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A Co-infection Model for Monkeypox and HIV/AIDS: Sensitivity and Bifurcation Analyses

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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ABSTRACT

Monkeypox can make people very sick. The skin becomes infected with bacteria, thus causing severe skin damage. This can lead to corneal infection with loss of vision, pneumonia, difficulty swallowing, diarrhoea and vomiting leading to harsh malnutrition or dehydration, several organs inflammation or death. HIV-AIDS is a life-threatening and chronic condition. In HIV-infected individuals whose immune systems have been compromised, monkeypox mortality alone may be much higher. The co-infection of monkeypox and HIV/AIDS infections has been studied from a mathematical perspective by constructing a 13-compartment deterministic model. Basic mathematical analyses were performed on the co-infection model and the sub-models. The disease equilibrium points, the non-negativity of solutions, the basic reproduction numbers, the invariant region and the stability patterns. When the basic reproduction number is less than unity, the disease-free equilibrium points of each sub-model are globally asymptomatically stable. Certain calculations were done using the maple 18 programming language. The sensitivity analysis reveals that the parameters of the basic reproduction of the monkeypox sub-model with positive sensitivity

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indices are the probability of catching the monkeypox virus, the rate of effective contact, the compartment I_m's coefficient of infection and the monkeypox vaccine's waning rate, while the parameters of the basic reproduction of the HIV/AIDS sub-model with positive sensitivity indices are the probability of catching HIV virus, the rate effective contact, the compartment I_h's coefficient of infection and the compartment A_h's coefficient of infection. Via the centre manifold theorem, the bifurcation analysis reveals a forward bifurcation pattern for the monkeypox sub-model and the HIV/AIDS sub-model, and under a certain condition, a critical value of the monkeypox basic reproduction exists such that an effective management and possible elimination of the monkeypox infection would require that the monkeypox basic reproduction number should be kept below unity and above the critical value.

Keywords: Bifurcation analyses; HIV/AIDS infections; co-infection model; monkeypox.

1. INTRODUCTION

According to World Health Organisation [1], the monkeypox virus causes monkeypox and it usually lasts between 2 weeks and 4 weeks. They further revealed that monkeypox has 6 to 13 days incubation period but can also range from 5 to 21 days, and the monkeypox infection is severe in persons with weak immune systems, pregnant women and children. Lack of energy, muscle aches, swollen lymph nodes, skin rash, headache and backache are typical symptoms of monkeypox. Although smallpox, measles and chickenpox may initially appear similar, the lymph nodes' swelling is a unique feature of monkeypox. The rash due to monkeypox often starts on the face, then extends to other parts. After the fever onset, the eruption of the skin starts within 1 to 3 days. At close contact with body fluids, lesions and contaminated materials, monkeypox virus is transferred from one individual to another. Normally, between 2 and 4 weeks, anyone with monkeypox is infectious as long as this individual manifests the symptoms. Transmission also occurs from mother to fetus via the placenta. Monkeypox can make people very sick. The skin becomes infected with bacteria, thus causing severe skin damage. This can lead to corneal infection with loss of vision, pneumonia, difficulty swallowing, diarrhoea and vomiting leading to harsh malnutrition or dehydration, several organs inflammation or death.

Kannan, Shaik, Sheeza [2] stated that monkeypox which was dominant in Western and Central African countries is a zoonotic disease, lately, human to human transfer was observed in Australia, developed European countries and North America. They further stated that the strain in Central African is relatively dangerous with high death rate, and advised suitable measures such as wearing of masks, hand hygiene and vaccination. Petersen, Kabamba, McCollum, Lushima, Wemakoy, Muyembe, Nguete, Hughes, Monroe and Reynolds [3] stated that monkeypox virus can infect diverse kinds of domestic animals and wild animals, and that wild squirrels, primates, mongoose, dormice and African pouched rodents are some of the natural hosts.

The immune cells also known as the CD4 cells are attacked by the human immunodeficiency virus (HIV). The immune cells are white blood cells which assist in spotting anomalies and infections in other cells. When there is no treatment, HIV can develop to AIDS (Acquired immunodeficiency syndrome). AIDS is a life-threatening and chronic condition. In HIV-infected individuals whose immune systems have been compromised, monkeypox mortality alone may be much higher. The impact and the risk of other diseases are increased by the presence of HIV. In Nigeria, the number of HIV/AIDS infected individuals is high, thus the human monkeypox outbreak in Nigeria demands a critical study on the co-infection of these two diseases.

According to Getachew [4], models are constructed to study the transmissions dynamics of infectious diseases and to suggest plans on the effective control. Tsetimi, Ossaiugbo and Atonuje [5] and Ossaiugbo and Okposo [6] constructed mathematical models for the study of Pneumonia and COVID-19 infection dynamics respectively. Ayele, Goufo, and Mugisha [7], Somma, Akinwande, and Chado [8], Usman and Adamu [9] and Bhunu, Mushayabasa and Mac Hyman [10] have done some research on HIV/AIDS and monkeypox.

Sensitivity and bifurcation analyses have been performed on several models including the Kumar,Basu,Ghosh,Santra,Mahapatra [11] analysis of COVID-19 epidemic model; Santra, Mahapatra and Phaijo [12] bifurcation analysis and chaos control of discrete pre-predator model;

Kumar, Basu, Santra, Ghosh and Mahapatra [13] optimal control design; Basu, Kumar, Santra, Mahapatra and Elsadany [14] Covid-19 pandemic's second wave's preventive control strategy; Kumar, Mahapatra, Parshad and Santra [15] model for dengue re-infection; Kumar, Santra and Mahapatra [16] stability and sensitivity analyses of the parameters of a SARS-CoV-2 model; Kumar,Basu, Santra, Elsadany, Elsonbaty, Mahapatraand Al-khedhairi [17] stability and sensitivity analyses of an Omicron variant epidemic's model parameters.

The co-infection of HIV/AIDS and Monkeypox is not a desirable condition. This work developed and mathematically analyzed a deterministic model of 13 classes with ordinary differential equations for HIV/AIDS and Monkeypox. The results shall help in the management and possible eradication of HIV/AIDS and Monkeypox co-infection.

2. MODEL DESCRIPTION AND FORMULATION

The animal population is divided into four compartments according to their monkeypox-infection status, namely the susceptible compartment (S_n), the exposed compartment (E_n), the infectious compartment (I_n) and the recovered compartment (R_n). Animals are recruited into the susceptible compartment at rate Λ_n. The animals become exposed at rate λ_n - the force of infection. Animals that are exposed to monkeypox infection become infectious at rate v_n. The exposed animals can recover from the monkeypox infection at rate ϱ_n, andanimals that are already infectious of monkeypox infection can recover at rate ρ_n. We accept that natural death occurs in all the animal compartments and we take the natural rate of death as μ_n. We also assume that death due to the monkeypox infection only occurs in the infectious compartment, and we take the death rate due to the monkeypox infection asd_n. The co-infection model also divides the humans into nine mutually exclusive compartments namely, the susceptible compartment (S), the monkeypox-vaccinated compartment (V_m), the monkeypox-exposed compartment (E_m), the monkeypox-infectious compartment (I_m), the monkeypox-recovered compartment (R_m), the HIV-only compartment (I_h), the HIV/AIDS-infectious compartment (A_h), the HIV-only and Monkeypox co-infectious compartment (I_{hm}), and the HIV/AIDS and Monkeypox co-infectious compartment (A_{hm}).

Relevant information has been provided by the Nigeria Centre for Disease Control and Prevention [18], the World Health Organisation [1] and the World Health Organisation [19] on the transmission dynamics of these diseases. Thus, we make the following assumptions. We assumed that permanent immunity is not conferred on humans upon recovery from monkeypox, and the monkeypox vaccine is not 100% effective, hence there is a waning effect which can cause vaccinated humans to become susceptible again. Furthermore, we assumed that monkeypox-exposed animals/humans and monkeypox-infectious animals/humans do not recover at the same rate. We have also assumed that no sexual activity exists between animals and humans; and monkeypox-recovery rates for the compartments I_{hm} and A_{hm} differ. Additionally, we have assumed the chance of vertical transmission for HIV infection.

