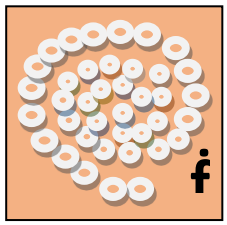

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A theoretical investigation of the impact of a 2-dose vaccination strategy on Monkeypox transmission dynamics at population level

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Abstract

In order to qualitatively evaluate the influence of a two-dose monkeypox (MPV) vaccination strategy when decreased human-to-human infection is accounted for, a new deterministic model for MPV is created and implemented. The backward bifurcation phenomena is demonstrated to occur in the model as a result of the parameter that describes how quickly susceptible individuals receive their first dose of the MPV vaccination. Additionally, a distinct cutoff point for the rate at which vulnerable individuals receive the first dose of the MPV vaccination was discovered An example of the model demonstrated that, in the absence of the rate at which susceptible individuals receive the first dose of the MPV vaccine, the disease-free equilibrium was globally asymptotically stable. The endemic equilibrium point of the distinguished case was also shown to globally asymptotically stable.

Keywords and phrases: Monkeypox virus; Backward bifurcation; Vaccination; Equilibria

1 Introduction

With a clinically-reduced severity than smallpox, monkeypox virus illness (MPV) is zoonotic with accompanying symptoms reminiscent of those seen in previous smallpox cases [1]. MPV tops the chart in terms of the significance of orthopoxviruses on public health since the eradication of smallpox and the resultant annulment of its corresponding vaccine circa 1980 [1]. Central Africa and West Africa regions are the basic habitatas for MPV due to the proximity to the tropical rainforests in the tropics, and has been reported to be more common in cities in this environment [1]. Assorted rodent species cum animal primates are examples of animal hosts [1].

The premier case of human MPV on record was in DR Congo in 1970. Since then, several cases have been reported with the majority of such infections arising from rural communities in the rainforests domiciled in the Congo Basin especially in the DR Congo [1].

Eleven African nations, Nigeria inclusive, have documented human cases of MPV since 1970 [1]. There has been a significant outbreak in Nigeria beginning from 2017, which accounts for ≥ 500 suspected cases, ≥ 200 confirmed cases, and a CFR of approximately 3%. Infections are still being reported as of right now [1].

Due to its widespread distribution throughout the world, MPV is a disease of worldwide public health significance [1]. It is not limited to nations in western and central Africa. According to [1], MPV cases have been reported in nations outside Africa, namely: in the USA since 2003 (and also in 2021),in Israel (in 2018), in the UK (2018, 2019, 2021 and 2022) and in Singapore in 2019 [1]. Numerous cases of monkeypox were reported in May 2022 in a number of non-endemic nations. Research is presently being conducted to gain additional insight into the epidemiology, infection origins, and transmission patterns [1].

Although the most likely natural reservoir for monkeypox is rats, this has not yet been determined [1].

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The consumption of meat and other products not properly prepared from contaminated animals is a major risk factor [1]. Living in or close to forested regions can expose people to sick animals indirectly or at a low level [1].
Close contact with bodily secretions, injuries of an infected case, or just contaminated materials result in human-to-human infection [1]. Health professionals, family members, and other close contacts of active patients are more at risk of contracting the disease since droplet respiratory particles typically require prolonged face-to-face contact for transmission. Nevertheless, from six to nine consecutive person-to-person infections, the longest known chain of transmission in a community has increased recently [1]. This might be a result of smallpox vaccinations ending in all areas, which is causing immunity to decline [1]. In addition, congenital monkeypox can be transmitted through the placenta from mother to fetus or through intimate contact both during and after delivery [1]. Despite the fact that intimate physical contact is a well-established risk factor for transmission, it is currently unknown whether sexual contact is a specific means of transmission for monkeypox. To fully comprehend this risk, more research is required [1].
The typical course of monkeypox is self-limited, with symptoms lasting two to four weeks [1]. Severe cases are more common in youngsters and are associated with factors such as the degree of viral exposure, the patient's health, and the type of sequelae. Poorer results could result from underlying immunological deficits [1]. Although smallpox vaccination was protective in the past, people under the age of 40 to 50 (depending on the country) may now be more vulnerable to monkeypox due to the worldwide discontinuation of smallpox vaccine campaigns following the disease's elimination [1]. Secondary infections, bronchopneumonia, sepsis, encephalitis, and corneal infection leading to visual loss are among the complications associated with monkeypox [1]. It is unknown how much an asymptomatic infection can spread [1]. Monkeypox has historically had a case fatality ratio in the general population ranging from 0 to 11%, with a greater rate among young children. The case fatality ratio has been approximately 3-6% in the recent past [1].
Other rash illnesses like chickenpox, measles, bacterial skin infections, scabies, syphilis, and medication-associated allergies are among the clinical differential diagnosis that need to be taken into account [1]. A clinical characteristic that can be used to differentiate smallpox or chickenpox from monkeypox is lymphadenopathy during the prodromal stage of the illness [1]. Antigen and antibody detection techniques do not yield monkeypox-specific confirmation because orthopoxviruses are serologically cross-reactive [1]. Therefore, in situations when resources are scarce, serology and antigen detection techniques are not advised for diagnosis or case investigation. Furthermore, false positive results may result from recent or distant immunization with a vaccinia-based vaccine (e.g., anyone immunized before to smallpox eradication, or more recently vaccinated due to heightened risk, such as orthopoxvirus laboratory personnel) [1].
The literature has been enhanced by the development of mathematical models by multiple authors addressing various topics related to the dynamics of MPV illness [2, 3, 4, 5, 6]. As the above makes clear, many mathematical models have been created to analyze the monkeypox virus disease. However, to the best of the authors' awareness, none of these models have taken into account the potential influence of a two-dose vaccination strategy on the disease burden in the population in the presence of decreased infection for human to human infection transmission on the population dynamics for monkeypox virus disease burden transmission.
In Section 2 of this paper, a mathematical model is developed to examine how a 2-dose monkeypox vaccination method affects the disease burden in the community in the context of a lower population-level infection rate for human-to-human MPV illness. Important thresholds regulating the dynamics of the disease are found in Section 3, along with the local and global asymptotic stabilities of equilibria and the paper's conclusion is provided in Section 4.

2 Methodology/Model formulation

The model accounts for the total animal population responsible for the disease at time t into mutually exclusive classes of susceptible to infections $(S_a(t))$, infectious $(I_a(t))$ and recovered $(R_a(t))$ given by

$$N_a(t) = S_a(t) + I_a(t) + R_a(t).$$

At any given time t, let $N_h(t)$ denote the the total number of human beings in the environment which is decomposed into classes that are exclusive mutually, represented by: individuals susceptible to infection $(S_h(t))$, vaccinated with the first dose of monkeypox vaccine $(V_{h1}(t))$, vaccinated with the second (and final) dose of monkeypox vaccine $(V_{h2}(t))$, exposed to MPV $(E_h(t))$, infected with MPV $(I_a(t))$, and infectious humans recovered from MPV $(R_h(t))$, individuals so that

$$N_h(t) = S_h(t) + V_{h1}(t) + V_{h2}(t) + E_h(t) + I_h(t) + R_h(t).$$

The susceptible animal class, $S_a(t)$, is created by the influx of animals (assumed wholly susceptible) into the population at a rate Λ_a. The population of susceptible animals is reduced due to MPV infection at the rate λ_a. The class is further reduced by natural mortality at a rate μ_a. Thus

$$S_a' = \Lambda_a - \lambda_a S_a - \mu_a S_a. \tag{1}$$

The infected animal population, $I_a(t)$, is generated by the influx of individuals from the susceptible animals class that are infected with the MPV at the rate λ_a. The class of infectious animals declines in size as they recover from infection arising from MPV at the rate r_a and infection-induced mortality d_a. This particular class is further shrunken in size by natural mortality at a rate μ_a. Thus

