Transactions of NAMP 23, (2025) 85-98

Transactions of
The Nigerian Association of
Mathematical Physics
Journal homepage: https://nampjournals.org.ng

MATHEMATICAL ANALYSIS OF A MONKEY-POX MODEL

BATUBO T.B.* AND YUSUF T.T

**Department of Mathematics, University of Benin, Benin City, Nigeria.*
Department of Mathemtical sciences, Federal University of Technology, Akure, Nigeria..

ARTICLE INFO

Article history:
Received xxxxx
Revised xxxxx
Accepted xxxxx
Available online xxxxx

Keywords:
Transmission dynamics,
Zoonotic disease,
Local
Global asymptotic stability.

ABSTRACT

Monkey-pox is a relatively rare disease but with a case fatality rate of 1-10%. In this work, we formulated and analyze a mathematical model for the transmission dynamics of monkey-pox. We showed the existence of two equilibria namely, disease free and endemic equilibria. Both equilibria were shown to be globally stable. Sensitivity analysis showed that control strategies should be geared towards reducing contacts between susceptible humans and infected humans and infected non-humans. Also, numerical simulations showed that though human to human transmission may contribute to the incidence of monkey-pox in a population, the non-human to human transmission is seen to play more significant role in sustaining the transmission of monkey pox in a human population.

1. INTRODUCTION

Monkey-pox (MPX) is a rare viral zoonotic disease with its symptoms similar to small pox but less severe [1]. It is a member of the orthopoxvirus genus in the family poxviridae and it is similar to smallpox in terms of the skin lesions (pox) seen in humans [1]. It belongs to the same family as small pox and cowpox [2], [3]. It is different from smallpox because unlike smallpox that is from (human to human) only, monkey- pox can be from human to human or animal to human [4]. Monkey pox is endemic in rodent populations in African [5]. It was first discovered in monkeys in 1958 [4]. The disease and eventually the causative virus was named monkey pox because the lesion (pox) seen in monkeys developed like other pox forming diseases [6]. The incubation period of MPX is usually from 6 to 16 days but it can range from 5 to 21 days which can be divided into two periods [5]. First, the invasion period (0 - 5days) and this is characterized by fever, malaise, intense headache, cough, nausea, back and muscle ache and shortness of breath. Secondly, we have the skin eruption period (within 1-3 days) after appearance of fever [7].

*Corresponding author: BATUBO T.B
E-mail address: toritsesan.batubo@uniben.edu
https://doi.org/10.60787/tnamp.v23.624

The rash appears often on the face (95% of cases) and then spreads to other parts of the body like the palms, feet and entire body [5]. During this period the lymph nodes swell. Some patients develop severe lymphenopathy before the appearance of the rash, which is a distinctive feature of MPV compared to other similar diseases [5]. Currently, there are no specific treatments or vaccines available for monkey pox infections, but outbreaks can be controlled (WHO). The vaccines used to eradicate small pox have been proven tobe 85% effective in preventing monkeypox [6].

The first mathematical model for MPV is credited to [7]. They developed a mathematical model for the dynamics of the transmission of MPV infection with control strategies of combined vaccine and treatment interventions. Using standard approaches, they determined two equilibria for the model namely: disease free and endemic. The disease- free equilibrium was proved to be both locally and globally asymptotically stable if $R_O < 1$ using the next generation matrix approach and comparison theorem. They computed the basic reproduction number for the humans and non-humans primates using some parameter values. Numerical simulations carried out revealed that the infectious individuals in the human and non-human primates' populations will die out in the course of the proposed interventions. Sensitivity analysis carried out on the model parameters showed that the basic reproduction numbers of the model which served as a threshold for measuring new infections in the host populations decrease with increase in the control parameters of vaccination and treatment. [8] formulated a mathematical model of monkey pox virus transmission dynamics with two interacting host populations; humans and rodents. They incorporated into the human population the quarantine class and public enlightenment campaign parameter as means of controlling the spread of the disease. They obtained the Disease Free Equilibrium (DFE) and Endemic Equilibrium (EE). The basic reproduction number $R_{oh}$and R_{on} were computed and used for the analysis after which they analyzed the Disease Free Equilibrium (DFE) for stability using Jacobian matrix techniques and Lyapunov function. In 2020, [9] analysed a compartmental model of MPX dynamics. Their goal was to see whether MPX can be controlled and eradicated by voluntary vaccinations. They showed that there are three equilibria: disease free, fully endemic and previously neglected semi-endemic (with disease existing only among humans). The existence of semi-endemic equilibrium has severe implications should the MPX virus mutate to increased viral fitness in humans. They discovered that MPX is controllable and can be eradicated in a semi-endemic equilibrium by vaccination. However, in a fully endemic equilibrium, MPX cannot be eradicated by vaccination alone.

Therefore, in this study, we seek to investigate how the rodent population affects the dynamics of monkey-pox in the human population. This paper is organized as follows. The model is formulation is given in Section 2 and the model is analyzed in Section 3. Numerical studies are considered in Section 4. The findings from this study are summarized in Section 5.

2. MODEL FORMULATION

A basic Monkey-pox virus model with human population at time t is formulated. The total human population size denoted by (N_h), is divided into four subclasses namely; $S_h(t)$which denotes the number of persons not yet infected with the Monkey-pox virus but are susceptible to the disease, $I_h(t)$ which denotes the number of infected individuals and are capable of spreading the disease to those to those in the susceptible class, $E_h(t)$ denoting the individuals that are exposed and $R_h(t)$ denoting the number of individuals who initially were infected but have now recovered. The total non-human population N_{NH} is divided into four subclasses which are the $S_{nh}(t)$denoting the number of rodents (Non-humans) not yet infected with the virus but are susceptible to the virus, $E_{nh}(t)$ denoting the number of rodents (Non-humans) that are exposed and $I_{nh}(t)$ which denotes the infected rodents and $R_{nh}(t)$the population of the recovered rodents. It is assumed that, the

susceptible human individuals increased by recruitment of individuals at a constant rate Λ_h. The susceptible individuals acquire the virus when they come into contact with infected individual and non-human individuals at the rate λ_h.

where $\lambda_h = \frac{\beta_{n1} I_{nh}}{N_{nh}} + \frac{\beta_h I_h}{N_h}$.

