

Development and exploration of a mathematical model for transmission of monkey-pox disease in humans

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Abstract. In this paper, mathematical model of Monkey-Pox transmission is developed and investigated, using ordinary differential equation. We verified the feasible region of the model and showed the positivity of the solutions. We obtained the disease free equilibrium (DFE). We computed and analysed the effective basic reproduction number (R_0) of the model.

Keywords: effective basic reproduction number, disease free equilibrium, mathematical modelling, monkey pox disease.

1. Introduction

Monkey-pox is known as pathogens, affecting livestock animals and humans and belongs to the orthopox virus, which included small pox cause infection in humans and cow pox viruses [1]. The monkey pox causes lymph nodes to swell. The symptoms are fever, headache, muscle aches, backache, swollen lymph nodes, a feeling of discomfort and exhaustion. The virus is similar to small pox in human. The virus can spread both from animal to human and from human to human. The increased transmission risk associated with factors involving introduction of virus to the oral mucosa [2].

The incubation period range from 7 to 14 days. After 1 to 3 days of the appearance of fever, the patient develops raised bumps and the illness lasted for 2 to 4 weeks [5]. Thus, the reported case fatality ratio is range from 1 % to 10 % [3]. Currently, there are no vaccines for monkey pox, however, evidence showed that small pox vaccine reduced the risk of monkey pox among previously vaccinated persons in Africa [4, 5]. Centre for disease control and prevention also recommended that persons investigating monkey pox outbreaks and caring for infected individuals should vaccinated with small pox vaccine, to protect against the monkey pox. Individuals who have had close contact with persons confirmed to have monkey pox should be vaccinated up to 14 days after exposure were recommended [4].

Mathematical models have played a central role to capture the dynamics of different Disease transmission [6]. The aim of this paper is therefore to the development and exploration of a mathematical model for transmission of monkey-pox disease in humans. Therefore, there is non-numerous work on mathematical modelling of monkey pox transmission. This Paper extends the work of [7] by incorporating Vaccination class for migrants, Exposed classes for both human and non-human population.

2. Model formulation

We formulate a model for the spread of Monkey-pox in human and primates (Monkey) population with the total population size at time t given by $N_h(t)$ and $N_p(t)$. The populations are further compartmentalized into epidemiological classes as shown in the model flow diagram in Fig. 1. The total human population is divided into five subgroups that is the Susceptible, $S_h(t)$, the Vaccinated, $V_h(t)$, the Exposed, $E_h(t)$, the Infected, $I_h(t)$, and the Recovery with permanent immunity, $R_h(t)$. The total primates (Monkey) population model divides into the Susceptible,

$S_p(t)$, the Infected primates, $I_p(t)$, the Exposed, $E_p(t)$ and the recovery with permanent immunity, $R_p(t)$. As indicated in the compartmental diagram in Fig. 1, people enter Susceptible class through birth and immigration, (Λ_h) , where a proportion of vaccinated human immigrants (f) enter to the vaccinated class and proportion of unvaccinated immigrants $(1 - f)$ enter to the susceptible class. We do not consider the immigration of infection person, because we assume that people who are coming from monkey-pox endemic zones have to be vaccinated. The susceptible individuals vaccinated at the rate γ and loss the vaccination at the rate ω . Susceptible human get contact from primate at rate σ_{p_1} , S_h are exposed to monkey-pox infection at the rate σ_h and infected at the rate β_h , with natural death μ_h and die due to the infection at the rate δ_h and recovery with permanent immunity at a rate ρ_h . The Susceptible primates, S_p class is generated from the daily recruitment of individuals through birth and immigration at the Λ_p and natural death rate μ_p . They become exposed to monkey-pox virus at the rate σ_{p_2} and leave the class to infected class at the rate β_p . Individuals primate infected die due to the infection at the rate δ_p and recovery with the permanent immunity at the rate ρ_p .

