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Analysis of An SEI1I2QRV S Epidemic Infectious Disease Model with Multiple Infection Stages and Virus

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ABSTRACT

In this study, we propose an SEI1I2QRV Smodel for epidemic infec- tious diseases, which simulates the process of virus transmission. The model demonstrates how the virus impacts individuals who are infected. It is a well-established fact that the spread of infectious diseases can contribute to the proliferation of the virus within a susceptible population. One method of managing infectious diseases is to raise the virus-related fatality rate. In order to explain the virus's growth and decline rates in the susceptible pop- ulation, the suggested model will be examined. We investigate the dynamic behaviour inside the model's framework. It is shown that the model has two equilibrium points: a disease-free equilibrium (DFE) and an endemic equi- librium (EE). According to our results, the basic reproduction number, or

R0, has a major impact on the model's dynamics. When R0 < 1, the DFE is asymptotically stable both globally and locally under specific conditions.

On the other hand, if R0 > 1, the internal equilibrium is asymptotically stable both globally and locally. Finally, we evaluate our analytical results by numerical simulations utilizing biologically relevant parameter values.

Keywords: *Two phases of infection, Epidemic model, Quarantine, Basic reproduction number, Virus Class, Global stability, Local stability*

1. INTRODUCTION

Mathematical modeling has emerged as an invaluable tool in the fight against infectious diseases, offering profound insights into their spread and effective management. These models aid in forecasting the course of an illness over time and emphasise the critical factors that influence disease transmis- sion and recovery rates [1, 31, 32]. Assessing stable states and their stability is one of the primary difficulties in examining the behaviour of epidemic models [2, 26, 32].It is believed that no late interventions are taken into consideration when the population in each compartment shows no structure, such as age or geographic location [32]. In a conventional SIR (Suscepti- ble, Infectious, Recovered) model, the time evolution is defined by ordinary differential equations (ODEs) [3, 26, 33]. The disease incubation period is frequently assumed to be insignificant in the literature currently under pub- lication. In this case, each vulnerable person becomes infectious right away after contracting the infection and gradually recovers, gaining temporary im- munity [32]. These presumptions form the basis of the SIR model. But in the natural world, several illnesses (including measles, influenza, and tuber- culosis) need a susceptible individual to come into intimate contact with an infected people in order for them to become exposed and sick but not yet contagious. Before becoming contagious, this individual stays in the exposed group for a predetermined amount of time [33]. It can be remarkably short for an organism to go from a latent condition to infectious status [4, 5, 6, 7, 8, 9, 10].

Public health organisations are quite concerned about emerging trans-

missible illnesses because they have a huge financial impact on communities and have a devastating effect on public health [31]. Therefore, it is crucial to assess possible strategies for managing these illnesses [11, 12, 13]. The foot-and-mouth disease, 2009 swine influenza pandemic, and severe acute respiratory syndrome (SARS) are only some of the examples of the develop- ing and reemerging illnesses that isolation and quarantine have recently been successfully implemented to prevent the spread of in humans and animals [10, 11, 12, 21]. The SARS out- breaks 2003 serve as an important exam- ple of how quarantine and isolation can effectively control a novel disease [10, 11, 12, 21]. However, implement- ing these measures can lead to signif- icant psychological costs and socio-economic[31]. Removing people from the general

community who are suspected of being infected but do not exhibit any clinical signs is known as quarantine [31]. Such individuals can have

a viral infection without any symptoms or infected asymptomatically. In contrast, isolation refers to separating infected individuals who exhibit clin- ical symptoms of the disease. Infected people are placed in isolation to stop them from interacting with others and spreading the infection [10, 11, 14, 15, 16]. This strategy is mostly used to suppress abrupt illness outbreaks. A successful case of isolation was the manage- ment of SARS during 2003-2004. Nonetheless, this strategy has drawbacks, including the challenges in detect- ing infected individuals and the costs asso- ciated with isolation. Generally, achieving perfect isolation on a large scale is difficult, leading to incomplete isolation and the risk of nosocomial infections [17, 18, 19, 20, 22].

In this model, we have taken two infectious stages: the first infectious stage I1 and the second infection stage I2. In the first infectious stage, indi- viduals are actively affected with the pathogen and can transmit it to suscep- tible persons. They may exhibit symptoms or remain asymptomatic with varying degrees of infectiousness. Transmission dynamics, such as contact rates and the efficacy of preventative measures, significantly impact the in- fection's spread, within the stage. In the second infectious stage, individuals have recovered and developed immunity but may still carry the pathogen and transmit it to susceptible individuals. While their infectiousness is typically lower than that of those in the first infectious stage, they can still contribute to transmitting the disease. The duration of immunity and the potential for reinfection or waning immunity impact the dynamics of this stage [23, 24].

The virus class V in this model represents the presence and dynamics of the virus within the population and the environment. The virus class encompasses all instances of the virus within the population, including vi- ral particles shed by infected individuals and viral particles present in the environment. This class accounts for the infectiousness of the virus and its potential to cause new infections. Infected individuals shed viral particles into their surroundings through various means, such as bodily fluids, res- piratory droplets, or contaminated surfaces. The virus class captures the replication and shedding dynamics of the virus, contributing to the infection transmission within the population. Public health initiatives like immuni- sation drives, personal hygiene routines, and isolation protocols, can affect the dynamics of the virus class, and these interventions aim to reduce the transmission potential of the virus and lessen its influence on public health [25].

