An Investigation on the Monkeypox Virus Dynamics in Human and Rodent Populations for a Deterministic Mathematical Model

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Research Article

Keywords: Monkeypox, Vaccination, Quarantine, Mathematical modelling, Reproduction number, Sensitivity analysis

Posted Date: August 24th, 2023

DOI: https://doi.org/10.21203/rs.3.rs-1972401/v3

Additional Declarations: No competing interests reported.

Version of Record: A version of this preprint was published at Informatics in Medicine Unlocked on August 25th, 2023. See the published version at https://doi.org/10.1016/j.imu.2023.101325.

An Investigation on the Monkeypox Virus Dynamics in Human and Rodent Populations for a Deterministic Mathematical Model

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[1,]*

Abstract

A mathematical deterministic model for the dynamics of Monkeypox disease is developed. Monkeypox is a viral zoonotic disease that can be transmitted to humans, through contact with infected rodents. The model captures both the human and rodent populations and incorporates control strategies such as vaccines and quarantine for the human population. The model is analysed for local and global stability of the equilibrium solutions. In addition, numerical simulations of the model equations and sensitivity analysis of the parameters are carried out. The solutions obtained show that an increase in vaccination and quarantine measures could reduce the number of reproductions and ultimately eradicate the virus.

Keywords: `Monkeypox; Vaccination, Quarantine; Mathematical modelling; Reproduction number; Sensitivity analysis`

1. Introduction

Monkeypox is a viral disease that affects rodents (e.g. rats) and is transmitted to humans through contact with the infected rodents. Monkeypox was

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first discovered in 1958 when two outbreaks of a pox-like disease occurred in colonies of monkeys kept for research; hence the name 'monkeypox'. The first human case of monkeypox was recorded in 1970 in the Democratic Republic of Congo, during a period of intensified efforts to eliminate smallpox. Since then, monkeypox has been reported in humans in other central and western African countries [1].

The most recent outbreak of the monkeypox virus was in May 2022, and the first case was confirmed on May 6th, 2022, by an individual with a travel history to Nigeria. According to the Centres for Disease Control and Prevention (CDC), there were 87,314 confirmed infected cases by February 2023, in more than 112 countries, since the outbreak. The outbreak is ongoing, and affected countries include the United Kingdom and some countries in Europe, North and South America, Asia, Australia and Africa. Due to the unexpected and large geographical spread of the disease, it is likely that the actual number of cases will be underestimated [2, 3]. This is in line with the Global Health Report which reported 98% of the cases confirmed were male of an average age of 41.

On 23 July 2022, the Director General of the World Health Organisation (WHO) declared the outbreak of the disease and called upon the public health under emergency [4, 3]. According to WHO[4] monkeypox virus is a type of orthopox virus in the same family of viruses that causes smallpox. The symptoms of monkeypox are similar, though less severe and less contagious, than those of smallpox.

In a study done by Mackenzie [5], the processes of zoonotic viral emergence, the intricacies of host virus interactions, and the distinct role of biological transitions and modifying factors are well documented. Additionally, the study showed that the process of emergence is conceptualised as having two transition stages typical of disease emergence: contact of humans with the infectious agent and cross-species transmission of the agent.

The report on the response to the monkeypox outbreak of the Nigeria Centre for Disease Control [6] highlighted that the primary route of transmission of the disease to humans was contact with infected animals or their bodily fluids or

the eating of inadequately cooked meat from infected animals.

In the book titled *"Learning from Experience: The Public Health Response to West Nile Virus, SARS, monkeypox, and Hepatitis A Outbreaks in the United States"* which was written by Stoto[7], there is a need to prepare resources in the health sector to accommodate people infected with emerging outbreaks such as the monkeypox virus. Additionally, there is a need to understand the dynamics and take precautionary measures well before the disease becomes out of control.

There is no licenced vaccine available specifically for the monkeypox virus. However, some experimental vaccines have been tested on animals and humans [8]. One example is the modified Vaccinia Ankara (MVA) vaccine, which has been shown to be effective in preventing monkeypox in non-human primates. A phase 1 clinical trial of the MVA vaccine was also conducted in humans and found to be safe and well-tolerated [9].

Presently, there are no clear treatments available for monkeypox infection, though vaccination against smallpox has been shown to be successful by 85 percent in the prevention of monkeypox. Therefore, vaccination remains the best way to reduce the incidence and spread of the virus, as shown in the studies by Shaban & Mofi[10] and Rwezaura [11] in other virally spread diseases. A study by Martcheva[12] has shown that in order to typically contain the disease, quarantine must be made compulsory. Quarantine is applied to any individual who has come into contact with an infectious individual. Infectious diseases are currently posing a huge challenge not only to individuals but also to the global community, demanding the development of mathematical modelling approaches to improve our understanding of their dynamics. This is particularly relevant in the context of diseases such as Monkeypox.

Recent literature has made valuable contributions to this domain, with notable work by Naik et al. [13] on the approximate solution of nonlinear fractional-order HIV models using the homotopy analysis method, Naik et al. [14] on the complex dynamics of a seasonally forced discrete-time SIR epidemic model, Farman et al. [15] on the numerical treatment of a nonlinear dynamical hepatitis B model using an evolutionary approach, and Ahmad et al. [16] on

the modelling and numerical investigation of fractional-order bovine babesiosis disease. These works have provided valuable insights into the application of mathematical modelling to understanding and combating infectious diseases.

In recent years, several studies have been conducted, including the work of Peter et al.[17]; Usman & Isa Adamu[18]; Lasisi et al [19]; Emeka et al.[20]; Al-Shomrani et al[21]; Marwa et al [22] and Stoto et al.[7], among others. However, more studies are needed to understand the dynamics and control of monkeypox.

According to the WHO [4], the monkeypox virus remains a significant threat to the world [23]. Therefore, the current study makes a significant contribution to mitigating the spread of the virus as it investigates the dynamics of the deterministic model of the monkeypox virus with quarantine and vaccination interventions, which have not been extensively explored before.

2. Material and Methods

2.1. Mathematical Model Formulation

A mathematical model is formulated with two sub-populations, namely, the human sub-population denoted by N_H and the rodent (the non-human primates) sub-population denoted by N_R.

The model assumptions are as follows:

- The human sub-population has six compartments, that is, the susceptible humans, S_H , exposed E_H, the quarantined, Q_H, infectious I_H, recovered R_H, and vaccinated individuals V_H. The rodent subpopulation has three compartments, namely susceptible S_R, exposed rodents E_R, and infectious rodents R_H.

- Immigrants contribute to an increase in susceptible humans S_H at a rate of π_H. This will later be taken as $\pi_H = 2.7$ per every 1000 human population as shown by [24] or as by the International Migration Report of 2020 (see more details in the link here `https://www.un.org/en/desa/international-migration-report-2020-highlights`).

SN	Variable	Description
1	S_H	Susceptible human population
2	S_R	Susceptible rodent population
3	E_H	Number of exposed human individuals
4	E_R	Number of exposed rodent
5	I_H	Number of infectious human
6	I_R	Number of infectious rodent
7	R_H	Number of recovered human beings
8	V_H	Number of vaccinated human beings
9	Q_H	Number of quarantined human beings

Table 1: Variable descriptions

- Only susceptible and recovered individuals can be vaccinated, at rates of ϑ_1 and ϑ_2, respectively. The vaccine used is that for smallpox, which is not efficient 100% for the monkeypox virus [25]. Thus, vaccinated individuals return to the susceptible class at a rate ω.

The contact rate between rodents and susceptible humans is indicated by ξ_1, while that between humans and humans is indicated by ξ_2 and the proportion of undetected cases from the quarantined population after diagnosis is ψ. A proportion (p) of immigrants are vaccinated, while the rest $1-p$ are not and therefore belong to the susceptible class.

From the exposed class, some individuals transition to either the infected class at the rate σ_1 or the quarantine class at the rate σ_2. Likewise, the progression from quarantined class to infected class is ζ and while the recovery rate from infection is λ. Humans die naturally at rate μ_H or due to monkeypox at rate δ_H. The recruitment rate for the rodent sub-population is denoted as π_R, while the rodent-to-rodent contact rate is ξ_3. The progression rate of rodents from exposed to infectious is denoted as σ_3. Rodents die naturally at the rate μ_R or due to disease at the rate of δ_R.

2.2. Schematic Diagram

The schematic diagram for monkeypox dynamics, based on the assumptions above, is shown in Figure 1. The solid arrows represent the transition of the disease from one compartment to another, while the dotted arrows represent the

interactions between the rodent subpopulation and the human subpopulation. The direction of transmission indicated by the dotted arrow is from rodents to humans, meaning the disease is from the rodent population to humans.

