

Vaccination-Based Modelling for the Dynamics of COVID-19

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Abstract—Coronavirus disease (COVID-19) was a serious health issue in late 2019 and caused a huge number of deaths worldwide. Various mathematical models were constructed to predict and analyse the dynamics of COVID-19. In this study, we formulate a deterministic model that incorporates a vaccination compartment and some other important parameters. The disease-free and disease-present equilibria were analysed, and the analytical expression for the basic reproduction number (R_0) was derived using the next-generation matrix approach. Stability of the system was checked using Jacobian and eigenvalue analysis. The analytical results establish that the system preserves biological feasibility, with all state variables remaining positive and bounded over time. The model dynamics and effect of key parameters on the transmission of COVID-19 are demonstrated through numerical simulations using the MATLAB ODE45 solver. We found that vaccination has a positive impact on controlling COVID-19 spread.

Keywords—mathematical modelling, vaccination, stability analysis, basic reproduction number, numerical simulation, nonlinear dynamical systems.

I. INTRODUCTION

COVID-19 is one of the contagious diseases, initially originating in Wuhan, China [6]. At first, it was considered that the disease could be severe acute respiratory syndrome (SARS). Chinese doctors tested the hypothesis, identified the cause of the new virus, and named it COVID-19 [1]. The uncontrolled growth and rapid expansion of the disease affect population health, education, the economy, social activities, etc [2, 11].

COVID-19 belongs to a family of viruses called coronaviruses. This family includes SARS and COVID-19, etc. These viruses have a high impact on human and animal health. Some of these viruses cause mild infections such as colds, fever, etc. Some cause severe infections like SARS and COVID-19. Due to rapid transmission among the population, these diseases are very difficult to control.

Due to changes in clinical symptoms of coronaviruses, from mild to severe, the viruses are divided into two categories. In mild cases, patients show weak symptoms like fever, cough, and fatigue, etc [3]. In severe cases, patients show strong

symptoms which can cause death and other serious respiratory diseases like pneumonia, organ failure, and SARS, etc.

COVID-19 virus is transmitted from person to person through coughs, sneezes, handshakes, and other contact [8]. This virus penetrates the body and targets the cells within the respiratory system. It binds to the angiotensin-converting enzyme 2 (ACE2), which is an important protein located on the cell membrane, allowing the coronavirus to enter the cell via ACE2. Inside the host cell, it releases its Ribonucleic Acid (RNA) and utilises the cell's machinery to replicate its RNA [16]. As a result of this process, new coronaviruses are generated, which then go on to infect neighbouring cells, thereby continuing the spread throughout the body.

The rapid expansion of COVID-19 in the world made it a leading cause of death worldwide. Due to the high rate of transmission, the disease spread globally, and the World Health Organisation (WHO) announced it as a pandemic. In January 2023, WHO stated that there were about 744 million confirmed cases and about 7 million deaths worldwide, and these numbers were expected to grow soon [11]. Countries all across the world closed their borders and implemented restrictions such as smart lockdowns, home isolation, social distancing, etc [5]. These interventions reduced the disease spread to a certain extent. But these policies were not enough to eliminate COVID-19.

In addition to these interventions, vaccination is also one of the most important factors that contribute to the reduction of disease spread [4, 9]. For almost all infectious diseases, vaccination is considered the key factor in controlling the disease. For COVID-19, different doses of vaccine were used in different countries; in Pakistan, three doses were administered to individuals [6].

The first confirmed death due to COVID-19 was officially recorded on January 9, and the number of deaths later increased rapidly. In the early stage, when the disease erupted in China, over 40 million cases were confirmed. Initially, the disease was limited to China, but later it spread around the world and affected all countries [10].

To understand the behaviour of COVID-19, mathematical modelling is a crucial tool to be used. From all across the world, different models were created to study the disease dynamics [12, 19]. Theoretical study of COVID-19 is very important to understand its behaviour across different parameters [15, 18]. It

answers the questions of what proportion of individuals should be vaccinated, isolated, and hospitalised to control the disease spread, and which interventions should be implemented [13, 19]. Every model has some limitations because of its assumptions, and no model captures the disease dynamics 100 per cent accurately.

Based on existing research and its limitations, the proposed study is motivated and adds a vaccination compartment to study the dynamics of COVID-19. Therefore, the proposed research work introduces vaccination for susceptible people. The impact of vaccination is represented through simulation curves, and the results show that increases in vaccination rate decrease disease transmission.

This research proposes a compartmental model to investigate COVID-19 dynamics by using a vaccination compartment. The proposed model also includes other main parameters such as waning immunity, isolation, quarantine, etc. It analyses their impact on COVID-19 transmission dynamics. The total population is divided into twelve mutually exclusive groups.

1. LIST OF AGGREGATED COMPARTMENTS

Table A. Compartments with their description are given below.

Symbol	Description
S_f	Susceptible freely
S_r	Susceptible restricted isolation
E_f	Exposed freely
E_r	Exposed restricted isolation
I_f	Infected freely
I_r	Infected restricted isolation
A_f	Asymptomatic freely
A_r	Asymptomatic restricted isolation
Q	Quarantined
V	Vaccinated
R	Recovered
D	Death

II. MATHEMATICAL MODEL OVERVIEW

We investigated the vaccination compartment and introduced some modifications to the mathematical model proposed in [8]. In the existing model, Aronna, M. S et al.[8], studied the impact of isolation, hospitalisation, and control strategies to analyse and predict the COVID-19 transmission dynamics. In the proposed model, we have integrated a vaccination compartment along with some additional parameters and analysed their impact on COVID-19 dynamics.

