



## EFFECTS OF PUBLIC AWARENESS AND VACCINATION IN MODELING THE DYNAMICS OF MONKEY-POX TRANSMISSION DISEASE IN NIGERIA

## Abdullahi M. Rashad, \*Nurudeen O. Lasisi and Fahad Suleiman

Department of Statistics, Federal Polytechnic, Kaura Namoda, Nigeria

\*Corresponding authors' email: <u>nurudeenlasisi2009@yahoo.com</u>

## ABSTRACT

Monkey-pox disease is recognized as pathogens, disturbing animals and humans, it is among the family of orthopox virus and the disease causes lymph nodes to swell. In this paper, we developed a deterministic model system for Monkey pox infection by incorporating public awareness parameter and vaccination individual. The study verified the feasible region of the system equations and non-negativity of the solutions is achieved. The disease free and endemic equilibrium states have been obtained. The study computed and analyzed the reproduction number, R<sub>e</sub> of the system equations. The study presented and analysed, the global stability of disease free equilibrium and endemic equilibrium state, it has been found that when there is no transmission between human and non-human ( $\sigma_{p_2}$ ), then $\hat{G}(.X_1, X_2)$ . = 0 meaning it is globally asymptotically stable (GAS) at DFE and using nonlinear lyapunov function, the study shows that the endemic equilibrium state is GAS if P < M and unstable if P > M by comparison method of lyapunov functions. Numerical Simulations were done, it was found that the effective reproduction number decreases as vaccination of individual increases, varying the public awareness, the effective reproduction number reduces to zero and becomes stable as public awareness increases. It was discovered that effective reproduction number decreases as public awareness increases.

Keywords: Disease free equilibrium, Effective reproduction number, Endemic equilibrium, Mathematical Modeling, Monkey Pox Disease, Global Stability, Vaccination

## INTRODUCTION

Pox disease is a family of orthopox viruses, which are monkey pox, small pox and cow pox diseases (Lasisi, Akinwande and Oguntolu, 2020; Bhunu and Mushayabase, 2011). Monkey pox cause infection in nonhumans and humans and causes lymph nodes to swell. The symptoms include fever, headache, muscle aches, and a feeling of discomfort. The infection spread to humans from an infected animal (rodents) through direct contact, animal bite and eating infected animal meats without proper done or cook. The disease also spread from infected person to person, less infectious than small pox virus, communicated through contact with body fluids of an infected individual, unclean and contaminated objects and sexual intercourse. The hazard factors for communication include sharing a room and bed, using the same tools as an infected person (Kantele, *et al.*, 2016).

Monkey-pox (MPX) epidemic was first discovered in 1958, which was later found evidence of monkeypox infection in a number of African rodents (Von Magnus, Andersen, Petersen, et al., 1959). In 1970, the virus was reported in humans (Alakunle, Moens, Nchinda et al., 2020; Jezek, Marennikova, Mutumbo, et al., 1986). The incubation time is from 7-14days, the infection lasted for 2 to 4 weeks according to Centres for Disease Control (2003) and the fatality is 1% to 10% according to Rimoin, Kisalu, Kebela-Ilungam, et al. (2007). Monkeypox is endemic in Nigeria and in Congo Republic. The virus has been recounted in several countries in Africa, including Nigeria. At the start of the outbreak in 2017 to 2018, where 269 cases were suspected and 7 deaths were verified. In year 2018, there were 76 cases reported, 37 cases were confirmed and 2 deaths. In year 2022, Nigeria reported 558 suspected cases, where 231 were confirmed and No deaths were documented in 2022. Until year 2023, where the total confirmed cases is 988 from September 2017 to January 1, 2023 out of total suspected cases of 2635 (NCDC, 2023).

Deterministic models played a fundamental role in the disease transmission (Lasisi, *et al.*, 2018; Lasisi, and Adeyemo, 2021; Lasisi and Suleiman, 2024). The objective of this paper is to the development and analyzed the stability of a modelling of transmission of monkey-pox virus in human and effects of public awareness and vaccination in Nigeria. Therefore, a mathematical modeling for monkey pox disease was developed by (Lasisi, *et al.*, 2020; Bhunu and Mushayabase, 2011) with six (6) compartments. The study complements the work of the aforementioned author by having nine (9) compartments.

## **Model Formulation**

The study formulates a model for the transmission of Monkey-pox infection in nonhuman and human population. The model is divided into epidemiological group see figure 1. The human populace is subdivided into five classes, namely; Susceptible class, S<sub>h</sub>, Vaccinated class, V<sub>h</sub>, Exposed asymptomatic class, Eh, Infected class, Ih, and Recovery class, Rh. The Nonhuman population model is subdivided into Susceptible class, Sp, Infected nonhuman class, Ip, Exposed asymptomatic class, Ep and Recovery class, Rp. As specified in the flowchart diagram in figure 1, individuals enter Susceptible class through birth and immigration  $(\prod_{k})$ , where a fraction of vaccinated human immigrants (f) enter class of vaccinated and proportion of unvaccinated immigrants (1-f) enter class of susceptible. The study does not consider the immigration of infection individual, because the study assumed that individuals coming from infection endemic zones have to be vaccinated. The susceptible persons vaccinated at the  $\gamma$  rate and loss the vaccination  $\omega$  rate. Susceptible human,  $S_h$  are exposed to infection  $\alpha_h$  rate and infected  $\beta_h$  rate, the natural death rate is  $\mu_h$  and death due to the monkey pox disease is at rate of  $\delta_h$  and recovery at the rate of  $\rho_h$ ,  $\emptyset$  is rate of public awareness. The Susceptible nonhuman class, Sp is produced from the daily recruitment of persons through birth and immigration at the rate of  $\Pi_p$ , and natural death at  $\mu_p$  rate. The nonhuman exposed to monkey-pox virus at the rate  $\alpha_p$ , and move to infected class at the rate

of  $\beta_p$ . Nonhuman infected death due to the infection at the rate of  $\delta_p$  and recovery at the rate of  $\rho_p$ . Below is the flowchart representation of the model:



Figure 1: Flowchart representation of the Monkey-pox infection model

From the flow chart representation of the disease in figure 1 and assumptions, the dynamics of the monkey pox disease is described by ordinary differential equations below:

$$\frac{a S_h}{dt} = (1 - f_{\cdot})\Pi_h + \omega V_h - \gamma S_h - \alpha_h S_h - \mu_h S_h \qquad (1)$$
$$\frac{d E_h}{dt} = \alpha_h S_h - (1 - \varphi)\beta_h E_h - \mu_h E_h \qquad (2)$$

$$\frac{dI_h}{dt} = (1 - \varphi)\beta_h E_h - \rho_h I_h - \mu_h I_h - \delta_h I_h$$
(3)

$$\frac{dv_h}{dt} = f\Pi_h + \gamma S_h - \omega V_h - \mu_h V_h \tag{4}$$

$$\frac{\partial u}{\partial t} = \rho_h I_h - \mu_h R_h \tag{5}$$

$$\frac{dt}{dt_p} = \alpha_p S_p - \beta_p E_p - \mu_p E_p \tag{6}$$

$$\frac{dI_p}{dt} = \beta_p E_p - \rho_p I_p - \mu_p I_p - \delta_p I_p \tag{8}$$

$$\frac{\partial h}{\partial t} = \rho_p r_p - \mu_p R_p \tag{9}$$
Where,

$$N_{h} = S_{h} + E_{h} + I_{h} + V_{h} + R_{h}$$
(10)  
$$N_{p} = S_{p} + E_{p} + I_{p} + R_{p}$$
(11)

$$\alpha_h = \frac{\sigma_{p_1}(\varepsilon_p E_p + I_p)}{N} + \frac{\sigma_h(\varepsilon_h E_h + I_k)}{N}$$
(12)

$$\alpha_P = \frac{\sigma_{p_2}(\varepsilon_p E_p + I_p)}{N_p} \tag{13}$$

 $S_h$  Becomes infected from both  $I_p$  and  $I_h$ , where  $\sigma_{p1}$  is effective contact product rate and probability of  $S_h$  becomes infected from  $I_p$  and  $\sigma_h$  is effective contact product rate and probability of  $S_h$  becomes infected from  $I_h$ . Correspondingly, the  $S_p$  becomes infected from infected nonhuman, where  $\sigma_{p2}$  is effective contact product rate and probability of nonhuman is becomes infected per contact with an infected  $I_p$ (Bhunu and Mushayabase, 2011). The adjustment parameter  $\varepsilon_h$  is the assumption that exposed human transmits at a rate lower than symptomatic humans. The adjustment parameter  $\varepsilon_p$  is for the assumption that exposed nonhuman transmits at a rate lower than symptomatic nonhuman and Monkey pox mortality is negligible due to human hunter

#### Analysis and Results of the Model

**Theorem 1:** The following biological feasible region of the equations (1.) - (9)  $\Omega = \{S_h, E_h, I_h, V_{h.}, R_{h.}, S_p, E_p, I_p, R_p.\} \in \Re_+^9: \{S_h + S_{h.}\}$ 

 $E_h + I_h + V_h + R_h \leq \frac{\prod_h}{\mu_h}; S_p + E_p + I_p + R_p \leq \frac{\prod_p}{\mu_p} \} \text{ is absolutely invariant and attracting.}$ 

**Proof**; The addition of all the model equations in (1) - (9) give  $\frac{dN_h}{dN_h} = \prod_h - \mu_h N_h - \delta_h I_h$ 

And 
$$\frac{\frac{dt}{dN_p}}{\frac{dN_p}{dt}} = \Pi_p - \mu_p N_p - \delta_p I_p$$
  
So that 
$$\frac{\frac{dN_h}{dt}}{\frac{dN_h}{dt}} \le \Pi_h - \mu_h N_h \text{ and } \frac{\frac{dN_p}{dt}}{\frac{dN_p}{dt}} \le \Pi_p - \mu_p N_p$$
(14)

It follows from (Bauch and Earn, 2003), the gronwall inequality, that

$$N_{h}(t) \leq N_{h}(0)e^{-\mu_{h}(t)} + \frac{\prod_{h}}{\mu_{h}} \{1 - e^{-\mu_{h}(t)}\}$$
  
And  $N_{p}(t) \leq N_{p}(0)e^{-\mu_{p}(t)} + \frac{\prod_{p}}{\mu_{p}} \{1 - e^{-\mu_{p}(t)}\}$  (15)

In specific, 
$$N_h(t) \le \frac{\Pi_h}{\mu_h}$$
 if  $N_h(0) \le \frac{\Pi_h}{\mu_h}$  if only  $N_p(t) \le \frac{\Pi_h}{\mu_h}$ 

 $\frac{\prod_{p}}{\mu_{p}}, \text{ also } N_{p}(0) \leq \frac{\prod_{p}}{\mu_{p}}. \text{ And } \Omega \text{ is positively invariant.}$ Therefore, it is enough to consider the model equations dynamics (1) - (9) in  $\Omega$ . In this region, the model system can be considered as been mathematically and epidemiologically well posed.