$$I_a' = \lambda_a S_a - (\mu_a + r_a + d_a) I_a. \tag{2}$$

The recovered animal population, $R_a(t)$, is generated by the influx of infectious individuals who recover from MPV at the rate r_a. The population of recovered animals is reduced by natural mortality at a rate μ_a. Thus

$$R_a' = r_a I_a - \mu_a R_a. \tag{3}$$

The wholly susceptible human population, $S_h(t)$, is generated by the recruitment of individuals (assumed susceptible) into the population at a rate Λ_h. The population of susceptible individuals is reduced due to MPV infection at the rate λ_h. The population is further reduced by natural mortality at a rate μ_h. Thus

$$S_h' = \Lambda_h - \lambda_h S_h - (\mu_h + \eta_v) S_h. \tag{4}$$

The population of humans vaccinated with the first dose for mpox V_{h1} is generated by at the rate η_v. This class is depleted by vaccinated individuals who relatively at risk of breakthrough infection with MPV at the rate $(1-\epsilon)\lambda_h$. The class is also depleted by humans who receive the second mandatory dose thus entering the class V_{h2} at the rate η_v. Furthermore, this class is reduced by the natural death rate μ_h. Thus

$$V_{h1}' = \eta_v S_h - (1-\epsilon)\lambda_h V_{h1} - (\mu_h + n_0) V_{h1}. \tag{5}$$

The population of humans vaccinated with the second mandatory dose for mpox V_{h2} is generated by at the rate n_0. This class is depleted by vaccinated individuals who are relatively at reduced risk of breakthrough infection with MPV at the rate $\psi(1-\epsilon)\lambda_h$. The class is reduced by the natural death rate μ_h. Thus

$$V'_{h2} = n_0 V_{h1} - \psi(1-\epsilon)\lambda_h V_{h2} - \mu_h V_{h2}. \tag{6}$$

The exposed human population, $E_h(t)$, is populated by the movement of individuals from susceptible individuals infected with MPV infection at the rate λ_h. The population is further populated by vaccinated individuals who are relatively at risk of breakthrough infection with MPV at the rates of $(1-\epsilon)\lambda_h$ and $\psi(1-\epsilon)\lambda_h$, from classes V_{h1} and V_{h2} respectively. The population is further reduced by individuals who become infectious at the rate π. The population is further reduced by the natural death rate μ_h. Thus

$$E'_h = \lambda_h S_h + (1-\epsilon)\lambda_h V_{h1} + \psi(1-\epsilon)\lambda_h V_{h2} - (\mu_h + \pi)E_h. \tag{7}$$

The infected human population, $I_h(t)$, is populated by the movement of individuals from the exposed class at the rate π. The population is depleted by individuals who recover from MPV infection at the rate r_h. The population is further reduced by individuals who suffer disease-induced mortality at the rate d_h and natural death rate μ_h. Thus

$$I'_h = \pi E_h - (\mu_h + r_h + d_h)I_h. \tag{8}$$

The recovered human population, $R_h(t)$, is populated by the movement of the population of infected individuals who recovered from MPV infection at the rate r_h. The recovered human population is reduced by individuals who succumb to natural death at the rate μ_h. Thus

$$R'_h = r_h I_h - \mu_h R_h. \tag{9}$$

On the basis of the foregoing, the MPV model is the following deterministic system of nine non-linear ordinary differential equations (the schematics of the model is shown Figure 1, and the state variables and parameters of the model are tabulated in Table 1 and Table 2 respectively):

$$\begin{aligned}
S'_a &= \Lambda_a - \lambda_a S_a - \mu_a S_a,\\
I'_a &= \lambda_a S_a - (\mu_a + r_a + d_a)I_a,\\
R'_a &= r_a I_a - \mu_a R_a,\\
S'_h &= \Lambda_h - \lambda_h S_h - (\mu_h + \eta_v)S_h,\\
V'_{h1} &= \eta_v S_h - (1-\epsilon)\lambda_h V_{h1} - (\mu_h + n_0)V_{h1},\\
V'_{h2} &= n_0 V_{h1} - \psi(1-\epsilon)\lambda_h V_{h2} - \mu_h V_{h2},\\
E'_h &= \lambda_h S_h + (1-\epsilon)\lambda_h V_{h1} + \psi(1-\epsilon)\lambda_h V_{h2} - (\mu_h + \pi)E_h,\\
I'_h &= \pi E_h - (\mu_h + r_h + d_h)I_h,\\
R'_h &= r_h I_h - \mu_h R_h,
\end{aligned} \tag{10}$$

where the force of infection associated with MPV animal to animal transmission is given below:

$$\lambda_a = \frac{\beta_{a0} I_a}{N_a}. \tag{11}$$

The corresponding infection force associated with MPV animal to human and human to human transmission, respectively, is given below:

$$\lambda_h = \frac{\beta_{a1} I_a}{N_a} + \frac{(1-\alpha)(1-\rho)\theta_0 \beta_h I_h}{N_h} \tag{12}$$

where

$$\frac{\beta_{a1}}{N_a}$$

is the animal to human contribution to the force of infection associated with MPV while

$$\frac{(1-\alpha)(1-\rho)\theta_0 \beta_h}{N_h}$$

is the human to human contribution to the force of infection associated with MPV.
Model flow diagram is seen in Figure 1:

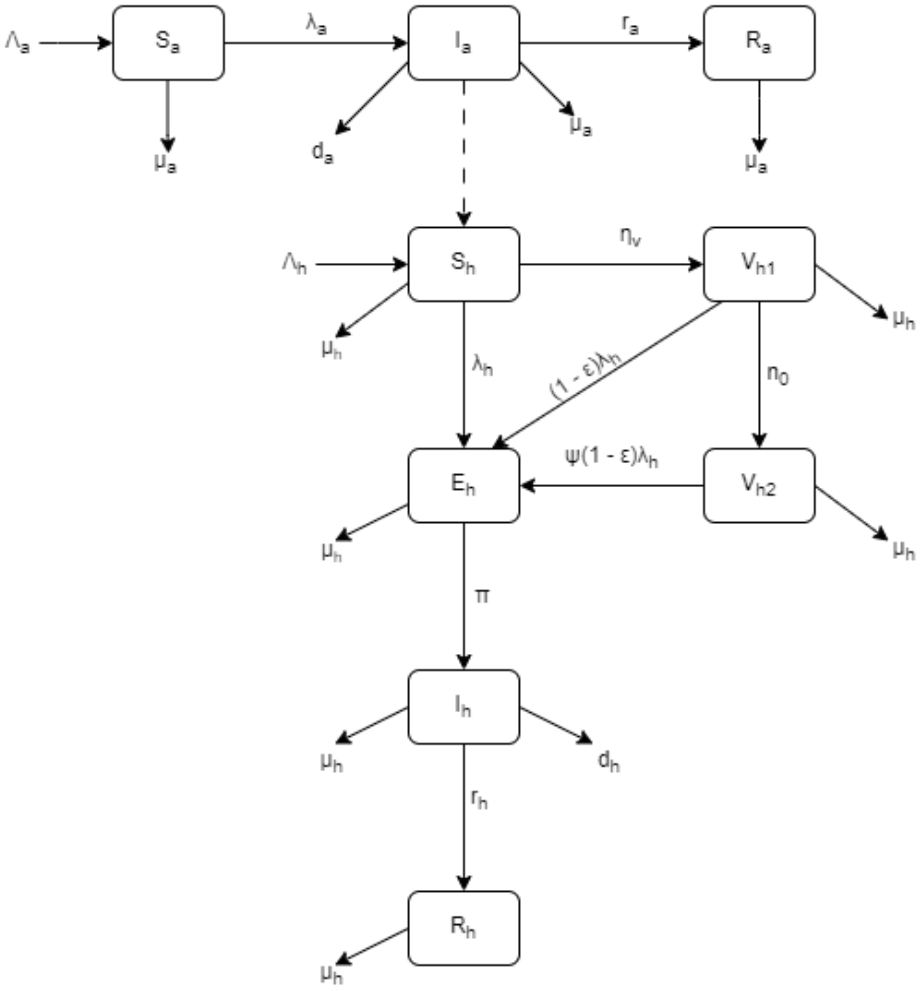

Figure 1: Schematic diagram for the MPV transmission dynamics model (10).

