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MONKEYPOX TRANSMISSION DYNAMICS IN A POPULATION OF MEN WHO HAVE SEX WITH MEN: A MATHEMATICAL MODELLING APPROACH

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Abstract: The spread of monkeypox within a human population is investigated in this study using a mathematical model that incorporates the administration of two vaccine doses. The model's fundamental properties including the non-negativity and boundedness of its solutions are established to ensure its biological and epidemiological significance. The effective reproduction number, an important threshold for assessing disease transmission, is derived using the next generation matrix operator and used to analyse the conditions for the existence of equilibrium states of the model. In addition, the normalized forward sensitivity is performed on the effective reproduction number to identify parameters that strongly influence the spread of the disease in the population. The results provides useful insights into effective control strategies and appropriate intervention measures in reducing monkeypox transmission within the population

1. INTRODUCTION

The 2022 global outbreak of monkeypox (mpox) marked a significant departure from the established epidemiological understanding of the disease, thrusting the dynamics of transmission among men who have sex with men (MSM) to the forefront of public health concern. Historically considered a zoonotic disease with sporadic human-to-human transmission in endemic regions of Central and West Africa, the virus's unprecedented spread across non-endemic countries such as Italy and Spain revealed a distinct clinical and epidemiological profile closely tied to sexual networks [1]. The outbreak, which led the World Health Organization to declare a Public Health Emergency of International Concern, was characterized by a predominance of cases among gay, bisexual, and other men who have sex with men, highlighting the critical role of sexual contact as a primary transmission route [2]. The epidemiological features of this outbreak were starkly different from previous ones. Before 2022, mpox transmission was largely understood to occur through close skin-to-skin contact, respiratory droplets, or contact with contaminated fomites [3]. However, the 2022 outbreak saw a high incidence of anogenital and perianal lesions, strongly indicating transmission through intimate sexual contact [1]. This shift in transmission dynamics is not merely a superficial observation; it has profound implications for modeling, intervention strategies, and public health messaging. As the Africa CDC notes, recent outbreaks in Africa have also shown a significant shift towards transmission linked to sexual contact, particularly among MSM, a pattern driven by a confluence of factors including high population mobility, socio-economic instability, and the virus's genetic evolution [1, 2].

A crucial factor explaining the rapid spread within MSM networks is the structure of these sexual networks themselves. Research using transmission models fitted to empirical sexual partnership data has shown that these networks are heavy-tailed, meaning a small subset of individuals have a disproportionately large number of sexual partners [4]. This core group of highly active individuals can sustain transmission chains even when the virus's basic reproduction number might be low in the general population. This finding fundamentally challenges traditional outbreak containment strategies, suggesting that for mpox among MSM, the effective reproduction number can be substantially above 1, underscoring the vulnerability of these communities and the need for rapid, targeted interventions.

The 2022 outbreak also brought to light the challenge of subclinical or undiagnosed infections. A prospective surveillance study in Los Angeles, which tested anorectal swabs from a cohort of MSM, estimated that documented mpox cases may represent only a fraction of total infections, with a reporting multiplier of approximately 33-fold [5]. This extensive cryptic circulation, likely driven by mild or asymptomatic cases, suggests that a substantial proportion of virus

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transmission is associated with subclinical infections, potentially challenging the feasibility of elimination targets based solely on reported cases [5]. This finding, coupled with the documented instances of pre-symptomatic transmission, paints a picture of a virus that can circulate efficiently within dense sexual networks, often flying under the radar of traditional surveillance systems [5].

Mathematical modelling has been a key tool for studying the transmission and dynamical spread of infectious diseases and is of critical importance in addressing the challenges posed by monkeypox in the human population [6, 7, 8]. To predict the future trend, several models have been formulated and analysed for effective intervention and access to vaccination, which would allow for a comprehensive and better proactive public health measures. Adepoju and Ibrahim [9] proposed and analyzed a model for monkeypox transmission with vaccination and immunity loss following recovery. Their model accounted for the animal reservoir and findings concluded that the use of personal protective equipment (PPE) will help to mitigate the spread of monkeypox in the population. A deterministic mathematical model incorporating pre-exposure vaccination, post-exposure vaccination and isolation was considered by Ahmad *et al* [10]. Their findings reveal that awareness and isolation of infected individual will help curb the spread of monkeypox. The impact of immunization, isolation, and hospitalization on disease management, as well as the interaction between humans and rodents was considered to study the transmission dynamics of monkeypox using fractional differential equations [11]. Olaniyi and Chuma [12] formulated and analysed a deterministic compartmental model featuring the influence of vertical transmission and vaccination on the transmission dynamics of Mpox. Their study showed that the combination of awareness campaign and antiviral therapy will help reduce the persistence of monkeypox in the population. An investigation into the transmission of Mpox dynamics considering the effects of vaccination and treatment in Sub-Sahara Africa was conducted by Bolaji *et al* [13] while Okongo and his group [14], considered the effect of environmental and direct transmission.

Additionally, the dynamics of monkeypox with age-structured human population was offered by by Okongo *et al* [15]. Their model considered two distinct subgroups of children and adults, and the study found that early diagnosis and treatment of critically ill infected individuals will help curb its transmission. In another development, Naandam *et al* [16] formulated a nonlinear mathematical model featuring the impact of imperfect vaccination and immunity degradation post-recovery on the transmission dynamics of monkeypox, while Yaga [17] considered temporal dynamics and waning immunity from smallpox vaccination. Idisi et al [18] investigated Mpox two strain dynamics using computational and data driven approaches. Omame et al [19] considered a mathematical model for Mpox and Covid-19 dynamics in a high risk population by assessing the impact of intervention measures. Ventakesh *et al* [20] presented fractional mathematical model for vaccinated humans with impairment of monkeypox transmission and Rabiu *et al* [21] looked into the impact sexual transmission on the dynamics of monkeypox in human population. Furthermore, Akinyemi et al [22] concluded that maintain proper personal hygiene and changing behavoiur will help combat Mpox. Advances in artificial intelligence have significantly enhanced Mpox diagnostic capabilities. For instance, an automated deep learning model employing transfer learning has been created to identify Mpox skin lesions with high accuracy, enhancing early diagnosis [23].

The above reviewed articles synthesize recent advances in the research of Mpox to provide a multi-disciplinary framework for fighting the disease in the human population. Therefore, a mathematical model with double doses of vaccination that considers the impact of sexual transmission among men who have sex with men is proposed to study Mpox strains, which is missing in the literature. The organization of this work is as follows: Section 2 provides the formulation of the model. The analysis of the model is carried out in Section 3. Numerical simulations of the model are performed in Section 4, while Section 5 wraps up the concluding remarks of the study.

2. MODEL FORMULATION

A deterministic compartmental model for the dynamical spread of monkeypox among men that have sex with men with double dosage of vaccination is considered in this work. The total population at time t, denoted by N(t) is subdivided into seven mutually exclusive compartments namely, susceptible individuals S(t), individuals who received the first dose of vaccine $V_A(t)$, individuals that were administered second dose $V_B(t)$, exposed individuals E(t), infected individuals I(t), hospitalized individuals $H_P(t)$, and recovered individuals R(t). Therefore, the total human population is given by $N(t) = S(t) + V_A(t) + V_B(t) + E(t) + I(t) + R(t)$.

