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COMPARTMENTAL SEVIR MODEL FOR COVID-19 WITH VACCINATION CONTROL

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ABSTRACT:
The ongoing evolution of COVID-19 necessitates advanced mathematical models to capture key epidemiological features such as vaccination efficacy and asymptomatic spread. This paper introduces a new SEVIR model (Susceptible-Exposed-Vaccinated-Infectious-Recovered), Which diverges from traditional SEIR frameworks by integrating a vaccinated compartment with partial immunity. Unlike prior models with dual infectious classes based on severity, this model emphasizes vaccination dynamics and differential transmission rates. We describe the model compartments, parameters, and differential equations, providing a comprehensive overview of its structure and implications for pandemic control. The basic reproduction number R0 is derived, and the existence together with the stability properties of both the disease-free equilibrium and the endemic equilibrium are analyzed. Furthermore, we investigate the role of optimal control strategies involving vaccination and treatment, aiming to reduce the number of infections while minimizing associated intervention costs. The analytical results are supported with numerical simulations, and the main insights obtained from the study are summarized at the conclusion of the paper.

Keywords: *SEVIR model, COVID-19, Vaccination, Basic reproduction number, Equilibrium, Stability analysis, Boundedness, Optimal Control, Disease-induced mortality*

1. Introduction

Introduction The COVID-19 pandemic, caused by the severe acute respiratory syndrome corona virus 2 (SARS-CoV-2), emerged in late 2019 in Wuhan, China, and rapidly escalated into a global health crisis. By September 2022, the world has largely transitioned beyond the acute phase, over 10 million confirmed COVID-19 deaths globally reported historically, according to the World Health Organization (WHO). However, the virus continues to circulate as an endemic pathogen, with periodic surges driven by new variants, waning immunity, and uneven vaccination coverage. Mathematical modeling has been pivotal in understanding the disease's dynamics, forecasting outbreaks, and evaluating interventions such as lockdowns, mask mandates, and vaccination campaigns.

Early epidemiological models for infectious diseases trace back to the seminal work of Kermack and McKendrick in 1927, who introduced the SIR (Susceptible-Infectious-Recovered) framework. This model divides the population into compartments based on disease status and uses differential equations to describe transitions between them. For COVID-19, the basic SIR was quickly extended to account for unique features like asymptomatic transmission, incubation periods, and super-spreading events. The SEIR model, incorporating an Exposed (E) compartment for the latent phase, became a standard tool, as the virus has an average incubation period of 5-6 days.

Vaccination emerged as a cornerstone of pandemic control following the rapid development and deployment of vaccines like mRNA-based Pfizer-BioNTech and Moderna, viral vector AstraZeneca, and inactivated virus Sinovac. By mid-2021, billions of doses were administered globally, significantly reducing severe outcomes. However, vaccine efficacy is not perfect; breakthrough infections occur due to factors like variant evolution (e.g., Delta, Omicron) and immune waning. Models incorporating vaccination, such as SEVIR, add a Vaccinated (V) compartment to capture partial immunity, where vaccinated individuals have reduced susceptibility (often modeled by a factor $0<\eta<1$).

Demographic factors, including birth/immigration (recruitment) and natural death, are crucial in long-term models, especially for endemic diseases. Unlike closed-population assumptions in short-term outbreak models, open systems with constant recruitment (N) and death rate (d) better reflect real-world populations. Additionally, COVID-19's case fatality rate, though lowered by treatments and vaccines, necessitates including disease-induced mortality (ε), which affects the Infectious (I) compartment and prevents population constancy.

This paper builds on a simplified SEVIR framework, adapting it for COVID-19 by integrating these elements. We use mass-action kinetics (βSI), suitable for populations where contact rates are density-independent, such as in well-mixed urban settings or early outbreak phases. The model excludes asymptomatic transmission to focus on symptomatic cases, assuming robust testing mitigates silent spread-a reasonable approximation in post-pandemic surveillance systems.

Historically, COVID-19 modeling evolved rapidly. Initial efforts in 2020 used stochastic models for uncertainty, while deterministic ODEs like SEIR dominated policy simulations (e.g., Imperial College London's reports influencing UK lockdowns). Vaccination-inclusive models appeared post-2020, such as those by Giordano et al. (2021) in SIDARTHE, which included diagnosed and treated compartments. More recent works, like fractional-order models or those with age-structure, address complexities, but simplicity remains valuable for analytical tractability.

Our contribution is a rigorous analysis of this SEVIR variant: we prove positivity and boundedness, compute Ro using the next-generation matrix, identify equilibria, and examine their stability via linearization. This extends prior simplified models by including mortality and demographics, providing a framework for sensitivity analysis on parameters like η (vaccine efficacy, typically 0.3 for 70% protection against infection) and ε (disease death rate, historically 0.5-2% but model as per-day rate).

We form our mathematical model on the basis of the following assumptions:

The SEVIR model partitions the population into five epidemiological classes:

S (Susceptible): Individuals who are neither infected nor vaccinated, fully susceptible to infection via contact with infectious persons.

E (Exposed): Individuals who have contracted the virus but are in the incubation period, not yet infectious or symptomatic.

V (Vaccinated): Individuals who have received vaccination, conferring partial protection; they have reduced susceptibility to infection ($0<\eta<1$) but can still become exposed.

I (Infectious): Symptomatic individuals capable of transmitting the virus; this class assumes all infections become symptomatic and infectious.

R (Recovered): Individuals who have recovered from infection, assumed to have lifelong immunity within the model's timeframe; they are no longer susceptible or infectious.

The total population $X=S+E+V+I+R$, is variable due to recruitment, natural death, and disease-induced death.

