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Mathematical Analysis of a Dynamic Model of Epidemic Influenced by Super-Spreaders [1]

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Abstract

In this paper, we develop a six dimensional compartment model to investigate the impact of super-spreader during an epidemic. Stability analysis of the model shows that the disease-free equilibrium is globally asymptotically stable if a certain threshold quantity, the basic reproduction number ($\mathcal{R}_0$), is less than unity. On the other hand, If $\mathcal{R}_0 > 1$, then the endemic equilibrium H^* is stable, and the disease persists. Sensitivity analysis indicates that the basic reproduction number $\mathcal{R}_0$ is most sensitive to the population recruitment rate Λ, the disease transmission rate β_1 and the disease super transmission rate β_2.

Keywords: super-spreader; stability; $SEIMQR$ model; sensitivity analysis

1 Introduction

The spread of infectious diseases is a serious threat to human life and health, and the number of deaths due to infectious diseases accounts for a quarter of the total deaths worldwide every year[1]. When an emerging infections disease occurs, a vaccine or effective drugs to treat the disease cannot be developed

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in time. This makes it very challenging to control the spread of the disease in both temporal and spatial dimensions, especially in some low-and middle-income countries, where medical resources are not fully guaranteed. Therefore, reducing the rate of disease transmission has become one of the most direct and effective means of prevention and control.

Reports of super-spreaders first appeared in the 20th century in the case of "Typhoid Mary" in 1900-1907 [2], when one person caused multiple infections as well as deaths. In addition, super spreaders have been seen in the transmission of several major infectious diseases (such as, tuberculosis, measles, Ebola hemorrhagic fever, malaria, SARS-COV, MERS-COV, etc [3–7, 12]). The presence of super spreaders can rapidly accelerate disease transmission in a short period of time, leading to mass infections and fatalities. This phenomenon is often linked to high contact rates or highly infectious viral strains and can help identify more transmissible variants. Studying superspreading events is essential for understanding the transmission mechanisms of infectious diseases, providing governments and health authorities with the scientific basis to develop effective policies and improve outbreak response. In summary, researching super spreaders aids in more effective disease control and mitigates their societal impact.

Mathematical modelling is a tool that can be used to visually simulate the impact of super-spreaders on disease, to predict disease trends and to assess the effectiveness of different prevention and control measures. In order to study the effect of super-spreaders on the disease, the following two main approaches have been used: One approach is to introduce a new independent compartment $M(t)$ to represent the disease information level related to super-spreader [8–10]. Ullah S, Zahir H et al. in [10] added an independent compartment $M(t)$, and uses common linear incidence $\frac{\beta SI}{N}$ to describe the disease transmission pattern.

The second approach introduces an expression for the transmission rate as a function of the transmission distance r. In [11], it is assumed that the normal probability of infection $w(r)$ is a decreasing function of the propagation distance r with a cut-off value r_0. Fujie R and Odagaki T argue that the probability of infection of a super-spreader $w(r)$ has the same cut-off value as the normal probability of infection r_0, but it is a constant rather than decreasing function. In [12], Walker D M et al. consider the rate of infection of an infected person to be constant, but the phenomenon of "super-spreading" cannot be ruled out. Mushanyu, J et al. consider different levels of spreaders in [13], giving the normal rate $\lambda_n(t)$ and the super-spreading rate $\lambda_{pi}(t)$ and classifying the super-spreader rate into two types and the propagation rate function is defined.

In the studies on superspreaders mentioned above, most of the models did not take into account the use of precautions by exposed and infected people.

There is currently no model that takes both factors into account, so we plan to develop a robust mathematical model to study the phenomenon of super-spreading that includes both exposed and isolated individuals.

The paper is organised as follows: In section 2 constructs the model and provides the relevant explanations. In section 3 describes the qualitative properties of the model including the non-negativity of the solutions, boundedness and the stability of the equilibrium points. In sections 4 and 5 provide the numerical results of the analysis of the model. Finally, the main results are summarised in the discussion section.

2 Model construction

Since the outbreak of SARS, the phenomenon of super transmission has become common in the process of disease transmission. Super carriers have a greater impact on susceptible individuals than general infected individuals. So it is necessary to divide infected individuals into general infected individuals and super infected individuals. As an extension of the basic model SEIQR, it is necessary to add a super propagator compartment M to obtain the SEIMQR model.

Then, the total population size at time t is $N(t) = S(t)+E(t)+I(t)+M(t)+Q(t)+R(t)$. We assume that the spread of the disease follows linear incidence, and that susceptible individuals can be infected by infected individuals as well as by super-spreaders. Then we have the model

$$\begin{cases} \dfrac{\mathrm{d}S}{\mathrm{d}t} = \Lambda - (\beta_1 I + \beta_2 M)S - dS, \\ \dfrac{\mathrm{d}E}{\mathrm{d}t} = (\beta_1 I + \beta_2 M)S - (k_1 + k_2 + d)E, \\ \dfrac{\mathrm{d}I}{\mathrm{d}t} = k_1 E - (\omega + \gamma + \nu + d)I, \\ \dfrac{\mathrm{d}M}{\mathrm{d}t} = k_2 E - (\mu + \delta + \nu + d)M, \\ \dfrac{\mathrm{d}Q}{\mathrm{d}t} = \omega I + \mu M - (d + \nu + \rho)Q, \\ \dfrac{\mathrm{d}R}{\mathrm{d}t} = \gamma I + \delta M + \rho Q - dR, \end{cases} \tag{2.1}$$

for subsequent calculations make $\varepsilon_1 = k_1 + k_2 + d; \varepsilon_2 = \omega + \gamma + \nu + d; \varepsilon_3 = \mu + \delta + \nu + d; \varepsilon_4 = \nu + \rho + d$.

