

Monkeypox Transmission through Human Interaction: A Compartmental Model Study

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ABSTRACT

Background: In this study, with a mathematical model, we provide an epidemic model for Mpox virus infection. This paper aims to develop a mathematical epidemic model that captures some of the key characteristics of Mpox. **Materials and Methods:** The endemic equilibrium, disease-free equilibrium, and basic Reproduction Number (R_0) of the model are investigated. Moreover, we focused on how the virus that causes Mpox behaves dynamically. To investigate the existence and uniqueness of the proposed system's solution for the Mpox viral infection, the Derrick and Grossman theorems are utilized. In addition, the solution pathways are studied numerically, emphasizing the influence of the parameters on the Mpox virus dynamics. The Mpox model is described by its fundamental mathematical properties, such as equilibrium, stability, and reproduction number. Using the Lyapunov technique, the Global Asymptotically Stable (GAS) human endemic equilibrium is only displayed when the human basic reproduction number is less than unity. It is demonstrated to be Locally Asymptotically Stable (LAS) when the basic reproduction number for humans is greater than unity by using the definite function of the endemic equilibrium point where the infection resides in the human-to-human population of the model. **Results:** Based on numerical simulations, it appears that people's immunological state varies based on how they recover from an Mpox virus infection. **Conclusion:** Our study emphasizes that treatment, vaccination, and public awareness are the only preventive strategies concerning Mpox. Moreover, our findings indicate that while the disease can be managed and controlled, attaining long-lasting immunity remains unfeasible due to the lack of conclusive treatments and vaccines for Mpox infection. For the special case, where there is only human-to-human transmission of Mpox, the disease-free equilibrium of the model is shown to be Locally Asymptotically Stable (LAS) whenever the associated reproduction number is less than unity.

Keywords: Global Asymptotically Stable (GAS), Locally Asymptotically Stable (LAS), Mathematical Modeling, Monkey Pox [Mpox], Monkey Pox Disease-Endemic Equilibrium [Mpoxdee], Monkey Pox Disease-Free Equilibrium [Mpoxdfe].

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INTRODUCTION

The survival of humans is seriously threatened by infectious diseases, but the development of safe vaccines, drugs and medical equipment has led to the reduction of the prevalence of some of the infectious diseases. Mpox is a zoonotic illness, indicating that it mainly affects animals but can also transmit to humans. It exhibits symptoms that are comparable to those experienced by those who have smallpox in humans. Mpox virus was first described as a cause of pox-like illnesses in monkeys in the late sixties of the last

century (Jothi *et al.*, 2024). The first known human case of Mpox was a nine-month-old boy in the Democratic Republic of the Congo (DRC, 1970). The virus was initially identified in 1958 in a study of monkeys housed in Denmark. Africa's Central and West regions are the main locations of infection (Grant *et al.*, 2020). As of December 2022, there were over 80,000 instances of Mpox in non-endemic nations, raising concerns about the disease on a worldwide scale. We include a brief overview of Mpox's ecology, history, and basic virology in this study, along with an analysis of the main variations in the virus's fitness features before and after 2022 (Sklenovská, and Van Ranst, 2018).

Using a One Health approach, we distinguish between models that focus on vaccine immunity, geography, climatic variables, and animal models, and we review and critically evaluate current knowledge from epidemiological mathematical models, within-host models, and between-host transmission models. To



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make comparisons among research easier, we include a variety of epidemiological parameters in a reduced style, such as the reproduction number (Usman 2017). We highlight the ways that research on mathematical modeling has produced new mechanistic understandings of the pathogenesis and transmission of Mpox (Rizk *et al.*, 2022; Korobeinikov and Wake 2002; Yuan *et al.*, 2023). Mpox, a zoonotic disease, is gradually posing a public health challenge to both developed and developing countries of the world. Therefore, preventative and control measures must be developed to slow the disease's spread across the population. In this study, a new Mathematical model with determinism for Mpox is developed to critically look at the impact of public awareness campaigns, treatment and vaccination on the transmission dynamics, prevention and control of Mpox (Kumbhar and Agarwala, 2022; Shah and Fulmali, 2023; Fischer *et al.*, 2022; Jothi *et al.*, 2023). Numerical simulation results show that increasing the rates of vaccination of the susceptible individuals, efficacy of public awareness campaigns and the treatment of symptomatic individuals could help in reducing Mpox burden in the population other critical stakeholders to educate the populace on the need to be vaccinated against Mpox infection, get to a health facility immediately when blisters show up on their body and the need to develop vaccines that will not wane within a short period (Peter *et al.*, 2022; Gong *et al.*, 2022).

In close quarters, the virus can also be transferred by droplets expelled during breathing, sneezing, or talking. Having intercourse with an infectious person can potentially spread it. Mpox can spread from infected humans to pets, according to a recent analysis (Ahmad *et al.*, 2024). People who are infected with Mpox have distinct clinical symptoms. Infected people typically experience headaches, fever, back discomfort, low energy, enlarged lymph nodes, and a rash that lasts for two to four weeks during epidemics (Jothi *et al.*, 2019). Mpox is first protected by a vaccination meant to thwart the smallpox eradication program. There are now three Mpox vaccinations available. It has been licensed for two of them to prevent this infection: MVA-BN and LC16 (Arita *et al.*, 1985; Sukhdeo *et al.*, 2022).

Between January 1, 2022, and October 31, 2023, a total of 91,788 authenticated Mpox cases and 167 fatalities were reported to the World Health Organization (WHO) across all WHO regions. The Region of the Americas accounted for the highest burden of disease, reporting 60,092 confirmed cases along with 134 fatalities, indicating that this region was the most affected during the study period.

The European Region reported 26,395 authenticated cases and 7 fatalities, representing the second-highest number of cases globally but with a comparatively lower fatality count. In the Western Pacific Region, 2,586 cases and 1 fatality were recorded, while the African Region reported 1,981 cases and 22 fatalities, reflecting a relatively higher fatality proportion compared to other regions.

The South-East Asia Region documented 639 cases with 2 fatalities, whereas the Eastern Mediterranean Region recorded the lowest numbers, with 95 authenticated cases and 1 fatality. Overall, these findings highlight significant regional disparities in Mpox transmission and mortality, emphasizing the need for region-specific surveillance, prevention, and control strategies.