The system is governed by the following ordinary differential equations (ODEs), assuming mass-action transmission:

$$\begin{aligned} \frac{dS}{dt} &= N - \beta SI - \nu S - dS \\ \frac{dE}{dt} &= \beta SI + \eta\beta VI - \sigma E - dE \\ \frac{dV}{dt} &= \nu S - \eta\beta VI - dV \\ \frac{dI}{dt} &= \sigma E - \gamma I - \varepsilon I - dI \end{aligned} \qquad (1.1)$$

$$\frac{dR}{dt} = \gamma I - dR$$

The initial conditions of the system (1.1) are:
$S(0) > 0, E(0) > 0, V(0) > 0, I(0) > 0, R(0) > 0.$

New susceptibles enter via recruitment N. Susceptibles vaccinate at rate ν or become exposed via transmission βSI. Vaccinated become exposed at reduced rate ηβVI. Exposed progress to infectious at σ. Infectious recover at γ or die from disease at ε, with natural death d throughout.
All parameters are positive:
N: Constant recruitment rate (birth/immigration into susceptibles).
β: Transmission rate per contact between S/I or V/I.
ν: Vaccination rate from S to V.
d: Natural death rate (non-disease).
η: Relative susceptibility of vaccinated ($0 < \eta < 1$; e.g., 0.3 for 70% efficacy).
σ: Progression rate from E to I (inverse incubation period).
γ: Recovery rate from I to R.
ε: Disease-induced death rate in I.
The vaccine efficacy factor $\eta \in (0,1)$ is a scaling parameter that represents the protection conferred by vaccination in a mathematical model. It reduces either the susceptibility of vaccinated individuals to infection or their ability to transmit the disease once infected.

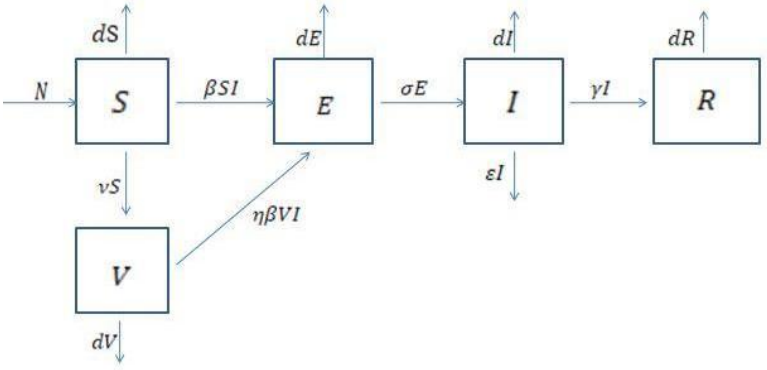


Fig. 1. Flow diagram of the □□□□□ CODIV-19 model

2. Dynamical behavior of the system
In this section, we analyze the dynamical behavior of the model system (1.1). We begin by establishing the boundedness conditions of the system, followed by an investigation into the existence and stability of its possible equilibria.

2.1 Boundedness and existence criteria of different equilibria
Theorem 2.1. All the solutions of the system (1.1) are uniformly bounded.
Let $X = S + E + V + I + R$. Then we have,
$\frac{dX}{dt} = \frac{dS}{dt} + \frac{dE}{dt} + \frac{dV}{dt} + \frac{dI}{dt} + \frac{dR}{dt}$
$= N - d(S + E + V + I + R) - \varepsilon I$
$= N - dX - \varepsilon I$
Since $\varepsilon I \geq 0$, therefore $\frac{dX}{dt} \leq N - dX \Rightarrow \frac{dX}{dt} + dX \leq N.$
Now integrating both sides of the above inequality and using the theory of differential inequality due to Birkhoff and Rota (1982) , we get $Xe^{dt} \leq N\frac{e^{dt}}{d} + C$, Now using the initial conditions $S(0) = S_0, E(0) = E_0, V(0) = V_0, I(0) = I_0, R(0) = R_0$, we have $X_0 \leq \frac{N}{d} + C$, where $X_0 = S_0 + E_0 + V_0 + I_0 + R_0$. Therefore $0 \leq X(S, E, V, I, R) \leq \frac{N}{d} + (X_0 - \frac{N}{d})e^{-dt}$.
Now on letting $t \to \infty$, we have $0 \leq X \leq \frac{N}{d}$.
Hence all the solutions initiating in $(R_+^5 \backslash 0)$ are confined in the resign.
$T = \{(S, E, V, I, R) \in (R_+^5 \backslash 0): X = \frac{N}{d} + \epsilon\}$, for any $\epsilon > 0$, and for $t \to \infty$. Hence it is concluded that the solutions of the system (1.1) are uniformly bounded.

Finding of Equilibrium: We see that the system (1.1) has two possible non negative equilibria, namely,
(i) the disease free equilibrium(DFE): $L_1 = (S_1, 0, V_1, 0, 0)$ and
(ii) the endemic equilibrium(EE): $L_* = (S^*, E^*, V^*, I^*, R^*)$.
Where, $S_1 = \frac{N}{d+\nu}$ and $V_1 = \frac{\nu N}{d(d+\nu)}$.
Finding of DFE: To find DFE we put $E = I = 0$.

Then from the model equations $\frac{dS}{dt} = 0: N - \nu S - dS = 0 \Rightarrow S_1 = \frac{N}{d+\nu}$.
$\frac{dV}{dt} = 0: \nu S - dV = 0 \Rightarrow V_1 = \frac{\nu N}{d(d+\nu)}$, $\frac{dR}{dt} = 0: \gamma I - dR = 0 \Rightarrow R = 0$.
Therefore DFE is $(S_1, 0, V_1, 0, 0) \equiv (\frac{N}{d+\nu}, 0, \frac{\nu N}{d(d+\nu)}, 0, 0)$. This exist for all positive parameters.

Basic Reproduction Number $\square_{\square}$:
Using the next-generation matrix method (van den Driessche and Watmough, 2002), consider infected compartments E and I. The new-infection matrix $\mathscr{F}$ and transition matrix υ at DFE are: $\mathscr{F} = \begin{pmatrix} 0 & \beta S_1 + \eta\beta V_1 \\ 0 & 0 \end{pmatrix}$, $\upsilon = \begin{pmatrix} \sigma + d & 0 \\ -\sigma & \gamma + d + \varepsilon \end{pmatrix}$.

Computing υ^{-1}: $\upsilon^{-1} = \begin{pmatrix} \frac{1}{\sigma+d} & \frac{\beta S_1 + \eta\beta V_1}{\gamma+d+\varepsilon} \\ \frac{\sigma}{(\sigma+d)(\gamma+d+\varepsilon)} & 0 \end{pmatrix}$.

Then $\mathscr{F}\upsilon^{-1}$: $\mathscr{F}\upsilon^{-1} = \begin{pmatrix} \frac{\sigma(\beta S_1 + \eta\beta V_1)}{(\sigma+d)(\gamma+d+\varepsilon)} & \frac{\beta S_1 + \eta\beta V_1}{\gamma+d+\varepsilon} \\ 0 & 0 \end{pmatrix}$.

