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A MATHEMATICAL MODEL OF MONKEY POX VIRUS TRANSMISSION DYNAMICS

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In this paper a mathematical model of monkey pox virus transmission dynamics with two interacting host populations; humans and rodents is formulate. The quarantine class and public enlightenment campaign parameter are incorporated into human population as means of controlling the spread of the disease. The Disease Free Equilibrium (DFE) and Endemic Equilibrium (EE) are obtained. The basic reproduction number R_{0h} and R_{0r} are computed and used for the analysis. The Disease Free Equilibrium (DFE) is analyzed for stability using Jacobian matrix techniques and Lyapunov function. Stability analysis shows that the DFE is stable if $R_{0h}<1$ and $R_{0r}<1$.

INTRODUCTION

Monkey pox is caused by a rodent virus, which occurs mostly in West and Central Africa. The identification of monkey pox virus is based on biological characteristics and endonuclease patterns of viral DNA. In contrast to smallpox, monkey pox virus can infect rabbit skin and can be transmitted serially by intracerebral inoculation of mice. The four orthopox viruses that may infect man produce macroscopically characteristic lesions on the inoculated chorioallantoic membrane of an embryonated chicken egg, (Jezek and Fenner, 1988).Monkey pox was limited to the rain forests of central and western Africa until 2003, when the first cases in the Western Hemisphere were reported. In late spring 2003, multiple persons were identified in the Midwestern United States who had developed fever, rash, respiratory symptoms, and lymphadenopathy following exposure to ill pet prairie dogs (a rodent of Cynomys species) infected with the monkey pox virus, (Reed, et al. 2004).

The virus can spread from human to human by both respiratory (airborne) contact and contact with infected person's bodily fluids. Risk factors for transmission include sharing a bed, room, or using the same utensils with an infected patient. Increased transmission risk associated with factors involving introduction of virus to the oral mucosa, (Kantele, et al. 2016). Incubation period is 10–14 days. Prodromal symptoms include swelling of lymph nodes, muscle pain, headache, fever, prior to the emergence of the rash. The rash is usually only present on the trunk but has the capacity to spread to the palms and soles of the feet, occurring in a centrifugal distribution. The initial macular lesions exhibit a papular, then vesicular and pustular appearance, (Kantele, et al. 2016).

Monkey pox, is a rare zoonosis that occurs sporadically in forested areas of Central and West Africa, is an orthopox virus that can cause fatal illness. The disease manifestations are similar to human smallpox (eradicated since 1980), however human monkey pox is less severe. The disease is self-limiting with symptoms usually resolving within 14–21 days, (Marriott, et al. 2008). Treatment is supportive. This is the first outbreak in Nigeria since 1978. The virus is transmitted through direct contact with blood, bodily fluids and cutaneous/mucosal lesions of an infected animals (rats, squirrels, monkeys, dormice, striped mice, chimpanzees amongst others rodents) Secondary human-to-human transmission is limited but can occur via exposure to respiratory droplets, contact with infected persons or contaminated materials. The primary route of infection is thought to be contact with the infected animals or their bodily fluids, (Meyer, et al. 2002).

On 20 September 2017, WHO was notified of a

suspected outbreak of human monkey pox in Bayelsa State. Laboratory investigations have been conducted by the Nigeria Centre for Disease Control (NCDC) National Reference Laboratory, Institut Pasteur de Dakar and the WHO Collaborating Center for orthopox viruses, the United States Centers for Disease Control and Prevention (US CDC) in Atlanta, (WHO, 2017).

From 4 September, 2017 through 9 December 2017, 172 suspected and 61 confirmed cases were reported in different parts of Nigeria. Laboratory-confirmed cases were reported from fourteen states (out of 36 states)/territory: Akwa Ibom, Abia, Bayelsa, Benue, Cross River, Delta, Edo, Ekiti, Enugu, Lagos, Imo, Nasarawa, Rivers and Federal Capital Territory (FCT). Suspected cases were reported from 23 states/territories including: Abia, Adamawa, Akwa Ibom, Bayelsa, Benue, Cross River, Delta, Edo, Ekiti, Enugu, Federal Capital Territory (FCT), Imo, Kaduna, Kano, Katsina, Kogi, Kwara, Lagos, Ondo, Oyo, Nasarawa, Niger, and Rivers, (WHO, 2017).

The majority of cases are male (75%) and aged 21–40 years old (median age = 30 years old). One death has been reported in an immune-compromised patient not receiving anti-retroviral therapy. Clustering of cases has occurred within states, however there is no known evidence of epidemiological linkages across states. Further, genetic sequencing results of the virus isolated within and across states suggest multiple sources of introduction of the virus into the human population. Further epidemiological investigation is ongoing, (WHO, 2017).

