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MATHEMATICAL MODELING AND ANALYSIS OF A MONKEYPOX MODEL

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Abstract. In this work, we present a continuous mathematical model, SEIQR, for monkeypox infection. We study the dynamical behaviour of this model and discuss the basic properties of the system. By constructing Lyapunov functions and using Routh-Hurwitz criteria, the stability analysis of the model confirms that the system is globally, as well as locally, asymptotically stable at the free equilibrium E_0 when $R_0 < 1$. When $R_0 > 1$, the endemic equilibrium E^* exists, and the system is globally, as well as locally, asymptotically stable at the endemic equilibrium E^* . Additionally, we conduct a sensitivity analysis of the model parameters to identify the parameters that have a significant impact on the reproduction number R_0 . Finally, we perform numerical simulations to confirm the theoretical analysis using Matlab.

Keywords: monkeypox model; analysis; stability.

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1. INTRODUCTION

Smallpox, caused by the simian orthopoxvirus, was one of the deadliest endemo-epidemic diseases for a long time. It is a viral zoonotic disease, meaning it can be transmitted to humans by animals. Another mode of transmission is through close contact with a person who has a rash due to monkey pox, including face-to-face and skin-to-skin contact. Monkeypox has been declared a global disease by the WHO, as it affects not only African countries but also the rest of the world. In 2003, the first outbreak of smallpox in the monkey population reached the United States of America. This outbreak was attributed to contact with infected domestic prairie dogs that had been housed with cricetoma savannas and dormice imported from Ghana. Nearly 70 cases were identified in the USA between July and November 2021 and in the United Kingdom in May 2022. The majority of monkeypox cases were reported in non-endemic countries. Numerous studies are being conducted to better understand the epidemiology, infection sources, and transmission modes [1].

Many specialists state that monkeypox is primarily manifested by fever, rashes, and swollen lymph nodes. It can lead to a range of medical complications, with severe cases occurring more frequently in children and being associated with the extent of exposure to the virus, the patient's health status, and the nature of the complications. Underlying immune deficiency can contribute to an unfavourable progression. Individuals under the age of 40 to 50 (varying by country) may be more susceptible to monkeypox due to the discontinuation of smallpox vaccination campaigns worldwide after the eradication of the disease, even though past vaccination against smallpox provided protection [1,2].

Monkeypox can lead to various complications such as secondary infections, bronchopneumonia, sepsis, encephalitis, and corneal infection that can result in vision loss. The extent to which the infection may be asymptomatic is not yet known. Historically, the case fatality rate of monkeypox has ranged from 0 to 11% in the general population, with higher rates in young children. In recent times, the case fatality rate has been approximately 3 to 6% [1].

Since the epidemic started spreading in early May 2022, the WHO has taken the situation very seriously, rapidly releasing clinical and public health guidance, actively engaging with societies, and bringing together hundreds of scientists and researchers to accelerate monkeypox

research and development, including the exploration of new diagnostic tools, vaccines, and treatments [3].

Over 16,700 confirmed cases of monkeypox have been identified in more than 75 countries, but the actual number is likely higher, according to the UN World Health Agency. Five deaths, all in Africa, have been reported, and there have been 81 cases involving children under the age of 17 reported internationally so far. According to the latest WHO count, the number of new cases reported each week in the world has increased by 48% between July 18 and 24 (4045 cases) compared to 2740 cases between July 11 and 17 [4].

As of August 22, the United States (14,049 cases) leads the ten countries with the highest number of cumulative cases worldwide, followed by Spain (6,119), Brazil (3,450), Germany (3,295), the United Kingdom (3,225), France (2,889), Canada (1,168), the Netherlands (1,090), Peru (937), and Portugal (810). These countries together account for almost 90% of the reported cases worldwide, totalling 44,464 cases of monkeypox. The WHO reports a total of 13 deaths as of August 25, 2022 [5].

Many studies and research in social, medical, and political sciences have focused on this topic and other related topics [6, 7, 8, 9, 15, 16]. However, the mathematical studies and research on this topic are still limited, with most of them focusing on the statistical and stochastic aspects of the disease [10, 11]. Mathematical models can be used to analyze the spread of infectious diseases or the social behaviour of individuals [12, 13].

