DOI Link: https://doi.org/10.61586/cKnGr
Vol.65, Issue.7, Part.1, July 2025, PP.2-24

Analysis of COVID-19 Disease Transmission: A Blended Appraisal of Quarantine and Vaccination Effects in Bangladesh

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Received May 2025 Accepted June 2025 Published July 2025

Highlights

- We developed a new compartmental model (SEQIRVP) that incorporates quarantine, vaccination, and deceased states for accurate simulation of infectious disease transmission.
- Analyzed stability and reproduction number (R_0) to identify threshold parameters for disease elimination or persistence.
- Applied the model on real-world COVID-19 data from Bangladesh to test the model prediction abilities across different public health intervention techniques.
- Performed a detail sensitivity analysis to find out the most influential parameters which are the responsible for spreading the infection.
- This paper showed that the combination of interventions such as vaccination, quarantine, and healthcare enhancement is much more beneficial than immunization alone in lowering infection and mortality rates.

Abstract

A precise understanding of infectious disease transmission patterns is critical for developing effective control methods and reducing the negative effect on public health. In this study, we introduce SEQIRVP, a unique mathematical epidemic model that incorporates the effects of quarantine, vaccination and death by dividing the population into distinct epidemiological compartments. A full stability analysis is performed in which equilibrium conditions are systematically analyzed and the calculation of basic reproduction number to determine whether the disease will persist or decline.
Numerical simulations are used to study how the disease spreads across different groups in the population. Furthermore, a sensitivity analysis is performed to identify which parameters most influence the outbreak's dynamics. Using COVID-19 data from Bangladesh, we simulate the model in MATLAB to see the disease progression under varying vaccination rates, quarantine effectiveness, and healthcare interventions. The results indicate that while vaccination alone may not lead to an immediate reduction in transmission, its combination with vaccination and quarantine and enhanced healthcare significantly lowers both transmission rates and mortality. This study strengthens the importance of strategic epidemic control and provides a strong foundation for influencing public health policy, enhancing social resilience, and ensuring economic stability.
Keywords: COVID-19, Sensitivity Analysis, Vaccination Strategy, Quarantine Measures.

Introduction:

One of the most recent and significant outbreaks, COVID-19 pandemic, was first detected towards the end of December of 2019 in Wuhan, China and then rapidly spread across China. It captured global attention due to its respiratory effects caused by a new coronavirus named SARS-COV-2[1]. By January 7, 2020, experts had confirmed that the disease was caused by this novel coronavirus. Soon after, cases of the same disease appeared in Thailand, Japan, and South Korea. As the virus spread quickly worldwide, the World Health Organization (WHO) officially classified it as a pandemic on March 11, 2020. According to WHO, by April 10, 2020, more than 1.6 million people had been infected worldwide, and nearly 100,000 had died. In China, about 82,000 cases were recorded, and Wuhan remained under lockdown for over two months. Countries such as the United States, Italy, Iran, Spain, France, and the United Kingdom reported some of the highest number of cases outside of China. The United States became the worst-affected country, with more than 500,000 cases and over 17,000 deaths. Italy had already recorded 18,000 deaths[2].

Between 2021 and 2023, the COVID-19 pandemic underwent significant changes due to new SARS-CoV-2 mutations, continued research, and the development of mathematical models for controlling the virus. Scientists and health experts worked hard to track these changes and develop better ways to manage the spread of the disease. During this time, several new variants of the virus were identified, each with unique mutations that affected how easily they spread and how well they could avoid immune responses. Alpha (B.1.1.7) first found in the UK in late 2020, this variant spread more easily than the original virus. Beta (B.1.351) discovered in South Africa; Beta had mutations that made some vaccines less effective. Gamma (P.1) variant, which appeared in Brazil, spread faster and had a higher risk of reinfection. Delta (B.1.617.2) first detected in India and it quickly became the dominant strain worldwide because it was highly contagious. Omicron (B.1.1.529) was found in late 2021. It had many mutations that made it spread rapidly and partially escape immunity. These variants mainly spread through the air when infected people talked, coughed, or sneezed. Changes in the virus, especially in its spike protein, made it easier for it to enter human cells and, in some cases, resist immunity from past infections or vaccinations. For example, studies showed that the Gamma variant was up to 2.4 times more contagious than earlier versions of the virus. Individuals who had previously recovered from COVID-19 had only 54-79% immunity against reinfection with the Gamma variant [3]. With new variants emerging, scientists needed better ways to predict how the virus would spread and how different strategies like lockdowns and vaccines could help control it. For this reason, researchers used mathematical models to analyze disease transmission and guide health policies [4] [5] [6].

In this study, we have built the SEQIRVP model, a seven compartmental model and an extension of several classical compartmental models in epidemiology, to understand the evolution of compartmental models including how it simulates the progression of infectious diseases within a population. We also conduct a sensitivity analysis and compare the results with statistical methods to determine how closely the model aligns with actual real-world data.

Model Formulation:

The mathematical modeling of infectious diseases has a rich history. In the early 20th century, Ronald Ross developed early compartmental models to describe the spread of malaria, laying the groundwork for future epidemiological modeling [7] [8]. Hamer contributed to the development of mathematical models in epidemiology, focusing on the quantification of disease transmission dynamics [9]. In 1927, Kermack and McKendrick introduced the SIR (Susceptible-Infectious-Recovered) model, a seminal framework that divides the population into compartments to study disease progression [10] [11] [12].

Some of the main models used were SIR (Susceptible-Infectious-Recovered) models. This is the simplest model that divides people into three groups—those who can get infected (S), those who are infected (I), and those who have recovered (R). It assumes that once someone recovers, they are fully immune. The SEIR (Susceptible-Exposed-Infectious-Recovered) model is more advanced than SIR because it includes an "Exposed" (E) group. It represents people who have been infected but are not yet spreading the virus [13]. This makes it more realistic since COVID-19 has an incubation period. COVID-19 does not Spread evenly. The virus spreads differently in crowded cities vs. rural areas, so scientists created spatial models to track outbreaks more accurately.

The need for new models came from the fact that the pandemic changed over time. The early SIR model was helpful, but as scientists learned more about COVID-19, they realized that the virus behaved in unexpected ways and SIR alone was too simple. It did not consider the incubation period, people who don't show symptoms but can still spread the virus, or the effect of vaccination. So more detailed models were needed to study different stages of the infection, public health measures, and vaccination campaigns. Some of the biggest challenges that led to new models were:

COVID-19 has an incubation period. People don't become infectious right away, so SEIR was needed to account for this [14]. Many Cases are Asymptomatic. Some people spread the virus without symptoms, so additional categories were added to the models. In this chapter, we will see that how the virus spreads when some remedies like quarantine, vaccination has been started. SEQIRVP model was created to measure how vaccinated individuals affect transmission. We want to see the effect of vaccination and what will happened after maintaining some health policies like quarantine and also, is it enough to take just vaccination to get rid of this disease.