In the same vein, the susceptible non-humans are infected at the rate λ_{nh} by other infected non-humans, where $\lambda_{nh} = \frac{\beta_{n2} I_{nh}}{N_{nh}}$. Where β_h is defined as the probability of a susceptible person getting infected from another human, β_{n1} is the probability of a susceptible human getting infected from an infected non-human and β_{n2} is the probability of a non-human getting infected by another non-human [10].The model for monkey –pox transmission dynamics in a population is given by the following system of deterministic non-linear differential equations (Table 1 and 2 describe the associated state variables and parameters in the model (1) while Figure 1 gives the flow diagram of model (1).)

$$\frac{dS_h}{dt} = \Lambda_h - \lambda_h S_h - \mu_h S_h$$

$$\frac{dE_h}{dt} = \lambda_h S_h - (\gamma + \mu_h) E_h$$

$$\frac{dI_h}{dt} = \gamma E_h - (\tau_h + \mu_h + \delta_1) I_h$$

$$\frac{dR_h}{dt} = \tau_h I_h - \mu_h R_h \qquad (1)$$

$$\frac{dS_{nh}}{dt} = \Lambda_{nh} - \lambda_{nh} S_{nh} - \mu_{nh} S_{nh}$$

$$\frac{dE_{nh}}{dt} = \lambda_{nh} S_{nh} - (\alpha + \mu_{nh}) E_{nh}$$

$$\frac{dI_{nh}}{dt} = \alpha E_{nh} - (\mu_{nh} + \tau_{nh} + \delta_2) I_{nh}$$

$$\frac{dR_{nh}}{dt} = \tau_{nh} I_{nh} - \mu_{nh} R_{nh}$$

$N_h = \mathrm{S}_h + \mathrm{E}_h + I_h + R_h, \quad N_{nh} = \mathrm{S}_{nh} + E_{nh} + I_{nh} + R_{nh}$

Subject to the following non-negative initial conditions $R_{nh}(0) \geq 0$

$S_h\,(0) \geq 0$, $E_h\,(0) \geq 0$, $I_h\,(0) \geq 0$, $R_h\,(0) \geq 0$, $S_{nh}\,(0) \geq 0$, $E_{nh}(0) \geq 0$, $I_{nh}(0) \geq 0$, $R_{nh}(0) \geq 0$

Table 1 : Description of variables and parameters in the model (1)

Variable	**Interpretation**
S_h	Population of Susceptible humans
E_h	Population of Exposed humans
I_h	Population of Infectious humans

R_h	Population of Recovered humans
S_{nh}	Population of Susceptible non-humans
E_{nh}	Population of Exposed non-humans
I_{nh}	Population of Infectious non-humans
R_{nh}	Population of Infected non-humans
Parameter	**Interpretation**
Λ_h	Recruitment rate for humans
Λ_{nh}	Recruitment rate for Non-humans
β_h	Probability of monkey pox transmission from infectious humans to susceptible humans
β_{n1}	Probability of monkey pox transmission from infectious non-humans to susceptible humans
β_{n2}	Probability of monkey pox transmission from infectious non-humans to susceptible non- humans
μ_h	Natural mortality rate for humans
μ_{nh}	Natural mortality rate for non-humans
δ_1	Disease induced death rates for humans
δ_2	Disease induced death rates for non-humans
τ_1	Recovery rate for humans
τ_2	Recovery rate for non-humans
Γ	Progression rate of exposed humans
A	Progression rate of exposed non-humans

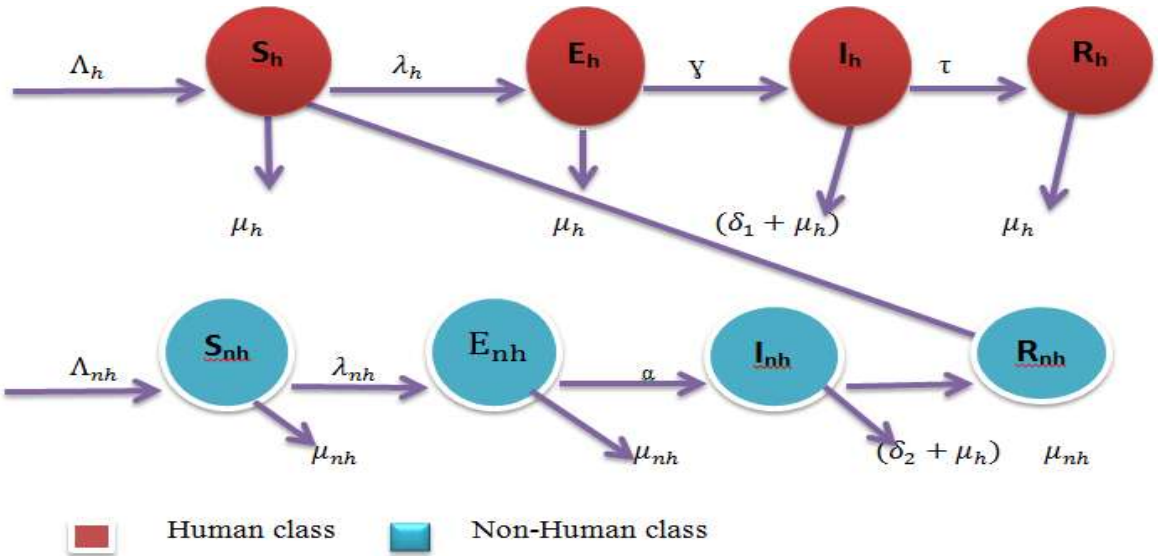

Figure 1: Schematic diagram of the model (1)

3. ANALYSIS OF THE MODEL

We will explore the qualitative properties of the in-host model (1) in this section.

3.1 Basic Properties of the model

In this section the basic qualitative properties such as positivity and boundedness of the solutions of the basic Monkey pox virus model (1) is explored. We claim the following result.

Theorem 0.1 *The model (1) preserves positivity of solutions. That is, solutions with positive initial conditions remain positive for all time t.*

Proof. Let

t_1 *sup{t > 0:* S_h *(t) > 0,* E_h *(t) > 0,* I_h *(t) > 0,* R_h *(t) > 0,* S_{nh} *(t) > 0,* E_{nh} *(t) > 0,* I_{nh} *(t) > 0,* R_{nh} *(t) > 0.*

From the first equation of the model (1), it follows that
$\frac{dS_h}{dt} = \Lambda_h - (\lambda_h + \mu_h)S_h$

when solved leads to $S_h(t_1) = S_h(0) \exp\left[-\mu_h t_1 - \int_0^{t_1} \lambda_h(\tau)d(\tau)\right] + \left\{\exp\left[-\mu_h t_1 - \int_0^{t_1} \lambda_h(\tau)d(\tau)\right]\right\} - \int_0^{t_1} \Lambda_h \left[\exp\left(\mu_h y + \int_0^{y} \lambda_h(\tau)\right)\right] dy > 0$.