The proposed model 1 is recruited by Λ , κ proportion is received by S_f and $(1 - \kappa)$ proportion enters into S_r compartment. S_f and S_r compartments are vaccinated at the same rate p , which results in growth in the population of V . V

class decay at the rate ω_v towards both S_f and S_r classes with different proportions. Susceptible individuals enter the exposed compartment at the rate β after coming into contact with infected people, and then move to infected classes after the incubation period. Natural mortality rate μ_n is introduced to all compartments, while the disease-induced death rate μ is only introduced to asymptomatic, infected and quarantined compartments. The quarantined class receives individuals only from the infected and asymptomatic classes at different rates. The recovered compartment is saturated by naturally recovered individuals from A_f and A_r at the rate γ_1 , and from Q at the rate γ_2 . Recovered individuals re-enter the susceptible compartments after losing immunity at the rate ω .

$$\frac{dS_f}{dt} = \Lambda\kappa - \beta [I_f + A_f + r(I_r + A_r)]S_f + \omega\phi R + \eta\omega_v V - (p + \mu_n)S_f,$$

$$\frac{dS_r}{dt} = \Lambda(1 - \kappa) - r\beta [I_f + A_f + r(I_r + A_r)]S_r + \omega(1 - \phi)R + \omega_v(1 - \eta)V - (p + \mu_n)S_r,$$

$$\frac{dE_f}{dt} = \beta [I_f + A_f + r(I_r + A_r)]S_f - (\tau + \mu_n)E_f,$$

$$\frac{dE_r}{dt} = r\beta [I_f + A_f + r(I_r + A_r)]S_r - (\tau + \mu_n)E_r,$$

$$\frac{dI_f}{dt} = \tau E_f - (\sigma + \mu + \mu_n)I_f,$$

$$\frac{dI_r}{dt} = \tau E_r - (\sigma + \mu + \mu_n)I_r, \quad (1)$$

$$\frac{dA_f}{dt} = \alpha\sigma I_f - (\gamma_1 + \mu + \mu_n)A_f,$$

$$\frac{dA_r}{dt} = \alpha\sigma I_r - (\gamma_1 + \mu + \mu_n)A_r,$$

$$\frac{dQ}{dt} = \sigma(1 - \alpha)(I_r + I_f) + (1 - \delta)\gamma_1(A_f + A_r) - (\mu_n + \gamma_2 + \mu)Q,$$

$$\frac{dV}{dt} = p(S_f + S_r) - (\omega_v + \mu_n)V,$$

$$\frac{dR}{dt} = \gamma_1\delta(A_f + A_r) + \gamma_2 Q - (\omega + \mu_n)R,$$

$$\frac{dD}{dt} = \mu_n(N + Q) + \mu(I_f + I_r + A_f + A_r + Q).$$

2. POSITIVITY AND BOUNDEDNESS OF THE MODEL

Theorem (Positivity). Assume that the initial conditions of the system (1) satisfy:

$$\begin{aligned} S_f(0) \geq 0, S_r(0) \geq 0, E_f(0) \geq 0, E_r(0) \geq 0, I_f(0) \geq 0, \\ I_r(0) \geq 0, A_f(0) \geq 0, A_r(0) \geq 0, Q(0) \geq 0, V(0) \geq 0, \\ R(0) \geq 0, D(0) \geq 0, \end{aligned}$$

and all the model parameters are non-negative. Then the solutions:

$$X(t) = (S_f, S_r, E_f, E_r, I_f, I_r, A_f, A_r, Q, V, R, D),$$

of the system remain nonnegative for all time greater than zero. Thus, the nonnegative orthant:

$$R^{12} = \{X \in R_+^{12} \mid X(t) \geq 0, \quad \forall t \geq 0\},$$

is positively invariant.

Proof: Consider the first equation of the system (1):

$$\begin{aligned} \frac{dS_f}{dt} = \Lambda\kappa - \beta [I_f + A_f + r(I_r + A_r)]S_f + \omega\phi R + \eta\omega_v V \\ - (p + \mu_n)S_f, \end{aligned}$$

It follows that

$$\begin{aligned} \frac{dS_f}{dt} \geq -(\beta [I_f + A_f + r(I_r + A_r)] + p + \mu_n)S_f, \\ \frac{dS_f}{dt} \geq -h(t)S_f, \end{aligned}$$

Using Gronwall's inequality, we obtain:

$$S_f(t) \geq S_f(0)e^{-\int_0^t h(u)du}$$

It follows that:

$$S_f(t) \geq 0, \quad \forall t \geq 0,$$

Similarly, each of the remaining state variables can be expressed in the form:

$$\frac{dx_i}{dt} = -c_i(t)x_i,$$

where

$$x_i \in X(t),$$

Therefore, every solution of the model with non-negative initial conditions remains non-negative for all time. Thus, the solution of the system satisfies:

$$S_r, E_f, E_r, I_f, I_r, A_f, A_r, Q, V, R, D \geq 0, \quad \forall t \geq 0.$$

Therefore, the region:

$$\Omega = \{S_f, S_r, E_f, E_r, I_f, I_r, A_f, A_r, Q, V, R, D \in R_+^{12}\},$$

is positively invariant.

Theorem: (Boundedness) Assume that all initial conditions are non-negative. Then every solution of the model is bounded for all $\forall t \geq 0$. Moreover, the total living population w.r.t. time is:

$$N(t) = S_f + S_r + E_f + E_r + I_f + I_r + A_f + A_r + Q + V + R + D,$$

satisfies:

$$0 \leq N(t) \leq \max\left\{N(0), \frac{\Lambda}{\mu_n}\right\}, \quad \forall t \geq 0.$$

Consequently, the feasible region is:

$$\Omega_1 = \left\{X \in R_+^{11} : N \leq \frac{\Lambda}{\mu_n}\right\},$$

is positively invariant.

Proof: The change in total population $N(t)$ over time is given by:

$$\frac{dN}{dt} = \Lambda - \mu_n N^* - \mu(I_f + I_r + A_f + A_r + Q),$$

Where N^* is the sum of all groups except S_f ,

as $\mu(I_f + I_r + A_f + A_r + Q) \geq 0$, therefore

$$\frac{dN}{dt} \leq \Lambda - \mu_n N^*,$$

the solution of the inequality gives:

$$N(t) \leq N(0)e^{-\mu_n t} + \frac{\Lambda}{\mu_n}(1 - e^{-\mu_n t}),$$

as $t \rightarrow \infty, N(t) \rightarrow \frac{\Lambda}{\mu_n}$ implies that $0 \leq N(t) \leq \frac{\Lambda}{\mu_n}$.

