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# Study of Monkeypox Virus Transmission Dynamics: Vaccination Effect and Sensitivity Analysis

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ABSTRACT: Monkeypox is an emerging infectious disease from Nigeria that has spread globally, affecting countries such as the USA and the United Kingdom. It belongs to the Poxviridae family under the Orthopoxvirus genus. In this study, we develop a deterministic compartmental model comprising eleven classes: susceptible, vaccinated, exposed, quarantined, hospitalized, and recovered humans, along with susceptible, exposed, infected, and recovered rodents. We analyze the disease-free equilibrium and calculate the basic reproduction number using the next-generation matrix approach. Sensitivity analysis identifies critical parameters influencing disease transmission. Numerical simulations conducted in MATLAB demonstrate that increasing vaccination coverage among humans significantly reduces infection prevalence. The results further reveal that timely quarantine and hospitalization effectively control the spread of the virus. Our findings provide quantitative evidence supporting vaccination and control interventions as vital strategies to mitigate monkeypox outbreaks.

Key Words: Monkeypox virus, mathematical model, reproduction number, sensitivity analysis.

#### Contents

1	Introduction	1			
2	Model formulation				
3	Qualitative assessment of the model         3.1 Boundedness	<b>4</b> 4			
4	Equilibria analysis 4.1 Equilibrium analysis	<b>7</b> 7 7			
5	Stability analysis of DFE				
6	6 Sensitivity analysis				
7	Numerical simulation and discussion	11			
8	8 Conclusion and future scope				

#### 1. Introduction

Over along with the few century, Novel illnesses have arisen in diverse places worldwide. Monkeypox (Mpox) disease is also one of them. Mpox is a viral disease caused by the MPXV [1], classified under the orthopox virus genus in the Poxviriade family. Three more human viruses belong to this category: the vaccinia virus, the variola virus, and the cowpox virus, which causes smallpox [2, 3]. Mpox and smallpox share similar clinical symptoms. The condition has developed as a severe public health concern throughout Africa, particularly in the central and western regions, because it primarily affects individuals who live near tropical rainforests. Researchers have increasingly focused on viruses over the past couple of years, seeking treatment for these ailments [4]. The first human case of the zoonotic ortho-pox DNA virus is Mpox. It occurred in the democratic republic of the Congo in 1970 [5], which had previously

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AGRAWAL ET AL.

been seen as smallpox. Since that time, the majority of cases have been recorded in rural regions of central and western Africa. This virus has spread to the Congo, Benin, Cameroon, Gabon, Nigeria, and elsewhere.

Nigeria saw one of the crucify eruption in 2017 [6,7], with 200 confirmed cases, 500 suspected cases, and a 3% death rate, and cases are still being recorded up to this day. A further outbreak outside of Africa happened in the USA in 2007 and was directly related to an infected pet, nominated as patient zero [8]. There were 70 confirmed cases as a result of this outbreak. With the continued identification of confirmed cases, more research is needed to appreciate the immunology, transmission patterns, and source of Mpox infection and develop treatments and vaccines. Mpox, a recently resurfaced virus since June 2022, has garnered significant interest among researchers striving to develop novel preventive measures to manage such viruses.

Between the crucial period (May 13 to May 21, 2022), a total of 92 obsessive cases were stated in many non-endemic regions, with no deaths reported so far. Mpox is a zoonotic viral disease. It mainly affects animals, especially monkeys, squirrels, and rodents. This illness can roll out from animals to people [9–11]. It can also roll out from person to person by close contact with infected people, typically through the transmission of gasping droplets or direct contact with skin pores and bodily fluids [12–14]. In addition to these mechanisms, it can be transferred by contact with contaminated surfaces or objects, referred to as ecological dissemination [15, 16].

The organization of this paper is as follows: In Section (2), we develop our model with the explanation. Section (3) provides the bounded solution with positivity. Section (4) describes equilibria analysis in which we have found some equilibrium points and basic reproduction numbers. Section (5) represents the stability analysis of the model. In Section (6), we investigate the sensitivity of the model. Section (7) describes numerical simulation with a graphical discussion of this proposed model. The last Section (8) concludes with the results and discussion of this paper.

#### 2. Model formulation

This section seeks to construct a compartmental system to examine the kinetics of MPXV spread in humans and primates who are not human (wild animals) [17]. We have categorized the two kinds whereas, the human populace is classified into seven compartments, namely susceptible human populace  $S_h(t)$ , vaccinated human populace  $V_h(t)$ , exposed human populace  $E_h(t)$ , quarantined human populace  $Q_h(t)$ , infectious human populace  $I_h(t)$ , recovered (removed) human populace  $R_h(t)$ , hospitalized human populace  $H_h(t)$  and the rodent populace are classified as susceptible rodent populace  $S_r(t)$ , exposed rodent populace  $E_r(t)$ , infectious rodent populace  $I_r(t)$ , and recovered (removed) rodent populace  $R_r(t)$ .

Birth and demise represent the two fundamental processes through which individuals are connected to humanity, occurring at rates of  $\Lambda_h$  and  $\mu_h$ , respectively. The rodent populace's birth and death rates are shown as  $\Lambda_r$  and  $\mu_r$ , respectively. Upon contact with an individual infected with MPXV, a healthy person may become infected. Upon the manifestation of symptoms associated with the illness within a time frame of 10 to 25 days, individuals are classified as part of the infected compartment. The parameter  $\alpha_h$  represents the transmission rate of the vaccine. In the event that the virus remains uncured, individuals in the vaccinated populace transition to the exposed populace at a rate of  $\gamma I_h$ . The susceptible populace transitions into the exposed class at rates of  $\beta_1$  and  $\beta_2$ . The exposed populace is categorized into three distinct compartments:  $I_h$ ,  $Q_h$ , and  $H_h$ . Each compartment is associated with rates of  $\alpha_1$ ,  $\alpha_2$ , and  $\alpha_3$ , respectively. Nonetheless, the quarantined human populace transitioned back to the susceptible compartment at a rate of  $\xi$ . At the rate of  $\tau_1$ , the quarantined human populace is admitted to a hospital, while at the rate of  $\tau_2$ , the quarantined populace experiences direct recovery. The admission rate of the infected human populace to the hospital is denoted as  $\zeta$ . The infected populace exhibited a recovery rate of  $\rho_h$ , while the hospitalized populace demonstrated a recovery rate of  $\beta$ .  $d_h$ presents the rate of disease spread among quarantined, hospitalized, and infected individuals. Rodents that are susceptible are exposed at a rate of  $\delta_r I_r$ . Exposed rodents transform infected  $\alpha_4$  is a rate of transform, and infected rodents recover at a rate of  $\rho_r$ . The parameter  $d_r$  represents the transmission rate of infectious diseases among rodent populaces. It is believed that individuals who have been vaccinated will not contract the disease at any point in their lives. In contrast, individuals who have not been successfully vaccinated are exposed at a rate denoted as  $\gamma$ . This group of humans is similarly affected,