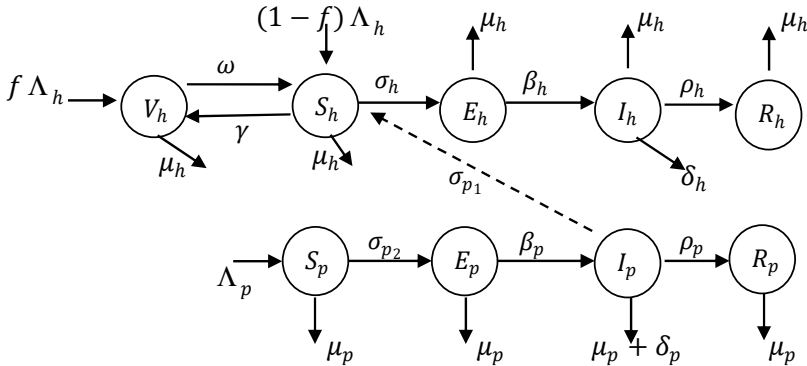


Fig. 1. Schematic diagram of transmission of monkey-pox disease

Base on the above assumptions and schematic diagram of Fig. 1, the model for transmission dynamics of Monkey-pox infection in human and primates (Monkey) is described by a system of Ordinary Differential Equations (ODEs) given below:

$$\frac{dS_h}{dt} = (1 - f)\Lambda_h + \omega V_h - \gamma S_h - \left(\frac{\sigma_{p_1}(\varepsilon_p E_p + I_p)}{N_p} + \frac{\sigma_h(\varepsilon_h E_h + I_h)}{N_h} \right) S_h - \mu_h S_h, \quad (1)$$

$$\frac{dE_h}{dt} = \left(\frac{\sigma_{p_1}(\varepsilon_p E_p + I_p)}{N_p} + \frac{\sigma_h(\varepsilon_h E_h + I_h)}{N_h} \right) S_h - \beta_h E_h - \mu_h E_h, \quad (2)$$

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

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

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

$$\frac{dS_p}{dt} = \Lambda_p - \left(\frac{\sigma_{p_2}(\varepsilon_p E_p + I_p)}{N_p} \right) S_p - \mu_p S_p, \quad (6)$$

$$\frac{dE_p}{dt} = \left(\frac{\sigma_{p_2}(\varepsilon_p E_p + I_p)}{N_p} \right) S_p - \beta_p E_p - \mu_p E_p, \quad (7)$$

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

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

where:

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

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

The susceptible human host get infected from both the infected primate (I_p) and infected human (I_h) which is called force of infection from primates to human as well as human to human [7]. The term σ_{p1} is the product of the effective contact rate and probability of the human being get infected from the infected primates (I_p) and σ_h is the product of the effective contact and probability of the human get infected from the infected persons (I_h). Similarly, the S_p get infected from infected primate, where σ_{p2} is the product of the effective contact rate and probability of the primates (monkey) get infected per contact with an infected primate (I_p) [7]. The modification parameter ε_h account for the assumption that exposed human transmits at a rate lower than symptomatic humans. The modification parameter ε_p account for the assumption that exposed non-human transmits at a rate lower than symptomatic non-humans. It is assumed here that mortality of monkeys due to being hunted by humans is negligible and can be safely ignored.

3. Analysis of the model equations

Theorem 1: (Invariant Region) the following biological feasible region of the model Eqs. (1)-(9) $\Omega = \{S_h, E_h, I_h, V_h, R_h, S_p, E_p, I_p, R_p\} \in \mathfrak{R}_+^9 : \{S_h(t) + E_h(t) + I_h(t) + V_h(t) + R_h(t) \leq \Lambda_h/\mu_h; Spt + Ept + Ipt + Rp(t) \leq \Lambda_p/\mu_p\}$ is positively invariant and attracting.