In the absence of specific treatment or vaccine, the only way to reduce infection in people is by raising public awareness of the risk factors, such as close contact with wildlife animals including rodents, and educating people about the measures they can take to reduce exposure to the virus, (WHO, 2017).

Surveillance measures and rapid identification of new cases is critical for outbreak containment. Public health educational messages should focus on the following risks: Reducing the risk of animal-to-human transmission. Efforts to prevent transmission in endemic regions should focus on avoiding eating or touching animals that are sick of found dead in the wild. Gloves and other appropriate protective clothing should be worn while handling sick animals or their infected tissues. And reducing the risk of human-to-human transmission. People infected with monkey pox should be isolated and infection prevention and control measures should be implemented in healthcare facilities caring for infected patients. Close physical contact with persons infected with monkey pox should be avoided until they have recovered. Gloves, face masks and protective gowns should be worn when taking care of ill people in any setting. Regular hand washing should be carried out after caring for or visiting sick people, (WHO, 2017).

Bhunu and Mushayabasa (2011), developed a mathematical modeling of pox- like infection, in their model they considered SIR model for both human and rodent/wild animals.

This paper reviews the paper of Bhunu and Mushayabasa (2011), by incorporating quarantine class and an enlightenment campaign parameter into the human population to control the spread of the disease in the population. The Disease Free Equilibrium (DFE) was obtained and the basic reproduction number of the model is computed. The Jacobian matrix stability techniques and Lyapunov function was used to analyzed the local and global stability of the DFE.

MATERIALS AND METHODS

Model Formulation

The model considers two populations of; humans and rodents. The human population is sub-divided into four compartments; Susceptible S_h, Infected I_h, Quarantined Q_h and Recovered R_h. While the rodents population is sub divided into two compartments; Susceptible S_r and infected I_r.

The human population is recruited into Susceptible S_h at the constant recruitment rate Λ_h, the susceptible human become infected and move to Infected I_h class by contacting the infected rodents or infected humans at the contact rates α_1

and α_2 respectively. Infected humans I_h move to quarantine class at the rate τ, and the Quarantined Q_h move to Recovered R_h class after treatment at recovery rate γ_h. The individual leave the population either by natural death rate μ_h or by disease induced death rate δ_h. ε measures the effectiveness of enlightenment campaign, where $0 \leq \varepsilon \leq 1$ and θ is the effectiveness of quarantine and treatment where $0 \leq \theta \leq 1$. It is assumed that the death in Q_h due to disease is influenced by the effectiveness of treatment, hence it is $(1-\theta)\delta_h$.

Rodent population is recruited into the Susceptible S_r at the constant recruitment rate Λ_r the susceptible rodents become infected and move to Infected I_r class by contacting infected rodent at contacting rate α_3. The Infected rodents the rodents also leave the population either by natural death rate μ_r or by disease induced death rate δ_r. We also assumed that since the wild rodents may not have access to treatment, they do not recovered from the disease.

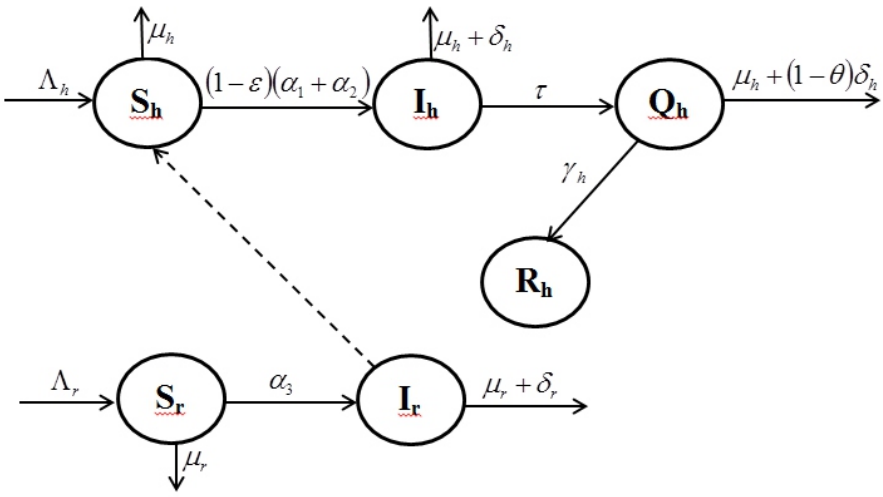

Figure 2.1: Schematic Diagram of the Model

Model Equations

$$\frac{dS_h}{dt} = \Lambda_h - (1-\varepsilon)\left(\frac{\alpha_1 I_r}{N_h} + \frac{\alpha_2 I_h}{N_h}\right)S_h - \mu_h S_h \quad (2.1)$$

$$\frac{dI_h}{dt} = (1-\varepsilon)\left(\frac{\alpha_1 I_r}{N_h} + \frac{\alpha_2 I_h}{N_h}\right)S_h - (\mu_h + \delta_h + \tau)I_h \quad (2.2)$$

