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ORIGINAL RESEARCH ARTICLE

Mathematical Transmission Dynamics and Intervention Strategies for Monkeypox: A Model-Based Approach Including Human-Rodent Interactions

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ABSTRACT
Monkeypox is now a major public health problem because of threats of infection, which include animal-to-human with human-to-human transmissions of the disease. In this paper, we proposed a SEIR-type model to understand different routes of disease spread by including human-to-animal transmission and intervention strategies. Isolation, hospitalization, and diagnosis parameters are incorporated into the human population as a means of reducing the spread of the disease. The model equations were first transformed to obtain the basic reproduction number Ro. The Disease-Free Equilibrium (DFE) and Endemic Equilibrium (EE) are obtained, and the equilibrium of differential systems free from the disease is stable when Ro < 1 and unstable otherwise. The findings indicated that quarantine, hospitalization of infected individuals in the human population, and diagnosis help to maintain a low disease transmission rate in reducing the Basic reproduction number of monkeypox to have a value of 0.9338.

INTRODUCTION
Particularly near tropical rainforests, in remote areas of sub-Saharan Africa, outbreaks of monkeypox, a major viral zoonotic disease, sporadically arise. This disease is caused by the Poxviridae family's monkeypox, under the category of poxviruses known as Orthopoxviruses (Jezek *et al.* 1988). First used in 1958, the name "monkeypox" came from two outbreaks of infections akin to those of the pox observed in populations of monkeys under use for research (Kaler *et al.* 2022). Though the Democratic Republic of Congo recorded the first human case in 1970, smallpox eradication activities generated significant interest in it. It affects a broad spectrum of mammals found in a specific geographical location, mainly involving bodily fluids such as blood, mucosal skin sores, respiratory droplets, and even sexual contact; transmission of the virus from person to human mainly results from direct contact with infected individuals(Guarner et al., 2022). Human-to-animal transmission can be transferred by direct physical contact; animal-to-human transmission occurs by bites from infected animals or by the ingestion of unprocessed bush meat and contaminated food by rodents, including rats, dogs, cats, monkeys, and squirrels. The virus can be transmitted to an animal through respiratory droplets and contact from skin to skin if a person with the virus comes into close range to the animal, particularly if the individual has exposed wounds or skin lesions. Small virus-containing droplets may be expelled into the air when an infected individual talks, coughs, or sneezes. An animal close by inhales these droplets and becomes afflicted. The time usually for the development of monkeypox is ten to fourteen days, with a mean of twelve days. However, the length of time varies according to the type of host. According to clinical studies, for infected black dogs is four to thirteen days, and for dogs in the prairie is eleven to eighteen days. Patients typically experience a mild fever, muscle aches, fatigue, headaches, chills, and backaches during the 7–14-day incubation period (Hutin et al., 2001). Patients frequently develop the characteristic rash, which begins on the face and continues throughout the body three days after developing a fever and chills.

In 2017, there was a widespread epidemic in Nigeria, with multiple states reporting confirmed cases. The bulk of those affected were men aged between 21 and 40. On September 20, 2017, the state of Bayelsa reported an outbreak of human monkeypox, with 196 suspected cases. Numerous organisations, including the Nigeria Centre for Disease Control (NCDC) National Reference Laboratory, the Institute Pasteur de Dakar, the United States Centres for Disease Control and Prevention (US CDC) in Atlanta, and the WHO Collaborating Centre for Orthopox Viruses, conducted laboratory investigations (WHO, 2017). (NCDC 2022), Nigeria Centre for Disease Control reports that between September 2017 and June 26, 2022, 716 cases and nine fatalities were confirmed across 25

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states. By April 13, 2023, the CDC Centre for Disease Control and Prevention had confirmed 86,956 cases worldwide (CDC 2023). Nigeria tops the African case count with 829 cases, followed by the Democratic Republic of the Congo with 439 confirmed cases. As of 2023, reports from the CDC and WHO indicate that there have been 30,347 confirmed cases in the United States, making it the nation with the highest number of cases worldwide. In order to investigate the epidemiological characteristics, transmission patterns, and potential impact of this newly discovered and reemerging infectious disease on worldwide public health, researchers and health organizations have turned to mathematical modeling. The World Health Organization, on July 23, 2022, declared monkeypox to be of international Concern to Public Health (PHEIC) (WHO 2022). One contributing factor to the limited understanding of this disease lies in the transmission mechanisms, which are poorly understood. The dynamics of monkeypox transmission among human and animal populations, the potential impact of Isolation for individuals with mild complications and hospitalization for severe cases on reducing disease transmission, the role of diagnosis in controlling monkeypox spread, and the interplay between human and rodent transmission dynamics was explored.

Existing research has explored various mathematical modeling approaches to understand monkeypox transmission. Bhunu and Mushayabasa (2011) demonstrated that through carefully designed treatment intervention, the disease is poised to be eliminated not only from human subjects but also from non-human primates. They only considered the primary mode without any intervention strategy. The SIR model by Bhunu and Mushayabasa was studied and expanded to include a scenario in which there are more than two populations, and the contact rate is a function of time rather than just a constant by finding the global asymptotical stability of the endemic equilibrium, which has been previously uncompleted (TeWinkel 2019). They used fractional calculus to study the nature of the monkeypox virus using actual data from Nigeria, and they debated the modeling structure by analyzing preventive measures that would help the general public understand better. Considering those who are subpopulation's mix of exposed and secluded areas as well as the effects of that interaction rate with the number of rodents, they investigated the several elements that might lead to a decrease in disease transmission and the effects of such factors on the fundamental reproduction number. (Peter *et al.* 2021) and (Maysaa et al. 2022) analyzed a novel fractional framework characterizing the Monkey-pox model under fractional-order differential operators depending on the generalised Mittag-Leffler (GML) kernel employing the Atangana–Baleanu in the Caputo environment. They investigated and verified the system's equilibrium conditions in order of robustness. The global stability of the endemic equilibrium is solved with the use of Jacobian matrix techniques and the Routh-Hurwitz threshold. Furthermore, they implement a fresh approach combining the two-step Lagrange polyn with the fundamental concept of fractional calculus. Multiple fractional order numerical simulations reveal that the fractional order drops from 1 reduces the virus's proliferation. They didn't include hospitalization and other modes of transmission that could aid in curtaining the disease. Further work by Samuel and Joseph (2023) proposed in order to investigate the virus's human-to-human transmission in Ghana, Atangana-Baleanu fractional-order derivatives were defined in the Caputo sense. The model employing this operator made use of the Atangana-Baleanu and Caputo derivatives, which included a nonlocal and nonsingular kernel and (Maria et al. 2023) studied cases were men who have sex with men (MSM), and it was examined if the decrease in monkeypox cases was related to a decrease in the number of susceptible males (MSM). They created a framework for the transmission and matched it to data on behavioural changes, such as fewer casual partners and a shorter period when cases of monkeypox in the Netherlands refrain from contact. Their model only considered the transmission of the virus among the human population and failed to include any intervention strategies.

While existing models focus primarily on human-to-human transmission, the inclusion of human-to-rodent dynamics provides a more comprehensive understanding of potential zoonotic reservoirs. This paper aims to fill this gap by modeling both transmission pathways and the basic reproduction number impact on the intervention strategies.

MATERIALS AND METHODS

Mathematical model and formulation

The model was divided into a system of ordinary differential equations with nine different compartments consisting of both human and rodent populations.