This model divides the population N into seven compartments based on the number of persons, providing a more realistic view of illness progression and the impact of inventions. For analyzing the disease dynamics, we created a flow graphic to show how the disease spreads across different compartments. The system is based on a set of ordinary differential equations (ODEs) that describe the changes in disease dynamics for each compartment over time (t) within the population [15].

Here, $S(t)$ indicate uninfected persons who are at risk of infection. $E(t)$ refers to people who have been exposed but are not yet infected, while $Q(t)$ are segregated to avoid further transmission. $I(t)$ refers to individuals who are actively infected and sick and can transmit viruses, $R(t)$ are those who have recovered from the

disease, $V(t)$ are those who have received a vaccination, and finally $P(t)$ are those who have died. The whole population is assumed constant. So, we can define,

$$N(t) = S(t) + E(t) + Q(t) + I(t) + R(t) + V(t) + P(t)$$

Each of these compartments are controlled by a system of ordinary differential equation.

Some Assumptions of the Model

To interpret SEQIRVP model, we have considered some following assumptions or properties:

1. Susceptible individuals become exposed with the disease and move to exposed class at a rate β_1 and a portion of susceptible individuals directly moved to quarantined class at the rate of β_2.
2. Quarantine individuals may return to the susceptible class at the rate of ε if they were not actually infected.
3. After being quarantined, people who have shown symptoms are moved to infected class at the rate of μ , while some progressed to death class at the rate of σ.
4. Exposed individuals moved to infected class at the rate of ω while some moved to death class at the rate of φ.
5. After being infected some people reach to recovery stage at the rate of γ according to their high immunity or after receiving proper treatment whereas some moved to death class at the rate of δ.
6. Recovered people may lose the immunity and returned to the susceptible class at the rate of α.
7. Individuals who take the vaccine at the rate of η are transferred to the vaccination group.
8. Vaccinated population can be exposed, quarantined and infected again after taking the vaccine at the rate of β_3, β_4 respectively. Additionally, some may move to death class at the rate of χ.

The following figure provides a detailed representation of our proposed SEQIRVP model. The model's assumption with notation and parameters are shown in the figure.

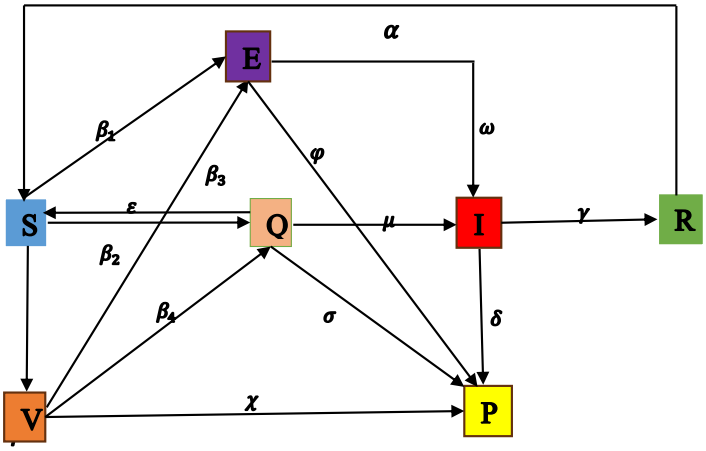

Figure 1 ***A Schematic flow illustration of compartmental SEQIRVP model***

To effectively simulate the spread of COVID-19, we consider specific transmission rate parameters. These parameters actually represent the rate at which individuals move between different compartments, for example susceptible to exposed, infected, quarantined, recovered, vaccinated. By using these parameters, we can see the real-world dynamics including the rate of infection, impact of vaccination, persistence of immunity and many more. In our model, the number of deceased individuals is considered as constant. These parameters determine the rate at which individual transition between groups, helping us to simulate different scenarios. For example, how fast a disease spreads, how long individuals remain sick, how many people may die from the disease, which public health measures should be appropriate to reduce the disease like vaccination, quarantine and many more. Adjusting some parameters values like vaccination rate or quarantine effectiveness helps researchers determine the best ways to reduce disease transmission. These parameters also help estimate the basic reproduction number, which indicates whether a disease will grow or fade out. If $R_0 > 1$, the infection spreads, but if $R_0 < 1$, it declines [16] [15] [17]. The parameters which we are utilizing in our model are fundamental to any mathematical models in biology. They help researchers to describe their processes, predict future trends, and design efficient strategies. Reliable models in domains like epidemiology depend on accurate parameter estimation and analysis, ecology, and biomedical research, ensuring better understanding and informed decision-making.
A detailed explanation of all parameters used in this model is provided below:

Table 1 ***Description of Parameters***

Parameters	Description
α	The rate of susceptibility
β_1	Contact rate of susceptible class to exposed class
β_2	Contact rate of susceptible class to quarantine class
β_3	Contact rate of vaccinated class to exposed class
β_4	Rate at which vaccinated individual moves to quarantine class
ω	Transmission rate of exposed individuals to infected class
μ	The mortality rate of exposed class to death class
ε	Transferred rate of quarantine class to susceptible class
η	Vaccination rate
χ	The mortality rate of vaccinated individual to death class
γ	Recovery rate
δ	The mortality rate of infected individual to death class
σ	The mortality rate of quarantine individual to death class
φ	Transferred rate of quarantine individual to infectious class

Depending on the proposed SEQIRVP model, the disease transmission dynamics are formulated with the following seven differential equations. The system of non-linear differential equations is presented as follows which is originated from the *Figure 1*,

$$\begin{aligned}
\frac{dS}{dt} &= -\frac{(\beta_1+\beta_2)}{N}SI + \varepsilon Q + \alpha R - \eta S \\
\frac{dE}{dt} &= \left(\frac{\beta_1 S + \beta_3 V}{N}\right) I - \omega E - \mu E \\
\frac{dQ}{dt} &= \left(\frac{\beta_2 S + \beta_4 V}{N}\right) I - \varepsilon Q - \varphi Q - \sigma Q \\
\frac{dI}{dt} &= \omega E - \gamma I - \delta I + \varphi Q \\
\frac{dR}{dt} &= \gamma I - \alpha R \\
\frac{dV}{dt} &= \eta S - \frac{(\beta_3+\beta_4)}{N}VI - \chi V
\end{aligned} \qquad (1)$$

$$\frac{dP}{dt} = \mu E + \sigma Q + \delta I + \chi V$$

where,

$S(0) \geq 0,\ E(0) \geq 0,\ I(0) \geq 0,\ Q(0) \geq 0,\ V(0) \geq 0,\ R(0) \geq 0,\ P(0) \geq 0$

and $S + E + Q + I + R + V + P = 1$ for all $t \geq 0$ when

$s(t) = \frac{S(t)}{N},\ e(t) = \frac{E(t)}{N},\ q(t) = \frac{Q(t)}{N},\ i(t) = \frac{I(t)}{N},\ r(t) = \frac{R(t)}{N},\ v(t) = \frac{V(t)}{N},$

$p(t) = \frac{P(t)}{N}$

and all parameters in the model are assumed to be positive when $t \geq 0$.