Several researchers have utilized mathematical analysis to study monkeypox dynamics. For instance, they have developed and analyzed a deterministic monkeypox mathematical model, and their results suggest that isolating infected individuals in the human population reduces disease transmission. In a similar vein, [7] established a mathematical model for the dynamics of the smallpox virus in monkey transmission, presenting it as a system of interactions represented by a system of nonlinear differential equations. Numerical simulations indicate that the individuals' immune status influences their recovery after being infected with the monkeypox virus. [8] Investigated the transmission dynamics and control of the monkeypox virus in the population using both a classical model and a fractional-order model, exploring how the fractional-order parameter affects monkeypox dynamics and whether it can be used as a control

parameter. Additionally, in [9]’s study, numerical simulations carried out on the model revealed that infectious individuals in human and non-human primate populations will disappear over time due to the proposed interventions during the study period.

We will propose a continuous mathematical model that describes the spread of monkeypox using differential equations. The virus primarily spreads through contact between humans and animals, particularly through direct contact with an infected person. First, we will test the local stability of the model at the disease-free equilibrium and the endemic equilibrium. Then, we will examine the overall stability of the model. Since data collection often involves errors and parameter values are assumed, we will also conduct sensitivity analysis of the model parameters to identify those that have a significant impact on the reproduction number R_0 .

This article is organized as follows: Section 1 presents the formulation of the proposed model and its basic properties. In Section 2, we discuss the equilibrium points of the model. Section 3 covers the analysis of local and global stability of the equilibrium point. The sensitivity analysis of the parameters is discussed in Section 4. Section 5 presents numerical simulations that confirm the theoretical results. Finally, we conclude by discussing the obtained results.

2. MATHEMATICAL MODEL FORMULATION AND PROPERTIES OF BASE

2.1. Mathematical model. In this section, we present a continuous mathematical model of the disease denoted by $S(t), E(t), I(t), Q(t)$, and $R(t)$, where the population under investigation is divided into five compartments:

The compartment S: Represents susceptible individuals who are at risk of acquiring the small-pox virus. This compartment is increased by the birth rate denoted as Λ and decreased by effective contact with asymptomatic infected cases at a rate β , as well as natural mortality at a rate μ .

The compartment E: Consists of asymptomatic infected cases. It is increased by susceptible individuals becoming asymptomatic infected people at a rate β . This compartment is reduced when asymptomatic infected people develop symptoms and become carriers of the virus at a rate α , and also decreases due to natural mortality at a rate μ .

The compartment I: Contains individuals with symptoms and carriers of the virus. It is increased by asymptomatic infected people developing symptoms at a rate α . This compartment

decreases when infected individuals are admitted to the hospital at a rate δ , and also decreases due to natural mortality at a rate μ and disease-related deaths at a rate λ .

The compartment Q: Represents infected individuals who are hospitalized. This compartment increases at a rate δ , which represents the transmission coefficient of infected persons to hospitalized cases. It decreases as people recover at a rate θ and due to natural mortality at a rate μ .

The compartment R: Contains individuals who have recovered. It increases with the recruitment of people who have been treated in the hospital at a rate θ and decreases due to natural mortality at a rate μ .

The total size of the population is denoted as N , given by the equation:

$$N(t) = S(t) + E(t) + I(t) + Q(t) + R(t),$$

and it is assumed to be constant.

The diagram in Figure 1 illustrates the flow of individuals among compartments, represented by directed arrows:

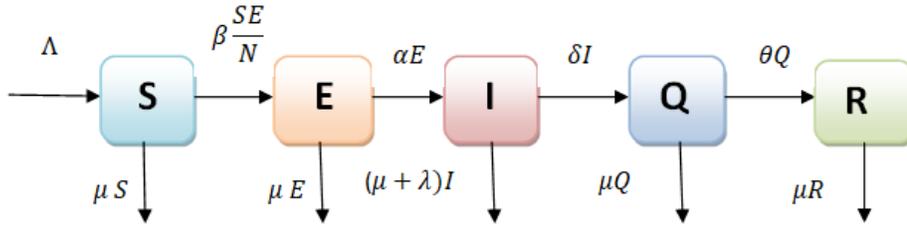


FIGURE 1 -Relations between the five compartments $S(t), E(t), I(t), Q(t), R(t)$

The dynamics of this model is governed by the following differential equation system:

$$1 \left\{ \begin{array}{l} \dot{S}(t) = \Lambda - \beta \frac{S(t)E(t)}{N} - \mu S(t) \\ \dot{E}(t) = \beta \frac{S(t)E(t)}{N} - (\alpha + \mu)E(t) \\ \dot{I}(t) = \alpha E(t) - (\delta + \mu + \lambda)I(t) \\ \dot{Q}(t) = \delta I(t) - (\theta + \mu)Q(t) \\ \dot{R}(t) = \theta Q(t) - \mu R(t) \end{array} \right.$$

Where $S(0) \geq 0, E(0) \geq 0, I(0) \geq 0, Q(0) \geq 0, R(0) \geq 0$ the given initial states.