Recruitment into the susceptible population is at a rate Π. The effective transmission rate with the probability that individuals infected with Mpox per contact with an infected individual is β_t. The incidence of Mpox $\frac{\beta_t SI}{N}$ increases

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the exposed human population and reduces it at the rate α. The population of those who received the first dose of vaccine increases at a rate $(1-d_1)\gamma_1$; where d_1 represents fraction of susceptible humans unwilling to take the first dose and γ_1 represents the vaccination rate of individuals in the susceptible class. V_A subgroup reduces when vaccine wane at θ and when second dose is administered at $(1-d_2)\gamma_2$. $(1-d_3)\gamma_3$ represents those who received single dose which increases V_B subgroup. Infected individuals with severe cases got hospitalized at σ and recovered population increased as result of treatment from infected, and hospitalized compartments at the rates τ and ω. Thus, the mathematical model describing the transmission of Mpox in a closed population of men that have sex with men is given by the following system of nonlinear ordinary differential equations:

$$\begin{aligned}
\frac{dS}{dt} &= \Pi - \frac{\beta_t SI}{N} - \left(\mu + (1-d_1)\gamma_1 + (1-d_3)\gamma_3\right)S + \theta V_A \\
\frac{dV_A}{dt} &= (1-d_1)\gamma_1 S - \left(\mu + \theta + (1-d_2)\gamma_2\right)V_A \\
\frac{dV_B}{dt} &= (1-d_3)\gamma_3 S + (1-d_2)\gamma_2 V_A - \mu V_B \\
\frac{dE}{dt} &= \frac{\beta_t SI}{N} - (\alpha + \mu)E \\
\frac{dI}{dt} &= \alpha E - (\mu + \delta + \sigma + \tau)I \\
\frac{dH_P}{dt} &= \sigma I - (\mu + \delta + \omega)H_P \\
\frac{dR}{dt} &= \tau I + \omega H_p - \mu R
\end{aligned} \tag{1}$$

with initial conditions

$$S(0) = S_0,\ V_A(0) = V_{A0},\ V_B(0) = V_{B0},\ E(0) = E_0,\ I(0) = I_0,\ H_P(0) = H_{P0}, R(0) = R_0 \tag{2}$$

The flow diagram describing the transmission of Mpox is given in Figure 1, while the description of variables and parameters of the nonlinear system (1) are given in Table 1 and Table 2, respectively.

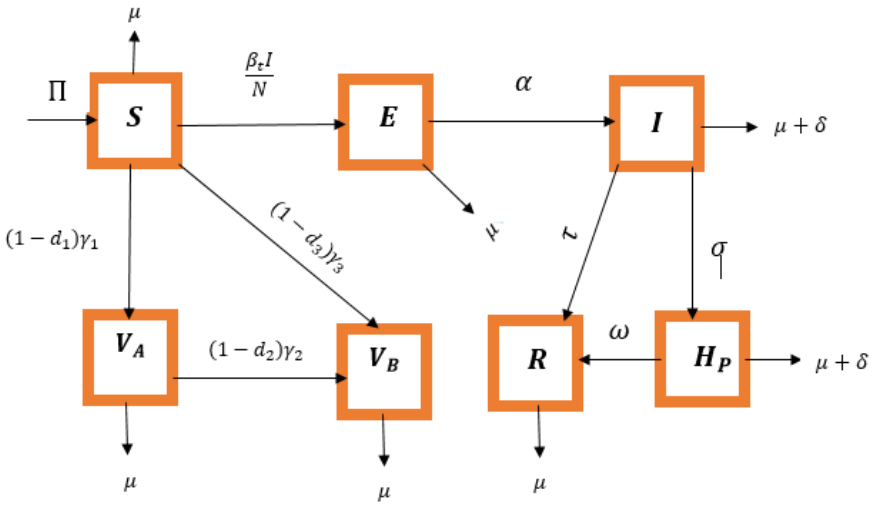

Figure 1: The flow diagram of the monkeypox model (1)

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Table 1: The description of variables of Mpox model (1)

Variable	Description
$S(t)$	Susceptible individuals
$V_A(t)$	Partially vaccinated individuals (first dose)
$V_B(t)$	Fully vaccinated individuals (second dose)
$E(t)$	Exposed individuals
$I(t)$	Infected individuals
$H_P(t)$	Hospitalized individuals
$R(t)$	Recovered individuals
$N(t)$	Total population

Table 2: The description of parameters of the Mpox model (1)

Parameter	Description
Π	Recruitment rate
β_t	Mpox transmission rate
γ_1	First dose of vaccine rate
μ	Natural mortality rate
d_1	Fraction of susceptible unwilling to take first dose
δ	Mpox induced death rate
ω	Recovery rate
α	Progression rate from exposed to infected class
σ	Hospitalized rate
γ_2	Vaccination rate of individuals in V_A subgroup with second dose
τ	Treatment rate
γ_3	Vaccination rate of humans in S with single dose
d_2	Fraction of vaccinated individuals unwilling to take second dose
d_3	Fraction of susceptible individuals with administered single dose

3. ANALYSIS OF THE MPOX MODEL

This section provides the rigorous analysis of the mathematical model governing the dynamics of Mpox transmission in human population

3.1 Invariant region

Theorem 1: *The feasible region* D *system (1) given by*

$$D=\left\{\left(S,V_A,V_B,E,I,H_P,R\right)\in R_+^7 : S+V_A+V_B+E+I+H_P+R\le\frac{\Pi}{\mu}\right\}$$

is positively invariant.

Proof: Since $N(t)=S(t)+V_A(t)+V_B(t)+E(t)+I(t)+R(t)$, then the derivative of $N(t)$ is given by

$$\frac{dN}{dt}=\frac{dS}{dt}+\frac{dV_A}{dt}+\frac{dV_B}{dt}+\frac{dE}{dt}+\frac{dI}{dt}+\frac{dH_P}{dt}+\frac{dR}{dt} \tag{3}$$

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Simplifying Equation (3) yields

$$\frac{dN}{dt} = \Pi - (I + H_P)\delta - \mu N \tag{4}$$

Since $\delta_h > 0$ and $I(t),\ H_P \geq 0$ for all $t \geq 0$, Equation (4) becomes

$$\frac{dN}{dt} \leq \Pi - \mu N \tag{5}$$

Integrating Equation (5) and solving by standard technique gives

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

$$N(t) \leq N(0)e^{-\mu_h t} + \frac{\Pi}{\mu}\left(1 - e^{\mu_h t}\right) \tag{6}$$

Therefore, at $t \geq 0$, $N(t) \leq \frac{\Pi}{\mu}$.

This implies that $\frac{\Pi}{\mu}$ is the upper bound for the human population $N(t)$. Thus, if $N(0) \leq \frac{\Pi}{\mu}$, then $N(t) \leq \frac{\Pi}{\mu}$.

Therefore, the region D is positively invariant. Additionally, if $N(0) \geq \frac{\Pi}{\mu}$, then the solution enters D in finite time as $N(t)$ approaches $\frac{\Pi}{\mu}$ asymptotically. Hence the feasible region is attracting. Biologically and mathematically, this implies that all solutions eventually enters D.