Now we can assume the system as follows,

$$\begin{aligned}
\frac{ds(t)}{dt} &= -(\beta_1 + \beta_2)s(t)i(t) + \varepsilon q(t) + \alpha r(t) \\
&\quad - \eta s(t) \\
\frac{de(t)}{dt} &= \big(\beta_1 s(t) + \beta_3 v(t)\big)i(t) - \omega e(t) - \mu e(t) \\
\frac{dq(t)}{dt} &= \big(\beta_2 s(t) + \beta_4 v(t)\big)i(t) - \varepsilon q(t) - \varphi q(t) \\
&\quad - \sigma q(t) \\
\frac{di(t)}{dt} &= \omega e(t) - \gamma i(t) - \delta i(t) + \varphi Q(t) \qquad (2) \\
\frac{dr(t)}{dt} &= \gamma i(t) - \alpha r(t) \\
\frac{dv(t)}{dt} &= \eta s(t) - (\beta_3 + \beta_4)v(t)i(t) - \chi v(t) \\
\frac{dp(t)}{dt} &= \mu e(t) + \sigma q(t) + \delta i(t) + \chi v(t)
\end{aligned}$$

Stability Analysis

In this part we are going to discuss the existence of SEQIRVP model and analyze its stability at different equilibrium points.

Equilibrium State

In an epidemic model, the basic reproduction number determines whether the population is in Disease-Free Equilibrium (ξ_0) or Endemic Equilibrium (ξ^*) [17] [18] [19].

Disease-free Equilibrium Point

The disease-free equilibrium point occurs when there is no active infection in the community. This indicates that the number of individuals-exposed, quarantined,

infected, or recovered is zero [17] [19]. So, the disease-free equilibrium of the system becomes

$$\xi_0 = (1,0,0,0,0,\frac{\eta}{\gamma})$$

Endemic Equilibrium Point

To get the endemic equilibrium, point of the system we set,

$$\frac{ds(t)}{dt} = 0, \frac{de(t)}{dt} = 0, \frac{dq(t)}{dt} = 0, \frac{di(t)}{dt} = 0, \frac{dr(t)}{dt} = 0, \frac{dv(t)}{dt} = 0, \frac{dp(t)}{dt} = 0$$

Then we get,

$$-(\beta_1 + \beta_2)s(t)i(t) + \varepsilon q(t) + \alpha r(t) - \eta s(t) = 0$$

$$(\beta_1 s(t) + \beta_3 v(t))i - \omega e(t) - \mu e(t) = 0$$

$$(\beta_2 s(t) + \beta_4 v(t))I - \varepsilon q(t) - \varphi q(t) - \sigma q(t) = 0$$

$$\omega e(t) - \gamma i(t) - \delta i(t) + \varphi Q(t) = 0$$

$$\gamma i(t) - \alpha r(t) = 0$$

$$\eta s(t) - (\beta_3 + \beta_4)v(t)i(t) - \chi v(t) = 0$$

$$\mu e(t) + \sigma q(t) + \delta i(t) + \chi v(t) = 0$$

After solving these equations simultaneously,

$$s = \frac{-\varepsilon q - \alpha r + \eta s}{-(\beta_1 + \beta_2)i} = S^*$$

$$e = \frac{(\beta_1 s + \beta_3 v)i}{\omega + \mu} = E^*$$

$$q = \frac{(\beta_2 s + \beta_4 v)i}{\varepsilon + \varphi + \sigma} = Q^*$$

$$i = \frac{\varphi + \omega}{\gamma + \delta} = I^*$$

$$r = \frac{\gamma}{\alpha} i = R^*$$

$$v = \frac{\lambda v - \eta s}{-(\beta_3 + \beta_4)i} = V^*$$

Thus, the endemic equilibrium points if the system is

$$\xi^* = \left(\frac{-\varepsilon q - \alpha r + \eta s}{-(\beta_1 + \beta_2)i}, \frac{(\beta_1 s + \beta_3 v)i}{\omega + \mu}, \frac{(\beta_2 s + \beta_4 v)I}{\varepsilon + \varphi + \sigma}, \frac{\varphi + \omega}{\gamma + \delta}, \frac{\gamma}{\alpha} i, \frac{\eta s}{\lambda + (\beta_3 + \beta_4)i}\right)$$

Basic Reproduction Number

The basic reproduction number determines the possibility of a disease spreading over an entire population. It is the average number of new infections induced by a single infected individual in a population. The value of R_0 influences if an outbreak spreads or dies. If $R_0 > 1$, the disease endemic equilibrium is asymptotically stable. This means that the illness will spread and persist in the population, perhaps leading to a massive epidemic. If $R_0 < 1$, the disease-free equilibrium is globally asymptotically stable. This indicates that infection will gradually drop and there will be no outbreak, implying that the population will finally disappear [12] [13]. We use Next Generation Matrix (NGM) method at disease free equilibrium point (at which there is no infection in the population) to find the basic reproduction number [16]. The basic reproduction number of the system is given below:

$$\frac{dx}{dt} = F(x) - V(x)$$

where, $f = \begin{bmatrix} (\beta_1 s + \beta_3 v)i \\ (\beta_2 s + \beta_4 v)i \\ 0 \end{bmatrix}$ and $v = \begin{bmatrix} (\omega + \mu)e \\ (\varepsilon + \varphi + \sigma)q \\ -\omega e + (\gamma + \delta)i - rv - \varphi q \end{bmatrix}$

For this we compute the product of two matrices F and V^{-1} where F represents transmission part and V represents transition part throughout the compartment. Here the matrices are,

$$F = \begin{bmatrix} 0 & 0 & (\beta_1 + \beta_3) \\ 0 & 0 & (\beta_2 + \beta_4) \\ 0 & 0 & 0 \end{bmatrix}, V = \begin{bmatrix} \omega + \mu & 0 & 0 \\ 0 & \varepsilon + \varphi + \sigma & 0 \\ -\omega & -\varphi & (\gamma + \delta) \end{bmatrix}$$

The next generation matrix for the system is

$$FV^{-1} = \begin{bmatrix} \frac{\omega(\beta_1 + \beta_3)}{(\omega + \mu)(\gamma + \delta)} & \frac{\varphi(\beta_1 + \beta_3)}{(\gamma + \delta)(\varepsilon + \varphi + \sigma)} & \frac{\beta_1 + \beta_3}{\gamma + \delta} \\ \frac{\omega(\beta_2 + \beta_4)}{(\omega + \mu)(\gamma + \delta)} & \frac{\varphi(\beta_2 + \beta_4)}{(\gamma + \delta)(\varepsilon + \varphi + \sigma)} & \frac{\beta_2 + \beta_4}{\gamma + \delta} \\ 0 & 0 & 0 \end{bmatrix}$$

The eigenvalues of the matrix FV^{-1} is obtained by evaluating the equation

$$|\lambda I - FV^{-1}| = 0$$

where is λ the eigenvalue and I is the identity matrix and we get

$$\lambda_1 = 0, \qquad \lambda_2 = 0, \qquad \lambda_3 = \frac{\omega(\beta_1 + \beta_3)}{(\omega + \mu)(\gamma + \delta)} + \frac{\varphi(\beta_2 + \beta_4)}{(\gamma + \delta)(\varepsilon + \varphi + \sigma)}$$

As R_O is the largest eigenvalue of the next generation matrix thus we have

$$R_0 = \frac{\omega(\beta_1 + \beta_3)}{(\omega + \mu)(\gamma + \delta)} + \frac{\varphi(\beta_2 + \beta_4)}{(\gamma + \delta)(\varepsilon + \varphi + \sigma)} \qquad (3)$$

which is the systems required basic reproduction number.

