



# SENSITIVITY ANALYSIS FOR THE MATHEMATICAL MODELLING OF MONKEY POX VIRUS INCORPORATING QUARANTINE AND PUBLIC ENLIGHTENMENT CAMPAIGN

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## ABSTRACT

In this paper sensitivity analysis was carried out for the mathematical modeling of Monkey pox virus incorporating quarantine and public enlightenment campaign into the human population. The model was formulated using first order ordinary differential equations. The model equation was divided into two populations of human and rodents. There are two equilibrium points that exist in the model; Disease Free Equilibrium (DFE) and Endemic Equilibrium (EE). The Local and Global stabilities of Disease Free Equilibrium (DFE) were analyzed. The basic reproduction numbers of human to human  $R_{0h}$  and rodent to rodent  $R_{0r}$  transmission was computed. The sensitivity analysis of the Basic reproduction number with the parameters was carried out. The Disease Free Equilibrium (DFE) is Locally and Globally Asymptotically Stable if  $R_{0h} < 1$  and  $R_{0r} < 1$ . The graphical presentation of the Basic reproduction number and the sensitive parameters shows that effective progression rate from infected class to Quarantine and effective public enlightenment campaign are the most sensitive parameters that will eradicate the disease from the population.

**Keywords:** *sensitivity analysis; monkey pox; equilibrium; reproduction number; public enlightenment campaign; quarantine.*

## INTRODUCTION

Monkeypox is caused by a rodent virus, which occurs mostly in West and Central Africa. Monkeypox virus can be identified through biological characteristics and endonuclease patterns of viral DNA. The monkeypox virus can infect rabbit skin and can be transmitted serially by intracerebral inoculation of mice (Jezek and Fenner, 1988). Monkeypox was first found in the rain forests of central and western Africa until 2003, that the first cases was reported in the Western Hemisphere. In 2003 also several people were identified in the Midwestern United States who had developed fever, rash, respiratory symptoms, and lymphadenopathy due to their exposure to ill pet prairie dogs infected with the monkeypox virus, (Reed, *et al.*, 2004).

The two major ways monkeypox virus spread from human to human is by respiratory (airborne) contact and contact with infected person's bodily fluids. The other ways of transmission include sharing a bed, room, or using the same utensils with an infected patient (Kantele, *et al.*, 2016). Incubation period is 10–14 days. The symptoms at the early stage of monkeypox include swelling of lymph nodes, muscle pain, headache, fever, prior to the emergence of the rash. The initial macular lesions exhibit a papular, then vesicular and pustular appearance, (Kantele, *et al.*, 2016).

Monkeypox, is a uncommon zoonosis that can cause fatal illness. The manifestations of the disease are similar to human smallpox, but human monkeypox is less severe (Marriott, *et al.*, 2008). The first outbreak occurred in Nigeria in 1978. The 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).

The suspected outbreak of human monkeypox in Bayelsa State was reported to WHO on 20 September 2017. Relevant agencies such as 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 carried out laboratory investigations (WHO, 2017).

About 172 suspected and 61 confirmed cases were reported in different parts of Nigeria from 4 September, 2017 through 9 December 2017. 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 only preventive measure of monkeypox virus transmission 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).

Investigation measures and rapid identification of new cases is critical for outbreak containment. Public health educational should focus on the risk of animal-to-human transmission. The preventive measure in endemic regions should focus on avoiding eating or touching animals that are sick or found dead in the bush. In reducing the risk of human-to-human transmission, infected people should be isolated from the population (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 research 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  $\Lambda_h$ , the susceptible human become infected and move to Infected  $I_h$  class by contacting the infected rodents or infected humans at the contact rates  $\alpha_1$  and  $\alpha_2$  respectively. Infected humans  $I_h$  move to quarantine class at the rate  $\tau$ , and the Quarantined  $Q_h$  move to Recovered  $R_h$  class after treatment at recovery rate  $\gamma_h$ . The individual leave the population either by natural death rate  $\mu_h$  or by disease induced death rate  $\delta_h$ .  $\varepsilon$  measures the effectiveness of enlightenment campaign, where  $0 \leq \varepsilon \leq 1$  and  $\theta$  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  $\Lambda_r$  the susceptible rodents become infected and move to Infected  $I_r$  class by contacting infected rodent at contacting rate  $\alpha_3$ . The Infected rodents  $I_r$  the rodents also leave the population either by natural death rate  $\mu_r$  or by disease induced death rate  $\delta_r$ . We also assumed that since the wild rodents may not have access to treatment, they do not recovered from the disease.