This paper introduces an intricate and comprehensive mathematical model

that captures the complexities of multiple infection stages and diverse virus

class. The primary objective of this research is to investigate the significant influence that an infectious disease's virus class can have on an entire neigh- bourhood. The following is an elegant arrangement of the paper's structure: The proposed model's formulation is explained in Section 2. We examine the presence and dynamic behaviours of endemic and disease-free equilibria on a local and global scale in Section 3. The insightful numerical simula- tions in Section 4 improve our model understanding. Finally, the concluding section engages in a thoughtful discussion of the results, illuminating their significance.

2. Model Formulation

Assumptions and Model description are the two subsections that further subdivide this section.

2.1. Assumptions

The following presumptions must be made in light of our biological back- ground:

1. The population is categorized into several mutually exclusive compart- ments, such as Susceptible(S), Exposed(E), First Infected Individuals(I1) and Second Infected Individuals(I2), Quarantine(Q), Virus(V), and Re- covered(R).

2. In each compartment population is devoid of any structure be it spatial age or location and does not take into account any delayed processes.

3. Population increases at a constant recruitment rate σ and experiences natural death at a rate $\mu 1$.

4. The population is engaging and blending seamlessly with one another, creating a vibrant and harmonious community.

5. Disease transmission occurs horizontally rather than vertically, and there is no population migration.

6. Recovered individuals develop disease temporary immunity acquired from the infection.

2.2. Model Description

A compelling epidemic model inspired by the saturation incidence of the virus is proposed in this section. Numerous researchers are fascinated by viruses' complex involvement in infectious illness transmission. We define

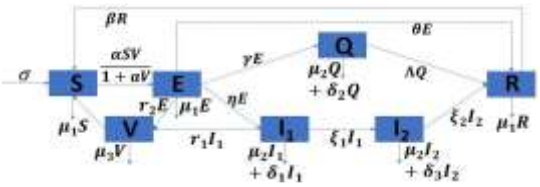

Figure 1: The suggested SEI_1I_2QRVS model system's schematic diagram

the non-linearity of the incidence as $\frac{\alpha S(t)V(t)}{\rho(V}$, where $\rho(0) = 1$ and $\rho'(V) \geq 0$. Effectively controlling infectious diseases requires that the growth rate of the virus is intricately linked to the levels of infection and exposure.

We present a sophisticated mathematical epidemic model, aptly named $SEI_1I_2QRV\,S$ and the accompanying schematic diagram of this model, ap- propriate for a homogeneous population, is illustrated in Figure 1, and nonlin- ear ordinary differential equations govern this dynamic system that capture the complexities of disease transmission and progression.

$$\begin{aligned}
\frac{dS}{dt} &= \sigma - \frac{\alpha SV}{1+\alpha V} - \mu_1 S + \beta R,\\
\frac{dE}{dt} &= \frac{\alpha SV}{1+\alpha V} - \gamma E - \eta E - \mu_1 E - \vartheta E,\\
\frac{dI_1}{dt} &= \eta E - \xi_1 I_1 - \mu_2 I_1 - \delta_1 I_1,\\
\frac{dI_2}{dt} &= \xi_1 I_1 - \mu_2 I_2 - \delta_3 I_2 - \xi_2 I_2,\\
\frac{dQ}{dt} &= \gamma E - \mu_2 Q - \delta_2 Q - \Lambda Q,\\
\frac{dR}{dt} &= \Lambda Q + \xi_2 I_2 + \vartheta E - \mu_1 R - \beta R,\\
\frac{dV}{dt} &= r_1 I_1 + r_2 E - \mu_3 V
\end{aligned} \tag{1}$$

with initial conditions:

$$S(0) = S_0 > 0,\ E(0) = E_0 > 0,\ I_1(0) = (I_1)_0 > 0,\ I_2(0) = (I_2)_0 > 0,$$
$$Q(0) = Q_0 > 0,\ R(0) = R_0 > 0,\ V(0) = V_0 > 0.$$

where $N = S + E + I_1 + I_2 + Q + V + R$, is the total population at time t. 1 summarises all of the parameter descriptions..

Table 1: Parameter description for the system 1

Parameter	Description (Unit)
σ	Recruitment rate of Susceptible (days)
α	Transmission coefficient of individuals exposed (days)
μ	Rate of natural death (days)
$\frac{1}{a}$	Half-saturation Constant of infected persons (days)
γ	Transition rate of exposed to quarantine individuals (days)
η	Transition rate of individuals exposed to first infection stage (days)
θ	Recovery rate of individuals exposed (days)
ξ_1	Infected class transition rate from first to second stage (days)
δ	Disease induced death rate (days)
ξ_2	Recovery rate from second infection stage (days)
Λ	Recovery rate of quarantine people (days)
β	Rate of transfer from recovered to vulnerable people (days)
r_1	Rate of virus birth from infected people (days)
r_2	Rate of virus birth from exposed people (days)

3. Model Analysis

In this section, we embark on a fascinating exploration of the dynamical behavior of the system outlined in equation 1. Here, we will evaluate all feasible steady states also determining the R_0 for this intriguing system.

