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MATHEMATICAL ANALYSIS OF A MONKEYPOX TRANSMISSION MODEL WITH CONTACT TRACING AND QUARANTINE AS NON-PHARMACEUTICAL INTERVENTIONS

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Abstract. This study develops and analyzes a deterministic compartmental model to describe the transmission dynamics of monkeypox with the inclusion of contact tracing and quarantine as non-pharmaceutical interventions. The human population is divided into seven mutually exclusive compartments: susceptible, exposed, traced exposed, untraced exposed, infectious, quarantined, and recovered. The model is shown to be mathematically well-posed, ensuring non-negativity and boundedness of solutions. Equilibrium points, such as the disease-free and endemic equilibria, are computed. Using the next-generation matrix method, the basic reproduction number R_0 is computed and identified as the threshold parameter that determines the persistence or eradication of the disease. The analysis establishes that the disease-free equilibrium is locally and globally asymptotically stable when $R_0 < 1$, while the endemic equilibrium becomes locally stable when $R_0 > 1$. Numerical simulations support the theoretical analysis and demonstrate the effectiveness of contact tracing and quarantine in reducing and ultimately eradicating monkeypox transmission.

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1. Introduction

Monkeypox (mpox) is an emerging zoonotic disease caused by the Monkeypox virus (MPXV), a double-stranded DNA virus belonging to the *Orthopoxvirus genus* of the Poxviridae family [1]. First detected in humans in 1970 in the Democratic Republic of Congo, mpox has been historically endemic in Central and West Africa, primarily resulting from zoonotic spillover through contact with infected animals such as rodents and primates [2]. Human-to-human transmission occurs mainly through

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direct contact with infectious lesions, body fluids, respiratory droplets, and contaminated materials, as well as close physical and sexual contact [3]. Since 2022, mpox has demonstrated sustained interhuman transmission across multiple continents, prompting the World Health Organization (WHO) to declare it a Public Health Emergency of International Concern (PHEIC) in July 2022 [4]. Clinically, the disease presents with fever, lymphadenopathy, and vesiculopustular rash and can cause severe complications among immunocompromised or vulnerable individuals [5,6].

In the absence of widespread vaccination coverage, non-pharmaceutical interventions (NPIs) such as contact tracing and quarantine remain critical tools in mitigating mpox transmission [7]. These interventions aim to break transmission chains by identifying infectious individuals early and preventing further exposure among close contacts. Contact tracing enables health authorities to detect potential secondary cases before symptom onset, reducing both the effective reproduction number and outbreak magnitude [8]. Empirical studies and modeling analyses have demonstrated that timely isolation and quarantine can substantially delay epidemic peaks and decrease cumulative incidence [9,10]. The effectiveness of these strategies, however, depends strongly on tracing efficiency, compliance rates, and delays between exposure and isolation [11,12]. Consequently, quantifying their epidemiological impact through mathematical modeling is essential for guiding evidence-based public health responses, especially in emerging infectious diseases such as mpox.

Several recent studies have explored different strategies in the mathematical modeling of monkeypox transmission dynamics. Khalid et al. [13] integrated quarantine and vaccination to evaluate the combined impact of pharmaceutical and non-pharmaceutical interventions, while Hassan et al. [16] emphasized indirect transmission through environmental contamination and analyzed the effectiveness of sanitation measures. Ameh et al. [14] investigated non-pharmaceutical interventions using real outbreak data but without explicit consideration of structured isolation or environmental reservoirs. Although these studies contributed valuable insights into mpox control, they addressed these mechanisms separately and under specific assumptions. To date, no existing model has simultaneously incorporated both contact tracing and quarantine within a unified analytical framework. This limitation underscores the importance of developing a comprehensive model that integrates these interventions, as their combined implementation has been shown to significantly reduce transmission chains and prevent outbreak persistence [15].

To address the identified gap, this study integrates contact tracing and quarantine into a deterministic compartmental model to capture the dynamics of mpox transmission. These non-pharmaceutical interventions are incorporated to reflect realistic control measures and to analyze their influence on disease behavior. The relevance of this study lies in providing a rigorous theoretical framework that enhances understanding of mpox mitigation through effective tracing and quarantine strategies.

2. Model Formulation

To capture the transmission dynamics of monkeypox in the presence of contact tracing, quarantine, and recovery heterogeneity, we propose a deterministic compartmental model structured around ordinary differential equations. The total human population at time t, denoted by $N(t)$, is stratified into seven mutually exclusive compartments: susceptible $S(t)$, exposed $E(t)$, traced exposed $E_t(t)$, untraced exposed $E_u(t)$, infectious $I(t)$, quarantined $Q(t)$, and recovered $R(t)$. Thus, the total population is given by:

$$N(t) = S(t) + E(t) + E_t(t) + E_u(t) + I(t) + Q(t) + R(t).$$

Susceptible individuals become infected at a rate proportional to their contact with infectious individuals, governed by a bilinear incidence term βSI, where $\beta > 0$ denotes the transmission coefficient. Upon infection, individuals enter the latent class $E(t)$, where they remain non-infectious during the incubation period.

A core feature of this model is the integration of contact tracing. A proportion $p \in [0, 1]$ of exposed individuals are successfully traced before becoming infectious and are routed to the traced exposed class $E_t(t)$ at a rate $p\alpha$, where α is the incubation rate. The remaining fraction $1-p$ of exposed individuals are not traced and are transferred to the untraced exposed class $E_u(t)$ at rate $(1-p)\alpha$. Individuals in $E_t(t)$ are moved to quarantine $Q(t)$ at a rate θ_1, while those in $E_u(t)$ progress to the infectious compartment $I(t)$ at rate δ.

Infectious individuals $I(t)$ can either be isolated via symptom-based detection and moved to quarantine at rate θ_2, or recover naturally without quarantine at rate γ_u. Quarantined individuals $Q(t)$ eventually recover at rate γ_q. All classes are subject to natural mortality at rate μ_h, and the susceptible population is replenished by a constant recruitment rate Λ.

The formulation of the model considers the following assumptions:

(1) The population is homogeneously mixed and closed to migration, except for recruitment and natural death.
(2) All individuals progress through a latent (exposed) period after infection, governed by a constant incubation rate α.
(3) A proportion p of exposed individuals are successfully traced and quarantined before symptom onset.
(4) Untraced individuals progress to become infectious and may be quarantined later upon detection.
(5) Recovery from infection or quarantine leads to permanent immunity.
(6) All transitions and processes are governed by constant, time-invariant rates.
(7) Natural death occurs uniformly across all compartments at rate μ_h.

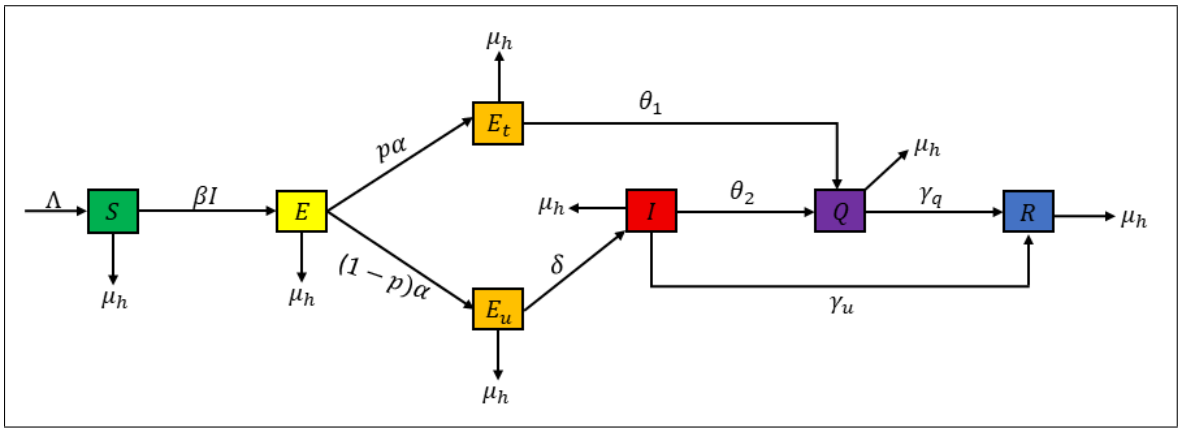

FIGURE 1. Transmission Diagram of Monkeypox

Parameter	Description
Λ	Recruitment rate into the susceptible population
μ_h	Natural death rate
β	Effective contact transmission rate
α	Incubation rate (rate of leaving exposed class)
p	Proportion of exposed individuals who are traced
θ_1	Quarantine rate of traced exposed individuals
δ	Rate at which untraced exposed individuals become infectious
θ_2	Quarantine rate of infectious individuals
γ_u	Recovery rate of unquarantined infectious individuals
γ_q	Recovery rate of quarantined individuals

TABLE 1. Description of the Model Parameters

Based on these assumptions, the system of differential equations governing the model is

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \beta SI - \mu_h S \\
\frac{dE}{dt} &= \beta SI - (\alpha + \mu_h)E \\
\frac{dE_t}{dt} &= p\alpha E - (\theta_1 + \mu_h)E_t \\
\frac{dE_u}{dt} &= (1-p)\alpha E - (\delta + \mu_h)E_u \\
\frac{dI}{dt} &= \delta E_u - (\theta_2 + \gamma_u + \mu_h)I \\
\frac{dQ}{dt} &= \theta_1 E_t + \theta_2 I - (\gamma_q + \mu_h)Q \\
\frac{dR}{dt} &= \gamma_u I + \gamma_q Q - \mu_h R
\end{aligned} \tag{1}$$

with the initial conditions $S(0) \geq 0$, $E(0) \geq 0$, $E_t(0) \geq 0$, $E_u(0) \geq 0$, $I(0) \geq 0$, $Q(0) \geq 0$, and $R(0) \geq 0$.