2.2. Basic Properties: System (1) describes the human population and therefore it is necessary to prove that all the solutions of system (1) with positive initial data will remain positive for all time $t > 0$ and are bounded. This will be established by the following theorem and lemma.

a) Positivity of the model solutions:

Theorem 1. *If $S(0) \geq 0$, $E(0) \geq 0$, $I(0) \geq 0$, $Q(0) \geq 0$, $R(0) \geq 0$, the Solutions $S(t), E(t), I(t), Q(t), R(t)$ of system (1) are positive for all $t > 0$.*

Demonstration:

It follows from the first equation of system (1) that:

$$\begin{aligned} \frac{dS}{dt} &= \lambda - \beta \frac{S(t)E(t)}{N} - \mu S(t) \\ \implies \frac{dS(t)}{dt} + \beta \frac{S(t)E(t)}{N} + \mu S(t) &= \Lambda \geq 0 \\ \implies \frac{dS(t)}{dt} + (\beta \frac{E(t)}{N} + \mu)S(t) &\geq 0 \end{aligned}$$

We multiply the inequality by

$$\exp\left(\int_0^t \beta \frac{E(s)}{N} + \mu ds\right)$$

We obtain:

$$\frac{dS(t)}{dt} \exp\left(\int_0^t \beta \frac{E(s)}{N} + \mu ds\right) + (\beta \frac{E(t)}{N} + \mu) \exp\left(\int_0^t \beta \frac{E(s)}{N} + \mu ds\right) S(t) \geq 0$$

So

$$\frac{d}{dt} \left(S(t) \exp\left(\int_0^t \beta \frac{E(s)}{N} + \mu ds\right) \right) \geq 0$$

Let's integrate this inequality

$$S(t) \geq S(0) \exp\left(-\int_0^t \beta \frac{E(s)}{N} + \mu ds\right)$$

Then $S(t)$ is positive.

Similarly for the other equations we find,

$$E(t) \geq E(0) \exp\left(-\int_0^t \beta \frac{S(s)}{N} - \alpha - \mu ds\right) \geq 0;$$

$$I(t) \geq I(0) e^{-(\delta+\lambda+\mu)t} \geq 0;$$

$$Q(t) \geq Q(0) e^{-(\theta+\mu)t} \geq 0;$$

$$R(t) \geq R(0) e^{-\mu t} \geq 0;$$

b) Invariant region:

Lemma: The feasible region Ω defined by:

$$\Omega = \{S(t), E(t), I(t), Q(t), R(t), S + E + I + Q + R \leq \frac{\Lambda}{\mu}\}$$

With the conditions $S(0) \geq 0, E(0) \geq 0, I(0) \geq 0, Q(0) \geq 0, R(0) \geq 0$.

Demonstration:

We add the system equations (1) we find:

$$\frac{dN}{dt} = \dot{S} + \dot{E} + \dot{I} + \dot{Q} + \dot{R}$$

$$\frac{dN}{dt} \leq \Lambda - \mu N$$

According to Gronwall's lemma we have:

$$N(t) \leq N(0) \exp(-\mu t) + \frac{\Lambda}{\mu} (1 - \exp(-\mu t))$$

Where $N(0)$ represents the initial values of the total population.

So $\limsup_{t \rightarrow +\infty} N(t) = \frac{\Lambda}{\mu}$. This implies that the region Ω a positively invariant set for system (1). We therefore only need to consider the dynamics of the system on the set Ω .

The first three equations of system (1) are independent of the variables Q and R . Therefore, the dynamics of the system of equations (1) is equivalent to the dynamics of the system of equations:

$$2 \begin{cases} \dot{S}(t) &= \Lambda - \beta \frac{S(t)E(t)}{N} - \mu S(t) \\ \dot{E}(t) &= \beta \frac{S(t)E(t)}{N} - (\alpha + \mu)E(t) \\ \dot{I}(t) &= \alpha E(t) - (\delta + \mu + \lambda)I(t) \end{cases}$$

3. THE EQUILIBRIUM POINTS:

System (1) has the following two equilibrium points:

3.1. Point of equilibrium without disease. Is giving by $E_{eq}^0(\frac{\Lambda}{\mu}, 0, 0)$ and is reached in the absence of disease $E(t) = I(t) = Q(t) = R(t) = 0$.

