

Mathematical Analysis of a Stochastic SEIARV Model for COVID-19 Disease Transmission with Intervention

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Abstract.

This study presents a stochastic SEIARV model for analysing COVID-19 transmission and vaccination intervention. The model incorporates symptomatic and asymptomatic infectious individuals, demographic recruitment and mortality, disease-induced mortality, incomplete vaccine protection, and stochastic fluctuations in epidemic dynamics. The basic reproduction number, R_0 , is derived using the next-generation matrix approach, and the disease-free and endemic equilibria are analysed. The stochastic model is solved numerically using the Euler–Maruyama method, and multiple sample paths are generated to examine uncertainty in epidemic trajectories. Numerical results show that vaccination reduces both the reproduction number and the peak infectious population. In particular, increasing the vaccination rate from the baseline level to five times its value reduces R_0 below unity and substantially decreases the epidemic peak. The results demonstrate the importance of vaccination in reducing COVID-19 transmission and highlight the value of stochastic modelling in capturing variability and uncertainty in epidemic outcomes.

Keywords: stochastics; SEIARV; intervention; COVID-19; modelling

1. Introduction

Coronavirus disease 2019 (COVID-19), caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), has stimulated extensive mathematical research aimed at understanding disease transmission and evaluating effective control strategies. Mathematical epidemic models provide a useful framework for describing disease progression, identifying transmission thresholds, and assessing interventions such as vaccination. Compartmental models are particularly useful because they divide a population into epidemiological classes and provide a systematic framework for analysing disease dynamics [1–3].

Vaccination has remained an important component of mathematical models of COVID-19. Several studies have demonstrated that vaccination can substantially reduce infection, hospitalisation, mortality, and the overall burden of COVID-19 [2–5]. However, vaccine protection may be incomplete and may decline over time. Consequently, models that explicitly distinguish vaccinated individuals from susceptible individuals and account for reduced, rather than complete, vaccine protection can provide a more realistic representation of transmission dynamics [3,4].

Another important consideration in COVID-19 modelling is the distinction between symptomatic and asymptomatic infections. Asymptomatic individuals may contribute to transmission without being readily detected, while symptomatic individuals may have different infectiousness and disease outcomes. Recent studies have therefore incorporated separate symptomatic and asymptomatic infectious classes, together with vaccination and other forms of population heterogeneity, to improve the representation of COVID-19 transmission [5–7]. Wang et al. [9], in particular, developed

a stochastic COVID-19 model incorporating vaccination classes and separate symptomatic and asymptomatic infectious populations, demonstrating the importance of accounting for stochastic effects and vaccination in epidemic modelling.

Although deterministic epidemic models are useful for describing average disease behaviour, COVID-19 transmission is influenced by random variations in human behaviour, contact patterns, mobility, environmental conditions, and other epidemiological factors. These sources of variability can produce substantial differences between epidemic trajectories even when the underlying model parameters are unchanged. Stochastic epidemic models address this limitation by incorporating random perturbations into the disease dynamics and consequently provide a framework for examining uncertainty and variability in epidemic outcomes [8–10].

Motivated by these developments, this study formulates a stochastic susceptible–exposed–infectious–symptomatic–asymptomatic–recovered–vaccinated (SEIARV) model for COVID-19 transmission. The model incorporates demographic recruitment and natural mortality, infection, progression from exposure to infectiousness, separate symptomatic and asymptomatic infectious classes, recovery, disease-induced mortality, vaccination, incomplete vaccine protection, and stochastic perturbations. Both symptomatic and asymptomatic individuals contribute to the force of infection, while vaccinated individuals retain a reduced susceptibility to infection because vaccine protection is assumed to be incomplete.

The mathematical analysis determines the basic reproduction number, (R_0), and investigates the disease-free and endemic equilibria together with their stability properties. The stochastic system is solved numerically using the Euler–Maruyama method. Multiple stochastic sample paths are generated to examine the effects of random fluctuations on the trajectories of the epidemiological compartments. Sensitivity analysis is conducted to identify parameters that have substantial effects on important model outcomes, while uncertainty analysis is used to assess the influence of parameter variability on epidemic behaviour.

The novel contribution of this study lies in the development of a stochastic SEIARV framework that extends existing COVID-19 models by incorporating incomplete vaccine protection while explicitly representing symptomatic and asymptomatic infectious populations. Unlike the model of Wang et al. [9], the present study combines reproduction-number and equilibrium analysis with Euler–Maruyama numerical simulation, sensitivity and uncertainty analyses, and the generation of multiple stochastic sample paths to examine the effects of random fluctuations on epidemic trajectories. Furthermore, the use of a synthetic monthly dataset provides a controlled computational framework for investigating the interaction between vaccination, disease transmission, and stochastic variability. The model therefore provides an integrated framework for assessing how incomplete vaccination protection and stochastic effects influence COVID-19 transmission and epidemic burden.

Since the numerical investigation is based on a synthetic dataset, the results are interpreted as simulation-based findings rather than direct empirical estimates of COVID-19 transmission in a specific population. Consequently, calibration and validation using observed epidemiological and vaccination data would be required before the model could be applied to population-specific forecasting or policy evaluation.

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stochastic effects influence COVID-19 transmission and epidemic burden.

2. Model Formulation

This section presents the stochastic Susceptible–Exposed–Infectious–Asymptomatic–Recovered–Vaccinated (SEIARV) model developed to describe the transmission dynamics of COVID-19 in a population subject to vaccination and demographic processes. The formulation distinguishes between symptomatic and asymptomatic infectious individuals and incorporates random perturbations to represent unobserved environmental, behavioural, and epidemiological variations. The inclusion of vaccination provides a framework for evaluating the effect of preventive intervention on disease transmission.

Recent epidemiological studies have shown that mathematical and stochastic models remain important tools for understanding infectious disease transmission, evaluating intervention strategies, and investigating uncertainty in epidemic outcomes [1–4]. In particular, vaccination and other non-pharmaceutical interventions can substantially modify transmission dynamics, while stochastic effects may become important when epidemic conditions vary over time [5–7].