Using the same approach, we can equally show that all other state variable of the model (1) will remain positive for all time t > 0

Lemma 3.1 *Consider the biologically-feasible region*

$D = \{(S_h, E_h, I_h, R_h, S_{nh}, E_{nh}, I_{nh}, R_{nh}) \in \mathbb{R}_+^8 ; \ N_h \leq \frac{\Lambda_h}{\mu_h}, N_{nh} \leq \frac{\Lambda_{nh}}{\mu_{nh}}\}$

The closed set D is positively invariant and a global attractor of all positive solution of the model (1).

Proof. Adding the first four equations of the model (1) gives
$N_h = \Lambda_h - \mu_h N_h - d_1 I_h$
Since the right hand side of the above equality is bounded by $\Lambda_h - \mu_h N_h$, a standard comparison theorem can be used to show that

$$N_h(t) \leq N_h(0)e^{-\mu_h t} + \frac{\Lambda_h}{\mu_h}(1 - e^{-\mu_h t})$$

Also, adding the last three equations of model (1), we have

$$N_{nh} = \Lambda_{nh} - \mu_{nh} N_{nh} - d_2 I_{nh}$$

And by a standard comparison theorem, it can be used to show that
$N_{nh}(t) \leq N_{nh}(0)e^{-\mu_{nh} t} + \frac{\Lambda_{nh}}{\mu_{nh}}(1 - e^{-\mu_{nh} t})$

In particular, $N_h(t) \leq \frac{\Lambda_h}{\mu_h}$ if $N_h(0) \leq \frac{\Lambda_h}{\mu_h}$ for all t > 0 and $N_{nh}(t) \leq \frac{\Lambda_{nh}}{\mu_{nh}}$ if $N_{nh}(0) \leq \frac{\Lambda_{nh}}{\mu_{nh}}$ for all t > 0. Thus, D is a positively invariant set under the flow described by the model. The solutions with initial condition in D remain in D with respect to the model. Hence, the system is mathematically and epidemiologically well posed in D.

3.2 Model Analysis

In this session, we analyse the MPV model to investigate the existence and stability of the equilibrium points.

3.2.1 Local Stability of the disease free equilibrium

We obtain the disease free equilibrium (DFE) of the model (1) by setting the right hand side of the equations in the model (1) to zero as well as the infected classes. This equilibrium is given by,

$\emptyset_0 = (S_h^*, E_h^*, I_h^*, R_h^*, S_{nh}^*, E_{nh}^*, I_{nh}^*, R_{nh}^*) = (\frac{\Lambda_H}{\mu_H}, 0, 0, 0, \frac{\Lambda_{NH}}{\mu_{NH}}, 0, 0, 0)$

The Jacobian matrix of the model (1) at the disease free equilibrium is given as

$$J(\phi_o) = \begin{pmatrix} -\mu_h & 0 & -\beta_h & 0 & 0 & 0 & -\beta_{n1}\frac{\Lambda_h\mu_{nh}}{\Lambda_{nh}\mu_h} & 0 \\ 0 & -g_1 & \beta_h & 0 & 0 & 0 & \beta_{n1}\frac{\Lambda_h\mu_{nh}}{\Lambda_{nh}\mu_h} & 0 \\ 0 & \gamma & -g_2 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \tau_h & -\mu_h & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\mu_{nh} & 0 & -\beta_{n2} & 0 \\ 0 & 0 & 0 & 0 & 0 & -g_3 & \beta_{n2} & 0 \\ 0 & 0 & 0 & 0 & 0 & \alpha & -g_4 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \tau_{nh} & -\mu_{nh} \end{pmatrix}$$

Where $g_1 = \gamma + \mu_h, g_2 = \tau_h + \delta_1 + \mu_h, g_3 = \alpha + \mu_{nh}, \ g_4 = \tau_{nh} + \delta_2 + \mu_{nh}$

The eigenvalues of the matrix are given below

$$\begin{aligned} \lambda_1 &= -\mu_h, \ \lambda_2 = -\mu_h, \lambda_3 = -\mu_{nh}, \lambda_4 = -\mu_{nh}, \lambda_5 = -((\gamma+\mu_h)^2 + (\delta_1+\tau_h+\mu_h)^2 + 2\gamma\beta_h), \\ \lambda_6 &= -((\alpha+\mu_{nh})^2 + (\delta_2+\tau_{nh}+\mu_{nh})^2 + 2\alpha\beta_{n2}), \quad \lambda_7 = \gamma\beta_h - (\gamma+\mu_h)(\delta_1+\tau_h+\mu_h), \\ &\text{and} \quad \lambda_8 = \alpha\beta_{n2} - (\alpha+\mu_{nh})(\delta_2+\tau_{nh}+\mu_{nh}). \end{aligned} \tag{2}$$

It is easy to see that the first six eigenvalues are less than zero. Therefore, for the local stability of ϕ_0 to be established, $\lambda_7 < 0$ and $\lambda_8 < 0$ which implies that $\gamma\beta_h - (\gamma+\mu_h)(\delta_1+\tau_h+\mu_h) < 0$ and $\alpha\beta_{n2} - (\alpha+\mu_{nh})(\delta_2+\tau_{nh}+\mu_{nh}) < 0$.

Thus we have that

$$\begin{aligned} \mathcal{R}_{oh} &= \frac{\gamma\beta_h}{(\gamma+\mu_h)(\delta_1+\tau_h+\mu_h)} < 1 \quad \text{and} \\ \mathcal{R}_{on} &= \frac{\alpha\beta_{n2}}{(\alpha+\mu_{nh})(\delta_2+\tau_{nh}+\mu_{nh})} < 1 \end{aligned} \tag{3}$$

We can define the reproduction number $\mathcal{R}_o$ associated with the model (1) as $\mathcal{R}_o = max\{\mathcal{R}_{oh}, \mathcal{R}_{on}\}$ hence we claim the following result

Theorem 3.2. The DFE, ϕ_o, of the model(1) is locally-asymptotically stable (LAS) if $\mathcal{R}_o < 1$, and unstable if $\mathcal{R}_o > 1$.

