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AN SEIR ENDEMIC MODEL FOR MONKEYPOX SPREAD IN UNITED STATES

S. SHALINI PRIYA AND K. GANESAN*

Abstract. In this paper, we construct a monkeypox model which is similar to smallpox infection. It is caused by a monkeypox virus which is related to Poxviridae family. It will occur mostly in West African communities and in remote Central. We develop a system of differential equations for an SEIR (Suspected, Exposed, Infected and Recovered) model and analyze the outbreak of monkeypox disease and its effect on United States(US) population. We establish theorems on asymptotical stability conditions for endemic equilibrium and disease-free equilibrium. The basic reproduction number R_0 has been determined using next generation matrix. We expect that this study will be effective at controlling monkeypox spread in United States. Our goal is to see whether monkeypox can be controlled and destroyed by smallpox vaccination. We find that monkeypox is controllable and can be fully destroyed in disease free state by vaccination. However, in the endemic state, monkeypox cannot be destroyed by vaccination alone.

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1. Introduction

Monkeypox is a dangerous zoonotic disease (i.e., the infection will transmit from animal to human) which occurs occasionally and mostly in Western African region near tropical rain forests and in remote Central areas. Monkeypox is within the Poxviridae which is a member of the genus Orthopox. The genus Orthopox virus also comprises the variola virus (which is the origin of smallpox) and vaccinia virus (which is used for eradication of smallpox in the vaccination). The monkeypox is mostly transferred from wild primates and rodents

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to humans, but most transmissions happen between human to human. Two pox-like disease outbreaks in monk colonies kept for research in 1958 and it led to the discovery of monkeypox. In 1970, the first human case was reported in the Democratic Republic of Congo during a time of increased attempts to fight the smallpox. Among other Western and Central African countries like Cote d Ivoire, Cameroon, Liberia, Congo, Sierra Leone and South Sudan, monkeypox has been since identified in humans in those regions. There are currently no effective treatment for monkeypox infection, although many snipping antiviral like Tecovirimat, Brincindofovir and vaccinia immunity globulin, it can be used to stop the disease's transmission. Due to the decline in smallpox herd immunity, there has been a large rise in monkeypox over the past ten years. Since smallpox has been eradicated worldwide, vaccination against it has been demonstrated to be 85 percent effective in preventing monkeypox. However, it is no longer readily accessible.

Hutson et al. [6] have discussed that monkeypox's incubation period is typically about 6 to 16 days but can range from 5 to 21 days. It is noticeable that secondary animals are exposed and appeared to manifest a more serious illness. Monkeypox was transmitted through exposure to contaminated co-housing, bedding or respiratory secretions/nasalmucous. Usman and Adamu [23] have discussed the dynamic of the monkeypox in rat and human hosts using stability analysis. Meyer et al. [9] have suggested that the severity of the illness may be reduced or prevented with the use of the post-exposure vaccine. Narayanamoorthy et al. [12] have derived a new numerical method to solve fuzzy differential equations of fractional order in the sense of Caputo-type. The test problem is given to verify the resilient and effectiveness of the proposed method. They noted that the small step size plays the main role in their proposed method and is more accurate and closer to the exact solutions. Muhammad Abdy et al. [11] created an SIR mathematical model for COVID 19 in Indonesia using the fuzzy parameters. They suggested that the treatment has a great effect in stopping or slowing the force of infection but not as much as implementing the health protocols and the effect of vaccination. Nguyen et al. [13] suggested that in 2017, Nigeria have significant returns of monkeypox which seems to have driven by a mixture of accumulation of the unvaccinated individuals, population growth and decline in smallpox vaccine immunity. The most of deaths are happening in children under ten years old. James Peter et al. [15] developed and analyzed a mathematically deterministic model of the monkeypox virus. The asymptotic stability criteria for endemic and disease-free equilibrium are established on both a global and local level. Rabab et al. [16] have demonstrated how the dynamics of monkeypox behaves as a result of changing input variables and their results indicate how mathematical modeling can offer special understanding into the dynamics. The model outlined seeks to provide a means to investigate the monkeypox dynamics. They used conditions for the dynamics of monkeypox virus Ulam-Hyers stability and demonstrated the effect of the various factors on the dynamics of monkeypox, their dynamical behavior of the system is examined

using a novel numerical scheme. Samayan et al. [18] have targeted to investigate the most accurate approximate solution of fuzzy linear differential equations using Homotopy Perturbation Method(HPM) and is found that the solution obtained by HPM is closer to the exact solution. They have done a comparison with the exact solution of fuzzy linear systems on Glucose-Insulin Regulatory System model and dynamics of drug therapy model.

In this study, we consider a mathematical model of SEIR in a normalised form with three control parameters, namely smallpox vaccination control, social distancing and following health protocols. Our goal is to look into the numerous elements that can reduce the spread of diseases and how they might affect the basic reproduction rate. Graphs are provided to show the efficiency of the proposed method for disease free equilibrium's stability and for the endemic equilibrium's stability.

2. Method

We propose a four compartmental model on the spread of monkeypox virus consisting of human population. The human population is further subdivided into susceptible S(t), exposed E(t), infected I(t) and recovered R(t) so that the total population $N(t) = S(t) + E(t) + I(t) + R(t)$. The compartmental model is used to construct an SEIR model by considering smallpox vaccination, people who are following the health protocols, force of infection and the rate of exposed human to infected human. Here we use smallpox vaccination and following health protocols as the monkeypox virus control parameters. An SEIR model analysis uses the next generation matrix technique to obtain the basic reproduction number R_0 and the stability of SEIR model for the spread of monkeypox. A discussion is made for SEIR model and we take data on the number of suspected, exposed, infected and recovered monkeypox cases in United States from May 2022 to September 2022. The parameters of smallpox vaccine effectiveness, the level of obedience of the people who are following the health protocols and monkeypox virus are taken in the example.

