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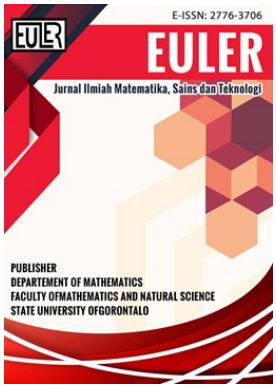

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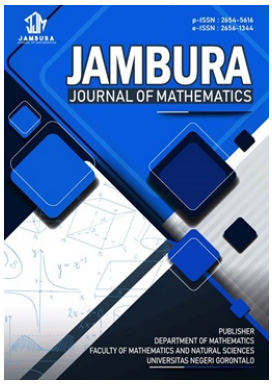

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Research Article

Analysis of Mpox Dynamic Model with Reinfection and Treatment

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ABSTRACT. *This research aims to develop a mathematical model of monkeypox disease spread with reinfection and hospitalization. This model divided the human population into five sub-populations: susceptible, exposed, infectious, hospitalized, and recovered. On the other hand, the animal population is divided into three: susceptible, exposed, and infectious. The results of the model analysis show that the stability of the two equilibrium points, disease-free and endemic, is asymptotically stable when $\mathcal{R}_0 < 1$ and $\mathcal{R}_0 > 1$, respectively. A sensitivity analysis was conducted on the parameter of the rate at which infected individuals are hospitalized. Based on numerical simulations, a disease-free state has been achieved when more than 68.87% of infected individuals receive hospital treatment. Hospital treatment has a positive impact on efforts to reduce the number of infected individuals in the population. The more individuals who are hospitalized, the greater the number of individuals who are exposed, hospitalized, and recover will increase.*

1. Introduction

Monkeypox (Mpox) is an infectious disease caused by the monkeypox virus. It causes a painful rash, enlarged lymph nodes, and fever. In May 2023, the monkeypox outbreak spread rapidly across Europe, the Americas, and six WHO regions, with 110 countries reporting approximately 87,000 cases and 112 deaths [1]. The proportion of patients who died varied between 0 and 11% and was higher in children. Mpox can spread from animal to human and from human to human through contact with infected individuals or animals with body fluids, skin lesions, or internal mucosal surfaces [2]. When someone gets infected with the monkeypox virus, they do not immediately show symptoms of infection. A person can only transmit the virus when symptoms of the disease appear [3]. The time between when someone gets infected and when they can spread the virus is called the incubation period, which for monkeypox is typically between 5 and 21 days.

A vaccine is one of the most effective measures to suppress the spread of the Mpox disease. The vaccinia virus-based smallpox vaccine has been proven effective in preventing Mpox disease [1]. Additionally, hospitalization can also be an alternative to reduce the number of Mpox cases. In [4], the global hospitalization rate was 14.1%, decreasing over time: 49.8% prior to 2017, 21.7% from 2017 to 2021, and 5.8% in 2022. The case fatality rate was estimated at 0.1% when hospital care was available. Vaccinated individuals or those who experienced smallpox were generally thought to produce a strong immunological response. However, Mpox reinfection is possible, with cases occurring quickly, highlighting the need for continued monitoring and prevention efforts [5].

There are many studies related to the spread of Mpox disease. One of them is using mathematical models. The mathematical model can accurately describe the spread of the disease in real-life scenarios. Mathematical models can be a valuable tool for analyzing the spread and control of infectious diseases through a set of equations. Modeling can help control contagious diseases by estimating epidemic scales, understanding transmission characteristics, evaluating the efficacy of interventions or policies, and forecasting disease outbreaks [6]. Peter et al. [7] formed a mathematical model of the spread of Mpox disease by dividing the human population into five compartments (susceptible, exposed, infected, isolated, and recovered) and the animal population into three (susceptible, exposed, and infected). In 2023, Kumar [8] conducted a case study of Mpox disease in the United States using a mathematical model with data. In 2024, Savinkina et al. [9] found that vaccinating children under 15 years old would be the most efficient use of vaccines. Ngungu et al. [10] researched the dynamics of monkeypox virus transmission with non-pharmaceutical interventions and simulated using data available in the UK. Liu et al. [11] studied the epidemiological aspects of the spread of the monkeypox virus using nonlinear differential equations. However, most existing models have not explicitly addressed reinfection and hospitalization as key factors influencing the disease dynamics.