1. Notably, the total population size N follows the compelling equation $\frac{dN}{dt} = \sigma - \mu N$, where $\mu = min(\mu_1, \mu_2, \mu_3)$ and thus $N(t) \to \frac{\sigma}{\mu}$ as, $t \to \infty$. Consequently, the biologically feasible region is not just preserved but positively invariant for the system 1. Therefore, we focus exclusively on solutions with initial conditions that lie within the captivating boundaries of the region Ω.

$$\Omega = \{(S, E, I_1, I_2, Q, V, R) : 0 \leq S, E, I_1, I_2, Q, R, N \leq \frac{\sigma}{\mu}\} \quad (2)$$

3.1. Basic Reproduction Number

The basic reproduction number, or R0, is the projected number of sec- ondary cases that a single typical infection would create in a population that is entirely susceptible [**26, 28**]. One of the most crucial threshold criteria for statistically describing the transmission of infectious diseases is this figure 1, which is also represented as the basic reproductive rate or ratio. Because it aids in predicting whether an infectious disease will spread throughout a population, R0 is very helpful. Similar to the method outlined in [**26, 28**]. We compute the R0

Given $x = (E, I_1, I_2, Q, V)$, then from model 1, it follows: $\frac{dx}{dt} = \mathrm{F} - \mathrm{V}$, where,

$$\mathrm{F} = \begin{bmatrix} \frac{\alpha SV}{1+\alpha V} \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix} \text{ and } \mathrm{V} = \begin{bmatrix} (-\gamma - \eta - \mu_1 - \vartheta)E \\ \eta E - \xi_1 I_1 - \mu_2 I_1 - \delta_1 I_1 \\ \xi_1 I_1 - \mu_2 I_2 - \delta_3 I_2 - \xi_2 I_2 \\ \gamma E - \mu_2 Q - \delta_2 Q - \Lambda Q \\ r_1 I_1 + r_2 E - \mu_3 V \end{bmatrix}.$$

We get

$$F = \begin{bmatrix} 0 & 0 & 0 & 0 & \alpha S^0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

$$\mathrm{V} = \begin{bmatrix} -(\gamma + \eta + \mu_1 + \vartheta) & 0 & 0 & 0 & 0 \\ \eta & -(\xi_1 + \mu_2 + \delta_1) & 0 & 0 & 0 \\ 0 & \xi_1 & -(\mu_2 + \delta_3 + \xi_2) & 0 & 0 \\ \gamma & 0 & 0 & -(\mu_2 + \delta_2 + \Lambda) & 0 \\ r_2 & r_1 & 0 & 0 & -\mu_3 \end{bmatrix}$$

where F = jacobian of F at DFE and V = jacobian of V at DFE.
Thus, the model's next generation matrix is

$$K = \mathrm{FV}^{-1} = \begin{bmatrix} \frac{\alpha S^0[\eta r_1 + r_2(\xi_1 + \mu_2 + \delta_1)]}{(\gamma+\eta+\vartheta+\mu_1)(\xi_1+\mu_2+\delta_1)\mu_3} & \frac{\alpha S^0 r_1}{(\xi_1+\mu_2+\delta_1)\mu_3} & 0 & 0 & \frac{\alpha S^0}{\mu_3} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

For the next generation matrix $K = \mathrm{FV}^{-1}$, the spectral radius $R_0 = \rho(\mathrm{FV}^{-1})$, hence

$$R_0 = \frac{\alpha\sigma[\eta r_1 + r_2(\xi_1 + \mu_2 + \delta_1)]}{\mu_1 \mu_3 (\gamma + \eta + \vartheta + \mu_1)(\xi_1 + \mu_2 + \delta_1)}. \quad (3)$$

3.2. Existence of Endemic Equilibrium

Furthermore, system 1 contains an internal equilibrium known as endemic equilibrium (EE), which is provided by

$$\bar{E} = (S^*, E^*, I_1^*, I_2^*, Q^*, R^*, V^*)$$

where,

$$E^* = \frac{\alpha\sigma(1-\frac{1}{R_0})}{\alpha\mu_1(\gamma+\eta+\mu_1+\vartheta)+\alpha(\gamma+\eta+\mu_1+\vartheta)-\frac{\alpha\beta}{\mu_1+\beta}\{\frac{\Lambda\gamma}{\Lambda+\mu_2+\delta_2}+\frac{\eta\xi_1\xi_2}{(\xi_1+\mu_2+\delta_1)(\xi_2+\mu_2+\delta_3)}+\vartheta\}}, \quad (4)$$

$$S^* = \frac{1}{\mu_1}[\sigma-(\gamma+\eta+\mu_1+\vartheta)E^*+\frac{\beta E^*}{\mu_1+\beta}\{\frac{\Lambda\gamma}{\Lambda+\mu_2+\delta_2}+\frac{\eta\xi_1\xi_2}{(\xi_1+\mu_2+\delta_1)(\xi_2+\mu_2+\delta_3)}+\vartheta\}], \quad (5)$$

$$I_1^* = \frac{\eta}{\xi_1+\mu_2+\delta_1}E^*, \quad (6)$$

$$I_2^* = \frac{\eta\xi_1}{(\xi_1+\mu_2+\delta_1)(\xi_2+\mu_2+\delta_3)}E^*, \quad (7)$$

$$Q^* = \frac{\gamma}{\Lambda+\mu_2+\delta_2}E^*, \quad (8)$$

$$R^* = \frac{E^*}{\mu_1+\beta}[\frac{\Lambda\gamma}{\Lambda+\mu_2+\delta_2}+\frac{\eta\xi_1\xi_2}{(\xi_1+\mu_2+\delta_1)(\xi_2+\mu_2+\delta_3)}+\vartheta], \quad (9)$$

$$V^* = \frac{E^*}{\mu_3}[\frac{\eta r_1}{\xi_1+\mu_2+\delta_1}+r_2]. \quad (10)$$

3.3. Local Stability of disease-free and endemic equilibrium

Considering $\frac{dN}{dt} = \sigma - \mu N$ is satisfied by the entire size of population, $N(t)$ Therefore, as $t \to \infty$, $N(t) \to \frac{\sigma}{\mu}$ By taking into account the restricting system 1, where the entire population is taken to be constant, we can get analytical results $N = N^0 = \frac{\sigma}{\mu}$.