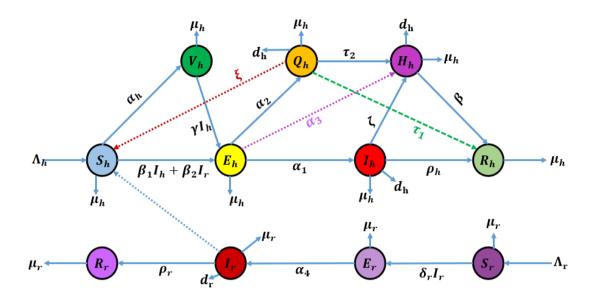


Figure 1: Schematic flow diagram of  $S_h V_h E_h Q_h I_h H_h R_h S_r E_r I_r R_r$ .

and it is also believed that transmission to rodents does not occur through human contact. Figure 1 illustrates the parameters transmitted from each compartment and the interactions occurring between the human and rodent populaces.

$$\frac{dS_{h}}{dt} = \Lambda_{h} - (\alpha_{h} + \mu_{h})S_{h}(t) + \xi Q_{h}(t) - (\beta_{1}I_{h}(t) + \beta_{2}I_{r}(t))S_{h}(t), 
\frac{dV_{h}}{dt} = \alpha_{h}S_{h}(t) - \mu_{h}V_{h}(t) - \gamma V_{h}(t)I_{h}(t), 
\frac{dE_{h}}{dt} = \gamma V_{h}(t)I_{h}(t) + (\beta_{1}I_{h}(t) + \beta_{2}I_{r}(t))S_{h}(t) - (\alpha_{2} + \mu_{h} + \alpha_{3} + \alpha_{1})E_{h}(t), 
\frac{dQ_{h}}{dt} = \alpha_{2}E_{h}(t) - (\xi + \mu_{h} + \tau_{1} + d_{h} + \tau_{2})Q_{h}(t), 
\frac{dI_{h}}{dt} = \alpha_{1}E_{h}(t) - (\zeta + \mu_{h} + \rho_{h} + d_{h})I_{h}(t), 
\frac{dH_{h}}{dt} = \zeta I_{h}(t) + \alpha_{3}E_{h}(t) + \tau_{2}Q_{h}(t) - (\beta + \mu_{h} + d_{h})H_{h}(t), 
\frac{dR_{h}}{dt} = \rho_{h}I_{h}(t) + \tau_{1}Q_{h}(t) + \beta H_{h}(t) - \mu_{h}R_{h}(t), 
\frac{dS_{r}}{dt} = \Lambda_{r} - \mu_{r}S_{r}(t) - \delta_{r}S_{r}(t)I_{r}(t), 
\frac{dE_{r}}{dt} = \delta_{r}S_{r}(t)I_{r}(t) - (\mu_{r} + \alpha_{4})E_{r}(t), 
\frac{dI_{r}}{dt} = \alpha_{4}E_{r}(t) - (\rho_{r} + \mu_{r} + d_{r})I_{r}(t), 
\frac{dR_{r}}{dt} = \rho_{r}I_{r}(t) - \mu_{r}R_{r}(t),$$
(2.1)

with starting conditions  $S_h(0) = S_{h0}$ ,  $V_h(0) = V_{h0}$ ,  $E_h(0) = E_{h0}$ ,  $Q_h(0) = Q_{h0}$ ,  $I_h(0) = I_{h0}$ ,  $H_h(0) = H_{h0}$ ,  $R_h(0) = R_{h0}$ ,  $S_r(0) = S_{r0}$ ,  $E_r(0) = E_{r0}$ ,  $I_r(0) = I_{r0}$ ,  $R_r(0) = R_{r0}$ .

## 3. Qualitative assessment of the model

This section, we discuss the topics of boundedness and positivity of solutions in more detail through essential theorems.

#### 3.1. Boundedness

The developed Mpox model (2.1) viable area is  $\Omega = \Omega_h \times \Omega_r$  is the proper set of  $\mathbb{R}^7_+ \times \mathbb{R}^4_+$ , where.

$$\Omega_{h} = \{(S_{h}(t), V_{h}(t), E_{h}(t), Q_{h}(t), I_{h}(t), H_{h}(t), R_{h}(t) \subseteq \mathbb{R}^{7}_{+} : 0 < N_{h} \leq \frac{\Lambda_{h}}{\mu_{h}}\} \text{ and } \Omega_{r} = \{(S_{r}, E_{r}, I_{r}, R_{r}) \subseteq \mathbb{R}^{4}_{+} : 0 < N_{r} \leq \frac{\Lambda_{r}}{\mu_{r}}\}.$$

**Theorem 3.1** The area indicated by  $\Omega = \Omega_h \times \Omega_r$  is a positively stable collection for model (2.1).

**Proof 3.1** The whole human population  $N_h = S_h + V_h + E_h + Q_h + I_h + H_h + R_h$ . Thus,

$$\begin{split} \frac{dN_h}{dt} &= \frac{dS_h}{dt} + \frac{dV_h}{dt} + \frac{dE_h}{dt} + \frac{dQ_h}{dt} + \frac{dI_h}{dt} + \frac{dH_h}{dt} + \frac{dR_h}{dt}, \\ \frac{dN_h}{dt} &\leq \Lambda_h - \mu_h \times N_h, \\ \lim_{t \to \infty} N_h(t) &\leq \frac{\Lambda_h}{\mu_h}. \end{split}$$

As a result, for all positive time values,  $N_h(t)$  converges as t approaches infinity. Consequently, the model's solution (2.1) with a starting condition in  $\Omega$  stays in  $\Omega$ . Incorporating the final four model (2.1) equations together, we now get

$$\begin{split} \frac{dN_r}{dt} &= \frac{dS_r}{dt} + \frac{dE_r}{dt} + \frac{dI_r}{dt} + \frac{dR_r}{dt}, \\ \frac{dN_r}{dt} &= \Lambda_r - \mu_r \times N_r, \\ \frac{dN_r}{dt} &+ \mu_r \times N_r \leq \Lambda_r. \end{split}$$

Thus,  $\lim_{t \to \infty} N_r(t) \leq \frac{\Lambda_r}{\mu_r}$ . Likewise, for all non-negative values of time,  $N_r(t)$  converges for t approaches to infinity; thus, the model's solution (2.1) with starting condition in  $\Omega$  stays in  $\Omega$ . This results in the deduction that, in  $\Omega = \Omega_h \times \Omega_r$ , the region is well-posed and epidemiologically viable.