2.1. Population Compartments

The total population is divided into six epidemiological compartments:

$$N(t) = S(t) + E(t) + I(t) + A(t) + R(t) + V(t), \quad (1)$$

where $S(t)$ represents the susceptible population, $E(t)$ represents individuals exposed to the infection but not yet infectious, $I(t)$ represents symptomatic infectious individuals, $A(t)$ represents asymptomatic infectious individuals, $R(t)$ represents recovered individuals, and $V(t)$ represents vaccinated individuals.

The model assumes that individuals enter the population at rate Λ and experience natural mortality at rate μ . Susceptible individuals may become infected through contact with symptomatic or asymptomatic infectious individuals. Vaccination transfers susceptible individuals into the vaccinated compartment at rate ν . Vaccine protection is represented by the parameter ϵ , where $0 \leq \epsilon \leq 1$.

2.2. Force of Infection

The force of infection is defined by

$$\lambda(t) = \frac{\beta_H [I(t) + \eta A(t)]}{N(t)}, \quad (2)$$

where β_H is the effective transmission rate and η is the relative infectiousness of asymptomatic individuals.

The parameter η accounts for the possibility that asymptomatic individuals may transmit infection at a different rate from symptomatic individuals. When $0 < \eta < 1$, asymptomatic individuals are assumed to be less infectious than symptomatic individuals. This formulation is consistent with epidemic models in which infectious individuals with different clinical characteristics contribute differently to transmission [2,4].

2.3. Deterministic SEIARV Model

Before introducing stochastic perturbations, the deterministic component of the model is expressed as

$$\frac{dS}{dt} = \Lambda - \lambda S - \nu S - \mu S, \quad (3)$$

$$\frac{dE}{dt} = \lambda S + (1 - \epsilon)\lambda V - (\sigma + \mu)E, \quad (4)$$

$$\frac{dI}{dt} = p\sigma E - (\gamma_I + \alpha_I + \mu)I, \quad (5)$$

$$\frac{dA}{dt} = (1 - p)\sigma E - (\gamma_A + \mu)A, \quad (6)$$

$$\frac{dR}{dt} = \gamma_I I + \gamma_A A - \mu R, \quad (7)$$

$$\frac{dV}{dt} = \nu S - (1 - \epsilon)\lambda V - \mu V. \quad (8)$$

Here, σ denotes the rate at which exposed individuals leave the latent stage. A proportion p of exposed individuals progress to the symptomatic infectious class, while the remaining proportion $1 - p$ enter the asymptomatic infectious class. The parameters γ_I and γ_A represent recovery rates for symptomatic and asymptomatic infectious individuals, respectively, while α_I represents disease-induced mortality among symptomatic infectious individuals.

The term

$$(1 - \epsilon)\lambda V \quad (9)$$

represents infection among vaccinated individuals who are not completely protected. Thus, when $\epsilon = 1$, vaccination provides complete protection against infection, whereas $\epsilon = 0$ corresponds to no vaccine protection.

2.4. Stochastic SEIARV Model

Epidemic transmission is influenced by random factors including changes in contact behaviour, environmental conditions, reporting variations, population mobility, and other unobserved sources of variability. To account for such effects, independent stochastic perturbations are incorporated into the six model compartments.

The stochastic SEIARV model is therefore given by

$$dS = (\Lambda - \lambda S - \nu S - \mu S) dt + \sigma_1 S dW_1(t), \quad (10)$$

$$dE = [\lambda S + (1 - \epsilon)\lambda V - (\sigma + \mu)E] dt + \sigma_2 E dW_2(t), \quad (11)$$

$$dI = [p\sigma E - (\gamma_I + \alpha_I + \mu)I] dt + \sigma_3 I dW_3(t), \quad (12)$$

$$dA = [(1 - p)\sigma E - (\gamma_A + \mu)A] dt + \sigma_4 A dW_4(t), \quad (13)$$

$$dR = [\gamma_I I + \gamma_A A - \mu R] dt + \sigma_5 R dW_5(t), \quad (14)$$

$$dV = [\nu S - (1 - \epsilon)\lambda V - \mu V] dt + \sigma_6 V dW_6(t), \quad (15)$$

where $W_1(t), \dots, W_6(t)$ are independent standard Brownian motions and $\sigma_1, \dots, \sigma_6$ are the corresponding stochastic intensity parameters.

The stochastic terms allow the model trajectories to deviate from their deterministic counterparts. In particular, the magnitude of σ_i determines the intensity of random fluctuations in the corresponding epidemiological compartment. This formulation is useful for representing uncertainty and temporal variability that cannot be adequately captured by a purely deterministic model [3,6,7].

2.5. Model Parameters

The principal parameters of the model are summarized as follows:

- Λ : recruitment or population-entry rate;
- β_H : effective disease transmission rate;
- η : relative infectiousness of asymptomatic individuals;
- σ : progression rate from the exposed to infectious classes;
- p : proportion of exposed individuals developing symptomatic infection;
- γ_I : recovery rate of symptomatic infectious individuals;
- γ_A : recovery rate of asymptomatic infectious individuals;
- α_I : disease-induced mortality rate;
- μ : natural mortality rate;
- ν : vaccination rate;
- ϵ : vaccine effectiveness;
- $\sigma_1, \dots, \sigma_6$: stochastic intensity parameters associated with the six epidemiological compartments.