Following that, the restricting dynamical systems that has been reduced is provided by

$$\frac{dS}{dt} = \sigma\left(1+\frac{\beta}{\mu_1}\right) - (\mu_1+\beta)S - \frac{\alpha SV}{1+aV} - \beta E - \beta I_2 - \beta Q, \quad (11)$$

$$\frac{dE}{dt} = \frac{\alpha SV}{1+aV} - \gamma E - \eta E - \mu_1 E - \vartheta E, \quad (12)$$

$$\frac{dI_1}{dt} = \eta E - \xi_1 I_1 - \mu_2 I_1 - \delta_1 I_1, \quad (13)$$

$$\frac{dI_2}{dt} = \xi_1 I_1 - \mu_2 I_2 - \delta_3 I_2 - \xi_2 I_2, \quad (14)$$

$$\frac{dQ}{dt} = \gamma E - \mu_2 Q - \delta_2 Q - \Lambda Q, \quad (15)$$

$$\frac{dV}{dt} = r_1 I_1 + r_2 E - \mu_3 V. \quad (16)$$

with initial conditions:

$$S(0) = S_0 > 0, E(0) = E_0 > 0, I_1(0) = (I_1)_0 > 0, I_2(0) = (I_2)_0 > 0, Q(0) = Q_0 > 0, V(0) = V_0 > 0. \quad (17)$$

For both the equilibria the local stability is determined follows:

Theorem 1 : The disease-free equilibrium (DFE) E^0 is

1. if $R_0 < 1$, locally asymptotically stable,

2. if $R_0 > 1$, unstable.

Proof. The variational matrix at DFE point is given by

$$J_0 = \begin{bmatrix} -(\mu_1+\beta) & -\beta & 0 & -\beta & -\beta & \frac{-\alpha\sigma}{\mu_1} \\ 0 & -(\gamma+\eta+\mu_1+\vartheta) & 0 & 0 & 0 & \frac{\alpha\sigma}{\mu_1} \\ 0 & \eta & -(\xi_1+\mu_2+\delta_1) & 0 & 0 & 0 \\ 0 & 0 & \xi_1 & -(\mu_2+\delta_3+\xi_2) & 0 & 0 \\ 0 & \gamma & 0 & 0 & -(\mu_2+\delta_2+\Lambda) & 0 \\ 0 & r_2 & r_1 & 0 & 0 & -\mu_3 \end{bmatrix}$$

The characteristic equation of J_0 is given by,

$$J_0 = -(\mu_1+\beta+\lambda)(\mu_2+\delta_3+\xi_2+\lambda)(\mu_2+\delta_2+\Lambda+\lambda)$$

The characteristic equation of J_0 is given by,

$$J_0 = -(\mu_1+\beta+\lambda)(\mu_2+\delta_3+\xi_2+\lambda)(\mu_2+\delta_2+\Lambda+\lambda)$$

$$-(\gamma+\eta+\mu_1+\vartheta+\lambda)(\xi_1+\mu_2+\delta_1+\lambda)(\mu_3+\lambda)+\frac{\alpha\sigma}{\mu_1}\{\eta r_1+r_2(\xi_1+\mu_2+\delta_1+\lambda)\} \quad (18)$$

Two roots are readily obtained from this: $\lambda = -\mu_1-\beta$, $\lambda = -\mu_2-\delta_3-\xi_2$ and $\lambda = -\mu_2-\delta_2-\Lambda$

Applying the Routh-Hurwtiz Criterion is necessary for the remaining three roots of the characteristics equations [**ref17**],

$A_1A_2 - A_3 > 0$,

where, $A_1 > 0$, $A_2 > 0$, $A_3 > 0$ and $A_1A_2 > A_3$.

Now,

$$\lambda^3+\lambda^2A_1+\lambda A_2+A_3 = 0$$

where,

$A_1 = \gamma+\delta_1+\vartheta+\mu_1+\mu_2+\mu_[3]+\xi_1+\eta$,

$A_2 = \gamma\delta_1+\eta\delta_1+\vartheta\delta_1-\frac{r_2\alpha\sigma}{\mu_1}+\mu_1\delta_1+\gamma\mu_2+\eta\mu_2+\vartheta\mu_2+\mu_1\mu_2+\gamma\mu_3+\delta_1\mu_3+\eta\mu_3+\vartheta\mu_3+\mu_1\mu_3+\mu_2\mu_3+\gamma\xi_1+\eta\xi_1+\vartheta\xi_1+\xi_1\mu_1+\xi_1\mu_3$

and

$A_3 = -\frac{r_2\alpha\sigma\alpha_1}{\mu_1}-\frac{r_1\alpha\sigma\eta}{\mu_1}-\frac{r_2\alpha\sigma\mu_2}{\mu_1}+\gamma\delta_1\mu_3+\delta_1\eta\mu_3+\delta_1\vartheta\mu_3+\delta_1\mu_1\mu_3+\gamma\mu_2\mu_3+\eta\mu_2\mu_3+\vartheta\mu_2\mu_3+\mu_1\mu_2\mu_3-\frac{r_2\alpha\sigma\xi_1}{\mu_1}+\gamma\xi_1\mu_3+\vartheta\xi_1\mu_3+\xi_1\mu_1\mu_3$

It is evident that $A_1 > 0$, $A_2 > 0$, $A_3 > 0$ and $(A_1A_2 - A_3) > 0$. Therefore, all six of $\mathbf{J_0}$ eigenvalues have negative real portions if $R_0 < 1$. On the other hand, one eigenvalue has a positive real portion if $R_0 > 1$, but five eigenvalues of $\mathbf{J_0}$ have negative real parts. Consequently, when $R_0 < 1$ the DFE is locally asymptotically stable; when $R_0 > 1$, the DFE is unstable.

