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Development and Simulations of a Mathematical Model for Monkey-Pox Transmission Disease in Nigeria

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ABSTRACT: Monkey pox causes a rash which can be uncomfortable, itchy, and painful and its early detection is vital to every control mechanisms. Hence, the objective of this paper was the development and simulation of a mathematical model for monkey-pox transmission disease in Nigeria using Ordinary Differential Equations. The feasible region of the model was verified and solutions positivity was shown. We achieved the disease free equilibrium and computed effective reproduction number, R_e of the model system. We show the global stability of disease free equilibrium and we found that the disease free equilibrium of the model system is globally asymptotically stable if Re < 1 and $\bar{G}(X_1, X_2) = 0$. The model system is considered mathematically and epidemiologically well posed. Furthermore, the simulations of the model shows that the average secondary cases of disease increases as exposed individual increases and rate of infection increases. Again, the effective reproduction number reduces as vaccination increases and it is observed that as exposed nonhuman transmits at low rate than symptomatic reduced, it reduces the secondary cases of the disease.

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Monkey-pox disease is recognized as pathogens, disturbing animals and humans, it is among the family of orthopox virus, other families are small pox and cow pox viruses (Essbauer *et al.*, 2009). The disease causes lymph nodes to swell. The backache, fever, muscle aches, headache and swollen lymph nodes are all the symptoms. The virus can is transmitted from non-human to human and as well as human to human. The incubation period is from 7-14 days. The appearance of fever displayed after 1-3 days and the infection lasted for 2-4 weeks (Essbauer *et al.*, 2009). Persons who have had close contact with individuals confirmed to have disease must be vaccinated for 14days after exposure (CDC, 2003a, 2003b). Monkey-pox (MPX) epidemic was first discovered in 1958 (Magnus, *et al.*, 1959). The disease was reported in humans in 1970 (Breman *et al.*, 1986). The incubation time is 7–14 days, the disease lasted for 2 to 4 weeks (Centres for Disease Control, 2003) and the fatality ratio is 1% to 10% (Rimoin, *et al.*, 2007). Monkey pox is endemic in Nigeria and the infection has been conveyed in numerous countries in Africa, including

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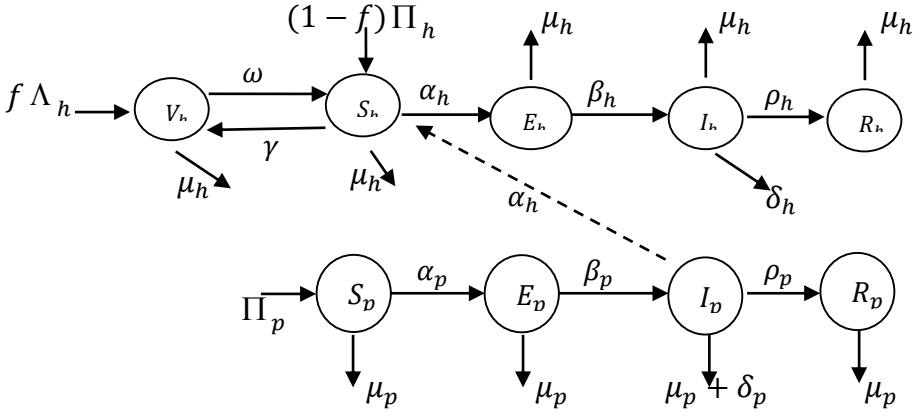
Nigeria. From the beginning of the outbreak in 2017 to 2018, there were 269 suspected cases in 25 states and one case in territory with the confirmed cases of 115 in 16 states were reported in Nigeria. 7 deaths were verified plus 4 in patients with pre-existing immune compromised situations. In 2018, a total of 76 were reported and 37 were confirmed and two deaths were recorded. Nigeria reported a total number of 558 in April 2022, as suspected cases, of which confirmed cases were 231. In 2022, Nigeria reported 46 suspected cases and no deaths recorded. 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).

Mathematical models have been helpful in gaining insight of transmission of disease (Lasisi, *et al.*, 2018; Lasisi, and Adeyemo, 2021; Lasisi and Fahad, 2024; Lasisi and Suleiman, 2024). The focus of this work is on global stability for mathematical model of transmission of monkey-pox infection and effects of public awareness and vaccination in Nigeria. Therefore, a mathematical modeling for monkey pox disease was developed by (Lasisi, *et al.*, 2011) with six (6) compartments. In this work, we therefore complement and extend the work of the aforementioned authors by having nine (9) compartments. The objective of this paper is the development and simulation of a mathematical model for monkey-pox transmission disease in Nigeria using Ordinary Differential Equations.

MATERIALS AND METHODS

Formulation of a model for the monkey pox disease in nonhuman and human population was done in this research, we have population size of both human and nonhuman as $N_h(t)$ and $N_p(t)$. The populations are compartmentalized into classes in Figure 1. The human population is subdivided into five class such as, susceptible, $S_h(t)$, vaccination class, $V_h(t)$, exposed class, $E_h(t)$, infected class, $I_h(t)$, and recovery class, $R_h(t)$. The total nonhuman population model subdivided into susceptible class, $S_p(t)$, infected class, $I_p(t)$, exposed, $E_p(t)$ and recovery, $R_p(t)$. As showed in the in Figure 1, individuals come into susceptible class through immigration and birth (Π_h), the proportion of vaccinated human immigrants (f) come into vaccinated class and proportion of unvaccinated immigrants (1-f) come into the susceptible class. The work does not consider the immigration of infection individual, because we assumed they have be vaccinated. The susceptible persons vaccinated at rate of γ and loss the vaccination at the rate of ω. Contact of susceptible human from primate is at the rate σ_{p_1}, S_h are exposed to disease at the rate of λ_h and infected at the rate of β_h, the natural death is μ_h and die due to the disease is at the rate of δ_h and recovery at a rate of ρ_h. The susceptible nonhuman (primates), S_p is generated from the daily recruitment of persons through births at the Π_p and natural death rate of μ_p. Individuals become exposed to the virus at the rate of λ_p and move to the infected class at the rate of β_p. Nonhuman (primate) infected die due to the disease at the rate of δ_p and recovery at the rate of ρ_p. Figure 1 is the flowchart representation of the 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 1 to 11.