Theorem 1. *Given non-negative initial conditions, the solutions* $S(t), E(t), E_t(t), E_u(t), I(t), Q(t), R(t)$ *of system* (1) *remain non-negative for all* $t \geq 0$.

Proof. Let $S(0) \geq 0$, $E(0) \geq 0$, $E_t(0) \geq 0$, $E_u(0) \geq 0$, $I(0) \geq 0$, $Q(0) \geq 0$, and $R(0) \geq 0$. Consider the first differential equation of system (1)

$$\frac{dS}{dt} = \Lambda - \beta SI - \mu_h S \quad \Rightarrow \quad \frac{dS}{dt} + (\beta I + \mu_h)S = \Lambda$$

Define the integrating factor

$$\phi_S(t) = e^{\int_0^t (\beta I(s) + \mu_h)\, ds}.$$

Multiplying the differential equation by $\phi_S(t)$, we get

$$\phi_S(t)\frac{dS}{dt} + \phi_S(t)(\beta I + \mu_h)S = \Lambda \phi_S(t) \quad \Rightarrow \quad \frac{d}{dt}\left[\phi_S(t)S(t)\right] = \Lambda \phi_S(t).$$

Integrating both sides from 0 to t, we have

$$\phi_S(t)S(t) - \phi_S(0)S(0) = \int_0^t \Lambda \phi_S(s)\, ds$$

Since $\phi_S(0) = 1$, we obtain

$$S(t) = S(0)e^{-\int_0^t (\beta I(s) + \mu_h)\, ds} + \int_0^t \Lambda\, e^{-\int_s^t (\beta I(r) + \mu_h)\, dr}\, ds$$

Since all terms are non-negative, $S(t) \geq 0, \forall t \geq 0$.

Next, consider the second differential equation

$$\frac{dE}{dt} = \beta SI - (\alpha + \mu_h)E \quad \Rightarrow \quad \frac{dE}{dt} + (\alpha + \mu_h)E = \beta SI$$

Define the integrating factor

$$\phi_E(t) = e^{\int_0^t (\alpha + \mu_h)\, ds} = e^{(\alpha + \mu_h)t}$$

Multiply both sides by $\phi_E(t)$:

$$\phi_E(t)\frac{dE}{dt} + \phi_E(t)(\alpha + \mu_h)E = \phi_E(t)\beta SI \quad \Rightarrow \quad \frac{d}{dt}\left[\phi_E(t)E(t)\right] = \phi_E(t)\beta S(t)I(t)$$

Integrate both sides

$$\phi_E(t)E(t) = E(0) + \int_0^t \beta S(s)I(s)\, \phi_E(s)\, ds$$

Then,

$$E(t) = E(0)e^{-(\alpha + \mu_h)t} + \int_0^t \beta S(s)I(s)\, e^{-(\alpha + \mu_h)(t-s)}\, ds$$

Hence, $E(t) \geq 0\ \forall t \geq 0$. By applying the same approach, the following results were obtained for the remaining compartmental variables.

$$E_t(t) = E_t(0)e^{-(\theta_1 + \mu_h)t} + \int_0^t p\alpha E(s)e^{-(\theta_1 + \mu_h)(t-s)}\, ds \geq 0$$

$$E_u(t) = E_u(0)e^{-(\delta + \mu_h)t} + \int_0^t (1-p)\alpha E(s)e^{-(\delta + \mu_h)(t-s)}\, ds \geq 0$$

$$I(t) = I(0)e^{-(\theta_2+\gamma_u+\mu_h)t} + \int_0^t \delta E_u(s)e^{-(\theta_2+\gamma_u+\mu_h)(t-s)}\,ds \geq 0$$

$$Q(t) = Q(0)e^{-(\gamma_q+\mu_h)t} + \int_0^t \left[\theta_1 E_t(s) + \theta_2 I(s)\right] e^{-(\gamma_q+\mu_h)(t-s)}\,ds \geq 0$$

$$R(t) = R(0)e^{-\mu_h t} + \int_0^t \left[\gamma_u I(s) + \gamma_q Q(s)\right] e^{-\mu_h(t-s)}\,ds \geq 0$$

Thus, the solutions of system (1) remain non-negative $\forall t \geq 0$. □

Theorem 2. *The solutions of the model system are bounded in the region*

$$\Omega = \left\{(S, E, E_t, E_u, I, Q, R) \in \mathbb{R}_+^7 :\ S + E + E_t + E_u + I + Q + R \leq \frac{\Lambda}{\mu_h}\right\}.$$

Proof. We define the total population as

$$N(t) = S(t) + E(t) + E_t(t) + E_u(t) + I(t) + Q(t) + R(t).$$

Differentiating with respect to time and summing the system equations gives

$$\begin{aligned}\frac{dN}{dt} &= \Lambda - \mu_h E - \mu_h E_t - \mu_h E_u - \mu_h I - \mu_h Q - \mu_h R - \mu_h S \\ &= \Lambda - \mu_h(S + E + E_t + E_u + I + Q + R) \\ &= \Lambda - \mu_h N(t)\end{aligned}$$

Using the integrating factor $e^{\mu_h t}$, we get

$$\frac{d}{dt}\left(N(t)e^{\mu_h t}\right) = \Lambda e^{\mu_h t}.$$

Integrating both sides

$$N(t)e^{\mu_h t} = N(0) + \int_0^t \Lambda e^{\mu_h s} ds = N(0) + \frac{\Lambda}{\mu_h}\left(e^{\mu_h t} - 1\right).$$

Solving for $N(t)$

$$N(t) = \left(N(0) - \frac{\Lambda}{\mu_h}\right) e^{-\mu_h t} + \frac{\Lambda}{\mu_h}.$$

Hence, as $t \to \infty$, $N(t) \to \frac{\Lambda}{\mu_h}$, and for all $t \geq 0$,

$$N(t) \leq \max\left\{N(0),\ \frac{\Lambda}{\mu_h}\right\}, \quad \text{for all } t \geq 0.$$

This proves that all solutions of the system (1) are bounded within the region. □

3. Model Analysis

3.1. **Equilibrium Points and Basic Reproduction Number.** This section presents the computation of the disease-free and endemic equilibrium points of the monkeypox model (1), along with the basic reproduction number.

Theorem 3. *The system (1) admits two equilibrium points: the disease-free equilibrium (DFE), given by*

$$\mathcal{E}_0 = \left(\frac{\Lambda}{\mu_h}, 0, 0, 0, 0, 0, 0\right),$$

and the endemic equilibrium (EE), represented by

$$\mathcal{E}_1 = (S^*, E^*, E_t^*, E_u^*, I^*, Q^*, R^*),$$

where

$$\begin{aligned}
S^* &= \frac{(\alpha+\mu_h)(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h)}{\beta(1-p)\alpha\delta},\\
E^* &= \frac{\Lambda}{\alpha+\mu_h} - \frac{\mu_h(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h)}{\beta(1-p)\alpha\delta},\\
E_t^* &= \frac{p\left(\Lambda\beta(1-p)\alpha\delta - \mu_h(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h)(\alpha+\mu_h)\right)}{(\theta_1+\mu_h)\beta(1-p)\delta(\alpha+\mu_h)},\\
E_u^* &= \frac{\Lambda\beta(1-p)\alpha\delta - \mu_h(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h)(\alpha+\mu_h)}{\beta\delta(\delta+\mu_h)(\alpha+\mu_h)},\\
I^* &= \frac{\Lambda\beta(1-p)\alpha\delta - \mu_h(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h)(\alpha+\mu_h)}{\beta(\delta+\mu_h)(\alpha+\mu_h)(\theta_2+\gamma_u+\mu_h)},\\
Q^* &= \frac{1}{\gamma_q+\mu_h}\left[\frac{\theta_1 p\left(\Lambda\beta(1-p)\alpha\delta - \mu_h(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h)(\alpha+\mu_h)\right)}{(\theta_1+\mu_h)\beta(1-p)\delta(\alpha+\mu_h)}\right.\\
&\quad \left.+\frac{\theta_2\left(\Lambda\beta(1-p)\alpha\delta - \mu_h(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h)(\alpha+\mu_h)\right)}{\beta(\delta+\mu_h)(\alpha+\mu_h)(\theta_2+\gamma_u+\mu_h)}\right],\\
R^* &= \frac{1}{\mu_h}\left[\frac{\gamma_u\left(\Lambda\beta(1-p)\alpha\delta - \mu_h(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h)(\alpha+\mu_h)\right)}{\beta(\delta+\mu_h)(\alpha+\mu_h)(\theta_2+\gamma_u+\mu_h)}\right.\\
&\quad + \frac{\gamma_q}{\gamma_q+\mu_h}\left(\frac{\theta_1 p\left(\Lambda\beta(1-p)\alpha\delta - \mu_h(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h)(\alpha+\mu_h)\right)}{(\theta_1+\mu_h)\beta(1-p)\delta(\alpha+\mu_h)}\right.\\
&\quad \left.\left.+\frac{\theta_2\left(\Lambda\beta(1-p)\alpha\delta - \mu_h(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h)(\alpha+\mu_h)\right)}{\beta(\delta+\mu_h)(\alpha+\mu_h)(\theta_2+\gamma_u+\mu_h)}\right)\right].
\end{aligned}$$