3.2 Positivity of solutions

Theorem 2: *The solution set,* $\{S, V_A, V_B, E, I, H_P, R\}$ *of the Mpox system (1) with nonnegative initial conditions (2) remain positive for all time,* $t > 0$.

Proof: employing the approach used in [24, 25, 26], it is obvious that

$$\left.\frac{dS}{dt}\right|_{S=0} = \Pi + \theta V_A > 0$$

$$\left.\frac{dV_A}{dt}\right|_{V_A=0} = (1 - d_1)\gamma_1 S_h \geq 0 \qquad \text{since } d_1 < 1,\ \gamma_1 > 0.$$

$$\left.\frac{dV_B}{dt}\right|_{V_B=0} = (1 - d_2)\gamma_2 V_A + (1 - d_3)\gamma_3 S \geq 0 \text{ since } r_2, r_3 < 1, \quad \theta_2, \theta_3 > 0$$

$$\left.\frac{dE}{dt}\right|_{E=0} = \frac{\beta_t SI}{N - E} \geq 0 \tag{7}$$

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$$\left.\frac{dI}{dt}\right|_{I=0} = \alpha E \geq 0$$

$$\left.\frac{dH_P}{dt}\right|_{H_P=0} = \sigma I \geq 0$$

$$\left.\frac{dR}{dt}\right|_{R=0} = \tau I + \omega H_P \geq 0$$

From Equation (7), it is obvious that the rate of change of the state variables of system (1) are non-negative. Given that the vector field's direction is inward on all bounding planes, this suggests that if solutions are launched inward in the bounding cone R^7, they will stay in the cone.

3.3 Existence and uniqueness of solution

The Mpox system (1) and the initial condition (2) can be written as an initial valued system given by

$$\begin{aligned} X'(t) &= F(X) \\ X(0) &= X_0 \end{aligned} \tag{8}$$

where $X = (S, V_A, V_B, E, I, H_P, R)^T$ and $F = (f_1, f_2, f_3, f_4, f_5, f_6, f_7)^T$.

Definition 1: $F(x)$ is Lipschitz continuous in x if there exists a constant $K > 0$ such that

$$\|F(X_1) - F(X_2)\| \leq K\|X_1 - X_2\| \tag{9}$$

Theorem 3: *The initial valued system (8) has a unique solution $x(t)$ corresponding to the Mpox model (1).*

Proof: It is important to show that the Lipschitz condition given in Equation (9) is satisfied. Let $X_1 = (S, V_A, V_B, E, I, H_P, R)$ and $X_2 = (\bar{S}, \bar{V}_A, \bar{V}_B, \bar{E}, \bar{I}, \bar{H}_P, \bar{R})$, then from the first compartment of system (1);

$$f_1(X_1) - f_1(X_2) =$$

$$\left(\left(\Pi - \frac{\beta_t SI}{N} - (\mu + (1-d_1)\gamma_1 + (1-d_3)\gamma_3)S + \theta V_A\right) - \left(\Pi - \frac{\beta_t \bar{S}\bar{I}}{N} - (\mu + (1-d_1)\gamma_1 + (1-d_3)\gamma_3)\bar{S} + \theta \bar{V}_A\right)\right) \tag{10}$$

$$= -\frac{\beta_t}{N}(SI - \bar{S}\bar{I}) - (\mu + (1-d_1)\gamma_1 + (1-d_3)\gamma_3)(S - \bar{S}) - \theta(V_A - \bar{V}_A) \tag{11}$$

Expanding the nonlinear term $(SI - \bar{S}\bar{I})$ in Equation (11) using cross correspondence yields

$$\begin{aligned} SI - \bar{S}\bar{I} &= SI - S\bar{I} + S\bar{I} - \bar{S}\bar{I} \\ &= S(I - \bar{I}) + \bar{I}(S - \bar{S}) \end{aligned} \tag{12}$$

Substituting Equation (12) into Equation (11) gives

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$$f_1(X_1)-f_1(X_2)=-\frac{\beta_t}{N}\left(S(I-\bar{I})+\bar{I}(S-\bar{S})\right)-\left(\mu+(1-d_1)\gamma_1+(1-d_3)\gamma_3\right)(S-\bar{S})-\theta(V_A-\bar{V}_A) \quad (13)$$

Taking the modulus of Equation (15) yields

$$\|f_1(X_1)-f_1(X_2)\|\le\left\|-\frac{\beta_t}{N}\left(S(I-\bar{I})+\bar{I}(S-\bar{S})\right)\right\|+\left\|-\left(\mu+(1-d_1)\gamma_1+(1-d_3)\gamma_3\right)(S-\bar{S})\right\|+\left\|-\theta(V_A-\bar{V}_A)\right\|$$

$$\le\frac{\beta_t}{N}\left\|\left(S(I-\bar{I})+\bar{I}(S-\bar{S})\right)\right\|+\left(\mu+(1-d_1)\gamma_1+(1-d_3)\gamma_3\right)\left\|(S-\bar{S})\right\|+\theta\left\|(V_A-\bar{V}_A)\right\| \quad (14)$$

Applying the limiting value $N\le\frac{\Pi}{\mu}$ in D such that $S\le\frac{\Pi}{\mu}$ and $\bar{I}\le\frac{\Pi}{\mu}$, then Equation (14) becomes

$$\|f_1(X_1)-f_1(X_2)\|\le\left(\beta_t+\left(\mu+(1-d_1)\gamma_1+(1-d_3)\gamma_3\right)\right)\|S-\bar{S}\|+\beta_t\|I-\bar{I}\|+\theta\|V_A-\bar{V}_A\| \quad (15)$$

Similarly, considering the remaining compartments of the model (1), it can be shown that

$$\begin{aligned}
&\|f_2(X_1)-f_2(X_2)\|\le(1-d_1)\gamma_1\|S-\bar{S}\|+\left(\mu+\theta+(1-d_2)\gamma_2\right)\|V_A-\bar{V}_A\|,\\
&\|f_3(X_1)-f_3(X_2)\|\le(1-d_3)\gamma_3\|S-\bar{S}\|+(1-d_2)\gamma_2\|V_A-\bar{V}_A\|+\mu\|V_B-\bar{V}_B\|,\\
&\|f_4(X_1)-f_4(X_2)\|\le\beta_t\|S-\bar{S}\|+\beta\|I-\bar{I}\|+(\alpha+\mu)\|E-\bar{E}\|, \qquad (16)\\
&\|f_5(X_1)-f_5(X_2)\|\le\alpha\|E-\bar{E}\|+(\mu+\delta+\sigma+\tau)\|I-\bar{I}\|,\\
&\|f_6(X_1)-f_6(X_2)\|\le\sigma\|I-\bar{I}\|+(\mu+\delta+\omega)\|H_P-\bar{H}_P\|,\\
&\|f_7(X_1)-f_7(X_2)\|\le\tau\|I-\bar{I}\|+\omega\|H_P-\bar{H}_P\|+\mu\|R-\bar{R}\|
\end{aligned}$$

Therefore, $K=\max\begin{Bmatrix}(2(\beta_t+(1-d_1)\gamma_1+(1-d_3)\gamma_3)+\mu),\ (2(\theta+(1-d_2)\gamma_2)+\mu),\ \mu,\ (2\alpha+\mu),\\ (2(\beta_t+\sigma+\tau)+\mu+\delta),\ (2\omega+\mu+\delta),\ \mu\end{Bmatrix}$

Since K is finite, then the system satisfies the Lipschitz condition. Hence, a unique solution exists and the model is well-posed.

