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Mathematical Modeling and Analysis of Monkeypox Transmission Dynamics with Treatment and Quarantine Interventions

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Abstract

This study presents a comprehensive mathematical model to understand the transmission dynamics of monkeypox, incorporating multiple compartments for both human and rodent populations, which are essential in the spread of the virus. The model captures zoonotic transmission (from rodents to humans)

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and human-to-human transmission, including compartments for susceptible, exposed, infected, quarantined, treated, and recovered humans, as well as susceptible, exposed, and infected rodents. Numerical simulations show how interventions such as reducing contact rates, quarantining infected individuals, and promoting effective treatment can significantly control the spread of the virus. Sensitivity analysis reveals that parameters with positive sensitivity indices, such as contact rates, enhance the spread of monkeypox, whereas parameters with negative sensitivity indices, like the treatment rate of infected humans, reduce transmission. The results demonstrate that reducing contact rates, especially between susceptible and infected humans and rodents, plays a crucial role in disease control. This study provides valuable insights for policymakers and public health officials to effectively manage monkeypox outbreaks.

Keywords: Monkeypox dynamics , Human-rodent interactions , Mathematical epidemiology, Sensitivity analysis, Endemic equilibrium

INTRODUCTION

Monkeypox is a zoonotic viral disease that has drawn significant attention due to its potential to cause outbreaks in humans, particularly in regions where there is close contact with wildlife or among individuals in close proximity to one another. First identified in laboratory monkeys in 1958 and reported in humans in the Democratic Republic of the Congo (DRC) in 1970, the virus belongs to the Orthopoxvirus genus, which also includes variola (smallpox) and vaccinia viruses (Ladnyj et al., 1972). Although smallpox was eradicated in 1980, monkeypox remains a global health threat, especially in Central and West Africa, where it continues to circulate in animal reservoirs and periodically spills over into human populations (Bunge et al., 2022).In recent years, monkeypox has resurfaced as a global concern due to rising infection rates and international spread, exacerbated by increased urbanization, deforestation, and climate change, which disturb wildlife habitats. These factors elevate the likelihood of zoonotic transmission, where humans come into closer contact with the animal species that host the monkeypox virus (Nguyen et al., 2022). Furthermore, the disruption of healthcare services and vaccination campaigns for smallpox has left many individuals susceptible to monkeypox, as cross-protection from the smallpox vaccine is no longer prevalent (Reynolds et al., 2019). This combination of environmental, ecological, and healthcare challenges underscores the need for thorough investigation into the transmission dynamics of monkeypox. The recent surge in monkeypox cases can be attributed to several interrelated factors, including environmental, social, and immunological changes. One of the primary causes is the increasing encroachment of humans into wildlife habitats due to deforestation and urbanization, which has heightened human exposure to the animals that naturally harbor the monkeypox virus (Nguyen et al., 2022). Additionally, declining immunity in populations once protected by smallpox vaccinations has left many individuals more vulnerable, as the cessation of routine smallpox immunization following its eradication has weakened cross-protection against monkeypox (Bunge et al., 2022). Furthermore, international travel and close-contact transmission in dense urban settings have facilitated the rapid spread of the virus across borders. Solutions to this growing public health concern involve a combination of strategies. These include reviving targeted vaccination programs, particularly for high-risk populations, enhancing surveillance and diagnostic capabilities, and promoting early quarantine and treatment interventions (Parker et al., 2022). Increasing public awareness about preventive hygiene measures and maintaining strong international cooperation to contain outbreaks are also vital to mitigating future epidemics. Transmission of the monkeypox virus typically occurs through direct contact with the body fluids, skin lesions, or respiratory droplets of infected individuals or animals. Human-to-human transmission, while less efficient than zoonotic transmission, has been observed primarily in households or healthcare settings where close contact is more frequent (Petersen et al., 2019). The incubation

period ranges from 6 to 13 days, though it can extend to 21 days in some cases. Symptoms often resemble those of smallpox, including fever, headache, muscle pain, and the characteristic rash that progresses from macules to pustules before scabbing over (World Health Organization, 2022).

Mathematical models provide a powerful tool for understanding the dynamics of infectious diseases like monkeypox. Such models can help researchers predict how the virus spreads within populations, identify the most critical factors that influence transmission, and evaluate the effectiveness of different interventions, such as quarantine and treatment. For example, by simulating how various intervention strategies reduce transmission rates or delay outbreaks, public health authorities can make informed decisions about resource allocation and preparedness (Koopman et al., 2021). This becomes especially relevant in regions where healthcare infrastructure is strained, and timely interventions can mean the difference between containment and widespread transmission.Current treatment and prevention strategies for monkeypox include the use of antiviral drugs like tecovirimat, which has shown promise in reducing the severity and duration of symptoms (Parker et al., 2022). In addition, supportive care and quarantine remain key strategies in limiting the spread of the virus, particularly in areas with limited access to vaccines or medical resources. The global community has also recognized the need to revive smallpox vaccination campaigns in populations at risk of monkeypox, as this can offer cross-protection against the virus.

The resurgence of monkeypox and its global spread has necessitated the use of mathematical models to understand its transmission dynamics and predict outbreak scenarios. Recent literature has explored different approaches and models to assess interventions, estimate transmission rates, and predict the spread of the disease under various conditions. Below is a review of five recent studies that focus on mathematical modelling of monkeypox: Endo et al. (2022) conducted a study that developed a stochastic compartmental model to estimate the reproduction number of monkeypox in non-endemic countries, focusing on the outbreak observed in 2022. The model separated populations into susceptible, exposed, infectious, and recovered compartments (SEIR) and analyzed contact rates and the role of human mobility in spreading the virus. They found that the basic reproduction number was higher in regions with dense populations and more frequent international travel. Their model also emphasized the role of early intervention, particularly quarantine and isolation, in reducing transmission (Endo et al., 2022). Grant et al. (2023) applied a mathematical model to evaluate the impact of vaccination strategies on monkeypox transmission. Using data from the 2022 outbreak in the UK, their model integrated vaccination coverage rates and timing of administration into a compartmental model. They demonstrated that even with limited vaccine availability, targeted vaccination of high-risk groups could significantly reduce transmission. The model also highlighted the importance of timely vaccine deployment and rapid case detection to curb outbreaks (Grant et al., 2023). Yinka-Ogunleye et al. (2022) developed a deterministic model to simulate monkeypox transmission in West Africa, considering zoonotic transmission from animal reservoirs and human-to-human transmission. Their model showed that while zoonotic transmission remains a significant source of infection, human-to-human transmission plays a growing role in the spread, particularly in urban areas. The model recommended enhancing surveillance and public health measures, especially in regions with high contact between humans and wildlife (Yinka-Ogunleye et al., 2022). Al-Najjar et al. (2023) focused on the effects of social behavior changes, such as reduced contact rates and increased hygiene practices, on monkeypox transmission. They created a dynamic model incorporating behavioral feedback mechanisms where public perception of risk influenced contact rates. Their findings suggested that social distancing and behavior changes