The Susceptible Human Compartment S represents the total count of people in the population who could potentially contract Mpox. It rises in number by λ and decreases by μ (natural death), $\frac{\beta_r I_r + \beta_h I_h}{N_h}$ (Susceptible human who contracted the virus by means of interaction with an infected rodent and a susceptible human with an infected human).

$$\frac{dS_h}{dt} = \lambda_h - \frac{\beta_r I_r + \beta_h I_h}{N_h} - \mu_h$$

The human Compartment E is showing the virus's infecting count of people representing the number of individuals infected by the virus but are asymptomatic (Infectious disease necessarily passes through this phase). It increases by $\frac{\beta_r I_r + \beta_h I_h}{N_h}$. , decreases by μ (natural death) and $\theta_1 E_h$ (the rate at which healthy persons become infected).

$$\frac{dE_h}{dt} = \frac{\beta_r I_r + \beta_h I_h}{N_h} - (\theta_1 + \mu_h)E_h$$

The Infected human Compartment I represent the total count of people infected with the virus and are now symptomatic. It increases by $\theta_1 E_h$ and decreases by μ (natural death) and, δ (death caused by the disease), θ_2 (The rate at which individuals who had serious complication from the disease and are moved to the hospital on admission), θ_3 (The rate at which individual with mild complication are being quarantine) and τ (diagnosis of infected individual parameter).

$$\frac{dI_h}{dt} = \theta_1 E_h - (\theta_2 + \theta_3 + \mu_h + \delta_h + \tau) I_h$$

The Isolated human Compartment Q represent the total count of people being isolated with a contact rate θ_3, decreases by μ (natural death) and with a recovery rate θ_5

$$\frac{dQ_h}{dt} = \theta_3 I_h - (\theta_5 + \mu_h + \delta_h) Q_h$$

The Human Compartment H is representing the number of individuals who are being treated in the hospital with a contact rate ofθ_2, decreases by μ (natural death) and with a recovery rate θ_4.

$$\frac{dH_h}{dt} = \theta_2 I_h - (\theta_4 + \delta_h + \mu_h) H_h$$

The human Compartment R is representing the number of individuals who have recovered from the hospital at the rate θ_4, recovered from Isolation at the rate θ_5 and decreases by μ (natural death).

$$\frac{dR_h}{dt} = (\theta_4 H_h + \theta_5 Q_h) - \mu_h R_h$$

Rodent population

The Susceptible rodent Compartment S represent the total count of rodent in the population who could potentially contract Mpox. It rises in number by λ_r and decreases by μ (natural death), $\frac{\beta_1 I_r + \beta_2 I_h}{N_r}$ (Susceptible rodent who contracted the virus by means of interaction with an infected human and a susceptible rodent who contracted the infection via coming into touch with an infected rodents).

$$\frac{dS_r}{dt} = \lambda_r - \frac{\beta_1 I_r + \beta_2 I_h}{N_r} - \mu_r$$

The rodent Compartment E is showing the virus's infecting count of rodent representing the number of rodent infected by the virus. It increases by $\frac{\beta_1 I_r + \beta_2 I_h}{N_r}$, decreases by μ (natural death) and γ_1 (the rate at which exposed rodents become infected).

$$\frac{dE_r}{dt} = \frac{\beta_1 I_r + \beta_2 I_h}{N_r} - (\mu_r + \gamma_1) E_r$$

The rodent Compartment I Compartment I represent the total count of rodent infected with the virus. It increases by the contact rate of $\gamma_1 E_h$ and decreases by μ (natural death).

$$\frac{dI_r}{dt} = \gamma_1 E_r - (\mu_r + \delta_r + \gamma_2) I_r$$

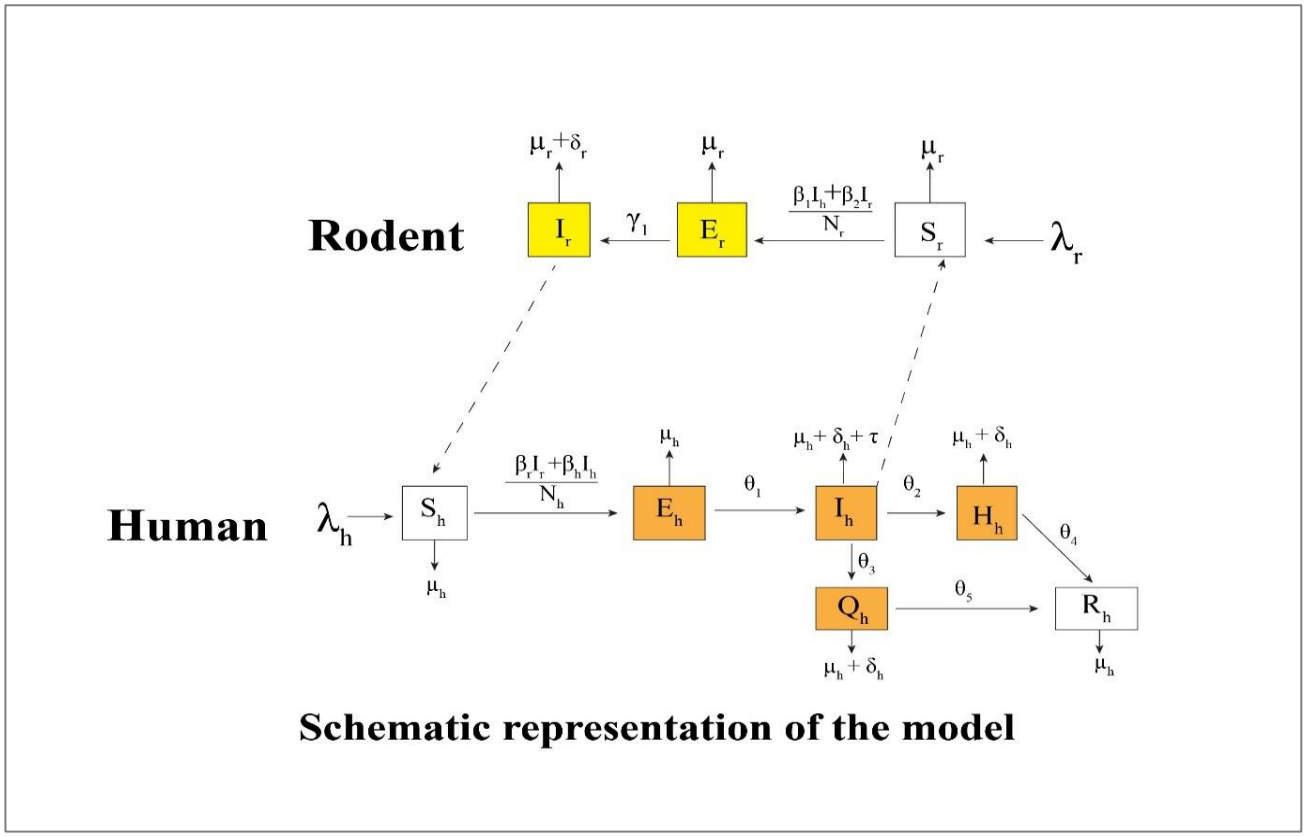

Figure 1. Monkeypox model

The Model's Basic Assumptions

The main presumptions that went into the construction of this representation are as follows:

I. The virus is expected to spread since the demographics of people and rodents are equally mixed. (Lioyd-Smith et al. 2005)

II. There is no available vaccination for the model, but Isolation, diagnosis, and hospitalization are assumed to be intervention measures.

