UNDER PEER REVIEW

# AN SVEIR COVID-19 MATHEMATICAL MODEL WITH DOUBLE DOSE VACCINATION

**Abstract**

In this study, the effects of a twofold vaccination dosage are examined using the Covid-19 mathematical model. In addition to obtaining the basic reproduction number and analyzing the model's stability, the sensitivity analysis was also performed. The results obtained suggested ways to manage the corona virus while taking the contact rate and vaccination into consideration. Finally, the numerical simulations demonstrated that the control intervention's primary goal should be to boost immunization rates using a vaccine with a high level of efficacy.

**Keywords:** Covid-19, Reproduction Number, sensitivity analysis, Stability Analysis.

## 1 Introduction

A class of viruses known as coroviruses can infect both humans and other mammals like pigs and bats . A new coronavirus called COVID-19 causes a respiratory infection that spreads between people via tiny droplets released during coughing, sneezing, or speaking [1, 2]. In December 2019, Wuhan City, Hebei Province, China, received the first reports of the virus. The World Health Organization (WHO) designated it a global epidemic after it spread to numerous nations. The symptoms of the COVID-19 virus, according to the WHO, include fever, dry cough, exhaustion, sore throat, pains, diarrhea, nasal congestion, and loss of taste or smell [3, 4, 5].

Many mathematical models have been developed by researchers since COVID-19 first surfaced in late 2019 to help in understanding the dynamic spread and control of the pandemic in various regions for example [1, 2, 4, 6, 7, 8] among others. An SEIR (Susceptible-Exposed-Infected-Recovered) model was used

by [6] and [7] to quantitatively predict the transmission of COVID-19. Without taking into account the fact that persons who have been vaccinated cannot spread disease to others at the same rate as those who are yet to be vaccinated.

Different health organizations came up with a number of vaccinations like:

1. The Pfizer, BioNTech Comirnaty vaccine,
2. The SII/COVISHIELD and AstraZeneca/AZD1222 vaccines,
3. The Janssen/Ad26.COV 2.S vaccine developed by Johnson & Johnson,
4. The Moderna COVID-19 vaccine (mRNA 1273),

among others [3].

Different COVID-19 variants can cause varying levels of infection, symptoms, transmission speed, and susceptibility. Additionally, it is clear that a medication that is effective for one variants may not be effective for another. Due to the advent of a novel variety with potential for immunological escape and reinfection, most countries have recommended booster doses in order to strengthen the population's defense against COVID-19 [8].

In this paper we formulate and analyze a model in which individuals can receive first vaccination or second vaccination doses

## 2 The Model

We formulate a model in which the total human population at any time $t$ denoted by $N$ is subdivided into classes, $S(t)$ the class of individuals susceptible to Covid-19 infection. Recruitment into susceptible class is done at a rate $\Lambda$. The class $V_1(t)$ consists of individuals who have received first vaccination, this vaccination occurs at the rate $a$. Second vaccination is done at the rate $b$ and this leads to the creation of the class $V_2(t)$. Susceptible individuals, individuals who have received the first and second vaccination dose can be exposed to Covid-19 infection at the rates $\lambda$, $\lambda_1$ and $\lambda_2$ respectively, thus progressing to the exposed class $E(t)$, where $\lambda_2 < \lambda_1 < \lambda$. The class $I(t)$ consist of individuals who are asymptomatically infected with Covid-19 infection, this infection occurs at the rate $\epsilon$. Recovery of Covid-19 infection occurs at the rate $\beta$ and thus the class $R(t)$ consist of individuals who have recovered. Mortality occurs among Covid-19 patients at the rate $\delta$ while natural death is assumed to occur in all classes at the rate $\mu$.

From the above definitions, the dynamics described can be represented mathematically as;

$$
\begin{aligned}
\dot{S}(t) &= \Lambda - \frac{\lambda SI}{N} - (a+\mu)S(t) \\
\dot{V_1}(t) &= aS(t) - (\mu+b)V_1(t) - \frac{\lambda_1 V_1 I}{N} \\
\dot{V_2}(t) &= bV_1 - \mu V_2(t) - \frac{\lambda_2 V_2 I}{N} \\
\dot{E}(t) &= \frac{(\lambda S + \lambda_1 V_1 + \lambda_2 V_2)I}{N} - (\epsilon+\mu)E(t) \\
\dot{I}(t) &= \epsilon E(t) - (\mu+\delta+\beta)I(t) \\
\dot{R}(t) &= \beta I(t) - \mu R(t)
\end{aligned} \tag{1}
$$