3.2. Point of equilibrium with disease. is given by $E_{eq}^*(S^*, E^*, I^*, Q^*, R^*)$ where

$$\begin{aligned} S^* &= \frac{\Lambda}{\mu R_0}, \\ E^* &= \frac{\mu N}{\beta}(R_0 - 1), \\ I^* &= \frac{\alpha \mu N(R_0 - 1)}{\beta(\delta + \lambda + \mu)}. \end{aligned}$$

and

$$R_0 = \frac{\beta \Lambda}{\mu N(\alpha + \mu)}$$

R_0 is the base reproduction number which measures the average number of newly infected individuals generated by a single infected individual in a population of susceptible individuals.

4. STABILITY AND SENSITIVITY ANALYSIS OF MODEL PARAMETERS

In this section, we will study system's stability behavior at equilibrium with and without disease.

4.1. Local stability analysis. We analyze the local stability of the equilibrium points E_{eq}^0 and E_{eq}^* .

4.1.1. Disease-Free Equilibrium Point:

Theorem 2. *Equilibrium points without disease $E_{eq}^0(\frac{\Lambda}{\mu}, 0, 0)$ of the system (2) is asymptotically stable if $R_0 < 1$ and is unstable if $R_0 > 1$.*

Demonstration: The Jacobian matrix at E_{eq}^0 is given by:

$$J(E_{eq}^0) = \begin{pmatrix} -\mu & -\frac{\beta \Lambda}{N\mu} & 0 \\ 0 & \frac{\beta \Lambda}{N\mu} - (\alpha + \mu) & 0 \\ 0 & \alpha & -(\delta + \lambda + \mu) \end{pmatrix}$$

The characteristic equation of this matrix is given by $\det(J(E_{eq}^0) - \zeta I_3) = 0$, where I_3 is an identity matrix of order 3.

$$\det(J(E_{eq}^0) - \zeta I_4) = -(\mu + \zeta) \left[\left(\frac{\beta \Lambda}{N\mu} - (\mu + \alpha) - \zeta \right) (-(\delta + \mu + \lambda) - \zeta) \right] = 0.$$

So, the eigenvalues of the characteristic equation of $J(E_{eq}^0)$ are:

$$\zeta_1 = -\mu$$

$$\zeta_2 = (\alpha + \mu)(R_0 - 1)$$

$$\zeta_3 = -(\delta + \mu + \lambda)$$

where

$$R_0 = \frac{\beta\Lambda}{\mu N(\alpha + \mu)}$$

Therefore, all eigenvalues of the characteristic equation $J(E_{eq}^0)$ are clearly negative real numbers if $R_0 < 1$.

So, we conclude that Equilibrium point without disease $E_{eq}^0(\frac{\Lambda}{\mu}, 0, 0)$ of system (2) is asymptotically stable if $R_0 < 1$ and is unstable if $R_0 > 1$.

4.1.2. Point of equilibrium with disease.

Theorem 3. *Equilibrium points with disease $E_{eq}^*(S^*, E^*, I^*, Q^*, R^*)$ of system (2) is asymptotically stable if $R_0 > 1$ and is unstable if $R_0 < 1$.*

Demonstration: The Jacobian matrix at E_{eq}^* is given by:

$$J(E_{eq}^*) = \begin{pmatrix} -\mu R_0 & -\frac{\beta\Lambda}{\mu NR_0} & 0 \\ \mu(R_0 - 1) & \frac{\beta\Lambda}{\mu NR_0} - (\alpha + \mu) & 0 \\ 0 & \alpha & -(\delta + \lambda + \mu) \end{pmatrix}$$

We notice that the characteristic equation $\varphi(\zeta)$ of $J(E_{eq}^*)$

$$\varphi(\zeta) = \zeta^3 + a_1\zeta^2 + a_2\zeta + a_3$$

where,

$$a_1 = \delta + \lambda + \mu(\Lambda + R_0)$$

$$a_2 = (\delta + \mu + \lambda)\mu R_0 - \frac{\beta\Lambda}{NR_0}(1 - R_0)$$

$$a_3 = -\frac{\beta\Lambda}{NR_0}(\delta + \mu + \lambda)(1 - R_0)$$

According to the Routh-Hurwitz criterion, the system (2) is locally asymptotically stable if $a_1 > 0, a_2 > 0, a_3 > 0$ and $a_1 a_2 > a_3$. Therefore, $E_{eq}^*(S^*, E^*, I^*, Q^*, R^*)$ of system (2) is asymptotically stable if $R_0 > 1$.

