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A model for the control of transmission dynamics of human monkeypox disease in Sub-Saharan Africa

Bolarinwa Bolaji[a,d,*], Favour Ani[b], Benjamin Omede[a,d], Godwin Acheneje[a,d], Abdullahi Ibrahim[c]

[a]*Department of Mathematical Sciences, Prince Abubakar Audu University, Anyigba, P.M.B 1008, Nigeria*
[b]*Department of Mathematics, National Open University of Nigeria, Nnamdi Azikiwe Expressway, Jabi, 901108, Abuja, Nigeria*
[c]*Department of Mathematics, Baze University, Jabi Airport Road Bypass (Ring Road), Jabi, Abuja, 901108, Nigeria*
[d]*Laboratory of Mathematical Epidemiology and Applied Sciences, Prince Abubakar Audu University, Anyigba, P.M.B 1008, Nigeria*

Abstract

The Human Monkeypox virus has received significant research interest in recent times. While few researchers have studied the effects of vaccination on human-to-animal or animal-to-human Monkeypox transmission, others just studied the effects of treatment on human Monkeypox. In this article, we made the proposition of a deterministic vaccine model that deals with the dynamics of the effects of vaccination and treatment on human Monkeypox in sub-Saharan Africa. We investigated the effects of vaccination on the various epidemiological classes qualitatively. The findings from the analysis of the model are that the model possesses two equilibria, locally asymptotically stable disease-free equilibrium (DFE) when an epidemiological threshold - the effective reproductive number is less than unity, and locally asymptotically stable endemic equilibrium when the number is greater than unity. We then corroborated the theoretical findings with numerical simulations, which reveal that when the rate of vaccination is increased resulting in many newly born persons in the populace being vaccinated, the prevalence of the deadly scourge is significantly reduced, while newly born individuals that miss vaccination experience a drastic torment of the deadly disease that often occasion death. Further revelation from the simulations is that when greater efforts is geared towards vaccination of individuals in the population, the loss of more people to the scourge of the virus would be greatly reduced.

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1. Introduction

Monkeypox virus has its origin dated back to 1959 as a result of a strange disease that invaded the colony of monkeys in Copenhagen, Denmark at their State Serum Institute [1–3]. Worthy of note is the fact that Monkeypox virus was actually identified as a distinct disease only after smallpox was eradicated in 1970-1971, when it was identified as a disease whose illness resembles that of smallpox in the suburb of western and central Africa [1–3]. Human Monkeypox virus, a zoonotic febrile rash illness with characteristic that it is smallpox-like in nature which belong to the family of genus Orthopox virus family poxviridae and it is of subfamily chordopoxvirinae with traits of disease in the category of cowpox, camelpox, ec-

*Corresponding author: Tel.: +234-803-499-5772
Email address: bolarinwa.s.bolaji@gmail.com (Bolarinwa Bolaji)

tromelia, vaccinia and smallpox otherwise called variola [1, 2]. During this period, it should be noted that in the countries of the sub-Saharan Africa, such as Nigeria, Liberia, Sierra Leone, and Democratic Republic of Congo (DRC), 6 cases were reported in Liberia, Sierra Lone, DRC and Nigeria while over the next decade in the same countries of sub-Saharan Africa, 53 additional cases of human Monkeypox virus disease were reported [2]. However, it is pertinent to note that 80% of this cases occurred in DRC with other remaining cases of Monkeypox virus infection afflicted the citizens of the Liberia, Sierra Leone, Nigeria, Gabon, Central African Republic, Cameroon and Cote d'Ivoire [3, 4]. Furthermore, it has been identified within the terminal regions via whole genome analysis and DNA sequencing of Monkeypox virus (MPXV), the presence of two geographically distinct MPXV classes which are of ninety nine percent similar and demonstrate greatest diversity [3–7]. There are two clades of the disease, namely, the Congo Basin clade which comprises MPXV isolates which are from Gabon, DRC, Republic of the Congo and the Cameroon, while on the other hand, isolates from Nigeria, Liberia and imports to United States from Ghana is referred to as the West African clade [5–7]. Of the two clades, the latter appears to be significantly less virulent and transmissible than the other, though the two of them appear not as virulent as variola [3].

Surveillance program were initiated in the DRC from 1981-1986 by the WHO as result of concern that the disease might fill the niche vacated by smallpox (viriola). From this program, though there was limitation brought about by lack of a robust MPXV antibody-specific immunoassay and similarity between Varicella Zoster Virus (VZV) and human Monkeypox virus, there was the identification of 404 human Monkeypox cases [1–3]. The good news is that, there is the reports of decline in reported cases of human Monkeypox virus disease at the conclusion of the surveillance program, from 1986-1992, only a paltry cases of 13 infections was reported, while from 1993-1995, there was no case of infection reported [6, 7]. However, number of reported cases rebounded in 1996 when from the countries in western and central Africa, there was a report of 133 infections while there was 511 reported cases in 1997. Sadly, from 1998-2002, there was a rise in reported number of cases of human Monkeypox infection. though absence of laboratory confirmation which could not be ruled out in some of these reported cases gave indication that some of these cases were as a result of VZV infections [2, 7]. On the other hand, in the spring of year 2003, in western hemisphere, there was emergence of human Monkeypox virus where there was a reportage of a cluster of cases in the United States [8, 9]. At the end of the outbreak, there was a reportage of some 72 cases made up of confirmed cases that were 37 in number in the Midwestern part of United States [9]. Whereas, it was ascertained that the origin of the virus was from importation of rodents from Ghana, however, it was ascertained that the origin of human exposure to the disease was from prairie dogs which were accommodated close to rodents that were infected with the disease prior to when they were taking to pet market [2, 3, 10]. The transmission of the virus to humans during this disease outbreak, from the vectors occurred primarily through direct contact with the vectors by indirect exposure via aerosol or fomites [3, 10].

It is noteworthy to state that illness due to Monkeypox virus is similar to that of smallpox, the illness of the disease which usually takes effect between 1-3 days is characterized with the symptoms that are: tiredness, fever, malaise, lymphadenopathy, and upper respiratory tract illness begin to manifest after incubation period of 10-14 days [2, 11]. This will be followed by 0.5-1.0 cm diameter lesions rashes over a 2-4 week period which progressed through some stages [1, 3, 11].

This rashes has its origin being trunk from where it spreads to the limbs and occasionally to the oral mucosa and genitalia [2, 3]. The major way human Monkeypox virus disease can be basically differentiated from smallpox (variola) and VZV is unilateral or bilateral lymphadenopathy [1–3]. It is pertinent to note the fact that arising from complications form human Monkeypox virus disease, the individuals under its attack will suffer from recurrent fever coagulation disorders, ocular lesions, secondary skin or soft tissue infection, encephalitis and multi-organ failure resulting to fatal outcome which is mortality commonly estimated to be 10% [2, 3, 11]. Indication from relevant data is that primates act only as incidental hosts while reservoir hosts are rodent species indigenous to West and Central Africa [12–15]. In the United States, the vaccine that has been approved and licensed for use is JYNNEOSTM also known as Imvamune or Imvanex while for its treatment for now, smallpox drugs such as tecovirimat (TPOXX) are adopted since there is no clinically proven drug for Monkeypox treatment for now [15]. Tremendous efforts on how human Monkeypox virus is transmitted in a heterogeneous population have been made, some of which includes Emeka *et al.* [16] who developed a model with vaccine compartment to study the dynamics of this disease. Sulaiman *et al.* [17] proposed a mathematical model incorporating control strategies made of combined vaccine and treatment parameters. Olumuyiwa *et al.* [18] formulated a model that considered human and rodent populations. Bankuru *et al.* [19] established a two-population model; including both the squirrel and human populations.

Whereas, there are many published works on models for animal-to-human and human-to-animal transmission of MPXV such as those in Refs. [15, 16] among others, despite the fact that there exist in literature human-to-human transmission of MPXV [15], to the best of our knowledge, only very few researchers have proposed epidemiological models on human-to-human transmission dynamics of MPXV. They include Olumuyiwa *et al.* [20] who formulated model with control strategies to combat the human-to-human spread of Monkeypox and mitigate the burden associated with the deadly disease. Olumuyiwa *et al.* [21] proposed an epidemiological model incorporating some effective optimal control strategies through which the deadly disease can be controlled in a human population. They went further to procure cost effective strategies that helped extend the frontiers of the control of transmission dynamics of Monkeypox disease in a region where the disease is endemic. Alakunle *et al.* [22] and Grant *et al.* [23] contributed significantly to formulation of model to control human-to-human spread of Monkeypox disease. It then becomes highly imperative that we extend studies in this area. Consequently, the key

contributions of this work is as follows:

(i) We carefully did a model-building that captures the transmission dynamics of human-to-human MPXV in the presence of vaccine, in a heterogeneous population.