Mpox was reportedly found in large numbers in non-endemic regions in October 2023. Early and accurate diagnosis of Mpox disease is very essential to overcoming this epidemic (Peter *et al.*, 2023). Artificial intelligence is a new and rapidly developing field that provides the foundation for a variety of scientific disciplines (Van Dijck *et al.*, 2023). This can be used as a method of learning and discovering patterns and relationships from a large set of sample models to improve the current method for decision-making in a specific domain. Intelligence technologies are used to improve surveillance, disease prevention, and infection control for vector-borne, person-to-person, healthcare-related, and bacterial diseases

We begin by analyzing the most basic of the epidemic models: the SIOTR ($M_s(t)$, $M_i(t)$, $M_o(t)$, $M_T(t)$, $M_R(t)$) model. The SIOTR model divides the total population into a susceptible compartment and an infected/infectious compartment, an observation compartment, under treatment compartment, and a recovered compartment. We then proceed to modify our model to more accurately portray the behaviour of Mpox. First, we consider an infection rate that varies periodically with time, since the spread of Mpox has been proven to be seasonal (Hossain *et al.*, 2024; Emeka *et al.*, 2018; Jothi *et al.*, 2024). Vaccinations make it possible for people to either start in the recovered category or to move from the susceptible to the recovered category without moving through the infected category first. Finally, our last improvement adds in the recovered compartment, which contains people has been infected with the disease but is not infectious yet (Somma *et al.*, 2019). We conclude with a discussion on the implications and the limitations of our models and provide suggestions for future research (Lahariya *et al.*, 2022; Rhodes *et al.*, 2020).

There is still much to learn about the epidemiological characteristics of the Mpox infection. Research is being done to learn more about how this sickness spreads and how to treat it (Din and Li2024). One of the best methods for researching the dynamic features of infectious diseases is the use of mathematical models. These models aid in the creation of successful interventions and offer the medical community and decision-makers valuable data for comprehending the dynamics and management of disease (Guirio *et al.*, 2018; Jothi *et al.*, 2024; Jothi *et al.*, 2025). A new Mpox transmission model based on a mathematical model was recently proposed in that Order differential equations are used to establish the majority of Mpox transmission models currently in use (Khan *et al.*, 2024). Mathematical modeling studies of Mpox can offer quick, useful insights into viral dynamics to direct public health initiatives and mitigation strategies, as the virus

is expected to cause further infection peaks in many formerly non-endemic nations (Tul, 2024).

A mathematical model with compartments was created and examined to better understand the dynamics of the Mpox virus's spread. Effective controls included in the model are contact tracking and appropriate surveillance (Shah *et al.*, 2024).

Model Description and Formulation

Terms and Postulates

$M_S(t)$: The number of Susceptible Humans.

$M_I(t)$: The number of Mpox Infectious humans.

$M_O(t)$: Number of Mpox patients under observation in a separate room.

$M_T(t)$: Number of Mpox patients under treatment.

$M_R(t)$: Number of patients recovered from Mpox.

N : Susceptible rate of population.

κ_1 : Infection rate of Mpox patients.

κ_2 : Observation rate of Mpox patients.

κ_3 : Treatment rate of Mpox patients.

κ_4 : The recovery rate of Mpox patients.

q : Natural death rate at all stages.

$q+\iota$: Human death rate caused by Mpox [$q+\iota>q$].

Postulates

The formulation foundation for the Mpox disease model is presented in this section. A compartmental Mpox model for the human population was suggested during the model's development ($M_S(t), M_I(t), M_O(t), M_T(t), M_R(t)$). Based on an abridged epidemiological history of the Mpox viral disease, we also considered the following hypotheses:

- The Mpox viral disease can infect everyone who joins the population.
- It is believed that intimate contact with infected animals is how the virus is spread to people. For instance, it's thought that handling or consuming diseased animals might cause skin injuries that lead to infections.
- Humans who are contagious can infect susceptible people.
- Individuals in Mpox endemic areas are vaccinated.
- All compartments of the human population are well-mixed.

On the one hand, the whole population of humans at time t is represented by $N(t)$, is subdivided into sub-populations of susceptible ($M_S(t)$), infectious ($M_I(t)$), observation ($M_O(t)$), treatment ($M_T(t)$), and recovered ($M_R(t)$) individuals such that

$$N(t) = M_S(t) + M_I(t) + M_O(t) + M_T(t) + M_R(t)$$

We generated the following model equations from the model's description and the schematic picture shown in Figure 1.

$$\frac{dM_S(t)}{dt} = \mathfrak{N} - (\kappa_1 + q)M_S(t) \quad (1)$$

$$\frac{dM_I(t)}{dt} = \kappa_1 M_S(t) - (\kappa_2 + q)M_I(t) \quad (2)$$

$$\frac{dM_O(t)}{dt} = \kappa_2 M_I(t) - (\kappa_3 + q)M_O(t) \quad (3)$$

$$\frac{dM_T(t)}{dt} = \kappa_3 M_O(t) - (\kappa_4 + q + \iota)M_T(t) \quad (4)$$

$$\frac{dM_R(t)}{dt} = \kappa_4 M_T(t) - qM_R(t) \quad (5)$$

With the following nonnegative initial conditions (ICs):

$$M_S(0) = \bar{M}_{S0} \geq 0, M_I(0) = \bar{M}_{I0} \geq 0, M_O(0) = \bar{M}_{O0} \geq 0,$$

$$M_T(0) = \bar{M}_{T0} \geq 0, M_R(0) = \bar{M}_{R0} \geq 0$$

Disease-free equilibrium of Mpox [Mpoxdfe]

Setting all of the equations on the right side of the model (1-5) to zero yields the Mpoxdfe of that model, which is represented by M_{dfe} .

$$\text{i.e., } \frac{dM_S(t)}{dt} = \frac{dM_I(t)}{dt} = \frac{dM_O(t)}{dt} = \frac{dM_T(t)}{dt} = \frac{dM_R(t)}{dt} = 0$$

These yields

$$\mathfrak{N} - (\kappa_1 + q)M_S(t) = 0$$

$$\mathfrak{N} - qM_S(t) = 0 \text{ [Since } \kappa_1 = 0]$$

$$M_S^0(t) = \frac{\mathfrak{N}}{q}$$

The Mpoxdfe for model (1-5) is given by

$$M_{dfe} = \{M_S^0, M_I^0, M_O^0, M_T^0, M_R^0\} = \left\{\frac{\mathfrak{N}}{q}, 0, 0, 0, 0\right\}$$