$$\frac{dS_h}{dt} = (1-f)\Pi_h + \omega V_h - \gamma S_h - \alpha_h S_h - \mu_h S_h \quad (1)$$

$$\frac{dE_h}{dt} = \lambda_h S_h - \beta_h E_h - \mu_h E_h \quad (2)$$

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

$$\frac{dV_h}{dt} = f\Pi_h + \gamma S_h - \omega V_h - \mu_h V_h \quad (4)$$

$$\frac{dR_h}{dt} = \rho_h I_h - \mu_h R_h \quad (5)$$

$$\frac{dS_p}{dt} = \Pi_p - \alpha_p S_p - \mu_p S_p \quad (6)$$

$$\frac{dE_p}{dt} = \alpha_p S_p - \beta_p E_p - \mu_p E_p \quad (7)$$

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

$$\frac{dR_p}{dt} = \rho_p I_p - \mu_p R_p \quad (9)$$

Where,

$\lambda_h = (\frac{\sigma_{p_1}(\varepsilon_p E_p + I_p)}{N_p} + \frac{\sigma_h(\varepsilon_h E_h + I_k)}{N_h})$ and

$\lambda_p = (\frac{\sigma_{p_2}(\varepsilon_p E_p + I_p)}{N_p})$

$N_h = S_h + E_h + I_h + V_h + R_h \quad (10)$

$N_p = S_p + E_p + I_p + R_p \quad (11)$

S_h Becomes infected from both I_p and I_h. σ_{p1} is effective contact product rate and probability of S_h becomes infected from I_p and σ_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 ε_h is the assumption that exposed human transmits at a rate lower than symptomatic humans. The adjustment parameter ε_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 of the Model Equations:

Theorem 1: The following biological feasible region of the model equations (1) - (9)

$\Omega = \{S_h, E_h, I_h, V_h, R_h, S_p, E_p, I_p, R_p) \in \Re_+^9 :$ $\{S_h + E_h + I_h + V_h + R_h \leq \frac{\Pi_h}{\mu_h}; S_p + E_p + I_p + R_p \leq \frac{\Pi_p}{\mu_p}\}$ is attracting and positively invariant.

Proof: Adding all the model equations in (1) - (9), we get

$\frac{dN_h}{dt} = \Pi_h - \mu_h N_h - \delta_h I_h$

And $\frac{dN_p}{dt} = \Pi_p - \mu_p N_p - \delta_p I_p$

So that $\frac{dN_h}{dt} \leq \Pi_h - \mu_h N_h$ and $\frac{dN_p}{dt} \leq \Pi_p - \mu_p N_p \quad (12)$

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

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

And $N_p(t) \leq N_p(0)e^{-\mu_p(t)} + \frac{\Pi_p}{\mu_p}\{1 - e^{-\mu_p(t)}\}$ (13)

In specific, $N_h \leq \frac{\Pi_h}{\mu_h}$, if only $N_h(0) \leq \frac{\Pi_h}{\mu_h}$, also $N_p \leq \frac{\Pi_p}{\mu_p}$, if only $N_p(0) \leq \frac{\Pi_p}{\mu_p}$. And Ω is positively invariant. Therefore, it is enough to consider the model equations dynamics (1) - (9) in Ω. 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 $S_h(0) > 0, E_h(0) > 0, I_h(0) > 0, V_h(0) > 0, R_h(0) > 0, S_p(0) > 0, E_p(0) > 0, I_p(0) > 0, R_p(0) > 0$, then the solutions $S_h(t), E_h(t), I_h(t), V_h(t), R_h(t), S_p(t), E_p(t), I_p(t), R_p(t)$ of the model equations (1)-(9) are nonnegative for all $t \geq 0$.

Proof:

Proving that for all $t \in [0, t_0]$, $S_h(t), E_h(t), I_h(t), V_h(t), R_h(t), S_p(t), E_p(t), I_p(t), R_p(t)$ will be nonnegative in $\Re_+^9$, Since all the parameters used in the system are positive. Thus, it is clear from equation (1) that

$\frac{dS_h}{dt} = (1 - f)\Pi_h + \omega V - \gamma S_h - \alpha_h S_h - \mu_h S_h \geq -(\gamma + \alpha_h + \mu_h)S_h$

So that,

$S_h(t) \geq S_h(0)e^{\{-\int(\gamma + \alpha_h + \mu_h)dt\}} \quad (14)$

The similar approach can be used to show that $E_h(t) > 0, I_h(t) > 0, V_h(t) > 0, R_h(t) > 0, S_p(t) > 0, E_p(t) > 0, I_p(t) > 0, R_p(t) > 0$. Thus, for all $t \in [0, t_0]$, $S_h(t), E_h(t), I_h(t), V_h(t), R_h(t), S_p(t), E_p(t), I_p(t), R_p(t)$ will be nonnegative and remain in $\Re_+^9$

The Equilibrium State: At equilibrium point, we setting the model equations to zero, we have

$\frac{dS_h}{dt} = \frac{dE_h}{dt} = \frac{dI_h}{dt} = \frac{dV_h}{dt} = \frac{dR_h}{dt} = \frac{dS_p}{dt} = \frac{dE_p}{dt} = \frac{dI_p}{dt} = \frac{dR_p}{dt} = 0$ (15)

From (3), we have

$E_h = \frac{(\rho_h+\mu_h+\delta_h)I_h}{\beta_h}$ (16)

Substitute (16) into (2), we have

$I_h\left(\frac{(\sigma_h\varepsilon_h(\rho_h+\mu_h+\delta_h)+\sigma_h\beta_h)S_h}{\beta_h N_h} - \frac{(\beta_h+\mu_h)(\rho_h+\mu_h+\delta_h)}{\beta_h}\right) = 0$ (17)