Theorem 1

When $R_0 < 1$, the disease-free equilibrium ζ_0 is locally asymptotically stable. When $R_0 > 1$, the disease-free equilibrium ζ_0 is an unstable saddle point.

Proof:

To determine the stability of DFE at the point ζ_0, we calculate the Jacobian matrix of the system,

$$J(\zeta_0) = \begin{pmatrix} -\eta & 0 & 0 & \varepsilon & -(\beta_1+\beta_2) & \alpha \\ \eta & -\chi & 0 & 0 & 0 & 0 \\ 0 & 0 & -(\omega+\mu) & 0 & \beta_1 & 0 \\ 0 & 0 & 0 & -(\varepsilon+\varphi+\sigma) & \beta_2 & 0 \\ 0 & 0 & \omega & \varphi & -(\gamma+\delta) & 0 \\ 0 & 0 & 0 & 0 & \gamma & -\alpha \end{pmatrix}$$

Now we have to demonstrate all the eigenvalues of $J(\zeta_0)$ which are negative. Now we have to solve the characteristic equation of $J(\zeta_0)$ which is

$$|\lambda I - J| = 0$$

$$\Rightarrow \begin{vmatrix} \lambda+\eta & 0 & 0 & \varepsilon & -(\beta_1+\beta_2) & \alpha \\ \eta & \lambda+\chi & 0 & 0 & 0 & 0 \\ 0 & 0 & \lambda+(\omega+\mu) & 0 & \beta_1 & 0 \\ 0 & 0 & 0 & \lambda+(\varepsilon+\varphi+\sigma) & \beta_2 & 0 \\ 0 & 0 & \omega & \varphi & \lambda+(\gamma+\delta) & 0 \\ 0 & 0 & 0 & 0 & \gamma & \lambda+\alpha \end{vmatrix} = 0$$

After solving this determinant, we get,

$$(\lambda+\eta)(\lambda+\chi)(\lambda+\alpha)[(\lambda+\omega+\mu)(\lambda+\gamma+\delta)(\lambda+\varepsilon+\varphi+\sigma) - \beta_2\varphi(\lambda+\omega+\mu) - \beta_1\omega(\lambda+\varepsilon+\varphi+\sigma)] = 0$$

Which gives us the $\lambda_1 = -\eta$, $\lambda_2 = -\chi$, $\lambda_3 = -\alpha$ and other eigenvalues are determined by the following equation

$$\lambda^3 + \lambda^2(X+Y+Z) + \lambda(XY+YZ+ZX-\beta_2\varphi-\beta_1\omega) + XYZ - \beta_2\varphi X - \beta_1\omega Z = 0$$

where, $X = \omega+\mu,\ Y = \gamma+\delta,\ Z = \varepsilon+\varphi+\sigma.$

It can be written as the polynomial equation of λ,

$\lambda^3 + M\lambda^2 + P\lambda + Q = 0$;

where, $M = X+Y+Z$

$$P = XY + YZ + ZX - \beta_2\varphi - \beta_1\omega$$

$$Q = XYZ - \beta_2\varphi X - \beta_1\omega Z$$

Now by using the Routh Hurwitz criterion test, all the roots of λ are either negative or have negative real part if and only if following conditions hold:

$$M > 0, P > 0, Q > 0 \; and \; MP > Q.$$

So now we get,

$M = X + Y + Z > 0$

$P = XY + YZ + ZX - \beta_2\varphi - \beta_1\omega = XY(1 - R_0) + YZ(1 - R_0) + XZ + \varphi\beta_4 + \omega\beta_3 + \frac{X}{Z}\varphi(\beta_4 + \beta_2) + \frac{Z}{X}\omega(\beta_1 + \beta_3) > 0$; if $R_0 < 1$.

$Q = XYZ - \beta_2\varphi X - \beta_1\omega Z = XYZ(1 - R_0) + \varphi X\beta_4 + \omega Z\beta_3 > 0$; if $R_0 < 1$.

and

$MP - Q = (X + Y + Z)(XY + YZ + ZX - \beta_2\varphi - \beta_1\omega) - XYZ + \beta_2\varphi X + \beta_1\omega Z = (X^2Y + +XY^2 + Y^2Z + YZ^2)(1 - R_0) + X^2Z + 2XYZ + XZ^2 + \omega\beta_3(X + Y) + \varphi\beta_4(Y + Z) + \frac{X^2+XY}{Z}\varphi(\beta_2 + \beta_4) + \frac{YZ+Z^2}{X}\omega(\beta_1 + \beta_3) > 0$; if $R_0 < 1$.

From the above expressions, it is proved that when $R_0 < 1$, the conditions of the Routh Hurwitz Criterion are satisfied. As a result, the Disease-Free Equilibrium ζ_0 of the system is Locally Asymptotically Stable [8] [20].

Theorem 2

When $R_0 > 1$, the endemic equilibrium is ζ^* is locally asymptotically stable.

Proof:

The Jacobian matrix of the system $\zeta^* = (S^*, E^*, Q^*, I^*, R^*, V^*, P^*)$ is J(ζ^*)

$$= \begin{bmatrix} -(\beta_1 + \beta_2)I - \eta & 0 & 0 & \varepsilon & -(\beta_1 + \beta_2)S & \alpha \\ \eta & -(\beta_3 + \beta_4)I - \chi & 0 & 0 & -(\beta_3 + \beta_4)V & 0 \\ \beta_1 I & \beta_3 I & -(\omega + \mu) & 0 & (\beta_1 S + \beta_3 V) & 0 \\ \beta_2 I & \beta_4 I & 0 & -(\varepsilon + \varphi + \sigma) & \beta_2 S + \beta_4 V & 0 \\ 0 & r & \omega & \varphi & -(\gamma + \delta) & 0 \\ 0 & 0 & 0 & 0 & \gamma & -\alpha \end{bmatrix}$$

Now, trace of $J(\zeta^*)$ is,

$trJ(\xi^*) = -(\beta_1 + \beta_2)I - \eta - \beta_3 I - \beta_4 I - \chi - (\omega + \mu) - (\varepsilon + \varphi + \sigma) - (\gamma + \delta) - \alpha < 0$