Therefore, this study proposes a novel mathematical model that incorporates the effects of reinfection and hospitalization in the transmission dynamics of monkeypox. Unlike previous models that generally simplify the recovery process or exclude the possibility of individuals becoming reinfected, this model explicitly accounts for the epidemiological feedback loop resulting from reinfection, as well as the clinical progression leading to hospitalization. The inclusion of these two critical yet often neglected components reflects real-world complexities more ac-

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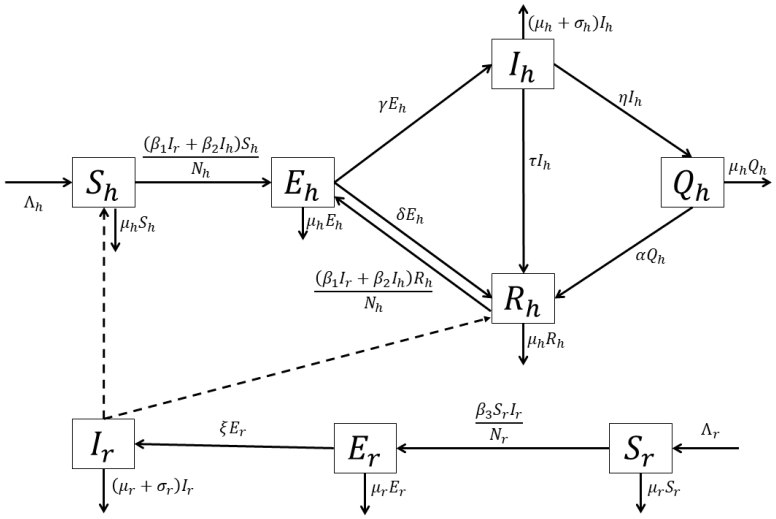

Figure 1. Diagram of Mpox transmission

curately, especially in the context of prolonged outbreaks and healthcare system burdens. Furthermore, by integrating these factors into a compartmental framework, the model enables more precise simulation of disease dynamics under various intervention scenarios, including hospital capacity and reinfection rates. This approach not only enriches the theoretical understanding of Mpox transmission but also offers practical implications for public health planning, especially in regions with limited vaccine coverage or delayed case detection. Ultimately, the insights derived from this model are expected to support more targeted, efficient, and adaptive control strategies in mitigating future monkeypox outbreaks.

2. Model

To develop the Mpox transmission model, the human population is divided into five sub-populations: susceptible (S_h), exposed (E_h), infectious (I_h), hospitalized (Q_h), and recovered (R_h) individuals. This model assumes a constant population. Additionally, the infection in animals is only transmitted by infectious animals. Newborns are considered susceptible individuals at a rate of Λ_h per unit of time. Individuals who have recovered from the Mpox have no permanent immunity. Thus, reinfection remains possible when these individuals come into contact with infectious individuals [12]. On the other hand, the animal population is divided into three sub-populations: susceptible (S_r), exposed (E_r), and infectious (I_r). Hence, the human and animal populations are given by $N_h = S_h + E_h + I_h + Q_h + R_h$ and $N_r = S_r + E_r + I_r$, respectively.

Infection occurs through direct contact between susceptible individuals and infected animals or humans, with transmission rates of β_1 and β_2, respectively. The susceptible humans who became infected will move to the exposed population. In this condition, individuals do not show symptoms of infection and are unable to spread the virus to others. Exposed individuals can recover without treatment if they have high immunity and move to the recovered population at a rate of δ. Conversely, individuals with low immunity will become infectious at a rate of γ. The description of the parameters used is presented in Table 1.

The number of infectious people declines due to a combination of factors, including hospital treatments, natural deaths, disease-related deaths, and recovery from illness without medical intervention. This underscores the effectiveness of these interventions in controlling the spread of Mpox. Based on that description, Figure 1 illustrates the transmission model of Mpox disease, which considers reinfection and hospitalization. From this transmission model, the mathematical model can be expressed in the system (1).

$$\begin{aligned}
\frac{dS_h}{dt} &= \Lambda_h - \frac{(\beta_1 I_r + \beta_2 I_h)S_h}{N_h} - \mu_h S_h \\
\frac{dE_h}{dt} &= \frac{(\beta_1 I_r + \beta_2 I_h)}{N_h}(S_h + R_h) - AE_h \\
\frac{dI_h}{dt} &= \gamma E_h - BI_h \\
\frac{dQ_h}{dt} &= \eta I_h - CQ_h \\
\frac{dR_h}{dt} &= \delta E_h + \tau I_h + \alpha Q_h - \frac{(\beta_1 I_r + \beta_2 I_h)R_h}{N_h} - \mu_h R_h \\
\frac{dS_r}{dt} &= \Lambda_r - \frac{\beta_3 S_r I_r}{N_r} - \mu_r S_r \\
\frac{dE_r}{dt} &= \frac{\beta_3 S_r I_r}{N_r} - DE_r \\
\frac{dI_r}{dt} &= \xi E_r - EI_r
\end{aligned} \tag{1}$$

with $A = \gamma + \delta + \mu_h$, $B = \eta + \tau + \mu_h + \sigma_h$, $C = \alpha + \mu_h$, $D = \xi + \mu_r$, and $E = \sigma_r + \mu_r$.