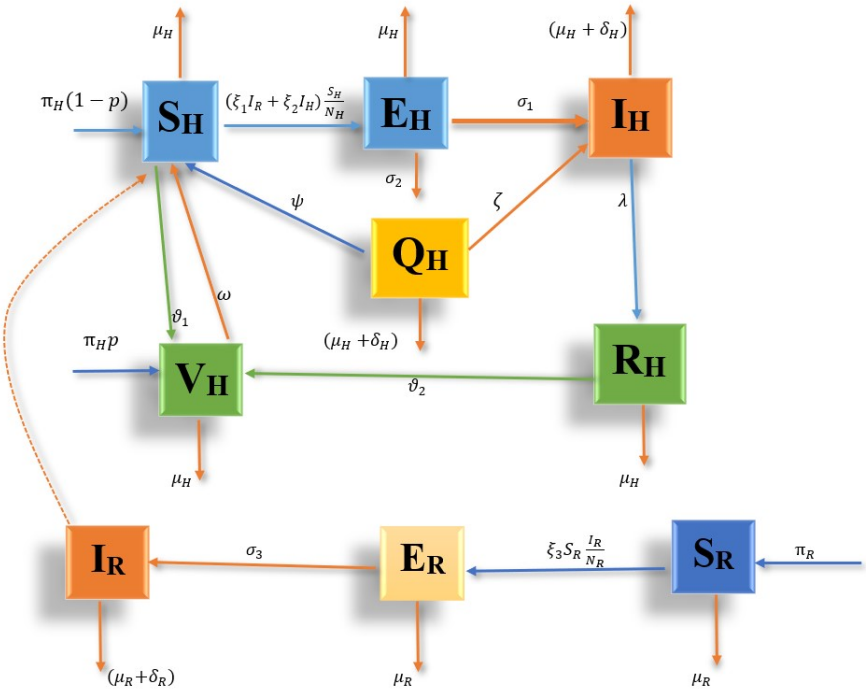

Figure 1: The Schematic diagram of Monkeypox virus transition dynamics

2.3. Model equations

From the schematic diagram in Figure 1 we obtain the following differential model equations

$$\begin{cases} \frac{dS_H}{dt} &= \pi_H(1-p) - (\xi_1 I_R + \xi_2 I_H)\frac{S_H}{N_H} - (\mu_H + \vartheta_1)S_H + \omega V_H + \psi Q_H \\ \frac{dE_H}{dt} &= (\xi_1 I_R + \xi_2 I_H)\frac{S_H}{N_H} - (\sigma_1 + \sigma_2 + \mu_H)E_H \\ \frac{dI_H}{dt} &= \sigma_1 E_H + \zeta Q_H - (\mu_H + \delta_H + \lambda)I_H \\ \frac{dQ_H}{dt} &= \sigma_2 E_H - (\psi + \zeta + \mu_H + \delta_H)Q_H \\ \frac{dR_H}{dt} &= \lambda I_H - (\mu_H + \vartheta_2)R_H \\ \frac{dV_H}{dt} &= \pi_H p + \vartheta_1 S_H + \vartheta_2 R_H - (\mu_H + \omega)V_H \\ \frac{dS_R}{dt} &= \pi_R - \xi_3 I_R \frac{S_R}{N_R} - \mu_R S_R \\ \frac{dE_R}{dt} &= \xi_3 I_R \frac{S_R}{N_R} - (\mu_R + \sigma_3)E_R \\ \frac{dI_R}{dt} &= \sigma_3 E_R - (\mu_R + \delta_R)I_R. \end{cases} \quad (1)$$

2.4. Values for each parameter used for simulation

The following Table 2 presents the parameter descriptions and values that have been used for the simulation:

Parameter	Description	Value	Unit	Source
π_H	Human Recruitment rate	1,160,000	$year^{-1}$	[Estimated]
π_R	Rodents Recruitment rate	200000	$year^{-1}$	[Estimated]
ξ_1	Force of infection from Rodents to Human	0.0025	$year^{-1}$	[26]
ξ_2	Force of infection from Human to Human	0.000063	$year^{-1}$	[26]
ξ_3	Force of infection from Rodents to Rodents	0.0027	$year^{-1}$	[26]
σ_1	rate of transition from exposed human to infected human	0.2	$year^{-1}$	[17]
σ_2	Transition rate from exposed to isolated cases	2.0	$year^{-1}$	[17]
σ_3	transition from exposed rodents to infected rodents	3.0	$year^{-1}$	[Assumed]
ψ	fraction of those isolated that is not infected	2.0	$year^{-1}$	[17]
ζ	Progression from isolated to infected class	0.52	$year^{-1}$	[17]
λ	Humans recovery rate	0.83	$year^{-1}$	[27]
μ_H	Natural death rate of human	1.5	$year^{-1}$	[26]
μ_R	Natural death rate of rodents	0.002	$year^{-1}$	[26]
δ_R	Disease induced death rate for rodents	0.5	$year^{-1}$	[17]
δ_H	Death rate due to disease for humans	0.2	$year^{-1}$	[28]
ϑ_1	Vaccination rate of humans from susceptible class	0.1	$year^{-1}$	[18]
ϑ_2	Vaccination rate of humans from Recovered class	0.01	$year^{-1}$	[Assumed]
ω	Rate loss of Vaccination effectiveness	0.003	$year^{-1}$	[Assumed]
p	fraction of vaccinated immigrants	0.0004	$year^{-1}$	[Assumed]

Table 2: Parameter Values used in Numerical simulation

2.5. Model Analysis

2.5.1. Invariant Domain

Theorem 2.1. *Let the initial conditions of the monkeypox disease dynamical system 1 be given as* $S_H(0) \geqslant 0, I_H(0) \geqslant 0, E_H(0) \geqslant 0, V_H(0) \geqslant 0, Q_H(0) \geqslant 0, R_H(0) \geqslant 0, S_R(0) \geqslant 0, E_R(0) \geqslant 0, I_R(0) \geqslant 0$ *Then, the solutions* $\Omega = (S_H, I_H, E_H, V_H, Q_H, R_H, S_R, E_R, I_R) \in R_+^9$ *of the model 1 are non-negative for all time* $t > 0$

Proof of Theorem 2.1. *From the model equations 1, it is clearly seen that*

$$\frac{dS_H(t)}{dt} \geqslant -(\xi_1 I_R(t) + \xi_2 I_H(t))\frac{S_H(t)}{N_H(t)} - (\mu_H + \vartheta_1)S_H(t) \tag{2}$$

Solving (2) we have,

$$S_H(t) \geqslant S_H(0)\exp\left\{ -\int_0^t ((\xi_1 I_R(\eta) + \xi_2 I_H(\eta))\frac{S_H(\eta)}{N_H(\eta)} + (\mu_H + \vartheta_1)S_H(\eta))d\eta \right\} \tag{3}$$

Before the onset of disease, $I_R = E_H = I_H = 0$ *and after the onset of disease when* $I_R > 0, E_H > 0, I_H > 0$ *as* $t \to \infty$ *in equation 3 we have;*

$$\lim_{t\to\infty} S_H(t) > 0 \tag{4}$$

Similarly, following the same procedure for the other equations of 1 gives $(S_H, I_H, E_H, V_H, Q_H, R_H, S_R, E_R, I_R) \in R_+^6 \times R_+^3$ *of the model 1 are non-negative for all time* $t > 0$. *For details of the proof one can read the work of [12, 17].*

2.6. Steady States of the Model

In this section the model system is analyzed qualitatively by determining the equilibrium points, finding out their corresponding stability (local and global for disease free equilibrium and endemic equilibrium) and subsequently interpreting the sensitivity of the model parameters.

2.6.1. Monkeypox Free Equilibrium state (MFE), ε_0

An equilibrium solution for the system of equations 1 is obtained by setting the derivatives equal to zero and solving the algebraic equations.

One of the equilibrium solutions obtaining from solving the system of algebraic equations represents no disease, $E_H^* = Q_H^* = R_H^* = E_R^* = I_R^* = 0$, $S_H^* = \dfrac{\pi_H(1-p)(\mu_H+\omega)+\pi_H p\omega}{(\mu_H+\vartheta_1)(\mu_H+\omega)+\omega\vartheta_1}$, $V_H^* = \dfrac{\vartheta_1 S_H^* + p\pi_H}{\mu_H(\mu_H+\vartheta_1)}$ and $S_R^* = \dfrac{\pi_R}{\mu_R}$.

We define this equilibrium solution as Monkeypox-free-equilibrium (MFE) and denote it using ε_0.

$$\varepsilon_0 = \left(\frac{\pi_H(1-p)(\mu_H+\omega)+\pi_H p\omega}{(\mu_H+\vartheta_1)(\mu_H+\omega)+\omega\vartheta_1}, 0, 0, 0, 0, \frac{\vartheta_1 S_H^* + p\pi_H}{\mu_H(\mu_H+\vartheta_1)}, \frac{\pi_R}{\mu_R}, 0, 0 \right) \quad (5)$$

Where,

$$S_H^* = \frac{\pi_H(1-p)(\mu_H+\omega)+\pi_H p\omega}{(\mu_H+\vartheta_1)(\mu_H+\omega)+\omega\vartheta_1}.$$

2.6.2. Monkeypox virus Endemic equilibrium (MEE)

A second equilibrium solution obtaining from solving the system of algebraic equations represents the case when $I_H, I_R \neq 0$. This solution is termed as the

endemic equilibrium solution and is given as

$$\begin{aligned}
S_H^* &= \frac{\pi_H(1-p)+\omega V_H^*+\psi Q_H^*}{\xi_1 I_R^*+\xi_2 I_H^*+\mu_H+\nu_1},\\
E_H^* &= \frac{\xi_1 I_R^*+\xi_2 I_H^* S_H^*}{\sigma_1+\sigma_2+\mu_H},\\
I_H^* &= \frac{\sigma_1 E_H^*+\zeta Q_H^*}{\mu_H+\delta_H+\lambda},\\
Q_H^* &= \frac{\sigma_2 E_H^*}{\psi+\zeta+\mu_H+\delta_H},\\
R_H^* &= \frac{\lambda I_H^*}{\mu_H+\nu_2},\\
V_H^* &= \frac{p\pi_H\nu_1 S_H^*+\nu_2 R_H^*}{\mu_H+\omega},\\
S_R^* &= \frac{(\mu_R+\sigma_3)(\mu_R+\delta_R)}{\xi_3\sigma_3},\\
E_R^* &= \frac{(\pi_R\sigma_3\xi_3)}{(\mu_R+\sigma_3)^2(\mu_R+\delta_R)}\left(\frac{(\pi_R\sigma_3\xi_3)}{(\mu_R+\sigma_3)(\mu_R+\delta_R)}-\mu_R\right),\\
I_R^* &= \frac{\pi_R\sigma_3}{(\mu_R+\sigma_3)(\mu_R+\delta_R)}-\frac{\mu_R}{\xi_3}.
\end{aligned} \tag{6}$$

Endemic equilibrium occurs when the infection (monkeypox virus) persists in the population.