(ii) The model so built was analysed for its features: showing positivity of its state variables and feasibility of the solution of the model at all times.

(iii) Thorough analysis of the vaccination model for its local and global asymptotic stability so as to ascertain the threshold that defines the spread of the disease in the heterogeneous population under study.

(iv) Numerical simulation of the model was carried out so as to corroborate the analytical results earlier obtained.

(v) A robust interpretation of the plots obtained from the numerical simulation of the model was done so as to draw meaningful epidemiological inferences from the study that helped in making recommendations to policy makers towards mitigating the effect of the affliction of the disease.

Organisation of the remaining part of the article are: Section 2 deals with model building. We carried out theoretical analysis of the model in Section 3, bifurcation analysis in Section 4 while Section 5 is all about numerical simulations of the model so as to corroborate some of the analytic findings. In Section 6, we presented the discussion, recommendations and conclusion of this work.

2. Model building

In the society under consideration the total number of individuals at time t, represented by $N(t)$, is broken into groups of susceptible new-borns $S(t)$, susceptible individuals vaccinated against the disease $V(t)$, exposed people latently infected $E(t)$, infected people that are asymptomatic (not showing symptoms of the disease) $I_A(t)$, people that are symptomatic $I_S(t)$, and those recovered from the disease $R(t)$. Consequently, we have:

$$N(t) = S(t) + V(t) + E(t) + I_A(t) + I_S(t) + R(t).$$

We incorporate parameters as follows. The population of susceptible S grows with a recruitment rate π (as an effect of the influx of new-borns), and is reduced by progression of susceptible individuals to vaccinated individuals at a per capita rate of α. Furthermore, the per capita rate of susceptible that become infected is λ, and the per capita death rate for the susceptible as well as for all other compartments is μ. The per capita progression rate from the class of exposed individuals to the class of (either asymptomatic or symptomatic) infected individuals is denoted with σ. The population of class I_A is reduced by individuals who progress from this class to the class I_S of symptomatic individuals, at a per capita rate γ; reduction also occurs at a per capita rate of δ due to death by affliction of the disease. The recovery rate from the asymptomatic and symptomatic infectious classes by virtue of treatment are represented by K_A and K_s, respectively. The fraction of people who progressed from class E to I_A is represented with η. Adopting the established notations and parameters, the vaccination model for human Monkeypox virus is given by:

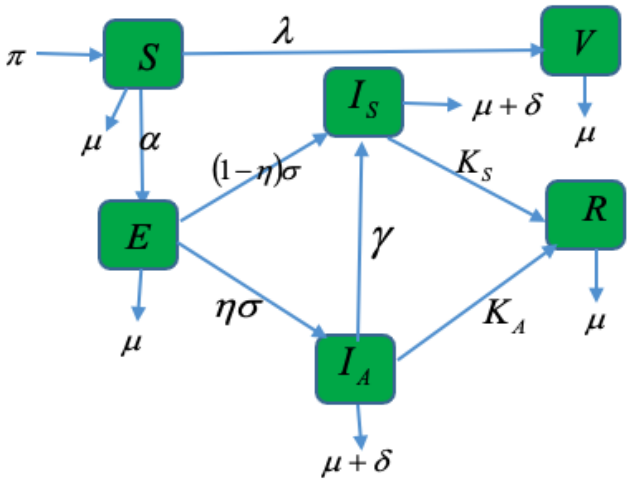

Figure 1. Schematic diagram of the model, showing state variables and parameters on each directed arrow.

$$\begin{aligned}
\frac{dS}{dt} &= \pi - (\alpha + \mu) S - \lambda S, \\
\frac{dV}{dt} &= \alpha S - \mu V, \\
\frac{dE}{dt} &= \lambda S - (\sigma + \mu) E, \\
\frac{dI_A}{dt} &= \eta\sigma E - (\gamma + K_A + \delta + \mu) I_A, \\
\frac{dI_S}{dt} &= (1 - \eta)\sigma E + \gamma I_A - (K_S + \delta + \mu) I_S, \\
\frac{dR}{dt} &= K_A I_A + K_S I_S - \mu R,
\end{aligned} \tag{1}$$

where

$$\lambda = \frac{\beta (I_A + I_S)}{N}. \tag{2}$$

The schematic diagram for model (1) is depicted in Figure 1, and the description of variables used in the model building and their corresponding values are presented in Table 1.

2.1. Model assumptions

(i) Vaccinated susceptible individuals (S) either receive the vaccine for the virus, thus acquiring vaccine-induced immunity or naturally recover from the virus after initially having been infected, or they may also die.

(ii) Once any individual take the vaccine it does not wane, thus recovery from the scourge of the virus confers permanent immunity against re-infection of the virus [17, 19].

Table 1. Description of the state Variables and Parameters.

Variable	Interpretation	Values (source)
$S(t)$	Susceptible individuals at any time t.	-
$V(t)$	Vaccinated Individuals.	-
$E(t)$	Number of individuals latently infected.	-
$I_A(t)$, $I_S(t)$	Individuals that are asymptomatically infectious and symptomatically infectious, respectively.	-
$R(t)$	Number of individuals who recovered from the disease.	-
Parameters	Interpretation	Values (source)
π	Recruitment rate	0.029 [16]
α	Vaccination rate	0.0455 [16]
λ	Force of infection	-
β	Transmission rate	0.00063 [16]
σ	Progression rate from exposed to infectious class	0.095 [16]
η	Part of individuals that progresses from the exposed class to the class of asymptomatically infected individuals	0.43 [17]
K_A, K_S	Recovery rate from the class of asymptomatically infected individuals and symptomatically infected individuals by virtue of treatment, respectively	0.0023 [16] 0.0015 [17]
γ	Progression rate from asymptomatic infectious to symptomatic infectious class	0.51 [17]
δ	Disease-induced death rate	0.1 [17]
μ	Natural death rate	0.000312 [16]

(iii) There are several reasons why individuals can miss vaccination for human Monkeypox; these include but are not limited to logistics, poor public health campaign (as it often occurs in some African countries), economics and religious sentiments.

(iv) Those in the exposed class (E) are individuals that are infected when they make contact with an infected individual. Thus, those in classes I_A and I_S comprises of those cohorts non-vaccine-derived human Monkeypox cases.

(v) Against a breakthrough infection, the vaccine against the virus is supposed to be perfect [17, 19].

(vi) There is natural death in all groups, which occurs at the same rate.

(vii) The death rates arising from affliction of the disease occurs only in the two classes of infected individuals I_A and I_S, and are taken to be the same.

2.2. Basic properties

In order for the established model (1) to be biologically valid, it is pertinent to show that the values of the variables used in the model formulation will be non-negative at all-time $t \geq 0$, as follows:

Given that the number of people living in the community under study at a time t is given by:

$$N(t) = \{(S(t) + V(t) + E(t) + I_A(t) + I_S(t) + R(t)) \geq 0\} \in R_+^6,$$

and

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dV}{dt} + \frac{dE}{dt} + \frac{dI_A}{dt} + \frac{dI_S}{dt} + \frac{dR}{dt}. \tag{3}$$

We consider the theorem below in order to obtain this region.

Theorem 1. *For the system (1) to have all its solutions feasible for all time $t > 0$, then the solution must be in the invariant region Ω, given by:*

$$\Omega = \left\{ \mathcal{I} \in R_+^6 : \ S > 0, V > 0, \ E > 0, I_A > 0, I_S > 0, \ R > 0, N \leq \frac{\pi}{\mu} \right\},$$

where $\mathcal{I} = (S, V, E, I_A, I_S, R)$.

Proof. Let $\Omega = (S, \ V, E, I_A, I_S, R) \in R_+^6$ be any solution with non-negative initial conditions. Suppose there is no disease-induced death (*i.e* $\delta = 0$). Equation (3) becomes:

$$\frac{dN}{dt} + \mu N \leq \pi.$$

Finding the integrating factors, $(IF) = e^{\int \mu dt} = e^{\mu t}$; we have:

$$\frac{d(Ne^{\mu t})}{dt} \leq \pi e^{\mu t}.$$

Integrating both sides yield:

$$N \leq \frac{\pi}{\mu} + ce^{-\mu t}, \tag{4}$$

where c is a constant of integration. Applying the initial conditions; where $t = 0$, $N(0) = N_0$, the inequality holds $N_0 - \frac{\pi}{\mu} \leq c$, then eqn. (4) becomes;

$$N \leq \frac{\pi}{\mu} + \left(N_0 - \frac{\pi}{\mu} \right) e^{-\mu t}. \tag{5}$$

Applying the theorem of inequality in Birkhoff *et al.* [24], we obtain $0 \leq N \leq \frac{\pi}{\mu}$ as $t \to \infty$. The total population approaches $k = \frac{\pi}{\mu}$ as $t \to \infty$, which is commonly termed as the carrying

capacity. Therefore, the feasible solution set of the model falls within the region Ω. Hence, the established model is biologically feasible, for all $N > \frac{\pi}{\mu}$. Then $N < 0$ implies the population diminishes asymptotically to the carrying capacity and for all $N \leq \frac{\pi}{\mu}$, all solutions whose initial conditions are contained in the region Ω will remain there at all time when $t > 0$, so the model is said to be well posed in Ω. Hence, the region Ω is said to be positively-invariant and biologically valid. □

3. Theoretical analysis

Next, we analyzed theoretically the stability (i.e., locally and globally) of the proposed model.

