GANIT

Available online at https://ganitjournal.bdmathsociety.org/

GANIT: Journal of Bangladesh Mathematical Society

GANIT *J. Bangladesh Math. Soc.* 43.1 (2023) 45–62
DOI: https://doi.org/10.3329/ganit.v43i1.67858

Vaccine efficacy of COVID-19 in Bangladesh: Does vaccination prevent the pandemic?

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ABSTRACT

We adopted a commonly used epidemiologic model known as Susceptible-Infectious-Recovered (SIR) by adding a compartment vaccination to locate the COVID-19 hotspot in Bangladesh. Using data from the Directorate General of Health Services (DGHS), we utilized the Susceptible-Vaccinated-Infectious-Recovered (SVIR) model to assess vaccine efficacy. Vaccination against COVID-19 has begun in industrialized nations and will be completed by early December 2020. Before 2023, mass vaccination for impoverished or low-income nations would be extremely difficult. Bangladesh, a lower-middle-income country, started a statewide COVID-19 immunization effort in early February 2021. This study aimed to recognize the effectiveness of vaccination on the infected population in Bangladesh. Additionally, the study aimed to create public awareness of the importance of vaccination during a pandemic.

Received: May 28, 2023 **Accepted:** July 14, 2023 **Published Online:** July 15, 2023

Keywords: SVIR model; vaccination; COVID-19; Bangladesh; data.
AMS Subject Classifications 2020: 92D25, 92D30, 97M60, 97M99.

1 Introduction

Of the three zoonotic viruses that have emerged in the last century and affect humans, Novel Coronavirus 2019 (nCoV-2019) is one, with the others being severe acute respiratory syndrome coronavirus (SARS-CoV) and Middle East respiratory syndrome coronavirus (MERS-CoV) [1]. Between 2002 and 2012, they expanded to 37 and 27 nations, respectively. According to the World Health Organization (WHO), since its discovery on December 31, 2019, in Wuhan City, Hubei Province of China, it has spread to more than 200 nations globally, with 687,868,896 confirmed cases up to May 9, 2023. WHO classified COVID-19 as a pandemic on March 11, 2020, due to its rapid spread. On March 8, 2020, two Italian returnees discovered the first instance of COVID-19 in Dhaka, and the afflicted lady was a family member of one of the two. The transmission rate was rapid from the time COVID-19 was exposed to the world. The death rate being low also helped the spread of the disease.

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In the end, the virus affected a vast number of populations. Even with a low fatality rate, COVID-19 killed more people than most other deadlier viruses [2].

The pandemic of COVID-19 is being brought under control through accelerated vaccination efforts. According to the data collected, 72.30% of the global population has been vaccinated (2^{nd} dose) as of March 2023. However, only 6% of individuals in low-income nations have been fully immunized against the coronavirus. In high-income nations, 70% of the population has received at least two doses of vaccination [3]. Most people were unconcerned about the outbreak in a heavily populated nation like Bangladesh. Not only did COVID-19 cause a health crisis, but it also severely impacted the economy. Bangladesh is yet to recover from the financial damages it caused. Despite some donations, Bangladesh had to buy many vaccines for its large population [4, 5].

To avoid and regulate the transmission of COVID-19, multiple models such as Susceptible-Infected (SI), Susceptible-Infected-Recovered (SIR), and Susceptible-Infected-Susceptible (SIS) have been employed to predict the spread of the virus within populations. In the past, various academics have attempted to anticipate epidemic situations using various epidemiological models. The authors, Dantas, Tosin, and Cunha Jr., are responsible for producing the text. This study aimed to delineate the Zika virus epidemic in Brazil by implementing the SEIR-SEIR model. Another researcher was investigating the efficacy of modeling tools during the epidemic and developing a SIR model using data from COVID-19. During the pandemic, modeling tools have been widely used to track and forecast the spread of COVID-19. In epidemiology, a significant number of SEIR model was considered, a commonly used mathematical model for epidemics, to predict the trajectory of the pandemic [6, 7]. These models certainly offer a more thorough picture of the epidemic spread, but identifying them is substantially more difficult. One reason is that they frequently have a vast number of parameters and factors, but the information is regularly boisterous and in restricted numbers, making these models inclined to overfit. At that point, taking into consideration the characteristics of this study's information, our investigation presents the SIR strategy, which is based on the visionary show of plague phenomena and is one of the foremost broadly acknowledged measurable models for determining inalienable disease scenarios and is extensively utilized to evaluate disease information between the different waves of the epidemic. These models use an ordinary differential equation that considers the population contaminated as well as the drift of individuals recouping from sickness over time [8, 9, 10].

Amid the ongoing pandemic, scholars have employed diverse analytical techniques to generate projections and forecasts pertaining to novel occurrences of COVID-19, fatalities, and recoveries. These methodologies encompass but are not limited to, statistical analysis, mathematical modeling, machine learning, and deep learning algorithms. Papastefanopoulos et al. examined different statistical and machine learning time series models to anticipate COVID-19 cases, including ARIMA, PROPHET, Holt-Winters additive model, TBAT, Deep-AR, and N-Beats. Facebook Prophet model is a well-known novel technique for its simple-to-use yet powerful concept [11]. Several research makes use of the Facebook Prophet model Forecasting Procedure. Cluster analysis techniques were performed on confirmed cases to assess whether there are homogenous groupings among districts [12, 13].

The assumption of constant parameters in compartmental models, such as SIR, SIRD, and SEIR, may only partially account for changes in real-world scenarios and can result in inaccurate predictions. To better capture the disease dynamics, consider time-varying parameter models. Due to variations in the data utilized to develop the SIRD model across different locations, its projections can be unreliable. SEIR is a complicated model that predicts less correctly as the number of model parameters grows. The SEIR model outperforms the SIR model regarding the number of occurrences anticipated. Researchers have leveraged the SIR model's superior ability in long-term forecasting compared to the regression model, along with machine learning, to develop an intelligent healthcare system to predict and prevent the spread of COVID-19. Simple models of epidemics could be better and quicker at showing how COVID-19 spreads than complicated models [14, 15].

According to the literature, the primary goal of this study is to assess the impacts of vaccination on a worldwide pandemic like COVID-19 in Bangladesh using an epidemiological model SVIR and data from the DGHS from February 2020 to October 2022 [16].

This study will also help public health decision-makers to make better preparations if another epidemic like this ever hits Bangladesh [17].

Background and Dynamics of the SVIR Model

Health issues and disease are strongly related to studying health and illness, encouraging people to give these subjects priority knowledge. Creating a healthy living environment, including access to high-quality medical care, is one of a country's main objectives. To do this, quality healthcare issues must be dealt with methodically, effectively, and effectively. Analyzing disease propagation is a crucial first step in solving these problems. By doing this, we can better grasp the issue at hand and implement the necessary solutions to raise the standard of healthcare.

