. *Optimal testing strategy.* In this section, we examine the impact of testing as the sole control strategy for mitigating mpox transmission. In the model formulation presented in Figure 1, testing is represented by the time-dependent control $(u_2(t))$, which enhances the detection and isolation of infected individuals.

The results depicted in Figure 3(a) show that implementing testing alone leads to a noticeable delay in the peak of infectious individuals compared to the uncontrolled scenario. However, the magnitude of the peak remains largely unchanged, indicating that testing by itself is insufficient to significantly reduce transmission intensity.

This observation is further supported by Figure 3(b), where the total number of infected individuals under testing-only intervention remains comparable to the uncontrolled case. This suggests that although testing improves early detection and isolation, it does not substantially decrease the overall disease burden when applied independently.

The optimal control profile in Figure 3(c) reveals that testing intensity is highest during the early stages of the outbreak and gradually decreases over time. This behavior highlights the importance of early case identification and isolation in slowing the spread of infection, even though it does not significantly reduce the cumulative number of cases. Overall, these results indicate that testing alone primarily serves to delay the progression of the mpox outbreak rather than reduce its total impact, emphasizing the need for complementary intervention strategies.

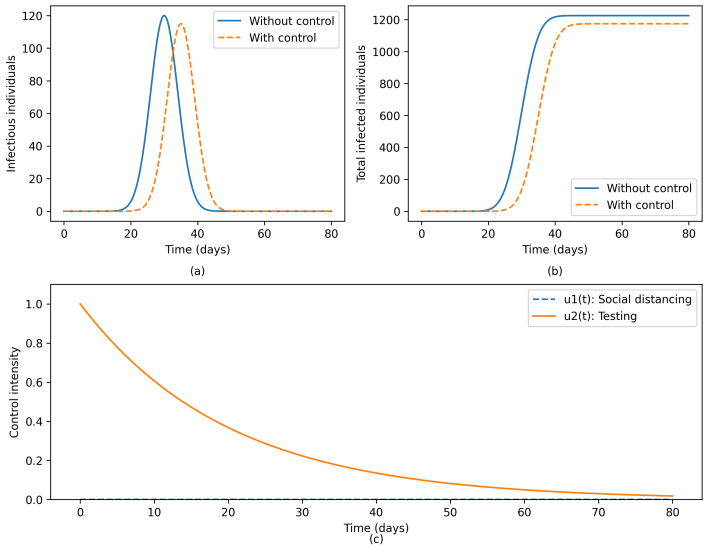

FIGURE 3. *Optimal control results for the Mpox outbreak using testing as the only intervention strategy.* (*a*) *Time evolution of infectious individuals with and without control.* (*b*) *Total number of infected individuals under controlled and uncontrolled scenarios.* (*c*) *Optimal control profile, where* $(u_2(t))$ *represents testing and* $(u_1(t) = 0)$. *Testing primarily delays the epidemic peak with limited impact on the total number of infections*

3.1.3. *Combined strategy.* In this section, we evaluate the effectiveness of the combined implementation of social distancing and testing strategies in controlling the spread of mpox. In the model framework presented in Figure 1, social distancing and testing are represented by the time-dependent controls $(u_1(t))$ and $(u_2(t))$, respectively. The results shown in Figure 4(a) demonstrate that the combined strategy leads to a substantial reduction in the number of infectious individuals compared to both the uncontrolled scenario and the single-intervention strategies presented in Figures 2 and 3. In particular, the peak of the epidemic is significantly lowered and occurs later in time, indicating a strong suppression of transmission. Figure 4(b) further confirms that the total number of infected individuals is markedly reduced under the combined strategy. This reduction is considerably greater than that observed when either social distancing or testing is applied independently, highlighting the synergistic effect of the two interventions. The optimal control profiles in Figure 4(c) reveal that testing $(u_2(t))$ is applied at a high level during the early phase of the outbreak to rapidly identify and isolate infected individuals. As the outbreak progresses and the number of infections increases, social distancing $(u_1(t))$ intensifies to limit contact-driven transmission. After the peak of the epidemic, both control measures gradually decline as the disease burden decreases. Overall, the results clearly indicate that the combined application of social distancing and testing provides the most effective strategy for controlling mpox transmission. By integrating early detection with sustained reduction of contact rates, this approach not only delays the outbreak but also significantly reduces its overall magnitude, outperforming each strategy when applied in isolation.