Theorem 2 : If $R_0 > 1$, then the EE of point $\bar{E}$ exists and is locally asymptotically stable.

Proof. We employ Lyapunov's Direct Method of Stability to assess system 3.3 EE global stability. Examine a positive definite function:

$$U_1 = \frac{1}{2}(D_1S^2+D_2E^2+D_3I_1^2+D_4I_2^2+D_5Q^2+D_6V^2), \quad (19)$$

Then using the system 3.3 in $\frac{dU_1}{dt}$, we get,

$$\frac{dU_1}{dt} = (D_1S)\sigma\left(1+\frac{\beta}{\mu_1}\right)-(\mu_1+\beta_1-\frac{\alpha SV}{1+aV}-\beta_1E+\gamma_1+I_2+Q)$$

$$+(D_2E)\left(\frac{\alpha SV}{1+aV}-\gamma E-\eta E-\mu_1E-\vartheta E\right)+(D_3I_1)(\eta E-\xi_1I_1-\mu_2I_1-\delta_1I_1)$$

$$(D_4I_2)(\xi_1I_1-\mu_2I_2-\delta_3I_2-\xi_2I_2)+(D_5Q)(\gamma E-\mu_2Q-\delta_2Q-\Lambda Q)$$

$$(D_6V)(r_1I_1+r_2E-\mu_3V), \quad (20)$$

$$\frac{dU_1}{dt} = D_1\left(\sigma S + \frac{\upsilon\sigma\varsigma}{\mu_1} - (\mu_1 + \beta)S^2 - \beta S(S + E + I_1 + I_2 + Q)\right.$$
$$+D_2(-\gamma E^2 - \eta E^2 - \mu_1 E^2 - \vartheta E^2) + D_3(\eta E I_1 - \xi_1 I^2 - \mu_2 I^2 - \delta_1 I^2)$$
$$+D_4(\xi_1 I_1 I_2 - \mu_2 I^2_2 - \delta_3 I^2_2 - \xi_2 I^2_2) + D_5(\gamma EQ - \mu_2 Q^2_1 - \delta_2 Q^2_1 - \Lambda Q^2_1)$$
$$\left. D_6(r_1 I_1 V + r_2 EV - \mu_3 V^2),\right. \quad (21)$$

$$\frac{dU_1}{dt} = \frac{a_{11}S^2}{3} - a_{12}SE + \frac{a_{22}E^2}{3} + \frac{a_{11}S^2}{3} - a_{13}SI_1 + \frac{a_{33}I_1^2}{3}$$
$$+ \frac{a_{11}S^2}{3} - a_{14}SI_2 + \frac{a_{44}I_2^2}{3} + \frac{a_{11}S^2}{3} - a_{15}SQ + \frac{a_{55}Q^2}{3}$$
$$+ \frac{a_{22}E^2}{3} - a_{23}EI_1 + \frac{a_{33}I^2}{3} + \frac{a_{33}I_1^2}{3} - a_{34}I_1I_2 + \frac{a_{44}I_2^2}{3}$$
$$+ \frac{a_{22}E^2}{3} - a_{25}EQ + \frac{a_{55}Q^2}{3} + \frac{a_{22}E^2}{3} - a_{26}EV + \frac{a_{66}V^2}{3}$$
$$+ \frac{a_{33}I_1^2}{3} - a_{36}I_1V + \frac{a_{66}V^2}{3}. \quad (22)$$

where,

$a_{11} = D_1(\mu_1 + \beta)$, $a_{22} = D_2(\gamma + \eta + \mu_1 + \vartheta)$, $a_{33} = D_3(\xi_1 + \mu_2 + \delta_1)$,

$a_{44} = D_4(\mu_2 + \delta_3 + \xi_2)$, $a_{55} = D_5(\mu_2 + \delta_2 + \Lambda)$, $a_{66} = \mu_3 D_6$, $a_{12} = \beta D_1$, $a_{13} = \beta D_1$, $a_{14} = \beta D_1$,

$a_{15} = \beta D_1$, $a_{23} = \eta D_3$, $a_{34} = \xi_1 D_4$, $a_{25} = \gamma D_5$, $a_{26} = r_2 D_6$, $a_{36} = r_1 D_6$

By applying Sylvester's criteria, it is apparent that $\frac{dU_1}{dt}$ is negative definite in the following circumstances:

1. $\frac{D_1\beta^2}{4} < \frac{D_2(\gamma+\eta+\mu_1+\vartheta)(\mu_1+\beta)}{9}$
2. $\frac{D_1\beta^2}{4} < \frac{D_4(\mu_2+\beta)(\mu_2+\delta_3+\xi_2)}{9}$
3. $\frac{D_1\beta^2}{4} < \frac{D_5(\mu_2+\beta)(\mu_2+\delta_2+\Lambda)}{9}$
4. $\frac{D_2\eta^2}{D_4\xi^2} < \frac{D_2(\gamma+\eta+\mu_1+\vartheta)(\mu_2+\delta_1+\xi_2)}{9}$
5. $\frac{1}{4} < \frac{\mu_3\mu_2+\mu_3\delta_3+\mu_3\xi_2+\mu_2\delta_1+\delta_3\delta_1+\xi_2\delta_1}{9}$
6. $\frac{D_5\gamma^2}{D_6r^2_2} < \frac{D_2(\gamma+\eta+\mu_1+\vartheta)(\mu_2+\delta_2+\Lambda)}{9}$
7. $\frac{2}{4} < \frac{\mu_3(\gamma+\eta+\mu_1+\vartheta)\mu_3}{9}$
8. $\frac{D_6r^2_1}{4} < \frac{D_2(\mu_2+\delta_2+\xi_2)\mu_3}{9}$

After choosing $D_1 = D_2 = 1$, then we have the final condition required for linear stability is:

$$\frac{\beta^2}{4} < \frac{(\gamma + \eta + \mu_1 + \vartheta)(\mu_1 + \beta)}{9} \quad (23)$$

It is evident that the EE point is only linearly asymptotically stable when the aforementioned requirement is met by Lyapunov's direct technique.