2.1 Description of State variables and Parameters

2.1.1 Description of State variables

The following Table 1 gives a summary of the state variables used herein:

Table 1: Description of state variables of the model (10)

Variables	Description
S_a	Susceptible animal population
I_a	Infected animal population
R_a	Recovered animal population
S_h	Susceptible human population
V_{h1}	Human population vaccinated (with the initial dose) against MPV
V_{h2}	Human population vaccinated (with the second and final dose) against MPV
E_h	Humans beings exposed to MPV
I_h	Humans beings infected with MPV
R_h	Humans beings recovered from MPV

2.1.2 Description of Parameters

The following Table 2 gives a summary of the parameters used herein:

Table 2: Description of parameters of the model (10)

Parameter	Description
Λ_a	Animal recruitment rate
μ_a	Natural death rate of animals
d_a	Infection-induced mortality rate for animals
r_a	Recovery rate for animals
β_{a0}	Animal to animal transmission rate
β_{a1}	Animal to human transmission rate
Λ_h	Human recruitment rate
μ_h	Natural death rate of humans
d_h	Infection-induced mortality rate for humans
r_h	Recovery rate for humans
β_h	Human to human transmission rate
θ_0	Modification parameter which accounts for reduced rate of infection of human to human transmission, with $0 < \theta_0 \leq 1$
η_v	Rate at which susceptible humans are inoculated with the first dose of the MPV vaccine
n_0	Rate at which humans vaccinated with the first dose are inoculated with the second and final dose of the MPV vaccine
π	Rate at which the exposed class progresses to the infectious class
ψ	Modification parameter which accounts for reduced rate of infection for humans vaccinated with the mandatory second dose of the MPV vaccine, with $0 < \psi \leq 1$
α	Proportion of humans maintaining the use of hand sanitizers, with $0 \leq \alpha < 1$
ρ	Proportion of humans using personal protection equipment, with $0 \leq \rho < 1$
ϵ	Relative risk of a human vaccinated with MPV vaccine experiencing a breakthrough infection, with $0 < \epsilon \leq 1$

2.2 Positivity and Boundedness of Solutions

Theorem 2.1. *Permit the initial data for the monkeypox virus transmission model (10) be given as* $S_a(0) > 0, I_a(0) > 0, R_a(0) > 0, S_h(0) > 0, V_{h1}(0) > 0, V_{h2}(0) > 0, E_h(0) > 0, I_h(0) > 0$ *and* $R_h(0) > 0$. *Therefore the solutions* $\big(S_a(t), I_a(t), R_a(t), S_h(t), V_{h1}(t), V_{h2}(t), E_h(t), I_h(t)$ *and* $R_h(t)\big)$ *of the model with initial conditions that are positive, will remain positive for every time* $t > 0$.

Proof: Permit $t_1 = \sup\{t > 0 : S_a(0) > 0, I_a(0) > 0,\ R_a(0) > 0,\ S_h(0) > 0,\ V_{h1}(0) > 0,\ V_{h2}(0) > 0,\ E_h(0) > 0,\ I_h(0) > 0,\ R_h(0) > 0\}$.
Looking at the premier equation of model (10), represented as

$$\frac{dS_a(t)}{dt} = \Lambda_a - (\lambda_a + \mu_a)S_a(t), \tag{13}$$

which can be re-expressed as

$$\frac{d}{dt}\Big[S_a(t)\exp\Big\{\mu_a t + \int_0^t \lambda_a(\tau)d\tau\Big\}\Big] \tag{14}$$

$$= \Lambda_a \exp\Big\{\mu_a t + \int_0^t \lambda_a(\tau)d\tau\Big\}. \tag{15}$$

$$S_a(t_1)\exp\Big\{\mu_a t_1+\int_0^{t_1}\lambda_a(\tau)d\tau\Big\}-S_a(0) \\ =\int_0^{t_1}\Lambda_a\Big[\exp\Big\{\mu_a y+\int_0^{y}\lambda_a(\tau)d\tau\Big\}\Big]dy, \tag{16}$$

So that,

$$S_a(t_1)=S_a(0)\exp\Big[-\mu_a t_1-\int_0^{t_1}\lambda_a(\tau)d\tau\Big] \\ +\Big[\exp\Big\{-\mu_a t_1-\int_0^{t_1}\lambda_a(\tau)d\tau\Big\}\Big] \\ \times\int_0^{t_1}\Lambda_a\Big[\exp\Big\{\mu_a y+\int_0^{y}\lambda_a(\tau)d\tau\Big\}\Big]dy>0. \tag{17}$$

Therefore $S_a(t)>0,\ \forall\, t>0$.
Correspondingly, other state variables in (10) can be shown to be positive, i.e., $I_a(t)>0,\ R_a(t)>0,\ S_h(t)>0,\ V_{h1}(t)>0,\ V_{h2}(t)>0,\ E_h(t)>0,\ I_h(t)>0,\ R_h(t)>0,\ \forall\, t>0$.
Hence, we have shown that all state variables in model (10) are positive always.

We proceed to establish the boundedness of solutions to the model (10).

Theorem 2.2. *Allow* $\big(S_a(t),I_a(t),R_h(t),S_h(t),V_{h1}(t),V_{h2}(t),E_h(t),I_h(t),R_h(t)\big)$ *to be flows of the system* (10) *with initial conditions cum the biological feasible region given by the set* $\mathcal{D}_o=\mathcal{D}_a\times\mathcal{D}_h\times\subset\mathbb{R}^3_+\times\mathbb{R}^6_+$, *where:*

$$\mathcal{D}_a=\{(S_a,I_a,R_a)\in\mathbb{R}^3_+:N_a\le\frac{\Lambda_a}{\mu_a}\}$$
$$\mathcal{D}_h=\{(S_h,V_{h1},V_{h2},E_h,I_h,R_h)\in\mathbb{R}^6_+:N_h\le\frac{\Lambda_h}{\mu_h}\}$$

is invariant positively, attracting every positive solutions of the model (10).

Proof: Summing up the first three equations on the right flank of the vector field representing the animal populace in (10), leads to

$$\frac{dN_h}{dt}=\Lambda_a-\mu_a N_a-d_a I_a. \tag{18}$$

From (18), it ensues that $\frac{dN_a}{dt}\le\Lambda_a-\mu_a N_a$. Hence, $\frac{dN_a}{dt}\le 0$ if $N_a(t)\ge\frac{\Lambda_a}{\mu_a}$. Employing [7] comparison theorem, we reveal that $N_a(t)\le N_a(0)e^{-\mu_a t}+\frac{\Lambda_a}{\mu_a}(1-e^{-\mu_a t})$. Specifically, if $N_a(0)\le\frac{\Lambda_a}{\mu_a}$, thus $N_a(t)\le\frac{\Lambda_a}{\mu_a}$ for every $t>0$. Hence, the set $\mathcal{D}_a$ is invariant positively. Moreover, if $N_a(0)>\frac{\Lambda_a}{\mu_a}$, then either the orbits penetrate the set $\mathcal{D}_a$ in fixed time or $N_a(t)$ asymptotically tends in the direction of $\frac{\Lambda_a}{\mu_a}$ as $t\to\infty$. Thus, the set $\mathcal{D}_a$ attracts every trajectory in $\mathbb{R}^3_+$.