For the numerical investigation, the parameter values used in the simulations were

$$\begin{aligned}\beta_H &= 0.4600000142, & \nu &= 0.0036884715, \\ \sigma_1 &= 0.0773018868, & \sigma_2 &= 0.0175675179, \\ \sigma_3 &= 0.0243317982, & \sigma_4 &= 0.0222895683, \\ \sigma_5 &= 0.0145873353, & \sigma_6 &= 0.0162260104.\end{aligned}$$

The remaining parameters were specified as

$$\begin{aligned}\Lambda &= 18, & \eta &= 0.50, & \sigma &= \frac{1}{5.2}, & p &= 0.70, \\ \gamma_I &= \frac{1}{10}, & \gamma_A &= \frac{1}{7}, & \alpha_I &= 0.0015, \\ \mu &= \frac{1}{70 \times 12}, & \epsilon &= 0.80.\end{aligned}$$

The initial population was specified by

$$\begin{aligned}S(0) &= 9800, & E(0) &= 80, & I(0) &= 50, \\ A(0) &= 40, & R(0) &= 30, & V(0) &= 0.\end{aligned}$$

Consequently,

$$N(0) = 10000.$$

2.6. Total Population Dynamics

Adding equations (3) – (8) gives the total population equation

$$\frac{dN}{dt} = \Lambda - \mu N - \alpha_I I. \quad (16)$$

Thus, population change is determined by recruitment, natural mortality, and disease-induced mortality. Vaccination and disease transmission redistribute individuals among epidemiological compartments but do not directly alter the total population.

For the stochastic formulation, summing equations (10)–(15) yields

$$dN = (\Lambda - \mu N - \alpha_I I) dt + \sum_{j=1}^6 \sigma_j X_j dW_j(t), \quad (17)$$

where

$$(X_1, X_2, X_3, X_4, X_5, X_6) = (S, E, I, A, R, V).$$

Equation (17) demonstrates that the total population is also subject to random fluctuations arising from the stochastic perturbations in the individual compartments.

2.7. Numerical Solution

The stochastic system is solved numerically using the Euler–Maruyama method. For a time step Δt , the general discretization of a stochastic differential equation

$$dX(t) = f(X(t))dt + g(X(t))dW(t)$$

is

$$X_{n+1} = X_n + f(X_n)\Delta t + g(X_n)\sqrt{\Delta t} Z_n, \quad (18)$$

where Z_n is a standard normally distributed random variable.

Accordingly, the stochastic SEIARV equations are simulated over the period January 2020 to January 2026 using a monthly time step. Multiple stochastic sample paths are generated to investigate the variability of epidemic trajectories. The numerical implementation was developed MATLAB, and the resulting trajectories are used in the simulation and sensitivity analyses presented in the results section.

3. Mathematical Analysis

This section establishes the principal mathematical properties of the proposed stochastic SEIARV model. The analysis focuses on positivity and boundedness of the epidemiological variables, the disease-free equilibrium, derivation of the basic reproduction number, local stability of the disease-free equilibrium, existence and uniqueness of the stochastic solution, and the numerical approximation of the stochastic system. These properties provide the mathematical foundation for the numerical simulations and intervention analysis presented in Section 4.

3.1. Positivity and Boundedness of Solutions

Since the variables of the SEIARV model represent population classes, biologically meaningful solutions must remain non-negative for all $t \geq 0$. Consider the deterministic system given by equations (3)–(8). We establish that solutions originating from non-negative initial conditions remain in the non-negative region.

For the susceptible population,

$$\frac{dS}{dt} = \Lambda - \lambda S - \nu S - \mu S. \quad (19)$$

At the boundary $S = 0$,

$$\left. \frac{dS}{dt} \right|_{S=0} = \Lambda \geq 0. \quad (20)$$

Therefore, the vector field does not point outside the non-negative region through the S -boundary.

Similarly, for the exposed population,

$$\frac{dE}{dt} = \lambda S + (1 - \epsilon)\lambda V - (\sigma + \mu)E. \quad (21)$$

At $E = 0$,

$$\left. \frac{dE}{dt} \right|_{E=0} = \lambda S + (1 - \epsilon)\lambda V \geq 0. \quad (22)$$

Thus, the exposed population cannot become negative.

For the symptomatic infectious population,

$$\frac{dI}{dt} = p\sigma E - (\gamma_I + \alpha_I + \mu)I. \quad (23)$$

At $I = 0$,

$$\left. \frac{dI}{dt} \right|_{I=0} = p\sigma E \geq 0. \quad (24)$$

Likewise, for the asymptomatic infectious population,

$$\frac{dA}{dt} = (1-p)\sigma E - (\gamma_A + \mu)A, \quad (25)$$

and hence

$$\left. \frac{dA}{dt} \right|_{A=0} = (1-p)\sigma E \geq 0. \quad (26)$$

For the recovered population,

$$\frac{dR}{dt} = \gamma_I I + \gamma_A A - \mu R, \quad (27)$$

which gives

$$\left. \frac{dR}{dt} \right|_{R=0} = \gamma_I I + \gamma_A A \geq 0. \quad (28)$$

Finally, for the vaccinated population,

$$\frac{dV}{dt} = \nu S - (1-\epsilon)\lambda V - \mu V, \quad (29)$$

and therefore

$$\left. \frac{dV}{dt} \right|_{V=0} = \nu S \geq 0. \quad (30)$$

Consequently, if

$$S(0), E(0), I(0), A(0), R(0), V(0) \geq 0, \quad (31)$$

then the deterministic solution remains in the non-negative region for all $t \geq 0$.

The total population is given by

$$N(t) = S(t) + E(t) + I(t) + A(t) + R(t) + V(t). \quad (32)$$

Adding the six deterministic equations gives

$$\frac{dN}{dt} = \Lambda - \mu N - \alpha_I I. \quad (33)$$

Since $I(t) \geq 0$,

$$\frac{dN}{dt} \leq \Lambda - \mu N. \quad (34)$$

The comparison equation

$$\frac{dY}{dt} = \Lambda - \mu Y \quad (35)$$

has solution

$$Y(t) = Y(0)e^{-\mu t} + \frac{\Lambda}{\mu} (1 - e^{-\mu t}). \quad (36)$$

It follows that

$$N(t) \leq N(0)e^{-\mu t} + \frac{\Lambda}{\mu} (1 - e^{-\mu t}). \quad (37)$$

Consequently,

$$N(t) \leq \max \left\{ N(0), \frac{\Lambda}{\mu} \right\}. \quad (38)$$

Thus, the feasible region

$$\Omega = \left\{ (S, E, I, A, R, V) \in \mathbb{R}_+^6 : N \leq \max \left(N(0), \frac{\Lambda}{\mu} \right) \right\} \quad (39)$$

is positively invariant for the deterministic model.