3. SEIR Model for Monkeypox

Consider an SEIR model for monkeypox which describes the change of direct and indirect transmission of monkeypox with the interaction between exposed and infected, birth/death rates, change from being infected to recovery, smallpox vaccine effectiveness, obedience in following health protocols and deaths due to the monkeypox infection. The schematic diagram of the monkeypox transmission flow is given in figure 1 and is mathematically described as follows:

The schematic SEIR model for the spread of monkeypox transmission flow is given by

$$\frac{dS}{dt} = \mu - (\mu + \pi + \tau_s)S - \left[\beta\beta_1 \frac{I}{N}(1-\pi)(1-\tau_s)\right] S \tag{1}$$

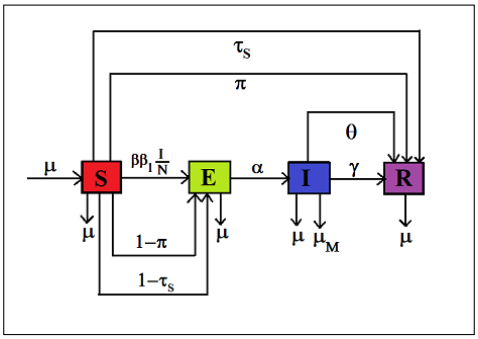

FIGURE 1. SEIR model for the spread of monkeypox transmission flow

$$\frac{dE}{dt} = \left[\beta\beta_1 \frac{I}{N}(1-\pi)(1-\tau_s)\right] S - (\mu+\alpha)E \tag{2}$$

$$\frac{dI}{dt} = -(\mu+\mu_M+\theta+\gamma)I + \alpha E \tag{3}$$

$$\frac{dR}{dt} = (\theta+\gamma)I + (\pi+\tau_S)S - \mu R \tag{4}$$

with $S(0) \geq 0, E(0) \geq 0, I(0) \geq 0, R(0) \geq 0$ as initial conditions.

TABLE 1. Description of parameters

Parameters	Description
β	The force of infection
β_1	Human to human contact rate
γ	Recovery rate
τ_S	Smallpox vaccination
μ	Natural birth/death rate
θ	Treatment effectiveness
π	Following the health protocols
μ_M	Death rate due to monkeypox
α	The rate of exposed human to infected human

4. The Equilibrium Points

There are two equilibrium points which are disease free equilibrium and endemic equilibrium. To find these two equilibrium points each of the equation $(1-4)$ must be equal to zero, where,

$$0 = \mu - (\mu+\pi+\tau_s)S - \left[\beta\beta_1 \frac{I}{N}(1-\pi)(1-\tau_s)\right] S \tag{5}$$

$$0 = \left[\beta\beta_1 \frac{I}{N}(1-\pi)(1-\tau_s)\right] S - (\mu+\alpha)E \tag{6}$$

$$0 = -(\mu+\mu_M+\theta+\gamma)I + \alpha E \tag{7}$$

$$0 = (\theta+\gamma)I + (\pi+\tau_S)S - \mu R \tag{8}$$

with $S(0) \geq 0, E(0) \geq 0, I(0) \geq 0, R(0) \geq 0$.

5. The Disease-free Equilibrium Points for SEIR Model

The points of disease free equilibrium are conditions when there is absence of spread in monkeypox, where E=E^0=0 and I= $I^0 = 0$. From (1) to (4), we get the equations for disease free equilibrium for the SEIR model as follows:
From (5), we have

$$\mu - (\mu+\pi+\tau_s)S - \left[\beta\beta_1 \frac{I}{N}(1-\pi)(1-\tau_s)\right] S = 0$$

$$\Rightarrow\ S = S^0 = \frac{\mu}{\mu+\pi+\tau_S} \tag{9}$$

From (6), we have

$$\left[\beta\beta_1 \frac{I}{N}(1-\pi)(1-\tau_s)\right] S - (\mu+\alpha)E = 0\ \Rightarrow\ (\mu+\alpha)E = 0$$

$$\Rightarrow\ E = E^0 = 0 \tag{10}$$

From (7), we have

$$I = I^0 = 0 \tag{11}$$

From (8), we have

$$(\theta+\gamma)I+(\pi+\tau_S)S-\mu R = 0\ \Rightarrow\ \mu R = (\theta+\gamma)I+(\pi+\tau_S)S\ \Rightarrow\ \mu R = (\pi+\tau_S)S$$

$$\Rightarrow\ R = R^0 = \frac{(\pi+\tau_S)S}{\mu} = \frac{(\pi+\tau_S)\left(\dfrac{\mu}{\mu+\pi+\tau_s}\right)}{\mu}, [\text{ using equation (9)}]$$

$$R = R^0 = \frac{\pi+\tau_S}{\mu+\pi+\tau_S} \tag{12}$$

6. The Endemic Equilibrium Points for SEIR Model

The points of equilibrium for endemic are conditions where there is possibilities of the spread of disease, where S=$S^* \neq 0$, E=$E^* \neq 0$, I=$I^* \neq 0$ and R=$R^* \neq 0$. From (1) to (4), we get the equations for endemic equilibrium for the SEIR model as follows:

$$\text{Equation (5)}\ \Rightarrow\ \mu - (\mu+\pi+\tau_s)S - \left[\beta\beta_1 \frac{I}{N}(1-\pi)(1-\tau_s)\right] S = 0$$