Proof. To solve for the equilibrium points, we set the derivatives in system (1) to zero. That is,

$$\frac{dS}{dt} = \Lambda - \beta SI - \mu_h S = 0 \tag{2}$$

$$\frac{dE}{dt} = \beta SI - (\alpha+\mu_h)E = 0 \tag{3}$$

$$\frac{dE_t}{dt} = p\alpha E - (\theta_1+\mu_h)E_t = 0 \tag{4}$$

$$\frac{dE_u}{dt} = (1-p)\alpha E - (\delta + \mu_h)E_u = 0 \tag{5}$$

$$\frac{dI}{dt} = \delta E_u - (\theta_2 + \gamma_u + \mu_h)I = 0 \tag{6}$$

$$\frac{dQ}{dt} = \theta_1 E_t + \theta_2 I - (\gamma_q + \mu_h)Q = 0 \tag{7}$$

$$\frac{dR}{dt} = \gamma_u I + \gamma_q Q - \mu_h R = 0 \tag{8}$$

Case 1: Assume that there is no infection in the population, i.e., $E = E_t = E_u = I = Q = R = 0$. Then from equation (2),

$$\Lambda - \mu_h S = 0$$

$$S = \frac{\Lambda}{\mu_h}.$$

Thus, the disease-free equilibrium point is

$$\mathcal{E}_0 = \left(\frac{\Lambda}{\mu_h}, 0, 0, 0, 0, 0, 0\right).$$

Case 2: If $I \neq 0$, then from equation (6),

$$I = \frac{\delta E_u}{\theta_2 + \gamma_u + \mu_h}.$$

From equation (5), we get

$$E_u = \frac{(1-p)\alpha E}{\delta + \mu_h}. \tag{9}$$

So,

$$I = \frac{\delta}{\theta_2 + \gamma_u + \mu_h} \cdot \frac{(1-p)\alpha E}{\delta + \mu_h} \tag{10}$$

$$= \frac{(1-p)\alpha\delta E}{(\delta + \mu_h)(\theta_2 + \gamma_u + \mu_h)}. \tag{11}$$

From equation (3), we have

$$S = \frac{(\alpha + \mu_h)E}{\beta I}. \tag{12}$$

Substituting equation (11) into (12), we get

$$S = \frac{(\alpha + \mu_h)E}{\beta \cdot \frac{(1-p)\alpha\delta E}{(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h)}} \tag{13}$$

$$= \frac{(\alpha + \mu_h)(\delta + \mu_h)(\theta_2 + \gamma_u + \mu_h)}{\beta(1-p)\alpha\delta}. \tag{14}$$

Substitute equations (11) and (14) into equation (2):

$$\Lambda - \beta SI - \mu_h S = 0.$$

Now,

$$\beta SI = (\alpha + \mu_h)E, \tag{15}$$

so equation (2) becomes

$$\Lambda - (\alpha + \mu_h)E - \mu_h S = 0.$$

Substitute equation (14) for S:

$$E = \frac{\Lambda - \mu_h \cdot \frac{(\alpha+\mu_h)(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h)}{\beta(1-p)\alpha\delta}}{\alpha+\mu_h} \tag{16}$$

$$= \frac{\Lambda}{\alpha+\mu_h} - \frac{\mu_h(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h)}{\beta(1-p)\alpha\delta}. \tag{17}$$

By equations (9) and (17), we get

$$E_u = \frac{(1-p)\alpha}{\delta+\mu_h} \cdot \left(\frac{\Lambda}{\alpha+\mu_h} - \frac{\mu_h(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h)}{\beta(1-p)\alpha\delta} \right)$$
$$= \frac{\Lambda\beta(1-p)\alpha\delta - \mu_h(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h)(\alpha+\mu_h)}{\beta\delta(\delta+\mu_h)(\alpha+\mu_h)}.$$

By equation (6), we have

$$I = \frac{\delta E_u}{\theta_2+\gamma_u+\mu_h}$$
$$= \frac{\Lambda\beta(1-p)\alpha\delta - \mu_h(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h)(\alpha+\mu_h)}{\beta(\delta+\mu_h)(\alpha+\mu_h)(\theta_2+\gamma_u+\mu_h)}.$$

From equation (4), we get

$$E_t = \frac{p\alpha E}{\theta_1+\mu_h}. \tag{18}$$

Substituting equation (17) into (18), we obtain

$$E_t = \frac{p\left(\Lambda\beta(1-p)\alpha\delta - \mu_h(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h)(\alpha+\mu_h)\right)}{(\theta_1+\mu_h)\beta(1-p)\delta(\alpha+\mu_h)}.$$

From equation (7),

$$Q = \frac{\theta_1 E_t + \theta_2 I}{\gamma_q+\mu_h}$$
$$= \frac{1}{\gamma_q+\mu_h}\left[\frac{\theta_1 p\left(\Lambda\beta(1-p)\alpha\delta - \mu_h(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h)(\alpha+\mu_h)\right)}{(\theta_1+\mu_h)\beta(1-p)\delta(\alpha+\mu_h)} \right.$$
$$\left. + \frac{\theta_2\left(\Lambda\beta(1-p)\alpha\delta - \mu_h(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h)(\alpha+\mu_h)\right)}{\beta(\delta+\mu_h)(\alpha+\mu_h)(\theta_2+\gamma_u+\mu_h)} \right].$$

Finally, by equation (8),

$$R = \frac{\gamma_u I + \gamma_q Q}{\mu_h}$$

$$R = \frac{1}{\mu_h}\left[\frac{\gamma_u\left(\Lambda\beta(1-p)\alpha\delta - \mu_h(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h)(\alpha+\mu_h)\right)}{\beta(\delta+\mu_h)(\alpha+\mu_h)(\theta_2+\gamma_u+\mu_h)} \right.$$
$$+ \frac{\gamma_q}{\gamma_q+\mu_h}\left(\frac{\theta_1 p\left(\Lambda\beta(1-p)\alpha\delta - \mu_h(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h)(\alpha+\mu_h)\right)}{(\theta_1+\mu_h)\beta(1-p)\delta(\alpha+\mu_h)} \right.$$
$$\left.\left. + \frac{\theta_2\left(\Lambda\beta(1-p)\alpha\delta - \mu_h(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h)(\alpha+\mu_h)\right)}{\beta(\delta+\mu_h)(\alpha+\mu_h)(\theta_2+\gamma_u+\mu_h)} \right) \right]$$

Thus, the endemic equilibrium point of system (1) is

$$\mathcal{E}_1 = (S, E, E_t, E_u, I, Q, R),$$

where

$$S = \frac{(\alpha+\mu_h)(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h)}{\beta(1-p)\alpha\delta},$$

the number of individuals still susceptible to infection, determined by recruitment, transmission, isolation, and natural death rates.

$$E = \frac{\Lambda}{\alpha+\mu_h} - \frac{\mu_h(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h)}{\beta(1-p)\alpha\delta},$$

the number of exposed individuals in the population, determined by transmission dynamics and compartment transitions.

$$E_t = \frac{p\left(\Lambda\beta(1-p)\alpha\delta - \mu_h(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h)(\alpha+\mu_h)\right)}{(\theta_1+\mu_h)\beta(1-p)\delta(\alpha+\mu_h)}, \quad \text{and}$$
$$E_u = \frac{\Lambda\beta(1-p)\alpha\delta - \mu_h(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h)(\alpha+\mu_h)}{\beta\delta(\delta+\mu_h)(\alpha+\mu_h)}.$$

the number of traced and untraced exposed individuals, respectively, based on proportions traced and isolation progression.

$$I = \frac{\Lambda\beta(1-p)\alpha\delta - \mu_h(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h)(\alpha+\mu_h)}{\beta(\delta+\mu_h)(\alpha+\mu_h)(\theta_2+\gamma_u+\mu_h)}, \quad \text{and}$$
$$Q = \frac{1}{\gamma_q+\mu_h}\left[\frac{\theta_1 p\left(\Lambda\beta(1-p)\alpha\delta - \mu_h(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h)(\alpha+\mu_h)\right)}{(\theta_1+\mu_h)\beta(1-p)\delta(\alpha+\mu_h)}\right.$$
$$\left.+\frac{\theta_2\left(\Lambda\beta(1-p)\alpha\delta - \mu_h(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h)(\alpha+\mu_h)\right)}{\beta(\delta+\mu_h)(\alpha+\mu_h)(\theta_2+\gamma_u+\mu_h)}\right],$$

the number of actively infected and quarantined individuals, depending on tracing, testing outcome, and recovery or removal.

$$R = \frac{1}{\mu_h}\left[\frac{\gamma_u\left(\Lambda\beta(1-p)\alpha\delta - \mu_h(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h)(\alpha+\mu_h)\right)}{\beta(\delta+\mu_h)(\alpha+\mu_h)(\theta_2+\gamma_u+\mu_h)}\right.$$
$$+\frac{\gamma_q}{\gamma_q+\mu_h}\left(\frac{\theta_1 p\left(\Lambda\beta(1-p)\alpha\delta - \mu_h(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h)(\alpha+\mu_h)\right)}{(\theta_1+\mu_h)\beta(1-p)\delta(\alpha+\mu_h)}\right.$$
$$\left.\left.+\frac{\theta_2\left(\Lambda\beta(1-p)\alpha\delta - \mu_h(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h)(\alpha+\mu_h)\right)}{\beta(\delta+\mu_h)(\alpha+\mu_h)(\theta_2+\gamma_u+\mu_h)}\right)\right],$$

the number of recovered individuals, influenced by the successful recovery rates of isolated and non-isolated infected individuals.