Similarly, adding up the right flank of last five equations of the vector field representing the human population in (10), yields

$$\frac{dN_h}{dt}=\Lambda_h-\mu_h N_h-d_h I_h. \tag{19}$$

From (19), it ensues that $\frac{dN_h}{dt}\le\Lambda_h-\mu_h N_h$. Hence, $\frac{dN_h}{dt}\le 0$ if $N_h(t)\ge\frac{\Lambda_h}{\mu_h}$. Employing [7] comparison theorem, we reveal that $N_h(t)\le N_h(0)e^{-\mu_h t}+\frac{\Lambda_h}{\mu_h}(1-e^{-\mu_h t})$. Specifically, if $N_h(0)\le\frac{\Lambda_h}{\mu_h}$,

thus $N_h(t) \leq \frac{\Lambda_h}{\mu_h}$ for all $t > 0$. Therefore, the set $\mathcal{D}_h$ is invariant positively. Furthermore, suppose that $N_h(0) > \frac{\Lambda_h}{\mu_h}$, then either the solutions penetrate the set $\mathcal{D}_h$ in fixed time or $N_h(t)$ asymptotically tends in the direction of $\frac{\Lambda_h}{\mu_h}$ as $t \to \infty$. Thus, the set $\mathcal{D}_h$ attracts every flow in $\mathbb{R}^6_+$.
Thus, it is enough to investigate the dynamics of the flows generated by the system (10) in $\mathcal{D}_o$. We come to the conclusion, thus, that the model (10) is altogether mathematically and epidemiologically well-posed.

3 Analysis of the model

3.1 Local Asymptotic Stability (LAS) of the Disease-free Equilibrium (DFE)

The disease-free equilibrium (DFE) of the system (10) is represented by

$$\begin{aligned}\mathcal{E}_0 &= (S_a^*, I_a^*, R_a^*, S_h^*, V_{h1}^*, V_{h2}^*, E_h^*, I_h^*, R_h^*) \\ &= (\frac{\Lambda_a}{\mu_a}, 0, 0, \frac{\Lambda_h}{\mu_h + \eta_v}, \frac{\eta_v \Lambda_h}{(\mu_h + \eta_v)(\mu_h + n_0)}, \frac{n_0 \eta_v \Lambda_h}{\mu_h(\mu_h + \eta_v)(\mu_h + n_0)}, 0, 0, 0)\end{aligned} \tag{20}$$

The linear stability of $\mathcal{E}_0$ is shown by employing the next-generation operator method on the system (10) [8]. Using the original notations given in [8], it ensues that matrices F and V, respectively, for the fresh infection terms and the other transition terms, are given by

$$F = \begin{bmatrix} \beta_{a0} & 0 & 0 \\ \frac{\Lambda_h \mu_a \beta_{a1}}{\Lambda_a \mu_h}\Big(\frac{\mu_h(\mu_h+n_0)+(1-\epsilon)\eta_v(\mu_h+\psi n_0)}{(\mu_h+\eta_v)(\mu_h+n_0)}\Big) & 0 & (1-\alpha)(1-\rho)\theta_0\beta_h\Big(\frac{\mu_h(\mu_h+n_0)+(1-\epsilon)\eta_v(\mu_h+\psi n_0)}{(\mu_h+\eta_v)(\mu_h+n_0)}\Big) \\ 0 & 0 & 0 \end{bmatrix}$$

and

$$G = \begin{bmatrix} (\mu_a + r_a + d_a) & 0 & 0 \\ 0 & (\mu_h + \pi) & 0 \\ 0 & -\pi & (\mu_h + r_h + d_h) \end{bmatrix}.$$

It ensues that the *effective reproduction number*, denoted by $\mathcal{R}_m = \rho\left(FG^{-1}\right)$, where $\rho\left(FG^{-1}\right)$ is the spectral radius belonging to the matrix FG^{-1}, is denoted by

$$\mathcal{R}_m = max\{\mathcal{R}_{ma}, \mathcal{R}_{mh}\} \tag{21}$$

where

$$\mathcal{R}_{ma} = \frac{\beta_{a0}}{\mu_a + r_a + d_a} \tag{22}$$

where $\mathcal{R}_{ma}$ represents the MPV transmission dynamics amongst the animal population. While

$$\begin{aligned}\mathcal{R}_{mh} &= \frac{(1-\alpha)(1-\rho)\pi\theta_0\beta_h}{(\mu_h+\pi)(\mu_h+r_h+d_h)}\Big(\frac{\mu_h(\mu_h+n_0)+(1-\epsilon)\eta_v(\mu_h+\psi n_0)}{(\mu_h+\eta_v)(\mu_h+n_0)}\Big) \\ &= \mathcal{R}_{0h}\Big(\frac{\mu_h(\mu_h+n_0)+(1-\epsilon)\eta_v(\mu_h+\psi n_0)}{(\mu_h+\eta_v)(\mu_h+n_0)}\Big) \\ &= \mathcal{R}_{0h}\Big((1-\epsilon).\frac{\eta_v}{\mu_h+\eta_v}.\frac{\mu_h+\psi n_0}{\mu_h+n_0} + \frac{\mu_h}{\mu_h+\eta_v}\Big)\end{aligned} \tag{23}$$

represents the control reproduction number of the MPV transmission dynamics amongst the human population [9]. Now, the quantity $\mathcal{R}_{mh} \geq 0$, since $\frac{\eta_v}{\mu_h+\eta_v} \leq 1$, $\frac{\mu_h+\psi n_0}{\mu_h+n_0} \leq 1$ and $\frac{\mu_h}{\mu_h+\eta_v} \leq 1$ and $0 < \psi, \epsilon \leq 1$ (hence, $1-\epsilon \geq 0$). The quantity $\mathcal{R}_{0h} = \frac{(1-\alpha)(1-\rho)\pi\theta_0\beta_h}{(\mu_h+\pi)(\mu_h+r_h+d_h)}$ represents the basic reproduction

number of the MPV transmission amongst humans in the absence of intervention (which is vaccination, in this case).
Therefore, the consequent result arises from the deductions of Theorem 2 in [8].

Lemma 3.1. *The DFE $\mathcal{E}_0$ of (10) is locally asymptotically stable given that $\mathcal{R}_m < 1$ and unstable given condition that $\mathcal{R}_m > 1$.*

The critical quantity, $\mathcal{R}_m$, is the basic reproduction ratio of the malady [8] . It represents the measure of the average number of secondary monkeypox virus infections generated by a typical infectious human in an entirely susceptible populace or at the DFE [8]. The epidemiological consequence of Lemma 3.1 implies that whenever $\mathcal{R}_{m<1}$, monkeypox virus infections can be extricated from the population if the initial sizes of the components of the system are in the basin of attraction of the DFE, $\mathcal{E}_0$. Hence, a small inflow of monkeypox virus-infected persons into the populace will not provoke large monkeypox virus outbreaks, leading to the gradual decline of the disease time goes on.