2.6.3. Computing effective reproduction number ($\mathcal{R}_v$)

The next-generation approach is used (see Martcheva [12] and Al-Shomrani[21]) as a techniques for computing effective reproduction number ($\mathcal{R}_v$). The idea for the next-generation approach rests on the observation that $\mathcal{R}_v$ is characterized by regarding the infection transmission as producing offspring in an epidemiological sense that is, giving birth to a new infected individual [19]. The approach used here is that developed by Van den Driessche and Watmough whereby, we divided the compartments into two broad categories: infected compartments and non-infected (healthy) compartments [29]. The compartments for which

individuals are infected but not infectious (such as Quarantined and Exposed individuals), are also among the infected compartments. Clearly, in this model the disease free compartments are S_H, R_H, V_H and S_R while infectious compartments are E_H, I_H, Q_H, E_R and I_R. Then by Next generation approach, the compartments of infected groups is taken then split and the model can be written as;

$$\frac{dX}{dt} = F(X) - V(X)$$

That is,

$$\frac{dX}{dt} = \begin{cases} (\xi_1 I_R + \xi_2 I_H)\frac{S_H}{N_H} - (\sigma_1 + \sigma_2 + \mu_H)E_H \\ \sigma_1 E_H + \zeta Q_H - (\mu_H + \delta_H + \lambda)I_H \\ \sigma_2 E_H - (\psi + \zeta + \mu_H + \delta_H)Q_H \\ \xi_3 I_R \frac{S_R}{N_R} - (\mu_R + \sigma_3)E_R \\ \sigma_3 E_R - (\mu_R + \delta_R)I_R \end{cases} \tag{7}$$

Then after splitting the right hand side in the infected compartments gives the following

$$F_i = \begin{cases} (\xi_1 I_R + \xi_2 I_H)\frac{S_H}{N_H} \\ 0 \\ 0 \\ \xi_3 I_R \frac{S_R}{N_R} \\ 0 \end{cases} \tag{8}$$

and

$$V_i = \begin{cases} (\sigma_1 + \sigma_2 + \mu_H)E_H \\ -\sigma_1 E_H - \zeta Q_H + (\mu_H + \delta_H + \lambda)I_H \\ -\sigma_2 E_H + (\psi + \zeta + \mu_H + \delta_H)Q_H \\ (\mu_R + \sigma_3)E_R \\ -\sigma_3 E_R + (\mu_R + \delta_R)I_R \end{cases} \tag{9}$$

Now, the partial derivatives with respect to the dependent variables E_H, I_H, E_R, I_R in equation 8 and 9 are taken and then upon substituting the MFE values we get

$$F = \begin{pmatrix} 0 & \xi_2 \frac{\pi_H(1-p)(\mu_H+w)+\pi_H p\omega}{N_H[(\mu_H+\nu_1)(\mu_H+\omega)+\omega\nu_1]} & 0 & 0 & \xi_1 \frac{\pi_H(1-p)(\mu_H+w)+\pi_H p\omega}{N_H[(\mu_H+\nu_1)(\mu_H+\omega)+\omega\nu_1]} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \frac{\xi_3\pi_R}{N_R\mu_R} \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix} \tag{10}$$

$$V = \begin{pmatrix} \sigma_1+\sigma_2+\mu_H & 0 & 0 & 0 & 0 \\ -\sigma_1 & \mu_H+\delta_H+\lambda & -\zeta & 0 & 0 \\ -\sigma_2 & 0 & \psi+\zeta+\mu_H+\delta_H & 0 & 0 \\ 0 & 0 & 0 & \mu_R+\sigma_3 & 0 \\ 0 & 0 & 0 & -\sigma_3 & \mu_R+\delta_R \end{pmatrix}. \tag{11}$$

We need to find FV^{-1}:

For simplicity we can let;

$$\begin{cases} v_{11} &= \sigma_1 + \sigma_2 + \mu_H \\ v_{22} &= \mu_H + \delta_H + \lambda \\ v_{33} &= \psi + \zeta + \mu_H + \delta_H \\ v_{44} &= \mu_R + \sigma_3 \\ v_{55} &= \mu_R + \delta_R \end{cases}$$

Then the matrix V in equation 11 becomes,

$$V = \begin{pmatrix} v_{11} & 0 & 0 & 0 & 0 \\ -\sigma_1 & v_{22} & -\zeta & 0 & 0 \\ -\sigma_2 & 0 & v_{33} & 0 & 0 \\ 0 & 0 & 0 & v_{44} & 0 \\ 0 & 0 & 0 & \sigma_3 & v_{55} \end{pmatrix} \tag{12}$$

Computing the determinant we get,

$$|V| = v_{11}v_{22}v_{33}v_{44}v_{55}$$

The inverse of V is given by:

$$V^{-1} = \frac{1}{|V|} \begin{pmatrix} v_{22}v_{33}v_{44}v_{55} & 0 & 0 & 0 & 0 \\ \sigma_1 v_{33}v_{44}v_{55} & v_{11}v_{33}v_{44}v_{55} & 0 & 0 & 0 \\ 0 & 0 & v_{11}v_{22}v_{44}v_{55} & 0 & 0 \\ 0 & 0 & 0 & v_{11}v_{22}v_{33}v_{55} & 0 \\ 0 & 0 & 0 & \sigma_3 v_{11}v_{22}v_{33} & v_{11}v_{22}v_{33}v_{44} \end{pmatrix} \tag{13}$$

Then,

$$FV^{-1} = \frac{1}{|V|}\begin{pmatrix} \sigma_1 v_{33}v_{44}v_{55}\xi_2 k & v_{11}v_{33}v_{44}v_{55}\xi_2 k & 0 & \sigma_3 v_{11}v_{22}v_{33}\xi_1 k & v_{11}v_{22}v_{33}v_{44}\xi_1 k \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \sigma_3 v_{11}v_{22}v_{33}\xi_3 l & v_{11}v_{22}v_{33}v_{44}\xi_3 l \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix} \tag{14}$$

where, $k = \dfrac{\pi_H(1-p)(\mu_H + w) + \pi_H p\omega}{N_H[(\mu_H + \nu_1)(\mu_H + \omega) + \omega\nu_1]}$ and $l = \dfrac{\pi_R}{N_R\mu_R}$

The basic reproduction number is defined as the largest eigenvalue (spectral radius) of the next generation matrix.

Definition 2.1. *The spectral radius of a matrix A is defined as the maximum of the absolute values of the eigenvalues of A:$\rho(A) = sup\{|\lambda| : \lambda \in \sigma(A)\}$, where $\sigma(A)$ denotes the eigenvalues of A.*

Consequently, it is easily shown that the eigenvalues of A are:

$$\begin{cases} \lambda_1 &= \frac{\sigma_1\xi_2 k}{v_{11}v_{22}} \\ \lambda_2 &= 0 \\ \lambda_3 &= 0 \\ \lambda_4 &= \frac{\sigma_3\xi_3 l}{v_{44}v_{55}} \\ \lambda_5 &= 0 \end{cases} \tag{15}$$

The Basic reproduction number is the largest eigenvalue of the next generation matrix, from Equation 15. The Effective reproduction number is

$$\mathcal{R}_v = max\left\{ \frac{\sigma_1\xi_2 k}{v_{11}v_{22}}, \frac{\sigma_3\xi_3 l}{v_{44}v_{55}} \right\}. \tag{16}$$

That is,

$$\mathcal{R}_v = max\left\{ \begin{array}{cc} \frac{\sigma_1\xi_2}{(\sigma_1+\sigma_2+\mu_H\mu_H+\delta_H+\lambda)}\frac{\pi_H(1-p)(\mu_H+w)+\pi_H p\omega}{(N_H[(\mu_H+\vartheta_1)(\mu_H+\omega)+\omega\vartheta_1])}, \\ \\ \frac{\sigma_3\xi_3}{(\mu_R+\sigma_3\mu_R+\delta_R)}\frac{\pi_R}{(N_R\mu_R)} \end{array} \right\}. \quad (17)$$

Since λ_1 is clearly on dynamics related to human population and λ_4 is for Rodent population, then

$$\begin{aligned} \mathcal{R}_H &= \frac{\sigma_1\xi_2}{(\sigma_1+\sigma_2+\mu_H)(\mu_H+\delta_H+\lambda)}.\frac{\pi_H(1-p)(\mu_H+w)+\pi_H p\omega}{(N_H[(\mu_H+\vartheta_1)(\mu_H+\omega)+\omega\vartheta_1])} \\ \mathcal{R}_R &= \frac{\sigma_3\xi_3}{(\mu_R+\sigma_3\mu_R+\delta_R)}\frac{\pi_R}{(N_R\mu_R)} \end{aligned} \quad (18)$$

In the absence of vaccine and quarantine, that is $\vartheta_1 = p = \omega = 0 = \vartheta_2 = \sigma_2$, then we have the basic reproduction number as

$$\mathcal{R}_0 = max\left\{ \frac{\sigma_1\xi_2}{(\sigma_1+\mu_H)(\mu_H+\delta_H+\lambda)}\frac{\pi_H\mu_H}{(N_H\mu_H)}, \quad \frac{\sigma_3\xi_3}{(\mu_R+\sigma_3\mu_R+\delta_R)}\frac{\pi_R}{(N_R\mu_R)} \right\}. \quad (19)$$

Therefore, since we are interested in reducing the transmission of the virus to the human population then the analysis done here considers all the possibilities of the basic reproduction number for the two populations such as the case when $\mathcal{R}_H > \mathcal{R}_R$, $\mathcal{R}_H < \mathcal{R}_R$ and $\mathcal{R}_H = \mathcal{R}_R$. Throughout this study the analysis has been done with the main objective of investigation on how to reduce the transmission to the human population when $\mathcal{R}_H < 1$and $\mathcal{R}_R < 1$.