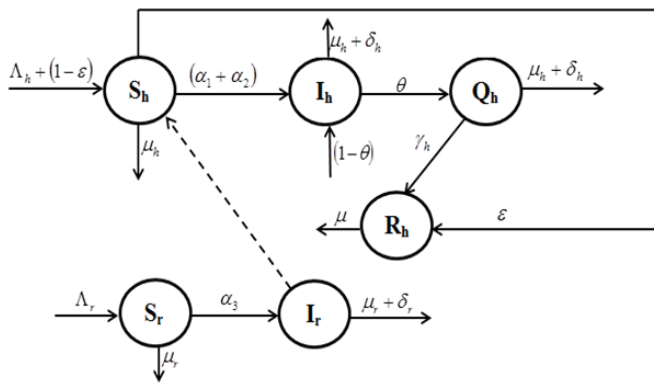


Figure 1: Schematic Diagram of the Model

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

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

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

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

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

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

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

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

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

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

$\Lambda_h$  = Recruitment Rate of Humans

$\Lambda_r$  = Recruitment Rate of Rodents

$\alpha_1$  = Contact Rate of Rodents to Humans

$\alpha_2$  = Contact Rate of Humans to Humans

$\alpha_3$  = Contact Rate of Rodents to Rodents

$\mu_h$  = Natural Death Rate of Humans

$\delta_h$  = Disease Induced Death Rate of Humans

$\gamma_h$  = Recovery Rate of Humans

$\varepsilon$  = Effectiveness Public Enlightenment Campaign

$\theta$  = Effectiveness of Quarantine and Treatment

$\mu_r$  = Natural Death Rate of Rodents

$\delta_r$  = Disease Induced Death Rate of Rodents

**Equilibrium State of the Model**

At equilibrium

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

Let

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

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

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

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

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

$$\gamma_h Q_h^* + \varepsilon S_h^* - \mu_h R_h^* \quad \dots(2.15)$$

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

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

Where,  $A_1 = (\mu_h - (1 - \varepsilon))$ ,  $A_2 = (\mu_h + \delta_h - (1 - \theta))$ ,  $A_3 = (\mu_h + \delta_h + \gamma_h)$ ,  $A_4 = (\mu_r + \delta_r)$  .....(2.18)

From (2.17)

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

From (2.17)

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

or

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

Substituting (2.21) into (2.13) gives

$$\left( \frac{\alpha_2 S_h'}{N_h'} - A_2 \right) I_h' = 0 \quad \dots\dots(2.22)$$

From (2.23)

$I_h^* = 0$   
or

$$\left( \frac{\alpha_2 S_h'}{N_h'} - A_2 \right) \dots\dots(2.23)$$

Hence,

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

Disease Free Equilibrium (DFE)

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

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

$$E^0 = (S_h^0, I_h^0, Q_h^0, R_h^0, S_r^0, I_r^0) = \left( \frac{\Lambda_h}{A_1}, 0, 0, \frac{\Lambda_h \varepsilon}{A_1 \mu_h}, \frac{\Lambda_r}{\mu_r}, 0 \right) \dots\dots(2.26)$$

At DFE

$$N_h^0 = \frac{\Lambda_h A_2}{A_1 \mu_h} \quad \dots\dots(2.27)$$

and

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

Where,  $A_5 = (\mu_h + \varepsilon)$

**Basic Reproduction Number**

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  $FV^{-1}$  is the basic reproduction number of the model.