In the model, all parameters are non-negative, where the recruitment rate of the susceptible population is Λ; the contact rate of the susceptible population with the infected population is β_1; the contact rate of the susceptible population with the super infected population is β_2; the proportions of exposed individuals transmitted to the infected and super infected populations

are the proportions k_1, k_2, respectively; the proportions of infected and super infected individuals entering the quarantined population are ω, μ, respectively; the disease mortality rate of the population is ν; the recovery rate of the super infected population is δ; the recovery rate of the quarantined population is ρ; the natural mortality rate of the population is d; the recovery rate of the infected population is γ. Then initial conditions are defined as follows,

$$S(0) > 0,\ E(0) > 0,\ I(0) > 0,\ M(0) > 0,\ Q(0) > 0,\ R(0) > 0. \tag{2.2}$$

3 Mathematical analysis

3.1 Non-negativity and Boundedness of solutions

In this section, we prove that every solution of system (2.1) is non-negative and uniformly bounded with initial conditions (2.2).

Theorem 3.1. *Every solution of model (2.1) with initial values (2.2) remains positive in* $\mathbb{R}^6_+$ *as* $t > 0$.

Proof. From the model system(2.1), we obtain

$$\begin{aligned}
\frac{\mathrm{d}S}{\mathrm{d}t}|_{S=0,E\geq 0,I\geq 0,M\geq 0,Q\geq 0,R\geq 0} &= \Lambda > 0,\\
\frac{\mathrm{d}E}{\mathrm{d}t}|_{S\geq 0,E=0,I\geq 0,M\geq 0,Q\geq 0,R\geq 0} &= (\beta_1 I + \beta_2 M)S \geq 0,\\
\frac{\mathrm{d}I}{\mathrm{d}t}|_{S\geq 0,E\geq 0,I=0,M\geq 0,Q\geq 0R\geq 0} &= k_1 E \geq 0,\\
\frac{\mathrm{d}M}{\mathrm{d}t}|_{S\geq 0,E\geq 0,I\geq 0,M=0,Q\geq 0.R\geq 0} &= k_2 E \geq 0,\\
\frac{\mathrm{d}Q}{\mathrm{d}t}|_{S\geq 0,E\geq 0,I\geq 0,M\geq 0,Q=0,R\geq 0} &= \omega I + \mu M \geq 0,\\
\frac{\mathrm{d}R}{\mathrm{d}t}|_{S\geq 0,E\geq 0,I\geq 0,M\geq 0,Q\geq 0,R=0} &= \gamma I + \delta M + \rho Q \geq 0.
\end{aligned} \tag{3.1}$$

Since the above rate is always non-negative on the boundary of a non-negative surface of $\mathcal{R}^6_+$, this means that the direction of the vector field is always pointing inwards. Therefore, from the non-negative cone, all solution trajectories will remain in the positive region and will not cross the boundary. This proves the correctness of the theorem.

Theorem 3.2. *Every solution of model (2.1) initiating in* $\mathbb{R}^6_+$ *is uniformly bounded in the region*
$\Delta_\epsilon = \left\{(S, E, I, M, Q, R) \in \mathbb{R}^6_+ : 0 \leq S + E + I + M + Q + R \leq \frac{\Lambda}{d} + \epsilon\right\}$ *for some* $\epsilon > 0$.

Proof. According to $N = S+E+I+M+Q+R$, then the time derivative

$$\begin{aligned}\frac{\mathrm{d}N}{\mathrm{d}t} &= \Lambda - dN - \nu(I+M+Q) \leq \Lambda - dN,\\ &\Rightarrow \frac{\mathrm{d}N}{\mathrm{d}t} + dN \leq \Lambda,\\ &\Rightarrow 0 \leq N \leq \frac{\Lambda}{d} + \left(N(0) - \frac{\Lambda}{d}\right) e^{-dt}.\end{aligned}$$

For $t \to +\infty, 0 \leq N \leq \frac{\Lambda}{d}$. Therefore, the solution trajectory initiating inside the region Δ_ϵ confined within the region and if it starts from outside Δ_ϵ then it enters into the region after a finite time and approaches towards $\frac{\Lambda}{d}$. Hence the theorem.