A virus or bacteria that enter the body can propagate and produce health problems that, in turn, affect how a group develops socioeconomically. It is crucial to go through multiple stages, including the study and development of various diagnostic tools, medications, and vaccines, to stop the spread of disease. We can maximize our efforts to stop disease spread and its detrimental effects by reaching these benchmarks.

The patterns of disease propagation can be reviewed and analyzed using mathematical models, which are a crucial tool. Models like the Susceptible-Infected-Recovered (SIR) and Susceptible-Infected-Recovered-Susceptible (SIRS) models can describe how infectious diseases propagate quantitatively. The compartment model, also known as the SIR model or the susceptible, infected, and recovered population model, is the simplest primary method for simulating illness transmission. The vaccination process can be conceptualized as incorporating a naturally existing constituent within the epidemic base model for certain diseases.

With the improvement of medical understanding, vaccination can be used to control the spread of epidemic diseases. The SIR model can be further modified to include a fourth class, the Vaccinated population, to incorporate vaccination into the modeling of disease propagation. The Susceptible-Vaccinated-Infected-Recovered (SVIR) model, an extension of the SIR model, offers a more accurate description of disease transmission in a population where vaccination is a crucial control tool. The four classes in the SVIR model are Susceptible, Vaccinated, Infected, and Recovered. We can better understand the efficacy of vaccination programs and other control measures by simulating the spread of disease in this way.

There are two methods we can add an extension of vaccination to the SIR model. both the SVIR and SIVR models. They are comparable in adding a population class for those who have received vaccinations to the SIR model. The definition of the "Removed" class is where the two models most significantly diverge. The "Removed" class in the SIVR model covers those who have either recovered from the illness or have gotten a vaccination and are disease-immune. In contrast, the "Removed" class is defined by the SVIR model as people who have recovered from the illness or got a vaccination but do not explicitly take immunity into account. The SVIR model, on the other hand, assumes that while those who have received vaccinations have a lower risk of contracting the disease, they can still spread it to others. In general, the disease being represented and the questions being posed determine which model should be used. Both the SIVR and SVIR models can shed light on the efficacy of vaccination campaigns and other preventative measures. However, they base their predictions on various hypotheses regarding how vaccination protects against disease.

Several academics have proposed models of vaccination dynamics. According to several studies, the value of the Basic Reproduction Number (R_0) is used to identify the presence of an endemic disease. If R_0 is less than 1, the disease distribution model will be locally stable, and if R_0 is more than 1, the illness will exist. The death rate of those who contract the disease is not taken into account by the SVIR epidemic model because it was first developed to study the spread of non-lethal diseases. We may, however, draw some conclusions about how this model might relate to the spread of a fatal disease in the public sphere by researching it. In this paper, we will examine the SVIR model's application to a lethal disease epidemic in this study and discuss how it might help direct efficient preventative measures.

2 Formulation of the SVIR Model

To consider and interpret the SVIR model, we have following assumptions:

1. There is no movement inside the closed population.
2. Every category of the population experiences births, and people move into the vulnerable category.
3. In every subgroup, natural death happens at the same rate whereas getting sick can cause someone to die.
4. Recipients of the cure are unable to re-join the weak. In addition, the incubation period is short.
5. Sub-populations that were at risk received vaccinations.

6. The populations who recover will eventually receive vaccinations once they have reached a certain immunity threshold.

7. The spread of disease can occur as a result of interaction between those who are healthy and vulnerable as well as contact between those who have had vaccines.

8. Vaccine recipients develop an infection if their immunity is weakened.

In our initial analysis, we divide the total population into four compartments by dividing it based on the number of individuals in each, such as $S(t)$ stands for susceptible, $V(t)$ stands for vaccinated, $I(t)$ stands for infected, and $R(t)$ stands for recovered individuals. Let β be the frequency of contact necessary to propagate disease. γ is the consistent rate of recovery. The frequency of immunization among the vulnerable population is α. Assuming that mortality from sickness is possible, we define it as the rate of death from disease, which is ω. In contrast, μ is the rate of disease-unrelated natural mortality. We assume that the μ entry rate into the susceptible class is the same for all babies. Let γ_1 be the average rate susceptible people develop immunity and join the population after recovery. Since vaccine-induced immunity can also be long-lasting, we do not differentiate between innate and vaccine-induced immunity. We hypothesize that vaccine recipients are still vulnerable to infection before developing immunity when interacting with carriers of the disease, with a β_1 disease transmission rate. β_1 may be regarded as being less than β since the vaccinated individuals may have some level of partial protection during the process, or they may comprehend the disease's transmission aspects and so minimize successful encounters with infected individuals. Figure 2.1 provides a detailed representation of the SVIR model's population transmission diagram.

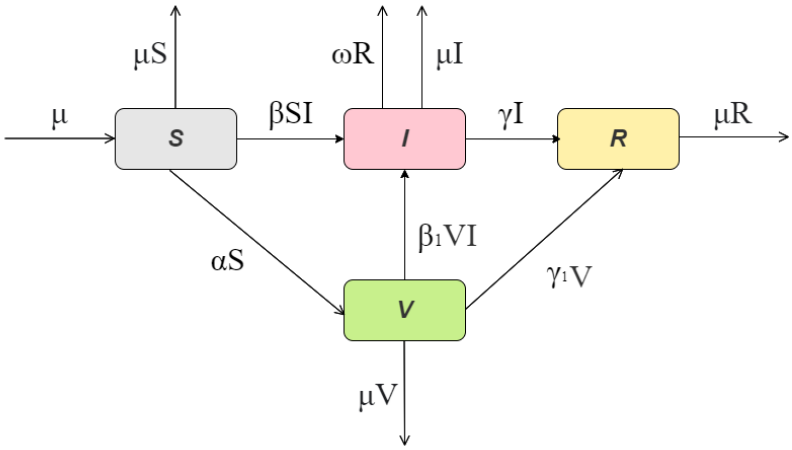


Figure 2.1: Transfer diagram of the SVIR Model.

Now, the differential equation system model is presented in the following form:

$$\begin{cases} \dfrac{\mathrm{dS}}{\mathrm{dt}} = \mu - \mu S - \beta SI - \alpha S \\ \dfrac{\mathrm{dV}}{\mathrm{dt}} = \alpha S - \beta_1 VI - \gamma_1 V - \mu V \\ \dfrac{\mathrm{dI}}{\mathrm{dt}} = \beta SI + \beta_1 VI - \gamma I - \mu I - \omega I \\ \dfrac{\mathrm{dR}}{\mathrm{dt}} = \gamma_1 V + \gamma I - \mu R \end{cases} \tag{2.1}$$

where $S(0) > 0$, $V(0) > 0$, $I(0) > 0$, $R(0) = 0$, $S + V + I + R = 1$ for all $t \geq 0$ and all parameters are positive. The interpretation of all parameters are prescribed in Table 2.1.