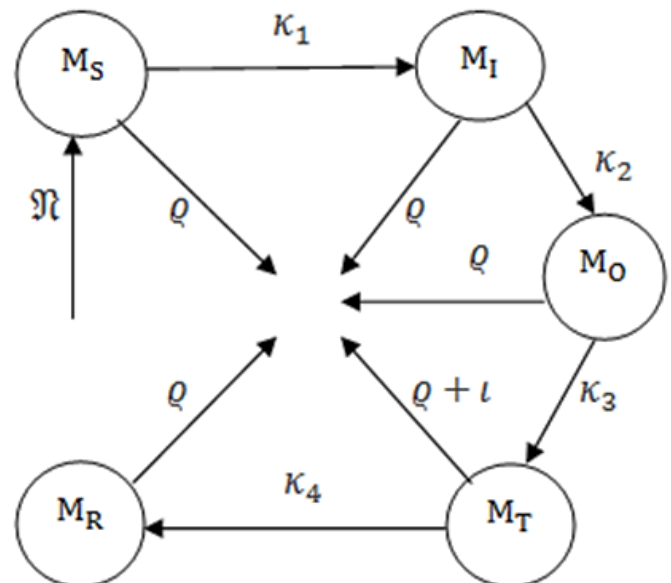


Figure 1: The systematic diagram of the model.

Basic Reproduction Number (R_0)

In this segment, we get the basic reproduction Number (R_0) which is defined as the quantity of secondary infection created by a single infective person when introduced into a susceptible populace. To get the R_0 , consider F and V of two matrices, with new infection terms represented by F and the remaining terms of transfer are represented by V (Saber *et al.*, 2025) (Saber and Alahmari, 2025).

Our model has only five compartments with only one infective compartment.

$$i.e., \frac{dM_I(t)}{dt} = \kappa_1 M_S(t) - (\kappa_2 + \varrho) M_I(t)$$

We have that $F = [\kappa_1 M_S]$ and $V = [(\kappa_2 + \varrho) M_I]$

At the equilibrium of disease-free, the matrix F becomes

$$F = \left[\kappa_1 \frac{\mathfrak{N}}{\varrho} \right]$$

At the equilibrium of disease-free, the matrix V becomes

$$V = (\kappa_2 + \varrho)$$

The inverse matrix of V becomes

$$V^{-1} = \frac{1}{(\kappa_2 + \varrho)}$$

Let $R_0 = FV^{-1}$

Then $R_0 = \left[\kappa_1 \frac{\mathfrak{N}}{\varrho} \right] \left[\frac{1}{(\kappa_2 + \varrho)} \right]$

$$R_0 = \frac{\kappa_1 \mathfrak{N}}{\varrho(\kappa_2 + \varrho)} \quad (6)$$

An important measure in epidemiology is the basic reproduction number, which indicates the likelihood of disease transmission within a population. Public health officials can more accurately assess the risk and put in place the necessary control measures if they are aware of the number of new illnesses that can result from a single infected person.

The solutions positivity

If all of the initial conditions are true, it may be demonstrated that the model's solutions are also positive ($M_S > 0$, $M_I > 0$, $M_O > 0$, $M_T > 0$, and $M_R > 0$). Let us examine the first equation in the system of equation (1-5):

$$\begin{aligned} \frac{dM_S(t)}{dt} &= \mathfrak{N} - (\kappa_1 + \varrho) M_S(t) \\ \Rightarrow \frac{dM_S(t)}{dt} &\geq -(\kappa_1 + \varrho) M_S(t) \end{aligned}$$

Integrating both sides yields,

$$(\kappa_1 + \varrho) \ln M_S \geq -t + C \quad (7)$$

Where C is an integration constant. Equation (7), which introduces the exponential and simplifies it, gives us:

$$M_S \geq Ae^{-(\kappa_1 + \varrho)t} \quad (8)$$

Where A is a constant in this case. After $t = 0$, solving equation (8) yields the value of A:

$$M_S(t) \geq M_S(0) e^{-(\kappa_1 + \varrho)t} \geq 0 \quad (9)$$

Taking into account the second equation, which is

$$\begin{cases} \frac{dM_I(t)}{dt} = \kappa_1 M_S(t) - (\kappa_2 + \varrho) M_I(t) \\ \Rightarrow \frac{dM_I(t)}{dt} \geq -(\kappa_2 + \varrho) M_I(t) \end{cases} \quad (10)$$

Using the initial conditions and integrating via separation of variables, the following result is obtained:

$$M_I(t) \geq M_I(0) e^{-(\kappa_2 + \varrho)t} \geq 0 \quad (11)$$

Comparably, the following is the evaluation of the third equation:

$$\begin{cases} \frac{dM_O(t)}{dt} = \kappa_2 M_I(t) - (\kappa_3 + \varrho) M_O(t) \\ \Rightarrow \frac{dM_O(t)}{dt} \geq -(\kappa_3 + \varrho) M_O(t) \\ \Rightarrow M_O(t) \geq M_O(0) e^{-(\kappa_3 + \varrho)t} \geq 0 \end{cases} \quad (12)$$

When the same procedure is used for the remaining equations, the following outcomes are obtained:

$$\begin{cases} M_S(t) \geq M_S(0) e^{-(\kappa_1 + \varrho)t} \geq 0 \\ M_I(t) \geq M_I(0) e^{-(\kappa_2 + \varrho)t} \geq 0 \\ M_O(t) \geq M_O(0) e^{-(\kappa_3 + \varrho)t} \geq 0 \\ M_T(t) \geq M_T(0) e^{-(\kappa_4 + \varrho + \iota)t} \geq 0 \\ M_R(t) \geq M_R(0) e^{-\varrho t} \geq 0 \end{cases}$$

Consequently, every solution to the model is in the affirmative (positive).

Positivity shows that as possible to protect humans against Mpox.

Theorem 1

If $0 < R_0 < 1$ is satisfied by the reproduction number, then the equilibrium of disease-free M_{dfe} is locally asymptotically stable. Otherwise, M_{dfe} is unstable.

Proof.