#### 3.2. Positiveness of Solution

**Theorem 3.2** For any  $t \in [0, \infty)$ , all solutions of the model (2.1) are non-negative, given that the beginning conditions meet  $S_h(0) \ge 0, V_h(0) \ge 0, E_h(0) \ge 0, I_h(0) \ge 0, Q_h(0) \ge 0, H_h(0) \ge 0, R_h(0) \ge 0, S_r(0) \ge 0, I_r(0) \ge 0, I_r(0) \ge 0, R_r(0) \ge 0$ .

**Proof 3.2** Let  $W = \sup\{t \geq 0 : S_h(t) \geq 0, \ V_h(t) \geq 0, \ E_h(t) \geq 0, \ I_h(t) \geq 0, \ Q_h(t) \geq 0, \ H_h(t) \geq 0, \ R_h(t) \geq 0, \ S_r(t) \geq 0, \ E_r(t) \geq 0, \ I_r(t) \geq 0, \ R_r(t) \geq 0\}$  there exist t is the element of  $[0, \infty)$ . Obviously W > 0 and  $W < \infty$  from system (2.1).

$$\frac{dS_h}{dt} + (\mu_h + \alpha_h + \beta_1 I_h + \beta_2 I_r) S_h(t) \ge \Lambda_h, \tag{3.1}$$

let  $\mu_h + \alpha_h + \beta_1 I_h + \beta_2 I_r = \lambda_1$ ,

$$\frac{dS_h}{dt} + \lambda_1 S_h(t) \ge \Lambda_h.$$

By applying the method of integrating factors, we have that

$$\frac{d}{dt} \Big( S_h \exp\Big( \int_0^t \lambda_1(t) dt \Big) \Big) \ge \Lambda_h \exp\Big( \int_0^t \lambda_1(t) dt \Big).$$

The following results will occur when you take an integral

$$S_h \ge \left(S_h(0) + \int_0^t \Lambda_h \exp\left(\int_0^\tau \lambda_1(\tau)d\tau\right)dt\right) \exp\left(\int_0^t -\lambda_1(t)dt\right) \ge 0.$$

Using the same method, it is easily demonstrated that,  $V_h(t) \geq 0$ ,  $E_h(t) \geq 0$ ,  $I_h(t) \geq 0$ ,  $Q_h(t) \geq 0$ ,  $P_h(t) \geq 0$ , P

**Theorem 3.3** Let us consider  $f: \mathbb{L} \subseteq \mathbb{R} \times \mathbb{R}^n \to \mathbb{R}$  be a continuous function for every point in the area  $\vartheta|t-t_0| \leq a, ||z-z_0|| \leq b$ , where  $z=(z_1,z_2,z_3,\cdots,z_n)$  and  $z_0=(z_{10},z_{20},z_{30},\cdots,z_{n0}), ||f(t,z)|| \leq M$  and f(x,z) satisfies Lipschitz condition,  $||f(t,\bar{z_1})-f(t,\bar{z_2})|| \leq \bar{K}||\bar{t_1}-\bar{t_2}||, \bar{K}>0$  then for any pairs  $(t,\bar{z_1}),(t,\bar{z_2}) \in \vartheta \exists \delta>0$  such that there is a unique solution z(t) of the system  $\frac{dz}{dt}=f(t,z)\in |t-t_0|<\delta$ .

**Theorem 3.4** If  $\frac{\partial f}{\partial z_i}$  is continuous on a closed, convex, and bounded domain in  $\mathbb{R}$ , then f is Lipschitz.

**Theorem 3.5** Suppose that  $\mathbb{L} \subseteq \mathbb{R} \times \mathbb{R}^n$  be the domain described in Theorem 3.3. According to Theorem 3.4, a unique solution exists in the system (2.1).

## Proof 3.3 Let

$$\begin{split} &M_{1} = \Lambda_{h} + \xi Q_{h}(t) - (\mu_{h} + \alpha_{h})S_{h}(t) - (\beta_{1}I_{h}(t) + \beta_{2}I_{r}(t))S_{h}(t), \\ &M_{2} = \alpha_{h}S_{h}(t) - \mu_{h}V_{h}(t) - \gamma V_{h}(t)I_{h}(t), \\ &M_{3} = (\beta_{1}I_{h}(t) + \beta_{2}I_{r}(t))S_{h}(t) - (\alpha_{1} + \mu_{h} + \alpha_{2} + \alpha_{3})E_{h}(t) + \gamma V_{h}(t)I_{h}(t), \\ &M_{4} = \alpha_{2}E_{h}(t) - (\xi + \mu_{h} + d_{h} + \tau_{1} + \tau_{2})Q_{h}(t), \\ &M_{5} = \alpha_{1}E_{h}(t) - (\mu_{h} + \rho_{h} + \zeta + d_{h})I_{h}(t), \\ &M_{6} = \zeta I_{h}(t) + \alpha_{3}E_{h}(t) + \tau_{2}Q_{h}(t) - (\beta + \mu_{h} + d_{h})H_{h}(t), \\ &M_{7} = \rho_{h}I_{h}(t) + \tau_{1}Q_{h}(t) + \beta H_{h}(t) - \mu_{h}R_{h}(t), \\ &M_{8} = \Lambda_{r} - \mu_{r}S_{r}(t) - \delta_{r}S_{r}(t)I_{r}(t), \\ &M_{9} = \delta_{r}S_{r}(t)I_{r}(t) - (\alpha_{4} + \mu_{r})E_{r}(t), \\ &M_{10} = \alpha_{4}E_{r}(t) - (d_{r} + \rho_{r} + \mu_{r})I_{r}(t), \\ &M_{11} = \rho_{r}I_{r}(t) - \mu_{r}R_{r}(t). \end{split}$$

We demonstrate the boundedness and continuity of  $\frac{\partial M_i}{\partial z_j}$ ,  $i, j = 1, 2, 3, \dots, n$ . We have looked at each model equation's partial derivatives (2.1):

$$\begin{split} \left|\frac{\partial M_1}{\partial S_h}\right| &= |-(\mu_h + \alpha_h) - (\beta_1 I_h + \beta_2 I_r)| < \infty, \ \, \left|\frac{\partial M_1}{\partial V_h}\right| = |0| < \infty, \quad \left|\frac{\partial M_1}{\partial E_h}\right| = |0| < \infty, \\ \left|\frac{\partial M_1}{\partial Q_h}\right| &= |\xi| < \infty, \ \, \left|\frac{\partial M_1}{\partial I_h}\right| = |-\beta_1 S_h| < \infty, \ \, \left|\frac{\partial M_1}{\partial H_h}\right| = |0| < \infty, \quad \, \, \left|\frac{\partial M_1}{\partial R_h}\right| = |0| < \infty, \\ \left|\frac{\partial M_1}{\partial S_r}\right| &= |0| < \infty, \quad \left|\frac{\partial M_1}{\partial I_r}\right| = |-\beta_2 S_h| < \infty, \left|\frac{\partial M_1}{\partial R_r}\right| = |0| < \infty. \end{split}$$