Hence, every solution of the model remains bounded for all $\forall t \geq 0$, and the feasible region:

$$\Omega_1 = \{X(t) \in R_+^{12} : N(t) \leq \frac{\Lambda}{\mu_n}\}.$$

Hence, the system (1) is biologically meaningful and mathematically correct.

a) INITIAL CONDITION

The initial condition used throughout the paper is given by:

$$\{S_f^0, S_r^0, E_f^0, E_r^0, I_f^0, I_r^0, A_f^0, A_r^0, Q^0, V^0, R^0\} = \{2500, 2000, 20, 20, 70, 70, 30, 30, 3, 10, 10\}.$$

b) STABILITY ANALYSIS

To check the stability of the model, first we determine two equilibrium points, namely disease-free and disease-present. Next, we analyse the local stability of the system at both equilibrium points [18, 19]. To find the equilibrium points of the system, we set the right-hand sides of all the derivatives of system (1) equal to zero:

1) DISEASE-FREE EQUILIBRIUM

The disease-free equilibrium (DFE) of the system (1) arises once the infected compartments become zero, i.e $E = I = A = 0$, R is also zero because it is saturated by A and Q . Under this condition, the system (1) reduces to:

$$\begin{aligned} \Lambda\kappa + \omega\phi R + \eta\omega_v V - (p + \mu_n)S_f &= 0, \\ \Lambda(1 - \kappa) + \omega\phi R + \omega_v(1 - \eta)V - (p + \mu_n)S_f &= 0, \\ p(S_f + S_r) - (\omega_v + \mu_n)V &= 0. \end{aligned} \quad (2)$$

The DFE of subsystem (2) is given by:

$$\Sigma^o = (S_f^o, S_r^o, V^o),$$

where

$$S_f^o = \frac{\Lambda\kappa + \omega_v\eta V^0}{p + \mu_n}, \quad S_r^o = \frac{\Lambda(1 - \kappa) + \omega_v(1 - \eta)V^0}{p + \mu_n},$$

$$V^0 = \frac{\Lambda p}{\mu_n(p + \omega_v + \mu_n)}.$$

The Jacobian matrix at the DFE associated with subsystem (2) is:

$$J(\Sigma^o) = \begin{pmatrix} -(p + \mu_n) & 0 & \omega_v\eta \\ 0 & -(p + \mu_n) & \omega_v(1 - \eta) \\ p & p & -(\omega_v + \mu_n) \end{pmatrix} \quad (3)$$

The eigenvalues of matrix (3) are:

$$\lambda_1 = -(p + \mu_n), \quad \lambda_2 = -\mu_n, \quad \lambda_3 = -(p + \omega_v + \mu_n),$$

where

$$p, \mu_n, \omega_v > 0,$$

Since all the eigenvalues are negative, DFE is locally asymptotically stable.

2) DISEASE PRESENT EQUILIBRIUM

The disease-present equilibrium (DPE) is obtained numerically by using the Newton-Raphson iterative method in MATLAB and is given by:

$$\Sigma^* = (S_f^*, S_r^*, E_f^*, E_r^*, I_f^*, I_r^*, A_f^*, A_r^*, Q^*, V^*, R^*, D^*).$$

The numerical value of DPE is as follows:

$$\Sigma^* = (91.9313, 75.3171, 141.5905, 47.5607, 97.2653, 32.6717, 94.6950, 31.8083, 177.6336, 1742.1707, 3651.7179).$$

The corresponding eigenvalues of the Jacobian matrix of system (1) at the DPE for the chosen parameter values given in Table 1 are:

$$\begin{aligned} \lambda_1 &= -0.297309111516405 + 0.021355391100933i, \\ \lambda_2 &= -0.297309111516405 - 0.021355391100933i, \\ \lambda_3 &= -0.206500817725189 + 0.000000000000000i, \\ \lambda_4 &= -0.073382763475098 + 0.061568943666431i, \\ \lambda_5 &= -0.073382763475098 - 0.061568943666431i, \\ \lambda_6 &= -0.155990701966552 + 0.019559924033108i, \\ \lambda_7 &= -0.155990701966552 - 0.019559924033108i, \\ \lambda_8 &= -0.129318618441039 + 0.000000000000000i, \\ \lambda_9 &= -0.079815164244949 + 0.000000000000000i, \\ \lambda_{10} &= -0.001948368962738 + 0.000000000000000i, \end{aligned}$$

$$\lambda_{11} = -0.004791826739767 + 0.000000000000000i.$$

Since the real parts of all the eigenvalues are negative, for the given parameter values mentioned in Table 1, the disease-present equilibrium is locally asymptotically stable.

a) BASIC REPRODUCTION NUMBER

Basic reproduction number (R_o) describes the average number of susceptible individuals infected by a single infectious person during his whole infectious period. Recalling (1), we can find R_o by using the next-generation matrix method.

Consider a vector 'X' of infectious compartments:

$$X = [E_f, E_r, I_f, I_r, A_f, A_r]^T,$$

Define two column vectors ' F_1 ' and ' V_1 ', where F_1 contains all new infection terms and V_1 contains all transition terms. In model (1), only exposed classes receive new infections, so

$$F_1 = \begin{pmatrix} \beta [I_f + A_f + r(I_r + A_r)] S_f \\ r\beta [I_f + A_f + r(I_r + A_r)] S_r \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix},$$

and

$$V_1 = \begin{pmatrix} (\tau + \mu_n) E_f \\ (\tau + \mu_n) E_r \\ (\sigma + \mu + \mu_n) I_f - \tau E_f \\ (\sigma + \mu + \mu_n) I_r - \tau E_r \\ (\gamma_1 + \mu + \mu_n) A_f - \alpha \sigma I_f \\ (\gamma_1 + \mu + \mu_n) A_r - \alpha \sigma I_r \end{pmatrix},$$

Next, we define ' F ' and ' V ' as Jacobian matrices of ' F_1 ' and ' V_1 ' evaluated at DFE.