Humans are born into the susceptible compartment (S) at rate Λ. The fraction of these humans which acquire the virus via vertical transmission is ε. Thus, this ε -fraction is recruitedinto the compartment I_h, and $(1-\varepsilon)$ -fraction into the compartment S. Humans are given the monkeypox vaccine at the rate α_m, and the vaccine's waning rate is ω_m. The rate in which humans are exposed to the monkeypox infection is λ_m - the force of infection. Monkeypox-exposed humans recover at rate ϱ_m.Humans move from the class E_m to the class I_m at rate v_m. Monkeypox-infectious humans recover at rate ρ_m. Individuals who recovered from the monkeypox infection return to the susceptible compartment at rate ζ. Humans acquire the HIV infection at rateλ_h. People in the compartments E_m and I_m acquire the HIV-infection at rate λ_h ,and move into the compartment I_{hm} .Humans in class R_m also acquire the HIV infection and move to compartment I_h .HIV-infectious humans develop the AIDS syndrome at rate ρ_1. The monkeypox exposure rates for the compartments$I_h$and $A_h$is $\sigma_1\lambda_m$and $\sigma_2\lambda_m$ respectively. σ_1 and $\sigma_2$justifiesthe monkeypox vulnerability increment due to an underlying HIV/AIDS infection. Individuals in the compartmentI_{hm}develop the AIDS syndrome at rate ρ_2 . People in the compartments I_{hm} and A_{hm} recover from the monkeypox infection at the rates τ_1 and τ_2 respectively. Humans die due to the HIV/AIDS infection at the rate d_h , while d_m is the monkeypox-induced death rate. μ - natural mortality rate.

$$
\begin{cases}
\frac{dS_n}{dt} = \Lambda_n - (\mu_n + \lambda_n)S_n, \\
\frac{dE_n}{dt} = \lambda_n S_n - (\mu_n + \varrho_n + v_n)E_n, \\
\frac{dI_n}{dt} = v_n E_n - (\mu_n + d_n + \rho_n)I_n, \\
\frac{dR_n}{dt} = \varrho_n E_n + \rho_n I_n - \mu_n R_n, \\
\frac{dS}{dt} = (1-\varepsilon)\Lambda + \omega_m V_m + \zeta R_m - (\mu + \lambda_m + \lambda_h + \alpha_m)S, \\
\frac{dV_m}{dt} = \alpha_m S - (\mu + \omega_m)V_m, \\
\frac{dE_m}{dt} = \lambda_m S - (\mu + v_m + \varrho_m + \lambda_h)E_m, \\
\frac{dI_m}{dt} = v_m E_m - (\mu + d_m + \rho_m + \lambda_h)I_m, \\
\frac{dR_m}{dt} = \varrho_m E_m + \rho_m I_m - (\mu + \zeta + \lambda_h)R_m, \\
\frac{dI_h}{dt} = \varepsilon\Lambda + \lambda_h(S + R_m) + \tau_1 I_{hm} - (\mu + \rho_1 + \sigma_1\lambda_m)I_h, \\
\frac{dA_h}{dt} = \rho_1 I_h + \tau_2 A_{hm} - (\mu + d_h + \sigma_2\lambda_m)A_h, \\
\frac{dI_{hm}}{dt} = \sigma_1\lambda_m I_h + \lambda_h(E_m + I_m) - (\mu + d_m + \rho_2 + \tau_1)I_{hm}, \\
\frac{dA_{hm}}{dt} = \rho_2 I_{hm} + \sigma_2\lambda_m A_h - (\mu + d_m + d_h + \tau_2)A_{hm}.
\end{cases} \tag{1}
$$

Initial conditions:

$$S_n(0) \geq 0, E_n(0) \geq 0, I_n(0) \geq 0, R_n(0) \geq 0, S(0) \geq 0, V_m(0) \geq 0, E_m(0) \geq 0, I_m(0) \geq 0, R_m(0) \geq 0, I_h(0) \geq 0, A_h(0) \geq 0, I_{hm}(0) \geq 0, A_{hm}(0) \geq 0.$$

where:

$$\lambda_n = \beta_n c_n \frac{I_n(t)}{S_n(t) + E_n(t) + I_n(t) + R_n(t)},$$

$$\lambda_m = (1-\delta_m)\left(\beta_n c_n \frac{I_n}{N_n} + \beta_m c_m \frac{\left(A_{hm}(t) + \theta_1 I_m(t) + \theta_2 I_{hm}(t)\right)}{S(t) + V_m(t) + E_m(t) + I_m(t) + R_m(t)}\right), \theta_1 < \theta_2,$$

$$\lambda_h = (1-\delta_h)\left(\beta_h c_h \frac{\left(A_{hm}(t) + \phi_1 I_h(t) + \phi_2 I_{hm}(t) + \phi_3 A_h(t)\right)}{S(t) + I_h(t) + A_h(t) + I_{hm}(t) + A_{hm}(t)}\right), \phi_1 < \phi_2 < \phi_3,$$

$$N_n(t) = S_n(t) + E_n(t) + I_n(t) + R_n(t).$$

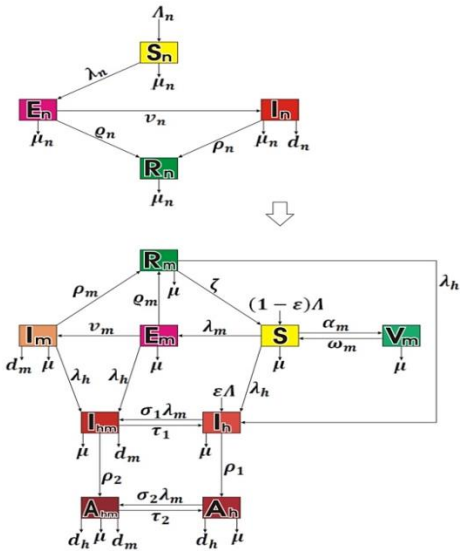

Fig. 1. Schematic diagram

Table 1.Variables and Parameters Descriptions

Parameter	Description
Λ_n	Recruitment rate into the class S_n.
Λ	Recruitment rate into the susceptible class S.
μ_n	Natural death rate among animals.
μ	Natural death rate among humans.
ϱ_n	Recovery rate for the class E_n.
ϱ_m	Recovery rate for the class E_m.
ρ_n	Recovery rate for the class I_n.
ρ_m	Recovery rate for the class I_m.
τ_1	Monkeypox-recovery rate for the class I_{hm}.
τ_2	Monkeypox-recovery rate for the class A_{hm}.
d_n	Monkeypox-induced death rate among animals.
d_m	Monkeypox-induced death rate among humans.
d_h	HIV/AIDS-induced death rate.
ε	The fraction born infected with HIV virus.
ω_m	Monkeypox vaccine's waning rate.
ζ	Rate at which humans who recover from monkeypox infection become susceptible again.
α_m	Monkeypox vaccination rate.
v_n	Progression rate from class E_n to class I_n.
v_m	Progression rate from class E_m to class I_m.
ρ_1	Progression rate from class I_h to class A_h.
ρ_2	Progression rate from class I_{hm} to class A_{hm}.
σ_1	Parameter accounting for increased monkeypox susceptibility because of an underlying HIV infection.
σ_2	Parameter accounting for increased monkeypox susceptibility because of underlying AIDS infection.
c_n	Animals' effective rate of contact for getting monkeypox
c_m	Humans' effective rate of contact for getting monkeypox
c_h	Humans' effective rate of contact for getting HIV infection
δ_m	Monkeypox prevention measure. $0 \leq \delta_m \leq 1$.
δ_h	HIV/AIDS prevention measure. $0 \leq \delta_h \leq 1$.
β_n	Animal's probability of catching monkeypox.
β_m	Human's probability of catching monkeypox.
β_h	Probability of getting infected with HIV per sexual contact with a partner that is infected.
θ_1	The infection coefficient of the class I_m.
θ_2	The infection coefficient of the class I_{hm}.
ϕ_1	The HIV infection coefficient of the class I_h.
ϕ_2	The HIV infection coefficient of the class I_{hm}.
ϕ_3	The HIV infection coefficient of the class A_h.