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

Where  $F_i$  is the rate of appearance of new infection in compartment  $i$ ,  $V_i$  is the transfer of infections

from one compartment to another and is the disease-free Equilibrium.

$$F = \begin{bmatrix} \frac{\alpha_2 \mu_h}{A_2} & \frac{\alpha_1 \mu_h}{A_3} \\ 0 & \alpha_3 \end{bmatrix} \quad \dots\dots(2.30)$$

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

$$V^{-1} = \begin{bmatrix} \frac{1}{A_2} & 0 \\ 0 & \frac{1}{A_4} \end{bmatrix} \quad \dots\dots(2.32)$$

$$FV^{-1} = \begin{bmatrix} \frac{\alpha_2 \mu_h}{A_2 A_2} & \frac{\alpha_1 \mu_h}{A_4 A_3} \\ 0 & \frac{\alpha_3}{A_4} \end{bmatrix} \quad \dots\dots(2.33)$$

$$|FV^{-1} - \lambda I| = 0 \quad \dots\dots(2.34)$$

$$\begin{vmatrix} \frac{\alpha_2 \mu_h}{A_2 A_2} - \lambda & \frac{\alpha_1 \mu_h}{A_4 A_3} \\ 0 & \frac{\alpha_3}{A_4} - \lambda \end{vmatrix} = 0 \quad \dots\dots(2.35)$$

The characteristic equation of (2.35) is given as

$$\left( \frac{\alpha_2 \mu_h}{A_2 A_2} - \lambda \right) \left( \frac{\alpha_3}{A_4} - \lambda \right) = 0 \quad \dots\dots(2.36)$$

Simplifying (2.36) gives

$$\lambda^2 - \left( \frac{\alpha_2 \mu_h}{A_2 A_2} + \frac{\alpha_3}{A_4} \right) \lambda + \frac{\alpha_2 \alpha_3 \mu_h}{A_2 A_4 A_2} = 0 \quad \dots\dots(2.37)$$

Solving (2.37) gives

$$\lambda = \frac{\left( \frac{\alpha_2 \mu_h}{A_2 A_2} + \frac{\alpha_3}{A_4} \right) \pm \sqrt{\left( \frac{\alpha_2 \mu_h}{A_2 A_2} + \frac{\alpha_3}{A_4} \right)^2}}{2} \dots\dots(2.38)$$

Therefore,

$$\lambda_1 = \frac{\alpha_2 \mu_h}{A_2 A_2} \text{ and } \lambda_2 = \frac{\alpha_3}{A_4} \quad \dots\dots(2.39)$$

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

$$R_{0h} = \frac{\alpha_2 \mu_h}{A_2 A_2} \quad \dots\dots(2.40)$$

which is the basic reproduction number of humans to humans and

$$R_{0r} = \frac{\alpha_3}{A_4} \quad \dots\dots(2.41)$$

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:

$$J(E^0) = \begin{bmatrix} -A_1 & -\frac{\alpha_2 \mu_h}{A_5} & 0 & 0 & 0 & -\frac{\alpha_1 \mu_h}{A_5} \\ 0 & \frac{\alpha_2 \mu_h - A_2 A_5}{A_5} & 0 & 0 & 0 & \frac{\alpha_1 \mu_h}{A_5} \\ 0 & \theta & -A_3 & 0 & 0 & 0 \\ 0 & 0 & \gamma_h & -\mu_h & 0 & 0 \\ 0 & 0 & 0 & 0 & -\mu_r & -\alpha_3 \\ 0 & 0 & 0 & 0 & 0 & \alpha_3 - A_4 \end{bmatrix} \tag{2.42}$$

Reducing (2.42) to upper triangular matrix gives

$$J(E^0) = \begin{bmatrix} -A_1 & -\frac{\alpha_2 \mu_h}{A_5} & 0 & 0 & 0 & -\frac{\alpha_1 \mu_h}{A_5} \\ 0 & \frac{\alpha_2 \mu_h - A_2 A_5}{A_5} & 0 & 0 & 0 & \frac{\alpha_1 \mu_h}{A_5} \\ 0 & 0 & -A_3 & 0 & 0 & \frac{-\alpha_1 \mu_h \theta}{\alpha_2 \mu_h - A_2 A_5} \\ 0 & 0 & 0 & -\mu_h & 0 & \frac{A_2 A_3 \alpha_1 \varepsilon \mu_h - A_1 \alpha_1 \gamma_h \theta \mu_h}{A_1 A_3 (\alpha_2 \mu_h - A_2 A_5)} \\ 0 & 0 & 0 & 0 & -\mu_r & -\alpha_3 \\ 0 & 0 & 0 & 0 & 0 & \alpha_3 - A_4 \end{bmatrix} \tag{2.43}$$