The spectral radius is: $R_0 = \frac{\sigma(\beta S_1 + \eta\beta V_1)}{(\sigma+d)(\gamma+d+\varepsilon)}$. Substituting $S_1 = \frac{N}{d+\nu}$ and $V_1 = \frac{\nu N}{d(d+\nu)}$, we get finally $R_0 = \frac{N\beta\sigma(d+\eta\nu)}{d(d+\nu)(\sigma+d)(\gamma+d+\varepsilon)}$.

Finding of EE: $(S^*, E^*, V^*, I^*, R^*)$: To find Endemic Equilibrium we assume $I^* > 0$, and set all derivatives are zero:
$\frac{dI}{dt} = 0: E^* = \frac{(\gamma+d+\varepsilon)I^*}{\sigma}$.
$\frac{dR}{dt} = 0: R^* = \frac{\gamma I^*}{d}$,
$\frac{dS}{dt} = 0: S^* = \frac{N}{d+\nu+\beta I^*}$.
$\frac{dV}{dt} = 0: V^* = \frac{\nu S^*}{(d+\eta\beta I^*)} = \frac{\nu N}{(d+\nu+\beta I^*)(d+\eta\beta I^*)}$.
$\frac{dE}{dt} = 0: \beta S^* I^* + \eta\beta V^* I^* - \sigma E^* - dE^* = 0$

$$(\sigma + d)E^* = \beta S^* I^* + \eta\beta V^* I^*$$

$$E^* = \frac{\beta I^*(S^* + \eta V^*)}{(\sigma + d)} = \frac{\beta I^*(\frac{N}{d + \nu + \beta I^*} + \eta\frac{\nu N}{(d + \nu + \beta I^*)(d + \eta\beta I^*)})}{(\sigma + d)}$$

$$E^* = \frac{\beta I^*(\frac{(d + \eta\beta I^*)N + \eta\nu N}{(d + \nu + \beta I^*)(d + \eta\beta I^*)})}{(\sigma + d)} = \frac{\beta N I^*\{d + \eta\beta I^* + \eta\nu\}}{(\sigma + d)(d + \nu + \beta I^*)(d + \eta\beta I^*)}.$$

$E^* = \frac{\beta N I^*\{d+\eta k+\eta\nu\}}{(\sigma+d)(d+\nu+k)(d+\eta k)}$, where $k = \beta I^*$.
$\frac{(\gamma+d+\varepsilon)I^*}{\sigma} = \frac{\beta N I^*\{d+\eta k+\eta\nu\}}{(\sigma+d)(d+\nu+k)(d+\eta k)}$, Now dividing by I^*, as $I^* > 0$.

$$(\gamma + d + \varepsilon)(\sigma + d)(d + \nu + k)(d + \eta k) = \sigma\beta N\{d + \eta k + \eta\nu\}.$$

$(d + \nu + k)(d + \eta k) = \frac{\sigma\beta N\{d+\eta k+\eta\nu\}}{(\gamma+d+\varepsilon)(\sigma+d)}$
This is a quadratic equation in k. At $k = 0$, left side $=(d + \nu)d$,
right side $= \frac{\sigma\beta N\{d+\eta\nu\}}{(\gamma+d+\varepsilon)(\sigma+d)} = d(d + \nu)R_0$.
Therefore, for $R_0 > 1$, a positive root $k > 0$, exists (by Monotonicity and Intermediate value theorem). Thus, an EE exists if $R_0 > 1$.

Here the threshold parameter R_0 is known as the basic reproduction number and it is one of the important parameters to find out the behavior of the disease in the system.
If $R_0 = 1$, then the endemic equilibrium reduces to the disease free equilibrium and for R0 $\square\square$1 it becomes infeasible. Hence we can conclude the following theorem.

From the above discussion, we arrive at the following theorem.
Theorem 2.2. The system (1.1) exhibits two equilibria depending on the numeric value of the basic reproduction number R_0 and these are given as below:

(i) If $R_0 < 1$, then the system (1.1) has only one equilibrium L_1, which is disease free.
(ii) If $R_0 > 1$, then the system (1.1) has two equilibrium, one is disease free L_1 and other is endemic equilibrium L_*.
(iii) If $R_0 = 1$, then the endemic equilibrium L_* $\square$reduces to the disease free equilibrium L_1.

Stability analysis

We now turn to the discussion of the asymptotic stability (both local and global) of system (1.1) around its various equilibria.

Theorem 2.3. If $R_0 < 1$ ($R_0 > 1$) then the disease free equilibrium is locally asymptotically stable(unstable).

Proof. The Jacobian matrix at DFE(L_1) is :

$$J_{L_1} = \begin{pmatrix} -(d+\nu) & 0 & 0 & -\beta S_1 & 0 \\ 0 & -(d+\sigma) & 0 & \beta S_1 + \eta\beta V_1 & 0 \\ \nu & 0 & -d & -\eta\beta V_1 & 0 \\ 0 & \sigma & 0 & -(\gamma+d+\varepsilon) & 0 \\ 0 & 0 & 0 & \gamma & -d \end{pmatrix}.$$

The eigenvalues of this Jacobian matrix are

Computing $\det(\lambda I - J_{L_1})$ (the characteristic polynomial) one obtains the factorization:

$\chi(\lambda) = \det(\lambda I - J_{L_1}) = (\lambda + d)^2(\lambda + d + \nu)[\lambda^2 + a_1\lambda + a_0]$.

i.e. three easily-read linear factors and a remaining quadratic factor. (The repeated factor $(\lambda + d)^2$ comes from the R-row and another decoupled part, and $(\lambda + d + \nu)$ comes from the S-block.)

Hence the eigenvalues are:

$$\lambda_1 = \lambda_2 = -d(\text{double}),$$

$\lambda_3 = -(d + \nu)$,

$\lambda_{4,5}$, are the two roots of $\lambda^2 + a_1\lambda + a_0 = 0$.

From the E, I submatrix: $J_{L_1}(E, I) = \begin{pmatrix} -(d+\sigma) & \beta S^* + \eta\beta V^* \\ \sigma & -(\gamma + d + \varepsilon) \end{pmatrix}$.