3. BASIC ANALYSIS OF THE MODEL

In order to ascertain the biological relevance of the model, in this section, we present the non-negativity of solutions and the invariant region.

3.1 Non-negativity of Solutions

Theorem 1:

Suppose

$$\Gamma = \{(S_n, E_n, I_n, R_n, S, V_m, E_m, I_m, R_m, I_h, A_h, I_{hm}, A_{hm}) \in \mathbb{R}_+^{13}: S_n(0) > 0, E_n(0) > 0, I_n(0) > 0, R_n(0) > 0, S(0) > 0, V_m(0) > 0, E_m(0) > 0, I_m(0) > 0, R_m(0) > 0, I_h(0) > 0, A_h(0) > 0, I_{hm}(0) > 0, A_{hm}(0) > 0\},$$ then
$\{S_n, E_n, I_n, R_n, S, V_m, E_m, I_m, R_m, I_h, A_h, I_{hm}, A_{hm}\}$ is non-negative $\forall\ t \geq 0$.

Proof:

$$\frac{dS_n}{dt} = \Lambda_n - (\mu_n + \lambda_n)S_n.$$

$$\frac{dS_n}{dt} \geq -(\mu_n + \lambda_n)S_n,$$

$$\int \frac{1}{S_n} dS_n \geq -\int (\mu_n + \lambda_n)dt,$$

$$S_n \geq e^{-\int(\mu_n+\lambda_n)dt}.$$

$$\therefore \quad S_n > 0 \quad \forall \ t \geq 0.$$

Similarly,

$$E_n(t) > 0, I_n(t) > 0, R_n(t) > 0, S(t) > 0, V_m(t) > 0, E_m(0t) > 0, I_m(t) > 0, R_m(t) > 0, I_h(t) > 0, A_h(t) > 0, I_{hm}(t) > 0, A_{hm}(t) > 0 \ \forall \ t \geq 0 \ \blacksquare$$

3.2Invariant Region and Boundedness

Theorem 2:

The sets

$\Gamma_1 = \left\{(S_n, E_n, I_n, R_n) \in \mathbb{R}_+^4 : 0 \leq S_n + E_n + I_n + R_n = N_n \leq \frac{\Lambda_n}{\mu_n}\right\}$ and
$\Gamma_2 = \left\{(S, V_m, E_m, I_m, R_m, I_h, A_h, I_{hm}, A_{hm}) \in \mathbb{R}_+^9 : 0 \leq S + V_m + E_m + I_m + R_m + I_h + A_h + I_{hm} + A_{hm} = N \leq \frac{\Lambda}{\mu}\right\}$ are positively invariant.

Proof:

$$N_n(t) = S_n(t) + E_n(t) + I_n(t) + R_n(t).$$

$$\frac{dN_n}{dt} = \Lambda_n - \mu_n N_n - d_n I_n \leq \Lambda_n - \mu_n N_n.$$

$$\therefore \quad N_n(t) \leq \frac{\Lambda_n}{\mu_n} + ke^{-\mu_n t}. \tag{2}$$

$$N(t) = S(t) + V_m(t) + E_m(t) + I_m(t) + R_m(t) + I_h(t) + A_h(t) + I_{hm}(t) + A_{hm}(t).$$

$$\frac{dN}{dt} = \Lambda - \mu N - d_m(I_m + I_{hm}) - d_h(A_h + A_{hm}) \leq \Lambda - \mu N.$$

$$\therefore \quad N(t) \leq \frac{\Lambda}{\mu} + ke^{-\mu t}. \tag{3}$$

The inequalities (2) is the threshold population level for the animal population, while the inequality (3) is the threshold population level for the human population respectively. Thus, Γ_1 and Γ_2 are positively invariant ■

3.3 Equilibrium Points

We obtain the disease-free equilibrium (DFE) by setting all the infected compartments and the derivatives in system (1) to zero and solving the resulting system.

I. The DFE$(\mathbb{E}_{0_m})$*of the sub-model for Monkeypox*

$$\left.\begin{array}{l}\Lambda_n-(\mu_n+\lambda_n)S_n=0,\\ \lambda_nS_n-(\mu_n+\varrho_n+v_n)E_n=0,\\ v_nE_n-(\mu_n+d_n+\rho_n)I_n=0,\\ \varrho_nE_n+\rho_nI_n-\mu_nR_n=0,\\ \Lambda+\omega_mV_m+\zeta R_m-(\mu+\lambda_m+\alpha_m)S=0,\\ \alpha_mS-(\mu+\omega_m)V_m=0,\\ \lambda_mS-(\mu+v_m+\varrho_m)E_m=0,\\ v_mE_m-(\mu+d_m+\rho_m)I_m=0,\\ \varrho_mE_m+\rho_mI_m-(\mu+\zeta)R_m=0,\end{array}\right\} \quad (4)$$

$$\lambda_n=\beta_nc_n\frac{I_n}{S_n(t)+E_n(t)+I_n(t)+R_n(t)},$$

$$\lambda_m=(1-\delta_m)\left(\beta_nc_n\frac{I_n}{N_n}+\beta_mc_m\frac{\theta_1I_m}{S(t)+V_m(t)+E_m(t)+I_m(t)+R_m(t)}\right).$$

$$\therefore\quad \mathbb{E}_{0_m}=\left(\frac{\Lambda_n}{\mu_n},0,0,0,\frac{\Lambda(\mu+\omega_m)}{\mu(\mu+\alpha_m+\omega_m)},\frac{\Lambda\alpha_m}{\mu(\mu+\alpha_m+\omega_m)},0,0,0\right). \quad (5)$$

II.HIV/AIDS sub-model's DFE

$$\left.\begin{array}{l}(1-\varepsilon)\Lambda-(\mu+\lambda_h)S=0,\\ \boldsymbol{\varepsilon}\Lambda+\lambda_hS-(\mu+\rho_1)I_h=0,\\ \rho_1I_h-(\mu+d_h)A_h=0.\end{array}\right\} (6)$$

$$\lambda_h=(1-\delta_h)\left(\beta_hc_h\frac{(\phi_1I_h+\phi_3A_h)}{S(t)+I_h(t)+A_h(t)}\right).$$

$$\therefore\quad \mathbb{E}_{0_h}=\left(\frac{(1-\varepsilon)\Lambda}{\mu},0,0\right) \quad (7)$$

3.4. Basic Reproduction Number

The method of the next generation matrix which was used by Ossaiugbo and Okposo [6] shall be employed in calculating the monkeypox-sub-model's basic reproduction R_{0_m}, the HIV/AIDS sub-model's basic reproduction numberR_{0_h}, and the co-infection model's basic reproduction number$R_{0_{hm}}$.

(i) CalculatingR_{0_m}.