Similarly for  $M_2$ 

$$\begin{split} \left|\frac{\partial M_2}{\partial S_h}\right| &= |\alpha_h| < \infty, \quad \left|\frac{\partial M_2}{\partial V_h}\right| = |-\mu_h - \gamma I_h| < \infty, \left|\frac{\partial M_2}{\partial E_h}\right| = |0| < \infty, \quad \left|\frac{\partial M_2}{\partial Q_h}\right| = |0| < \infty, \\ \left|\frac{\partial M_2}{\partial I_h}\right| &= |-\gamma V_h| < \infty, \quad \left|\frac{\partial M_2}{\partial H_h}\right| = |0| < \infty, \quad \left|\frac{\partial M_2}{\partial R_h}\right| = |0| < \infty, \quad \left|\frac{\partial M_2}{\partial S_r}\right| = |0| < \infty, \\ \left|\frac{\partial M_2}{\partial E_r}\right| &= |0| < \infty, \quad \left|\frac{\partial M_2}{\partial I_r}\right| = |0| < \infty, \quad \left|\frac{\partial M_2}{\partial R_r}\right| = |0| < \infty. \end{split}$$

Similarly for  $M_3$ 

$$\begin{split} \left| \frac{\partial M_3}{\partial S_h} \right| &= \left| \beta_1 I_h + \beta_2 I_h \right| < \infty, \quad \left| \frac{\partial M_3}{\partial V_h} \right| = \left| \gamma I_h \right| < \infty, \quad \left| \frac{\partial M_3}{\partial E_h} \right| = \left| - \left( \alpha_1 + \alpha_2 + \alpha_3 + \mu_h \right) \right| < \infty, \\ \left| \frac{\partial M_3}{\partial Q_h} \right| &= \left| 0 \right| < \infty, \quad \left| \frac{\partial M_3}{\partial I_h} \right| = \left| \beta_1 S_h + \gamma V_h \right| < \infty, \quad \left| \frac{\partial M_3}{\partial H_h} \right| = \left| 0 \right| < \infty, \quad \left| \frac{\partial M_3}{\partial R_h} \right| = \left| 0 \right| < \infty, \\ \left| \frac{\partial M_3}{\partial S_r} \right| &= \left| 0 \right| < \infty, \quad \left| \frac{\partial M_3}{\partial E_r} \right| = \left| 0 \right| < \infty, \quad \left| \frac{\partial M_3}{\partial I_r} \right| = \left| \beta_2 S_h \right| < \infty, \\ \left| \frac{\partial M_3}{\partial R_r} \right| &= \left| 0 \right| < \infty. \end{split}$$

Similarly for  $M_4$ 

$$\begin{split} \left|\frac{\partial M_4}{\partial S_h}\right| &= |0| < \infty, \quad \left|\frac{\partial M_4}{\partial V_h}\right| = |0| < \infty, \quad \left|\frac{\partial M_4}{\partial E_h}\right| = |\alpha_2| < \infty, \quad \left|\frac{\partial M_4}{\partial Q_h}\right| = |-(\tau_1 + \tau_2 + d_h + \xi + \mu_h)| < \infty, \\ \left|\frac{\partial M_4}{\partial I_h}\right| &= |0| < \infty, \quad \left|\frac{\partial M_4}{\partial H_h}\right| = |0| < \infty, \quad \left|\frac{\partial M_4}{\partial R_h}\right| = |0| < \infty, \quad \left|\frac{\partial M_4}{\partial S_r}\right| = |0| < \infty, \\ \left|\frac{\partial M_4}{\partial E_r}\right| &= |0| < \infty, \quad \left|\frac{\partial M_4}{\partial I_r}\right| = |0| < \infty, \quad \left|\frac{\partial M_4}{\partial R_r}\right| = |0| < \infty. \end{split}$$

Similarly for M<sub>5</sub>

$$\begin{split} \left|\frac{\partial M_5}{\partial S_h}\right| &= |0| < \infty, \quad \left|\frac{\partial M_5}{\partial V_h}\right| = |0| < \infty, \quad \left|\frac{\partial M_5}{\partial E_h}\right| = |\alpha_1| < \infty, \quad \left|\frac{\partial M_5}{\partial Q_h}\right| = |0| < \infty, \\ \left|\frac{\partial M_5}{\partial I_h}\right| &= |-(d_h + \mu_h + \zeta + \rho_h)| < \infty, \quad \left|\frac{\partial M_5}{\partial H_h}\right| = |0| < \infty, \quad \left|\frac{\partial M_5}{\partial R_h}\right| = |0| < \infty, \\ \left|\frac{\partial M_5}{\partial S_r}\right| &= |0| < \infty, \quad \left|\frac{\partial M_5}{\partial E_r}\right| = |0| < \infty, \quad \left|\frac{\partial M_5}{\partial I_r}\right| = |0| < \infty, \quad \left|\frac{\partial M_5}{\partial R_r}\right| = |0| < \infty. \end{split}$$

Similarly for  $M_6$ 

$$\begin{split} \left|\frac{\partial M_6}{\partial S_h}\right| &= |0| < \infty, \quad \left|\frac{\partial M_6}{\partial V_h}\right| = |0| < \infty, \quad \left|\frac{\partial M_6}{\partial E_h}\right| = |\alpha_3| < \infty, \quad \left|\frac{\partial M_6}{\partial Q_h}\right| = |\tau_2| < \infty, \\ \left|\frac{\partial M_6}{\partial I_h}\right| &= |\zeta| < \infty, \quad \left|\frac{\partial M_6}{\partial H_h}\right| = |-(\beta + \mu_h + d_h)| < \infty, \quad \left|\frac{\partial M_6}{\partial R_h}\right| = |0| < \infty, \\ \left|\frac{\partial M_6}{\partial S_r}\right| &= |0| < \infty, \quad \left|\frac{\partial M_6}{\partial E_r}\right| = |0| < \infty, \quad \left|\frac{\partial M_6}{\partial I_r}\right| = |0| < \infty, \quad \left|\frac{\partial M_6}{\partial R_r}\right| = |0| < \infty. \end{split}$$