Proof; the addition of all the equations model in Eqs. (1)-(9) give:

$$\begin{aligned} \frac{dN_h}{dt} &= \Lambda_h - \mu_h N_h - \delta_h I_h, \\ \frac{dN_p}{dt} &= \Lambda_p - \mu_p N_p - \delta_p I_p. \end{aligned}$$

So that:

$$\frac{dN_h}{dt} \leq \Lambda_h - \mu_h N_h, \quad \frac{dN_p}{dt} \leq \Lambda_p - \mu_p N_p. \quad (12)$$

It follows from [8], the Gronwall inequality, that:

$$\begin{aligned} N_h(t) &\leq N_h(0)e^{-\mu_h(t)} + \frac{\Lambda_h}{\mu_h} \{1 - e^{-\mu_h(t)}\}, \\ N_p(t) &\leq N_p(0)e^{-\mu_p(t)} + \frac{\Lambda_p}{\mu_p} \{1 - e^{-\mu_p(t)}\}. \end{aligned} \quad (13)$$

In particular, $N_h(t) \leq \Lambda_h/\mu_h$ if $N_h(0) \leq \Lambda_h/\mu_h$ and $N_p(t) \leq \Lambda_p/\mu_p$ if $N_p(0) \leq \Lambda_p/\mu_p$. Thus, Ω is positively invariant. Hence, it is sufficient to consider the model dynamics Eqs. (1)-(9) in Ω , in this region, the model equations can be considered as been epidemiologically and mathematically well posed.

Theorem 2: (Positivity of the Solution for the Model). Let $t_0 > 0$ and 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 Eqs. (1)-(9) are positive 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 positive in \mathfrak{R}_+^9 , Since all the parameters used in the system are positive. Thus, it is clear from Eq. (1) that:

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

So that:

$$S_h(t) \geq S_h(0)e^{-(\gamma + \alpha_h + \mu_h)t - f \int dt} \geq 0 \quad \text{or} \quad S_h(t) \geq S_h(0) \exp\left\{-\int (\gamma + \alpha_h + \mu_h)dt\right\}. \quad (14)$$

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 positive and remain in \mathfrak{R}_+^9 .

4. Existence of the equilibrium

At equilibrium state, we let:

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

From Eq. (3), we have:

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

Substitute Eq. (16) into Eq. (2), we have:

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

Equation (17) gives:

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

From Eq. (8), we have:

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

Substitute Eq. (19) into Eqs. (7), we have:

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

Equation Eq. (20) gives:

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

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

$$E_h = R_h = 0. \quad (22)$$

Making V_h subject of expression in Eq. (4) and Eq. (1), and equating them, we have:

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

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 $I_h = 0$, implies $\alpha_h = 0$, then we have:

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

Reduced to:

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

If there is no vaccination, then $S_h^0 = \frac{\Pi_h\mu_h}{\mu_h^2} = \frac{\Pi_h}{\mu_h}$ as $\omega = \gamma = f = 0$.

Substitute Eq. (25) into Eq. (4), we have:

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

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

$$E_p = R_p = 0. \quad (27)$$

From Eq. (6), we have:

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

The disease free equilibrium (DFE) state is given as:

$$E_0 = \{ S_h^*, E_h^*, V_h^*, I_h^*, R_h^*, S_p^*, E_p^*, I_p^*, R_p^* \} \\ = \left\{ \frac{\Pi_h\omega + \Pi_h\mu_h - f\Pi_h\mu_h}{\gamma\mu_h + \mu_h\omega + \mu_h^2}, 0, \frac{f\Pi_h\mu_h\omega + f\Pi_h\mu_h^2 + \gamma\Pi_h\omega + \gamma\Pi_h\mu_h}{(\gamma\mu_h + \mu_h\omega + \mu_h^2)(\omega + \mu_h)}, 0, 0, \frac{\Pi_p}{\mu_p}, 0, 0, 0 \right\}. \quad (29)$$

5. Effective basic reproduction number (R_e)

We applying next generation matrix operator to compute the effective basic reproduction number [9]. The largest Eigenvalue or spectral radius of FV^{-1} is the effective basic reproduction

number of the model:

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

where F_i is the rate of appearance of new infection in compartment i , V_i is the transfer of infection from one compartment i to another and E^0 is the Disease-Free Equilibrium.