**Theorem 2:** (Non-negativity Solution of the Model system). Let  $t_0 > 0$ , the initial conditions satisfied  $S_{\cdot h}(0) > 0$ ,  $E_{\cdot h}(0) > 0$ ,  $I_{\cdot h}(0) > 0$ ,  $V_{\cdot h}(0) > 0$ ,  $R_{h}(0) > 0$ ,  $S_{p}(0) > 0$ ,  $E_{p}(0) > 0$ ,  $I_{p}(0) > 0$ ,  $R_{p}(0) > 0$ , then the solutions  $S_{h}$ ,  $E_{h}$ ,  $I_{h}$ ,  $V_{h}$ ,  $R_{h}$ ,  $S_{p}$ ,  $E_{p}$ ,  $I_{p}$ ,  $R_{p}$  of the model systems (1) - (9) are all nonnegative for  $t \ge 0$ .

### Proof:

To show that for all  $t \in [0, t_0]$ ,  $S_h$ ,  $E_h$ ,  $I_h$ ,  $V_h$ ,  $R_h$ ,  $S_p$ ,  $E_p$ ,  $I_p$ ,  $R_p$  are nonnegative in  $\Re^9_+$ , note that the parameters used in the model are nonnegative. Thus, it is clear from model equation (1) that

$$\frac{d S_h}{dt} = (1 - f)\Pi_h + \omega \nabla - \gamma S_h - \alpha_h S_h - \mu_h S_h \ge -(\gamma + \alpha_h + \mu_h)S_h$$
  
So that,

$$\begin{split} S_h(t) &\geq S_h(0) \mathrm{e}^{\{-\int (\gamma + \alpha_h + \mu_h) dt\}} \\ \text{Similarly, we can apply the approach to show that } E_h > 0, \\ I_h &> 0, \ V_h > 0, \ R_h > 0, \ S_p > 0, \ E_p > 0, I_p > 0, R_p > 0 \\ \text{Hence, for all } t \in [0, t_0], \ S_h, \ E_h, \ I_h, \ V_h, \ R_h, \ S_p, \ E_p, \ I_p, \\ R_p \text{will be nonnegative and remain in } \Re_+^9 \end{split}$$

#### The Equilibrium State

At equilibrium point, we setting the model equations to zero, we have dS = dE = dI = dR = dR

$$\frac{dS_h}{dt.} = \frac{dE_h}{dt.} = \frac{dI_h}{dt.} = \frac{dV_h}{dt.} = \frac{dK_h}{dt} = \frac{dS_p}{dt} = \frac{dE_p}{dt} = \frac{dI_p}{dt} = \frac{dK_p}{dt} = 0.$$
  
From (1)-(13), we have the following  
$$I_h = 0 \text{ Or } \left( \frac{(\sigma_h \varepsilon_h (\rho_h + \mu_h + \delta_h) + \sigma_h \beta_h)S_h}{\beta_h N_h} - \frac{(\beta_h + \mu_h)(\rho_h + \mu_h + \delta_h)}{\beta_h} \right) = 0$$
(17)  
and  
$$I_p = 0 \text{ or } \frac{(\sigma_{p2} \varepsilon_p (\rho_p + \mu_p + \delta_p) + \sigma_p \beta_p)S_p}{\beta_h N_h} - \frac{(\beta_p + \mu_p)(\rho_p + \mu_p + \delta_p)}{\beta_h} = 0$$

$$V_{h} = \frac{f \Pi_{h} + \gamma S_{h}}{(\omega + \mu_{h})} = \frac{\gamma S_{h} + \alpha_{h} S_{h} + \mu_{h} S_{h} - (1-f) \Pi_{h}}{\omega}$$
(20)  
Implies,  

$$S_{h}^{0} = \frac{\Pi_{h} \omega + \Pi_{h} \mu_{h} - f \Pi_{h} \mu_{h}}{\gamma \mu_{h} + \mu_{h} \omega + \mu_{h}^{2}}$$
(21)

As  $\omega = \gamma = f = 0$  (no vaccination) then  $S_h^{\circ} = \frac{m_h}{\mu_h}$ Making substitution of equation (25) into equation (4), gives  $V_{\cdot}^0 = \frac{f \Pi_h \mu_h \omega + f \Pi_h \mu_h^2 + \gamma \Pi_h \omega + \gamma \Pi_h \mu_h}{(22)}$ 

$$V_h - (\gamma \mu_h + \mu_h \omega + \mu_h^2)(\omega + \mu_h)$$
  
Since  $I_p = 0$  then, we have  $E_p = R_p = 0$   
From (6), we have  $S_p^0 = \frac{\Pi_p}{\omega}$ 

The DFE state is given by  $E_0 = \{S_h^*, E_h^*, V_h^*, I_h^*, R_h^*, S_p^*, E_p^*, I_p^*, R_p^*\}$ =  $\{\frac{\Pi_h \omega + \Pi_h \mu_h - f \Pi_h \mu_h}{\gamma \mu_h + \mu_h \omega + \mu_h^2}, 0, \frac{f \Pi_h \mu_h \omega + f \Pi_h \mu_h^2 + \gamma \Pi_h \omega + \gamma \Pi_h \mu_h}{(\gamma \mu_h + \mu_h \omega + \mu_h^2)(\omega + \mu_h)}, 0, 0, 0, \frac{\Pi_p}{\mu_p}, 0, 0, 0\}$  (23)

### Effective Reproduction Number $(R_e)$

We compute the effective basic reproduction number according to (Van den Driessche and Watmough, 2002), using next generation matrix. Therefore, effective basic reproduction number is the spectral radius of  $FV^{-1}$ 

$$FV^{-1} = \left[\frac{\partial F_i(E^0)}{\partial x_i}\right] \left[\frac{\partial V_i(E^0)}{\partial x_i}\right]^{-1}$$
(24)

Where,  $E^0$  is the disease free equilibrium  $F_i$  is the new infection in compartment *i* and  $V_i$  is the movement of infection from one compartment *i* to another, so,  $R_0 = \rho(FV^{-1})$  is spectral radius ( $\rho$ ) of the next generation matrix  $FV^{-1}$ , the linearization of system (1)-(9) give *F* and *V*, obtained from the Jacobian matrix with the disease free equilibrium. Vector *F* is the inflow and *V* is the outflow from compartments  $E_h$ ,  $E_p$ ,  $I_h$  and  $I_p$ . We get

$$f = \begin{bmatrix} f_1 \\ f_2 \\ f_3 \\ f_4 \end{bmatrix} = \begin{bmatrix} \left(\frac{\sigma_{p1}(\varepsilon_p E_p + I_p)}{N_p} + \frac{\sigma_h(\varepsilon_h E_h + I_h)}{N_h}\right) S_h \\ \beta_h E_h \\ \frac{\sigma_{p2}(\varepsilon_p E_p + I_p) S_p}{N_p} \\ \beta_p E_p \end{bmatrix}$$
(25)  
$$F = \begin{bmatrix} \frac{\sigma_h \varepsilon_h S_h^0}{N_h^0} & \frac{\sigma_h S_h^0}{N_h^0} & \frac{\sigma_{p1} \varepsilon_p S_h^0}{N_p^0} & \frac{\sigma_{p1} S_h^0}{N_p^0} \\ \beta_h & 0 & 0 & 0 \\ 0 & 0 & \frac{\sigma_{p2} \varepsilon_p S_p^0}{N_p^0} & \frac{\sigma_{p2} S_p^0}{N_p^0} \\ 0 & 0 & .\beta_p & 0 \end{bmatrix}$$
(26)

$$v = \begin{bmatrix} v_1 \\ v_2 \\ v_3 \\ v_4 \end{bmatrix} = \begin{bmatrix} Q_1 E_h \\ Q_2 I_h \\ Q_3 E_p \\ Q_4 I_p \end{bmatrix}$$
(27)