3.1. Local Asymptotic stability of Disease-free Equilibrium

The Disease-Free Equilibrium (DFE) of the model for Monkeypox is its disease-free state, that is, a point at which there is absence of disease in the system [18]. In the absence of Monkeypox infection, all infection-related parameters are set to zero, i.e., $E = I_A = I_s = 0$.
For example, we derive the DFE for the susceptible class as follow,

$$\frac{dS}{dt} = \pi - (\alpha + \mu) S = 0,$$

$$\pi - (\alpha + \mu) S = 0,$$

$$\therefore S^* = \frac{\pi}{\alpha + \mu}.$$

In a similar reasoning, we compute the DFE for every other compartment in equation (1). See Sowole *et al.* [25] for a step-by-step guide on DFE computation.
Given that the DFE of the system (1) is,

$$\varepsilon_0 = (S^*, V^*, E^*, {I_A}^*, {I_S}^* R^*) = \left(\frac{\pi}{\alpha + \mu}, \frac{\mu\alpha\pi + \alpha\pi}{\mu(\alpha + \mu)}, 0, 0, 0, {}^1\!/\!_\mu V^*\right).$$

The local stability of DFE (ε_0) can be determined using the next generation matrix approach [26]. Thus,

$$F = \begin{pmatrix} 0 & \frac{\beta S^*}{S^*+V^*+R} & \frac{\beta S^*}{S^*+V^*+R} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

and

$$V = \begin{pmatrix} P_1 & 0 & 0 \\ -\eta\sigma & P_2 & 0 \\ -P_3 & -\gamma & P_4 \end{pmatrix},$$

where $P_1 = (\sigma + \mu)$, $P_2 = (\delta + \gamma + K_A + \mu)$, $P_3 = (1 - \eta)\sigma$ and $P_4 = (\delta + K_S + \mu)$.
The threshold quantity called the basic reproduction number R_0 is defined as:

$$R_0 = \rho\left(FV^{-1}\right),$$

where ρ is the largest eigenvalue of FV^{-1}. Hence,

$$R_0 = \frac{\beta\mu\left[\alpha+\mu\right]\left[\eta\sigma(\delta+\gamma+\kappa_S+\mu)+(1-\eta)\sigma(\delta+\gamma+\kappa_A+\mu)\right]}{\mu(\alpha+\mu)(\rho+\mu)(\delta+\gamma+\kappa_A+\mu)(\delta+\kappa_S+\mu)}. \quad (6)$$

In mathematics, R_0 is a threshold parameter for the stability of DFE and is closely related to an epidemic's final size and its peak. The reproduction number R_0 can be defined as the expected number of subsequent cases of infection in a susceptible population as a result of the primary case [27]. If $R_0 < 1 (R_0 > 1)$, then the number of infected persons brought into a susceptible population will on average die out/reduce (spread out/increase). Yet, in many epidemiological models, the prevalence of infected hosts and the final size of the epidemic are brought about by increase in R_0 making it a useful measure of spread. To check the stability of the model, we will apply the Theorem 2 of Van den Driessche *et al.* [26].

Theorem 2. *The Disease-free equilibrium (ε_0) of the model (1) is Locally-Asymptotically stable (LAS) if $R_0 < 1$ and unstable if $R_0 > 1$.*

Proof. The Jacobian matrix of system (1) evaluated at ε_0 is given as:

$$J_1(\varepsilon_0) = \begin{bmatrix} -(\alpha+\mu) & 0 & 0 & \frac{\beta S^*}{S^*+V^*+R^*} & \frac{\beta S^*}{S^*+V^*+R^*} & 0 \\ \alpha & -\mu & 0 & 0 & 0 & 0 \\ 0 & 0 & -P_1 & \frac{\beta S^*}{S^*+V^*+R^*} & \frac{\beta S^*}{S^*+V^*+R^*} & 0 \\ 0 & 0 & \eta\sigma & -P_2 & 0 & 0 \\ 0 & 0 & P_3 & \gamma & -P_4 & 0 \\ 0 & 0 & 0 & \kappa_A & \kappa_S & -\mu \end{bmatrix},$$

where $P_1 = (\sigma + \mu), P_2 = (\delta + \gamma + K_A + \mu), P_3 = (1 - \eta)\sigma$ and $P_4 = (\delta + K_S + \mu)$.
It is necessary to show that the entire Eigen values of the Jacobian matrix at (ε_0) is negative. The second column of $J_1(\varepsilon_0)$ contain only the diagonal term which form the negative eigenvalue $-\mu$ needed. Therefore, the other five eigenvalues can be obtained from the sub-matrix $J_2(\varepsilon_0)$, which is formed by excluding the second row and column of $J_1(\varepsilon_0)$. We have:

$$J_2(\varepsilon_0) = \begin{bmatrix} -(\alpha+\mu) & 0 & 0 & \frac{\beta S^*}{S^*+V^*+R^*} & \frac{\beta S^*}{S^*+V^*+R^*} \\ \alpha & -\mu & 0 & 0 & 0 \\ 0 & 0 & -P_1 & \frac{\beta S^*}{S^*+V^*+R^*} & \frac{\beta S^*}{S^*+V^*+R^*} \\ 0 & 0 & \eta\sigma & -P_2 & 0 \\ 0 & 0 & P_3 & \gamma & -P_4 \end{bmatrix}.$$

Similarly, the second column of $J_2(\varepsilon_0)$ contain only the diagonal term which form the negative eigenvalue $-(\phi + \mu)$ needed. Therefore, the other four eigenvalues can be obtained from the sub-matrix $J_3(\varepsilon_0)$, which is formed by excluding the second row and column of $J_2(\varepsilon_0)$.

$$J_3(\varepsilon_0) = \begin{bmatrix} -(\alpha+\mu) & 0 & 0 & \frac{\beta S^*}{S^*+V^*+R^*} & \frac{\beta S^*}{S^*+V^*+R^*} \\ 0 & 0 & -P_1 & \frac{\beta S^*}{S^*+V^*+R^*} & \frac{\beta S^*}{S^*+V^*+R^*} \\ 0 & 0 & \eta\sigma & -P_2 & 0 \\ 0 & 0 & P_3 & \gamma & -P_4 \end{bmatrix}.$$

The first column of $J_3(\varepsilon_0)$ contain only the diagonal term which form the negative eigenvalue $-(\alpha+\mu)$ needed. Hence, the other three eigenvalues can be obtained from the sub-matrix $J_4(\varepsilon_0)$, which is formed by excluding the first row and column of $J_3(\varepsilon_0)$. We obtained

$$J_4(\varepsilon_0) = \begin{bmatrix} -P_1 & \frac{\beta\mu(\alpha+\mu)}{\mu(\alpha+\mu)} & \frac{\beta\mu(\alpha+\mu)}{\mu(\alpha+\mu)} \\ \eta\sigma & -P_2 & 0 \\ P_3 & \gamma & -P_4 \end{bmatrix}.$$

The eigenvalues of the matrix $J_4(\varepsilon_0)$ are the root of the characteristic polynomial given below:

$$\lambda^3 + a_1\lambda^2 + a_2\lambda + P_1P_2P_4(1-R_0) = 0, \tag{7}$$

where

$$a_1 = P_1 + P_2 + P_4, a_2 = P_1(P_2+P_4) + P_2P_4 - \frac{\beta\mu(\alpha+\mu)(\eta\sigma+P_3)}{\mu(\alpha+\mu)}.$$

Applying the Routh-Hurwitz criterion [28], which asserts that all roots of the polynomial (7) have negative real parts if and only if the coefficient $a_1 > 0$, $P_1P_2P_4 > 0$ and $a_1a_2 > P_1P_2P_4$. All these conditions will be satisfied if $R_0 < 1$. Therefore, by Routh-Hurwitz criterion, the DFE of Model (1) is locally asymptotically stable. □

Epidemiological significance of the Theorem 2 is that whenever the reproduction number of the disease is less than unity, an influx of at least a single Monkeypox infected individuals will will fail to replicate itself and consequently will not lead to epidemic of the disease in the human population.