Equation (17) gives,

$I_h = 0$ Or $\left(\frac{(\sigma_h\varepsilon_h(\rho_h+\mu_h+\delta_h)+\sigma_h\beta_h)S_h}{\beta_h N_h} - \frac{(\beta_h+\mu_h)(\rho_h+\mu_h+\delta_h)}{\beta_h}\right) = 0$ (18)

From (8), we have

$E_p = \frac{(\rho_p+\mu_p+\delta_p)I_p}{\beta_p}$ (19)

Substitute (19) into (7), we have

$I_p\left(\frac{(\sigma_{p2}\varepsilon_p(\rho_p+\mu_p+\delta_p)+\sigma_p\beta_p)S_p}{\beta_p N_p} - \frac{(\beta_p+\mu_p)(\rho_p+\mu_p+\delta_p)}{\beta_p}\right) = 0$ (20)

Equation (20) gives

$I_p = 0$ or $\frac{(\sigma_{p2}\varepsilon_p(\rho_p+\mu_p+\delta_p)+\sigma_p\beta_p)S_p}{\beta_p N_p} - \frac{(\beta_p+\mu_p)(\rho_p+\mu_p+\delta_p)}{\beta_p} = 0$ (21)

Substitute $I_h = 0$ in (9) into (16) and (5), we have

$E_h = R_h = 0$ (22)

Find $V_{.h}$ in (4) and (1), and equating them, we get

$V_h = \frac{f.\Pi_h+\gamma.S_h}{(\omega+\mu_h)} = \frac{\gamma S_h+\alpha_h S_h+\mu_h S_h-(1-f)\Pi_h}{\omega}$ (23)

Implies,

$(f\Pi_h + \gamma S_h)\,\omega = [\gamma S_h + \alpha_h S_h + \mu_h S_h - (1 - f)\Pi_h]\,(\omega + \mu_h)$

Since $I_h = 0$, then $\alpha_h = 0$, and we have

$S_h = \frac{f\omega\Pi_h+[\Pi_{.h}\omega+\Pi_h\mu_h-f\Pi_h\omega-f\Pi_h\mu_h]}{\gamma\omega+\gamma\mu_h+\mu_h\omega+{\mu_h}^2-\gamma\omega}$ (24)

Reduced to

$S_h{}^0 = \frac{\Pi_h\omega+\Pi_h\mu_h-f\Pi_h\mu_h}{\gamma\mu_h+\mu_h\omega+{\mu_h}^2}$ (25)

If the absence of vaccination, then $S_h{}^0 = \frac{\Pi_h}{\mu_h}$

Putting equation (25) into (4), we get

$V_h^0 = \frac{f\Pi_h\mu_h\omega+f\Pi_h{\mu_h}^2+\gamma\Pi_h\omega+\gamma\Pi_h\mu_h}{(\gamma\mu_h+\mu_h\omega+{\mu_h}^2)(\omega+\mu_h)}$ (26)

Substitute $I_p = 0$ in (21) into (19) and (9), we have

$E_p = R_p = 0$ (27)

From (6), we have

$S_p^0 = \frac{\Pi_p}{\mu_p}$ (28)

The DFE state is derived below,

$E_0 = \{S_h^*,\ E_h^*,\ V_h^*,\ I_h^*,\ R_h^*,\ S_p^*,\ E_p^*,\ I_p^*,\ R_p^*\}$

$= \{\frac{\Pi_h\omega+\Pi_h\mu_h-f\Pi_h\mu_h}{\gamma\mu_h+\mu_h\omega+{\mu_h}^2}, 0, \frac{f\Pi_h\mu_h\omega+f\Pi_h{\mu_h}^2+\gamma\Pi_h\omega+\gamma\Pi_h\mu_h}{(\gamma\mu_h+\mu_h\omega+{\mu_h}^2)(\omega+\mu_h)}, 0,\ 0,\ \frac{\Pi_p}{\mu_p}, 0, 0, 0\}$ (29)

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}

$FV^{-1} = \left[\frac{\partial F_i(E^0)}{\partial x_i}\right]\left[\frac{\partial V_i(E^0)}{\partial x_i}\right]^{-1}$ (30)

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 (ρ) 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

$$f = \begin{pmatrix} f_1 \\ f_2 \\ f_3 \\ f_4 \end{pmatrix} = \begin{pmatrix} \left(\frac{\sigma_{p1}(\varepsilon_p E_p+I_p)}{N_p} + \frac{\sigma_h(\varepsilon_h E_h+I_h)}{N_h}\right)S_h \\ \beta_h E_h \\ \frac{\sigma_{p2}(\varepsilon_p E_p+I_p)S_P}{N_p} \\ \beta_p E_p \end{pmatrix} \quad (31)$$

$$F = \begin{bmatrix} \frac{\sigma_h\varepsilon_h S_h^0}{N_h^0} & \frac{\sigma_h S_h^0}{N_h^0} & \frac{\sigma_{p_1}\varepsilon_p S_h^0}{N_p^0} & \frac{\sigma_{p_1} S_h^o}{N_p^0} \\ \beta_h & 0 & 0 & 0 \\ 0 & 0. & \frac{\sigma_{p_2}\varepsilon_p S_p^0}{N_p^0} & \frac{\sigma_{p_2} S_p^0}{N_p^0} \\ o & 0 & \beta_p & 0 \end{bmatrix} \quad (32)$$

$$v = \begin{pmatrix} v_1 \\ v_2 \\ v_3 \\ v_4 \end{pmatrix} = \begin{bmatrix} A_1E_h \\ A_2I_h \\ A_3E_p \\ A_4I_p \end{bmatrix} \quad (33)$$