And

$det\, J(\zeta^*) = [(\beta_1+\beta_2)I+\eta]\{((\beta_3+\beta_4)I+\chi)(\omega+\mu)(\varepsilon+\varphi+\sigma)\alpha(\gamma+\delta)+(\beta_3+\beta_4)v\beta_3 I(\varepsilon+\varphi+\sigma)\omega\alpha+(\beta_3+\beta_4)v(\omega+\mu)\beta_4 I\alpha\varphi\}+\varepsilon\eta(\beta_1 S+\beta_3 V)\beta_4 I\omega\alpha+\varepsilon\{(\beta_3+\beta_4)I+\chi\}[\beta_1 I(\beta_2 S+\beta_4 V)\omega\alpha+(\beta_1 S+\beta_3 V)\beta_2 I\omega\alpha]+\varepsilon(\beta_3+\beta_4)v\beta_1 I\beta_4 I\omega\alpha+(\beta_1+\beta_2)\delta\eta[\beta_3 I(\varepsilon+\varphi+\sigma)\omega\alpha+(\omega+\mu)\beta_4 I\alpha\varphi]+(\beta_1+\beta_2)\delta\beta_1 I\{(\beta_3+\beta_4)I+\chi\}[(\varepsilon+\varphi+\sigma)\omega\alpha+(\omega+\mu)\beta_2 I\alpha\varphi]-[((\beta_1+\beta_2)I+\eta)((\beta_3+\beta_4)I+\chi)\{(\omega+\mu)(\beta_2 S+\beta_4 V)\alpha\varphi+(\beta_1 S+\beta_3 V)(\varepsilon+\varphi+\sigma)\omega\alpha\}+\varepsilon\eta\{\beta_3 I(\beta_2 S+\beta_4 V)\omega\alpha+(\omega+\mu)\beta_4 I(\gamma+\delta)\alpha\}+\varepsilon\{(\beta_3+\beta_4)I+\chi\}(\omega+\mu)\beta_2 I(\gamma+\delta)\alpha+\varepsilon(\beta_3+\beta_4)v\beta_3 I\beta_2 I\omega\alpha+\alpha\eta\{\beta_3 I(\varepsilon+\varphi+\sigma)\omega\gamma+(\omega+\mu)\beta_4 I\varphi\gamma\}+\alpha\{(\beta_3+\beta_4)I+\chi\}\gamma\{\beta_1 I(\varepsilon+\varphi+\sigma)\omega+(\omega+\mu)\beta_2 I\varphi\}]=$

$P-Q>0$.

Provided the condition $P>Q$ then $R_0>1$ where

$P=[(\beta_1+\beta_2)I+\eta]\{((\beta_3+\beta_4)I+\chi)(\omega+\mu)(\varepsilon+\varphi+\sigma)\alpha(\gamma+\delta)+(\beta_3+\beta_4)v\beta_3 I(\varepsilon+\varphi+\sigma)\omega\alpha+(\beta_3+\beta_4)v(\omega+\mu)\beta_4 I\alpha\varphi\}+\varepsilon\eta(\beta_1 S+\beta_3 V)\beta_4 I\omega\alpha+\varepsilon\{(\beta_3+\beta_4)I+\chi\}[\beta_1 I(\beta_2 S+\beta_4 V)\omega\alpha+(\beta_1 S+\beta_3 V)\beta_2 I\omega\alpha]+\varepsilon(\beta_3+\beta_4)v\beta_1 I\beta_4 I\omega\alpha+(\beta_1+\beta_2)\delta\eta[\beta_3 I(\varepsilon+\varphi+\sigma)\omega\alpha+(\omega+\mu)\beta_4 I\alpha\varphi]+(\beta_1+\beta_2)\delta\beta_1 I\{(\beta_3+\beta_4)I+\chi\}[(\varepsilon+\varphi+\sigma)\omega\alpha+(\omega+\mu)\beta_2 I\alpha\varphi]$ and $Q=[((\beta_1+\beta_2)I+\eta)((\beta_3+\beta_4)I+\chi)\{(\omega+\mu)(\beta_2 S+\beta_4 V)\alpha\varphi+(\beta_1 S+\beta_3 V)(\varepsilon+\varphi+\sigma)\omega\alpha\}+\varepsilon\eta\{\beta_3 I(\beta_2 S+\beta_4 V)\omega\alpha+(\omega+\mu)\beta_4 I(\gamma+\delta)\alpha\}+\varepsilon\{(\beta_3+\beta_4)I+\chi\}(\omega+\mu)\beta_2 I(\gamma+\delta)\alpha+\varepsilon(\beta_3+\beta_4)v\beta_3\beta_2 I^2\omega\alpha+\alpha\eta\{\beta_3 I(\varepsilon+\varphi+\sigma)\omega\gamma+(\omega+\mu)\beta_4 I\varphi\gamma\}+\alpha\{(\beta_3+\beta_4)I+\chi\}\gamma\{\beta_1 I(\varepsilon+\varphi+\sigma)\omega+(\omega+\mu)\beta_2 I\varphi\}]$

According to [15] [19], the endemic equilibrium ξ^* of the system (4.1) has a negative real part. Thus, when $R_0>1$, the endemic equilibrium ξ^* of the system (4.1) is locally asymptotically stable.

Sensitivity Analysis

Sensitivity analysis is crucial in epidemiological modeling as it identifies key parameters that significantly impact outcomes. This method is essential for understanding how model inputs affect predictions, as correct predictions are vital for building effective control strategies. It identifies the characteristics that have a major impact on disease transmission, allowing for targeted control efforts like vaccination programs, quarantine protocols, thereby improving public health strategies. Additionally, it helps to identify those areas where additional data or

research is needed to refine the model and improve the accuracy of model's prediction. This approach is particularly important for any infectious diseases, where inaccurate predictions can lead to severe public health consequences.

The study aims to assess the sensitivity index for the basic reproduction number (R_0), a fundamental epidemiological metric that determines whether an infectious disease will spread or decline in a population. We followed the process of finding sensitivity analysis as in [19]. Here, R_0 represents that how many people, on average, a single infected person is likely to infect in a completely susceptible population. For understanding how R_0 influenced by the various factors that change disease transmission—things like how easily the disease spreads, how long someone remains infectious, or how often people come into contact with one another, we use sensitivity index, which measures the relative impact of each parameter on R_0. This allows us to identify which parameters have the greatest potential to decrease the spread of infectious disease. For example, if the sensitivity analysis shows that reducing contact rates has a huge impact on R_0, then measures like social distancing or lockdowns might be prioritized. On the other hand, if improving recovery rates through better healthcare is more influential, then investing in medical resources becomes the key strategy. By analyzing the rate of characteristics, we may better understand the necessary measures to minimize disease transmission. Changing a parameter with a high sensitivity index can significantly impact disease spread rate. If a parameter has a low sensitivity index, tiny modifications may not have significant influence.