Using this technique, we have spectral radius (ρ) of the next generation matrix, FV^{-1} that is, $R_0 = \rho(FV^{-1})$. Both F and V are obtained from the Jacobian matrix of the linearization of system Eqs. (1)-(9) disease free equilibrium. Therefore, the vector F and V representing inflow and outflow from compartments E_h, E_p, I_h and I_p are given by:

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

$$F = \begin{bmatrix} \frac{\sigma_h \varepsilon_h S_h^0}{N_h^0} & \frac{\sigma_h S_h^0}{N_h^0} & \frac{\sigma_{p1} \varepsilon_p S_h^0}{N_p^0} & \frac{\sigma_{p1} S_h^0}{N_p^0} \\ \beta_h & 0 & 0 & 0 \\ 0 & 0 & \frac{\sigma_{p2} \varepsilon_p S_p^0}{N_p^0} & \frac{\sigma_{p2} S_p^0}{N_p^0} \\ 0 & 0 & \beta_p & 0 \end{bmatrix}, \quad (32)$$

$$v = \begin{pmatrix} v_1 \\ v_2 \\ v_3 \\ v_4 \end{pmatrix} = \begin{bmatrix} A_1 E_h \\ A_2 I_h \\ A_3 E_p \\ A_4 I_p \end{bmatrix}, \quad (33)$$

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

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

From Eqs. (34), we have:

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

At DFE, and since $N_h(t) \leq \Pi_h/\mu_h$ and $N_p(t) \leq \Pi_p/\mu_p$ we have:

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

From Eq. (36), we have:

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

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

Multiplying Eq. (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:

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

The characteristics equation of Eq. (40), gives $|FV^{-1} - \lambda I| = 0$:

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

where:

$$\begin{aligned}
 K_1 &= \frac{\sigma_h \varepsilon_h \mu_h S_h^0}{\Pi_h A_1}, & K_2 &= \frac{\sigma_h \mu_h}{\Pi_h A_2}, & K_3 &= \frac{\sigma_{p_1} \varepsilon_p \mu_p}{\Pi_p A_3}, & K_4 &= \frac{\sigma_{p_1} \mu_p}{\Pi_p A_4}, \\
 K_5 &= \frac{\beta_h}{A_1}, & K_6 &= \frac{\sigma_{p_2} \varepsilon_p}{A_3}, & K_7 &= \frac{\sigma_{p_2}}{A_4}, & K_8 &= \frac{\beta_p}{A_3}.
 \end{aligned} \tag{42}$$

Determinant of Eq. (41) gives:

$$(\lambda^2 - K_6 \lambda - K_7 K_8) = 0 \quad \text{or} \quad (\lambda^2 - K_1 S_h^0 \lambda - K_2 K_5 S_h^0) = 0. \tag{43}$$

To solve Eq. (43) with completing the square method, we have:

$$\begin{aligned}
 \lambda_1 &= \frac{K_6 \pm \sqrt{K_6^2 + 4K_7 K_8}}{2}, \\
 \lambda_1 &= \frac{\frac{\sigma_{p_2} \varepsilon_p}{(\beta_p + \mu_p)} \pm \sqrt{\frac{(\sigma_{p_2} \varepsilon_p)^2}{(\beta_p + \mu_p)^2} + \frac{4\sigma_{p_2} \beta_p}{(\beta_p + \mu_p)(\rho_p + \mu_p + \delta_p)}}}{2}.
 \end{aligned} \tag{44}$$

λ_1 is the spectral radius of $\rho(FV^{-1})$ and the reproduction number is the largest eigenvalue from Eq. (44):

$$\begin{aligned}
 R_p &= \frac{\frac{\sigma_{p_2} \varepsilon_p}{(\beta_p + \mu_p)} + \sqrt{\frac{(\sigma_{p_2} \varepsilon_p)^2}{(\beta_p + \mu_p)^2} + \frac{4\sigma_{p_2} \beta_p}{(\beta_p + \mu_p)(\rho_p + \mu_p + \delta_p)}}}{2}, \\
 (\lambda^2 - K_1 S_h^0 \lambda - K_2 K_5 S_h^0) &= 0.
 \end{aligned} \tag{45}$$