Table 2.1: Description of parameter values used in the model (2.1).

Parameter	Description
μ	The percentage of deaths and births that occur naturally.
α	The rate of migration of vulnerable individuals toward vaccination.
β	The frequency of disease transmission when susceptible individuals come into contact with infected individuals.
β_1	The probability that vaccinated people will contract the disease before gaining immunity.
γ	The recovery rate.
γ_1	The typical rate at which vaccine recipients develop immunity and enter the recovered population.
ω	The death rate brought on by sickness.

After ignoring the R class population as described before, the modified main model is

$$\begin{cases} \dfrac{\mathrm{dS}}{\mathrm{dt}} = \mu - \mu S - \beta SI - \alpha S \\ \dfrac{\mathrm{dV}}{\mathrm{dt}} = \alpha S - \beta_1 VI - \gamma_1 V - \mu V \\ \dfrac{\mathrm{dI}}{\mathrm{dt}} = \beta SI + \beta_1 VI - \gamma I - \mu I - \omega I \end{cases} \tag{2.2}$$

Additionally, we conducted a stability analysis of the model at the system (2.2) equilibrium point. We start by determining the system equilibrium points (endemic and disease-free points). The disease-free equilibrium point and the endemic equilibrium point are typically the two equilibrium points in the epidemic model. We analyze both equilibrium points in the context of the evolution of the disease in a population. Since the diseased population is equal to zero for $t \to \infty$, the disease-free equilibrium point is the point at which a disease is least likely to spread. The disease must spread ($I > 0$) for $t \to \infty$ to reach the endemic equilibrium point in a given constrained area. It is noted that all the auxiliary results can be found in Appendix A. The summarized important results are

- the equilibrium point of disease-free:

$$E_0 = (\ S_0,\ V_0,\ I_0) = \left(\tfrac{\mu}{\alpha+\mu}, \tfrac{\mu\alpha}{(\mu+\alpha)(\gamma_1+\mu)}, 0\right)$$

- the endemic equilibrium point:

$$E^* = (\ S^*,\ V^*,\ I^*) = \left(\tfrac{\mu}{\alpha+\mu+\beta I^*}, \tfrac{\mu\alpha}{(\mu+\alpha+\beta I^*)(\gamma_1+\mu+\beta I^*)}, \beta I^*\right)$$

- the basic reproduction number:

$$R_0 = \tfrac{\mu\beta}{(\alpha+\mu)(\gamma+\mu+\omega)} + \tfrac{\mu\alpha\beta_1}{(\mu+\alpha)(\gamma_1+\mu)(\gamma+\mu+\omega)}.$$

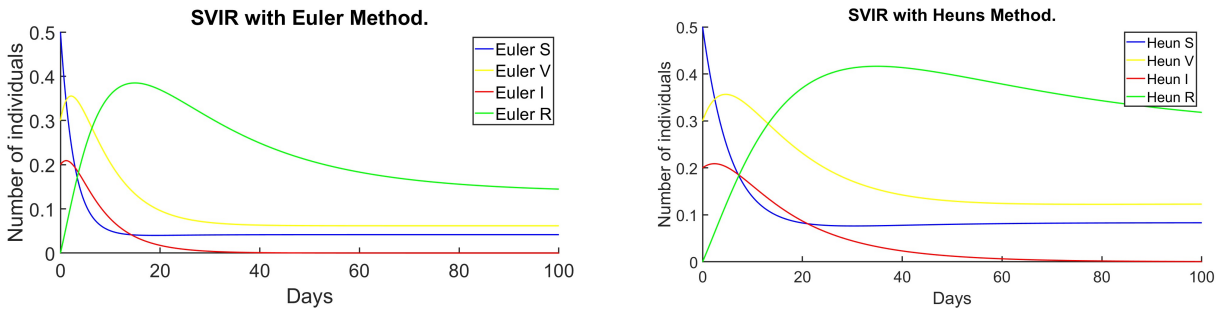
3 Computational Methods and Typical Results

To visualize the results, we used numerical solution methods such as Euler's Method and Heun's Method.

Euler's Method: The Euler method is a numerical technique used in mathematics and computational science to solve ordinary differential equations (ODEs) with a given initial value. It is considered the most basic explicit method for numerical integration of ODEs and a member of the Runge-Kutta family of methods [18].

Heun's Method: Heun's technique is a term used in mathematics and computing science to describe the enhanced or modified Euler's method (i.e., the explicit trapezoidal rule) or a related two-stage Runge-Kutta approach. Ordinary differential equations (ODEs) can be solved numerically using a predetermined beginning value. Both variations can be considered two-stage second-order Runge-Kutta procedures extending the Euler method [19].

Observing Accuracy of Graphical Comparison: We solved the SVIR model in the graph below using these two numerical techniques.
The first-order methods, such as the Euler method, have local errors proportional to the square of the step size and global errors proportional to the step size. Employing data from additional points in the interval can lead to higher-order convergence; the more points we use, our approach to solving ODEs will be more precise. Heun's approach increases accuracy by employing two points instead of Euler's single point. As a result, we can observe from our visual output that Heun's method supports our ideas more precisely than Euler's method (3.1). There are other ways to solve models, including SIR, SEIR, and SVIR.



(a) Solving the model with Euler's method.

(b) Solving the model with Heun's method.

Figure 3.1: Comparison of Accuracy.

Using the Runge-Kutta order four methods, we examine the numerical solution of problem (2.2) in this section. The initial conditions for the state variables are the same. Figures 3.2 and 3.3 display the numerical outcomes. When the value of the basic reproduction number is more than unity, Figure 3.3 illustrates this fact. When the basic reproduction number is less than one, Figure 3.2 demonstrates this fact.

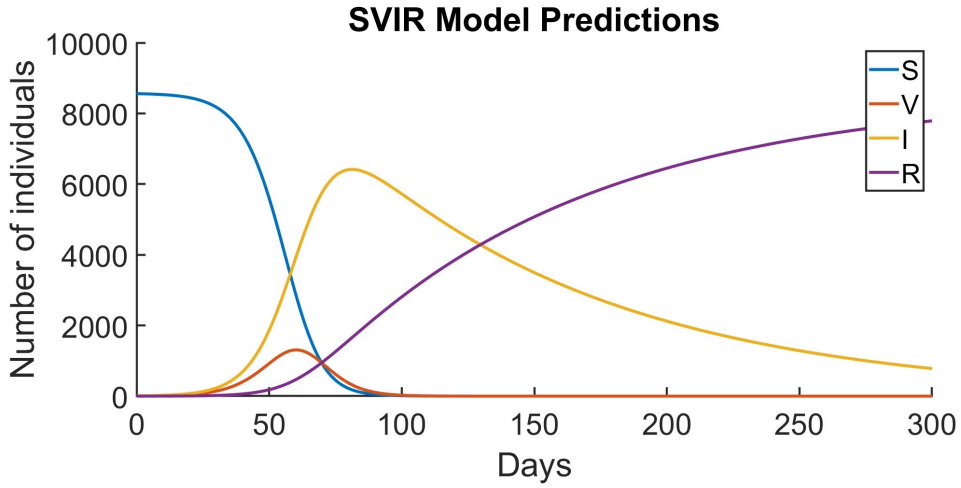


Figure 3.2: SVIR Model with $\mu = 1, \beta = 10, \beta_1 = 2, \gamma = 4, \gamma_1 = 8, \alpha = 10, \omega = 0.01$.