3.4. Global Stability of DFE and EE

The global stability of the EE and DFE stable states is examined in this section. First, we focus on the global stability of the DFE point. Castillo-Chavez et al. [**12**, **29**]. devised the method that we implement. Two conditions ensuring global disease-free state stability are listed below. As a result, we will additionally update model system 3.3.

$$\begin{aligned} \frac{dX}{dt} &= F(X, Z), \\ \frac{dZ}{dt} &= G(X, Z), G(X, 0) = 0 \end{aligned} \tag{24}$$

where $Z = (E, I_1, I_2, Q, V)$ and $X = (S)$. Let $Q_0 = (X^0, 0)$ represents the DFE. To provide local asymptotic stability, the subsequent requirements (H1) and (H2) must be satisfied:

1. H1 For $\frac{dX}{dt} = F(X, 0)$, X^0 is globally asymptotically stable,
2. H2 $G(X, Z) = BZ - \hat{G}(X, Z)$, where $\hat{G}(X, Z) \geq 0$, $\forall (X, Z) \in \Omega$,

where Ω is the region where the model makes biological sense and $B = D_z G(X^0, 0)$ is an M-matrix. Then the lemma that follows is true:

Lemma 1 : If the criteria (H1) and (H2) are met and $R_0 < 1$, the fixed point $Q_0 = (X^0, 0)$ is a globally stable asymptotic equilibrium of 3.4.

We assert the subsequent theorem:

Theorem 3 : assume that $R_0 < 1$. E_0, the DFE, is globally asymptotically stable.

Proof. Let $X = (S)$ and $Z = (E, I_1, I_2, Q, V)$, and

$$Q_0 = (X^0, 0), \text{ where } X^0 = \frac{\Lambda}{\mu_1}. \tag{25}$$

Then,

$$\frac{dX}{dt} = F(X, Z) = \sigma\left(1 + \frac{\beta}{\mu_1}\right) - (\mu_1 + \beta)S - \frac{\alpha SV}{1 + \sigma V} - \beta(E + I_1 + I_2 + Q)$$

At $S = S^0$, $G(X, 0) = 0$ and $\frac{dX}{dt} = G(X, 0) = \sigma\left(1 + \frac{\beta}{\mu_1}\right) - (\mu_1 + \beta)X$.

As $X \to X^0$, $t \to \infty$. Therefore, $X = X^0 (= S^0)$ is g.a.s.

Now,

$$G(X, Z) = \begin{bmatrix} -(\gamma + \eta + \mu_1 + \vartheta) & 0 & 0 & 0 & \alpha S^0 \\ \eta & -(\xi_1 + \mu_2 + \delta_1) & 0 & 0 & 0 \\ 0 & \xi_1 & -(\mu_2 + \delta_3 + \xi_2) & 0 & 0 \\ \gamma & 0 & 0 & -(\mu_2 + \delta_2 + \Lambda) & 0 \\ r_2 & r_1 & 0 & 0 & -\mu_3 \end{bmatrix} \begin{bmatrix} E \\ I_1 \\ I_2 \\ Q \\ V \end{bmatrix} - \begin{bmatrix} \alpha S^0 V - \frac{\alpha SV}{1+\sigma V} \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}$$

$G(X, Z) = BZ - \hat{G}(X, Z)$,
where,

$$B = \begin{bmatrix} -(\gamma+\eta+\mu_1+\vartheta) & 0 & 0 & 0 & \alpha S^0 \\ \eta & -(\xi_1+\mu_2+\delta_1) & 0 & 0 & 0 \\ 0 & \xi_1 & -(\mu_2+\delta_3+\xi_2) & 0 & 0 \\ \gamma & 0 & 0 & -(\mu_2+\delta_2+\Lambda) & 0 \\ r_2 & r_1 & 0 & 0 & -\mu_3 \end{bmatrix}$$

and

$$\hat{G}(X, Z) = \begin{bmatrix} \alpha S^0 V - \frac{\alpha SV}{1+\sigma V} \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}$$

The entire population in the streamlined dynamical system 3.3 is enclosed by N^0 i.e., $S, E, I_1, I_2, Q, V \leq N^0$. Since $S^0 \geq N^0 \geq S \geq \frac{S}{1+\sigma V}$ and consequently $\hat{G}(X, Z) \geq 0$. Clearly, B is an M-matrix, the previously mentioned requirements (H1) and (H2) are satisfied, and the DFE is thus established by the aforementioned lemma. If $R_0 < 1$, then E_0 is globally asymptotically stable. □

Theorem 4 : The global asymptotic stability of the endemic equilibrium $\bar{E}$ is considered if $R_0 > 1$.

