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A Fractional Optimal Control Problem for Mpox Integrating Vaccination, Treatment and Awareness Campaign

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Abstract

The aim of the present study is to propose a new mathematical model of compartment type for an epidemic problem using fractional order derivatives. This epidemic model takes into account vaccination, hospitalization, asymptomatic infection, and health awareness programs. Caputo fractional derivatives are used to model the temporal non-locality of epidemic phenomena in the proposed model. The qualitative analysis of the model includes the characterization of equilibrium points and their stability. The disease-free equilibrium (DFE) is shown to be locally asymptotically stable when the basic reproduction number $R_0 < 1$, and unstable otherwise. Conversely, an endemic equilibrium emerges when $R_0 > 1$, corresponding to the instability of the DFE. Periodic oscillation is observed for a higher rate of infection transmission. A fractional optimal control problem is formulated to minimize disease prevalence through vaccination, hospitalization, and treatment strategies, supported by sustained awareness campaigns. The results emphasize the role of vaccination, treatment and awareness campaign in controlling Mpox outbreaks, showing their success in minimizing the epidemic. In addition, a fractional optimal control model is proposed to reduce disease prevalence using preventive measures such as vaccinations and treatments coupled with awareness impacts. From these results, one can clearly understand that vaccinations and continuous public health awareness are essential in reducing Mpox cases, which help flatten epidemic trends.

Keywords: mathematical model; awareness campaign; basic reproduction number (R_0); equilibria and stability; fractional optimal control problem (FOCP); RMSE analysis; numerical simulations

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1. Introduction

Mpox, formerly referred to as monkeypox, is one of the major diseases affecting the international health scene in the contemporary era [1]. The disease has spread to many countries that have never experienced mpox cases before and has led to its declaration as a Public Health Emergency of International Concern by WHO on 23rd July 2022 [2]. The disease started as isolated cases that later spread to epidemic, which was not previously experienced in such areas. Its mode of transmission can be complicated as it involves symptomatic and asymptomatic infected individuals. Its levels range from mild to severe, requiring hospital admission for some patients [3]. The public health approach, including immunization, has been influential in determining the course of the disease [4,5].

Community awareness plays an essential role in mpox control, as it provides communities the necessary information on mpox symptoms, transmission methods, and preventive

measures [4,6,7]. Being aware of the transmission methods for the virus helps individuals take proper safety measures, visit clinics, and participate in vaccination programs [8,9]. Additionally, community awareness initiatives can help prevent misinformation and stigma surrounding the disease, encouraging patients to disclose their infections and prevent further spread of the disease [10,11].

Mathematical modeling is essential in controlling mpox cases [12,13], allowing the spread of mpox to be predicted, analyzed, and effective ways of controlling mpox identified [5,14]. Mathematical modeling allows us to simulate various scenarios and analyze the likely outcome for mpox in the future by looking at issues such as vaccination, isolation, and changes in behavior patterns [15]. However, conventional models used in epidemic analysis often lack the ability to model memory effects and dependence on long-time intervals [16,17].

The fractional model approach enables one to establish such an operator that incorporates the non-local effect taking into consideration the past states [18–21]. This is what makes the fractional-order approach suitable for the modeling of mpox transmission dynamics in situations where past vaccination drives, hospitalization efforts, and campaigns continue to affect current population behavior. It is different from other models since the memory effect can be incorporated through fractional calculus [22]. The Caputo fractional derivative is often preferred over the Riemann–Liouville and Grünwald–Letnikov forms because it allows the use of classical initial conditions expressed in terms of integer-order derivatives (e.g., position, velocity), which are physically measurable and intuitive. In contrast, the Riemann–Liouville derivative requires fractional initial conditions that lack clear physical meaning, while the Grünwald–Letnikov approach, though conceptually simple, is computationally less efficient. Thus, Caputo provides a more natural extension of classical differential equations to fractional order systems [23,24].

In mpox modeling, optimal control theory using fractional order models plays a crucial role in designing and evaluating intervention strategies that minimize disease burden while balancing costs and resources [25–28]. By incorporating controls such as vaccination, treatment, isolation, or public awareness campaigns into the model, researchers can determine the most effective timing and intensity of interventions [29–31]. The goal is to reduce transmission and infection prevalence without excessive expenditure or disruption. Thus, this study presents a fractional Optimal control to guide policymakers in implementing targeted, efficient, and sustainable measures for mpox prevention and management. Unlike prior fractional SEIR-type models, our study integrates vaccination, hospitalization, treatment, and awareness campaigns simultaneously. We divided the infected human class into asymptomatic infectious and symptomatic infectious individual. Also, we adopted an optimal control approach using maximum principle to minimize mpox transmission cost-effectively. This comprehensive framework provides a new and more realistic representation of public health interventions for Mpox.

Basic reproduction numbers (R_0) have a critical role in understanding disease dynamics [32]. It represents the expected number of secondary infections caused by a single infectious individual in a fully susceptible population. It is a threshold parameter: it determines when the infection can spread and persist, and when the disease can be eliminated [33]. Sensitivity analysis of parameters using R_0 allows us to identify which parameters—such as contact rate, transmission probability, or duration of infectiousness—most strongly influence disease spread [34]. By quantifying these sensitivities, researchers can prioritize interventions (e.g., reducing contact rates or shortening infectious periods) to effectively lower R_0 and control mpox outbreaks [35]. Thus, this study determines the basic reproduction number, R_0 and provides a sensitivity analysis of the parameters of the proposed mpox model using R_0 .

In this study, a new compartmental fractional-order mathematical model of the mpox disease transmission is introduced, which goes beyond the traditional SEIR approach. The new model not only includes the conventional compartments but also adds some others related to asymptomatic infection, hospitalized/quarantined people, mortality caused by infection, and time-dependent awareness. The presence of these additional compartments makes it possible to simulate important factors such as silent transmission, treatment measures, mortality, and behavioral response. Interestingly, the *level of awareness* is simulated as a growing subpopulation based on the number of infected patients and people who get hospitalized. The application of Caputo derivatives brings memory effects to the development of infection as well as the changing dynamics of awareness, in other words, how previous statuses have affected the ongoing process. It is important to emphasize here that fractional-order modeling is well suited to the problem of infectious diseases and behavioral response since both show memory and hereditary properties.

The main objective of this study is to introduce a novel mathematical framework for modeling mpox virus transmission through a fractional-order derivative, explicitly incorporating the role of public awareness. The model distinguishes eight epidemiologically relevant subpopulations—susceptible, vaccinated, exposed but non-infectious, asymptomatic infectious, symptomatic infectious, hospitalized, and recovered—while uniquely treating awareness itself as an additional dynamic subpopulation. Analytical and numerical investigations are conducted to examine the properties of the fractional-order system dynamics. Furthermore, the model integrates diverse control strategies, enabling rigorous evaluation of medical and behavioral interventions. This approach provides a new perspective on policy assessment, offering insights into the optimal combination of clinical and social measures for effective disease management.

The article is organized as follows: A fractional order mathematical model for mpox transmission and the impact of awareness is developed and analyzed that integrates epidemiological and behavioral components (Section 2). The basic properties such as existence and uniqueness of solutions, nonnegativity and boundedness, and basic reproduction number are determined (Section 3). Stability of equilibria is discussed (Section 4.3). Through the application of optimal control theory, three control functions, vaccination, hospitalization, and awareness, are introduced to examine their combined impact on outbreak management (Section 5). Sensitivity analysis of the model parameters is presented in Section 6. The proposed framework offers valuable insights for numerical simulations and policy design, contributing to improved preparedness and more effective strategies for controlling future mpox epidemics (Section 7). The main results are discussed to conclude the paper (Section 8).

2. Model Formulation

We propose a fractional-order SEIR-type model with extended compartments to capture the complex dynamics of disease transmission and control. Fractional calculus is an extension of differential and integral operators into arbitrary real order, other than integers. Herein, we utilize the Caputo fractional derivative operator of order $\alpha \in (0, 1]$ for the differentiable function $f(t)$ given by

$$D_t^\alpha f(t) = \frac{1}{\Gamma(1-\alpha)} \int_0^t \frac{f'(s)}{(t-s)^\alpha} ds,$$

where $\Gamma(\cdot)$ represents the Gamma function. This fractional operator facilitates the inclusion of memory effects in the system where the current behavior is affected by the past behavior of the system.

The proposed model includes the following compartments: $S(t)$ representing the susceptible population, $V(t)$ for vaccinated individuals, $E(t)$ for those exposed but not yet infectious, $A(t)$ for asymptomatic infectious cases, $I(t)$ for symptomatic infectious individuals, $H(t)$ for hospitalized or isolated cases, $R(t)$ for recovered individuals, and $W(t)$ denoting the awareness level in the population.

These compartments allow us to incorporate both epidemiological and behavioral aspects of disease spread, with fractional-order derivatives providing a more flexible framework to describe memory and hereditary properties in the system.

The assumptions underlying this model are discussed below.

- (H1) Recruitment into the susceptible class occurs at a constant rate Λ , while all individuals experience natural mortality at rate μ . Disease transmission occurs through contact between susceptible individuals and either symptomatic or asymptomatic infectious individuals, with transmission rates β_1 and β_2 , respectively.
- (H2) An increase in the level of awareness is expected to reduce the transmission rate. Accordingly, the reduction in transmission can be expressed by the terms $\frac{\beta_1}{1+\phi W}$ and $\frac{\beta_2}{1+\phi W}$, where ϕ denotes the saturation effect of awareness. It is acknowledged, however, that in practice awareness efficacy may be only partial, and this simplifying assumption has been adopted to facilitate the modeling framework.
- (H3) Susceptible individuals are vaccinated at rate v and thus move to the vaccinated class. Exposed individuals progress to infectious classes at rate σ , with a fraction p becoming asymptomatic and the remaining $(1-p)$ becoming symptomatic.
- (H4) Asymptomatic individuals recover at rate γ_A , while symptomatic individuals recover at rate γ_I , die at rate δ , or are hospitalized at rate θ .
- (H5) Hospitalized individuals recover at rate γ_H or die at rate δ_H . Recovered individuals are assumed to have permanent immunity.
- (H6) Awareness increases due to symptomatic cases (ηI) and hospitalized cases (ξH), but decays at rate ω in the absence of new cases.
- (H7) The system is governed by Caputo fractional derivatives of order $\alpha \in (0, 1]$, which capture memory and hereditary effects in disease transmission and progression.