$$F = \begin{pmatrix} 0 & 0 & \beta S_f^o & r\beta S_f^o & \beta S_r^o & r\beta S_r^o \\ 0 & 0 & r\beta S_r^o & r^2\beta S_r^o & r\beta S_r^o & r^2\beta S_r^o \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix},$$

and

$$V = \begin{pmatrix} (\tau + \mu_n) & 0 & 0 & 0 & 0 & 0 \\ 0 & (\tau + \mu_n) & 0 & 0 & 0 & 0 \\ -\tau & 0 & \Psi_1 & 0 & 0 & 0 \\ 0 & -\tau & 0 & \Psi_1 & 0 & 0 \\ 0 & 0 & -\alpha\sigma & 0 & \Psi_2 & 0 \\ 0 & 0 & 0 & -\alpha\sigma & 0 & \Psi_2 \end{pmatrix},$$

where

$$\Psi_1 = (\sigma + \mu + \mu_n), \quad \Psi_2 = (\gamma_1 + \mu + \mu_n).$$

The basic reproduction number is the spectral radius (largest eigenvalue) of the next-generation matrix: $R_o = \rho(FV^{-1})$ [17].

Therefore,

$$R_o = \frac{\beta\tau(\alpha\sigma + \gamma_1 + \mu + \mu_n)(S_f^o + r^2S_r^o)}{(\tau + \mu_n)(\sigma + \mu + \mu_n)(\gamma_1 + \mu + \mu_n)(p + \mu_n)}$$

b) NUMERICAL SOLUTION

Because of the nonlinear and complex structure of the proposed model, it was difficult to find analytical solutions. Therefore, the proposed model was solved numerically using the ODE45 solver. This solver implements the RK45 method. It computes seven slopes at different locations and combines all these slopes using a weighted mean. It computes two approximations of the solutions at the average point: approximations of order 4 and 5. The fourth-order approximation is used for error, while the fifth-order approximation provides the final solutions. The difference between the two solutions gives the error. To illustrate the analytical results and to gain further insight into the dynamics of system (1), we now carry out numerical simulations. The model equations are solved using a standard numerical solver for systems of ordinary differential equations with biologically relevant parameter values [18, 19]. All computations are performed with nonnegative initial conditions, ensuring consistency with the positivity and boundedness properties established in the previous section.

The parameter values used in the simulations are summarised in Table 1. These values are chosen from the literature or selected to be within biologically realistic ranges.

Table 1: Model parameters used in numerical simulations.

Parameter	Description	Value	Unit
Λ	Recruitment	12	day ⁻¹
r	Reduction coefficient	0.41	day ⁻¹
κ	Proportion of recruited population S_f	0.70	day ⁻¹
β	Contact rate	0.001	day ⁻¹
τ	Rate of infection	0.14	day ⁻¹
ω	Waning rate for R class	0.005	day ⁻¹
σ	Transition rate for A classes	0.2	day ⁻¹
α	Proportion of from I into A classes	0.70	day ⁻¹
ϕ	Proportion of recovered into susceptible freely	0.70	day ⁻¹
γ_1	Recovery rate for A class	0.14	day ⁻¹
γ_2	Recovery rate for Q class	0.07	day ⁻¹

δ	Proportion of A into R	0.70	day ⁻¹
μ	Mortality rate	0.002	day ⁻¹
p	Vaccination rate	0.05	day ⁻¹
μ_n	Natural death rate	0.0018	day ⁻¹
Proportion from V in S_f			
η	waning rate for vaccinated class	0.70	day ⁻¹
ω_v		0.003	day ⁻¹

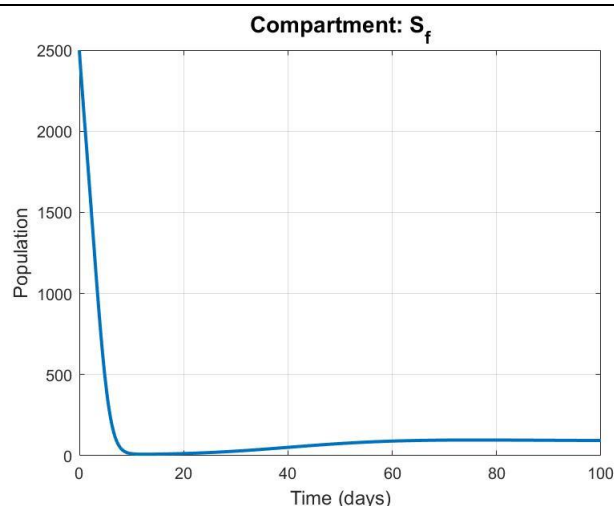


Figure 1: Time Evolution of the Susceptible freely moving individuals.

The freely moving population (S_f) dynamics are depicted in Figure (1). Initially, S_f drops quickly because susceptible people are moved to the vaccinated compartment and get infected through effective contact with infectious people. The sharp drop in the first ten days shows intense disease transmission. Due to the continuous recruitment of individuals and the decline of vaccine-induced and natural immunity, the compartment gradually grows and stabilises after reaching a minimum. This behaviour suggests that even when the epidemic reaches equilibrium, there is still a small population.

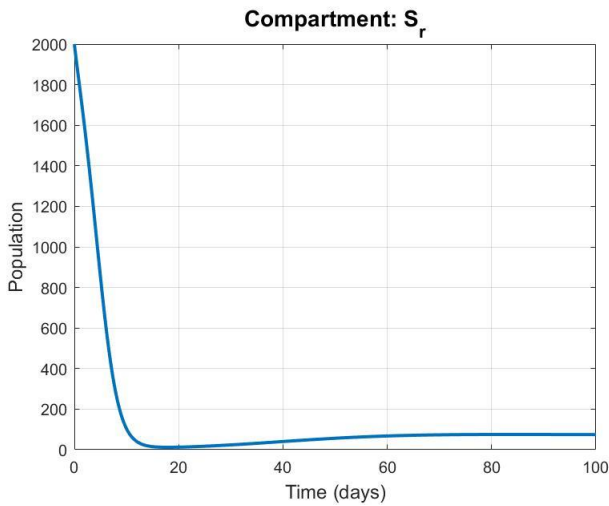


Figure 2: Dynamics of susceptible restricted-isolated individuals over time.