This completes the proof for the existence of the endemic equilibrium point for the model (1). □

The basic reproduction number, denoted by R_0, is defined as the expected number of secondary infections generated by a single infectious individual introduced into a wholly susceptible population [23,31]. To compute R_0, we apply the *Next-Generation Matrix* (*NGM*) method developed by van den Driessche and Watmough (2002). First, we identify the infected compartments involved in disease transmission, which are:

$$x = (E, E_t, E_u, I, Q)^T.$$

The new infection terms and transition dynamics are defined as:

$$\mathcal{F}(x) = \begin{pmatrix} \beta\dfrac{\Lambda}{\mu_h} I \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}, \qquad \mathcal{V}(x) = \begin{pmatrix} (\alpha+\mu_h)E \\ -p\alpha E + (\theta_1+\mu_h)E_t \\ -(1-p)\alpha E + (\delta+\mu_h)E_u \\ -\delta E_u + (\theta_2+\gamma_u+\mu_h)I \\ -\theta_1 E_t - \theta_2 I + (\gamma_q+\mu_h)Q \end{pmatrix}.$$

The Jacobian matrices of $\mathcal{F}$ and $\mathcal{V}$, evaluated at the disease-free equilibrium (DFE), where $S = \dfrac{\Lambda}{\mu_h}$ and all infected compartments are 0, are:

$$F = \begin{pmatrix} 0 & 0 & 0 & \beta\dfrac{\Lambda}{\mu_h} & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}, \quad V = \begin{pmatrix} \alpha+\mu_h & 0 & 0 & 0 & 0 \\ -p\alpha & \theta_1+\mu_h & 0 & 0 & 0 \\ -(1-p)\alpha & 0 & \delta+\mu_h & 0 & 0 \\ 0 & 0 & -\delta & \theta_2+\gamma_u+\mu_h & 0 \\ 0 & -\theta_1 & 0 & -\theta_2 & \gamma_q+\mu_h \end{pmatrix}.$$

The next-generation matrix is given by:

$$K = FV^{-1}.$$

Since only the first row of F is non-zero, only the first row of K is relevant. Computing this row, we obtain:

$$FV^{-1} = \begin{pmatrix} \frac{\beta\Lambda\alpha p}{\mu_h(\alpha+\mu_h)(\theta_1+\mu_h)} + \frac{\beta\Lambda\alpha(1-p)\delta}{\mu_h(\alpha+\mu_h)(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h)} & \frac{\beta\Lambda}{\mu_h(\theta_1+\mu_h)} & \frac{\beta\Lambda}{\mu_h(\theta_2+\gamma_u+\mu_h)} & \frac{\beta\Lambda}{\mu_h(\theta_2+\gamma_u+\mu_h)} & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

Accordingly, the basic reproduction number is defined as the spectral radius of the matrix FV^{-1}. Hence, the basic reproduction number is:

$$R_0 = \frac{\beta\Lambda\left[p(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h) + (1-p)\delta(\theta_1+\mu_h)\right]}{\mu_h(\alpha+\mu_h)(\theta_1+\mu_h)(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h)}.$$

Theorem 4. *The system* (1) *has a unique endemic equilibrium point if and only if* $R_0 > 1$, *where* R_0 *is the basic reproduction number defined by*

$$R_0 = \frac{\beta\Lambda\left[p(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h)+(1-p)\delta(\theta_1+\mu_h)\right]}{\mu_h(\alpha+\mu_h)(\theta_1+\mu_h)(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h)}.$$

Proof. Suppose that the point $\varepsilon^* = (S^*, E^*, E_t^*, E_u^*, I^*, Q^*, R^*)$ is the endemic equilibrium of the system. Then, from the model equations, we have the steady-state conditions:

$$\Lambda - \beta S^* I^* - \mu_h S^* = 0 \tag{19}$$

$$\beta S^* I^* - (\alpha+\mu_h)E^* = 0 \tag{20}$$

$$p\alpha E^* - (\theta_1+\mu_h){E_t}^* = 0 \tag{21}$$

$$(1-p)\alpha E^* - (\delta+\mu_h){E_u}^* = 0 \tag{22}$$

$$\delta {E_u}^* - (\theta_2+\gamma_u+\mu_h)I^* = 0 \tag{23}$$

$$\theta_1 {E_t}^* + \theta_2 I^* - (\gamma_q+\mu_h)Q^* = 0 \tag{24}$$

$$\gamma_u I^* + \gamma_q Q^* - \mu_h R^* = 0 \tag{25}$$

From (19), solve for S^*:

$$S^* = \frac{\Lambda}{\beta I^* + \mu_h} \tag{26}$$

From (20), solve for E^*:

$$E^* = \frac{\beta S^* I^*}{\alpha+\mu_h} \tag{27}$$

From equation (21), we solve for E_t^*:

$$E_t^* = \frac{p\alpha E^*}{\theta_1+\mu_h} \tag{28}$$

From equation (22), we solve for E_u^*:

$$E_u^* = \frac{(1-p)\alpha E^*}{\delta+\mu_h} \tag{29}$$

From equation (23), we solve for I^*:

$$I^* = \frac{(1-p)\alpha\delta E^*}{(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h)} \tag{30}$$

Substituting (27) into (30):

$$I^* = \frac{(1-p)\alpha\delta}{(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h)} \cdot \frac{\beta S^* I^*}{\alpha+\mu_h}$$

Assuming $I^* \neq 0$, divide both sides by I^*:

$$1 = \frac{(1-p)\alpha\delta\beta S^*}{(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h)(\alpha+\mu_h)} \tag{31}$$

Solving for S^*:

$$S^* = \frac{(\delta + \mu_h)(\theta_2 + \gamma_u + \mu_h)(\alpha + \mu_h)}{(1-p)\alpha\delta\beta} \tag{32}$$

Now substitute (32) into (26):

$$\frac{\Lambda}{\beta I^* + \mu_h} = \frac{(\delta + \mu_h)(\theta_2 + \gamma_u + \mu_h)(\alpha + \mu_h)}{(1-p)\alpha\delta\beta}$$

Solving for I^*:

$$I^* = \frac{\Lambda(1-p)\alpha\delta\beta - \mu_h(\delta + \mu_h)(\theta_2 + \gamma_u + \mu_h)(\alpha + \mu_h)}{\beta(\delta + \mu_h)(\theta_2 + \gamma_u + \mu_h)(\alpha + \mu_h)} \tag{33}$$

To ensure $I^* > 0$, the numerator in (33) must be positive. This leads to the inequality:

$$\Lambda(1-p)\alpha\delta\beta > \mu_h(\delta + \mu_h)(\theta_2 + \gamma_u + \mu_h)(\alpha + \mu_h)$$

Now incorporate the contribution of the traced infections (from E_t through Q) to form the complete reproduction number. Using the next-generation matrix method, the basic reproduction number is given by:

$$R_0 = \frac{\beta\Lambda\left[p(\delta + \mu_h)(\theta_2 + \gamma_u + \mu_h) + (1-p)\delta(\theta_1 + \mu_h)\right]}{\mu_h(\alpha + \mu_h)(\theta_1 + \mu_h)(\delta + \mu_h)(\theta_2 + \gamma_u + \mu_h)}$$

Clearly, all parameters are positive. Hence, the condition $I^* > 0$ holds if and only if $R_0 > 1$. Therefore, the system admits a unique endemic equilibrium if and only if $R_0 > 1$. □

Theorem 5. *If $R_0 < 1$, then the system (1) is locally asymptotically stable at the disease-free equilibrium $\mathcal{E}_0$ and unstable otherwise.*

Proof. First, we compute the Jacobian matrix J of the system (1), given by:

$$J = \begin{pmatrix}
\frac{\partial f_1}{\partial S} & \frac{\partial f_1}{\partial E} & \frac{\partial f_1}{\partial E_t} & \frac{\partial f_1}{\partial E_u} & \frac{\partial f_1}{\partial I} & \frac{\partial f_1}{\partial Q} & \frac{\partial f_1}{\partial R} \\
\frac{\partial f_2}{\partial S} & \frac{\partial f_2}{\partial E} & \frac{\partial f_2}{\partial E_t} & \frac{\partial f_2}{\partial E_u} & \frac{\partial f_2}{\partial I} & \frac{\partial f_2}{\partial Q} & \frac{\partial f_2}{\partial R} \\
\frac{\partial f_3}{\partial S} & \frac{\partial f_3}{\partial E} & \frac{\partial f_3}{\partial E_t} & \frac{\partial f_3}{\partial E_u} & \frac{\partial f_3}{\partial I} & \frac{\partial f_3}{\partial Q} & \frac{\partial f_3}{\partial R} \\
\frac{\partial f_4}{\partial S} & \frac{\partial f_4}{\partial E} & \frac{\partial f_4}{\partial E_t} & \frac{\partial f_4}{\partial E_u} & \frac{\partial f_4}{\partial I} & \frac{\partial f_4}{\partial Q} & \frac{\partial f_4}{\partial R} \\
\frac{\partial f_5}{\partial S} & \frac{\partial f_5}{\partial E} & \frac{\partial f_5}{\partial E_t} & \frac{\partial f_5}{\partial E_u} & \frac{\partial f_5}{\partial I} & \frac{\partial f_5}{\partial Q} & \frac{\partial f_5}{\partial R} \\
\frac{\partial f_6}{\partial S} & \frac{\partial f_6}{\partial E} & \frac{\partial f_6}{\partial E_t} & \frac{\partial f_6}{\partial E_u} & \frac{\partial f_6}{\partial I} & \frac{\partial f_6}{\partial Q} & \frac{\partial f_6}{\partial R} \\
\frac{\partial f_7}{\partial S} & \frac{\partial f_7}{\partial E} & \frac{\partial f_7}{\partial E_t} & \frac{\partial f_7}{\partial E_u} & \frac{\partial f_7}{\partial I} & \frac{\partial f_7}{\partial Q} & \frac{\partial f_7}{\partial R}
\end{pmatrix}$$

which simplifies at the disease-free equilibrium $\mathcal{E}_0 = (\frac{\Lambda}{\mu_h}, 0, 0, 0, 0, 0, 0)$ to:

$$J_{\mathcal{E}_0} = \begin{pmatrix}
-\mu_h & 0 & 0 & 0 & -\beta\frac{\Lambda}{\mu_h} & 0 & 0 \\
0 & -(\alpha + \mu_h) & 0 & 0 & \beta\frac{\Lambda}{\mu_h} & 0 & 0 \\
0 & p\alpha & -(\theta_1 + \mu_h) & 0 & 0 & 0 & 0 \\
0 & (1-p)\alpha & 0 & -(\delta + \mu_h) & 0 & 0 & 0 \\
0 & 0 & 0 & \delta & -(\theta_2 + \gamma_u + \mu_h) & 0 & 0 \\
0 & 0 & \theta_1 & 0 & \theta_2 & -(\gamma_q + \mu_h) & 0 \\
0 & 0 & 0 & 0 & \gamma_u & \gamma_q & -\mu_h
\end{pmatrix}$$