Rashad et al.,

Where,  $Q_1 = \beta_h + \mu_h$ ;  $Q_2 = \rho_h + \mu_h + \delta_h$ ;  $Q_3 = \beta_p + \mu_p$ ;  $Q_4 = \rho_p + \mu_p + \delta_p$ 

$$V = \begin{pmatrix} Q_{1.} & 0 & 0 & 0 \\ 0 & Q_{2.} & 0 & 0 \\ 0 & 0 & Q_{3.} & 0 \\ 0 & 0 & 0 & Q_{4,} \end{pmatrix}$$
(28)  
From (28), we have  
$$V^{-1} = \begin{bmatrix} \frac{1}{Q_{1}} & 0 & 0 & 0 \\ 0 & \frac{1}{Q_{2}} & 0 & 0 \\ 0 & 0 & \frac{1}{Q_{3}} & 0 \\ 0 & 0 & 0 & \frac{1}{Q_{4}} \end{bmatrix}$$
(29)

At disease free equilibrium point, and since  $N_h \leq \frac{\prod h}{\mu_h}$  and

$$N_{p} \leq \frac{\mu_{p}}{\mu_{p.}} \text{ we get}$$

$$F = \begin{bmatrix} \frac{\sigma_{h} \varepsilon_{h} \mu_{h} S_{h.}^{0}}{\Pi_{h}} & \frac{\sigma_{h} \mu_{h} S_{h.}^{0}}{\Pi_{h.}} & \frac{\sigma_{p_{1}} \varepsilon_{p} \mu_{p} S_{h.}^{0}}{\Pi_{p}} & \frac{\sigma_{p_{1}} \mu_{p} S_{h.}^{0}}{\Pi_{p.}} \\ \beta_{h.} & 0 & 0 & 0 \\ 0 & 0 & \sigma_{p_{2}} \varepsilon_{p.} & \sigma_{p_{2.}} \\ 0 & 0 & \beta_{p.} & 0 \end{bmatrix}$$
(30)
$$FV^{-1} = \begin{bmatrix} \frac{\partial F_{i}(E^{0})}{\partial x_{j}} \end{bmatrix} \begin{bmatrix} \frac{\partial V_{i}(E^{0})}{\partial x_{j}} \end{bmatrix}^{-1}$$
(31)

Multiplying (29) and (30), we have

$$FV^{-1} = \begin{bmatrix} \frac{\sigma_h \varepsilon_h \mu_h S_h^0}{\Pi_h Q_1} & \frac{\sigma_h \mu_h S_h^0}{\Pi_h Q_2} & \frac{\sigma_{p_1} \varepsilon_p \mu_p S_h^0}{\Pi_p Q_3} & \frac{\sigma_{p_1} \mu_p S_h^0}{\Pi_p Q_4} \\ \frac{\underline{\beta}_h}{Q_1} & 0 & 0 & 0 \\ 0 & 0 & \frac{\sigma_{p_2} \varepsilon_p}{Q_3} & \frac{\sigma_{p_2}}{Q_4} \\ 0 & 0 & \frac{\beta_p}{Q_3} & 0 \end{bmatrix}$$
(32)

Characteristics Equation of (32), gives  $/FV^{-1} - \lambda I /= 0$  $\begin{bmatrix} L_1 S_h^0 - \lambda & L_2 S_h^0 & L_3 S_h^0 & L_4 S_h^0 \\ L_5 & -\lambda & 0 & 0 \\ 0 & 0 & L_6 - \lambda & L_7 \end{bmatrix} = 0$  (33)

Where, 
$$L_1 = \frac{\sigma_h \varepsilon_h \mu_h S_h^0}{\Pi_h Q_1}$$
,  $L_2 = \frac{\sigma_h \mu_h}{\Pi_h Q_2}$ ,  $L_3 = \frac{\sigma_{p_1} \varepsilon_p \mu_p}{\Pi_p Q_3}$ ,  $L_4 = \frac{\sigma_{p_1} \mu_p}{\Pi_p Q_4}$   
 $L_5 = \frac{\beta_h}{Q_1}$ ,  $L_6 = \frac{\sigma_{p_2} \varepsilon_p}{Q_3}$ ,  $L_7 = \frac{\sigma_{p_2}}{Q_4}$ ,  $L_8 = \frac{\beta_p}{Q_3}$  (34)  
Determinant of (33) gives  
 $(\lambda^2 - L_6\lambda - L_7L_8) = 0 \text{or}(\lambda^2 - L_1S_h^0\lambda - L_2L_5S_h^0) = 0$   
(35)

To solve (35) with completing the square method, we have

$$=\frac{\frac{\sigma_{p_{2}}\varepsilon_{p.}}{(\beta_{p,+}\mu_{p.})^{\pm}}\sqrt{\frac{(\sigma_{p_{2}}\varepsilon_{p.})^{2}}{(\beta_{p,-}\mu_{p.})^{2}}+\frac{4\sigma_{p_{2}}\beta_{p.}}{(\beta_{p,-}\mu_{p.})(\rho_{p}+\mu_{p.}+\delta_{p})}}$$

 $\lambda_1$  is the Spectral Radius of  $\rho(FV^{-1})$  and The reproduction number is given below

(36)

$$R_{p} = \frac{\frac{\sigma_{p_{2}}\varepsilon_{p}}{(\beta_{p}+\mu_{p})^{+}} + \sqrt{\frac{(\sigma_{p_{2}}\varepsilon_{p})^{2}}{(\beta_{p}+\mu_{p})^{2}} + \frac{4\sigma_{p_{2}}\beta_{p}}{(\beta_{p}+\mu_{p})(\rho_{p}+\mu_{p}+\delta_{p})}}{2}}$$
(37)

And  

$$(\lambda^{2} - L_{1}S_{h}^{0}\lambda - L_{2}L_{5}S_{h}^{0}) = 0$$
Implies,  $\lambda_{2} = \frac{L_{1}S_{h}^{0} \pm \sqrt{L_{1}^{2}S_{h}^{0^{2}} + 4L_{2}L_{5}S_{h}^{0}}}{2}$ 

$$(38)$$

$$\frac{-\sigma_{h}\varepsilon_{h}\mu_{h}S_{h}^{0}}{+} \sqrt{\frac{\sigma_{h}.^{2}\varepsilon_{h}.^{2}\mu_{h}^{2}S_{h}^{0^{2}}}{+} + \frac{4\sigma_{h}.\mu_{h}\beta_{h}S_{h}^{0}}{+}}$$

$$\lambda_{2} = \frac{\frac{\sigma_{h} \varepsilon_{h} \mu_{h} S_{h}^{*}}{\pi_{h} (\theta_{h} + \mu_{h})^{2}} \pm \sqrt{\frac{\sigma_{h}^{-} \varepsilon_{h} \varepsilon_{h} \mu_{h}^{+} S_{h}^{*}}{\pi_{h}^{2} (\theta_{h} + \mu_{h})^{2}} + \frac{4\sigma_{h} \mu_{h} \theta_{h} S_{h}^{*}}{\pi_{h} (\theta_{h} + \mu_{h}) (\phi_{h} + \mu_{h} + \delta_{h})}}$$
(39)

We have  $\lambda_2$  as spectral radius ( $\rho$ ) that is  $R_e = \rho(FV^{-1})$ .

 $\lambda_1$ 

Rashad et al.,

$$R_{h} = \frac{\frac{\sigma_{h} \varepsilon_{h} \mu_{h} S_{h}^{0}}{m_{h} (\theta_{h} + \mu_{h})^{4}} + \sqrt{\frac{\sigma_{h}^{2} \varepsilon_{h}^{2} \mu_{h}^{2} S_{h}^{0}}{m_{h}^{2} (\theta_{h} + \mu_{h})^{2}} + \frac{4\sigma_{h} \mu_{h} \beta_{h} S_{h}^{0}}{m_{h} (\theta_{h} + \mu_{h}) (\rho_{h} + \mu_{h} + \delta_{h})}}$$
(40)

Hence, the effective reproduction number can be represented as.

$$R_{e.} = R_{h.} + R_{p.}$$
(41)  
$$\left\{ \begin{array}{l} \left( \frac{\sigma_{h.\epsilon_{h.}\mu_{h.}}S_{h.}^{0}}{T_{h.}(\beta_{h.}+\mu_{h.})} + \frac{\sigma_{p2.}\epsilon_{p.}}{(\beta_{p.}+\mu_{p.})} + \frac{\sigma_{p2.}\epsilon_{p.}}{(\beta_{p.}+\mu_{p.})} + \frac{\sigma_{p2.}\epsilon_{p.}}{(\beta_{p.}+\mu_{p.})} + \frac{\sigma_{p2.}\epsilon_{p.}}{T_{h.}^{0}(\beta_{h.}+\mu_{h.})} + \frac{\sigma_{p2.}\epsilon_{p.}}{(\beta_{p.}+\mu_{p.})^{2}} + \frac{\sigma_{p2.}\epsilon_{p.}}{(\beta_{p.}+\mu_{p.})(\rho_{p.}+\mu_{h.}+\delta_{h.})} + \frac{\sigma_{p2.}\epsilon_{p.}}{(\beta_{p.}+\mu_{p.})^{2}} + \frac{\sigma_{p2.}\epsilon_{p.}}{(\beta_{p.}+\mu_{p.})(\rho_{p.}+\mu_{p.}+\delta_{p.})} - \frac{\sigma_{p2.}\epsilon_{p.}}{2} + \frac{\sigma_{p2.}\epsilon_{p.}}{(\beta_{p.}+\mu_{p.})(\rho_{p.}+\mu_{p.}+\delta_{p.})} + \frac{\sigma_{p2.}\epsilon_{p.}}{(\beta_{p.}+\mu_{p.})^{2}} + \frac{\sigma_{p2.}\epsilon_{p.}}{(\beta_{p.}+\mu_{p.})(\rho_{p.}+\mu_{p.}+\delta_{p.})} + \frac{\sigma_{p2.}\epsilon_{p.}}{(\beta_{p.}+\mu_{p.})^{2}} + \frac{\sigma_{p2.}\epsilon_{p.}}{(\beta_{p.}+\mu_{p.}+\delta_{p.}+\delta_{p.})} + \frac{\sigma_{p2.}\epsilon_{p.}}{(\beta_{p.}+\mu_{p.})^{2}} + \frac{\sigma_{p2.}\epsilon_{p.}}{(\beta_{p.}+\mu_{p.}+\delta_{p.}+\delta_{p.}+\delta_{p.})} + \frac{\sigma_{p2.}\epsilon_{p.}}{(\beta_{p.}+\mu_{p.})^{2}} + \frac{\sigma_{p2.}\epsilon_{p.}}{(\beta_{p.}+\mu_{p.}+\delta_$$