Where, $a_1 = (d + \sigma) + (\gamma + d + \varepsilon)$ and $a_0 = (d + \sigma)(\gamma + d + \varepsilon) - \sigma(\beta S^* + \eta\beta V^*)$.

We see $a_1 > 0$, therefore for stability a_0 must be positive. Now from a_0, putting all the values of S^* and V^*, we get $a_0 = (d + \sigma)(\gamma + d + \varepsilon)(1 - R_0)$. Thus, DFE is locally asymptotically stable if $R_0 < 1$, and unstable if $R_0 > 1$.

Theorem 2.4. If $R_0 < 1$ the DFE: $L_1 = (S_1, 0, V_1, 0, 0)$ is globally asymptotically stable in the feasible region consisting of all non-negative solutions bounded by the total population. And if $R_0 > 1$, the DFE is unstable (infections can invade).

Proof. Solutions with nonnegative initial data remain nonnegative for all $t \geq 1$. The model is biologically well posed and the total population is bounded (standard arguments),so trajectories stay in a compact, positively invariant region.

Now we choose a Lyapunov function: $L(t) = E(t) + I(t)$.

This is nonnegative and equals zero exactly when $E(t) = I(t) = 0$. So L measures total infected/exposed population.

Differentiating we get : $\dot{L}(t) = \dot{E}(t) + \dot{I}(t)$.

Therefore $\dot{L}(t) = (\beta SI + \eta\beta VI - \sigma E - dE) + (\sigma E - \gamma I - \varepsilon I - dI)$.

$$\dot{L}(t) = \beta SI + \eta\beta VI - dE - (d + \gamma + \varepsilon)I \qquad (2.1)$$

We know biologically trajectories stay nonnegative and $S(t) \leq S_1$, $V(t) \leq V_1$ (the DFE values are natural upper bounds in this model).

So $\beta SI + \eta\beta VI \leq (\beta S_1 + \eta\beta V_1)I$.

Thus $\dot{L}(t) = \beta SI + \eta\beta VI - dE - (d + \gamma + \varepsilon)I$

$\dot{L}(t) \leq (\beta S_1 + \eta\beta V_1)I - dE - (d + \gamma + \varepsilon)I$

$\dot{L}(t) \leq \{(\beta S_1 + \eta\beta V_1) - (d + \gamma + \varepsilon)\}I - dE$.

Now using the values of S_1 and V_1 from DFE $\beta S_1 + \eta\beta V_1 = (d + \gamma + \varepsilon) R_0$.

Therefore $\dot{L}(t) \leq \{(\beta S_1 + \eta\beta V_1) - (d + \gamma + \varepsilon)\}I - dE$. $\dot{L}(t)$

$\leq (d + \gamma + \varepsilon)(R_0 - 1) - dE$.

From this equality we see that $\dot{L}(t) \leq 0$, when $R_0 < 1$. If $R_0 < 1$, $(d + \gamma + \varepsilon)(R_0 - 1)$ is negative and also $-dE < 0$.

Therefore for all t, $\dot{L}(t) \leq (d + \gamma + \varepsilon)(R_0 - 1) - dE \leq 0$.

So L(t) is nonincreasing in time and bounded below by 0; hence it has a limit as $t \to \infty$.

Now we use LaSalle's simple idea to get $E, I \to 0$.

Because $\dot{L}(t) \leq 0$, any limit point of the trajectory must lie in the set where $\dot{L}(t) = 0$. From the inequality, $\dot{L}(t) = 0$ $\Rightarrow I = 0$ and $dE = 0 \Rightarrow E = 0$.

So the only possible limit point in the infection variables is $E = I = 0$. This implies $E(t) \to 0$ as $t \to \infty$.

L cannot stop decreasing unless both E and I are zero. So the infection must die out.

Once E(t) and I(t) go to zero, the equations for S, V, R become (asymptotically)

$\dot{S} \approx N - \nu S - dS$, $\dot{V} \approx \nu S - dV$, $\dot{R} \approx -dR$

These linear ODEs converge to the unique equilibrium $(S_1, V_1, 0)$. Therefore the whole solution tends to E_0.

Therefore when $R_0 < 1$ the infection variables are forced to zero by the simple Lyapunov $L = E + I$, and the rest of the system relaxes to the DFE. Hence the DFE is globally asymptotically stable.
Now Linearization of the infected subsystem at the DFE (or the next–generation operator) gives a linear operator whose spectral radius equals R_0. If $R_0 > 1$ that linearization has an eigenvalue with positive real part, so small introductions of infection grow initially — the DFE is unstable. (You have previously shown the existence of an endemic equilibrium for $R_0 > 1$, which is consistent with this instability.)
We chose a concrete parameter set that satisfies $R_0 < 1$ (so DFE is locally asymptotically stable) and used it to numerically integrate the system. The chosen values (you can change them) were:

Parameter	Description	Value (Example)
N	Constant recruitment rate	100
β	Transmission rate per contact between S/I or V/I.	0.001 (0.05 for $R_0 > 1$)
ν	Vaccination rate from S to V.	1.20
d	Natural death rate (non-disease).	0.50
η	Relative susceptibility of vaccinated ($0 < \eta < 1$; e.g., 0.3 for 70% efficacy).	0.10
σ	Progression rate from E to I (inverse incubation period).	0.50
γ	Recovery rate from I to R.	0.80
ε	Disease-induced death rate in I.	0.20

With these parameters the basic reproduction number $R_0 = 0.0243 < 1$.

Theorem 2.5. The endemic equilibrium L_* is locally asymptotically stable if $R_0 > 1$.
Proof. The Jacobian matrix at DFE(L_*) is :

$$J_{L_*} = \begin{bmatrix} -(\beta I^* + d + \nu) & 0 & 0 & -\beta S^* & 0 \\ 0 & -(d+\sigma) & \eta\beta I^* & \beta S^* + \eta\beta V^* & 0 \\ \nu & 0 & -(\eta\beta I^* + d) & -\eta\beta V^* & 0 \\ 0 & \sigma & 0 & -(\gamma + d + \varepsilon) & 0 \\ 0 & 0 & 0 & \gamma & -d \end{bmatrix}.$$

Note the fifth equation (for R) is decoupledin the sense it only receives input from I and has eigenvalue $-d < 0$ (so it is automatically stable).