3. Results and Discussion

In this section, we analyze the model and perform a numerical simulation. Model analysis involves determining the equilibrium point and the basic reproduction number. Then, we analyze the stability of the disease-free equilibrium.

For all time $t \geq 0$, as long as the initial values are positive, all variables in model (1) are also positive. We define the possible region of the model (1) as

$$\mathcal{D} = \left\{ (S_h,\ E_h,\ I_h,\ Q_h,\ R_h,\ S_r,\ E_r,\ I_r) \in \mathbb{R}^8_+ ; N_h \leq \frac{\Lambda_h}{\mu_h} \right\}.$$

Table 1. Description of parameters for the model

Parameters	Description	Value (per day)	Sources
Λ_h	Human recruitment rate	$\frac{1000}{79\times365}$	[8]
Λ_r	Animal recruitment rate	$\frac{1000}{5\times365}$	[8]
β_1	Infection rate from animal to human	0.3045	[13]
β_2	Infection rate from human to human	0.747322	[8]
β_3	Infection rate from animal to animal	0.025	[13]
μ_h	The death rate of humans	$\frac{1}{79\times365}$	[8]
μ_r	The death rate of animals	$\frac{1}{5\times365}$	[8]
σ_h	The Mpox-induced death rate of humans	0.0011	[13]
σ_r	The Mpox-induced death rate of the animal	0.057	[13]
γ	The rate of progression from the exposed human to infectious	0.99	[13]
η	The rate of hospitalization of infectious human	Assumed	-
δ	The recovery rate of humans after exposure	0.01262	[14]
τ	The recovery rate of humans after being infected	0.048	[13]
α	The recovery rate of hospitalized infected humans	0.056	[13]
ξ	The rate of progression from the exposed animal to infectious	0.025289	[8]

Next, we show that model (1) is well-posed.

$$\frac{dN}{dt} = \Lambda_h - \mu_h N_h - \sigma_h I_h \leq \Lambda_h - \mu_h N_h.$$

If $N_h > \frac{\Lambda_h}{\mu_h}$, then we have $\frac{dN}{dt} < 0$. Since $\frac{dN}{dt} \leq \Lambda_h - \mu_h N_h$, we obtain $N_h(t) \leq N(0)\, e^{-\mu_h t} + \frac{\Lambda_h}{\mu_h}(1 - e^{-\mu_h t})$. Furthermore, we have $N_h(0) > \frac{\Lambda_h}{\mu_h}$ such that $N_h(t) \leq \frac{\Lambda_h}{\mu_h}$. Additionally, it can be seen that every solution in model (1) will remain in $\mathcal{D}$ for all $t > 0$. This implies that $\mathcal{D}$ is both positively and attracting. Consequently, the model (1) is mathematically well-posed.

3.1. Disease Free Equilibrium and the Basic Reproduction Number of the Model

Model (1) has a disease-free equilibrium given by

$$\begin{aligned} DFE &= (S_h^*,\ E_h^*,\ I_h^*,\ Q_h^*,\ R_h^*,\ S_r^*,\ E_r^*,\ I_r^*) \\ &= \left(\frac{\Lambda_h}{\mu_h},\ 0,\ 0,\ 0,\ 0,\ \frac{\Lambda_r}{\mu_r},\ 0,\ 0\right). \end{aligned} \tag{2}$$

This study defines the basic reproduction number $(\mathcal{R}_0)$ as the estimated number of newly infected cases resulting from an initial infection in a closed population during one infection period [15]. We construct the basic reproduction number for system (1) using the next-generation matrix (NGM), denoted by K [16]. System (1) has five infected states, E_h, I_h, Q_h, E_r, and I_r; and three uninfected states, S_h, R_h, and S_a. Regarding the linearized infection subsystem in the system (1), we obtain the transmission matrix T and transition matrix Σ is evaluated in the disease-free equilibrium.

$$T = \begin{bmatrix} 0 & \frac{(S_h+R_h)\beta_2}{N_h} & 0 & 0 & \frac{(S_h+R_h)\beta_1}{N_h} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \frac{\beta_3 S_r}{N_r} \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix},$$

and

$$\Sigma = \begin{bmatrix} -A & 0 & 0 & 0 & 0 \\ \gamma & -B & 0 & 0 & 0 \\ 0 & \eta & -C & 0 & 0 \\ 0 & 0 & 0 & -D & 0 \\ 0 & 0 & 0 & \xi & -E \end{bmatrix}.$$