Figure 3.2 demonstrates the dynamical behavior of the system (2.2), under identical initial circumstances but with various parameter values: the disease-free equilibrium point is locally asymptotically stable, where $\mu = 1, \beta = 10, \beta_1 = 2, \gamma = 4, \gamma_1 = 8, \alpha = 10, \omega = 0.01$, when $R_0 = 0.22 \leq 1$.

For $R_0 = 1.13 \geq 1$, figure 3.3 displays the dynamic behavior of system (2.2) with different parameters but

the same initial conditions, the disease-free equilibrium point is locally asymptotically stable, where $\mu = 1, \beta = 60, \beta_1 = 2, \gamma = 4, \gamma_1 = 8, \alpha = 10, \omega = 0.01$,.

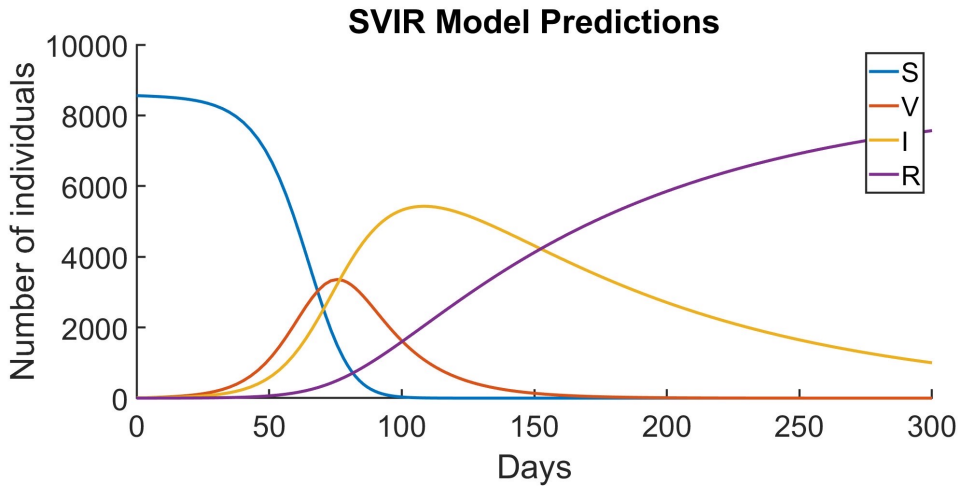


Figure 3.3: SVIR Model with $\mu = 1, \beta = 60, \beta_1 = 2, \gamma = 4, \gamma_1 = 8, \alpha = 10, \omega = 0.01$.

The basic reproduction number parameter is defined based on the analysis of the endemic equilibrium point results, as follows:

$$R_0 = \frac{\mu\beta}{(\alpha+\mu)(\gamma+\mu+\omega)} + \frac{\mu\alpha\beta_1}{(\mu+\alpha)(\gamma_1+\mu)(\gamma+\mu+\omega)}$$

Moreover, the existence and local stability of both equilibrium points is dependent on R_0 as a necessary condition.

If $R_0 \leq 1$, there exists only one unique equilibrium point which is disease-free:

$$E_0 = (\ S_0,\ V_0,\ I_0) = (\frac{\mu}{\alpha+\mu}, \frac{\mu\alpha}{(\mu+\alpha)(\gamma_1+\mu)}, 0)$$

Conversely, if $R_0 > 1$, then there are two equilibrium points, that is, E_0 and an endemic equilibrium point

$$E^* = (\ S^*,\ V^*,\ I^*) = (\frac{\mu}{\alpha+\mu+\beta I^*}, \frac{\mu\alpha}{(\mu+\alpha+\beta I^*)(\gamma_1+\mu+\beta I^*)}, \beta I^*)$$

where I^* is the positive root of the equation $A_1 I^2 + A_2 I + A_3(1 - R_0) = 0$

If basic reproduction number $R_0 \leq 1$, then the disease-free equilibrium point E_0 is locally asymptotically stable. In other words, the epidemic would end if the conditions $R_0 \leq 1$ were met since there would be a protracted period of no disease propagation among susceptible and immunized sub-populations. In contrast, the endemic equilibrium point E^* is locally asymptotically stable if $R_0 > 1$. It implies that the disease will constantly exist in the population throughout a long period of time with proportions of S^*, V^*, and I^* for each sub-population [20].

4 Vaccinated data and analysis: Case study of Bangladesh

The analysis of numerical data can be achieved through graphical representation, which involves presenting the relationships between data, ideas, information, and concepts in a diagram. It is a practical and easily understandable learning strategy dependent on the nature of the information being conveyed in a special domain [21]. This section will show the graphical representation of infected, vaccinated, and recovered population numbers concerning time.

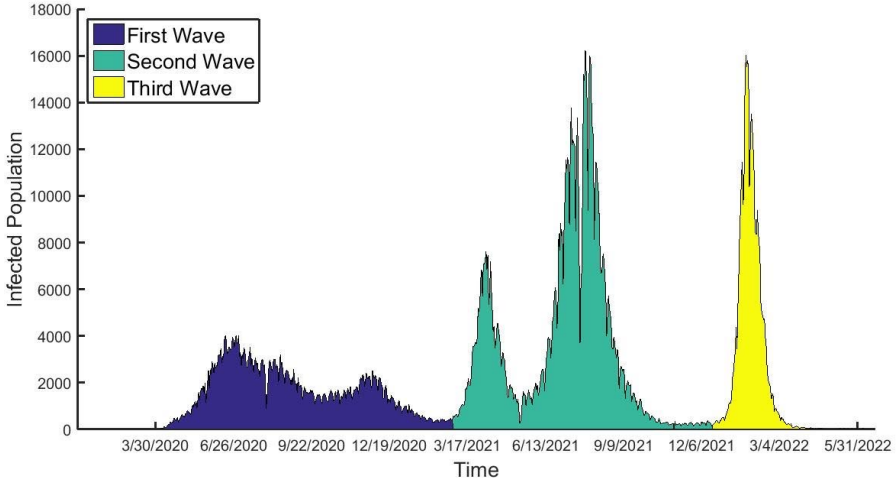


Figure 4.1: Infected vs Dates.