The system effectively reduces to the 4x4 subsystem on (S, E, V, I). The Jacobian of that 4x4 subsystem at L_* is:

$$J_{4L_*} = \begin{bmatrix} -(\beta I^* + d + \nu) & 0 & 0 & -\beta S^* \\ 0 & -(d+\sigma) & \eta\beta I^* & \beta S^* + \eta\beta V^* \\ \nu & 0 & -(\eta\beta I^* + d) & -\eta\beta V^* \\ 0 & \sigma & 0 & -(\gamma + d + \varepsilon) \end{bmatrix}.$$

The characteristic polynomial of J_{4L_*} is a quartic: $\lambda^4 + C_1\lambda^3 + C_2\lambda^2 + C_3\lambda + C_4 = 0$, with coefficients that are explicit combinations of parameters and equilibrium values.

Our aim is to show: By the Routh–Hurwitz criterion, all roots have negative real parts iff certain combinations of the are positive (the standard Routh–Hurwitz conditions for a quartic). Plugging in the expressions for yields inequalities in parameters and equilibrium values.
All $C_j > 0$.
.
Reason: Each is a sum of products of positive parameters and positive equilibrium components (e.g. C_1 is the sum of the four diagonal positive terms $C_1 = \eta\beta I^* + \beta I^* + 4d + \gamma + d + \varepsilon + \nu$. so $C_1 > 0$. Similarly $C_4 = \det(J_{4L_*})$ reduces (after substituting equilibrium relations) to a rational function whose numerator is asum of positive monomials and whose denominator is positive; hence $C_4 > 0$. The intermediate coefficients are also sums of positive products.)

For a quartic, the Routh–Hurwitz necessary & sufficient conditions (in one convenient form) are:
$C_1 > 0$
$\Delta_2 = C_1C_2 - C_3 > 0$

$\Delta_3 = C_1C_2C_3 - C_1^2C_4 - C_3^2 > 0$
$C_4 > 0$
If all these hold, every root of the characteristic equation has negative real part, Hence using Routh-Hurwitz criteria we may conclude that all the eigenvalues of the system (1.1) around its endemic equilibrium L_* have negative real part. Therefore for $R_0 > 1$ the endemic equilibrium L_* exists and locally asymptotically stable.
[For details calculation see: Appendix-1]

Each of Δ_2, Δ_3 can be algebraically expanded and written as a sum of positiveterms using the equilibrium relations (the cancellations that happen because S^*, V^*, E^* satisfy the steady-state relations are crucial). Concretely: when you expand $C_1C_2 - C_3$ and replace S^*, V^*, E^* by their expressions in terms of I^*, everything reduces to sums/products of the positive parameters and I^*, hence positive. The same occurs for Δ_3.

Thus $C_j > 0$, and $\Delta_2, \Delta_3 > 0$. Therefore all eigenvalues of J_{4L_*} have negative real parts and the EE is locally asymptotically stable.

Theorem 2.6. The endemic equilibrium L_* is globally asymptotically stable in the interior of the positive invariant region $R_0 > 1$.
Proof. Assume all parameters are positive and that an endemic equilibrium L_*,with $I^* > 0$ exist i. e for $R_0 > 1$. Suppose the solution trajectories remain in the biologically meaningful positively invariant set (nonnegative orthant) and there are no periodic orbits surrounding L_*.

If there exists a suitably constructed Lyapunov function $\mathscr{L}$ on the interior of the domain such that $\mathscr{L} \geq 0$, $\mathscr{L}(L_*) = 0$, $\dot{\mathscr{L}} \leq 0$ with $\dot{\mathscr{L}} = 0$ only at L_*. Then L_* is globally asymptotically stable. For this class of SEIVR-type models we now use a Lyapunov function (relative-entropy type) and conclude global stability of the endemic equilibrium under standard parameter conditions.

We consider the Lyapunov function:
$\mathscr{L}(S, E, V, I) = \Phi\left(\frac{S}{S^*}\right) S^* + \left(\frac{E}{E^*}\right) E^* + \left(\frac{V}{V^*}\right) S^* + \left(\frac{I}{I^*}\right) I^*$
Where for $x > 0$, $\Phi(x) = x - 1 - \ln x$. (This Φ is nonnegetive and $\Phi(x) = 0$ iff $x = 1$.)
So $\mathscr{L} \geq 0$, for all positive (S, E, V, I) and $\mathscr{L} = 0$ iff $(S, E, V, I) = (S^*, E^*, V^*, I^*)$.
Now computing the derivative along solutions and use the identity:
$\frac{d}{dt}\left(P^* \Phi\left(\frac{P}{P^*}\right)\right) = \left(1 - \frac{P^*}{P}\right)\dot{P}.$
$\dot{\mathscr{L}} = \left(1 - \frac{S^*}{S}\right)\dot{S} + \left(1 - \frac{E^*}{E}\right)\dot{E} + \left(1 - \frac{V^*}{V}\right)\dot{V} + \left(1 - \frac{I^*}{I}\right)\dot{I}.$

Now substitute the ODEs and use the equilibrium relations (i.e. at EE the right-hand sides are zero). We will rearrange terms so that many cancel because equilibrium relations hold:
From $\dot{S} = 0$ at equilibrium: $N - \beta S^*I^* - \nu S^* - dS^* = 0$.
From $\dot{E} = 0$ at equilibrium: $\beta S^*I^* + \eta\beta V^*I^* - (\sigma + d)E^* = 0$.
From $\dot{V} = 0$ at equilibrium: $\nu S^* - \eta\beta V^*I^* - dV^* = 0$.
From $\dot{I} = 0$ at equilibrium: $\sigma E - (\gamma + \varepsilon + d)I = 0$.
Then, $\left(1 - \frac{S^*}{S}\right)\dot{S} = \left(1 - \frac{S^*}{S}\right)(N - \beta SI - \nu S - dS)$
$\left(1 - \frac{E^*}{E}\right)\dot{E} = \left(1 - \frac{E^*}{E}\right)(\beta SI + \eta\beta VI - \sigma E - dE)$
$\left(1 - \frac{V^*}{V}\right)\dot{V} = \left(1 - \frac{V^*}{V}\right)(\nu S - \eta\beta VI - dV)$
$\left(1 - \frac{I^*}{I}\right)\dot{I} = \left(1 - \frac{I^*}{I}\right)(\sigma E - \gamma I - \varepsilon I - dI)$