2.6.4. Analysis of the basic effective reproduction number ($\mathcal{R}_v$)

Theorem 2.2. *The possibility that the monkeypox virus could be eradicated from the community is when $\mathcal{R}_v < 1$ which implies that $\mathcal{R}_H < 1$ and $\mathcal{R}_R < 1$ and the derivatives with respect to the transmission rate ξ_2 and ξ_3 should always be positive to reduce the transmission rate, also the derivative with respect to quarantine, σ_2 and vaccination, ϑ_1 rate should always be negative to reduce the reproduction number [30]*

Proof of Theorem 2.2. *Now taking the partial derivatives of $\mathcal{R}_v$ with respect to ξ_2 and ξ_3 we have:*

For ξ_2

$$\frac{\partial \mathcal{R}_v}{\partial \xi_2} = \frac{\sigma_1}{(\sigma_1+\sigma_2+\mu_H)(\mu_H+\delta_H+\lambda)} \frac{\pi_H(1-p)(\mu_H+\omega)+\pi_H p\omega}{(N_H[(\mu_H+\vartheta_1)(\mu_H+\omega)+\omega\vartheta_1])} \quad (20)$$

For ξ_3

$$\frac{\partial \mathcal{R}_v}{\partial \xi_3} = \frac{\sigma_3}{(\mu_R+\sigma_3\mu_R+\delta_R)} \frac{\pi_R}{(N_R\mu_R)} \quad (21)$$

which are always positive and this implies that the transmission could reduce the reproduction number linearly.

Moreover, this could be accomplished by isolation or quarantine the exposed cases in human population. That is, taking the partial derivative of $\mathcal{R}$ *with respect to* σ_2 *and* ϑ_1 *we have,*

$$\frac{\partial \mathcal{R}_v}{\partial \sigma_2} = -\frac{\sigma_1\xi_2}{(\sigma_1+\sigma_2+\mu_H)^2(\mu_H+\delta_H+\lambda)} \frac{\pi_H(1-p)(\mu_H+\omega)+\pi_H p\omega}{(N_H[(\mu_H+\vartheta_1)(\mu_H+\omega)+\omega\vartheta_1])} \quad (22)$$

which is always negative which implies that the increase in quarantine for suspected cases will reduce the reproduction number.

The same applies to the impacts of Vaccination, when analyzing we see that,

$$\frac{\partial \mathcal{R}_v}{\partial \vartheta_1} = -\frac{\sigma_1\xi_2}{(\sigma_1+\sigma_2+\mu_H)(\mu_H+\delta_H+\lambda)} \frac{(\pi_H(1-p)(\mu_H+\omega)+\pi_H p\omega)(\mu_H+2\omega)}{(N_H[(\mu_H+\vartheta_1)(\mu_H+\omega)+\omega\vartheta_1])^2} \quad (23)$$

which also is always negative, which implies that increasing the Vaccination could also reduce the reproduction number.

2.7. Global stability of the Monkey-pox virus Free Equilibrium

We used the approach as that done by Mumbu[31] where he used the Lyapunov function, in detail it has also been shown by Martcheva [12] and Li [32] to determine the global stability of the monkeypox virus free equilibrium one needs to prove that the Lyapunov function L is asymptotically stable.

Theorem 2.3. *If a function* $L(x)$ *is globally positive definite and radially unbounded, and its time derivative is globally negative,* $L'(x) < 0$ *for all* $x \neq x^*$ *then the equilibrium* x *is globally stable.*

Proof of Theorem 2.3. *For the proof details see in [12, 31]*
Suppose $\mathcal{R}_H > 1$ *and* $\mathcal{R}_R > 1$, *in which the endemic equilibrium point exists, then to prove the global stability, the Lyapunov function* L *is define and derive as follows: The approach used here is by letting the non-negative constants,* C_i *for* $i = 1, 2, \ldots, 6$ *and multiply to all the compartments which give zero values at the* MFE.

$$\begin{aligned} L = (S_H - S_H^* - S_H^* \ln \frac{S_H}{S_H^*}) + C_1 E_H + C_2 I_H + C_3 Q_H + C_4 R_H + \\ +(V_H - V_H^* - V_H^* \ln \frac{V_H}{V_H^*}) + (S_R - S_R^* - S_R^* \ln \frac{S_R}{S_R^*}) + C_5 E_R + C_6 I_R \end{aligned} \tag{24}$$

Then differentiating with respect to t *and supposing,* $S_H \leqslant S_H^*$, $V_H \leqslant V_H^*$ *and* $S_R \leqslant S_R^*$ *then the equation reduces to*

$$\frac{dL}{dt} = C_1 \frac{dE_H}{dt} + C_2 \frac{dI_H}{dt} + C_3 \frac{dQ_H}{dt} + C_4 \frac{dR_H}{dt} + C_5 \frac{dE_R}{dt} + C_6 \frac{dI_H}{dt} \tag{25}$$

Substituting the values from Equation1 we get,

$$\begin{aligned} \tfrac{dL}{dt} = C_1((\xi_1 I_R + \xi_2 I_H)\frac{S_H}{N_H} - (\sigma_1 + \sigma_2 + \mu_H)E_H) + C_2(\sigma_1 E_H + \zeta Q_H - (\mu_H + \delta_H + \lambda)I_H) + \\ +C_3(\sigma_2 E_H - (\phi + \zeta + \mu_H + \delta_H)Q_H) + C_4(\lambda I_H - (\mu_H + \vartheta_2)R_H) + \\ +C_5(\xi_3 I_R \frac{S_R}{N_R} - (\mu_R + \sigma_3)E_R) + C_6(\sigma_3 E_R - (\mu_R + \delta_R)I_R) \end{aligned} \tag{26}$$

Rearranging terms with trivial solution in MFE by collecting like terms we get,

$$\begin{aligned} \tfrac{dL}{dt} \leqslant (C_1 \xi_1 \tfrac{S_H^*}{N_H} + C_5 \xi_3 \tfrac{S_R^*}{N_R} - C_6(\mu_R + \delta_R))I_R + (C_1 \xi_2 \tfrac{S_H^*}{N_H} - C_2(\mu_H + \delta_H + \lambda) + C_4 \lambda)I_H \\ +(C_1(-(\sigma_1 + \sigma_2 + \mu_H)) + C_2 \sigma_1 + C_3 \sigma_2)E_H + (C_3(-(\psi + \zeta + \mu_H + \delta_H)) + C_2 \zeta)Q_H + \\ +(-C_4(\mu_H + \vartheta_2)R_H) + C_5(-(\mu_R + \sigma_3) + C_6 \sigma_3)E_R \end{aligned} \tag{27}$$

Then equating each term to zero and substituting the values of S_H^* and S_R^*, as we are looking for the points in the system state space where the function L is at an equilibrium, we obtain the following

$$\begin{cases} C_1\xi_1\frac{S_H^*}{N_H} + C_5\xi_3\frac{S_R^*}{N_R} - C_6(\mu_R + \delta_R) = 0 \\ C_1\xi_2\frac{S_H^*}{N_H} - C_2(\mu_H + \delta_H + \lambda) + C_4\lambda = 0 \\ -C_1(\sigma_1 + \sigma_2 + \mu_H) + C_2\sigma_1 + C_3\sigma_2 = 0 \\ -C_3(\psi + \zeta + \mu_H + \delta_H) + C_2\zeta = 0 \\ -C_4(\mu_H + \vartheta_2) = 0 \\ -C_5(\mu_R + \sigma_3) + C_6\sigma_3 = 0 \end{cases} \tag{28}$$

Solving for the constants, we have $C_4 = 0$ and $C_3 = 0$ substituting them back we have

$$\begin{cases} C_1\xi_2\frac{S_H^*}{N_H} - C_2(\mu_H + \delta_H + \lambda) = 0 \\ -C_1(\sigma_1 + \sigma_2 + \mu_H) + C_2\sigma_1 = 0 \end{cases} \tag{29}$$

Making C_2 the subject we get,

$$C_2 = C_1\frac{\sigma_1 + \sigma_2 + \mu_H}{\sigma_1} \tag{30}$$

Similarly for C_6

$$C_6 = C_5\frac{\sigma_3 + \mu_R}{\sigma_3} \tag{31}$$

Substituting back to Equation 27 and Simplifying we get,

$$\frac{dL}{dt} \leqslant C_1^*\left(\frac{\sigma_1\xi_2}{(\sigma_1+\sigma_2+\mu_H)(\mu_H+\delta_H+\lambda)}\frac{\pi_H(1-p)(\mu_H+w)+\pi_H p\omega}{(N_H[(\mu_H+\vartheta_1)(\mu_H+\omega)+\omega\vartheta_1])}-1\right)$$
$$+C_5^*\left(\frac{\sigma_3\xi_3}{(\mu_R+\sigma_3\mu_R+\delta_R)}\frac{\pi_R}{(N_R\mu_R)}-1\right) \tag{32}$$

That is,

$$\frac{dL}{dt} \leqslant C_1^*(\mathcal{R}_H-1)+C_5^*(\mathcal{R}_R-1) \tag{33}$$

Clearly, $\frac{dL}{dt} < 0$ *if and only if when* $\mathcal{R}_H < 1$ *and* $\mathcal{R}_R < 1$ *, which proves the theorem, therefore the* MFE *is globally stable.*

2.7.1. ***Global stability of the Monkeypox Endemic Equilibrium (MEE)***

Theorem 2.4. *Endemic equilibrium is asymptotically stable when* $\mathcal{R}_H > 1$ *(for Human) and* $\mathcal{R}_R > 1$ *(for rodents) and unstable when* $\mathcal{R}_R < 1$ *and* $\mathcal{R}_R < 1$ *[33]*

Proof of Theorem 2.4. *For this study we used the approach as that of Masandawa et.al [34], with Logarithmic Lyapunov function as* $p = \sum_{i=1}^{9} C_i(x_i - x_i^*)\ln(x_i)$ *Where,* C_i *is a positive constant* x_i *is population of the compartment* i *and* x_i^* *is the equilibrium level value.*

$$p(S_H,E_H,I_H,Q_H,R_H,V_H,S_R,E_R,I_R) = C_1((S_H-S_H^*)\ln S_H)+$$
$$+C_2((E_H-E_H^*)\ln E_H)+C_3((I_H-I_H^*)\ln I_H)+C_4((Q_H-Q_H^*)\ln Q_H)+$$
$$+C_5((R_H-R_H^*)\ln R_H)+C_6((V_H-V_H^*)\ln V_H)+C_7((S_R-S_R^*)\ln S_R)+ \tag{34}$$
$$+C_8((E_R-E_R^*)\ln E_R)+C_9((I_R-I_R^*)\ln I_R)$$