$$\frac{dQ_h}{dt} = \tau I_h - [\mu_h + \gamma_h + (1-\theta)\delta_h]Q_h \quad (2.3)$$

$$\frac{dR_h}{dt} = \gamma_h Q_h - \mu_h R_h \quad (2.4)$$

$$\frac{dS_r}{dt} = \Lambda_r - \frac{\alpha_3 I_r S_r}{N_r} - \mu_r S_r \quad (2.5)$$

$$\frac{dI_r}{dt} = \frac{\alpha_3 I_r S_r}{N_r} - (\mu_r + \delta_r)I_r \quad (2.6)$$

$$\left.\begin{aligned} 0 \leq \varepsilon \leq 1 \\ 0 \leq \theta \leq 1 \end{aligned}\right\} \quad (2.7)$$

$$N_h = S_h + I_h + Q_h + R_h \quad (2.8)$$

$$N_r = S_r + I_r \quad (2.9)$$

Where

S_h = susceptible Humans
I_h = Infected Humans
Q_h = Quarantine Infected Humans
R_h = Recovered Humans
S_r = Susceptible Rodents
I_r = Infected Rodents
Λ_h = Recruitment Rate of Humans
Λ_r = Recruitment Rate of Rodents
α_1 = Contact Rate of Rodents to Humans
α_2 = Contact Rate of Humans to Humans
α_3 = Contact Rate of Rodents to Rodents
μ_h = Natural Death Rate of Humans
δ_h = Disease Induced Death Rate of Humans
γ_h = Recovery Rate of Humans
τ =Progression Rate from Infected to Quarantine
ε = Effectiveness Public Enlightenment Campaign

θ= Effectiveness of Quarantine and Treatment
μ_r= Natural Death Rate of Rodents
δ_r= Disease Induced Death Rate of Rodents

Equilibrium State of the Model
At equilibrium

$$\frac{dS_h}{dt}=\frac{dI_h}{dt}=\frac{dQ_h}{dt}=\frac{dR_h}{dt}=\frac{dS_r}{dt}=\frac{dI_r}{dt}=0 \quad (2.10)$$

Let

$$(S_h, I_h, Q_h, R_h, S_r, I_r)= (S_h^*, I_h^*, Q_h^*, R_h^*, S_r^*, I_r^*) \quad (2.11)$$

be arbitrarily equilibrium point
substituting (2.11) into (2.1) to (2.7) gives

$$\Lambda_h - A_1\left(\frac{\alpha_1 I_r^*}{N_h^*}+\frac{\alpha_2 I_h^*}{N_h^*}\right)S_h^* - \mu_h S_h^* = 0 \quad (2.12)$$

$$A_1\left(\frac{\alpha_1 I_r^*}{N_h^*}+\frac{\alpha_2 I_h^*}{N_h^*}\right)S_h^* - A_2 I_h^* = 0 \quad (2.13)$$

$$\tau I_h^* - A_3 Q_h^* \quad (2.14)$$

$$\gamma_h Q_h^* - \mu_h R_h^* \quad (2.15)$$

$$\Lambda_r - \frac{\alpha_3 I_r^* S_r^*}{N_r^*} - \mu_r S_r^* = 0 \quad (2.16)$$

$$\frac{\alpha_3 I_r^* S_r^*}{N_r^*} - A_4 I_r^* = 0 \quad (2.17)$$

Where,

$$A_1=(1-\varepsilon),\ A_2=(\mu_h+\delta_h+\tau),\ A_3=[\mu_h+\delta_h+(1-\theta)\gamma_h],\ A_4=(\mu_r+\delta_r) \quad (2.18)$$

From (2.17)

$$\left(\frac{\alpha_3 S_r^*}{N_r^*}-A_4\right)I_r^* = 0 \quad (2.19)$$

From (2.17)

$$I_r^* = 0 \quad (2.20)$$

or

$$\left(\frac{\alpha_3 S_r^*}{N_r^*}-A_4\right) = 0 \quad (2.21)$$

Substituting (2.21) into (2.13) gives

$$\left(\frac{A_1\alpha_2 S_h^*}{N_h^*}-A_2\right)I_h^* = 0 \quad (2.22)$$

From (2.23)

$$I_h^* = 0$$

or

$$\left(\frac{A_1\alpha_2 S_h^*}{N_h^*}-A_2\right) \quad (2.23)$$

Hence,

$$I_h^* = I_r^* = 0 \quad (2.24)$$

Disease Free Equilibrium (DFE)