The threshold quantity $\mathcal{R}_o$ is the basic reproduction number for the model (1) . Biologically speaking, Theorem 0.2 implies that monkey-pox can be eliminated from the population when $\mathcal{R}_o < 1$ if the initial sizes of the population of the model (1) are in the region of attraction of $\mathcal{D}$.

3.3 Global Stability Analysis (GAS) for the Disease Free Equilibrium

We now prove the global asymptotic stability of the DFE of the model (1) We claim the following result.

Theorem 3.3 *The DFE of the model (1) is globally asymptotically stable in $\mathcal{D}$ if $\mathcal{R}_o \leq 1$.*

Proof. By the comparison theorem, the rate of change of the variables representing the infectious classes in the model can be compared in the following inequality:

$$\begin{pmatrix} \dot{E}_h \\ \dot{I}_h \\ \dot{E}_{nh} \\ \dot{I}_{nh} \end{pmatrix} \leq (F - V) \begin{pmatrix} E_h \\ I_h \\ E_{nh} \\ I_{nh} \end{pmatrix} - M_1\theta_1 \begin{pmatrix} E_h \\ I_h \\ E_{nh} \\ I_{nh} \end{pmatrix} - M_2\theta_2 \begin{pmatrix} E_h \\ I_h \\ E_{nh} \\ I_{nh} \end{pmatrix}$$

Where F and V are given below

$$F = \begin{pmatrix} 0 & \beta_h & 0 & \dfrac{\beta_{n1}\Lambda_h\mu nh}{\Lambda_{nh}\mu_h} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \beta_{n2} \\ 0 & 0 & 0 & 0 \end{pmatrix} \quad \text{and} \quad V = \begin{pmatrix} g_1 & 0 & 0 & 0 \\ -\gamma & g_2 & 0 & 0 \\ 0 & 0 & g_3 & 0 \\ 0 & 0 & -\alpha & g_4 \end{pmatrix}$$

where $g_1 = \gamma + \mu_h$, $g_2 = \tau_h + \delta_1 + \mu_h$, $g_3 = \alpha + \mu_{nh}$ and $g_4 = \tau_{nh} + \delta_2 + \mu_{nh}$. Also, θ_1 and θ_2 are given below

$$\theta_1 = \begin{pmatrix} 0 & \beta_h & 0 & \dfrac{\beta_{n1}\Lambda_h\mu nh}{\Lambda_{nh}\mu_h} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \quad \text{and} \quad \theta_2 = \begin{pmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \beta_{n2} \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

Given that $M_1 = 1 - \frac{S_h^*}{N_h^*}$, $M_1 = 1 - \frac{S_{nh}^*}{N_{nh}^*}$ and θ_1 and θ_2 are non-negative matrices. Since $S_h^* \leq N_h^*$, and $S_{nh}^* \leq N_{nh}^*$ we can say that

$$\begin{pmatrix} \dot{E}_h \\ \dot{I}_h \\ \dot{E}_{nh} \\ \dot{I}_{nh} \end{pmatrix} \leq (F - V) \begin{pmatrix} E_h \\ I_h \\ E_{nh} \\ I_{nh} \end{pmatrix}$$

Therefore, the matrix $F - V$ is given as

$$F - V = \begin{pmatrix} -g_1 & \beta_h & 0 & \dfrac{\beta_{n1}\Lambda_h\mu nh}{\Lambda_{nh}\mu_h} \\ \gamma & -g_2 & 0 & 0 \\ 0 & 0 & -g_3 & \beta_{n2} \\ 0 & 0 & -\alpha & -g_4 \end{pmatrix}$$

Let ψ be the eigenvalues of the matrix above. Thus, the characteristic equation gives the following eigenvalues

$$\psi_1 = -(g_1g_2 - \beta_h\gamma) \quad \psi_2 = -g_2 \quad \psi_3 = -(g_3g_4 - \beta_{n2}\alpha) \quad \psi_4 = -g_4$$

All the eigenvalues have negative real part if $\mathcal{R}_o < 1$. Hence, $(E_h, I_h, E_{nh}, I_{nh}) \to (0,0,0,0)$ as $t \to \infty$. By the comparison theorem used in $(E_h, I_h, E_{nh}, I_{nh}) \to (0,0,0,0)$ as $t \to \infty$. Now, when $E_h =$

$I_h = E_{nh} = I_{nh} = 0$, we have that $S_h^* = \frac{\Lambda_h}{\mu_h}$ and $S_{nh}^* = \frac{\Lambda_{nh}}{\mu_n h}$ and $(R_h, R_{nh}) \to (0,0)$ as $t \to \infty$ for $\mathcal{R}_o < 1$. Hence, the disease free equilibrium is globally asymptotically stable for $\mathcal{R}_o < 1$.

3.3.1 Existence of Endemic equilibrium point (EEP)

In this section, the existence of endemic equilibria (that is, equilibria where the infected compartments of the model are non-zero) of the model (1) is established. Let,

$$\phi_1 = (S_h^{**}, E_h^{**}, I_h^{**}, R_h^{**}, S_{nh}^{**}, E_{nh}^{**}, I_{nh}^{**}, R_{nh}^{**})$$

represents any arbitrary endemic equilibrium point (EEP) of the model (1) . It follows, by solving the equations of the model at steady-state, that

$$\begin{aligned}
S_h^{**} &= \frac{\Lambda_h}{\lambda_h^{**} + \mu_h}, \quad E_h^{**} = \frac{\Lambda_h \lambda_h^{**}}{(\lambda_h^{**} + \mu_h)(\gamma + \mu_h)}, \quad I_h^{**} = \frac{\Lambda_h \lambda_h^{**} \gamma}{(\lambda_h^{**} + \mu_h)(\gamma + \mu_h)(\tau_h + \delta_1 + \mu_h)}, \\
R_h^{**} &= \frac{\Lambda_h \lambda_h^{**} \gamma \tau_h}{(\lambda_h^{**} + \mu_h)(\gamma + \mu_h)(\tau_h + \delta_1 + \mu_h)\mu_h}, \quad S_{nh}^{**} = \frac{\Lambda_{nh}}{\lambda_{nh}^{**} + \mu_{nh}}, \quad E_{nh}^{**} = \frac{\Lambda_{nh} \lambda_{nh}^{**}}{(\lambda_{nh}^{**} + \mu_{nh})(\alpha + \mu_{nh})}, \\
I_{nh}^{**} &= \frac{\Lambda_{nh} \lambda_{nh}^{**} \alpha}{(\lambda_{nh}^{**} + \mu_{nh})(\alpha + \mu_{nh})(\delta_2 + \tau_{nh} + \mu_{nh})}, \quad R_{nh}^{**} = \frac{\Lambda_{nh} \lambda_{nh}^{**} \alpha \tau_{nh}}{(\lambda_{nh}^{**} + \mu_{nh})(\alpha + \mu_{nh})(\tau_{nh} + \delta_2 + \mu_{nh})\mu_{nh}}.
\end{aligned} \tag{4}$$