Finally, the system of fractional differential equations is as follows:

$$\begin{aligned}
 D_t^\alpha S &= \Lambda - \frac{\beta_1 SI}{1 + \phi W} - \frac{\beta_2 SA}{1 + \phi W} - vS - \mu S, \\
 D_t^\alpha V &= vS - \mu V, \\
 D_t^\alpha E &= \frac{\beta_1 SI}{1 + \phi W} + \frac{\beta_2 SA}{1 + \phi W} - (\sigma + \mu)E, \\
 D_t^\alpha A &= p\sigma E - (\gamma_A + \mu)A, \\
 D_t^\alpha I &= (1 - p)\sigma E - (\gamma_I + \delta + \mu)I - \theta I, \\
 D_t^\alpha H &= \theta I - (\gamma_H + \delta_H + \mu)H, \\
 D_t^\alpha R &= \gamma_A A + \gamma_I I + \gamma_H H - \mu R, \\
 D_t^\alpha W &= \eta(I + H) - \omega W,
 \end{aligned} \tag{1}$$

with the following initial conditions:

$$\begin{aligned}
 S(0) = S_0 \geq 0, V(0) &= V_0 \geq 0, E(0) = E_0 \geq 0, A(0) &= A_0 \geq 0, \\
 I(0) = I_0 \geq 0, H(0) &= H_0 \geq 0, R(0) = R_0 \geq 0, W(0) &= W_0 \geq 0.
 \end{aligned} \tag{2}$$

Here, S_0 , V_0 , E_0 , A_0 , I_0 , H_0 , R_0 , and W_0 denote the nonnegative initial population sizes in each compartment. These initial conditions ensure that the system starts with biologically meaningful values. Short descriptions of the model parameters are listed in Table 1.

Table 1. Model parameters with short description and baseline values [9,36–42].

Parameter	Description	Value (Unit)
Λ	Recruitment rate of susceptible human	20 humans day ^{−1}
β_1	Transmission rate (symptomatic)	0.0026 day ^{−1}
β_2	Transmission rate (asymptomatic)	0.00025 day ^{−1}
v	Vaccination rate	0.05 day ^{−1}
σ	Transmission rate from exposed → infectious	0.20 day ^{−1}
p	Fraction exposed becoming asymptomatic	0.30 (dimensionless)
γ_A	Recovery rate of asymptomatic human	0.10 day ^{−1}
γ_I	Recovery rate of symptomatic human	0.08 day ^{−1}
γ_H	Recovery rate of hospitalized human	0.07 day ^{−1}
δ	Disease-induced death rate of symptomatic human	0.02 day ^{−1}
δ_H	Disease-induced death rate of hospitalized human	0.01 day ^{−1}
θ	Hospitalization/isolation rate	0.05 day ^{−1}
μ	Natural death rate	0.01 day ^{−1}
ϕ	Effectiveness of awareness	0.002 (dimensionless)
η	Awareness growth from infected cases	0.001 day ^{−1}
ω	Awareness decay rate	0.015 day ^{−1}

3. Basic Properties

Theorem 1 (Local Existence and Uniqueness for System (1)). *Let $0 < \alpha < 1$. Consider the fractional-order epidemic system (1) with initial condition $X(0) = X_0 \in \mathbb{R}_+^8$, where $X(t) = (S(t), V(t), E(t), A(t), I(t), H(t), R(t), W(t))^T$. Then there exists $T > 0$ such that the system admits a unique solution $X(t)$ on $[0, T]$.*

Proof. We first define the vector field $F : \mathbb{R}^8 \rightarrow \mathbb{R}^8$ by

$$F(X) = \begin{pmatrix} \Lambda - \frac{\beta_1 SI}{1+\phi W} - \frac{\beta_2 SA}{1+\phi W} - vS - \mu S \\ vS - \mu V \\ \frac{\beta_1 SI}{1+\phi W} + \frac{\beta_2 SA}{1+\phi W} - (\sigma + \mu)E \\ p\sigma E - (\gamma_A + \mu)A \\ (1-p)\sigma E - (\gamma_I + \delta + \mu)I - \theta I \\ \theta I - (\gamma_H + \delta_H + \mu)H \\ \gamma_A A + \gamma_I I + \gamma_H H - \mu R \\ \eta(I + H) - \omega W \end{pmatrix}.$$

The system can be written compactly as

$$D_t^\alpha X(t) = F(X(t)), \quad X(0) = X_0.$$

For Caputo derivatives of order $\alpha \in (0, 1)$, the system is equivalent to the Volterra integral equation

$$X(t) = X_0 + \frac{1}{\Gamma(\alpha)} \int_0^t (t-s)^{\alpha-1} F(X(s)) ds.$$

Each component of $F(X)$ is a polynomial in the variables S, V, E, A, I, H, R, W . Polynomials are continuous functions on $\mathbb{R}^8$. Hence, F is continuous.

On any bounded domain $D \subset \mathbb{R}^8$, the Jacobian matrix $\nabla F(X)$ exists and is continuous because F is polynomial. By continuity, $\nabla F(X)$ is bounded on D . Therefore, there exists $L > 0$ such that

$$\|F(X) - F(Y)\| \leq L\|X - Y\|, \quad \forall X, Y \in D.$$

Thus, F is locally Lipschitz.

According to the fractional Picard–Lindelöf theorem [43], if F is continuous and locally Lipschitz, then the integral equation admits a unique local solution on some interval $[0, T]$. Since F satisfies these conditions, the theorem applies directly. Therefore, there exists $T > 0$ such that the system (1) admits a unique solution $X(t)$ on $[0, T]$. $\square$

Nonnegativity and Boundedness of Solutions

We now establish that the solutions of system (1) remain nonnegative for all $t \geq 0$. The proof relies on the quasi-positivity of the system rather than assuming the positivity of other compartments.

Consider the system in the form

$$D_t^\alpha X(t) = F(X(t)), \quad X(0) \geq 0,$$

where $X(t) = (S, V, E, A, I, H, R, W)^\top$. A system is said to be *quasi-positive* if, for each component x_i , the right-hand side $F_i(X)$ evaluated at $x_i = 0$ and $x_j \geq 0$ ($j \neq i$) is nonnegative. We verify this property for each equation:

- (i) For S : $D_t^\alpha S|_{S=0} = \Lambda \geq 0$;
- (ii) For V : $D_t^\alpha V|_{V=0} = vS \geq 0$;
- (iii) For E : $D_t^\alpha E|_{E=0} = \frac{\beta_1 SI}{1+\phi W} + \frac{\beta_2 SA}{1+\phi W} \geq 0$ if $(1 - \phi W) \geq 0$;
- (iv) For A : $D_t^\alpha A|_{A=0} = p\sigma E \geq 0$;
- (v) For I : $D_t^\alpha I|_{I=0} = (1 - p)\sigma E \geq 0$ as $p < 1$;
- (vi) For H : $D_t^\alpha H|_{H=0} = \theta I \geq 0$;
- (vii) For R : $D_t^\alpha R|_{R=0} = \gamma_A A + \gamma_I I + \gamma_H H \geq 0$;
- (viii) For W : $D_t^\alpha W|_{W=0} = \eta(I + H) \geq 0$.

Thus, each equation satisfies the quasi-positivity condition: whenever a variable is zero, its fractional derivative is nonnegative, independent of the positivity of other compartments. By the standard invariance result for fractional-order systems, this implies that the nonnegative orthant,

$$\mathbb{R}_+^8 = \{(S, V, E, A, I, H, R, W) \in \mathbb{R}^8 : S, V, E, A, I, H, R, W \geq 0\},$$

is positively invariant under the dynamics of system (1).

Next, we establish the boundedness of solutions of system (1). Let

$$N(t) = S(t) + V(t) + E(t) + A(t) + I(t) + H(t) + R(t)$$

denote the total population at time t . Summing the first seven equations of system (1), we obtain

$$D_t^\alpha N = \Lambda - \mu N - \delta I - \delta_H H.$$

Since $\delta I \geq 0$ and $\delta_H H \geq 0$, it follows that

$$D_t^\alpha N \leq \Lambda - \mu N.$$

This inequality implies that $N(t)$ is bounded above by the solution of the scalar fractional differential equation

$$D_t^\alpha y = \Lambda - \mu y, \quad y(0) = N(0).$$

The solution of this linear equation remains bounded and, in particular,

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

where $E_\alpha(\cdot)$ denotes the Mittag-Leffler function. Thus, $N(t)$ is uniformly bounded by $\frac{\Lambda}{\mu}$ for all $t \geq 0$.

For the environmental reservoir $W(t)$, we have

$$D_t^\alpha W = \eta(I + H) - \omega W.$$

Since $I(t), H(t) \leq N(t)$, it follows that

$$D_t^\alpha W \leq \eta N(t) - \omega W \leq \frac{\eta \Lambda}{\mu} - \omega W.$$

Thus, $W(t)$ is bounded above by $\frac{\eta \Lambda}{\omega \mu}$.

Finally, we conclude that all solutions are bounded for all $t > 0$.

4. Dynamics of the System

This section contains the determination of equilibria, basic reproduction derivation and stability analysis of equilibria.

4.1. Equilibria

The proposed model has two equilibria, namely

- (i) The disease-free equilibrium (DFE) is given by $\mathcal{E}_0 = \left(\frac{\Lambda}{v+\mu}, \frac{v\Lambda}{\mu(v+\mu)}, 0, 0, 0, 0, 0, 0 \right)$;
- (ii) The endemic equilibrium is given by

$$\mathcal{E}^* = (S^*, V^*, E^*, A^*, I^*, H^*, R^*, W^*),$$

where

$$E^* = \frac{\Lambda \sigma \left(\frac{\beta_1(1-p)}{k_I} + \frac{\beta_2 p}{k_A} \right) - (v+\mu)(\sigma+\mu)}{(\sigma+\mu)\sigma \left(\frac{\beta_1(1-p)}{k_I} + \frac{\beta_2 p}{k_A} \right) + (v+\mu)(\sigma+\mu)\phi c_W},$$

$$S^* = \frac{(\sigma+\mu)(1+\phi c_W E^*)}{\sigma \left(\frac{\beta_1(1-p)}{k_I} + \frac{\beta_2 p}{k_A} \right)}, \quad V^* = \frac{v}{\mu} S^*, \quad A^* = \frac{p\sigma}{k_A} E^*,$$

$$I^* = \frac{(1-p)\sigma}{k_I} E^*, \quad H^* = \frac{\theta(1-p)\sigma}{k_I k_H} E^*, \quad W^* = c_W E^*,$$

and

$$R^* = \frac{\sigma E^*}{\mu} \left[\frac{\gamma_A p}{k_A} + \frac{\gamma_I(1-p)}{k_I} + \frac{\gamma_H \theta(1-p)}{k_I k_H} \right].$$

Here,

$$k_A = \gamma_A + \mu, \quad k_I = \gamma_I + \delta + \mu + \theta, \quad k_H = \gamma_H + \delta_H + \mu,$$

and

$$c_W = \frac{\eta(1-p)\sigma}{\omega k_I} \left(1 + \frac{\theta}{k_H}\right).$$

Note that E^* is positive when

$$\Lambda\sigma \left(\frac{\beta_1(1-p)}{k_I} + \frac{\beta_2 p}{k_A} \right) > (v + \mu)(\sigma + \mu). \quad (3)$$

Thus, a feasible endemic equilibrium exists when (3) is satisfied.

