



MATHEMATICAL MODELLING AND STABILITY ANALYSIS OF MONKEY POX TRANSMISSION DYNAMICS IN NIGERIA

*Fatoba O. J., Atuji, S. S., Dashe, N. E., Akogwu, B. O., Ukoh, E. E., Udoh, I. J.

Applied Mathematics and Simulation Advanced Research Centre, Sheda Science and Technology Complex, Sheda-Kwali, FCT, Nigeria

*Corresponding authors' email: oluwaseun.fatoba@shestco.gov.ng

ABSTRACT

In this paper, we proposed a mathematical model for monkey pox disease dynamics. This model is divided into two sub-population which is a system of non-linear differential equations. It is made up of seven (7) compartments such as the Susceptible, the Infectious, the Treatment, the Recovery, the Susceptible, the Infectious, and the Recovery (SITR-SIR). The model is formulated with the aid of a schematic diagram using appropriate parameters. The model analysis was carried out to show the feasible region, the disease-free equilibrium points, the basic reproduction number, and the local stability of the model. The model was solved to show the effect of the parameters.

Keywords: Monkey Pox, Non-linear Differential Equations, Jacobian Matrix, Maple21, SITR-SIR, Nigeria

INTRODUCTION

Monkey pox is a viral illness caused by the *monkey pox virus*, a specie of the genus *Orthopoxvirus* which includes camel pox, cowpox, vaccinia, and variola viruses. The virus is the foremost *Orthopoxvirus* affecting human populations since the extinction of smallpox, confirmed by the World Health Organization in 1980 (Andrea & Inger, 2013).

Monkey pox is caused by a rodent virus, which occurs mostly in West and Central Africa close to tropical rainforests. It is caused by the monkey pox virus in the *Poxviridae* family, which belongs to the genus *Orthopoxvirus*. The identification of the monkey pox virus is based on biological characteristics and endonuclease patterns of viral DNA. In contrast to smallpox, the monkey pox virus can infect rabbit skin and can be transmitted serially by intra-cerebral inoculation of mice (Olumuyiwa et al, 2021)

It was first reported in 1959 as an outbreak of a pox-like disease in monkeys kept at a research institute in Copenhagen, Denmark. The first human Monkey Pox case in medical history was recognized on the 1st of September 1970, when a nine-month-old child was admitted to the Basankusu Hospital in the Democratic Republic of Congo. Six cases of humans were described in Liberia, Nigeria, and Sierra Leone between October 1970 and May 1971.

The first index case in Nigeria was recorded in 1971 and subsequently, 10 Monkey Pox cases were reported between 1971 and 1978. (Emmanuel et al, 2020). Monkey pox re-emerged on 24th of September 2017 with a total of 88 cases confirmed in Bayelsa state. Between September 2017 to June 19th in the year 2022, the Nigeria Centre for Disease control (NCDC) recorded a total of 674 suspected cases, a total 267 confirmed cases, and a total of 9 death (NCDC, 2022).

The primary mode of infection is when a person has been in contact with the infected animals or their body fluids (Silas & Ikechukwu, 2019). It can spread to anyone through close contact including direct contact with monkey pox rash, sores, or scabs. Contact with objects, fabrics (clothing, bedding, or towels), and surfaces that have been used by someone with monkeypox, when an infected person comes in contact with a human, animal, or material contaminated with the virus, the infection can gain entrance through eyes, nose, mouth, broken skin. Infections have been known to occur through the contact of infected animals such as rats, monkeys, and squirrels since such rodents are the main carriers for Monkey Pox Virus. It is

therefore important for individuals to be careful not to eat uncooked meat to avoid ingesting an infected animal.

The incubation period for monkey pox is usually 7 to 14 days but can also range between 5 to 21 days; it begins with swollen lymph nodes, Fever, Chills, Headache, Muscle aches, and Exhaustion. After the patient has developed fever and chills, within a few days, the patient usually begins to develop the usual rash that comes with the Monkey Pox Virus, the rash starts from the face and spreads to the rest of the body.

The lesions progress through the body in the following stages - Macules, Papules, Vesicles, Pustules, and Scabs before falling off. The disease cycles last about 2 to 4 weeks for the many that survive it. As many as 1 in 10 infected patients die from this virus.

To have a robust knowledge of the disease dynamics of monkey pox, various studies that involved mathematical models of infectious diseases are reviewed. The mathematical model gives a thorough analysis of the dynamics of the disease to assist the public health authorities makes proper decisions and policies. These studies are also powerful tools for predicting the future effect of a particular disease (Silesh, Henok, & Tadesse, 2023). Since the inception of monkeypox, only a few mathematical models have been proposed (Emeka et al, 2018).

(Somma et al, 2019) developed a mathematical model of monkey pox virus transmission dynamics with two interacting host populations, which are the humans and the rodent host. They incorporated the quarantine class and the public enlightenment campaign parameters into the human population as a means of controlling the spread of the disease. They obtained the Disease Free Equilibrium (DFE), Endemic Equilibrium (EE) and as well computed the basic reproduction numbers for the analysis. The authors analyzed the Disease Free Equilibrium (DFE) for stability by using Jacobian matrix techniques and the Lyapunov function. They concluded that the local and global stability of the DFE are stable if $R_{0r} < 1$ and $R_{0h} < 1$ which implies that the disease will not persist in the population.

(Bhunu & Mushayabasa, 2011) developed a mathematical model for the transmission dynamic of monkeypox. They used the Lyapunov approach to show the global stability of the non-human endemic equilibrium. By using the center manifold theory, they were able to show that the endemic equilibrium point in both the human and non-human

population of the model is locally asymptotically stable. The numerical simulations carried suggested that the immune status of the human tends to vary in the way humans recover following infection with the orthopoxvirus.

(Sulaiman & Ibrahim, 2017) developed a mathematical model for the dynamics of the transmission of monkeypox virus infection with control strategies of combined vaccine and treatment interventions. Using standard approaches, they established two equilibria for the model namely: disease-free and endemic. The disease-free equilibrium was proven to be both locally and globally asymptotically stable. They used a next-generation matrix and the comparison theorem to compute the basic reproduction number and the linearization plus row-reduction method to prove the local stability. Numerical simulations carried out on the model revealed that the infectious individuals in the human and non-human primates' populations will die out in the course of the proposed interventions. They also carried out the sensitivity analysis on the model parameters to show that the basic reproduction numbers of the model which served as a threshold for measuring new infections in the host populations decreased, while the control parameters of vaccination and treatment increased.