3.2 Endemic Equilibrium Point (EEP)

We define the endemic equilibrium point, $\mathcal{E}_M^*$, of the system (10) by

$$\mathcal{E}_m^* = \left(S_a^{**},\ I_a^{**},\ R_a^{**}, S_h^{**},\ V_{h1}^{**},\ V_{h2}^{**},\ E_h^{**},\ I_h^{**},\ R_h^{**}\right) \tag{24}$$

where

$$\begin{aligned}
S_a^{**} &= \frac{\Lambda_a(\beta_{a0} - d_a\mathcal{R}_{ma})}{\mu_a(\beta_{a0} - d_a)\mathcal{R}_{ma}},\\
I_a^{**} &= \frac{\Lambda_a}{\beta_{a0} - d_a}(\mathcal{R}_{ma} - 1),\\
R_a^{**} &= \frac{\Lambda_a r_a}{\mu_a(\beta_{a0} - d_a)}(\mathcal{R}_{ma} - 1),\\
S_h^{**} &= \frac{\Lambda_h}{\lambda_h^{**} + \mu_h + \eta_v},\\
V_{h1}^{**} &= \frac{\Lambda_h\eta_v}{(\lambda_h^{**} + \mu_h + \eta_v)((1-\epsilon)\lambda_h^{**} + \mu_h + n_0)},\\
V_{h2}^{**} &= \frac{\Lambda_h n_0\eta_v}{(\lambda_h^{**} + \mu_h + \eta_v)((1-\epsilon)\lambda_h^{**} + \mu_h + n_0)(\psi(1-\epsilon)\lambda_h^{**} + \mu_h)},\\
E_h^{**} &= \frac{\lambda_h^{**}S_h^{**} + (1-\epsilon)\lambda_h^{**}V_{h1}^{**} + \psi(1-\epsilon)\lambda_h^{**}V_{h2}^{**}}{\mu_h + \pi},\\
I_h^{**} &= \frac{\pi E_h^{**}}{\mu_h + r_h + d_h},\\
R_h^{**} &= \frac{r_h}{\mu_h}I_h^{**}.
\end{aligned} \tag{25}$$

The forces of infection are:

$$\lambda_a^{**} = \frac{\beta_{a0}I_a^{**}}{N_a^{**}}, \tag{26}$$

and

$$\lambda_h^{**} = \frac{\beta_{a1}I_a^{**}}{N_a^{**}} + \frac{\theta_0(1-\alpha)(1-\rho)\beta_h I_h^{**}}{N_h^{**}} \tag{27}$$

Substituting both (26) and (27) in (25), after multiple calculations, we have that the EEP connected with the system (10) satisfies the polynomial (in terms of λ_h^{**})

$$\mathcal{M}_1(\lambda_h^{**})^2 + (\mathcal{M}_2 + \mathcal{M}_3)(\lambda_h^{**}) + \mathcal{M}_4 = 0. \tag{28}$$

where,

$$\begin{aligned}
\mathcal{M}_1 &= \Lambda_h\mu_h(1-\epsilon)^2\psi(\mu_h+\pi)(\mu_h+r_h+d_h)(1-\mathcal{R}_{0h})\\
&\quad+\Lambda_h(1-\epsilon)^2\psi\frac{\beta_{a1}\mu_a}{\mathcal{R}_{ma}}(1-\mathcal{R}_{ma}),\\
\mathcal{M}_2 &= \Lambda_h\Big(\mu_h(\mu_h+r_h+d_h+\pi)+r_h)+\mu_h(\mu_h+\pi)(\mu_h+r_h+d_h)(1-\epsilon)\psi\eta_v\epsilon\Big),\\
\mathcal{M}_3 &= \Lambda_h\mu_h(1-\epsilon)\big(\mu_h+\psi(\mu_h+n_0)+(1-\epsilon)\psi\eta_v\big)(\mu_h+\pi)(\mu_h+r_h+d_h)(1-\mathcal{R}_{0h})\\
&\quad+\Lambda_h\Big(\mu_h(\mu_h+\pi)(\mu_h+r_h+d_h)\psi(1-\epsilon)^2\\
&\qquad+(1-\epsilon)(\mu_h+(\mu_h+n_0)\psi)+\psi(1-\epsilon)^2\eta_v\Big)\frac{\beta_{a1}\mu_a}{\mathcal{R}_{ma}}(1-\mathcal{R}_{ma}),\\
\mathcal{M}_4 &= \Lambda_h\mu_h(\mu_h+\eta_v)(\mu_h+n_0)(\mu_h+\pi)(\mu_h+r_h+d_h)(1-\mathcal{R}_{mh})\\
&\quad+\Lambda_h\big(\mu_h(\mu_h+n_0)+(1-\epsilon)\eta_v(\mu_h+\psi n_0)\big)\frac{\beta_{a1}\mu_a}{\mathcal{R}_{ma}}(1-\mathcal{R}_{ma}).
\end{aligned} \tag{29}$$

We summarize the results obtained above in the following theorem expressed below.

Theorem 3.2. *The model system (10) has:*

1. *two endemic equilibria given that $\mathcal{M}_1<0$, $\mathcal{M}_2>\mathcal{M}_3$, $\mathcal{M}_4<0$ or $\mathcal{M}_1>0$, $\mathcal{M}_2<\mathcal{M}_3$, $\mathcal{M}_4>0$ and $\mathcal{R}_m>1$,*

2. *one unique endemic equilibrium given that $\mathcal{M}_1>0$, $\mathcal{M}_2<\mathcal{M}_3$, $\mathcal{M}_4<0$ or $\mathcal{M}_1<0$, $\mathcal{M}_2>\mathcal{M}_3$, $\mathcal{M}_4>0$,*

3. *nil endemic steady state otherwise, whenever $\mathcal{R}_m<1$.*

It is crucial to state right here, that the first item (1) of Theorem 3.1 is reveals the existence of the backward bifurcation in (10). The backward bifurcation phenomenon, defined as the simultaneous existence of a DFE and EEP that both exhibit stability whenever $\mathcal{R}_m<1$. The implication of this is that the recognized and accepted requirement for the control of MPV disease ($\mathcal{R}_m<1$) is no longer sufficient but necessary criterion. In this situation, successful strategies for MPV control will now depend on the initial conditions of various components of the system under scrutiny [10]. It is seen that the model (10)'s EEP subject to $\mathcal{R}_m>1$ (and does not exist given that $\mathcal{R}_m<1$), and thus, no likelihood of backward bifurcation given that $\mathcal{R}_m<1$).

3.2.1 Backward Bifurcation Analysis

Theorem 3.3. *The model (10) experiences backward bifurcation at $\mathcal{R}_m=1$ whenever $\eta_v>\eta_v^c$, with η_v^c defined as*

$$\eta_v^c=\frac{(\mu_h+n_0)}{\beta_h\theta_h\Lambda_h}\frac{\Omega_2}{\Omega_1}>0, \tag{30}$$

Proof: We employ the ensuing alteration of variables. Let $S_a=x_1$, $I_a=x_2$, $R_a=x_3$, $S_h=x_4$, $V_{h1}=x_5$, $V_{h2}=x_6$, $E_h=x_7$, $I_h=x_8$ and $R_h=x_9$, so that $N_a=x_1+x_2+x_3$ and $N_h=$

$x_4 + x_5 + x_6 + x_7 + x_8 + x_9$ and; therefore, model (10) is re-expressed as

$$\begin{aligned}
\dot{x}_1 &\equiv f_1 = \Lambda_a - \Big(\frac{\beta_{a0} x_1 x_2}{x_1 + x_2 + x_3}\Big) - \mu_a x_1,\\
\dot{x}_2 &\equiv f_2 = \Big(\frac{\beta_{a0} x_1 x_2}{x_1 + x_2 + x_3}\Big) - (\mu_a + r_a + d_a) x_2,\\
\dot{x}_3 &\equiv f_3 = r_a x_2 - \mu_a x_3,\\
\dot{x}_4 &\equiv f_4 = \Lambda_h - \Big(\frac{\beta_{a1} x_2}{x_1 + x_2 + x_3} + \frac{\theta_h \beta_h x_8}{x_4 + x_5 + x_6 + x_7 + x_8 + x_9}\Big) x_4 - (\mu_h + \eta_v) x_4,\\
\dot{x}_5 &\equiv f_5 = \eta_v x_4 - (1-\epsilon)\Big(\frac{\beta_{a1} x_2}{x_1 + x_2 + x_3} + \frac{\theta_h \beta_h x_8}{x_4 + x_5 + x_6 + x_7 + x_8 + x_9}\Big) x_5 - (\mu_h + n_0) x_5,\\
\dot{x}_6 &\equiv f_6 = n_0 x_5 - (1-\epsilon)\psi\Big(\frac{\beta_{a1} x_2}{x_1 + x_2 + x_3} + \frac{\theta_h \beta_h x_8}{x_4 + x_5 + x_6 + x_7 + x_8 + x_9}\Big) x_6 - \mu_h x_6,\\
\dot{x}_7 &\equiv f_7 = (x_4 + (1-\epsilon)(x_5 + \psi x_6))\Big(\frac{\beta_{a1} x_2}{x_1 + x_2 + x_3} + \frac{\theta_h \beta_h x_8}{x_4 + x_5 + x_6 + x_7 + x_8 + x_9}\Big) - (\mu_h + \pi) x_7,\\
\dot{x}_8 &\equiv f_8 = \pi - (\mu_h + r_h + d_h) x_8,\\
\dot{x}_9 &\equiv f_9 = r_h x_8 - \mu_h x_9.
\end{aligned} \tag{31}$$