4.2. global stability.

4.2.1. *global stability without disease.* To show that the system (2) is globally asymptotically stable, we use the Lyapunov's function theory for equilibrium points without disease and equilibrium with disease. First, we present the global stability of the disease-free equilibrium E_{eq}^0 .

Theorem 4. *Equilibrium points without disease E_{eq}^0 of system (2) is globally asymptotically stable if $R_0 \leq 1$ and is unstable if $R_0 > 1$.*

Demonstration: We consider the following Lyapunov function:

$$V : \Gamma \longrightarrow \mathbb{R}$$

$$V(S, E, I) = E$$

where $\Gamma = \{(S, E, I) \in \Gamma / S > 0, E > 0, I > 0\}$ Then the derivative of the Lyapunov function is given by:

$$\begin{aligned} \frac{dV}{dt} &= \frac{dE}{dt} = \left(\frac{\beta\Lambda}{\mu N} - (\alpha + \mu) \right) E \\ \frac{dV}{dt} &= (\alpha + \mu)(R_0 - 1)E \end{aligned}$$

So, $\frac{dV}{dt} \leq 0$ if $R_0 \leq 1$ also $\frac{dV}{dt} = 0$ if and only if $E = 0$ then, E_{eq}^0 is globally asymptotically.

4.2.2. *global stability with disease.*

Theorem 5. *Equilibrium point with disease E_{eq}^* of system (2) is globally asymptotically stable if $R_0 > 1$.*

Demonstration: We consider the following Lyapunov function:

$$V : \Gamma \longrightarrow \mathbb{R}$$

$$V(S, E) = S - S^* \ln\left(\frac{S}{S^*}\right) + E - E^* \ln\left(\frac{E}{E^*}\right)$$

where $\Gamma = \{(S, E, I) \in \Gamma / S > 0, E > 0\}$ Then, the derivative of the Lyapunov function is given by:

$$\frac{dV(S, E)}{dt} = \left(-\frac{\Lambda(S - S^*)}{SS^*} - \frac{\beta}{N}(E - E^*) \right) (S - S^*) + \frac{\beta}{N}(S - S^*)(E - E^*)$$

Then,

$$\frac{dV(S, E)}{dt} = -\frac{\Lambda(S - S^*)^2}{SS^*} \leq 0$$

also,

$$\frac{dV(S, E)}{dt} = 0 \text{ if } S = S^*$$

4.3. Sensitivity analysis of R_0 . Sensitivity analysis is commonly used to help us know the parameters that have a high impact on the reproduction number R_0 (because there are usually errors in data collection and assumed parameter values). Using the approach of Chitnis and al [14], we calculate the normalized forward sensitivity indices of R_0 which is defined as

$$\gamma_n^{R_0} = \frac{\partial R_0}{\partial n} * \frac{n}{R_0}$$

let's Note the sensitivity index of R_0 with respect to the parameter n, we obtain:

$$R_0 = \frac{\beta \Lambda}{\mu N(\alpha + \mu)}$$

$$\gamma_\beta^{R_0} = 1$$

$$\gamma_\alpha^{R_0} = -\frac{\alpha}{\alpha + \mu}$$

$$\gamma_\mu^{R_0} = -\frac{\mu}{\alpha + \mu} - 1$$

With the sensitivity values are:

γ_β	1
γ_α	-0.2
γ_μ	-1.8

The sensitivity values

We observe from above that the basic reproduction number R_0 is the most sensitive to changes in β . Indeed, if β increases R_0 will also increase in the same proportion and if β decreases in the same proportion, μ and α will have an inversely proportional relationship with R_0 . Thus, an increase in one of them will lead to a decrease in R_0 .

5. NUMERICAL SIMULATIONS

In this section, we illustrate some numerical solutions of model (1) for different values of the parameters. We use the following different initial values such that $S + E + I + Q + R = 26000$.

5.1. Disease-free equilibrium: We use and present some numerical simulations of the system (1) to illustrate our results, estimating $\Lambda = 1500$, $\mu = 0.04$, $\beta = 0.05$, $\alpha = 0.09$, $\delta = 0.05$, $\lambda = 0.5$, $\theta = 0.05$, and different initial values for each state variable, we have the disease-free equilibrium (monkey pox) $R_0 = 0.1545 < 1$.