The most challenging part of mathematical modeling in biology is estimating parameters due to several complexities. We have obtained our data from some reference; some are estimated using information from credible sources and others have been assumed [21].

***Table 2* Initial value of variables**

Notations	Values	Data source	Notations	Values	Data source
N	174748372	World meter	I	533444	[21]
S	174648322	N-Q-E-I-R-V-P	R	478836	[21]
Q	100000	Assumed	V	27	[21]
E	20500	Assumed	P	8072	[21]

The sensitivity index of the basic reproduction number with respect to its associated parameter can be formulated as [15] [19],

$$I_{P_i}^{R_0} = \frac{\partial R_0}{\partial P_i}\frac{P_i}{R_0} \quad (4)$$

Now we are calculating the sensitivity index of reproduction number for our systems parameters. We have used the parameters value from the ***Table 3*** and also used the model's reproduction number that we have calculated which is

$$R_0 = \frac{\omega(\beta_1+\beta_3)}{(\omega+\mu)(\gamma+\delta)} + \frac{\varphi(\beta_2+\beta_4)}{(\gamma+\delta)(\varepsilon+\varphi+\sigma)}$$

Table 3 ***Values of parameters***

Parametres	Value	Data source	Parametres	Value	Data source
$\boldsymbol{\alpha}$	0.00001	Assumed	$\boldsymbol{\mu}$	0.125	[22]
$\boldsymbol{\beta_1}$	0.002824	Estimated	$\boldsymbol{\varphi}$	0.67	Assumed
$\boldsymbol{\beta_2}$	0.24	Assumed	$\boldsymbol{\sigma}$	0.01	Assumed
$\boldsymbol{\beta_3}$	0.00059	Assumed	$\boldsymbol{\gamma}$	0.0105	Estimated
$\boldsymbol{\beta_4}$	0.00001	Assumed	$\boldsymbol{\delta}$	0.000044	Estimated
$\boldsymbol{\varepsilon}$	1.2	Assumed	$\boldsymbol{\eta}$	0.0056	Assumed
$\boldsymbol{\omega}$	0.1735	Assumed	$\boldsymbol{\chi}$	0.00041	Assumed

Now we have got

$I_{\beta_1}^{R_0} = \frac{\partial R_0}{\partial \beta_1}\frac{\beta_1}{R_0} = \frac{\omega}{(\omega+\mu)(\gamma+\delta)}\frac{\beta_1}{R_0}$, and we put the values of the parameter and we get 0.0187

$I_{\beta_2}^{R_0} = \frac{\partial R_0}{\partial \beta_2}\frac{\beta_2}{R_0} = \frac{\varphi}{(\gamma+\delta)(\varepsilon+\varphi+\sigma)}\frac{\beta_2}{R_0}$, and we get 0.9754

$I_{\beta_3}^{R_0} = \frac{\partial R_0}{\partial \beta_3}\frac{\beta_3}{R_0} = \frac{\omega}{(\omega+\mu)(\gamma+\delta)}\frac{\beta_3}{R_0}$, we get 0.00589

$I_{\gamma}^{R_0} = \frac{\partial R_0}{\partial \gamma}\frac{\gamma}{R_0} = -\frac{1}{(\gamma+\delta)^2}\left[\frac{\omega(\beta_1+\beta_3)}{\omega+\mu} + \frac{\varphi(\beta_2+\beta_4)}{\varepsilon+\varphi+\sigma}\right]\frac{\gamma}{R_0}$, and we obtain -0.996

$I_{\delta}^{R_0} = \frac{\partial R_0}{\partial \delta}\frac{\delta}{R_0} = -\frac{1}{(\gamma+\delta)^2}\left[\frac{\omega(\beta_1+\beta_3)}{\omega+\mu} + \frac{\varphi(\beta_2+\beta_4)}{\varepsilon+\varphi+\sigma}\right]\frac{\delta}{R_0}$, and we get the value -0.0042

$I_{\varphi}^{R_0} = \frac{\partial R_0}{\partial \varphi}\frac{\varphi}{R_0} = -\frac{1}{\gamma+\delta}\frac{(\beta_2+\beta_4)(\varepsilon+\varphi+\sigma)-\varphi(\beta_2+\beta_4)}{(\varepsilon+\varphi+\sigma)^2}\frac{\varphi}{R_0}$, we obtain -0.628

$I_{\sigma}^{R_0} = \frac{\partial R_0}{\partial \sigma}\frac{\sigma}{R_0} = -\frac{1}{\gamma+\delta}\left[\frac{\varphi(\beta_2+\beta_4)}{(\varepsilon+\varphi+\sigma)^2}\right]\frac{\sigma}{R_0}$, we get -0.0052

The sensitivity indices $I_{\beta_1}^{R_0}$, $I_{\beta_2}^{R_0}$, $I_{\beta_3}^{R_0}$, $I_{\gamma}^{R_0}$, $I_{\varphi}^{R_0}$, $I_{\sigma}^{R_0}$ and $I_{\delta}^{R_0}$ measure that how much changes in specific parameters influenced the basic reproduction number, R_0 and which factors have the biggest impact on Covid-19 transmission. A positive

sensitivity index for the transmission rate $I_{\beta_1}^{R_0} = 0.0187$, which is quite low. It means that the changes of β_1 have a minimal impact on R_0. This might seem surprising because early public health interventions to control the Covid-19 was social distancing and wearing masks, were mainly aimed for reducing the spread of disease among the general public. But in our study, we started to collect our data from January 2021, when vaccines were already uprising in Bangladesh and people already spent a year with preventive behaviors i.e. mask-wearing, social distancing and hygiene practices had become common habits. So, even if β_1 is high it doesn't have that much impact on reproduction number.

One of the most significant parameters according to our result is β_2. The sensitivity index of β_2 is 0.9754; represents that slightly changes in this parameter had the most impact on reproduction number. It suggests that quarantine policies were most important for controlling the spread of disease. As our data is counted from 27th January of 2021, a small portion of the population got vaccinated, it took time to develop the immunity and its impact on transmission wasn't visible. So, this result suggests that transmission within quarantine settings was a significant factor which highlights the importance of quarantine protocols-for example ensuring isolation, reducing overcrowding from high-risk environments like factories, markets, public transport, and workplaces which remained major sources of transmission, as many people had no choice but to be in close contact, keeping the virus circulating.