At the disease-free equation

$$M_{dfe} = (M_S^0, M_I^0, M_O^0, M_T^0, M_R^0) = \left(\frac{\mathfrak{N}}{\varrho}, 0, 0, 0, 0 \right) \text{ the Jacobian}$$

matrix is:

$$J(M_{dfe}) = \begin{bmatrix} -(\kappa_1 + \varrho) & 0 & 0 & 0 & 0 \\ \kappa_1 & -(\kappa_2 + \varrho) & 0 & 0 & 0 \\ 0 & \kappa_2 & -(\kappa_3 + \varrho) & 0 & 0 \\ 0 & 0 & \kappa_3 & -(\kappa_4 + \varrho + \iota) & 0 \\ 0 & 0 & 0 & \kappa_4 & -\varrho \end{bmatrix} \quad (13)$$

Presently, we process the equation (13) of eigenvalues. The results of the eigenvalues will determine whether the disease-free equation (M_{dfe}) is locally asymptotic stable or not.

Let $|J(M_{dfe}) - \lambda I| = 0$, be a characteristic equation.

Then,

$$|J(M_{dfe}) - \Lambda I| = \begin{vmatrix} -(\kappa_1 + \varrho) - \Lambda & 0 & 0 & 0 & 0 \\ \kappa_1 & -(\kappa_2 + \varrho) - \Lambda & 0 & 0 & 0 \\ 0 & \kappa_2 & -(\kappa_3 + \varrho) - \Lambda & 0 & 0 \\ 0 & 0 & \kappa_3 & -(\kappa_4 + \varrho + \iota) - \Lambda & 0 \\ 0 & 0 & 0 & \kappa_4 & -\varrho - \Lambda \end{vmatrix}$$

From this characteristic equation, we get the five real roots as follows:

$$\Lambda_1 = -(\kappa_1 + \varrho), \Lambda_2 = -(\kappa_2 + \varrho), \Lambda_3 = -(\kappa_3 + \varrho), \Lambda_4 = -(\kappa_4 + \varrho + \iota), \Lambda_5 = -\varrho$$

Since all the eigenvalues of $J(M_{dfe}) < 1$ that is $\Lambda_1 = \Lambda_2 = \Lambda_3 = \Lambda_4 = \Lambda_5 < 1$, Therefore the disease-free equation (M_{dfe}) is locally asymptotic stable (LAS) since $R_0 < 1$.

Boundedness of solutions

For biological reasons, we suppose that the variables of the initial values of the systems (1-5) are non-negative. Then, we prove that all of its solutions are also non-negative, feasible, and bounded (Saber and Alahmari, 2025).

Theorem 2

In systems (1-5) given that the initial values are $M_S(0) > 0, M_I(0) > 0, M_O(0) > 0, M_T(0) > 0$, and $M_R(0) > 0$, for every $t \geq 0$ the solution to $M_S(t), M_I(t), M_O(t), M_T(t)$, and $M_R(t)$ will remain non-negative.

Proof

From Eqs. (1-5) of the Mpox model, the following outcome results from initial conditions on each variable being non-negative:

$$\left| \frac{dM_S(t)}{dt} M_S(0) \geq 0, M_I(0) \geq 0, M_O(0) \geq 0, M_T(0) \geq 0, M_R(0) > 0 = \Re \geq 0 \right. \quad (14)$$

$$\left| \frac{dM_I(t)}{dt} M_S(0) \geq 0, M_I(0) \geq 0, M_O(0) \geq 0, M_T(0) \geq 0, M_R(0) > 0 = \kappa_1 \geq 0 \right. \quad (15)$$

$$\left| \frac{dM_O(t)}{dt} M_S(0) \geq 0, M_I(0) \geq 0, M_O(0) \geq 0, M_T(0) \geq 0, M_R(0) > 0 = \kappa_2 \geq 0 \right. \quad (16)$$

$$\left| \frac{dM_T(t)}{dt} M_S(0) \geq 0, M_I(0) \geq 0, M_O(0) \geq 0, M_T(0) \geq 0, M_R(0) > 0 = \kappa_3 \geq 0 \right. \quad (17)$$

$$\left| \frac{dM_R(t)}{dt} M_S(0) \geq 0, M_I(0) \geq 0, M_O(0) \geq 0, M_T(0) \geq 0, M_R(0) > 0 = \kappa_4 \geq 0 \right. \quad (18)$$

The estimation at the boundary or limit of each variable above shows that all variable rates are positive on the limit planes of $\mathbb{R}_+^5$. This leads us to the conclusion that every vector field in the limit points inward all the time. Consequently, the solution will always stay non-negative for all times $t \geq 0$ if the initial condition is non-negative in $\mathbb{R}_+^5$. Therefore, the proof is complete.

The model's non-negativity invariant region

Total of the human-size

$$\mathcal{N}(t) = M_S(t) + M_I(t) + M_O(t) + M_T(t) + M_R(t) \quad (19)$$

$$\frac{d\mathcal{N}(t)}{dt} = \frac{dM_S(t)}{dt} + \frac{dM_I(t)}{dt} + \frac{dM_O(t)}{dt} + \frac{dM_T(t)}{dt} + \frac{dM_R(t)}{dt}$$

$$\frac{d\mathcal{N}(t)}{dt} = \Re - \varrho(M_S + M_I + M_O + M_T + M_R) - \iota M_T$$

$$\frac{d\mathcal{N}(t)}{dt} = \Re - \varrho\mathcal{N}(t) - \iota M_T \quad (20)$$

Theorem 3

In the systems (1-5) of the Mpox model is feasible for all $t > 0$ for the subsequent solutions of the provided analytically and the solutions of model (1-5) are ultimately bounded.

Proof. Let $D = \{M_S, M_I, M_O, M_T, M_R\} \in \mathbb{R}^5$ culminate the results of the system (1-5) with circumstances greater than zero. Assumed that no infection at the stage of primitive say $\iota=0$, then (20) gives

$$\frac{d\mathcal{N}(t)}{dt} \leq \Re - \varrho\mathcal{N}(t) \quad (21)$$

Rearranging in equation (21), we get,

$$\frac{d\mathcal{N}(t)}{dt} + \varrho\mathcal{N}(t) \leq \Re$$

Which is linear with integrating factor $I.F = e^{\int \varrho dt} = e^{\varrho t}$.

