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On the Effects of Vaccination against COVID-19 on Herd Immunity in Kenya between March 2020 to March 2022

Wechuli N. Ralph*
Multimedia University of Kenya
Supervisors: Dr. George Mocheche, Dr. Iyaya Wanjala, Dr. Irene Watanga

Abstract: This study was undertaken to establish the effect of the vaccination drive against COVID-19 on the herd immunity of Kenyan citizens.

Two mathematical tools key in this area were used to determine Basic Reproduction Number (R0) which in turn was used to establish herd immunity. The two are the compartmental basic model: Susceptible, Infected, and Removed (SIR) model and the Next Generation Matrix (NGM). The necessary parameters required to calculate the R0 were computed in Python Software using the Ordinary Least Squares (OLS) technique using the data obtained from the Ministry of Health, Kenya.

Lastly, Stability analysis was carried out on the SIR model with and without vaccination. The Lyapunov function was used in determining the global stability of the SIR model. The results from the calculations have depicted a significant drop in the value of R0 for a period after the vaccination campaigns began as compared to the period before the vaccination process. This points towards an increased herd immunity to a level way above the Herd immunity Threshold. The results from this work will be useful to the government (both at local and national levels) in planning vaccination plans to protect the population against COVID-19 as well as other possible emerging epidemics in the future. The study will also be relevant and useful in future research in the epidemiological field.

Keywords: Basic Reproduction Number, Herd Immunity, SIR Model, Vaccination, Equilibrium points.

1. Introduction

1.1 Essentials of Herd Immunity.

The term herd immunity was first used by Topley and Wilson [1] in 1923. It has since helped to serve as the bedrock for vaccines and their applications, vaccination programs, cost analysis, and eradication of diseases such as smallpox.

Acquired immunity is developed at the individual level either through immunization with a vaccine or via natural infection with a pathogen[2]. Herd immunity stems from the effects of individual immunity to that of the entire population of a particular region. As such as long as a sizable percentage of a population has been vaccinated, immunity is rolled out to the entire population, even those who have not been vaccinated. This population-level effect aims to establish a population immunity so that individuals who cannot be vaccinated such as the young and immune compromised are still protected against the disease.

The herd immunity threshold depends on a single parameter known as the Basic Reproduction number, R0. The R0 refers to the average number of secondary infections caused by a single infectious individual introduced into a completely susceptible population.

If a pathogen with an R0 of 3 is considered for example, it means on average, one infected person is capable of infecting three others on average during the infectious period.

Mathematically, the Herd Immunity Threshold is defined by:

$$HIT = 1 - \frac{1}{R_0} \tag{1.1.1}$$

E.g. If:

$$R_0 = 3$$

$$HIT = 1 - \frac{1}{3} = 0.6667$$

The basic reproduction number, R_0, is a necessary parameter when dealing with an epidemic under control with vaccination [3]. One of the ways to reduce the reproduction rate of a disease is to reduce the number of susceptible in a population. Vaccination is the best way of achieving this. For example, it was successful in eradicating smallpox in the world in 1979[9]. Similarly, substantial progress has been made through vaccination to reduce and eventually eliminate polio in the world. In 1988, polio paralyzed an estimated 350,000 individuals per year in more than 125 countries. However, by 2019, 125 cases caused by wild poliovirus were reported globally[4] .

Mass vaccination is the cheapest and most effective means to control infectious diseases. Vaccination not only provides immunity to the individual but also provides it for the com-munity at large since it keeps the effective reproductive rate below the level that would allow the epidemic to grow. This is the basis for herd immunity.

Not all vaccinations provide herd immunity e.g. vaccinations against tetanus.[1]

1.2 Epidemiology

Epidemiological modeling of infectious disease transmission has had an increasing influence on the theory and practice of disease management and control. Mathematical modeling of the spread of infectious diseases has become part of epidemiology policy decision-making in many countries. Epidemiology is the study of the distribution and determinants of disease prevalence in humans [7].

In mathematical modeling, two techniques are often used:

1. Deterministic Model:

These models ignore random variation, and so always predict the same outcome from a given starting point. Deterministic models allow one to calculate a future event exactly without the involvement of randomness. An epidemiological model is not reality; it is an extreme simplification of reality. An example is a model using an equation such as:

$$y = x^2 \tag{1.2.1}$$

2. Stochastic Model:

These are more statistical and so may predict the distribution of possible outcomes. They can handle uncertainties in the inputs applied. They possess some inherent randomness in that the same set of parameter values and initial conditions will lead to sets of different outputs. Example: Finance (stock markets), earthquakes (variation in displacement and amplitude).

1.2.1 Purposes of epidemiological Modelling

1. Modeling provides concepts such as a threshold, reproduction number, etc.
2. Models with appropriate complexity can be constructed to answer specific questions.
3. Modeling can be used to estimate key parameters by fitting data.
4. Models provide structures for organizing, coalescing, and cross-checking diverse pieces of information.
5. Models can be used to assess the sensitivity of results to changes in parameter val-ues.
6. Modeling can suggest crucial data that needs to be collected.
7. Modeling can contribute to the design and analysis of epidemiological surveys.
8. Identifying the causes and risk factors of the disease spread.

1.2.2 Limitations of epidemiological Models

1. An epidemiological model is not reality; it is an extreme simplification of reality.
2. Deterministic models do not reflect the role of chance in disease spread and do not provide confidence intervals on results.
3. Stochastic models incorporate chance but are usually harder to analyze than the corresponding deterministic model.

By epidemiological model we refer to dynamic, deterministic modeling where the population is divided into compartments based on their epidemiological status such as susceptible, infectious, and recovered). The movement between compartments is specified using differential or difference equations.

Mathematical models have become important tools in analyzing the spread and control of infectious diseases. The model formulation process clarifies assumptions, variables, and parameters; moreover, models provide conceptual results such as thresholds, basic reproduction numbers, contact numbers, and replacement numbers.

Comparisons can lead to a better understanding of the processes of disease spread. Modeling can often be used to compare different diseases in the same population, the same disease in different populations, or the same disease at different times. Epidemiological models are useful in comparing the effects of prevention or control procedures. [8]

A lot has been published about the COVID-19 pandemic ever since its discovery in the Wuhan province of China. Being an infectious disease with a rampant spread among people due to close interaction, researchers in the Mathematics field have used various techniques available in the field to curb its spread. Such techniques include: SIR, SEIR, etc.

1.3 COVID-19 Background

Coronaviruses (CoVs) are a family of viruses that cause respiratory and intestinal illnesses in humans and animals. There are seven Human Coronaviruses that have been identified to date. Four of them are common, less high risk, and cause only mild respiratory illnesses in healthy humans. They include: HKU1, HCoV-OC43, HCoV-229E and HCoV-NL63. The other three are known to cause more severe illnesses in humans. These include:

1. Middle East Respiratory Syndrome (MERS-CoV)
2. Severe Acute Respiratory Syndrome (SARS-CoV)
3. Severe Acute Respiratory Syndrome 2 (SARS-CoV-2) [5]

The disease caused by SARS-CoV-2 was named COVID-19 by the World Health Organization. This virus was first identified in the Wuhan province of China in December 2019[6]. The Virus is known to transmit from human to human through body fluids. Patients with COVID-19 exhibit clinical symptoms that appear in the form of no symptoms at all (patients being asymptomatic) to having: fever, cough, sore throat, general weakness, fatigue, muscular pains, etc. [4].

The first case of COVID-19 was confirmed in Kenya on 12th March 2020 in Nairobi city.

Ever since the virus has spread over the country so far with 323, 921 confirmed cases, out of which 5, 649 succumbed to COVID-19 while 318, 097 recovered from the virus [21]. As a country, the government of Kenya tried its best to vaccinate its population against COVID-19. Kenya received 17,871,145 [21] doses of vaccines. Assuming each individual was to get two doses, it implies that in a population of 55, 944, 116 [39], approximately 16% percent were vaccinated. Among all the forty-seven counties in Kenya, Nairobi City County was the worst hit by the virus, with reported 129,123 cases as of 31st March 2022 [37].

The government of Kenya implemented severe measures to curb the spread of the disease including a lockdown of the city, social distance observance in public places, wearing of face masks as well and hygiene measures (washing of hands and sanitization practices). The implementation of these measures was implemented to the letter in Nairobi more than in any other place in Kenya.

According to research conducted in 2020, approximately 80% of those who have a mild case of COVID-19, close to the common cold, recover without needing any special treatment. This largely depends on the strength of the immune system of the particular individual. Still, as per the World Health Organization (WHO), one in every six people who get infected becomes seriously ill. The elderly and people with underlying medical conditions such as high blood pressure, heart problems, diabetes, etc. are at greater risk of serious illness from COVID-19. This is because to such individuals vaccinations will do much more harm than good.[6].