Figure 2 captures the movement of the (S_r) people over time. Initially, the number of restricted susceptible persons rapidly declines when they contract the disease or receive vaccinations. However, the long-term equilibrium is reached at a constant level due to decreased contact behaviour and immunity loss in the recovered and vaccinated compartments.

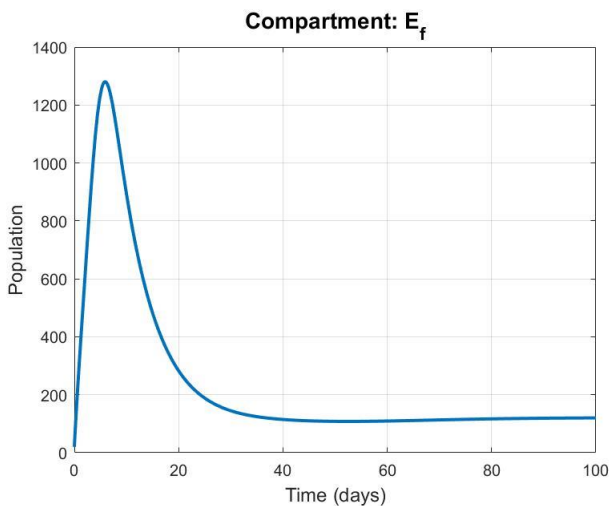


Figure 3: Time Evolution of exposed freely moving individuals.

The dynamics of the exposed freely moving population (E_f) are seen in Figure 3. As many susceptible people contract the infection but do not yet show symptoms, the exposed class grows quickly during the first week. Then, as people move into the infectious compartment, the exposed population gradually declines. At the end, E_f reaches the equilibrium state, signifying ongoing but managed disease transmission.

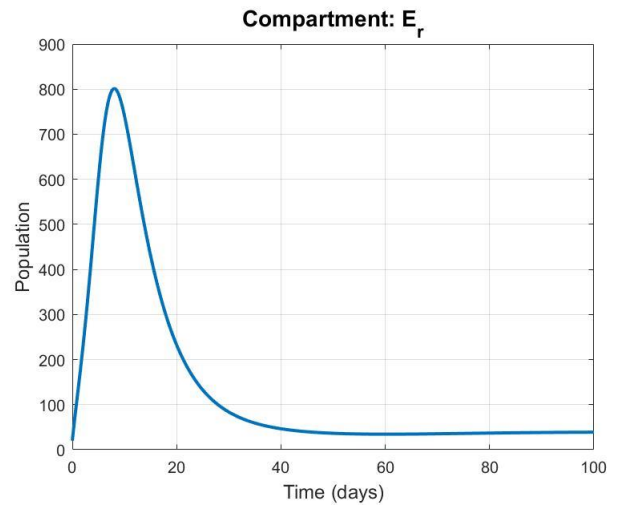


Figure 4: Dynamics of exposed restricted-isolated individuals over time.

The change in the exposed restricted population (E_r) over time is shown in Figure 4. The compartment behaves similarly to E_f , but with a lower peak level, since those that are restricted have fewer effective contacts. Reduced contact rates effectively limit the spread of disease, as shown by the exposed population's progressive fall and stabilisation at a low equilibrium level after the peak.

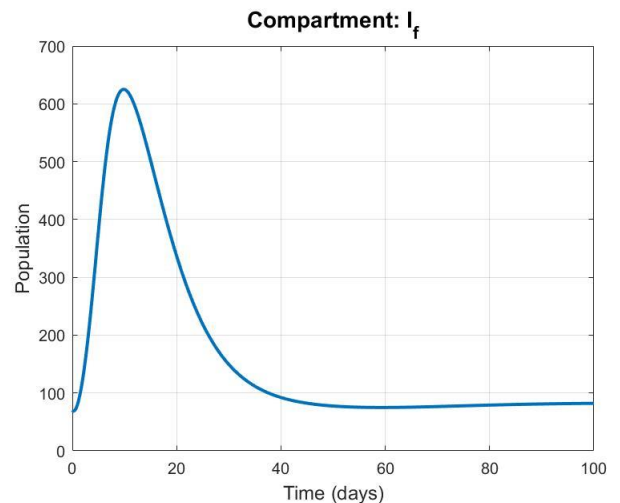


Figure 5: Dynamics of infected freely moving individuals over time.

The dynamics of the infected freely moving population (I_f) are described in Figure 5. Initially, the number of infectious individuals increases and reaches a peak level on day 10. After the peak level, as infected people recover, are placed under quarantine, are removed by A_f or pass away, the infectious population decreases. The compartment eventually stabilises at a constant level, indicating the continued existence of a tiny endemic infectious population.

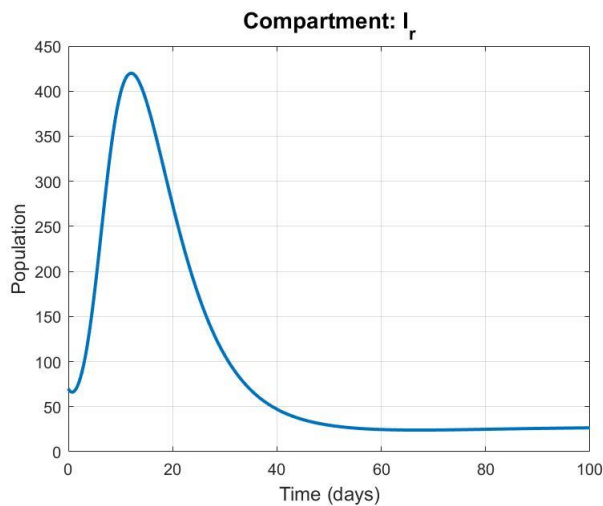


Figure 6: Dynamics of infected restricted-isolated individuals over time.