Similarly for M7

$$\begin{split} \left|\frac{\partial M_7}{\partial S_h}\right| &= |0| < \infty, \quad \left|\frac{\partial M_7}{\partial V_h}\right| = |0| < \infty, \quad \left|\frac{\partial M_7}{\partial E_h}\right| = |0| < \infty, \quad \left|\frac{\partial M_7}{\partial Q_h}\right| = |\tau_1| < \infty, \\ \left|\frac{\partial M_7}{\partial I_h}\right| &= |\rho_h| < \infty, \quad \left|\frac{\partial M_7}{\partial H_h}\right| = |\beta| < \infty, \quad \left|\frac{\partial M_7}{\partial R_h}\right| = |-\mu_h| < \infty, \quad \left|\frac{\partial M_7}{\partial S_r}\right| = |0| < \infty, \\ \left|\frac{\partial M_7}{\partial E_r}\right| &= |0| < \infty, \quad \left|\frac{\partial M_7}{\partial I_r}\right| = |0| < \infty, \quad \left|\frac{\partial M_7}{\partial R_r}\right| = |0| < \infty. \end{split}$$

Similarly for  $M_8$ 

$$\begin{split} \left|\frac{\partial M_8}{\partial S_h}\right| &= |0| < \infty, \quad \left|\frac{\partial M_8}{\partial V_h}\right| = |0| < \infty, \quad \left|\frac{\partial M_8}{\partial E_h}\right| = |0| < \infty, \quad \left|\frac{\partial M_8}{\partial Q_h}\right| = |0| < \infty, \\ \left|\frac{\partial M_8}{\partial I_h}\right| &= |0| < \infty, \quad \left|\frac{\partial M_8}{\partial H_h}\right| = |0| < \infty, \quad \left|\frac{\partial M_8}{\partial R_h}\right| = |0| < \infty, \quad \left|\frac{\partial M_8}{\partial S_r}\right| = |-\mu_r - \delta_r I_r| < \infty, \\ \left|\frac{\partial M_8}{\partial E_r}\right| &= |0| < \infty, \quad \left|\frac{\partial M_8}{\partial I_r}\right| = |-\delta_r S_r| < \infty, \quad \left|\frac{\partial M_8}{\partial R_r}\right| = |0| < \infty. \end{split}$$

Similarly for M<sub>9</sub>

$$\begin{split} \left|\frac{\partial M_9}{\partial S_h}\right| &= |0| < \infty, \qquad \left|\frac{\partial M_9}{\partial V_h}\right| = |0| < \infty, \qquad \left|\frac{\partial M_9}{\partial E_h}\right| = |0| < \infty, \qquad \left|\frac{\partial M_9}{\partial Q_h}\right| = |0| < \infty, \\ \left|\frac{\partial M_9}{\partial I_h}\right| &= |0| < \infty, \qquad \left|\frac{\partial M_9}{\partial H_h}\right| = |0| < \infty, \qquad \left|\frac{\partial M_9}{\partial R_h}\right| = |0| < \infty, \qquad \left|\frac{\partial M_9}{\partial S_r}\right| = |\delta_r I_r| < \infty, \\ \left|\frac{\partial M_9}{\partial E_r}\right| &= |-(\alpha_4 + \mu_r)| < \infty, \qquad \qquad \left|\frac{\partial M_9}{\partial I_r}\right| = |\delta_r S_r| < \infty, \quad \left|\frac{\partial M_9}{\partial R_r}\right| = |0| < \infty. \end{split}$$

Similarly for  $M_{10}$ 

$$\begin{split} \left|\frac{\partial M_{10}}{\partial S_h}\right| &= |0| < \infty, \qquad \left|\frac{\partial M_{10}}{\partial V_h}\right| = |0| < \infty, \qquad \left|\frac{\partial M_{10}}{\partial E_h}\right| = |0| < \infty, \qquad \left|\frac{\partial M_{10}}{\partial Q_h}\right| = |0| < \infty, \\ \left|\frac{\partial M_{10}}{\partial I_h}\right| &= |0| < \infty, \qquad \left|\frac{\partial M_{10}}{\partial H_h}\right| = |0| < \infty, \qquad \left|\frac{\partial M_{10}}{\partial R_h}\right| = |0| < \infty, \qquad \left|\frac{\partial M_{10}}{\partial S_r}\right| = |0| < \infty, \\ \left|\frac{\partial M_{10}}{\partial E_r}\right| &= |\alpha_4| < \infty, \qquad \left|\frac{\partial M_{10}}{\partial I_r}\right| = |-(d_r + \mu_r + \rho_r)| < \infty, \qquad \qquad \left|\frac{\partial M_{10}}{\partial R_r}\right| = |0| < \infty. \end{split}$$

Similarly for  $M_{11}$ 

$$\begin{split} \left|\frac{\partial M_{11}}{\partial S_h}\right| &= |0| < \infty, \quad \left|\frac{\partial M_{11}}{\partial V_h}\right| = |0| < \infty, \quad \left|\frac{\partial M_{11}}{\partial E_h}\right| = |0| < \infty, \quad \left|\frac{\partial M_{11}}{\partial Q_h}\right| = |0| < \infty, \\ \left|\frac{\partial M_{11}}{\partial I_h}\right| &= |0| < \infty, \quad \left|\frac{\partial M_{11}}{\partial H_h}\right| = |0| < \infty, \quad \left|\frac{\partial M_{11}}{\partial R_h}\right| = |0| < \infty, \quad \left|\frac{\partial M_{11}}{\partial S_r}\right| = |0| < \infty, \\ \left|\frac{\partial M_{11}}{\partial E_r}\right| &= |0| < \infty, \quad \left|\frac{\partial M_{11}}{\partial I_r}\right| = |\rho_r| < \infty, \quad \left|\frac{\partial M_{11}}{\partial R_r}\right| = |-\mu_r| < \infty. \end{split}$$

We have proved that these partial derivatives are bounded and continuous; hence, in the region  $\mathbb{L}$ , there is a unique solution, the system (2.1), as per Theorem 3.5.

## 4. Equilibria analysis

This section, we have discussed equilibrium points, reproduction numbers, and stability of reproduction numbers in detail.