T has three rows consisting entirely of zeros, then the NGM is calculated as $K = -\Omega' T \Sigma^{-1} \Omega$ [16]. The number of rows in matrix Ω is equal to that of matrix T. For every non-zero row of T, there is a single column of Ω. That column of Ω has a one in the row that corresponds to the non-zero row of T, and zeros elsewhere. We derive the matrix Ω as

$$\Omega = \begin{bmatrix} 1 & 0 \\ 0 & 0 \\ 0 & 0 \\ 0 & 1 \\ 0 & 0 \end{bmatrix}.$$

Hence, the NGM of the system (1) is given by

$$\begin{aligned} K &= -\Omega' T \Sigma^{-1} \Omega \\ &= \begin{bmatrix} \frac{\Lambda_h \beta_2 \gamma}{\mu_h N_h AB} & \frac{\Lambda_h \beta_1 \xi}{\mu_h N_h DE} \\ 0 & \frac{\Lambda_r \beta_1 \xi}{\mu_h N_h DE} \end{bmatrix}. \end{aligned}$$

The basic reproduction number is defined as the largest eigenvalue of the NGM, $\mathcal{R}_0 = \rho(K)$. Based on [16], for a 2×2 matrix, $\mathcal{R}_0$ can be calculated from the trace and determinant of the matrix K as,

$$\begin{aligned} \mathcal{R}_0 &= \rho(K) \\ &= \frac{1}{2}(\text{trace}\,(NGM) + \sqrt{\text{trace}(NGM)^2 - 4\text{det}(NGM)} \\ &= \frac{\beta_2 \Lambda_h \gamma}{\mu_h N_h AB} \\ &= \frac{\beta_2 \Lambda_h \gamma}{\mu_h N_h (\eta + \tau + \mu_h + \sigma_h)(\gamma + \delta + \mu_h)}. \end{aligned} \tag{3}$$

Using the parameter values in Table 1, $N_h = 1000$, and $\eta = 0.75$, the bar chart for the sensitivity indices of $\mathcal{R}_0$ is obtained in Figure 2.

From Figure 2, it can be seen that the parameters β_2, Λ_h, μ_h, N_h, and η have a significant influence on the changes in $\mathcal{R}_0$. Parameters β_2 and Λ_h have positive results, indicating that the larger the value of these parameters, the higher the value $\mathcal{R}_0$ will be. This means that the higher the infection

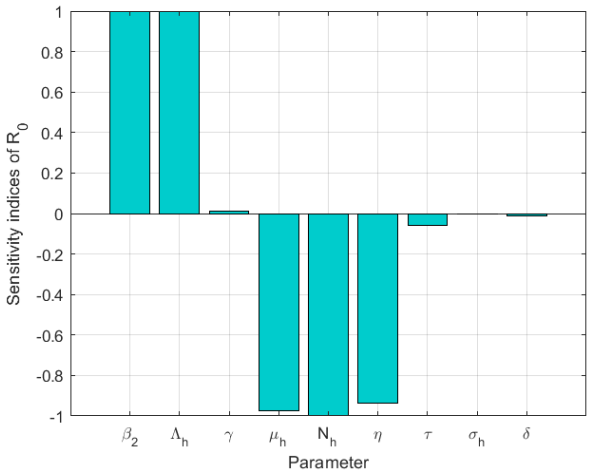

Figure 2. Sensitivity indices of the parameters to $\mathcal{R}_0$

rate from human to human (β_2) and human recruitment rate (Λ_h) the more the disease will spread. Conversely, the parameters μ_h, N_h, and η are inversely related to the value of $\mathcal{R}_0$. The larger the value of these parameters, the smaller the value of $\mathcal{R}_0$ will be. The death rate of humans (μ_h), total human population (N_h), and the rate of hospitalization of infectious human (η) that increase will result in a decrease in the spread of the disease. Furthermore, the sensitivity graph of the parameter η against $\mathcal{R}_0$ is obtained in Figure 3.