(Emeka et al, 2018) developed a deterministic mathematical model for the transmission dynamics of the Monkey pox virus. Their model incorporates an imperfect vaccine compartment for the human sub-population. The equilibrium states of the model equation were derived and analyzed for stability. The system was shown to have one unique endemic equilibrium which is stable when $R_{0v} < 1$, this eradicated the possibility of backward bifurcation, which indicates that interventions capable of reducing the basic effective reproductive (R_{0v}) less than unity will be adequate to contain the infection. Numerical simulation was carried out to underscore the role of weak, medium, and strong immune systems of the epidemiological states, as well as the effect of infection and vaccination rates on the prevalence and vulnerability respectively.

(Olumuyiwa et al, 2021) developed and analyzed a deterministic mathematical model for the monkeypox virus. Both local and global asymptotic stability conditions for disease-free and endemic equilibria are determined. It was shown that the model went through backward bifurcation, where the locally stable disease-free equilibrium coexists with an endemic equilibrium. Furthermore, they determined conditions under which the disease-free equilibrium of the model was globally asymptotically stable. Finally, numerical simulations were carried out to demonstrate their findings. Their findings indicated that isolation of infected individuals in the human population helps to reduce the spread of the disease.

TeWinkel (2019) expanded an existing model to incorporate a situation where the contact rate is a function of time and not simply a constant and they add more than two populations to the model. They carried out the global and local asymptotic stability of the model's equilibrium points. They proved that

the global asymptotic stability of the endemic equilibrium has been previously incomplete. The results of their numerical simulations for the original model and the modified models were compared. In this study, we aimed at developing a new model to incorporate the treatment class for the monkey pox disease transmission dynamics in Nigeria using the available information from the NCDC dataset.

MATERIALS AND METHODS

Monkey Pox Model

The mathematical model of the Monkey Pox (MPX) disease dynamics is a system of non-linear differential equations. The model formulation is made up of two populations, the host population (Humans) and the non-human primates (Rodents) population. The Human population is subdivided into four (4) compartments, the Susceptible (S_h), the Infectious (I_h), the Treated (T_h), and the Recovery (R_h). In our model formulations we assumed that, for Susceptible humans (S_h), the population is increased by recruitment rate Λ_h . The susceptible individual becomes exposed to the monkeypox virus after getting into contact with an infected human or infected rodent at a rate. Our force of infection for the human population is given as

$$\lambda_h = a \frac{I_r}{N_r} + b \frac{I_h}{N_h} \tag{1}$$

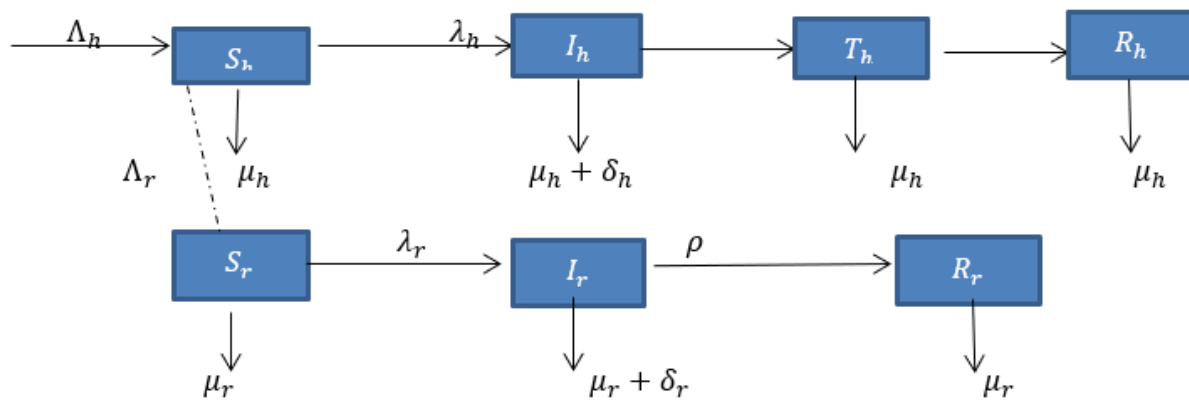
where a is the product of the contact rate and probability of the human being infected per contact with an infectious rodent, and b is the product of the contact rate and the probability of the human being infected with monkey pox virus after getting into contact with an infectious human per contact. After the incubation period, the infected individual (I_h) either dies die naturally, or gets treated at a rate (α), and the treated humans either die naturally or recover with permanent immunity after receiving treatment at (ξ)The Rodents population is subdivided into three (3) compartments, the Susceptible (S_r), the Infectious (I_r), and the Recovery (R_r). We assumed that rodent population are recruited into the susceptible population (S_r) at a constant birth rate of Λ_r and becomes exposed to the monkeypox virus after getting into contact with an infected rodent at a rate λ_r . Therefore, the force of infection for the rodent is given as

$$\lambda_r = \frac{m I_r}{N_r} \tag{2}$$

where m is the product of contact rate and probability of the rodent getting infected per contact. The susceptible rodent moved to the infected population I_r at a rate m . The infected rodents I_r are capable of either; infecting other rodents when they come into contact, dying due to the disease at a rate, or recovering naturally with permanent immunity at a rate ρ and moving into the recovery population R_r .

Schematic Representation of the Model

A schematic representation of our assumptions for the dynamics of the monkeypox virus to both the human and rodent population is shown below



The Model Equations

The mathematical equations for the model is derived from the schematic representation in figure 2 above, we have the following equations for the human and rodents compartments.

$$\frac{dS_h}{dt} = \Lambda_h - (\lambda_h + \mu_h)S_h \tag{3}$$

$$\frac{dI_h}{dt} = \lambda_h S_h - (\alpha + S_h + \mu_h)I_h \tag{4}$$

$$\frac{dT_h}{dt} = \alpha I_h - (\xi_h + \mu_h)T_h \tag{5}$$

$$\frac{dR_h}{dt} = \xi_h T_h - \mu_h R_h \tag{6}$$

$$\frac{dS_r}{dt} = \Lambda_r - (\lambda_r + \mu_r)S_r \tag{7}$$

$$\frac{dI_r}{dt} = \lambda_r S_r - (\rho + \delta_r + \mu_r)I_r \tag{8}$$

$$\frac{dR_r}{dt} = \rho I_r - \mu_r R_r \tag{9}$$

The initial conditions that are used are the cumulative values for the various classes, which are taken from the Nigeria Centre for Disease Control (NCDC, 2022). The initial conditions and parameters are described in the table below.