## 4.1. Equilibrium analysis

Equilibrium points for the model (2.1) be equalize all the right hand side will be zero. Then we get Mpox free equilibrium points for both population are free from disease and it is expressed by  $E^0$  described below:

$$E^{0} = (S_{h}^{0}, V_{h}^{0}, E_{h}^{0}, Q_{h}^{0}, I_{h}^{0}, H_{h}^{0}, R_{h}^{0}, S_{r}^{0}, E_{r}^{0}, I_{r}^{0}, R_{r}^{0}) = \left(\frac{\Lambda_{h}}{\alpha_{h} + \mu_{h}}, \frac{\alpha_{h} \Lambda_{h}}{\mu_{h} (\alpha_{h} + \mu_{h})}, 0, 0, 0, 0, 0, \frac{\Lambda_{r}}{\mu_{r}}, 0, 0, 0\right).$$

### 4.2. Fundamental reproduction number

The fundamental reproduction number for the proposed model can be regulated through the utilization of the next-generation matrix [18]. In the sense, we think about the non-negative matrix  $\mathbb{F}$  and the non-singular matrix  $\mathbb{V}$  indicating the generation of infection with the transition part in that order, Our  $S_h V_h E_h Q_h I_h H_h R_h S_r E_r I_r R_r$  system (2.1) is characterized as follows:

where 
$$Z = (E_h, Q_h, I_h, H_h, E_r, I_r)^T$$
,

$$F = \begin{bmatrix} (\beta_1 I_h + \beta_2 I_r) S_h + \gamma V_h I_h \\ 0 \\ 0 \\ 0 \\ \delta_r S_r I_r \\ 0 \end{bmatrix}$$

$$V = \begin{bmatrix} (\mu_h + \alpha_1 + \alpha_2 + \alpha_3)E_h \\ -\alpha_2 E_h + (\xi + \mu_h + d_h + \tau_1 + \tau_2)Q_h \\ -\alpha_1 E_h + (\mu_h + d_h + \zeta + \rho_h)I_h \\ -\alpha_3 E_h - \zeta I_h - \tau_2 Q_h + (\beta + \mu_h + d_h)H_h \\ (\mu_r + \alpha_4)E_r \\ -\alpha_4 E_r + (\mu_r + d_r + \rho_r)I_r \end{bmatrix}.$$

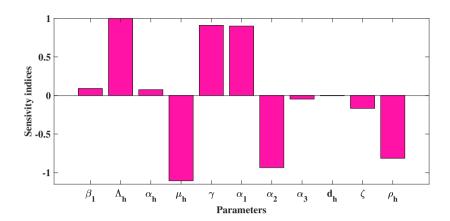


Figure 2: Sensitivity of  $R_0$  with respect to the human parameters defining it.

Now  $\mathbb F$  and  $\mathbb V$  can be represented as

$$\mathbb{V} = \begin{bmatrix} \kappa_1 & 0 & 0 & 0 & 0 & 0 \\ -\alpha_2 & \kappa_2 & 0 & 0 & 0 & 0 \\ -\alpha_1 & 0 & \kappa_3 & 0 & 0 & 0 \\ \alpha_3 & -\tau_2 & -\zeta & \kappa_4 & 0 & 0 \\ 0 & 0 & 0 & 0 & \kappa_5 & 0 \\ 0 & 0 & 0 & 0 & -\alpha_4 & \kappa_6 \end{bmatrix}.$$

Where

$$\begin{split} \kappa_1 &= (\mu_h + \alpha_1 + \alpha_2 + \alpha_3), \\ \kappa_2 &= (\zeta + \mu_h + d_h + \tau_1 + \tau_2), \\ \kappa_3 &= (\mu_h + d_h + \zeta + \rho_h), \\ \kappa_4 &= (\beta + \mu_h + d_h), \\ \kappa_5 &= (\mu_r + \alpha_4), \\ \kappa_6 &= (\mu_r + d_r + \rho_r). \end{split}$$

The fundamental reproduction number is denoted by  $R_0$ , and according to Dikemann [19],  $R_0$  is regarded as the spectral region of the next-generation matrix. Upon analyzing the aforementioned matrix, we obtain the two largest eigenvalues, which are as follows:

$$R_{0,h} = \frac{(\beta_1 S_h^0 + \gamma V_h^0)\alpha_1}{(\alpha_1 + \alpha_2 + \alpha_3 + \mu_h)(d_h + \mu_h + \zeta + \rho_h)}, \qquad R_{0,r} = \frac{\delta_r S_r^0 \alpha_4}{(\alpha_4 + \mu_r)(d_r + \mu_r + \rho_r)}$$

$$R_0 = \{R_{0,h}, R_{0,r}\}.$$

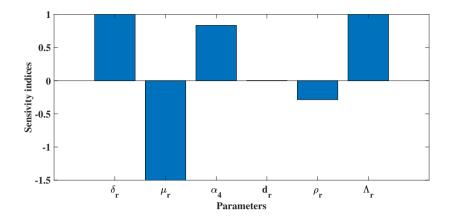


Figure 3: Sensitivity of  $R_0$  with respect to the rodent parameters defining it.

## 5. Stability analysis of DFE

**Theorem 5.1** When  $R_0 > 1$  for the DFE point  $E^0\left(\frac{\Lambda_h}{\alpha_h + \mu_h}, \frac{\alpha_h \Lambda_h}{\mu_h(\alpha_h + \mu_h)}, 0, 0, 0, 0, \frac{\Lambda_r}{\mu_r}, 0, 0, 0\right)$  are exists and it is locally asymptotically stable, otherwise it is unstable.

**Proof 5.1** The Jacobian matrix of the system is calculated to assess the local stability of  $E^0\left(\frac{\Lambda_h}{\alpha_h + \mu_h}, \frac{\alpha_h \Lambda_h}{\mu_h(\alpha_h + \mu_h)}, 0, 0, 0, 0, 0, \frac{\Lambda_r}{\mu_r}, 0, 0, 0\right)$  for the DFE is given by

Where,

$$\begin{split} & \mu_h + \alpha_h = A, \\ & \mu_h + \alpha_1 + \alpha_2 + \alpha_3 = B, \\ & \tau_1 + \tau_2 + \alpha_h + d_h + \mu_h + \xi = C, \\ & \zeta + d_h + \mu_h + \rho_h = D, \\ & \mu_h + \beta + d_h = E, \\ & \mu_r + \alpha_4 = F, \\ & d_r + \mu_r + \rho_r = G. \end{split}$$

Based on the matrix presented, we can readily identify seven eigenvalues as outlined below:  $\lambda_1 = -\mu_r, \quad \lambda_2 = -\mu_r, \quad \lambda_3 = -\mu_h, \quad \lambda_4 = -\mu_h, \quad \lambda_5 = -(\mu_h + \beta + d_h) \quad \lambda_6 = -\mu_h - \alpha_h, \quad \lambda_7 = -(\tau_1 + \tau_2 + \alpha_h + d_h + \mu_h + \xi).$ 

The provided equations can be utilized to determine the remaining eigenvalues  $\lambda_i$  (for i = 8, 9, 10, 11),

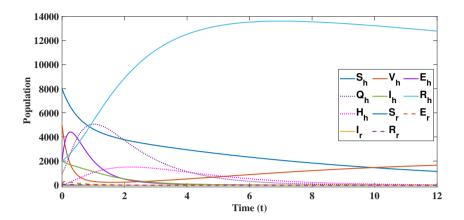


Figure 4: Dynamical behaviour of both population on time.