4.2. The Basic Reproduction Number

To compute the basic reproduction number R_0 , we apply the Next Generation Matrix (NGM) method [44]. The infected compartments are

$$x = (E, A, I)^T.$$

The new infection terms are given by

$$F(x) = \begin{bmatrix} \beta_1 SI + \beta_2 SA \\ 0 \\ 0 \end{bmatrix},$$

and the transition terms are

$$V(x) = \begin{bmatrix} (\sigma + \mu)E \\ (\gamma_A + \mu)A - p\sigma E \\ (\gamma_I + \delta + \mu + \theta)I - (1-p)\sigma E \end{bmatrix}.$$

Evaluating the Jacobian matrices at the disease-free equilibrium (DFE), where $S = \bar{S} = \frac{\Lambda}{v + \mu}$ and $W = 0$, we obtain

$$F = \begin{bmatrix} 0 & \beta_2 \bar{S} & \beta_1 \bar{S} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}, \quad V = \begin{bmatrix} \sigma + \mu & 0 & 0 \\ -p\sigma & \gamma_A + \mu & 0 \\ -(1-p)\sigma & 0 & \gamma_I + \delta + \mu + \theta \end{bmatrix}.$$

The next generation matrix is then

$$K = FV^{-1},$$

and the basic reproduction number R_0 is defined as the spectral radius of K . After simplification, we obtain

$$R_0 = \frac{\bar{S}}{\sigma + \mu} \left(\frac{p\sigma\beta_2}{\gamma_A + \mu} + \frac{(1-p)\sigma\beta_1}{\gamma_I + \delta + \mu + \theta} \right).$$

Substituting $\bar{S} = \frac{\Lambda}{v + \mu}$, the final expression of R_0 becomes

$$R_0 = \frac{\Lambda}{(v + \mu)(\sigma + \mu)} \left(\frac{p\sigma\beta_2}{\gamma_A + \mu} + \frac{(1-p)\sigma\beta_1}{\gamma_I + \delta + \mu + \theta} \right). \quad (4)$$

Remark 1. Note that E^* is positive when condition (3) holds, which is equivalent to $R_0 > 1$. Therefore, the endemic equilibrium $\mathcal{E}^*$ is feasible whenever $R_0 > 1$.

4.3. Stability Analysis

Here, the stability analysis of the equilibria is studied.

Stability of DFE

The Jacobian matrix at $\mathcal{E}_0$ is

$$J(\mathcal{E}_0) = \begin{bmatrix} -(v + \mu) & 0 & 0 & 0 & -\beta_1 \bar{S} & 0 & 0 & 0 \\ v & -\mu & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -(\sigma + \mu) & \beta_2 \bar{S} & \beta_1 \bar{S} & 0 & 0 & 0 \\ 0 & 0 & p\sigma & -(\gamma_A + \mu) & 0 & 0 & 0 & 0 \\ 0 & 0 & (1-p)\sigma & 0 & J_{55} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \theta & -(\gamma_H + \delta_H + \mu) & 0 & 0 \\ 0 & 0 & 0 & \gamma_A & \gamma_I & \gamma_H & -\mu & 0 \\ 0 & 0 & 0 & 0 & \eta & \eta & 0 & -\omega \end{bmatrix},$$

with $J_{55} = -(\gamma_I + \delta + \mu + \theta)$.

From the block structure of $J(\mathcal{E}_0)$, five eigenvalues come directly from the diagonal entries as follows: $\lambda_1 = -(v + \mu)$, $\lambda_2 = -\mu$, $\lambda_3 = -(\gamma_H + \delta_H + \mu)$, $\lambda_4 = -\mu$, $\lambda_5 = -\omega$ and the remaining eigenvalues satisfy the cubic equation

$$\lambda^3 + a_1 \lambda^2 + a_2 \lambda + a_3 = 0, \quad (5)$$

with coefficients:

$$\begin{aligned} a_1 &= \sigma + \gamma_A + 2\mu + J_{55}, \\ a_2 &= (\sigma + \mu)(\gamma_A + \mu) + J_{55}(\sigma + \gamma_A + 2\mu) - p\sigma\beta_2\bar{S} - (1-p)\sigma\beta_1\bar{S}, \\ a_3 &= J_{55}(\sigma + \mu)(\gamma_A + \mu) - p\sigma\beta_2\bar{S}J_{55} - (1-p)\sigma\beta_1\bar{S}(\gamma_A + \mu). \end{aligned}$$

For fractional-order systems, the equilibrium $\mathcal{E}_0$ is locally asymptotically stable if all eigenvalues λ of $J(\mathcal{E}_0)$ satisfy

$$|\arg(\lambda)| > \frac{\alpha\pi}{2}. \quad (6)$$

which holds precisely when $R_0 < 1$. Thus, we have the following theorem.

If $R_0 < 1$, all eigenvalues λ of the Jacobian matrix $J(\mathcal{E}_0)$ satisfy

$$|\arg(\lambda)| > \frac{\alpha\pi}{2},$$

and hence the disease-free equilibrium $\mathcal{E}_0$ is locally asymptotically stable. Also, if $R_0 > 1$, there exists at least one eigenvalue λ of $J(\mathcal{E}_0)$ with $\Re(\lambda) > 0$, so that

$$|\arg(\lambda)| \leq \frac{\alpha\pi}{2}, \quad (7)$$

and the disease-free equilibrium $\mathcal{E}_0$ becomes unstable.

Remark 2. R_0 serves as a threshold parameter. If $R_0 < 1$, the disease will eventually die out; if $R_0 > 1$, the disease persists. This provides practical guidance for policymakers to evaluate intervention effectiveness.

We use the following result [45].

Proposition 1.

1. If $D(P) > 0$, then the Routh–Hurwitz conditions (5) are the necessary and sufficient conditions for every $\alpha \in [0, 2)$ to have (7) satisfied:

$$a_1 > 0, \quad a_3 > 0, \quad a_1 a_2 > a_3.$$

2. If $D(P) < 0$ and $\alpha \in [0, 2)$, then

(a) If $a_1 \geq 0$, $a_2 \geq 0$, $a_3 > 0$, then

If $\alpha < \frac{\pi}{3}$, then (7) is satisfied, but if $\alpha > \frac{\pi}{3}$, then (7) is not satisfied.

(b) If $a_1 > 0$, $a_2 > 0$, $a_1 a_2 = a_3$, then

(7) is satisfied for all $\alpha \in [0, 1)$, and not satisfied for all $\alpha \in (1, 2)$.

4.4. Forward Bifurcation Analysis

In this section, the nature of the bifurcation occurring at the critical threshold $R_0 = 1$ is investigated. Using the center manifold theory of Castillo-Chavez and Song [46], it is established here that the system undergoes a forward (transcritical) bifurcation at the disease-free equilibrium $\mathcal{E}_0$ when the basic reproduction number crosses unity.

Theorem 2. For the system (1), let the transmission rate β_1 be the bifurcation parameter and denote by β_1^* its critical value that corresponding to $R_0 = 1$. Then, the disease-free equilibrium $\mathcal{E}_0 = \left(\frac{\Lambda}{v+\mu}, \frac{v\Lambda}{\mu(v+\mu)}, 0, 0, 0, 0, 0 \right)$ undergoes a forward (transcritical) bifurcation at $R_0 = 1$.

Proof. Let $x = (E, A, I, H)^T$ denote the infected subsystem. Then the new infection terms are given by

$$\mathcal{F} = \begin{pmatrix} \frac{\beta_1 SI}{1 + \phi W} + \frac{\beta_2 SA}{1 + \phi W} \\ 0 \\ 0 \\ 0 \end{pmatrix},$$

while the transition terms are

$$\mathcal{V} = \begin{pmatrix} (\sigma + \mu)E \\ (\gamma_A + \mu)A - p\sigma E \\ (\gamma_I + \delta + \mu + \theta)I - (1 - p)\sigma E \\ (\gamma_H + \delta_H + \mu)H - \theta I \end{pmatrix}.$$

At the disease-free equilibrium $\mathcal{E}_0$, the Jacobian matrix associated with the infected subsystem is $J = F - V$, where

$$F = \begin{pmatrix} 0 & \beta_2 \bar{S} & \beta_1 \bar{S} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix},$$

and

$$V = \begin{pmatrix} k_1 & 0 & 0 & 0 \\ -p\sigma & k_2 & 0 & 0 \\ -(1 - p)\sigma & 0 & k_3 & 0 \\ 0 & 0 & -\theta & k_4 \end{pmatrix},$$

with

$$\begin{aligned} k_1 &= \sigma + \mu, & k_2 &= \gamma_A + \mu, \\ k_3 &= \gamma_I + \delta + \mu + \theta, & k_4 &= \gamma_H + \delta_H + \mu. \end{aligned}$$

Let $\beta_1 = \beta_1^*$ be such that $R_0 = 1$. Then, the Jacobian $J(\mathcal{E}_0, \beta_1^*)$ possesses a simple zero eigenvalue, while all remaining eigenvalues have negative real parts. Hence, the hypotheses of the center manifold theorem are satisfied.

To determine the direction of bifurcation, we compute the coefficients

$$a = \sum_{k,i,j} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(\mathcal{E}_0, \beta_1^*), \quad (8)$$

and

$$b = \sum_{k,i} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta_1}(\mathcal{E}_0, \beta_1^*), \quad (9)$$

where $w = (w_i)$ and $v = (v_i)$ are the right and left eigenvectors corresponding to the zero eigenvalue.