Table 1: Description of variables and parameters

SYMBOL	DESCRIPTION OF VARIABLES AND PARAMETERS	SOURCES
S_h	Individuals who are susceptible to the disease = 674	NCDC,2022
I_h	Individuals per unit of time who are infected with the disease = 267	NCDC,2022
R_h	Individuals per unit of time who recovered from the disease = 258	NCDC,2022
T_H	Individuals per unit of time who are treated from the disease = 267	NCDC,2022
S_r	Rodents that are susceptible to the disease = 1000	Assumed
I_r	Rodents per unit of time that are infected with the disease = 400	Assumed
R_r	Rodents per unit of time that recovered from the disease = 400	Assumed
Λ_h	Recruitment Rate of Humans = 0.02	Usman and Adamu, (2017)
Λ_r	Recruitment Rate of Rodents = 0.1	Usman and Adamu, (2017)
μ_h	Death Rate of Humans = 0.1	Usman and Adamu, (2017)
μ_r	Death Rate of Rodent = 0.3961	Estimated
λ_h	Infectious Rate of humans = 0.00058	Estimated
λ_r	Infectious Rate of Rodents = 0.0006	Estimated
α	Treatment Rate = 0.001	Estimated
ξ_h	Recovery rate of humans = 0.9663	Estimated
ρ	Recovery rate of rodent = 0.01	Usman and Adamu, (2017)
a	Contact rate of human to rodent = 0.00252	Usman and Adamu, (2017)

b	Contact rate of human to human = 0.000063	Usman and Adamu, (2017)
w	Contact rate of human to human = 0.000063	Usman and Adamu, (2017)

Analysis of the Model:

Feasible Region:

Let the region $\varphi = \{(S_h, I_h, T_h, R_h, S_r, I_r, R_r) \in \mathbb{R}_+^7\}$ be the solution of the problem for the model equation (3-9) with the initial conditions in the biological feasible region $\omega = \omega^h \times \omega^r$. Then, for Human population, $\omega^h = S_h, I_h, T_h, R_h \in \mathbb{R}_+^4: N_h(t) \leq \frac{\Lambda_h}{\mu_h}$, and, for Rodent population, $\omega^r = S_r, I_r, R_r \in \mathbb{R}_+^3: N_r(t) \leq \frac{\Lambda_r}{\mu_r}$. Therefore, ω is a non-negative invariant region.

Proof:

For the human population, $N_h = S_h + I_h + T_h + R_h$ (10)

Then, the rate of change of equation (10) is given by $\frac{dN_h}{dt} = \frac{dS_h}{dt} + \frac{dI_h}{dt} + \frac{dT_h}{dt} + \frac{dR_h}{dt} \geq 0$ (11)

or

$$N_h' = S_h' + I_h' + T_h' + R_h'$$

Substituting equation (3-7) into equation (10), we obtain

$$N_h' = \Lambda_h - (\lambda_h + \mu_h)S_h + \lambda_h S_h - (\alpha + \delta_h + \mu_h)I_h + \alpha I_h - (\xi_h + \mu_h)T_h + \xi_h T_h - \mu_h R_h \geq 0$$
 (12)

$$N_h' = \Lambda_h - \mu_h S_h - S_h I_h - \mu_h I_h - \mu_h T_h - \mu_h R_h \geq 0$$

$$N_h' = \Lambda_h - \mu_h(S_h + I_h + T_h + R_h) - S_h I_h \geq 0$$

$$N_h' = \Lambda_h - \mu_h N_h - S_h I_h \leq \Lambda_h - \mu_h N_h \geq 0$$

$$N_h' = \Lambda_h - \mu_h N_h \geq 0$$

$$N_h' = N_h \leq \frac{\Lambda_h}{\mu_h}$$
 (13)

We set the initial time at $t = 0$, we have

$$N_h(0) \geq 0$$

Clearly $S_h(0) \geq 0, I_h(0) \geq 0, T_h(0) \geq 0$ and $R_h(0) \geq 0$ are the non-negative initial condition

Similarly for the Rodents population we have $N_r = S_r + I_r + R_r$ (14)

Then, the rate of change of equation (14) is given by

$$\frac{dN_r}{dt} = \frac{dS_r}{dt} + \frac{dI_r}{dt} + \frac{dR_r}{dt} \geq 0$$
 (15)

or

$$N_r' = S_r' + I_r' + R_r'$$

Also, by substituting equations (7-9) into equation (15), we obtain

$$\Lambda_r - (\lambda_r + \mu_r)S_r + \lambda_r S_r - (\rho + \delta_r + \mu_r)I_r + \rho I_r - \mu_r R_r \geq 0$$

$$\Lambda_r - \mu_r S_r - \delta_r I_r - \mu_r I_r - \mu_r R_r \geq 0$$

$$\Lambda_r - \mu_r(S_r + I_r + R_r) - \delta_r I_r \geq 0$$

$$\Lambda_r - \mu_r N_r - \delta_r I_r \geq 0$$

But

$$\Lambda_r - \mu_r N_r - \delta_r I_r \leq \Lambda_r - \mu_r N_r \geq 0$$

$$= N_r \leq \frac{\Lambda_r}{\mu_r}$$
 (16)

Setting the initial time at $t = 0$, we have

$$= N_r(0) \geq 0$$
 (17)

Clearly $S_r(0) \geq 0, I_r(0) \geq 0$ and $R_r(0) \geq 0$ are the non-negative initial condition

Hence the set ω is positive invariant. Therefore the model is feasible since it is positive invariant on the domain over time t.

Model Disease Free Equilibrium Points

This an equilibrium state where at least one of the infected compartments of the model is non-zero. To obtain the Disease free equilibrium point ϵ_0 , we set the systems of equation (3-9) to zero to obtain a nonlinear system

$$\frac{dS_h}{dt} = \frac{dI_h}{dt} = \frac{dT_h}{dt} = \frac{dR_h}{dt} = \frac{dS_r}{dt} = \frac{dI_r}{dt} = \frac{dR_r}{dt} = 0$$

$$\Lambda_h - (\lambda_h + \mu_h)S_h = 0$$

$$\lambda_h S_h - (\alpha + \delta_h + \mu_h)I_h = 0$$

$$\alpha I_h - (\xi_h + \mu_h)T_h = 0$$
 (18)

$$\xi_h T_h - \mu_h R_h = 0$$

$$\Lambda_r - (\lambda_r + \mu_r)S_r = 0$$

$$\lambda_r S_r - (\rho + \delta_r + \mu_r)I_r = 0$$

$$\rho I_r - \mu_r R_r = 0$$

Setting $I_h = 0$ and $I_r = 0$, we obtain $T_h, R_h = 0$ and $R_r = 0$. We then set $\lambda_h = 0$ and $\lambda_r = 0$,

Then equation (3) and (7) becomes

$$S_h = \frac{\Lambda_h}{\mu_h} \text{ and } S_r = \frac{\Lambda_r}{\mu_r}$$

hence

$$\epsilon_0 = (S_h, I_h, T_h, R_h, S_r, I_r, R_r) = \left(\frac{\Lambda_h}{\mu_h}, 0, 0, 0, \frac{\Lambda_r}{\mu_r}, 0, 0\right)$$
 (19)