The Jacobian for the system (31) at the DFE is given by

$$J_{\beta_h^*} = \begin{bmatrix}
-\mu_a & -\beta_{a0} & 0 & 0 & 0 & 0 & 0 & 0 & 0\\
0 & -(\mu_a + r_a + d_a) + \beta_{a0} & 0 & 0 & 0 & 0 & 0 & 0 & 0\\
0 & r_a & -\mu_a & 0 & 0 & 0 & 0 & 0 & 0\\
0 & -\frac{\beta_{a1}\Lambda_h \mu_a}{\Lambda_a(\mu_h+\eta_v)} & 0 & -(\mu_h+\eta_v) & 0 & 0 & 0 & -\frac{\beta_h \theta_h \mu_h}{\mu_h+\eta_v} & 0\\
0 & -\frac{(1-\epsilon)\beta_{a1}\eta_v \Lambda_h \mu_a}{\Lambda_a(\mu_h+\eta_v)(\mu_h+n_0)} & 0 & \eta_v & -(\mu_h+n_0) & 0 & 0 & -\frac{(1-\epsilon)\beta_h \theta_h \eta_v \mu_h}{(\mu+\eta_v)(\mu_h+n_0)} & 0\\
0 & -\frac{(1-\epsilon)\psi\beta_{a1} n_0 \eta_v \mu_a}{\Lambda_a \mu_h(\mu+\eta_v)(\mu_h+n_0)} & 0 & 0 & n_0 & -\mu_h & 0 & -\frac{(1-\epsilon)\psi\beta_{a1} n_0 \eta_v}{(\mu+\eta_v)(\mu_h+n_0)} & 0\\
0 & B_0 & 0 & 0 & 0 & 0 & -(\mu_h+\pi) & B_1 & 0\\
-\mu_a & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0
\end{bmatrix} \tag{32}$$

where $B_0 = \frac{\beta_{a1}\Lambda_h \mu_a \big(\mu_h(\mu_h+n_0)+(1-\epsilon)\eta_v(\mu_h+\psi n_0)\big)}{\Lambda_a \mu_a(\mu+\eta_v)(\mu_h+n_0)}$ and $B_1 = \frac{\beta_h \beta_h \big(\mu_h(\mu_h+n_0)+(1-\epsilon)\eta_v(\mu_h+\psi n_0)\big)}{(\mu+\eta_v)(\mu_h+n_0)}$.

Observing the particular situation when $\mathcal{R}_{mh} = 1$. Calculating the value for $\beta_h = \beta_h^*$ from $\mathcal{R}_{mh} = 1$ gives

$$\beta_h = \beta_h^* = \frac{(\mu_h + \pi)(\mu_h + \eta_v)(\mu_h + n_0)(\mu_h + r_h + d_h)}{\mu_h\big(\beta_h(\mu_h + n_0) + (1-\epsilon)\eta_v(\mu_h + \psi n_0)\big)} \tag{33}$$

Matrix $J_{\beta_h^*}$ possesses a right eigenvector given by $\mathbf{w} = (\omega_1, \omega_2, ..., \omega_9)^T$, such that

$$\begin{aligned}
\omega_1 &= \omega_2 = \omega_3 = 0,\\
\omega_4 &= -\frac{\beta_h \theta_h \mu_h^2}{r_h(\mu_h + \eta_v)^2}\omega_9,\\
\omega_5 &= -\frac{\beta_h \theta_h \eta_v \mu_h^2\big((1-\epsilon)(\mu_h + \eta_v) + (\mu_h + n_0)\big)}{r_h(\mu_h + \eta_v)^2(\mu_h + n_0)^2}\omega_9,\\
\omega_6 &= -\frac{\beta_h \theta_h n_0 \eta_v\big((1-\epsilon)(\mu_h + \eta_v)(\psi\Lambda_h(\mu_h + n_0) + \mu_h) + \mu_h(\mu_h + n_0)\big)}{r_h(\mu_h + \eta_v)^2(\mu_h + n_0)^2}\omega_9,\\
\omega_7 &= \frac{\mu_h(\mu_h + r_h + d_h)}{\pi r_h}\omega_9,\\
\omega_8 &= \frac{\mu_h}{r_h}\omega_9,\\
\omega_9 &= \omega_9 > 0.
\end{aligned} \tag{34}$$

In addition, $J_{\beta_h^*}$ possesses a left eigenvector $\mathbf{v} = (\nu_1, \nu_2, ..., \nu_9)$ satisfying **v.w = 1**, with

$$\begin{aligned}
&\nu_1 = 0,\\
&\nu_2 = -\frac{\beta_{a1}\Lambda_h\mu_a\big(\mu_h(\mu_h+n_0)+(1-\epsilon)\eta_v(\psi n_0+\mu_h)\big)}{\Lambda_a\mu_h(\beta_{a0}-(\mu_a+r_a+d_h))(\mu_h+n_0)(\mu_h+\eta_v)}\nu_7,\\
&\nu_3 = \nu_4 = \nu_5 = \nu_6 = 0,\\
&\nu_7 = \nu_7 > 0, \nu_8 > 0,\\
&\nu_9 = 0
\end{aligned} \tag{35}$$

The application of the *Center Manifold Theory* given by [11], deriving the connected non-zero partial derivatives of the right sides of the changed system (31), *(without infection with $\beta_h = \beta_h^*$)* we have that the associated bifurcation coefficients, a and b, are represented by

$$a = \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0), \qquad \text{and} \qquad b = \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta^*}(0,0), \tag{36}$$

The bifurcation coefficient a for the model system (10) (or (31)) (after several algebraic calculations), is:

$$\begin{aligned}
a &= v_7 \sum_{i,j=1}^{9} w_i w_j \frac{\partial^2 f_7}{\partial x_i \partial x_j}\\
&= \frac{2\beta_h\theta_h\mu_h^2}{r_h^2(\mu_h+\eta_v)(\mu_h+n_0)^2}\Big(-\frac{\Omega_2}{\Lambda_h}+\frac{\theta_h\beta_h\eta_v\Omega_1}{\mu_h+n_0}\Big)\nu_7\omega_9^2.
\end{aligned} \tag{37}$$

where

$$\begin{aligned}
\Omega_1 &= \big((1-\epsilon)(\mu_h+\eta_v)+(\mu_h+n_0)+n_0((1-\epsilon)(\mu_h+\eta_v)(\psi\Lambda_h(\mu_h+n_0)+\mu_h)+\mu_h(\mu_h+n_0))\big),\\
\Omega_2 &= \big((\mu_h(\mu_h+n_0)+(1-\epsilon)\eta_v(\mu_h+\psi n_0))+((\mu_h+r_h)(\mu_h+\eta_v)^2(\mu_h+n_0)\\
&\quad+\beta_h\theta_h\Lambda_h(\mu_h(\mu_h+n_0)+\eta_v(\mu_h+n_0))\\
&\quad+\beta_h\theta_h n_0\eta_v\Lambda_h(1-\epsilon)\psi(\mu_h+\eta_v)((1-\epsilon)(\mu_h+\eta_v)(\psi\Lambda_h(\mu_h+n_0)+\mu_h)+\mu_h(\mu_h+n_0))\big)
\end{aligned} \tag{38}$$