3.2. Global Asymptotic Stability

Consider the following theorem:

Theorem 3. *The DFE of the model (1) is globally asymptotically stable if $R_0 \le 1$ and unstable if $R_0 \ge 1$.*

Proof. Constructing a Lyapunov function as follows:

$$\begin{aligned} V &= [\mu(\alpha+\mu)(\eta\sigma(\gamma+P_4)+P_2P_3)]E \\ &+ [(\alpha+\mu)\mu P_1(\gamma+P_4)]I_A \\ &+ [(\alpha+\mu)\mu P_1P_2]I_S, \end{aligned}$$

where, $P_1 = (\sigma+\mu)$, $P_2 = (\delta+\gamma+K_A+\mu)$, $P_3 = (1-\eta)\sigma$ and $P_4 = (\delta+K_S+\mu)$,

$$\begin{aligned} \dot{V} &= [\mu(\alpha+\mu)(\eta\sigma(\gamma+P_4)+P_2P_3)]\dot{E} \\ &+ [(\alpha+\mu)\mu P_1(\gamma+P_4)]\dot{I}_A \\ &+ [(\alpha+\mu)\mu P_1P_2]\dot{I}_S, \\ \dot{V} &= [\mu(\alpha+\mu)(\eta\sigma(\gamma+P_4)+P_2P_3)](\lambda S - P_1E) \\ &+ [(\alpha+\mu)\mu P_1(\gamma+P_4)](\eta\sigma E - P_2I_A) + [(\alpha+\mu)\mu P_1P_2] \\ &+ [(\alpha+\mu)\mu P_1P_2](P_3E + \gamma I_A - P_4I_S), \\ \dot{V} &= [\mu(\alpha+\mu)(\eta\sigma(\gamma+P_4)+P_2P_3)](\lambda S) \\ &- [(\alpha+\mu)\mu(\eta\sigma(\gamma+P_4))]P_1E \\ &+ ((\mu(\alpha+\mu)P_1(\gamma+P_4))\eta\sigma E - (\alpha+\mu)\mu P_1(\gamma+P_4))P_2I_A \\ &+ ((\alpha+\mu)\mu P_1P_2)P_1E \\ &+ ((\alpha+\mu)\mu P_1P_2)\gamma E - ((\alpha+\mu)\mu P_1P_2)P_4I_S, \\ \dot{V} &= [\mu(\alpha+\mu)(\eta\sigma(\gamma+P_4)+P_2P_3)](\lambda S) \\ &- [(\alpha+\mu)\mu P_1P_2P_4](I_A+I_S), \end{aligned}$$

note that $S(t) \le N(t)$ in Ω, so that

$$\begin{aligned} \dot{V} &\le \beta\mu(\alpha+(\alpha+\mu))(\eta\sigma(\gamma+P_4)+P_2P_3)(I_A+I_S) \\ &- ((\alpha+\mu)\mu P_1P_2P_4)(I_A+I_S), \\ &= (I_A+I_S)((\alpha+\mu)\mu P_1P_2P_4)\left(\frac{P_X}{\mu(\alpha+\mu)P_1P_2P_4} - 1\right), \end{aligned}$$

where $P_X = \beta\mu(\alpha+(\alpha+\mu))(\eta\sigma(\gamma+P_4)+P_2P_3)$ and

$$\dot{V} = (I_A+I_S)((\alpha+\mu)\mu P_1P_2P_4)(R_0-1) \le 0, \tag{8}$$

for $R_0 \le 1$. □

Since we have been able to show that the established model parameters will never be negative at all time, consequently, $\dot{V} \le 0$ when $R_0 \le 1$. Therefore, V is a Lyapunov function in the invariant region. By the LaSalle's invariance principle [29], every solution to systems (1), with condition in the invariant region, will approach ε_0 as $t \to \infty$. Arising from Theorem 3 is the fact that, epidemiologically, for the elimination of this disease, Monkeypox virus from the population, the necessary and sufficient condition is that $R_0 \le 1$. This means that the disease will be eradicated from the population so long that the value of reproduction number can be kept less than unity.

3.3. Endemic Equilibrium (EE)

The endemic equilibrium (EE) can be derived for the model (1) when $\left(S^{**}, V^{**}, E^{**}, I_A^{**}, I_S{}^{**}, R^{**}\right) = 0$. To establish the necessary conditions for endemic equilibrium point, we solve the system (1) in terms of the force infection, that is,

$$\lambda^{**} = \frac{\beta(I_A{}^{**} + I_S{}^{**})}{N^{**}}, \tag{9}$$

with

$$\begin{aligned} S^{**} &= \frac{\pi}{\alpha+\mu}, \\ V^{**} &= \frac{1}{\mu}\left(\frac{\mu\alpha\pi + \alpha\pi}{(\alpha+\mu)\mu^2}\right), \\ E^{**} &= \frac{\lambda^{**}\pi(\lambda^{**}+\mu+\alpha)}{(\lambda^{**}+\mu)(\lambda^{**}+\alpha+\mu)P_1}, \\ I_A{}^{**} &= \frac{\lambda^{**}\pi\eta\sigma(\lambda^{**}+\mu+\alpha)}{(\lambda^{**}+\mu)(\lambda^{**}+\alpha+\mu)P_1P_2}, \\ I_S{}^{**} &= \frac{\lambda^{**}\pi(\lambda^{**}+\mu+\alpha)(\eta\sigma\gamma+P_2P_3)}{(\lambda^{**}+\mu)(\lambda^{**}+\alpha+\mu)P_1P_2}, \\ R^{**} &= \frac{Z_1 + \pi P_1P_2P_4[\alpha(\lambda^{**}+\mu)+\alpha]}{(\lambda^{**}+\mu)(\lambda^{**}+\alpha+\mu)\mu P_1P_2P_4}, \end{aligned} \tag{10}$$

and $P_1 = (\sigma+\mu)$, $P_2 = (\delta+\gamma+K_A+\mu)$, $P_3 = (1-\eta)\sigma$,$P_4 = (\delta+K_S+\mu)$, and

$Z_1 = \lambda^{**}\pi(\sigma+\mu)(\lambda^{**}+\mu+\alpha)((\eta\sigma\gamma+P_2P_3)\kappa_S+\eta\sigma\kappa_A P_4)$. Substituting the variables in eqn. (10) into eqn. (9), we obtain:

$$f(\lambda^{**}) = A(\lambda^{**})^2 + B\lambda^{**} + C = 0, \tag{11}$$

where

$$A = \pi(\sigma+\mu)((\eta\sigma\gamma+P_2P_3)(\kappa_S+\mu) + \eta\sigma\kappa_A P_4 + \mu P_4(P_2+\eta\sigma)),$$

$$B = \pi(\sigma+\mu)((\mu+\alpha)(\eta\sigma\gamma+P_2P_3)(\kappa_S+\mu) + \eta\sigma\kappa_A P_4 + \mu P_4(P_2+\eta\sigma)) + P_1P_2P_4(\alpha+\mu) - \beta\mu(\eta\sigma\kappa_A P_4 + \eta\sigma\gamma + P_2P_3),$$

$$C = \pi(\alpha+\mu)(\sigma+\mu)\mu P_1P_2P_4(1-R_0).$$

From eqn. (11), the root $\lambda^{**} = 0$ corresponds to the DFE point (in Subsection 3.1).

It follows from here that, the model (1) whose equilibria are different from zero is satisfied by eqn. (11). Consequently, regardless of the sign of B in eqn. (11), the quadratic equation has a unique and non-negative root for all $R_0 > 1$.

Remark 1. *From polynomial in equation (11), it could be observed that B has a positive coefficient always, and so do C whenever $R_0 < 1$, while it will be negative whenever $R_0 > 1$ respectively. Structure of the polynomial (11) is suggestive of the phenomenon of bifurcation, which is typically characterized by existence of a stable DFE and a stable EE at the same time whenever the associated reproductive number is not up to 1. The major public health implication of this phenomenon is that the classical epidemiological requirement of having $R_0 < 1$, as necessary as it is, it is no longer sufficient for the effective and adequate control of the spread of the disease in the given population under this scenario.*

4. Bifurcation analysis

A bifurcation can be described as a qualitative change in the nature of the solution trajectories of epidemiological models occasioned by a parameter change. The bifurcation point is the point at which this change occurs. To inquire into the nature of the bifurcation exhibited by our model, we consider the method introduced by Castillo-Chavez *et al.* [30].