Now taking the derivative of p with respect to time and simplifying we have,

$$\begin{aligned}\frac{dp}{dt} = C_1(\frac{(S_H - S_H^*)}{S_H}\frac{dS_H}{dt}) + C_2(\frac{(E_H - S_H^*)}{E_H}\frac{dE_H}{dt}) + C_3(\frac{(I_H - I_H^*)}{I_H}\frac{dI_H}{dt}) + \\ + C_4(\frac{(Q_H - Q_H^*)}{Q_H}\frac{dQ_H}{dt}) + C_5(\frac{(R_H - R_H^*)}{R_H}\frac{dR_H}{dt}) + C_6(\frac{(V_H - V_H^*)}{V_H}\frac{dV_H}{dt}) + \\ + C_7(\frac{(S_R - S_R^*)}{S_R}\frac{dS_R}{dt}) + C_8(\frac{(E_R - E_R^*)}{E_R}\frac{dE_R}{dt}) + C_9(\frac{(I_R - I_R^*)}{I_R}\frac{dI_R}{dt})\end{aligned} \tag{35}$$

Then substitute Equation 1 into Equation 35 we have,

$$\begin{aligned}\frac{dp}{dt} = C_1(\frac{(S_H - S_H^*)}{S_H}\pi_H(1-p) - (\xi_1 I_R + \xi_2 I_H)\frac{S_H}{N_H} - (\mu_H + \vartheta_1)S_H + \\ + \omega V_H + \psi Q_H) + C_2(\frac{(E_H - S_H^*)}{E_H}\xi_1 I_R + \xi_2 I_H)\frac{S_H}{N_H} - (\sigma_1 + \sigma_2 + \mu_H)E_H) + \\ + C_3(\frac{(I_H - I_H^*)}{I_H}\sigma_1 E_H + \zeta Q_H - (\mu_H + \delta_H + \lambda)I_H) + \\ + C_4(\frac{(Q_H - Q_H^*)}{Q_H}\sigma_2 E_H - (\psi + \zeta + \mu_H + \delta_H)Q_H) + \\ + C_5(\frac{(R_H - R_H^*)}{R_H}\lambda I_H - (\mu_H + \vartheta_2)R_H) + \\ + C_6(\frac{(V_H - V_H^*)}{V_H}\pi_H p + \vartheta_1 S_H + \vartheta_2 R_H - (\mu_H + \omega)V_H) + \\ + C_7(\frac{(S_R - S_R^*)}{S_R}\pi_R - \xi_3 I_R\frac{S_R}{N_R} - \mu_R S_R) + \\ + C_8(\frac{(E_R - E_R^*)}{E_R}\xi_3 I_R\frac{S_R}{N_R} - (\mu_R + \sigma_3)E_R) + \\ + C_9(\frac{(I_R - I_R^*)}{I_R}\sigma_3 E_R - (\mu_R + \delta_R)I_R)\end{aligned} \tag{36}$$

As done by Martcheva [12] one of the classical first steps here is to replace π_H

and π_R with its equal from the equilibria equations, that is,

$$\begin{aligned}\pi_H &= \frac{1}{(1-p)}((\xi_1 I_R^* + \xi_2 I_H^*)\frac{S_H^*}{N_H} + (\mu_H + \vartheta_1)S_H^* - \omega V_H^* - \psi Q_H^*) \\ \pi_R &= \xi_3 I_R^* \frac{S_R^*}{N_R} - \mu_R S_R^*\end{aligned} \tag{37}$$

Then $((\mu_H + \sigma_1)S_H^* - \psi S_H^*) - ((\mu_H + \sigma_1)S_H - \psi S_H)$can be combined with the first term in the product to yield a negative term. We multiply out all other products: We now have to apply the Krasovkii-LaSalle theorem. We consider the set where the Lyapunov function is equal to zero, that is

$$L = \{x \in \Re^n | V'(x) = 0\} \tag{38}$$

Clearly from equation 36 the $V^{'}(x) = 0$ if and only if $S_H - S_H^* = 0$, $E_H = E_H^*$, $I_H = I_H^*$, $Q_H = Q_H^*$, $R_H = R_H^*$, $V_H = V_H^*$, $S_R = S_R^*$, $E_R = E_R^*$and $I_R = I_R^*$, other terms are negative when $(E_H, I_H, Q_H, R_H, V_H, S_R, E_R, I_R) > 0$. Therefore, the MEE is globally asymptotically stable when $\mathcal{R}_H > 1$ and $\mathcal{R}_R > 1$ this results concludes and proves the theorem.

3. Model simulation and Discussion

3.1. Sensitivity analysis

The goal of sensitivity analysis is to qualitatively decide which parameters are most influential in the model output. Sensitivity analysis can be performed on a dynamical system or on static quantities such as the reproduction number or equilibria prevalence [11, 35]. We compute the sensitivity indices of the basic reproductive number $\mathcal{R}_0$ to the parameters in model. These indices allow us to see how important each parameter is to disease transmission [36]. The normalized forward sensitivity index of $\mathcal{R}$ that depends differentiability on the parameter p is defined as $\gamma_p^{\mathcal{R}} = \frac{\partial \mathcal{R}}{\partial p} \times \frac{p}{\mathcal{R}}$, where $\gamma_p^{\mathcal{R}}$ is the sensitivity index of $\mathcal{R}$

with respect to parameter p is positive if $\mathcal{R}$ is increasing with respect to p and is negative if $\mathcal{R}$ is decreasing with respect to p [10, 18, 34, 37, 35].

Using the effective reproduction number $\mathcal{R}_v$ we can take partial derivative with respect to each parameter and compute sensitivity, for human and rodent reproduction numbers populations, $\mathcal{R}_H$ and $\mathcal{R}_R$, respectively and then was filled in the Table 3

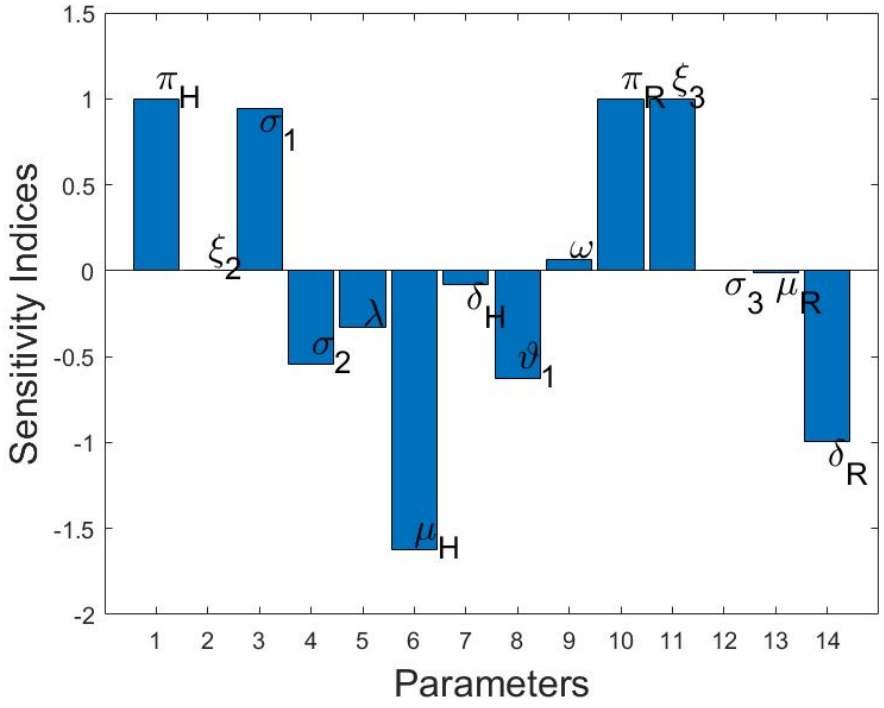

Figure 2: The Sensitivity Indices

Table 3: Sensitivity expression and index for each parameter

SN	Parameter	Expression of the Sensitivity parameter	Sensitivity Value
1	π_H	1	1
2	ξ_2	$\frac{\xi_2\sigma_1\pi_H(1-p)(\mu_H+\omega)}{\sigma_1\xi_2\pi_H(1-p)(\mu_H+\omega)+\pi_H p\omega}$	0.00005999976
3	σ_1	$1-\frac{\sigma_1}{\sigma_1+\sigma_2+\mu_H}$	0.945945945
4	σ_2	$-\frac{\sigma_2}{\sigma_1+\sigma_2+\mu_H}$	-0.54054054
5	λ	$-\frac{\lambda}{(\mu_H+\delta_H+\lambda)}$	-0.328063241
6	μ_H	$\frac{\mu_H}{(\mu_H+\omega)+\pi_H p\omega}+(1+\frac{1}{(\mu_H+\delta_H+\lambda)})\times\frac{\mu_H}{(\sigma_1\xi_3\pi_H(1-p)(\mu_H+\omega)+\pi_H p\omega)}$	1.622222519
7	δ_H	$-\frac{\delta_H}{(\mu_H+\delta_H+\lambda)}$	-0.0790514
8	ϑ_1	$-\frac{2\omega+\mu_H}{(\mu_H+\vartheta_1)(\mu_H+\omega)+\omega\vartheta_1}$	-0.62616939
9	ω	$\frac{\xi_2\sigma_1\pi_H(1-p)(\mu_H+\omega)}{\sigma_1\xi_2\pi_H(1-p)(\mu_H+\omega)+\pi_H p\omega}-\frac{\omega(\mu_H+2\vartheta_1)}{(\sigma_1+\sigma_2+\mu_H)(\mu_H+\delta_H+\gamma)((\mu_H+\vartheta_1)(\mu_H+\omega)+\omega\vartheta_1)}$	0.064051357
10	π_R	1	1
11	ξ_3	1	1
12	σ_3	$1-\frac{\sigma_3}{(\mu_R+\sigma_3)}$	0.000666222
13	μ_R	$-\frac{\mu_R^2(3\mu_R+2\delta_R+2\sigma_3)}{(\mu_R+\sigma_3)(\mu_R+\delta_R)\mu_R}$	-0.009297918
14	δ_R	$-\frac{\delta_R}{\mu_R+\delta_R}$	-0.996015936

The results from the sensitivity analysis clearly indicate that increasing the values of parameters π_H, ξ_2, ω, and σ_1 will lead to an increase in the spread of the monkeypox virus in the human population (for details see Figure 2). Conversely, increasing the values of parameters σ_2, λ, μ_H, δ_H, and ϑ_1 will contribute to a decrease in the spread of the virus among humans. In the case of the rodent population, increasing the values of parameters π_R, ξ_3, and σ_3 will result in an increased infection rate of the monkeypox virus among rodents, while increasing the parameters δ_R and μ_R will lead to the elimination of the virus in the rodent population.