Basic Reproduction Number and Local Stability Analysis of the Model

i. Basic Reproduction Number Point

The Basic Reproduction Number (R_0) is an important parameter that defines the average number of secondary infections caused by an individual in a susceptible population. This number indicated whether the infection will spread through the population or not. R_0 is established using the next generation operator method on the DFE (ϵ_0) using the next generation matrix method as described in (Driessche & Wathmough, 2002). The matrices F and for the new infection terms and the remaining transfer terms are given by

$$\frac{dI_h}{dt} = \lambda_h S_h - (\alpha + S_h + \mu_h)I_h$$

$$\frac{dT_h}{dt} = \alpha I_h - (\xi_h + \mu_h)T_h$$
 (20)

$$\frac{dI_r}{dt} = \lambda_r S_r - (\rho + \delta_r + \mu_r)I_r$$

Substituting

$$\lambda_h = a \frac{I_r}{N_r} + b \frac{I_h}{N_h} \text{ and } \lambda_r = \frac{m I_r}{N_r}, \text{ we obtain}$$

$$\frac{dI_h}{dt} = \left(a \frac{I_r}{N_r} + b \frac{I_h}{N_h}\right) S_h - (\alpha + S_h + \mu_h)I_h = \phi_1$$

$$\frac{dT_h}{dt} = \alpha I_h - (\xi_h + \mu_h)T_h = \phi_2$$
 (21)

$$\frac{dI_r}{dt} = \frac{mI_r S_r}{N_r} - (\rho + \delta_r + \mu_r)I_r = \phi_3$$

Let $x = (I_h, T_h, I_r)^T$, then the equation () can be written as

$$\frac{dx}{dt} = F(x) - V(x) \tag{22}$$

where

$$F(x) = \begin{pmatrix} (a \frac{I_r}{N_r} + b \frac{I_h}{N_h}) S_h \\ 0 \\ \frac{mI_r S_r}{N_r} \end{pmatrix}, V(x) = \begin{pmatrix} -(\alpha + S_h + \mu_h)I_h \\ \alpha I_h - (\xi_h + \mu_h)T_h \\ -(\rho + \delta_r + \mu_r)I_r \end{pmatrix} \tag{23}$$

Evaluating the derivative of F and V at the DFE (ϵ_0) point, gives

$$F = \begin{pmatrix} \frac{d\phi_1}{dI_h} & \frac{d\phi_1}{dT_h} & \frac{d\phi_1}{dI_r} \\ \frac{d\phi_2}{dI_h} & \frac{d\phi_2}{dT_h} & \frac{d\phi_2}{dI_r} \\ \frac{d\phi_3}{dI_h} & \frac{d\phi_3}{dT_h} & \frac{d\phi_3}{dI_r} \end{pmatrix} = F = \begin{pmatrix} \frac{b S_h}{N_h} & 0 & \frac{\alpha S_h}{N_r} \\ 0 & 0 & 0 \\ 0 & 0 & \frac{m S_r}{N_r} \end{pmatrix}, F(\epsilon_0) = \begin{pmatrix} \frac{\Lambda_h b}{\mu_h N_h} & 0 & \frac{\alpha \Lambda_h}{\mu_h N_r} \\ 0 & 0 & 0 \\ 0 & 0 & \frac{m \Lambda_r}{\mu_r N_r} \end{pmatrix} \tag{24}$$

$$V = \begin{pmatrix} -(\alpha + \delta_h + \mu_h) & 0 & 0 \\ \alpha & -(\xi + \mu_h) & 0 \\ 0 & 0 & -(\rho + \delta_r + \mu_r) \end{pmatrix} \tag{25}$$

Let $c_1 = \alpha + \delta_h + \mu_h$, $c_2 = \xi + \mu_h$ and $c_3 = \rho + \delta_r + \mu_r$

Thus

$$V = \begin{pmatrix} -c_1 & 0 & 0 \\ \alpha & -c_2 & 0 \\ 0 & 0 & -c_3 \end{pmatrix}$$

$$V^{-1} = \begin{pmatrix} -\frac{1}{c_1} & 0 & 0 \\ -\frac{\alpha}{c_1 c_2} & -\frac{1}{c_2} & 0 \\ 0 & 0 & -\frac{1}{c_3} \end{pmatrix}$$

$$FV^{-1} = \begin{pmatrix} \frac{\Lambda_h b}{\mu_h N_h} & 0 & \frac{\alpha \Lambda_h}{\mu_h N_r} \\ 0 & 0 & 0 \\ 0 & 0 & \frac{m \Lambda_r}{\mu_r N_r} \end{pmatrix} \begin{pmatrix} -\frac{1}{c_1} & 0 & 0 \\ -\frac{\alpha}{c_1 c_2} & -\frac{1}{c_2} & 0 \\ 0 & 0 & -\frac{1}{c_3} \end{pmatrix} \tag{26}$$

Also, let $d_1 = \frac{\Lambda_h b}{\mu_h N_h}$, $d_2 = \frac{\alpha \Lambda_h}{\mu_h N_r}$, and $d_3 = \frac{m \Lambda_r}{\mu_r N_r}$

$$FV^{-1} = \begin{pmatrix} -\frac{d_1}{c_1} & 0 & -\frac{d_2}{c_1} \\ -\frac{\alpha d_1}{c_1 c_2} & -\frac{\alpha}{c_2} & \frac{\alpha d_2}{c_1 c_2} \\ 0 & 0 & -\frac{d_3}{c_3} \end{pmatrix} = \begin{pmatrix} -\frac{d_1}{c_1} & 0 & -\frac{d_2}{c_1} \\ -\frac{\alpha(d_1 + c_1)}{c_1 c_2} & -\frac{1}{c_2} & \frac{\alpha d_2}{c_1 c_2} \\ 0 & 0 & -\frac{d_3}{c_3} \end{pmatrix}$$

$$= \begin{pmatrix} -\frac{\frac{b \Lambda_h}{\mu_h N_h}}{\alpha + \delta_h + \mu_h} & 0 & -\frac{\frac{\alpha \Lambda_h}{\mu_h N_r}}{\alpha + \delta_h + \mu_h} \\ -\frac{\alpha(\frac{\Lambda_h b}{\mu_h N_h} + (\alpha + \delta_h + \mu_h))}{(\alpha + \delta_h + \mu_h)(\xi + \mu_h)} & -\frac{1}{(\xi + \mu_h)} & \frac{\alpha(\frac{\alpha \Lambda_h}{\mu_h N_r})}{(\alpha + \delta_h + \mu_h)(\xi + \mu_h)} \\ 0 & 0 & -\frac{\frac{m \Lambda_r}{\mu_r N_r}}{\rho + \delta_r + \mu_r} \end{pmatrix} \tag{27}$$

The Reproduction number R_0 is the spectral- radius of the product of FV^{-1} which is given as

$$R_{0,h} = -\frac{\frac{b \Lambda_h}{\mu_h N_h}}{\alpha + \delta_h + \mu_h}, R_{0,r} = -\frac{\frac{m \Lambda_r}{\mu_r N_r}}{\rho + \delta_r + \mu_r} \tag{28}$$

where $R_{0,h}$ and $R_{0,r}$ are the monkey pox induced reproduction numbers for humans and rodents

Therefore, Theorem 1: The disease free equilibrium is locally asymptotically stable when $R_0 < 1$ and unstable when $R_0 > 1$.