The first case of COVID-19 in Bangladesh was reported on **March 8, 2020** [22]. There has not been a single day without this infectious disease. As of today **(May 9, 2023)**, there have been 2,038,250 confirmed cases and 29,446 deaths in Bangladesh [23]. There were mainly three waves of a surge in infection during a two-and-a-half-year span from April 2020 to February 2022. The first wave started in around **April 2020**. Although it was the most extended wave of the three, the infection rate was not as high. The highest number of confirmed cases occurred in the last week of June 2020. There were 25,481 infected that week. The curve went down in the first week of October 2020, then again increased a bit in the last week of November. Eventually, it flattened out in **February 2021**.

The second wave lasted from **March 2021** to **August 2021**. This wave had a higher number of infected than the first one. It had two spikes. The first spike occurred in the second week of April 2021, where the confirmed cases were 47,392 per week. After that, the wave was well controlled, and the weekly case count decreased to around 6,000 in the second week of May. Nevertheless, the situation quickly changed, and the curve hit one of its highest points in the last week of July 2021. The confirmed cases in that following week were around 100,000. The situation gradually became stable, and by the time it was November, there were very minimal cases of Covid-19. The third wave had a brief span. It started at the end of **October 2021** and lasted till **February 2022**. Despite the short period, the infection rate was relatively high. The wave peaked in the last week of January, having around 100,000 confirmed cases [24].

4.1 Comparison of Multiple Waves and Actual Data

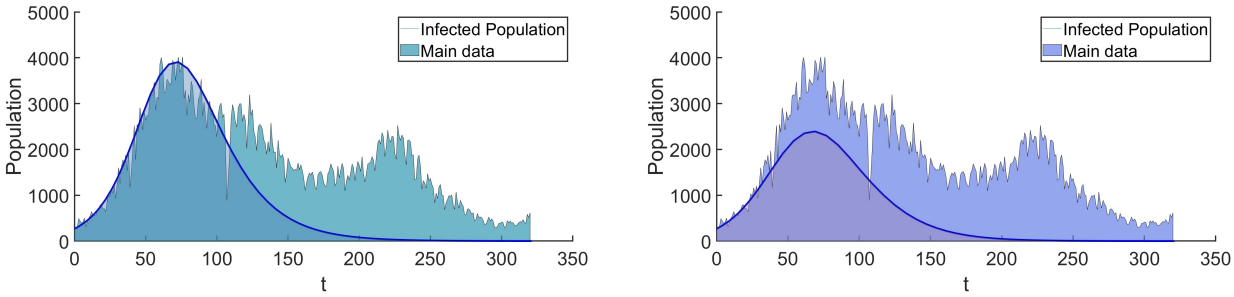
In this section, we will compare the results of the SVIR model with our actual data. We will also discuss the accuracy of our model. When evaluating mathematical models through empirical data, it is common to face scenarios in which an assessment of the suitability of the data for an equation or a graphical model is required. We took the actual data from a very trusted link DGHS [16], where they update the daily data with authenticity about different national COVID-related health issues. The data is presented here in the following graphs. Here, β represents the disease transmission rate when susceptible individuals come into contact with infected individuals, γ represents the recovery rate, and α represents the vaccination rate.

Table 4.1: Parameter values for multi-wave as used in the model (2.1).

Parameter	1st Wave	2nd Wave	3rd Wave	References
μ	1	1	1	[20]
β	10	10	10	[20]
β_1	2	2	2	[20]
γ	3	3	3	Assumed
γ_1	6	6	6	Assumed
ω	0.02	0.02	0.02	Assumed
α	[0-0.003]	[0.0008-0.01]	[0.002-0.04]	Assumed

1st Wave

We took the total number of infected people in the first 320 days from **April 2020 to February 2021**. From Figure 6a it is clear that the logistic growth prediction almost reflects the actual graph. During the first wave, vaccination had yet to be started in Bangladesh. So, we took the vaccination rate $\alpha = 0$. Here, both graphs almost match. We can see how the SVIR model predicts the ups and downs of the infected individuals.



(a) 1st wave with vaccination rate=0

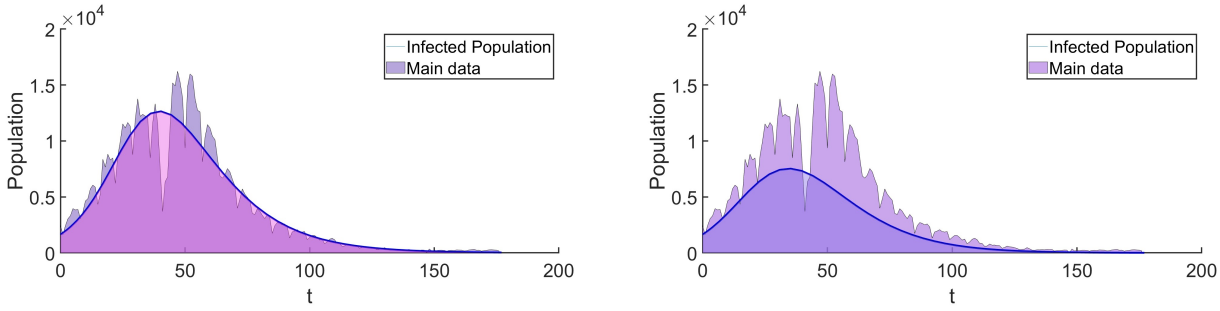
(b) 1st wave with vaccination rate=0.003

Figure 4.2: First Wave Infected Population Comparison with Real Data.

We were increasing the vaccination rate (α) just by 0.003, significantly impacting the infected population. We can see in Figure 6b how rapidly the number of infected populations would decrease if we could start vaccinating earlier. Also, the number of infected individuals would be less.

2nd Wave

We took the total number of infected people for the next 180 days from **March 2021 to August 2021**. From Figure 7a, it is clear that, in almost every case, the logistic growth prediction matches precisely with the actual graph on which it is based. During the time of the second wave, vaccination just started in Bangladesh. So, we took the vaccination rate $\alpha = 0.0008$. Here, both graphs almost match. We can observe how the SVIR model predicts infected individuals' fluctuations.