The general solution of (21) is obtained as;

$$\mathcal{N}(t). (I.F) \leq \int \Re(I.F) dt + k$$

Equivalently,

$$\mathcal{N}(t). e^{\varrho t} \leq \int \Re e^{\varrho t} dt + k$$

$$\mathcal{N}(t). e^{\varrho t} \leq \Re \frac{e^{\varrho t}}{\varrho} + k$$

$$\mathcal{N}(t) \leq \frac{\Re}{\varrho} + k e^{-\varrho t} \quad (22)$$

Using $\mathcal{N}(t=0) = \mathcal{N}_0$ we have;

$$\mathcal{N}(0) = \frac{\Re}{\varrho} + k$$

$$\left[\mathcal{N}(0) - \frac{\Re}{\varrho} \right] = k \quad (23)$$

From equation (22) and (23) we have;

$$\mathcal{N}(t) \leq \frac{\Re}{\varrho} + \left(\mathcal{N}_0 - \frac{\Re}{\varrho} \right) e^{-\varrho t} \quad (24)$$

As $t \rightarrow \infty$ in the (24), the human population reduces to $\mathcal{N}(t) \leq \frac{\Re}{\varrho}$

Thus, $\forall t > 0$, the region is given by;

$$\mathcal{D} = \left\{ \begin{array}{l} (M_S(t), M_I(t), M_O(t), M_T(t), M_R(t)) \in \mathbb{R}^5 \geq 0 \\ M_S(t) + M_I(t) + M_O(t) + M_T(t) + M_R(t) \leq \mathcal{N}(t), \mathcal{N}(t) \leq \frac{\Re}{\varrho} \end{array} \right\}$$

Represents the feasible region of the model. This shows that the vaccination is possible to control an Mpox-bound region.

Solution of the boundedness

The formulated systems of the equation denote the first-order human ODE and its general solution. $\mathcal{N}(t) \leq \frac{\Re}{\varrho} + \left(\mathcal{N}_0 - \frac{\Re}{\varrho} \right) e^{-\varrho t}$

$$\mathcal{N}_0 = \mathcal{N}(t=0)$$

Thus, the population of the total human is bounded $\Rightarrow \mathcal{N}_0 \leq \mathcal{N}(t) \leq \frac{\Re}{\varrho}$

Theorem 4

For the Mpox model in Eqs. (1-5), all solutions $M_S(0), M_I(0), M_O(0), M_T(0)$, and $M_R(0)$ are bounded. i.e., if $\limsup_{t \rightarrow \infty} \mathcal{N}(t) \leq \frac{\Re}{\varrho}$, then

$$\mathcal{N}(t) = M_S(t) + M_I(t) + M_O(t) + M_T(t) + M_R(t).$$

Proof. To establish the solution's boundedness, $0 < M_I(t) \leq \mathcal{N}(t)$.

We get by adding each equation in model (1-5), we acquire

$$\frac{dN(t)}{dt} = \mathfrak{N} - \varrho N(t) - \iota M_T. \quad (25)$$

The suggested model's solutions in (1-5) are bounded.

Because of eq. (25),

$$\mathfrak{N} - (\varrho + \iota)N(t) \leq \frac{dN(t)}{dt} \leq \mathfrak{N} - \varrho N(t)$$

and this may now be expressed as

$$\frac{\mathfrak{N}}{(\varrho + \iota)} \leq \liminf_{t \rightarrow \infty} N(t) \leq \limsup_{t \rightarrow \infty} N(t) \leq \frac{\mathfrak{N}}{\varrho}.$$

This bound shows that, region-wise, public health awareness campaigns become more critical in preventing outbreaks and mitigating transmission within communities.

Lemma 1. (Existence)

Solution of the model equations (1-5) together with the initial circumstances $M_S(t) > 0$, $M_I(t) > 0$, $M_O(t) > 0$, $M_T(t) > 0$, and $M_R(t) > 0$ exist and is unique in $\mathbb{R}_+^5$. i.e., the model variables $M_S(t)$, $M_I(t)$, $M_O(t)$, $M_T(t)$, and $M_R(t)$ exist $\forall t$ and will remain in $\mathbb{R}_+^5$.

Proof. The system of equations (1-5) on the right-hand side can be expressed as follows.

$$\phi_1(M_S, M_I, M_O, M_T, M_R) = \mathfrak{N} - (\kappa_1 + \varrho)M_S \quad (26)$$

$$\phi_2(M_S, M_I, M_O, M_T, M_R) = \kappa_1 M_S - (\kappa_2 + \varrho)M_I \quad (27)$$

$$\phi_3(M_S, M_I, M_O, M_T, M_R) = \kappa_2 M_I - (\kappa_3 + \varrho)M_O \quad (28)$$

$$\phi_4(M_S, M_I, M_O, M_T, M_R) = \kappa_3 M_O - (\kappa_4 + \varrho + \iota)M_T \quad (29)$$

$$\phi_5(M_S, M_I, M_O, M_T, M_R) = \kappa_4 M_T - \varrho M_R \quad (30)$$

As per the Derrick and Grossman theorem,

Let D indicate the region

$$\mathcal{D} = \{(M_S, M_I, M_O, M_T, M_R) \in \mathbb{R}_+^5 : N(t) \leq \frac{\mathfrak{N}}{\varrho}\}$$

Then equations (1-5) have a unique solution if $\left(\frac{\partial \phi_i}{\partial y_j}\right)$, $i, j=1,2,3,4,5$ are bounded and continuous in D . Here, $y_1 = M_S$, $y_2 = M_I$, $y_3 = M_O$, $y_4 = M_T$ and $y_5 = M_R$. Under this analysis, the boundedness and continuity of the system solutions are established.