$$\Rightarrow\ S^* = \frac{\mu}{\mu+\pi+\tau_s+\beta\beta_1 \dfrac{I}{N}(1-\pi)(1-\tau_s)} \tag{13}$$

$$\text{Equation (6)} \Rightarrow \left[\beta\beta_1 \frac{I}{N}(1-\pi)(1-\tau_s)\right] S - (\mu+\alpha)E = 0$$

$$\Rightarrow E = \frac{\left[\beta\beta_1 \frac{I}{N}(1-\pi)(1-\tau_s)\right] S}{\mu+\alpha} \text{ [using equation (13)]}$$

$$\Rightarrow E^* = \frac{\mu(\beta\beta_1 \frac{I}{N}(1-\pi)(1-\tau_s))}{(\mu+\alpha)(\mu+\pi+\tau_s+\beta\beta_1 \frac{I}{N}(1-\pi)(1-\tau_s))} \tag{14}$$

$$\text{Equation (7)} \Rightarrow -(\mu+\mu_M+\theta+\gamma)I + \alpha E = 0 \Rightarrow (\mu+\mu_M+\theta+\gamma)I = \alpha E$$

$$\Rightarrow I = \frac{\alpha E}{\mu+\mu_M+\theta+\gamma} \text{ [using equation (14)]}$$

$$\Rightarrow I^* = \frac{\alpha\mu(\beta\beta_1 \frac{I}{N}(1-\pi)(1-\tau_s))}{(\mu+\mu_M+\theta+\gamma)[\mu+\alpha(\mu+\pi+\tau_s+\beta\beta_1 \frac{I}{N}(1-\pi)(1-\tau_s))]} \tag{15}$$

$$\text{Equation (8)} \Rightarrow (\theta+\gamma)I + (\pi+\tau_S)S - \mu R = 0 \Rightarrow \mu R = (\theta+\gamma)I + (\pi+\tau_S)S$$

$$\Rightarrow R = \frac{(\theta+\gamma)I + (\pi+\tau_S)S}{\mu}$$

We cannot substitute I^* and S^* because it is not easy to evaluate R^*. Hence we have

$$R^* = \frac{(\theta+\gamma)I^* + (\pi+\tau_S)S^*}{\mu} \tag{16}$$

7. Basic Reproduction Number for SEIR Model

In our proposed model, E and I compartments are the disease free state whereas S, E, I and R compartments are the endemic state. The basic reproduction number is denoted by R_0, an infection where there is expected number of cases directly caused by one to many cases in a human population, whereas all the individuals are suscepted to infection. This is one of the important parameter to consider the long term behavior of an endemic. It can be described as the total number of secondary cases that a single infected person causes during the course of their infectious agent's whole life. The basic reproduction number for a compartmental model of the spread of pathogens is derived from the next-generation matrix. It is used to determine the basic reproduction number for structured population models for both communicable and non-communicable diseases. It is also used in various analogous computations in multi-type branching models. The basic reproduction number R_0 for the SEIR model (1) - (4) is determined using the next generation matrix.

We consider only the disease class E and I and the differential equations (2) and (3)

$$\frac{dE}{dt} = \left[\beta\beta_1 \frac{I}{N}(1-\pi)(1-\tau_s)\right] S - (\mu+\alpha)E$$
$$\frac{dI}{dt} = -(\mu+\mu_M+\theta+\gamma)I + \alpha E$$

In the disease free equilibrium, since $(S, E, I, R) = (1, 0, 0, 0)$, the Jacobian matrix of $\mathcal{F}$ is $F = \begin{pmatrix} 0 & \frac{\beta\beta_1}{N}(1-\pi)(1-\tau_s)S \\ 0 & 0 \end{pmatrix} = \begin{pmatrix} 0 & \beta\beta_1(1-\pi)(1-\tau_s) \\ 0 & 0 \end{pmatrix}$ and the Jacobian matrix of $\mathcal{V}$ is $V = \begin{pmatrix} -(\mu+\alpha) & 0 \\ \alpha & -(\mu+\mu_M+\theta+\gamma) \end{pmatrix}$, where $\mathcal{F} = \begin{pmatrix} f \\ g \end{pmatrix} = \begin{pmatrix} [\beta\beta_1 \frac{I}{N}(1-\pi)(1-\tau_S)]S \\ 0 \end{pmatrix}$ and
$\mathcal{V} = \begin{pmatrix} -(\mu+\alpha)E \\ -(\mu+\mu_M+\theta+\gamma)I + \alpha E \end{pmatrix}$.

Now $V^{-1} = \begin{pmatrix} \frac{1}{-(\mu+\alpha)} & 0 \\ \frac{-\alpha}{-(\mu+\mu_M+\theta+\gamma)(-(\mu+\alpha))} & \frac{1}{-(\mu+\mu_M+\theta+\gamma)} \end{pmatrix}$ and

$$FV^{-1} = \begin{pmatrix} \frac{(-\alpha)(\beta\beta_1(1-\pi)(1-\tau_s))}{-(\mu+\mu_M+\theta+\gamma)(-(\mu+\alpha))} & \frac{\beta\beta_1(1-\pi)(1-\tau_s)}{-(\mu+\mu_M+\theta+\gamma)} \\ 0 & 0 \end{pmatrix}.$$