The infectious restricted population (I_r) is shown in Figure 6. The number of infectious restricted individuals rises initially until it reaches a peak level. The lower peak compared to I_f shows a decrease in the spread of disease among those who are restricted. Recovery, quarantined, and disease-related death are the causes of the drop, which finally reaches an equilibrium.

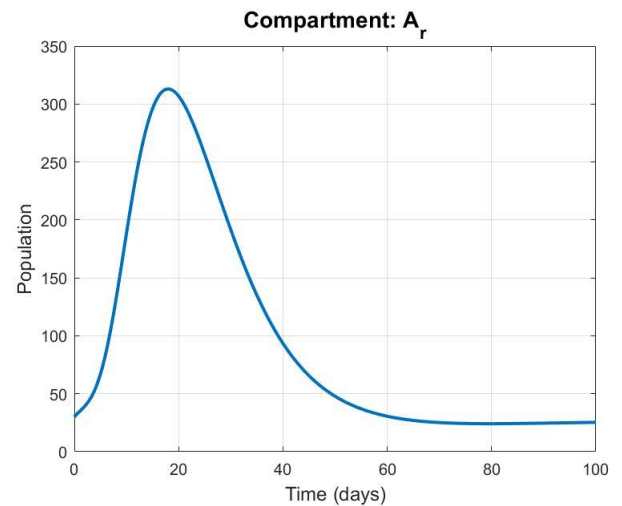


Figure 8: Dynamics of asymptomatic restricted-isolated individuals over time.

The asymptomatic restricted population (A_r) dynamics are shown in Figure 8. Initially, the number of asymptomatic individuals rises and reaches a peak before progressively declining. The lower peak in relation to A_f is in line with lower exposure among those who are restricted. In the end, the compartment stabilises at a low endemic level.

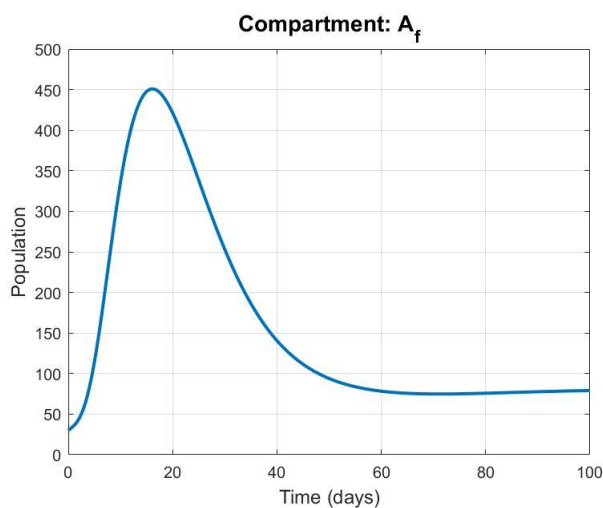


Figure 7: Dynamics of asymptomatic freely moving individuals over time.

The asymptomatic freely moving population (A_f) behaviour is depicted in Figure 7. As a percentage of infectious people experience asymptomatic infection, the compartment grows, peaking at a certain point. The asymptomatic population steadily declines and approaches a steady equilibrium level as recovery and other transition processes take individuals out of A_f .

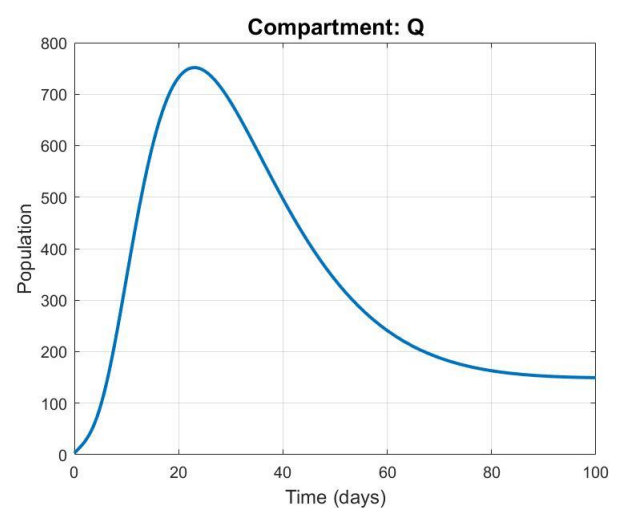


Figure 9: Dynamics of quarantined individuals over time.

The population under quarantine (Q) is represented in Figure 9. The quarantine compartment is initially empty, but as contagious persons are found and isolated, the number under quarantine grows quickly and reaches a peak level. The population then declines as those in quarantine either recover or pass away. A comparatively limited number of infectious individuals continue to be isolated during the endemic stage; at the end, the compartment stabilises at a steady state.

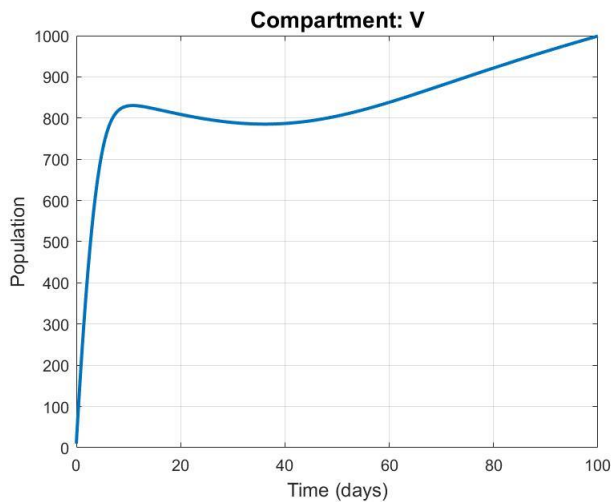


Figure 10: Dynamics of vaccinated individuals over time.

The vaccinated population (V) is shown in Figure 10. During the early stages of the epidemic, vaccination rates rise quickly, indicating fast immunisation. Waning vaccine-induced immunity causes a minor fall during the intermediate period, following which ongoing vaccination causes the vaccinated population to rise gradually once more. About 1000 people have received the vaccination by day 100, proving the long-term efficacy of persistent immunisation campaigns.