Theorem 4. *Given a general system of ordinary differential equations with parameter φ:*

$$\frac{dx}{dt} = f(x,\varphi), \qquad f: R \to R^n, f \in C^2(R^n \times R), \tag{12}$$

where $x = 0$ is an equilibrium point for eqn. (12). That is, $f(0,\varphi) \equiv 0$ for all φ.
With the following assumption:

$$M_1 : A = D_x f(0,0) = (\partial f/\partial x_j)(0,0),$$

being the value of system given by eqn. (12) when linearized around the equilibrium 0 and φ evaluated at 0. The simple eigenvalue of A is zero while the values of other eigenvalues of A have non-positive real parts.
M_2 : Matrix A has a positive right eigenvector w combine with a left eigenvector corresponding to the zero eigenvalue. Suppose that f_k is the k^{th} component of f and

$$a = \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0) \text{ and}$$

$$b = \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta^*}(0,0). \tag{13}$$

Conventionally, a and b determines the local dynamics of equation (12) around 0.

(i) For $a > 0$, $b > 0$. When parameter $\varphi < 0$ with $|\varphi| << 1$, 0 is locally asymptotically stable and there is existence of a nonnegative unstable equilibrium; when $0 < \varphi << 1$, 0 is unstable and there is existence of a non-positive, locally asymptotically stable equilibrium.

(ii) For $a < 0$, $b < 0$. When parameter $\varphi < 0$with $|\varphi| << 1$, 0 is not stable; when $0 < \varphi << 1$,0 is locally asymptotically stable equilibrium, and there is existence of a nonnegative non-stable equilibrium.

(iii) For $a > 0$, $b < 0$. When $\varphi < 0$ with $|\varphi| << 1$, 0 is not stable, and there is existence of a locally asymptotically stable non-positive equilibrium, when $0 < \varphi << 1$, 0 is stable, and there is an appearance of a positive non-stable equilibrium.

(iv) For $a < 0$, $b > 0$. When there is a change in the value of φ from negative to positive, 0 changes its stability from stable to unstable. Corresponding non-positive non-stable equilibrium becomes positive and locally asymptotically stable.

In particular, we have forward bifurcation if $a < 0$ and $b > 0$, while we have backward bifurcation if $a > 0$ and $b > 0$. By using the approach stated above as contained, we claimed the following result.

Theorem 5. *The model (1) exhibits backward bifurcation at R_0=1 whenever a bifurcation coefficient as denoted by a in theorem 4 is positive.*

Proof. Let $\varepsilon_a = (S^{**}, V^{**}, E^{**}, I_A{}^{**}, I_S{}^{**}, R^{**})$ denotes an arbitrary endemic equilibrium point of the complete model system in model (1). We investigate the existence of backward bifurcation using the centre manifold theory [30].
For convenience, we carry out the following change of variables before applying the theory: let $S = x_1, V = x_1, E = x_3, I_A = x_4, I_S = x_5$, and $R = x_6$ so that the total population becomes:

$$N = \sum_{i=1}^{6} x_i,$$

It then follows that the model (1) can be rewritten as:

$$\dot{x_1} = \pi - (\alpha+\mu)x_1 - \lambda x_1 \equiv f_1,$$

$$\begin{aligned}
\dot{x_2} &= f\alpha - (\rho+\mu)\,x_2 \equiv f_2,\\
\dot{x_4} &= \lambda x_1 - (\sigma+\mu)\,x_4 \equiv f_4,\\
\dot{x_5} &= \eta\sigma x_4 - (\gamma + K_A + \delta + \mu)\,x_5 \equiv f_5,\\
\dot{x_6} &= (1-\eta)\,\sigma x_4 + \gamma x_5 - (K_S + \delta + \mu)\,x_6 \equiv f_6,\\
\dot{x_7} &= K_A x_5 + K_S x_6 + \rho x_2 - \mu x_7 \equiv f_7,
\end{aligned} \tag{14}$$

where $\lambda = \frac{\beta(x_5 + x_6)}{\sum_{i=1}^{6} x_i}$.

We consider the case with $\beta = \beta^*$, a bifurcation parameter. By solving for $\beta = \beta^*$ from R_0 yields:

$$\beta = \beta^* = \frac{(\alpha+\mu)(\phi+\mu)P_1P_2P_4}{\mu\left((1-f)\alpha\theta + (\phi+\mu)\right)(\eta\sigma(P_4+\gamma) + P_2P_3)}, \tag{15}$$

where $P_1 = (\sigma+\mu)$, $P_2 = (\delta+\gamma+K_A+\mu)$, $P_3 = (1-\eta)\,\sigma$ and $P_4 = (\delta + K_S + \mu)$.

Evaluating the Jacobian of the transformed system (13) estimated at DFE (ε_0) with $\beta = \beta^*$ to obtain:

$$J^* = J(\varepsilon_0)|_{\beta\neq\beta^*} = \begin{bmatrix}
-(\alpha+\mu) & 0 & 0 & \frac{\beta^* x_1^*}{x_1^*+x_2^*+x_3^*} & \frac{\beta^* x_1^*}{x_1^*+x_2^*+x_3^*} & 0\\
\alpha & -\mu & 0 & 0 & 0 & 0\\
0 & 0 & -P_1 & \frac{\beta^* x_1^*}{x_1^*+x_2^*+x_3^*} & \frac{\beta^* x_1^*}{x_1^*+x_2^*+x_3^*} & 0\\
0 & 0 & \eta\sigma & -P_2 & 0 & 0\\
0 & 0 & P_3 & \gamma & -P_4 & 0\\
0 & 0 & 0 & \kappa_A & \kappa_S & -\mu
\end{bmatrix},$$

with $P_1 = (\sigma+\mu)$, $P_2 = (\delta+\gamma+K_A+\mu)$, $P_3 = (1-\eta)\,\sigma$ and $P_4 = (\delta + K_S + \mu)$.

The matrix J^* above has a simple zero eigenvalue and the remaining eigenvalues having real part, indicating that the *"center manifold theory"* is applicable. It is noted that matrix J^* has a right eigenvector given by: $w = (w_1, w_2, ...w_7)^T$, where

$$\begin{aligned}
w_1 &= -\frac{\mu\beta^*(\eta\sigma(P_4+\gamma) + P_2P_3)}{(\alpha+\mu)^2 P_2P_4}w_3,\\
w_2 &= -\frac{\mu\beta^*(\eta\sigma(P_4+\gamma) + P_2P_3)(\alpha\mu + (\alpha+\mu)\alpha)}{\mu^2(\alpha+\mu)\rho(\rho+\mu)P_2P_4}w_3,\\
w_3 &> 0, w_4 = -\frac{\eta\sigma}{P_2}w_3,\\
w_5 &= -\frac{(\eta\sigma\gamma + P_2P_3)}{P_2P_4}w_3,\\
w_6 &= -\frac{\mu^2(\alpha+\mu)^2(\rho+\mu)(\eta\sigma(P_4+\gamma)+P_2P_3) - \mu\rho\beta^*(\alpha\mu+\alpha(\alpha+\mu))(\eta\sigma(P_4+\gamma)+P_2P_3)}{\mu^2(\alpha+\mu)^2(\rho+\mu)P_2P_4}w_3.
\end{aligned}$$

Similarly, J^* has a left Eigen vectors $v = (v_1, v_2, \ldots, v_6)$ satisfying $v \cdot w = 1$ with

$$\begin{aligned}
v_1 = 0,\ v_2 = 0,\ v_3 = v_3 > 0,&\\
v_4 &= -\frac{(\mu(\alpha+\mu)P_1P_4 - \mu\beta^*P_3(\alpha+\mu))}{\mu(\alpha+\mu)\eta\sigma P_4}v_3,\\
v_5 &= -\frac{\mu\beta^*(\alpha+\mu)}{\mu(\alpha+\mu)P_4}v_3,\\
v_6 &= 0.
\end{aligned} \tag{16}$$

It then follows that there is a need to compute the associated partial derivatives of $f(x)$ that are different from zero evaluated at the DFE (ε_0) with $\beta = \beta^*$, by taking into account the expression for $\beta^* = 1$, where v_3 is computed to ensure that the eigenvectors satisfy the condition $v \cdot w = 1$. Since the first two components of v are zero, there is no need for the differential coefficients f_1 and f_2. From the remaining differential coefficients f_4 and f_5 the nonzero are as follows:

$$\frac{\partial^2 f_3}{\partial x_1 \partial x_4}(0,0) = \frac{\partial^2 f_3}{\partial x_4 \partial x_1}(0,0) = \frac{\beta^*}{x_1^*+x_2^*+\ldots x_6^*} - \frac{\beta^* x_1^*}{\left(x_1^*+x_2^*+\ldots x_6^*\right)^2},$$

$$\frac{\partial^2 f_3}{\partial x_1 \partial x_5}(0,0) = \frac{\partial^2 f_3}{\partial x_5 \partial x_1}(0,0) = \frac{\beta^*}{x_1^*+x_2^*+\ldots x_6^*} - \frac{\beta^* x_1^*}{\left(x_1^*+x_2^*+\ldots x_6^*\right)^2} \tag{17}$$

By putting these values in eqn. (17) into eqn. (12) we obtained:

$$a = \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0) = v_4 w_1 w_5 \frac{\partial^2 f_4}{\partial x_1 \partial x_5} + v_4 w_1 w_6 \frac{\partial^2 f_4}{\partial x_1 \partial x_6},$$

where $N^* = {x_1}^* + {x_2}^* + {x_6}^*$.