Despite the acquisition of COVID-19 vaccines by the government, few Kenyans responded to the government's appeal to get vaccinated. Below are some of the reasons that led to low vaccination drive turnout:

1. Some believed the drugs had been developed in haste.
2. Lack of trust in the vaccines. Those infected could get reinfected.
3. Religious reasons: some denominations don't believe in modern medication.
4. Fear: some saw how others reacted after being vaccinated and kept off themselves.

2. Literature Review

In 1985, Anderson and May [9], in their study on herd immunity pointed out that the point at which the proportion of susceptible people falls below the threshold needed for transmission is known as the Herd Immunity Threshold (HIT). Above this level, herd immunity begins to take effect and susceptible individuals benefit from the indirect protection from infection.

In 2014, Ochoche and Gweryina [12], in their research on vaccination against measles in Nigeria, concluded that not all susceptibles in a population need to be vaccinated against a disease before the disease can be eliminated. There is a threshold level above which an infectious disease will cease to persist. When this level

is exceeded by vaccination as well as natural immunity the disease can be eliminated without necessarily vaccinating the entire population. This concept is known as herd immunity.

In 2020, Haley E. R. et al. [2], proposed two ways of establishing herd immunity against Covid-19:

1. Mass vaccination campaign, which requires the development of an effective and safe vaccine.
2. Natural immunization of global population over time. However, the consequences of the latter are serious and far-reaching since a large population of humans would need to become infected with the virus, out of whom millions may succumb to it.

They thus concluded that in the absence of vaccines, establishing herd immunity should not be the ultimate goal. Instead, an emphasis should be placed on policies that would safeguard the vulnerable in the population in the hope that herd immunity would be achieved as a byproduct of such measures, although not a primary objective itself.

In 2020, Martin Nyamu et al. [13] in their work on modeling COVID-19 in Kenya obtained a basic reproduction number of 1.2 from the data available on the Kenyan population. This implied that about 16.67% of the Kenyan population needed to be vaccinated to stop the spread of the virus and achieve the desired herd immunity.

In 2020, Imam A. F. et al. [14], in their paper, concluded that applying the concept of herd immunity to the Indonesians would be controversial because till then no vaccine had been found.

In 2020, Robinson J. et al. [15] Analyzed the situation in a couple of countries regarding the situation of COVID-19 and the effects it had on their economies. In the study, they used Sweden as an example of an economy that relied heavily on herd immunity for survival. It never underwent any lockdown during the entire period of COVID-19.

In 2021, Randy et al. [16], in their study on the effect of vaccination on COVID-19 progression and herd immunity in the Philippines concluded that herd immunity could be achieved faster using vaccination as opposed to naturally induced herd immunity. They did a comparison of the various available vaccines in the Philippines to determine which was the most effective; Pfizer-BioNTech is the best vaccine for decreasing the number of susceptible infections while increasing the number of fully immune individuals. It is fol-lowed closely by Moderna and Sputnik.

In 2021, Hoque et al. [17], in their study on the progression of COVID-19 in Bangladesh came to the conclusion that to achieve herd immunity in the said country, 31% of the total population has to be vaccinated.

In 2021, Chowdhary S. et al. [18], in their work on universality and herd immunity threshold through a study over several countries concluded that the COVID-19 epidemic will start decreasing when Herd immunity exceeds the Herd Immunity Threshold, which means the herd immunity has greater value than the herd immunity threshold. They hence proposed two different ways of disease reduction:

1. By increasing herd immunity of the population over the herd immunity threshold. or
2. By decreasing the herd immunity threshold below the herd immunity of the population.

In 2021, Soni M. et al. [19], in their paper on the basic reproduction number and herd immunity for COVID-19 in India using data from March 2020 to January 2021 established that the R_0 ranged from 1. 2561 to 3 translating to a herd immunity ranging from 20% to 66%. They arrived at a mean value of 2.0546 for the basic reproduction number translating to 51% herd immunity In 2021, Garcia D. et al. [20], in their study of herd immunity in the Spanish population considered different combinations of elements that R_0 depends on such as the virus itself, characteristics of the populations and their environment. Further, they established that R_0 still depends on the methodology used, the accuracy of the data, and the generation time distribution. Estimates of R_0 for the population of Spain were established to range from 1.39 to 3.10. With these values, the herd immunity threshold ranges from 28.1% to 67.7% which makes 70% a realistic upper bound for Spain.

2.1 Statement of the Problem

A lot has been written on infectious diseases especially recently when the COVID-19 pandemic hit the world. Studies on enhancing herd immunity for epidemiological diseases such as smallpox, Influenza, and measles have been carried out extensively. However, little has been done on COVID-19's herd immunity, especially in developing sub-Saharan African countries in the said period. This study builds on existing work but with a focus on Kenya, to establish that the infection levels within the country could have gone down due to herd immunity developed by the population after a substantial number of people were vaccinated. The herd immunity could have well been acquired naturally by the populace who may have acquired the pathogens and recovered on their own without seeking medical attention. This can be attributed to the COVID-19's Delta

variant which was very dominant in late July 2021[21]. Acquisition of this led the majority of individuals to develop natural immunity against COVID-19 which contributed to herd immunity in the country.

2.2 Objective of the Study

2.2.1 Main Objective:

To establish the relationship between vaccination and herd immunity of the Kenyan population during the COVID-19 pandemic period.

2.2.2 Specific objectives:

1. To determine the Basic Reproduction Numbers (R_0) before and after COVID-19 vaccination exercise.
2. To use the calculated R_0 numbers above to establish the trend in Herd Immunity.
3. To carry out the stability analysis to determine the behaviour of the SIR model with and without vaccination.

2.3 Significance of the Study

The COVID-19 pandemic resulted in significant deaths of Kenyans since its first occurrence in the country in March 2020. Aside from the deaths, the pandemic has also stalled economic progress for many people most of whom have no formal employment and rely on day-to-day casual jobs to make ends meet. Such like a mass it becomes difficult to lock it up due to the pandemic. As some blatantly put it persuading the government to open up the economy (removal of curfews, lockdowns): "Better let us die of COVID-19 while looking for food for our households than die locked up in the house due to starvation." In the study, we have been able to establish that indeed vaccination exercises against COVID-19 exercise boosted herd immunity in the Kenyan population and thus lowered the infection rates. Such a positive result would encourage people to go for vaccination not only for COVID-19 but also for similar epidemics that may arise in the future. Furthermore, the result from this work will be useful to the government and other relevant authorities in planning for vaccinations to protect the population against COVID-19, and other diseases of similar infectious nature in the future. It will also be of benefit to the researchers in the same field.

3. Methodology

3.1 Introduction

This chapter outlines various expounds on the SIR model and the associated techniques and methods that will be employed to achieve our objectives as outlined in chapter two.

3.2 Study Design

To obtain herd immunity, one needs to calculate the R_0 first. The best way to arrive at the herd immunity of a population is to employ the use of compartmental models. Compartmental models are crucial mathematical tools used to establish the spread of infectious diseases. There are several such models in use since their inception. In a nutshell, compartmental models divide the population into different compartments which gives rise to various differential equations which are manipulated to give the desired output depending on the data given.

3.2.1 Susceptible, Infected and Removed (SIR) Model

For this study, to describe the dynamics of COVID-19 in Kenya we will use the SIR model whose initials stand for: Susceptible, Infected, and Removed. At any given time, individuals in a population, depending on their current status can be placed into different model compartments of the SIR Structure. SIR model together with the Next Generation Matrix will help us establish the Basic Reproduction Number in a population and hence determine the Herd immunity.

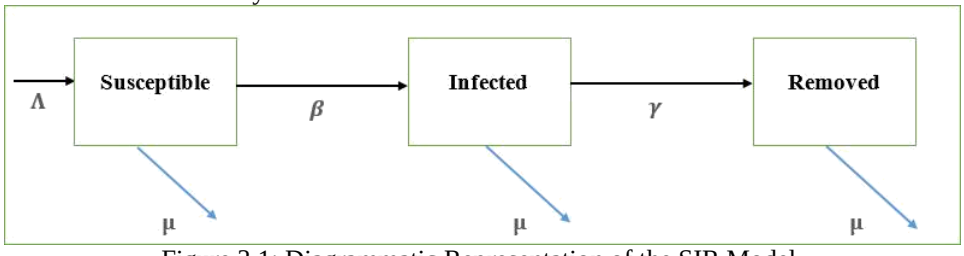


Figure 3.1: Diagrammatic Representation of the SIR Model.

In 1927, Kermack and McKendrick formulated a mathematical theory of the epidemic processes whose basis was a simple deterministic model i.e. SIR model [10]. Many other models have been extended from it to date.

The SIR model was selected for this work due to its simplicity and the nature of the data collected for this study.