3.2 Existence of equilibrium

In order to study the properties of the relevant dynamics of model (2.1), it is first necessary to determine the existence of the model equilibrium point. The disease-free equilibrium corresponds to the extinct state of the disease, while the local equilibrium corresponds to the state in which the disease will eventually persist in the population for a period of time. As mentioned earlier, model (2.1) has disease-free equilibrium (DFE), $H_0 = (\frac{\Lambda}{d}, 0, 0, 0, 0, 0)$. Linearizing model (2.1) at the DFE and using the next generation matrix method, we find the basic reproduction number $\mathcal{R}_0$, is given by

$$\mathcal{R}_0 = \frac{\Lambda(\beta_1 k_1 \varepsilon_3 + \beta_2 k_2 \varepsilon_2)}{d\varepsilon_1\varepsilon_2\varepsilon_3}.$$

Note, positive equilibrium $H^* = (S^*, E^*, I^*, M^*, Q^*, R^*)$ is exists and satisfies

$$\begin{cases} \Lambda - (\beta_1 I^* + \beta_2 M^*)S^* - dS^* = 0,\\ (\beta_1 I^* + \beta_2 M^*)S^* - \varepsilon_1 E^* = 0,\\ k_1 E^* - \varepsilon_2 I^* = 0,\\ k_2 E^* - \varepsilon_3 M^* = 0,\\ \omega I^* + \mu M^* - \varepsilon_4 Q^* = 0,\\ \gamma I^* + \delta M^* + \rho Q^* - dR^* = 0, \end{cases} \tag{3.2}$$

which gives

$$\begin{cases} E^* = \frac{\varepsilon_2 I^*}{k_1},\\ M^* = \frac{k_2\varepsilon_2 I^*}{k_1\varepsilon_3},\\ Q^* = \frac{[\omega k_1\varepsilon_3 + \mu k_2\varepsilon_2]I^*}{k_1\varepsilon_3\varepsilon_4},\\ R^* = \frac{[\gamma k_1\varepsilon_3\varepsilon_4 + \delta k_2\varepsilon_2\varepsilon_4 + \rho(\omega k_1\varepsilon_3 + \mu k_2\varepsilon_2)]I^*}{dk_1\varepsilon_3\varepsilon_4},\\ S^* = \frac{\Lambda}{d + \left[\left(\beta_1 + \frac{\beta_2 k_2\varepsilon_2}{k_1\varepsilon_3}\right)\right]I^*} = \frac{\varepsilon_1\varepsilon_2\varepsilon_3}{\beta_1 k_1\varepsilon_3 + \beta_2 k_2\varepsilon_2}. \end{cases} \tag{3.3}$$

From the two expressions of S^* in equation (3.2), can get

$$I^* = \frac{\Lambda k_1(\beta_1 k_1 \varepsilon_3 + \beta_1 k_2 \varepsilon_2) - d k_1 \varepsilon_1 \varepsilon_2 \varepsilon_3}{\varepsilon_1 \varepsilon_2 (\beta_1 k_1 \varepsilon_3 + \beta_1 k_2 \varepsilon_2)} = \frac{\Lambda k_1}{\varepsilon_1 \varepsilon_2}(1 - \frac{1}{\mathcal{R}_0}). \tag{3.4}$$

Now, using the I^* in equations (3.3), can obtain the positive values of S^*, E^*, M^*,Q^* and R^*, respectively. Thus, the model (2.1) consists of a positive endemic steady state H^* when $\mathcal{R}_0 > 1$.

3.3 Stability of the equilibrium point

In this subsection, the steady-state situation of the equilibrium point will be discussed.

In determining the local stability of the disease free equilibrium point, the equations of the system (2.1) is linearized by using Jacobian matrix(J). The trace-determinant technique as presented by [15], is used to determine local stability of disease-free equilibrium point.

Theorem 3.3. *The DFE H_0 is locally asymptotically stable if $\mathcal{R}_0 < 1$, and is unstable if $\mathcal{R}_0 > 1$.*

Proof. The Jacobian matrix of model (2.1) at H_0 is as follow

$$J(H_0) = \begin{pmatrix} -d & 0 & -\frac{\beta_1 \Lambda}{d} & -\frac{\beta_2 \Lambda}{d} & 0 & 0 \\ 0 & -\varepsilon_1 & \frac{\beta_1 \Lambda}{d} & \frac{\beta_2 \Lambda}{d} & 0 & 0 \\ 0 & k_1 & -\varepsilon_2 & 0 & 0 & 0 \\ 0 & k_2 & 0 & -\varepsilon_3 & 0 & 0 \\ 0 & 0 & \omega & \mu & -\varepsilon_4 & 0 \\ 0 & 0 & \gamma & \delta & \rho & -d \end{pmatrix}.$$

According to the linearized of the Jacobian matrix $J(H_0)$, the trace and determinant are calculated as follows:

1)Trace of the Jacobian matrix $J(H_0)$, the $TrJ(H_0)$ is given by

$$TrJ(H_0) = -2d - \varepsilon_1 - \varepsilon_2 - \varepsilon_3 - \varepsilon_4 < 0.$$

2)By computing the determinant $DetJ(H_0)$ and the result is given by

$$DetJ(H_0) = -d(\Lambda \beta_1 k_1 \varepsilon_3 \varepsilon_4 - d\varepsilon_1 \varepsilon_2 \varepsilon_3 \varepsilon_4 + \Lambda \beta_2 k_2 \varepsilon_2 \varepsilon_4),$$

simplified, become

$$DetJ(H_0) = d^2 \varepsilon_1 \varepsilon_2 \varepsilon_3 \varepsilon_4 \Big(1 - \frac{\Lambda(\beta_1 k_1 \varepsilon_3 + \beta_2 k_2 \varepsilon_2)}{d\varepsilon_1 \varepsilon_2 \varepsilon_3}\Big) = d^2 \varepsilon_1 \varepsilon_2 \varepsilon_3 \varepsilon_4 (1 - \mathcal{R}_0).$$

When $\mathcal{R}_0 < 1$, the $DetJ(H_0) > 0$, the DFE is locally asymptotically stable by the trace determinant technique. Hence the theorem.