3.4 Equilibrium points and stability analysis

Mpox-free equilibrium point

At Mpox-free equilibrium, compartments $E=I=H_P=R=0$.

The disease-free equilibrium point of the model $E_0=(S,\ V_A,\ V_B\ E,\ I,\ H_P,\ R)$ is given as

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$$E_0 = \left(\frac{k_2\Pi}{k_1k_2 - \theta(1-d_1)\gamma_1}, \frac{\Pi(1-d_1)\gamma_1}{k_1k_2 - (1-d_1)\gamma_1\theta}, \frac{\Pi(\gamma_1\gamma_2(1-d_1)(1-d_2) + k_2\gamma_3(1-d_3))}{\mu(k_1k_2 - (1-d_1)\gamma_1\theta)}, 0,0,0,0 \right) \quad (17)$$

To compute the effective reproduction number $\Re_0$, the next generation matrix approach is applied [27, 28]. This means that the transmission matrix F and the transition matrix V are obtained as follows

$$F = \begin{pmatrix} 0 & \frac{\beta_t S^0}{N} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \text{ and } V = \begin{pmatrix} k_3 & 0 & 0 \\ -\alpha & k_4 & 0 \\ 0 & -\sigma & k_5 \end{pmatrix} \quad (18)$$

Then the effective reproduction number that measures the threshold under which the incidence of Mpox will persist is obtained as

$$\Re_0 = \frac{k_2\alpha\beta_t\mu}{k_3k_4(k_1k_2 - \gamma_1\theta(1-d_1))} \quad (19)$$

where $k_1 = (\mu + (1-d_1)\gamma_1 + (1-d_3)\gamma_3)$, $k_2 = (\mu + (1-d_2)\gamma_2 + \theta)$, $k_3 = (\mu + \alpha)$, $k_1 = (\mu + \delta + \sigma + \tau)$ and $k_1 = (\mu + \delta + \omega)$

***Lemma* 1:** *The mpox-free equilibrium point of system (1) is locally asymptotically stable whenever the effective reproduction number $\Re_0$, is less than unity.*

Proof: The Jacobian matrix of the mpox model (1) evaluated at disease-free equilibrium E_0 is obtained as

$$\Im_{(E_0)} = \begin{pmatrix} -k_1 & \theta & 0 & 0 & -\frac{\beta_t S^0}{N} & 0 & 0 \\ (1-d_1)\gamma_1 & -k_2 & 0 & 0 & 0 & 0 & 0 \\ (1-d_3)\gamma_3 & (1-d_2)\gamma_2 & -\mu & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -k_3 & \frac{\beta_t S^0}{N} & 0 & 0 \\ 0 & 0 & 0 & \alpha & -k_4 & 0 & 0 \\ 0 & 0 & 0 & 0 & \sigma & -k_5 & 0 \\ 0 & 0 & 0 & 0 & \tau & \omega & -\mu \end{pmatrix} \quad (20)$$

Obviously, three of the eigenvalues of Equation (20) as $-\mu$, $-\mu$, k_5, while the remaining are obtained from the following quartic polynomial given by

$$\lambda^4 + Q_1\lambda^3 + Q_2\lambda^2 + Q_3\lambda + Q_4 = 0 \quad (21)$$

where

$Q_1 = k_1k_2k_3k_4$

$Q_2 = k_1k_2 + (k_1 + k_2)(k_3 + k_4) + \theta\gamma_1(1-d_1) + k_3k_4(1-\Re_0)$

$Q_3 = k_3k_4(k_1k_2 + \theta\gamma_1(1-d_1)) + (k_1 + k_2)k_3k_4(1-\Re_0)$

$Q_4 = k_3k_4(k_1k_2 + \theta\gamma_1(1-d_1))(1-\Re_0)$

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Using Routh-Hurwitz criterion, the quartic polynomial (21) will have roots with negative real parts provided $Q_1 > 0$, $Q_2 > 0$, $Q_3 > 0$, $Q_1 Q_4 > 0$, $Q_1 Q_2 > Q_3$ and $Q_1 Q_2 > Q_3$. Clearly, $Q_1 > 0$ and $Q_2, Q_3, Q_4 > 0$ provided $\Re_0 < 1$. As a consequence, the mpox-free equilibrium point is locally asymptotically stable whenever $\Re_0 < 1$.

Global stability of mpox-free equilibrium

In order to carry out the global asymptotic stability of the mpox-free equilibrium of system (1) using the approach of [29, 30]. The model can be restructured in the form

$$\begin{aligned} \frac{dX}{dt} &= F(X, Z) \\ \frac{dZ}{dt} &= G(X, Z), \quad G(X, 0) = 0 \end{aligned} \tag{22}$$

where $X \in R_+^4$ and $Z \in R_+^3$. The X component represents the uninfected compartments, while the Z component represents the infected compartments of system (1). Given that the mpox-free equilibrium point of system (1) is $E_0 = (X^*, 0)$, then the following properties must be satisfied in order to establish the global asymptotic stability of mpox-free equilibrium

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

P_2: $G(X, Z) = AZ - \hat{G}(X, Z)$, $\hat{G}(X, Z) \geq 0$ for $(X, Z) \in D$

where $A = \frac{\partial G}{\partial Z}$ is an M-matrix with non-negative off-diagonal elements evaluated $(X^*, 0)$.

Theorem 4: *The mpox-free equilibrium point of system (1) is globally asymptotically stable whenever* $\Re_0 < 1$ *and properties* P_1 *and* P_2 *are satisfied*

Proof: The functions $F(X, Z)$ and $G(X, Z)$ of are obtained from the compact system (22) as follows

$$F(X, Z) = \begin{pmatrix} \Pi - \frac{\beta_t SI}{N} - (\mu + (1-d_1)\gamma_1 + (1-d_3)\gamma_3)S + \theta V_A \\ (1-d_1)\gamma_1 S - (\mu + \theta + (1-d_2)\gamma_s)V_A \\ (1-d_3)\gamma_3 S + (1-d_2)\gamma_2 V_A - \mu V_B \\ \tau I + \omega H_P - \mu R \end{pmatrix} \tag{23}$$

And

$$G(X, Z) = \begin{pmatrix} \frac{\beta_t SI}{N} - (\alpha + \mu)E \\ \alpha E - (\mu + \delta + \sigma + \tau)I \\ \sigma I - (\mu + \delta + \omega)H_P \end{pmatrix} \tag{24}$$

Then, $\frac{dX}{dt} = F(X, 0)$ implies

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Impact Factor 8.066 ⁜ Peer-reviewed & Refereed journal ⁜ Vol. 13, Issue 9, September 2026

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$$\begin{aligned}
\frac{dS}{dt} &= \Pi - \frac{\beta_t SI}{N} - \left(\mu + (1-d_1)\gamma_1 + (1-d_3)\gamma_3\right)S + \theta V_A \\
\frac{dV_A}{dt} &= (1-d_1)\gamma_1 S - \left(\mu + \theta + (1-d_2)\gamma_2\right)V_A \\
\frac{dV_B}{dt} &= (1-d_3)\gamma_3 S + (1-d_2)\gamma_2 V_A - \mu V_B \\
\frac{dR}{dt} &= \tau I + \omega H_p - \mu R
\end{aligned} \tag{25}$$