3.2.2 Differential Equations:

The SIR model results in the following differential equations:

$$\begin{aligned} \frac{dS}{dt} &= \Lambda - \beta SI - \mu S \\ \frac{dI}{dt} &= \beta SI - \gamma I - \mu I \\ \frac{dR}{dt} &= \gamma I - \mu R \end{aligned} \tag{3.2.1}$$

$$\mathbf{N=S+I+R}$$

3.2.3 Description of Variables and Parameters used in the model.

1. **S**: The fraction of susceptible individuals (those able to contract the disease),
2. **I**: The fraction of infectious individuals (those capable of transmitting the disease),
3. **R**: The fraction of recovered individuals (those who have become immune).
4. **N** : The total population size.
5. Λ: Per-capita entry rate.
6. β: Disease transmission rate.
7. γ: Per- capita recovery rate.
8. μ: Per-capita removal rate.

Assumptions

The following assumptions hold:

1. Constant/closed population size, N.
2. Constant rates i.e. transmission and removal rates.
3. A well-mixed population i.e. one where an infected person has a probability of contacting any susceptible individual.

3.3 Next Generation Matrix(NGM) Method

In mathematical epidemiology, the Next Generation Matrix is used for deriving the Basic Reproduction Number in a compartmental model depicting the spread of infectious diseases. It is not only exclusive to epidemiology but it is also used in population dynamics to calculate the basic reproduction number for structured population models.[11] In the NGM, the R_0 is defined as the spectral radius of the Next Generation Operator. In Mathematics, the **spectral radius** of a square matrix is the maximum of the absolute values of its eigenvalues.

To calculate the basic reproduction number by using a next-generation matrix, the whole population is divided into several compartments

3.3.1 Next Generation Matrix Procedure:

Here are the steps followed to derive the R_0 Using NGM:

1. Regroup the model equations into infected and non-infected classes
2. For the infected class, we rearrange as shown:

$$\frac{dX}{dt} = \mathcal{F}(X,Y) - \mathcal{V}(X,Y); \tag{3.3.1}$$

Where

F= the rate at which new infected people enter the compartment

V= the transfer of individuals out of the compartment

F and V are m x m matrices which are obtained as follows:

3. We get F by differentiating the term in F partially with respect to the dependent variables.

$$F = \frac{\partial \mathcal{F}}{\partial I} \tag{3.3.2}$$

4. We get V by differentiating the terms in V partially with respect to the dependent variables.

$$V = \frac{\partial \mathcal{V}}{\partial I} \tag{3.3.3}$$

5. Find matrix FV^{-1}. This matrix is known as the Next Generation Matrix.
6. From the matrix result above, we find the eigenvalues. The dominant eigenvalue yields the R_0

Next-generation matrices are computationally worked out from the data collected, which is often the most productive approach where there are large numbers of compartmental.

3.3.2 Finding R_0 Using NGM

The Infected class is:

$$\frac{dI}{dt} = \beta SI - (\gamma + \mu)I \tag{3.3.4}$$

Which corresponds to:

$$\frac{dX}{dt} = \mathcal{F}(X, Y) - \mathcal{V}(X, Y); \tag{3.3.5}$$

Where

F= the rate at which new infected people enter the compartment
V= the transfer of individuals out of the compartment

Assumptions

The following assumptions hold:

1. $F_i(0, y)=V_i(0, y)=0$; for all $y > 0$
   (no new infections if no new infected people)
2. $F_i(x, y) \geq 0$; for all $x_i \geq 0$ and $y_i \geq 0$
   (no new infections if no new infected people)
3. $V_i(0, y) \geq 0$; for all $y_i \geq 0$ (empty compartments can only have inflows)
4. $\sum_i V_i(x, y) \geq 0$; for all $x_i \geq 0$ and $y_i \geq 0$ (Sum is net outflow)
5. System $y' = V_i(0, y)$ has unique asymptotically stable equilibrium, y*

Thus:

$$\mathcal{F}(I) = \beta SI$$
$$\mathcal{V}(I) = (\mu + \gamma)I \tag{3.3.6}$$

To obtain F and V:

$$F = \frac{d\mathcal{F}}{dI} = \beta S$$
$$V = \frac{d\mathcal{V}}{dI} = (\mu + \gamma) \tag{3.3.7}$$

Evaluating F and V at the disease-free equilibrium point, i.e. (S*, I*, R*)=(1,0,0):

We obtain

$$F = \beta(1) = \beta$$
$$V = (\mu + \gamma) \tag{3.3.8}$$

The inverse of V will be:

$$V^{-1} = \frac{1}{(\mu + \gamma)} \tag{3.3.9}$$

Thus:

$$FV^{-1} = \beta * \frac{1}{(\mu + \gamma)} = \frac{\beta}{(\mu + \gamma)} \tag{3.3.10}$$

This gives our R_0 i.e.

$$R_0 = \frac{\beta}{(\mu + \gamma)} \tag{3.3.11}$$

3.4 Ordinary Least Square Method

Regression analysis is a fundamental statistical technique that entails modeling the relationship between a dependent variable and one or more independent variables. The Ordinary Least Squares (OLS) method is one of the most commonly used techniques for regression analysis [11].

OLS is a linear regression technique used to find the best-fitting line for a set of data points by minimizing the residuals i.e. the differences between the observed and predicted values. An alternative word for residual could be **error**. It does so by estimating the coefficients of a linear regression model by minimizing the sum of the squared differences between the observed values of the dependent variable and the predicted values from the model. It is easy to use and produces decent results hence its popularity.

Other techniques include:

1. Weighted Least Squares (WLS)
2. Alternating Least Squares (ALS)
3. Partial Least Squares (PLS)

The OLS method is used to estimate the unknown parameters in a model. The method relies on minimizing the sum of the squared residuals between the actual values and the predicted values from the model.

The sum of the squared differences is called the **Residual Sum of Squares (RSS**). OLS works to minimize the RSS by finding the values of the coefficients that result in the smallest possible RSS. The resulting line is called the **regression line**. This represents the best fit for the data. Mathematically, OLS can be represented as:

$$\textbf{Minimize } \Sigma(y_i - \tilde{y}_i)^2 \tag{3.4.1}$$

Where $\mathbf{y_i}$ is the actual value and $\tilde{y}_\mathbf{i}$ is the predicted value.
The linear regression model used for determining the value of the response variable, $\mathbf{y_i}$, is represented as the equation below:

$$\mathbf{y = b_0 + b_1x_1 + b_2x_2 + ... + b_nx_n + e} \tag{3.4.2}$$

Where:

1. **y** is the dependent variable
2. $\mathbf{b}_0$ is the intercept
3. $\mathbf{b}_1$, $\mathbf{b}_2$, ...,$\mathbf{b}_n$ are the coefficients of the independent variables $\mathbf{x}_1$, $\mathbf{x}_2$, ...,$\mathbf{x}_n$
4. **e** is the error term

The OLS method is used to estimate the unknown parameters ($\mathbf{b}_1$, $\mathbf{b}_2$, ...,$\mathbf{b}_n$) by minimizing the sum of the squared residuals (RSS).

Assumptions of OLS:
The following assumption ought to be valid when working with OLS:

1. **Linearity**: There must be a linear relationship between the dependent and indepen-dent variables.
2. **Independence**: The observations must be independent of each other.
3. **Homogeneity of Variances**: The variance of the residuals should be constant across all the levels of the independent variables.
4. **Normality**: The residuals should be normally distributed.

3.5 Data Collection

The necessary data required for calculation of the Basic Reproduction Number for COVID-19 in Kenya and hence determine the herd immunity was obtained from Kenya's Ministry of Health website [21]. It is a collection of data that was issued daily by the ministry officials with respect to the COVID-19 scenario in the country. It can thus be classified as the secondary data.

3.6 Study Population

The sample data used in the study as obtained from the Ministry of Health, Kenya, was distributed across the country. The population of Kenya as of the year 2022 was approximately 54, 027, 487 [39]. However, for this study sample population used in our study period indicated was 2, 926,470 people. The pandemic did not affect the country uniformly; urban areas were adversely affected compared to rural areas.