# 3 Model analysis

Based on the fact that the model deals with human population, all the state variables and parameters are assumed to be non-negative $\forall t > 0$. This model is studied in the feasible region $\mathbb{R}$ where $(S(t), V_1(t), V_2(t), E(t), I(t), R(t)) \in \Omega \in \mathbb{R}^6_+$ and it can be shown that as $t$ tends to infinity;

$$0 \leq N(t) \leq \frac{\Lambda}{\mu} \tag{2}$$

Which shows that the set of solutions is bounded. Thus, the model Equation (1) is epidemiologically well posed in the region $\Omega$.

The basic reproduction number $R_0$ is defined as the average number of secondary Covid-19 infections produced by a single infectious individual over the course of their infectious period when introduced into an entirely susceptible population. The basic reproduction number, $R_0$, for model (1) computed using the next generation matrix method is given by;

$$R_0 = \frac{\lambda\epsilon}{(\epsilon+\mu)(\delta+\beta+\mu)} \tag{3}$$

# 4 Disease-free Equilibrium Point

The disease-free equilibrium point is a steady-state solution for which there is no disease or infection in the population [11]. To obtain the disease-free equilibrium point we set the normalised model system (1) equal to zero as shown below, $E^0 = \{S(t), V_1(t), V_2(t), E(t), I(t), R(t)\} = (\frac{\Lambda}{\mu}, 0, 0, 0, 0, 0)$.

# 5 Local stability of the Disease free equilibrium

**Theorem 5.1.** *The infection free equilibrium $E^0$ is locally asymptotically stable if and only if $R_0 < 1$*

.

*Proof.* The Jacobian matrix of Equation (1) is given by

$$J = \begin{pmatrix} -(a+\mu+\frac{\lambda I}{N}) & 0 & 0 & 0 & -\frac{\lambda S}{\mu} & 0 \\ a & -(\mu+b+\frac{\lambda_1 I}{N}) & 0 & 0 & -\frac{\lambda_1 V_1}{N} & 0 \\ 0 & b & -(\mu+\frac{\lambda_2 I}{N}) & 0 & -\frac{\lambda_2 V_2}{N} & 0 \\ \frac{\lambda I}{N} & \frac{\lambda_1 I}{N} & \frac{\lambda_2 I}{N} & -(\epsilon+\mu) & \frac{\lambda S}{N}+\frac{\lambda_1 V_1}{N}+\frac{\lambda_2 V_2}{N} & 0 \\ 0 & 0 & 0 & \epsilon & -(\mu+d+\delta) & 0 \\ 0 & 0 & 0 & 0 & \beta & -\mu \end{pmatrix} \quad (4)$$

Clearly $-\mu$ is an eigenvalue. We analyse the reduced matrix

$$J = \begin{pmatrix} -(a+\mu+\frac{\lambda I}{N}) & 0 & 0 & 0 & -\frac{\lambda S}{\mu} \\ a & -(\mu+b+\frac{\lambda_1 I}{N}) & 0 & 0 & -\frac{\lambda_1 V_1}{N} \\ 0 & b & -(\mu+\frac{\lambda_2 I}{N}) & 0 & -\frac{\lambda_2 V_2}{N} \\ \frac{\lambda I}{N} & \frac{\lambda_1 I}{N} & \frac{\lambda_2 I}{N} & -(\epsilon+\mu) & \frac{\lambda S}{N}+\frac{\lambda_1 V_1}{N}+\frac{\lambda_2 V_2}{N} \\ 0 & 0 & 0 & \epsilon & -(\mu+d+\delta) \end{pmatrix} \quad (5)$$

Applying the Routh-Hurwitz criterion [12], for stability analysis, then matrix $J$ in equation (6) will have negative real roots if and only if the $tr(J) < 0$ and $det(J) > 0$, and thus the trace of Equation (5) is negative and the determinant is given by

□

# 6 Global stability of the disease-free equilibrium

The Castillo Chavez theorem [9] is applied to study the global stability of the disease-free equilibrium. We rewrite model (1) in the form;

$$\frac{dX}{dt} = H(X, Z)$$
$$\frac{dZ}{dt} = G(X, Z), G(X, 0) = 0 \quad (6)$$

Where $X \in \mathbb{R}^4$ denotes the number of susceptible individual and $Z \in \mathbb{R}^2$ denotes the number of infected individuals.