Proof. We apply Lyapunov's Direct Method of Stability to evaluate the global stability of the EE of system 3.3. Such that

$$L_1 = \frac{1}{2}(D_1 S^2 + D_2 E^2 + D_3 I_1^2 + D_4 I_2^2 + D_5 Q^2 + D_6 V^2), \quad (26)$$

consequently the system 3.3 in $\frac{dV_1}{dt}$, we get,

$$\begin{aligned} \frac{dV_1}{dt} &= (D_1 S)\left(\sigma\left(1+\frac{\beta}{\mu_1}\right) - (\mu_1 + \vartheta\beta - \frac{\alpha SV}{1+\sigma V} - \vartheta(E+I_1+I_2+Q)\right) \\ &+(D_2 E)\left(\frac{\alpha SV}{1+\sigma V} - \gamma E - \eta E - \mu_1 E - \vartheta E\right) + (D_3 I_1)(\eta E - \xi_1 I_1 - \mu_2 I_1 - \delta_1 I_1) \\ &(D_4 I_2)(\xi_1 I_1 - \mu_2 I_2 - \delta_3 I_2 - \xi_2 I_2) + (D_5 Q)(\gamma E - \mu_2 Q - \delta_2 Q - \Lambda Q) \\ &(D_6 V)(r_1 I_1 + r_2 E - \mu_3 V), \end{aligned} \quad (27)$$

$$\begin{aligned} \frac{dV_1}{dt} &= D_1\left(\sigma S + \frac{\alpha\beta S}{\mu_1} - (\mu_1+\beta)S^2 - \beta S(S+E+I_1+I_2+Q)\right) \\ &+D_2(-\gamma E^2 - \eta E^2 - \mu_1 E^2 - \vartheta E^2) + D_3(\eta E I_1 - \xi_1 I_1^2 - \mu_2 I_1^2 - \delta_1 I_1^2) \\ &+D_4(\xi_1 I_1 I_2 - \mu_2 I_2^2 - \delta_3 I_2^2 - \xi_2 I_2^2) + D_5(\gamma EQ - \mu_2 Q^2 - \delta_2 Q^2 - \Lambda Q^2) \\ &D_6(r_1 I_1 V + r_2 EV - \mu_3 V^2), \end{aligned} \quad (28)$$

Using region Ω in [29] and the inequality $\pm 2ab \leq (a^2+b^2)$, we now obtain:

$$\begin{aligned} \frac{dV_1}{dt} \leq\ & \frac{b_{11}S^2}{3} - b_{12}SE + \frac{b_{22}E^2}{4} + \frac{b_{11}S^2}{3} - b_{13}SI_1 + \frac{b_{33}I_1^2}{3} \\ &+ \frac{b_{11}S^2}{3} - b_{14}SI_2 + \frac{b_{44}I_2^2}{3} + \frac{b_{11}S^2}{3} - b_{15}SQ + \frac{b_{55}Q^2}{3} \\ &+ \frac{b_{22}E^2}{4} - b_{23}EI_1 + \frac{b_{33}I_1^2}{3} + \frac{b_{33}I_1^2}{3} - b_{34}I_1I_2 + \frac{b_{44}I_2^2}{3} \\ &+ \frac{b_{22}E^2}{4} - b_{25}EQ + \frac{b_{55}Q^2}{3} + \frac{b_{22}E^2}{4} - b_{26}EV + \frac{b_{66}V^2}{2} \\ &+ \frac{b_{33}I_1^2}{3} - b_{36}I_1V + \frac{b_{66}V^2}{2}. \end{aligned} \quad (29)$$

wnere,

$b_{11} = D_1\ \mu_1 + \beta - \frac{\alpha\sigma}{2\mu_1}$, $b_{22} = D_2\ \gamma + \eta + \mu_1 + \vartheta - \frac{\alpha\sigma}{2\mu_1}$, $b_{33} = D_3(\xi_1 + \mu_2 + \delta_1)$,

$b_{44} = D_4(\mu_2 + \delta_3 + \xi_2)$, $b_{55} = D_5(\mu_2 + \delta_2 + \Lambda)$, $b_{66} = \mu_3 D_6$, $b_{12} = \beta D_1$, $b_{13} = \beta D_1$, $b_{14} = \beta D_1$,

$b_{15} = \beta D_1$, $b_{23} = \eta D_3$, $b_{34} = \xi_1 D_4$, $b_{25} = \gamma D_5$, $b_{26} = r_2 D_6$, $b_{36} = r_1 D_6$

Therefore, using Lyapunov's direct technique of stability, we determine that if the following criteria are met, the EE is either non-linearly or globally stable:

1. $D_2\ \mu_1 + \beta - \frac{\alpha\sigma}{2\mu_1}\ \gamma + \eta + \mu_1 + \vartheta - \frac{\alpha\sigma}{2\mu_1} > \beta^2 D_1$
2. $D_3\ \mu_1 + \beta - \frac{\alpha\sigma}{2\mu_1}\ (\xi_1 + \mu_2 + \delta_1) > \beta^2 D_1$
3. $D_4\ \mu_1 + \beta - \frac{\alpha\sigma}{2\mu_1}\ (\mu_2 + \delta_3 + \xi_2) > \beta^2 D_1$
4. $D_5\ \mu_1 + \beta - \frac{\alpha\sigma}{2\mu_1}\ (\mu_2 + \delta_2 + \Lambda) > \beta^2 D_1$