Day	Date	Total	Infected	Discharged	Deaths
1	19-Mar-20	173	7	0	0
2	24-Mar-20	82	9	0	0
3	26-Mar-20	74	3	1	0
4	28-Mar-20	81	7	0	1
5	29-Mar-20	69	4	0	0
6	30-Mar-20	260	8	0	0
7	31-Mar-20	234	9	0	0
8	01-Apr-20	380	22	2	0
9	02-Apr-20	662	29	0	2
10	03-Apr-20	362	12	0	1
11	04-Apr-20	372	4	0	1
12	05-Apr-20	530	16	0	0
13	07-Apr-20	696	14	3	0
14	09-Apr-20	308	5	4	1
15	10-Apr-20	504	5	10	0
16	11-Apr-20	491	2	2	0
17	12-Apr-20	766	6	1	0
18	13-Apr-20	674	11	15	1
19	14-Apr-20	694	8	1	0
20	15-Apr-20	803	9	12	1

Table 3.1: A snapshot of COVID-19 Data for the first twenty days
Source: Ministry of Health, Kenya

Day	Date	Total	Infected	Discharged	Deaths
1	03-Apr-21	7139	1184	220	20
2	04-Apr-21	6045	911	533	18
3	05-Apr-21	2753	460	178	20
4	06-Apr-21	2923	394	2217	14
5	07-Apr-21	7423	1523	616	18
6	08-Apr-21	11352	1698	456	16
7	09-Apr-21	7300	1091	533	17
8	12-Apr-21	2989	486	115	20
9	13-Apr-21	6417	991	370	26
10	14-Apr-21	7529	981	655	26
11	15-Apr-21	5958	1091	392	4
12	16-Apr-21	7753	1041	343	19

13	17-Apr-21	7184	1024	382	20
14	18-Apr-21	3664	366	280	18
15	19-Apr-21	2515	241	636	20
16	20-Apr-21	5832	629	1560	18
17	22-Apr-21	5673	904	88	20
18	23-Apr-21	7036	773	762	23
19	24-Apr-21	9316	1153	191	20
20	25-Apr-21	4194	469	304	19

Table 3.2: A snapshot of COVID-19 Data for the first twenty days in Kenya after the introduction of the Vaccines

Source: Ministry of Health, Kenya

4. Findings, Data Analysis and Discussion

4.1 Introduction

This chapter deals with the calculation of the R_0 and herd immunity using the techniques outlined in the previous chapter. Thereafter, stability analysis on the SIR model is carried out; i.e. without and with vaccination.

4.2 Findings

4.2.1 Pre-Vaccination Findings

COVID-19 pre-vaccination data between March 2020 to March 2021 was fed into the Python program to determine the average of the parameters necessary for determining the R_0. The code used to accomplish this is shown in the appendix.

The resulting values were:
$\beta = 0.0394399$
$\gamma = 0.0275976$
$R_0 = 1.42874$

Using equation (1.1.1), we get the resulting Herd Immunity Threshold as 0.3001 i.e. 30.01% of the population needed to be vaccinated to control the spread of the virus in the country. We also determined the R_0 numbers after each month for the said period prior to the vaccination drive. This together with their resulting Herd Immunity Threshold is tabulated in Table 4.1 below.

R_0 and Herd Immunity Before Vaccination			
S/no:	**Period(Days)**	**R_0**	**HIT(1-1/R_0)**
1	30	1.4504	0.3105
2	60	2.7061	0.6305
3	90	1.7015	0.4123
4	120	1.6337	0.3879
5	150	2.4101	0.5851
6	180	1.7256	0.4205
7	210	1.4738	0.3215
8	240	1.4607	0.3154
9	270	1.421	0.2963

Figure 4.1: R_0 and Herd immunity figures before Vaccination Exercise.

4.2.2 Post-Vaccination Findings

Similarly, COVID-19 data from April 2021 to March 2022 was also fed into Python soft-ware for the computation of parameters necessary to determine the R_0 number. It should be noted that at this period vaccination drive against COVID-19 was underway.

We obtained the following values:
$\beta = 0.14131$
$\gamma = 0.13307$

$R_0 = 1.06191$

Again, using equation (1.1.1), we got the resulting Herd Immunity to be 0.05829. This translates to 5.829%. i.e. the portion of the population that now needs to be vaccinated to prevent the spread of the COVID-19 virus in the population.

The accompanying figures for the R_0 numbers as well as the resulting Herd Immunity

Threshold for each month are shown in Table 4.2 below:

R_0 and Herd Immunity After Vaccination			
S/no:	**Period(Days)**	**R_0**	**HIT(1-1/R_0)**
1	30	1.0914	0.0837
2	60	1.0389	0.0374
3	90	1.0391	0.0376
4	120	1.0581	0.0549
5	150	1.0787	0.0729
6	180	1.0719	0.0671
7	210	1.0722	0.0673
8	240	1.1241	0.1104
9	270	1.1743	0.1484
10	300	1.0708	0.0661
11	330	1.061	0.0575

Figure 4.2: R_0 and Herd immunity figures during Vaccination Exercise.

4.3 Stability Analysis For SIR Model Without Vaccination

Susceptible, Infected, and Removed model's equations form a dynamical system; since all three variables vary over time. The stability analysis exercise helps us get answers to the following questions:

1. Do we have constant solutions?
2. If that is the case, do the solutions near the constant move towards or away from the constant solutions?
3. How do the solutions behave as time, t approaches infinity?
4. Do any solutions oscillate?

The constant solution is generally referred to as equilibrium. The phase portraits of the dynamical system will either show the solutions having vectors moving towards the equilibrium value or away from the equilibrium value. If the solutions tend toward the equilibrium value, such point will be considered **stable** or **an attractor.**

On the other hand, if the solutions of the system near the equilibrium value all tend away from the value, such point is said to be **unstable**, or **a repelling point.**

We may have a situation where both phenomena happen i.e. some values tend towards the equilibrium point while some move away from it. This is called **a saddle point**. It is generally unstable.

Terminologies in Stability Analysis

1. **Local Stability**- Local stability means that all solutions of the system that have initial values within a particular domain of the feasible region approach the equilibrium point. Local stability of an equilibrium point means that if you put the system some-where near the point then it will move itself to the equilibrium point in some time. If DFE or EE is locally stable then all the solutions near the stable equilibrium will evolve with time to the equilibrium point. It also means that the equilibria are stable to small perturbations i.e. if you push the situation a bit out from the equilibrium point, then the situation will return on its own.
2. **Global Stability**-Global stability means that all solutions of the system approach the equilibrium point independent of the initial values. It means the system will come to the equilibrium point point from any possible starting point.

Some cases show an equilibrium point at the origin but all trajectories near the equilibrium point stay a small distance away. This is a stable equilibrium point, but it is not globally asymptotically stable.

The case where both eigenvalues are real, negative, and distinct produces a phase portrait that shows all trajectories tending toward the equilibrium point as $t \to \infty$, the value of x(t) gets small, so it is a globally stable equilibrium point.

4.3.1 Determining Local Stability of the System

From our model, we had the following system of equations:

$$\frac{dS}{dt} = \Lambda - \beta SI - \mu S \tag{4.3.1}$$

$$\frac{dI}{dt} = \beta SI - \gamma I - \mu I \tag{4.3.2}$$

$$\frac{dR}{dt} = \gamma I - \mu R \tag{4.3.3}$$

Where N=S(t)+I(t)+R(t)

With the initial conditions:

1. $S(0) \geq 0$
2. $I(0) \geq 0$
3. $R(0) \geq 0$

It is obvious that only two variables are listed in the above system of equations, therefore it is enough to consider the first two equations only i.e.:

$$\frac{dS}{dt} = \Lambda - \beta SI - \mu S \tag{4.3.4}$$

$$\frac{dI}{dt} = \beta SI - \gamma I - \mu I \tag{4.3.5}$$

The following set is positively invariant for the above system of equations:
$\Omega=\{(S(t), I(t)) \in \mathbf{R}^2_+, S(t) + I(t) \leq 1\}$

We get the equilibrium points by setting the equations (4.2.4) and (4.2.5) above equal to zero and solving the system for S and I. For our model, two equilibrium points exist:

1. Disease Free Equilibrium Point E_0 (S=1 and I=0). This is at the very beginning.
2. Endemic Equilibrium Point, $E^*(S = \frac{\gamma+\mu}{\beta}, I = \frac{\beta\Lambda-\mu(\gamma+\mu)}{\beta(\gamma+\mu)})$

We solve for I to obtain the positive solution from equation (4.2.4):

$$I = \frac{\Lambda - \mu S}{\beta S} \tag{4.3.6}$$

We substitute the equation (4.2.6) into equation (4.2.5) at equilibrium point:

$$\beta IS - \gamma I - \mu I = 0 \tag{4.3.7}$$

$$(\frac{\Lambda - \mu S}{\beta S})\beta S - \gamma(\frac{\Lambda - \mu S}{\beta S}) - \mu(\frac{\Lambda - \mu S}{\beta S}) = 0 \tag{4.3.8}$$

Simplifying:

$$\Lambda - \mu S - \frac{\gamma\Lambda}{\beta S} + \frac{\gamma\boldsymbol{\mu}}{\beta} - \frac{\mu\Lambda}{\beta S} + \frac{\boldsymbol{\mu}^2}{\beta} = 0 \tag{4.3.9}$$

Multiplying by -S:

$$-\Lambda S+\mu S^2+\frac{\gamma\Lambda}{\beta}-\frac{\gamma\boldsymbol{\mu}S}{\beta}+\frac{\mu\Lambda}{\beta}-\frac{\boldsymbol{\mu}^2S}{\beta}=0 \tag{4.3.10}$$

But $\mu=\Lambda$

Therefore:

$$\mu S^2-\boldsymbol{\mu}S-\frac{\gamma\boldsymbol{\mu}S}{\beta}-\frac{\boldsymbol{\mu}^2S}{\beta}+\frac{\gamma\boldsymbol{\mu}}{\beta}+\frac{\mu^2}{\beta}=0 \tag{4.3.11}$$