Where, $A_1 = \beta_h + \mu_h$; $A_2 = \rho_h + \mu_h + \delta_h$; $A_3 = \beta_p + \mu_p$; $A_4 = \rho_p + \mu_p + \delta_p$

$$V = \begin{pmatrix} A_1 & 0 & 0 & 0 \\ 0 & A_2 & 0 & 0 \\ 0 & 0 & A_3 & 0 \\ 0 & 0 & 0 & A_4 \end{pmatrix} \quad (34)$$

From (34), we have

$$V^{-1} = \begin{bmatrix} \frac{1}{A_1} & 0 & 0 & 0 \\ 0 & \frac{1}{A_2} & 0 & 0 \\ 0 & 0 & \frac{1}{A_3} & 0 \\ 0 & 0 & 0. & \frac{1.}{A_4} \end{bmatrix} \quad (35)$$

At disease free equilibrium point, and since $N_h \leq \frac{\Pi h}{\mu_{h.}}$ and $N_p \leq \frac{\Pi p.}{\mu_{p.}}$ we get

$$F = \begin{bmatrix} \frac{\sigma_h.\varepsilon_h.\mu_h.S_h^0}{\Pi_h} & \frac{\sigma_h.\mu_h.S_h^0}{\Pi_h} & \frac{\sigma_{p_1}.\varepsilon_p.\mu_p.S_h^0}{\Pi_p} & \frac{\sigma_{p_1}.\mu_p.S_h^0}{\Pi_p} \\ \beta_h & 0 & 0 & 0 \\ 0 & 0 & \frac{\sigma_{p_2}.\varepsilon_p.\mu_p.S_p^0}{\Pi_p} & \frac{\sigma_{p_2}.\mu_p.S_p^0}{\Pi_p} \\ 0 & 0 & \beta_{p.} & 0 \end{bmatrix} \quad (36)$$

from (36), we get

$$F = \begin{bmatrix} \frac{\sigma_h.\varepsilon_h.\mu_h.S_h^0}{\Pi_h} & \frac{\sigma_h.\mu_h.S_h^0}{\Pi_h} & \frac{\sigma_{p_1}.\varepsilon_p.\mu_p.S_h^0}{\Pi_p} & \frac{\sigma_{p_1}.\mu_p.S_h^0}{\Pi_p} \\ \beta_h & 0 & 0 & 0 \\ 0 & 0 & \sigma_{p_2}.\varepsilon_p & \sigma_{p_2} \\ 0 & 0 & \beta_p & 0 \end{bmatrix} \quad (37)$$

$$FV^{-1} = \left[\frac{\partial F_i(E^0)}{\partial x_j}\right] \left[\frac{\partial V_i(E^0)}{\partial x_j}\right]^{-1} \quad (38)$$

Multiplying (37) and (35) together, we have

$$FV^{-1} = \begin{bmatrix} \frac{\sigma_h\varepsilon_h\mu_h S_h^0}{\Pi_h} & \frac{\sigma_h\mu_h S_h^0}{\Pi_h} & \frac{\sigma_{p_1}\varepsilon_p\mu_p S_h^0}{\Pi_p} & \frac{\sigma_{p_1}\mu_p S_h^0}{\Pi_p} \\ \beta_h & 0 & 0 & 0 \\ 0 & 0. & \sigma_{p_2}.\varepsilon_p & \sigma_{p_2} \\ 0 & 0. & \beta_p & 0 \end{bmatrix} \begin{bmatrix} \frac{1}{A_1} & 0 & 0 & 0 \\ 0 & \frac{1}{A_2} & 0 & 0 \\ 0 & 0 & \frac{1}{A_3} & 0 \\ 0 & 0 & 0 & \frac{1}{A_4} \end{bmatrix} \quad (39)$$

Equation (39) implies,

$$FV^{-1} = \begin{bmatrix} \frac{\sigma_h.\varepsilon_h.\mu_h.S_h^0}{\Pi_h A_1} & \frac{\sigma_h.\mu_h.S_h^0}{\Pi_h A_2} & \frac{\sigma_{p_1}.\varepsilon_p.\mu_p.S_h^0}{\Pi_p A_3} & \frac{\sigma_{p_1}.\mu_p.S_h^0}{\Pi_p A_4} \\ \frac{\beta_h}{A_1} & 0 & 0 & 0 \\ 0 & 0 & \frac{\sigma_{p_2}\varepsilon_p}{A_3} & \frac{\sigma_{p_2}}{A_4} \\ 0 & 0 & \frac{\beta_p}{A_3} & 0 \end{bmatrix} \quad (40)$$

Characteristics equation of (40), gives $/FV^{-1} - \lambda I/ = 0$

$$\begin{bmatrix} K_1.S_h^0 - \lambda & K_2.S_h^0 & K_3.S_h^0 & K_4.S_h^0 \\ K_5 & -\lambda & 0 & 0 \\ 0 & 0 & K_6 - \lambda & K_7 \\ 0 & 0 & K_8 & -\lambda \end{bmatrix} = 0 \quad (41)$$

Since $K_1 = \frac{\sigma_h \varepsilon_h \mu_h S_h^0}{\Pi_h A_1}, K_2 = \frac{\sigma_h \mu_h}{\Pi_h A_2}, K_3 = \frac{\sigma_{p_1} \varepsilon_p \mu_p}{\Pi_p A_3}, K_4 = \frac{\sigma_{p_1} \mu_p}{\Pi_p A_4}$

$$K_5 = \frac{\beta_h}{A_1}, K_6 = \frac{\sigma_{p_2} \varepsilon_p}{A_3}, K_7 = \frac{\sigma_{p_2}}{A_4}, K_8 = \frac{\beta_p}{A_3} \quad (42)$$

Determinant of (41) gives

$$(\lambda^2 - K_6\lambda - K_7K_8) = 0 \text{ or } (\lambda^2 - K_1S_h^0\lambda - K_2K_5S_h^0) = 0 \quad (43)$$