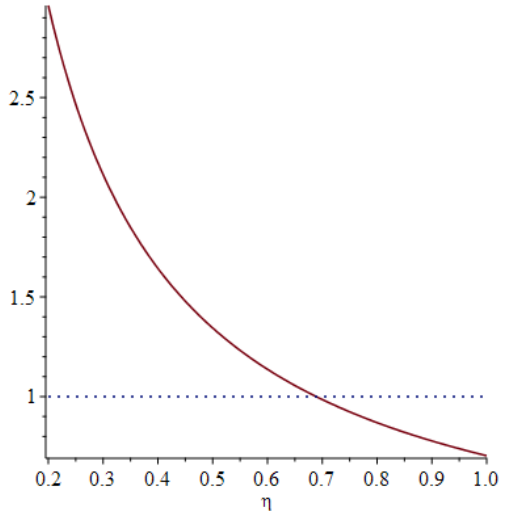

Figure 3. Sensitivity of the parameter η to $\mathcal{R}_0$

From Figure 3, it can be seen that as the value of η increases, the resulting value of $\mathcal{R}_0$ becomes smaller. It shows that the more hospital treatments conducted, the closer the situation will be to disease-free. A disease-free state will be achieved when more than 68.87% of infected individuals receive hospital treatment.

3.2. *Endemic Equilibrium of the Model*

Endemic equilibrium is the equilibrium where the number of infectious populations is non-zero. This equilibrium is given by

$$EE = (S_h^{**},\ E_h^{**},\ I_h^{**},\ Q_h^{**},\ R_h^{**},\ S_r^{**},\ E_r^{**},\ I_r^{**}),$$

where,

$$\begin{aligned}
S_h^{**} &= \frac{\Lambda_h N_h}{\beta_2 I_h + \beta_1 I_r + N_h \mu_h},\\
E_h^{**} &= \frac{B I_h}{\gamma},\\
Q_h^{**} &= \frac{\eta I_h}{C},\\
R_h^{**} &= \frac{I_h N_h \left(BC\delta + C\gamma\tau + \alpha\eta\gamma\right)}{C\gamma\left(\beta_2 I_h + \beta_1 I_r + N_h\mu_h\right)},\\
S_r^{**} &= \frac{\Lambda_r N_r}{\beta_3 I_r + N_r \mu_r},\\
E_r^{**} &= \frac{\Lambda_r \beta_3 I_r}{\left(I_r\beta_3 + N_r\mu_r\right) D}.
\end{aligned}$$

I_h^{**} and I_r^{**} are taken from the positive solution of the following equations:

$$\begin{aligned}
P_1\left(I_h^{**},\ I_r^{**}\right) &= DE\beta_3\ I_r^2 + \left(DEN_r\mu_r - \xi\Lambda_r\beta_3\right) I_r = 0,\\
P_2\left(I_h^{**},\ I_r^{**}\right) &= \left(ABC\beta_2 - BC\delta\beta_2 - C\gamma\tau\beta_2 - \alpha\eta\gamma\beta_2\right) I_h^2\\
&\quad + \left(ABCI_r\beta_1 + ABCN_h\mu_h - BC\delta I_r\beta_1\right.\\
&\quad \left. -C\gamma\tau I_r\beta_1 - \alpha\eta\gamma I_r\beta_1 - C\gamma\Lambda_h\beta_2\right) I_h\\
&\quad - C\gamma I_r\Lambda_h\beta_1 = 0.
\end{aligned}$$

Theorem 1. *System (1) always has the Mpox endemic equilibrium point whenever* $\mathcal{R}_0 > 1$.

Proof. From $P_1\left(I_h^{**},\ I_r^{**}\right)$ and $P_2\left(I_h^{**},\ I_r^{**}\right)$, we obtain a positive solution $I_r^{**} = 0$ and

$$I_h^{**} = \frac{\left(\alpha+\mu_h\right)\left(-\mu_h N_h(\eta+\tau+\mu_h+\sigma_h)(\gamma+\delta+\mu_h) + \Lambda_h\gamma\beta_2\ \right)}{i_h^{**}},$$

where

$$\begin{aligned}
i_h^{**} &= \mu_h^3\ +\ \left(\eta+\tau+\alpha+\gamma+\sigma_h\right)\mu_h^2\\
&\quad + \left(\left(\eta+\tau+\gamma+\sigma_h\right)\alpha + \gamma\left(\eta+\sigma_h\right)\right)\mu_h + \alpha\gamma\sigma_h)\ \beta_2.
\end{aligned}$$

When $\mathcal{R}_0 > 1$, we have $\beta_2\Lambda_h\gamma > \mu_h N_h(\eta+\tau+\mu_h+\sigma_h)(\gamma+\delta+\mu_h)$. This shows that I_h^{**}exist and $I_h^{**} > 0 \Leftrightarrow \mathcal{R}_0 > 1$. □