during outbreaks significantly decreased the effective reproduction number (Al-Najjar et al., 2023). The model underscored the need for public health campaigns that encourage behavior modification during outbreaks. Hebert-Dufresne et al. (2022) introduced a network-based model to simulate monkeypox spread within social and sexual networks. Unlike traditional compartmental models, their approach accounted for the heterogeneity in contact patterns, particularly in sexual transmission contexts, such as the disproportionate impact of monkeypox on men who have sex with men (MSM). The model provided insights into the super-spreader potential within tightly knit communities and emphasized the importance of targeting specific subpopulations for intervention strategies like vaccination and education (Hebert-Dufresne et al., 2022).The present study considered a comprehensive mathematical model to understand the transmission dynamics of monkeypox by incorporating multiple compartments for both the human and rodent populations, which play a crucial role in the spread of the virus. The model integrates human and rodent hosts to capture both zoonotic transmission (from rodents to humans) and human-to-human transmission.

Materials and Methods

We develop a deterministic compartmental model on the transmission dynamics of monkeypox consisting of human and rodent population. The human population comprises of six epidemiological groups, Susceptible humans $S_h(t)$, exposed humans $E_h(t)$, infected humans $I_h(t)$, quarantined humans $Q_h(t),$, treated humans $T_h(t)$ and recovered humans $R_h(t)$. The rodent population is subdivided into three compartments namely: Susceptible rodents $S_r(t)$, exposed humans $E_r(t)$, infected rodents $I_r(t)$. The recruitment rate of human population is given as Λ_h and β_{rh} is the effective contact rate with the probability of human being infected with monkeypox virus due to contact with infected rodent. β_{hh} denotes effective contact rate and the probability human been infected with monkeypox virus due to contact with infectious human and ω_h is the progression rate from exposed human to highly infected human whereas the treatment rate of infected humans is ρ_h and the recovery rate of treated humans is τ_h. θ_h is the quarantined rate of exposed humans whereas γ_h is the rate at which quarantined humans become susceptible and the recovery rate of quarantined humans is ψ_h. Natural death occurs in the humans and rodents population at the rates μ_h and μ_r respectively. The infected humans and rodents population decrease by the disease induced death rates δ_h and δ_r respectively. Λ_r is rodents recruitment rate and β_{rr} is the effective contact rate with the probability of rodent being infected per contact rate with already infected rodent. ω_r is the progression rate from exposed rodent population to highly infectious rodent population and σ_h is immunity loss rate. The transition from one epidemiological compartment to another is illustrated in figure 1 below.

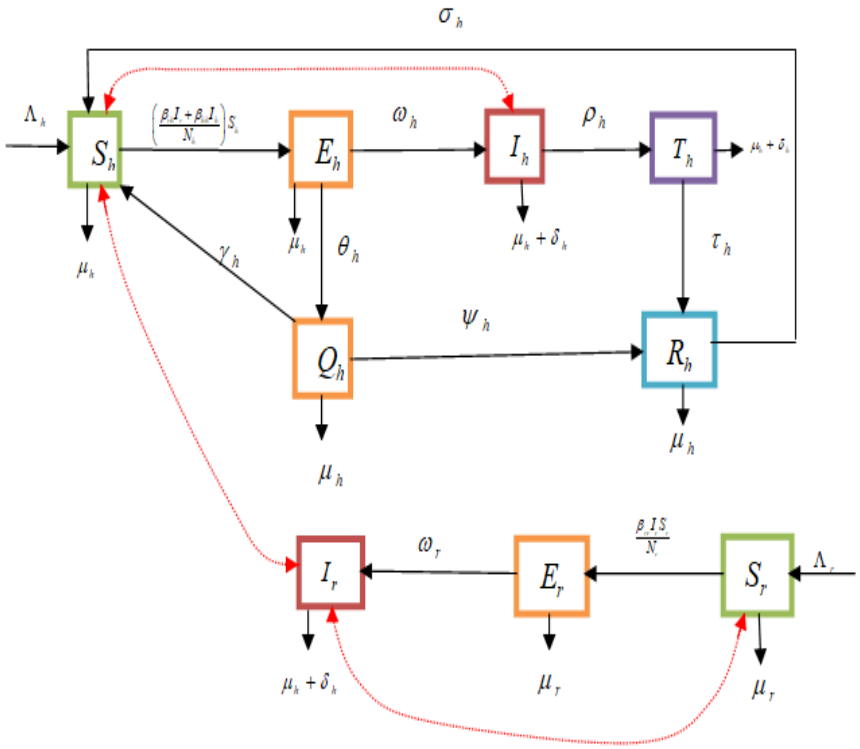


Figure 1. Schematic diagram for the model.

Table 1. Description of variables and parameters

Variables	Description
$S_h(t)$	Susceptible human
$E_h(t)$	Exposed human
$Q_h(t)$	Isolated/quarantined human
$I_h(t)$	Infected human
$T_h(t)$	Treated human
$R_h(t)$	Recovered human
$S_r(t)$	Susceptible rodent
$E_r(t)$	Exposed rodent
$I_r(t)$	Infected rodents
Λ_h	Human recruitment rate
β_{rh}	Rodent contact rate to humans
β_{hh}	Human to human contact rate
β_{rr}	Rodent to rodent to contact rate
ω_h	Progression rate from exposed human to infected human
ρ_h	Human treatment rate
θ_h	Quarantined rate of exposed humans
γ_h	Quarantine rate of susceptible humans

ψ_h	Recovery rate of quarantined individual
τ_h	Recovery rate of treated humans
γ_h	Rate at which quarantined human become susceptible
σ_h	Immunity loss rate for human population
δ_h	Disease induced death rate for humans
δ_r	Disease induce death rate for rodents
μ_h	Natural death rate for human
μ_r	Natural death rate for rodents
ω_r	Progression rate from exposed rodent to infected rodent

Model Equations

The model is governed by the following set of differential equations.