Factoring out μ, we get:

$$\mu(S^2-S-\frac{\gamma S}{\beta}-\frac{\boldsymbol{\mu}S}{\beta}+\frac{\gamma}{\beta}+\frac{\mu}{\beta})=0 \tag{4.3.12}$$

This results in:

$$S^2-(1+\frac{\gamma}{\beta}+\frac{\boldsymbol{\mu}}{\beta})S+\frac{\gamma}{\beta}+\frac{\mu}{\beta}=0 \tag{4.3.13}$$

Or:

$$S^2-(1+\frac{1}{R_0})S+\frac{1}{R_0}=0 \tag{4.3.14}$$

For $R_0=\frac{\beta}{\mu+\gamma}$

Solving the above, the discriminant, D, of the equation:

$D=(1+\frac{1}{R_0})^2-\frac{4}{R_0}$

For the positive solution of the equation $D\geq 0$ or

$(1+\frac{1}{R_0})^2-\frac{4}{R_0}\geq 0$

Or:

$(1+\frac{\gamma}{\beta}+\frac{\boldsymbol{\mu}}{\beta})^2-4(\frac{\gamma}{\beta}+\frac{\mu}{\beta})\geq 0$

We apply Jacobian to look at the linear stability of the equilibrium points:

$$J(S,I)=\begin{bmatrix}\frac{\partial}{\partial S}(\Lambda-\beta SI-\mu S) & \frac{\partial}{\partial I}(\Lambda-\beta SI-\mu S)\\ \frac{\partial}{\partial S}(\beta IS-\gamma I-\mu I) & \frac{\partial}{\partial I}(\beta IS-\gamma I-\mu I)\end{bmatrix}$$

$$J(S,I)=\begin{bmatrix}-\beta I-\boldsymbol{\mu} & -\beta S\\ \beta I & \beta S-\gamma-\boldsymbol{\mu}\end{bmatrix}$$

Disease Free Equilibrium Point

Evaluating the Jacobian matrix at E_0:

At E_0, S=1 and I=0:

$$J(1,0)=\begin{bmatrix}-\boldsymbol{\mu} & -\beta\\ 0 & \beta-\gamma-\boldsymbol{\mu}\end{bmatrix}$$

The corresponding characteristic equation for the Jacobian matrix at E_0 is:

$$\begin{vmatrix}-\boldsymbol{\mu}-\lambda & -\beta\\ 0 & \beta-\gamma-\boldsymbol{\mu}-\lambda\end{vmatrix}=0$$

Solving the above, we get the following eigenvalues:

$$\lambda_1 = -\boldsymbol{\mu} \qquad (4.3.15)$$
$$\lambda_2 = \beta - \gamma - \boldsymbol{\mu}$$

λ_1 is obviously less than 0

Looking at λ_2:

If:

$\beta - \gamma - \boldsymbol{\mu} < 0$, then

$\beta < \gamma + \boldsymbol{\mu}$ Which implies:

$$\frac{\beta}{\gamma+\boldsymbol{\mu}} < 1$$

Or

$R_0<1$

This shows that both eigenvalues are negative hence the Disease Free Equilibrium point is locally asymptotically stable. This implies that the disease will vanish from the population.

The Disease Free Equilibrium Point will be unstable if $\beta - \gamma - \boldsymbol{\mu} > 0$

Endemic Equilibrium Point

$$J(S,I) = \begin{bmatrix} -\beta I - \boldsymbol{\mu} & -\beta S \\ \beta I & \beta S - \gamma - \boldsymbol{\mu} \end{bmatrix}$$

At E^*:

$$S = \frac{\gamma+\mu}{\beta}$$

$$I = \frac{\beta\Lambda - \mu(\gamma+\mu)}{\beta(\gamma+\mu)}$$

Given earlier stated conditions: $\Lambda = \mu$

$$I = \frac{\boldsymbol{\mu}(\beta-\gamma-\mu)}{\beta(\gamma+\mu)}$$

Putting the above into the Jacobian matrix:

$$J(S,I) = \begin{bmatrix} -\beta I - \boldsymbol{\mu} & -\beta S \\ \beta I & \beta S - \gamma - \boldsymbol{\mu} \end{bmatrix}$$

$$J(S,I) = \begin{bmatrix} -\beta\left(\frac{\boldsymbol{\mu}(\beta-\gamma-\mu)}{\beta(\gamma+\mu)}\right) - \boldsymbol{\mu} & -\beta\left(\frac{\gamma+\mu}{\beta}\right) \\ \beta\left(\frac{\boldsymbol{\mu}(\beta-\gamma-\mu)}{\beta(\gamma+\mu)}\right) & \beta\left(\frac{\gamma+\mu}{\beta}\right) - \gamma - \boldsymbol{\mu} \end{bmatrix}$$

$$J(S,I) = \begin{bmatrix} -\left(\frac{\mu(\beta-\gamma-\mu)}{(\gamma+\mu)}\right) - \mu & -(\gamma+\mu) \\ \left(\frac{\mu(\beta-\gamma-\mu)}{(\gamma+\mu)}\right) & (\gamma+\mu) - \gamma - \mu \end{bmatrix}$$

$$J(S,I) = \begin{bmatrix} -\frac{\mu(\beta-\gamma-\mu+\gamma+\mu)}{\gamma+\mu} & -(\gamma+\mu) \\ \frac{\mu(\beta-\gamma-\mu)}{(\gamma+\mu)} & 0 \end{bmatrix}$$

$$J(S,I) = \begin{bmatrix} -\frac{\mu\beta}{\gamma+\mu} & -(\gamma+\mu) \\ \frac{\mu(\beta-\gamma-\mu)}{(\gamma+\mu)} & 0 \end{bmatrix}$$

From the above, we get the characteristic equation:

$$\begin{vmatrix} -\frac{\mu\beta}{\gamma+\mu} - \lambda & -(\gamma+\mu) \\ \frac{\mu(\beta-\gamma-\mu)}{(\gamma+\mu)} & 0-\lambda \end{vmatrix} = 0$$

Evaluating, we get:

$$\lambda^2 + \left(\frac{\mu\beta}{\gamma+\mu}\right)\lambda + \mu(\beta-\gamma-\mu) = 0 \qquad (4.3.16)$$

Solving equation (4.2.16):

$$\lambda = \frac{1}{2}\left[-\frac{\mu\beta}{\gamma+\mu} \pm \left\{\left(\frac{\mu\beta}{\gamma+\mu}\right)^2 - 4\mu(\beta-\gamma-\mu)\right\}^{\frac{1}{2}}\right]$$

Alternatively:

$$\lambda = \frac{1}{2}\left[-\mu R_0 \pm \left\{\mu^2 R_0^2 - 4\mu(\beta-\gamma-\mu)\right\}^{\frac{1}{2}}\right]$$

Since both coefficients in equation (4.2.16) above are both positive, it follows that the quantity of the discriminant under the square root is either smaller than $\mu^2R^2_0$, or greater than $\mu^2R^2_0$:

- If $\mu^2R^2_0 < 4\mu(\beta-\gamma-\mu)$

Then the eigenvalues are complex with the real part $-\mu R_0$ which is a negative value also.

- If $\mu^2R^2_0 > 4\mu(\beta-\gamma-\mu)$

Then the discriminant value under the square root must be smaller in absolute value than $\mu^2R^2_0$, but still the real part is negative. As such, either way, we conclude that the Endemic Equilibrium is stable since the real parts of both eigenvalues are negative. This shows that the endemic equilibrium point is stable.

The susceptible and infected population will survive in either of the cases and the phase planes will move toward the Endemic Equilibrium Point.

Conclusion

1. From the linear stability of the equilibrium points above, it can be observed that disease-free equilibrium and Endemic Equilibrium points cannot exist at the same time.
2. If $R_0 < 1$, then the Disease Free Equilibrium point is stable while if $R_0 > 1$ then the Endemic Equilibrium point is stable.

4.3.2 Determining Global Stability of the System

We analyze the global stability of the disease-free equilibrium and Endemic Equilibrium using the Lyapunov function.

Definitions:

- A function V(x,y) is said to be **positive definite** on a region Ω containing the origin if ∀(x, y)≠ 0, V(x, y) > 0.
- A function V(x,y) is said to be **negative definite** on a region Ω containing the origin if ∀(x, y)≠ 0, V(x, y) < 0.
- A function V(x,y) is said to be a **Lyapunov Function** on an open region Ω if the function is continuous, positive definite and has a continuous first Order Partial Derivatives on Ω.
- LaSalle's Invariance Principle is a criterion for the asymptotic stability of an au-tonomous dynamical system.