The most sensitive parameters in the analysis are the vaccination rate of susceptible individuals, the progression rate from exposed to infectious individuals, and the progression rate from exposed to individuals in quarantine, as well as the death rate of the rodents.

3.2. Model simulation

The proposed model was simulated and analysed using MATLAB R2018a with the parameter values stated in Table 2. The following initial population densities for people and rodents were considered for the analysis:
$N_H = [S_H, E_H, I_H, Q_H, R_H, V_H] = [4000000, 2000000, 100000, 500000, 200000, 400000]$
$N_R = [S_R, E_R, I_R] = [1000000, 500000, 300000]$ respectively which was simulated for a time span of one year to the developed model. The results of reproduction numbers $\mathcal{R}_v = 5.374900247$, while $\mathcal{R}_H = 2.4768 \times 10^{-7}$ and $\mathcal{R}_R = 5.374900247$ Clearly, the obtained reproduction numbers for the data simulated indicate that the main contribution to transmission comes from rodents, as their transmission can result in more than five (5) new secondary infections. This clearly demonstrates that the primary mode of virus transmission is from rodents to humans, while the transmission from human to human is minimal, with a rate less than one. Consequently, by effectively controlling the transmission among the rodent population, the virus can be eradicated in the human population. This observation is well depicted in Figure 5, Figure 4 and Figure 7, where the formulated model shows that the disease will eventually die out in the human population while persisting in the rodent population. Furthermore, the graphs illustrating the population dynamics in both humans (Figure 3) and rodents (Figure 4) provide additional support for this fact, showcasing the proportional relationship between the populations.

To gain a better understanding of the dynamics, the initial conditions were adjusted to a smaller population size. The model was further analyzed by modifying the initial conditions

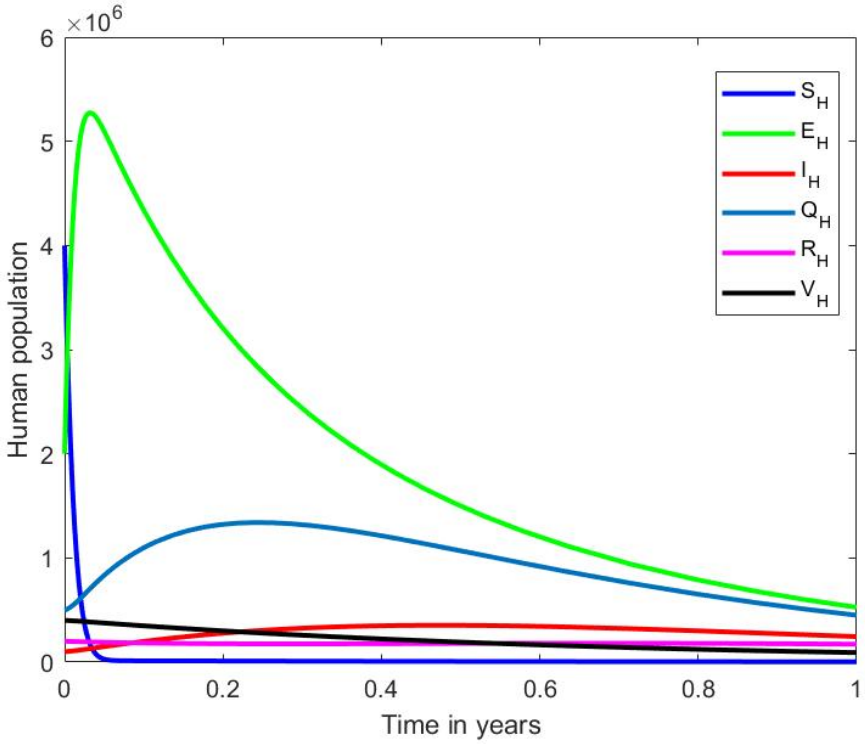

Figure 3: Human population Proportions evolution over time

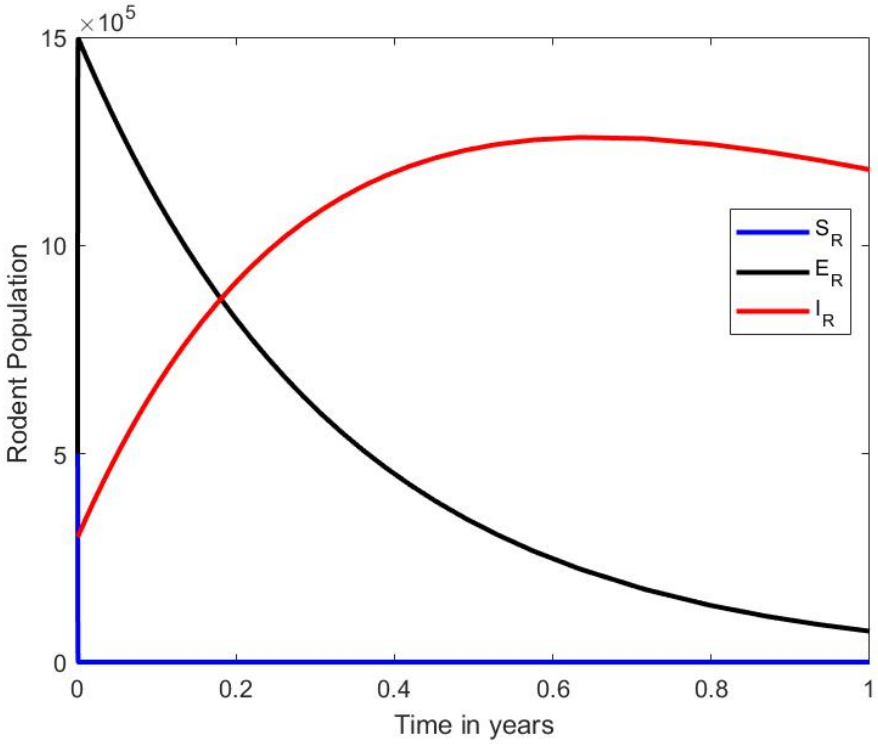

Figure 4: Rodent evolution against time for the proposed mathematical model

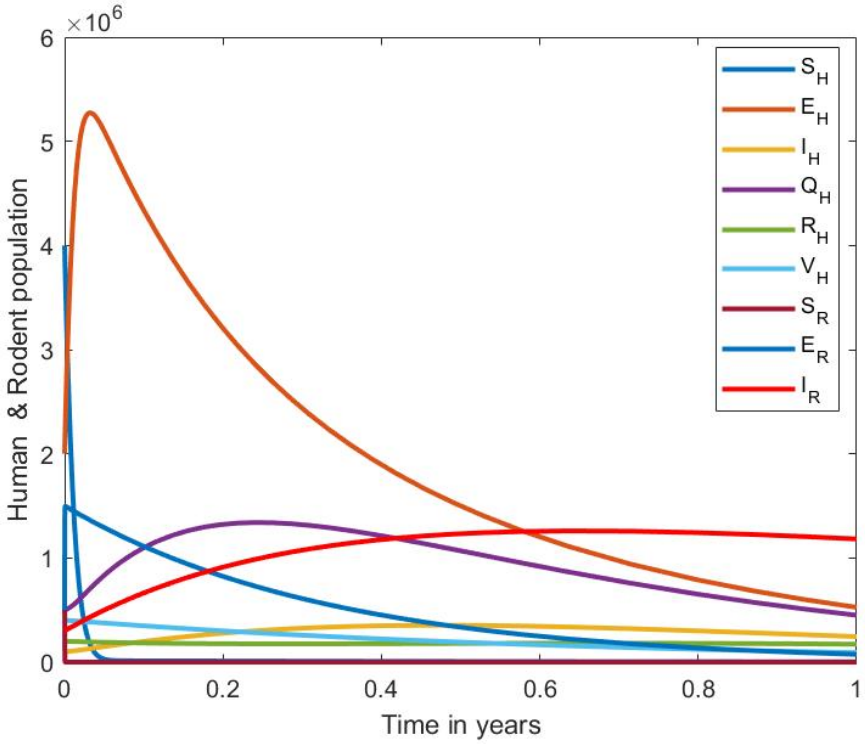

Figure 5: Human and rodent population evolution against time for the proposed model

as follows: For the human population, we set $S_H = 40$, $E_H = 3$, $I_H = 2$, $Q_H = 0$, $R_H = 0$, and $V_H = 0$. Similarly, for the rodent population, we set $S_R = 10$, $E_R = 2$, and $I_R = 1$. These adjustments allow for a more detailed exploration of the system's behavior and its response to different initial conditions. The details are explained in Figure 6 and Figure 7.