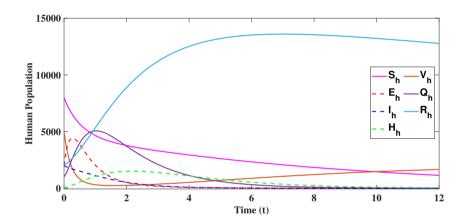


Figure 5: Dynamical behaviour of human population on time.

$$\lambda^{2} + (B+D)\lambda + BD - \alpha_{1} \left( \frac{\beta_{1} \Lambda_{h}}{\alpha_{h} + \mu_{h}} + \frac{\gamma \alpha_{h} \Lambda_{h}}{\mu_{h} (\alpha_{h} + \mu_{h})} \right) = 0, \tag{5.1}$$

$$\lambda^2 + (F+G)\lambda + FG - \alpha_4 \frac{\delta_r \Lambda_r}{\mu_r} = 0.$$
 (5.2)

As B+D>0,  $BD-\alpha_1\left(\frac{\beta_1\Lambda_h}{\alpha_h+\mu_h}+\frac{\gamma\alpha_h\Lambda_h}{\mu_h(\alpha_h+\mu_h)}\right)>0$ , utilizing the Routh-Hurwitz criteria, Equation (5.1) possesses the pair of roots with a negative real part. Furthermore, since F+G>0,  $FG-\alpha_4\frac{\delta_r\Lambda_r}{\mu_r}>0$  when  $R_0^h<1$ , applying the Routh-Hurwitz criterion, It can be concluded that Equation (5.2) possesses two roots with negative real parts as well.

All eigenvalues derived from the Jacobian matrix  $J_{E^0}$  demonstrate a characteristic where their non-negative elements are negative where  $R_0$  is less than 1, a condition satisfied by both  $R_0^h$  and  $R_0^r$ . This brings us to the theorem.

## 6. Sensitivity analysis

We analyse the effects of the parameter values, which should be categorized for dominating the infectious diseases, It may be accomplished via ascertaining the toughness of coordinate among every parameter in the model to control the fundamental reproduction number  $R_0$  by using the partial order

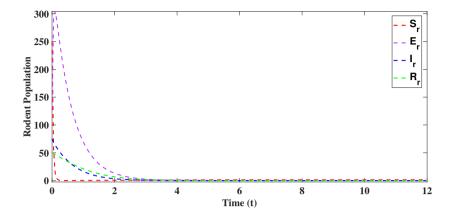


Figure 6: Dynamical behaviour of rodent population on time.

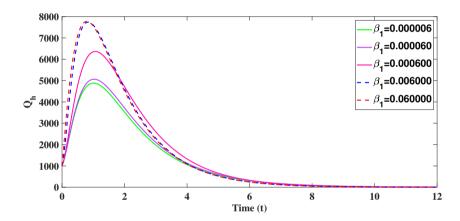


Figure 7: Variation of human quarantined population on time at the rate of  $\beta_1$ .

correlation coefficient [23,24]. Now we tested a significant parameters of  $R_0$ . The findings of the sensitivity analysis are shown in Figures 2 and 3. Now we obtain the sensitivity analysis of  $R_0$  for each parameters by using analytical expression given as

$$S_p^{R_0} = \frac{p}{R_0} \times \frac{\partial R_0}{\partial p},$$

Where p is any parameter contained in  $R_0$ . With the regarding of the above formulae, we analyse sensitivity indices and results are shown in Figures 2 and 3.

## 7. Numerical simulation and discussion

For the vital result of this  $S_h V_h E_h Q_h I_h H_h R_h S_r E_r I_r R_r$  model (2.1) of the MPXV, we also use ODE45 test in MATLAB and evaluate the variables that are best fit in our  $S_h V_h E_h Q_h I_h H_h R_h S_r E_r I_r R_r$  model. The suggested  $S_h V_h E_h Q_h I_h H_h R_h S_r E_r I_r R_r$  model system (2.1) has 23 parameters listed above with their references and values. We describe results of the model (2.1) graphically by applying some estimated and discussed parameters from Table 1.

Figure 4 illustrates the temporal dynamics of the total population (human and rodent) over several months. The susceptible human population steadily decreases while the recovered human population correspondingly increases, indicating successful disease progression towards recovery. Figure 5 further highlights the positive impact of vaccination, where an increase in recovered humans corresponds with vaccine rollout. In contrast, Figure 6 shows a general decline in the rodent population compartments over time, reflecting the effect of disease and control measures on the vector population.

12 Agrawal et al.

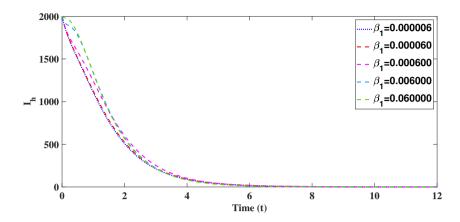


Figure 8: Variation of infected human population on time at the rate of  $\beta_1$ .

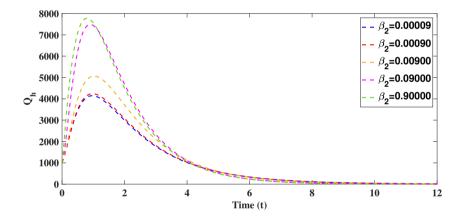


Figure 9: Variation of human quarantined population on time at the rate of  $\beta_2$ .

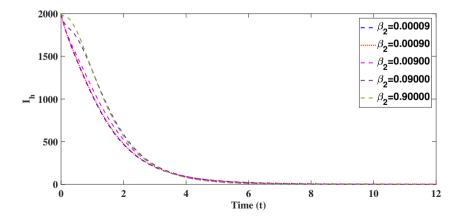


Figure 10: Dynamical behaviour of human infected population on time at the rate of  $\beta_2$ .

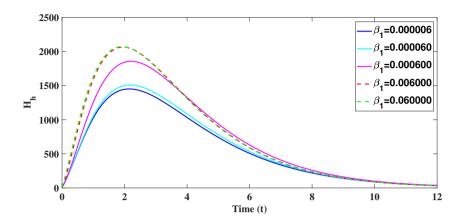


Figure 11: Dynamical behaviour of human hospitalised population on time at the rate of  $\beta_1$ .