In this case, according to theorem (4), the monkeypox disease-free equilibrium E_0 of system (1) is globally asymptotically stable on Ω . (See figures (2))

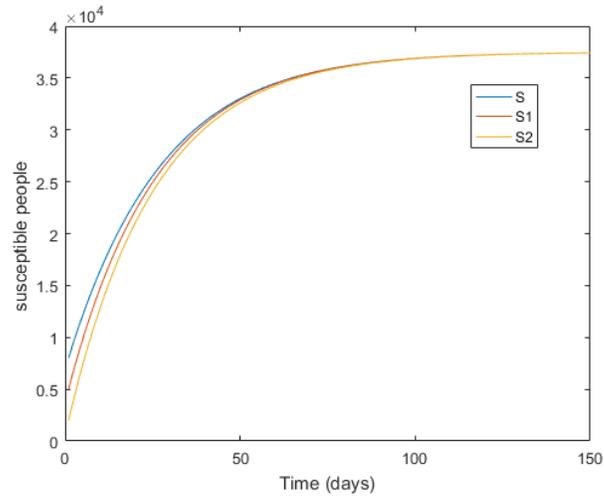


FIGURE 2a

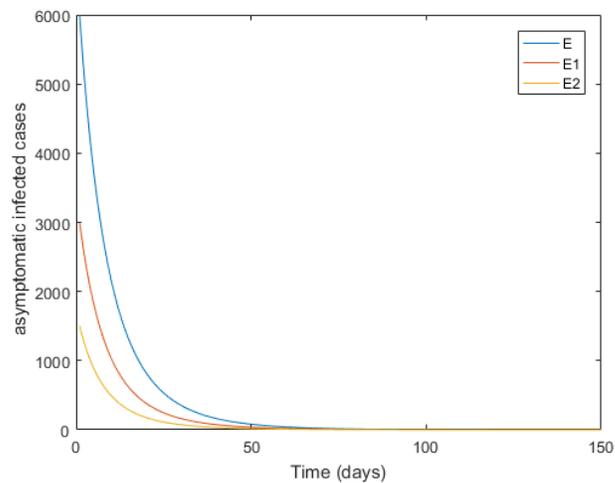


FIGURE 2b

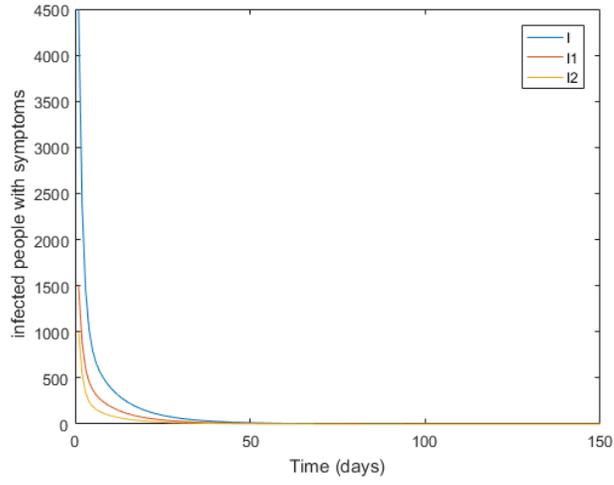


FIGURE 2c

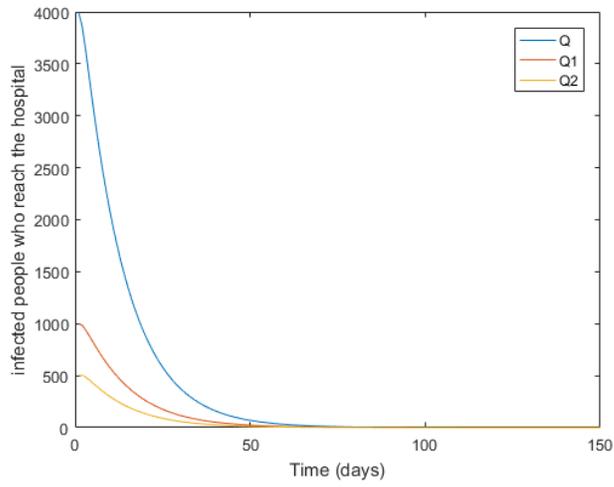


FIGURE 2d

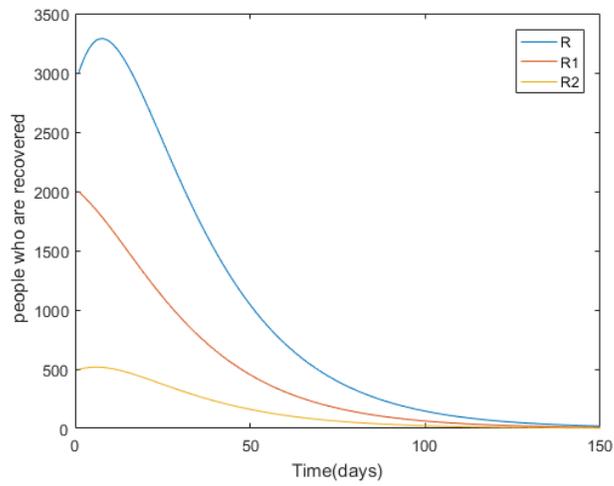


FIGURE 2e

From these Figures, using the different values of the initial variables S_0, E_0, I_0, Q_0 and R_0 , we obtained the following remarks:

Remarks:

- The number of potential individuals increases and approaches that of the population $S_0 = 1.3867e + 05$ (See figure 2a).
- the number of asymptomatic infected cases decreases and converges towards zero (See figure 2b).
- The number of infected people with symptoms and carriers of the virus first increases, then decreases and approaches zero (see Figure 2c).
- The number of hospitalized cases is decreasing and approaching zero (see Figure 2d).
- The number of recovered cases decreases and approaches zero (see Figure 2e).

Therefore, the solution curves towards the equilibrium $E_0^{eq}(S_0, 0, 0, 0, 0)$ when $R_0 < 1$. Therefore, the model (1) is globally asymptotically stable.

5.2. Point of equilibrium with disease: Also, we estimate for $\Lambda = 1500, \mu = 0.4, \beta = 0.09, \alpha = 0.01, \delta = 0.05, \lambda = 0.5, \theta = 0.05$, we have equilibrium point with monkeypox disease E_{eq}^* and $R_0 = 2.5755 > 1$, In this case, according to theorem (5), the equilibrium with monkeypox disease E^* of system (1) is globally asymptotically stable on Ω . (See Figures (3))