(a) 2nd wave with vaccination rate=0.0008

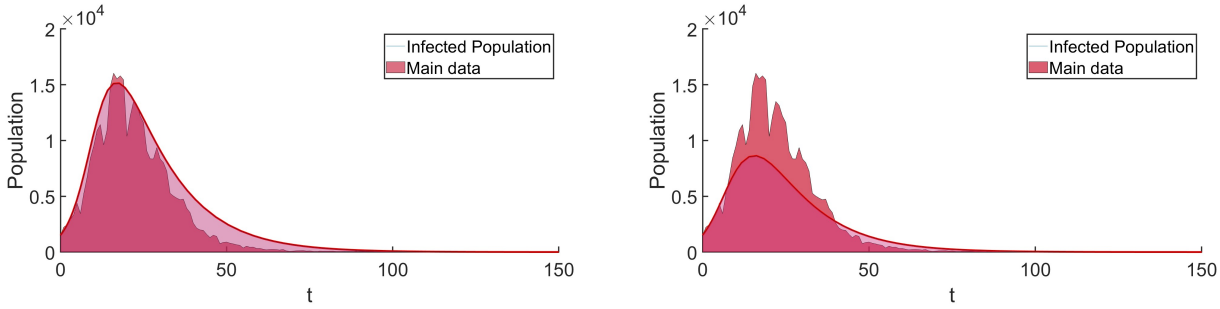
(b) 2nd wave with vaccination rate=0.01

Figure 4.3: Second Wave Infected Population Comparison with Real Data.

We are increasing the vaccination rate (α) just by 0.0092 leaves a significant impact on the infected population. We can see from Figure 7b how quickly the number of infected people will decrease if we immunize more people in vaccination programs. In addition, the number of infected individuals will be lower.

3rd Wave

We took the total number of infected people for the next 160 days from **September 2021 to February 2022**. During the time of the third wave, vaccination was going on in an increasing amount. So, we took the vaccination rate $\alpha = 0.002$. Here, the output of the SVIR model almost matched our actual data. We can observe how accurately this model predicts infected individuals' fluctuations.



(a) 3rd wave with vaccination rate=0.002

(b) 3rd wave with vaccination rate=0.04

Figure 4.4: Third Wave Infected Population Comparison with Real Data.

Suppose we increase the vaccination rate (α) just by 0.038. We can see from Figure 8b that it has an enormous impact on infected populations. It encompasses an expansive effect on contaminated populations. If able to begin inoculating more individuals, we will see how rapidly the number of infected individuals will drop. In expansion, the number of tainted individuals will diminish.

4.2 Comparing Vaccination and Recovered Rates

We can see no vaccination from March 2020 to January 2021. So, the curve is similar to the x-axis. As the days went by vaccination rate increased in a significant manner. This increasing rate of vaccination helped us to control a pandemic like COVID.

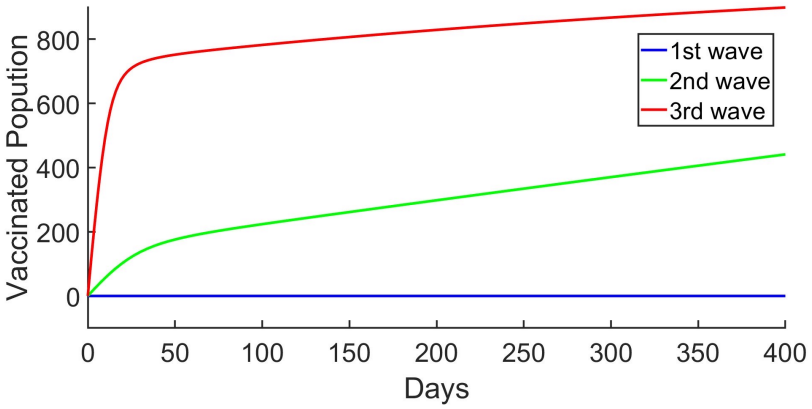


Figure 4.5: Vaccination Rate during Three Waves.

As vaccination had not been started during the first wave, the recovery rate was so slow compared to the other two waves. As days passed, we increased the vaccination rate, which greatly impacted our recovery curve. It is crystal clear how rapidly the rate of recovered population increased from the following graph of the second and third waves.

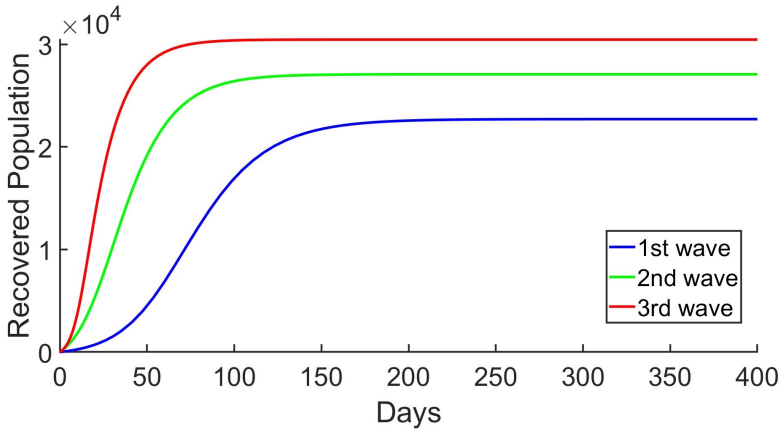


Figure 4.6: Recovery Rate during Three Waves.

4.3 Negative Impact on Accepting or Rejecting Vaccines

Negative emotions can indeed play a role in shaping an individual's acceptance or rejection of vaccines. Emotions such as fear, anxiety, skepticism, and distrust can influence people's attitudes and behaviors toward vaccination. Here are some ways in which negative emotions may impact vaccine acceptance or rejection: Fear of side effects, mistrust in the healthcare system, conspiracy theories and misinformation, emotional biases, personal autonomy and control.

It's important to note that while negative emotions can impact vaccine acceptance or rejection, they are not the sole determining factors. Various other factors, including access to accurate information, social influences, cultural norms, and personal experiences, also shape individual decisions regarding vaccines. Effective communication, education, and addressing concerns with empathy can help mitigate the influence of negative emotions and promote vaccine acceptance [25].