Since, the infection term is $\frac{\beta_1 SI}{1+\phi W} + \frac{\beta_2 SA}{1+\phi W}$, the only nonzero second derivatives at the disease-free equilibrium are obtained as

$$\frac{\partial^2 f_1}{\partial S \partial I} = \beta_1^*, \quad \frac{\partial^2 f_1}{\partial S \partial A} = \beta_2,$$

and

$$\frac{\partial^2 f_1}{\partial I \partial W} = -\phi \beta_1^* \bar{S}, \quad \frac{\partial^2 f_1}{\partial A \partial W} = -\phi \beta_2 \bar{S}.$$

Substituting these derivatives into (8) and (9), one can obtain $a < 0$, while $b > 0$. Thus, according to the theorem of Castillo-Chavez and Song, a forward (supercritical transcritical) bifurcation occurs at $R_0 = 1$. $\square$

Stability of Endemic Equilibrium (EE)

Stability of EE is determined by the infection-related compartments (E, A, I, H, W) , thus we restrict the Jacobian to this subsystem. The Jacobian of the subsystem is as follows:

$$J = \begin{bmatrix} a_{11} & 0 & 0 & -\frac{\beta_2 S}{1+\phi W} & -\frac{\beta_1 S}{1+\phi W} & 0 & 0 & a_{18} \\ v & -\mu & 0 & 0 & 0 & 0 & 0 & 0 \\ a_{31} & 0 & -(\sigma + \mu) & \frac{\beta_2 S}{1+\phi W} & \frac{\beta_1 S}{1+\phi W} & 0 & 0 & a_{38} \\ 0 & 0 & p\sigma & -(\gamma_A + \mu) & 0 & 0 & 0 & 0 \\ 0 & 0 & (1-p)\sigma & 0 & -a_{55} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \theta & a_{66} & 0 & 0 \\ 0 & 0 & 0 & \gamma_A & \gamma_I & \gamma_H & -\mu & 0 \\ 0 & 0 & 0 & 0 & \eta & \eta & 0 & -\omega \end{bmatrix},$$

with $a_{11} = -\frac{\beta_1 I}{1+\phi W} - \frac{\beta_2 A}{1+\phi W} - v - \mu$, $a_{31} = \frac{\beta_1 I}{1+\phi W} + \frac{\beta_2 A}{1+\phi W}$, $a_{66} = -(\gamma_H + \delta_H + \mu)$, $a_{18} = \phi(\beta_1 SI + \beta_2 SA)/(1+\phi W)^2$, $a_{38} = -a_{18}$, $a_{55} = (\gamma_I + \delta + \mu + \theta)$.

The characteristic equation is

$$\chi(\lambda) = (\lambda + \mu)^2(\lambda - a_{66})(\lambda + \omega)P(\lambda) = 0,$$

where

$$P(\lambda) = \lambda^4 + a_1 \lambda^3 + a_2 \lambda^2 + a_3 \lambda + a_4 = 0,$$

where

$$\begin{aligned}
 a_1 &= \left(-a_{11} + (\sigma + \mu) + (\gamma_A + \mu) + a_{55} \right), \\
 a_2 &= (\sigma + \mu)(\gamma_A + \mu) - (\sigma + \mu)a_{55} - (\gamma_A + \mu)a_{55} + a_{11}((\sigma + \mu) + (\gamma_A + \mu) + a_{55}) \\
 &\quad - a_{31}\beta_2 S/(1 + \phi W) - p\sigma\beta_2 S/(1 + \phi W) - (1 - p)\sigma\beta_1 S/(1 + \phi W), \\
 a_3 &= -a_{11}\left((\sigma + \mu)(\gamma_A + \mu) + (\sigma + \mu)a_{55} + (\gamma_A + \mu)a_{55}\right) + (\sigma + \mu)(\gamma_A + \mu)a_{55} \\
 &\quad - a_{31}\left(p\sigma a_{55} + (1 - p)\sigma(\gamma_A + \mu)\right) - a_{18}\left(p\sigma\beta_1 S/(1 + \phi W) + (1 - p)\sigma\beta_2 S/(1 + \phi W)\right) \\
 &\quad - a_{38}\left(p\sigma\beta_1 S/(1 + \phi W) + (1 - p)\sigma\beta_2 S/(1 + \phi W)\right), \\
 a_4 &= a_{11}(\sigma + \mu)(\gamma_A + \mu)a_{55} - a_{31}\left(p\sigma a_{55}\beta_2 S/(1 + \phi W) + (1 - p)\sigma(\gamma_A + \mu)\beta_1 S/(1 + \phi W)\right) \\
 &\quad + a_{18}\left(p\sigma\beta_1 S/(1 + \phi W)a_{55} + (1 - p)\sigma\beta_2 S/(1 + \phi W)(\gamma_A + \mu)\right) \\
 &\quad + a_{38}\left(p\sigma\beta_1 S/(1 + \phi W)a_{55} + (1 - p)\sigma\beta_2 S/(1 + \phi W)(\gamma_A + \mu)\right).
 \end{aligned}$$

Consider the fractional-order epidemic model with the Caputo derivative of order $\alpha \in (0, 1]$. The equilibrium $\mathcal{E}^*$ is locally asymptotically stable if and only if

$$|\arg(\lambda_i)| > \frac{\alpha\pi}{2}, \quad \forall i = 1, \dots, 5. \quad (10)$$

Local stability of fractional-order systems requires that the eigenvalues of the Jacobian lie outside the sector defined by angle $\alpha\pi$ around the positive real axis. Therefore, the endemic equilibrium is locally asymptotically stable if and only if the above inequality holds.

$D(p)$ is the discriminant of the characteristic polynomial.

Proposition 2. For $\alpha \in [0, 2)$. If $D(P) < 0$, then the necessary and sufficient conditions to have (10) satisfied are as follows:

$$a_3 > 0, \quad \tan^{-1}\left(\frac{-3\sqrt{3}(u - v)}{3(u + v) + 2a_1}\right) > \alpha\frac{\pi}{2},$$

where

$$u = \sqrt[3]{-\frac{n}{2} + \sqrt{\frac{m^3}{27} + \frac{n^2}{4}}}, \quad v = \sqrt[3]{-\frac{n}{2} - \sqrt{\frac{m^3}{27} + \frac{n^2}{4}}},$$

with

$$m = a_2 - \frac{a_1^2}{3}, \quad n = \frac{2a_1^3}{27} - \frac{a_1a_2}{3} + a_3.$$

5. Optimal Control Approach

In this section, we derive the optimal control strategy to minimize Mpox transmission. To reduce disease spread, we introduce three time-dependent control variables, namely

- (i) $u_1(t)$ for vaccination effort (enhancing v);
- (ii) $u_2(t)$ for awareness campaign effort (enhancing W growth);
- (iii) $u_3(t)$ for treatment/isolation effort (enhancing hospitalization rate θ).

The controlled system is as follows:

$$\begin{aligned}
 D_t^\alpha S &= \Lambda - \frac{\beta_1 SI}{1 + \phi W} - \frac{\beta_2 SA}{1 + \phi W} - (v + e_1 u_1)S - \mu S, \\
 D_t^\alpha V &= (v + e_1 u_1)S - \mu V, \\
 D_t^\alpha E &= \frac{\beta_1 SI}{1 + \phi W} + \frac{\beta_2 SA}{1 + \phi W} - (\sigma + \mu)E, \\
 D_t^\alpha A &= p\sigma E - (\gamma_A + \mu)A, \\
 D_t^\alpha I &= (1 - p)\sigma E - (\gamma_I + \delta + \mu)I - (\theta + e_3 u_3)I, \\
 D_t^\alpha H &= (\theta + e_3 u_3)I - (\gamma_H + \delta_H + \mu)H, \\
 D_t^\alpha R &= \gamma_A A + \gamma_I I + \gamma_H H - \mu R, \\
 D_t^\alpha W &= e_2 u_2 + \eta(I + H) - \omega W.
 \end{aligned} \tag{11}$$

The aim of the control problem is to minimize both the total number of infected individuals and the overall cost of implementing public health interventions. To ensure convexity and mathematical tractability, we adopt quadratic cost terms in the objective functional, which also reflect the increasing marginal cost of intensifying control measures. Hence, our objective functional is defined as

$$J(u_1, u_2, u_3) = \int_0^T \left[C_I I(t) + C_H H(t) + \frac{1}{2} (C_1 u_1^2(t) + C_2 u_2^2(t) + C_3 u_3^2(t)) \right] dt, \tag{12}$$

where C_I and C_H are the weights applied for the burden of infection in the community and hospitalization burden, respectively. Likewise, C_1 , C_2 , and C_3 are the cost factors associated with the cost of vaccination, cost of awareness campaign, and cost of treatment interventions, respectively. The choice of the weights and cost factors is made considering realistic public health considerations.

5.1. Existence and Uniqueness of Optimal Controls

Under the standard assumptions that the right sides of the fractional state system (11) is bounded and Lipschitz continuous with respect to the state and control variables, and that the admissible control set is compact, the existence of an optimal control is guaranteed. Furthermore, since the quadratic cost functional (12) is convex in the control variables, strict convexity ensures the uniqueness of the optimal control solution [47,48].

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To formalize this, let $\mathcal{U}$ denote the set of admissible controls:

$$\mathcal{U} = \{(u_1, u_2, u_3) \mid u_i(t) \in [0, 1], i = 1, 2, 3, t \in [0, T]\}.$$

Since $\mathcal{U}$ is nonempty, convex, and compact in $L^2([0, T])$, and the state system (11) admits a unique solution for each admissible control due to Lipschitz continuity, the mapping from $\mathcal{U}$ to the state trajectories is continuous.

Moreover, the cost functional $J(u_1, u_2, u_3)$ in (12) is strictly convex in (u_1, u_2, u_3) because of the quadratic terms. Therefore, by standard results in fractional optimal control theory, there exists a unique optimal control $(u_1^*, u_2^*, u_3^*) \in \mathcal{U}$ such that

$$J(u_1^*, u_2^*, u_3^*) = \min_{(u_1, u_2, u_3) \in \mathcal{U}} J(u_1, u_2, u_3).$$

Hence, the optimal control problem defined by (11) and (12) is well-posed, with both existence and uniqueness of the optimal solution guaranteed under the stated assumptions.

5.2. The Hamiltonian and Adjoint System

In order to find out the optimal control policy, the Pontryagin Maximum Principle is used. The Hamiltonian for this problem can thus be written as follows:

$$\mathcal{H} = C_I I + C_H H + \frac{1}{2}(C_1 u_1^2 + C_2 u_2^2 + C_3 u_3^2) + \sum_{j=1}^8 \lambda_j f_j,$$

where $\lambda_j(t)$ are adjoint variables and f_j are the right-hand sides of the controlled system equations.