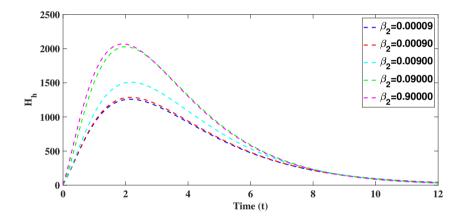


Figure 12: Dynamical behaviour of human hospitalised population on time at the rate of  $\beta_2$ .

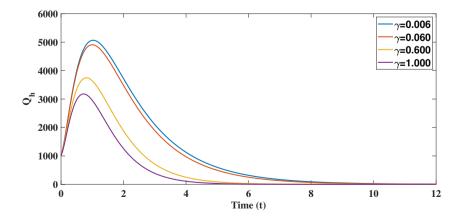


Figure 13: Dynamical behaviour of quarantined human population on time at the rate of  $\gamma$ .

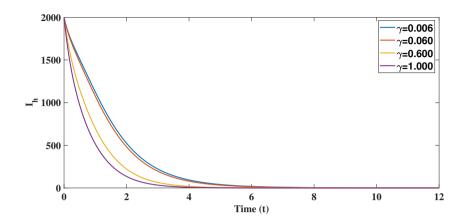


Figure 14: Dynamical behaviour of infected human population on time at the rate of  $\gamma$ .

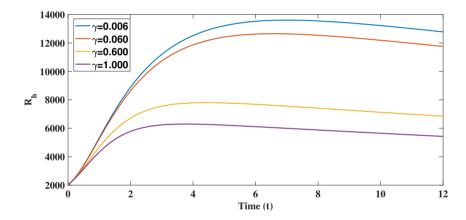


Figure 15: Dynamical behaviour of recovered human population on time at the rate of  $\gamma$ .

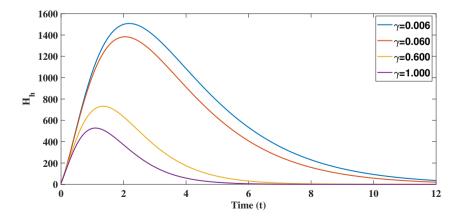


Figure 16: Dynamical behaviour of hospitalised human population on time at the rate of  $\gamma$ .

Parameter	Value	References	Parameter	Value	References
$\Lambda_h$	0.029	[20]	$\mu_h$	0.02	[20]
$\alpha_h$	0.1	[21]	$ ho_r$	0.6	[20]
ξ	0.2	[22]	$\gamma$	0.006	Assumed
$\alpha_1$	0.2	[17]	$\alpha_2$	2	[17]
$\alpha_3$	0.1	Assumed	$\alpha_4$	0.3	[21]
$d_h$	0.0002	Estimated [17]	$ ho_h$	0.83	[20]
ζ	0.17	Assumed	$ au_1$	0.52	[17]
$ au_2$	0.2	Assumed	$\beta$	0.5	Assumed
$\beta_1$	0.00006	[17, 20]	$\beta_2$	0.009	[17, 20]
$\Lambda_r$	2	[20]	$\mu_r$	1.5	[20]
$\delta_r$	0.4	[20]	$d_r$	0.0023	Assumed

Table 1: Description of parameters in the model.

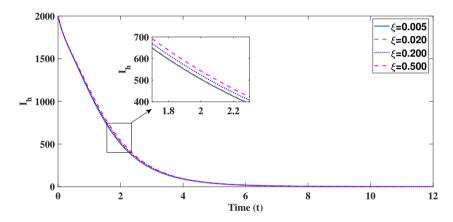


Figure 17: Dynamical behaviour of infected human population on time at the rate of  $\xi$ .

Figures 7 and 8 explore the influence of the parameter  $\beta_1$  on quarantined and infected humans. As  $\beta_1$  increases, more individuals enter quarantine, leading to a reduction in active infections, which underscores the effectiveness of quarantine as a control strategy. Similarly, Figures 9 and 10 demonstrate that increasing  $\beta_2$  also raises quarantine numbers while decreasing infections, reinforcing the importance of timely isolation measures. Hospitalized population dynamics depicted in Figures 11 and 12 show an increasing trend with rising  $\beta_1$  and  $\beta_2$ , highlighting the critical role of healthcare capacity in managing disease burden.

The parameters' effects on quarantined, infectious, recovered, and hospitalized populations are further analyzed in Figures 13 to 16 at varying rates  $\gamma$ . These results reveal that quarantined and hospitalized populations initially rise and then decline over time, while infectious cases steadily decrease and recovered cases increase, suggesting effective control interventions. Figure 17 illustrates the decrease in infected human population with an increase in the recovery rate  $\xi$ , emphasizing the importance of improving treatment and recovery processes.

The model has been simulated using the initial conditions:  $S_h(0) = 8000$ ,  $V_h(0) = 5000$ ,  $E_h(0) = 2000$ ,  $Q_h(0) = 1000$ ,  $I_h(0) = 2000$ ,  $R_h(0) = 2000$ ,  $S_r(0) = 250$ ,  $E_r(0) = 125$ ,  $I_r(0) = 75$ ,  $R_r(0) = 50$ ,  $H_h(0) = 10$ . The simulation results indicate that integrating vaccination, quarantine, and hospitalization strategies can effectively reduce the spread of monkeypox. These findings highlight the importance of achieving high vaccination coverage, prompt isolation of infected individuals, and sufficient hospital resources to control outbreaks. Additionally, managing the rodent vector population plays a critical role in preventing zoonotic transmission.

AGRAWAL ET AL.

#### 8. Conclusion and future scope

This study presents a comprehensive compartmental model for the transmission dynamics of monkeypox virus (MPXV) in human and rodent populations. Our analysis shows that maintaining high vaccination coverage and robust quarantine and hospitalization protocols effectively reduce disease spread and help contain potential epidemics, as indicated by the basic reproduction number  $(R_0)$ . If  $R_0 > 1$ , MPXV has epidemic potential, whereas  $R_0 < 1$  suggests eventual disease elimination or endemic stability. From a policy perspective, our findings advocate for integrated public health interventions combining vaccination campaigns, early case detection with quarantine, and strengthening healthcare infrastructure to manage hospitalizations. Additionally, vector control through rodent population management should be prioritized to minimize zoonotic spillover risks.

Future work may extend this model by incorporating age-structured populations and delay differential equations to capture latency and incubation periods better, as demonstrated in [25]. Furthermore, control strategies like awareness programs can enhance understanding of their role in transmission reduction [26]. Incorporating fractional-order derivatives may also provide deeper insight into memory effects in disease progression, potentially improving predictive accuracy and intervention planning [9, 27].

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18 Agrawal et al.

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