Let $(S_h^*, I_h^*, Q_h^*, R_h^*, S_r^*, I_r^*)=$

$$(S_h^0, I_h^0, Q_h^0, R_h^0, S_r^0, I_r^0)= E^0 \quad (2.25)$$

Substituting (2.25) and (2.26) into (2.12) to (2.18) and solve gives

$$E^0=(S_h^0, I_h^0, Q_h^0, R_h^0, S_r^0, I_r^0)= \left(\frac{\Lambda_h}{\mu_h}, 0, 0, 0, \frac{\Lambda_r}{\mu_r}, 0\right) \quad (2.26)$$

at DFE

$$N_h^0 = \frac{\Lambda_h}{\mu_h} \quad (2.27)$$

and

$$N_r^0 = \frac{\Lambda_r}{\mu_r} \quad (2.28)$$

Basic Reproduction Number R_0
Using the Next Generation Matrix approach as in, Diekmann, et al., 1990 and Driessche et al., (2002). Basic Reproduction Number, is the largest eigenvalue or spectral radius of $1\text{-}FV$is the basic reproduction number of the model.

$$FV^{-1} = \left[\frac{\partial F_i(E^0)}{\partial x_i}\right]\left[\frac{\partial V_i(E^0)}{\partial x_i}\right]^{-1} \quad (2.29)$$

Where F_i is the rate of appearance of new infection in compartment i, V_i is the transfer of infections from one compartment i to another and E_0 is the disease-Free Equilibrium.

$$F=\begin{bmatrix} A_1\alpha_2 & A_1\alpha_1 \\ 0 & \alpha_3 \end{bmatrix} \quad (2.30)$$

$$V=\begin{bmatrix} A_2 & 0 \\ 0 & A_4 \end{bmatrix} \quad (2.31)$$

$$V^{-1}=\begin{bmatrix}\frac{1}{A_2} & 0\\ 0 & \frac{1}{A_4}\end{bmatrix} \tag{2.32}$$

$$FV^{-1}=\begin{bmatrix}\frac{A_1\alpha_2}{A_2} & \frac{A_1\alpha_1}{A_4}\\ 0 & \frac{\alpha_3}{A_4}\end{bmatrix} \tag{2.33}$$

$$\left|FV^{-1}-\lambda I\right|=0 \tag{2.34}$$

$$\begin{vmatrix}\frac{A_1\alpha_2}{A_2}-\lambda & \frac{A_1\alpha_1}{A_4}\\ 0 & \frac{\alpha_3}{A_4}-\lambda\end{vmatrix}=0 \tag{2.35}$$

The characteristic equation of (2.35) is given as

$$\left(\frac{A_1\alpha_2}{A_2}-\lambda\right)\left(\frac{\alpha_3}{A_4}-\lambda\right)=0 \tag{2.36}$$

Simplifying (2.36) gives

$$\lambda^2-\left(\frac{A_1\alpha_2}{A_2}+\frac{\alpha_3}{A_4}\right)\lambda+\frac{A_1\alpha_2\alpha_3}{A_2A_4}=0 \tag{2.37}$$

Solving (2.37) gives

$$\lambda=\frac{\left(\frac{A_1\alpha_2}{A_2}+\frac{\alpha_3}{A_4}\right)\pm\left(\frac{A_1\alpha_2}{A_2}-\frac{\alpha_3}{A_4}\right)}{2} \tag{2.38}$$

Therefore,

$$\lambda_1=\frac{A_1\alpha_2}{A_2}\text{ and }\lambda_2=\frac{\alpha_3}{A_4} \tag{2.39}$$

From (2.39) there exist two reproduction numbers since the transmission is between; rodents to humans and humans to humans. Hence,

$$R_{0h}=\frac{A_1\alpha_2}{A_2} \tag{2.40}$$

which is the basic reproduction number of humans to humans and

$$R_{0r}=\frac{\alpha_3}{A_4} \tag{2.41}$$

which is the basic reproduction number of rodents to rodents

Local Stability of Disease Free Equilibrium (DFE)

Theorem 2.1: The Disease Free Equilibrium of the model system (2.1) to (2.7) is locally asymptotically stable (LAS) if $R_{0h}<1$ and $R_{0r}<1$.

Proof: using Jacobian stability techniques, as in, (Somma, et al., 2015)

The Jacobian Matrix at DFE is given as:

$$J(E^0)=\begin{bmatrix}-\mu_h & -A_1\alpha_2 & 0 & 0 & 0 & -A_1\alpha_1\\ 0 & A_1\alpha_2-A_2 & 0 & 0 & 0 & A_1\alpha_1\\ 0 & \tau & -A_3 & 0 & 0 & 0\\ 0 & 0 & \gamma_h & -\mu_h & 0 & 0\\ 0 & 0 & 0 & 0 & -\mu_r & -\alpha_3\\ 0 & 0 & 0 & 0 & 0 & \alpha_3-A_4\end{bmatrix} \tag{2.42}$$