By Pontryagin's Maximum Principle, the adjoint system is given by

$$D_t^\alpha \lambda_j(t) = -\frac{\partial \mathcal{H}}{\partial x_j}, \quad j = 1, \dots, 8,$$

with transversality conditions

$$\lambda_j(T) = 0, \quad j = 1, \dots, 8.$$

Explicitly, the adjoint equations are as follows:

$$\begin{aligned} D_t^\alpha \lambda_1 &= \lambda_1 \left(\frac{\beta_1 I}{1 + \phi W} + \frac{\beta_2 A}{1 + \phi W} + v + e_1 u_1 + \mu \right) - \lambda_2 (v + e_1 u_1) \\ &\quad - \lambda_3 \left(\frac{\beta_1 I}{1 + \phi W} + \frac{\beta_2 A}{1 + \phi W} \right), \end{aligned} \quad (13)$$

$$D_t^\alpha \lambda_2 = \mu \lambda_2, \quad (14)$$

$$D_t^\alpha \lambda_3 = (\sigma + \mu) \lambda_3 - p \sigma \lambda_4 - (1 - p) \sigma \lambda_5, \quad (15)$$

$$D_t^\alpha \lambda_4 = \lambda_1 \frac{\beta_2 S}{1 + \phi W} - \lambda_3 \frac{\beta_2 S}{1 + \phi W} + (\gamma_A + \mu) \lambda_4 - \gamma_A \lambda_7, \quad (16)$$

$$\begin{aligned} D_t^\alpha \lambda_5 &= C_I + \lambda_1 \frac{\beta_1 S}{1 + \phi W} - \lambda_3 \frac{\beta_1 S}{1 + \phi W} + (\gamma_I + \delta + \mu + \theta + e_3 u_3) \lambda_5 \\ &\quad - (\theta + e_3 u_3) \lambda_6 - \gamma_I \lambda_7 - \eta \lambda_8, \end{aligned} \quad (17)$$

$$D_t^\alpha \lambda_6 = C_H + (\gamma_H + \delta_H + \mu) \lambda_6 - \gamma_H \lambda_7 - \eta \lambda_8, \quad (18)$$

$$D_t^\alpha \lambda_7 = \mu \lambda_7, \quad (19)$$

$$D_t^\alpha \lambda_8 = \lambda_1 \left(\frac{\phi(\beta_1 S I + \beta_2 S A)}{(1 + \phi W)^2} \right) - \lambda_3 \left(\frac{\phi(\beta_1 S I + \beta_2 S A)}{(1 + \phi W)^2} \right) + \omega \lambda_8. \quad (20)$$

with terminal conditions

$$\lambda_j(T) = 0, \quad j = 1, \dots, 8.$$

5.3. Characterization of Optimal Controls

The optimal controls are obtained from the minimization condition:

$$\frac{\partial \mathcal{H}}{\partial u_i} = 0, \quad i = 1, 2, 3.$$

This gives the optimal controls as follows:

$$\begin{aligned} u_1^*(t) &= \min \left\{ 1, \max \left\{ 0, -\frac{e_1 S(t)(\lambda_1(t) - \lambda_2(t))}{C_1} \right\} \right\}, \\ u_2^*(t) &= \min \left\{ 1, \max \left\{ 0, -\frac{e_2 \lambda_8(t)}{C_2} \right\} \right\}, \\ u_3^*(t) &= \min \left\{ 1, \max \left\{ 0, -\frac{e_3 I(t)(\lambda_5(t) - \lambda_6(t))}{C_3} \right\} \right\}. \end{aligned} \quad (21)$$

The control problem is actually the state system, adjoint system, and optimal controls. Thus, it is a two-point boundary value problem. Numerical simulation of this system provides the optimal vaccination, awareness, and treatment strategies to minimize infection burden and control costs.

To sum up, the optimal control problem is fully determined by the following three key parts: the controlled state equation system along with the initial conditions, the adjoint equation system under transversality constraints, and the explicit formulas of the optimal control inputs u_1^*, u_2^*, u_3^* . Taken together, these ingredients fully determine the optimal control problem.

5.4. Numerical Considerations and Alternative Strategies

The numerical solution of the optimality system is obtained using a forward–backward sweep algorithm coupled with the Grünwald–Letnikov approximation of the fractional derivatives. This combination has been widely employed in the numerical solution of fractional optimal control problems due to its simplicity and computational effectiveness.

Let Δt denote the time step and D_t^α the fractional derivative operator approximated through the Grünwald–Letnikov scheme:

$$D_t^\alpha x(t) \approx \frac{1}{\Delta t^\alpha} \sum_{k=0}^N (-1)^k \binom{\alpha}{k} x(t - k\Delta t). \quad (22)$$

For sufficiently small time steps, the numerical solutions obtained in our simulations exhibit stable behavior and produce consistent approximations of the state, adjoint, and control variables. However, it should be emphasized that the present work does not provide a rigorous convergence or stability proof for the coupled fractional optimality system. In particular, no theorem is established guaranteeing a specific convergence rate or error estimate for the proposed implementation.

Consequently, the numerical results should be interpreted as computational evidence supporting the effectiveness of the adopted scheme rather than as a formal proof of convergence. Establishing rigorous convergence, stability, and error bounds for the Grünwald–Letnikov discretization combined with the forward–backward sweep method in the context of the present fractional optimal control problem remains an open research question and an important direction for future investigation.

Furthermore, although the quadratic optimal control formulation provides a practical framework for evaluating intervention strategies, comparison with alternative control paradigms such as bang–bang control, model predictive control, robust control, or adaptive control may offer additional insights into efficiency and robustness. Such comparative studies are beyond the scope of the present work but constitute promising avenues for future research [49,50].

6. Sensitivity Analysis

Here, the sensitivity indices of the parameters involved with the basic reproduction number (R_0) are determined. In this study, we find R_0 as

$$R_0 = \frac{\Lambda}{(v + \mu)(\sigma + \mu)} \left(\frac{p\sigma\beta_2}{\gamma_A + \mu} + \frac{(1-p)\sigma\beta_1}{\gamma_I + \delta + \mu + \theta} \right). \quad (23)$$

To quantify the influence of each parameter on R_0 , we compute the normalized forward sensitivity index:

$$Y_x^{R_0} = \frac{\partial R_0}{\partial x} \cdot \frac{x}{R_0}, \quad (24)$$

where x denotes a given parameter. The sensitivity indices of R_0 with respect to each parameter are expressed as follows:

$$\begin{aligned} Y_{\Lambda}^{R_0} &= 1, \\ Y_v^{R_0} &= -\frac{v}{v + \mu}, \\ Y_{\mu}^{R_0} &= -\frac{\mu}{v + \mu} - \frac{\mu}{\sigma + \mu} - \frac{\mu}{\gamma_A + \mu} \cdot \frac{p\sigma\beta_2}{p\sigma\beta_2 + (1-p)\sigma\beta_1} \\ &\quad - \frac{\mu}{\gamma_I + \delta + \mu + \theta} \cdot \frac{(1-p)\sigma\beta_1}{p\sigma\beta_2 + (1-p)\sigma\beta_1}, \\ Y_{\sigma}^{R_0} &= \frac{\sigma}{\sigma + \mu}, \\ Y_p^{R_0} &= \frac{p\sigma\beta_2}{p\sigma\beta_2 + (1-p)\sigma\beta_1} - \frac{(1-p)\sigma\beta_1}{p\sigma\beta_2 + (1-p)\sigma\beta_1}, \\ Y_{\beta_1}^{R_0} &= \frac{(1-p)\sigma\beta_1}{p\sigma\beta_2 + (1-p)\sigma\beta_1}, \\ Y_{\beta_2}^{R_0} &= \frac{p\sigma\beta_2}{p\sigma\beta_2 + (1-p)\sigma\beta_1}, \\ Y_{\gamma_A}^{R_0} &= -\frac{\gamma_A}{\gamma_A + \mu} \cdot \frac{p\sigma\beta_2}{p\sigma\beta_2 + (1-p)\sigma\beta_1}, \\ Y_{\gamma_I}^{R_0} &= -\frac{\gamma_I}{\gamma_I + \delta + \mu + \theta} \cdot \frac{(1-p)\sigma\beta_1}{p\sigma\beta_2 + (1-p)\sigma\beta_1}, \\ Y_{\delta}^{R_0} &= -\frac{\delta}{\gamma_I + \delta + \mu + \theta} \cdot \frac{(1-p)\sigma\beta_1}{p\sigma\beta_2 + (1-p)\sigma\beta_1}, \\ Y_{\theta}^{R_0} &= -\frac{\theta}{\gamma_I + \delta + \mu + \theta} \cdot \frac{(1-p)\sigma\beta_1}{p\sigma\beta_2 + (1-p)\sigma\beta_1}. \end{aligned}$$

Parameters that increase transmission or recruitment (Λ , β_1 , β_2 , σ) have positive sensitivity indices, meaning they increase R_0 . Parameters that increase removal from infectious classes (v , μ , γ_A , γ_I , δ , θ) have negative sensitivity indices, meaning they reduce R_0 . The fraction p shifts the balance between asymptomatic and symptomatic transmission, with its sensitivity depending on the relative contributions of β_1 and β_2 . Sensitivity indices, obtained using the values of parameters in Table 1, are listed in Table 2.

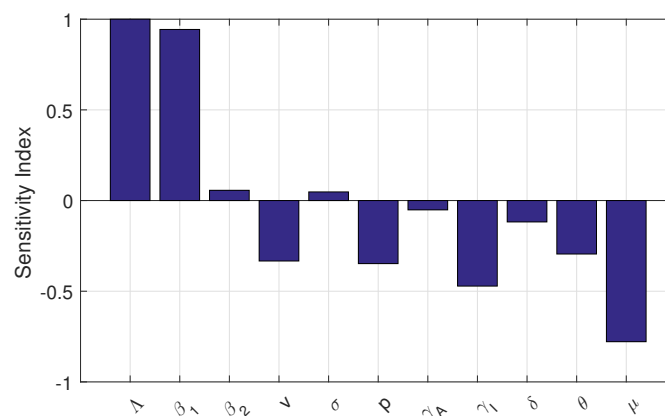
Table 2. Sensitivity indices of model parameters at baseline values.