Substituting the expressions in (4) into $\lambda_{nh}^{**} = \frac{\beta_{n2} I_{nh}^{**}}{N_{nh}^{**}}$ and $\lambda_h^{**} = \frac{\beta_{n1} I_{nh}^{**} + \beta_h I_h^{**}}{N_h^{**}}$ and solving gives $\lambda_{nh}^{**} = \frac{g_3 g_4 \mu_{nh} (\mathcal{R}_{on} - 1)}{\mu_{nh} g_3 + \alpha g_5}$ and

$$\begin{aligned}
g_6 \quad & (\alpha g_5 + \mu_{nh} g_4 + g_7(\mathcal{R}_{on} - 1))\lambda_h^{**2} + (g_1 g_2 \mu_h - \beta_h \Lambda_h \gamma \mu_h (\alpha g_5 + \mu_{nh} g_4 + g_7(\mathcal{R}_{on} - 1)) - \\
g_6 \quad & \beta_{n1} \Lambda_{nh} \alpha \mu_{nh} (\mathcal{R}_{on} - 1))\lambda_h^{**} - \beta_{n1} \alpha \Lambda_{n1} g_1 g_2 \mu_h \mu_{nh} (\mathcal{R}_{on} - 1).
\end{aligned} \tag{5}$$

where $g_1 = \gamma + \mu_h$, $g_2 = \tau_h + \delta_1 + \mu_h$, $g_3 = \alpha + \mu_{nh}$, $g_4 = \tau_{nh} + \delta_2 + \mu_{nh}$,

$g_5 = \mu_{nh} + \tau_{nh}$, $g_6 = \mu_h g_2 + \mu_h \gamma + \gamma \tau_h$, $g_7 = \mu_{nh} g_4 + \mu_{nh} \alpha + \alpha \tau_{nh}$.

It is clear to see that λ_{nh}^{**} exist if $\mathcal{R}_{on} > 1$. Also, the quadratic equation in terms of λ_h^{**} has a single positive root if $\mathcal{R}_{on} > 1$. Thus, the endemic equilibria of the of the model (1) can then be obtained by solving for λ_h^{**} from the above polynomial and substituting the positive values of λ_h^{**} into the expressions for the state variables.

3.4 Global stability of endemic equilibrium point (EEP)

The global asymptotic stability of the endemic equilibrium (ϕ_1) of the model (1) will now be explored. We claim the following result.

Theorem 3.4 The endemic equilibrium (ϕ_1) of the model (1) , with $\delta_1 = \delta_2 = 0$, is GAS in whenever $\mathcal{R}_o^m > 1$.

The proof of Theorem 3.4 based on using a non-linear Lyapunov function is given below

Proof. Consider the model with $\delta_1 = \delta_2 = 0$. We then have a mass action model where $\lambda_h^{**} = (\beta_{n1} I_n h^{**} + \beta_h I_h^{**}) S_h^{**}$ and $\lambda_{nh}^{**} = \beta_{n2} I_n h^{**} S_{nh}^{**}$
Let

$$\phi_1 \quad = (S_h^{**}, E_h^{**}, I_h^{**}, R_h^{**}, S_{nh}^{**}, E_{nh}^{**}, I_{nh}^{**}, R_{nh}^{**})$$

represent this unique endemic equilibrium. Consider the non-linear Lyapunov function of Goh-Volterra type:

$$\begin{aligned} V_2 \quad &= S_h - S_h^{**}\ln S_h + E_h - E_h^{**}\ln E_h + \frac{\gamma+\mu_h}{\gamma}(I_h - I_h^{**}\ln I_h) + \frac{(\gamma+\mu_h)(\tau_h+\mu_h)}{\gamma\tau_h}(R_h - R_h^{**}\ln R_h) \\ &+ S_{nh} - S_{nh}^{**}\ln S_{nh} + E_{nh} - E_{nh}^{**}\ln E_{nh} + \frac{\alpha+\mu_{nh}}{\alpha}(I_{nh} - I_{nh}^{**}\ln I_{nh}) + \\ &\frac{(\alpha+\mu_{nh})(\tau_{nh}+\mu_{nh})}{\alpha\tau_{nh}}(R_{nh} - R_{nh}^{**}\ln R_{nh}), \end{aligned}$$

We define the following steady state relations (obtained from the mass action version of the model at the endemic steady state ϕ_e):

$$\begin{aligned} \Lambda_h = (\beta_{n1} I_n h^{**} \quad &+ \beta_h I_h^{**}) S_h^{**} + \mu_h S_h^{**}, \quad (\gamma+\mu_h) E_h^{**} = (\beta_{n1} I_n h^{**} + \beta_h I_h^{**}) S_h^{**}, \\ (\tau_h+\mu_h) I_h^{**} \quad &= \gamma E_h^{**}, \quad \mu_h R_h^{**} = \tau_h I_h^{**}, \quad \Lambda_{nh} = \beta_{n2} I_{nh}^{**} S_{nh}^{**} + \mu_{nh} S_{nh}^{**}, \\ (\alpha+\mu_{nh}) E_{nh}^{**} \quad &= \beta_{nh} I_{nh}^{**} S_{nh}^{**}, \quad (\tau_{nh}+\mu_{nh}) I_{nh}^{**} = \alpha E_{nh}^{**}, \quad \mu_{nh} R_{nh}^{**} = \tau_{nh} I_{nh}^{**}. \end{aligned} \tag{6}$$