Now, evaluating at the disease-free equilibrium point $\mathcal{E}_0 = \left(\frac{\Lambda}{\mu_h}, 0, 0, 0, 0, 0, 0\right)$, we obtain the Jacobian matrix:

$$J_{\mathcal{E}_0} = \begin{pmatrix} -\mu_h & 0 & 0 & 0 & -\beta\frac{\Lambda}{\mu_h} & 0 & 0 \\ 0 & -(\alpha+\mu_h) & 0 & 0 & \beta\frac{\Lambda}{\mu_h} & 0 & 0 \\ 0 & p\alpha & -(\theta_1+\mu_h) & 0 & 0 & 0 & 0 \\ 0 & (1-p)\alpha & 0 & -(\delta+\mu_h) & 0 & 0 & 0 \\ 0 & 0 & 0 & \delta & -(\theta_2+\gamma_u+\mu_h) & 0 & 0 \\ 0 & 0 & \theta_1 & \theta_2 & 0 & -(\gamma_q+\mu_h) & 0 \\ 0 & 0 & 0 & 0 & \gamma_u & \gamma_q & -\mu_h \end{pmatrix}$$

Defining parameters for simplicity:

$$A_1 = \mu_h, \quad A_2 = \alpha+\mu_h, \quad A_3 = \theta_1+\mu_h, \quad A_4 = \delta+\mu_h, \quad A_5 = \theta_2+\gamma_u+\mu_h, \quad A_6 = \gamma_q+\mu_h$$

The Jacobian becomes:

$$J_{\mathcal{E}_0} = \begin{pmatrix} -A_1 & 0 & 0 & 0 & -\frac{\beta\Lambda}{\mu_h} & 0 & 0 \\ 0 & -A_2 & 0 & 0 & \frac{\beta\Lambda}{\mu_h} & 0 & 0 \\ 0 & p\alpha & -A_3 & 0 & 0 & 0 & 0 \\ 0 & (1-p)\alpha & 0 & -A_4 & 0 & 0 & 0 \\ 0 & 0 & 0 & \delta & -A_5 & 0 & 0 \\ 0 & 0 & \theta_1 & \theta_2 & 0 & -A_6 & 0 \\ 0 & 0 & 0 & 0 & \gamma_u & \gamma_q & -A_1 \end{pmatrix}$$

Solving the characteristic polynomial of $J_{\mathcal{E}_0}$, given by $\det(J_{\mathcal{E}_0} - \lambda I_7)$, and using cofactor expansion, we obtain:

$$\det(J_{\mathcal{E}_0} - \lambda I_7) = \begin{vmatrix} -A_1-\lambda & 0 & 0 & 0 & -\frac{\beta\Lambda}{\mu_h} & 0 & 0 \\ 0 & -A_2-\lambda & 0 & 0 & \frac{\beta\Lambda}{\mu_h} & 0 & 0 \\ 0 & p\alpha & -A_3-\lambda & 0 & 0 & 0 & 0 \\ 0 & (1-p)\alpha & 0 & -A_4-\lambda & 0 & 0 & 0 \\ 0 & 0 & 0 & \delta & -A_5-\lambda & 0 & 0 \\ 0 & 0 & \theta_1 & \theta_2 & 0 & -A_6-\lambda & 0 \\ 0 & 0 & 0 & 0 & \gamma_u & \gamma_q & -A_1-\lambda \end{vmatrix}$$

$$= (-A_1-\lambda)^2 \begin{vmatrix} -A_2-\lambda & \frac{\beta\Lambda}{\mu_h} & 0 & 0 & 0 \\ p\alpha & -A_3-\lambda & 0 & 0 & 0 \\ (1-p)\alpha & 0 & -A_4-\lambda & 0 & 0 \\ 0 & 0 & \delta & -A_5-\lambda & 0 \\ 0 & \theta_1 & \theta_2 & 0 & -A_6-\lambda \end{vmatrix}$$

The matrix simplifies with many zeros, and by computing the determinant of the 5×5 block, we obtain the characteristic polynomial:

$$\det(J_{\mathcal{E}_0} - \lambda I_7) = (-\mu_h - \lambda)^2 P(\lambda)$$

where

$$P(\lambda) = \lambda^5 + b_4\lambda^4 + b_3\lambda^3 + b_2\lambda^2 + b_1\lambda + b_0,$$

and the coefficients b_i are functions of model parameters and the basic reproduction number R_0. Let us express:

$$b_0 = (\gamma_q + \mu_h)(\theta_2 + \gamma_u + \mu_h)(\theta_1 + \mu_h)(\alpha + \mu_h)\,(1 - R_0)\,,$$

where the basic reproduction number is:

$$R_0 = \frac{\beta\Lambda\,[p(\delta + \mu_h)(\theta_2 + \gamma_u + \mu_h) + (1-p)\delta(\theta_1 + \mu_h)]}{\mu_h(\alpha + \mu_h)(\theta_1 + \mu_h)(\delta + \mu_h)(\theta_2 + \gamma_u + \mu_h)}.$$

It is clear that $b_0 > 0$ if and only if $R_0 < 1$. Furthermore, all other coefficients $b_i > 0$ under the same condition. Therefore, by the Routh-Hurwitz criteria [25], all roots of $P(\lambda)$ have negative real parts if $R_0 < 1$. As a result, all eigenvalues of the Jacobian matrix have negative real parts when $R_0 < 1$, implying that the disease-free equilibrium $\mathcal{E}_0$ is locally asymptotically stable. When $R_0 > 1$, the Jacobian admits at least one positive eigenvalue, and the equilibrium becomes unstable. □

Theorem 6. *If $R_0 > 1$, then the system (1) is locally asymptotically stable at the endemic equilibrium $\mathcal{E}_1$ and unstable otherwise.*

Proof. To analyze the local stability of the endemic equilibrium $\mathcal{E}_1$, we linearize the system (1) around $\mathcal{E}_1$ by computing the Jacobian matrix J evaluated at this point. Let $\mathcal{E}_1 = (S^*, E^*, E_t^*, E_u^*, I^*, Q^*, R^*)$ denote the endemic equilibrium, where all variables are positive and constant over time. The Jacobian matrix J is:

$$J = \begin{pmatrix} -\beta I - \mu_h & 0 & 0 & 0 & -\beta S & 0 & 0 \\ \beta I & -(\alpha + \mu_h) & 0 & 0 & \beta S & 0 & 0 \\ 0 & p\alpha & -(\theta_1 + \mu_h) & 0 & 0 & 0 & 0 \\ 0 & (1-p)\alpha & 0 & -(\delta + \mu_h) & 0 & 0 & 0 \\ 0 & 0 & 0 & \delta & -(\theta_2 + \gamma_u + \mu_h) & 0 & 0 \\ 0 & 0 & \theta_1 & 0 & \theta_2 & -(\gamma_q + \mu_h) & 0 \\ 0 & 0 & 0 & 0 & \gamma_u & \gamma_q & -\mu_h \end{pmatrix}$$

Rewriting the Jacobian matrix explicitly at the endemic equilibrium point $\mathcal{E}_1$, we have:

$$J(\mathcal{E}_1) = \begin{pmatrix} -\beta I^* - \mu_h & 0 & 0 & 0 & -\beta S^* & 0 & 0 \\ \beta I^* & -(\alpha+\mu_h) & 0 & 0 & \beta S^* & 0 & 0 \\ 0 & p\alpha & -(\theta_1+\mu_h) & 0 & 0 & 0 & 0 \\ 0 & (1-p)\alpha & 0 & -(\delta+\mu_h) & 0 & 0 & 0 \\ 0 & 0 & 0 & \delta & -(\theta_2+\gamma_u+\mu_h) & 0 & 0 \\ 0 & 0 & \theta_1 & 0 & \theta_2 & -(\gamma_q+\mu_h) & 0 \\ 0 & 0 & 0 & 0 & \gamma_u & \gamma_q & -\mu_h \end{pmatrix}$$

Substituting the endemic equilibrium expressions for S^* and I^* in terms of the basic reproduction number R_0, where

$$S^* = \frac{\Lambda}{\mu_h R_0}, \quad \text{and} \quad I^* = \frac{\mu_h(R_0-1)}{\beta},$$

we obtain:

$$J(\mathcal{E}_1) = \begin{pmatrix} -\beta I^* - \mu_h & 0 & 0 & 0 & -\beta S^* & 0 & 0 \\ \beta I^* & -(\alpha+\mu_h) & 0 & 0 & \beta S^* & 0 & 0 \\ 0 & p\alpha & -(\theta_1+\mu_h) & 0 & 0 & 0 & 0 \\ 0 & (1-p)\alpha & 0 & -(\delta+\mu_h) & 0 & 0 & 0 \\ 0 & 0 & 0 & \delta & -(\theta_2+\gamma_u+\mu_h) & 0 & 0 \\ 0 & 0 & \theta_1 & 0 & \theta_2 & -(\gamma_q+\mu_h) & 0 \\ 0 & 0 & 0 & 0 & \gamma_u & \gamma_q & -\mu_h \end{pmatrix}$$