The following system of equations (1-9) are obtained from the model schematic diagram in Figure 1.

Model diagram

The flow diagram in Figure 1 illustrates the state changes of humans and rodents in a population over time, represented by solid lines and their interactions by dot lines.

Mathematical formulations

The schematic representation of the differential equation is given as:

$$\frac{dS_h}{dt} = \lambda_h - \frac{\beta_r I_r + \beta_h I_h}{N_h} - \mu_h \quad (1)$$

$$\frac{dE_h}{dt} = \frac{\beta_r I_r + \beta_h I_h}{N_h} - (\theta_1 + \mu_h)E_h \quad (2)$$

$$\frac{dI_h}{dt} = \theta_1 E_h - (\theta_2 + \theta_3 + \mu_h + \delta_h + \tau)I_h \quad (3)$$

$$\frac{dQ_h}{dt} = \theta_3 I_h - (\theta_5 + \mu_h + \delta_h)Q_h \quad (4)$$

$$\frac{dH_h}{dt} = \theta_2 I_h - (\theta_4 + \delta_h + \mu_h)H_h \quad (5)$$

$$\frac{dR_h}{dt} = (\theta_4 H_h + \theta_5 Q_h) - \mu_h R_h \quad (6)$$

$$\frac{dS_r}{dt} = \lambda_r - \frac{\beta_1 I_r + \beta_2 I_h}{N_r} - \mu_r \quad (7)$$

$$\frac{dE_r}{dt} = \frac{\beta_1 I_r + \beta_2 I_h}{N_r} - (\mu_r + \gamma_1)Er \quad (8)$$

$$\frac{dI_r}{dt} = \gamma_1 E_r - (\mu_r + \delta_r + \gamma_2)I_r \quad (9)$$

Table 1. The overview of the model's variable used in the model

Variables	Description
$N_h(t)$	Total population of human compartment
$S_h(t)$	Susceptible human
$E_h(t)$	Exposed human
$I_h(t)$	Infected human
$H_h(t)$	Hospitalized human class
$Q_h(t)$	Isolated human class
$R_h(t)$	Recovered human class
$N_r(t)$	Total population of rodent compartment
$S_r(t)$	Susceptible rodent
$E_r(t)$	Exposed rodent
$I_r(t)$	Infected rodent

Table 2. The parameters description of the model

Parameters	Descriptions
λ_h	Reproduction rate of the human
λ_r	Population of the species of rodent
$\beta_r I_r$	Rate at which infections spread from diseased rodents to human
$\beta_h I_h$	Rate from infectious individuals to healthy ones
θ_1	Rate at which the exposed humans are moving to the infected class
θ_2	Rate at which human with severe symptoms are hospitalized
θ_3	Rate at which human with milder symptoms are being isolated
θ_4	Rate of recovery of human from hospitalization
θ_5	Rate of recovery of human from Isolation
$\beta_1 I_h$	Transmission rate from infected humans to susceptible rodents
$\beta_2 I_r$	Transmission rate from infected rodents to susceptible rodents
γ_1	Rate at which the exposed rodents are moving to the infected class
τ	Diagnosis of infected human
μ_h, μ_r	Natural death rate for humans and rodents
δ_h, δ_r	Death due to the disease for humans and rodents

MODEL ANALYSIS

Existence and Boundedness of solutions

Human Compartment

Positively invariant is the set $\Omega = \{(S, E, I, H, Q, R) \in IR6+)\}$: $0 \leq S+E+I+H+Q+R \leq \frac{\pi}{\mu}$ under system of equation (1-9) having a starting point ≥ 0.

Proof:

Adding the equations in human compartment 2.2

$$\frac{dN(t)}{dt} = \frac{dS(t)}{dt} + \frac{dE(t)}{dt} + \frac{dI(t)}{dt} + \frac{dH(t)}{dt} + \frac{dQ(t)}{dt} + \frac{dR(t)}{dt}$$

$$= \lambda_h - \mu_h N_h - \delta_h (I + \tau + H + Q + R) \leq \lambda_h - \mu_h N_h$$

$$\frac{dN(t)}{dt} \leq \lambda_h - \mu_h N_h$$

$$\frac{dN(t)}{dt} + \mu_h N_h \leq \lambda_h$$

Using the integrating factor (I.F) = $e^{\mu t}$

$$e^{\mu t}\frac{dN(t)}{dt} + e^{\mu t}\mu_h N_h \leq e^{\mu t}\lambda_h$$

$$\frac{d}{dt}(N(t)e^{\mu t}) \leq e^{\mu t}\lambda_h$$

Integrating with respect to t, we have

$$\int \frac{d}{dt}(N_h(t)e^{\mu t})dt \leq \lambda_h \int e^{\mu t}dt$$

$$N_h(t)e^{\mu t} \leq \frac{\lambda_h}{\mu_h}e^{\mu t} + C$$

$$N_h(t) \leq e^{-\mu t}(\frac{\lambda_h}{\mu_h}e^{\mu t} + C)$$

$$\Rightarrow N_h(t) \leq \frac{\lambda_h}{\mu_h} + Ce^{-\mu t}$$

At t= 0,

$$N_h(0) \leq \frac{\lambda_h}{\mu_h} + C$$

$$N_h(0) - \frac{\lambda_h}{\mu_h} \leq C$$

$$\Rightarrow N_h(t) \leq \frac{\lambda_h}{\mu_h} + (N(0) - \frac{\lambda_h}{\mu_h})e^{-\mu t}$$

$$N_h(t) \leq \frac{\lambda_h}{\mu_h} + N(0)e^{-\mu t} - \frac{\lambda_h}{\mu_h}e^{-\mu t}$$

Taking limit as $t \longrightarrow \infty$

$$0 \leq N(t) \leq \frac{\lambda_h}{\mu_h} \quad (10)$$

Therefore, the solution of equation (1 – 6) with non-negative initial value are bounded and exist within $[0, +\infty)$

Rodent Compartment

The set $\Omega = \{(S, E, I) \in IR3+)\}$: $0 \leq S+E+I \leq \frac{\lambda_r}{\mu_r}$ is positively invariant under system of equation (7-9) with initial conditions $S(0) \geq 0$, $E(0) \geq 0$ and $I(0) \geq 0$

Proof:

Adding the equations in rodent compartment 2.2,

$$\frac{dNr(t)}{dt} = \frac{dS_r(t)}{dt} + \frac{dE_r(t)}{dt} + \frac{dI_r(t)}{dt}$$

$$= \lambda_r - \mu_r Nh - \delta r(Er+Ir) - \gamma_2 I_r \leq \lambda_r - \mu_r Nr$$

$$\frac{dN(t)}{dt} \leq \lambda_r - \mu_r Nr$$

$$\Rightarrow \frac{dN(t)}{dt} + \mu_r Nr \leq \lambda_r$$

Using the integrating Factor (I.F) = $e^{\mu_r t}$

$$\Rightarrow e^{\mu_r t}\frac{dN(t)}{dt} + e^{\mu_r t}\mu_r N_r \leq e^{\mu_r t}\lambda_r$$

$$\frac{d}{dt}(Nr(t)\, e^{\mu_r t}) \leq e^{\mu_r t}\lambda_r$$

Integrating with respect to t,

$$\int \frac{d}{dt}(N_r(t)e^{\mu_r t})dt \leq \lambda_r \int e^{\mu_r t}dt$$

$$\Rightarrow Nr(t)\, e^{\mu_r t} \leq \frac{\lambda_r}{\mu_r}e^{\mu_r t} + C$$

$$Nr(t) \leq e^{-\mu_r t}(\frac{\lambda_r}{\mu_r}e^{\mu_r t} + C)$$

$$\Rightarrow Nr(t) \leq \frac{\lambda_r}{\mu_r} + Ce^{-\mu_r t}$$

At t=0

$$N(0) \leq \frac{\lambda_r}{\mu_r} + C$$

$$N(0) - \frac{\lambda_r}{\mu_r} \leq C$$

$$\Rightarrow Nr(t) \leq \frac{\lambda_r}{\mu_r} + (N(0) - \frac{\theta_r}{\mu_r})e^{-\mu_r t}$$

$$Nr(t) \leq \frac{\lambda_r}{\mu_r} + (N(0)\, e^{-\mu_r t} - \frac{\lambda_r}{\mu_r}e^{-\mu_r t}$$

Taking limit as $t \longrightarrow \infty$

$$0 \leq Nr(t) \leq \frac{\lambda_r}{\mu_r} \quad (11)$$

Therefore, the solution of system (7 – 9) with non-negative initial value are bounded and exist within $[0, +\infty)$

Positivity of solutions

If $S_h(0) \geq 0$, $E_h(0) \geq 0$, $I_h(0) \geq 0$, $H_h(0) \geq 0$, $Q_h(0) \geq 0$, $R_h(0) \geq 0$, $S_r(0) \geq 0$, $E_r(0) \geq 0$ and $I_r(0) \geq 0$ then the solutions $S_h(t)$, $E_h(t)$, $I_h(t)$, $H_h(t)$, $Q_h(t)$, $R_h(t)$, $S_r(t)$, $E_r(t)$ and $I_r(t)$ are all positive with every $t \geq 0$

Proof

$$\frac{dS_h(t)}{dt} = \lambda_h - \left(\frac{\beta_r I_r + \beta_h I_h}{N_h}\right) S_h - \mu_h S$$

$$\frac{dS_h(t)}{dt} = \lambda_h - S_h \left(\frac{\beta_r I_r + \beta_h I_h}{N_h} + \mu_h\right)$$

Let $G(t) = \frac{\beta_r I_r + \beta_h I_h}{N_h} + \mu_h$

$$\frac{dS_h(t)}{dt} = \lambda_h - G(t)S_h(t)$$

$$\Rightarrow \frac{dS_h(t)}{dt} + G(t)S_h(t) \geq \lambda_h$$

Using the Integrating factor, we have I. F= $e^{\int_0^t G(s)ds}$

$$e^{\int_0^t G(s)ds}\frac{dS_h(t)}{dt} + e^{\int_0^t G(s)ds} G(t)S_h(t) \geq 0$$

$$\Rightarrow \frac{d}{dt}(S_h(t)\, e^{\int_0^t G(s)ds}) \geq 0$$

$$\int_0^t \frac{d}{ds}(S_h(s) e^{\int_0^t G(s)ds}) \geq 0$$

$$\Rightarrow S_h(t)\, e^{\int_0^t G(s)ds} - S_h(0) \geq 0$$

$$S_h(t) \geq S_h(0) e^{-\int_0^t G(s)ds}$$

$$\Rightarrow S_h(t) \geq 0 \quad (12)$$

$$\frac{dE_h(t)}{dt} = \frac{\beta_r I_r + \beta_h I_h}{N_h} - (\theta_1 + \mu_h)E_h \geq -(\theta_1 + \mu_h) E_h$$

Let $F = \theta_1 + \mu_h$

$$\Rightarrow \frac{dE_h(t)}{dt} + F(t)E(t) \geq 0$$

Using Integrating factor, we have I.F= $e^{\int_0^t F(s)ds}$

$$e^{\int_0^t F(s)ds}\frac{dE_h(t)}{dt} + e^{\int_0^t F(s)ds} F(t)E(t) \geq 0$$

$$\Rightarrow \frac{d}{dt}(E_h(t)\, e^{\int_0^t F(s)ds}) \geq 0$$

$$\int_0^t \frac{d}{ds}(E_h(s) e^{\int_0^t F(s)ds}) \geq 0$$

$$\Rightarrow E_h(t)\, e^{\int_0^t F(s)ds} - E_h(0) \geq 0$$

$$\Rightarrow E_h(t) \geq 0 \quad (13)$$

$$\frac{dI_h(t)}{dt} = \theta_1 E_h - (\theta_2 + \theta_3 + \mu_h + \delta_h + \tau)I_h \geq -(\theta_2 + \theta_3 + \mu_h + \delta_h + \tau)I_h$$

Let $M = \theta_2 + \theta_3 + \mu_h + \delta_h + \tau$

$$\Rightarrow \frac{dI_h(t)}{dt} + M(t)I(t) \geq 0$$

Using Integrating factor, we have I. F= $e^{\int_0^t M(s)ds}$

$$e^{\int_0^t M(s)ds}\frac{dI_h(t)}{dt} + e^{\int_0^t M(s)ds} M(t)I(t) \geq 0$$

$$\Rightarrow \frac{d}{dt}(I_h(t)\, e^{\int_0^t M(s)ds}) \geq 0$$

$$\int_0^t \frac{d}{ds}(I_h(s) e^{\int_0^t M(s)ds}) \geq 0$$

$$\Rightarrow I_h(t)\, e^{\int_0^t M(s)ds} - I(0) \geq 0$$

$$I_h(t) \geq I_h(0) e^{-\int_0^t M(s)ds}$$

$$\Rightarrow I_h(t) \geq 0 \quad (14)$$

$$\frac{dH_h(t)}{dt} = \theta_2 I_h - (\theta_4 + \delta_h + \mu_h)H_h \geq -(\theta_4 + \delta_h + \mu_h)H_h$$

Let $Y = \theta_4 + \delta_h + \mu_h$

$$\Rightarrow \frac{dH_h(t)}{dt} + Y(t)H(t) \geq 0$$

Using Integrating factor, we have I.F= $e^{\int_0^t Y(s)ds}$

$$e^{\int_0^t Y(s)ds}\frac{dH_h(t)}{dt} + e^{\int_0^t Y(s)ds} M(t)H(t) \geq 0$$

$$\Rightarrow \frac{d}{dt}(H_h(t)\, e^{\int_0^t Y(s)ds}) \geq 0$$

$$\int_0^t \frac{d}{ds}(H_h(s) e^{\int_0^t Y(s)ds}) \geq 0$$

$$\Rightarrow H_h(t)\, e^{\int_0^t Y(s)ds} - H_h(0) \geq 0$$

$$H_h(t) \geq H_h(0) e^{-\int_0^t Y(s)ds}$$

$$\Rightarrow H_h(t) \geq 0 \quad (15)$$

$$\frac{dQ_h(t)}{dt} = \theta_3 I_h - (\theta_5 + \mu_h + \delta_h)Q_h \geq -(\theta_5 + \mu_h + \delta_h)Q_h$$