The classes that are infected are denoted by$X(t)$and represented as

$$X'=\mathcal{F}(t,X)-\mathcal{V}(t,X)$$

where $\mathcal{F}$are the new infection terms and $\mathcal{V}$are the remaining terms.

$$X=\begin{pmatrix}E_m\\I_m\end{pmatrix},\qquad \mathcal{F}=\begin{pmatrix}\lambda_mS\\0\end{pmatrix},\qquad \mathcal{V}=\begin{pmatrix}(\mu+v_m+\boldsymbol{\varrho_m})E_m\\-v_mE_m+(\mu+d_m+\rho_m)I_m\end{pmatrix}.$$

$$F=\begin{bmatrix}0 & -\dfrac{(-1+\delta_m)\beta_mc_m\theta_1(\mu+\omega_m)}{\mu+\alpha_m+\omega_m}\\ 0 & 0\end{bmatrix},V=\begin{bmatrix}\mu+v_m+\varrho_m & 0\\ -v_m & \mu+d_m+\rho_m\end{bmatrix}.$$

F and V which are the next generation matrices. They are the Jacobian matrices of $\mathcal{F}$ and $\mathcal{V}$evaluated at the DFE.

$$FV^{-1} = \begin{bmatrix} \frac{(1-\delta_m)\beta_m c_m \theta_1 (\mu+\omega_m) v_m}{(\mu+\alpha_m+\omega_m)(\mu+v_m+\varrho_m)(\mu+d_m+\rho_m)} & \frac{(1-\delta_m)\beta_m c_m \theta_1 (\mu+\omega_m)}{(\mu+\alpha_m+\omega_m)(\mu+d_m+\rho_m)} \\ 0 & 0 \end{bmatrix}.$$

$$\therefore \quad R_{0_m} = (\beta_m c_m \theta_1 (1-\delta_m) v_m) \left(\frac{\mu+\omega_m}{\mu+\alpha_m+\omega_m}\right)\left(\frac{1}{(\mu+v_m+\varrho_m)(\mu+d_m+\rho_m)}\right). \tag{8}$$

(ii) Calculating R_{0_h}.

$$X = \begin{pmatrix} I_h \\ A_h \end{pmatrix}, \qquad \mathcal{F} = \begin{pmatrix} \varepsilon\Lambda + \lambda_h S \\ 0 \end{pmatrix}, \mathcal{V} = \begin{pmatrix} (\mu+\rho_1) I_h \\ -\rho_1 I_h + (\mu + d_h) A_h \end{pmatrix}.$$

$$F = \begin{bmatrix} -(-1+\delta_h)\beta_h c_h \phi_1 & -(-1+\delta_h)\beta_h c_h \phi_3 \\ 0 & 0 \end{bmatrix}, V = \begin{bmatrix} \mu+\rho_1 & 0 \\ -\rho_1 & d_h+\mu \end{bmatrix}.$$

$$FV^{-1} = \begin{bmatrix} -\frac{(-1+\delta_h)\beta_h c_h \phi_1}{\mu+\rho_1} - \frac{(-1+\delta_h)\beta_h c_h \phi_3 \rho_1}{(\mu+\rho_1)(d_h+\mu)} & -\frac{(-1+\delta_h)\beta_h c_h \phi_3}{d_h+\mu} \\ 0 & 0 \end{bmatrix}.$$

$$\therefore \qquad R_{0_h} = \beta_h c_h (1-\delta_h)(\phi_1 (d_h+\mu) + \phi_3 \rho_1) \frac{1}{(\mu+\rho_1)(d_h+\mu)}. \tag{9}$$

(iii) Calculating $R_{0_{hm}}$

Similarly, we can show that

$$\therefore \quad R_{0_{hm}} = max\left(\frac{(1-\delta_m)\beta_m c_m \theta_1 (\mu+\omega_m) v_m}{(\mu+\alpha_m+\omega_m)(\mu+v_m+\varrho_m)(\mu+d_m+\rho_m)}, \frac{(1-\delta_h)\beta_h c_h ((d_h+\mu)\phi_1 + \phi_3 \rho_1)}{(\mu+\rho_1)(d_h+\mu)}\right).$$

$$i.e. \quad R_{0_{hm}} = max\left(R_{0_m}, R_{0_h}\right).$$

4. SENSITIVITY ANALYSIS

In this section, we examine the sensitivity of the parameters of R_{0_m} and R_{0_h} given in equations (8) and (9) respectively. We employ the method used by Tsetimi, Ossaiugbo and Atonuje [5]. This method helps us to easily quantify the importance of each parameter of the basic reproduction number. It quickly allows us to calculate the relative change in the basic reproduction number with change in the value of a parameter. The forward sensitivity index of a parameter, say μ, of R_0 is given by:

$$\mathfrak{S}_{\mu}^{R_0} = \frac{\partial R_0}{\partial \mu} \times \frac{\mu}{R_0}.$$

I. Sensitivity indices of parameters of R_{0_m}

$$\mathfrak{S}_{\beta_m}^{R_0} = 1 > 0, \qquad \mathfrak{S}_{c_m}^{R_0} = 1 > 0, \qquad \mathfrak{S}_{\theta_1}^{R_0} = 1 > 0,$$

$$\mathfrak{S}_{v_m}^{R_0} = \frac{\mu+\varrho_m}{\mu+v_m+\varrho_m} > 0, \qquad \mathfrak{S}_{\omega_m}^{R_0} = \frac{\alpha_m \omega_m}{(\mu+\alpha_m+\omega_m)(\mu+\omega_m)} > 0,$$

$$\mathfrak{S}_{\mu}^{R_0} = -2((\mu^3 + (\varrho_m/2 + d_m/2 + v_m/2 + \rho_m/2 + \alpha_m/2 + 2\omega_m)\mu^2 + \omega_m(\varrho_m + d_m + v_m + \rho_m + \alpha_m + \omega_m)\mu + (\varrho_m/2 + d_m/2 + v_m/2 + \rho_m/2){\omega_m}^2 + 1/2\alpha_m(\varrho_m + d_m + v_m + \rho_m)\omega_m - 1/2\alpha_m(d_m + \rho_m)(\varrho_m + v_m))\mu)/((\mu+\alpha_m+\omega_m)(\mu+v_m+\varrho_m)(\mu + d_m + \rho_m)(\mu+\omega_m)) < 0,$$

$$\mathfrak{S}_{\alpha_m}^{R_0} = -\frac{\alpha_m}{\mu+\alpha_m+\omega_m} < 0, \quad \mathfrak{S}_{\varrho_m}^{R_0} = -\frac{\varrho_m}{\mu+v_m+\varrho_m} < 0, \quad \mathfrak{S}_{\delta_m}^{R_0} = -\frac{\delta_m}{1-\delta_m} < 0,$$

$$\mathfrak{S}_{d_m}^{R_0} = -\frac{d_m}{\mu+d_m+\rho_m} < 0, \quad \mathfrak{S}_{\rho_m}^{R_0} = -\frac{\rho_m}{\mu+d_m+\rho_m} < 0.$$

The parameters of R_{0_m} with positive sensitivity indices are the

β_m -probability of humans catching monkeypox through an effective contact,
c_m - effective contact rate for catching monkeypox by humans,
θ_1 - compartment I_m's monkeypox-infection coefficient,
ω_m -monkeypox vaccine's waning rate, and
v_m - rate at which humans move from class E_m to class I_m.

Increasing the values of these parameters with positive sensitivity indices increases the value of R_{0_m} and thereby resulting in a high risk of the outbreak. Now, the parameters of R_{0_m} with negative sensitivity indices are the:

μ - natural mortality rate among humans.
d_m -death rate due to monkeypox humans,
ϱ_m - compartment E_m's recovery rate,
ρ_m -compartment I_m's recovery rate,
α_m - rate of monkeypox vaccination, and
δ_m - monkeypox prevention rate, $0 \le \delta_m \le 1$.

Although, it is not recommended to increase the values of d_m and μ, we note here that increasing the values of the parameters of R_{0_m} with negative sensitivity indices is an approach in the elimination of monkeypox infection.