Thus, $a > 0$ if and only if

$$\eta_v^c = \eta_v > \frac{(\mu_h+n_0)}{\beta_h\theta_h\Lambda_h}\frac{\Omega_2}{\Omega_1}. \tag{39}$$

The bifurcation coefficient b for the model system (10) (or (31)) is:

$$\begin{aligned}
b &= v_7 \sum_{i=1}^{9} w_i \frac{\partial^2 f_7}{\partial x_i \partial \beta_h^*},\\
&= \frac{\theta_h\mu_h\big(\mu_h(\mu_h+n_0)+(1-\epsilon)\eta_v(\mu_h+\psi n_0)\big)}{r_h(\mu+\eta_v)(\mu_h+n_0)}\nu_7\omega_9.
\end{aligned} \tag{40}$$

Obviously $b > 0$ for every biologically reasonable parameter values. Therefore, backward bifurcation appears if and only if the rate at which susceptible humans are inoculated with the first dose of the MPV vaccine (η_v), is large enough such that $a > 0$. Therefore, the implication is that if the rate at which susceptible humans are inoculated with the first dose of the MPV vaccine is less than the quantity, (η_v^c),

the effective reproduction ratio then becomes a necessary and sufficient tool for facilitating control measures that will culminate in the eradication of MPV disease. □

As a derivative of the results from Theorem 3.2, we declare this result.

Theorem 3.4. *(Non-existence of backward bifurcation) The model (10) (or (31)) does not manifest backward bifurcation in the direction $\mathcal{R}_m = 1$, on the condition that $\eta_v = 0$.*

Proof: Looking at the special case of (10) where the rate at which susceptible humans are inoculated with the first dose of the MPV vaccine is negligible (i.e., $\eta_v = 0$). Therefore, the backward bifurcation coefficient, a, in (37) shrinks to:

$$a = -\frac{2\beta_h\theta_h\mu_h^2\Omega_2}{\Lambda_h r_h^2(\mu_h+\eta_v)(\mu_h+n_0)^2}\nu_7\omega_9^2 < 0. \tag{41}$$

Consequently, this study proved that the presence of the rate at which susceptible humans are inoculated with the first dose of the MPV vaccine causes backward bifurcation in the epidemic dynamics of MPV disease. □

3.2.2 Global Asymptotic Stability (GAS) of DFE

Looking at the situation of model (10) with the condition that $\eta_v = 0$ (i.e., eliminating the parameter responsible backward bifurcation as stated earlier). Under this condition, the model shrinks to

$$\begin{aligned}
S_a' &= \Lambda_a - \lambda_a S_a - \mu_a S_a,\\
I_a' &= \lambda_a S_a - (\mu_a + r_a + d_a)I_a,\\
R_a' &= r_a I_a - \mu_a R_a,\\
S_h' &= \Lambda_h - \lambda_h S_h - \mu_h S_h,\\
E_h' &= \lambda_h S_h - (\mu_h + \pi)E_h,\\
I_h' &= \pi E_h - (\mu_h + r_h + d_h)I_h,\\
R_h' &= r_h I_h - \mu_h R_h.
\end{aligned} \tag{42}$$

The following result holds.

Theorem 3.5. *The DFE of the model (42), without the rate at which susceptible humans are inoculated with the first dose of the MPV vaccine (i.e., $\eta_v = 0$) is GAS in $\mathcal{D}_o$ if $\mathcal{R}_m \leq 1$ and unstable on the condition that $\mathcal{R}_m > 1$.*

Proof: Looking at the peculiar Lyapunov function below

$$\mathcal{V} = \mathcal{K}_1 I_a + \mathcal{K}_2 E_h + \mathcal{K}_3 I_h, \tag{43}$$

where

$$\mathcal{K}_1 = 1, \quad \mathcal{K}_2 = \pi, \quad \mathcal{K}_3 = \mu_h + \pi, \tag{44}$$

alongside Lyapunov derivatives (in this case, the time derivative is represented by a dot)

$$\dot{\mathcal{V}} = \mathcal{K}_1 \dot{I}_a + K_2 \dot{E}_h + \mathcal{K}_3 \dot{I}_h. \tag{45}$$

Transferring the right hand side of (42) to (45) leads to

$$\begin{aligned}\dot{\mathcal{V}} &= \mathcal{K}_1\lambda_a S_a \\ &\quad - \big[\mathcal{K}_1(\mu_a + r_a + d_a)\big] I_a \\ &\quad + \mathcal{K}_2\lambda_h S_h \\ &\quad - \big[\mathcal{K}_2(\mu_h + \pi) - \mathcal{K}_3\pi\big] E_h \\ &\quad - \big[\mathcal{K}_3(\mu_h + r_h + d_h)\big] I_h, \\ &= (\mu_a + r_a + d_a) I_a \Big[\frac{S_a}{N_a}.\frac{\beta_{a0}}{\mu_a + r_a + d_a} - 1\Big] \\ &\quad + (\mu_h + \pi)(\mu_h + r_h + d_h) I_h \Big[\frac{S_h}{N_h}.\frac{(1-\alpha)(1-\rho)\theta_0\beta\pi}{(\mu_h + \pi)(\mu_h + r_h + d_h)} - 1\Big] \\ &\quad + \beta_{a1}\pi.\frac{S_a}{N_a} I_a.\end{aligned} \tag{46}$$

Substituting $(N_a - S_a)$ for I_a in (46), we obtain

$$\begin{aligned}\dot{\mathcal{V}} &= (\mu_a + r_a + d_a) I_a \Big[\frac{S_a}{N_a}.\frac{\beta_{a0}}{\mu_a + r_a + d_a} - 1\Big] \\ &\quad + (\mu_h + \pi)(\mu_h + r_h + d_h) I_h \Big[\frac{S_h}{N_h}.\frac{(1-\alpha)(1-\rho)\theta_0\beta\pi}{(\mu_h + \pi)(\mu_h + r_h + d_h)} - 1\Big] \\ &\quad + \beta_{a1}\pi.\frac{S_a}{N_a}(N_a - S_a).\end{aligned} \tag{47}$$

At DFE, $S_h \leq \Lambda_h/\mu_h$ and $S_a \leq N_a$. Hence

$$\begin{aligned}\therefore \dot{\mathcal{V}} &\leq (\mu_a + r_a + d_a) I_a \big(\mathcal{R}_{ma} - 1\big) \\ &\quad + (\mu_h + \pi)(\mu_h + r_h + d_h) I_h \big(\mathcal{R}_{mh} - 1\big).\end{aligned} \tag{48}$$

Hence, $\dot{\mathcal{V}} \leq 0$ whenever $\mathcal{R}_{ma} \leq 1$ and $\mathcal{R}_{mh} \leq 1$ with $\dot{\mathcal{V}} = 0$ if and only if $I_a = I_h = 0$. Therefore, $\mathcal{V}$ satisfies the condition for a Lyapunov function in $\mathcal{D}_o$. Hence, it is established from *LaSalle's Invariance Principle* [12] that:

$$(I_a(t), E_h(t), I_h(t)) \to (0, 0, 0) \quad \text{as} \quad t \to \infty.$$

As a consequence, every flow of the equations of the model (42), with the condition that $\eta_v = 0$, advances in the direction of the DFE of the model (42), as $t \to \infty$ for $\mathcal{R}_{ma} \leq 1$ and $\mathcal{R}_{mh} \leq 1$.
This outcome reveals that in an environment that accommodates both *recovery from and medical intervention for MPV cases*, predicated on the premise we have a negligible that there is negligible rate at which susceptible humans are inoculated with the first dose of the MPV vaccine , that is, $\eta_v = 0$, the DFE will be GAS on the condition that $\mathcal{R}_m \leq 1$. Therefore, MPV disease can be eradicated from the populace on the condition that $\mathcal{R}_m \leq 1$, regardless of the initial sizes of the sub-populations.