To solve (43), we get

$$\lambda_1 = \frac{K_6 \pm \sqrt{K_6^2 + 4K_7K_8}}{2}$$

$$\lambda_1 = \frac{\frac{\sigma_{p_2}.\varepsilon_p}{(\beta_p + \mu_p)} \pm \sqrt{\frac{(\sigma_{p_2}.\varepsilon_p)^2}{(\beta_p + \mu_p)^2} + \frac{4.\sigma_{p_2}.\beta_p}{(\beta_p + \mu_p).(\rho_p + \mu_p + \delta_p)}}}{2} \quad (44)$$

The reproduction number is given below,

$$R_p = \frac{\frac{\sigma_{p_2}.\varepsilon_p}{(\beta_p + \mu_p)} + \sqrt{\frac{(\sigma_{p_2}.\varepsilon_p)^2}{(\beta_p + \mu_p)^2} + \frac{4.\sigma_{p_2}.\beta_p}{(\beta_p + \mu_p).(\rho_p + \mu_p + \delta_p)}}}{2} \quad (45)$$

And

$$(\lambda^2 - K_1S_h^0\lambda - K_2K_5S_h^0) = 0$$

Implies, $\lambda_2 = \frac{K_1S_h^0 \pm \sqrt{K_1^2S_h^{0^2} + 4K_2K_5S_h^0}}{2}$ (46)

$$\lambda_2 = \frac{\frac{\sigma_h.\varepsilon_h.\mu_h.S_h^0}{\Pi_h.(\beta_h + \mu_h)} \pm \sqrt{\frac{\sigma_h^{\ 2}.\varepsilon_h^{\ 2}.\mu_h^{\ 2}.S_h^{0^2}}{\Pi_h^2(\beta_h + \mu_h)^2} + \frac{4.\sigma_h.\mu_h.\beta_h.S_h^0}{\Pi_h.(\beta_h + \mu_h)(\rho_h + \mu_h + \delta_h)}}}{2} \quad (47)$$

λ_2 is the spectral radius of $\rho(FV^{-1})$

$$R_h = \frac{\frac{\sigma_h.\varepsilon_h.\mu_h.S_h^0}{\Pi_h(\beta_h + \mu_h)} + \sqrt{\frac{\sigma_h^{\ 2}.\varepsilon_h^{\ 2}.\mu_h^{\ 2}.S_h^{0^2}}{\Pi_h^2(\beta_h + \mu_h)^2} + \frac{4.\sigma_h.\mu_h.\beta_h.S_h^0}{\Pi_h(\beta_h + \mu_h).(\rho_h + \mu_h + \delta_h)}}}{2} \quad (48)$$

Hence, the effective reproduction number can be represented as,

$$R_0 = R_h + R_p \quad (49)$$

$$R_0 = \frac{\left(\begin{array}{l} \dfrac{\sigma_h.\varepsilon_h.\mu_h.S_h^0}{\Pi_h(\beta_h+\mu_h)} + \dfrac{\sigma_{p_2}.\varepsilon_p}{(\beta_p+\mu_p)} + \\ \sqrt{\dfrac{\sigma_h{}^2.\varepsilon_h{}^2.\mu_h{}^2.S_h^{0^2}}{\Pi_h^2(\beta_h+\mu_h.)^2} + \dfrac{4.\sigma_h.\mu_h.\beta_h.S_h^0}{\Pi_h(\beta_h+\mu_h).(.\rho_h+\mu_h+\delta_h)}} \\ + \sqrt{\dfrac{(\sigma_{p_2}.\varepsilon_p)^2}{(\beta_p+\mu_p)^2} + \dfrac{4.\sigma_{p_2}.\beta_p}{(\beta_p+\mu_p).(\rho_p+\mu_p+\delta_p)}} \end{array} \right)}{2} \quad (50)$$

Global Stability of Disease Free Equilibrium (DFE):

Theorem 3: The disease free equilibrium of the model system is GAS if Re < 1

Proof:

$$\frac{dX_1}{dt} = F(X_1, X_{2.}) \quad (53)$$

$$\frac{dX_2}{dt} = G(X_1, X_2); G(X_1, 0) = 0 \quad (54)$$

$$X_1 = (S_h^0, V_h^0, R_h^0, S_p^0, R_p^0), \text{ AND } X_2 = (E_h^0, I_h^0, E_p^0, I_p^0) \quad (55)$$

The DFE is now denoted as,

$$E^0 = (X_1^*, 0) \text{ where } X_1^* = (N^0, 0) \quad (56)$$

The first condition is GAS of X_1^*, we get,

$$\frac{dX_1}{dt} = F(X_1, 0) = \left\{ \begin{array}{l} (1-f)\Pi_h + \omega V_h^0 - \gamma S_h^0 - (0)S_h^0 - \mu_h S_h^0 \\ f\Pi_h + \gamma S_h^0 - \omega V_h^0 - \mu_h V_h^0 \\ -\mu_h R_h^0 \\ \Pi_p - (0)S_p^0 - \mu_p S_p^0 \\ \rho_p I_p^0 - \mu_p R_p^0 \end{array} \right\} \quad (57)$$

A linear differential equation solving gives,

$$S_h^0(t) = \frac{(1-f)\Pi_h + \omega V_h^0}{k_1} - \frac{(1-f)\Pi_h + \omega V_h^0}{k_1}.e^{-k_1.t} + S_h^0(0).e^{-k_1.t} \quad (58)$$

$$V_h^0(t) = \frac{f\Pi_h + \gamma S_h^0}{k_3} - \frac{f\Pi_h + \gamma S_h^0}{k_3}e^{-k_3 t} + V_h^0(0)e^{-k_3 t} \quad (59)$$

$$R_h^0(t) = R_h^0(0)e^{-\mu_h t} \quad (60)$$

$$S_p^0(t) = \frac{\Pi_P}{\mu_P} - \frac{\Pi_P}{\mu_P}e^{-\mu_P t} + s_P^0(0)e^{-\mu_P t} \quad (61)$$

$$R_p^0(t) = R_P^0(0)e^{-\mu_P t} \quad (62)$$

This shows that $S_h^0 + V_h^0 + R_h^0 + S_p^0 + R_p^0 -> N^0$ 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: $\widehat{G}(X_1, X_2) = AX_2 - G(X_1, X_2)$