$\left \left(\frac{\partial \phi_1}{\partial M_S}\right)\right = -(\kappa_1 + \varrho) < \infty$	$\left \left(\frac{\partial \phi_2}{\partial M_S}\right)\right = \kappa_1 < \infty$
$\left \left(\frac{\partial \phi_1}{\partial M_I}\right)\right = 0 < \infty$	$\left \left(\frac{\partial \phi_2}{\partial M_I}\right)\right = -(\kappa_2 + \varrho) < \infty$
$\left \left(\frac{\partial \phi_1}{\partial M_O}\right)\right = 0 < \infty$	$\left \left(\frac{\partial \phi_2}{\partial M_O}\right)\right = 0 < \infty$
$\left \left(\frac{\partial \phi_1}{\partial M_T}\right)\right = 0 < \infty$	$\left \left(\frac{\partial \phi_2}{\partial M_T}\right)\right = 0 < \infty$
$\left \left(\frac{\partial \phi_1}{\partial M_R}\right)\right = 0 < \infty$	$\left \left(\frac{\partial \phi_2}{\partial M_R}\right)\right = 0 < \infty$

$\left \left(\frac{\partial \phi_3}{\partial M_S}\right)\right = 0 < \infty$	$\left \left(\frac{\partial \phi_4}{\partial M_S}\right)\right = 0 < \infty$
$\left \left(\frac{\partial \phi_3}{\partial M_I}\right)\right = \kappa_2 < \infty$	$\left \left(\frac{\partial \phi_4}{\partial M_I}\right)\right = 0 < \infty$
$\left \left(\frac{\partial \phi_3}{\partial M_O}\right)\right = -(\kappa_3 + \varrho) < \infty$	$\left \left(\frac{\partial \phi_4}{\partial M_O}\right)\right = \kappa_3 < \infty$
$\left \left(\frac{\partial \phi_3}{\partial M_T}\right)\right = 0 < \infty$	$\left \left(\frac{\partial \phi_4}{\partial M_T}\right)\right = -(\kappa_4 + \varrho + \iota) < \infty$
$\left \left(\frac{\partial \phi_3}{\partial M_R}\right)\right = 0 < \infty$	$\left \left(\frac{\partial \phi_4}{\partial M_R}\right)\right = 0 < \infty$
$\left \left(\frac{\partial \phi_5}{\partial M_S}\right)\right = 0 < \infty$	
$\left \left(\frac{\partial \phi_5}{\partial M_I}\right)\right = 0 < \infty$	
$\left \left(\frac{\partial \phi_5}{\partial M_O}\right)\right = 0 < \infty$	
$\left \left(\frac{\partial \phi_5}{\partial M_T}\right)\right = \kappa_4 < \infty$	
$\left \left(\frac{\partial \phi_5}{\partial M_R}\right)\right = -\varrho < \infty$	

Therefore, each and every partial derivative $\left(\frac{\partial \phi_i}{\partial y_j}\right)$, $i, j=1,2,3,4,5$ exists, bounded and continuous in D . According to the Derrick and Grossman theorem, the solution's model (1-5) is unique and exists. This shows that effective strategies for vaccination against Mpox exist.

Mpox disease-endemic equilibrium (Mpoxdee)

Mpoxdee of model (1-5), denoted by M_{dee} , is obtained by the point where there is Mpox in the population. Here, infected classes of all are not equal to zero.

$$M_S^*(t) = \frac{\mathfrak{N}}{(\kappa_1 + \varrho)}$$

$$M_I^*(t) = \frac{\kappa_1}{(\kappa_2 + \varrho)} M_S^*(t)$$

By substituting the value of $M_I^*(t)$ in $M_S^*(t)$ we getting,

$$M_S^*(t) = \frac{\mathfrak{N}}{(\kappa_1 + \varrho)}$$

Also,

$$M_O^*(t) = \frac{\kappa_2}{(\kappa_3 + \varrho)} M_I^*(t)$$

By putting the value of $M_I^*(t)$ in $M_O^*(t)$ we have,

$$M_O^*(t) = \frac{\kappa_1 \kappa_2 \mathfrak{N}}{(\kappa_1 + \varrho)(\kappa_2 + \varrho)(\kappa_3 + \varrho)}$$

Furthermore,

$$M_T^*(t) = \frac{\kappa_3}{(\kappa_4 + \varrho + \iota)} M_O^*(t)$$

By substituting the value of $M_O^*(t)$ in $M_T^*(t)$ we have,

$$M_T^*(t) = \frac{\kappa_1 \kappa_2 \kappa_3 \mathfrak{N}}{(\kappa_1 + \varrho)(\kappa_2 + \varrho)(\kappa_3 + \varrho)(\kappa_4 + \varrho + \iota)}$$

Consequently,

$$M_R^*(t) = \frac{\kappa_4}{\varrho} M_T^*(t)$$

By putting the value of $M_T^*(t)$ in $M_R^*(t)$ we have,

$$M_R^*(t) = \frac{\kappa_1 \kappa_2 \kappa_3 \kappa_4 \mathfrak{N}}{(\kappa_1 + \varrho)(\kappa_2 + \varrho)(\kappa_3 + \varrho)(\kappa_4 + \varrho)\varrho}$$

Hence, the Mpox Endemic equilibrium satisfies

$$M_{dee} = \{M_S^*, M_I^*, M_O^*, M_T^*, M_R^*\}$$

$$= \left(\frac{\mathfrak{N}}{(\kappa_1 + \varrho)}, \frac{\kappa_1 \mathfrak{N}}{(\kappa_1 + \varrho)(\kappa_2 + \varrho)}, \frac{\kappa_1 \kappa_2 \mathfrak{N}}{(\kappa_1 + \varrho)(\kappa_2 + \varrho)(\kappa_3 + \varrho)}, \frac{\kappa_1 \kappa_2 \kappa_3 \mathfrak{N}}{(\kappa_1 + \varrho)(\kappa_2 + \varrho)(\kappa_3 + \varrho)(\kappa_4 + \varrho)}, \frac{\kappa_1 \kappa_2 \kappa_3 \kappa_4 \mathfrak{N}}{(\kappa_1 + \varrho)(\kappa_2 + \varrho)(\kappa_3 + \varrho)(\kappa_4 + \varrho)\varrho} \right) \quad (31)$$

Theorem 5

The endemic equilibrium point $M_{dee} = (M_S^*, M_I^*, M_O^*, M_T^*, M_R^*)$ is globally asymptotically stable [GAS].

Proof.