The autonomous systems to which LaSalle's Invariance Principle is applicable should be of the form:

$$x^{\cdot} = f(x),\ f(0) = 0 \tag{4.3.17}$$

Theorem 4.3.1. Let x = 0 be an equilibrium point for the autonomous system (4.2.17) above. Let

V: Ω → R be a **continously differentiable positive definite** function on a domain $\Omega \subset R^n$ containing the origin, such that $\dot{V}(x(t)) \leq 0$ in Ω. Let $S = \{x \in \Omega : \dot{V}(x) = 0\}$ and suppose that no other solution can stay in S, other than the trivial solution x(t) ≡ 0. Then, the origin is **locally asymptotically stable**. If, in addition, V(x) is radially unbounded, then the origin is **globally asymptotically stable**

Disease Free Equilibrium Point

To establish the global stability of the Disease Free Equilibrium point we will use the Lyapunov function:

Theorem 4.3.2. If $R_0 < 1$, then the Disease Free Equilibrium point of the system is globally asymptotically stable on Ω.

Proof. To establish the global stability of the Disease Free Equilibrium point, we construct the following Lyapunov function V : Ω → R

Where V(S, I)=I(t).

The Time derivative of V is:

$$\frac{dV}{dt}(S,I) = \frac{dI}{dt} \tag{4.3.18}$$

$$\frac{dV}{dt}(S,I) = \beta SI - \gamma I - \mu I \tag{4.3.19}$$

$$\frac{dV}{dt}(S,I) = \beta SI - (\gamma + \mu)I \tag{4.3.20}$$

$$\frac{dV}{dt}(S,I) = I\{\beta S - (\gamma + \mu)\} \tag{4.3.21}$$

$$\frac{dV}{dt}(S,I) = I(\gamma + \mu)\{\frac{\beta}{\mu + \gamma}S - 1\} \tag{4.3.22}$$

But $R_0 = \frac{\beta}{\mu+\gamma}$, hence:

$$\frac{dV}{dt}(S,I) = I(\gamma + \mu)\{R_0 S - 1\} \tag{4.3.23}$$

Thus $\frac{dV}{dt}(S,I) \leq 0$ for $R_0 \leq 1$

Furthermore:

- If $R_0 < 1$, then

  $\frac{dV}{dt}(S, I) = 0$; for at Disease Free Equilibrium point I(t)=0.

- If $R_0 = 1$, then,

  $\frac{dV}{dt}(S, I) = 0$, for at Disease Free Equilibrium point, S(t)=1.

Thus by LaSalle's Invariance Principle, the Disease Free Equilibrium point is asymptotically stable.

Endemic Equilibrium Point

Theorem 4.3.3. If there exists a Lyapunov function V(x,y), dependent on a system $\frac{dx}{dt}$ =f(x,y) and $\frac{dy}{dt}$ =g(x,y with equilibrium point (x,y)=(0,0), and $\frac{dV}{dt}$ is negative definitive on an open region R containing the origin, then the zero solution of the system is asymptotically stable.

If R contains all the possible values of (x,y) and satisfies theorem 4.3.3, the resulting stability of the system is said to be **globally stable**.

The derivative of V with respect to the system $\frac{dx}{dt}$ =f(x,y) and $\frac{dy}{dt}$ =g(x,y) is defined as:

$$\frac{dV}{dt} = \frac{\partial V}{\partial x}\frac{dx}{dt} + \frac{\partial V}{\partial y}\frac{dy}{dt}$$

Alternatively, we can determine the Endemic Equilibrium point using the theorem below:

Theorem 4.3.4. The Endemic Equilibrium point $E^*(S^*, I^*)$ of the system is globally asymptotically stable on Ω.

Proof. We use a Lyapunov function $V : \Omega_+ \rightarrow \mathbf{R}$, where $\Omega_+ = \{S(t), I(t) \in \Omega$ such that S(t) >

0 and I(t) > 0}.

Our function V is given by:

$$V(S, I) = \Phi[S - S^* ln(\frac{S}{S^*})] + \Psi[I - I^* ln(\frac{I}{I^*})] \tag{4.3.24}$$

Where Φ and Ψ are constants.
Differentiating the Lyapunov function with respect to time, we get:

$$\frac{dV}{dt} = \frac{\partial V}{\partial S}\frac{dS}{dt} + \frac{\partial V}{\partial I}\frac{dI}{dt} \tag{4.3.25}$$

$$\frac{dV}{dt} = \Phi[(1 - \frac{S^*}{S})(\Lambda - \beta SI - \mu S)] + \Psi[(1 - \frac{I^*}{I})(\beta SI - \gamma I - \mu I)] \tag{4.3.26}$$

$$\frac{dV}{dt} = \Phi[(\frac{S - S^*}{S}) * S(\frac{\Lambda}{S} - \beta I - \mu)] + \Psi[(\frac{I - I^*}{I}) * I(\beta S - \gamma - \mu)] \tag{4.3.27}$$

$$\frac{dV}{dt} = \Phi[(S - S^*)(\frac{\Lambda}{S} - \beta I - \mu)] + \Psi[(I - I^*)(\beta S - \gamma - \mu)] \tag{4.3.28}$$

Considering the equilibrium point, we get:

$$\mu = \frac{\mu}{S^*} - \beta I^* \quad \text{and} \quad \gamma + \mu = \beta S^*$$

This results in the following equation:

$$\frac{dV}{dt} = \beta(S - S^*)(I - I^*)(\Phi - \Psi) - \mu\Phi\frac{(S - S^*)^2}{SS^*} \quad (4.3.29)$$

Further:

- If $\Phi = \Psi = 1$, then

$\frac{dV}{dt} = -\mu\Phi\frac{(S-S^*)^2}{SS^*} \leq 0$

- If $S = S*$, then,

$\frac{dV}{dt} = 0$

Hence, by Lasalle's Invariance Principle, the endemic equilibrium point is globally asymptotically stable.

4.4 Stability Analysis For the SIR Model With Vaccination

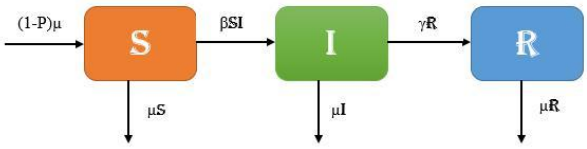


Figure 4.3: Diagrammatic Representation of the SIR Model with Vaccination.

In this model, we analyze the stability of the model with induced vaccination. With vaccination, we will have a system of equations as follows:

$$\frac{dS}{dt} = (1 - P)\Lambda - \beta SI - \mu S \quad (4.4.1)$$

$$\frac{dI}{dt} = \beta SI - \gamma I - \mu I \quad (4.4.2)$$

$$\frac{dR}{dt} = \gamma I - \mu R \quad (4.4.3)$$

$$\frac{dV}{dt} = P\mu - \mu V \quad (4.4.4)$$

Where:

a. **S** is the **S**usceptible population.
b. **I** is the **I**nfected Population.
c. **R** is the **R**ecovered population.
d. **V** is the **V**accinated Population.
e. Λ is the admission rate into the population..
f. **μ** is the mortality rate Population.
g. **P** is the Vaccination rate.
h. γ is the **R**ecovery rate.

And:

S+I+R+V=1

Only three variables are listed in the system of equations above. Therefore, equation (4.3.3) may be ignored.

But $\mu=\Lambda$

Therefore, we only consider:

$$\frac{dS}{dt} = (1-P)\mu - \beta SI - \mu S \tag{4.4.5}$$

$$\frac{dI}{dt} = \beta SI - \gamma I - \mu I \tag{4.4.6}$$

$$\frac{dV}{dt} = P\mu - \mu V \tag{4.4.7}$$

The following set is positively invariant for the above system of equations:
$\Omega=\{(S(t), I(t), V(t)) \in \mathbf{R}^3_+, S(t) + I(t) + V(t) \leq 1\}$

4.4.1 Determining Local Stability of the System

Two equilibrium points exist for the above model:

a. Disease Free Equilibrium point $E_0(S = 1 - P, I = 0, V = P)$

b. Endemic Equilibrium point $E^*(S = \frac{\gamma+\mu}{\beta}, I = \frac{\mu(\beta(1-P)-\gamma-\mu)}{\beta(\gamma+\mu)}, V = P)$

To show the existence of an endemic equilibrium point, we determine the value of I from equation (41). This value we substitute in equation (42), to get:

$$S^2 - (1 - P + \frac{\gamma}{\beta} + \frac{\mu}{\beta})S + \frac{(1-P)\gamma}{\beta} + \frac{(1-P)\mu}{\beta} = 0 \tag{4.4.8}$$

Whose discriminant, D, is:

$$D = (1 - P + \tfrac{\gamma}{\beta} + \tfrac{\mu}{\beta})^2 - 4(\tfrac{(1-P)\gamma}{\beta} + \tfrac{(1-P)\mu}{\beta})$$

For the positive solution, $D \geq 0$ i.e.

$$D = (1 - P + \tfrac{\gamma}{\beta} + \tfrac{\mu}{\beta})^2 \geq 0$$

The new reproduction number is $R_v = R_0(1 - P)$ and Endemic Equilibrium point will only exist if $R_v > 1$