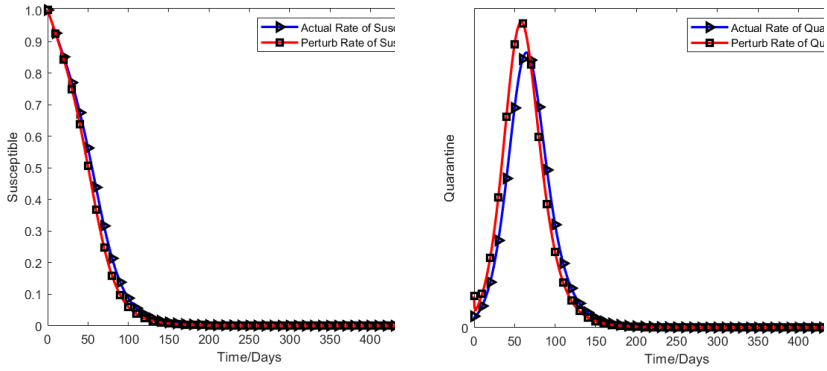

Figure 2 ***Actual and perturbated rates of susceptibility (left side) and quarantine (right side)***

The sensitivity of β_3 is 0.00589 which represents the transition from vaccinated individuals to exposed. It suggests that increasing vaccination rates had only a minor immediate effect on transmission of the disease. This could be because vaccines were already reducing susceptibility, preventing infections, making vaccinated individuals who had already received their doses were already benefiting some level of protection. Even if they were exposed, they might have had shorter infection period. This aligns with real-world scenario where vaccines not only prevent infections but also limit onward transmission. Therefore, rather than viewing the low sensitivity of β_3 as an indication that vaccination had little effect, this likely reflects the fact that vaccinated individuals were playing a much

smaller role to spread the transmission and indeed it was a crucial long-term strategy, and as more people became vaccinated, its effect on slowing down transmission over time.

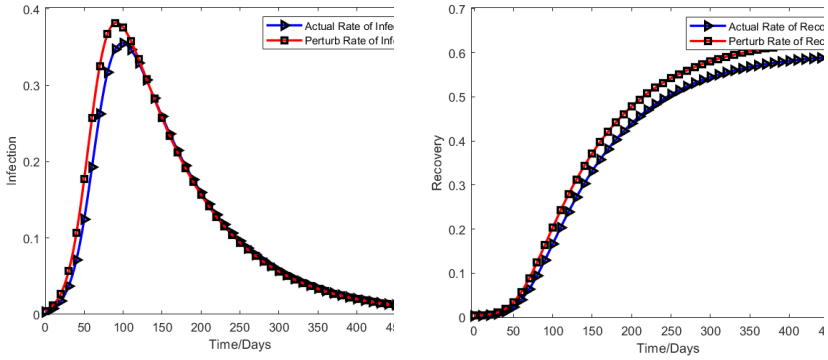

Figure 3 ***Actual and perturbated rates of infection (left side) and recovery (right side)***

One of the most critical parameters influencing reproduction number is γ, which represents the transition from infected to recovered. With a strong negative sensitivity index of -0.996 means that higher recovery rate led to fewer infection as people spend less time being infectious and spreading the virus which means it strongly reduces the reproduction number. It shows the significance of the improved healthcare access, and effective treatments in reducing transmission rates. So, this sensitivity index reinforces the importance of strengthening healthcare system during a pandemic to ensure that infected individuals receive effective treatments.

The sensitivity index of φ, which represents the transition from quarantine to becoming infectious, had a negative moderate sensitivity index of -0.628. This index highlights the importance of effective quarantine strategies i.e. if we can slow down this transition, we can reduce R_0. If quarantined individuals were isolated properly and did not progress to the infectious stage while still in contact with others, the overall transmission rate will decrease. This suggests that strict quarantine enforcement, timely testing was crucial in keeping transmission under control. This finding emphasizes the need for efficient testing and isolation protocols to minimize the risk of transmission during quarantine.

The parameter δ representing the transition from infected to death, has a small negative sensitivity index of -0.0042. This suggests that deaths alone had a low impact on transmission as it only removes a small portion of the infectious population and also individuals in severe or fatal cases were typically isolated in hospitals or at home, meaning they were not contributing significantly to further infections. However, it shows that reducing deaths alone won't necessarily slow the spread of the virus.

These outcomes give a clear idea of how COVID-19 was spreading in Bangladesh and what factors were most effective in controlling it after the starting of

vaccination. The biggest factor driving transmission was quarantine effectiveness. Vaccination had a limited immediate impact, likely because the rollout had just started, so not enough people were protected yet. Another key issue was the quarantine-to-infected transition, showing that people breaking quarantine or not getting tested in time contributed to continued spread. Recovery rate was one of the most important factors in reducing transmission, highlighting how critical good healthcare and treatment were in bringing down case numbers. On the other hand, deaths had little effect on transmission, since severely ill patients were already isolated and not spreading the virus. Overall, these results show that effective quarantine, early testing, better treatment, and expanding vaccination efforts were the key to controlling COVID-19 in Bangladesh during this period.

Numerical Simulation

This graph represents the dynamic behavior of SEQIRVP model. This model simulates and predicts how an infectious disease spreads and affects a population considering some important factors like vaccination, quarantine and many more. To make the simulation more accurate we used the RK4 (Runge kutta 4th order) method in MATLAB [23]. This RK4 method was chosen because it provides more precise and stable solution when solving the differential equations which makes our prediction more reliable [24], [25], [26]. After that we have compared this result with real world data from Bangladesh so that we can analyze how accurately this model represents the reality and what factors matter more for this fluctuation in Covid-19.

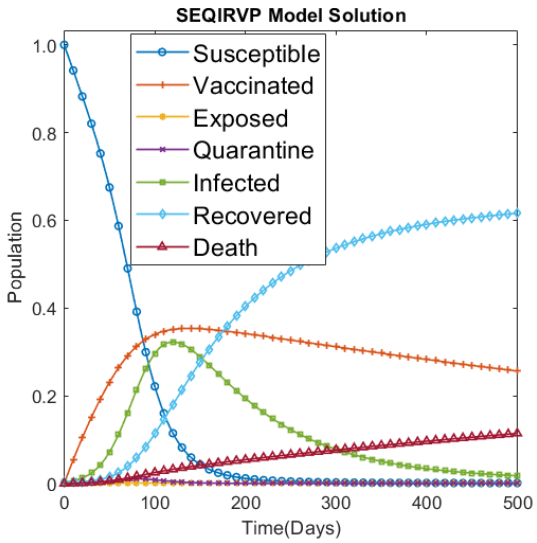

Figure 4 ***Simulation of SEQIRVP model***

This figure shows that at first almost the entire population are considering as susceptible and after sometime the rate of susceptible are rapidly decreasing when the disease starts to spread as they are moving into infected or exposed compartment and also for getting vaccinated. From this figure we can also see that the infection rate has an initial slow rise in the first 50 days and then rises sharply to its peak around 150 day and then gradually decreases as the disease is bought to under control. The rate of infection was going to be its peak position which was in around 150 days (in the month of July). This sharp increase occurs as more people who were exposed to the virus becomes infectious. Based on the actual circumstances in Bangladesh, we can see that in 28th July (counting from January 2021) there was highest number of active cases. After almost 250 days the rate of infection starts to decline steadily. This declination happened because of an increase in the recovery rate due to vaccination. We can see this from our real data that infection rate was decreasing slowly at the month of September. But in some cases, there might be some fluctuations in infection rate due to new variants or decreasing of immunity from vaccines over time. This might cause the temporary increase of infection rate but overall vaccination leads to a reduction in infection rate.