3.3. *Local Stability of Disease and Endemic Equilibrium*

The equilibrium point is locally stable if the real part of all its eigenvalues is negative. Linearized system (1) with Jacobian matrix J:

$$J = \begin{bmatrix}
j_{11} & 0 & 0 & j_{14} & 0 & 0 & 0 & j_{18}\\
j_{21} & -A & 0 & j_{24} & j_{25} & 0 & 0 & j_{28}\\
0 & \gamma & 0 & -B & 0 & 0 & 0 & 0\\
0 & 0 & -C & \eta & 0 & 0 & 0 & 0\\
0 & \delta & \alpha & j_{54} & j_{55} & 0 & 0 & j_{58}\\
0 & 0 & 0 & 0 & 0 & j_{66} & 0 & j_{68}\\
0 & 0 & 0 & 0 & 0 & j_{76} & -D & j_{78}\\
0 & 0 & 0 & 0 & 0 & 0 & \xi & -E
\end{bmatrix},$$

where

$$j_{11} = -\frac{I_h\beta_2 + \beta_!I_r}{N_h} - \mu_h, \quad j_{14} = -\frac{\beta_2 S_h}{N_h},$$
$$j_{18} = -\frac{\beta_1 S_h}{N_h}, \quad j_{21} = \frac{I_h\beta_2 + \beta_!I_r}{N_h},$$
$$j_{24} = \frac{\beta_2 S_h}{N_h} + \frac{\beta_2 R_h}{N_h}, \quad j_{25} = \frac{I_h\beta_2 + \beta_!I_r}{N_h},$$
$$j_{28} = \frac{\beta_1 S_h}{N_h} + \frac{\beta_1 R_h}{N_h}, \quad j_{54} = \tau - \frac{\beta_2 R_h}{N_h},$$
$$j_{55} = -\frac{I_h\beta_2 + \beta_!I_r}{N_h} - \mu_h, \quad j_{58} = -\frac{\beta_1 R_h}{N_h},$$
$$j_{66} = -\frac{\beta_3 I_r}{N_r} - \mu_r, \quad j_{68} = -\frac{\beta_3 S_r}{N_r},$$
$$j_{76} = \frac{\beta_3 I_r}{N_r}, \quad j_{78} = \frac{\beta_3 S_r}{N_r}.$$

Theorem 2. *The disease-free equilibrium DFE of the Mpox transmission model in the system (1) is locally asymptotically stable if* $\mathcal{R}_0 < 1$ *and unstable if* $\mathcal{R}_0 > 1$.

Proof. Let disease-free equilibrium DFE exist, which corresponds to $\mathcal{R}_0 < 1$. Substituting DFE into the Jacobian matrix J yields:

$$J(DFE) = \begin{bmatrix} -\mu_h & 0 & 0 & j_{14} & 0 & 0 & 0 & j_{18} \\ 0 & -A & 0 & j_{24} & 0 & 0 & 0 & j_{28} \\ 0 & \gamma & 0 & -B & 0 & 0 & 0 & 0 \\ 0 & 0 & -C & \eta & 0 & 0 & 0 & 0 \\ 0 & \delta & \alpha & \tau & -\mu_h & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -\mu_r & 0 & j_{68} \\ 0 & 0 & 0 & 0 & 0 & 0 & -D & j_{78} \\ 0 & 0 & 0 & 0 & 0 & 0 & \xi & -E \end{bmatrix},$$

where

$$j_{14} = -\frac{\beta_2 S_h}{N_h}, \quad j_{18} = -\frac{\beta_1 S_h}{N_h},$$
$$j_{24} = \frac{\beta_2}{N_h} S_h, \quad j_{28} = \frac{\beta_1}{N_h} S_h,$$
$$j_{68} = -\frac{\beta_3 S_r}{N_r}, \quad j_{78} = \frac{\beta_3 S_r}{N_r}.$$

The eigenvalues of the Jacobian matrix $J\,(DFE)$ are:

$$\lambda_1 = -\mu_h < 0,$$
$$\lambda_2 = -\mu_h < 0,$$
$$\lambda_3 = -\mu_r < 0,$$
$$\lambda_4 = \frac{-(D+E)\,\mu_r N_r + \sqrt{\mu_r N_r\left((D-E)^2\mu_r N_r + 4\xi\Lambda_r\beta_3\right)}}{2\mu_r N_r} < 0,$$
$$\lambda_5 = \frac{-(D+E)\,\mu_r N_r - \sqrt{\mu_r N_r\left((D-E)^2\mu_r N_r + 4\xi\Lambda_r\beta_3\right)}}{2\mu_r N_r} < 0,$$

while λ_6, λ_7, and λ_8 are taken from the root of the cubic equation below:

$$\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0, \tag{4}$$

where

$$a_1 = A - \eta > 0,$$
$$a_2 = -A\eta - BC,$$
$$a_3 = \frac{C\,(ABN_h\mu_h - \gamma\Lambda_h\beta_2)}{N_h\mu_h}.$$