Proof:

Since $R_{0,h}$ and $R_{0,r}$ are negative, the endemic equilibrium is locally asymptotically stable. That shows the proof.

ii. Local Stability of the Endemic Equilibrium (EE) Point

To obtain the Jacobian matrix J and evaluate it at ϵ_θ , then let

$$\phi_1 = \Lambda_h - \frac{\beta_1 I_r S_h}{N_h} - \frac{\beta_2 I_h S_h}{N_h} - \mu_h S_h \tag{29}$$

$$\begin{aligned}
 \phi_2 &= \frac{\beta_1 I_r S_h}{N_h} + \frac{\beta_2 I_h S_h}{N_h} - (\alpha + \delta_h + \mu_h) I_h \\
 \phi_3 &= \alpha I_h - (\xi_h + \mu_h) T_h \\
 \phi_4 &= \xi_h T_h - \mu_h R_h \\
 \phi_5 &= \Lambda_r - \frac{\beta_3 I_r S_r}{N_r} - \mu_r S_r \\
 \phi_6 &= \frac{\beta_2 I_r S_r}{N_r} - (\rho + \delta_r + \mu_r) I_r \\
 \phi_7 &= \rho I_r - \mu_r R_r
 \end{aligned} \tag{30}$$

Evaluating the derivative of equation (30), we have

$$J = \begin{pmatrix} \frac{\partial \phi_1}{\partial S_h} & \frac{\partial \phi_1}{\partial I_h} & \frac{\partial \phi_1}{\partial T_h} & \frac{\partial \phi_1}{\partial R_h} & \frac{\partial \phi_1}{\partial S_r} & \frac{\partial \phi_1}{\partial I_r} & \frac{\partial \phi_1}{\partial R_r} \\ \frac{\partial \phi_2}{\partial S_h} & \frac{\partial \phi_2}{\partial I_h} & \frac{\partial \phi_2}{\partial T_h} & \frac{\partial \phi_2}{\partial R_h} & \frac{\partial \phi_2}{\partial S_r} & \frac{\partial \phi_2}{\partial I_r} & \frac{\partial \phi_2}{\partial R_r} \\ \frac{\partial \phi_3}{\partial S_h} & \frac{\partial \phi_3}{\partial I_h} & \frac{\partial \phi_3}{\partial T_h} & \frac{\partial \phi_3}{\partial R_h} & \frac{\partial \phi_3}{\partial S_r} & \frac{\partial \phi_3}{\partial I_r} & \frac{\partial \phi_3}{\partial R_r} \\ \frac{\partial \phi_4}{\partial S_h} & \frac{\partial \phi_4}{\partial I_h} & \frac{\partial \phi_4}{\partial T_h} & \frac{\partial \phi_4}{\partial R_h} & \frac{\partial \phi_4}{\partial S_r} & \frac{\partial \phi_4}{\partial I_r} & \frac{\partial \phi_4}{\partial R_r} \\ \frac{\partial \phi_5}{\partial S_h} & \frac{\partial \phi_5}{\partial I_h} & \frac{\partial \phi_5}{\partial T_h} & \frac{\partial \phi_5}{\partial R_h} & \frac{\partial \phi_5}{\partial S_r} & \frac{\partial \phi_5}{\partial I_r} & \frac{\partial \phi_5}{\partial R_r} \\ \frac{\partial \phi_6}{\partial S_h} & \frac{\partial \phi_6}{\partial I_h} & \frac{\partial \phi_6}{\partial T_h} & \frac{\partial \phi_6}{\partial R_h} & \frac{\partial \phi_6}{\partial S_r} & \frac{\partial \phi_6}{\partial I_r} & \frac{\partial \phi_6}{\partial R_r} \\ \frac{\partial \phi_7}{\partial S_h} & \frac{\partial \phi_7}{\partial I_h} & \frac{\partial \phi_7}{\partial T_h} & \frac{\partial \phi_7}{\partial R_h} & \frac{\partial \phi_7}{\partial S_r} & \frac{\partial \phi_7}{\partial I_r} & \frac{\partial \phi_7}{\partial R_r} \end{pmatrix}$$

The local stability will be established using linearization method (Usman and Adamu, 2017) Therefore, the Jacobian matrix J of the model equations is given as

$$\begin{aligned}
 &= \begin{pmatrix} -\frac{\beta_1 I_r}{N_h} - \frac{\beta_2 I_h}{N_h} - \mu_h & -\frac{\beta_2 I_h}{N_h} & 0 & 0 & 0 & 0 & -\frac{\beta_1 S_h}{N_h} & 0 \\ \frac{\beta_1 I_r}{N_h} + \frac{\beta_2 I_h}{N_h} & \frac{\beta_2 S_h}{N_h} - (\alpha + \delta_h + \mu_h) & 0 & 0 & 0 & 0 & \frac{\beta_1 S_h}{N_h} & 0 \\ 0 & \alpha & -(\xi_h + \mu_h) & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \xi & -\mu_h & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\frac{\beta_3 I_r}{N_r} - \mu_r & -\frac{\beta_3 S_r}{N_r} & 0 & 0 \\ 0 & 0 & 0 & 0 & \frac{\beta_3 I_r}{N_r} & \frac{\beta_3 S_r}{N_r} - (\rho + \delta_r + \mu_r) & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \rho & -\mu_r & -\mu_r \end{pmatrix} \\
 J(\varepsilon_\theta) &= \begin{pmatrix} -\mu_h & -\frac{\beta_2 \Lambda_h}{\mu_h N_h} & 0 & 0 & 0 & -\frac{\beta_1 \Lambda_h}{\mu_h N_h} & 0 \\ 0 & -(\alpha + \delta_h + \mu_h) & 0 & 0 & 0 & \frac{\beta_1 \Lambda_h}{\mu_h N_h} & 0 \\ 0 & \alpha & -(\xi_h + \mu_h) & 0 & 0 & 0 & 0 \\ 0 & 0 & \xi & -\mu_h & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\mu_r & \frac{\beta_3 \Lambda_r}{\mu_r N_r} & 0 \\ 0 & 0 & 0 & 0 & 0 & -(\rho + \delta_r + \mu_r) & 0 \\ 0 & 0 & 0 & 0 & 0 & \rho & -\mu_r \end{pmatrix} \tag{31}
 \end{aligned}$$