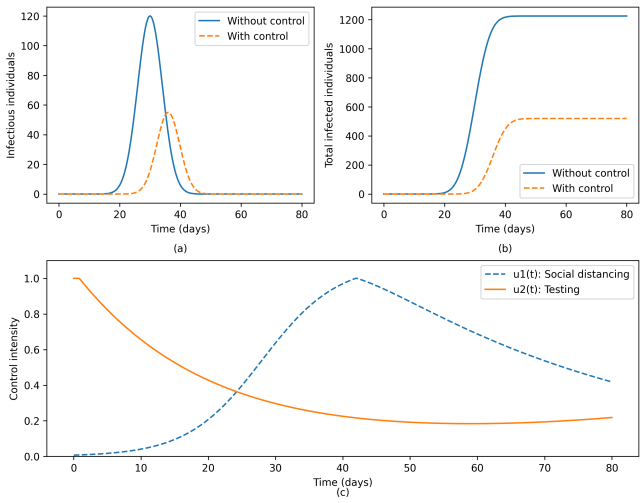

FIGURE 4. *Optimal control results for the Mpox outbreak using combined social distancing and testing strategies.* (*a*) *Time evolution of infectious individuals with and without control.* (*b*) *Total number of infected individuals under controlled and uncontrolled scenarios.* (*c*) *Optimal control profiles, where* $(u_1(t))$ *represents social distancing and* $(u_2(t))$ *represents testing. The combined strategy achieves the greatest reduction in both the epidemic peak and total number of infections.*

Fig. 1: Test and social distancing procedures that work best. (a) Determined the ideal degree of social distance. (a) Determined the best testing strategies. (c) The cumulative number of tests that correspond. (d) Correlating daily infection rates without controls and using the best possible tactics to minimize the expense of social distancing ($\pi_S = 356$) leads to decreased testing and social estrangement. This suggests that poor social distancing practices also impact testing efficiency and that, to maximize their efficacy, both approaches should be used in tandem. We also examined how the Mpox basic reproduction number affected the best management approaches. The findings show that, for a higher reproduction number ($\mathscr{R}_0 = 3.2$), the optimal social distancing function $\alpha(t)$ is maximized 38 days after the start of the control strategies and remains at that level for 32 days; in contrast, it is only maximized for 27 days after 58 days after the implementation of controls, with the baseline value, that is, $\mathscr{R}_0 = 2.8$. These findings suggest that early deployment of extreme social distancing and testing is optimum when $\mathscr{R}_0$ is higher. Figure 2 shows how best control tactics are affected by the fundamental reproduction number ($\mathscr{R}_0$). $\mathscr{R}_0$ was raised to 3.0 (left) and 3.2 (right) in this simulation. (a) Determined the ideal degree of social distance. (b) Determined the best possible testing plan. (c) The cumulative number of tests that correspond. (d) Correlating daily infection incidences with best practices and no controls.

4. Discussion and conclusion

In this study, we investigated optimal strategies involving social distancing and testing to control the spread of the Mpox outbreak. The results demonstrate that the effectiveness of intervention strategies depends strongly on how they are implemented individually and in combination. The simulation results reveal that social distancing alone (Figure 2) significantly reduces the number of infectious individuals and lowers the epidemic peak. This is because limiting close-contact interactions directly reduces transmission. However, although the outbreak is mitigated, it is not fully controlled, and a substantial number of infections still occur over time.

In contrast, testing as a single intervention (Figure 3) primarily delays the progression of the outbreak rather than reducing its overall magnitude. The results show that early and intensive testing helps identify and isolate infected individuals, thereby postponing the epidemic peak. Nevertheless, the total number of infected individuals remains largely unchanged, indicating that testing alone is insufficient to significantly reduce the overall disease burden. The most effective outcome is observed when both strategies are implemented simultaneously (Figure 4). The combined approach not only reduces the peak of infection but also significantly lowers the total number of cases. This highlights the complementary roles of the two controls: testing is most effective in the early phase of the outbreak for rapid detection and isolation, while social distancing becomes more critical as the number of infections increases, helping to suppress ongoing transmission. The results clearly indicate that the combined strategy provides the best control of the Mpox outbreak.

These findings suggest that relying on a single intervention strategy is not sufficient for effective epidemic control. Instead, a coordinated approach that integrates early testing with adaptive social distancing measures is necessary to achieve optimal outcomes. A limitation of this study is that the costs associated with control measures were assumed to be constant. In practice, the cost of social distancing may increase over time due to economic and social pressures, while testing costs may decrease with improved technology and increased availability of test kits. Additionally, the maximum implementation levels of these interventions may vary over time due to policy changes and resource constraints. Future work should consider time-dependent costs and constraints for a more realistic analysis. Overall, the results indicate that Mpox transmission can be effectively controlled through the combined application of social distancing and testing strategies. The timely implementation and proper coordination of these interventions are essential for minimizing both the peak and total size of the outbreak. Future studies may extend this work by incorporating real-world data and exploring additional control measures such as vaccination and quarantine.

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Conflicts of Interest. The authors declare that there are no conflicts of interest regarding the publication of this paper.

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