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

$$\begin{vmatrix} -A_1 - \lambda & -\frac{\alpha_2 \mu_h}{A_5} & 0 & 0 & 0 & -\frac{\alpha_1 \mu_h}{A_5} \\ 0 & \frac{\alpha_2 \mu_h - A_2 A_5}{A_5} - \lambda & 0 & 0 & 0 & \frac{\alpha_1 \mu_h}{A_5} \\ 0 & 0 & -A_3 - \lambda & 0 & 0 & \frac{-\alpha_1 \mu_h \theta}{\alpha_2 \mu_h - A_2 A_5} \\ 0 & 0 & 0 & -\mu_h - \lambda & 0 & \frac{A_2 A_3 \alpha_1 \varepsilon \mu_h - A_1 \alpha_1 \gamma_h \theta \mu_h}{A_1 A_3 (\alpha_2 \mu_h - A_2 A_5)} \\ 0 & 0 & 0 & 0 & -\mu_r - \lambda & -\alpha_3 \\ 0 & 0 & 0 & 0 & 0 & \alpha_3 - A_4 - \lambda \end{vmatrix} = 0 \tag{2.45}$$

The characteristic equation of (2.45) is given as

$$(-A_1 - \lambda) \left( \frac{\alpha_2 \mu_h - A_2 A_5}{A_5} - \lambda \right) (-A_3 - \lambda) (-\mu_h - \lambda) (-\mu_r - \lambda) (\alpha_3 - A_4 - \lambda) = 0 \tag{2.46}$$

From (2.46)

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

It is observe from (2.47) that all the eigenvalues  $\lambda_{i_s}$  are less than zero (i.e.  $\lambda_{i_s} < 0$  ) except  $\lambda_2$  and  $\lambda_6$  .

For  $\lambda_2 < 0$  It implies that

$$\left. \begin{aligned} \frac{\alpha_2 \mu_h - A_2 A_3}{A_3} &< 0 \\ \frac{\alpha_2 \mu_h}{A_2 A_3} &< 1 \end{aligned} \right\} \dots\dots\dots(2.48)$$

The Left Hand Side (LHS) of second equation of (2.48) is the same as Right Hand Side of equation (2.40). Therefore,

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

For  $\lambda_6 < 0$

It implies that

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

The Left Hand Side (LHS) of second inequality of (2.50) is the same as Right Hand Side of equation (2.41). 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 .

$$R_{0r} < 1 \text{ and } R_{0h} < 1$$

Proof:

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

Consider the Lyapunov function

$$L(S_h, I_h, Q_h, R_h, S_r, I_r) = A_4 \alpha_2 I_h + A_2 I_r \dots\dots(2.52)$$

Differentiating (2.52) with respect to time gives

$$\frac{dL}{dt} = A_4 \alpha_2 \frac{dI_h}{dt} + A_2 \frac{dI_r}{dt} \dots\dots\dots(2.53)$$

$$\begin{aligned} \frac{dL}{dt} &= A_4 \alpha_2 \left[ \left( \frac{\alpha_1 I_r + \alpha_2 I_h}{N_h} \right) S_h - A_2 I_h \right] \\ &\quad + A_2 \left[ \frac{\alpha_3 I_r S_r}{N_r} - A_4 I_r \right] \dots\dots\dots(2.54) \end{aligned}$$

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

Equation (2.54) becomes

$$\begin{aligned} \frac{dV}{dt} &\leq \left[ \frac{A_4 \alpha_2^2 \mu_h}{A_3} - A_2 A_4 \alpha_2 \right] I_h \\ &\quad + \left[ \frac{A_4 \alpha_1 \alpha_2 \mu_h}{A_3} + A_2 \alpha_3 - A_2 A_4 \right] I_r \dots\dots\dots(2.55) \end{aligned}$$