$$\frac{dS_h}{dt} = \Lambda_h + \sigma_h R_h + \alpha_h Q_h - \frac{(\beta_{rh} \mathrm{I}_r + \beta_{hh} \mathrm{I}_h)\mathrm{S}_h}{N_h} - \mu_h S_h$$

$$\frac{dE_h}{dt} = \frac{(\beta_{rh} \mathrm{I}_r + \beta_{hh} \mathrm{I}_h)\mathrm{S}_h}{N_h} - (\omega_h + \theta_h + \mu_h)\mathrm{E}_h$$

$$\frac{dI_h}{dt} = \omega_h E_h - (\rho_h + \delta_h + \mu_h) I_h$$

$$\frac{dQ_h}{dt} = \theta_h E_h - (\alpha_h + \psi_h + \mu_h)\mathrm{Q}_h \qquad (1)$$

$$\frac{dT_h}{dt} = \rho_h I_h - (\tau_h + \delta_h + \mu_h)\mathrm{T}_h$$

$$\frac{dR_h}{dt} = \psi_h Q_h + \tau_h T_h - (\sigma_h + \mu_h)\mathrm{R}_h$$

$$\frac{dS_r}{dt} = \Lambda_r - \frac{\beta_{rr} I_r S_r}{N_r} - \mu_r \mathrm{S}_r$$

$$\frac{dE_r}{dt} = \frac{\beta_{rr} I_r S_r}{N_r} - (\omega_r + \mu_r)\mathrm{E}_r$$

$$\frac{dI_r}{dt} = \omega_r \mathrm{E}_r - (\delta_r + \mu_r)\mathrm{I}_r$$

Model Analysis

For the human population,

$$N_h = S_h + E_h + I_h + \mathrm{Q}_h + T_h + R_h$$

The differential equation yields

$$\frac{dN_h}{dt} = \Lambda_h - \delta_h I_h - \mu_h N_h \qquad (2)$$

For the rodent population,

$$N_r = S_r + E_r + I_r$$

The differential equation also gives

$$\frac{dN_r}{dt} = \Lambda_r - (\mu_r + \Lambda_r) N_r \qquad (3)$$

Lemma 1

Let $\left(S_h, E_h, I_h, \mathrm{Q}_h, T_h, R_h, S_r, E_r, I_r\right)$ be the solution of the model (1) with initial conditions in a epidemiological feasible region $D = D_h \times D_r$ with:

$$D_h = S_h, E_h, I_h, \mathrm{Q}_h, T_h, R_h \in R_+^6 : N_h \le \frac{\Lambda_h}{\mu_h} \qquad (4)$$

and

$$D_r = S_r, E_r, I_r \in R_+^3 : N_r \le \frac{\Lambda_r}{\mu_r} \qquad (5)$$

Then D is non-negative invariant
From the result of Somma et al (2019), we obtain

$$0 \le N_h(t) \le N_h(0)e^{-\mu_h(t)} + \frac{\Lambda_h}{\mu_h}\left(1 - e^{-\mu_h(t)}\right) \qquad (6)$$

and

$$0 \le N_r(t) \le N_r(0)e^{-(\mu_r + \Lambda_r)t} + \frac{\Lambda_h}{\mu_h}\left(1 - e^{-(\mu_r + \Lambda_r)t}\right) \qquad (7)$$

Therefore, the set D is positively invariant for all t

Asymptotic Stability of the Disease Free Equilibrium of the Monkeypox Model

The disease-free equilibrium (DFE) refers to a state where the disease is completely absent from the population, meaning no individual is infected. This represents a situation where the disease has either been eradicated or is not capable of spreading (Watmough, 2002).. If small changes in the system do not trigger a return of the disease, the DFE is considered stable, indicating that eradication is possible. The DFE point, as shown below, is pivotal in guiding public health strategies.

$$\eta_0 = \left\{S_h^*, E_h^*, I_h^*, \mathrm{Q}_h^*, T_h^*, R_h^*, S_r^*, E_r^*, I_r^*\right\} = \left\{\frac{\Lambda_h}{\mu_h}, 0, 0, 0, 0, 0, \frac{\Lambda_r}{\mu_r}, 0, 0\right\}$$

Basic Reproduction Number

The basic reproduction number, often referred to as (R_0), is the average number of new infections caused by a single infected person in a population where no one has immunity. This is an important measure in epidemiology for evaluating how easily a disease can spread. When R_0 is greater than 1, it shows that each infected person is likely to pass the disease to more than one individual, increasing the chance of an outbreak (Diekmann&Heesterbeek, 2000). If R_0 is less than 1, the disease is expected to fade away over time. Understanding R_0 is essential for creating effective public health strategies, as it helps predict the likelihood of an epidemic and informs decisions on interventions like vaccination programs and social distancing measures (Watmough, 2002). In this study, we calculate R_0 using the next generation operator method on the dynamic system (1), as detailed below.
Hence, it follows that

$R_0 = \rho\left(FV^{-1}\right)$ where ρ is the dominant eigenvalue of FV^{-1}

$$F=\begin{bmatrix} 0 & \beta_{hh} & 0 & 0 & 0 & \beta_{rh} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \beta_{rr} \\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}, V=\begin{bmatrix} P_1 & 0 & 0 & 0 & 0 & 0 \\ -\omega_h & P_2 & 0 & 0 & 0 & 0 \\ \theta_h & 0 & P_3 & 0 & 0 & 0 \\ 0 & -\rho_h & 0 & P_4 & 0 & 0 \\ 0 & 0 & 0 & 0 & P_6 & 0 \\ 0 & 0 & 0 & 0 & -\omega_t & P_7 \end{bmatrix}$$

$$J=\begin{bmatrix} \frac{\beta_{hh}\omega_h}{P_2P_1} & \frac{\beta_{hh}}{P_2} & 0 & 0 & \frac{\beta_{rh}\omega_r}{P_6P_7} & \frac{\beta_{rh}}{P_7} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \frac{\beta_{rr}\omega_r}{P_6P_7} & \frac{\beta_{rr}}{P_7} \\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

The eigenvalues of FV^{-1}are

$$R_0^r=\frac{\beta_{rr}\omega_r}{P_6P_7}$$

$$R_0^h=\frac{\beta_{hh}\omega_h}{P_2P_1}$$

$$R_0=\left(R_0^h,R_0^r\right)$$

Jacobian Matrix (LAS)

$$J=\begin{bmatrix} \frac{\beta_{hh}\omega_h}{P_2P_1} & \frac{\beta_{hh}}{P_2} & 0 & 0 & \frac{\beta_{rh}\omega_r}{P_6P_7} & \frac{\beta_{rh}}{P_7} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \frac{\beta_{rr}\omega_r}{P_6P_7} & \frac{\beta_{rr}}{P_7} \\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