Evaluating Equation (25) at mpox-free equilibrium yields

$$\begin{aligned}
\frac{dS}{dt} &= \Pi - \left(\mu + (1-d_1)\gamma_1 + (1-d_3)\gamma_3\right)S + \theta V_A \\
\frac{dV_A}{dt} &= (1-d_1)\gamma_1 S - \left(\mu + \theta + (1-d_2)\gamma_2\right)V_A \\
\frac{dV_B}{dt} &= (1-d_3)\gamma_3 S + (1-d_2)\gamma_2 V_A - \mu V_B \\
\frac{dR}{dt} &= -\mu R
\end{aligned} \tag{26}$$

Solving Equation (26) simultaneously by standard technique gives

$$S(t) = \frac{k_2\Pi}{k_1k_2 - \theta\gamma_1(1-d_1)} + S(0)e^{\frac{-(k_1k_2-\theta\gamma_1(1-d_1))t}{k_2}} - \frac{k_2\Pi}{k_1k_2 - \theta\gamma_1(1-d_1)}e^{\frac{-(k_1k_2-\theta\gamma_1(1-d_1))t}{k_2}}$$

$$V_A(t) = \frac{(1-d_1)\gamma_1}{k_2}\left(\frac{k_2\Pi}{k_1k_2 - \theta\gamma_1(1-d_1)} + S(0)e^{\frac{-(k_1k_2-\theta\gamma_1(1-d_1))t}{k_2}} - \frac{k_2\Pi}{k_1k_2 - \theta\gamma_1(1-d_1)}e^{\frac{-(k_1k_2-\theta\gamma_1(1-d_1))t}{k_2}}\right) \tag{27}$$

$$V_B(t) = \frac{(1-d_3)\gamma_3}{\mu}\left(\frac{k_2\Pi}{k_1k_2 - \theta\gamma_1(1-d_1)} + S(0)e^{\frac{-(k_1k_2-\theta\gamma_1(1-d_1))t}{k_2}} - \frac{k_2\Pi}{k_1k_2 - \theta\gamma_1(1-d_1)}e^{\frac{-(k_1k_2-\theta\gamma_1(1-d_1))t}{k_2}}\right)$$
$$+ \frac{(1-d_2)\gamma_2}{\mu}\frac{(1-d_1)\gamma_1}{k_2}\left(\frac{k_2\Pi}{k_1k_2 - \theta\gamma_1(1-d_1)} + S(0)e^{\frac{-(k_1k_2-\theta\gamma_1(1-d_1))t}{k_2}} - \frac{k_2\Pi}{k_1k_2 - \theta\gamma_1(1-d_1)}e^{\frac{-(k_1k_2-\theta\gamma_1(1-d_1))t}{k_2}}\right)$$

$$R(t) = R(0)e^{-\mu t}$$

Hence,

$$(S, V_A, V_B, R) \to \left(\frac{k_2\Pi}{k_1k_2 - \theta\gamma_1(1-d_1)}, \frac{\Pi\gamma_1(1-d_1)}{k_1k_2 - \theta\gamma_1(1-d_1)}, \frac{\Pi\left(\gamma_1\gamma_2(1-d_1)(1-d_2) + k_2\gamma_3(1-d_3)\right)}{\mu\left(k_1k_2 - \theta\gamma_1(1-d_1)\right)}, 0\right)$$

irrespective of the initial values of the state variables as $t \to \infty$. Therefore, X^* is globally asymptotically stable satisfying property P_1. Furthermore, the matrix A with non-negative off-diagonal elements is given as

IARJSET ISSN (O) 2393-8021, ISSN (P) 2394-1588

International Advanced Research Journal in Science, Engineering and Technology

Impact Factor 8.066 ⁜ Peer-reviewed & Refereed journal ⁜ Vol. 13, Issue 9, September 2026

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$$A=\frac{\partial G}{\partial Z}\left(X^*,0\right)=\begin{pmatrix}-(\alpha+\mu) & \frac{\beta_t S}{N} & 0\\ \alpha & -(\mu+\delta+\sigma+\tau) & 0\\ 0 & \sigma & -(\mu+\delta+\omega)\end{pmatrix}\begin{pmatrix}E\\ I\\ H_P\end{pmatrix} \tag{28}$$

Then, $\hat{G}(X,\,Z)=AZ-G(X,\,Z)$ gives

$$\begin{pmatrix}\frac{\beta_t S^0 I}{N}-(\alpha+\mu)E\\ \alpha E-(\mu+\delta+\sigma+\tau)I\\ \sigma I-(\mu+\delta+\omega)H_P\end{pmatrix}-\begin{pmatrix}\frac{\beta_t SI}{N}-(\alpha+\mu)E\\ \alpha E-(\mu+\delta+\sigma+\tau)I\\ \sigma I-(\mu+\delta+\omega)H_P\end{pmatrix} \tag{29}$$

$$\hat{G}(X,\,Z)=\begin{pmatrix}\frac{\beta_t I}{N}\left(S^0-S\right)\\ 0\\ 0\end{pmatrix} \tag{30}$$

Since $0\le S\le S^0$, $0\le V_A\le V_A^0$, and $0\le V_B\le V_B^0$, it is obvious that $\hat{G}(X,\,Z)\ge 0$. Therefore, property P_2 holds. Hence, the mpox-free equilibrium is globally asymptotically stable.

Endemic equilibrium

Let the endemic equilibrium point be $\Delta^{**}=\left(S^{**},V_A^{**},V_B^{**},E^{**},I^{**},H_P^{**},R^{**}\right)$ and let $\lambda^{**}=\frac{\beta_t I}{N}$ be the force of infection of the system. Then, solving model (1) simultaneously at steady state gives the following

$$S^{**}=\frac{k_2\Pi}{k_2\left(k_1+\lambda^{**}\right)-(1-d_1)\gamma_1\theta}$$

$$V_A^{**}=\frac{\Pi(1-d_1)\gamma_1}{k_2\left(k_1+\lambda^{**}\right)-\theta(1-d_1)\gamma_1}$$

$$V_B^{**}=\frac{\Pi\left((1-d_1)\gamma_1(1-d_2)\gamma_2+k_2\left((1-d_3)\gamma_3\right)\right)}{\mu\left(k_2\left(k_1+\lambda^{**}\right)-\theta(1-d_1)\gamma_1\right)}$$

$$E^{**}=\frac{k_2\Pi\lambda^{**}}{k_3\left(k_2\left(k_1+\lambda^{**}\right)-\theta(1-d_1)\gamma_1\right)} \tag{31}$$

$$I^{**}=\frac{k_2\alpha\Pi\lambda^{**}}{k_3k_4\left(k_2\left(k_1+\lambda^{**}\right)-\theta(1-d_1)\gamma_1\right)}$$

IARJSET ISSN (O) 2393-8021, ISSN (P) 2394-1588

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$$H_P^{**} = \frac{k_2\sigma\alpha\Pi\lambda^{**}}{k_3k_4k_5\left(k_2\left(k_1+\lambda^{**}\right)-\theta(1-d_1)\gamma_1\right)}$$

$$R^{**} = \frac{k_2\alpha\Pi\lambda^{**}\left(k_5\tau+\sigma\omega\right)}{k_3k_4k_5\left(k_2\left(k_1+\lambda^{**}\right)-\theta(1-d_1)\gamma_1\right)}$$

Substituting the value I^{**} in Equation (31) into the force of infection gives

$$\lambda^{**} = \frac{k_1k_2-(1-d_1)\gamma_1(\Re_0-1)}{k_2} \tag{32}$$

Hence endemic equilibrium point exists whenever $\Re_0 > 1$. Epidemiologically, this implies that mpox will persist in the population and would be difficult to control whenever the effective reproduction number is greater than unity.