Let us assume the Lyapunov function

$$\mathcal{L}_f^*(t) = \frac{1}{2} [(M_S - M_S^*) + (M_I - M_I^*) + (M_O - M_O^*) + (M_T - M_T^*) + (M_R - M_R^*)]^2$$

$$\frac{d\mathcal{L}_f^*}{dt} = [(M_S - M_S^*) + (M_I - M_I^*) + (M_O - M_O^*) + (M_T - M_T^*) + (M_R - M_R^*)] \left[\frac{dM_S(t)}{dt} + \frac{dM_I(t)}{dt} + \frac{dM_O(t)}{dt} + \frac{dM_T(t)}{dt} + \frac{dM_R(t)}{dt} \right]$$

$$\frac{d\mathcal{L}_f^*}{dt} = [(M_S - M_S^*) + (M_I - M_I^*) + (M_O - M_O^*) + (M_T - M_T^*) + (M_R - M_R^*)] [\mathfrak{N} - \varrho(M_S + M_I + M_O + M_T + M_R) - \iota M_T]$$

$$\frac{d\mathcal{L}_f^*}{dt} = - [(M_S - M_S^*) + (M_I - M_I^*) + (M_O - M_O^*) + (M_T - M_T^*) + (M_R - M_R^*)] [\varrho [(M_S - M_S^*) + (M_I - M_I^*) + (M_O - M_O^*) + (M_T - M_T^*) + (M_R - M_R^*)]]$$

$$\frac{d\mathcal{L}_f^*}{dt} = -\varrho [(M_S - M_S^*) + (M_I - M_I^*) + (M_O - M_O^*) + (M_T - M_T^*) + (M_R - M_R^*)]^2$$

$$\frac{d\mathcal{L}_f^*}{dt} \leq 0 \text{ Where } \mathfrak{N} = \varrho(M_S^* + M_I^* + M_O^* + M_T^* + M_R^*) + \iota M_T$$

Here, $\frac{d\mathcal{L}_f^*}{dt} \leq 0$. Hence, by the LaSalle Invariance principle the EE point is GAS.

Theorem 6

The equilibrium endemic point $M_{dee} = (M_S^*, M_I^*, M_O^*, M_T^*, M_R^*)$ of the dynamical system of Mpox, model of (1-5) is locally asymptotically stable [LAS] when the system of basic reproduction number $R_0 > 1$.

Proof.

Consider the following: M_{dee}^* is transformed into $M_S = M_S^* + m_s$, $M_I = M_I^* + m_i$, $M_O = M_O^* + m_o$, $M_T = M_T^* + m_t$ and $M_R = M_R^* + m_r$ where m_s , m_i , m_o , m_t and m_r are small perturbations.

Using a Lyapunov function

$$\mathcal{H} = \frac{1}{2} (h_1 m_s^2 + h_2 m_i^2 + h_3 m_o^2 + h_4 m_t^2 + h_5 m_r^2) \quad (32)$$

Differentiating equation (31) with respect to time t, we obtain:

$$\frac{d\mathcal{H}}{dt} = h_1 m_s \frac{dM_S}{dt} + h_2 m_i \frac{dM_I}{dt} + h_3 m_o \frac{dM_O}{dt} + h_4 m_t \frac{dM_T}{dt} + h_5 m_r \frac{dM_R}{dt} \quad (33)$$

The values $\frac{dM_S}{dt}, \frac{dM_I}{dt}, \frac{dM_O}{dt}, \frac{dM_T}{dt}, \frac{dM_R}{dt}$ may now be substituted into equation (33) to obtain

$$\frac{d\mathcal{H}}{dt} = h_1 m_s [\mathfrak{N} - (\kappa_1 + \varrho)M_S] + h_2 m_i [\kappa_1 M_S - (\kappa_2 + \varrho)M_I] + h_3 m_o [\kappa_2 M_I - (\kappa_3 + \varrho)M_O] + h_4 m_t [\kappa_3 M_O - (\kappa_4 + \varrho + \iota)M_T] + h_5 m_r [\kappa_4 M_T - \varrho M_R]$$

$$\frac{d\mathcal{H}}{dt} = -h_1 (\kappa_1 + \varrho) m_s^2 - h_2 (\kappa_2 + \varrho) m_i^2 - h_3 (\kappa_3 + \varrho) m_o^2 - h_4 (\kappa_4 + \varrho + \iota) m_t^2 - h_5 \varrho m_r^2 + h_1 m_s \mathfrak{N} + h_2 m_i \kappa_1 M_S + h_3 m_o \kappa_2 M_I + h_4 m_t \kappa_3 M_O + h_5 m_r \kappa_4 M_T$$

Let Choosing $h_1 = h_2 = h_3 = h_4 = h_5 = 1$

$$\frac{d\mathcal{H}}{dt} = -(\kappa_1 + \varrho) m_s^2 - (\kappa_2 + \varrho) m_i^2 - (\kappa_3 + \varrho) m_o^2 - (\kappa_4 + \varrho + \iota) m_t^2 - \varrho m_r^2 + m_s \mathfrak{N} + m_i \kappa_1 M_S + m_o \kappa_2 M_I + m_t \kappa_3 M_O + m_r \kappa_4 M_T \quad (34)$$

Because $\frac{d\mathcal{H}}{dt} \leq 0$ is negative definite within the attraction area D . As a result, endemic if $R_0 > 1$, the endemic equilibrium point M_{dee} is LAS.

Numerical Simulation

One of the most important parameters in the model, the transmission rate parameter, needs to be minimized to successfully control the spread of the Mpox virus. To study the dynamics of the model (1-5), MATLAB software was used to simulate it.

The model classes are assumed to have the following initial values:

$$N = 1.1752; \kappa_1 = 0.135; \kappa_2 = 0.250; \kappa_3 = 0.350; \kappa_4 = 0.200; \varrho = 0.520; \iota = 0.370;$$

The model classes are assumed to have the following values:

$$N = 100; \kappa_1 = 0.135; \kappa_2 = 0.250; \kappa_3 = 0.350; \kappa_4 = 0.200; \varrho = 0.520; \iota = 0.970;$$

$$M_S(t) = 10000; M_I(t) = 4000; M_O(t) = 3000; M_T(t) = 2000; M_R(t) = 1500$$

The model classes are assumed to have the following values:

$$N = 300; \kappa_1 = 0.135; \kappa_2 = 0.250; \kappa_3 = 0.350; \kappa_4 = 0.200; \varrho = 0.520; \iota = 0.370;$$