Theorem 3.4. *The DFE H_0 is globally asymptotically stable if $\mathcal{R}_0 < 1$, otherwise, it's unstable when $\mathcal{R}_0 > 1$.*

Proof. Constructing a suitable Lyapunov function $L : \mathbb{R}^4_+ \to \mathbb{R}$, defined as

$$L(t) = E + \frac{\beta_1 S_0}{\varepsilon_2} I + \frac{\beta_2 S_0}{\varepsilon_3} M, \tag{3.5}$$

suppose that: $S(t) \leq S_0$, then take the derivative of $L(t)$ along the solution path of model (2.1), and get

$$\begin{aligned} \frac{\mathrm{d}L(t)}{\mathrm{d}t} &\leq \beta_1 I S_0 + \beta_2 M S_0 - \varepsilon_1 E + \frac{\beta_1 S_0}{\varepsilon_2}\left(k_1 E - \varepsilon_2 I\right) + \frac{\beta_2 S_0}{\varepsilon_3}\left(k_2 E - \varepsilon_3 M\right) \\ &= \frac{\beta_1 S_0 k_1}{\varepsilon_2} E + \frac{\beta_2 S_0 k_2}{\varepsilon_3} E - \varepsilon_1 E \\ &= \varepsilon_1 E\left(\frac{\beta_1 S_0 k_1}{\varepsilon_1 \varepsilon_2} + \frac{\beta_2 S_0 k_2}{\varepsilon_1 \varepsilon_3} - 1\right) = \varepsilon_1 E\left(\mathcal{R}_0 - 1\right). \end{aligned} \tag{3.6}$$

So, when $\mathcal{R}_0 < 1$, $\frac{\mathrm{d}L(t)}{\mathrm{d}t} \leq 0$, $\frac{\mathrm{d}L(t)}{\mathrm{d}t} = 0$ if and only if $E = I = M = 0$. Therefore, the $\{(S, E, I.M, Q, R) \in \Delta\}$ maximal invariant set is $\{H_0\}$. By the LaSalle's invariant set principle, the DFE H_0 is globally asymptotically stable if $\mathcal{R}_0 < 1$. This concludes the proof of the Theorem 3.4.

Theorem 3.5. *When $\mathcal{R}_0 > 1$, the endemic equilibrium H^* of the model (2.1) is locally asymptotically stable provided that the following conditions hold simultaneously*

$$A_1 > 0, A_2 > 0, A_1 A_2 > A_3, A_4 > 0, A_1 A_2 A_3 - A_1^2 A_4 > A_3^2.$$

Proof. The Jacobian matrix of model (2.1) at $H^* = (S^*, E^*, I^*, M^*, Q^*, R^*)$ is as follow

$$J(H^*) = \begin{pmatrix} -d - \beta_1 I^* - \beta_2 M^* & 0 & -\beta_1 S^* & -\beta_1 S^* & 0 & 0 \\ \beta_1 I^* + \beta_2 M^* & -\varepsilon_1 & \beta_1 S^* & \beta_2 S^* & 0 & 0 \\ 0 & k_1 & -\varepsilon_2 & 0 & 0 & 0 \\ 0 & k_2 & 0 & -\varepsilon_3 & 0 & 0 \\ 0 & 0 & \omega & \mu & -\varepsilon_4 & 0 \\ 0 & 0 & \gamma & \delta & \rho & -d \end{pmatrix}.$$

Using the determinant expansion of algebra to obtain the characteristic polynomial of the matrix, can get

$$\begin{aligned} g_1(\lambda) &= (\lambda + d)(\lambda + \varepsilon_4) \begin{pmatrix} \lambda + \beta_1 I^* + \beta_2 M^* + d & 0 & \beta_1 S^* & \beta_2 S^* \\ -\beta_1 I^* - \beta_2 M^* & \lambda + \varepsilon_1 & -\beta_1 S^* & -\beta_2 S^* \\ 0 & -k_1 & \lambda + \varepsilon_2 & 0 \\ 0 & -k_2 & 0 & \lambda + \varepsilon_3 \end{pmatrix} \\ &= (\lambda + d)(\lambda + \varepsilon_4) g_2(\lambda). \end{aligned}$$

The $g_2(\lambda)$ can be expressed as follows

$$g_2(\lambda) = (\lambda + \beta_1 I^* + \beta_2 M^* + d) \begin{pmatrix} \lambda+\varepsilon_1 & -\beta_1 S^* & -\beta_2 S^* \\ -k_1 & \lambda+\varepsilon_2 & 0 \\ -k_2 & 0 & \lambda+\varepsilon_3 \end{pmatrix} + (\beta_1 I^* + \beta_2 M^*) \begin{pmatrix} 0 & \beta_1 S^* & \beta_2 S^* \\ k_1 & \lambda+\varepsilon_2 & 0 \\ k_2 & 0 & \lambda+\varepsilon_3 \end{pmatrix}.$$