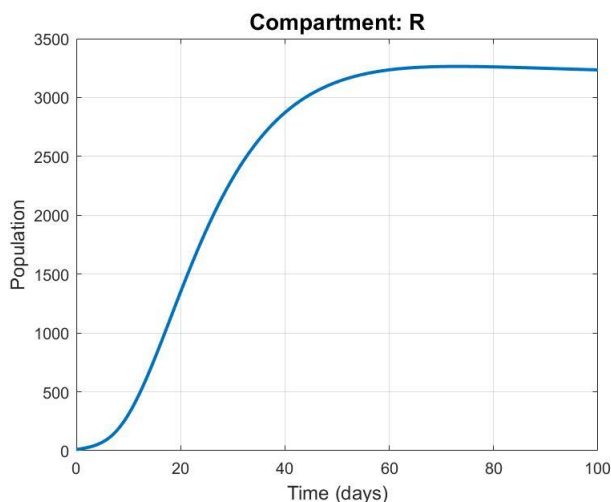


Figure 11: Dynamics of recovered (R) individuals over time.

The recovered population (R) is shown in Figure 11. As asymptomatic and isolated people recover from the illness, the recovered compartment keeps growing. As the epidemic spreads, the curing process picks up speed. The recovered population eventually reaches a peak, suggesting that most affected people eventually recover and develop transient immunity.

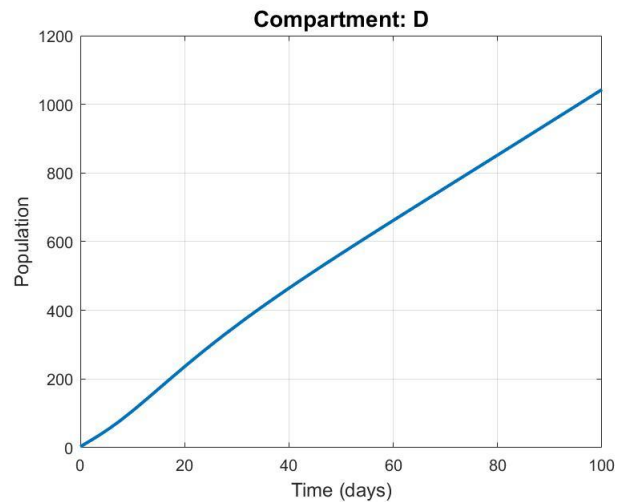


Figure 12: Time Evolution of the Death compartment.

The total number of deaths (D) caused by the disease is displayed in Figure 12. The death compartment grows monotonically over the course of the simulation since it is cumulative. After the first stage, the growth is almost linear, indicating that disease-related deaths continue to occur. The total death toll by the simulation's conclusion surpasses 1000, indicating the epidemic's long-term mortality burden.

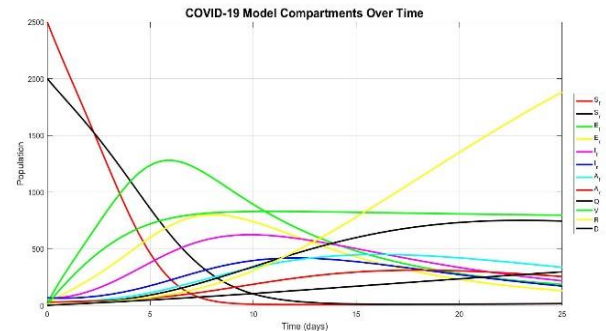


Figure 13: Combined behaviour of all state variables.

The numerical simulations demonstrate the complete progression of the epidemic predicted by the proposed twelve-compartment model. The susceptible populations (S_f, S_r) decline rapidly during the initial outbreak due to infection and vaccination. Consequently, the exposed (E_f, E_r), infectious (I_f, I_r), asymptomatic (A_f, A_r), and quarantined (Q) populations increase, each reaching a distinct peak before declining as recovery, vaccination, quarantine, and disease-induced mortality become dominant. The vaccinated (V) and recovered (R) compartments increase throughout the simulation, indicating the accumulation of immunity within the population, while the cumulative death compartment (D) increases continuously. By approximately 70–80 days, all non-cumulative compartments approach steady-state values, demonstrating that

the epidemic converges to a disease-present (endemic) equilibrium. These results indicate that the proposed model successfully captures the transition from the epidemic growth phase to a long-term endemic state while accounting for vaccination, quarantine, immunity waning, and heterogeneous contact behaviour.

Impact of Vaccination Rate (p) on Infectious Compartments

The impact of p is illustrated in the figures given below. All other model parameters are considered constant.

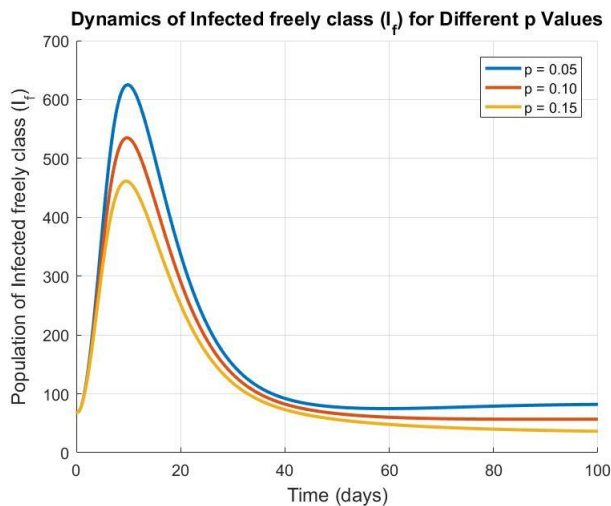


Figure 14: Comparison of the infected freely compartment at different vaccination rates.

The influence of vaccination rate p on the infected free compartment I_f is described in Figure 14. The simulations are conducted for three different vaccination rates ($p=0.05, 0.10$, and 0.15) while all other parameters are constant. The results show that increasing the vaccination rate significantly reduces both the epidemic peak and steady state of the system. The infected individuals reach the highest peak at $p = 0.05$, while these peak levels decrease with an increase in vaccination rate. This behaviour occurs because higher p transfers more population into the p compartment, which results in less availability of people for infection.