Consequently, by putting the value of w_1, w_5 *and* w_6 into this and tidy up, we obtained:

$$a = 2v_4 w_4^2 \left(\frac{\beta^* x_1^*}{N^{*2}} - \frac{\beta^*}{N^{*2}}\right)\left(\frac{\mu\beta^*(\eta\sigma(P_4+\gamma)+P_2P_3)}{(\alpha+\mu)^2 P_2P_4}\right)\times \left(\frac{\eta\sigma}{P_2} + \frac{\eta\sigma\gamma + P_2P_3}{P_2P_4}\right). \tag{18}$$

From the reference point, the derivatives f_4, f_5 *and* f_6 are nonzero, and the non-zero derivatives are:

$$\frac{\partial^2 f_3}{\partial x_4 \partial \beta^*}(0,0) = \frac{x_1^*}{x_1^* + x_2^* + \ldots x_6^*} \quad \text{and}$$

$$\frac{\partial^2 f_3}{\partial x_5 \partial \beta^*}(0,0) = \frac{x_1^*}{\left(x_1^* + x_2^* + \ldots x_6^*\right)^2}$$

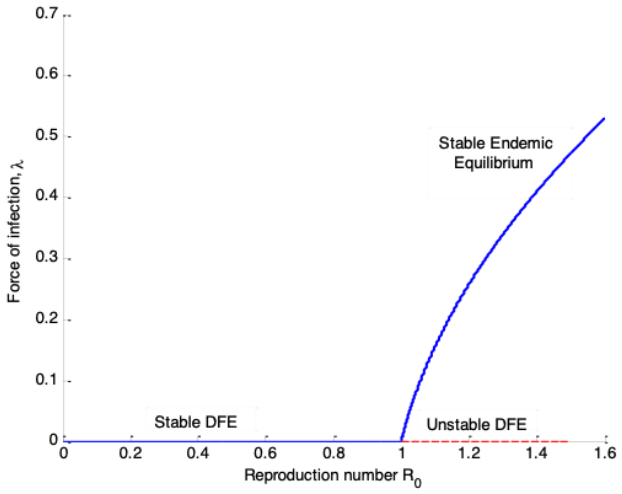

Figure 2. Forward bifurcation diagram for the model showing force of infection against R_0.

By substituting these values into eqn. (12) likewise and noting that $N^* = {x_1}^* + {x_2}^* + {x_6}^*$, to obtain b is as follows:

$$b = \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta^*}(0,0) = v_4 w_5 \frac{\partial^2 f_3}{\partial x_4 \partial \beta^*} + v_4 w_6 \frac{\partial^2 f_3}{\partial x_5 \partial \beta^*}$$
$$= v_4 w_5 \frac{{x_1}^*}{N^{*2}} + v_4 w_6 \frac{{x_1}^*}{N^{*2}},$$

$$\therefore b = v_4 w_4^2 \frac{x_1^*}{N^{*2}} \left(\frac{\eta\sigma\,(P_4 + \gamma) + P_2 P_3}{P_2 P_4} \right) > 0. \tag{19}$$

□

Biologically, one observes that for all feasible parameters, $b > 0$. Consequently, the direction of the bifurcation at $\beta = \beta^*$ for $R_0 = 1$ depends only on the sign of a. From eqn. (18), sincew_5,w_6, andw_1 are positive and if the negative term dominates the positive term, then a will be negative. Hence, there is an exhibition of forward bifurcation by our model and there is existence of not less than one stable endemic equilibrium when $R_0 > 1$. The forward bifurcation exhibited by model (1) is as shown in Figure 2:

5. Numerical results

Using the parameter values as obtained from previous works in the literature as contained in Table 1, and by adopting MATLAB software, we conducted numerical simulation of the model so as to be able to observe the transmission dynamics of human Monkeypox virus model over time. By using this simulation, we were able to do illustration of some theoretical results that were earlier obtained in the previous sections of this study.

Figure 3 verifies the effect of vaccination on each of these classes, invariably it affects the population of the inhabitants of the community under study at DFE. From Figure 3(a) – 3(c), it is observed that the increase in the rate of vaccination on the susceptible, vaccinated, and exposed populations, the slower the rate of progression to infectious classes. The lower the rate of vaccination, the faster the progression to infectious classes. From Figure 3(c), it is observed that among the exposed individuals, it is seen that as many individuals progress from the susceptible class to the exposed class, at increased vaccination rate, it decreases the time it takes for infectiousness with the virus. In Figure 3(d), the observation is that, the number of symptomatically infected individuals decreases exponentially with time. From Figure 3(e), the observation is that, the number of asymptomatically infected individuals increases (grow exponentially) in few days after being infected up to equilibrium level but gradually start decreasing with time perhaps due to the efficacy of the vaccination. From Figure 3(f), it is observed that at high vaccination rate, the number of people who recovered from the Monkeypox infection increases with time.

Intuitively, as vaccine are been administered, the individuals in susceptible class becomes vaccinated, while the vaccinated individuals at equilibrium points decreases. In addition, the susceptible class also experience natural death [17]. The recovered class on the other hand witnessed significant increase in the population as a result of the vaccination intervention.

In Figures 4(a) and 4(f), one would observe that there is significant impact of change in the vaccination rate and recovery from symptomatic infectious class by virtue of treatment of the population of the susceptible population and recovered population as shown in the wide gap between the two curves. Here, with increase in the three parameters, the population of susceptible individuals rapidly decrease exponentially while the population of the recovered individuals grows exponentially. In Figures 4(c) and 4(d), observe that there is very little impact of change in the vaccination rate and recovery from symptomatic infectious class by virtue of treatment of the population of the susceptible population and recovered population as shown in the near overlapping of the two curves. This means that with increase in the two parameter values, the population of Exposed and infected asymptomatic remains almost unchanged. In Figures 4(c) and 4(d), observe that there is no impact of change in the vaccination rate and recovery from symptomatic infectious class by virtue of treatment of the population of the exposed and infected asymptomatic population as shown in the overlapping of the two curves. This means that with increase in the three parameter values, the population of Exposed and infected asymptomatic remains unchanged.

6. Summary, Findings, Recommendations and Conclusion

6.1. Summary

A new deterministic compartmental vaccination model was proposed and rigorously analysed to gaining insight into human-to-human transmission dynamics of Monkeypox virus in a population in sub-Saharan Africa.

Proposed model was discovered to exhibit two equilibria, the disease-free equilibrium which is asymptotically stable (i.e., locally and globally) when the associated reproductive number is less than one and, likewise, it was discovered that when the