Divide (2.55) by  $A_2 A_4$  gives

$$\frac{dV}{dt} \leq A_2 A_4 \left[ \begin{aligned} &\alpha_2 \left( \frac{\alpha_2 \mu_h}{A_2 A_3} - 1 \right) I_h \\ &+ \left( \frac{\alpha_1 \alpha_2 \mu_h}{A_2 A_3} + \frac{\alpha_3}{A_4} - 1 \right) I_r \end{aligned} \right] \dots\dots\dots(2.56)$$

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

$$\frac{dV}{dt} \leq A_2 A_4 \left[ \alpha_2 (R_{0h} - 1) I_h + (\alpha_1 R_{0h} + (R_{0r} - 1)) I_r \right] \dots\dots\dots(2.57)$$

From (2.57)

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

$$\frac{dV}{dt} = 0 \text{ if } I_h = I_r = 0$$

Hence, the DFE is globally asymptotically stable.

**RESULTS AND DISCUSSION**

**Sensitivity Analysis of the Basic Reproduction Number, with Some Parameter of the Model**

Sensitivity indices allow us to measure the relative change in a variable when a parameter changes. The normalized forward sensitivity index of a variable with respect to a parameter is the ratio of the relative change in the variable to the relative change in the parameter. When the variable is a differentiable function of the parameter, the sensitivity index may be alternatively defined using partial derivatives.

In determining how best to reduce human mortality and morbidity due to measles, the sensitivity indices of the basic reproduction number to the parameters of the model was calculated following

similar approaches as in Arriola and Hyman (2005), Chitnis *et al.* (2008), Mikuchi *et al.* (2012) and Abdulrahman *et al.* (2013). The normalized forward sensitivity indices with respect to a parameter value,

$$S_P^{R_0} = \frac{\partial R_0}{\partial P} \times \frac{P}{R_0} \quad \dots\dots(4.1)$$

Where,

$$P = \{\alpha_2, \varepsilon, \theta\} \quad \dots\dots(4.2)$$

The sensitivity indices of the parameters of the basic reproduction number  $R_0$  were calculated using Maple 13 software. The sensitivity analysis was carried out using three different proportion of the parameters; low = 0.25, moderate = 0.50 and high = 0.75. The sensitivity indices are shown in table 1 below.

Table 1: Sensitivity Indices of To Parameters of the Model, Evaluated at The Different Proportion of the Parameters

Parameters	Low	Moderate	High
Contact Rate of Human to Human Transmission $\alpha_2$	1.0000	1.0000	1.0000
Effectiveness of Public Enlightenment Campaign $\varepsilon$	-0.9960	-0.9980	-0.9987
Effectiveness of Quarantine and Treatment $\theta$	-0.2002	-0.3336	-0.4288

Table 4.1 shows that all the parameters have either positive or negative effects on the basic reproduction number,  $R_{0h}$ . The positive parameter will increase the basic reproduction number while the negative parameters will decrease the basic reproduction number. The Contact Rate of Human to Human Transmission,  $\alpha_2$  has the highest sensitivity index follow by Effectiveness of Public Enlightenment Campaign, and Effectiveness of Quarantine and Treatment, has the lowest sensitivity indices.

**Graphical Representation of Basic Reproduction Number with Sensitive Parameter**

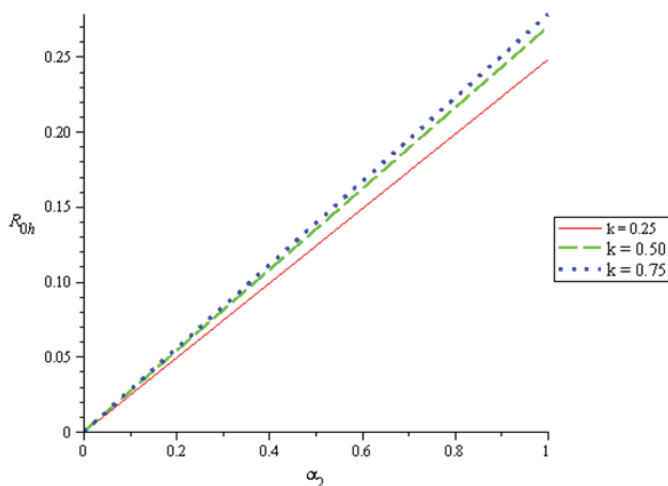


Figure 2: Graph of Basic Reproduction Number of Human to Human Transmission against different proportions of Contact Rate of Human to Human