At the beginning, the recovery rate remains relatively low as the infection is still spreading among the population. People take time to improve from exposed to infected and finally to recovered. In this scenario, the recovery curve begins to rise more sharply in around 100^{th} day. As the infection rate peaks, people started to recover and it happened when recovery rate rises sharply; as many people who were infected earlier are now becoming recovered. According to our data, we can see the same thing. From march 2021, recovered rate is increasing due to vaccination or quarantine. At one point recovery curve surpasses the infected curve; this is when more people are recovering than getting infected. It indicates the epidemic has mostly been under control which means most of the people who were infected have either recovered due to vaccination or in some cases, passed away. Abd that's why our model shows a steady rise in the recovered population after 250 days.

The death rate represents the increasing calamity due to the infection. The death population grow more slowly and steadily over time. At the beginning of the epidemic the death rate was lowest as the infection is just beginning to spread. As the infection spread and the number of infectious populations increases, the death rate gradually rises throughout the 500-day period but at a slower rate. This eventually slower rate of death is a positive indicator as its showing that the epidemic is no longer causes high mortality. By the end of the period, most of the population are either vaccinated or recovered or deceased.

At the beginning, vaccination curve starts low as the vaccination campaign just begins after recognizing the epidemics severity. The gradual rise in the vaccination rate reflects an increase in the number of vaccinated populations throughout the time. As a result, the curve rises slowly which means that a small percent of the population has been vaccinated. In this graph around day 50, the vaccination rate begins to start upward and this represents the increasing of vaccination campaign among general population. After 150 days, we can see an excessive rise in the vaccination curve which is actually in the end of July 2021 and it crosses over other compartments. This crossover means a large number of populations are being

vaccinated daily which helps to preventing new infections and reducing the spread of the virus. Between day 150 and 300, this curve starts to flatten, showing a gradual increase. It represents that a portion of population has been vaccinated already and fewer unvaccinated population remain. After 300 days, the vaccination curve begins to level off, indicates that majority of the population has been vaccinated. Consequently, new infection decreases, and the epidemic stabilizes.

The following line plot represents a comparative analysis of COVID-19, from 2021 to 2023 using four major key measures that is monthly new cases, monthly reproduction rate, monthly new deaths, and monthly people Vaccinated.

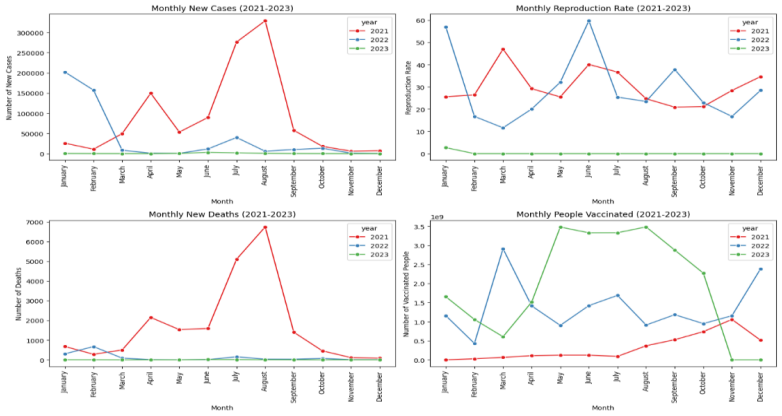

Figure 5 ***Line graph of COVID-19 new cases (top left side), reproduction rate (top right side), death (bottom left side) and vaccinated (bottom right side)***

The monthly new cases graph (top-left) highlights a significant outbreak in mid-2021, with cases reaching at over 300,000 in July-August. However, from 2022, the number of cases declined significantly, reflecting the impact of vaccination campaigns and health measures improved. Although occasional fluctuations in new cases occurred, hospitalizations and severe cases were notably lower compared to the previous year. The monthly reproduction rate (top-right) follows a similar trend, showing high fluctuations in 2021 which indicates rapid virus transmission. But from 2022 onwards, the reproduction rate stayed low stabilizes near zero. As mass vaccination efforts expanded in 2022, both infection rates and fatalities showed a significant decline. The monthly new deaths graph (bottom-left) follows similar pattern to new cases, with a sudden rise in mid-2021, rising at nearly 7,000 deaths in August. As vaccination efforts increased, deaths dropped steadily, reflecting improved protection and medical
interventions. The monthly people vaccinated graph (bottom-right) represents an inverse relationship with new cases and deaths. The number of vaccinated

individuals saw a sharp rise from early 2021, peaking between mid-2022 and early 2023 [21].
The data suggests that vaccinations played a crucial role in reducing both infections and fatalities. The overall trend confirms that vaccinations were a critical factor in reducing both infections and mortality, highlighting their role in controlling public health crises and preventing future outbreaks [27], [28].

Conclusion:

This study introduces a newly constructed SEQIRVP model, which has proven to be a useful tool for investigating the transmission of COVID-19. This model has a more refined approach by including compartments for vaccinated individuals and fatalities, allowing for a better understanding of disease progression and the impact of various public health measures such as quarantine and vaccinations. Through this model, it became clear that quarantine tactics and recovery rates were critical in limiting the virus's spread in Bangladesh. Quarantine methods were especially crucial in limiting transmission, particularly in the early phases of the epidemic.
While vaccination initially had a minimal effect on transmission, its importance grew as more people in Bangladesh were immunized. Because of the delayed distribution, the effect of vaccination was more subtle at first. However, as vaccination campaigns increased and more individuals were protected, the infection rate in Bangladesh gradually decreased. This progressive decrease in instances is consistent with real-world data from Bangladesh, demonstrating the long-term importance of vaccination as a crucial method for epidemic control [29].
The sensitivity analysis also found that in Bangladesh, quarantine measures and recovery rates were the most important factors determining COVID-19 transmission. The data showed that vaccination alone, especially early on, was insufficient to prevent transmission. This shows that controlling the pandemic requires a combination of methods [30], [31], [32] [33], like as effective quarantine, healthcare advancements, and a progressive vaccine program. By modelling these tactics and using actual data from Bangladesh, we were able to gain a better understanding of how these factors combine to limit the virus spread overall. Although the study focuses on Bangladesh, the SEQIRVP model and its findings are widely applicable. The methodology can be used to other countries, particularly low- and middle-income regions that face similar challenges in health infrastructure and policy execution [34].
Overall, this study adds a useful tool to the field of mathematical epidemiology by emphasizing the significance of multi-layered interventions in pandemic response initiatives. The findings highlight that no single strategy is sufficient on its own, and that a multifaceted approach that includes quarantine, vaccinations, and healthcare preparedness is critical to preventing disease spread and reducing societal disturbance.

Acknowledgement:

The research grant PS/2023/1/23 has offered support to S. M. Saydur Rahman and Dr. Pabel Shahrear. Furthermore, the SUST Research Center has fund available to offer additional help.

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