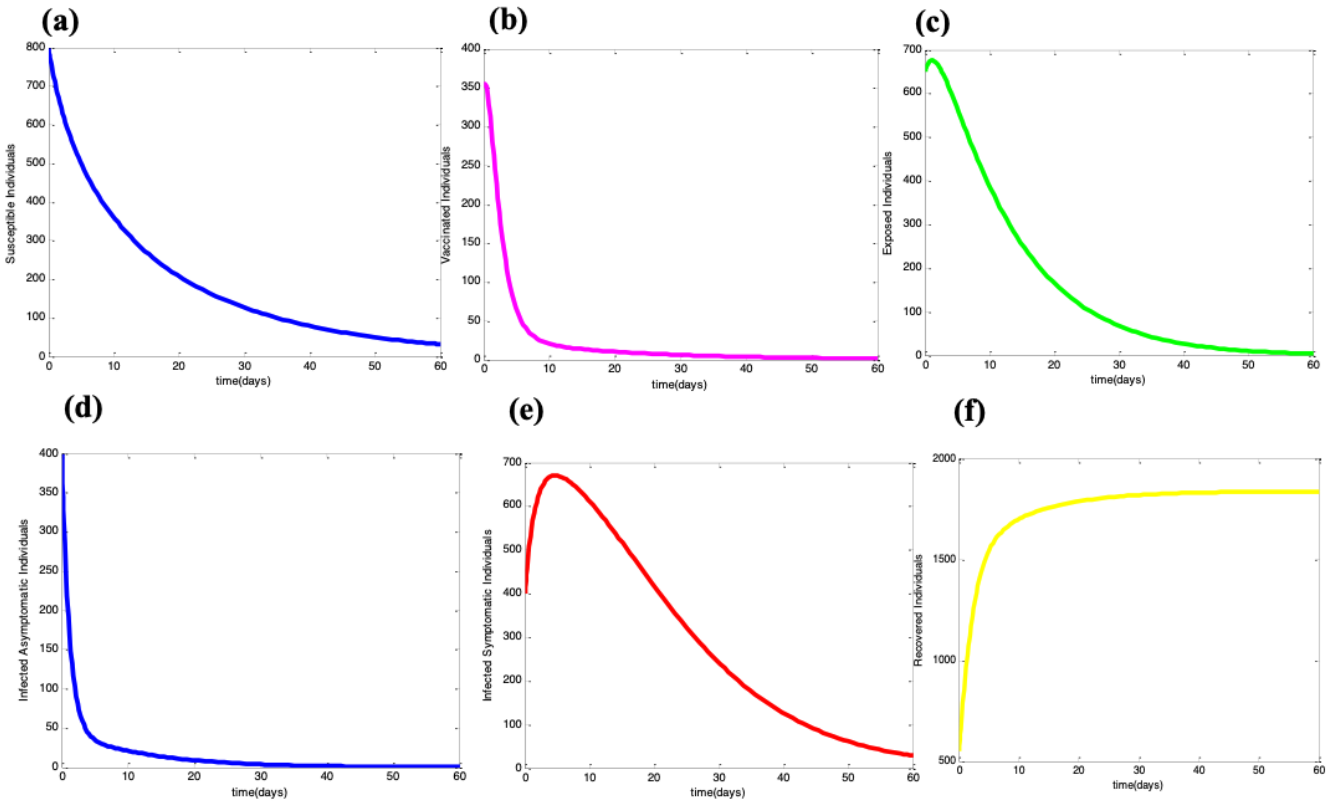

Figure 3. The dynamics of each state variables showing the change in the dynamics of each sub-population with respect to time in the model by using the parameters in Table 1. In the Figure (a) susceptible population, (b) Vaccinated population, (c) exposed population, (d) Simulation of Infected Asymptomatic population, (e) Simulation of Infected symptomatic population, (f) recovered population.

reproductive number was greater than one, the endemic equilibrium is locally asymptotically stable. By adopting parameter values as obtained from the literature, we carried out the numerical simulation of the proposed model so as to confirm some analytical results earlier gotten in the study.

6.2. *Findings*

1) The model was found to undergo forward bifurcation meaning that the classical epidemiological requirement that its reproduction number be less than unity for the control of the disease is not sufficient for its control in the population.

2) It is discovered that among the vaccinated individuals and those that missed vaccination, the difference is clear, in the sense that, among individuals with increased vaccination, the loss of individuals to infectiousness is invariably lower to those with a lower rate of vaccination.

3) Expectedly, it was discovered that at high vaccination rate, the number of people who recovered from the Monkeypox infection increases (grows exponentially) with time.

4) When there is increase in the two parameters: vaccination rate (α) and Recovery rate from symptomatic infectious class by virtue of treatment (K_S), in the model, this has great impact on susceptible population and recovered population; while it is of no impact on exposed population and infected asymptomatic population.

6.3. *Recommendations*

Over the years Monkeypox virus has become a threat and causing great havoc in the developing and under developing countries, especially in sub-Saharan Africa, consequently, arising from this study and findings, it is recommended that:

1) There should be more public awareness campaign on the dangers of Monkeypox and policy makers in the sector should set strategies on how to control its spread and cure.

2) There should be establishment of a strong policy on the vaccination against Monkeypox and make it mandatory in hospitals and health centres for new-born babies and other age categories.

3) Strong health education on personal hygiene should be carried out.

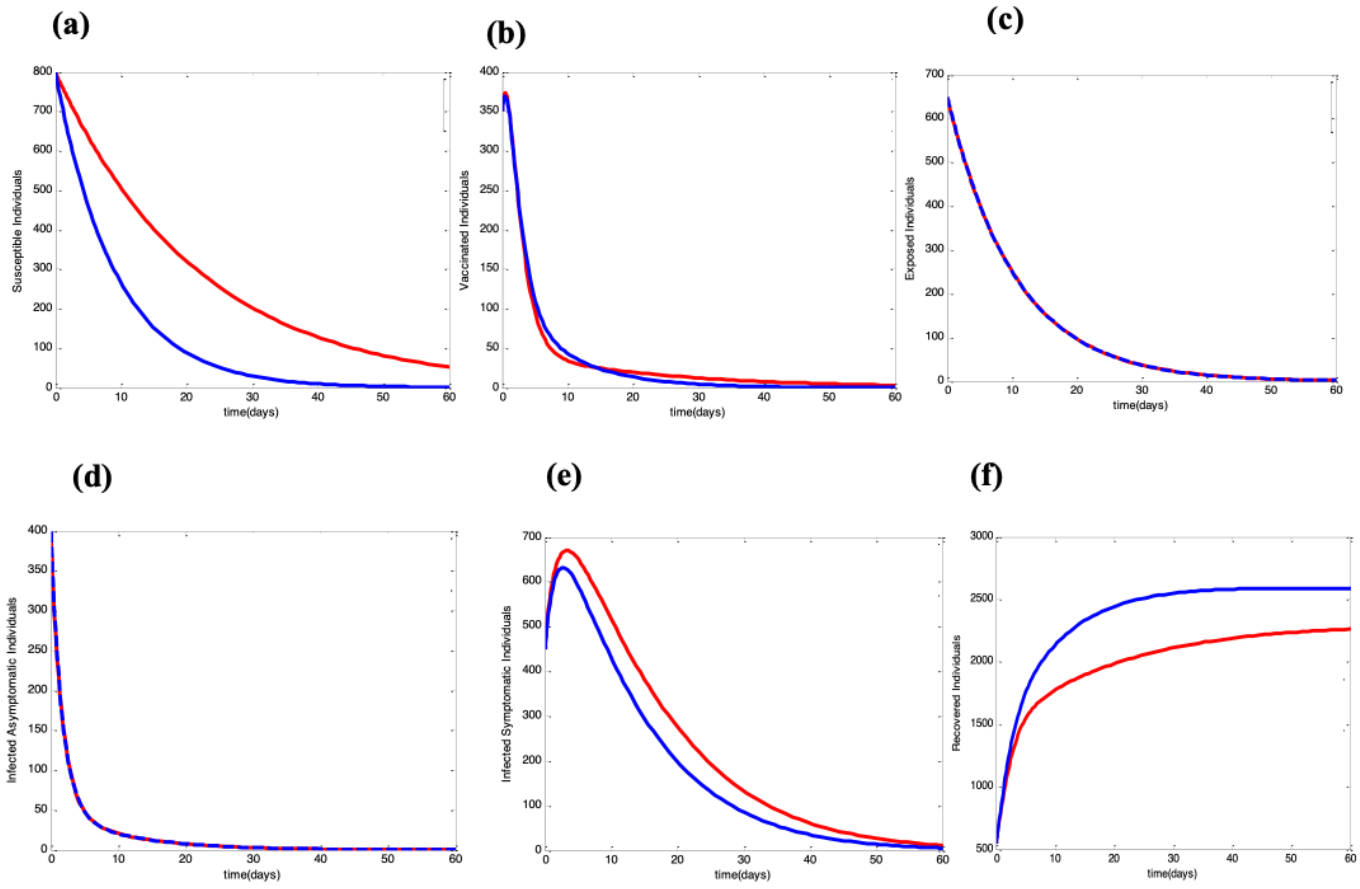

Figure 4. Plots showing the change in the dynamics of each sub-population with respect to time by varying the vaccination rate (α) of the model and Recovery rate from symptomatic infectious class by virtue of treatment (K_S), where panel (a) susceptible population, (b) Vaccinated population, (c) exposed population, (d) Simulation of Infected Asymptomatic population, (e) Simulation of Infected symptomatic population, (f) removed population.

6.4. Conclusion

In this paper, we studied the Monkeypox virus and designed a novel vaccination model for the transmission dynamics of the virus. As a major contribution, we focused on human-to-human transmission model which to the best of our knowledge, this is the first time this is done, thus this is the novelty of our work. We proved theoretically the proposed model stability conditions (i.e., local and global stability) at equilibrium level. It is discovered that the model undergoes forward bifurcation which is described as a qualitative change in the nature of the solution trajectories of epidemiological models occasioned by a parameter change. Based on the findings from the study, expectedly, it is observed that if there are no pharmaceutical interventions (i.e., vaccination) to control the spread of the virus, the virus will not be wiped out of the population. Conclusively, we observed that when efforts are geared at adequate vaccination of individuals in the heterogeneous population, the loss of people to the scourge of the virus would be greatly reduced. Unlike other works that considers human-to-animal models, it is discovered that when there is increase in vaccination rate, vaccination-induced recovery rate and Recovery rate from symptomatic infectious class by virtue of treatment in the proposed model has great impact on susceptible population and recovered population.