Let $J = \theta_5 + \mu_h + \delta_h$

$$\Rightarrow \frac{dQ_h(t)}{dt} + J(t)Q(t) \geq 0$$

Using Integrating factor, we have I.F= $e^{\int_0^t J(s)ds}$

$$e^{\int_0^t J(s)ds}\frac{dQ_h(t)}{dt} + e^{\int_0^t J(s)ds} J(t)Q(t) \geq 0$$

$$\Rightarrow \frac{d}{dt}(Q_h(t)\, e^{\int_0^t J(s)ds}) \geq 0$$

$$\int_0^t \frac{d}{ds}(Q_h(s) e^{\int_0^t J(s)ds}) \geq 0$$

$$\Rightarrow Q(t)\, e^{\int_0^t J(s)ds} - Q(0) \geq 0$$

$$Q(t) \geq Q(0) e^{-\int_0^t J(s)ds}$$

$$\Rightarrow Q(t) \geq 0 \quad (16)$$

$$\frac{dR_h(t)}{dt} = (\theta_4 H_h + \theta_5 Q_h) - \mu_h R_h \geq -\mu_h R_h$$

$$\Rightarrow \frac{dQ_h(t)}{dt} + \mu_h(t)R_h(t) \geq 0$$

Using Integrating factor, we have I.F= $e^{\int_0^t \mu_h(s)ds}$

$e^{\int_0^t \mu_h(s)ds}\frac{dR_h(t)}{dt} + e^{\int_0^t \mu_h(s)ds}\,\mu_h(t)R(t) \geq 0$

$\Longrightarrow \frac{d}{dt}(R_h(t)\,e^{\int_0^t \mu_h(s)ds}) \geq 0$

$\int_0^t \frac{d}{ds}(R_h(s)e^{\int_0^t \mu_h(s)ds}) \geq 0$

$\Longrightarrow R(t)\,e^{\int_0^t \mu_h(s)ds} - R(0) \geq 0$

$R(t) \geq R(0)e^{-\int_0^t \mu_h(s)ds}$

$\Longrightarrow R(t) \geq 0$ (17)

$\frac{dS_r}{dt} = \theta_r - (\frac{\beta_1 I_r + \beta_2 I_h}{N_r})S - \mu_r S$

$\frac{dS_h(t)}{dt} = \theta_r - S_r(\frac{\beta_1 I_r + \beta_2 I_h}{N_r} + \mu_r)$

Let $P(t) = \frac{\beta_1 I_r + \beta_2 I_h}{N_r} + \mu_h$

$\frac{dS_h(t)}{dt} = \theta_r - P(t)S_r(t)$

$\Longrightarrow \frac{dS_r(t)}{dt} + P(t)S_r(t) \geq 0$

Using the Integrating factor, we have I.F= $e^{\int_0^t P(s)ds}$

$e^{\int_0^t P(s)ds}\frac{dS_r(t)}{dt} + e^{\int_0^t P(s)ds}\,P(t)S_r \geq 0$

$\Longrightarrow \frac{d}{dt}(S_r(t)\,e^{\int_0^t P(s)ds}) \geq 0$

$\int_0^t \frac{d}{ds}(S_r(s)e^{\int_0^t P(s)ds}) \geq 0$

$\Longrightarrow S_r(t)\,e^{\int_0^t P(s)ds} - S(0) \geq 0$

$S_r(t) \geq S_r(0)e^{-\int_0^t P(s)ds}$

$\Longrightarrow S_r(t) \geq 0$ (18)

$\frac{dE_r(t)}{dt} = \frac{\beta_1 I_r + \beta_2 I_h}{N_r} - (\gamma_1 + \mu_r)E_r \geq -(\gamma_1 + \mu_r)E_r$

Let $N = \gamma_1 + \mu_h$

$\Longrightarrow \frac{dE_r(t)}{dt} + N(t)\,E_r(t) \geq 0$

Using Integrating factor, we have I.F= $e^{\int_0^t N(s)ds}$

$e^{\int_0^t N(s)ds}\frac{dE_r(t)}{dt} + e^{\int_0^t N(s)ds}\,N(t)E_r(t) \geq 0$

$\Longrightarrow \frac{d}{dt}(E_r(t)\,e^{\int_0^t N(s)ds}) \geq 0$

$\int_0^t \frac{d}{ds}(E_r(s)e^{\int_0^t N(s)ds}) \geq 0$

$\Longrightarrow E_r(t)\,e^{\int_0^t N(s)ds} - E_r(0) \geq 0$

$E_r(t) \geq E_r(0)e^{-\int_0^t F(s)ds}$

$\Longrightarrow E_r(t) \geq 0$ (19)

$\frac{dI_r(t)}{dt} = \gamma_1 E_r - (\mu + \delta + \gamma_2)I \geq -(\mu + \delta + \gamma_2)I$

Let $V = \mu + \delta + \gamma_2$

$\Longrightarrow \frac{dI_r(t)}{dt} + V(t)I(t) \geq 0$

Using Integrating factor, we have I.F= $e^{\int_0^t V(s)ds}$

$e^{\int_0^t V(s)ds}\frac{dI_r(t)}{dt} + e^{\int_0^t V(s)ds}\,V(t)I(t) \geq 0$

$\Longrightarrow \frac{d}{dt}(I_r(t)\,e^{\int_0^t V(s)ds}) \geq 0$

$\int_0^t \frac{d}{ds}(I_r(s)e^{\int_0^t V(s)ds}) \geq 0$

$\Longrightarrow I_r(t)\,e^{\int_0^t V(s)ds} - I(0) \geq 0$

$I_r(t) \geq I_r(0)e^{-\int_0^t V(s)ds}$

$\Longrightarrow I_r(t) \geq 0$ (20)

then the solutions $S_h(t)$, $E_h(t)$, $I_h(t)$, $H_h(t)$, $Q_h(t)$, $R_h(t)$, $S_r(t)$, $E_r(t)$ and $I_r(t)$ are all positive for all $t \geq 0$

Equilibrium Point Analysis

These points are generated by setting the equations (1-9) above to zero, which will absolutely yield Equilibrium of Differential Systems Free from Diseases and the endemic equilibrium (DEE). At equilibrium states we have, $\frac{dS_h}{dt} = \frac{dSE_h}{dt} = \frac{dIS_h}{dt} = \frac{dH_h}{dt} = \frac{dQ_h}{dt} = \frac{dR_h}{dt} = \frac{dS_r}{dt} = \frac{dE_r}{dt} = \frac{dI_r}{dt} = 0$

Let $\alpha_h = \frac{\beta_r I_r + \beta_h I_h}{N_h}$ and $\alpha r = \frac{\beta_1 I_r + \beta_2 I_h}{N_r}$

Therefore:

$\lambda_h - (\alpha_h + \mu)S_h = 0$

$\alpha_h Sh - (\theta_1 + \mu_h)E_h = 0$

$\theta_1 E_h - (\theta_2 + \theta_3 + \mu_h + \delta_h + \tau)I_h = 0$

$\theta_2 I_h - (\theta_4 + \delta_h + \mu_h)H_h = 0$

$\theta_3 I_h - (\theta_5 + \mu_h + \delta_h)Q_h = 0$

$(\theta_4 H_h + \theta_5 Q_h) - \mu_h R_h = 0$

$\lambda_r - (\alpha r + \mu_r)S_r = 0$

$\alpha r S_r - (\mu + \gamma_1)Er = 0$

$\gamma_1 E_r - (\mu_r + \delta_r)I_r = 0$ (21)