$$E^0 = (\frac{\Lambda}{\mu}, 0, 0, 0, 0, 0) \tag{7}$$

denotes the disease free equilibrium point of this system where

$X^* = \frac{\Lambda}{\mu}$

The conditions below must be met to guarantee global asymptotic stability
$\frac{dX}{dt} = H(X, 0), X^0$ is globally Asymptotically stable (GAS)

$$G(X, Z) = PZ - \hat{G}(X, Z), \hat{G}(X, Z) \geq 0, for(X, Z) \in \Omega \tag{8}$$

Where $P = D_z G(X^0, 0)$ is an M- matrix (the off diagonal elements of $P$ are nonnegative) and $\Omega$ is the region where the model makes biological sense. If system (6) satisfies conditions in (8) then the following theorem holds:

**Theorem 6.1.** *The fixed point $E_0 = (X^0, 0, 0, 0, 0, 0)$ is a Globally Asymptotically Stable equilibrium point of model (1) provided that $R_0 < 1$ and the conditions in (8) are satisfied.*

*Proof.*

$$H(X, 0) = \Lambda - (\mu + a)S, aS - (\mu + b)V_1, bV_1 - \mu V_2 \tag{9}$$

And $G(X, Z) = PZ - \hat{G}(X, Z)$ where

$$P = \begin{pmatrix} -(\epsilon + \mu) & 0 \\ \epsilon & -(\mu + \delta + \beta) \end{pmatrix} \tag{10}$$

$$\hat{G}(X, Z) = \begin{pmatrix} \hat{G}_1(X, Z) \\ \hat{G}_2(X, Z) \end{pmatrix} = \begin{pmatrix} -\frac{(\lambda S + \lambda_1 V_1 + \lambda_2 V_2) I}{N} \\ 0 \end{pmatrix} \tag{11}$$

Considering the Jacobian matrix, and replacing $S(t) = \frac{\Lambda}{\mu}$, $I(t) = 0$, $E(t) = 0$, we obtain $\hat{G}_1(X, Z) = 0$ and so the conditions in (8) are met so $E^0$ is globally asymptotically stable when $R_0 < 1$. Epidemiologically, any perturbation of the model by the introduction of infectives shows that the model solutions will converge to the DFE whenever $R_0 < 1$. Global asymptotic stability shows that regardless of any starting solution, the solutions of the model will converge to DFE whenever $R_0 < 1$. This implies that we do not expect the disease outbreak for life. Thus, the epidemic will die out or will not develop in the population.

□

This implies that given a large perturbation of the DFE by the introduction of free virus particles, the solutions of model (1) will eventually converge to the DFE whenever $R_{0w} < 1$.

# 7 Local stability of endemic equilibrium point

For an infection to be endemic in a population, $E^* > 0$. At the endemic equilibrium, persistence of infection occurs and thus at least one of the infected classes is greater than zero.
The Jacobian of Equation (1) at endemic state $E^*(S^*(t), V_1^*(t), V_2^*(t), E^*(t), I^*(t), R^*(t))$ is given by

$$J = \begin{pmatrix} -(a+\mu+\frac{\lambda I^*}{N}) & 0 & 0 & 0 & -\frac{\lambda S^*}{N} & 0 \\ a & -(\mu+b+\frac{\lambda_1 I^*}{N}) & 0 & 0 & -\frac{\lambda_1 V_1^*}{N} & 0 \\ 0 & b & -(\mu+\frac{\lambda_2 I^*}{N}) & 0 & -\frac{\lambda_2 V_2^*}{N} & 0 \\ \frac{\lambda I^*}{N} & \frac{\lambda_1 I^*}{N} & \frac{\lambda_2 I^*}{N} & -(\epsilon+\mu) & \frac{\lambda S^*}{N}+\frac{\lambda_1 V_1^*}{N}+\frac{\lambda_2 V_2^*}{N} & 0 \\ 0 & 0 & 0 & \epsilon & -(\mu+d+\delta) & 0 \\ 0 & 0 & 0 & 0 & \beta & -\mu \end{pmatrix} \quad (12)$$