Reducing (2.42) to upper triangular matrix gives

$$J(E^0)=\begin{bmatrix}-\mu_h & -A_1\alpha_2 & 0 & 0 & 0 & -A_1\alpha_1\\ 0 & A_1\alpha_2-A_2 & 0 & 0 & 0 & A_1\alpha_1\\ 0 & 0 & -A_3 & 0 & 0 & \frac{-A_1\alpha_1\tau}{A_1\alpha_2-A_2}\\ 0 & 0 & 0 & -\mu_h & 0 & \frac{-A_1\alpha_1\gamma_h\tau}{A_3(A_1\alpha_2-A_2)}\\ 0 & 0 & 0 & 0 & -\mu_r & -\alpha_3\\ 0 & 0 & 0 & 0 & 0 & \alpha_3-A_4\end{bmatrix} \tag{2.43}$$

$$\left|J(E^0)-\lambda I\right|=0 \tag{2.44}$$

$$\begin{vmatrix}-\mu_h-\lambda & -A_1\alpha_2 & 0 & 0 & 0 & -A_1\alpha_1\\ 0 & A_1\alpha_2-A_2-\lambda & 0 & 0 & 0 & A_1\alpha_1\\ 0 & 0 & -A_3-\lambda & 0 & 0 & \frac{-A_1\alpha_1\tau}{A_1\alpha_2-A_2}\\ 0 & 0 & 0 & -\mu_h-\lambda & 0 & \frac{-A_1\alpha_1\gamma_h\tau}{A_3(A_1\alpha_2-A_2)}\\ 0 & 0 & 0 & 0 & -\mu_r-\lambda & -\alpha_3\\ 0 & 0 & 0 & 0 & 0 & \alpha_3-A_4-\lambda\end{vmatrix}=0 \tag{2.45}$$

The characteristic equation of (2.45) is given as

$$(-\mu_h-\lambda)(A_1\alpha_2-A_2-\lambda)(-A_3-\lambda)(-\mu_h-\lambda)(-\mu_r-\lambda)(\alpha_3-A_4-\lambda)=0 \tag{2.46}$$

$$\left.\begin{aligned}\lambda_1&=-\mu_h\\ \lambda_2&=A_1\alpha_2-A_2\\ \lambda_3&=-A_3\\ \lambda_4&=-\mu_h\\ \lambda_5&=-\mu_r\\ \lambda_6&=\alpha_3-A_4\end{aligned}\right\} \tag{2.47}$$

It is observe from (2.47) that all the eigen values λ_{is} are less than zero (i.e. $\lambda_{is}<0$) except λ_2 and λ_6.

For $\lambda_2<0$

It implies that

$$\left.\begin{aligned}&A_1\alpha_2-A_2<0\\ &\frac{A_1\alpha_2}{A_2}<1\end{aligned}\right\} \tag{2.48}$$

The Left Hand Side (LHS) of second inequality of (2.48) is the same as Right Hand Side of equation (2.41). Therefore,

$$R_{0h} < 1 \quad (2.49)$$

For $\lambda_6 < 0$

It implies that

$$\left.\begin{aligned} \alpha_3 - A_4 < 0 \\ \frac{\alpha_3}{A_4} < 1 \end{aligned}\right\} \quad (2.50)$$

The Left Hand Side (LHS) of second inequality of (2.50) is the same as Right Hand Side of equation (2.40). Therefore,

$$R_{0r} < 1 \quad (2.51)$$

Hence, the DFE is locally asymptotically stable.

This proof the theorem 2.1, equations (2.49) and (2.51) implies that the disease will not persist in the population.

Global Stability of Disease Free Equilibrium (DFE)

Theorem 2.2: The DFE, E_0 of the model system is globally asymptotically stable (GAS) if $R_{0h} < 1$ and $R_{0r} < 1$.

Proof:

In using the LaSalle's invariance principle as in, (Somma et al., 2017).

Consider the Lyapunov function

$$V(S_h, I_h, Q_h\ R_h, S_r,\ I_r) = A_4\alpha_3 I_h + A_2 I_r \quad (2.52)$$

Differentiating (2.52) with respect to time gives

$$\frac{dV}{dt} = A_4\alpha_3 \frac{dI_h}{dt} + A_2 \frac{dI_r}{dt} \quad (2.53)$$

$$\frac{dV}{dt} = A_4\alpha_3 \left[A_1 \left(\frac{\alpha_1 I_r}{N_h} + \frac{\alpha_2 I_h}{N_h} \right) S_h - A_2 I_h \right] + A_2 \left[\frac{\alpha_3 I_r S_r}{N_r} - A_4 I_r \right] \quad (2.54)$$