Equilibrium of Differential Systems Free from Diseases

The equilibrium of Differential Systems Free from Diseases refers to the complete absence of a disease in the population. We consider all disease compartments to be zero. Therefore, we have $I_h = I_h = H_h = Q_h = E_r = I_r = 0$. On putting these terms in equation (13) and solving simultaneously, we get the Equilibrium of differential systems free from the disease in the model.

$$S_h = \frac{\lambda_h}{\mu_h}, E_h = 0, I_h = 0, Q_h = 0, H_h = 0, R_h = 0, S_r = \frac{\lambda_r}{\mu_r}, E_r = 0, I_r = 0 \quad (22)$$

Endemic equilibrium of the population

When disease cannot be eliminated but still exists in the population, it is said to be an endemic equilibrium state. At this point, monkeypox is present in the susceptible population. Since there are both humans and rodents who are prone to being exposed to the disease, all compartments in the model will be taken into account in this scenario. If it is the endemic equilibrium state, then $E_h = I_h = H_h = Q_h = E_r = I_r \neq 0$ while taking into account the outcome for each compartment in consideration as follows:

$$S_h^* = \frac{\lambda_h}{\alpha_h + \mu_h} \quad (23)$$

$$E_h^* = \frac{\alpha_h \lambda_h}{(\lambda_h + \mu)(\theta_1 + \mu_h)} \quad (24)$$

$$I_h^* = \frac{\theta_1 \alpha_h \lambda_h}{(\lambda_h + \mu)(\theta_1 + \mu_h)(\theta_2 + \theta_3 + \mu_h + \delta_h + \tau_h)} \quad (25)$$

$$H_h^* = \frac{\theta_1 \alpha_h \lambda_h \theta_2}{(\lambda_h + \mu)(\theta_1 + \mu_h)(\theta_2 + \theta_3 + \mu_h + \delta_h + \tau_h)(\theta_4 + \delta + \mu)} \quad (26)$$

$$Q_h^* = \frac{\theta_1 \alpha_h \lambda_h \theta_3}{(\lambda_h + \mu)(\theta_1 + \mu_h)(\theta_2 + \theta_3 + \mu_h + \delta_h + \tau_h)} \quad (27)$$

$$R_h^* = \frac{\theta_1 \alpha_h \lambda_h \theta_2 \Theta_4 + \theta_1 \alpha_h \lambda_h \theta_3 \theta_5}{\mu(\lambda_h + \mu)(\theta_1 + \mu_h)(\theta_2 + \theta_3 + \mu_h + \delta_h + \tau_h)(\theta_4 + \delta + \mu)(\lambda_h + \mu)(\theta_1 + \mu_h)(\theta_2 + \theta_3 + \mu_h + \delta_h + \tau_h)} \quad (28)$$

$$S_r^* = \frac{\lambda_r}{\alpha_r + \mu_r} \quad (29)$$

$$E_r^* = \frac{\lambda_r \alpha_r}{(\alpha_r + \mu_r)(\mu_r + \gamma_1)} \quad (30)$$

$$I_r^* = \frac{\gamma_1 \lambda_r \alpha_r}{(\alpha_r + \mu_r)(\mu_r + \gamma_1)(\mu_r + \delta_r)} \quad (31)$$

BASIC REPRODUCTION NUMBER

As Diekmann et al. (1990) pointed out, the Next Generation Matrix approach sees the Reproduction number (Ro) as the highest eigenvalue model.

Using the spectral radius ϱ, Ro= ϱ(FV-1.

Theorem 1

Examine the model of disease transmission provided by and see whether it meets the following presumptions $\frac{dx_i}{dt} = f_i(x) = \mathcal{F}_i(x) - \mathcal{V}_i(x), i = 1, \ldots, n, where\ \mathcal{V}_i = \mathcal{V}_i^- - \mathcal{V}_i^+$

should x ≥ 0, then (should a compartment be empty, there cannot be any individual transfer out of the compartment)

if $x_i = 0$, the incidence of infection for the non-infected compartment is zero.

$\mathcal{F}_i = 0\ if\ i > m.$ (The population is free of the disease)

If x_o is an Equilibrium of differential systems free from the disease in the model, then x_o is stable if Ro < 1, but unstable if Ro > 1, where Ro= ϱ(FV-1).

Proof

Define $Z_1 = F - V$. With V a nonsingular M-matrix and F non-negative, - $Z_1 = V - F$. This means,
$s(Z_1)$ constitutes a nonsingular M-matrix that displays the biggest true value among the matrix eigenvalues Z_1. - $Z_1V^{-1}=1$ - FV^{-1} ; FV^{-1} is nonnegative.
Z_1 is a non-singular M-matrix. FV^{-1} is non-singular in M-matrix form.
FV^{-1} is a non-negative hence all eigenvalues of FV^{-1} have either less magnitudes than $\varrho(FV^{-1})$.
FV^{-1} is a nonsingular M-matrix whose, ⇔ $\varrho(FV^{-1})$ is 1. $s(Z_1) < 0$ only hence if Ro < 1. Moreover, follows:
1 - FV^{-1} is a single M-matrix; $\varrho(FV^{-1}) = 1$. $s(Z_1) = 0$ ⇔ - Z_1

Using equation (1-9), the infection States of the model are E_h, I_h, H_h, Q_h, E_r and I_r. At Disease Free equilibrium, we have $(\frac{\lambda_h}{\mu_h}, 0,0,0,0,0, \frac{\lambda_r}{\mu_r}, 0,0)$ then the transmission matrix F and the Transition matrix V are given below.

$$F=\begin{bmatrix} \frac{\beta_r I_r+\beta_h I_h}{N_h} S_h \\ 0 \\ 0 \\ 0 \\ \frac{\beta_1 I_r+\beta_2 I_h}{N_r} S_r \\ 0 \end{bmatrix} \text{ and } V=\begin{bmatrix} (\theta_1+\mu_h)E_h \\ (\theta_2+\theta_3+\mu_h+\delta_h+\tau)I_h-\theta_1 E_h \\ (\theta_4+\delta_h+\mu_h)H_h-\theta_2 I_h \\ (\theta_5+\mu_h+\delta_h)Q_h-\theta_3 I_h \\ (\mu_r+\gamma_1)Er \\ (\mu_r+\delta_r)I_r-\gamma_1 E_r \end{bmatrix} \quad (32)$$

Then

$$F=\begin{bmatrix} 0 & \beta_h & 0 & 0 & 0 & \beta_r \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \beta_2 & 0 & 0 & 0 & \beta_1 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix} \text{ and } V=\begin{bmatrix} K_1 & 0 & 0 & 0 & 0 & 0 \\ -\theta_1 & K_2 & 0 & 0 & 0 & 0 \\ 0 & -\theta_2 & K_3 & 0 & 0 & 0 \\ 0 & -\theta_3 & 0 & K_4 & 0 & 0 \\ 0 & 0 & 0 & 0 & K_6 & 0 \\ 0 & 0 & 0 & 0 & -\gamma_1 & K_7 \end{bmatrix} \quad (33)$$