Parameter	Sensitivity Index, $Y_x^{R_0}$
Λ	1.0000
v	−0.8333
μ	−1.0000
σ	0.9524
p	−0.6923
β_1	0.9231
β_2	0.0769
γ_A	−0.0550
γ_I	−0.7385
δ	−0.1846
θ	−0.4615

7. Numerical Simulations

This section contains the detailed simulations of the analytical results presented in the previous sections. Using the parameter values as in Table 1, the simulations are performed. To solve model (1) numerically, the Fractional Adams–Bashforth–Moulton method [51] is used. Using the parameter values as in Table 1, the simulations are performed. Parameter values listed in Table 1 are estimated from published articles [36–41]. Awareness-related parameters such as η and ω are estimated from other infectious diseases [9,42].

The results of the sensitivity analysis are shown in Figure 1. The positively sensitive parameters include Λ , β_1 , β_2 and σ , while the negatively sensitive parameters are v , γ_I , γ_A and μ . These findings indicate that increases in recruitment and transmission rates strongly promote disease persistence, whereas improvements in vaccination, recovery, and natural mortality reduce infection levels. Such insights can guide policymakers in prioritizing interventions that most effectively reduce disease burden.

**Figure 1.** Sensitivity of model parameters is plotted. Parameter values are taken from Table 1.

The solution of system (1) is plotted for a set of parameters satisfying the condition $R_0 < 1$ in Figure 2. The results show that the system converges to the disease-free equilibrium state, $\mathcal{E}_0$. This outcome is achieved by considering lower infection rates in conjunction with higher vaccination rates, thereby ensuring the eradication of the disease. These findings confirm that appropriate parameter choices can drive the system toward a disease-free situation, highlighting the critical role of vaccination in controlling Mpox transmission. In practice, this suggests that sustained vaccination coverage, combined with reduced transmission opportunities, can eliminate outbreaks before they become endemic.

A forward bifurcation diagram is presented in Figure 3. DFE is stable for $R_0 < 1$ and endemic equilibrium exists for $R_0 > 1$. A forward transcritical bifurcation occurs at $R_0 = 1$ (Theorem 2).

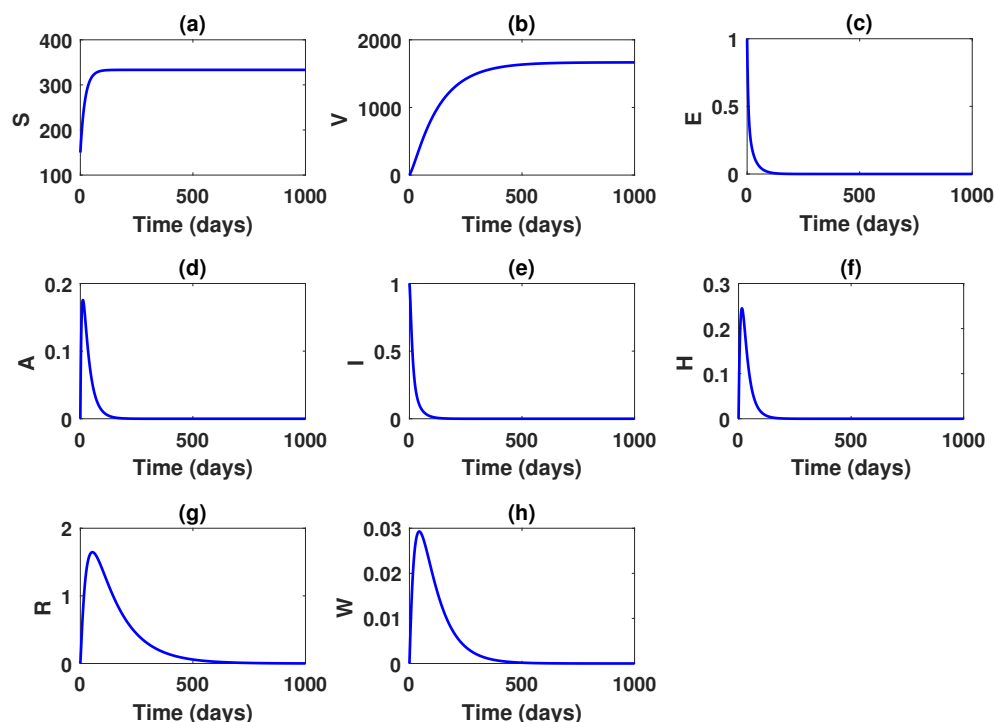


Figure 2. (a–h): Time series solutions are plotted for $R_0 < 1$. Parameter values are taken from Table 1 except $\beta_1 = 0.00035$.

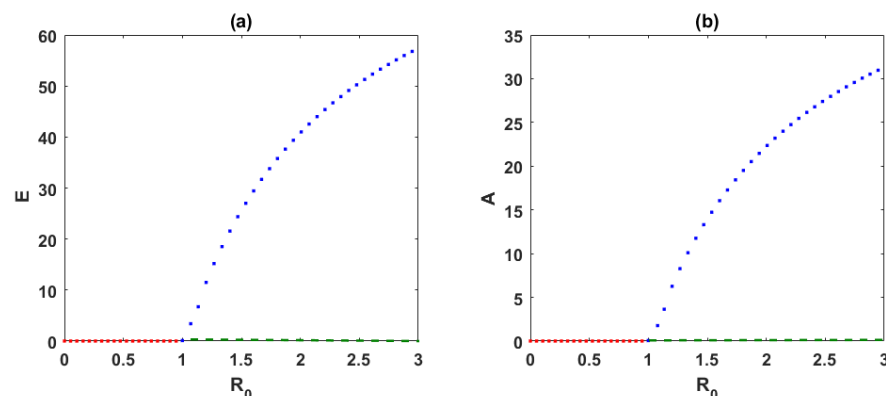


Figure 3. (a,b): Forward bifurcation at $R_0 = 1$: steady state values of infected population are plotted with respect to R_0 . Parameters values are taken from Table 1 and β_1 is varied. Red dotted lines represent stable DFE and green dashed lines stand for unstable DFE. Blue dotted lines show the stable endemic equilibrium.

The eigenvalues of Jacobian obtained at DFE: -0.0100 , -0.0600 , -0.0100 , -0.0150 , -0.0900 , -0.3117 , -0.1278 , and -0.0405 . Thus, DFE is locally asymptotically stable for $\alpha = 0.9$.

For the case of $R_0 > 1$, the graph of the solution of the system is shown in Figure 4. The trajectories of the system in this scenario tend towards the endemic steady state $\mathcal{E}^*$. It can thus be concluded that for relatively high infection rates, the disease persists, thereby reaching the endemic equilibrium point. This result emphasizes that once the

basic reproduction number exceeds unity, Mpox cannot be eradicated without strong interventions, underscoring the importance of early detection and rapid response strategies.

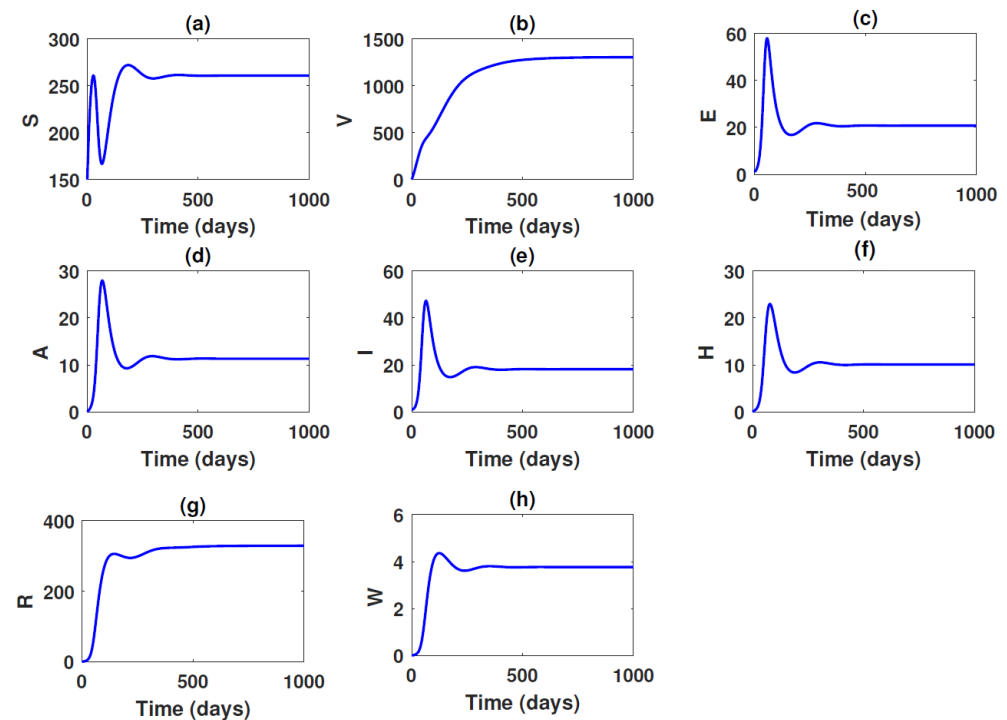


Figure 4. (a–h): Time series solutions are plotted using parameter values from Table 1. In this case, $R_0 > 1$.

We obtained the endemic equilibrium as $S^* = 260.7$, $V^* = 1302$, $E^* = 20.77$, $A^* = 11.33$, $I^* = 18.17$, $H^* = 10.09$, $R^* = 329.2$, $W^* = 3.769$. Eigenvalues of the Jacobian matrix is obtained as follows: $-0.0100 - 0.0100i$, -0.5704 , 0.1211 , -0.0060 , -0.0508 , -0.0911 , and -0.1178 . The conditions $|\arg(\lambda)| > \frac{\alpha\pi}{2}$ is also satisfied for all eigenvalues. Thus, the endemic equilibrium is asymptotically stable.

In Figure 5, trajectories are plotted in $S - E - I$ plane for different initial conditions. It is noticed that all phase-portraits are converged to the same endemic equilibrium $\mathcal{E}^*$. This shows the nonlinear stability of the endemic equilibrium.