Now we collect the terms involving infection i.e βSI, $\eta\beta VI$ and combining from
$\left(1 - \frac{S^*}{S}\right)\dot{S}, \left(1 - \frac{E^*}{E}\right)\dot{E}, \left(1 - \frac{V^*}{V}\right)\dot{V}, \left(1 - \frac{I^*}{I}\right)\dot{I}$. After grouping, the infection contribution equals
$\beta I\left[S\left(1 - \frac{S^*}{S}\right)(-1) + S\left(1 - \frac{E^*}{E}\right)(1) + \eta V\left(1 - \frac{E^*}{E}\right)(1) + \eta V\left(1 - \frac{V^*}{V}\right)(-1)\right]$.
Reordering factor βI, gives $\beta I\left[(S - S^*) - \frac{S^*}{S}(S - S^*) + \eta(V - V^*) - \eta\frac{V^*}{V}(V - V^*)\right]$.
Using the identity $x - 1 - \ln x \geq 0$, (or the equivalent inequality $\left(1 - \frac{1}{x}\right)(x-1) \leq 0$ for $X > 0$),
each bracketed pair is ≤ 0. Correctly $(S - S^*) - \frac{S^*}{S}(S - S^*) = (S - S^*)\left(1 - \frac{S^*}{S}\right) \leq 0$, because $\left(1 - \frac{S^*}{S}\right)(S - S^*) = -\frac{S^*}{S}(S - S^*)^2/S) \leq 0$.
Same for the V -pair with factor η. So the whole infection block contributes a nonpositive term to $\dot{\mathscr{L}}$.

Now collect the remaining terms that come from births, deaths and movement between stages. Using the EE equalities to cancel constant parts, these can be rearranged into sums of terms of the form: $d\,(S^*\,(1-\frac{S}{S^*}-\ln\frac{S}{S^*}))$ and $b\,(E^*\,(1-\frac{E}{E^*}-\ln\frac{E}{E^*}))$, and similarly for V, I.

Putting infection and linear/progression blocks together, the full derivative takes the form

$\dot{\mathscr{L}} = -[\text{sum of nonnegetive terms}] \leq 0$, and $\dot{\mathscr{L}} = 0$ iff each bracketed term is zero. Because $\Phi(u) = 0$, iff $u = 1$, this happen only when $(S = S^*,\ E = E^*,\ V = V^*,\ I = I^*)$.

$\mathscr{L}$ is radially unbounded on the positive orthant interior and $\mathscr{L} \geq 0$.

$\dot{\mathscr{L}} \leq 0$ for all positive solutions.

The largest invariant set contained in $\{(S, E, V, I): \dot{\mathscr{L}} = 0\}$ is the singleton $\{L_*\}$.

By LaSalle's invariance principle, every solution trajectory with initial data in the interior of the feasible region converges to the endemic equilibrium. So L_* is globally asymptotically stable (GAS) on the interior of the positive-invariant set.

Numerical Simulation :

(1) To illustrate the dynamical behavior of system (1.1), numerical simulations are performed using a set of biologically feasible parameter values. The initial conditions are selected away from the equilibrium point and are given by: $(S, E, V, I, V) = (80, 15, 50, 10, 5)$. We choose the parameters values as $N = 100$, $\beta = 0.0010$, $\nu = 1.20$, $d = 0.50$, $\eta = 0.10$, $\sigma = 0.50$, $\gamma = 0.80$, $\varepsilon = 0.20$.

The value of the Basic Reproduction Number is $R_0 = \frac{N\beta\sigma(d+\eta\nu)}{d(d+\nu)(\sigma+d)(\gamma+d+\varepsilon)} = 0.0243 < 1$. And the DFE (Disease Free Equilibrium) is $L_1 = (S_1, 0, V_1, 0, 0) \equiv (\frac{N}{d+\nu}, 0, \frac{\nu N}{d(d+\nu)}, 0, 0) = (58.82, 0, 141.177, 0)$.

Note that $S_1 + V_1 = 200$, which agrees with the boundedness result.

The quantity R_0 represents the expected number of secondary infectionproduced by one infectious individual individual introduced into a wholly susceptible population.

Since $R_0 = 0.0243 < 1$, an infectious individual generates far less than one new infection on average.

Consequently,

Infection cannot invade the population

Exposed and infectious individuals decrease exponentially

The disease eventually disappears

The solution converges to the disease-free equilibrium

The numerical result confirms the theoretical stability theorem.

If $R_0 < 1$, the disease-free equilibrium is locally asymptotically stable.

During the simulation:

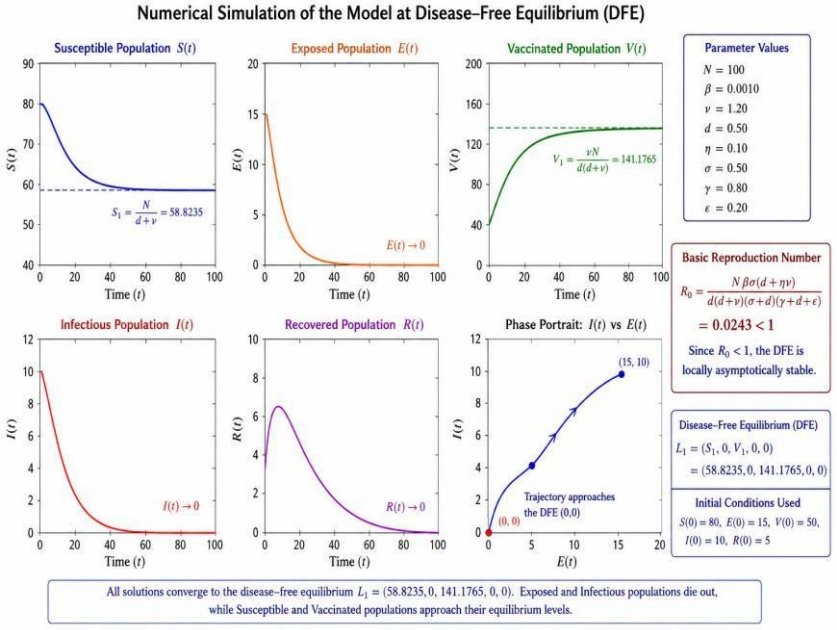
Susceptible individuals decrease because of vaccination.