Where k1= $\theta_1+\mu_h$, k2=$(\theta_2+\theta_3+\mu_h+\delta_h+\tau)$, k3= $\theta_4+\delta_h+\mu_h$, k4= $\theta_5+\mu_h+\delta_h$, k6= $\mu_r+\gamma_1$, k7= $\mu_r+\delta_r$

$$V^{-1}=\begin{bmatrix} \frac{1}{K_1} & 0 & 0 & 0 & 0 & 0 \\ \frac{\theta_1}{K_1K_2} & \frac{1}{K_2} & 0 & 0 & 0 & 0 \\ \frac{\theta_1\theta_2}{K_1K_2K_3} & \frac{\theta_2}{K_2K_3} & \frac{1}{K_3} & 0 & 0 & 0 \\ \frac{\theta_1\theta_3}{K_1K_2K_4} & \frac{\theta_3}{K_2K_4} & 0 & \frac{1}{K_4} & 0 & 0 \\ 0 & 0 & 0 & 0 & \frac{1}{K_6} & 0 \\ 0 & 0 & 0 & 0 & \frac{\gamma_1}{K_6K_7} & \frac{1}{K_7} \end{bmatrix} \text{ Then, } FV^{-1}=\begin{bmatrix} \frac{\beta_h\theta_1}{K_1K_2} & \frac{\beta_h}{K_2} & 0 & 0 & \frac{\beta_r\gamma_1}{K_6K_7} & \frac{\beta_r}{K_7} \\ O & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ \frac{\beta_1\theta_1}{K_1K_2} & \frac{\beta_1}{K_2} & 0 & 0 & \frac{\beta_2\gamma_1}{K_6K_7} & \frac{\beta_2}{K_7} \\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix} \quad (34)$$

Now we have,

$$R0=\frac{1}{2}\frac{\beta_2\gamma_1K_1K_2+\beta_h\theta_1K_6K_7+\sqrt{4\beta_1\beta_r\gamma_1K_1K_2K_6K_7\theta_1+\beta_2^2\gamma_1^2K_1^2K_2^2-2\beta_2\beta_h\gamma_1K_1K_2K_6K_7\theta_1+\beta_h^2K_6^2K_7^2\theta_1^2}}{K_1K_2K_6K_7} \quad (35)$$

The Disease-free Equilibrium exhibits both locally asymptotically stability and instability when R0 < 1 and otherwise.

Where k1= $\theta_1+\mu_h$, k2=$(\theta_2+\theta_3+\mu_h+\delta_h+\tau)$, k3=$\theta_4+\delta_h+\mu_h$, k4= $\theta_5+\mu_h+\delta_h$, k6= $\mu_r+\gamma_1$, k7= $\mu_r+\delta_r$

SENSITIVITY ANALYSIS OF R_0

Using the standardized forward sensitivity index as the ratio of variable change to parameter change, sensitivity analysis evaluates variable relative change when parameter changes, it also evaluates, under a certain set of assumptions, the effects of varying values of an independent variable on a specific dependent (Kalyan et al., 2021). Partially derivative products may be used as an alternate definition of the awareness indicator when the variable is an identifiable component of the parameter in question. The factors that impact the dynamics of the monkeypox virus model are weighted based on their sensitivity indices, we can identify the most sensitive characteristics that significantly affect the basic reproduction and demonstrate how specific parameters can impact the growth or decline of the basic reproductive number. To obtain the sensitivity analysis of the basic reproductive numberR_0, we use the procedure in (Chitnis et al., 2008). The standardized forward sensitivity index of a variable ζ under the parameter υ can be defined through the following formula as,

$$S_\upsilon^\zeta=\frac{\partial\zeta}{\partial\upsilon}\text{ x }\frac{\upsilon}{\zeta} \quad (36)$$

Having considered the above formula (36), we can have the expression for R_0 as,

$$S_\upsilon^{R_0}=\frac{\partial R_0}{\partial\upsilon}\text{ x }\frac{\upsilon}{R_0} \quad (37)$$

For each parameters R_0 and their numerical values are given in Table 3 are used to get the sensitivity indices. We shall have the sensitivity index of R_0 with respect to the parameter μ_r which is as follows,

$$S_{\mu_r}^{R_0} = \frac{\partial R_0}{\partial \mu_r} \times \frac{\mu_r}{R_0} = -1.87524853710^{-7} \quad (38)$$

We use the above formula to obtain the sensitivity indices of the remaining 11 different parameters which include $\beta_h, \beta_2, \mu_h, \tau, \theta_1, \delta_h, \theta_3, \theta_2, \delta_r, \beta_r, \beta_1$.

Table 3. Parameters and their sensitivity indices

Parameters	Values	Sensitivity indices	Reference Value
μ_r	0.002	-1.87524853710^{-7}	Bhunu and Mushayabasa(2011)
β_h	0.000063	-1.73182805710^{-7}	Bhunu and Mushayabasa(2011)
μ_h	1.5	-0.4624230659	Bhunu and Mushayabasa(2011)
τ	2.0	0.1643704234	Assumed
θ_1	0.2	0.03866711564	Peter et al. (2022)
δ_h	0.2	0.01643704234	Odom et al. (2009)
θ_3	0.1375	0.01130046661	Assumed
θ_2	0.001	0.0001779936876	Assumed
β_2	0.027	0.009355014423	Bhunu and Mushayabasa(2011)
δ_r	0.4	0.03264884276	Emeka et al. (2018)
β_r	0.0025	0.00003916740274	Bhunu and Mushayabasa(2011)
β_1	0.075	0.00003916740274	Assumed