In study of dynamics of how communicable diseases are transmitted, scientists have been deploying mathematical models which has proven to be a very useful tool for understanding the dynamics of diseases, works such as those in Refs. [1–7, 19, 25, 31]. In future work, this work can be extended by incorporating time dependent optimal control strategies into the model with a view to procuring optimal strategies to combating the transmission dynamics of the disease. Furthermore, the model can be reformulated as Caputo based or Atagana Baleanu based fractional order model and solve the resulting system of non-linear fractional order model using appropriate numerical schemes towards obtaining more novel findings that will ultimately help in curtailment of the spread of the deadly disease in sub-Saharan Africa.

References

[1] S. Arita, L. Jezek & K. R. Khodakevich, “Human Monkeypox: A newly emerged orthopox virus zoonosis in the tropical rain forests of Africa”, Am. J Trop Med Hyg. **34** (1985) 781. https://doi.org/10.4269/ajtmh.1985.34.781.

[2] J. G. Breman, “Monkeypox: An emerging infection for humans?”, in *Emerging infections*, W. M. Scheld, W. A. Craig, and J. M. Hughes (eds.),

ASM Press, Washington, D.C., 2000, pp. 45–67. https://doi.org/10.1128/9781555816971.ch5.

[3] A. Nalca, A. W. Rimoin, S. Bavari & C. A. Whitehouse, "Re-emergence of monkey pox: prevalence, diagnostics, and countermeasures", Clin. Infect Dis. **41** (2005) 1765. https://doi.org/10.1086/498155.

[4] A. Mackett & J. Archard, "Conservation and variation in orthopox virus genome structure", J. Gen. Virol. **45** (1979) 683. https://doi.org/10.1099/0022-1317-45-3-683.

[5] J. Esposito & K. Knight, "Orthopox virus DNA: a comparison of restriction profiles and maps", Virology **143** (1985) 230. https://doi.org/10.1016/0042-6822(85)90111-4.

[6] A. M. Likos *et al.*, "A tale of two Clades: Monkey pox viruses", J. Gen. Virol. **86** (2005) 2661. https://doi.org/10.1099/vir.0.81215-0.

[7] K. D. Reed *et al.*, "The detection of monkey pox in humans in the western hemisphere", New England Journal of Medicine **350** (2004) 342. https://doi.org/10.1056/NEJMoa032299.

[8] M. Stanford, G. Mcfadden, G. Karupiah & G. Chaudhri, "Immuno pathogenesis of pox virus infections: Forecasting the impending storm", Immunology and Cell Biology **85** (2007) 93. https://doi.org/10.1038/sj.icb.7100033.

[9] G. D. Huhn, A. M. Bauer, K. Yorita, M. B. Graham, J. Sejvar, A. Likos, I. K. Damon, M. G. Reynolds & M. J. Kuehnert, "Clinical characteristics of human monkey pox and risk factors for severe disease", Clinical Infectious Diseases **41** (2005) 1742. https://doi.org/10.1086/498115.

[10] E. Sbrana, S.-Y. Xiao, P. C New,an & R. B. Tesh, "Comparative pathology of North American and Central African strains of monkey pox virus in a ground squirrel model of the disease", Am. J. Trop. Med. Hyg. **76** (2007) 155. https://doi.org/10.4269/ajtmh.2007.76.155.

[11] M. G. Reynolds, K. L. Yorita, M. J. Keuhnert, W. B. Davidson, G. D. Huhn, R. C. Holman & I. K. Damon, "Clinical manifestations of human monkey pox influenced by route of infection", Journal of Infectious Diseases **194** (2006) 773. https://doi.org/10.1086/505880.

[12] L. Khodakevich, Z. Jezek & K. Kinzanzka, "Isolation of monkey pox virus from wild squirrel infected in nature", Lancet **1** (1986) 98. https://doi.org/10.1016/S0140-6736(86)90748-8.

[13] L. Khodakevich, Z. Jezek & D. Messinger, "Monkey pox virus: ecology and public health significance", Bull World Health Organ **66** (1988) 47. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2491157/.

[14] T. A. Sale, J. W. Melski & E. J. Stratmen, "Monkey pox: an epidemiologic and clinical comparison of African and US Disease", Journal of the American Academy of Dermatology **55** (2006) 478. https://doi.org/10.1016/j.jaad.2006.05.061.

[15] Centers for Disease Control and Prevention (CDC), "About monkey pox", 2022. https://www.cdc.gov/poxvirus/monkeypox/about.html, assessed on April 16, 2022.

[16] P. C. Emeka, M. O. Onuorah, F. Y. Eguda, and B. G. Babangida, "Mathematical model for monkey pox virus transmission dynamics", Epidemiology (Sunnyvale) **8** (2017) 348. https://doi.org/10.4172/2161-1165.1000348.

[17] S. Usman & I. Adamu, "Modeling the transmission dynamics of the monkey pox virus infection with treatment and vaccination interventions", Journal of Applied Mathematics and Physics **5** (2017) 2335. https://doi.org/10.4236/jamp.2017.512191.

[18] O. J. Peter, C. E. Madubueze, M. M. Ojo, F. A. Oguntolu & T. A. Ayoola, "Transmission dynamics of Monkeypox virus: a mathematical modelling approach", Modeling Earth Systems and Environment **8** (2022) 3423. https://doi.org/10.1007/s40808-021-01313-2.

[19] S. V. Bankuru, S. Kossol, W. Hou, P. Mahmoudi, J. Rychtar & D. Taylor, "A game-theoretic model of Monkeypox to assess vaccination strategies", PeerJ **8** (2020) e9272. https://doi.org/10.7717/peerj.9272.

[20] O. J. Peter, A. Abidemi, M. M. Ojo & T. A. Ayoola, "Mathematical model and analysis of Monkeypox with control strategies", Eur. Phys. J. Plus **138** (2023) 242. https://doi.org/10.1140/epjp/s13360-023-03865-x.

[21] O. J. Peter. C. E. Madubueze, M. M. Ojo, F. A. Oguntolu & T. A. Ayoola, "Modelling and optimal control of Monkeypox with cost-effective strategies", Model. Earth Syst. Environ. **9** (2023) 1989. https://doi.org/10.1007/s40808-022-01607-z.

[22] E. Alakunle, U. Moens, G. Nchinda & M. Okeke, "Monkeypox virus in Nigeria: infection biology, epidemiology, and evolution", Viruses **12** (2020) 1257. https://doi.org/10.3390/v12111257.

[23] R. Grant, L. L. Nguyen & R. Breban, "Modelling human-to-human transmission of Monkeypox", Bull World Health Organ **98** (2020) 638. https://doi.org/10.2471/BLT.19.242347.

[24] G. Birkhoff & G. Rota, *Ordinary differential equations*, Ginn and Company, Boston, 1962.

[25] S. O. Sowole, D. Sangare, A. A. Ibrahim & I. A. Paul, "On the existence, uniqueness, stability of solution and numerical simulations of a mathematical model for measles disease", International Journal of Advances in Mathematics **4** (2019) 84. https://adv-math.com/wp-content/uploads/2019/07/adv-manuscript.pdf.pdf.

[26] P. Van den Driessche & J. Watmough, "Reproduction numbers and sub-threshold endemic equilibria for compartmental; models of disease transmission", Math. Biosci. **180** (2002) 29. https://doi.org/10.1016/S0025-5564(02)00108-6.

[27] O. Diekman, J. A. P. Heesterbeek & J. A. J. Metz, "On the definition and computation of the basic reproduction numbers in the models for infectious diseases in heterogeneous populations", J. Math. Biol. **28** (1990) 365. https://doi.org/10.1007/BF00178324.

[28] J. D. Murray, *Mathematical Biology I, An introduction, 3rd Ed.*, SpringerVerlag Berlin Heidelberg, 2001. http://pcleon.if.ufrgs.br/pub/listas-sistdin/MurrayI.pdf

[29] J. La Salle, *The stability of dynamical systems*, SIAM, Philadelphia, 1976. https://doi.org/doi/10.1137/1.9781611970432.fm

[30] C. Castillo-Chavez & B. Song, "Dynamical models of tuberculosis and their applications", Mathematical Biosciences and Engineering **1** (2004) 361. https://www.aimspress.com/article/10.3934/mbe.2004.1.361.

[31] B. Bolaji, U. B. Odionyenma, B. I. Omede, P. B. Ojih & A. A. Ibrahim, "Modelling the transmission dynamics of Omicron variant of COVID-19 in densely populated city of Lagos in Nigeria", J. Nig. Soc. Phys. Sci. **5** (2023) 1055. https://doi.org/10.46481/jnsps.2023.1055.