II. Sensitivity indices of parameters of R_{0_h}

$$\mathfrak{S}_{\beta_h}^{R_0} = 1 > 0, \quad \mathfrak{S}_{c_h}^{R_0} = 1 > 0, \quad \mathfrak{S}_{\phi_1}^{R_0} = \frac{\phi_1(\mu+d_h)}{\phi_1(\mu+d_h)+\phi_3\rho_1} > 0,$$

$$\mathfrak{S}_{\phi_3}^{R_0} = \frac{\phi_3\rho_1}{(\mu+d_h)\phi_1+\phi_3\rho_1} > 0, \quad \mathfrak{S}_{\delta_h}^{R_0} = -\frac{\delta_h}{1-\delta_h} < 0,$$

$$\mathfrak{S}_{d_h}^{R_0} = -\frac{\phi_3\rho_1 d_h}{(\phi_1(\mu+d_h)+\phi_3\rho_1)(\mu+d_h)} < 0,$$

$$\mathfrak{S}_{\mu}^{R_0} = -\frac{\mu\left(\mu^2\phi_1+(2d_h\phi_1+2\phi_3\rho_1)\mu+{d_h}^2\phi_1+\phi_3\rho_1(d_h+\rho_1)\right)}{(\mu+\rho_1)(\mu+d_h)(d_h\phi_1+\mu\phi_1+\phi_3\rho_1)} < 0,$$

$$\mathfrak{S}_{\rho_1}^{R_0} = -\frac{\left((\mu+d_h)\phi_1-\phi_3\mu\right)\rho_1}{\left((\mu+d_h)\phi_1+\phi_3\rho_1\right)(\mu+\rho_1)} < 0.$$

The parameters of R_{0_h} with positive sensitivity indices are the

β_h - probability of humans catching HIV through an effective contact,
c_h - effective contact rate for catching HIV infection by humans,
ϕ_1 - compartment I_h's HIV-infection coefficient, and
ϕ_3 - compartment A_h's HIV-infection coefficient.

Increasing the values of these parameters with positive sensitivity indices increases the value of R_{0_h} and thereby resulting in an increased chance of the disease spread. Now, the parameters of $R_{0_h}$with negative sensitivity indices are the:

d_h -death rate due to HIV/AIDS,
ρ_1 - rate of progression from compartment I_h to compartmentA_h.
δ_h - HIV/AIDS prevention rate,$0 \leq \delta_h \leq 1$,
μ - natural mortality rate among humans,

Again, it is not recommended to increase the values of d_h and μ,we note here that increasing the values of the parameters of R_{0_h} with negative sensitivity indices is an approach in the effective management and possible elimination of HIV-AIDS infection.

5. BIFURCATION ANALYSIS

In analyzing the dynamical system, a powerful and systematic framework is required in studying the bifurcation pattern, and this framework should offer insights into the system's qualitative behavior near bifurcation points, and assisting the analysis of stability. Therefore, we employ the centre manifold the theorem as presented by Castillo-Chavez and Song [20] to determine the bifurcation pattern of the sub-models. The Center Manifold Theorem gives a systematic way to reduce the dimensionality of the problem, thereby concentrating on the most important variables close to a bifurcation point.

Theorem 3 (Castillo-Chavez and Song, 2004)

Consider the following general system of ODEs with a parameter ϕ:

$$\frac{dx}{dt} = f(x,\phi), \quad f: \mathbf{R^n} \times \mathbf{R} \to \mathbf{R^n}, \ f \in \mathbf{C^2}(\mathbf{R^n} \times \mathbf{R}), \tag{10}$$

where 0 is an equilibrium point of the system, that is, $f(0,\phi) \equiv 0$ for all ϕ. Assume the following:

A1. $\mathcal{A} = D_x f(0,0) = \left(\frac{\partial f_i}{\partial x_j}(0,0)\right)$ *is the linearization matrix of system (4.1) around the equilibrium 0 with ϕ evaluated at 0. Zero is a simple eigenvalue of $\mathcal{A}$, and other eigenvalues have negative real parts.*

A2. The matrix $\mathcal{A}$ has a nonnegative right eigenvector w and a left eigenvector v each corresponding to the zero eigenvalue.

Let f_k be the kth component of f and

$$\left.\begin{aligned} a &= \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0) \\ b &= \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \phi}(0,0) \end{aligned}\right\} \tag{11}$$

The local dynamics of the system around 0 are completely determined by the signs of a and b:

i. $a > 0$, $b > 0$. When $\phi < 0$ with $|\phi| \ll 1$, 0 is locally asymptotically stable, and there exists a positive unstable equilibrium; when $0 < \phi \ll 1$, 0 is unstable, and there exists a negative and locally asymptotically stable equilibrium.
ii. $a < 0, b < 0$. When $\phi < 0$ with $|\phi| \ll 1$, 0 is unstable; when $0 < \phi \ll 1$, 0 is locally asymptotically stable, and there exists a positive unstable equilibrium;

iii. $a > 0, b < 0$. When $\phi < 0$ *with* $|\phi| \ll 1$, *0 is unstable, and there exists a locally asymptotically stable negative equilibrium; when* $0 < \phi \ll 1$, *0 is stable, and a positive unstable equilibrium appears;*

iv. $a < 0, b > 0$. *When* ϕ *changes from negative to positive, 0 changes its stability from stable to unstable. Correspondingly, a negative unstable equilibrium becomes positive and locally asymptotically stable.*

Particularly, if $a > 0$ *and* $b > 0$, *then a backward bifurcation occurs at* $\phi = 0$.

Proof:

I. Bifurcation of the monkeypox sub-model:

$$\left.\begin{aligned}
S_n{}' &= \Lambda_n - (\mu_n + \lambda_n)S_n,\\
E_{n'} &= \lambda_n S_n - (\mu_n + \varrho_n + v_n)E_n,\\
I_n{}' &= v_n E_n - (\mu_n + d_n + \rho_n)I_n,\\
R_n{}' &= \varrho_n E_n + \rho_n I_n - \mu_n R_n,\\
S' &= \Lambda + \omega_m V_m + \zeta R_m - (\mu + \lambda_m + \alpha_m)S,\\
V_m{}' &= \alpha_m S - (\mu + \omega_m)V_m,\\
E_m{}' &= \lambda_m S - (\mu + v_m + \varrho_m)E_m,\\
I_m{}' &= v_m E_m - (\mu + d_m + \rho_m)I_m,\\
R'_m &= \varrho_m E_m + \rho_m I_m - (\mu + \zeta)R_m.
\end{aligned}\right\} \tag{12}$$

$$\lambda_n = \beta_n c_n \frac{I_n}{S_n + E_n + I_n + R_n},$$

$$\lambda_m = (1 - \delta_m)\left(\beta_n c_n \frac{I_n}{N_n} + \beta_m c_m \frac{\theta_1 I_m}{S + V_m + E_m + I_m + R_m}\right),$$

we set $x_1 = S_n$, $x_2 = E_n$, $x_3 = I_n$, $x_4 = R_n$, $x_5 = S$, $x_6 = V_m$, $x_7 = E_m$, $x_8 = I_m$, $x_9 = R_m$. Thus, system (12) becomes:

$$\left.\begin{aligned}
x_1{}' &= \Lambda_n - (\mu_n + \lambda_n)x_1,\\
x_2{}' &= \lambda_n x_1 - (\mu_n + \varrho_n + v_n)x_2,\\
x_3{}' &= v_n x_2 - (\mu_n + d_n + \rho_n)x_3,\\
x_4 &= \varrho_n x_2 + \rho_n x_3 - \mu_n x_4,\\
x_5 &= \Lambda + \omega_m x_6 + \zeta x_9 - (\mu + \lambda_m + \alpha_m)x_5,\\
x_6{}' &= \alpha_m x_5 - (\mu + \omega_m)x_6,\\
x_7{}' &= \lambda_m x_5 - (\mu + v_m + \varrho_m)x_7,\\
x_8{}' &= v_m x_7 - (\mu + d_m + \rho_m)x_8,\\
x_9' &= \varrho_m x_7 + \rho_m x_8 - (\mu + \zeta)x_9.
\end{aligned}\right\} \tag{13}$$

From

$$R_{0_m} = (\beta_m c_m \theta_1 (1 - \delta_m) v_m)\left(\frac{\mu + \omega_m}{\mu + \alpha_m + \omega_m}\right)\left(\frac{1}{(\mu + v_m + \varrho_m)(\mu + d_m + \rho_m)}\right) = 1,$$

we obtain

$$\beta_m{}^* = \frac{(\mu + \alpha_m + \omega_m)(\mu + v_m + \varrho_m)(\mu + d_m + \rho_m)}{v_m c_m \theta_1 (1 - \delta_m)(\mu + \omega_m)}.$$