Global asymptotic stability of endemic equilibrium

The investigation into the global asymptotic dynamics of the mpox endemic equilibrium point of model (1) would be carried out here using the approach of [31, 32]

Theorem 5: *The endemic equilibrium point, Δ^{**}, of the mpox model (1) is globally asymptotically stable whenever the effective reproduction number is greater than unity $(\Re_0 > 1)$.*

Proof: Consider the positive definite quadratic Lyapunov function defined by

$$\mathrm{P} = \frac{1}{2}\left(\left(S-S^{**}\right)+\left(V_A-V_A^{**}\right)+\left(V_B-V_B^{**}\right)+\left(E-E^{**}\right)+\left(I-I^{**}\right)+\left(H_P-H_P^{**}\right)+\left(R-R^{**}\right)\right)^2 \tag{33}$$

Differentiating Equation (33) with respect to time gives

$$\begin{aligned}\frac{d\mathrm{P}}{dt} &= \left(\left(S-S^{**}\right)+\left(V_A-V_A^{**}\right)+\left(V_B-V_B^{**}\right)+\left(E-E^{**}\right)+\left(I-I^{**}\right)+\left(H_P-H_P^{**}\right)+\left(R-R^{**}\right)\right)\\ &\quad\times\frac{d}{dt}\left(S+V_A+V_B+E+I+H_P+R\right)\\ &= \left(\left(S-S^{**}\right)+\left(V_A-V_A^{**}\right)+\left(V_B-V_B^{**}\right)+\left(E-E^{**}\right)+\left(I-I^{**}\right)+\left(H_P-H_P^{**}\right)+\left(R-R^{**}\right)\right)\\ &\quad\times\left(\Pi-\mu\left(S+V_A+V_B+E+I+H_P+R\right)-\delta\left(I+H_P\right)\right)\\ &\le \left(\left(S-S^{**}\right)+\left(V_A-V_A^{**}\right)+\left(V_B-V_B^{**}\right)+\left(E-E^{**}\right)+\left(I-I^{**}\right)+\left(H_P-H_P^{**}\right)+\left(R-R^{**}\right)\right)\\ &\quad\times\left(\Pi-\mu\left(S+V_A+V_B+E+I+H_P+R\right)\right)\\ &\le -\mu\left(\left(S-S^{**}\right)+\left(V_A-V_A^{**}\right)+\left(V_B-V_B^{**}\right)+\left(E-E^{**}\right)+\left(I-I^{**}\right)+\left(H_P-H_P^{**}\right)+\left(R-R^{**}\right)\right)\\ &\quad\times\left(\left(S+V_A+V_B+E+I+H_P+R\right)-\frac{\Pi}{\mu}\right)\end{aligned} \tag{34}$$

Substituting the limiting value , $N^{**} = \dfrac{\Pi}{\mu}$ at invariant region, then Equation (34) becomes

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International Advanced Research Journal in Science, Engineering and Technology

Impact Factor 8.066 ⁜ Peer-reviewed & Refereed journal ⁜ Vol. 13, Issue 9, September 2026

DOI: 10.17148/IARJSET.2026.13902

$$\frac{d\dot{P}}{dt} \le -\mu\left(\left(S - S^{**}\right) + \left(V_A - V_A^{**}\right) + \left(V_B - V_B^{**}\right) + \left(E - E^{**}\right) + \left(I - I^{**}\right) + \left(H_P - H_P^{**}\right) + \left(R - R^{**}\right)\right)^2 \quad (35)$$

Correlatively, the derivative of the continuously differentiable function $\dot{P}$ is negative semi-definite, that is $\dot{P} \le 0$. This implies that P is a Lyapunov function. Therefore, $\dot{P} = 0$ provided $S = S^{**}$, $V_A = V_A^{**}$, $V_B = V_B^{**}$, $E = E^{**}$, $I = I^{**}$, $H_P = H_P^{**}$ and $R = R^{**}$. Then, conjuring LaSalle's invariance principle (LaSalle, 1976), the largest invariance set for which $\dot{P} = 0$ is the singleton set $\left\{\Delta^{**}\right\}$, which means that the endemic equilibrium point of the mpox model (1) is globally asymptotically stable.

Sensitivity analysis

The sensitivity analysis of the parameters associated with the effective reproduction number are calculated to observe the changes that will occur in the dynamics of mpox transmission. This is obtained by using the normalized for sensitivity index (chitnis et al). The normalized forward sensitivity indices of the effective reproduction number relative to its paramerer is given as

$$\Psi_p^{\Re_0} = \frac{\partial \Re_0}{\partial p} \times \frac{p}{\Re_0} \quad (36)$$

where p represents the parameter in the effective reproduction number. Applying Equation (33), the sensitivity index of each parameter of $\Re_0$ are calculated with parameters values given in Table 3 and the S.I results are given in Table 4.

Table 3: Values of parameters of the Mpox model

Parameter	Value	Source
Π	10	Olaniyi and Chuma (2023)
β_t	0.09	Olaniyi and Chuma (2023)
α	0.14	Wangari *et al*., (2023)
ω	0.07143	Olaniyi and Chuma (2023)
μ	0.0000548	Olaniyi and Chuma (2023)
τ	0.05	Assumed
σ	0.04762	Wangari *et al*., (2023)
δ	0.002	Assumed
γ_1	0.07	Wangari *et al*., (2023)
γ_2	0.05	Wangari *et al*., (2023)
γ_3	0.07	Wangari *et al*., (2023)
d_1	0.35	Wangari *et al*., (2023)
d_2	0.25	Wangari et al., (2023)
d_3	0.35	Wangari et al., (2023)
θ	5.56×10^{-3}	Assumed

IARJSET ISSN (O) 2393-8021, ISSN (P) 2394-1588

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Impact Factor 8.066 ⁜ Peer-reviewed & Refereed journal ⁜ Vol. 13, Issue 9, September 2026

DOI: 10.17148/IARJSET.2026.13902

Table 4: Sensitivity indices of $\Re_0$ relative to the values of its parameters

Parameter	Sensitivity Indices
Π	+0.0377
β_t	+1.0000
α	+0.8772
μ	-0.0001
τ	-0.0471
σ	-0.0448
δ	-0.0019
γ_1	-0.0407
γ_2	-0.0000099
γ_3	-0.0607
d_1	+0.0219
d_2	+0.000003
d_3	+0.0220
θ	+0.0002