Let $d_1 = \frac{\beta_2 \Lambda_h}{\mu_h N_h}$, $d_2 = \frac{\beta_1 \Lambda_h}{\mu_h N_h}$, $d_3 = \alpha + \delta_h + \mu_h$, $d_4 = \xi_h + \mu_h$, $d_5 = \frac{\beta_3 \Lambda_r}{\mu_r N_r}$, $d_6 = \rho + \delta_r + \mu_r$

$$J(\varepsilon_\theta) = \begin{pmatrix} -\mu_h & -d_1 & 0 & 0 & 0 & -d_2 & 0 \\ 0 & -d_3 & 0 & 0 & 0 & d_2 & 0 \\ 0 & \alpha & -d_4 & 0 & 0 & 0 & 0 \\ 0 & 0 & \xi & -\mu_h & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\mu_r & d_5 & 0 \\ 0 & 0 & 0 & 0 & 0 & -d_6 & 0 \\ 0 & 0 & 0 & 0 & 0 & \rho & -\mu_r \end{pmatrix} \tag{32}$$

The eigenvalues are

$$\lambda_1 = -d_6, \quad \lambda_2 = -d_4, \quad \lambda_3 = -d_3, \quad \lambda_4 = -\mu_r, \quad \lambda_5 = -\mu_h, \quad \lambda_6 = -\mu_h, \quad \lambda_7 = -\mu_r$$

Thus

$$\lambda_1 = -(\rho + \delta_r + \mu_r)$$

$$\lambda_2 = -(\xi_h + \mu_h)$$

$$\lambda_3 = -(\alpha + \delta_h + \mu_h)$$

$$\lambda_4 = -\mu_r \quad \text{twice}$$

$$\lambda_5 = -\mu_h \quad \text{twice}$$

RESULTS AND DISCUSSION

The Sitr-SIR Monkey Pox model was solved using the inbuilt classical Runge- Kutta method of Maple21 programming software. For the numerical simulation, the initial conditions with the values of the parameters for the model in table1 was used, some of these parameters were

sourced from existing literatures where available, and some were assumed for the purpose of illustrations to fit the model analysis where otherwise. We considered a duration of 6 years (t) starting from September 2017 to 19th June 2022 and we take the fixed time step as, h=0.1

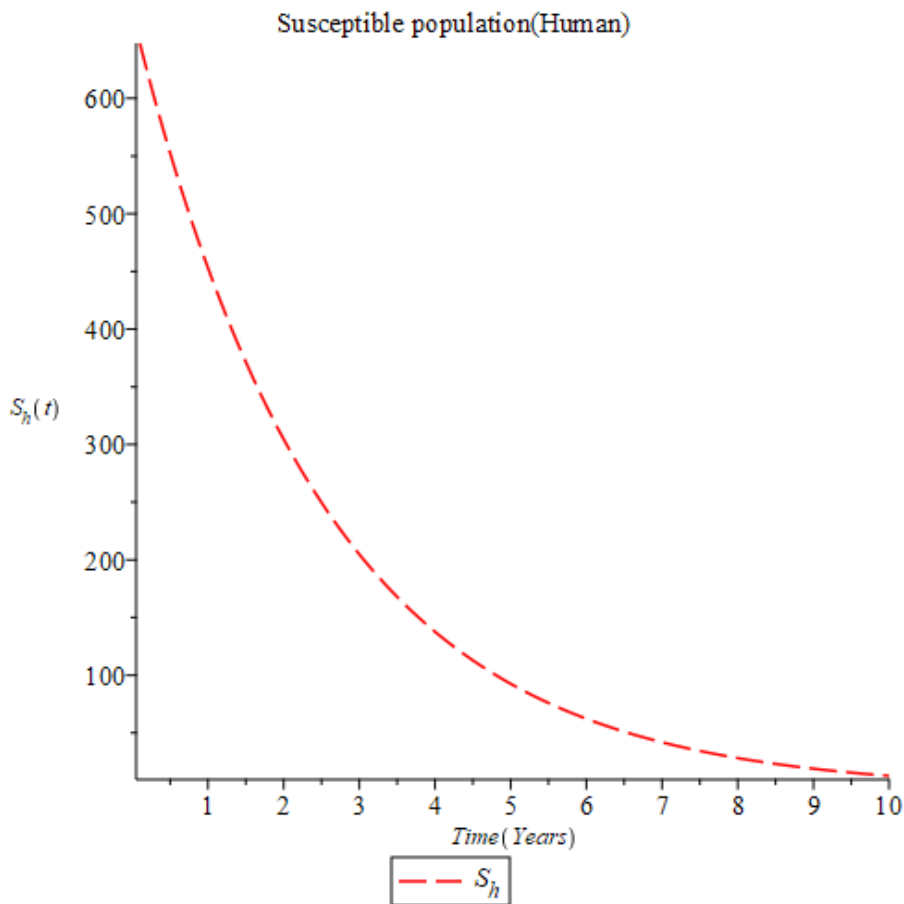


Figure. 1: Plot showing the solutions of the Susceptible Humans

Figure 1 shows that the susceptible human population decreasing exponentially, this decrease is due to the migration to the infected population.