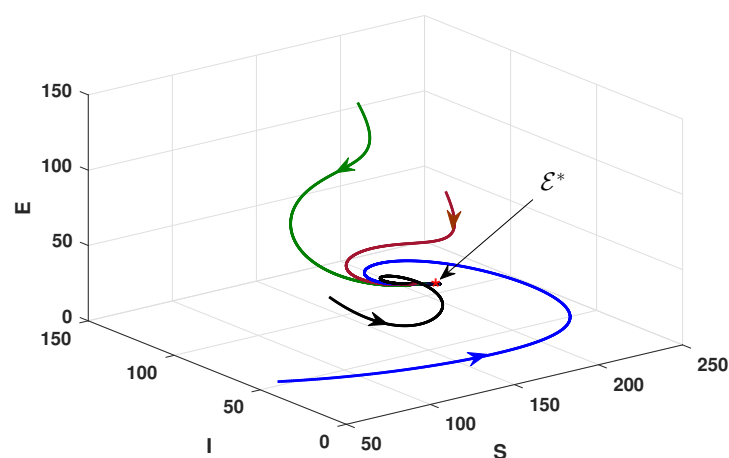


Figure 5. Phase portraits are plotted for different initial conditions in $S - I - E$ plane. Parameters are same as taken in Figure 4.

The revised Figure 6 illustrates the sensitivity of the infection dynamics to variations in the fractional order α . As α decreases from the classical case $\alpha = 1.0$ to lower values, the infection curve becomes flatter and more prolonged, with a delayed peak and slower decline. This behavior reflects the memory effect inherent in fractional-order models: smaller values of α capture long-term persistence of infection and awareness, leading to extended epidemic duration and reduced peak size. In contrast, when α approaches unity, the system recovers the classical sharp epidemic trajectory characterized by a rapid rise, higher peak, and faster resolution. In Table 3, the peak of the infected population and the corresponding time is presented. Also, for recovery population, the final values are given. These results confirm that the fractional order α plays a crucial role in shaping epidemic outcomes and must be carefully considered in model calibration and interpretation. In terms of epidemic models, memory properties represent the contribution of past events or information to the current epidemic pattern. Thus, fractional order models are more appropriate representations of the epidemic situation.

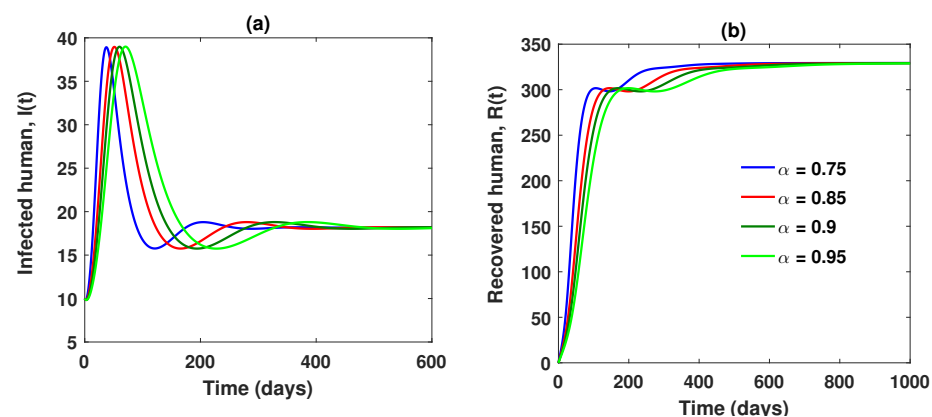


Figure 6. (a,b): Solution trajectories are plotted for two values of α . Parameter values are the same as in Figure 4.

Table 3. Sensitivity of α : Simulation results for varying α values.

α	I_{max}	t_{peak} (Days)	R_{final}
0.75	38.92	37.40	329.30
0.85	38.96	51.20	329.29
0.90	38.97	60.00	329.26
0.95	38.99	70.40	329.17

The system with and without awareness campaign is compared in Figure 7. This figure shows that awareness is important for managing mpox transmission. Infected population is reduced, whereas susceptible and recovered human increased significantly by the influence of awareness campaigns.

The impact of the awareness campaign on the endemic system is illustrated in Figure 8. Here, the parameter of awareness impact is changed for its effect to be seen on the solutions. The higher the awareness impact rate, the lower the number of infected people, including both hospitalized and asymptomatic patients. Therefore, the findings suggest that awareness campaigns are vital in controlling the disease. In practice, this supports investment in public health communication and education as complementary tools to vaccination and treatment.

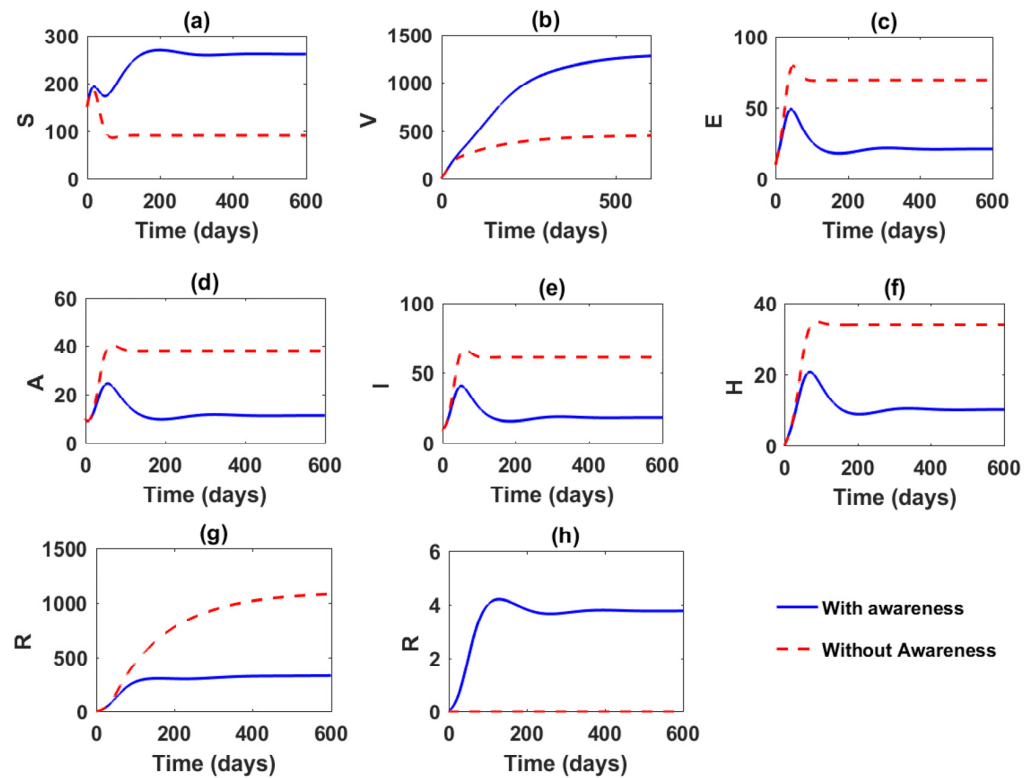


Figure 7. (a–h): Numerical solution for the system with and without awareness are plotted. Parameter values are taken from Table 1.

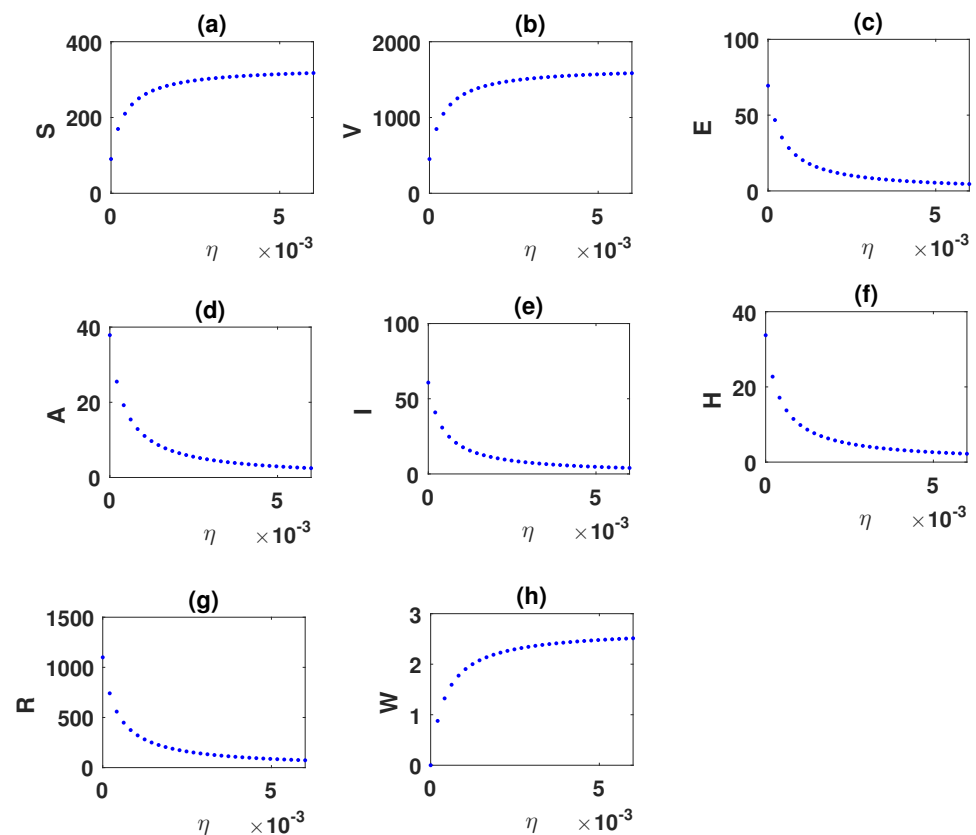


Figure 8. (a–h): Steady-state values are plotted versus the rate of organizing awareness campaigns η . Other parameter values are taken from Figure 7.

7.1. Root Mean Square Error (RMSE) Analysis

In order to study the impact of memory on the solution, the output of the integer-order model ($\alpha = 1$) will be compared with that of the fractional-order model ($\alpha = 0.9$) based on the same set of parameters. The difference will be measured by computing the root mean square error (RMSE) for the infected compartments $I(t)$ as follows:

$$\text{RMSE} = \sqrt{\frac{1}{N} \sum_{k=1}^N (I_{\alpha=1}(t_k) - I_{\alpha=0.9}(t_k))^2},$$

where t_k denotes the k -th discrete time point in the numerical simulation grid. A nonzero RMSE indicates that the fractional-order system generates epidemic dynamics distinct from the classical case.

The graph shown in Figure 9 shows the dynamics of infections according to the integer-order system ($\alpha = 1$) and the fractional-order system ($\alpha = 0.9$). It can be observed that the integer-order system reaches its peak quickly after which there is a fast decrease, while the fractional-order system is smooth, with a late peak and a slow decrease.