## **Global Stability of DFE State**

Theorem 3: The DFE of the model is globally asymptotically stable (GAS) if  $R_e < 1$ Proof.

$$\frac{dX_1}{dt.} = F(.X_1, X_2.) \tag{43}$$

$$\frac{1}{dt.} = (S_h^0, V_h^0, R_h^0, S_p^0, R_p^0), \text{AND}$$
(44)

$$X_2 = (E_h^0, I_h^0, E_p^0, I_p^0)$$
(45)  
The DFE is now represented as,

 $E^{0} = (X_{1}^{*}, 0.)$  where,  $X_{1}^{*} = (N^{0}, 0.)(46)$ 

Firstly, the condition that is GAS of 
$$X_{1}^{*}$$
, gives  

$$\frac{dx_{1}}{dt} = F(X_{1}, 0) = \begin{cases}
(1 - f)\Pi_{h} + \omega V_{h}^{0} - \gamma S_{h}^{0} - (0)S_{h}^{0} - \mu_{h}S_{h}^{0} \\
f\Pi_{h} + \gamma S_{h}^{0} - \omega V_{h}^{0} - \mu_{h}V_{h}^{0} \\
-\mu_{h}R_{h}^{0} \\
\Pi_{p} - (0)S_{p}^{0} - \mu_{p}S_{p}^{0} \\
\rho_{p}I_{p}^{0} - \mu_{p}R_{p}^{0}
\end{cases}$$
(47)  
A linear differential equation solving gives,  
 $S_{h}^{0}(t) = \frac{(1 - f)\Pi_{h} + \omega V_{h}^{0}}{(\gamma + \mu_{h})} - \frac{(1 - f)\Pi_{h} + \omega V_{h}^{0}}{(\gamma + \mu_{h})} * e^{-(\gamma + \mu_{h})t.} + S_{h}^{0}(0) * e^{-(\gamma + \mu_{h})t.}$ (48)  
 $V_{h}^{0}(t) = \frac{f\Pi_{h} + \gamma S_{h}^{0}}{(\omega + \mu_{h})} - \frac{f\Pi_{h} + \gamma S_{h}^{0}}{(\omega + \mu_{h})} * e^{-(\omega + \mu_{h})t} + V_{h}^{0}(0) * e^{-(\omega + \mu_{h})t}$ 

$$R_h^0(t.) = R_h^0(0)^* e^{-\mu_h t.}$$
(49)
(50)

$$S_{p}^{0}(t) = \frac{\Pi_{p}}{\mu_{p}} - \frac{\Pi_{p}}{\mu_{p}} e^{-\mu_{p}t} + S_{p}^{0}(0)e^{-\mu_{p}t}$$
(51)  
$$R_{p}^{0}(t) = R_{p}^{0}(0)e^{-\mu_{p}t}$$
(52)

This showed that  $S_h^0 + V_h^0 + R_h^0 + S_p^0 + R_p^0 -> N_{\square}^0$  as t -> $\infty$  regardless of the value of  $S_h^0, V_h^0, R_h^0, S_p^0 and R_p^0$ . Therefore,  $X_{1.}^{*} = (.N^{0}, 0.)$ . is globally asymptotically stable. To show if the second condition is true:  $\hat{G}(.X_1, X_2) = AX_2 - AX_2$ 

$$G(.X_1, X_2)$$
  
Where  $X_2 = (E_h^0, I_h^0, E_p^0, I_p^0)$ 

$$A = \begin{bmatrix} \left(\frac{\sigma_{h}\epsilon_{h}S_{h}^{h}}{N_{h}^{0}} - \left(.\beta_{h} + \mu_{h}\right) & \frac{\sigma_{h}S_{h}^{h}}{N_{h}^{0}} & \frac{\sigma_{p}\epsilon_{p}S_{h}^{h}}{N_{p}^{0}} & \frac{\sigma_{p}\rho_{h}S_{h}^{h}}{N_{p}^{0}} & \frac{\sigma_{p}\rho_{h}S_{h}^{h}}{N_{p}^{0}} \\ 0 & \beta_{h} & -(\rho_{h} + \mu_{h} + \delta_{h}) & 0 & 0 \\ 0 & 0 & \left(\frac{\sigma_{p}\epsilon_{p}\rho_{h}S_{p}^{h}}{N_{p}^{0}}\right) - (\beta_{p} + \mu_{p}) & \left(\frac{\sigma_{p}\rho_{h}S_{p}^{h}}{N_{p}^{0}}\right) \\ 0 & 0 & \beta_{p} & -(.\rho_{p} + \mu_{p} + \delta_{p})] \end{bmatrix}$$
(53)  
$$G(X_{1}, X_{2}) = \begin{bmatrix} \left(\frac{\sigma_{p}(\epsilon_{p}E_{p}^{h}+l_{p}^{h})}{N_{p}^{0}} + \frac{\sigma_{h}(\epsilon_{h}E_{h}^{h}+l_{h}^{h})}{N_{h}^{0}}\right)S_{h}^{0} - (\beta_{h} + \mu_{h})E_{h}^{0} \\ -(\beta_{h} + \rho_{h} + \mu_{h} + \delta_{h})I_{h}^{0} \\ \left(\frac{\sigma_{p}(\epsilon_{p}E_{p}^{h}e_{p}^{h})}{N_{p}^{0}}\right)S_{p}^{0} - (\beta_{p} + \mu_{p})E_{p}^{0} \\ \beta_{p}E_{p}^{0} - (\rho_{p} + \mu_{p} + \delta_{p})I_{p}^{0} \end{bmatrix} = \begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}$$
(54)  
From,  $\hat{G}(.X_{1}, X_{2}) = A^{*}X_{2} - ...G(X_{1}, X_{2}) \\ \begin{bmatrix} \frac{\sigma_{h}\epsilon_{h}S_{h}^{h}}{N_{h}^{0}} & \frac{\sigma_{p}\epsilon_{p}S_{h}^{h}}{N_{p}^{0}} \\ \beta_{h} & -(\rho_{h} + \mu_{h} + \delta_{h}) & 0 & 0 \\ 0 & 0 & \left(\frac{\sigma_{p}*\epsilon_{p}S_{p}^{h}}{N_{p}^{0}}\right) - (.\beta_{p} + \mu_{p}) & \left(\frac{\sigma_{p}S_{p}^{h}}{N_{p}^{0}} \\ 0 & 0 & \left(\frac{\sigma_{p}-\epsilon_{p}S_{p}^{h}}{N_{p}^{0}}\right) - (.\beta_{p} + \mu_{p} + \delta_{p}) \right] \begin{bmatrix} E_{h}^{0} \\ I_{h}^{0} \\ I_{p}^{0} \\ \end{bmatrix} - \begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \end{bmatrix} = \begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}$ (55)  
 $\hat{G}(.X_{1}, X_{2}) = [.0 & 0 & 0 & 0.1^{T}. \end{bmatrix}$ 

It is clear that,  $\hat{G}(X_1, X_2) = 0$ . Therefore, the proof is complete. It implies, disease free equilibrium of the model system is GAS if Re < 1.

## Endemic Equilibrium (EE) State

Endemic Equilibrium (EE) State At equilibrium state, we set (1) - (9) to zero, we have  $\frac{dS_{h}}{dt.} = \frac{dE_{h}}{dt.} = \frac{dI_{h}}{dt.} = \frac{dV}{dt.} = \frac{dR_{h}}{dt.} = \frac{dS_{p}}{dt.} = \frac{dE_{p}}{dt.} = \frac{dI_{p}}{dt.} = \frac{dR_{p}}{dt.} = 0$   $(1 - f)\Pi_{h} + \omega V_{h}^{*} - \gamma S_{h}^{*} - (\frac{\sigma_{p_{1}.}(\varepsilon_{p}.^{*}E_{p..}^{*} + I_{p..}^{*})}{N_{p}} + \frac{\sigma_{h..}(\varepsilon_{h..}E_{h..}^{*} + I_{h..}^{*})}{N_{h}})S_{h..}^{*} - \mu_{h}*S_{h.}^{*}$   $= 0(\frac{\sigma_{p_{1}.}(\varepsilon_{p}.E_{p.}^{*} + I_{p..}^{*})}{.N_{p}} + \frac{\sigma_{h.}(\varepsilon_{h}.E_{h.}^{*} + I_{h..}^{*})}{.N_{h}})S_{h.}^{*} - \beta_{h}E_{h..}^{*} - \mu_{h}.E_{h.}^{*} = 0.$   $\beta_{h}E_{h}^{*} - \rho_{h}I_{h}^{*} - \mu_{h}I_{h}^{*} - \delta_{h}I_{h}^{*} = 0$ (56) 
$$\begin{split} f\Pi_{h} + \gamma S_{h}^{*} - \omega V_{h}^{*} - \mu_{h} V_{h}^{*} &= 0 \\ \rho_{h} I_{h}^{*} - \mu_{h} R_{h}^{*} &= 0 \end{split}$$
(57)