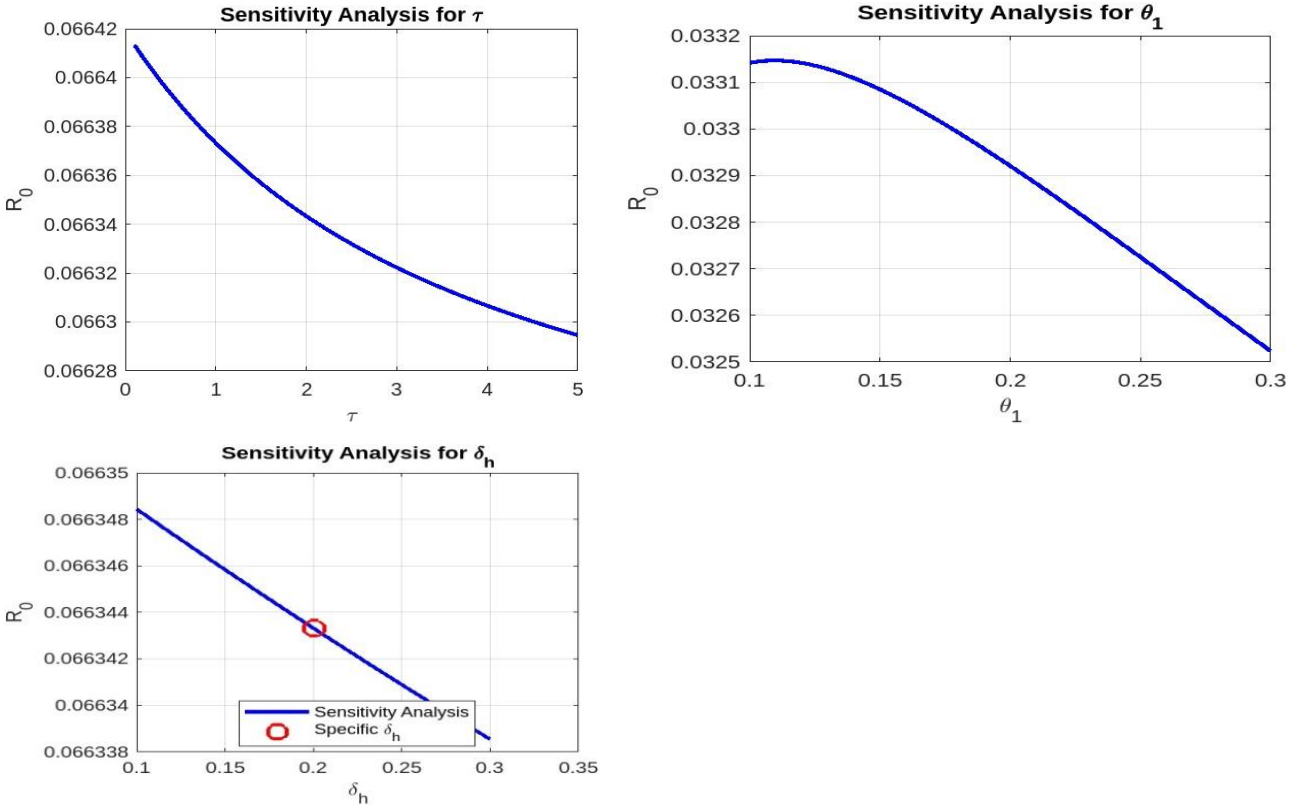

Figure 2. Sensitivity of some important parameters

RESULTS AND DISCUSSIONS

The essence of the formulated model in Figure 1 with the model equations (1 - 11) explains the transmission dynamics of monkeypox in the two population and their interaction as it relates to the variables and parameters in Table 1 and Table 2. This shows the reality of how monkeypox is being transmitted and takes into detail the incubation stage of both rodent and human populations and the rate at which the disease progresses therefore, the need for prevention and intervention. By the process of evaluation and derivation, we obtain the basic reproduction number as seen in equation (27). We use this as a key variable to determine if monkeypox can be eradicated or will continue to evade the population. The basic reproduction number is a threshold below which the generation of secondary cases is insufficient to maintain the spread of the monkeypox virus within the population. If the basic reproduction number is less than 1, then the number of infected humans and rodents will decrease from the population, and the monkeypox virus will die off;

should the count of those with the disease rise, the monkeypox virus will continue to spread (Driesche and Watmough, 2022). From the analysis of the model, it is clear that the model satisfies stability, positivity, drug-free equilibrium, and other properties. The model can also become endemic, as seen in equation (15 to 23). When the sensitivity indices provided in Table 3 are reviewed, the index of the natural death rate of rodents and the transmission rate from diseased humans to vulnerable humans is extremely near to zero. This means that their alterations have the least predictive power of the results of the model. It implies that changes in the population dynamics of rodents as a result of natural mortality may not be a more determinate of the expanding tendencies in the spread of the illness, and hence, this mechanism of transmission is not a more likely trend in the monkeypox disease dynamics. A rise in the natural death rate of humans is also a factor of the monkeypox disease.

The parameter of the transmission rate from diseased rats to humans, has a moderate impact on the model outputs. It implies that actions targeting this transmission channel may influence the overall spread of monkeypox. Monkeypox's spread is influenced in part by the speed at which infected humans infect rodents. A more important influence on the outcomes of the model is indicated by a higher positive sensitivity index for the parameter assessing the rate of transmission from infected to susceptible rodents. It shows that this transmission route considerably influences the dynamics of monkeypox. A positive sensitivity index clearly shows that the capacity of recovered infected rats to spread the infection determines the dynamics of monkeypox mostly. The death rate for infected people underlines the need of knowing how monkeypox affects the population. The death rate of infected rodents shows a greater positive sensitivity index when compared to the mortality rate of infected humans, therefore stressing the need of treating the disease's effect on the rodent population. Understanding the mechanics of transmission from exposed to infected individuals and changes in the rate at which exposed individuals become infected can help one to control the spread of monkeypox. Though it might not have as great of an influence as other intervention elements, the rate at which infected people are admitted to the hospital determines the general dynamics of monkeypox. The encouraging sensitivity index of quarantine individuals with modest problems is a key intervention tactic for stopping the spread of the disease. As the diagnosis of an infected person parameter shows, changes in the efficacy or precision of spotting infected people have the most impact on the model results. A timely and precise diagnosis is essential for both efficient therapy and control of monkeypox. The positive sensitivity index of Isolating persons with modest problems is an essential intervention technique in reducing the spread of the disease, as we can see from (Somma et al. 2019) how it reduces the spread of monkeypox in the population. Diagnosis of the infected person parameter has the highest positive sensitivity index, indicating that changes in the efficiency or accuracy of diagnosing infected individuals have the most substantial impact on the model outputs. Similar studies like that of (Huang et al. 2022) and (Peter et al. 2021) have shown that timely and correct laboratory diagnosis and Isolation of infected humans are critical for efficient care and control of monkeypox within a susceptible population and human-to-human interaction was the most sensitive parameter in their sensitivity analysis which further support the notion that diagnosis, hospitalization and Isolation help in reducing our model's basic reproduction number.

The basic reproduction number is sensitive to changes in diagnosis (τ), contact rate (θ_1), and death rate (δ_h). A diagnosis in disease progression leads to a reduction in the basic reproduction number, suggesting that it can reduce the spread of the disease. Initially increasing the transmission potential, higher contact rates later decline in response to intervention strategies. As death rates resulting from the illness increase, the sensitivity of the basic reproduction number falls linearly. Increasing diagnosis results in a reduction in R_o and with a slight increase of the basic reproduction number, which still reduces as the contact rate θ_1 keeps increasing shows that because of the intervention strategies, the possibility of transmission of new infection is reduced. Public health experts must improve on the understanding of the disease transmission, laboratory diagnosis, and prevention which can be achieved through awareness of diagnosis, Isolation, and hospitalization so as to curtail the spread of the disease within the population. The value of R_o also shows that public health must put in all effort in maintaining a low transmission rate, as also pointed out by (Allehiany et al. 2023), and also address any factor that could lead to an increase of disease transmission.

CONCLUSION

This study develop a mathematical model that has nine compartmental models developed using ordinary differential equations. When the fundamental reproductive number is smaller than unity, it was demonstrated that the model exhibited a drug-free equilibrium point that was locally asymptotically stable. This indicates that if the interventions are not adhered to, the disease will continue to evade human and rodent population as seen in equation (15 to 23). MATLAB was used to plot the graph in Figure 2, and Maple software was used in the sensitivity analysis which offers helpful information on the relative significance of various components in the transmission model of monkeypox. The diagnosis, rates of infection transmission for humans and rodents, rates of natural and disease-related mortality for humans and rodents, and the rates at which infected humans moved to the intervention strategies are all highly sensitive to the model (for instance, the rates of contacts from infected to quarantine demonstrate that individuals with robust immune systems can fend off the illness). It is evident that the implementation of a diagnosis strategy will significantly mitigate the transmission of monkeypox to both humans and rats; nonetheless, controlling the disease's spread within the rodent population will provide

challenges and so future research should investigate the role of vaccination and rodent control measures in reducing transmission. The impact of interventions related to these traits on stopping the disease's spread will be greater. Future research should investigate the role of vaccination and rodent control measures in reducing transmission among the rodent population.

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