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

$$A=\begin{bmatrix} (\frac{\sigma_h \varepsilon_h S_h^0}{N_h^0}-(\beta_h+\mu_h) & \frac{\sigma_h S_h^0}{N_h^0} & \frac{\sigma_p \varepsilon_p S_h^0}{N_p^0} & \frac{\sigma_p S_h^0}{N_p^0} \\ \beta_h & -(\rho_h+\mu_h+\delta_h) & 0 & 0 \\ 0 & 0 & (\frac{\sigma_p.\varepsilon_p.S_p^0}{N_p^0})-(.\beta_p+\mu_{p.}) & (\frac{\sigma_p.S_p^0}{.N_p^0} \\ 0 & 0 & .\beta_p & -(.\rho_p+\mu_p+\delta_p) \end{bmatrix} \quad (63)$$

$$G(X_1,X_2)=\begin{bmatrix} (\frac{\sigma_p.(\varepsilon_p E_p^0+I_p^0)}{N_p^0}+\frac{\sigma_h.(\varepsilon_h E_h^0+I_h^0)}{N_h^0}).S_h^0-(\beta_h+\mu_h)E_h^0 \\ -(\beta_h+\rho_h+\mu_h+\delta_h)I_h^0 \\ (\frac{\sigma_p.(\varepsilon_p E_p^0+I_p^0)}{N_p^0}).S_p^0-(\beta_p+\mu_p)E_p^0 \\ \beta_p.E_p^0-(\rho_p+\mu_p+\delta_p)I_p^0 \end{bmatrix}=\begin{bmatrix}0\\0\\0\\0\end{bmatrix} \quad (64)$$

Then, $\widehat{G}(X_1,X_2)=A.X_2-G(X_1,X_2)$

$$\begin{bmatrix} (\frac{\sigma_h \varepsilon_h S_h^0}{N_h^0}-(\beta_h+\mu_h) & \frac{\sigma_h S_h^0}{N_h^0} & \frac{\sigma_p \varepsilon_p S_h^0}{N_p^0} & \frac{\sigma_p S_h^0}{N_p^0} \\ \beta_h & -(.\rho_h+\mu_h+\delta_{h.}) & 0 & 0 \\ 0 & 0 & (\frac{\sigma_p.\varepsilon_p.S_p^0}{N_p^0})-(.\beta_p+\mu_p) & (\frac{.\sigma_p S_p^0}{N_p^0} \\ 0 & 0 & \beta_p & -(.\rho_p+\mu_p+\delta_p) \end{bmatrix}\begin{bmatrix}E_h^0\\I_h^0\\E_p^0\\I_p^0\end{bmatrix}-\begin{bmatrix}0\\0\\0\\0\end{bmatrix}=\begin{bmatrix}0\\0\\0\\0\end{bmatrix} \quad (65)$$

$$\widehat{G}(.X_1,X_2)=\begin{bmatrix}0 & 0 & 0 & 0\end{bmatrix}^T.$$

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

RESULTS AND DISCUSSION

The calculation and estimation of the parameters values was done based on the availability of information from the Nigeria Centre for Disease control (NCDC), on situation report of Monkey-pox disease between 2017 to 2023, as mentioned 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)

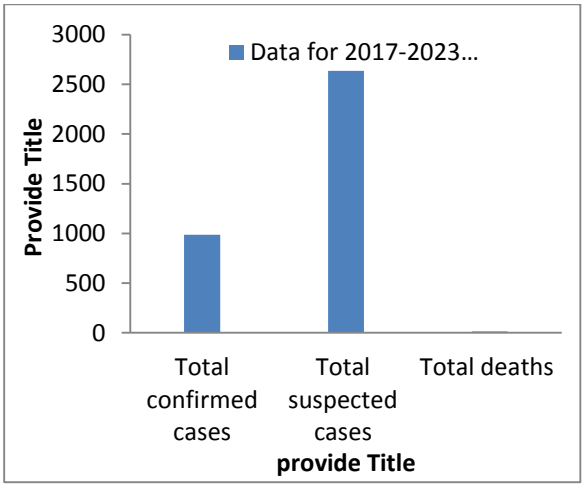


Fig. 2: Chart for data of confirmed cases, suspected cases and death for monkey pox for 2017-2023

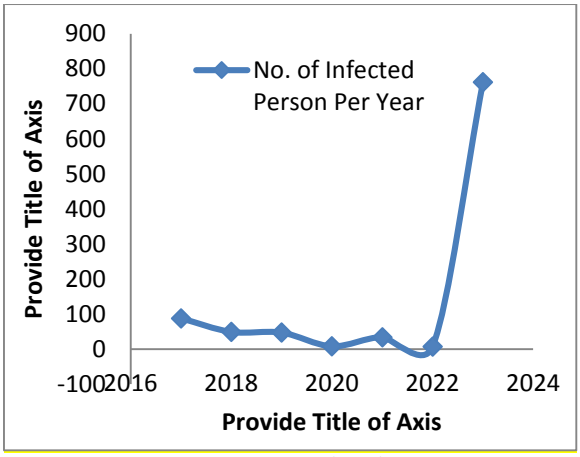


Fig. 3: Graphical Representation for infected monkey pox individual per year for 2017-2023

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 is $\mu_h = \frac{1}{56.05} = 0.01784$ per year. The birth rate is 38.03 per year per 1000 people; this gives the rate as $\frac{38.03}{1000} = 0.03803$/year. However, the recruitment rate due to birth in Nigeria is $\Pi_h = N_h * \mu_h = 3{,}568{,}000$. 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, $\mu_m = \frac{1}{30}\ or\ \frac{1}{15} = (0.033\ or\ 0.067)$. 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, $\Pi_m = N_m * \mu_m = 1{,}665$. The vaccination rate of monkeypox infection is 10.1%, so fraction of vaccination against monkeypox (f) is $f = \frac{10.1}{100} = 0.0101$. Vaccination last upto 3-5 years and can also protect around 85% from monkeypox. 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 2: Values of the parameters