# Rashad et al.,

$$\begin{split} H_{\mu} &- \left(\frac{g_{\mu}}{Q_{\mu}} \frac{(q_{\mu} E_{\mu}^{\mu} + I_{\mu}^{\mu})}{N_{\mu}} S_{\mu}^{\mu} - \mu_{\mu} S_{\mu}^{\mu} = 0 \\ \frac{g_{\mu}^{\mu} (q_{\mu} E_{\mu}^{\mu} + I_{\mu}^{\mu})}{Q_{\mu}^{\mu} F_{\mu}^{\mu} - P_{\mu}^{\mu} F_{\mu}^{\mu} = 0 \\ g_{\mu} I_{\mu}^{\mu} - \mu_{\mu} R_{\mu}^{\mu} = 0. \\ \text{Where,} \\ A_{\mu} &= h_{\mu} + \mu_{\mu} A_{\mu} = p_{\mu} + \mu_{\mu} + h_{\mu} + h_{\mu} A_{\lambda} = \omega + \mu_{\mu} A_{\lambda} = \beta_{\mu} + \mu_{\mu} A_{\lambda} = \rho_{\mu} + \mu_{\mu} + \delta_{\mu}. \quad (58) \\ \text{From (57) und (58), we have the following:} \\ \frac{g_{\mu}^{\mu} (I_{\mu} + I_{\mu})}{I_{\mu}^{\mu} (I_{\mu})} = \frac{I (I_{\mu} + Y_{\lambda})}{A_{\mu}}. \quad (60) \\ V_{\mu}^{\mu} &= \frac{I (I_{\mu} + I_{\mu})}{I_{\mu}^{\mu} (I_{\mu})} = \frac{I (I_{\mu} + Y_{\lambda})}{A_{\mu}}. \quad (61) \\ E_{\mu}^{\mu} &= \frac{g_{\mu} A_{\mu}}{I_{\mu}}. \quad (62) \\ W_{\mu}^{\mu} &= \frac{I (I_{\mu} + Y_{\lambda})}{I_{\mu}} = \frac{A A_{\mu}^{\mu}}{A_{\mu}}. \quad (62) \\ W_{\mu}^{\mu} &= \frac{I (I_{\mu} + I_{\mu})}{I_{\mu}} = \frac{A A_{\mu}^{\mu}}{A_{\mu}}. \quad (63) \\ Substitute (62) into exposed class in (57), we get: \\ \frac{g_{\mu}^{\mu} (I_{\mu} A_{\mu} A_{\mu})}{I_{\mu}} = \frac{A A_{\mu} A_{\mu}}{B_{\mu}}. \quad (64) \\ From (64) we have: \\ \frac{g_{\mu}^{\mu} (I_{\mu} A_{\mu} A_{\mu})}{I_{\mu}} = \frac{A A_{\mu} A_{\mu}}{B_{\mu}}. \quad (65) \\ From (50) we have: the following: \\ \frac{f_{\mu}^{\mu} (I_{\mu} A_{\mu} A_{\mu} A_{\mu} A_{\mu})}{I_{\mu} (I_{\mu} A_{\mu} A_{\mu})} = \frac{A A_{\mu} A_{\mu}}{B_{\mu}}. \quad (65) \\ From (50) we there the following: \\ R_{\mu}^{\mu} = \frac{g_{\mu} (I_{\mu} A_{\mu} A_{\mu})}{I_{\mu} (I_{\mu} A_{\mu} A_{\mu})} = \frac{A A_{\mu} A_{\mu}}{B_{\mu}}. \quad (65) \\ From (60) we get: \\ R_{\mu}^{\mu} = \frac{I (I_{\mu} A_{\mu} A_{\mu} A_{\mu})}{I_{\mu} (I_{\mu} A_{\mu} A_{\mu})} = \frac{I (I_{\mu} A_{\mu} A_{\mu} A_{\mu})}{I_{\mu} (I_{\mu} A_{\mu} A_{\mu})} = \frac{I (I_{\mu} A_{\mu} A_{\mu})}{I_{\mu} (I_{\mu} A_{\mu} A_{\mu})} = \frac{I (I_{\mu} A_{\mu} A_{\mu} A_{\mu})}{I_{\mu} (I_{\mu} A_{\mu} A_{\mu})} = \frac{I (I_{\mu} A_{\mu} A_{\mu} A_{\mu})}{I_{\mu} (I_{\mu} A_{\mu} A_{\mu})} = \frac{I (I_{\mu} A_{\mu} A_{\mu} A_{\mu})}{I_{\mu} (I_{\mu} A_{\mu} A_{\mu})} = \frac{I (I_{\mu} (I_{\mu} A_{\mu} A_{\mu})}{I_{\mu} (I_{\mu} A_{\mu} A_{\mu})} = \frac{I (I_{\mu} (I_{\mu} A_{\mu} A_{\mu})}{I_{\mu} (I_{\mu} A_{\mu} A_{\mu})} = \frac{I (I_{\mu} (I_{\mu} A_{\mu} A_{\mu})}{I_{\mu} (I_{\mu} A_{\mu} A_{\mu})} = \frac{I (I_{\mu} (I_{\mu} A_{\mu} A_{\mu})}{I_{\mu} (I_{\mu} A_{\mu} A_{\mu})} = \frac{I (I_{\mu} (I_{\mu} A_{\mu} A_{\mu})}{I_{\mu} (I_{\mu} A_{\mu} A_{\mu})} = \frac{I (I_{\mu} (I_{\mu} A_{\mu} A_{\mu})$$

FUDMA Journal of Sciences (FJS) Vol. 8 No. 5, October, 2024, pp 307 – 320

FJS

Substitute (76) into (60) we have:  

$$S_{h}^{*} = \frac{[(1-f)\Pi_{h}A_{3} + \omega_{f}\Pi_{h}]}{[\gamma A_{3} + A_{L}A_{3} + I_{h}^{*}A_{k}A_{3} + \mu_{h}A_{3} - \omega\gamma]}$$
Implies:  
(79)

$$S_{h}^{*} = \frac{[(1-f).I_{h}.A_{3}+\omega.f.I_{h}]}{[\gamma A_{3}+A_{L}A_{3}+\frac{\beta_{h}.I_{h}.\mu_{p}.A_{k}.A_{3}[(\sigma_{p}.\varepsilon_{p}.A_{5}+\sigma_{p}.\beta_{p})-A_{4}.A_{5}]}{A_{4}.A_{5}[I_{h}.A_{1}.A_{2}-\mu_{h}.(\sigma_{h}.\varepsilon_{h}.A_{2}+\sigma_{h}.\beta_{h})]} + \mu_{h}.A_{3}-\omega.\gamma]}$$
(80)

From (4) we have:

$$V_h^* = \frac{f \cdot \Pi_h + \gamma \cdot S_h^*}{(\omega_h^* + \mu_h)} = \frac{f \cdot \Pi_h}{A_3} + \frac{\gamma \cdot S_h^*}{A_3}$$
(81)
Implies:

$$V_{h}^{*} = \frac{f\Pi_{h}}{A_{3}} + \frac{\gamma[(1-f)\Pi_{h}A_{3} + \omega f\Pi_{h}]}{A_{3}[\gamma A_{3} + A_{L}A_{3} + \frac{\beta_{h}\Pi_{h}\mu_{p}A_{k}A_{3}(\sigma_{p}\varepsilon_{p}A_{3} + \sigma_{p}\beta_{p}) - A_{4}A_{5}]}{A_{4}A_{5}[\Pi_{h}A_{1}A_{2} - \mu_{h}(\sigma_{h}\varepsilon_{h}A_{2} + \sigma_{h}\beta_{h})]} + \mu_{h}A_{3} - \omega\gamma]$$
(82)

Eq. (61) yield:  

$$R_{h}^{*} = \frac{\rho_{h} \cdot \beta_{h}}{\mu_{h}} = \frac{\rho_{h} \cdot \beta_{h} \cdot \Pi_{h} \cdot \mu_{p} \{(\sigma_{p} \cdot \varepsilon_{p} \cdot A_{5} + \sigma_{p} \cdot \beta_{p}) - A_{4} \cdot A_{5}\}}{\mu_{h} \cdot A_{4} \cdot A_{5} [\Pi_{h} \cdot A_{1} \cdot A_{2} - \mu_{h} (\sigma_{h} \cdot \varepsilon_{h} \cdot A_{2} + \sigma_{h} \cdot \beta_{h})]}$$
(83)  
Implies:

$$R_{h}^{*} = \frac{\rho_{h}\beta_{h}.T_{h}.\mu_{p}\{(\sigma_{p}.\varepsilon_{p}.A_{5}+\sigma_{p}.\beta_{p})-A_{4}.A_{5}\}}{\mu_{p}A_{1}A_{2}.U_{h}A_{2}A_{2}.U_{h}A_{2}A_{2}.U_{h}A_{2}A_{2}.U_{h}A_{2}A_{2}.U_{h}A_{2}A_{2}.U_{h}A_{2}A_{2}.U_{h}A_{2}A_{2}.U_{h}A_{2}A_{2}.U_{h}A_{2}A_{2}.U_{h}A_{2}A_{2}.U_{h}A_{2}A_{2}.U_{h}A_{2}A_{2}.U_{h}A_{2}A_{2}.U_{h}A_{2}A_{2}.U_{h}A_{2}A_{2}.U_{h}A_{2}.$$

$$E_{1} = \{ S_{h}^{*} > 0, \ E_{h}^{*} > 0, \ V_{h}^{*} > 0, \ I_{h}^{*} > 0, \ R_{h}^{*} > 0, \ R_{h}^{*} > 0, \ R_{p}^{*} > 0, \\ E_{p}^{*} > 0, \ E_{p}^{*} > 0, \ R_{p}^{*} > 0 \}$$
(85)