Clearly $-\mu$ is an eigenvalue. We analyse the reduced matrix

$$J = \begin{pmatrix} -(a+\mu+\frac{\lambda I^*}{N}) & 0 & 0 & 0 & -\frac{\lambda S^*}{N} \\ a & -(\mu+b+\frac{\lambda_1 I^*}{N}) & 0 & 0 & -\frac{\lambda_1 V_1^*}{N} \\ 0 & b & -(\mu+\frac{\lambda_2 I^*}{N}) & 0 & -\frac{\lambda_2 V_2^*}{N} \\ \frac{\lambda I^*}{N} & \frac{\lambda_1 I^*}{N} & \frac{\lambda_2 I^*}{N} & -(\epsilon+\mu) & \frac{\lambda S^*}{N}+\frac{\lambda_1 V_1^*}{N}+\frac{\lambda_2 V_2^*}{N} \\ 0 & 0 & 0 & \epsilon & -(\mu+d+\delta) \end{pmatrix} \quad (13)$$

An important criterion by Routh-Hurwitz gives the necessary and sufficient conditions for all the roots of the characteristic polynomial (with real coefficients) to lie in the left half of the complex plane. In other words, all the roots of the polynomial are negative or have negative real roots if the determinants of all Hurwitz matrices are positive [12].
From the Jacobian matrix (13), the trace is negative and the determinant is given by
$detJ_2(E^*) =$
The determinant $detJ_2(E^*) > 0$ provided that;

Thus, by Routh-Hurwitz criterion, the endemic state $E^*(S^*(t), V_1^*(t), V_2^*(t), E^*(t), I^*(t), R^*(t))$ is locally asymptotically stable. Therefore if $R_0 > 1$ and given a small infective population, each infected individual in the entire period of infectivity will produce more than one infected individual on average, which shows that the disease will persist in the population and thus the disease transmission levels can be kept quite low or manageable with minimal deaths.

# 8 Global stability of endemic equilibrium point

The global stability of the equilibrium is obtained by means of Lyapunov's direct method and LaSalle's invariance principle De Leon [10]. Consider the

non-linear Lyapunov function
$V : (S(t), V_1(t), V_2(t), E(t), I(t), R(t)) \in \Omega \subset \mathbb{R}^6_+ : S(t), V_1(t), V_2(t), E(t), I(t), R(t) > 0$

defined as

$$V = S - S^* \ln S + V_1 - V_1^* \ln V_1 + V_2 - V_2^* \ln V_2 + E - E^* \ln E + I - I^* \ln I + R - R^* \ln R \quad (14)$$

where $V$ is in the interior of the region $\Omega$. $E^*$ is the global minimum of $V$ on $\Omega$ and $V : \{S(t), V_1(t), V_2(t), E(t), I(t), R(t)\} = 0$. Differentiating $V$ with respect to time gives

$\frac{dV}{dt} = \dot{V} = \dot{S}(1 - \frac{S^*}{S}) + \dot{V_1}(1 - \frac{V_1^*}{V_1}) + \dot{V_2}(1 - \frac{V_2^*}{V_2}) + \dot{E}(1 - \frac{E^*}{E}) + \dot{I}(1 - \frac{I^*}{I}) + \dot{R}(1 - \frac{R^*}{R})$

Replacing $\dot{S}(t), \dot{V_1}, \dot{V_2}, \dot{E}, \dot{I}(t), \dot{R}$ from Equation (1), we obtain

$\dot{V} = [\Lambda - (\frac{\lambda I}{N} + a + \mu)S](1 - \frac{S^*}{S}) + [aS - (\mu + b + \frac{\lambda_1 I}{N})V_1](1 - \frac{V_1^*}{V_1})[bV_1 - (\mu + \frac{\lambda_2 I}{N})V_2](1 - \frac{V_2^*}{V_2})[\frac{(\lambda S + \lambda_1 V_1 + \lambda_2 V_2)I}{N} - (\epsilon + \mu)E](1 - \frac{E^*}{E})[\epsilon E - (\mu + \delta + \beta)I](1 - \frac{I^*}{I}) + [\beta I - \mu R](1 - \frac{R^*}{R})$

At the boundary conditions $N \leq \frac{\Lambda}{\mu}$, then we let $N = \frac{\Lambda}{\mu}$

$\dot{V} = [\Lambda - (\frac{\lambda \mu I}{\Lambda} + a + \mu)S](1 - \frac{S^*}{S}) + [aS - (\mu + b + \frac{\lambda_1 I \mu}{\Lambda})V_1](1 - \frac{V_1^*}{V_1}) + [bV_1 - (\mu + \frac{\lambda_2 I \mu}{\Lambda})V_2](1 - \frac{V_2^*}{V_2}) + [\frac{(\lambda S + \lambda_1 V_1 + \lambda_2 V_2)I\mu}{\Lambda} - (\epsilon + \mu)E](1 - \frac{E^*}{E}) + [\epsilon E - (\mu + \delta + \beta)I](1 - \frac{I^*}{I}) + [\beta I - \mu R](1 - \frac{R^*}{R})$