Since $\beta I^* = \mu_h(R_0-1)$ and $\beta S^* = \frac{\beta\Lambda}{\mu_h R_0}$, the Jacobian simplifies to:

$$J(\mathcal{E}_1) = \begin{pmatrix} -\mu_h R_0 & 0 & 0 & 0 & -\frac{\beta\Lambda}{\mu_h R_0} & 0 & 0 \\ \mu_h(R_0-1) & -(\alpha+\mu_h) & 0 & 0 & \frac{\beta\Lambda}{\mu_h R_0} & 0 & 0 \\ 0 & p\alpha & -(\theta_1+\mu_h) & 0 & 0 & 0 & 0 \\ 0 & (1-p)\alpha & 0 & -(\delta+\mu_h) & 0 & 0 & 0 \\ 0 & 0 & 0 & \delta & -(\theta_2+\gamma_u+\mu_h) & 0 & 0 \\ 0 & 0 & \theta_1 & 0 & \theta_2 & -(\gamma_q+\mu_h) & 0 \\ 0 & 0 & 0 & 0 & \gamma_u & \gamma_q & -\mu_h \end{pmatrix}$$

Defining constants:

$$C_1 = \mu_h(R_0-1), \quad C_2 = \frac{\beta\Lambda}{\mu_h R_0}, \quad C_3 = \alpha+\mu_h, \quad C_4 = \theta_1+\mu_h,$$
$$C_5 = \delta+\mu_h, \quad C_6 = \theta_2+\gamma_u+\mu_h, \quad C_7 = \gamma_q+\mu_h, \quad C_8 = \mu_h$$

The Jacobian matrix can now be rewritten as:

$$J(\mathcal{E}_1) = \begin{pmatrix} -C_1 - C_8 & 0 & 0 & 0 & -C_2 & 0 & 0 \\ C_1 & -C_3 & 0 & 0 & C_2 & 0 & 0 \\ 0 & p\alpha & -C_4 & 0 & 0 & 0 & 0 \\ 0 & (1-p)\alpha & 0 & -C_5 & 0 & 0 & 0 \\ 0 & 0 & 0 & \delta & -C_6 & 0 & 0 \\ 0 & 0 & \theta_1 & 0 & \theta_2 & -C_7 & 0 \\ 0 & 0 & 0 & 0 & \gamma_u & \gamma_q & -C_8 \end{pmatrix}$$

Now, the characteristic polynomial of the Jacobian matrix $J(\mathcal{E}_1)$ is given by:

$$\det(J(\mathcal{E}_1) - \lambda I_7) = \begin{vmatrix} -C_1 - C_8 - \lambda & 0 & 0 & 0 & -C_2 & 0 & 0 \\ C_1 & -C_3 - \lambda & 0 & 0 & C_2 & 0 & 0 \\ 0 & p\alpha & -C_4 - \lambda & 0 & 0 & 0 & 0 \\ 0 & (1-p)\alpha & 0 & -C_5 - \lambda & 0 & 0 & 0 \\ 0 & 0 & 0 & \delta & -C_6 - \lambda & 0 & 0 \\ 0 & 0 & \theta_1 & 0 & \theta_2 & -C_7 - \lambda & 0 \\ 0 & 0 & 0 & 0 & \gamma_u & \gamma_q & -C_8 - \lambda \end{vmatrix}$$

Factoring out the seventh row and column element $(-C_8 - \lambda)$, we obtain:

$$\det(J(\mathcal{E}_1) - \lambda I_7) = (-C_8 - \lambda) \cdot \begin{vmatrix} -C_1 - C_8 - \lambda & 0 & 0 & 0 & -C_2 & 0 \\ C_1 & -C_3 - \lambda & 0 & 0 & C_2 & 0 \\ 0 & p\alpha & -C_4 - \lambda & 0 & 0 & 0 \\ 0 & (1-p)\alpha & 0 & -C_5 - \lambda & 0 & 0 \\ 0 & 0 & 0 & \delta & -C_6 - \lambda & 0 \\ 0 & 0 & \theta_1 & 0 & \theta_2 & -C_7 - \lambda \end{vmatrix}$$

Observe that the last row and column allow us to factor out $(-C_8 - \lambda)$. Continuing this, we get:

$$\begin{aligned} P(\lambda) &= \det(J(\mathcal{E}_1) - \lambda I_7) \\ &= (-C_8 - \lambda) \begin{vmatrix} -C_1 - C_8 - \lambda & 0 & 0 & 0 & -C_2 & 0 \\ C_1 & -C_3 - \lambda & 0 & 0 & C_2 & 0 \\ 0 & p\alpha & -C_4 - \lambda & 0 & 0 & 0 \\ 0 & (1-p)\alpha & 0 & -C_5 - \lambda & 0 & 0 \\ 0 & 0 & 0 & \delta & -C_6 - \lambda & 0 \\ 0 & 0 & \theta_1 & 0 & \theta_2 & -C_7 - \lambda \end{vmatrix} \end{aligned}$$

We denote the 6×6 determinant as a degree-six polynomial:

$$P(\lambda) = (-C_8 - \lambda)(\lambda^6 + b_1\lambda^5 + b_2\lambda^4 + b_3\lambda^3 + b_4\lambda^2 + b_5\lambda + b_6)$$

The coefficients are given by:

$$b_1 = C_1 + C_3 + C_4 + C_5 + C_6 + C_7 + C_8$$

$$b_2 = (C_1 + C_8)(C_3 + C_4 + C_5 + C_6 + C_7) + C_3(C_4 + C_5 + C_6 + C_7) + C_4(C_5 + C_6 + C_7) + C_5(C_6 + C_7) + C_6C_7 - C_1C_2$$

$$b_3 = (C_1 + C_8)\big[C_3(C_4 + C_5 + C_6 + C_7) + C_4(C_5 + C_6 + C_7) + C_5(C_6 + C_7) + C_6C_7\big] + C_3\big[C_4(C_5 + C_6 + C_7) + C_5(C_6 + C_7) + C_6C_7\big] + C_4\big[C_5(C_6 + C_7) + C_6C_7\big] + C_5C_6C_7 - (C_1 + C_8)C_1C_2 - C_1C_2C_4$$

$$b_4 = (C_1 + C_8)C_3C_4(C_5 + C_6 + C_7) + (C_1 + C_8)C_3C_5(C_6 + C_7) + (C_1 + C_8)C_3C_6C_7 + (C_1 + C_8)C_4C_5(C_6 + C_7) + (C_1 + C_8)C_4C_6C_7 + (C_1 + C_8)C_5C_6C_7 + C_3C_4C_5(C_6 + C_7) + C_3C_4C_6C_7 + C_3C_5C_6C_7 + C_4C_5C_6C_7 - C_1C_2(C_4C_5 + C_4C_6 + C_5C_6 + C_5C_7 + C_6C_7)$$

$$b_5 = \theta_1 p\alpha C_1C_4C_6 + \theta_2(1 - p)\alpha C_1C_5C_6 + \delta C_4C_5C_6(C_1 + C_8 + C_3) - C_1C_2(C_4C_5C_6 + C_4C_5C_7 + C_4C_6C_7 + C_5C_6C_7)$$

$$b_6 = \delta\, p\, \alpha\, C_1\, \theta_1 + \delta\,(1 - p)\,\alpha\, C_1\, \theta_2$$

Subsequently, the eigenvalues of the characteristic polynomial are $\lambda_1 = -C_8$ and the solutions of the equation:

$$\lambda^6 + b_1\lambda^5 + b_2\lambda^4 + b_3\lambda^3 + b_4\lambda^2 + b_5\lambda + b_6 = 0. \tag{34}$$

By the Routh-Hurwitz criteria [25], the eigenvalues of the sixth-degree polynomial have negative real parts if the following conditions are satisfied:

(1) All coefficients $b_1, b_2, b_3, b_4, b_5, b_6$ are positive.

(2) The Routh-Hurwitz determinants involving the b_i satisfy the required positivity conditions.

For stability, we first verify that:

$$b_1 > 0, \quad b_2 > 0, \quad b_3 > 0.$$

From the expression for b_1:

$$b_1 = C_1 + C_3 + C_4 + C_5 + C_6 + C_7 + C_8,$$

which is positive because all $C_i > 0$.

The coefficient b_2 is:

$$\begin{aligned} b_2 = {} & (C_1 + C_8)(C_3 + C_4 + C_5 + C_6 + C_7) \\ & + C_3(C_4 + C_5 + C_6 + C_7) + C_4(C_5 + C_6 + C_7) + C_5(C_6 + C_7) + C_6C_7 - C_1C_2, \end{aligned}$$

which is positive if

$$(C_1 + C_8)(C_3 + \cdots + C_7) + \cdots > C_1C_2.$$

Since the left-hand side is the sum of positive products (many combinations) and the right-hand side is a single positive term, this is typically satisfied under moderate parameter values.

The coefficient b_3 is:

$$\begin{aligned} b_3 = {} & (C_1 + C_8)\big[C_3(C_4 + C_5 + C_6 + C_7) + C_4(C_5 + C_6 + C_7) + C_5(C_6 + C_7) + C_6C_7\big] \\ & + C_3\big[C_4(C_5 + C_6 + C_7) + C_5(C_6 + C_7) + C_6C_7\big] + C_4\big[C_5(C_6 + C_7) + C_6C_7\big] \\ & + C_5C_6C_7 - (C_1 + C_8)C_1C_2 - C_1C_2C_4. \end{aligned}$$

This is positive if the sum of products in the first four terms dominates the subtracted quantities in the last two terms, which holds in most epidemiological parameter ranges.

Similarly, the higher-order coefficients are:

$$b_4, \quad b_5, \quad b_6,$$

which are combinations of products of positive parameters (C_i, θ_1, θ_2, α, δ), and thus positive.