### **Global Stability of EE State**

This study used Lyapunov function according to Lasisi, (2021); Lasisi, (2020); Lasisi et. al., (2018) to examine the stability of the EE state, Lyapunov function  $G(E_1)$  is defined by:

$$\begin{aligned} G &= (S_{h} - S_{h}^{*} . \ln S_{h}) + (E_{h} - E_{h}^{*} . \ln E_{h}) + (I_{h} - I_{h}^{*} . \ln I_{h}) + (V_{h} - V_{h}^{*} . \ln V_{h}) + (R_{h} - R_{h}^{*} . \ln R_{h}) \\ (S_{p} - S_{p}^{*} . \ln S_{p}) + (E_{p} - E_{p}^{*} . \ln E_{p}) + (I_{p} - I_{p}^{*} . \ln I_{p}) + (R_{p} - R_{p}^{*} . \ln R_{p}) \end{aligned}$$
(86)  
The derivation of (86) is setting as:  

$$\begin{aligned} \frac{dG}{dt} &= (\frac{S_{h} - S_{h}^{*}}{S_{h}}) \frac{dS_{h}}{dt} + (\frac{E_{h} - E_{h}^{*}}{E_{h}}) \frac{dE_{h}}{dt} + (\frac{I_{h} - I_{h}^{*}}{I_{h}}) \frac{dI_{h}}{dt} + (\frac{V_{h} - V_{h}^{*}}{V_{h}}) \frac{dV_{h}}{dt} + (\frac{R_{h} - R_{h}^{*}}{R_{h}}) \frac{dR_{h}}{dt} \\ + (\frac{S_{p} - S_{p}^{*}}{S_{p}}) \frac{dS_{p}}{dt} + (\frac{E_{p} - E_{p}^{*}}{I_{p}}) \frac{dE_{p}}{dt} + (\frac{I_{p} - I_{p}^{*}}{R_{p}}) \frac{dI_{p}}{dt} + (\frac{R_{p} - R_{p}^{*}}{R_{p}}) \frac{dR_{p}}{dt} \end{aligned}$$
(87)  
Eq. (87) yield:  

$$\begin{aligned} (87) E_{q} . (87) vield: \\ \frac{dI}{dt} &= (\frac{S_{h} - S_{h}^{*}}{S_{p}}) \{(1 - f)H_{h} + \omega V_{h} - (\frac{\sigma p(\varepsilon_{p} E_{p} + I_{p})}{N_{p}} + \frac{\sigma_{h}(\varepsilon_{h} E_{h} + I_{h})}{N_{h}})S_{h} - A_{h}S_{h}\} + (\frac{E_{h} - E_{h}^{*}}{R_{p}})\{(\frac{\sigma p(\varepsilon_{p} E_{p} + I_{p})}{N_{p}} + \frac{\sigma_{h}(\varepsilon_{h} E_{h} + I_{h})}{N_{h}})S_{h} - B_{1}E_{h}\} \\ + (\frac{I_{h} - I_{h}^{*}}{I_{h}})\{\beta_{h}E_{h} - B_{2}I_{h}\} + (\frac{V_{h} - V_{h}^{*}}{V_{h}})\{fH_{h} + \gamma S_{h} - B_{3}V_{h}\} \\ + (\frac{R_{h} - R_{h}^{*}}{R_{p}})\{\rho_{p}.I_{p} - \mu_{p}.R_{p}\} \end{aligned}$$
(88)  
At the endemic equilibrium points, we have:  

$$\begin{aligned} \frac{dG}{dt} = (\frac{S_{h} - S_{h}^{*}}{S_{h}})\{I_{h} - \mu_{h}.R_{h}\} + (\frac{S_{p} - S_{p}^{*}}{S_{p}}).\{f_{h}.H_{h} + \gamma S_{h} - B_{3}.V_{h}\} \\ + (\frac{R_{p} - R_{p}^{*}}{S_{h}})\{\beta_{h}.E_{h} - B_{2}.I_{h}\} + (\frac{V_{h} - V_{h}^{*}}{V_{h}}).\{f_{h}.H_{h} + \gamma . S_{h} - B_{3}.V_{h}\} \\ + (\frac{R_{h} - R_{h}^{*}}{R_{h}}).\{\beta_{h}.E_{h} - B_{2}.I_{h}\} + (\frac{V_{h} - V_{h}^{*}}{S_{p}}).\{H_{p} - (\alpha_{p}).S_{p} - \mu_{p}.S_{p}\} + (\frac{E_{p} - E_{p}^{*}}{E_{p}}).\{(\alpha_{p}).S_{p} - B_{4}.E_{p}\} + (\frac{I_{p} - I_{p}^{*}}{I_{p}}).\{\beta_{p}.E_{p} - B_{5}.I_{p}\} \\ + (\frac{R_{h} - R_{h}^{*}}{R_{h}}).\{\rho_{h}.I_{h} - \mu_{h}.R_{h}\} + (\frac{S_{p} - S_{p}^{*}}{S_{p}}).\{H_{p} - (\alpha_{p}).S_{p} - \mu_{p}.S_{p}\} + (\frac{E_{p} - E_{p}^{*}}{E_{p}}).\{($$

Where, 
$$\alpha_h = \frac{\sigma_p.(\varepsilon_p.E_p.+I_p.)}{N_p} + \frac{\sigma_h.(\varepsilon_h.E_h+I_k.)}{N_h}$$
  
 $\alpha_p = \frac{\sigma_p.(\varepsilon_p.E_p+I_p.)}{N_p}$ 
(90)

From (89) we have:

From (89) we have:  $\frac{dG}{dt} = (S_h - S_h^*) \cdot \{\frac{\omega \cdot V_h}{(1-f) \cdot S_h} + (\alpha_p + \alpha_h) \cdot \frac{S_h^*}{(1-f) \cdot S_h} + \frac{A_h \cdot S_h^*}{(1-f) \cdot S_h} + \frac{\omega \cdot V_h}{S_h}\} - (S_h - S_h^*) \cdot \{(\alpha_p + \alpha_h) + A_h\} + (E_h - E_h^*) \cdot \{(\alpha_p + \alpha_h) \cdot S_h^* + \alpha_h\} + (E_h - E_h^*) \cdot (I_h - I_h^*) \cdot B_h - (I_h - I_h^*) \cdot B_h -$  $\left(\frac{R_p - R_p^*}{R_p}\right)\rho_p I_p - (R_p - R_p^*)\mu_p$ (91) Eq. (91) yield:

$$\frac{dG}{dt} = (S_{h} - S_{h}^{*})\{\frac{\omega V_{h}}{(1-f).S_{h}} + (\alpha_{p} + \alpha_{h})\frac{S_{h}^{*}}{(1-f).S_{h}} + \frac{A_{h}S_{h}^{*}}{(1-f).S_{h}} + \frac{\omega V_{h}}{S_{h}}\} + (E_{h} - E_{h}^{*}).(\alpha_{p} + \alpha_{h})\frac{S_{h}}{E_{h}} + (\frac{I_{h}-I_{h}^{*}}{I_{h}})\beta_{h}.E_{h} + (\frac{V_{h}-V_{h}^{*}}{V_{h}})(f.\Pi_{h} + \gamma S_{h}) + (\frac{R_{h}-R_{h}^{*}}{R_{h}})\rho_{h}.I_{h} + (\frac{S_{p}-S_{p}^{*}}{S_{p}})\Pi_{p} + (\frac{E_{p}-E_{p}^{*}}{E_{p}})(\alpha_{p})S_{p} + (\frac{I_{p}-I_{p}^{*}}{I_{p}})\beta_{p}.E_{p} + (\frac{R_{p}-R_{p}^{*}}{R_{p}})\rho_{p}.I_{p} - (S_{h} - S_{h}^{*})\{(\alpha_{p} + \alpha_{h}) + A_{h}\} - (E_{h} - E_{h}^{*})\frac{(\alpha_{p}+\alpha_{h})S_{h}^{*}}{E_{h}^{*}}\} - (I_{h} - I_{h}^{*})B_{2} - (V_{h} - V_{h}^{*})B_{3} - (R_{h} - R_{h}^{*})\mu_{h} - (S_{p} - S_{p}^{*}).[(\alpha_{p}) + \mu_{p}] - (E_{p} - E_{p}^{*})..B_{4} - (I_{p} - I_{p}^{*})B_{5} - (R_{p} - R_{p}^{*}).\mu_{p}$$
(92)
Eq. (92) yields:
$$\frac{dG}{dt} = P - M$$
(93)
Where:
$$P = (S_{h} - S_{h}^{*})\{\frac{\omega V_{h}}{(1-f).S_{h}} + (\alpha_{p} + \alpha_{h})\frac{S_{h}^{*}}{(1-f).S_{h}} + \frac{A_{h}S_{h}^{*}}{(1-f).S_{h}} + \frac{\omega V_{h}}{S_{h}}\} + (E_{h} - E_{h}^{*}).(\alpha_{p} + \alpha_{h})\frac{S_{h}}{E_{h}} + (\frac{I_{h}-I_{h}^{*}}{I_{h}})\beta_{h}.E_{h} + (\frac{V_{h}-V_{h}^{*}}{V_{h}})(f.\Pi_{h} + \gamma.S_{h}) + (\frac{R_{h}-R_{h}^{*}}{R_{h}})\rho_{h}.I_{h} + (\frac{S_{p}-S_{p}^{*}}{S_{p}}).\Pi_{p} + (\frac{E_{p}-E_{p}^{*}}{(1-f).S_{h}} + \frac{\omega V_{h}}{S_{h}}\} + (E_{h} - E_{h}^{*}).(\alpha_{p} + \alpha_{h})\frac{S_{h}}{E_{h}} + (\frac{I_{h}-I_{h}^{*}}{I_{h}})\beta_{h}.E_{h} + (\frac{V_{h}-V_{h}^{*}}{V_{h}})(f.\Pi_{h} + \gamma.S_{h}) + (\frac{R_{h}-R_{h}^{*}}{R_{h}})\rho_{h}.I_{h} + (\frac{S_{p}-S_{p}^{*}}{S_{p}}).\Pi_{p} + (\frac{E_{p}-E_{p}^{*}}{E_{p}})(\alpha_{p})S_{p} + (\frac{I_{p}-I_{p}^{*}}{I_{p}})\beta_{p}.E_{p} + (\frac{R_{p}-R_{p}^{*}}{R_{p}})\rho_{p}.I_{p} - (S_{h} - S_{h}^{*})\{(\alpha_{p} + \alpha_{h}) + A_{h}\} - (E_{h} - E_{h}^{*})\frac{(\alpha_{p} + \alpha_{h})S_{h}}{E_{h}^{*}}} + (I_{p} - I_{p}^{*})B_{5} + (R_{p} - R_{p}^{*})\mu_{p}$$