This result is epidemiologically important because it establishes that the model produces physically meaningful population trajectories and that the total population remains bounded.

3.2. Disease-Free Equilibrium

The disease-free equilibrium (DFE) corresponds to a state in which there are no exposed or infectious individuals. Hence,

$$E^* = I^* = A^* = 0. \quad (40)$$

At the disease-free state, the susceptible and vaccinated populations satisfy

$$0 = \Lambda - \nu S^* - \mu S^* \quad (41)$$

and

$$0 = \nu S^* - \mu V^*. \quad (42)$$

Therefore,

$$S^* = \frac{\Lambda}{\mu + \nu}, \quad (43)$$

and

$$V^* = \frac{\nu \Lambda}{\mu(\mu + \nu)}. \quad (44)$$

Since there is no infection at the disease-free state,

$$R^* = 0. \quad (45)$$

Consequently, the disease-free equilibrium is

$$\mathcal{E}_0 = \left(\frac{\Lambda}{\mu + \nu}, 0, 0, 0, 0, \frac{\nu \Lambda}{\mu(\mu + \nu)} \right). \quad (46)$$

The corresponding total population at the disease-free equilibrium is

$$N^* = S^* + V^*. \quad (47)$$

Substitution gives

$$N^* = \frac{\Lambda}{\mu + \nu} + \frac{\nu \Lambda}{\mu(\mu + \nu)}. \quad (48)$$

Hence,

$$N^* = \frac{\Lambda}{\mu}. \quad (49)$$

The disease-free equilibrium therefore represents a demographic balance between recruitment, natural mortality, and vaccination in the absence of infection.

3.3. Basic Reproduction Number

The basic reproduction number R_0 is derived using the next-generation-matrix approach. The infected subsystem consists of the exposed, symptomatic infectious, and asymptomatic infectious compartments,

$$x = \begin{pmatrix} E \\ I \\ A \end{pmatrix}. \quad (50)$$

The new-infection terms are

$$\mathcal{F} = \begin{pmatrix} \lambda S + (1 - \epsilon)\lambda V \\ 0 \\ 0 \end{pmatrix}. \quad (51)$$

At the disease-free equilibrium,

$$S^* = \frac{\Lambda}{\mu + \nu}, \quad V^* = \frac{\nu\Lambda}{\mu(\mu + \nu)}, \quad (52)$$

and

$$N^* = \frac{\Lambda}{\mu}. \quad (53)$$

The effective susceptible fraction at the disease-free equilibrium is

$$q = \frac{S^* + (1 - \epsilon)V^*}{N^*}. \quad (54)$$

Substitution of the equilibrium values yields

$$q = \frac{\mu + (1 - \epsilon)\nu}{\mu + \nu}. \quad (55)$$

The transmission contribution is consequently

$$\mathcal{F} = \begin{pmatrix} \beta_H q(I + \eta A) \\ 0 \\ 0 \end{pmatrix}. \quad (56)$$

The transition terms are

$$\mathcal{V} = \begin{pmatrix} (\sigma + \mu)E \\ (\gamma_I + \alpha_I + \mu)I - p\sigma E \\ (\gamma_A + \mu)A - (1 - p)\sigma E \end{pmatrix}. \quad (57)$$

The Jacobian matrices of $\mathcal{F}$ and $\mathcal{V}$ evaluated at the disease-free equilibrium are

$$F = \begin{pmatrix} 0 & \beta_H q & \beta_H \eta q \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \quad (58)$$

and

$$V = \begin{pmatrix} \sigma + \mu & 0 & 0 \\ -p\sigma & \gamma_I + \alpha_I + \mu & 0 \\ -(1 - p)\sigma & 0 & \gamma_A + \mu \end{pmatrix}. \quad (59)$$

The next-generation matrix is

$$K = FV^{-1}. \quad (60)$$

The spectral radius of K gives the basic reproduction number. After simplification,

$$R_0 = \frac{\beta_H \sigma q}{\sigma + \mu} \left[\frac{p}{\gamma_I + \alpha_I + \mu} + \frac{(1-p)\eta}{\gamma_A + \mu} \right]. \quad (61)$$

Using equation (55), this can also be written as

$$R_0 = \frac{\beta_H \sigma}{\sigma + \mu} \cdot \frac{\mu + (1-\epsilon)\nu}{\mu + \nu} \left[\frac{p}{\gamma_I + \alpha_I + \mu} + \frac{(1-p)\eta}{\gamma_A + \mu} \right]. \quad (62)$$

The expression demonstrates that vaccination affects disease transmission through the factor

$$\frac{\mu + (1-\epsilon)\nu}{\mu + \nu}. \quad (63)$$

Thus, increasing the vaccination rate ν or increasing vaccine effectiveness ϵ reduces the fraction of the population effectively susceptible to infection.

The two terms inside the square brackets represent the contributions of symptomatic and asymptomatic infectious individuals. The first term,

$$\frac{p}{\gamma_I + \alpha_I + \mu}, \quad (64)$$

represents transmission through symptomatic individuals, whereas

$$\frac{(1-p)\eta}{\gamma_A + \mu} \quad (65)$$

represents transmission through asymptomatic individuals.

Therefore, the model explicitly incorporates both symptomatic and asymptomatic transmission in the threshold quantity.

3.4. Local Stability of the Disease-Free Equilibrium

The local stability of the disease-free equilibrium is determined by the eigenvalues of the Jacobian matrix associated with the infected subsystem. The infected subsystem near the disease-free equilibrium is

$$\frac{dE}{dt} = \beta_H q(I + \eta A) - (\sigma + \mu)E, \quad (66)$$

$$\frac{dI}{dt} = p\sigma E - (\gamma_I + \alpha_I + \mu)I, \quad (67)$$

$$\frac{dA}{dt} = (1-p)\sigma E - (\gamma_A + \mu)A. \quad (68)$$

The Jacobian matrix is therefore

$$J_I = \begin{pmatrix} -(\sigma + \mu) & \beta_H q & \beta_H \eta q \\ p\sigma & -(\gamma_I + \alpha_I + \mu) & 0 \\ (1-p)\sigma & 0 & -(\gamma_A + \mu) \end{pmatrix}. \quad (69)$$

The disease-free equilibrium is locally asymptotically stable when all eigenvalues of J_I have negative real parts. According to the next-generation-matrix criterion, this condition is equivalent to

$$\rho(FV^{-1}) < 1, \quad (70)$$

where $\rho(\cdot)$ denotes the spectral radius. Since

$$\rho(FV^{-1}) = R_0, \quad (71)$$

the disease-free equilibrium is locally asymptotically stable whenever

$$R_0 < 1. \quad (72)$$

Conversely, when

$$R_0 > 1, \quad (73)$$

the disease-free equilibrium loses stability and infection invasion becomes possible.