Implies:

$$\lambda_2 = \frac{K_1 S_h^0 \pm \sqrt{K_1^2 S_h^{0^2} + 4K_2 K_5 S_h^0}}{2}. \tag{46}$$

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

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

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

Then, we obtained effective basic reproduction number as Eqs. (48) and (45). There are two host populations and it was shown from the model flow diagram in Fig. 1 that the monkey transmits the infection to human host and human to human. Hence, the effective basic reproduction number can be represented as:

$$R_0 = R_h + R_p, \tag{49}$$

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

5.1. Analysis of effective basic reproduction number on R_p and R_h

Form Eq. (45), we have:

$$R_p = \frac{M_1 + \sqrt{M_1^2 + M_2}}{2},$$

where:

$$M_1 = \frac{\sigma_{p_2} \varepsilon_p}{(\beta_p + \mu_p)}, \quad M_2 = \frac{4\sigma_{p_2} \beta_p}{(\beta_p + \mu_p) (\rho_p + \mu_p + \delta_p)}. \quad (51)$$

Since the disease will not be established in the population if the secondary cases on average is less than a unit ($R_p \leq 1$), this implies:

$$R_p = \frac{M_1 + \sqrt{M_1^2 + M_2}}{2} \leq 1, \quad (52)$$

$$M_1 + \sqrt{M_1^2 + M_2} \leq 2, \quad R_p \leq 1. \quad (53)$$

From Eqs. (53), we have:

$$M_2 = 4 - 4M_1. \quad (54)$$

Implies:

$$\frac{4\sigma_{p_2} \beta_p}{(\beta_p + \mu_p) (\rho_p + \mu_p + \delta_p)} \leq \frac{4(\beta_p + \mu_p) - 4\sigma_{p_2} \varepsilon_p}{(\beta_p + \mu_p)}. \quad (55)$$

From Eqs. (55), we make the product of the effective contact rate and probability of the primates (monkey) get infected per contact with an infected primate as a subject, we have:

$$\sigma_{p_2} \leq \frac{4(\beta_p + \mu_p) (\rho_p + \mu_p + \delta_p)}{4\beta_p + 4\varepsilon_p (\rho_p + \mu_p + \delta_p)}, \quad R_p \leq 1, \quad (56)$$

$$\sigma_{p_2} \leq P_p, \quad R_p \leq 1, \quad (57)$$

Where:

$$P_p = \frac{4(\beta_p + \mu_p) (\rho_p + \mu_p + \delta_p)}{4\beta_p + 4\varepsilon_p (\rho_p + \mu_p + \delta_p)}. \quad (58)$$

Also, we make the infected at the rate β_p as a subject:

$$4\sigma_{p_2}\beta_p - 4\beta_p(\rho_p + \mu_p + \delta_p) \leq 4\mu_p(\rho_p + \mu_p + \delta_p) - 4\sigma_{p_2}\varepsilon_p(\rho_p + \mu_p + \delta_p),$$

$$\beta_p \leq \frac{\mu_p(\rho_p + \mu_p + \delta_p) - \sigma_{p_2}\varepsilon_p(\rho_p + \mu_p + \delta_p)}{\sigma_{p_2} - (\rho_p + \mu_p + \delta_p)}, \quad R_p \leq 1, \quad (59)$$

$$\beta_p \leq L_p, \quad R_p \leq 1. \quad (60)$$

Where:

$$L_p = \frac{\mu_p(\rho_p + \mu_p + \delta_p) - \sigma_{p_2}\varepsilon_p(\rho_p + \mu_p + \delta_p)}{\sigma_{p_2} - (\rho_p + \mu_p + \delta_p)}. \quad (61)$$

In a similar case, since the disease will not be established in the population if the secondary case on average is less than a unit ($R_h \leq 1$).