The DFE is

$$\left(x_1^* = \frac{\Lambda_n}{\mu_n}, x_2^* = 0, x_3^* = 0, x_4^* = 0, x_5^* = \frac{\Lambda(\mu+\omega_m)}{\mu(\mu+\alpha_m+\omega_m)}, x_6^* = \frac{\Lambda\alpha_m}{\mu(\mu+\alpha_m+\omega_m)}, x_7^* = 0, x_8^* = 0, x_9^* = 0\right).$$

The matrix of linearization around the DFE evaluated at β_m^* is

$$\mathcal{A} = \begin{pmatrix} -\mu_n & 0 & -\beta_n c_n & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & -\mu_n-\varrho_n-v_n & \beta_n c_n & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & v_n & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \varrho_n & \rho_n & -\mu_n & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & l_1 & 0 & -\mu-\alpha_m & \omega_m & 0 & l_3 & \zeta \\ 0 & 0 & 0 & 0 & \alpha_m & -\mu-\omega_m & 0 & 0 & 0 \\ 0 & 0 & l_2 & 0 & 0 & 0 & -\mu-v_m-\varrho_m & l_4 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & v_m & -\mu-d_m-\rho_m & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \varrho_m & \rho_m & -\mu-\zeta \end{pmatrix}.$$

where

$$l_1 = \frac{(-1+\delta_m)\beta_n c_n \mu_n \Lambda(\mu+\omega_m)}{\Lambda_n \mu(\mu+\alpha_m+\omega_m)}, l_2 = \frac{(1-\delta_m)\beta_n c_n \mu_n \Lambda(\mu+\omega_m)}{\Lambda_n \mu(\mu+\alpha_m+\omega_m)},$$

$$l_3 = \frac{(-1+\delta_m)\beta_m^* c_m \theta_1(\mu+\omega_m)}{\mu+\alpha_m+\omega_m}, l_4 = -\frac{(-1+\delta_m)\beta_m^* c_m \theta_1(\mu+\omega_m)}{\mu+\alpha_m+\omega_m}.$$

Now, $|\mathcal{A}-\lambda I| = 0$expands to

$$(2\mu+\varrho_m+d_m+v_m+\lambda+\rho_m)(\lambda+\mu_n)^2(\mu+\alpha_m+\omega_m+\lambda)(\mu+\zeta+\lambda)(-\beta_n c_n v_n+\lambda\mu_n+\lambda^2+\lambda v_n+\lambda\varrho_n)\lambda(\mu+\lambda)=0$$

The solutions are:

$$\lambda_1 = 0, \lambda_2 = -\mu_n, \lambda_3 = -\mu_n, \lambda_4 = -\mu, \lambda_5 = -\mu-\zeta, \lambda_6 = -\mu-\alpha_m-\omega_m,$$

$$\lambda_7 = -\frac{\mu_n}{2}-\frac{v_n}{2}-\frac{\varrho_n}{2}-\frac{1}{2\sqrt{{v_n}^2+(4\beta_n c_n+2\mu_n+2\varrho_n)v_n+(\mu_n+\varrho_n)^2}},$$

$$\lambda_8 = -\frac{\mu_n}{2}-\frac{v_n}{2}-\frac{\varrho_n}{2}+\frac{1}{2\sqrt{{v_n}^2+(4\beta_n c_n+2\mu_n+2\varrho_n)v_n+(\mu_n+\varrho_n)^2}},$$

$$\lambda_9 = -2\mu-\varrho_m-d_m-v_m-\rho_m.$$

0 is a simple eigenvalue of $\mathcal{A} = D_x f(0,0)$. To get a right eigenvector w, we solve the system $\mathcal{A}w = 0$. Assume $w = (w_1, w_2, w_3, w_4, w_5, w_6, w_7, w_8, w_9)^T$. Then

$$\left.\begin{aligned} -\beta_n c_n w_3 - \mu_n w_1 &= 0 \\ (-\mu_n-\varrho_n-v_n)w_2+\beta_n c_n w_3 &= 0 \\ v_n w_2 &= 0 \\ -\mu_n w_4+\varrho_n w_2+\rho_n w_3 &= 0 \\ \frac{(-1+\delta_m)\beta_n c_n \mu_n \Lambda(\mu+\omega_m)w_3}{\Lambda_n\mu(\mu+\alpha_m+\omega_m)} - (\mu+\alpha_m)w_5+\omega_m w_6+\frac{(-1+\delta_m)\beta_m^* c_m\theta_1(\mu+\omega_m)w_8}{\mu+\alpha_m+\omega_m}+\zeta w_9 &= 0 \\ \alpha_m w_5+(-\mu-\omega_m)w_6 &= 0 \\ -\frac{(-1+\delta_m)\beta_n c_n \mu_n \Lambda(\mu+\omega_m)w_3}{\Lambda_n\mu(\mu+\alpha_m+\omega_m)} - (\mu+v_m+\varrho_m)w_7-\frac{(-1+\delta_m)\beta_m^* c_m\theta_1(\mu+\omega_m)w_8}{\mu+\alpha_m+\omega_m} &= 0 \\ v_m w_7+(-\mu-d_m-\rho_m)w_8 &= 0 \\ \varrho_m w_7+\rho_m w_8+(-\mu-\zeta)w_9 &= 0 \end{aligned}\right\} \quad (14)$$

Solving the system with $w_6 = \alpha_m$ and $w_8 = v_m$ we obtain

$$w = \left(0,0,0,0,\mu+\omega_m,\alpha_m,\mu+d_m+\rho_m,v_m,\frac{2\mu^2+(d_m+v_m+\rho_m+\alpha_m+\omega_m+\varrho_m)\mu+(v_m+\varrho_m)(d_m+\rho_m)}{\zeta}\right)^T.$$

w is nonnegative. To obtain the left eigenvector we solve the system $v\mathcal{A} = 0$. Assume $v = (v_1, v_2, v_3, v_4, v_5, v_6, v_7, v_8, v_9)$. Then

$$\left.\begin{aligned}
-v_1\mu_n &= 0\\
v_2(-\mu_n-\varrho_n-v_n)+v_3v_n+v_4\varrho_n &= 0\\
-v_1\beta_nc_n+v_2\beta_nc_n+v_4\rho_n+\frac{v_5(-1+\delta_m)\beta_nc_n\mu_n\Lambda(\mu+\omega_m)}{\Lambda_n\mu(\mu+\alpha_m+\omega_m)}-\frac{v_7(-1+\delta_m)\beta_nc_n\mu_n\Lambda(\mu+\omega_m)}{\Lambda_n\mu(\mu+\alpha_m+\omega_m)} &= 0\\
-\mu_nv_4 &= 0\\
(-v_5+v_6)\alpha_m-v_5\mu &= 0\\
(v_5-v_6)\omega_m-v_6\mu &= 0\\
v_7(-\mu-v_m-\varrho_m)+v_8v_m+v_9\varrho_m &= 0\\
\frac{v_5(-1+\delta_m)\beta_m{}^*c_m\theta_1(\mu+\omega_m)}{\mu+\alpha_m+\omega_m}+\frac{v_7(1-\delta_m)\beta_m{}^*c_m\theta_1(\mu+\omega_m)}{\mu+\alpha_m+\omega_m}-v_8(\mu+d_m+\rho_m)+v_9\rho_m &= 0\\
(v_5-v_9)(\zeta)-v_9\mu &= 0
\end{aligned}\right\} \quad (15)$$

Solving the system, we obtain the components of v as:

$v_1 = 0,$

$$v_2 = -((\mu+\omega_m)\mu_n\Lambda(\mu^3v_5+((\zeta+d_m+v_m+\rho_m+\varrho_m)v_5+v_8v_m)\mu^2+(((d_m+v_m+\rho_m+\varrho_m)(\zeta)+(v_m+\varrho_m)(d_m+\rho_m))v_5+v_8v_m(\zeta+d_m+\rho_m))\mu+(\zeta)((v_md_m+\varrho_m(d_m+\rho_m))v_5+v_8v_m(d_m+\rho_m))-(v_5(\zeta+\mu)\varrho_m(\mu+d_m+\rho_m)+v_5(\zeta+\mu)v_m(\mu+d_m+\rho_m)+v_5(\zeta+\mu)\mu(\mu+d_m+\rho_m)))(1-\delta_m))/((\zeta+\mu)(\mu(\mu+d_m+\rho_m)+v_m(\mu+d_m+\rho_m)+\varrho_m(\mu+d_m+\rho_m))\Lambda_n\mu(\mu+\alpha_m+\omega_m)),$$

$$v_3 = -(\mu_n(\mu+\omega_m)(\mu^3v_5+((\zeta+d_m+v_m+\rho_m+\varrho_m)v_5+v_8v_m)\mu^2+(((d_m+v_m+\rho_m+\varrho_m)(\zeta)+(v_m+\varrho_m)(d_m+\rho_m))v_5+v_8v_m(\zeta+d_m+\rho_m))\mu+(\zeta)((v_md_m+\varrho_m(d_m+\rho_m))v_5+v_8v_m(d_m+\rho_m))-(v_5(\zeta+\mu)\varrho_m(\mu+d_m+\rho_m)+v_5(\zeta+\mu)v_m(\mu+d_m+\rho_m)+v_5(\zeta+\mu)\mu(\mu+d_m+\rho_m)))\Lambda(\mu_n+\varrho_n+v_n)(1-\delta_m))/((\zeta+\mu)(\mu(\mu+d_m+\rho_m)+v_m(\mu+d_m+\rho_m)+\varrho_m(\mu+d_m+\rho_m))\Lambda_n\mu(\mu+\alpha_m+\omega_m)v_n),$$

$$v_4 = 0, v_5 = v_5 > 0, \qquad v_6 = \frac{v_5(\mu+\alpha_m)}{\alpha_m},$$

$$v_7 = (\mu^3v_5+((\zeta+d_m+v_m+\rho_m+\varrho_m)v_5+v_8v_m)\mu^2+(((d_m+v_m+\rho_m+\varrho_m)(\zeta)+(v_m+\varrho_m)(d_m+\rho_m))v_5+v_8v_m(\zeta+d_m+\rho_m))\mu+(\zeta)((v_md_m+\varrho_m(d_m+\rho_m))v_5+v_8v_m(d_m+\rho_m)))/((\zeta+\mu)(\mu+v_m+\varrho_m)(\mu+d_m+\rho_m)),$$

$$v_8 = v_8 > 0, \qquad v_9 = \frac{\zeta v_5}{\zeta+\mu}.$$

Substituting w and v into equation (11), we obtain

$$a = -4(\mu(\mu^2v_8+v_8(\zeta+d_m+\rho_m)\mu+(\zeta)((-v_5+v_8)\rho_m+d_mv_8))(\mu^2+(\alpha_m/2+\omega_m/2+\varrho_m/2+\zeta/2+d_m/2+v_m/2+\rho_m/2)\mu+1/2(d_m+\rho_m)(\varrho_m+\zeta+v_m))(1-\delta_m)(\mu+\omega_m)\beta_m{v_m}^2c_m\theta_1)/((\mu+\alpha_m+\omega_m)\Lambda(\mu+\zeta)(\mu+v_m+\varrho_m)(\mu+d_m+\rho_m)(\zeta)),$$

$$b = (\theta_1(1-\delta_m){v_m}^2(\mu+\omega_m)(\mu^2v_8+v_8(\zeta+d_m+\rho_m)\mu+(\zeta)((d_m+\rho_m)v_8-v_5\rho_m))c_m)/((\mu+\alpha_m+\omega_m)(\mu+\zeta)(\mu+v_m+\varrho_m)(\mu+d_m+\rho_m)).$$

Observe that $a > 0, \text{and } b < 0$ if we choose $v_8 < v_5$, say

$$v_8 = \frac{1}{\mu^2 + (\zeta + d_m + \rho_m)\mu + (d_m + \rho_m)(\zeta)}, \qquad v_5 = \frac{1}{\rho_m},$$

so that

$\mu^2 v_8 + v_8(\zeta + d_m + \rho_m)\mu + \zeta\big((d_m + \rho_m)v_8 - v_5\rho_m\big)$is negative and we get$a > 0, b < 0$if

$$\zeta > 1. \tag{16}$$

Also, Observe that $a < 0,\ b > 0$ if we put$v_8 \geq v_5$, say $v_8 = 1,\ v_5 = 1$, so that $\mu^2 v_8 + v_8(\zeta + d_m + \rho_m)\mu + \zeta\big((d_m + \rho_m)v_8 - v_5\rho_m\big)$is positive and we obtain $a < 0,\ b > 0$if

$$\zeta < \frac{\mu^2 + (\zeta + d_m + \rho_m)\mu}{d_m}. \tag{17}$$

Thus, case iii and case iv of the theorem 3 captures the dynamics of the monkeypox sub-model around the DFE, according to inequalities (16) and (17) respectively. Considering the inequality (17), a bifurcation plot is presented in Fig.2.It requires that an infective management of the monkeypox infection shouldR_{0_m}in the interval$R_{0_{m*}} < R_{0_m} < 1$.

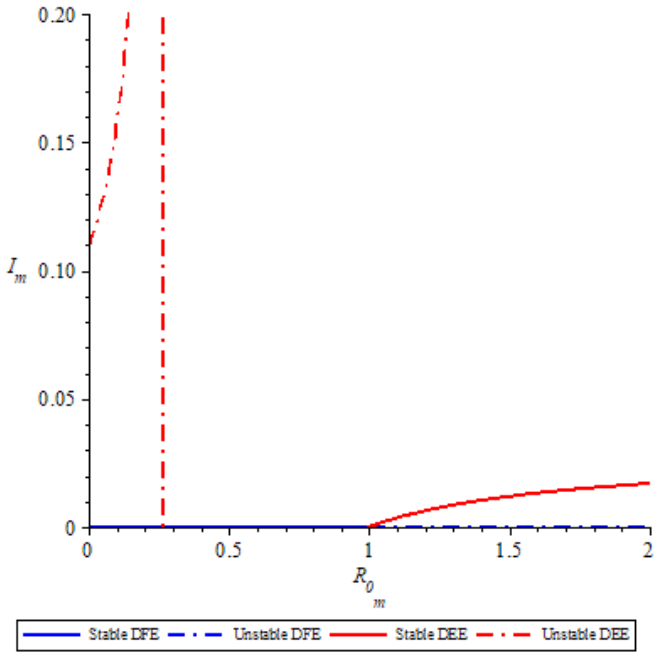

Fig. 2. Bifurcation plot for the monkeypox sub-model

II. Bifurcation of the HIV-AIDS sub-model:

$$\left.\begin{aligned} (1-\varepsilon)\Lambda - (\mu + \lambda_h)S = 0, \\ \boldsymbol{\varepsilon}\Lambda + \lambda_h S - (\mu + \rho_1)I_h = 0, \\ \rho_1 I_h - (\mu + d_h)A_h = 0. \end{aligned}\right\} \tag{18}$$

Where

$$\lambda_h = (1-\boldsymbol{\delta_h})\left(\beta_h c_h \frac{(\phi_1 I_h + \phi_3 A_h)}{S(t)+I_h(t)+A_h(t)}\right).$$

we set $x_1 = S$, $x_2 = I_h$, $x_3 = A_h$. Thus, system (18) becomes:

$$\left.\begin{array}{c} x_1' = (1-\varepsilon)\Lambda - (\mu+\lambda_h)S, \\ x_2' = \boldsymbol{\varepsilon}\Lambda + \lambda_h S - (\mu+\rho_1)I_h, \\ x_3' = \rho_1 I_h - (\mu+d_h)A_h. \end{array}\right\} \quad (19)$$