The corresponding characteristic polynomial equation of J_{H^*} is given by

$$g_2(\lambda) = \lambda^4 + A_1\lambda^3 + A_2\lambda^2 + A_3\lambda + A_4, \tag{3.7}$$

the four parameters in equation (3.7) are as follows:

$$\begin{aligned}
A_1 &= \beta_1 I^* + \beta_2 M^* + d + \varepsilon_1 + \varepsilon_2 + \varepsilon_3, \\
A_2 &= (\beta_1 I^* + \beta_2 M^* + d)(\varepsilon_1 + \varepsilon_2 + \varepsilon_3) + \varepsilon_1\varepsilon_2 + \varepsilon_2\varepsilon_3 + \varepsilon_1\varepsilon_3 - (k_1\beta_1 S^* + k_2\beta_2 S^*), \\
A_3 &= \varepsilon_1\varepsilon_2\varepsilon_3 - (\varepsilon_3 + d)\beta_1 k_1 S^* - (\varepsilon_2 + d)\beta_2 k_2 S^* + (d + \beta_1 I^* + \beta_2 M^*)(\varepsilon_1\varepsilon_2 + \varepsilon_2\varepsilon_3 + \varepsilon_1\varepsilon_3), \\
A_4 &= (\beta_1 I^* + \beta_2 M^*)\varepsilon_1\varepsilon_2\varepsilon_3 + d\Big(\varepsilon_1\varepsilon_2\varepsilon_3 - (\varepsilon_3\beta_1 k_1 S^* - \varepsilon_2\beta_2 k_2 S^*)\Big).
\end{aligned}$$

Hence, according to the Routh-Hurwitz criterion, the necessary and sufficient conditions for H^* to be locally asymptotically stable are $A_1 > 0, A_2 > 0, A_1A_2 > A_3, A_4 > 0$, and $A_1A_2A_3 - A_1^2A_4 > A_3^2$.

The above is an analysis of the local stability of the endemic equilibrium point, and the following content is a discussion of the global stability of the endemic equilibrium point. We consider the global asymptotically stability of the model (2.1) in the absence of equation Q, R, as it is independent on the rest of the equation of the model (2.1). We first give endemic equilibrium in the following at a steady-state at Δ_ϵ, for system (2.1)

$$\begin{cases}
\Lambda = (\beta_1 I^* + \beta_2 M^*)S^* + dS^*, \\
\varepsilon_1 E^* = (\beta_1 I^* + \beta_2 M^*)S^*, \\
\varepsilon_2 I^* = k_1 E^*, \\
\varepsilon_3 M^* = k_2 E^*.
\end{cases} \tag{3.8}$$

The expression shown in (3.8) will be used later in the proof of the globally asymptotically stable of endemic equilibrium point.

Theorem 3.6. *The endemic equilibrium H^* of the model (2.1) is globally asymptotically stable when $\mathcal{R}_0 > 1$.*

Proof. Constructing a suitable Lyapunov function $V : \mathbb{R}^4_+ \to \mathbb{R}$, defined as

$$\begin{aligned}
V(t) = {} & S - S^* - S^* \ln \frac{S}{S^*} + E - E^* - E^* \ln \frac{E}{E^*} + \frac{\beta_1 I^* S^*}{k_1 E^*}\Big(I - I^* - I^* \ln \frac{I}{I^*}\Big) \\
& + \frac{\beta_2 M^* S^*}{k_2 E^*}\Big(M - M^* - M^* \ln \frac{M}{M^*}\Big).
\end{aligned} \tag{3.9}$$

The time differentiation of equation (3.9) gives

$$V(t)' = \Big(1-\frac{S^*}{S}\Big)S' + \Big(1-\frac{E^*}{E}\Big)E' + \frac{\beta_1 I^* S^*}{k_1 E^*}\Big(1-\frac{I^*}{I}\Big)I' + \frac{\beta_2 M^* S^*}{k_2 E^*}\Big(1-\frac{M^*}{M}\Big)M'. \tag{3.10}$$

The expressions on the right hand side of equation (3.10) are obtained using the equations from the model (2.1) as follows

$$\begin{aligned}\Big(1-\frac{S^*}{S}\Big)S' &= \Big(1-\frac{S^*}{S}\Big)\Big(\Lambda - \Big(\beta_1 I + \beta_2 M\Big)S - dS\Big)\\ &= \Big(1-\frac{S^*}{S}\Big)\Big(\Big(\beta_1 I^* + \beta_2 M^*\Big)S^* + dS^* - \Big(\beta_1 I + \beta_2 M\Big)S - dS\Big)\\ &\le \beta_1 I^* S^*\Big(1 - \frac{S^*}{S} - \frac{SI}{S^*I^*} + \frac{I}{I^*}\Big) + \beta_2 M^* S^*\Big(1 - \frac{S^*}{S} - \frac{SM}{S^*M^*} + \frac{M}{M^*}\Big),\end{aligned} \tag{3.11}$$