Based on Routh-Hurwitz criteria in [17], all eigenvalues in eq. (4) are negative if $a_1 > 0$, $a_3 > 0$, and $a_1a_2 > a_3$. Since $\mathcal{R}_0 < 1$, then $\mu_h N_h AB - \beta_2\Lambda_h\gamma > 0$. It was shown that $a_3 > 0$. To proof $a_1a_2 > a_3$, we must show that $\frac{a_3}{a_1a_2} < 1$. Since $\mathcal{R}_0 < 1$, we obtained

$$\frac{C\,(ABN_h\mu_h - \gamma\Lambda_h\beta_2)}{-(A-\eta)\,(A\eta + BC)\,N_h\mu_h} < 1.$$

It was proven that $a_1 > 0$, $a_3 > 0$, and $a_1a_2 > a_3$. So, we can conclude that the model is locally stable at the disease-free equilibrium. □

Numerically, the stability of the disease-free equilibrium point is analyzed using the parameter values in Table 1 and selecting $\eta = 0.8$ such that $\mathcal{R}_0 = 0.869 > 1$. We obtained that all eigenvalues of the Jacobian matrix are negative: -0.000035, -0.000035, -0.056035, -0.000548, -1.789459, -0.062333, -0.011967, and -0.071418.

The local stability of the endemic equilibrium point is evaluated numerically by using $\eta = 0.6$ such that $\mathcal{R}_0 = 1.137 > 1$. We obtained the endemic equilibrium point $EE = (S_h^{**}, E_h^{**}, I_h^{**}, Q_h^{**}, R_h^{**}, S_r^{**}, E_r^{**}, I_r^{**}) = (16.723,\ 1.789, 2.728,\ 29.216,\ 862.998,\ 500,\ 0,\ 0)$. Substitute the point EE into the Jacobian matrix, so that we have eigenvalues $\lambda_1 = -0.000548$, $\lambda_2 = -1.652269$, $\lambda_3 = -0.028754 + 0.003972I$, $\lambda_4 = -0.028754 - 0.003972I$, $\lambda_5 = -0.000124$, $\lambda_6 = -0.002074$, $\lambda_7 = -0.011967$, $\lambda_8 = -0.071418$. We found that all the real parts of the eigenvalues are negative. Therefore, system (1) is locally stable at both the disease-free and endemic states.

3.4. Numerical Simulation

In this section, we discussed the numerical simulation of the Mpox transmission model in the system (1). In this simulation, the total initial population used is $N_h = 1000$, $N_r = 500$, $S_h = 700$, $E_h = 100$, $I_h = 200$, $Q_h = 0$, $R_h = 0$, $S_r = 300$, $E_r = 100$, and $I_r = 100$. In the autonomous system, we analyze the sensitivity of hospital treatment rates using several strategies, $\eta = 0$, $\eta = 0.25$, $\eta = 0.75$, and $\eta = 1$. This means that the total number of infectious individuals receiving treatment in hospitals is 0%, 25%, 75%, and 100%. Using these strategies, the number of susceptible, exposed, infectious, hospitalized, and recovered individuals in the human population is presented in Figure 4.

Figure 4b, Figure 4d, and Figure 4e show that the number of infectious individuals receiving treatment in the hospital is directly proportional to the population of exposed, hospitalized,