Since $S_h^0 \le N_h^0$ and $S_r^0 \le N_r^0$

Equation (2.54) becomes

$$\frac{dV}{dt} \le [\alpha_1\alpha_3 A_1 A_4 + \alpha_3 A_2 - A_2 A_4] I_r + [\alpha_2\alpha_3 A_1 A_4 - A_2 A_4\alpha_3] I_h \quad (2.55)$$

$$\frac{dV}{dt} \le A_2 A_4 \left[\left[\frac{A_1 A_4 \alpha_1 \alpha_3}{A_2 A_4} + \left(\frac{\alpha_3}{A_4} - 1 \right) \right] I_r + \alpha_3 \left(\frac{A_1 \alpha_2}{A_2} - 1 \right) I_h \right] \quad (2.56)$$

comparing (2.56) with (2.40) and (2.41) gives

$$\frac{dV}{dt} \le A_2 A_4 \left[\left[\frac{A_1 A_4 \alpha_1}{A_2} R_{0r} + (R_{0r} - 1) \right] I_r + \alpha_3 (R_{0h} - 1) I_h \right] \quad (2.57)$$

From (2.57)

$$\frac{dV}{dt} < 0 \text{ if } R_{0r} < 1 \text{ and } R_{0h} < 1 \text{ and } \frac{dV}{dt} = 0$$

or $I_h = I_r = 0$

Hence, the DFE is globally asymptotically stable.

RESULT AND DISCUSSION

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. Hence, the graphical simulation of basic reproduction number R_{0h} and some parameters of the model are show below in order to understand the effect of these parameters in the spread and control of the monkey pox virus. The parameters that were consider in this simulation are; Contact Rate of Humans with Humans, α_2, Natural Death Rate of Humans μ_h, Disease Induced Death Rate of Humans δ_h, Progression Rate from Infected to Quarantine τ and Public Enlightenment Campaign ε. The Contact Rate of Humans to Humans α_2, Progression Rate from Infected to Quarantine τ and Public Enlightenment Campaign ε were varied while other parameters remain constant. k is the different proportions of public enlightenment campaign $\varepsilon$and progression rate from Infected to Quarantine τ.

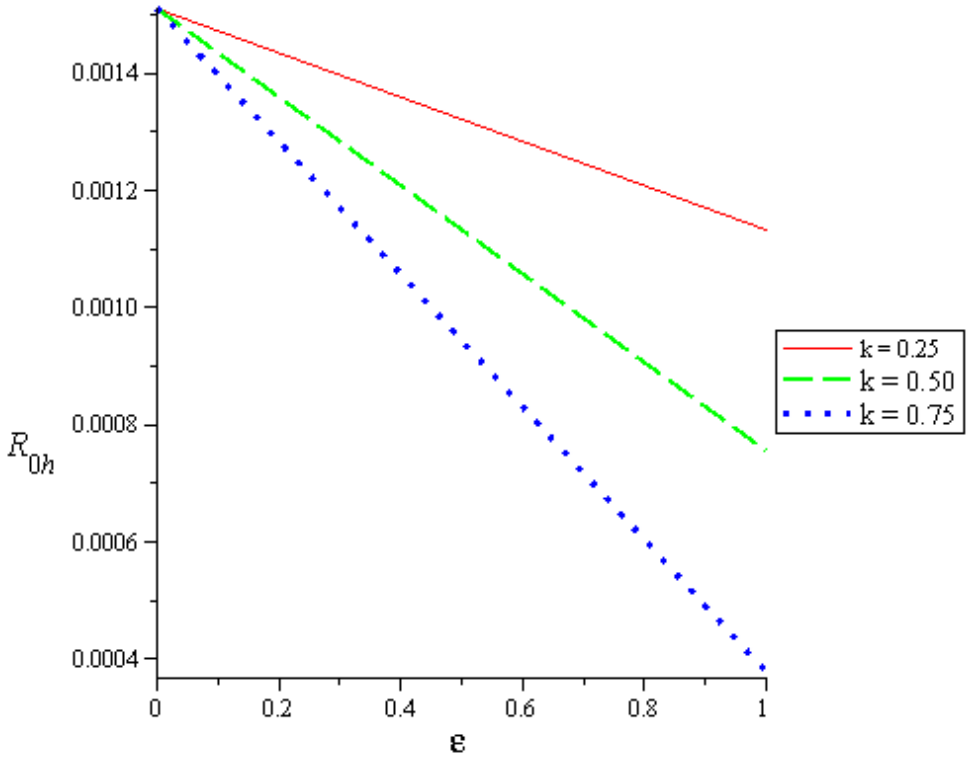

Figure 3.1: Simulation of Basic Reproduction Number of Humans R_{0h} against different Proportions of Public Enlightenment Campaigh ε with Low Contact Rate of Humans to Humans, α_2.