$$\begin{aligned}\Big(1-\frac{E^*}{E}\Big)E' &= \Big(1-\frac{E^*}{E}\Big)\Big(\Big(\beta_1 I + \beta_2 M\Big)S - \varepsilon_1 E\Big)\\ &= \Big(1-\frac{E^*}{E}\Big)\Big(\Big(\beta_1 I + \beta_2 M\Big)S - \Big(\beta_1 I^* + \beta_2 M^*\Big)S^*\frac{E}{E^*}\Big)\\ &= \beta_1 I^* S^*\Big(1 - \frac{E}{E^*} + \frac{SI}{S^*I^*} - \frac{SIE^*}{S^*I^*E}\Big)\\ &\quad + \beta_2 M^* S^*\Big(1 - \frac{E}{E^*} + \frac{SM}{S^*M^*} - \frac{MSE^*}{M^*S^*E}\Big),\end{aligned} \tag{3.12}$$

$$\begin{aligned}\frac{\beta_1 I^* S^*}{k_1 E^*}\Big(1-\frac{I^*}{I}\Big)I' &= \frac{\beta_1 I^* S^*}{k_1 E^*}\Big(1-\frac{I^*}{I}\Big)\Big(k_1 E - \varepsilon_2 I\Big)\\ &= \frac{\beta_1 I^* S^*}{k_1 E^*}\Big(1-\frac{I^*}{I}\Big)\Big(k_1 E - \frac{k_1 E^* I}{I^*}\Big)\\ &= \beta_1 I^* S^*\Big(1 + \frac{E}{E^*} - \frac{I}{I^*} - \frac{I^*E}{IE^*}\Big),\end{aligned} \tag{3.13}$$

$$\begin{aligned}\frac{\beta_2 M^* S^*}{k_2 E^*}\Big(1-\frac{M^*}{M}\Big)M' &= \frac{\beta_2 M^* S^*}{k_2 E^*}\Big(1-\frac{M^*}{M}\Big)\Big(k_2 E - \varepsilon_3 M\Big)\\ &= \frac{\beta_2 M^* S^*}{k_2 E^*}\Big(1-\frac{M^*}{M}\Big)\Big(k_2 E - \frac{k_2 E^* M}{M^*}\Big)\\ &= \beta_2 M^* S^*\Big(1 + \frac{E}{E^*} - \frac{M}{M^*} - \frac{M^*E}{ME^*}\Big).\end{aligned} \tag{3.14}$$

Substituting equation (3.11) to (3.14) into equation (3.10) and after simplifications, we get

$$V' = \beta_1 I^* S^*\Big(3 - \frac{S^*}{S} - \frac{I^*E}{IE^*} - \frac{ISE^*}{I^*S^*E}\Big) + \beta_2 M^* S^*\Big(3 - \frac{S^*}{S} - \frac{M^*E}{ME^*} - \frac{MSE^*}{M^*S^*E}\Big). \tag{3.15}$$

Here, we using the properties of arithmetic geometric averages, we have

$$3 - \frac{S^*}{S} - \frac{I^*E}{IE^*} - \frac{ISE^*}{I^*S^*E} \le 0; \qquad 3 - \frac{S^*}{S} - \frac{M^*E}{ME^*} - \frac{MSE^*}{M^*S^*E} \le 0. \tag{3.16}$$

From equations (3.15) and (3.16), it is clear that, Lyapunov asymptotic stability theorem satisfied as $\frac{dL(t)}{dt} \leq 0$ is strictly a Lyapunov function which implies that, the endemic equilibrium point H^* is globally asymptotically stable contained in the region Δ_ϵ. From a biological perspective, this means that the disease is stable and will persist in the population for a long time.

Therefore, $\frac{dV}{dt}$ is a Lyapunov function converging in the positive region Δ such that $S \to S^*, E \to E^*, I \to I^*, M \to M^*, Q \to Q^*$ and $R \to R^*$ as $t \to \infty$. Hence, this concludes the proof of Theorem 3.6.

4 Sensitivity analysis

Considering the importance of the basic regeneration number in the transmission dynamics of infectious diseases. It is important to study how the parameters in the model affect $\mathcal{R}_0$. Sensitivity analysis was performed for $\mathcal{R}_0$ according to the method described in [16].

Definition 4.1. *The normalized forward sensitivity index of a variable $\mathcal{R}_0$ to a parameter σ is defined as*

$$\Upsilon_\sigma^{\mathcal{R}_0} := \frac{\partial \mathcal{R}_0}{\partial \sigma} \times \frac{\sigma}{\mathcal{R}_0}.$$

We summarize the computed sensitivity indices of $\mathcal{R}_0$ to several important model parameters in Table 1 and present them in Figure 1.

Table 1: Sensitivity indexes of $\mathcal{R}_0$

Parameter	Value	Sensitivity index	Source of the data
Λ	0.01138	1	[17]
β_1	0.2944	0.499493	[18]
β_2	0.45	0.500507	[19]
k_1	0.117	0.00582248	Assume
k_2	0.1	0.0785657	Assume
ω	0.2944	-0.365374	Assume
γ	$\frac{1}{15}$	-0.0827387	[20]
μ	0.45	-0.429224	Assume
δ	$\frac{1}{30}$	-0.0317943	[20]
ν	0.0214	-0.0469711	[21]
d	0.02	-1.12829	[22]

For example, we calculate $\Upsilon_{\beta_2}^{\mathcal{R}_0} = 0.500507$, it indicates if β_2 is increased by 10% then the basic reproduction number $\mathcal{R}_0$ also increased by 5.00507%. Further, $\Upsilon_\mu^{\mathcal{R}_0} = -0.429224$, signifies that 10% increment in μ will decrease $\mathcal{R}_0$