The threshold condition

$$R_0 = 1 \quad (74)$$

represents the critical epidemiological boundary. Therefore, intervention strategies that reduce R_0 below unity can theoretically suppress sustained disease transmission.

For the proposed model, reductions in R_0 can be achieved through increases in vaccination coverage and vaccine effectiveness, reductions in the effective transmission parameter β_H , reductions in the relative infectiousness η of asymptomatic individuals, and improvements in recovery rates.

3.5. Existence and Uniqueness of the Stochastic Solution

Consider the stochastic SEIARV system in the vector form

$$dX(t) = F(X(t)) dt + G(X(t)) dW(t), \quad (75)$$

where

$$X(t) = (S(t), E(t), I(t), A(t), R(t), V(t))^T. \quad (76)$$

The drift vector $F(X)$ is defined by the deterministic components of equations (10)–(15), while the diffusion matrix is

$$G(X) = \begin{pmatrix} \sigma_1 S & 0 & 0 & 0 & 0 & 0 \\ 0 & \sigma_2 E & 0 & 0 & 0 & 0 \\ 0 & 0 & \sigma_3 I & 0 & 0 & 0 \\ 0 & 0 & 0 & \sigma_4 A & 0 & 0 \\ 0 & 0 & 0 & 0 & \sigma_5 R & 0 \\ 0 & 0 & 0 & 0 & 0 & \sigma_6 V \end{pmatrix}. \quad (77)$$

The force of infection is

$$\lambda(X) = \frac{\beta_H(I + \eta A)}{S + E + I + A + R + V}. \quad (78)$$

On the biologically relevant region where the total population is positive, the function $\lambda(X)$ is locally Lipschitz. Consequently, the drift and diffusion functions are locally Lipschitz on the feasible state space.

Furthermore, the diffusion coefficients satisfy a linear-growth condition of the form

$$|G(X)|^2 \leq C(1 + |X|^2) \quad (79)$$

for some positive constant C . The drift term also satisfies an analogous linear-growth bound on the biologically feasible region.

Under these conditions, standard results for stochastic differential equations imply that for every non-negative initial condition there exists a unique local strong solution to the stochastic SEIARV

system. Together with the boundedness properties established above and the non-negative numerical treatment used in the simulations, this provides the mathematical basis for the stochastic simulation framework.

The multiplicative form of the stochastic terms is particularly appropriate for epidemiological modelling because random fluctuations increase with the size of the corresponding population class. For example, the stochastic contribution to the symptomatic infectious class is

$$\sigma_3 I(t) dW_3(t), \quad (80)$$

which becomes zero when $I(t) = 0$ and increases in magnitude as the symptomatic infectious population increases.

3.6. Euler–Maruyama Numerical Approximation

Analytical solutions of the nonlinear stochastic SEIARV system are generally not available. Numerical simulation is therefore employed to investigate the temporal behaviour of the model.

Let the time interval be divided into equally spaced points

$$t_n = n\Delta t, \quad n = 0, 1, \dots, M, \quad (81)$$

where

$$\Delta t = \frac{T}{M}. \quad (82)$$

For each Brownian motion,

$$\Delta W_{j,n} = W_j(t_{n+1}) - W_j(t_n) \quad (83)$$

satisfies

$$\Delta W_{j,n} = \sqrt{\Delta t} Z_{j,n}, \quad (84)$$

where

$$Z_{j,n} \sim N(0, 1) \quad (85)$$

are independent standard normal random variables.

The Euler–Maruyama approximation for the susceptible population is

$$S_{n+1} = S_n + [\Lambda - \lambda_n S_n - \nu S_n - \mu S_n] \Delta t + \sigma_1 S_n \sqrt{\Delta t} Z_{1,n}. \quad (86)$$

Similarly, the exposed population is approximated by

$$E_{n+1} = E_n + [\lambda_n S_n + (1 - \epsilon)\lambda_n V_n - (\sigma + \mu)E_n] \Delta t + \sigma_2 E_n \sqrt{\Delta t} Z_{2,n}. \quad (87)$$

The symptomatic infectious population is approximated by

$$I_{n+1} = I_n + [p\sigma E_n - (\gamma_I + \alpha_I + \mu)I_n] \Delta t + \sigma_3 I_n \sqrt{\Delta t} Z_{3,n}. \quad (88)$$

The asymptomatic infectious population satisfies

$$A_{n+1} = A_n + [(1 - p)\sigma E_n - (\gamma_A + \mu)A_n] \Delta t + \sigma_4 A_n \sqrt{\Delta t} Z_{4,n}. \quad (89)$$

The recovered population is approximated by

$$R_{n+1} = R_n + [\gamma_I I_n + \gamma_A A_n - \mu R_n] \Delta t + \sigma_5 R_n \sqrt{\Delta t} Z_{5,n}. \quad (90)$$

Finally, the vaccinated population is approximated by

$$V_{n+1} = V_n + [\nu S_n - (1 - \epsilon)\lambda_n V_n - \mu V_n] \Delta t + \sigma_6 V_n \sqrt{\Delta t} Z_{6,n}. \quad (91)$$

At each time step, the force of infection is evaluated as

$$\lambda_n = \frac{\beta_H(I_n + \eta A_n)}{S_n + E_n + I_n + A_n + R_n + V_n}. \quad (92)$$

For numerical stability and biological consistency, the implementation applies a non-negativity correction after each time step:

$$X_{n+1} = \max(X_{n+1}, 0), \quad (93)$$

for each epidemiological compartment.