The reduced Jacobian matrix becomes

$$J_1 = \begin{bmatrix} -P_1 & \beta_{hh} & 0 & \beta_{rh} \\ \omega_h & -P_2 & 0 & 0 \\ 0 & 0 & -P_6 & \beta_{rr} \\ 0 & 0 & \omega_r & -P_7 \end{bmatrix}$$

The characteristic polynomial becomes

$$\lambda^4 + \left(P_7 + P_6 + P_2 + P_1\right)\lambda^3 + \left(P_2P_1 + P_6P_1 + P_7P_1 + P_6P_2 + P_7P_2 + P_7P_6 - \beta_{hh}\omega_h - \omega_r\beta_{rr}\right)\lambda^2$$
$$+\left(P_1P_2P_6 + P_1P_2P_7 + P_1P_6P_7 - \omega_r\beta_{rr}P_1 + P_2P_6P_7 - \omega_r\beta_{rr}P_2 - P_6\beta_{hh}\omega_h - P_7\beta_{hh}\omega_h\right)\lambda$$
$$+P_1P2\left(1-R_0^r\right)+P_6P_7\left(1-R_0^h\right)$$

Applying the Routh Hurwitz criterion, we observe that the DFE is locally asymptotically stable.

$P_1 = (\omega_h + \theta_h + \mu_h)\ P_2 = (\rho_h + \delta_h + \mu_h)\ P_3 = (\alpha_h + \psi_h + \mu_h)\ P_4 = (\tau_h + \delta_h + \mu_h)\ P_5 = (\sigma_h + \mu_h)$
$P_6 = (\omega_r + \mu_r)\ P_7 = (\delta_r + \mu_r)$

Global Asymptotic Stability of the Disease Free Equilibrium Point of the Monkeypox Model.

Global asymptotic stability of the disease-free equilibrium (DFE) in the monkeypox model means that, over time, the entire system will naturally move towards a state where the disease is eradicated, regardless of the initial number of infected individuals. This occurs if the basic reproduction number R_0 is less than 1, indicating that, on average, an infected person transmits the virus to fewer than one other person. In such cases, even if a small number of infections occur, they will gradually die out as the infection cannot sustain itself in the population (Odeh et al, 2024). Proving global asymptotic stability ensures that, under current conditions or interventions, any outbreak will eventually diminish, leading the system back to the DFE. This concept is crucial for determining whether long-term control measures, such as quarantine, treatment, and vaccination, can permanently eliminate monkeypox from the population. If the DFE is globally asymptotically stable, we can be confident that the disease will not re-emerge.

To investigate the global stability of the disease free equilibrium, we use the technique implemented by Castillo-Chavez and song (2004).

To do this, we write the equation in the uninfected class as

$$\frac{dX}{dt} = F\left(X, Z\right)$$

And we re-write the equation in the infected class as

$$\frac{dZ}{dt} = G(X, Z)$$

Where $X = \left(S_h, R_h, S_r\right) \in R^3_+$ denotes the uninfected population and

$Z = \left(E_h, Q_h, I_h, T_h, E_r, I_r\right) \in R^6_+$ denotes the infected population

$\eta_0 = (X^*, 0)$ represent the disease free equilibrium of the system, and it is globally asymptotically stable if it satisfies the following conditions:

$H_1 : \frac{dX}{dt} = F\left(X^*, 0\right), X^*$ is globally asymptotically stable

$$H_2 : \frac{dZ}{dt} = D_Z G\left(X^*, 0\right) Z - \hat{G}\left(X, Z\right)$$

$\hat{G}\left(X, Z\right) \geq 0$ for all $\left(X, Z\right) \in D$ and where $D_Z G\left(X^*, 0\right)$ is an M- matrix (i.e the diagonal elements are non-negative and it is also the Jacobian of $\hat{G}\left(X, Z\right) \geq 0$ evaluated at $(X^*, 0)$.

If the system satisfies the above condition, then the theorem below holds.

Theorem 2

The equilibrium point $\eta_0 = (X^*, 0)$. *is globally asymptotically stable if* $R_0 \leq 1$, *conditions* H_1 *and* H_2 *are satisfied*

$$F(X,Z) = \begin{bmatrix} \Lambda_h + \sigma_h R_h + \alpha_h Q_h - \dfrac{(\beta_{rh} \mathrm{I}_r + \beta_{hh} \mathrm{I}_h)\mathrm{S}_h}{N_h} - \mu_h S_h, \\ \psi_h Q_h + \tau_h T_h - (\sigma_h + \mu_h)\mathrm{R}_h, \\ \Lambda_r - \dfrac{\beta_{rr} I_r S_r}{N_r} - \mu_r \mathrm{S}_r \end{bmatrix},$$

$$G(X,Z) = \begin{bmatrix} \dfrac{(\beta_{rh} I_r + \beta_{hh} I_h)S_h}{N_h} - (\omega_h + \theta_h + \mu_h)E_h \\ \omega_h E_h - (\rho_h + \delta_h + \mu_h)I_h \\ \theta_h E_h - \left(\gamma_h + \mu_h + \delta_h\right) I_h \\ \theta_h E_h - (\alpha_h + \psi_h + \mu_h)Q_h \\ \rho_h I_h - (\tau_h + \delta_h + \mu_h)T_h \\ \dfrac{\beta_{rr} I_r S_r}{N_r} - (\omega_r + \mu_r)E_r \\ \omega_r E_r - (\delta_r + \mu_r)I_r \end{bmatrix}$$

At disease free equilibrium,

H_1:

$$\frac{dS_h}{dt} = \Lambda_h - \mu_h S_h$$

$$\frac{dS_r}{dt} = \Lambda_r - \mu_r S_r$$

$$\frac{dR_h}{dt} = 0$$

H_2:

$$D_z G(X^*, 0)Z = \begin{bmatrix} (\beta_{rh} I_r + \beta_{hh} I_h) - (\omega_h + \theta_h + \mu_h)E_h \\ \omega_h E_h - (\rho_h + \delta_h + \mu_h)I_h \\ \theta_h E_h - \left(\gamma_h + \mu_h + \delta_h\right) I_h \\ \theta_h E_h - (\alpha_h + \psi_h + \mu_h)Q_h \\ \rho_h I_h - (\tau_h + \delta_h + \mu_h)T_h \\ \beta_{rr} I_r S_r - (\omega_r + \mu_r)E_r \\ \omega_r E_r - (\delta_r + \mu_r)I_r \end{bmatrix}$$

$$\hat{G}(X,Z) = D_Z G(X^*,0)Z - G(X,Z)$$

$$\hat{G}(X,Z) = \begin{bmatrix} (\beta_{rh}I_r + \beta_{hh}I_h)\left(1-\frac{S_h}{N_h}\right) \\ 0 \\ 0 \\ 0 \\ \beta_{rr}I_r\left(1-\frac{S_r}{N_r}\right) \\ 0 \end{bmatrix}$$

Clearly, $1 \geq \frac{S_h}{N_h}, 1 \geq \frac{S_h}{N_h}$ this implies that $\hat{G}(X,Z) \geq 0$.