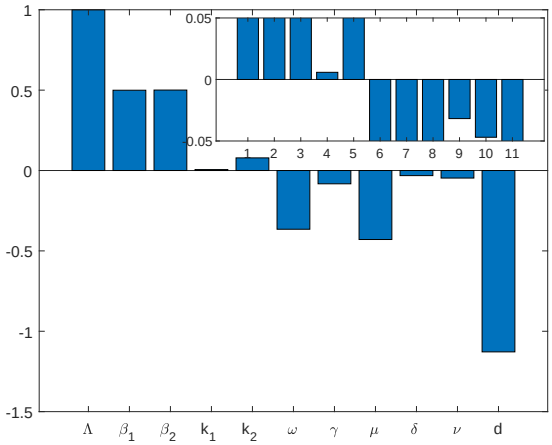

Figure 1: Sensitivity plot for the basic reproduction number $\mathcal{R}_0$

by 4.29224%. Table 1 also shows that $\Lambda, \beta_1, \beta_2, \mu$ and d have strong correlation with the basic reproduction number $\mathcal{R}_0$. In practice, control measures should therefore focus on reducing the disease transmission rate β_2.

5 Numerical simulations

5.1 The influence of the parameters contained in $\mathcal{R}_0$ on the spread of infectious diseases

According to the expression of the basic reproduction number, we analyze the change of $\mathcal{R}_0$ when some parameters change.

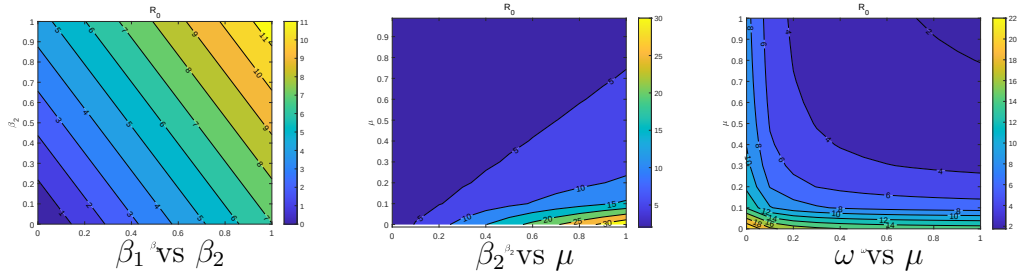

Figure 2: Contour plots of $\mathcal{R}_0$ affected by parameters β_1 vs β_2, β_2 vs μ, ω vs μ.

The contour plots of the basic reproduction number $\mathcal{R}_0$ with respect to parameters β_1 vs β_2, β_2 vs μ, ω vs μ are presented in Figure 2. As can be seen from Figure 2 that when other parameters related to $\mathcal{R}_0$ are fixed, $\mathcal{R}_0$ increases with the increase of β_1 and β_2. When μ increases or β_2 decreases, $\mathcal{R}_0$ decreases. μ and ω have similar effects on $\mathcal{R}_0$. This is consistent with the sensitivity analysis presented in Table 1.

5.2 Stability of the equilibrium point

Our system (2.1) possess a disease free equilibrium point $H_0(0.569, 0, 0, 0, 0, 0)$ for $\rho = 0.02$ and other parameters are as specified in Table 1. We find the corresponding eigenvalues of the jacobian matrix J_{H_0} as $-0.02, -0.02, -0.0614, -0.1087, -0.6145, -0.4409$. All the eigenvalues are negative and hence H_0 is locally asymptotically stable. In Figure 3, we plot the solution trajectories of system (2.1) with initial value (1.3951.0141.0391.0090.091.31) which converges to the

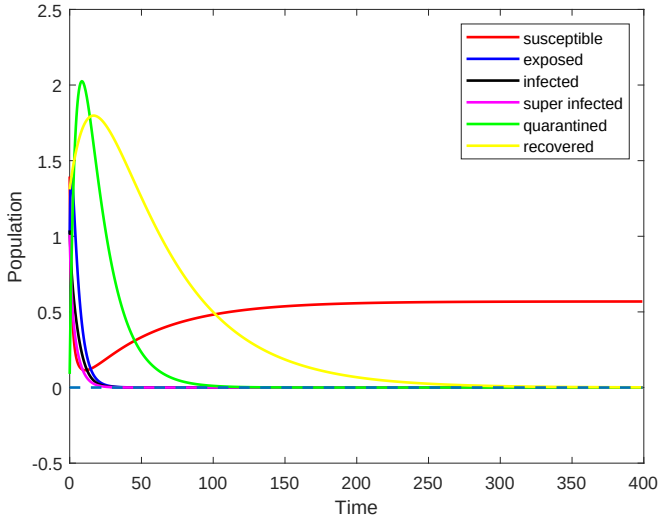

Figure 3: A numerical solution of model (2.1) with $\mathcal{R}_0 < 1$.

infection free steady state H_0. Further, we note that the corresponding basic reproduction number $\mathcal{R}_0 = 0.4114 < 1$. Therefore, disease can be eradicated for $\mathcal{R}_0 < 1$ but it depends on initial size of the population.