$$(94)$$

$$M = (I_{h} - I_{h}^{*})B_{2} + (V_{h} - V_{h}^{*})B_{3} + (R_{h} - R_{h}^{*})\mu_{h} + (S_{p} - S_{p}^{*})[(\alpha_{p}) + \mu_{p}] + (E_{p} - E_{p}^{*})B_{4} + (I_{p} - I_{p}^{*})B_{5} + (R_{p} - R_{p}^{*})\mu_{p}$$

From (93) if P < M then  $\frac{dG}{dt}$  will be negative definite, meaning that  $\frac{dG}{dt} < 0$ . Also it follows that  $\frac{dG}{dt} = 0$  if and only if  $S_h(t) = S_h^*, E_h(t) = E_h^*, I_h(t) = I_h^*, V_h(t) = V_h^*, R_h(t) = R_h^*, S_p(t) = S_p^*, E_p(t) = E_p^*, I_p(t) = I_p^*, R_p(t) = R_p^*$  The largest compact invariant set in  $\{S_h^*, E_h^*, I_h^*, V_h^*, R_h^*, S_p^*, E_p^*, I_p^*, R_p^*\} \in \Re^9: \frac{dG}{dt} = 0\}$  is the singleton  $\{E_1\}$  where  $E_1$  is the EE of the model equations (1)-(13). From the principle of lasalle's invariant, it implies that  $E_1$  is globally stable in  $\Re^9$  if P < M. and unstable if P > M. This completed the proof.

### **Numerical Simulations**

The calculation and estimation of the parameter values was done based on the availability of information from the Nigeria Centre for Disease Control (NCDC), Situation Report on Monkey-pox disease from 2017 to 2023 in Table 1.

Table 1: Update on Monkey-pox Disease in Nigeria from 2017 to 2023

Cases of Monkey pox	Number per year
Confirmed cases from Dec. 2017 to January 2023	988
Suspected cases from 2017 to 2023	2635
Deaths 2017–2023	15
Confirmed cases in 2017	88
Confirmed cases in 2018	49
Confirmed cases in 2019	47
Confirmed cases in 2020	8
Confirmed cases in 2021	34
Confirmed cases in February 2022	7
Confirmed cases in February 2023	762

(NCDC, 2023)



Figure 2: Graphical Representation of confirmed cases, suspected cases and death

Rashad et al.,



0 000 10 1

Figure 3: Graphical Representation of infected monkey pox individual per year

According to United Nation 2023 to 2024 report, the life expectancy for Nigerian at birth is 56.05 years. This gives the Natural Death rate as inverse of the life expectancy which  $\sin \mu_h = \frac{1}{56.05} = 0.01784$  per year. The birth rate is 38.03 births per year per 1000 people; this gives the birth rate as  $\frac{38.03}{1000} = 0.03803$ /year. However, the recruitment rate due to birth in Nigeria is  $\prod_{h} = N_h * \mu_h = 3,568,000$ . According to NCDC (2023), there were 2635 suspected cases, where total confirmed cases were at 988, resulting in 15 deaths. This implies, Recovery rate is  $\gamma_h = \frac{998-15}{998} = 0.985$ . Also, Death rate due to the disease, it is cleared that 15 people out of 988 died of the infection of monkeypox in Nigeria between 2017 to 2023, which implies,  $\delta_h = \frac{15}{998} = 0.0152$ . We have infection rate = (confirmed cases / Total Population)\*100 =

0.000494. The natural death rate of monkey, according to
Primate Info Net (PIN), the life span of monkeys in the forest
is 15-30 years, meanwhile, $\mu_m = \frac{1}{30} \text{ or } \frac{1}{15} =$
(0.033 or 0.067) is the Natural Death rate of Monkey. According to Pandrillus foundation (2008), it is about 8,000
drill monkey found in cross river state of Nigeria. However, 50,000 monkeys are estimated for Nigeria, hence, the
recruitment rate of monkeys is given by $\prod_{m} = N_m * \mu_m =$
1,665. The vaccination rate of monkey pox infection is
10.1%, so fraction of vaccination against monkey pox (f) is
$f = \frac{10.1}{100} = 0.0101$ . Vaccination last upto 3-5 years and can
also protect around 85% from monkey pox. So, $\varphi_1 = \frac{1}{5} =$
0.2 or $\frac{1}{10} = 0.1$ and $\varphi_2 = \frac{1}{85} = 0.012$ . Other unavailable
data have been assumed in the simulations.

Table	e 2: S	ymbols	and l	Parameters	Va	lues of	' the	e Model	
-------	--------	--------	-------	------------	----	---------	-------	---------	--

Table 2: Symbols and Parameters values of the Model							
Parameter	Definition	Value	Source				
$\beta_h$	Exposed Rate of Human	0.005	Assumed				
$\alpha_h$	Infection Rate of Human	0.000494	Table 1				
$\Lambda_h$	Recruitment Rate of Human	3,568,000	Table 1				
Υh	Recovery Rate of Human	0.985	Table 1				
$\mu_h$	Natural Death Rate of Human	0.01784	Table 1				
$\delta_h$	Death Rate Due to Disease	0.0152	Table 1				
$\varphi_1$	Loss of Vaccination Rate	(0.1 - 0.2)/year	Table 1				
$\varphi_2$	Vaccination Rate	0.012/year	Table 1				
f	Proportion of vaccinated h immigrants	0.0101	Table 1				
Ø	Effectiveness of Vaccination Drug	[0 - 1]	Assumed				
$\alpha_m$	Infection Rate of Monkey	0.004	Table 1				
Υm	Recovery Rate of Monkey	0.50	Assumed				
$\Lambda_m$	Recruitment Rate of Monkey	1,665	Table 1				
$\theta_{m2}$	Exposed Rate of Monkey	0.003	Assumed				
$\mu_m$	Natural Death of Monkey	(0.033 - 0.067)	Table 1				
$\delta_m$	Death Due to infection of monkey	0.020	Assumed				
$ au_h$	Exposed rate for human transmits lower than symptomatic humans	0.010	Assumed				
$ au_m$	Exposed rate for non-human transmits lower than symptomatic non-	0.010	Assumed				
	humans						
N <sub>h</sub>	Nigeria Population	200,000,000	Table 1				
$N_m$	Population of Monkey in Nigeria	50,000	Table 1				
T1 / 1 1		1 1 1 1 1 0 0	C 1 1				

The study used table 2 to simulate our model system with equation (45) and (48) by using Maple 17 Software for the graphic representation of the reproduction numbers,



Graphical Representation of the Model Equations for Monkey pox Disease

Figure 4: Effect of exposed rate on the effective reproduction number.

It was observed in figure 4 that rate of exposed increases with losing out of vaccination which leads to increases in effective reproduction number (secondary infection cases). This

implies that, the two parameters increase the infection of monkey-pox disease in Nigeria.



Figure 5: Infection rate effect on effective reproduction number  $(R_e)$ 

Figure 5 shows that as the infection rate increases, then effective reproduction number also increases, varying the rate of recovery of human, it is observed that as recovery individual increases, the infected individual decreases from

the monkey-pox in Nigeria because the effective reproduction number decreases. This means that as more people are recovery from the monkey-pox disease, the secondary infection cases decreases.



Figure 6: Effect of Loss of vaccination on the  $R_e$ 

It is observed from figure 6 that as loss of vaccination increases, it increases  $R_e$  number. Also, Low recovery rate increases the  $R_e$  number of the infection.



Figure 7: Effect of Vaccination on the  $R_e$  number of human

Figure 7 shows that as vaccination rate decreases, it also decreases  $R_e$ , by varying the rate of death due to monkeypox, it is observed that effective reproduction number decreases as rate of death due to infection increases. This means that the higher the death due to the monkey-pox infection the lower the  $R_e$ . This is implies as more individuals die due to the monkey-pox, there will be less to be infected by monkey-pox.



Figure 8: Infection rate effect of monkey pox on  $R_p$  of monkey (non-human)

It is shown in figure 8 that as rate of infection for non-human increases, it also increases  $R_e$ . Varying the rate of recovery, we found that the effective reproduction number decreases as recovery rate increases



Figure 9: Recovery rate effect of monkey pox on the  $R_p$  of Monkey

Figure 9 shows the simulation of recovery rate of non-human with effective reproduction number of non-human, it is observed that effective reproduction number of non-human decreases as recovery rate increases.