The Lyapunov derivative of V is given as

$$\begin{aligned} \dot{V} \quad &= (1 - \frac{S_h^{**}}{S_h})(\Lambda_h - (\beta_{n1} I_{nh} + \beta_h I_h) S_h - \mu_h S_h) \\ &+ (1 - \frac{E_h^{**}}{E_h})((\beta_{n1} I_{nh} + \beta_h I_h) S_h - (\gamma+\mu_h) E_h) \\ &+ \frac{\gamma+\mu_h}{\gamma}(1 - \frac{I_h^{**}}{I_h})(\gamma E_h - (\tau+\mu_h) I_h) \\ &+ \frac{(\gamma+\mu_h)(\tau_h+\mu_h)}{\gamma\tau_h}(1 - \frac{R_h^{**}}{R_h})(\tau_h I_h - \mu_h R_h) \\ &+ (1 - \frac{S_{nh}^{**}}{S_{nh}})(\Lambda_{nh} - \beta_{n2} I_{nh} S_{nh} - \mu_{nh} S_{nh}) \\ &+ (1 - \frac{E_{nh}^{**}}{E_{nh}})(\beta_{n2} I_{nh} S_{nh} - (\alpha+\mu_{nh}) E_{nh}) + \frac{\alpha+\mu_{nh}}{\alpha}(1 - \frac{I_{nh}^{**}}{I_{nh}})(\alpha E_{nh} - \mu_{nh} I_{nh}) + \\ &\frac{(\alpha+\mu_{nh})(\tau_{nh}+\mu_{nh})}{\alpha\tau_{nh}}(1 - \frac{R_{nh}^{**}}{R_{nh}})(\tau_{nh} I_{nh} - \mu_{nh} R_{nh}). \end{aligned} \tag{7}$$

Substituting the steady state relations in (6) and in (7) (after some algebraic simplifications) gives the following in $\mathcal{D}$ and noting that the state variables of the model are bounded by the values of their endemic steady state in $\mathcal{D}$,

$$\dot{V} \leq \mu_h S_h^{**} \left[2 - \frac{S_h^{**}}{S_h} - \frac{S_h}{S_h^{**}}\right] + (\gamma + \mu_h)E_h^{**}\left[5 - \frac{S_h^{**}}{S_h} - \frac{E_h^{**}S_h}{E_h S_h^{**}} - \frac{I_h^{**}E_h}{I_h E_h^{**}} - \frac{R_h^{**}I_h}{R_h I_h^{**}} - \frac{R_h}{R_h^{**}}\right]$$
$$+\mu_{nh} S_{nh}^{**} \left[2 - \frac{S_{nh}^{**}}{S_{nh}} - \frac{S_{nh}}{S_{nh}^{**}}\right] + (\alpha + \mu_{nh})E_{nh}^{**}\left[5 - \frac{S_{nh}^{**}}{S_{nh}} - \frac{E_{nh}^{**}S_{nh}}{E_{nh}S_{nh}^{**}} - \frac{I_{nh}^{**}E_{nh}}{I_{nh}E_{nh}^{**}} - \frac{R_{nh}^{**}I_{nh}}{R_{nh}I_{nh}^{**}} - \frac{R_{nh}}{R_{nh}^{**}}\right].$$

Finally, since the arithmetic mean exceeds the geometric mean, the following inequalities hold.

$$\left[2 - \frac{S_h^{**}}{S_h} - \frac{S_h}{S_h^{**}}\right] \leq 0, \quad \left[5 - \frac{S_h^{**}}{S_h} - \frac{E_h^{**}S_h}{E_h S_h^{**}} - \frac{I_h^{**}E_h}{I_h E_h^{**}} - \frac{R_h^{**}I_h}{R_h I_h^{**}} - \frac{R_h}{R_h^{**}}\right] \leq 0$$
$$\left[2 - \frac{S_{nh}^{**}}{S_{nh}} - \frac{S_{nh}}{S_{nh}^{**}}\right] \leq 0, \quad \left[5 - \frac{S_{nh}^{**}}{S_{nh}} - \frac{E_{nh}^{**}S_{nh}}{E_{nh}S_{nh}^{**}} - \frac{I_{nh}^{**}E_{nh}}{I_{nh}E_{nh}^{**}} - \frac{R_{nh}^{**}I_{nh}}{R_{nh}I_{nh}^{**}} - \frac{R_{nh}}{R_{nh}^{**}}\right] \leq 0.$$

Thus, we have that $\dot{V} \leq 0$. Hence, V is a Lyapunov function in $\mathcal{D}$.

4 NUMERICAL SIMULATION

The model (1) will be simulated to illustrate the impact of some parameters on the transmission dynamics of monkey pox. The numerical simulations are carried out using the parameters values in Table 2.

Table 2: Description of variables and parameters in the model (1)

Parameter	Value	References
Λ_h	0.029/yr	**[11]**
μ_h	0.02/yr	**[11]**
β_h	0.75/yr	**[11]**
β_{n1}	1.5/yr	**[11]**
β_{n2}	3/yr	**[11]**
γ	30/yr	**[5]**
τ_h	0.45/yr	**[7]**
τ_{nh}	0.3/yr	**[7]**
δ_1	0.15/yr	**[11]**
Λ_{nh}	2000/yr	**[11]**
μ_{nh}	1.5/yr	**[11]**
α	120/yr	**[5]**
δ_2	0.4/yr	**[5]**