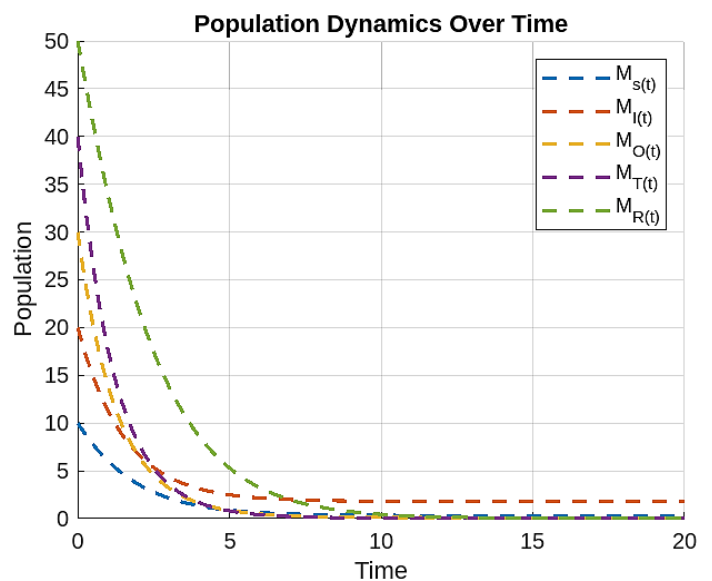


Figure 2: Temporal Dynamics of the Model Populations.



Figure 3: 3D Surface Plot of Monkeypox Model State Variable over Time.

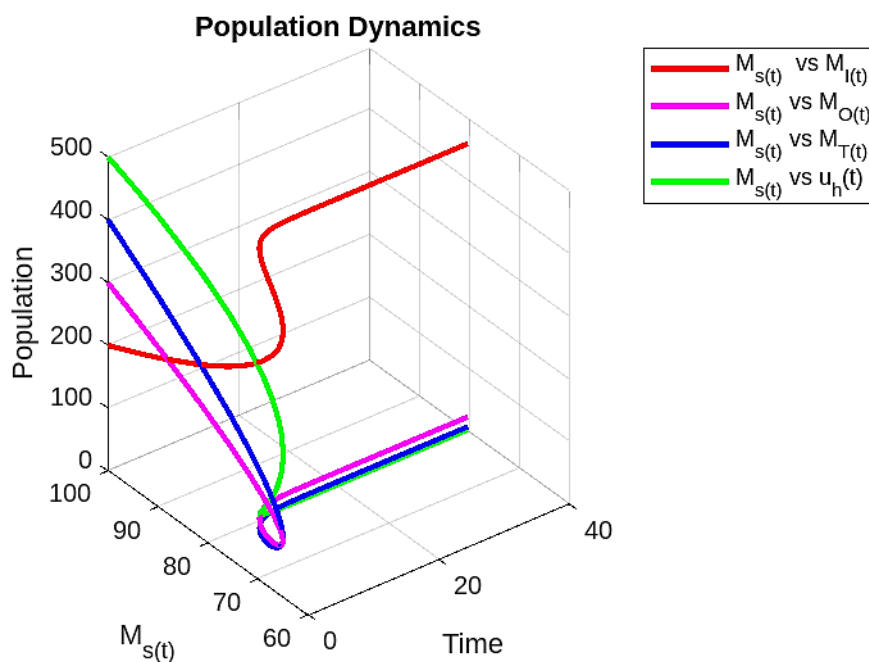


Figure 4: 3D Phase-Space Dynamics of the Monkeypox Transmission Model.

RESULTS

Figure 2 confirms the stability of the proposed Mpox transmission model and highlights the role of control measures in reducing infection prevalence over time.

Figure 3 illustrates a three-dimensional surface of the Mpox model variable, showing its variation with respect to time and population, and demonstrating convergence toward a stable equilibrium under control measures.

Figure 4 presents 3D phase-space trajectories showing the interaction between the susceptible population and other compartments of the Mpox model, illustrating convergence toward a stable equilibrium.

Numerical simulations suggest that recovery does not guarantee long-term immunity, allowing for possible reinfection. These findings highlight the importance of integrated control measures, including treatment, vaccination, and public awareness. Although effective management is achievable, complete eradication remains challenging due to uncertainties in lasting immunity.

CONCLUSION

In this paper, the possibility of creating a mathematical model for Mpox infection is investigated. The Lyapunov function, Derrick and Grossman theorem, and LaSalle invariance principle have all been used to analyze the existence, boundedness, non-negativity, continuity, and uniqueness conclusions for this Mpox model. Furthermore examined are the equilibriums of endemics and disease-free states. The ODE point is locally asymptotically stable when the reproduction number ($R_0 < 1$) and unstable when $R_0 < 1$, whereas the endemic equilibrium point is stable when $R_0 < 1$ and does not exist otherwise. The ODE point is both locally and globally asymptotically stable if $R_0 < 1$ and unstable if $R_0 < 1$, whereas the endemic equilibrium point is both locally and globally asymptotically stable if $R_0 < 1$. Numerous factors can impact the severity and pace of transmission of infectious diseases, according to numerical simulations on the spread and control of these diseases.

We submit that effective strategies for vaccination against Mpox exist, even though the Mpox reservoir remains unknown: for example, the strategy of universal vaccination against smallpox. Another strategy would be to vaccinate individuals community by community, depending on the Mpox epidemiology in each community. The vast majority of communities is not in touch with the Mpox reservoir and still maintains low levels of herd immunity against Mpox, due to their smallpox epidemiology.

We utilized parameter values from existing literature to conduct numerical simulations and perform sensitivity analyses on the model and its parameters. The results indicated that if the model's recommended actions are implemented, the disease can be eliminated from both humans and primates over time. The sensitivity analysis revealed that increasing the control rates of vaccination and treatment offers optimal protection against Mpox virus infections within the human population. This model is suggested for examining the dynamics of the Mpox virus epidemic. Current studies generally affirm that taking measures such as enhancing vaccination programs, enforcing quarantine measures, establishing suitable treatment facilities, and reducing contact with infected rodents can significantly diminish virus transmission.

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ABBREVIATIONS

Mpox: Monkey Pox; **Mpoxdfe:** Monkey Pox Disease-Free Equilibrium; **Mpoxdee:** Monkey Pox Disease-Endemic Equilibrium; **R_0 :** Basic Reproduction Number; **ODE:** Ordinary differential equations; **SIOTR:** Susceptible, Infected, Observation,

Treatment, Recovered; **DRC:** Democratic Republic of the Congo; **IRB:** Institutional Review Board.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

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