3.2.3 Global Asymptotic Stability (GAS) of EEP

The stable manifold of the DFE of the system (42) is assumed to be

$$\mathcal{D}_0 = \{(S_a, I_a, R_a, S_h, E_h, I_h, R_h) \in \mathcal{D}_1 : S_a = I_a = R_a = S_h = E_h = I_h = R_h = 0\}. \tag{49}$$

The following result is established.

Theorem 3.6. *The unique EEP, $\mathcal{E}_L^*$, of model (42) with $\eta_v = 0$ is globally asymptotically stable in $\mathcal{D}_1 \backslash \mathcal{D}_0$ at any time $\mathcal{R}_m > 1$.*

Proof: Again, looking at the non-linear Lyapunov function

$$\begin{aligned}\mathcal{L} &= S_a - S_a^{**}\ln\Big(\frac{S_a}{S_a^{**}}\Big) + A_1\Big(I_a - I_a^{**}\ln\frac{I_a}{I_a^{**}}\Big) + A_2\Big(R_a - R_a^{**}\ln\frac{R_a}{R_a^{**}}\Big)\\ &+ S_h - S_h^{**}\ln\Big(\frac{S_h}{S_h^{**}}\Big) + A_3\big(E_h - E_h^{**}\ln\frac{E_h}{E_h^{**}}\big) + A_4\Big(I_h - I_h^{**}\ln\frac{I_h}{I_h^{**}}\Big)\\ &+ A_5\Big(R_h - R_h^{**}\ln\frac{R_h}{R_h{}^{`**}}\Big),\end{aligned} \tag{50}$$

where

$$\begin{aligned}A_1 = 0, \quad A_2 = \frac{\mu_h+\pi}{\pi}, \quad A_3 = 0\\ \mu_a + r_a + d_a = \tilde{\beta}_{a0}S_a^{**} + \tilde{\beta}_{a1}S_h^{**}\\ ,\mu_h + r_h + d_h = \frac{\pi\theta_h\tilde{\beta}_h S_h^{**}}{\mu_h+\pi}\end{aligned} \tag{51}$$

$\mathcal{L}$ has Lyapunov derivatives, given as

$$\begin{aligned}\dot{\mathcal{L}} = \Big(1-\frac{S_a^{**}}{S_a}\Big)\dot{S}_a + \Big(1-\frac{I_a^{**}}{I_a}\Big)\dot{I}_a + A_1\Big(1-\frac{R_a^{**}}{R_a}\Big)\dot{R}_a + \Big(1-\frac{S_h^{**}}{S_h}\Big)\dot{S}_h\\ + \Big(1-\frac{E_h^{**}}{E_h}\Big)\dot{E}_h + A_2\Big(1-\frac{I_h^{**}}{I_h}\Big)\dot{I}_h + A_3\Big(1-\frac{R_h^{**}}{R_h}\Big)\dot{R}_h.\end{aligned} \tag{52}$$

Substituting the right right hand sides of the equations in model (42) corresponding to $\dot{S}_h$, $\dot{E}_h$, $\dot{I}_a$, $\dot{I}_s$, $\dot{R}_h$, $\dot{S}_r$, $\dot{I}_r$, and (51) into (52), and after several algebraic calculations gives:

$$\begin{aligned}\dot{\mathcal{L}} &= \mu_a S_a^{**}\Big(2 - \frac{S_a^{**}}{S_a} - \frac{S_a}{S_a^{**}}\Big)\\ &+ \tilde{\beta}_{a0}S_a^{**}I_a^{**}\Big(2 - \frac{S_a}{S_a^{**}} - \frac{S_a^{**}}{S_a}\Big)\\ &+ \tilde{\beta}_{a1}S_h^{**}I_a^{**}\Big(3 - \frac{S_h^{**}}{S_h} - \frac{E_h}{E_h^{**}}\frac{I_a^{**}}{I_a} - \frac{S_h}{S_h^{**}}\frac{E_h^{**}}{E_h}\frac{I_a}{I_a^{**}}\Big)\\ &+ \mu_h S_h^{**}\Big(2 - \frac{S_h^{**}}{S_h} - \frac{S_h}{S_h^{**}}\Big)\\ &+ \theta_h\tilde{\beta}_h S_h^{**}I_h^{**}\Big(3 - \frac{S_h^{**}}{S_h} - \frac{E_h}{E_h^{**}}\frac{I_h^{**}}{I_h} - \frac{S_h}{S_h^{**}}\frac{E_h^{**}}{E_h}\frac{I_h}{I_h^{**}}\Big).\end{aligned} \tag{53}$$

In the situation that the *arithmetic mean is greater than the geometric mean*, the following inequalities hold

$$\begin{aligned}&2 - \frac{S_a^{**}}{S_a} - \frac{S_a}{S_a^{**}} \le 0, \quad 2 - \frac{S_a}{S_a^{**}} - \frac{S_a^{**}}{S_a} \le 0, \quad 3 - \frac{S_h^{**}}{S_h} - \frac{E_h}{E_h^{**}}\frac{I_a^{**}}{I_a} - \frac{S_h}{S_h^{**}}\frac{E_h^{**}}{E_h}\frac{I_a}{I_a^{**}} \le 0,\\ &2 - \frac{S_h^{**}}{S_h} - \frac{S_h}{S_h^{**}} \le 0, \quad 3 - \frac{S_h^{**}}{S_h} - \frac{E_h}{E_h^{**}}\frac{I_h^{**}}{I_h} - \frac{S_h}{S_h^{**}}\frac{E_h^{**}}{E_h}\frac{I_h}{I_h^{**}}.\end{aligned} \tag{54}$$

Thus, $\dot{\mathcal{L}} \le 0$ whenever $\mathcal{R}_m > 1$.

As far as the important variables in the equations of I_a and I_h respectively are at the endemic equilibrium, substituting them into the equations are represent I_a and I_h, respectively, in (42) such that

$$I_a(t) \to I_a^{**}, \quad I_h(t) \to I_h^{**} \quad \text{as} \quad t \to \infty. \tag{55}$$

Therefore, $\mathcal{L}$ represents a Lyapunov function in $\mathcal{D}_1 \setminus \mathcal{D}_0$. □

This result shows that in MPV population, if $\eta_v = 0$, the EEP will be GAS on the condition that $\mathcal{R}_m > 1$. Hence, MPV will persist in the human community regardless of the initial sizes of the sub-populations on the condition that $\mathcal{R}_m > 1$.

4 Discussions and Conclusion

In order to qualitatively evaluate the impact of a 2-dose monkeypox vaccination strategy on the disease burden in the population in the presence of reduced infection for human to human infection transmission for the disease, a deterministic model for the transmission dynamics of the monkeypox virus (MPV) malady was specifically developed and used theoretically. It was demonstrated that the existence of the parameter that describes the rate at which susceptible persons receive the first dose of the MPV vaccine causes the model to experience the backward bifurcation concept. It was also possible to determine a significant threshold for the rate at which susceptible individuals receive their first dose of the MPV vaccination. When vulnerable humans are vaccinated against MPV at a minimal rate, the DFE is globally asymptotically stable (GAS), according to a particular case study of the model. It was also demonstrated that the endemic equilibrium point of the model system's special case is globally asymptotically stable.

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