The characteristic equation of FV^{-1} is

$$\begin{vmatrix} \frac{(-\alpha)(\beta\beta_1(1-\pi)(1-\tau_s))}{-(\mu+\mu_M+\theta+\gamma)(-(\mu+\alpha))} - \lambda & \frac{\beta\beta_1(1-\pi)(1-\tau_s)}{-(\mu+\mu_M+\theta+\gamma)} \\ 0 & 0-\lambda \end{vmatrix} = 0$$

$$\Rightarrow \left(\frac{(-\alpha)(\beta\beta_1(1-\pi)(1-\tau_s))}{-(\mu+\mu_M+\theta+\gamma)(-(\mu+\alpha))} - \lambda\right)(-\lambda) = 0$$

$$\Rightarrow \left(\frac{(-\alpha)(\beta\beta_1(1-\pi)(1-\tau_s))}{-(\mu+\mu_M+\theta+\gamma)(-(\mu+\alpha))} - \lambda\right) = 0 \quad \text{(or)} \quad (-\lambda) = 0$$

$$\Rightarrow \boxed{\lambda_1 = \frac{\alpha(\beta\beta_1(1-\pi)(1-\tau_s))}{(\mu+\mu_M+\theta+\gamma)(\mu+\alpha)}} \quad \text{and} \quad \boxed{\lambda_2 = 0}.$$

Since λ_1 is the dominant eigen value and the reproduction number is,

$$\boxed{R_0 = \frac{\alpha(\beta\beta_1(1-\pi)(1-\tau_s))}{(\mu+\mu_M+\theta+\gamma)(\mu+\alpha)}} \tag{17}$$

8. Stability Analysis

Theorem 8.1. *If $R_0 < 1$, then the disease free equilibrium D^0 for system (1) - (4) is asymptotically stable and if $R_0 > 1$ then the disease free equilibrium D^0 of the system is unstable.*

Proof. From the equation (1) - (4), we frame the Jacobian matrix J as,

$$J = \begin{bmatrix} [-(\mu+\pi+\tau_S) - \beta\beta_1 \frac{I}{N}(1-\pi)(1-\tau_S)] & 0 & -\left[\frac{\beta\beta_1}{N}(1-\pi)(1-\tau_s)\right]S & 0 \\ \beta\beta_1\frac{I}{N}(1-\pi)(1-\tau_S) & -(\mu+\alpha) & \left[\frac{\beta\beta_1}{N}(1-\pi)(1-\tau_s)\right]S & 0 \\ 0 & \alpha & -(\mu+\mu_M+\theta+\gamma) & 0 \\ \pi+\tau_S & 0 & \theta+\gamma & -\mu \end{bmatrix}$$

Since $I = E = 0$ in disease free equilibrium, the Jacobian matrix J becomes

$$J_0 = \begin{bmatrix} -(\mu+\pi+\tau_S) & 0 & -\left[\frac{\beta\beta_1}{N}(1-\pi)(1-\tau_s)\right]S & 0 \\ 0 & -(\mu+\alpha) & \left[\frac{\beta\beta_1}{N}(1-\pi)(1-\tau_s)\right]S & 0 \\ 0 & \alpha & -(\mu+\mu_M+\theta+\gamma) & 0 \\ \pi+\tau_S & 0 & \theta+\gamma & -\mu \end{bmatrix} \tag{18}$$

The characteristic equation of the Jacobian matrix J_0 given by

$$\begin{vmatrix} -(\mu+\pi+\tau_S)-\lambda & 0 & -\left[\frac{\beta\beta_1}{N}(1-\pi)(1-\tau_s)\right]S & 0 \\ 0 & -(\mu+\alpha)-\lambda & \left[\frac{\beta\beta_1}{N}(1-\pi)(1-\tau_s)\right]S & 0 \\ 0 & \alpha & -(\mu+\mu_M+\theta+\gamma)-\lambda & 0 \\ \pi+\tau_S & 0 & \theta+\gamma & -\mu-\lambda \end{vmatrix} = 0$$

$$\begin{aligned} \Rightarrow (\mu+\pi+\tau_S+\lambda)(\mu+\lambda)[(\mu+\alpha+\lambda)(\mu+\mu_M+\theta+\gamma+\lambda) \\ - \frac{\beta\beta_1}{N}(1-\pi)(1-\tau_s)S\alpha] = 0 \end{aligned} \tag{19}$$

Solving equation (19), we have $\lambda_1 = -(\mu+\pi+\tau_S)$, $\lambda_2 = -\mu$ and

$$\begin{aligned} &(\mu+\alpha+\lambda)(\mu+\mu_M+\theta+\gamma+\lambda) - \frac{\beta\beta_1}{N}(1-\pi)(1-\tau_s)S\alpha = 0 \\ \Rightarrow\ &\lambda^2+\lambda(2\mu+\alpha+\mu_M+\theta+\gamma)+\mu^2+\mu\mu_M+\mu\theta \\ &+\mu\gamma+\alpha\mu+\alpha\mu_M+\alpha\theta+\alpha\gamma-\frac{\beta\beta_1}{N}(1-\pi)(1-\tau_S)S\alpha = 0 \end{aligned} \tag{20}$$

Since $S = 1$ and $N = 1$ in disease free equilibrium, equation (20) becomes

$$\lambda^2+\lambda(2\mu+\alpha+\mu_M+\theta+\gamma)+\mu^2+\mu\mu_M+\mu\theta+\mu\gamma+\alpha\mu+\alpha\mu_M+\alpha\theta+\alpha\gamma-$$

$$\beta\beta_1(1-\pi)(1-\tau_S)\alpha = 0$$

$$\Rightarrow \lambda^2+\lambda(2\mu+\alpha+\mu_M+\theta+\gamma)+(\mu+\alpha)(\mu+\mu_M+\theta+\gamma)-\beta\beta_1(1-\pi)(1-\tau_S)\alpha = 0$$