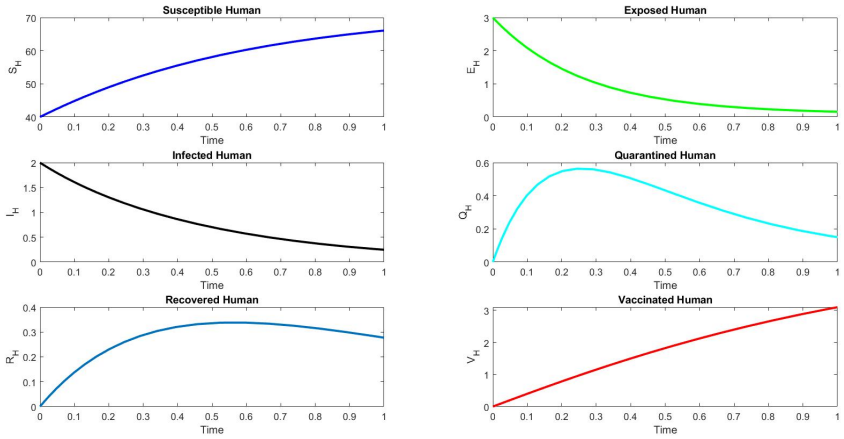

Figure 6: Evolution of the human population over time in the proposed model: It is evident that implementing the suggested model leads to a significant reduction in the number of infected and exposed individuals within the human population. At the same time, it effectively increases the number of individuals who have recovered from the infection and enhances the size of the susceptible population, contributing to overall population health and resilience. The model highlights the potential benefits of the proposed interventions in controlling the spread of the disease and mitigating its impact on the human population.

3.3. Effects of Quarantine and Vaccination on the Transmission of Monkeypox Virus

This is well depicted in Figure 8, Figure 9, Figure 10 Figure 11 and Figure 12 with detailed explanation in their caption. From Figure 9, it is evident that increasing the vaccination rate and implementing both quarantine measures and vaccination (Figure 8) have a significant impact on reducing the number of infectious individuals in the human population. Additionally, controlling the spread of the virus among rodents is also crucial, as demonstrated in Figure 10. By decreasing the force of infection from rodents to humans, the number of infectious individuals in the human population is effectively reduced. This outcome is clearly depicted in Figure 12, where the reduction in the progression rate from exposed rodents to infectious rodents successfully reduces the infectious human population.

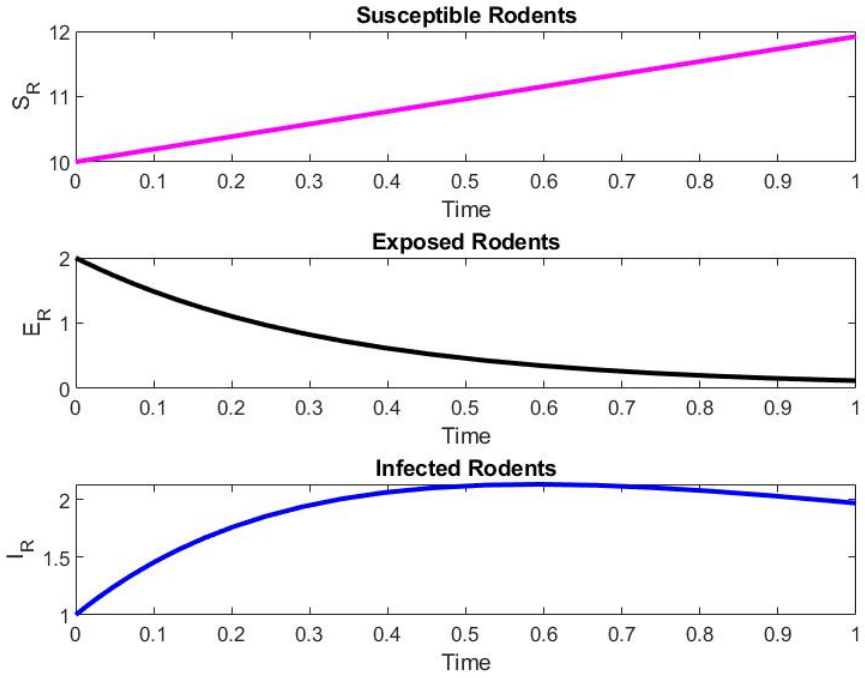

Figure 7: Evolution of the rodent population over time in the proposed model: In the absence of interventions, the dynamics demonstrate a persistent infected population within the rodent population. This highlights the need to address the human-to-rodent contact to effectively eliminate the spread of monkeypox. We recommend implementing measures to reduce human-rodent interactions as a crucial step in controlling the disease and safeguarding public health. By minimizing contact, we can mitigate the transmission of monkeypox from rodents to humans and contribute to the overall prevention and eradication efforts.

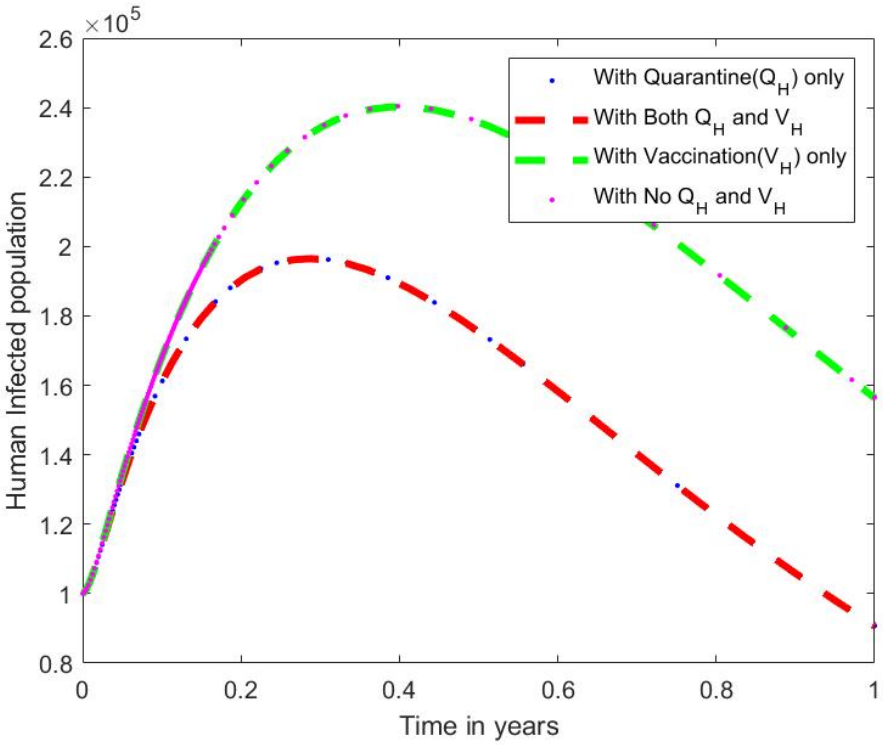

Figure 8: The human infected population increases when no intervention is taken but reduces significantly when both quarantine and vaccination measures are implemented. This observation highlights the effectiveness of these interventions in controlling the spread of monkeypox. Without any intervention, the disease can rapidly propagate within the human population, leading to a higher number of infected individuals. However, with the implementation of quarantine measures and vaccination, virus transmission is significantly curtailed, resulting in a notable reduction in the population of infected humans. This underscores the importance of proactive measures, such as quarantine and vaccination, in mitigating the impact of monkeypox and protecting public health.

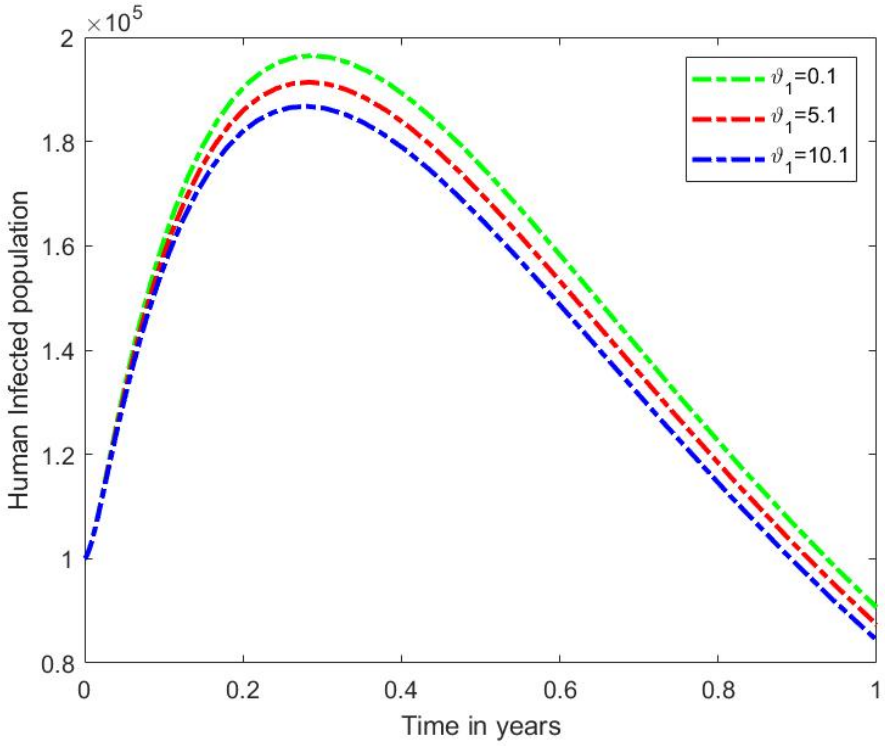

Figure 9: Infected human dynamics under varying vaccination rates: Figure 9 demonstrates the relationship between the vaccination rate and the infected human population. As the vaccination rate increases, there is a noticeable reduction in the number of infected humans. This finding highlights the significance of a higher vaccination rate in mitigating the spread and impact of monkeypox among the human population.