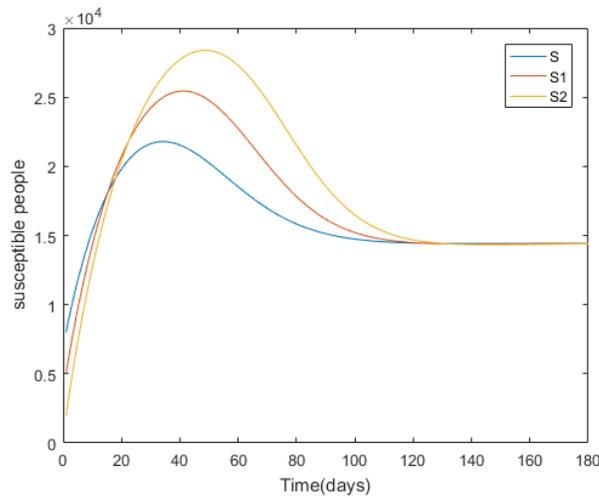


FIGURE 3a

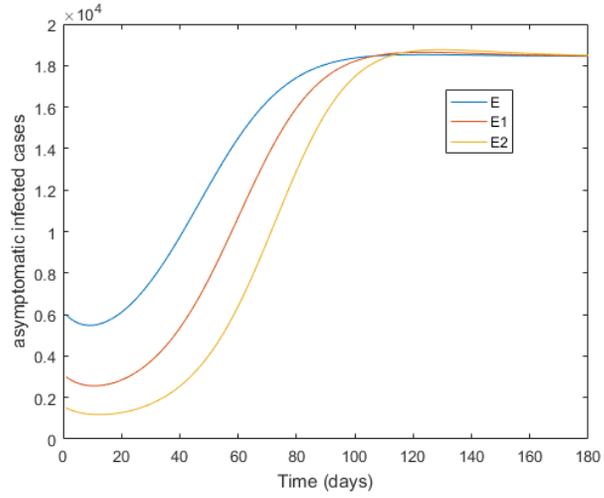


FIGURE 3b

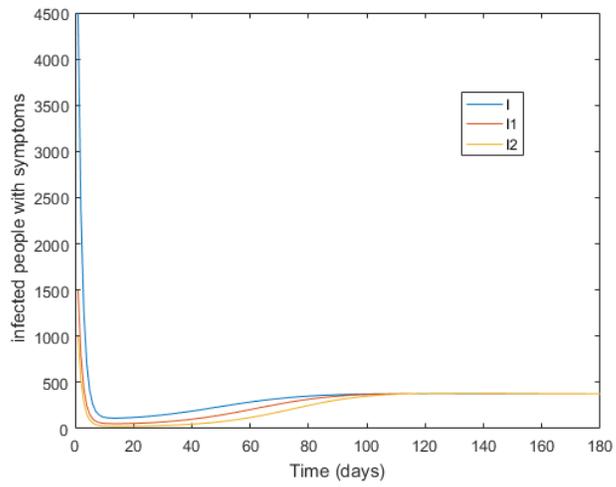


FIGURE 3c

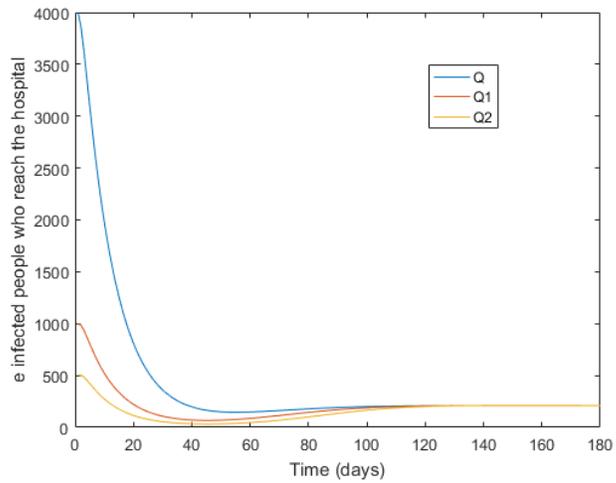


FIGURE 3d

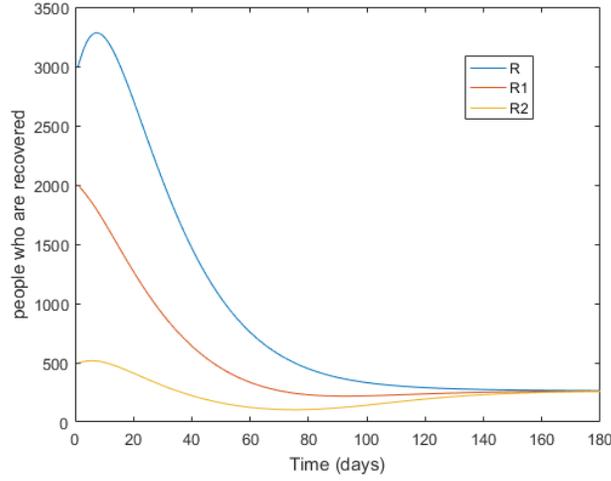


FIGURE 3e

Remarks:

- the number of potential individuals first increases, then it decreases slightly and approaches the $S^* = 1.4444e + 04$ value (see figure 3a)
- the number of asymptomatic infected cases increases and converges towards the value of $E^* = 1.8444e + 04$ (see figure 3b).
- The number of infected people with symptoms and carriers of the virus decreases, then increases slightly towards the value of $I^* = 376.4172$ (See figure 3c).
- The number of hospitalized cases decreases and approaches the value of $Q^* = 209.1207$ (see figure 3d).
- number of recovered cases decreases and converges towards the value of $R^* = 261.4009$ (see figure 3e).

Therefore, the solution curves towards the equilibrium $E_*^{eq}(S^*, E^*, I^*, Q^*, R^*)$ when $R_0 > 1$. Therefore, model (1) is globally asymptotically stable.

6. CONCLUSION

In this work, we have formulated and presented a continuous $SEIQR$ mathematical model of monkeypox that describes the dynamics of individuals infected with this disease. We have determined the base reproduction number of the system (1) as $R_0 = \frac{\beta\Lambda}{\mu N(\alpha + \mu)}$ which helps us

understand the dynamics of the system. Additionally, we have conducted a sensitivity analysis of the model parameters to identify the parameters that have a significant impact on the reproduction number R_0 . Using the theory of stability analysis for nonlinear systems, we have analyzed the mathematical model of monkeypox and examined its local and global stability. The disease-free equilibrium point E_{eq}^0 can exhibit local stability if the reproduction number $R_0 \leq 1$. Conversely, if $R_0 > 1$, the equilibrium points with disease E_{eq}^* are locally asymptotically stable. We have demonstrated global asymptotic stability of E_{eq}^0 using a Lyapunov function when $R_0 \leq 1$. Similarly, we have shown global asymptotic stability of E_{eq}^* using a Lyapunov function when $R_0 > 1$.

CONFLICT OF INTERESTS

The author(s) declare that there is no conflict of interests.

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