Therefore the disease free equilibrium of the Monkeypox only model is globally asymptotically stable.

Endemic Equilibrium Point of the Monkeypox Model

The endemic equilibrium point in the monkeypox model represents a steady state where the disease persists in the population at a constant level, rather than being completely eradicated. At this point, the number of new infections balances with the number of recoveries or deaths, meaning the disease remains consistently present without growing into an outbreak or dying out (Watmough, 2002). This occurs when the basic reproduction number R_0 is greater than or equal to 1, indicating that each infected individual, on average, transmits the virus to at least one other person. In the endemic equilibrium, monkeypox continues circulating in the population, with some level of ongoing transmission despite interventions like quarantine or treatment. Understanding the endemic equilibrium helps public health authorities assess the long-term presence of the disease and develop strategies to reduce its prevalence or keep it at manageable levels, preventing large-scale outbreaks.

Theorem 3

The endemic equilibrium point of the Monkeypox model in (1) is stable if $R_0^h > 1, R_0^r > 1$ *and unstable if* $R_0^h < 1, R_0^r < 1$.

Proof

To obtain the endemic equilibrium we set the RHS of the differential equations in (1) to zero and solve for the state variables.

Thus, at the endemic equilibrium point,

$$\frac{dS_h}{dt} = \frac{dE_h}{dt} = \frac{dQ_h}{dt} = \frac{dI_h}{dt} = \frac{dT_h}{dt} = \frac{dR_h}{dt} = \frac{dS_r}{dt} = \frac{dE_r}{dt} = \frac{dI_r}{dt} = 0.$$

Let $\eta^{**} = \left(S_h^{**}, E_h^{**}, Q_h^{**}, I_h^{**}, T_h^{**}, R_h^{**}, S_r^{**}, E_r^{**}, I_r^{**}\right)$ be the endemic equilibrium point.

Let $\lambda_h = \frac{(\beta_{rh}I_r + \beta_{hh}I_h)}{N_h}$, $\lambda_r = \frac{\beta_{rr}I_r}{N_r}$ be the forces of infection of the monkeypox model.

We have that,

$$S_h^{**} = \frac{\Lambda_h P_1 P_2}{P_1 P_2(\lambda_h + \mu_h) - \omega_h \lambda_h \phi_h}$$

$$E_h^{**} = \frac{\Lambda_h P_2 \lambda_h^{**}}{P_2 P_1 \mu_h + P_1 P_2 \lambda_h^{**} - \omega_h \lambda_h^{**} \rho_h}$$

$$Q_h^{**} = \frac{\Lambda_h \lambda_h^{**} \rho_h}{P_2 P_1 \mu_h + P_1 P_2 \lambda_h^{**} - \omega_h \lambda_h^{**} \rho_h}$$

$$I_h^{**} = \frac{\theta_h \lambda_h^{**} P_2 \Lambda_h}{\left(P_2 P_1 \mu_h + P_1 P_2 \lambda_h^{**} - \omega_h \lambda_h^{**} \rho_h\right) P_3}$$

$$T_h^{**} = \frac{\Lambda_h \lambda_h^{**} \left(P_2 \gamma_h \theta_h + P_3 \alpha_h \phi_h\right)}{\left(P_2 P_1 \mu_h + P_1 P_2 \lambda_h^{**} - \omega_h \lambda_h^{**} \rho_h\right) P_3 P_4}$$

$$R_h^{**} = \frac{\Lambda_h \lambda_h^{**} \left(P_2 \gamma_h \theta_h + P_3 \alpha_h \phi_h\right) \varepsilon_h}{\left(P_2 P_1 \mu_h + P_1 P_2 \lambda_h^{**} - \omega_h \lambda_h^{**} \psi_h\right) P_3 P_4 \mu_h}$$

$$S_r^{**} = \frac{\Lambda_r}{\lambda_r + \mu_r},\ E_r^{**} = \frac{\Lambda_r \lambda_r}{\left(\lambda_r + \mu_r\right) P_5},\ I_r^{**} = \frac{\Lambda_r \lambda_r \theta_r}{\left(\lambda_r + \mu_r\right) P_5 P_6}.$$

Substituting them into the force of infection for the Monkeypox disease transmission in the rodents' population, $\lambda_r^{**} = \frac{\beta I_r^{**}}{N_r^{**}}$, we obtained the following:

$\lambda_r^{**} = \frac{\beta_r \theta_r - P_5 P_6}{P_6 + \theta_r}$. Substituting the value of λ_r^{**} into the force of infection for the Monkeypox disease transmission in the human population, $\lambda_h = \frac{(\beta_{rh} I_r + \beta_{hh} I_h)}{N_h}$. We obtained,

$$A\lambda_h^{**2} + B\lambda_h^{**} + C = 0 \qquad (2)$$

Where,

$$A = P_5 \Lambda_h \left(\left(\left(P_3 P_4 + \theta_h \left(\gamma_h + P_4\right)\right) P_2 + P_3 \omega_h \left(\theta_h + P_4\right)\right) \mu_h + \varepsilon_h \left(P_2 \gamma_h \theta_h + P_3 \theta_h \omega_h\right)\right)$$
$$\left(\left(-P_5 + \mu_r\right) P_6 + \theta_r \left(\beta_r + \mu_r\right)\right) P_6$$

$$B = -\begin{pmatrix} -P_6\left(\left(-P_1\left(\left(-\beta_{rr} - \mu_r\right)\Lambda_h + \beta_{rh}\Lambda_r\right) P_3 - \beta_b \theta_h \Lambda_h \left(\beta_{rr} + \mu_r\right)\right) P_2 + \phi_h \beta_{rr} \omega_h \Lambda_r P_3\right) P_5 \theta_r \\ \beta_{rh} \beta_r \Lambda_r P_3 \left(-P_1 P_2 + \omega_h \phi_h\right) \theta_r{}^2 \\ -\Lambda_h P_2 P_5 P_6{}^2 \left(P_5 - \mu_r\right)\left(-P_1 P_3 + \beta_{rh} \theta_h\right) \end{pmatrix} \mu_h P_4$$