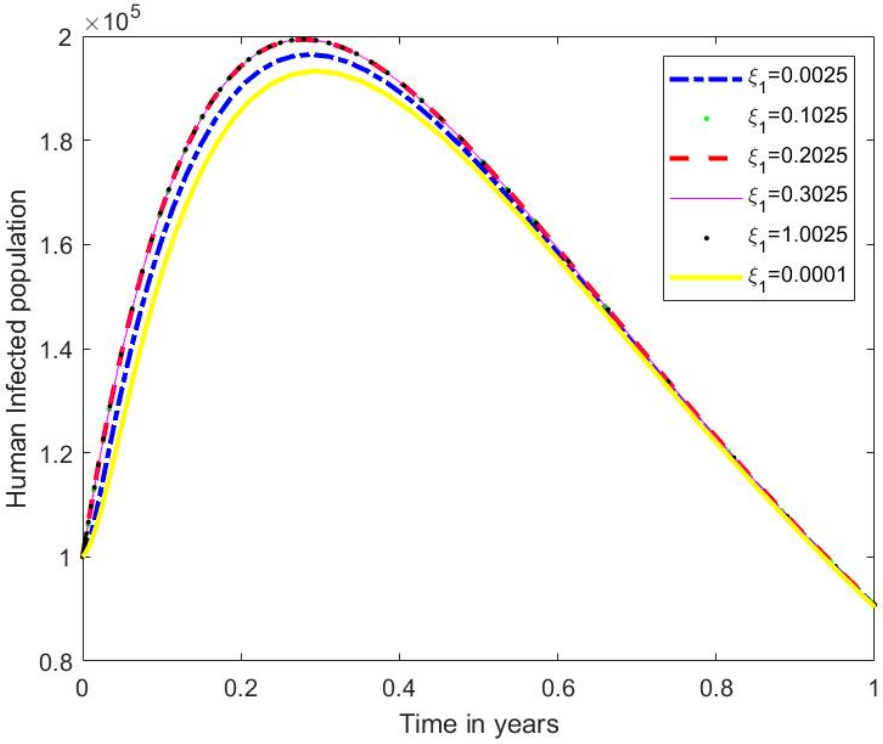

Figure 10: Infected human dynamics under the variation of force of infection from rodents, which shows that an increase in the force of infection will lead to an increase in the population of infected humans. This observation highlights the crucial role of rodents as a source of transmission of monkeypox to humans. As the force of infection from rodents increases, the likelihood of transmission to humans also increases, resulting in a larger population of infected individuals. This emphasizes the importance of effective control measures targeting rodents, such as improved rodent control practices and habitat modification, to mitigate the risk of monkeypox transmission and reduce the burden of human infections.

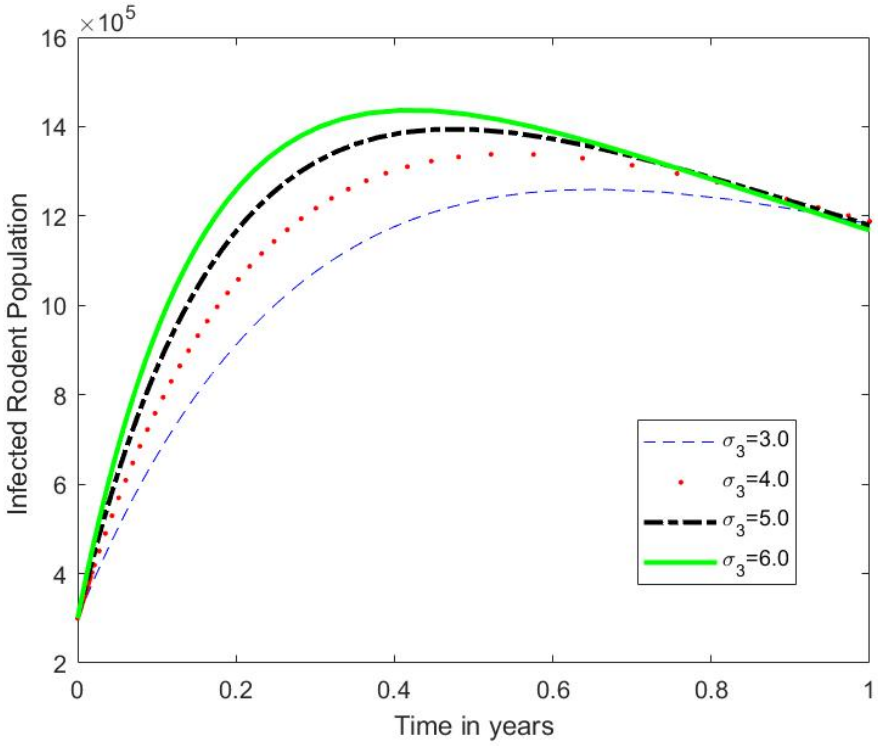

Figure 11: Dynamics of infected rodents with varying progression rates from the exposed to infectious population: The plot illustrates a direct proportional relationship between the increase in the progression rate and the number of infected humans. As the progression rate from the exposed to infectious population in rodents increases, it leads to a corresponding increase in the number of infected humans. This finding emphasizes the importance of understanding and managing the transmission dynamics within the rodent population to effectively control and prevent the spread of monkeypox to humans.

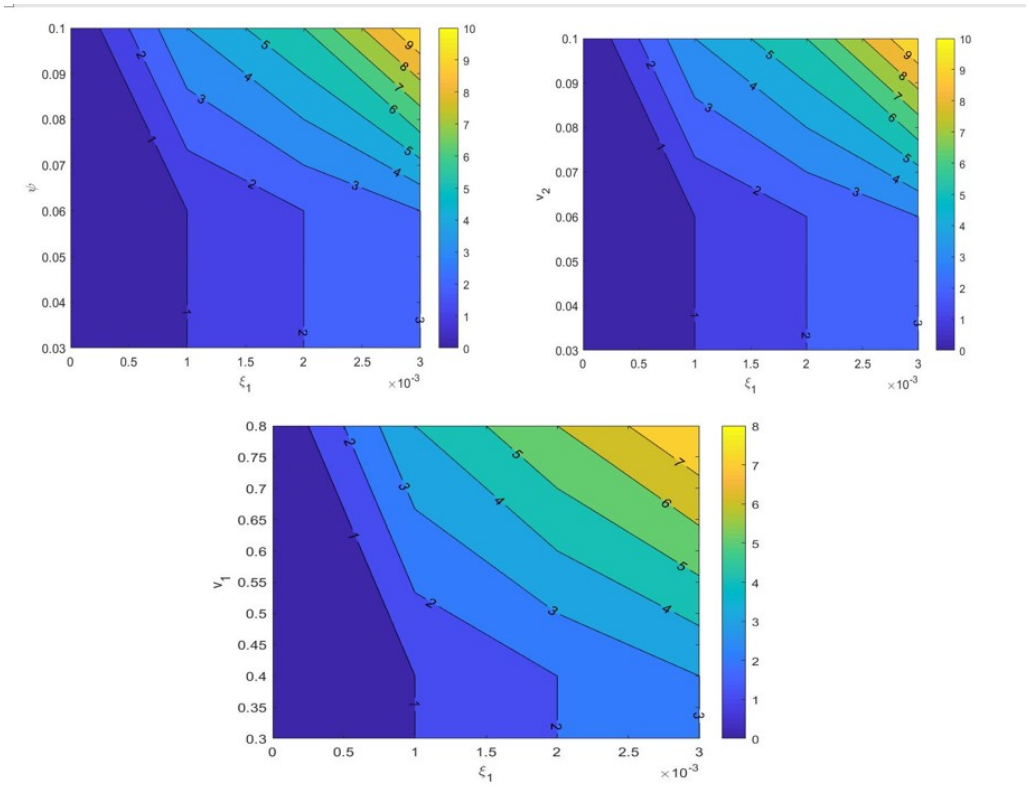

Figure 12: The contour plots demonstrates the contribution of the proposed interventions. Starting from the top left, it shows that increasing the rate of quarantine and reducing the human-to-human contact rate help to reduce the reproduction number. The same principle applies to the vaccine in the top right and bottom sections. They exhibit the same property.

4. Conclusions

In our study, we formulated a deterministic dynamic model for monkeypox virus transmission in which we calculated the DFE. The reproduction number is $\mathcal{R}_v = 5.3749$, with $\mathcal{R}_H = 2.4768 \times 10^{-7}$ for for humans and $\mathcal{R}_R = 5.3749$ for rodents, for the simulated results, which shows that the main contribution of infection by this virus is from the rodent population. The global stability of the model at MFE and MEE was analysed, which revealed it to be stable. The sensitivity analysis was done for the reproduction numbers of both human and rodent populations. The most sensitive parameters that should be taken into account were the rate of vaccination of susceptibles, the progression rate from exposed to infectious and exposed to quarantine, and the mortality rate of the rodent population. Generally, the study shows that for the proposed mathematical model, increasing vaccination, quarantine, and avoiding contact with infected rodents will eradicate the spread of the virus; therefore, we propose this model be used to explore the dynamics of the monkeypox virus outbreak.

In summary, this study contributes to understanding the dynamics of monkeypox virus transmission and provides information for effective control measures. The proposed deterministic dynamic model, with its calculated reproduction numbers and stability analysis, offers valuable guidance for targeted interventions. The sensitivity analysis identifies key parameters for control, while the proposed numerical scheme enhances the model's predictive capacity. Overall, this research has practical implications for outbreak management and can inform public health interventions to eradicate the spread of the monkeypox virus.

Acknowledgments

The authors express their gratitude for the support received from the NORHED II project and the Department of Mathematics at the University of Dar es Salaam in Tanzania, as well as the University of Bergen in Norway. These institutions provided essential facilities that contributed to the successful completion of this research. Additionally, the authors acknowledge the anonymous reviewers for their valuable comments that greatly contributed to the improvement of this work.

Declarations

Ethical Approval

Not applicable

Competing interests

The authors have no conflict of interest.

Authors' contributions

- **Leonce Leandry:** Writing original draft, Conceptualization, Methodology, Formal analysis, Software and Review.
- **Eunice Mureithi:** Validation, Writing review, editing and Supervision.

Funding

This work has been supported by the Mathematics for Sustainable Development (MATH4SDG) project at the University of Dar es Salaam -Tanzania funded through the NORHED II programme under the Norwegian Agency for Development Cooperation (NORAD, project no. 68105).

Availability of data and materials

The numerical data used to support the findings of this study have been taken from previously published articles and are cited in Table 2 of this article. Also, others are available online in `https://www.cdc.gov/poxvirus/mpox/response/2022/world-map.html` and `https://ourworldindata.org/monkeypox`.

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