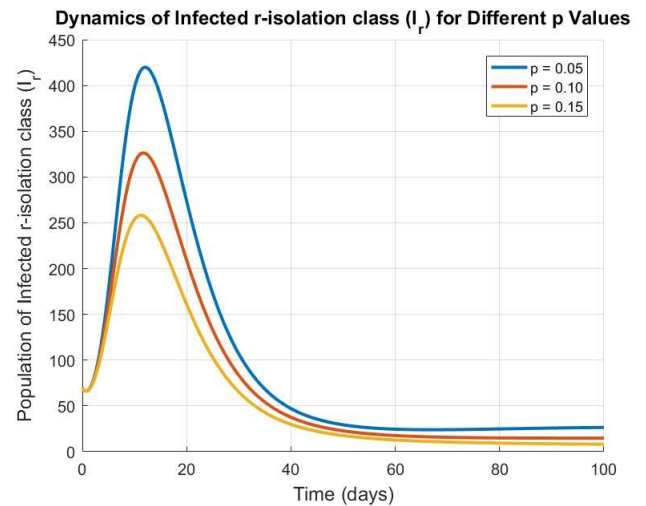


Figure 15: Comparison of infected restricted-isolated compartment at different vaccination rates.

Figure 15 represents the impact of vaccination rate p on the dynamics of the infected restricted compartment (I_r). Three different vaccination rates ($p = 0.05, 0.10$, and 0.15) are considered, while all other parameters are kept constant. The numerical simulations show three different peaks for the corresponding p values. At $p = 0.05$, the individuals reach the highest peak of endemicity, while increasing the vaccination rate reduces both the endemic peak and the equilibrium state. This behaviour occurs because a higher p rate reduces the number of susceptible people available for infection.

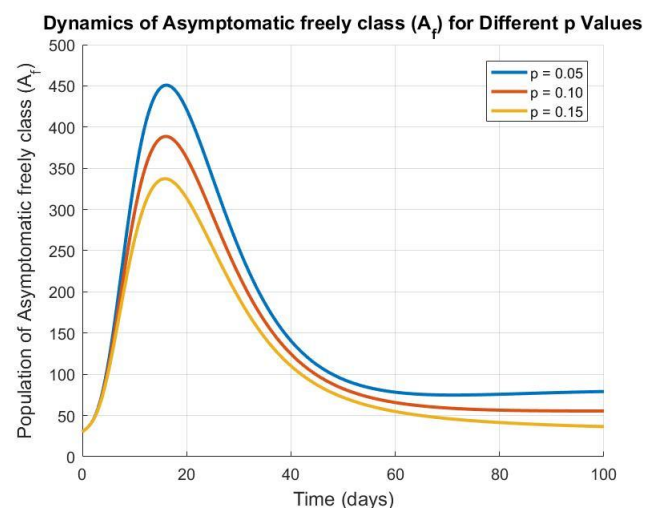


Figure 16: Comparison of the asymptomatic freely compartment at different vaccination rates.

The influence of vaccination rate (p) on the asymptomatic freely class A_f is presented in Figure 16. Vaccination rate p takes different values $p = 0.05, 0.10$, and 0.15 , keeping all other parameters constant. The simulation results record a noticeable reduction in the asymptomatic compartment. The A_f reaches the highest peak at the lowest vaccination rate, $p = 0.05$; both

endemic peaks and endemic equilibrium decrease with an increase in p . The reduction occurs because vaccination reduces the susceptible population that can become infected and asymptomatic.

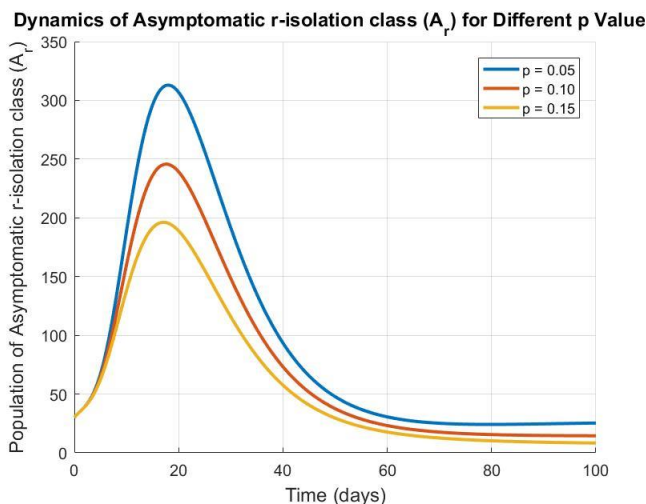


Figure 17: Comparison of the asymptomatic restricted-isolated compartment at different vaccination rates.

Figure 17 illustrates the vaccination effect on the asymptomatic restricted population (A_r). The numerical simulations were obtained for three different vaccination rates $p = 0.05, 0.10$, and 0.15 , and all other parameters are considered constant. The numerical results elaborate the significant decrease in the A_r population with increasing p . The largest peak value at $p = 0.05$ is followed by two consecutive lower peaks at $p = 0.10$ and 0.15 ; the corresponding endemic equilibrium points are reduced. The result indicates that effective vaccination controls the progression of susceptible individuals into asymptomatic A_r infection.

ONCLUSION

This research developed a twelve-compartment model to analyse and predict COVID-19 transmission in a heterogeneous population. The total population was divided into twelve compartments. The proposed model incorporated vaccination, waning immunity, natural mortality, and disease-induced mortality to provide a comprehensive representation of COVID-19 transmission. The mathematical properties of the proposed model were first established. The positivity and boundedness analyses confirmed that the solutions remain non-negative and bounded for all time. The disease-free equilibrium was derived analytically, while the disease-present equilibrium was obtained numerically. Jacobian matrix and eigenvalue analysis were used to ensure the local stability of the equilibrium; the equilibrium is locally asymptotically stable if all the eigenvalues possess negative real parts. Numerical simulations were performed using MATLAB's ODE45 solver to examine the dynamics of all compartments.

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