5 Conclusion

Mathematical and computational models are being used more frequently to understand better the biomedical data generated by high-throughput genomics and proteomics programs. Using sophisticated computer models that simulate complicated biological processes leads to the generation of hypotheses and recommendations for

studies. Models must be interfaced adequately with biological databases to allow quick access to information sharing via data mining and knowledge discovery techniques [26, 27]. Here, we have proposed an SVIR epidemic model for the COVID-19 pandemic in Bangladesh where S, V, I, and R represent susceptible, vaccinated, infected, and recovered populations, respectively. Initially, an examination of the fundamental characteristics of the introduced model, such as its positivity and boundedness, was conducted. Subsequently, the computation of the fundamental reproduction number, denoted by R_0, is undertaken for the model in question. Based on our theoretical analysis, it is evident that the model comprises two distinct types of equilibrium points: namely, the disease-free equilibrium point and the endemic equilibrium point. This study substantiates the existence of the disease-free equilibrium. A point of stability is achieved in a population when the basic reproduction number falls below one, signifying the successful eradication of the disease. This study examines the dataset spanning from April 2020 to October 2022, pertaining to Bangladesh. During the nascent phase, the illness was disseminated expeditiously within a brief timeframe due to the general population's lack of awareness regarding contagion. The commencement of the COVID-19 vaccination campaign in Bangladesh took place on the 27th of January 2021, followed by the subsequent instigation of mass vaccination measures on the 7th of February 2021 [28, 29]. By continuing lockdown, enforcing house quarantines, washing hands, and donning masks, the disease's power has begun to wane daily. Comparing the models with actual data also reveals how mass immunization has contributed to the containment of this pandemic. It's worth noting that the effectiveness of vaccination in preventing pandemics relies on factors such as vaccine coverage, vaccine uptake, public health measures, and the emergence of new variants. Continued monitoring, adherence to public health guidelines, and timely vaccine distribution are vital to maximize the impact of vaccination efforts in preventing and controlling pandemics.

Acknowledgments

The research by M. Kamrujjaman was partially supported by the University Grants Commission (UGC), and the University of Dhaka, Bangladesh.

Conflict of interest

The authors declare no conflict of interest.

Data sharing

Data is publicly available online.

Ethical approval

No consent is required to publish this manuscript.

Author contributions

Conceptualization, MK and NNK; methodology, NNK and SAB; software, NNK, RA and SAB; validation, MK; formal analysis, NNK, SAB and MK; investigation, RA; resources, SAB; data curation, NNK; original draft preparation, NNK, RA and SAB; review and editing, MK; supervision, MK. All authors have read and agreed to the published version of the manuscript.

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A Auxiliary Results and Stability Analysis

A.0.1 Finding Equilibrium Points

The equilibrium point of system (2.2) exists when $\frac{dS}{dt} = 0, \frac{dV}{dt} = 0, \frac{dI}{dt} = 0$ so that:

$$\mu - \mu S - \beta SI - \alpha S = 0 \tag{A.1}$$
$$\alpha S - \beta_1 VI - \gamma_1 V - \mu V = 0 \tag{A.2}$$
$$\beta SI + \beta_1 VI - \gamma I - \mu I - \omega I = 0 \tag{A.3}$$

From equation (A.3), we have I($\beta S+\beta_1 V-\gamma-\mu-\omega) = 0$, and the solutions are I=0 or ($\beta S+\beta_1 V-\gamma-\mu-\omega = 0$, consequently, we have two situations, namely:

(a) The disease-free equilibrium point; Situation at $I = 0$: It is the necessary condition for the disease-free equilibrium point. Note that, From equation (A.1), $\mu - \mu S - \alpha S = 0$ such that $S = \frac{\mu}{\alpha+\mu}$. Similarly, from (A.2), $\alpha S - \beta_1 VI - \gamma_1 V - \mu V = 0$ which implies $V = \frac{\alpha\mu}{(\alpha+\mu)(\gamma_1+\mu)}$. So we get the equilibrium point of disease-free, that is,

$$E_0 = (\ S_0,\ V_0,\ I_0) = (\tfrac{\mu}{\alpha+\mu}, \tfrac{\mu\alpha}{(\mu+\alpha)(\gamma_1+\mu)}, 0)$$

(b) The endemic equilibrium point; Situation at $I \neq 0$ or $I > 0$: This circumstance is a required prerequisite for an endemic equilibrium point. Note that:

- From equation (A.1), $\mu - \mu S - \beta SI - \alpha S = 0 \Longleftrightarrow S^* = \frac{\mu}{(\alpha+\mu+\beta I^*)}$
- From (A.2), $\alpha S - \beta_1 VI - \gamma_1 V - \mu V = 0 \Longleftrightarrow V^* = \frac{\mu\alpha}{(\mu+\alpha+\beta I^*)(\gamma_1+\mu+\beta I^*)}$
- From (A.3), if $I \neq 0$, then $\beta S + \beta_1 V - \gamma - \mu - \omega = 0$, consequently, with substitute $S^*$and V^* we have:

$$\frac{\mu\alpha\beta_1}{(\mu+\alpha+\beta I^*)(\gamma_1+\mu+\beta I^*)} = \gamma + \mu + \omega - \frac{\beta\mu}{(\alpha+\mu+\beta I^*)} \Longleftrightarrow$$

$$\beta_1\beta(\gamma+\mu+\omega)(I^*)^2((\gamma+\mu+\omega)((\alpha+\mu)\beta_1+(\mu+\gamma_1)\beta)\beta_1\beta\mu)I^* + (\gamma+\mu+\omega)(\mu+\gamma_1)(\alpha+\mu)(1 - (\tfrac{\mu\beta}{(\alpha+\mu)(\gamma+\mu+\omega)} + \tfrac{\mu\alpha\beta_1}{(\mu+\alpha)(\gamma_1+\mu)(\gamma+\mu+\omega)}))$$

By applying :

$$A_1 = (\gamma+\mu+\omega)\beta\beta_1 > 0$$
$$A_2 = (\gamma+\mu+\omega)((\alpha+\mu)\beta_1 + (\gamma_1+\mu)\beta)) - \beta_1\beta_\mu$$
$$A_3 = (\gamma+\mu+\omega)(\alpha+\mu)(\gamma_1+\mu) > 0$$
$$C = \frac{\mu\beta}{(\alpha+\mu)(\gamma+\mu+\omega)} + \frac{\mu\alpha\beta_1}{(\mu+\alpha)(\gamma_1+\mu)(\gamma+\mu+\omega)}$$

the above equation becomes:

$$A_1 I^2 + A_2 I + A_3(1-C) = 0 \tag{A.4}$$

Thus, we obtain an endemic equilibrium point, that is,

$$E^* = (\ S^*,\ V^*,\ I^*) = (\tfrac{\mu}{\alpha+\mu+\beta I^*}, \tfrac{\mu\alpha}{(\mu+\alpha+\beta I^*)(\gamma_1+\mu+\beta I^*)}, \beta I^*).$$

A.0.2 Basic Reproduction Number (R_0)

The value of the basic reproduction number (R_0), or the number of susceptible individuals who contract the infection after coming into contact with patients from a completely vulnerable population, affects the stability analysis of the equilibrium point in the SVIR model. By keeping an eye on the state of the endemic equilibrium point, this number is calculated.

Consider Eq.(6), an endemic equilibrium point only applies to non-negative roots $I^* > 0$ when $C > 1$. Finally, because the magnitude of the positive endemic equilibrium point is in the value of C, it may be defined as the value of the basic reproduction number, that is,

$$R_0 = \frac{\mu\beta}{(\alpha+\mu)(\gamma+\mu+\omega)} + \frac{\mu\alpha\beta_1}{(\mu+\alpha)(\gamma_1+\mu)(\gamma+\mu+\omega)}.$$

A.0.3 Local Stability of Equilibrium Point

We will assess the stability of the endemic equilibrium point and the disease-free equilibrium in the SVIR model. Since equations (A.1), (A.2), and (A.3), are not linear, the Taylor series will result in a set of linear differential equations at each equilibrium point.