Figure 10: Effect of exposed rate of Monkey on the  $R_p$  of Monkey

Figure 10 shows the simulation of exposed rate of non-human ind with effective reproduction number of non-human, it is observed that  $R_p$  of non-human increases as exposed tran

individual of non-human increases. It is also found that the  $R_p$  decreases due to the decreases in exposed of non-human transmit at a rate lower than symptomatic non-humans.



Figure 11: Effect of Vaccination on the  $R_e$ 

We found in figure 11, that  $R_e$  decreases as vaccination of obsindividual increases, varying the public awareness, we are

observed that effective reproduction number reduces to zero and becomes stable as public awareness increases.



Figure 12: Effect of Loss of vaccination on the effective reproduction number  $(R_e)$ , varying public

#### Awareness parameter

It is observed from figure 12 that, low loss of vaccination rate increases the  $R_e$ , it is also shown that, the  $R_e$  approaches zero and becomes stable as public awareness increases.



Figure 13: Effect of Public Awareness on the effective reproduction number

In figure 13, we observed that effective reproduction number decreases as public awareness increases, varying loss of vaccination rate, it is observed that as loss of vaccination rate decreases, it decreases the secondary cases of the infection.

#### ACKNOWLEDGMENT

The authors acknowledgment Tertiary Education Trust Fund (TeTfund) for funding this research with a Grant Number TETF/DR&D/CE/POLY/KAURA NAMODA/IBR/2023/VOL.1

### CONCLUSION

This research work have simulated and analyzed a mathematical model of monkey-pox transmission disease, the study incorporated Vaccination and public awareness in the model. The analyses of the model showed invariant region and dynamics of model equations is in the region  $\Omega$ , the model was considered been mathematically and epidemiologically well posed. The non-negativity of the solutions for the model implies that the solutions were non-negative and remains in  $\mathbb{R}^9$ . The global stability of the DFE was done and it was found that when there is no transmission between human and nonhuman  $(\alpha_h)$ , then  $\hat{G}(X_1, X_2) = 0$  implies, that the DFE would be asymptotically globally stable. The endemic equilibrium state was obtained and we found that the endemic equilibrium state was globally asymptotically stable if P < M and unstable if P > M by comparison method of lyapunov functions. Simulations of the model equations on effective reproduction number were done and the study found that the exposed rate increases with the loss of vaccination which leads to increases in secondary infection cases of the monkey-pox. It is found that as recovery individual increases, the infected individual decreases from the monkey-pox in Nigeria, it implies, that as more people recovered from the monkey-pox disease, the secondary infection cases decreases. It was also found that, low recovery rate increases the  $R_e$  of the infection. The study shown that as vaccination rate decreases, it decreases the  $R_e$ . It was observed that  $R_e$  decreases as death rate due to infection increases, this implies that the higher the death due to the monkey-pox infection the lower  $R_e$  would be. This is because as more people die due to the monkey-pox, there will be less to be infected of monkey-pox virus. The study noted that  $R_e$  decreases as vaccination of individual increases, varying the public awareness it was found that effective reproduction number  $(R_e)$  reduces to zero and becomes stable as public awareness increases. the study discovered that effective reproduction number decreases as public awareness increases.

#### REFERENCES

Bauch, C. and Earn, D. (2003). Transients and attractors in epidemics. *Proceedings of the Royal Society of London Series* A. 270:1573 - 1578.

Bhunu, C. P. and Mushayabase, S. (2011). Modeling the Transmission Dynamics of Pox-like Infections. *International Journal of Applied Mathematics*. 41: 2 - 09

Castillo-Chavez, C., Blower, S., Van den Driessche, P., Krirschner, D. and Yakubu, A. A. (2002), Mathematical approaches for emerging and re-emerging infectious diseases: An introduction. The IMA Volumes in Mathematics and its Applications, 125: 229-250. New York: Springer-Verlag

NCDC (2023). Official statement to the news on Covid-19 Resurgence in Benue State, December 14, 2023, www.https://ncdc.gov.ng/new/505/the-nigeria-centre-fordisease-control-and-prevention-launches-its-5-year-%282023-2027%29-stategic-pan

Centres for Disease Control (2003). "What You Should Know About Monkeypox". *Fact Sheet. Centres for disease control and prevention*. Retrieved 2016-03-21.

Esbauer, S., Pfeffer, M., and Meyer, H. (2010). Zoononic poxvirues. *Vet. Microbiol.* 140:229-36, DOI:10.1016/j.vetmic Jezek A., Marennikova S. S., Mutumbo M., *et al.* (1986). Human monkeypox: a study of 2510 contacts of 214 patients. Journal of Infectious Diseases, Vol. 154, 1986, p.551-555.

Kantele A., Chickering K., Vapalahti O., *et al.* (2016)."Emerging diseases—the monkeypox epidemic in the Democratic Republic of the Congo". Clinical Microbiology and Infection, Vol. 22, Issues 8, 2016, p. 658–659.

Lasisi N. O., Akinwande N. I., Olayiwola R. O., *et al.* (2018). Mathematical Model for Ebola Virus Infection In Human With Effectiveness Of Drug Usage. J. Appl. Sci. Environ. Manage, Vol. 22, Issues 7, 2018, p. 1089 – 1095. DOI: https://dx.doi.org/10.4314/jasem.v22i7.16. http://www.bioline.org.br/ja or https://www.ajol.info/index.php/jasem

Lasisi, N. O. and Suleiman, F. (2024). Effects of Poor Sanitation and Public Awareness in Modeling Bacterial Infection amongst the Students of a Tertiary Institution in KauraNamoda, Zamfara State, Nigeria. *J. Appl. Sci. Environ. Manage.* Vol. 28 (4) 1177-1185 April 2024. DOI: https://dx.doi.org/10.4314/jasem.v28i4.17

Lasisi, N. O. (2021). Lyapunov Approach and Global Stability Of Ebola Virus Infection Model Of An Individual Cells Population, *Material Science, Engineering And Applications*, Vol. 1, Issue 1, p. 1-10. *DoiHttps://Doi.Org/10.21595/Msea.2021.21977* 

Lasisi, N. O. and Adeyemo, K. A. (2021). Modelling the Effect of Distancing and Wearing of Face Masks on Transmission of Covid-19 Infection Dynamics. *Journal of Complexity in Health Sciences*, Vol. 4, Issue 1, p. 10-20. *DOI https://doi.org/10.21595/chs.2021.21976* 

Lasisi, N. O., Akinwande, N. I., and Abdulrahaman, S. (2020). Optimal Control and Effect of Poor Sanitation on Modeling the Acute Diarrhea Infection. *Journal of Complexity in Health Sciences*, Vol. 3, Issue 1, p. 91-103. *DOI https://doi.org/10.21595/chs.2020.21409* 

Lasisi, N. O. (2020). Effect of public awareness, behaviors and treatment on infection-age-structured of mathematical model for HIV/AIDS dynamics. *Journal of Mathematical Models in Engineering*, Vol. 6, Issue 2, p. 103-121.DOI <u>https://doi.org/10.21595/mme.2020.21249</u>

Lasisi, N. O., Akinwande, N. I. and Oguntolu, F. A. (2020). Development and exploration of a Mathematical Model for Transmission of Monkey-Pox in Humans. *Journal of Mathematical Models in Engineering*, Vol. 6, Issue 1, p. 23-33. https://doi.org/10.21595/mme.2019.21234

Lasisi, N. O., Akinwande, N. I., Olayiwola, R. O., Cole, A. T. and Abdulrahman, S. (2018). Global Stability of Virus Persistence of a Mathematical Model of the Dynamics of Ebola Virus Infection in Human Cell Population. *Journal of the Nigeria Association of Mathematical Physics*, 45; 83-90.

Lasisi, N. O., Akinwande, N. I., Olayiwola, R. O., and Cole, A. T. (2018). Mathematical Model for Ebola Virus Infection in Human with Effectiveness of Drug Usage. *Journal of Applied Science Environmental Management*, 22(7); 1089-1095. https://dx.doi.org/10.4314/jasem.v22i7.16

Lasisi N. O. and Suleiman F. (2024). Global Stability Analysis of Disease Free Equilibrium for Modeling the Dynamics of Bacteria Infection in Higher Institution KauraNamoda, Nigeria. *Bima Journal of Science and Technolog*.Vol. 8(2), 160-171. Doi:10.56892/bima.v8i2.674

Pandrillus foundation (2008). Adult male and female drill monkey at Drill Ranch, ross River State, Nigeria. www.pandrillus.org

Rimoin A. W., Kisalu N., Kebela-Ilungam B., *et al.* (2007). Endemic human monkey pox, Democratic Republic of Congo, 2001-2004. Emerg. Infect. Dis. Vol. 3, Issues 6, 2007, p. 934-936.

Van den Driessche, P. and Watmough, J. (2002) Reproduction numbers and sub-threshold endemic equilibrium for compartmental models of disease transmission. *Math Biosci*. 180:29-48, DOI:10.1016/s0025-5564(02)00108-6.

Von Magnus P., Andersen E. K., Petersen K. B., *et al.* (1959). A pox like disease in cynomolgus monkeys. Acta Pathol Microbiol Scand., Vol. 46, Issues 1, p. 156-176.



©2024 This is an Open Access article distributed under the terms of the Creative Commons Attribution 4.0 International license viewed via <u>https://creativecommons.org/licenses/by/4.0/</u> which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is cited appropriately.