Parameter	Definition	Value	Source
β_h	Exposed Rate of Human	0.005	Assumed
α_h	Infection Rate of Human	0.000494 per year	Table 1
Π_h	Recruitment Rate of Human	3,568,000	Table 1
γ_h	Recovery Rate of Human	0.985 per year	Table 1
μ_h	Natural Death Rate of Human	0.01784 per year	Table 1
δ_h	Death Rate Due to Disease	0.0152 per year	Table 1
φ_1	Loss of Vaccination Rate	0.1 - 0.2 /year	Table 1
φ_2	Vaccination Rate	0.012/year	Table 1
F	Proportion of vaccinated human immigrants	0. .0101	Table 1
∅	Effectiveness of Vaccination Drug	0 – 1	Assumed
α_m	Infection Rate of Monkey	0.004	Table 1
γ_m	Recovery Rate of Monkey	0.50	Assumed
Π_m	Recruitment Rate of Monkey	1,665	Table 1
θ_{m_2}	Exposed Rate of Monkey	0.003	Assumed
μ_m	Natural Death of Monkey	0.033 – 0.067	Table 1
δ_m	Death Due to infection of monkey	0.020	Assumed
τ_h	Exposed rate for human transmits lower than symptomatic humans	0.010	Assumed
τ_m	Exposed rate for non-human transmits lower than symptomatic non-humans	0.010	Assumed
N_h	Nigeria Population	200,000,000	Estimated
N_m	Population of Monkey in Nigeria	50,000	Assumed

We 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. In fig. 4, we simulated the vaccination rate on effective reproduction number, we found in figure 4, that average secondary cases of disease increases as exposed individual increases, varying the vaccination, we observed that effective reproduction number reduces as vaccination increases. Figure 5 shows the simulation of Monkey pox infection rate with changing in recovery rate of infected individual, it is observed that average secondary cases of infection increase as infection rate increases. Figure 6 shows the simulation of loss of vaccination against effective reproduction number, it is observed that average effective reproduction number of monkey pox infection increases as loss of vaccination of individual increases.

Figure 7 shows the simulation of Infection rate of non-human with effective reproduction number of non-human, it is observed that effective reproduction number of monkey infection increases as infection individual of non-human increases. In the figure 8, we observed that effective reproduction number of the monkey pox disease among the non-human increases as exposed rate of non-human increases.

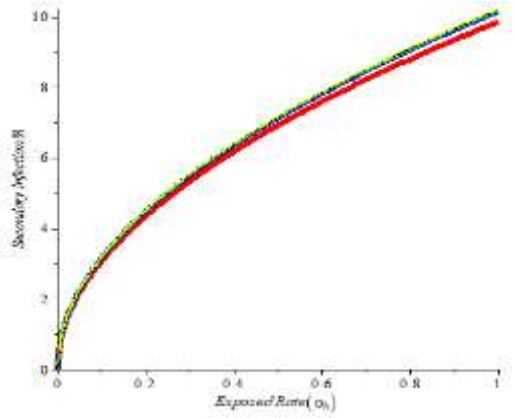


Fig. 4: Varying Rate of Vaccination (γ),

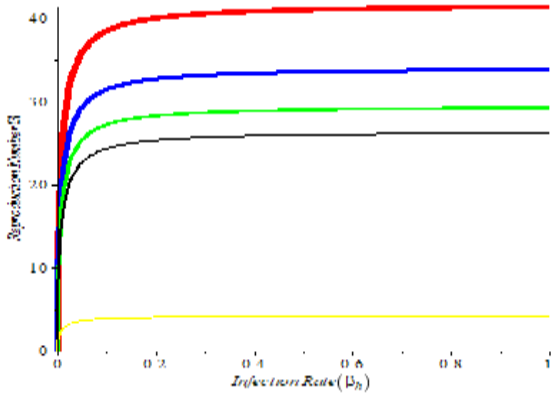


Fig. 5: Varying Rate of Recovery

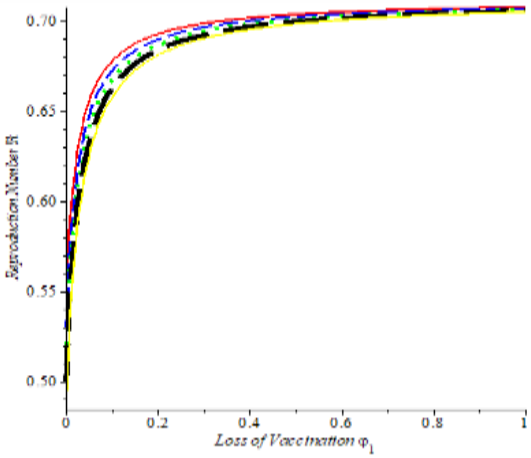


Fig. 6: Varying Rate of Vaccination

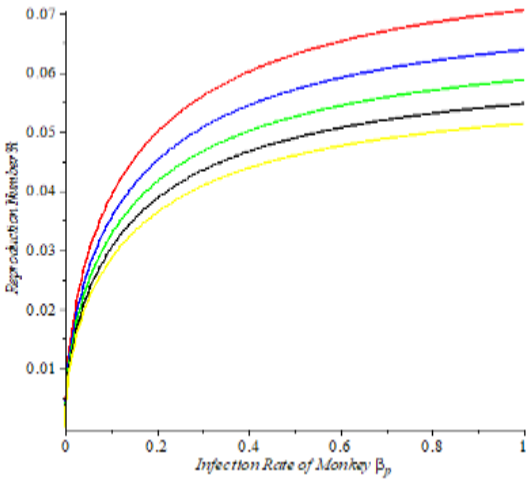


Fig. 7: Varying Rate of Recovery for the primate

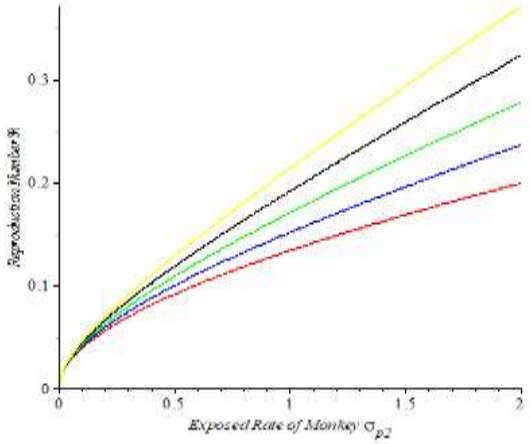


Fig. 8: varying the exposed rate for non-human transmits lower than symptomatic non-humans.