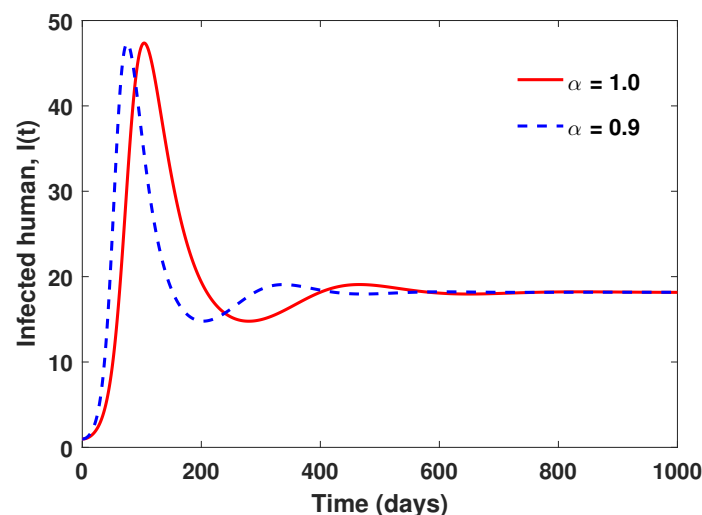


Figure 9. Comparison of integer and fractional order model. Parameters values from Figure 6.

In comparing the results obtained for the proposed model to that of the classical case, the RMSE is estimated at 6.5065. The outcome shows the high level of sensitivity of the process under consideration to small changes in the order of the equation. Although the model incorporates hereditary property and fractal property, incorrect values of α will reduce its accuracy greatly.

7.2. Numerical Solutions for the FOCP

We solve the fractional optimal control problem (FOCP) with memory effect by the forward–backward sweep method using Grünwald–Letnikov approximation [52,53].

Optimal control and no-control comparisons are presented in Figure 10. Optimal control involves a combination of vaccination, awareness, and treatment, giving the mathematical possibility of eradicating the disease from the population. According to our model, implementing all the three controls after twenty days leads to the eradication of the disease in ten more days. Although this gives the mathematical feasibility of eradication, it fails to include real-life factors like vaccine availability, hospital resources, budget, and behavior of individuals. It is important to note that the findings presented above do not necessarily translate to real-life strategies for eradicating Mpox.

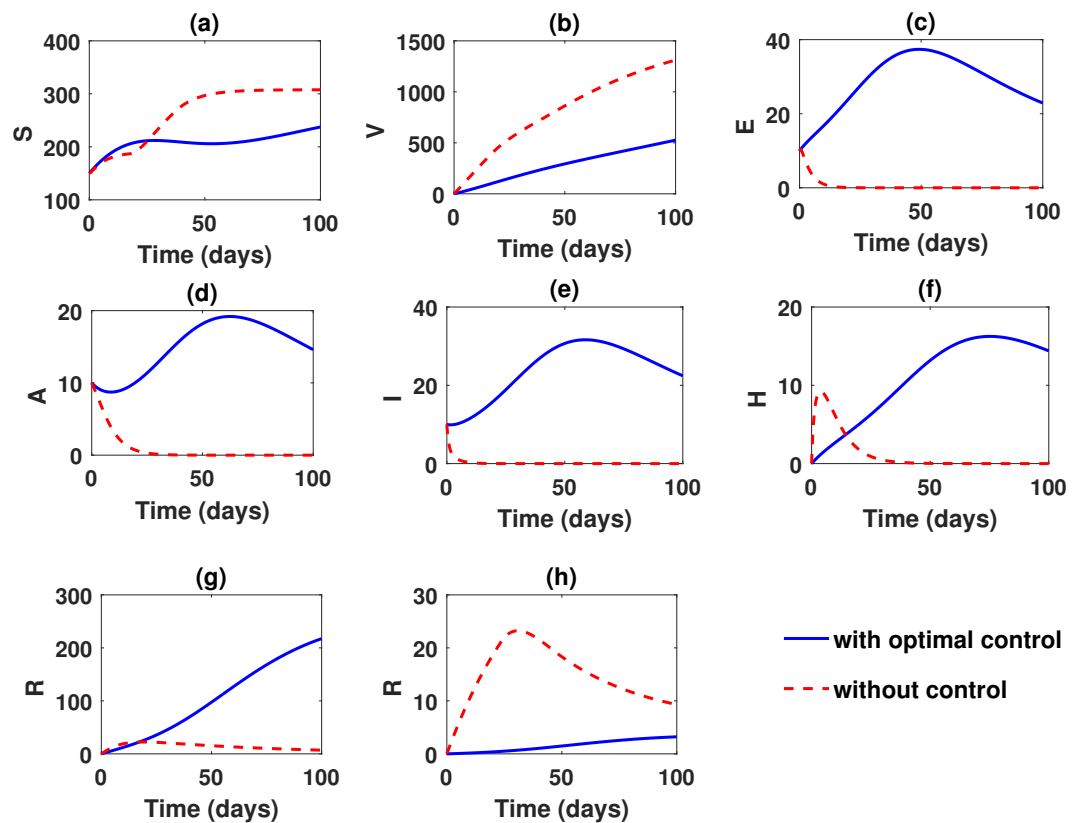


Figure 10. (a–h): Solutions of the optimal system and the system (1) are compared. Parameters are the same as in Figure 4.

Based on the control measures indicated in Figure 11, it is clear that a lot of efforts need to be put forth within the first fifty days to ensure that the system moves towards achieving a disease-free environment. After fifty day, awareness campaign from external sources like television, radio, etc. is required. However, maintaining such a system in real-life would necessitate feasible measures of vaccination, awareness, and treatment delivery, taking into account economic and logistical limitations. This brings about the need for striking a balance between intensive measures over a short period and the sustainability of such efforts over time.

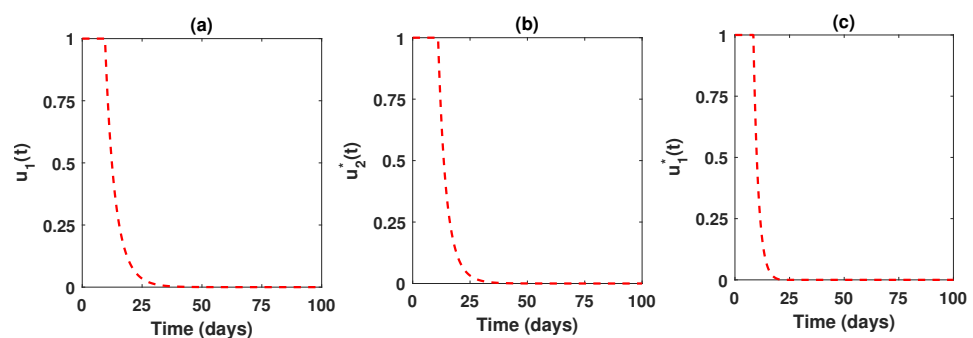


Figure 11. (a–c): Solutions of the optimal control functions (1) are plotted. Parameter values are same as in Figure 10.

7.3. Comparative Study with Existing Models

Employment of fractional-order modeling proves its merit, the current paper requires a more explicit comparative analysis in relation to existing works on this topic [5,13]. Indeed, traditional compartmental models in epidemiology, such as the classical SIR and SEIR

frameworks, rely on integer order dynamics and lack the ability to model the memory effect. By contrast, our fractional model takes into account the properties of heredity and memory in the course of disease transmission, which makes it an even closer representation of Mpox dynamics than any traditional compartmental model.

While the existing literature on Mpox predominantly relies on centralized control strategies, our research advances beyond this conventional framework by addressing optimal control of fractional-order problems that incorporate memory effects. By combining the forward-backward sweep method with the Grünwald–Letnikov approximation [52,53], we developed and implemented a fractional optimal control policy that integrates vaccination, awareness campaigns, and treatment interventions. This comprehensive approach has demonstrated its effectiveness in significantly reducing infection levels and, under appropriate conditions, achieving eradication within a finite time horizon.

8. Discussion

A fractional order model for the Mpox dynamics is proposed and analyzed here. This fractional-order mathematical model not only gives an insight into the dynamics of Mpox virus transmission but also considers human behavior. By considering the awareness programs, we have included a feedback mechanism: an increase in the number of cases increases the awareness level, which leads to a decrease in transmission rate. However, the awareness level decreases due to the fatigue of human behavior. Using fractional derivatives, we include the property of memory in our mathematical model, where past events can impact future results. For instance, past epidemics can affect the future epidemic spread. Here, we simulate our mathematical model at equilibrium, where $\alpha < 1$.

The administration of vaccines becomes important at this point. If the value of R_0 is below unity, the system tends towards the disease-free equilibrium point $\mathcal{E}_0$ through decreased infection incidence and higher vaccination rates. On the other hand, if the value of R_0 is above unity, then the system settles into the endemic equilibrium point $\mathcal{E}^*$. Consequently, forward transcritical bifurcation occurs at $R_0 = 1$. The endemic equilibrium $\mathcal{E}^*$ when exists is nonlinearly asymptotically stable. The sensitivity analysis reveals that Λ , β_1 , β_2 , and σ contribute to increasing the infection transmission, whereas the vaccination rate v , γ_I , γ_A , and μ decrease it. It can be emphasized that the vaccine being administered provides total protection against the disease, which is a simplifying assumption.

Awareness campaigns play an important part in the control of mpox transmission. The more impact the campaign awareness factor has, the less the number of individuals who are either admitted to hospital or asymptomatic. This is the preventive effect that knowledge can bring about. Most importantly, the model shows that by integrating awareness, vaccination, and treatment, it may be possible, at least theoretically, to eradicate Mpox in a matter of days. For instance, it is eradicated after fifty days in the presence of a hundred-day optimal strategy implementation period.

In summary, the eradication of Mpox is impossible without resorting to the implementation of a combination of strategies that include vaccination, awareness programs, and early treatment. The implementation of all these strategies is imperative to bring the basic reproduction number below unity. In other words, this is evidence to support the fact that interdisciplinary methods are critical in eradicating Mpox and establishing epidemiological stability.

There exist several limitations in the current research that deserve further exploration. To begin with, the suggested modeling framework will be improved with the help of data-based calibration and uncertainty quantification. These measures will make the framework more reliable and robust. Secondly, this paper addresses only one particular approach to control design. Hence, the investigation of other approaches like bang–bang control, model

predictive control, robust control, and adaptive control can shed new light on the matter. Last but not least, in terms of theory development, proving convergence and stability of the coupled fractional optimality system is a highly relevant problem. Moreover, decentralized optimal control could be another interesting way to tackle an epidemic situation [54]. Our current focus was on deterministic fractional dynamics. In future work, we plan to extend the model to include stochastic terms, and we will note this limitation explicitly in the revised manuscript.

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