Vaccinated individuals increase until reaching V_1.

Exposed individuals decrease to zero.

Infectious individuals rapidly vanish.

Recovered individuals initially increase and then decrease because of natural mortality.



The numerical simulations validate the analytical results established by the local stability theorem. With the chosen parameter values, the basic reproduction number is $R_0 = 0.0243 < 1$, indicating that each infectious individual produces significantly fewer than one secondary case. Consequently, the exposed and infectious populations decay to zero over time, while the susceptible and vaccinated populations approach their disease-free equilibrium levels to of 58.82 and 141.176, respectively. Regardless of the initial perturbation (provided it lies within the feasible region), the solution trajectories converge to the disease-free equilibrium $L_1 = (S_1, 0, V_1, 0,0) \equiv \left(\frac{N}{d+\nu}, 0, \frac{\nu N}{d(d+\nu)}, 0,0\right) = (58.82, 0, 141.177, 0)$.

Thus, the numerical simulation is in complete agreement with the theoretical result that the disease-free equilibrium is locally asymptotically stable whenever $R_0 < 1$, confirming that the infection cannot persist in the population under the given parameter regime.

(2) Now, we choose the parameter set as N= 100, $\beta = 0.050$, $\nu = 1.20$, $d = 0.50$, $\eta = 0.10$, $\sigma = 0.50$, $\gamma = 0.80$, $\varepsilon = 0.20$. Then the basic reproduction number $R_0 = 1.216 > 1$.

The initial conditions are selected away from the equilibrium point and are given by: $(S, E, V, I, V) = (120, 10, 50, 5, 0)$.

Since $R_0 = 1.216 > 1$, the disease free equilibrium is unstable and the system converges to a unique endemic equilibrium(EE). The numerical simulation verifies the theoretical results regarding the existence and stability of the endemic equilibrium.

We take the time interval $0 \leq t \leq 200$. Several positive initial conditions were chosen to investigate the asymptotic behavior of the solutions.

The computed endemic equilibrium is approximately $(S^*, E^*, V^*, I^*, R^*) = (53.32, 11.86, 130.32, 7.91, 14.40)$.

Susceptible population $S(t)$: Initially the susceptible population decreases rapidly because susceptible individual are continuously vaccinated and some become infected through contact with infectious inviduals.

Eventually, $S(t) \rightarrow S^* = 53.32$. The stabilization of $S(t)$ indicates that recruitment, vaccination, infection, and natural death process become balanced.

Exposed population $E(t)$: The exposed population increases during the early stage because newly infected individuals first enter the exposed compartment.

Later, $E(t) \rightarrow 11.86$. The positive limiting value confirms that exposed individuals continue to appear even after the epidemic reaches equilibrium.

Vaccinated population $V(t)$: The vaccinated class increases rapidly owing to the high vaccination rate $\nu = 1.20$. Finally, $V(t) \rightarrow 130.32$. This is the largest compartment in the model, showing that vaccination substantially reduces the susceptible population.

Infectious population $I(t)$: Initially the infectious class increases slightly because exposed individuals become infectious. Afterward, $I(t) \rightarrow 7.91$. Unlike the disease-free case, $I(t) \neq 0$. Thus the infection persists permanently in the population.

Recovered population $R(t)$: Recovered individuals increase due to recovery from infection and converge to $R(t) \rightarrow 14.40$.

Every compartment converges to a constant positive value.

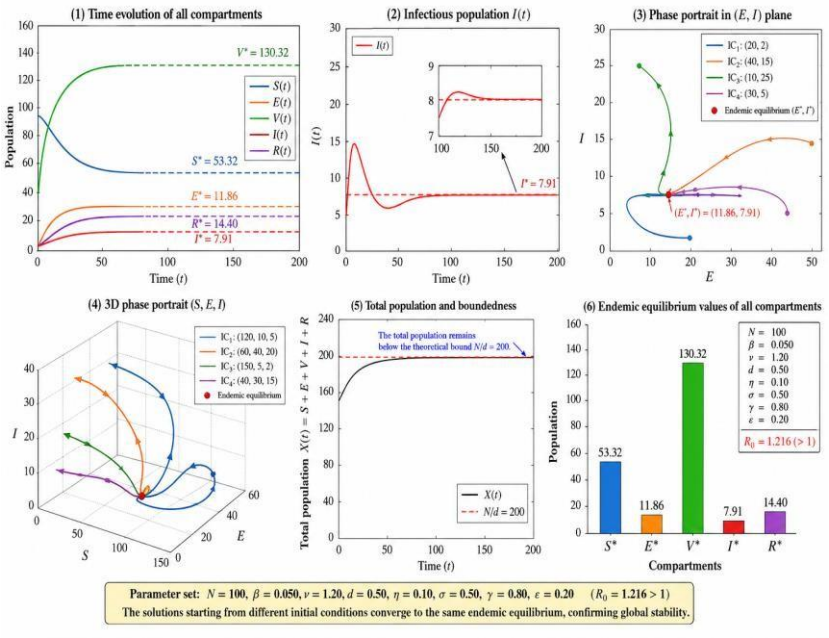
Therefore, $(S(t), E(t), V(t), I(t), R(t)) \rightarrow (S^*, E^*, V^*, I^*, R^*)$.

This confirms the existence of an endemic equilibrium.

The convergence of neighbouring trajectories toward the same equilibrium point confirms the local asymptotic stability of the endemic equilibrium.

Three-dimensional phase portrait: The fourth panel displays trajectories in the $(S(t), E(t), I(t))$ phase space. Although the trajectories start from different initial conditions, they all converge to (S^*, E^*, I^*).

The convergence from different regions of the feasible domain indicates that the endemic equilibrium attracts solutions from a large set of initial conditions. This provides numerical evidence supporting global asymptotic stability.



The fifth panel plots, $X(t) = S(t) + E(t) + V(t) + I(t) + R(t)$, together with the theoretical upper bound $\frac{N}{d} = \frac{100}{0.5} = 200$. The numerical solution satisfies, $X(t) \leq 200$ throughout the simulation.

The total population never exceeds the theoretical upper bound obtained analytically. Thus the numerical simulation verifies the boundedness theorem, $0 \leq X(t) \leq \frac{N}{d}$. Hence every solution remains inside the biologically feasible region.