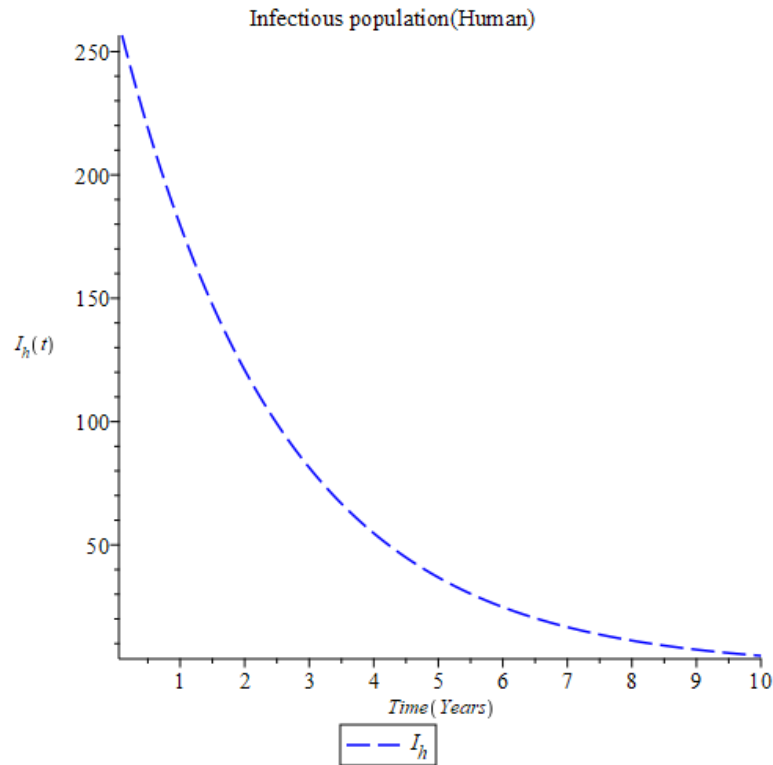


Figure 2: Plot showing the solutions of the Infected Humans

A decrease is observed in figure 2, as the infected human receive treatment they are being move to the recovery population.

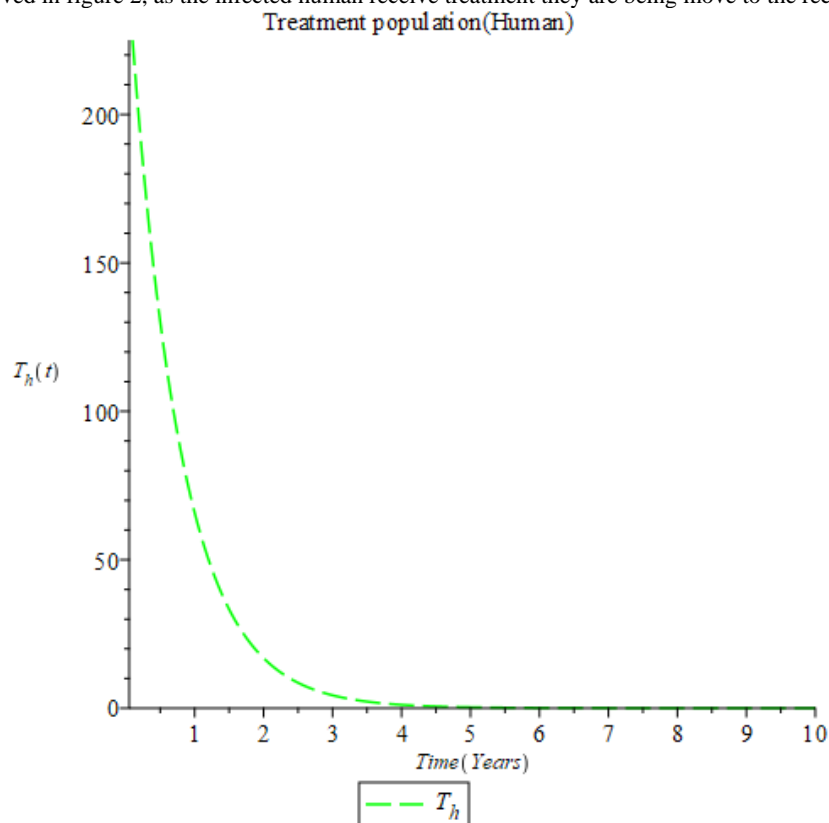


Figure 3: Plot showing the solutions of Humans who are treated

It is observed in figure 3 that the treated human population decreases exponentially. This decrease is due to the migration of treated humans to the recovery population. Also, the treated population suffers natural mortality.

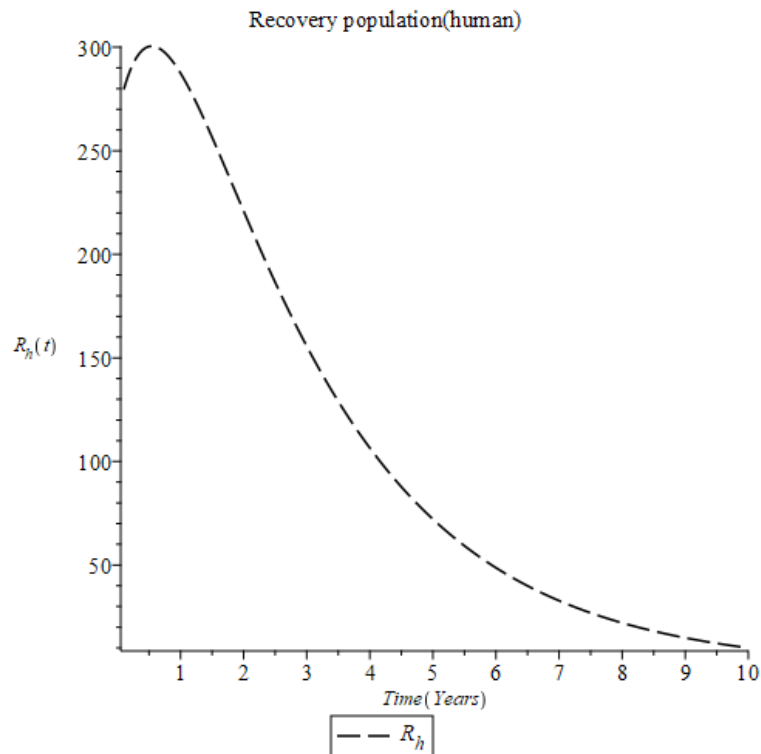


Figure 4: Plot showing the solutions of the Recovered Humans

Figure 4 shows that the recovery population grows exponentially up to equilibrium level, and which then started decreasing. This decrease is due to the treatment received by the infected humans. And this means that, when the infected human population approaches zero, the recovered class dies out exponentially, and besides, humans recover with permanent immunity and that recovered class also suffers natural mortality.