On solving this quadratic equation to find λ_3 and λ_4, we have

$$\begin{aligned}\lambda &= -\frac{1}{2}\Big[(2\mu+\alpha+\mu_M+\theta+\gamma) \pm \sqrt{(2\mu+\alpha+\mu_M+\theta+\gamma)^2} \\ &\quad \overline{-4[(\mu+\alpha)(\mu+\mu_M+\theta+\gamma)-\beta\beta_1(1-\pi)(1-\tau_S)\alpha]}\Big] \\ &= -\frac{1}{2}\Big[(2\mu+\alpha+\mu_M+\theta+\gamma) \pm \sqrt{(2\mu+\alpha+\mu_M+\theta+\gamma)^2} \\ &\quad \overline{-4[(\mu+\alpha)(\mu+\mu_M+\theta+\gamma)+4(\mu+\alpha)(\mu+\mu_M+\theta+\gamma)R_0}\Big]\end{aligned}$$

$$\begin{aligned}\Rightarrow \quad \lambda &= -\frac{1}{2}\left[(2\mu+\alpha+\mu_M+\theta+\gamma)\right. \\ &\quad \left. \pm \sqrt{(2\mu+\alpha+\mu_M+\theta+\gamma)^2+4(\mu+\alpha)(\mu+\mu_M+\theta+\gamma)(R_0-1)}\right].\end{aligned}$$

From this, we see that
(i). When $R_0 = 1$, we have $\lambda_1 < 0, \lambda_2 < 0, \lambda_3 = 0$ and $\lambda_4 < 0$
(ii). When $R_0 > 1$ we have $\lambda_1 < 0, \lambda_2 < 0, \lambda_3 > 0$ and $\lambda_4 < 0$
(iii). When $R_0 < 1$ we have $\lambda_1 < 0, \lambda_2 < 0, \lambda_3 < 0$ and $\lambda_4 < 0$.

Therefore, for the disease free equilibrium D^0 is asymptotically stable when $R_0 < 1$ and is unstable when $R_0 > 1$. □

Theorem 8.2. *If $R_0 > 1$, then the endemic equilibrium D^* for the system* (1) - (4) *is asymptotically stable.*

Proof. From the equation (1) - (4), we obtain the Jacobian matrix J_1 as follows:

$$J_1 = \begin{bmatrix} [-(\mu+\pi+\tau_S)-\beta\beta_1 \frac{I}{N}(1-\pi)(1-\tau_S)] & 0 & -\left[\frac{\beta\beta_1}{N}(1-\pi)(1-\tau_s)\right]S & 0 \\ \beta\beta_1\frac{I}{N}(1-\pi)(1-\tau_S) & -(\mu+\alpha) & \left[\frac{\beta\beta_1}{N}(1-\pi)(1-\tau_s)\right]S & 0 \\ 0 & \alpha & -(\mu+\mu_M+\theta+\gamma) & 0 \\ \pi+\tau_S & 0 & \theta+\gamma & -\mu \end{bmatrix}$$

We assume that
$j_1 = (\mu+\pi+\tau_S)+\beta\beta_1\frac{I}{N}(1-\pi)(1-\tau_S),\ j_2 = \left[\frac{\beta\beta_1}{N}(1-\pi)(1-\tau_s)\right]S,$
$j_3 = \beta\beta_1\frac{I}{N}(1-\pi)(1-\tau_S),\ j_4 = (\mu+\alpha),\ j_5 = \left[\frac{\beta\beta_1}{N}(1-\pi)(1-\tau_s)\right]S$
$j_6 = \alpha,\ j_7 = (\mu+\mu_M+\theta+\gamma),\ j_8 = \pi+\tau_S$ and $j_9 = \theta+\gamma.$

Then the Jacobian matrix J_1 as becomes:

$$J_1 = \begin{bmatrix} -j_1 & 0 & -j_2 & 0 \\ j_3 & -j_4 & j_5 & 0 \\ 0 & j_6 & -j_7 & 0 \\ j_8 & 0 & j_9 & -\mu \end{bmatrix} \tag{21}$$

The characteristic equation of the Jacobian matrix J_1 given by

$$|J_1 - \lambda I| = \begin{vmatrix} -j_1 - \lambda & 0 & -j_2 & 0 \\ j_3 & -j_4 - \lambda & j_5 & 0 \\ 0 & j_6 & -j_7 - \lambda & 0 \\ j_8 & 0 & j_9 & -\mu - \lambda \end{vmatrix} = 0$$

$$\Rightarrow (-j_1 - \lambda)[(-j_4 - \lambda)[(-j_7 - \lambda)(-\mu - \lambda)] - j_5(j_6(-\mu - \lambda))] = 0$$

$$\Rightarrow (\lambda + j_7)(\lambda + \mu)[(\lambda^2 + \lambda(j_1 + j_4) + j_1 j_4 + j_5 j_6] = 0$$

$$\Rightarrow (\lambda + j_7) = 0,\ (\lambda + \mu) = 0 \text{ and } [(\lambda^2 + \lambda(j_1 + j_4) + j_1 j_4 + j_5 j_6] = 0$$

$$\Rightarrow \lambda_1 = -j_7,\ \lambda_2 = -\mu \text{ and } [(\lambda^2 + \lambda(j_1 + j_4) + j_1 j_4 + j_5 j_6] = 0$$

It is easy to see that $\lambda_1 = -(\mu + \mu_M + \theta + \gamma)$ and $\lambda_2 = -\mu$ are the two eigenvalues of J_1. A negative real part is present in the other two roots, $j_1 + j_4 > 0$ and $j_1 j_4 + j_5 j_6 > 0$

$$j_1 + j_4 = [(\mu + \pi + \tau_S) + \beta\beta_1 \frac{I}{N}(1 - \pi)(1 - \tau_S)] + (\mu + \alpha) \tag{22}$$