$$C = -2\mu_h P_4 \begin{pmatrix} -P_6\left(\left(-P_1\left(\beta_{rh}\Lambda_r(-1+k) - \Lambda_h\left(\beta_r + \mu_r\right)\right) P_2 + \phi_h \beta_{hh} \omega_h \Lambda_r (-1+k)\right) P_3 - \beta_b \theta_h \Lambda_h P_2 \left(\beta_r + \mu_r\right)\right) P_5 \theta_r \\ \beta_a \beta_r \Lambda_r P_3 \left(-P_1 P_2 + \omega_h \phi_h\right) \theta_r{}^2 \\ +\Lambda_h P_2 P_5 P_6{}^2 \mu_r \left(-P_1 P_3 + \beta_{hh} \theta_h\right) \\ +\mu_h{}^2 P_1 P_2 P_3 P_4 k \Lambda_r \beta_{rh} \theta_r \left(1 - R_0^r\right) \\ +\mu_h P_2 P_4 P_5{}^2 P_6{}^2 \Lambda_h \left(1 - R_0^h\right) \end{pmatrix}$$

For λ_h^{**} to be positive, we have that

$\left(R_0^h - 1\right) > 0$ and $\left(R_0^r - 1\right) > 0$

$\Rightarrow R_0^h > 1$ and $R_0^r > 1$

Thus the endemic equilibrium point of the monkeypox model is said to be stable.

Sensitivity Analysis of the Monkeypox Model

Sensitivity analysis is carried out to determine the parameters that enhances the spread as well as control of an infection in a population.

The sensitivity index of the reproduction number of the Monkeypox model with respect to any parameter say p is given by:

$$\Im_p^{R_0^h} = \frac{\partial R_0^h}{\partial p} \times \frac{p}{R_0^h}, \quad \Im_p^{R_0^r} = \frac{\partial R_0^r}{\partial p} \times \frac{p}{R_0^r}$$

Given that

$$R_0^h = \frac{\beta_{hh}\omega_h}{P_1 P_2}, \quad R_0^h = \frac{\beta_{rr}\omega_r}{P_5 P_6}$$

$$\Im_{\beta_{hh}}^{R_0^h} = 1.0000, \quad \Im_{\omega_h}^{R_0^h} = \frac{\omega_h + \mu_h}{\omega_h + \theta_h + \mu_h} = 0.1832$$

$$\Im_{\delta_h}^{R_0^h} = -\frac{\delta_h}{\gamma_h + \mu_h + \delta_h} = -0.4962, \Im_{\mu_h}^{R_0^h} = -\frac{\left(\psi_h + 2\mu_h + \delta_h + \omega_h + \theta_h\right)\mu_h}{\left(\omega_h + \theta_h + \mu_h\right)\left(\omega_h + \mu_h + \delta_h\right)} = -0.0167$$

$$\Im_{\rho_h}^{R_0^h} = -\frac{\omega_h}{\rho_h + \mu_h + \delta_h} = -0.4962 \; \Im_{\beta_{rr}}^{R_0^r} = 1.0000, \; \Im_{\omega_r}^{R_0^r} = \frac{\mu_r}{\omega_r + \mu_r} = 0.0244,$$

$$\Im_{\mu_r}^{R_0^r} = -\frac{\left(\delta_r + 2\mu_r + \theta_r\right)\mu_r}{\left(\theta_r + \mu_r\right)\left(\delta_r + \mu_r\right)} = -0.0284, \Im_{\delta_r}^{R_0^r} = -\frac{\delta_r}{\delta_r + \mu_r} = -0.9960$$

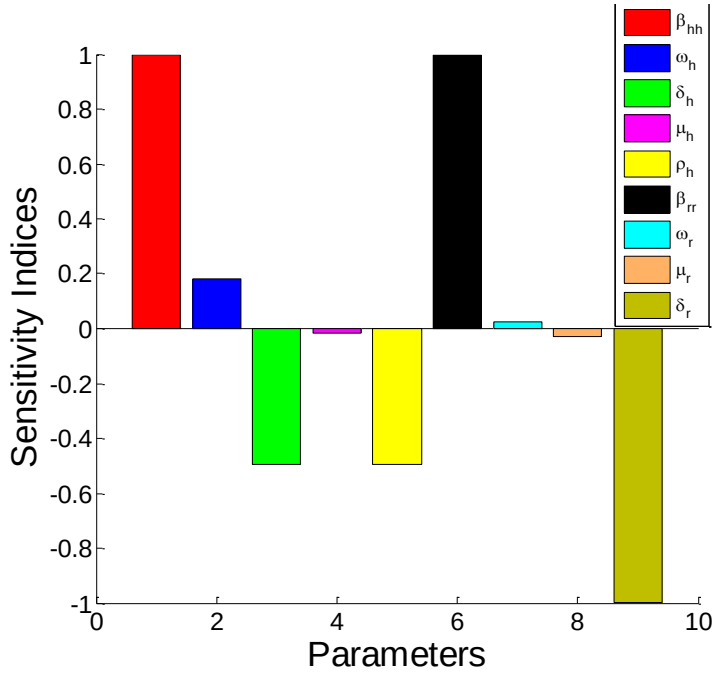


Figure 2. Bar chat of Monkeypox sensitivity Indices

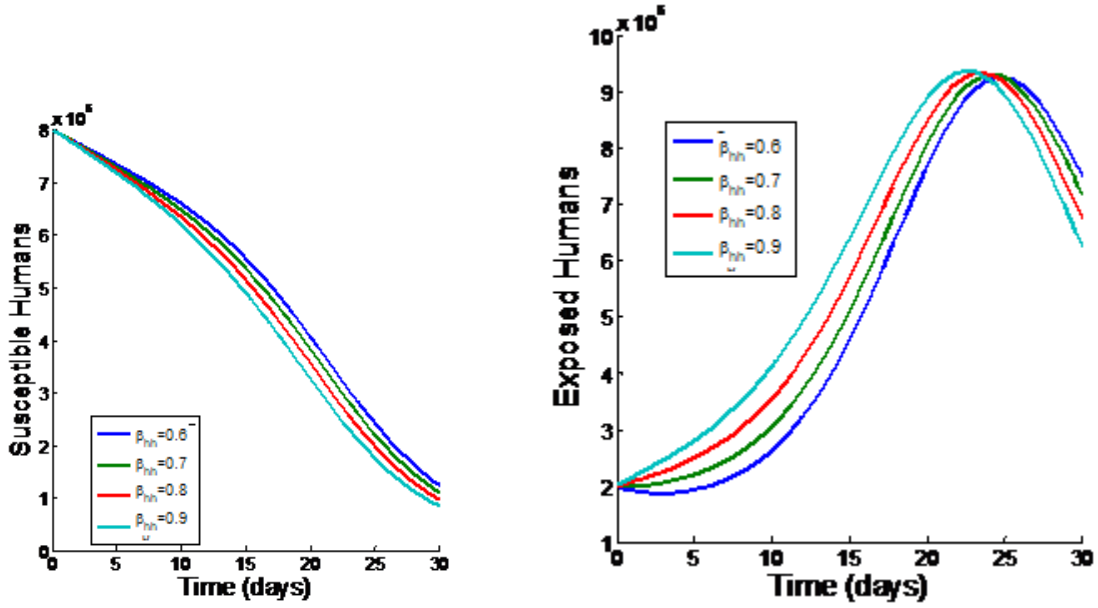
Interpretation of the Monkeypox Sensitivity Analysis