4. NUMERICAL SIMULATION

Here, numerical simulation of the monkeypox model are performed to corroborate the qualitative analysis of performed in Section 3 using MATLAB computing software and the results are displayed graphically. The impact of effective transmission rate and progression rate of exposed individuals on effective reproduction number are depicted in Figure 2. It can be seen that both have corresponding increase on $\Re_0$. This implies that there would be persistence of monkeypox in the population as more exposed individuals' progresses to active infectious class. The effect of treatment rate and effective contact rate are depicted on $\Re_0$ are depicted in Figure 3. One can see that when active infectious individuals are treated properly, it will stem down the tide of the disease in the population. The impact of varying values of effective transmission rate on the infectious population are carried out in Figure 4a, b and c. it is observed that as the value of β_t increases, the infectious population increased. This means that there would be prevalence of monkeypox in the population if necessary actions are not taken in controlling its transmission

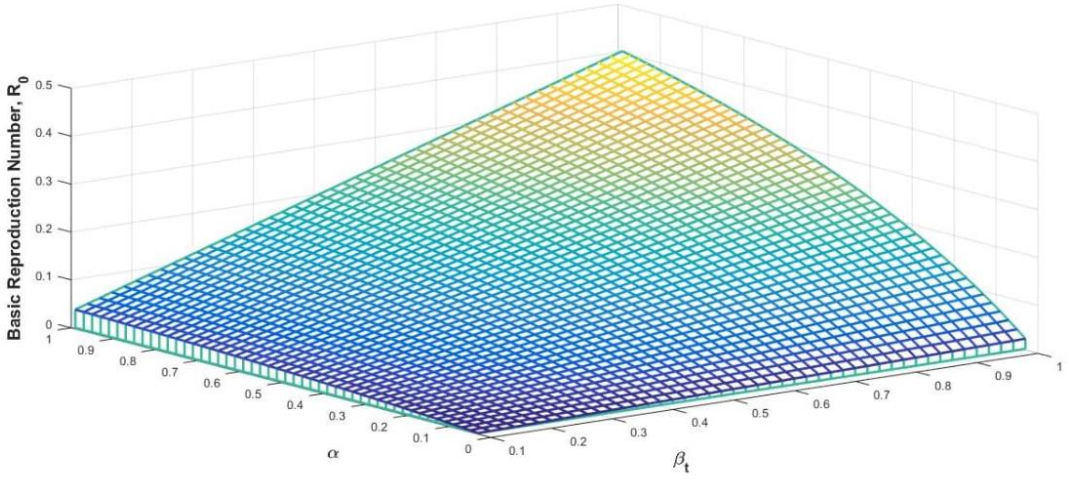

Figure 2: Impact of α and β_t on the effective reproduction number

IARJSET ISSN (O) 2393-8021, ISSN (P) 2394-1588

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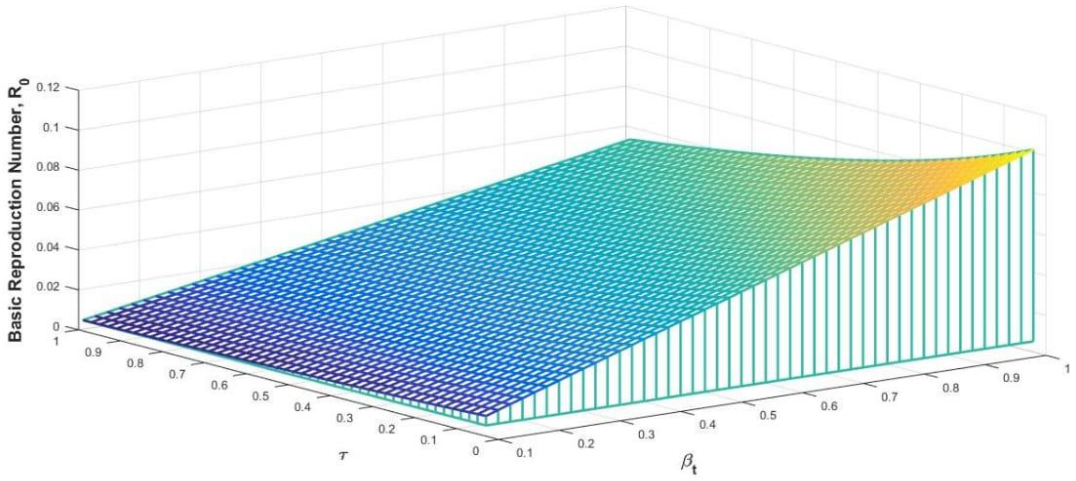
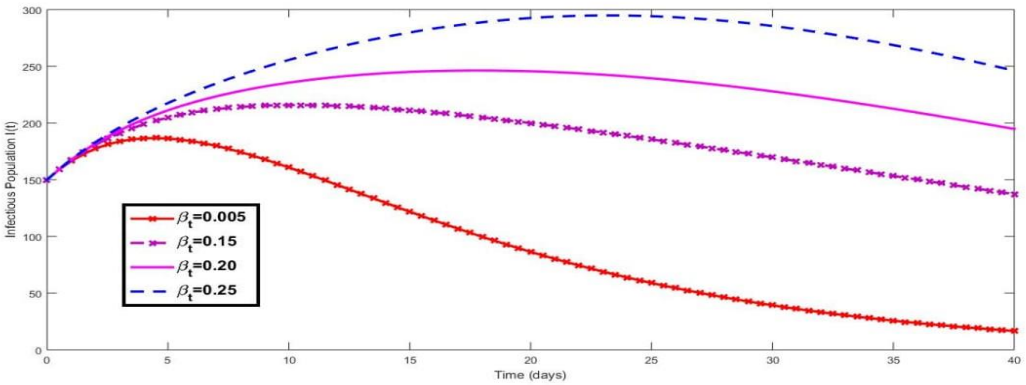


Figure 3: Impact of τ and β_t on the effective reproduction number

Furthermore, the global asymptotic behavior of infectious populations around the monkeypox-free equilibrium are shown in Figures 5a, b and c. It is observed that regardless of the initial size of the infectious populations, they will converge to a monkeypox-free equilibrium state. Biologically, this means that a monkeypox-free equilibrium state is possible in the human population.

5. CONCLUSION

A nonlinear deterministic compartmental model describing the dynamical spread of monkeypox in a closed population of men that have sex with men was formulated and analyzed in this study. A set of ordinary differential equations differential equations divided into seven mutually exclusive compartments namely, susceptible population, partially vaccinate population, fully vaccinated population, exposed individuals, infected population, hospitalized population and recovered population was used to govern the model. The Lipschitz condition was used to show that the model has a unique solution and the effective reproduction number was obtained using the next generation matrix operator. Using the linearized Jacobian matrix approach, the monkeypox-free equilibrium was shown to be locally asymptotically stable whenever the effective reproduction number is less than unity, and globally asymptotically stable using the M-matrix method. Additionally, the global asymptotic dynamics of the monkeypox-present equilibrium was carried out using a well-constructed Lyapunov function, and the impact of parameters associated with the effective reproduction number was carried out using the normalized forward sensitivity index. Parameters with positive sensitivity indices indicate that when increased will increase the effective reproduction number, while those with negative indices will reduce it when increased. Numerical simulation was carried out using MATLAB computing software. It is instructive to state that parameters used for simulation in this study are obtained from existing literature on the dynamics of monkeypox transmission.