Substitute (15) in equation (22), we have

$$j_1 + j_4 = \Bigg[(\mu + \pi + \tau_S) + \frac{\beta\beta_1}{N}(1 - \pi)(1 - \tau_S) \frac{\alpha\mu \left[\beta\beta_1 \frac{I}{N}(1 - \pi)(1 - \tau_s)\right]}{(\mu + \mu_M + \theta + \gamma)\left[(\mu + \alpha)(\mu + \pi + \tau_S + \beta\beta_1 \frac{I}{N}(1 - \pi)(1 - \tau_s))\right]}\Bigg] + (\mu + \alpha) \tag{23}$$

Now substitute equation (17) in (23), we have $j_1 + j_4$

$$= (\mu + \pi + \tau_S) + (\mu + \alpha)(\mu + \mu_M + \theta + \gamma) R_0 \left[\beta\beta_1 \frac{I}{N}(1 - \pi)(1 - \tau_s)\right](\mu + \mu_M + \theta + \gamma)\left[(\mu + \alpha)(\mu + \pi + \tau_S + \beta\beta_1 \frac{I}{N}(1 - \pi)(1 - \tau_s))\right] + (\mu + \alpha)$$

$$= (\mu + \pi + \tau_S) + R_0 \frac{\mu\beta\beta_1 \frac{I}{N}(1 - \pi)(1 - \tau_S)}{\mu + \pi + \tau_S + \beta\beta_1 \frac{I}{N}(1 - \pi)(1 - \tau_S)} + (\mu + \alpha)$$

$$\boxed{j_1 + j_4 > 0} \quad \text{if} \quad R_0 > 0$$

$$\begin{aligned} j_1j_4 + j_5j_6 &= (\mu+\pi+\tau_S) + \beta\beta_1\frac{I}{N}(1-\pi)(1-\tau_S) + \frac{\beta\beta_1}{N}(1-\pi)(1-\tau_S)S\alpha \\ &= [(\mu+\pi+\tau_S) + \beta\beta_1\frac{I}{N}(1-\pi)(1-\tau_S)] + (\mu+\alpha) \\ &\quad (\mu+\mu_M+\theta+\gamma)R_0S \end{aligned} \tag{24}$$

Substitute (13) and (15) in equation (24), we have $j_1j_4 + j_5j_6$

$$\begin{aligned} &= (\mu+\pi+\tau_S) + \frac{\beta\beta_1}{N}(1-\pi)(1-\tau_S) \\ &\left(\frac{\alpha\mu\left[\beta\beta_1\frac{I}{N}(1-\pi)(1-\tau_s)\right]}{(\mu+\mu_M+\theta+\gamma)\left[(\mu+\alpha)(\mu+\pi+\tau_S+\beta\beta_1\frac{I}{N}(1-\pi)(1-\tau_s))\right]} \right) \\ &+ (\mu+\alpha)(\mu+\mu_M+\theta+\gamma)R_0\left(\frac{\mu}{\mu+\pi+\tau_S+\beta\beta_1\frac{I}{N}(1-\pi)(1-\tau_S)} \right) \\ &= \frac{\alpha\mu\left[\beta\beta_1\frac{I}{N}(1-\pi)(1-\tau_s)\right]}{(\mu+\mu_M+\theta+\gamma)\left[(\mu+\alpha)(\mu+\pi+\tau_S+\beta\beta_1\frac{I}{N}(1-\pi)(1-\tau_s))\right]} \\ &+ R_0\mu \end{aligned}$$

That is $j_1j_4 + j_5j_6$

$$= \mu\left(\frac{\alpha\left[\beta\beta_1\frac{I}{N}(1-\pi)(1-\tau_s)\right]}{(\mu+\mu_M+\theta+\gamma)\left[(\mu+\alpha)(\mu+\pi+\tau_S+\beta\beta_1\frac{I}{N}(1-\pi)(1-\tau_s))\right]} + R_0 \right) \tag{25}$$

In the endemic equilibrium $I = I^* \neq 0$, so we take $I = 1$ and we substitute equation (17) in (25).

$$\begin{aligned} j_1j_4 + j_5j_6 &= \mu\left[\frac{(\mu+\alpha)(\mu+\mu_M+\theta+\gamma)R_0}{\mu+\pi+\tau_S+\frac{\beta\beta_1}{N}(1-\pi)(1-\tau_S)} + R_0 \right] \\ &= \mu R_0\left[\frac{(\mu+\alpha)(\mu+\mu_M+\theta+\gamma)}{\mu+\pi+\tau_S+\frac{\beta\beta_1}{N}(1-\pi)(1-\tau_S)} + 1 \right] \end{aligned}$$

It is easy to see that $\boxed{j_1j_4 + j_5j_6 > 0 \quad \text{if} \quad R_0 > 1}$

When $R_0 < 1$ it is stable in disease free equilibrium and when $R_0 > 1$ it is unstable. Whereas in the endemic equilibrium when $R_0 > 1$ it is stable and

hence we say that it is locally asymptotically stable.

□

9. Results and Discussion

A discussion is executed using the initial values for S, E, I, R and N which is evaluated from the data of United states during May 06, 2022 to September 07, 2022 and is given in the table below. The parameters are calculated based on the current cases due to monkeypox.

The system $(1) - (4)$ is simulated for various set of parameters satisfying the conditions of the stability of disease free equilibrium and endemic equilibrium. In the numerical simulation, we have represented the x axis as months from May to September and y axis as the total number of cases who are affected by monkeypox virus in the United States. We have taken the four compartments as suscepted, exposed, infected and recovered.