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4. Numerical Results

This section presents the numerical simulation of the stochastic SEIARV model using the parameter values and initial conditions specified in Section 2.4. The results include the deterministic trajectories of the six epidemiological compartments, an ensemble of stochastic sample paths, the resulting epidemic indicators, and a comparison of vaccination scenarios that illustrates the effect of the intervention on transmission dynamics.

4.1. Simulation Setup

The deterministic system (3)–(8) was integrated using a fourth-order Runge–Kutta scheme with a fine internal step size, sampled at monthly intervals for comparison with the stochastic simulations. The stochastic system (10)–(15) was simulated using the Euler–Maruyama scheme of Section 3.8, with a monthly step size $\Delta t = 1$, consistent with the monthly reporting interval described in Section 2. Both systems were simulated over 72 time steps, corresponding to the period January 2020 to January 2026, from the initial condition $(S(0), E(0), I(0), A(0), R(0), V(0)) = (9800, 80, 50, 40, 30, 0)$. For the stochastic model, an ensemble of $n = 30$ independent sample paths was generated using independent standard normal increments for each of the six Wiener processes $W_1, \dots, W_6$, with a non-negativity correction applied at every step as described by the projection in Section 3.8.

4.2. Deterministic versus Stochastic Trajectories

Figure 1 shows the deterministic trajectories of the six epidemiological compartments under the baseline parameter values, including the vaccination rate $\nu = 0.0036884715$. The susceptible population declines steadily as individuals move into the exposed, vaccinated, and (following infection) recovered classes, while the exposed, symptomatic, and asymptomatic populations rise to a single epidemic peak before declining as the epidemic progresses.

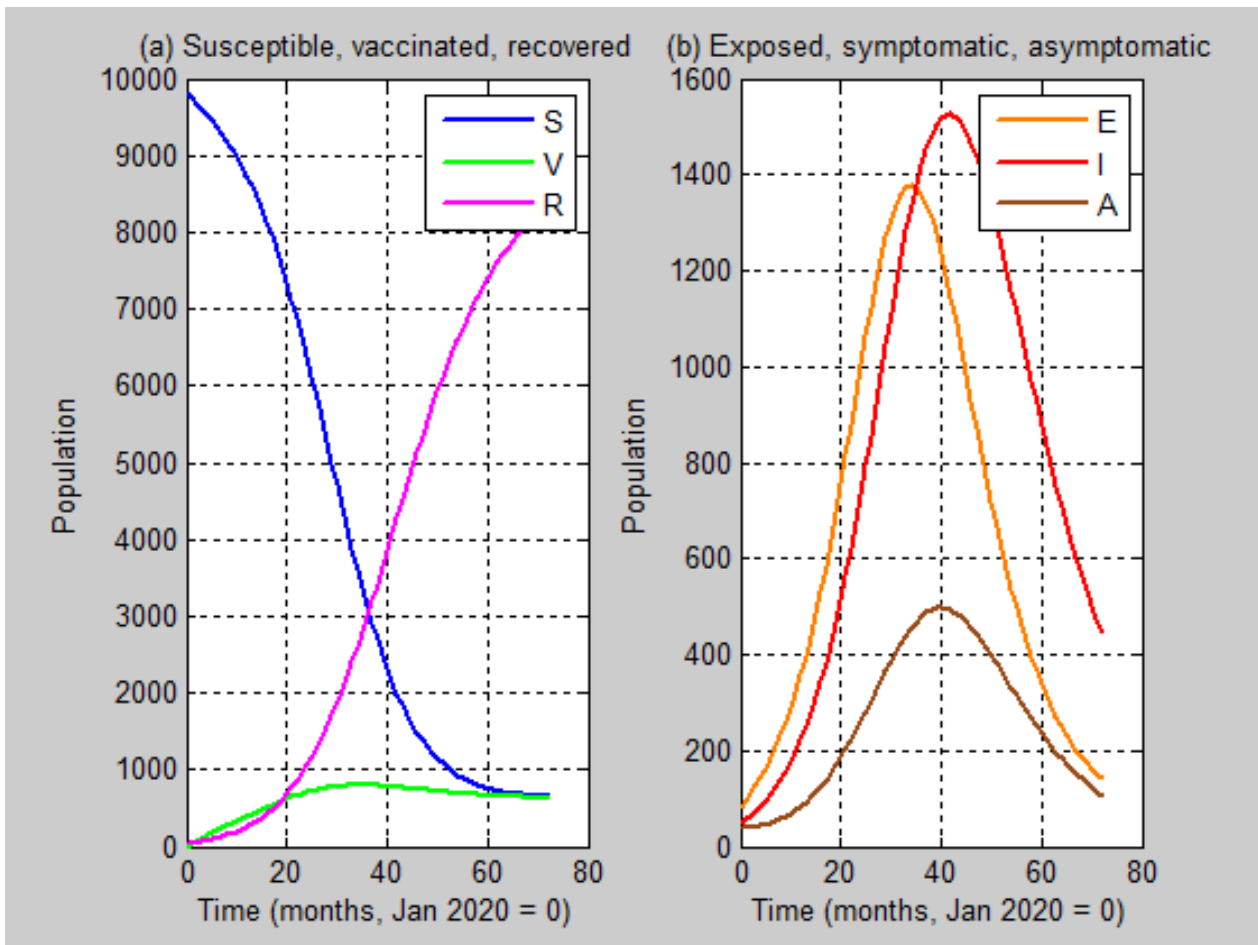


Figure 1: Deterministic trajectories of the SEIARV compartments under the baseline parameter values. (a) Susceptible, vaccinated, and recovered populations. (b) Exposed, symptomatic, and asymptomatic infectious populations.