Using the following relations at the steady state

$\Lambda = \frac{\lambda \mu I^* S^*}{\Lambda} + aS^* + \mu S^*,\ aS^* = \mu V_1^* + bV_1^* + \frac{\lambda_1 \mu I^* V_1^*}{\Lambda},\ bV_1^* = \mu V_2^* + \frac{\lambda_2 I^* V_2^* \mu}{\Lambda},\ \frac{(\lambda S^* + \lambda_1 V V_1^* + \lambda_2 V_2^*)I^* \mu}{\Lambda} = \epsilon E^* + \mu E^*,\ \epsilon E^* = \mu I^* + \delta I^* + \beta I^*,\ \beta I^* - \mu R^*$

After simplification we get

$\dot{V} = [\frac{\lambda \mu I^* S^*}{\Lambda} + aS^* + \mu S^* - (\frac{\lambda \mu}{I\Lambda} + a + \mu)S](1 - \frac{S^*}{S}) + [\mu V_1^* + bV_1^* + \frac{\lambda_1 \mu I^* V_1^*}{\Lambda} - (\mu + b + \frac{\lambda_1 I \mu}{\Lambda})V_1](1 - \frac{V_1^*}{V_1}) + [\mu V_2^* + \frac{\lambda_2 I^* V_2^* \mu}{\Lambda} - (\mu + \frac{\lambda_2 I \mu}{\Lambda})V_2](1 - \frac{V_2^*}{V_2}) + [\epsilon E^* + \mu E^* - (\epsilon + \mu)E](1 - \frac{E^*}{E}) + [\mu I^* + \delta I^* + \beta I^* - (\mu + \delta + \beta)I](1 - \frac{I^*}{I}) + [\mu R^* - \mu R](1 - \frac{R^*}{R})$

At endemic states:
$\dot{V} = (\frac{\lambda\mu I^* S^*}{\Lambda} + aS^* + \mu S^*)(2 - \frac{S}{S^*} - \frac{S^*}{S}) + aS^*(1 - \frac{V_1^*}{V_1}\frac{S}{S^*}) + \frac{\lambda\mu I^* S^*}{\Lambda}(1 - \frac{S}{S^*}\frac{I}{I^*}\frac{E^*}{E}) + \frac{\lambda_1\mu I^* V_1^*}{\Lambda}(1 - \frac{I}{I^*}\frac{V_1}{V_1^*}\frac{E^*}{E}) + \frac{\lambda_2 I^* V_2^* \mu}{\Lambda}(1 - \frac{I}{I^*}\frac{V_2}{V_2^*}\frac{E^*}{E}) + bV_1^*(1 - \frac{V_1}{V_1^*}\frac{V_2^*}{V_2}) + \epsilon E^*(1 - \frac{E}{E^*}\frac{I^*}{I}) + \beta I^*(1 - \frac{I}{I^*}\frac{R^*}{R}) \leq 0$
Hence $V < 0$. We see that $V = 0$ iff $S = S^*, I = I^*$, $V_1 = V_1^*$, $V_2 = V_2^*$, $E = E^*$, $I_t = I_t^*$ and $R = R^*$. Thus the largest compact invariant set in $\{S(t), V_1(t), V_2(t), E(t), I(t), R(t)\} \in \Omega : V = 0$ is the Singleton $E^*$, where $E^*$ is the endemic equilibrium. Thus $E^*$ is globally asymptotically stable in the interior of the region $\Omega$. Global asymptotic stability shows that regardless of any starting solution, the solutions of the model will converge to $E^*$ whenever $R_0 > 1$. Epidemiologically, any perturbation of the model by the introduction of infectives shows that the model solutions will converge to the $E^*$ whenever $R_0 > 1$. This implies that the disease transmission levels can be kept quite low or manageable with minimal deaths at the peak times of the re-occurrence.