From the sensitivity analysis above, it is observed that the parameters like $\beta_{rh}, \beta_{rr}, \beta_{hh}, \omega_h, \omega_r$ with positive sensitivity indices enhances the spread of Monkeypox within the human

population, hence any effort taken to prevent contact with any of these parameters will reduce spread of the disease. Conversely, the parameters $\delta_h, \mu_h, \mu_r, \rho_h$ with negative sensitivity indices reduce the prevalence of Monkeypox within the human population. For instance, the negative sensitivity of treatment rate of infected humans (ρ_h) implies any effort taken to promote effective treatment would reduce transmission of monkeypox within the population.

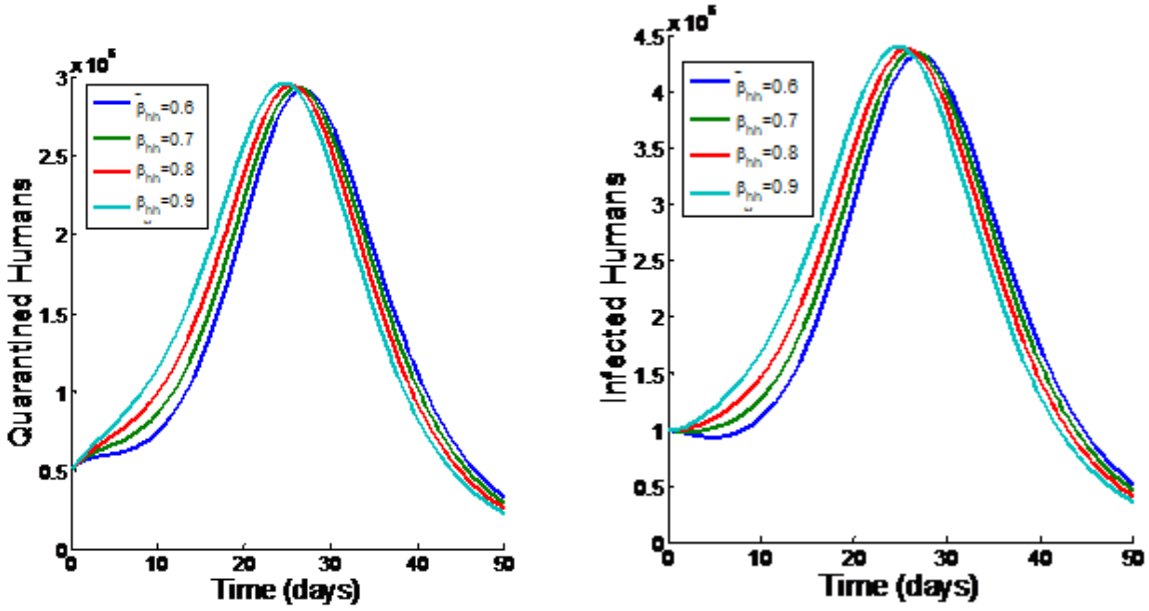
Table 2. **Parameter Values Used in the Model**

Parameters	Value	Sources
Λ_h	0.029	Assumed
Λ_r	0.9	Assumed
β_{rh}	0.00025	Odeh et al, 2024
β_{hh}	0.00006	Olumuyiwa et al, 2021
β_{rr}	0.027	Olumuyiwa et al, 2021
ω_h	0.07	Acheneje et al, 2024
ρ_h	0.002	Olumuyiwa et al, 2021
θ_h	0.2	Olumuyiwa et al, 2021
μ_r	0.00200	Assumed
τ_h	0.02	Olumuyiwa et al, 2021
α_h	0.0001	Agbata et al, 2019
σ_h	0.00001	Agbata, et al 2024
δ_h	0.02	Olumuyiwa et al, 2021
δ_r	0.5	Agbata et al, 2023
μ_h	0.020435	Bolaji et al, 2024
ω_r	0.007	Assumed



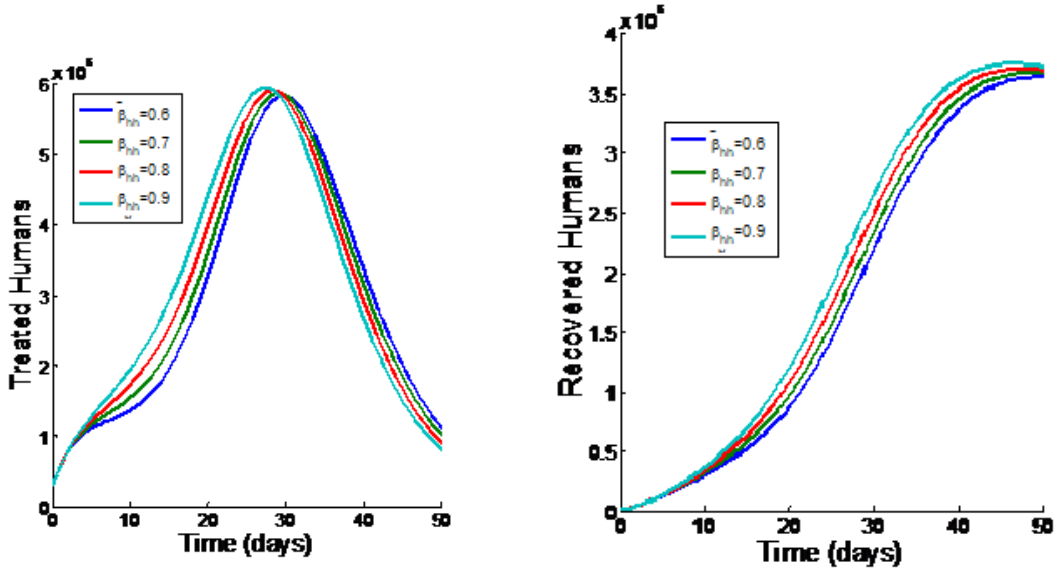
a. **Effect of varying β_{hh} on susceptibleHumans** b. **Effect of varying β_{hh} on Exposed humans**