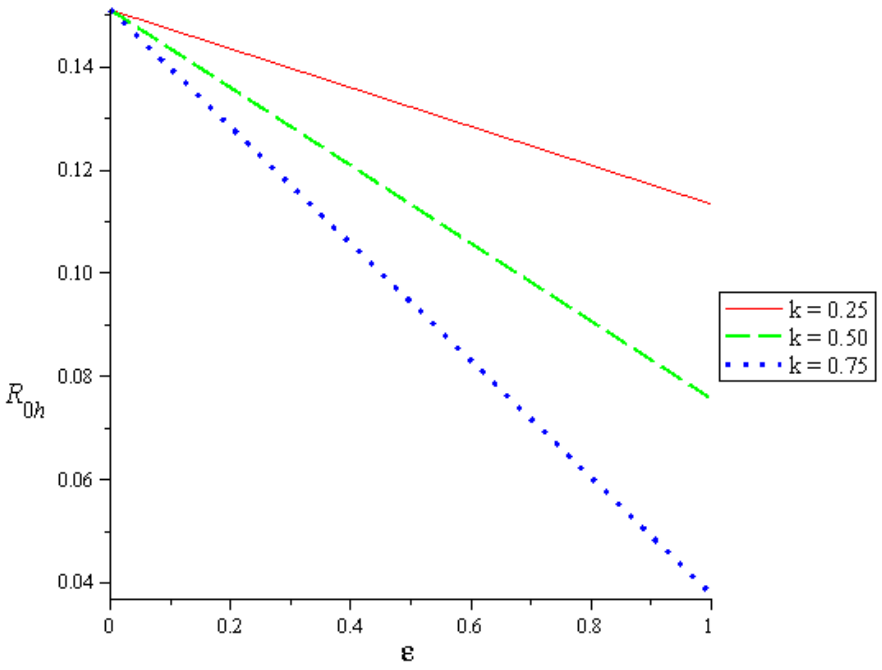

Figure 3.2: Simulation of Basic Reproduction Number of Humans R_{0h} against different Proportions of Public Enlightenment Campaigh ε with High Contact Rate of Humans to Humans, α_2.

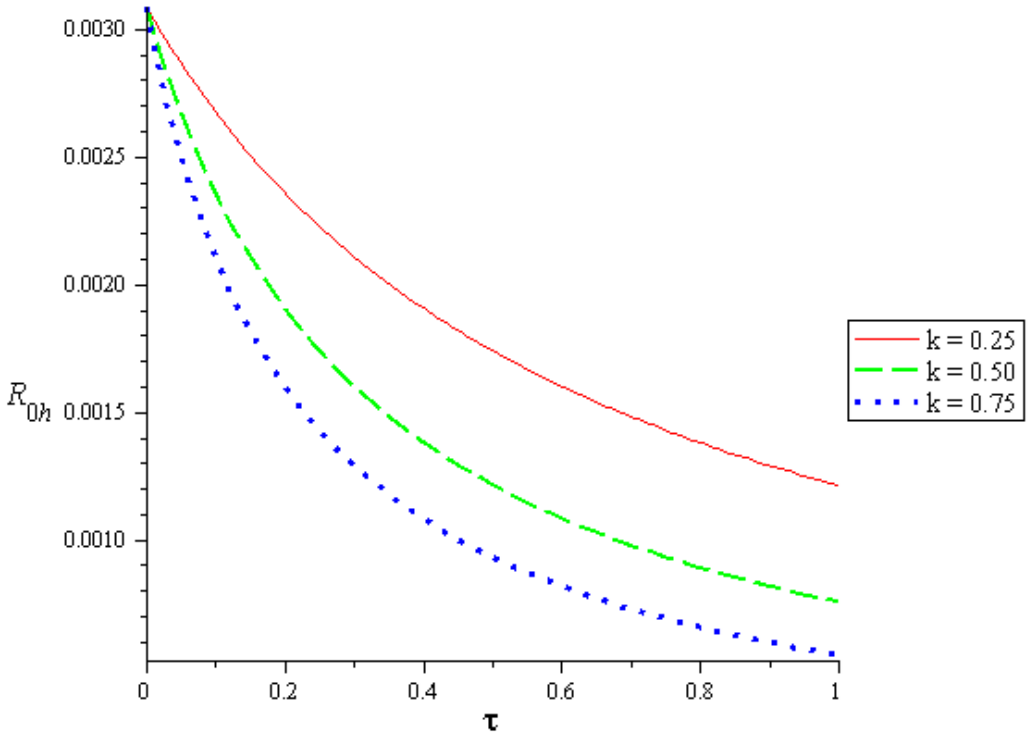

Figure 3.3: Simulation of Basic Reproduction Number of Humans R_{0h} and different Proportions of Progression Rate from Infected to Quarantine τ with low contact rate of Humans to Humans, α_2.