# 9 Sensitivity analysis

Parameter sensitivity is the degree to which an input parameter influences a model's output. Sensitivity analysis of $R_0$ can be used to develop a mitigation strategy that will slow the spread of COVID-19 by lowering $R_0$. Sensitive parameter include those that have a significant impact in the transmission dynamics of the infection. The sensitivity indices with respect to a parameter $X$ values are given in form of:

$$\chi_{R_0}^X = \frac{\partial R_0}{\partial X} \times \frac{X}{R_0} \tag{15}$$

Table 1 gives a summary of the sensitivity indices of $R_0$ evaluated at the baseline parameters values given in Table 2.

**Table 1:** *Sensitivity Index*

| Parameter | Description | Sensitivity Index |
|---|---|---|
| $\lambda$ | Transmission rate from $S$ to $E$ | $+1$ |
| $\epsilon$ | Transition rate from $E$ to $I$ | $\frac{\mu}{\epsilon+\mu} = +0.7915$ |
| $\beta$ | Human recovery rate | $\frac{-\beta}{\beta+\delta+\mu} = -0.9996$ |
| $\mu$ | Natural death rate | $\frac{-\mu}{\beta+\delta+\epsilon+2\mu} = -6.255 \times 10^{-5}$ |
| $\delta$ | Disease mortality rate | $\frac{-\delta}{\beta+\delta+\mu} = -8.237 \times 10^{-5}$ |

**Table 2:** *Parameter values and Sources*

| Parameter | Description | Unit/Unit Value |
|---|---|---|
| $\beta$ | Human recovery rate | 0.125 $\text{day}^{-1}$ |
| $\mu$ | Natural death rate | $3.91\times10^{-5}$ $\text{day}^{-1}$ |
| $\Lambda$ | Recruitment rate | $3.178\times10^{-5}$ $\text{day}^{-1}$ |
| $\lambda$ | Transmission rate from $S$ to $E$ | 0.02 $\text{day}^{-1}$ |
| $\lambda_1$ | Transmission rate from $V_1$ to $E$ | 0.01 $\text{day}^{-1}$ |
| $\lambda_2$ | Transmission rate from $V_2$ to $E$ | 0.005 $\text{day}^{-1}$ |
| $a$ | Rate of first dose vaccine | 0.4[0-1.0] |
| $b$ | Rate of second dose vaccine | 0.5[0-1.0] |
| $\delta$ | Disease mortality rate | $0.103\times10^{-5}$ $\text{day}^{-1}$ |
| $\epsilon$ | Transition rate from $E$ to $I$ | 0.5 $\text{day}^{-1}$ |

The reproduction number $R_0$ increases as the average number of contacts between infected/exposed individuals per unit time increases. On the other hand $\epsilon$, $\mu$, $\delta$ and $\beta$ are inversely proportional to $R_0$ . This implies that, increasing them wound decrease the $R_0$ even when the effects are not extreme. For instance, the sensitivity index for $R_0$ with respect to rate of moving from exposed class to infected class, $\epsilon$ is +0.7915 implying increasing (or decreasing) $\epsilon$ by 10 percent increases (or decreases) $R_0$ by 7.915 percent.

# 10 Numerical analysis

Numerical analysis is carried out using parameter values given in Table 2 Figure 1 shows that when $R_0 > 1$, then the population exposed to the virus

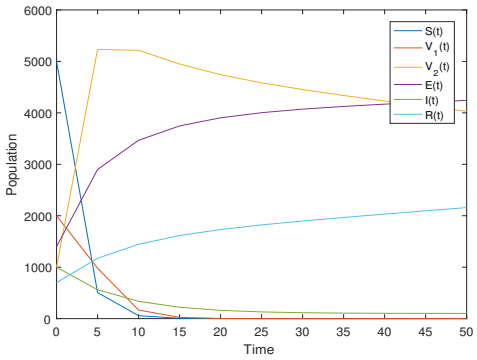


Figure 1: $R_0 > 1$

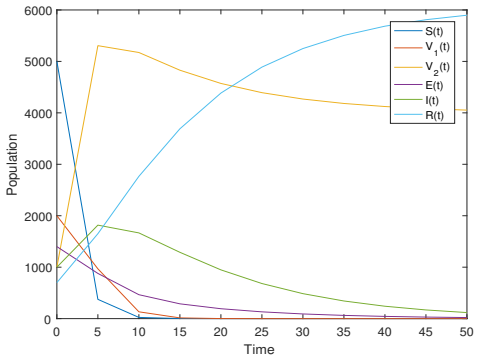


Figure 2: $R_0 < 1$

will be increasing meaning that the disease will be persistent in the population.

When $R_0 < 1$, then the people who have recovered from the disease will be increasing compared to all other compartments.

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