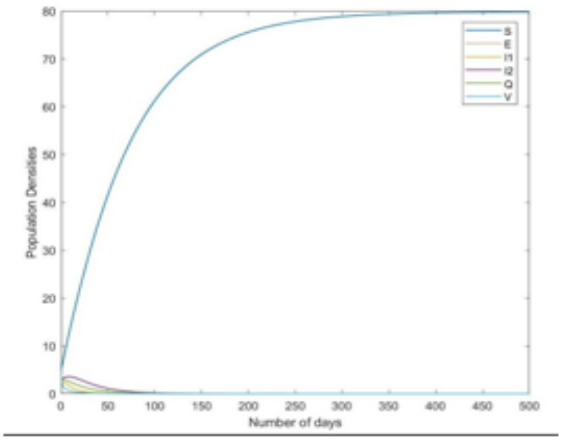

Figure 2: Densities of Population at virus rate $r_2 = 0.001$

5. $D_2\ \gamma + \eta + \mu_1 + \vartheta - \frac{\alpha\sigma}{2\mu_1}\ (\xi_1 + \mu_2 + \delta_1) > \eta^2 D_3$
6. $D_3(\xi_1 + \mu_2 + \delta_1)(\mu_2 + \delta_3 + \xi_2) > \xi_1^2 D_4$
7. $D_2\ \gamma + \eta + \mu_1 + \vartheta - \frac{\alpha\sigma}{2\mu_1}\ (\mu_2 + \delta_2 + \Lambda) > \gamma^2 D_5$
8. $D_2\ \gamma + \eta + \mu_1 + \vartheta - \frac{\alpha\sigma}{2\mu_1}\ \mu_3 > r_2^2 D_6$
9. $D_3(\xi_1 + \mu_2 + \delta_1)\mu_3 > r_1^2 D_6$

Lastly, the following requirements must be met for the EE to be globally stable:

1. $\mu_1 + \beta - \frac{\alpha\sigma}{2\mu_1} > 0$
2. $\gamma + \eta + \mu_1 + \vartheta - \frac{\alpha\sigma}{2\mu_1} > 0$
3. $\mu_1 + \beta - \frac{\alpha\sigma}{2\mu_1}\ \gamma + \eta + \mu_1 + \vartheta - \frac{\alpha\sigma}{2\mu_1} > \beta^2$

We demonstrate that the EE point $\bar{E}$ is globally stable under the specified parameters. □

4. Numerical Simulations

In order to demonstrate previously established conclusions, we present numerical simulations in this part using the feasible parametric settings with

Table 2: Simulation parameters system 1

Parameter	Value
σ	0.4
α	0.008
γ	0.1
η	0.1
Λ	0.04
ξ_1	0.03 - 0.1 (variable)
ξ_2	0.03
θ	0.001
β	0.01
μ_1	0.005
μ_2	0.008
μ_3	0.8
δ_1	0.01
δ_2	0.01
δ_3	0.01
r_1	0.3
r_2	0.001 - 0.1 (variable)
a	0.1

time units in days, as indicated in Table 2. Most parameter values are taken from the literature [**12**, **30**] but the remaining values for parameters are taken into consideration for numerical computation. To validate the analytical conclusions of the preceding sections, numerical simulations are carried out using Matlab ODE solver. Here, Figure 2 - Figure 3 show how the system responds for various virus growth rate values.

1. We determine the $R_0 = 0.991213 < 1$ with a viral growth rate of $r_2 = 0.001$. The DFE, $E^0 = (80, 0, 0, 0, 0, 0)$ is globally asymptotically stable according to Theorems $1 - 3$. Refer to Figure 2.
2. For virus growth rate $r_2 = 0.1$ we calculate $R_0 = 2.81553 > 1$. From Theorem 2 the EE $\bar{E} = (35.4078, 2.71614, 5.65862, 3.53664, 4.683, 2.4615)$. Also the conditions for global stability $\mu_1 + \beta - \frac{\alpha\sigma}{2\mu_1} > 0$, $\gamma + \eta + \mu_1 + \vartheta - \frac{\alpha\sigma}{2\mu_1} > 0$ and

$\mu_1 + \beta - \frac{\alpha\sigma}{2\mu_1}$ $\gamma + \eta + \mu_1 + \vartheta - \frac{\alpha\sigma}{2\mu_1} > \beta^2$ holds and therefore, the conditions of Theorem 4 are verified. Thus the EE $\bar{E}$ is globally asymptotically stable.(see Figure 3)

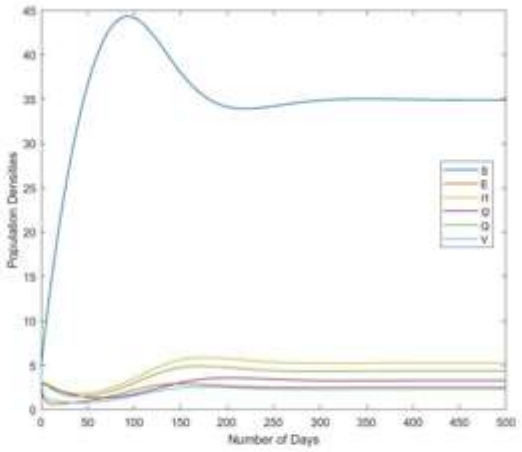

Figure 3: Densities of Population at virus rate $r_2 = 0.1$

5. Results and Discussion

This paper proposes and evaluates a mathematical model for the spread of contagious diseases that includes multiple infection stages and virus clas- sifications. For the sake of mathematical simplicity, let us consider the popu- lation exists in a homogeneous environment, meaning that individuals do not have any specific structure (including age, location, etc.) and can shift instantly between different compartments. The time evolution of these com- partments is described by a system of ordinary differential equations. We consider that acquired immunity is temporary, allowing individuals who re- cover from an infection to become susceptible again over time. This model focuses on nonlinear mathematical principles. We specifically analyze the transmission dynamics of contagious diseases with two infection stages and virus classes, using COVID-19 as an example. According to our analytical analysis, this model experiences a transcritical bifurcation at R0 = 1, sug- gesting that EE is reached when R0 is greater than 1. This highlights the necessity of lowering infection levels over time in order to mitigate the illness burden, even while it has no effect on the model's qualitative behaviour. Al- though previous research has explored the dynamics of infectious diseases, they generally do not account for multiple infection stages with varying virus classes. The primary mathematical finding of this research is the significant impact of virus classification on the spread of infectious diseases within a population that experiences multiple infection stages.

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