Figure 3



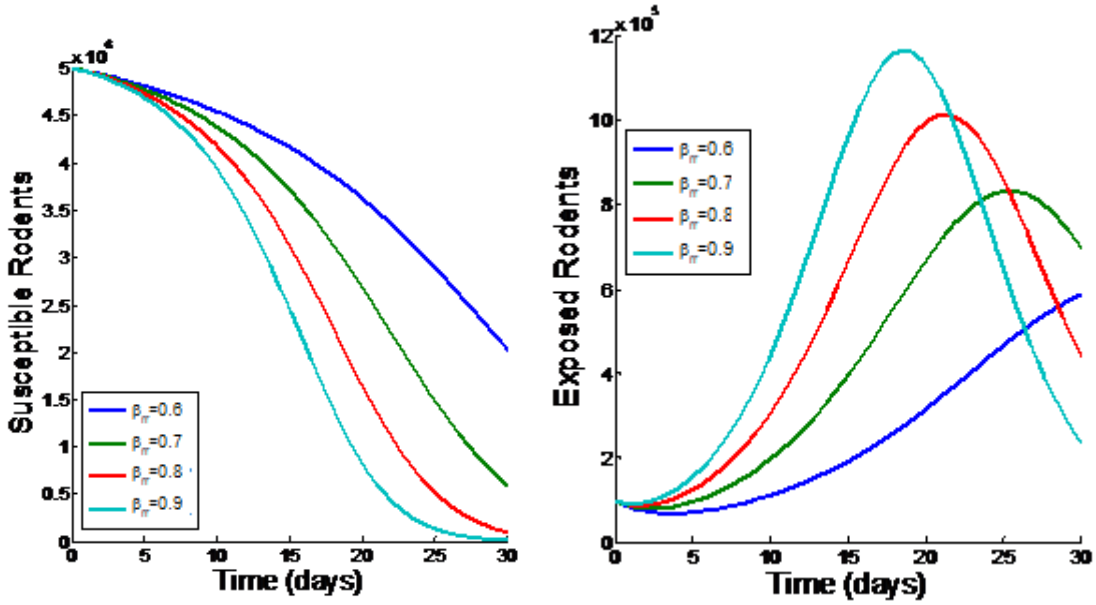
a. Effect of varying β_{hh} on Quarantined Humansb. Effect of varying β_{hh} on Infected humans

Figure 4



a. Effect of varying β_{hh} on Treated Humansb. Effect of varying β_{hh} on Recovered humans

Figure 5



a. Effect of varying β_{rr} on SusceptibleRodentsb. Effect of varying β_{rr} on Exposedhumans

Figure 6

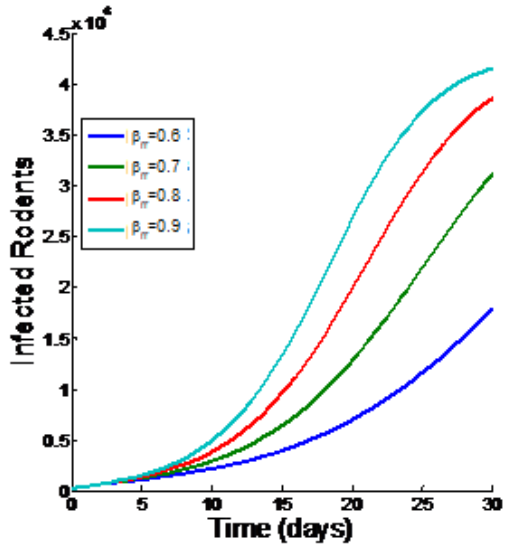


Figure 7: Effect of varying β_{rr} on infected Rodents

Discussion

Figures 3a and 3b illustrate the dynamics of susceptible and exposed humans, respectively. In figure 3a, the number of susceptible humans decreases as the contact rate between susceptible and infectious humans declines. This suggests that reducing contact between these two groups plays a crucial role in disease control. Figure 3b shows that the number of exposed humans initially increases but eventually begins to decline, indicating that as control measures are implemented and exposure is reduced, the spread of the disease is curbed over time. In figure 4a, we observe the effects of quarantining infected humans. The number of quarantined individuals increases initially, demonstrating effective isolation measures. However, as the number of infected humans decreases (as shown in Figure 4b), the need for

quarantine also diminishes. This is due to a reduction in the contact rate between susceptible and infectious individuals, which lowers transmission rates.

These results imply that reducing contact with infected humans significantly reduces the spread of Monkeypox within the population. Figure 5a highlights the number of treated individuals, which rises rapidly at first but later declines toward zero. This trend corresponds with the decrease in the number of infected humans seen in figure 4b, suggesting that as the infection subsides, fewer treatments are needed. Additionally, the high treatment rate depicted in figure 5a leads to a high recovery rate for infected individuals, as shown in figure 5b. Moving on to rodents, figures 6a and 6b present the dynamics of susceptible and exposed rodents, respectively. In figure 6a, the number of susceptible rodents declines to zero as contact between susceptible and infected rodents decreases. Meanwhile, figure 6b shows that the number of exposed rodents initially rises but eventually declines, indicating effective disease control as contact between these groups is minimized. Lastly, figure 7 displays the number of infected rodents. Initially, this number increases rapidly but later drops as the contact rate between susceptible and infected rodents decreases. The numerical simulation results confirm that reducing the contact rate between susceptible and infectious humans helps control the spread of Monkeypox within the population. Similarly, reducing contact between susceptible and infected rodents decreases the prevalence of the disease among rodents, further contributing to overall disease control.

Conclusion

Our study provides a clear understanding of how monkeypox spreads within and between human and rodent populations. The results show that reducing contact between infected and susceptible individuals—both human and rodent—is key to controlling the virus. Quarantine and effective treatment, particularly for infected humans, play an essential role in reducing transmission. The sensitivity analysis reveals that certain factors, like human-to-human contact and zoonotic transmission from rodents, drive the spread of the disease, making them critical targets for intervention. Meanwhile, promoting treatments that improve recovery can significantly reduce the number of infections. Overall, the findings highlight the need for a multi-faceted public health approach, one that not only reduces human contact with infected rodents but also strengthens medical interventions to stop the spread of monkeypox. This model offers a valuable framework for policymakers and health officials to manage resources and implement effective control measures during monkeypox outbreaks, particularly in regions where the disease is endemic.

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