Further, we increase the recruitment rate of the susceptible Λ and set it as 0.1138, $\rho = 0.02$ and other parameters are as specified in Table 1. Then our system (2.1) possess two equilibria: (i) disease free equilibrium point $H_0(0.569, 0, 0, 0, 0, 0)$ and (ii) endemic equilibrium $H^*(1.3832, 0.3634, 0.1057, 0.0693, 1.0142, 1.4818)$. The corresponding basic reproduction number is calculated as $\mathcal{R}_0 = 4.1137 > 1$. The corresponding eigenvalues of the jacobian matrix J evaluated at H_0 and H^* are respectively 0.3301, -0.02, -0.02, -0.0614, -0.4537, -1.0406 and -0.02, -0.0614, -0.7206, -0.4533, $-0.0363 \pm 0.0607i$. Clearly, H_0 is unstable as one of the eigenvalues of J_{H_0} is positive. All the eigenvalues of J_{H^*} are negative or have negative real part. Hence, H^* is locally asymptotically stable. We plot the solution trajectories in Figure 4, with initial data (1.3951.0141.0391.0090.091.31). The trajectories converges to an endemic equilibrium point H^* Figure 5.

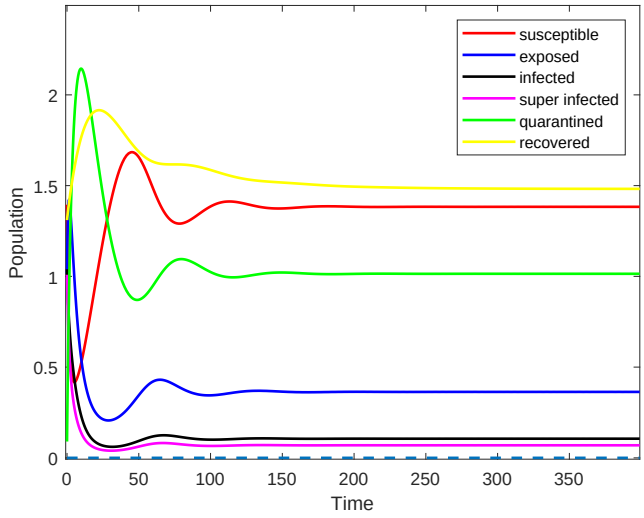

Figure 4: A numerical solution of model (2.1) with $\mathcal{R}_0 > 1$.

6 Discussion

In this article, we study an $SEIMQR$ infectious disease model affected by super-spreaders, which divides the total population into six compartments: susceptible $S(t)$, exposed $E(t)$, infected $I(t)$, super-spreaders $M(t)$, quarantine $Q(t)$ and recovered $R(t)$. The threshold quantity, known as the basic reproduction number $\mathcal{R}_0$, is obtained through the next-generation matrix method. Numerically, the sensitivity index of the associated parameters are in shown in Table 1, in order to determine the robustness of $\mathcal{R}_0$. In addition, the local asymptotic stability of the disease-free equilibrium point verified by the trace-determinant technique for $\mathcal{R}_0 < 1$; the local asymptotic stability of the endemic equilibrium point verified by the Routh-Hurwitz discriminant method for $\mathcal{R}_0 > 1$. The global asymptotic stability of equilibrium points is also discussed by constructing an appropriate Lyapunov function and combining them with Lasalle's invariant set principle. Finally, the conditions for the global stability of the equilibrium point are obtained.

We also performed sensitivity analysis on parameters related to $\mathcal{R}_0$ and we found that parameters $\Lambda, \beta_1, \beta_2, k_1$ and k_2 have positive effects on $\mathcal{R}_0$, while other parameters $\omega, \gamma, \nu, \mu, \delta, d$ all have negative effects on $\mathcal{R}_0$. Among all relevant parameters, $\Lambda, \beta_1, \beta_2, \mu$ and d have a strong correlation with the basic reproduction number $\mathcal{R}_0$. However, under actual control conditions, we cannot change the recruitment rate of the susceptible Λ and natural mortality rate d through external measures, which leads us to focus on reducing the disease transmission rates β_1, β_2 and increasing the isolation rate ω, μ.

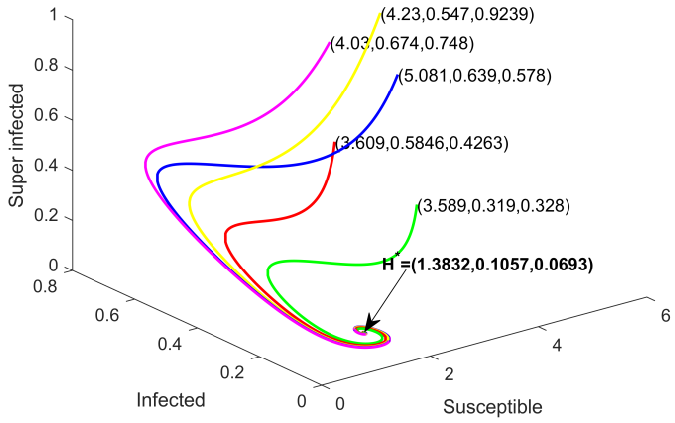

Figure 5: Numerical trajectories of model (2.1) showing that the endemic equilibrium H^* is globally asymptotically stable.

Competing interests. The authors declare that they have no competing interests.

Authors' contributions. JX and YW conceived of the studies, and drafted the manuscript. JZ participated in the discussion. All authors read and approved the final manuscript.

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