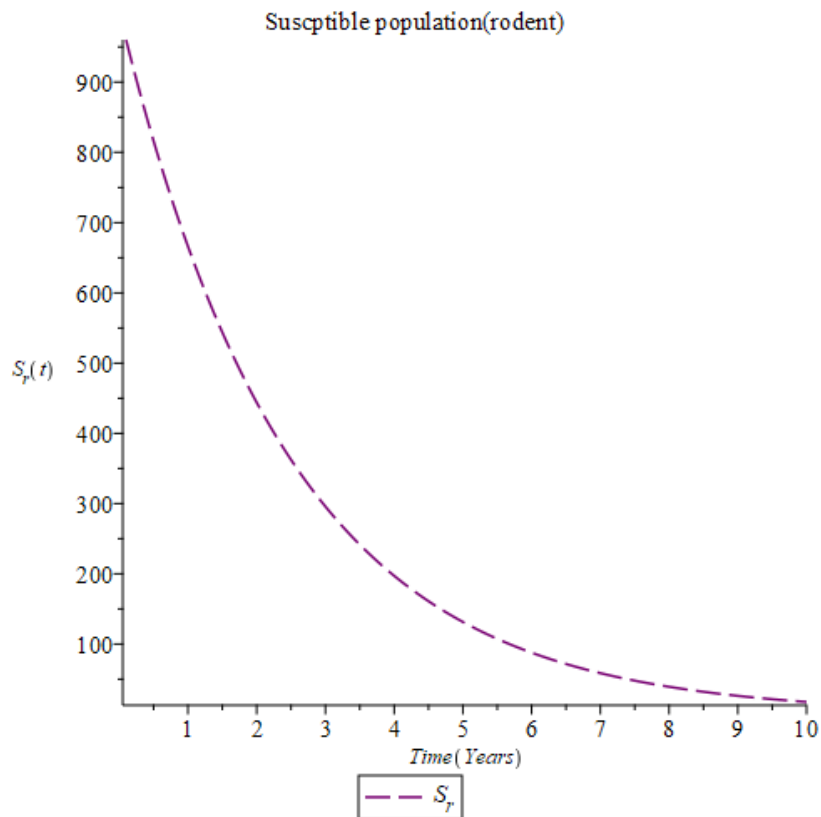


Figure 5: Plot showing the solutions of the Susceptible Rodents

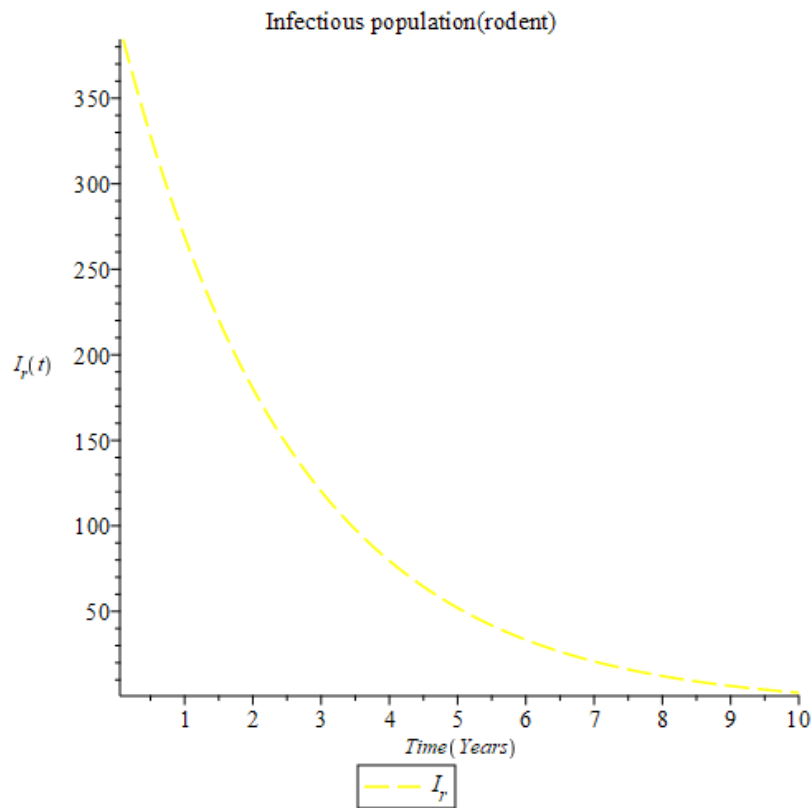


Figure 6: Plot showing the solutions of the Infected Rodents

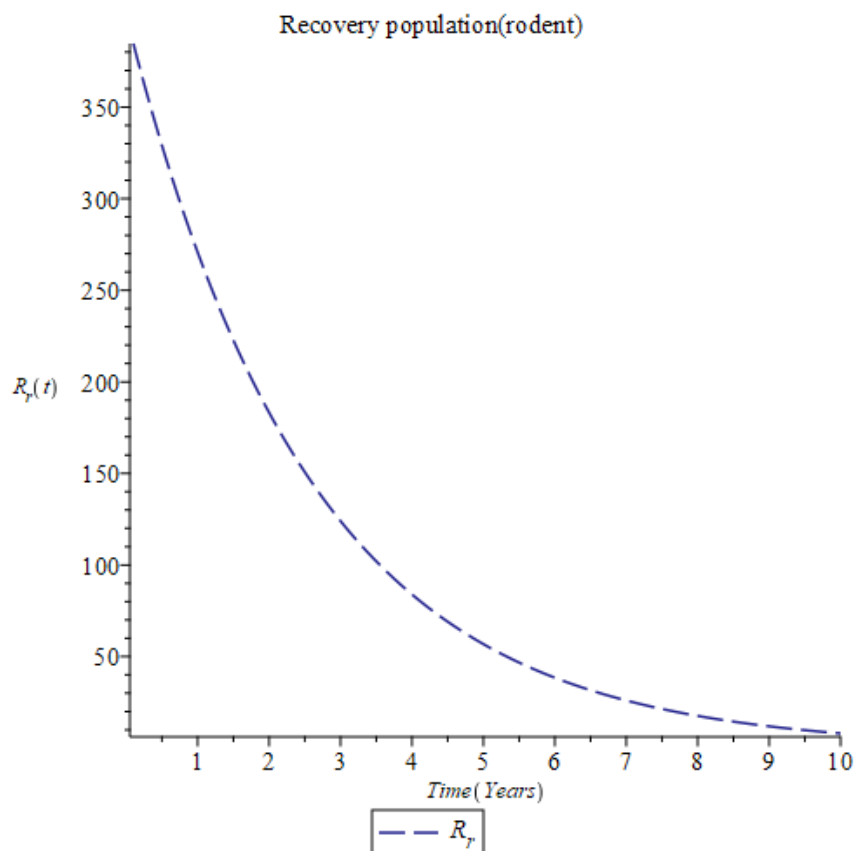


Figure 7: Plot showing the solutions of the Recovered Rodents

Figure 5-7 shows that the rodent susceptible, infected and recovery population decrease due to various migration to the next population. Also the rodent recovery population dies out exponentially in the absence of an infected rodent.

CONCLUSION

In this paper, we developed a mathematical model for monkey pox disease dynamics in Nigeria using the available information from the NCDC dataset. The model is formulated with the aid of a schematic diagram using appropriate parameters. The model analysis was carried out to show the feasible region, the disease-free equilibrium points, the basic reproduction number, and the local stability of the model. We proved that the disease-free equilibrium is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$. The model was solved to show the effect of the parameters using the Maple 21 programming software. The simulations results revealed that, the disease will be eradicated from both humans and the non-human primates with the developed model in due time.

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