Theorem 1. *1. The disease-free equilibrium point E_0 exists if $R_0 \leq 1$, making it a unique equilibrium of system (2.2). Additionally, if $R_0 > 1$, then system (2.2) has two equilibrium points, namely, the disease-free equilibrium points E_0 and E^*.*

2. The equilibrium point E_0 is locally asymptotically stable if $R_0 \leq 1$, and unstable if $R_0 > 1$.

3. The endemic equilibrium E^* is locally asymptotically stable if $R_0 > 1$.

Proof. **Case 1.**
The presence of the disease-free equilibrium point has already been covered. We find an equilibrium point in the discussion when $I = 0$, and it is unique if $R_0 \leq 1$. The disease-free equilibrium represents the state of equilibrium in which there are no cases of the disease.

Let us now observe the equation $A_1I^2 + A_2I + A_3(1 - R_0) = 0$, which has two real roots (positive and negative) if $R_0 > 1$, then $A_1 > 0$ and $A_3(1 - R_0) < 0$. The fulfillment of a condition that guarantees the existence and uniqueness of equilibrium point E^* has resulted in this outcome. Moreover, we shall demonstrate that the aforementioned quadratic equation has no positive roots when $R_0 \leq 1$. Recall that,

$R_0 \leq 1 \Rightarrow \mu\beta \leq (\alpha+\mu)(\gamma+\mu+\omega) \Rightarrow A_2 \geq (\gamma+\mu+\omega)\Big((\alpha+\mu)\beta_1 + (\gamma_1+\mu)\beta\Big) - \beta_1(\gamma+\mu+\omega)(\alpha+\mu) > \beta(\gamma_1+\mu)(\gamma+\mu+\omega) > 0$

So if $A_1 > 0$, $A_3(1 - R_0) > 0$ then the function of $A_1I^2 + A_2I + A_3(1 - R_0)$ will rise and $A_1I^2 + A_2I + A_3(1 - R_0) > A_3(1 - R_0) \geq 0$, for $I > 0$, which means the quadratic function has no positive roots.

Case 2.
The Jacobian Matrix of the system (2.2) is :

$$J(S,V,I) = \begin{bmatrix} -\mu-\alpha-\beta I & 0 & -\beta S \\ \alpha & -\mu-\gamma_1-\beta_1 I & \beta_1 V \\ \beta I & \beta_1 I & \beta S+\beta_1 V-\mu-\gamma-\omega \end{bmatrix}$$

Therefore, the Jacobian matrix evaluated at point $E_0 = (S_0, V_0, I_0) = (\frac{\mu}{\alpha+\mu}, \frac{\mu\alpha}{(\mu+\alpha)(\gamma_1+\mu)}, 0)$ is:

$$J(\ E_0) = \begin{bmatrix} -\mu-\alpha & 0 & -\beta S_0 \\ \alpha & -\mu-\gamma_1 I & \beta_1 V_0 \\ 0 & 0 & \beta S_0+\beta_1 V_0-\mu-\gamma-\omega \end{bmatrix}$$

The eigenvalues are :

$$\kappa_1 = -(\mu+\alpha) < 0$$
$$\kappa_2 = -(\mu+\gamma_1) < 0$$
$$\kappa_3 = \beta S_0+\beta_1 V_0-\mu-\gamma-\omega = (\gamma+\mu+\omega)(R_0-1)$$

It is obvious that if $R_0 < 1$, all of $J(E_0)$'s eigenvalues are negative, and as a result, the point E_0 is locally asymptotically stable. The point E_0 is unstable if $R_0 > 1$ since there is a positive eigenvalue in that case.

Case 3.

The existence of the endemic point E^* is evident for $R_0 > 1$. Consequently, the Jacobian matrix evaluated at point $E^* = (\ S^*, V^*, I^*)$ is:

$$J(E^*) = \begin{bmatrix} -\mu-\alpha-\beta I^* & 0 & -\beta S \\ \alpha & -\mu-\gamma_1-\beta_1 I^* & \beta_1 V \\ \beta I^* & \beta_1 I^* & \beta S^*+\beta_1 V^*-\mu-\gamma-\omega \end{bmatrix}$$

It is possible to alter the elements a_{11}, a_{22}, a_{33} of the matrix mentioned earlier to:

$$-\mu-\alpha-\beta I^* = -\frac{\mu}{S^*}$$
$$-\mu-\gamma_1-\beta_1 I^* = -\frac{\alpha S^*}{V^*}$$

The outcome that we are obtaining is based on the evaluation outcomes around the endemic equilibrium point: $\beta S^*+\beta_1 V^*-\mu-\gamma-\omega = 0$, as a result, the matrix is transformed into:

$$J(E^*) = \begin{bmatrix} -\frac{\mu}{S^*} & 0 & -\beta S \\ \alpha & -\mu-\gamma_1-\beta_1 I^* & \beta_1 V \\ \beta I^* & \beta_1 I^* & -\frac{\alpha S^*}{V^*} \end{bmatrix}$$

As per the Routh-Hurwitz criterion, all the roots of the characteristic equations mentioned above have a real component that is negative. As a result, the equilibrium point $E^* = (\ S^*, V^*, I^*)$ is locally stable. □

We shall interpret the results biologically in the following ways based on the findings:

- If $R_0 \leq 1$, the solution of system (2.2) will progress to E_0=(S_0,V_0,I_0) for $t \to \infty$ and (S,V,I) that close to E_0=(S_0,V_0,I_0). It means that if $R_0 \leq 1$, the disease will not spread and has a tendency to vanish for an unlimited amount of time for the population that is susceptible, immunized, and infected close to E_0=(S_0,V_0,I_0). In this case, the equilibrium point E_0=(S_0,V_0,I_0) is referred to be asymptotically stable, which can be interpreted as a disease-free equilibrium point.
- The solution of system (2.2) will shift to $E^* = (\ S^*, V^*, I^*)$ if $R_0 \geq 1$, then for $t \to \infty$ and (S,V,I) that close enough to $E^* = (\ S^*, V^*, I^*)$. This means that if $R_0 > 1$, the illness will continue to spread but won't be wiped out indefinitely for the population that is susceptible, immunized, and infected close to $E^* = (\ S^*, V^*, I^*)$. Around the equilibrium point $E^* = (\ S^*, V^*, I^*)$, which we interpret as an endemic equilibrium point, this state is thus referred to as asymptotically stable.