Form Eq. (48), we have:

$$R_h = \frac{Z_1 + \sqrt{Z_1^2 + Z_2}}{2}, \quad (62)$$

where:

$$Z_1 = \frac{\sigma_h \varepsilon_h \mu_h S_h^0}{\Pi_h(\beta_h + \mu_h)}, \quad Z_2 = \frac{4\sigma_h \mu_h \beta_h S_h^0}{\Pi_h(\beta_h + \mu_h)(\rho_h + \mu_h + \delta_h)}, \quad (63)$$

$$R_h = \frac{Z_1 + \sqrt{Z_1^2 + Z_2}}{2} \leq 1, \quad (64)$$

$$Z_1 + \sqrt{Z_1^2 + Z_2} \leq 2, \quad R_h \leq 1. \quad (65)$$

From Eqs. (65), we have:

$$Z_2 \leq 4 - 4Z_1. \quad (66)$$

Implies:

$$\frac{4\sigma_h \mu_h \beta_h S_h^0}{\Pi_h(\beta_h + \mu_h)(\rho_h + \mu_h + \delta_h)} \leq \frac{4\Pi_h(\beta_h + \mu_h) - 4\sigma_h \varepsilon_h \mu_h S_h^0}{\Pi_h(\beta_h + \mu_h)}. \quad (67)$$

From Eqs. (67), we make the product of the effective contact and probability of the human get infected from the infected persons as a subject, we have:

$$\sigma_h \leq \frac{4\Pi_h(\beta_h + \mu_h)(\rho_h + \mu_h + \delta_h)}{4\mu_h \beta_h S_h^0 + 4\varepsilon_h \mu_h S_h^0(\rho_h + \mu_h + \delta_h)}, \quad R_h \leq 1, \quad (68)$$

$$\sigma_h \leq P_h, \quad R_h \leq 1, \quad (69)$$

where:

$$P_h = \frac{4\Pi_h(\beta_h + \mu_h)(\rho_h + \mu_h + \delta_h)}{4\mu_h \beta_h S_h^0 + 4\varepsilon_h \mu_h S_h^0(\rho_h + \mu_h + \delta_h)}. \quad (70)$$

Also, we make the infected at the rate β_h as a subject:

$$\beta_h \leq \frac{\Pi_h \mu_h (\rho_h + \mu_h + \delta_h) - \sigma_h \varepsilon_h \mu_h S_h^0 (\rho_h + \mu_h + \delta_h)}{\sigma_h \mu_h S_h^0 - \Pi_h (\rho_h + \mu_h + \delta_h)}, \quad R_h \leq 1, \quad (71)$$

$$\beta_h \leq L_h, \quad R_h \leq 1, \quad (72)$$

where:

$$L_h = \frac{\Pi_h \mu_h (\rho_h + \mu_h + \delta_h) - \sigma_h \varepsilon_h \mu_h S_h^0 (\rho_h + \mu_h + \delta_h)}{\sigma_h \mu_h S_h^0 - \Pi_h (\rho_h + \mu_h + \delta_h)}. \quad (73)$$

6. Conclusions

The Monkey pox will be eradicated since the term P_p is greater than the product of the effective contact rate and probability of the primates (monkey) σ_{p_2} getting infected and the term L_p is greater than infected rate β_p as shown in Eq. (60), provided $R_p \leq 1$. However, since $\sigma_{p_1} = 0$, it means that there is no transmission between primates and humans population at disease free equilibrium. Similarly, the monkey pox will be eradicated among human population, since the term P_h is greater than σ_h and L_h is greater than β_h as shown in Eq. (72), provided $R_h \leq 1$. Epidemiological implication is that if the secondary cases of the monkey pox infection and human cases are on average less than one unit, then the disease will die out on the long run.

In this paper, we developed a mathematical model of monkey-pox transmission disease. We analysed the invariant region and shown that the dynamics of model Eqs. (1)-(9) is in the region Ω , the model equations was considered as been epidemiologically and mathematically well posed. The positivity of the solutions for the model was showed which implies that the solutions were positive and remains in R_+^9 . The disease free equilibrium and effective basic reproduction number of the model were obtained. Analyses of effective basic reproduction number were done.

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