Figure 2 shows the SEIR graph of monkeypox in United States from May 06, 2022 to September 07, 2022, figure 3 shows the disease free equilibrium's stability for the suscepted monkeypox population, figure 4 shows the disease free equilibrium's stability for the exposed monkeypox population, figure 5 shows the disease free equilibrium's stability for the infected monkeypox population, figure 6 shows the disease free equilibrium's stability for the recovered monkeypox population, figure 7 shows the disease free equilibrium's stability for the four compartment model suscepted, exposed, infected and recovered when $R_0 < 1$, figure 8 shows the endemic equilibrium's stability for the suscepted monkeypox population, figure 9 shows the endemic equilibrium's stability for the exposed monkeypox population, figure 10 shows the endemic equilibrium's stability for the infected monkeypox population, figure 11 shows the endemic equilibrium's stability for the recovered monkeypox population and figure 12 shows the endemic equilibrium's stability for the four compartment model suscepted, exposed, infected and recovered when $R_0 > 1$. When comparing the figure 7 and 12 we can clearly see that the suscepted cases decreases at a certain month from July and stays constant from August in figure 7 and in figure 12 we can clearly see that the suscepted cases decreases at a certain month from August and keep on going. In figure 7 we can clearly see that the exposed cases gradually decreases at a certain month from June and keep on going and in figure 12 we can clearly see that the exposed cases decreases from September and keep on going. In figure 7 we can clearly see that the infected cases decreases rapidly from July and stops in mid of September and in figure 12 we can clearly see that the infected cases decreases from June and it stays constant from July. In figure 7 we can clearly see that the recovered cases increases and from June it gradually decreases and stays constant. In figure 12 we can clearly see that the recovered cases increases from may and stays constant from August.

TABLE 2. Initial values of the SEIR model for monkeypox in US

Variable	Value	Reference
$N(0)$	412,065	[21]
$S(0)$	148,343	[19]
$E(0)$	81,336	[7]
$I(0)$	20,700	[3]
$R(0)$	12,420	[7]

TABLE 3. Parameter value of the SEIR model for monkeypox in US

Parameter	value	Description	Reference
β	0.0416	The force of infection	[4]
β_1	0.00006	Human to human contact rate	[20]
γ	0.99	Recovery rate	[3]
τ_S	0.622	Smallpox vaccination	[24]
μ	0.0058	Natural birth/death rate	[21]
θ	0	Treatment effectiveness	[14]
π	0.5	Following the health protocols	Assumption
μ_M	0.01	Death rate due to monkeypox	[3]
α	0.2	The rate of exposed human to infected human	[15]