We apply the Jacobian to determine the linear stability of the equilibrium points:

$$J(S,I,V) = \begin{bmatrix} \frac{\partial}{\partial S}((1-P)\mu - \beta SI - \mu S) & \frac{\partial}{\partial I}((1-P)\mu - \beta SI - \mu S) & \frac{\partial}{\partial V}((1-P)\mu - \beta SI - \mu S) \\ \frac{\partial}{\partial S}(\beta SI - \gamma I - \mu I) & \frac{\partial}{\partial I}(\beta SI - \gamma I - \mu I) & \frac{\partial}{\partial V}(\beta SI - \gamma I - \mu I) \\ \frac{\partial}{\partial S}(P\mu - \mu V) & \frac{\partial}{\partial I}(P\mu - \mu V) & \frac{\partial}{\partial V}(P\mu - \mu V) \end{bmatrix}$$

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

Disease Free Equilibrium

At $E_0(S = 1 - P, I = 0, V = P)$, Hence:

$$J(S,I,V) = \begin{bmatrix} -\mu & -\beta(1-P) & 0 \\ 0 & \beta(1-P) - \gamma - \mu & 0 \\ 0 & 0 & -\mu \end{bmatrix}$$

The characteristic equation corresponding to the Disease Free Equilibrium points is:

$$\begin{vmatrix} -\mu - \lambda & -\beta(1-P) & 0 \\ 0 & \beta(1-P) - \gamma - \mu - \lambda & 0 \\ 0 & 0 & -\mu - \lambda \end{vmatrix} = 0$$

From the above determinant, we get three eigenvalues:

$\lambda_1 = -\mu$

$\lambda_2 = -\mu$

$\lambda_3 = \beta(1-P) - \gamma - \boldsymbol{\mu}$

It is evident that λ_1 and λ_2 are negative. Checking λ_3:

a. If $\lambda_3 > 0$, it implies, $\beta(1 - P) > \gamma + \boldsymbol{\mu}$

Meaning:

$R_0(1 - P) > 1$ or

$R_v > 1$

This means the disease-free equilibrium point is not locally asymptotically stable.

b. If $\lambda_3 < 0$, this implies, $\beta(1 - P) < \gamma + \boldsymbol{\mu}$

Meaning:

$R_0(1 - P) < 1$ or

$R_v < 1$

This means the system is stable since all eigenvalues are negative. This will imply there will be no epidemic.

The trajectories will approach the disease-free equilibrium point.

Endemic Equilibrium

$$J(S,I,V) = \begin{bmatrix} -\beta I - \boldsymbol{\mu} & -\beta S & 0 \\ \beta I & \beta S - \gamma - \boldsymbol{\mu} & 0 \\ 0 & 0 & -\boldsymbol{\mu} \end{bmatrix}$$

At E^*:

$S = \frac{\gamma+\mu}{\beta}$

$I = \frac{\boldsymbol{\mu}(\beta(1-P)-\gamma-\mu)}{\beta(\gamma+\mu)}$

$V = P$

Putting the above into the Jacobian matrix:

$$J(S,I,V) = \begin{bmatrix} -\beta I - \boldsymbol{\mu} & -\beta S & 0 \\ \beta I & \beta S - \gamma - \boldsymbol{\mu} & 0 \\ 0 & 0 & -\boldsymbol{\mu} \end{bmatrix}$$

$$J(S,I,V) = \begin{bmatrix} -\beta\left(\frac{\boldsymbol{\mu}(\beta(1-P)-\gamma-\mu)}{\beta(\gamma+\mu)}\right) - \boldsymbol{\mu} & -\beta\left(\frac{\gamma+\mu}{\beta}\right) & 0 \\ \beta\left(\frac{\boldsymbol{\mu}(\beta(1-P)-\gamma-\mu)}{\beta(\gamma+\mu)}\right) & \beta\left(\frac{\gamma+\mu}{\beta}\right) - \gamma - \boldsymbol{\mu} & 0 \\ 0 & 0 & -\boldsymbol{\mu} \end{bmatrix}$$

$$J(S,I,V) = \begin{bmatrix} -\left(\frac{\boldsymbol{\mu}(\beta(1-P)-\gamma-\mu)}{(\gamma+\mu)}\right) - \boldsymbol{\mu} & -(\gamma+\mu) & 0 \\ \left(\frac{\boldsymbol{\mu}(\beta(1-P)-\gamma-\mu)}{(\gamma+\mu)}\right) & (\gamma+\mu) - \gamma - \boldsymbol{\mu} & 0 \\ 0 & 0 & -\boldsymbol{\mu} \end{bmatrix}$$

$$J(S,I,V) = \begin{bmatrix} -\frac{\boldsymbol{\mu}(\beta(1-P)-\gamma-\mu+\gamma+\mu)}{\gamma+\boldsymbol{\mu}} & -(\gamma+\mu) & 0 \\ \frac{\boldsymbol{\mu}(\beta(1-P)-\gamma-\mu)}{(\gamma+\mu)} & 0 & 0 \\ 0 & 0 & -\boldsymbol{\mu} \end{bmatrix}$$

$$J(S,I,V) = \begin{bmatrix} -\frac{\boldsymbol{\mu\beta(1-P)}}{\gamma+\boldsymbol{\mu}} & -(\gamma+\mu) & 0 \\ \frac{\boldsymbol{\mu}(\beta(1-P)-\gamma-\mu)}{(\gamma+\mu)} & 0 & 0 \\ 0 & 0 & -\boldsymbol{\mu} \end{bmatrix}$$

From the above, we get the characteristic equation:

$$\begin{vmatrix} -\frac{\boldsymbol{\mu}\beta(1-P)}{\gamma+\boldsymbol{\mu}} - \lambda & -(\gamma+\mu) & 0 \\ \frac{\boldsymbol{\mu}(\beta(1-P)-\gamma-\mu)}{(\gamma+\mu)} & -\lambda & 0 \\ 0 & 0 & -\boldsymbol{\mu} - \lambda \end{vmatrix} = 0$$

Evaluating the above determinant, we get:

$$(\mu+\lambda)\{\lambda^2 + (\frac{\boldsymbol{\mu}\beta(1-P)}{\gamma+\boldsymbol{\mu}})\lambda + \boldsymbol{\mu}[(\beta-\gamma-\mu)]\} = 0 \qquad (4.4.9)$$

It is noted that both coefficients $(\frac{\boldsymbol{\mu}\beta(1-P)}{\gamma+\boldsymbol{\mu}})\lambda$ and $\boldsymbol{\mu}[(\beta-\gamma-\mu)]$ are positive.

On solving the above equation we get the following eigenvalues:

$$\lambda_1 = -\mu$$

$$\lambda_2 = -\frac{\boldsymbol{\mu}\beta(1-P)}{(\gamma+\boldsymbol{\mu})} \pm \{\frac{\boldsymbol{\mu}^2\beta^2(1-P)^2}{(\gamma+\boldsymbol{\mu})^2} - 4\boldsymbol{\mu}[\beta(1-P)-\gamma-\mu]\}^{\frac{1}{2}}$$

Which is equivalent to:

$$\lambda_2 = -\boldsymbol{\mu}R_v \pm \{\boldsymbol{\mu}^2R_v^2 - 4\mu(\mu+\gamma)(R_v-1)\}^{\frac{1}{2}}$$

Since $\frac{\beta(1-P)}{(\gamma+\boldsymbol{\mu})} = R_0(1-P) = R_v$

Since μ[β(1 − P) − γ − μ] is positive, the quantity under the square root is either smaller than $\boldsymbol{\mu}^2R^2_v$, or it is greater.

If it is greater, then the solutions are complex with real part $-\boldsymbol{\mu}R_v$. If it is smaller in value than $\boldsymbol{\mu}^2R^2_v$, still the real part of the eigenvalue will be negative.

Thus, either way, it can be concluded that the Endemic Equilibrium is stable because both real parts of the eigenvalues have negative values and λ_1 is also negative.

This implies that the Endemic Equilibrium point is locally stable; both the susceptible and the infected will survive in either case.

It can also be seen that the infection rate has reduced because of the vaccination.

4.4.2 Determining Global Stability of the System

Disease Free Equilibrium

Theorem 4.4.1. The Disease Free Equilibrium point of the system is globally asymptotically stable on Ω

Proof. To establish the global stability of the Disease Free point, we make use of the following Lyapunov function L : Ω → R and L(S, I, V) = S(t) + I(t) + V(t).