Conclusion: This research work have developed and simulated a mathematical model for monkey pox transmission disease. The model was considered as been mathematically and epidemiologically well posed. The non-negativity of the solutions for the model system implies that the solutions were positive and remains in region. The disease free equilibrium and effective reproduction number of the model were obtained. The disease free equilibrium of the model equations is asymptotically stable globally (GAS) if Re < 1. It is observed that the average secondary cases of monkey pox infection increases as loss of vaccination of individual increases and reduces as vaccination increases

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REFERENCES

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

Bhunu, CP; Mushayabase, S (2011). Modeling the Transmission Dynamics of Pox-like Infections. *Intern. J. Appl. Math*. 41: 2-09

Castillo-Chavez, C; Feng, Z; Huang, W (2002). On the computation of Ro and its role on global stability. In C. Castillo-Chavez S., Blower P., Van den Driessche D. Krirschner, A. A. Yakubu (Eds), Mathematical approaches for emerging and re-emerging infectious diseases: An introduction. The IMA Volumes in Mathematics and its Applications, Vol. 125, p. 229-250. New York: Springer-Verlag.

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

Essbauer, S; Pfeffer, M; Meyer, H (2009). Zoonotic poxviruses. *Vet. Microbiol.* doi:10.1016/j.vetmic.

Breman, JG; Ruti, K; Steniowski, MV; Zanotto, E; Gromyko, AI; Arita, I (1986). Human monkeypox: a study of 2510 contacts of 214 patients. *J. Infectious Dis.* 154(4): 551-555.

Kantele, A; Chickering, K; Vapalahti, O; Rimoin, AW (2016). Emerging diseases—the monkeypox epidemic in the Democratic Republic of the Congo. *Clinical Microbiology and Infection*. 22(8): 658–659.

Lasisi, NO; Suleiman, F (2024). Effects of Poor Sanitation and Public Awareness in Modeling Bacterial Infection amongst the Students of a Tertiary Institution in Kaura Namoda, Zamfara State, Nigeria. *J. Appl. Sci. Environ. Manage.* 28(4): 1177-1185 April 2024. DOI: https://dx.doi.org/10.4314/jasem.v28i4.17

Lasisi, NO (2021). Lyapunov Approach and Global Stability Of Ebola Virus Infection Model Of An Individual Cells Population, *Mat. Sci. Eng. & Applic.*. 1(1): 1-10. *Doi Https://Doi.Org/10.21595/Msea.2021.21977*

Lasisi, NO; Adeyemo, KA (2021). Modelling the Effect of Distancing and Wearing of Face Masks on Transmission of Covid-19 Infection Dynamics. *J. Complex. Health Sci*. 4(1): 10-20. *DOI https://doi.org/10.21595/chs.2021.21976*

Lasisi, NO; Akinwande, NI; Abdulrahaman, S (2020). Optimal Control and Effect of Poor Sanitation on Modeling the Acute Diarrhea Infection. *J. Complex. Health Sci*. 3(1): 91-103. *DOI https://doi.org/10.21595/chs.2020.21409*

Lasisi, NO (2020). Effect of public awareness, behaviours and treatment on infection-age-structured of mathematical model for HIV/AIDS dynamics. *J. Mathematical Models in Eng*. 6(2): 103-121.*DOI https://doi.org/10.21595/mme.2020.21249*

Lasisi, NO; Akinwande, NI; Oguntolu, FA (2020). Development and exploration of a Mathematical Model for Transmission of Monkey-Pox in Humans. *J. Mathematical Models in Eng*. 6(1): 23-33. https://doi.org/10.21595/mme.2019.21234

Lasisi, NO; Akinwande, NI; Olayiwola, RO; Cole, AT; Abdulrhaman, S (2018). Global Stability of Virus Persistence of a Mathematical Model of the Dynamics of Ebola Virus Infection in Human Cell Population. *J. Nig. Associ. Mathematical Phy*. 45: 83-90.

Lasisi, NO; Akinwande, NI; Olayiwola, RO; Cole, AT (2018). Mathematical Model for Ebola Virus Infection in Human with Effectiveness of Drug Usage. *J. Appl. Sci. Environ. Manage.* 22(7): 1089–1095. https://dx.doi.org/10.4314/jasem.v22i7.16

Lasisi, NO; Suleiman, F (2024). Global Stability Analysis of Disease Free Equilibrium for Modeling the Dynamics of Bacteria Infection in Higher Institution Kaura Namoda, Nigeria. *Bima J. Sci. & Tech.* 8(2), 160-171. Doi:10.56892/bima.v8i2.674

Nigeria Centre for Disease Control (2023)**.** *Situation Report*: Update on Monkey-pox in Nigeria, January 1, 2023

Rimoin, AW; Kisalu, N; Kebela-Ilunga, B; Mukaba, T; Wright, LL; Formenty, P; Wolfe, ND; Hennessey, M (2007). Epidemic human monkey pox, Democratic Republic of Congo, 2001-2004. *Emerg. Infect. Dis.* 13(6): 934-936.

Magnus, PV; Andersen, EK; Petersen, KB; Birch-Andersen, A (1959). A pox like disease in cynomolgus monkeys. *Acta Pathol. Microbiol. Scand.* 46(2): 156-176