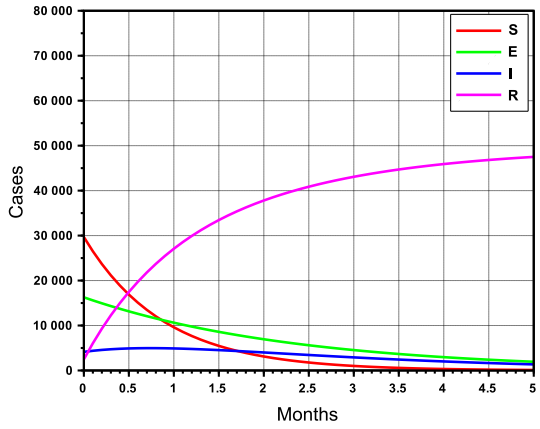

FIGURE 2. SEIR graph of Monkey Pox in US from May 06 2022 to September 07 2022

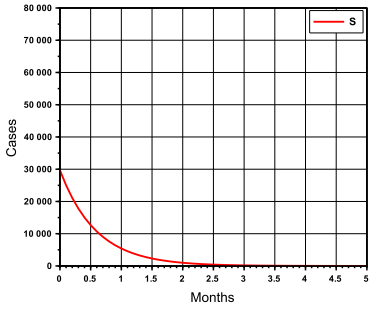

FIGURE 3. Variation of the suspected cases in disease free equilibrium

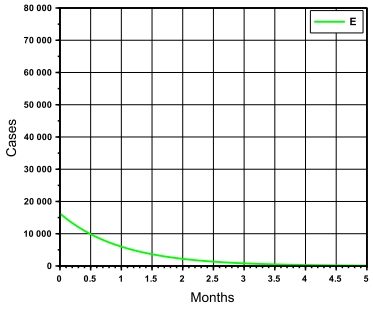

FIGURE 4. Variation of the exposed cases in disease free equilibrium

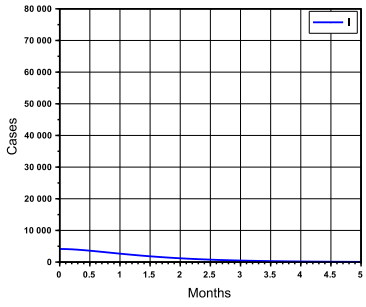

FIGURE 5. Variation of the infected cases in disease free equilibrium

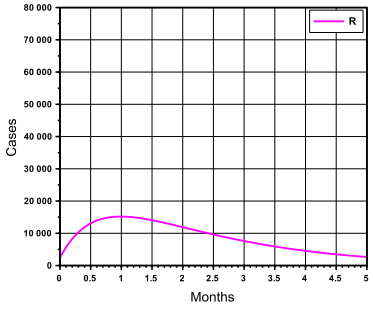

FIGURE 6. Variation of the recovered cases in disease free equilibrium

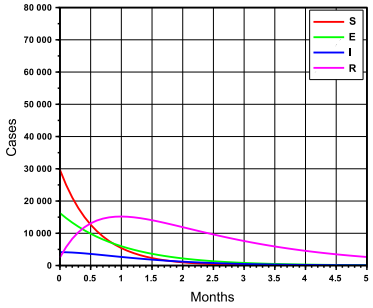

FIGURE 7. Stability of the disease free equilibrium when $R_0 < 1$

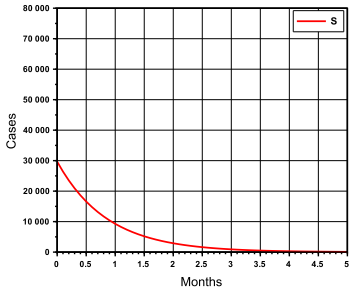

FIGURE 8. Variation of the suspected cases in endemic equilibrium

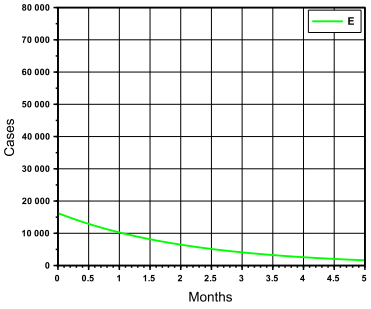

FIGURE 9. Variation of the exposed cases in endemic equilibrium

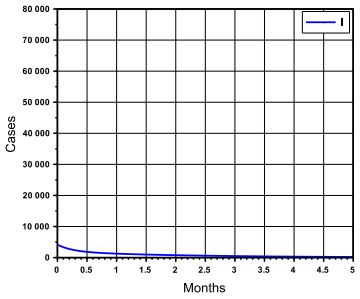

FIGURE 10. Variation of the infected cases in endemic equilibrium

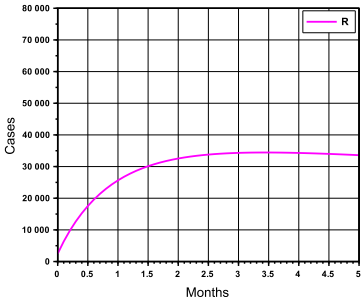

FIGURE 11. Variation of the recovered cases in endemic equilibrium

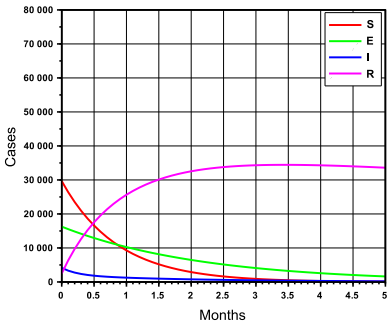

FIGURE 12. Stability of endemic equilibrium when $R_0 > 1$

10. Conclusion

A study has been done using an SEIR model for the spread of monkeypox in United states taking into account the factors of smallpox vaccination and implementation of following the health protocols has been performed. The points of disease-free equilibrium and endemic equilibrium are asymptotically stable for $R_0 < 1$ and $R_0 > 1$ respectively. Based on our discussion, it is found that smallpox vaccination and following the health protocols have a significant effect in stopping or slowing the spread of monkeypox virus in United States because United States is the 1^{st} among the world outbreak [25]. Likewise, smallpox vaccination has an effect in stopping or slowing the rate of infection of monkeypox virus but not as much as following the health protocols. Our SEIR model predicts, lowering transmission to reduce the number of new confirmed cases and illnesses. The parameter τ_S shows that the people who get smallpox vaccination due to monkeypox infection will soon be recovering soon, the parameter π shows that the people who are following the health protocols will not get affected soon and it may reduce the infection rate. We have shown this fact in our result and discussion part when there is a disease free equilibrium and endemic equilibrium. As there is a decrease in the monkeypox infection, soon United States will overcome from it, if the consumption of smallpox vaccination percentage increases in the human population. If smallpox vaccination is not provided to the people with monkeypox infection it may lead to severeness and it may cause death. The most important is following the health protocols and social distancing from the people who has got the symptoms of monkeypox and who has monkeypox because monkeypox is a communicable disease and it spread rapidly from one person to another. So we suggest the people to follow the health protocols, social distancing and vaccinate in the beginning of the monkeypox infection as soon as possible to stop spreading, to reduce the number of death cases, to become less severe among people and the effect of monkeypox in United States. Since

monkeypox outbreak in the United States is part of the larger outbreak. The United States was the fourth country outside of the African countries with endemic monkeypox. As of August 2022, monkeypox has spread to all 50 states in the United States. The United States has the highest number of monkeypox cases in the world. The first death was reported in United states on August 30, 2022. The burden of monkeypox virus infection has been increased due to the quick spread in United states and our model would help to reduce monkeypox spread and reduce the number of deaths.

Conflicts of interest : The authors declare no conflict of interest.

Data availability : Not applicable

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S. Shalini Priya received M.Sc. from SRM Institute of Science and Technology and currently pursuing Ph.D. at SRM Institute of Science and Technology. Her research interests include fuzzy optimization, operation research, mathematical biology and mathematical modelling.

Department of Mathematics, SRM Institute of Science and Technology, Kattankulathur, Chennai 603-203, India.
e-mail: ss1305@srmist.edu.in

K. Ganesan received M.Sc. from Bharathidasan University and Ph.D. from University of Madras. He is currently a professor at SRM Institute of Science and Technology. His research interests are Fuzzy Optimization, Operations Research, Interval Analysis, Ordinary and Partial Differential Equations.

Department of Mathematics, SRM Institute of Science and Technology, Kattankulathur, Chennai 603-203, India.
e-mail: ganesank@srmist.edu.in