Figure 2 compares the deterministic trajectory of the total infectious population $I(t) + A(t)$ with an ensemble of 30 stochastic sample paths generated under the same baseline parameters. The stochastic trajectories fluctuate around the deterministic curve, with the largest dispersion occurring near the epidemic peak, consistent with the multiplicative structure of the noise terms in equations (12) and (13), whose magnitude scales with the size of the infectious compartments. Across the ensemble, the peak value of $I(t) + A(t)$ has mean 2020.55 and standard deviation 752.23, with individual sample paths attaining peak values ranging from approximately 1128 to 4054. This variability illustrates that a single deterministic trajectory can substantially understate the range of epidemic outcomes that are plausible under environmental and demographic randomness.

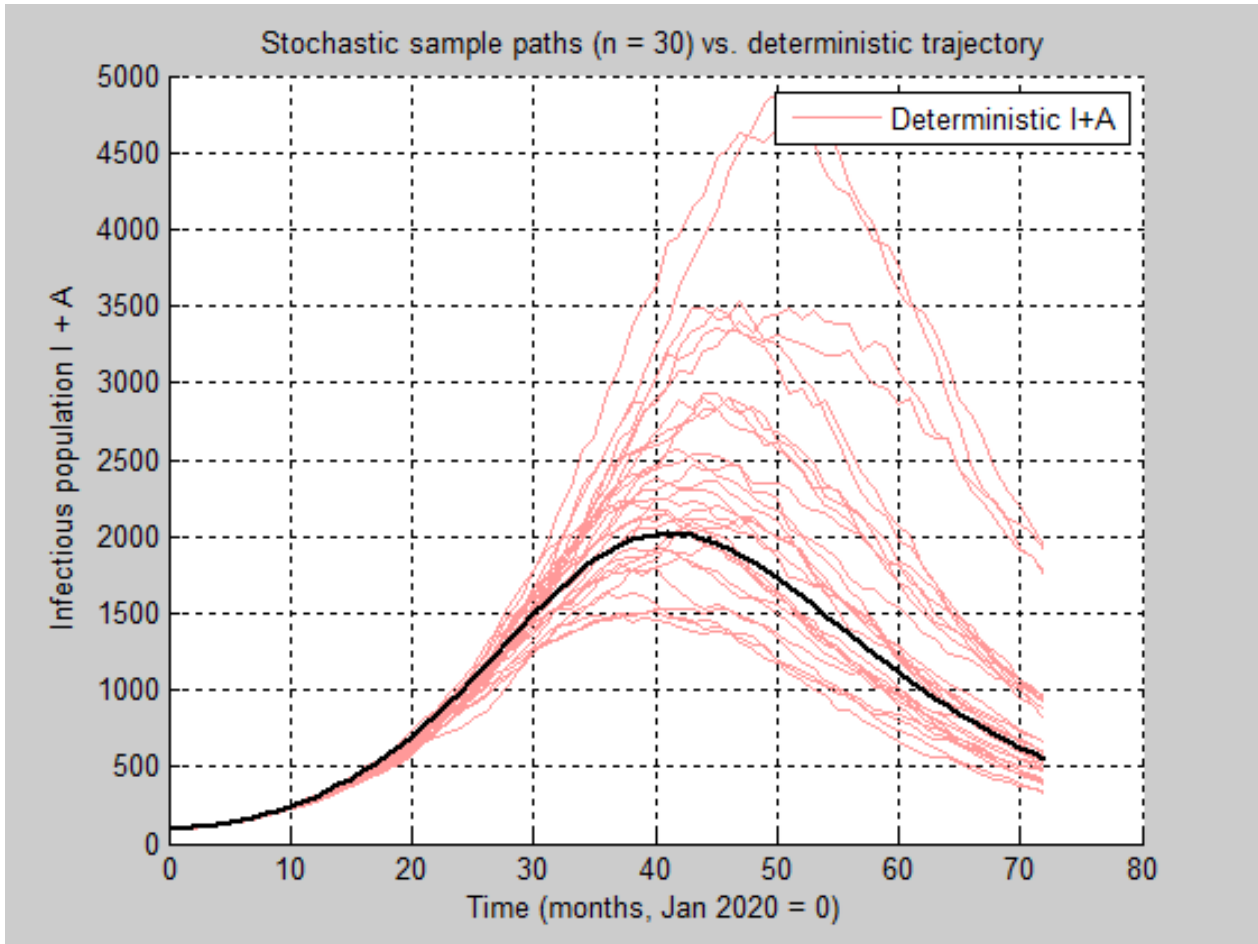


Figure 2: Stochastic sample paths ($n = 30$) of the total infectious population $I(t) + A(t)$ compared with the deterministic trajectory, under the baseline vaccination rate.

4.3. Epidemic Indicators

Table 1 summarizes the principal epidemic indicators obtained from the deterministic simulation under the baseline vaccination rate. The basic reproduction number computed from equation (62) using the baseline parameter values is $R_0 \approx 1.42$, confirming that the disease-free equilibrium is unstable and that the epidemic is expected to invade the population, consistent with the results of Section 3.3–3.6.

Table 1: Epidemic indicators under the baseline vaccination rate ($\nu = 0.0036884715$, $R_0 \approx 1.42$).

Indicator	Value
Peak infectious population, $I + A$	2020.62
Month of peak infectious population	Month 41
Final infectious population, $I(72) + A(72)$	553.63
Final vaccinated population, $V(72)$	636.15
Final recovered population, $R(72)$	8341.73

The epidemic peak occurs at month 41 (approximately mid-2023 within the simulated horizon), after which the infectious population declines gradually. By the end of the 72-month simulation window, the infectious population has fallen substantially from its peak but has not yet reached

zero, and the recovered population accounts for the majority of the total population, reflecting the cumulative burden of transmission over the simulated period.

4.4. Effect of Vaccination on Transmission Dynamics

To evaluate the effect of the vaccination intervention, three scenarios were compared: (i) no vaccination ($\nu = 0$), (ii) the baseline vaccination rate ($\nu = 0.0036884715$), and (iii) an accelerated vaccination rate five times the baseline value ($\nu = 0.0184423575$). Table 2 reports the basic reproduction number and the peak infectious population obtained under each scenario, and Figure 3 shows the corresponding trajectories of the infectious population and the vaccinated population.