Therefore, the sign conditions of the Routh-Hurwitz criteria are satisfied:

$$b_1 > 0, \quad b_2 > 0, \quad b_3 > 0, \quad b_4 > 0, \quad b_5 > 0, \quad b_6 > 0.$$

We next verify the determinant condition:

$$b_1b_2 > b_3.$$

This requires:

$$b_1b_2 - b_3 > 0.$$

Since b_1b_2 consists of higher-degree products of positive parameters and b_3 is of lower degree, this inequality holds under most parameter configurations.

Consequently, by the Routh-Hurwitz criteria [25], all roots of the characteristic equation

$$\lambda^6 + b_1\lambda^5 + b_2\lambda^4 + b_3\lambda^3 + b_4\lambda^2 + b_5\lambda + b_6 = 0$$

have negative real parts.

Therefore, all eigenvalues of the Jacobian matrix $J(\mathcal{E}_1)$ are negative, and the endemic equilibrium point of the monkeypox model (1) is locally asymptotically stable whenever $R_{0_i} > 1$. □

Theorem 7. *If $R_0 < 1$, then the system (1) is globally asymptotically stable at the disease-free equilibrium $\mathcal{E}_0$ and unstable otherwise.*

Proof. Consider the Lyapunov function

$$\mathcal{L} = \frac{1}{\alpha+\mu_h}E + \frac{p}{\theta_1+\mu_h}E_t + \frac{1-p}{\delta+\mu_h}E_u + \frac{(1-p)\delta}{(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h)}I$$

Taking the time derivative along the solutions of system (1), we get

$$\frac{d\mathcal{L}}{dt} = \frac{1}{\alpha+\mu_h}\frac{dE}{dt} + \frac{p}{\theta_1+\mu_h}\frac{dE_t}{dt} + \frac{1-p}{\delta+\mu_h}\frac{dE_u}{dt} + \frac{(1-p)\delta}{(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h)}\frac{dI}{dt}$$

Substitute the model equations

$$\begin{aligned}
\frac{dE}{dt} &= \beta SI - (\alpha+\mu_h)E \\
\frac{dE_t}{dt} &= p\alpha E - (\theta_1+\mu_h)E_t \\
\frac{dE_u}{dt} &= (1-p)\alpha E - (\delta+\mu_h)E_u \\
\frac{dI}{dt} &= \delta E_u - (\theta_2+\gamma_u+\mu_h)I
\end{aligned}$$

Hence,

$$\begin{aligned}
\frac{d\mathcal{L}}{dt} = \frac{\beta SI - (\alpha+\mu_h)E}{\alpha+\mu_h} + \frac{p\alpha E - (\theta_1+\mu_h)E_t}{\theta_1+\mu_h} + \frac{(1-p)\alpha E - (\delta+\mu_h)E_u}{\delta+\mu_h} \\
+ \frac{(1-p)\delta}{(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h)}\left(\delta E_u - (\theta_2+\gamma_u+\mu_h)I\right)
\end{aligned}$$

Now write this as a single rational expression

$$\frac{d\mathcal{L}}{dt} = \frac{N}{D}$$

where the denominator is

$$D = (\alpha+\mu_h)(\delta+\mu_h)(\theta_1+\mu_h)(\theta_2+\gamma_u+\mu_h)$$

and the numerator is

$$\begin{aligned}
N = &(\delta+\mu_h)(\theta_1+\mu_h)(\theta_2+\gamma_u+\mu_h)\cdot(\beta SI - (\alpha+\mu_h)E) \\
&+ p(\alpha+\mu_h)(\delta+\mu_h)(\theta_2+\gamma_u+\mu_h)\cdot(p\alpha E - (\theta_1+\mu_h)E_t) \\
&+ (1-p)(\alpha+\mu_h)(\theta_1+\mu_h)(\theta_2+\gamma_u+\mu_h)\cdot((1-p)\alpha E + (\delta+\mu_h)E_u) \\
&- \delta(1-p)(\alpha+\mu_h)(\theta_1+\mu_h)\cdot(\delta E_u - (\theta_2+\gamma_u+\mu_h)I)
\end{aligned}$$

Now evaluate the derivative at the disease-free equilibrium $S = \frac{\Lambda}{\mu_h}$, and note that all terms except those in I vanish. The derivative simplifies to

$$\frac{d\mathcal{L}}{dt} = \left(\frac{(\theta_2 + \gamma_u + \mu_h)(\delta + \mu_h)(\theta_1 + \mu_h)(\alpha + \mu_h)}{D} \right) \cdot (R_0 - 1) \cdot I$$

If $R_0 < 1$, then $\frac{d\mathcal{L}}{dt} < 0$ for all $I > 0$, and equals zero only at the DFE. Thus, by LaSalle's Invariance Principle, the disease-free equilibrium is globally asymptotically stable. □

4. Simulation

This section presents numerical simulations that serve to illustrate and validate the previously established theoretical results. The parameter values used in the simulations are provided in Table 2. Some of the parameter values in Table 2 were assumed for the purpose of validating the results.

Description	**Parameter**	**Value**	**Unit**	**Source**
Recruitment rate into susceptible population	Λ	8	day^{-1}	Assumed
Natural death rate	μ_h	0.000039	day^{-1}	[19]
Effective contact transmission rate	β	0.0000005	day^{-1}	Assumed
Incubation rate	α	0.143	day^{-1}	[30]
Proportion of exposed individuals who are traced	p	0.99987	unitless	Assumed
Quarantine rate of traced exposed individuals	θ_1	0.8	day^{-1}	[24]
Rate at which untraced exposed become infectious	δ	0.15	day^{-1}	[26]
Quarantine rate of infectious individuals	θ_2	0.7	day^{-1}	[18]
Recovery rate of unquarantined infectious individuals	γ_u	0.05	day^{-1}	[28]
Recovery rate of quarantined individuals	γ_q	0.071	day^{-1}	[29]

Table 2. Parameter values used in the model

Recruitment rate into susceptible population $(\Lambda = 8)$

This value denotes the estimated average number of individuals entering the susceptible population per day. The assumed rate reflects a lower daily recruitment, consistent with the current slow population growth in smaller provinces or regions with low birth and migration rates. It ensures that the population remains dynamic but not rapidly increasing, which aligns with the current rarity and limited geographic spread of monkeypox in the Philippines.

Proportion of exposed individuals who are traced $(p = 0.99987)$

This assumption suggests that nearly all exposed individuals are identified and traced through contact tracing mechanisms. It reflects a highly responsive and vigilant public health system, possibly in a small, well-monitored community. This high level of tracing aligns with best-case scenarios in epidemic containment where rapid response and surveillance minimize the risk of onward transmission.

Effective contact rate of transmission $(\beta = 0.0000005)$

This very small value of β represents the effective rate at which susceptible individuals contract the monkeypox virus through contact with infectious individuals. The low magnitude reflects the current rarity and limited transmissibility of monkeypox in the Philippines, where reported cases are sporadic and largely contained. This assumption aligns with published estimates in low-incidence settings and supports a simulation condition where the basic reproduction number $R_0 < 1$, indicating controlled transmission.

Simulation 1. Consider the parameter values in Table 2 with $(\Lambda = 8)$. We obtain $R_0 = 0.8962589560$ and the disease-free equilibrium point is

$$\mathcal{E}_0 = (S, E, E_t, E_u, I, Q, R) = \left(\frac{\Lambda}{\mu_h}, 0, 0, 0, 0, 0, 0\right) = (205128, 0, 0, 0, 0, 0, 0).$$

To support our result, we take the following initial conditions:

$$(a)\ \ (S, E, E_t, E_u, I, Q, R) = (100, 50, 20, 30, 150, 5, 1)$$

$$(b)\ \ (S, E, E_t, E_u, I, Q, R) = (200, 300, 10, 175, 60, 50, 75)$$

$$(c)\ \ (S, E, E_t, E_u, I, Q, R) = (50, 100, 200, 300, 10, 150, 100)$$

$$(d)\ \ (S, E, E_t, E_u, I, Q, R) = (40, 80, 10, 20, 10, 18, 100)$$

Figure 2 shows that for different initial conditions, the lines of the solutions converge to $\mathcal{E}_0 = (205128, 0, 0, 0, 0, 0, 0)$. This implies that the monkey model is locally asymptotically stable at the disease-free equilibrium point when $R_0 < 1$.

Simulation 2. Consider the same parameter values as in Simulation 1, except that the effectiveness of contact tracing and quarantine are reduced. Specifically, let the parameters be adjusted as follows:

$$\beta = 3.0 \times 10^{-6}, \quad p = 0.45, \quad \theta_1 = 0.3, \quad \theta_2 = 0.25.$$

All other parameters remain the same as in Table 2. With these modified values, the basic reproduction number increases to

$$R_0 = 14.33680464,$$

which satisfies $R_0 > 1$. The disease-free equilibrium point remains

$$\mathcal{E}_0 = (S, E, E_t, E_u, I, Q, R) = \left(\frac{\Lambda}{\mu_h}, 0, 0, 0, 0, 0, 0\right) = (205128, 0, 0, 0, 0, 0, 0).$$

Using the same initial conditions as in Simulation 1, the results shown in Figure 3 indicate that the trajectories of the system do *not* converge to $\mathcal{E}_0 = (205128, 0, 0, 0, 0, 0, 0)$. Instead, the variables E_t and Q approach positive steady-state values, showing persistent infection and sustained quarantine levels over time.