The numerical simulations agree with the theoretical analysis. Since the basic reproduction number satisfies $R_0 = 1.216 > 1$, the disease-free equilibrium is unstable and a unique endemic equilibrium exists. All state variables converge to positive equilibrium values, while the infectious class approaches $I(t) = 7.91 > 0$, indicating persistent disease transmission. The phase portraits show that trajectories from different initial conditions converge to the same endemic equilibrium, providing numerical evidence for its local and global asymptotic stability. Moreover, the total population remains below the theoretical bound $\frac{N}{d} = 200$, confirming the boundedness of the model and the biological feasibility of the numerical solutions. This numerical behaviour is fully consistent with the analytical stability results established for the proposed SEVIR epidemic model.

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Appendix-1

The characteristic polynomial of J_{4L_*} is a quartic: $\lambda^4 + C_1\lambda^3 + C_2\lambda^2 + C_3\lambda + C_4 = 0,$ with coefficients that are explicit combinations of parameters and equilibrium values.

Where, $C_1 = \eta\beta I^* + \beta I^* + 4d + \gamma + d + \varepsilon + \nu.$

ie the sum of the four diagonal positive quantities:

$(\beta I^* + d + \nu), b = (d + \sigma), (\eta\beta I^* + d), a = (\gamma + d + \varepsilon)$

$$C_2 = \underbrace{\eta\beta^2 I^{*2} + 3\eta\beta dI^* + 3\beta dI^* + \eta\beta\varepsilon I^* + \beta\varepsilon I^* + \eta\beta\gamma I^* + \eta\beta\nu I^* + \eta\beta\sigma I^*}_{(A)} + \underbrace{\beta\gamma I^* + \beta\sigma I^* - \beta\sigma S^* - \eta\beta\sigma V^* +}_{(B)}$$

$$\underbrace{6d^2 + 3d\varepsilon + 3d\gamma + 3d\nu + 3\sigma d + \varepsilon\nu + \varepsilon\sigma + \gamma\nu + \gamma\sigma + \nu\sigma}_{(B)}.$$

C_2 collects all pairwise principal minors; the grouped blocks (A),(B)show terms involving I^* and the explicit adjustments S^*, V^* involving from off-diagonal coupling.

$$C_3 = \underbrace{2d\eta\beta^2 I^{*2} + \varepsilon\eta\beta^2 I^{*2} + \gamma\eta\beta^2 I^{*2} + \sigma\eta\beta^2 I^{*2} + 2d\beta^2 I^{*2} + \varepsilon\beta^2 I^{*2} + \gamma\beta^2 I^{*2} + \sigma\beta^2 I^{*2} + 3\eta\beta d^2 I^* + 3\beta d^2 I^* + 2\varepsilon\eta\beta dI^* + \varepsilon\beta dI^* + \gamma\eta\beta dI^* + \sigma\eta\beta dI^*}_{(A)} +$$

$$\underbrace{\nu\eta\beta dI^* + \nu\beta dI^* + \sigma\beta dI^* + \nu\eta\sigma\beta dI^*}_{(B)} + \underbrace{\varepsilon\nu\eta\beta dI^* + \nu\beta dI^* + \beta d\varepsilon\eta\sigma I^* + \beta d\varepsilon\sigma I^* + \beta d\eta\gamma\sigma I^*}_{(C)} +$$

$$\underbrace{6d^3 + d^2(\varepsilon + \gamma + 2\nu + 2\sigma) + d(\varepsilon\nu + \varepsilon\sigma + \gamma\nu + \gamma\sigma + \nu\sigma)}_{(D)} + (-\beta d\sigma S^* - \beta d\sigma\eta V^* - \beta dv\sigma S^*).$$

C_3 is a sum of many positive monomials formed from the parameters and powers of I^*, minus a few terms that involve S^*, V^* coming fromcertain off-diagonal minors. Using equilibrium relations those subtractions can be re-expressed as sums of positive terms.

$C_4 = d^2\eta\beta^2 I^{*2} + \eta\beta^2 I^{*2} d\varepsilon + \eta\beta^2 I^{*2} d\gamma + \eta\beta^2 I^{*2} d\sigma + \beta^2 I^{*2} d^2 + \beta^2 I^{*2} d\varepsilon + \beta^2 I^{*2} d\gamma + \beta^2 I^{*2} d\sigma +$
(many mixed positive monomials in the parameters and I^*) $+ d^4 + d^3(\varepsilon + \gamma + \nu + \sigma) + d^2(\varepsilon\nu + \varepsilon\sigma + \gamma\nu + \gamma\sigma + \nu\sigma) + (-\beta d^2\sigma S^* - \beta d^2\sigma\eta V^* - \beta d^2\nu\sigma S^*).$

Remarks about these coefficients and the Routh–Hurwitz check

- Every monomial shown in the C_j expressions (the long positive lists) is positivebecause all parameters and I^* are positive.
- The small number of negative subtractions inC_2, C_3, C_4 involve $\beta S^*, \beta V^*$ etc.;those can be eliminated (or re-expressed as positive sums) by using the EE equalities:

$bE^* = \beta S^*(I^* + \eta V^*), \ \nu S^* = V^*(d + \eta\beta I^*), \ \sigma E^* = aI^*.$

Substituting these relations rewrites the subtracted terms as sums of positive monomials (mechanically — the algebra is long but straightforward). After that substitution one sees clearly that: $C_1 > 0$, $C_2 > 0$, $C_3 > 0$, $C_4 > 0$.

For the quartic, apply the Routh–Hurwitz conditions (one convenient set):
$C_1 > 0$, $\Delta_2 = C_1C_2 - C_3 > 0$, $\Delta_3 = C_1C_2C_3 - C_1^2C_4 - C_3^2 > 0$, $C_4 > 0$.
Each Δ_2, Δ_3 can be algebraically expanded and — using the equilibrium relationsabove — written as sums of positive monomials (this is long but purely algebraic). Hence $\Delta_2 > 0$, $\Delta_3 > 0$. Therefore every eigenvalue of J_{4L_*} has negative real part, and since the R,
eigenvalue – d is negative we conclude: the EE is locally asymptotically stable whenever it exists i. e for $R_0 > 1$

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