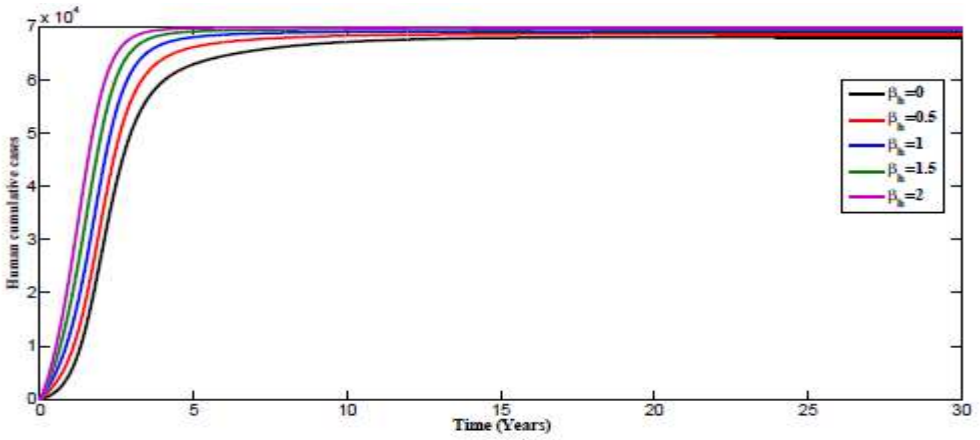

Figure 2: Simulation of the model (1) with varying values of β_h

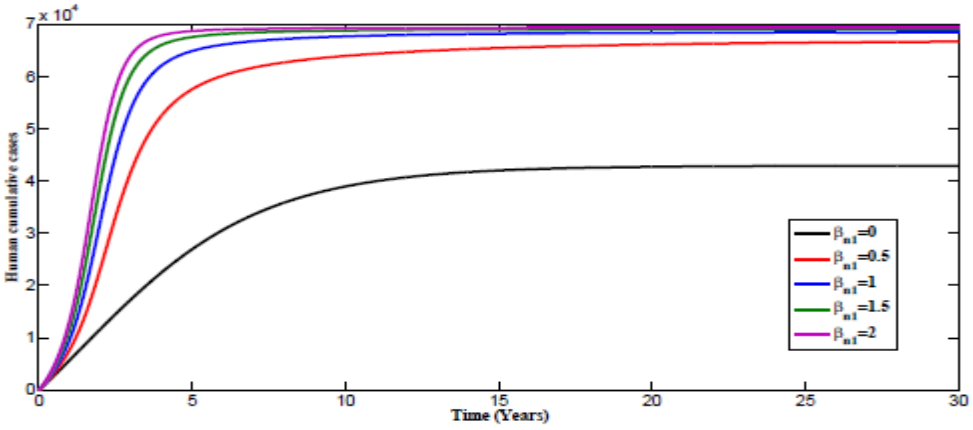

Figure 3: Simulation of the model (1) with varying values of β_{n1}

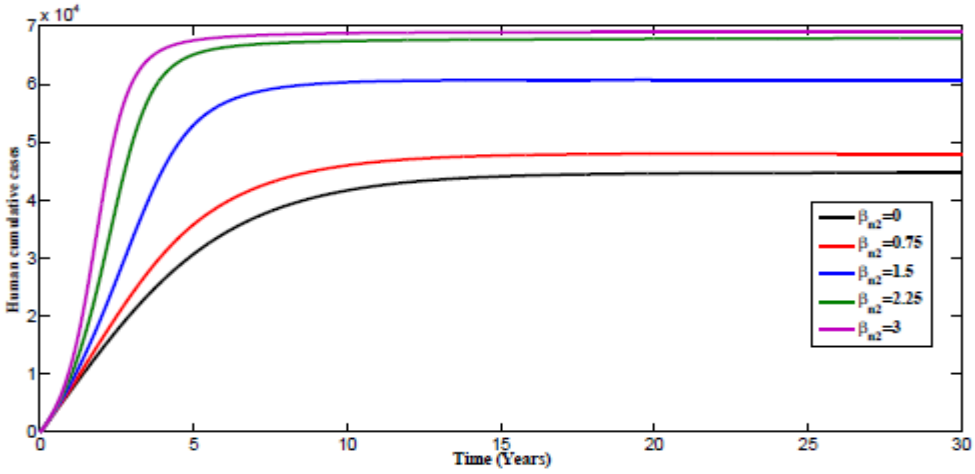

Figure 4: Simulation of the model (1) with varying values of β_{n2}

We see from Figure 2 that varying the value of β_h has minimal impact on the human cumulative cases of monkey pox. However, we see from Figures 3 and 4 that β_{n1} and β_{n2} has greater impact

on the human monkey pox dynamics. These results suggest that controlling the disease in the human population also depends on reducing the contacts between humans and the reservoirs an well as the controlling the spread in the non-human population

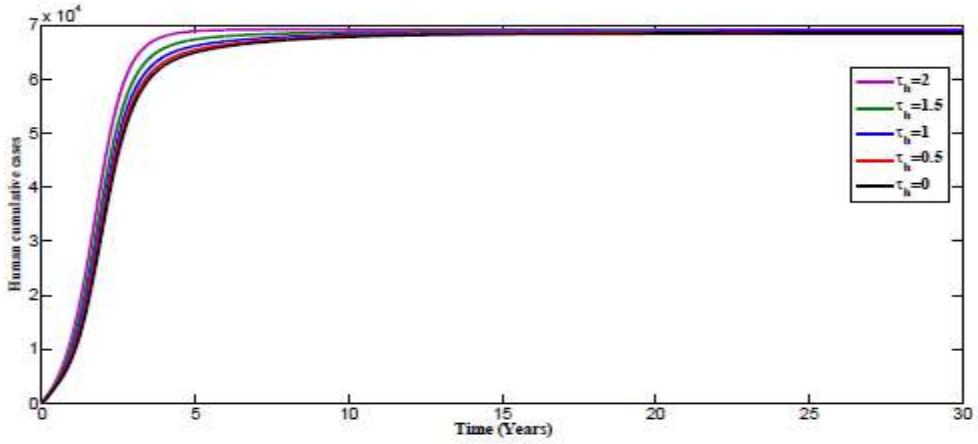

Figure 5: Simulation of the model (1) with varying values of τ_h

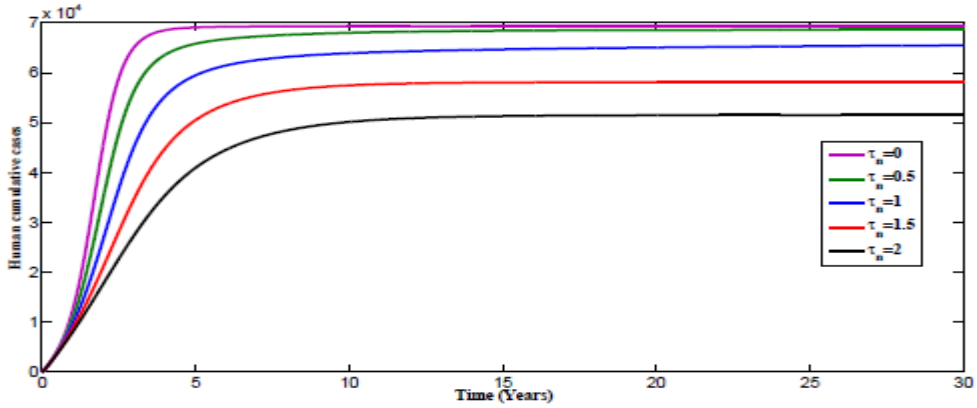

Figure 6: Simulation of the model (1) with varying values of τ_n

Again, Figures 5 and 6 further establishes this point as the recovery of infectious non-humans was seen to be more significant in reducing the cumulative number of cases in the human population than the recovery of infectious humans.