(a)

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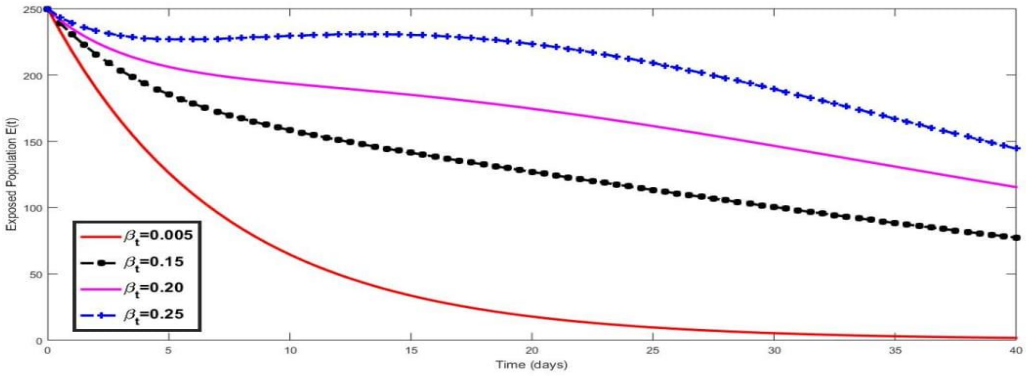

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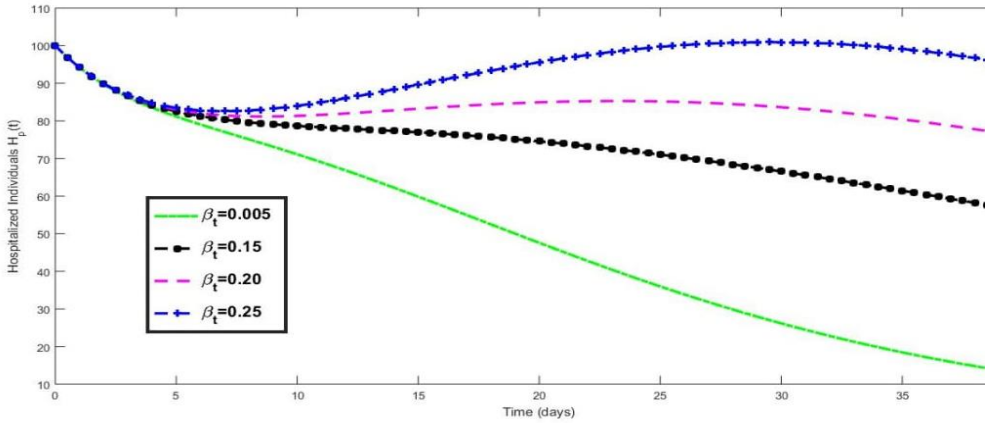
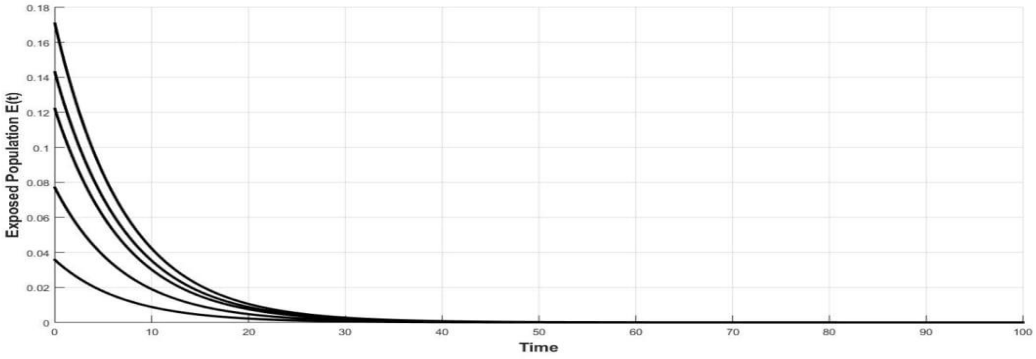


(c)

Figure 4: Influence of varying values of β_t on the infectious population



(a)

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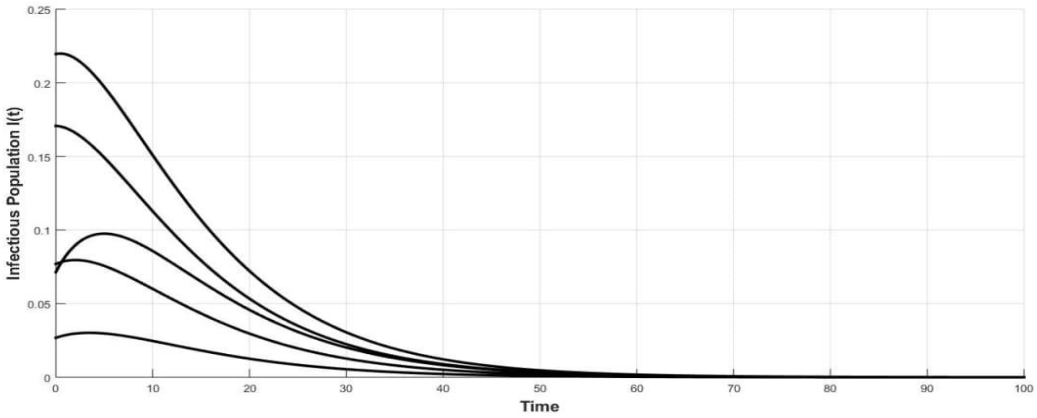

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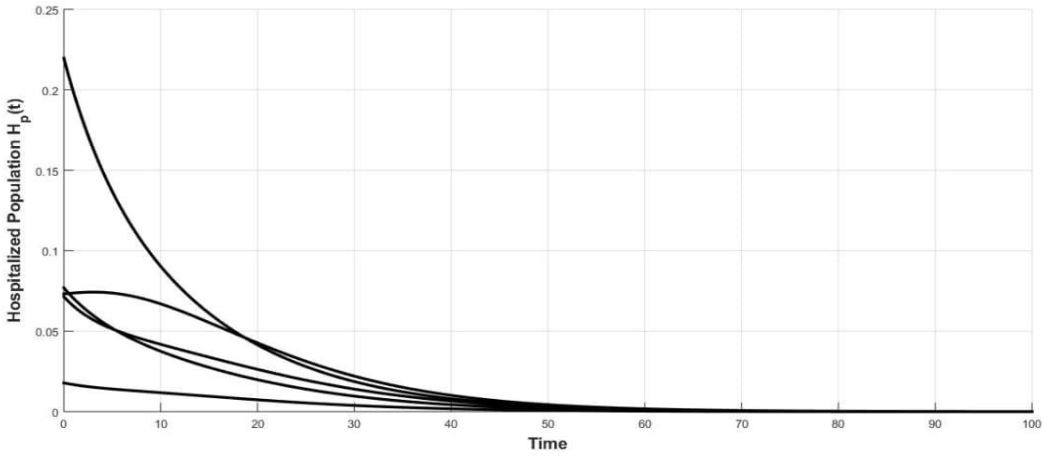

(c)

Figure 5: Convergence of global asymptotic dynamics of infectious population around monkeypox-free equilibrium regardless of the initial size

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