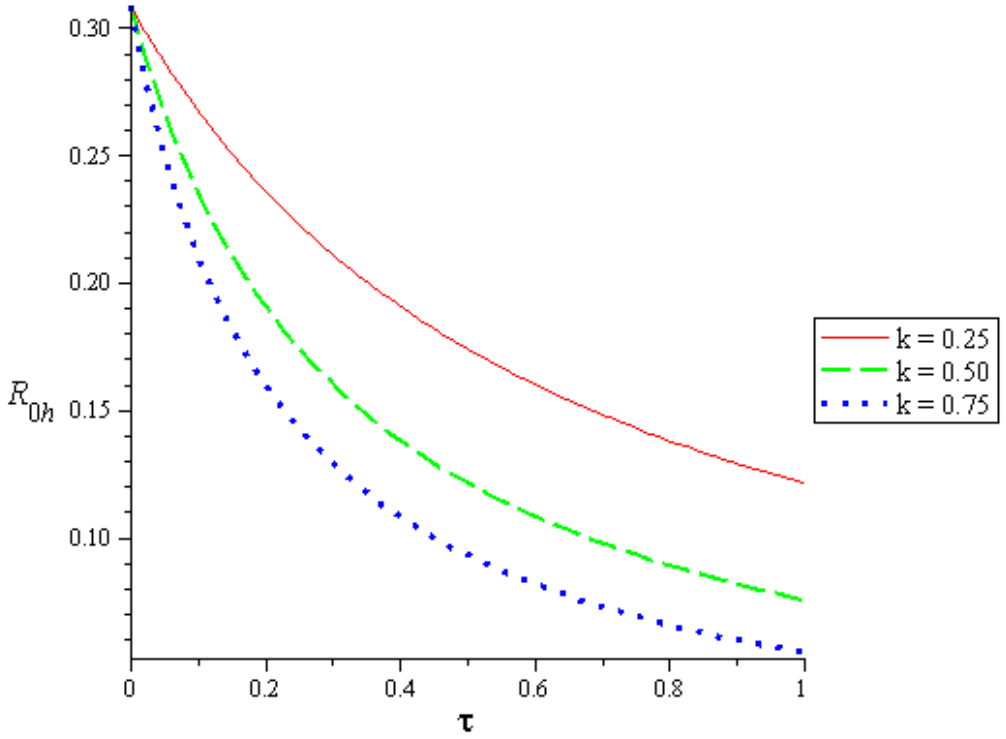

Figure 3.4: Simulation of Basic Reproduction Number of Humans R_{0h} and different Proportions of Progression Rate from Infected to Quarantine τ with High contact rate of Humans to Humans, α_2.

Figure 3.1 is the graph of basic reproduction number of humans R_{0h} and enlightenment campaign ε at low contact rate α_2. It shows that, the low contacting rate give rise to low basic reproduction number. It was also observe that the higher the enlightenment campaign ε the lower the basic reproduction number R_{0h}.

Figure 3.2 is the graph of basic reproduction number of humans R_{0h} and enlightenment campaign ε at high contact rate α_2. It shows that, the low contacting rate give rise to high basic reproduction number compare to that of low contacting rate. It was also observe that the higher the enlightenment campaign ε the lower the basic reproduction number R_{0h} despite the high contacting rate.

Figure 3.3 is the graph of basic reproduction number of humans R_{0h} and Progression Rate from Infected to Quarantine τ at low contact rate α_2. It reveals that, the low contacting rate give rise to low basic reproduction number. It was also observe that the higher the progression rate from Infected to Quarantine τ the lower the basic reproduction number R_{0h}.

Figure 3.4 is the graph of basic reproduction number of humans R_{0h} and Progression Rate from Infected to Quarantine τ at high contact rate α_2. It shows that, the low contacting rate give rise to high basic reproduction number compare to that of low contacting rate. It was also observe that the higher the progression rate from Infected to Quarantine τ the lower the basic reproduction number R_{0h} despite the high contacting rate.

CONCLUSION

The model of monkey pox virus incorporating quarantine class and public enlightenment campaign parameter to control the spread of the disease was developed using first order ordinary differential equations. Two equilibrium states exist in the model; Disease Free Equilibrium (DFE) and Endemic Equilibrium (EE). There are also two reproduction numbers in the model; rodent to human transmission reproduction number and human to human reproduction number.

The stability analysis of the DFE shows that, the DFE is locally and globally stable if $R_{0r}<1$ and $R_{0h}<1$ which implies that the disease will not persist in the population.

The graphs also show that, the low and high contact rate give rise to low and high reproduction number R_{0h}. It was also revealed that, the higher the public enlightenment campaign and progression rate from Infected to Quarantine the lower the basic reproduction number R_{0h}. This implies that public enlightenment campaign and isolation of infected people from susceptible people will go a long way to reduce the spread of monkey pox in the population.

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