Sensitivity Analysis

We assess the impact of the parameters in each of the basic reproduction numbers by computing the sensitivity indices. According to the approach in [12], the sensitivity index $\mathcal{R}_0$ to a parameter say 'a' where a is any parameter in the basic reproduction number $\mathcal{R}_0$ is given by

$$\Gamma_a^{\mathcal{R}_0} = \frac{\delta \mathcal{R}_0}{\delta a} \times \frac{a}{\mathcal{R}_0}$$

These indices can be used to quantify the relative changes in the basic reproduction number in response to the changes in each of the parameters in it. Thus, we can identify critical parameters that are significant for disease control. Table 3 gives the sensitivity indices of the parameters in the basic reproduction numbers ($\mathcal{R}_{0h}$ and $\mathcal{R}_{0n}$)using the parameters values in Table 2.

Table 3: Sensitivity indices of parameters in the model (1)

Parameter	Sensitivity index
μ_h	-0.0329
β_h	+1
β_{n2}	+1
γ	+0.0007
τ_h	-0.726
τ_{nh}	-0.136
δ_1	-0.242
μ_{nh}	-0.694
α	+0.0123
δ_h	-0.182

We can see from Table 3 that the basic reproduction number is most sensitive to the transmission rates β_h and β_{n2} with a sensitivity index of +1 each. This means that 1% increase in the transmission rates (β_h and β_{n2}) will also lead to 1% increase in $\mathcal{R}_{oh}$ and $\mathcal{R}_{on}$ respectively. It is also important to state that the human basic reproduction number $\mathcal{R}_{oh}$ is also sensitive to human recovery rate τ_h while the non-human basic reproduction number $\mathcal{R}_{on}$ is also sensitive to the natural mortality rate μ_{nh} of non-humans. While β_h is positively correlated with$\mathcal{R}_{oh}$, τ_h is negatively correlated with $\mathcal{R}_{oh}$. Also, while is positively correlated with $\mathcal{R}_{on}, \mu_h$ is negatively correlated with $\mathcal{R}_{on}$

DISCUSSIONS

A new mathematical model for the transmission dynamics of monkey-pox in a community is designed (and rigorously analyzed) and used to assess how monkey-pox can be controlled or managed in a population. Some of the theoretical findings are given below.

(i) The model (1) has a disease free equilibrium (DFE) (when there are no infection in the population). The disease free equilibrium is locally asymptotically stable whenever the associated reproduction number is less than unity. Also, the DFE is globally asymptotically stable in the absence of disease induced mortality for humans whenever the associated reproduction number is less than unity.
(ii) The model (1) has a globally asymptotically stable endemic equilibrium in the absence of disease induced mortality for humans whenever the associated reproduction number is greater than unity.

Numerical simulations of the model (1), showed that the transmission rates from non-human to non-human (β_{n2}) and non-human to human (β_{n1}) have greater impact on the cumulative number of monkey-pox cases amongst humans that the transmission rate from human to human (β_h). Also, we saw that the recovery rate of non-humans had more significant impact on the cumulative number of cases of monkey-pox than the human recovery rate; re-emphasizing the first result. Again, sensitivity analysis showed that the transmission rates β_h and β_{n2} had the most significant influence on the reproduction number.
Thus we can see that though human to human transmission may contribute to the incidence of monkey pox in a population, the non-human to human transmission is seen to play more significant role in sustaining the transmission of monkey pox in a human population. Therefore, the need to educate people on how to curb the spread of monkey-pox from person-to-person may be important. However, educating people on proper handling of infected animals is crucial in limiting the spread

of this disease among humans. Thus, public health policies should focus more on reducing transmission among the non-human population and also transmission from non-humans to humans. Raising awareness of risk factors and enlightening people about measures that will lead to reduced exposure is the main strategy for monkey-pox prevention and control.

REFERENCES

[1] Durski K.N., McCollum A.M., Nakazawa Y., Petersen B.W., Reynolds M.G., Briand S., Djingarey M.H., Olson V., Damon I.K. & Khalakdina A. (2018) Emergence of Monkey-pox West and Central Africa, 1970-2017.Morbidity and Mortality Weekly Report 67(10):306-310

[2] Shchelkunov S.N., Marennikova S.S. & Moyer R.W. (2006) Orthopox viruses pathogenic for humans. New York: Springer Science & Business Media

[3] SklenovskÃ¡ N. & Van Ranst M. (2018) Emergence of monkey-pox as the most important orthopoxvirus infection in humans. Frontiers in Public Health 6:241.

[4] Jezek Z., Szczeniowski M., Paluku K. & Mutombo M. (1987) Human Monkey-pox: clinical features of 282 patients. Journal of Infectious Diseases 156(2):293-298

[5] Bankuru S.V., Kossol S., Hou W., Mahmoudi P., RychtÃ¡ÅTM J., Taylor D. (2020) A game-theoretic model of Monkeypox to assess vaccination strategies. PeerJ 8:e9272 http://doi.org/10.7717/peerj.9272

[6] Eteng W.E., Mandra A., Doty J., Yinka-Ogunleye A., Aruna S., Reynolds M.G., McCollum A.M., Davidson W., Wilkins K. & Saleh M.(2018) Notes from the Field: Responding to an Outbreak of Monkey-pox using the One Health Approach Nigeria, 2017-2018. Morbidity and Mortality Weekly Report 67(37):1040-1041

[7] Di Giulio D.B. & Eckburg P.B. (2004) Human monkey-pox: an emerging zoonosis. The Lancet Infectious Diseases 4(1):15-25

[8] Usman, S. and Adamu, I.I. (2017) Modeling the Transmission Dynamics of the Monkey-pox Virus Infection with Treatment and Vaccination Interventions. Journal of Applied Mathematics and Physics, 5, 2335-2353.

[9] Somma, S. A, Akinwande, N. I. and Chado, U. D A Mathematical Model of Monkey pox Virus Transmission Dynamics Ife Journal of Science vol.21, no. 1 (2019)

[10] Bhunu, C.P. and Mushayabasa, S. (2011) Modeling the Transmission Dynamics of Pox-Like Infections. International Journal of Applied Mathematics,41, 2.

[11] Chitnis N., Hyman, J.M., Cushing, J.M., (2008). Determining important parameters in the spread of malaria through the sensitivity analysis of a mathematical model. Bulletin of Mathematical Biology 70, 1272-1296.