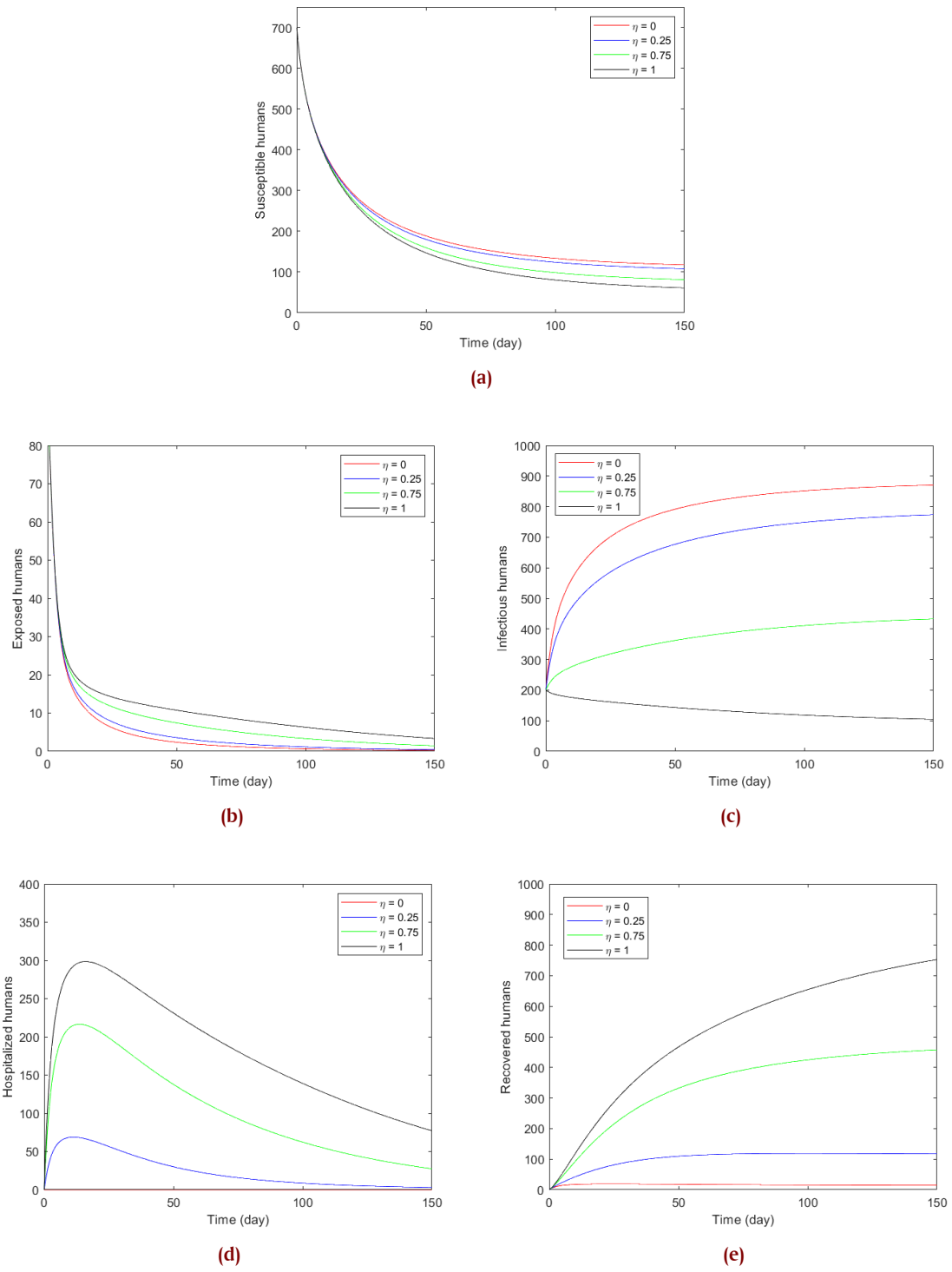

Figure 4. The number of (a) susceptible (S_h), (b) exposed (E_h), (c) infectious (I_h), (d) hospitalized (Q_h), and (e) recovered (R_h) population with different strategies

and recovered individuals. The more individuals are treated, the larger the total population of exposed, hospitalized, and recovered individuals will be. Conversely, it is inversely proportional to the population of susceptible and infectious individuals. From Figure Figure 4a, it can be seen that although the total population decreases as the number of treated infectious individuals increases, this does not result in a significant difference. Therefore, the rate of hospitalization of infectious humans does not have a significant impact on changes in the susceptible population. Figure 4c shows that the contagious population increases further when treatment is given to 25% and 75% of the contagious individuals. The number of contagious individuals decreases when the entire infectious population is treated.

4. Conclusion

Based on the discussion, the Mpox dynamic model with reinfection and treatment has a disease-free equilibrium $DFE = \left(\frac{\Lambda_h}{\mu_h}, 0, 0, 0, 0, \frac{\Lambda_r}{\mu_r}, 0, 0\right)$ and always has the endemic equilibrium point whenever $\mathcal{R}_0 > 1$. Using this disease-free equilibrium, we obtained the basic reproduction number $\mathcal{R}_0 = \frac{\beta_2 \Lambda_h \gamma}{\mu_h N_h (\eta+\tau+\mu_h+\sigma_h)(\gamma+\delta+\mu_h)}$. The results of the model analysis show that the stability of the two equilibrium points, disease-free and endemic, is asymptotically stable when $\mathcal{R}_0 < 1$ and $\mathcal{R}_0 > 1$, respectively. Hospital treatment has a positive impact on efforts to reduce the number of infected individuals. The more hospital treatments conducted, the closer the situation will be to disease-free. A disease-free state will be achieved when more than 68.87% of infected individuals receive hospital treatment. The number of individuals receiving treatment is directly proportional to the number of individuals exposed, hospitalized, and recovered. Conversely, the more individuals who receive treatment, the fewer the susceptible and infectious population will be.

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