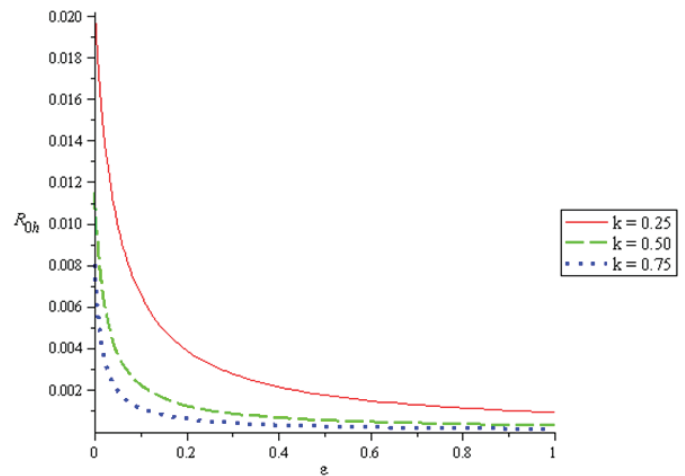


Figure 3: Graph of Basic Reproduction Number of Human to Human Transmission against different proportions of Effectiveness of Public Enlightenment Campaign

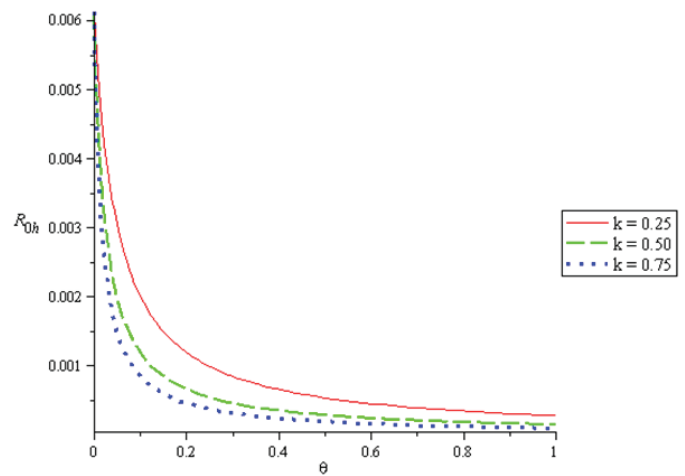


Figure 4: Graph of Basic Reproduction Number of Human to Human Transmission against different proportions of Effectiveness of Quarantine and Treatment.

The Disease Free Equilibrium (DFE) is local and global asymptotically stable if  $R_{0r} < 1$  and  $R_{0h} < 1$  which implies that the disease will not persist in the population.

Figure 2 shows that as contact rate of human to human increases the basic reproduction number of human to human transmission increases. This implies that the contact between the infected and susceptible individuals in the population will make the disease endemic.

It is observed from figure 3 that as

effectiveness of public enlightenment campaign increases the basic reproduction number of human to human transmission decreases. This shows that the more people are enlightened about the risk of monkey pox the less the transmission and this will result to the eradication of the disease in the population.

Figure 4 reveals that, as effectiveness of quarantine and treatment increases the Basic reproduction number of human to human transmission decreases. The effort to quarantine the infected people from the entire population and treat them will also help to eradicate the monkey pox.

## CONCLUSION

The model of monkey pox virus incorporating quarantine class and public enlightenment campaign reproduction number . 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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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 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 . It was also revealed that, the higher the public enlightenment campaign and progression rate from Infected to Quarantine the lower the basic reproduction number .