Table 2: Effect of the vaccination rate ν on the basic reproduction number and peak infectious population.

Scenario	ν	R_0	Peak ($I + A$)
No vaccination	0	3.59	2267.20
Baseline vaccination	0.0036884715	1.42	2020.62
Accelerated vaccination	0.0184423575	0.89	1251.94

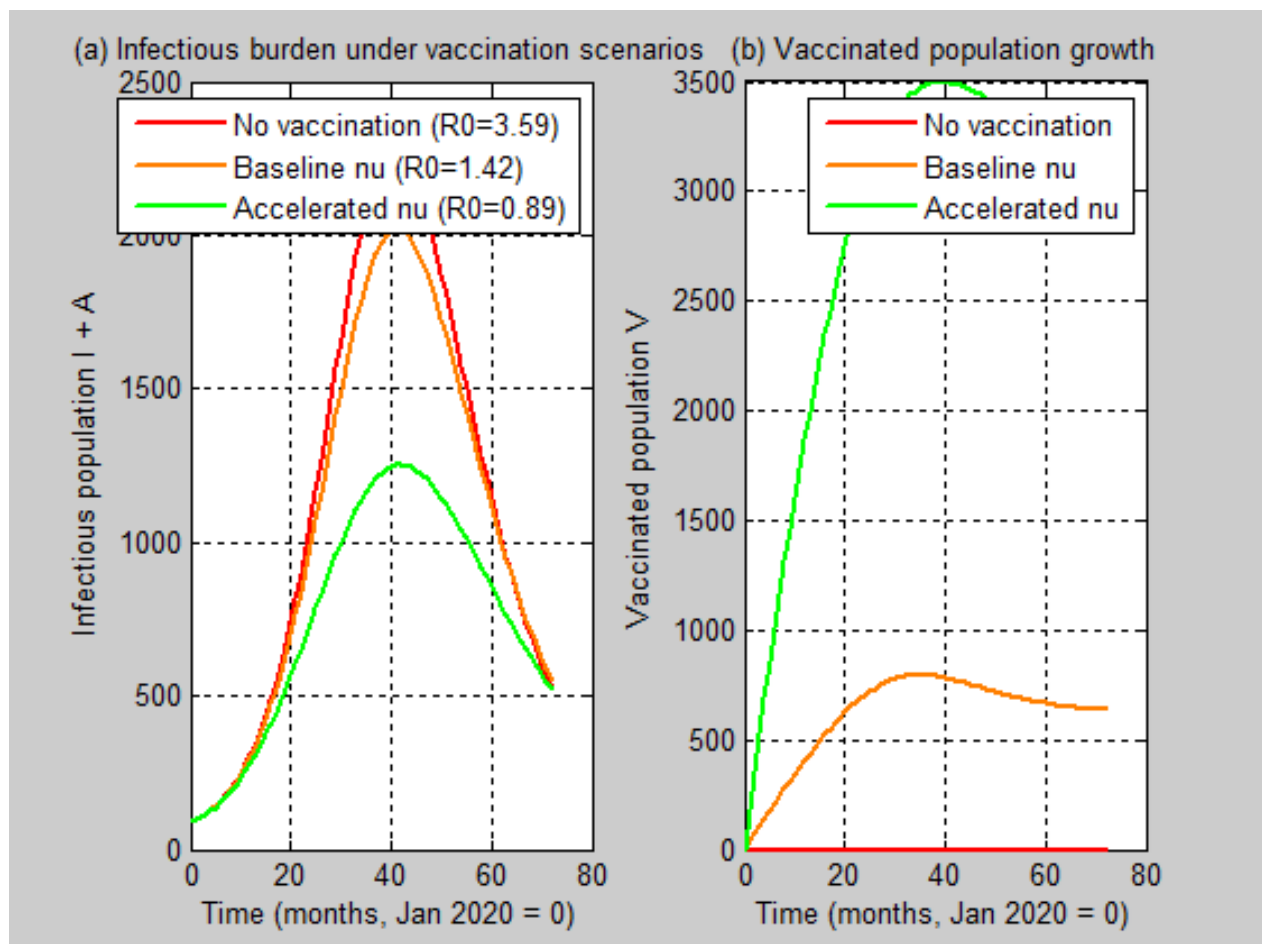


Figure 3: Effect of the vaccination rate on (a) the infectious population $I(t) + A(t)$ and (b) the vaccinated population $V(t)$, under three scenarios: no vaccination, the baseline vaccination rate, and an accelerated vaccination rate five times the baseline value.

In the absence of vaccination, the basic reproduction number rises to $R_0 \approx 3.59$ and the epi-

demic peak increases to approximately 2267 infectious individuals, compared with 2021 under the baseline vaccination rate, a reduction of approximately 11% in peak infectious burden attributable to vaccination at the baseline rate. Under the accelerated vaccination scenario, R_0 falls below the epidemic threshold to approximately 0.89, and the peak infectious population is reduced to approximately 1252, a reduction of roughly 45% relative to the no-vaccination scenario. Because the natural mortality rate μ is small relative to the 72-month simulation horizon, the infectious population under the accelerated scenario has not yet fully decayed to the disease-free equilibrium by the end of the simulated period, even though $R_0 < 1$; this is consistent with the slow demographic timescale, of order $1/\mu \approx 840$ months, that governs convergence to the disease-free equilibrium once R_0 is reduced below unity.

These results demonstrate that increasing the vaccination rate ν (with vaccine effectiveness ϵ held fixed) both reduces the basic reproduction number, in accordance with equation (62), and lowers the peak infectious burden observed in simulation. The results are consistent with the analytical role of vaccination identified in Section 3.3, in which increasing ν or ϵ reduces the effective susceptible fraction q and hence R_0 , and provide numerical confirmation that sufficiently intensive vaccination can, in principle, drive the basic reproduction number below the epidemic threshold identified in Section 3.4.

5. Declarations

Funding: The authors received no specific funding for this research.

Competing interests: The authors declare no competing interests.

Ethics and consent: This study used synthetic computational data and involved no human participants or identifiable personal data. Ethical approval and informed consent were therefore not required.

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