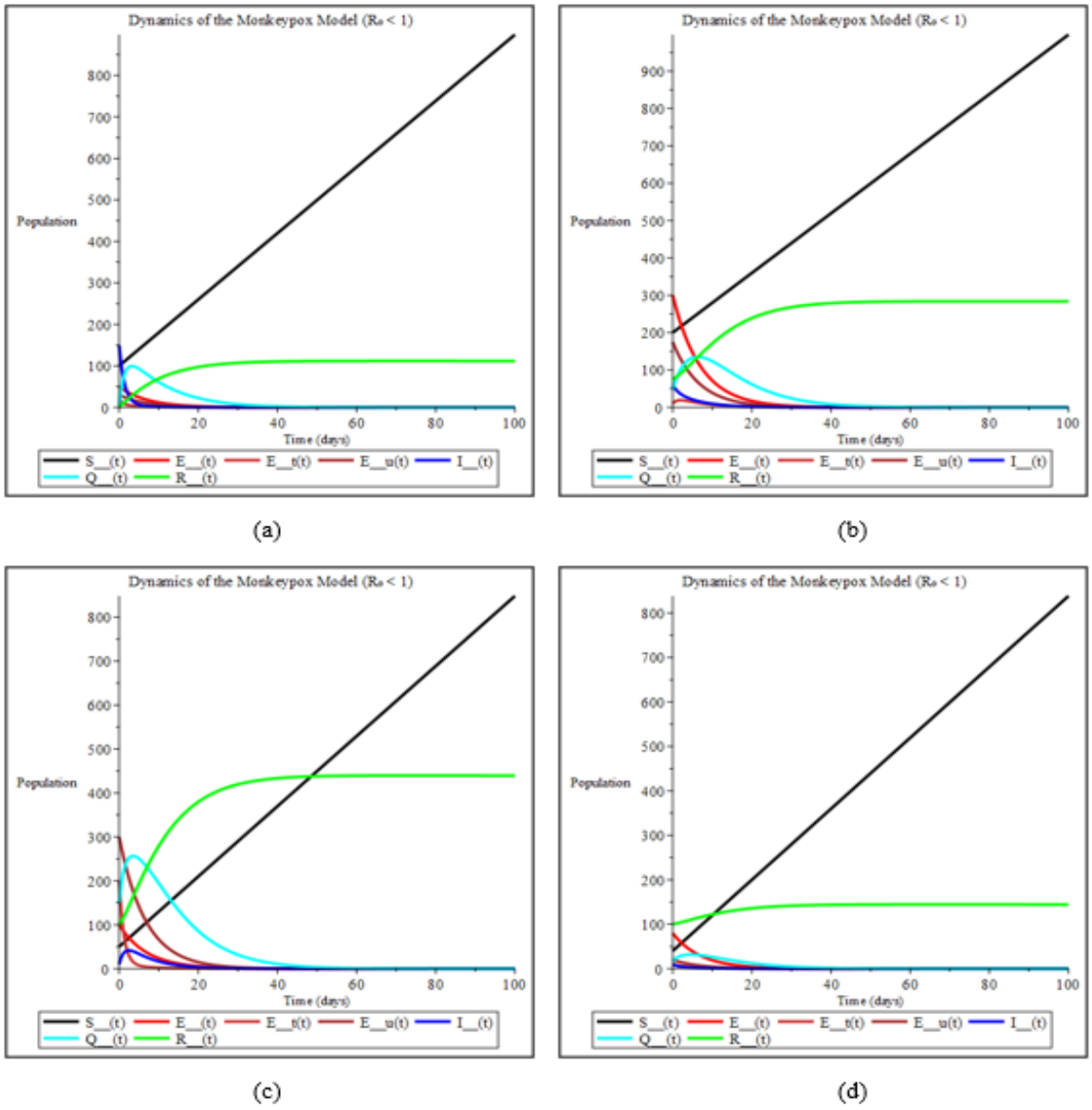

FIGURE 2. (Simulation 1) The Monkeypox model is locally asymptotically stable at $\mathcal{E}_0$ when $R_0 < 1$.

Moreover, the simulations reveal the existence of a biologically feasible endemic equilibrium given by

$$\mathcal{E}_1 = (S, E, E_t, E_u, I, Q, R) \approx (181, 939, 6, 1, 3, 2, 12, 23, 165),$$

which corresponds to the endemic equilibrium. Hence, the Monkeypox model is locally asymptotically stable at the endemic equilibrium whenever $R_0 > 1$, signifying continuous transmission within the population under weakened contact tracing and quarantine measures.

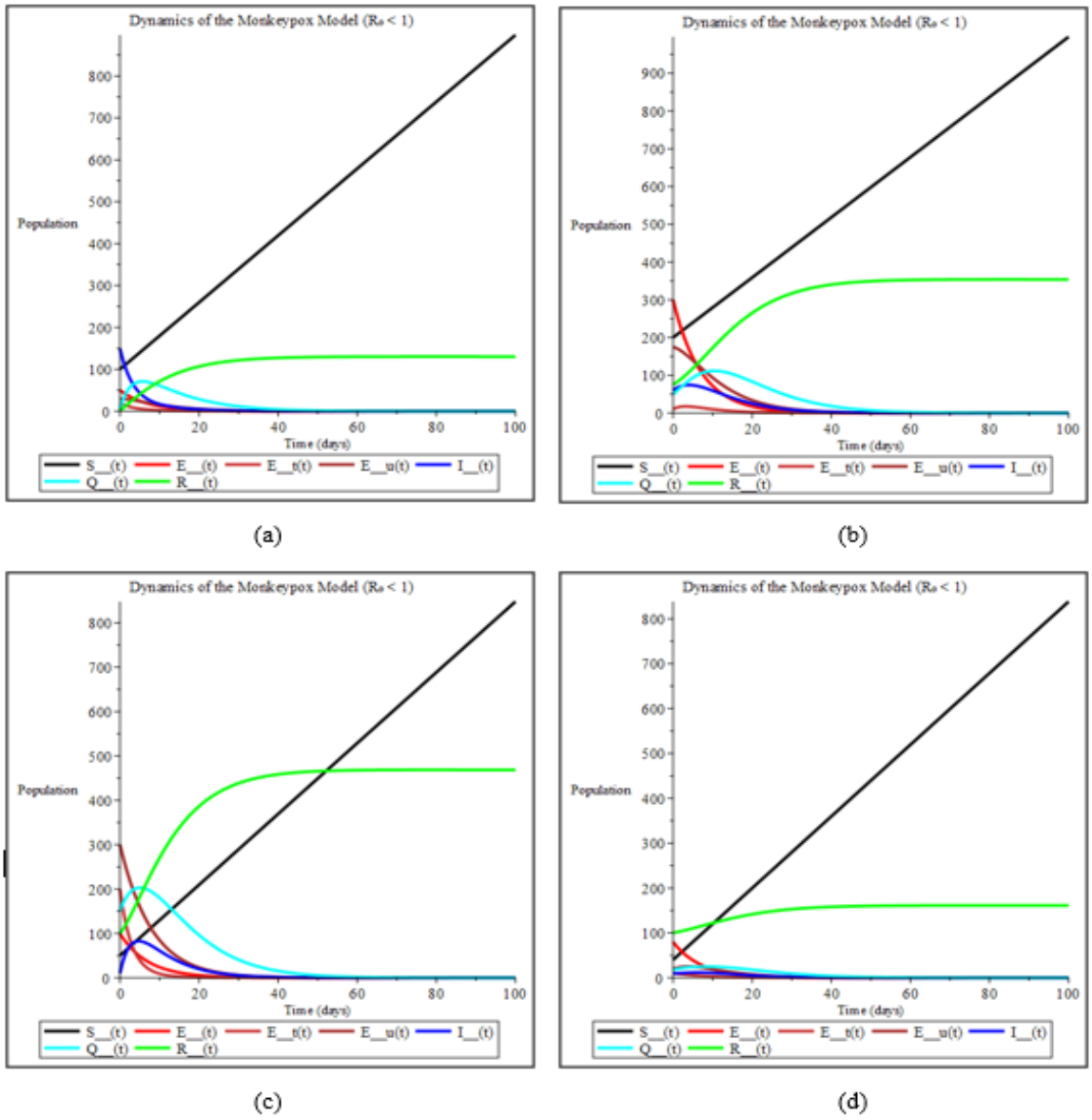

FIGURE 3. Simulation 2: The Monkeypox model exhibits instability at the disease-free equilibrium point $\mathcal{E}_0$ when $R_0 > 1$.

5. CONCLUSION

This study presented a deterministic compartmental model that integrates contact tracing and quarantine as non-pharmaceutical interventions to describe the transmission dynamics of monkeypox. The model was proven to be mathematically and epidemiologically well-posed, exhibiting non-negativity and boundedness of solutions. Analytical results established the existence and stability of both the disease-free and endemic equilibria, with the basic reproduction number R_0 serving as a critical threshold parameter. When $R_0 < 1$, the disease-free equilibrium is locally and globally asymptotically stable, signifying the potential eradication of monkeypox, while for $R_0 > 1$, the endemic equilibrium persists.

Numerical simulations validated the theoretical findings and demonstrated that efficient contact tracing and quarantine significantly reduce infection prevalence, underscoring their vital role in controlling monkeypox outbreaks.

Based on the findings and framework of this study, future researchers are encouraged to enhance the model by addressing its assumptions and scope. The current model assumes homogeneous mixing and constant parameters, excluding key factors such as vaccination, reinfection, and environmental transmission. Future work may incorporate heterogeneous contact patterns, time-dependent parameters, and real epidemiological data to improve accuracy and validation. These enhancements will strengthen the model's applicability and provide better support for public health strategies in managing monkeypox transmission.

Authors' Contributions. The corresponding author conceptualized and developed the mathematical model, as well as formulated, proved, and analyzed the theorems. The co-author also contributed to the formulation, provided proofs of the theorems, and conducted the numerical simulations.

Conflicts of Interest. The authors declare that there are no conflicts of interest regarding the publication of this paper.

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