From

$$R_{0_h} = \beta_h c_h (1-\delta_h)(\phi_1(d_h+\mu)+\phi_3\rho_1)\frac{1}{(\mu+\rho_1)(d_h+\mu)} = 1. \quad (20)$$

we obtain

$$\beta_h{}^* = \frac{(\mu+\rho_1)(\mu+d_h)}{(1-\delta_h)((\mu+d_h)\phi_1+\phi_3\rho_1)c_h}.$$

The DFE is

$$\left(x_1{}^* = \frac{(1-\varepsilon)\Lambda}{\mu}, x_2{}^* = 0, x_3{}^* = 0\right).$$

The matrix of linearization around the DFE evaluated at $\beta_m{}^*$ is

$$\mathcal{A} = \begin{pmatrix} -\mu & -(1-\delta_h)\beta_h{}^* c_h\phi_1 & -(1-\delta_h)\beta_h{}^* c_h\phi_3 \\ 0 & (1-\delta_h)\beta_h{}^* c_h\phi_1 - \mu - \rho_1 & (1-\delta_h)\beta_h{}^* c_h\phi_3 \\ 0 & \rho_1 & -\mu - d_h \end{pmatrix}.$$

Now$|\mathcal{A}-\lambda I| = 0$expands to

$$((\mu+d_h)(\mu+d_h+\lambda)\phi_1 + 2\phi_3\rho_1(\mu+d_h/2+\lambda/2+\rho_1/2))(\mu+\lambda)\lambda = 0$$

The solutions are: $\lambda_1 = 0, \lambda_2 = -\mu,$

$$\lambda_3 = -\frac{d_h{}^2\phi_1 + 2d_h\mu\phi_1 + d_h\phi_3\rho_1 + \mu^2\phi_1 + 2\mu\phi_3\rho_1 + \phi_3\rho_1{}^2}{d_h\phi_1 + \mu\phi_1 + \phi_3\rho_1}.$$

0 is a simple eigenvalue of $\mathcal{A} = D_x f(0,0)$. To get a right eigenvector $w = (w_1, w_2, w_3)^T$, we consider the system

$$\left.\begin{array}{c} -\mu w_1 - \frac{(\mu+\rho_1)(\mu+d_h)\phi_1 w_2}{(\mu+d_h)\phi_1+\phi_3\rho_1} - \frac{(\mu+\rho_1)(\mu+d_h)\phi_3 w_3}{(\mu+d_h)\phi_1+\phi_3\rho_1} = 0 \\ \frac{(\mu+\rho_1)\phi_3(-\rho_1 w_2 + (\mu+d_h)w_3)}{(\mu+d_h)\phi_1+\phi_3\rho_1} = 0 \\ \rho_1 w_2 + (-\mu-d_h)w_3 = 0 \end{array}\right\} \quad (21)$$

Solving the system with$w_3 = \rho_1$, we obtain

$$w = (-(\mu+\rho_1)(\mu+d_h)/\mu, (\mu+d_h), \rho_1)^T.$$

The negative component of w is acceptable because it corresponds to the first entry of the DFE which is strictly positive. Now, to obtain the left eigenvector$v = (v_1, v_2, v_3)$, we solve the system

$$\left.\begin{array}{r} -v_1\mu = 0 \\ \dfrac{-((v_1 - v_3)\rho_1 + v_1\mu)(\mu + d_h)\phi_1 - ((v_2 - v_3)\rho_1 + \mu v_2)\rho_1\phi_3}{(\mu + d_h)\phi_1 + \phi_3\rho_1} = 0 \\ -\dfrac{(\mu + d_h)(((v_1 - v_2)\mu + \rho_1(v_1 - v_2 + v_3))\phi_3 + v_3\phi_1(\mu + d_h))}{(\mu + d_h)\phi_1 + \phi_3\rho_1} = 0 \end{array}\right\} \quad (22)$$

Solving the system with $v_3 = (\mu + \rho_1)\phi_3$, we obtain

$$v = \big(0, \big((\mu + d_h)\phi_1 + \phi_3\rho_1\big), (\mu + \rho_1)\phi_3\big).$$

Substituting w and v into equation (11), we obtain

$$a = -2(((1/2{\rho_1}^2 + (\mu + d_h)\rho_1 + (\mu + d_h)^2)\phi_1 + \phi_3\rho_1(\mu + d_h + \rho_1/2))c_h(1 - \delta_h)\beta_h((\mu + d_h)\phi_1 + \phi_3\rho_1)\mu)/((1 - \epsilon)\Lambda) < 0,$$

$$b = c_h(1 - \delta_h)\big((\mu + d_h)\phi_1 + \phi_3\rho_1\big)^2 > 0.$$

Thuscase iv of the theorem 3 captures the local dynamics of the HIV/AIDS sub-model around the DFE. Hence, the sub-model exhibits a forward bifurcation, which guarantees that the condition $R_{0_h} < 1$ is enough to effectively manage the disease.

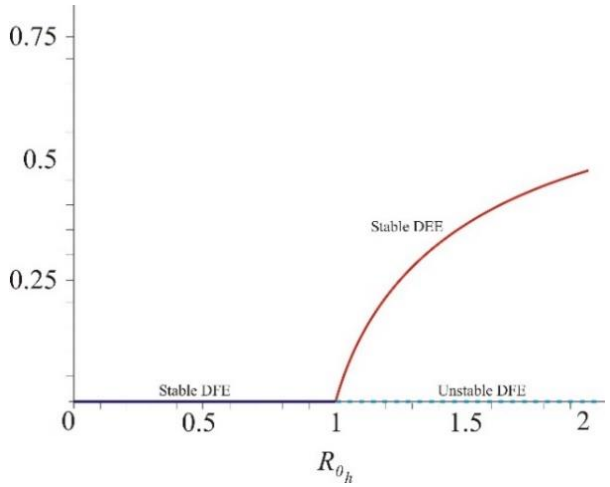

Fig. 3. Bifurcation plot for the HIV/AIDS sub-model

6. DISCUSSION OF RESULTS AND CONCLUSION

A 13-compartment deterministic model has been constructed and used for the analyses of the co-infection of monkeypox and HIV/AIDS infections. Epidemiological analyses were performed on model, and results obtained. In this study, calculations and numerical simulations were done with the Maple 18 programming language. It was shown that under some critical conditions, monkeypox and HIV/AIDS diseases can be properly managed and possibly eliminated. Lyapunov functions were employed in the stability analysis and the disease-free equilibrium (DFE) and disease-endemic equilibrium (DEE) of the sub-models are globally asymptomatically stable when the basic reproduction number (R_0) satisfies the condition $R_0 < 1$ .The centre manifold theorem as given by Castillo-Chavez and Song (2004) was employed in the bifurcation analysis.

The sensitivity analysis approach used by Tsetimi, Ossaiugbo and Atonuje [5] was employed in obtaining the sensitivity indices of the parameters of the basic reproduction number. The probability (β_m) of catching the monkeypox infection, the rate of effective contact (c_m) , the compartment I_m 's coefficient of infection (θ_1) and the monkeypox vaccine's waning rate (ω_m) are the parameters of monkeypox R_0 that havepositive sensitivity indices, while the probability $(\beta_h)$of catching HIV

virus, the rate effective contact (c_h) , the compartment I_h's coefficient of infection and the compartment A_h's coefficient of infection are the parameters of HIV/AIDS R_0 with positive sensitivity indices.

The bifurcation analysis revealed a forward bifurcation for the sub-model for monkeypox and the HIV/AIDS sub-model. It was further revealed that when $\zeta < \frac{\mu^2+(\zeta+d_m+\rho_m)\mu}{d_m}$, then a critical value $R_{0_{m*}}$ exists such that an effective management and possible elimination of the monkeypox disease would require that the basic reproduction number (R_{0_m}) satisfies$R_{0_m} \in (R_{0_{m*}}, 1)$.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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