Its derivative will be:

$$\frac{dL}{dt}(S,I,V) = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dV}{dt} \tag{4.4.10}$$

$$\frac{dL}{dt}(S,I,V) = (1-P)\mu - \beta SI - \mu S + \beta SI - \gamma I - \mu I + P\mu - \mu V \tag{4.4.11}$$

$$\frac{dL}{dt}(S,I,V) = \mu - P\mu - \mu S - \gamma I - \mu I + P\mu - \mu V \tag{4.4.12}$$

$$\frac{dL}{dt}(S,I,V) = \mu - \mu S - \gamma I - \mu I - \mu V \tag{4.4.13}$$

$$\frac{dL}{dt}(S,I,V) = (1-S)\mu - \mu V - (\gamma+\mu)I \tag{4.4.14}$$

$$\frac{dL}{dt}(S,I,V) = (\gamma+\mu)[\frac{(1-S)\mu R_0}{\beta} - \frac{\mu R_0 V}{\beta} - I] \tag{4.4.15}$$

$$\frac{dL}{dt}(S,I,V) = \frac{(\gamma+\mu)}{\beta}\{\mu R_0(1-S) - \mu V R_0 - I\} \tag{4.4.16}$$

$$\frac{dL}{dt}(S,I,V) = \frac{(\gamma+\mu)}{\beta}\{\mu R_0(1-S-V) - I\} \tag{4.4.17}$$

Thus:

If $R_0 < 1$, then

$\frac{dL}{dt}(S, I, V) < 0$; implying Disease Free Equilibrium is globally asymptotically stable.

Endemic Equilibrium

Theorem 4.4.2. The Endemic Equilibrium point $E^*(S^*, I^*, V^*)$ of the system is globally asymptotically stable on Ω

Proof. We use a Lyapunov function $L : \Omega_+ \rightarrow R$, where $\Omega_+ = \{S(t), I(t), V(t) \in \Omega$ such that $S(t) > 0$, $I(t) > 0$ and $V(t) > 0\}$ Our function L is given by:

$$L(S, I, V) = \Phi[S - S^* ln(\frac{S}{S^*})] + \Psi[I - I^* ln(\frac{I}{I^*})] + \Theta[V - V^* ln(\frac{V}{V^*})] \quad (4.4.18)$$

Where Φ, Ψ and Θ are constants.

Differentiating the Lyapunov function about time, we get:

$$\frac{dL}{dt} = \frac{\partial L}{\partial S}\frac{dS}{dt} + \frac{\partial L}{\partial I}\frac{dI}{dt} + \frac{\partial L}{\partial V}\frac{dV}{dt} \quad (4.4.19)$$

$$\frac{dL}{dt} = \Phi[(1 - \frac{S^*}{S})((1-P)\mu - \beta SI - \mu S)] + \Psi[(1 - \frac{I^*}{I})(\beta SI - \gamma I - \mu I)] + \Theta[(1 - \frac{V^*}{V})(\mu P - \mu V)] \quad (4.4.20)$$

$$\frac{dL}{dt} = \Phi[(\frac{S - S^*}{S}) * S(\frac{(1-P)\mu}{S} - \beta I - \mu)] + \Psi[(\frac{I - I^*}{I}) * I(\beta S - \gamma - \mu)] + \Theta[(\frac{V - V^*}{V}) * V(\frac{\mu P}{V} - \mu)$$

(4.4.21)

$$\frac{dL}{dt} = \Phi[(S - S^*)(\frac{(1-P)\mu}{S} - \beta I - \mu)] + \Psi[(I - I^*)(\beta S - \gamma - \mu)] + \Theta[(V - V^*)(\frac{\mu P}{V} - \mu)] \quad (4.4.22)$$

Considering the equilibrium point, we get:

$\mu = \frac{(1-P)\mu}{S^*} - \beta * I^*, \quad \gamma + \mu = \beta S^*, \quad V^* = P$

Putting these values into the equation above, we get:

$$\frac{dL}{dt} = -\mu\Phi(1-P)\frac{(S - S^*)^2}{SS^*} + \beta(\Phi - \Psi)(I - I^*)(S - S^*) - \mu\Theta(V - V^*)^2 \quad (4.4.23)$$

Further:

If $\Phi = \Psi + \Theta = 1$, then

$\frac{dL}{dt} = -\mu\Phi\frac{(S-S^*)^2}{SS^*} - \mu\Theta(V - V^*)^2 \leq 0$

If $S = S*$ and $V = V*$ then,

$\frac{dL}{dt} = 0$

Hence, by Lasalle's Invariance Principle, the endemic equilibrium point is globally asymptotically stable.

4.5 Simulations

We will simulate the results of both models using Python's Matplot function to display the relationship between the Susceptible population and the Infected population four months after the pandemic was reported in the country as well as four months after the inception of the vaccination program in the country.

4.5.1 Simulations for Period before Vaccinations

At the inception, we have the Disease Free Equilibrium where S = 1 and i = 0. This automatically implies that R = 0 also. This when run in Python, produces the graph 4.12 shown below. For the first month

since the inception of COVID-19, the calculated parameters are: $\beta = 0.0487299$, $\gamma = 0.0202825$, $\mu = 0.000813$ and $R_0 = 2.40256$

The endemic equilibrium points for the first four months are shown in the figure 4.5 be-low.

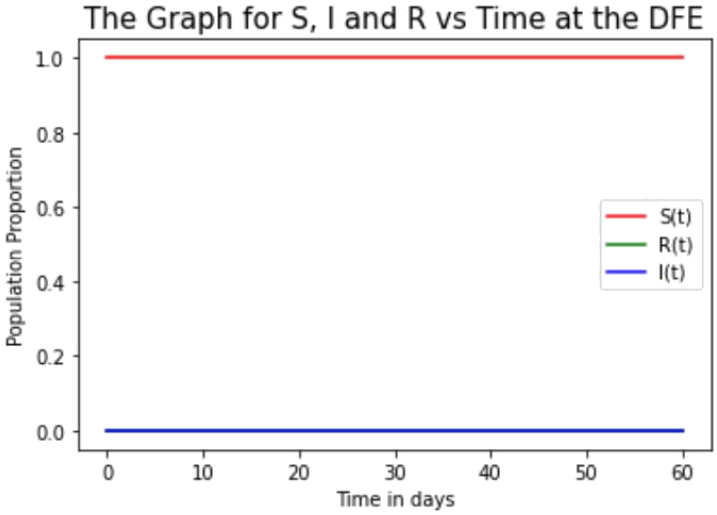


Figure 4.4: S, I and R relationship at DFE

Endemic Equilibrium Points Before Vaccinations.	
Month	**Endemic Equilibrium Point,E*(S,I)**
1	E*(0.432907, 0.021855)
2	E*(0.437873, 0.024769)
3	E*(0.359884, 0.036686)
4	E*(0.397177, 0.033888)

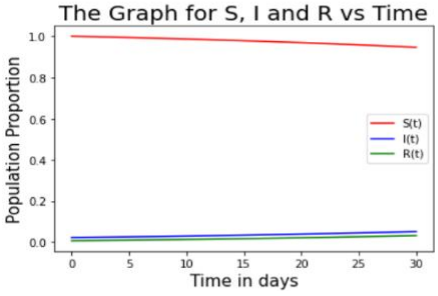
Figure 4.5: Endemic Points for the first four months of COVID-19

Remark. The susceptible population decreases due to the presence of infection whereas the infected population rises because of infection. The linear stability for both points is calculated. Using equation (4.2.15) eigenvalues the disease-free equilbrium are given by: $\lambda_1 = 0$ while $\lambda_2 = 2.142857$.

This implies that $R_0 = 2.142857 > 0$ hence the trajectories do not approach the disease free equilibrium point. Using the equation(4.2.16), the characteristic equation for the Endemic Equilibrium Point is given by: $\lambda^2 + 0.0019\lambda + 0.00002 = 0$

Its eigenvalues are: $\lambda_1 = -0.00095 + 0.00437i$ while $\lambda_2 = -0.00095 - 0.00437i$.

The real part for both eigenvalues is negative, hence the Endemic Equilibrium point is linearly stable as claimed from the theoretical results that if $R_0 > 0$ then the Endemic



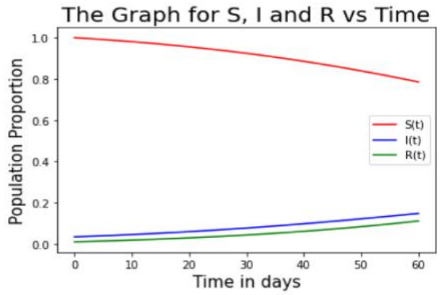


Figure 4.6: SIR Simulation for the 1st Month.

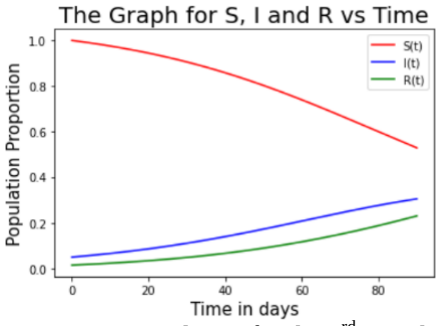


Figure 4.7: SIR Simulation for the 2nd Month.

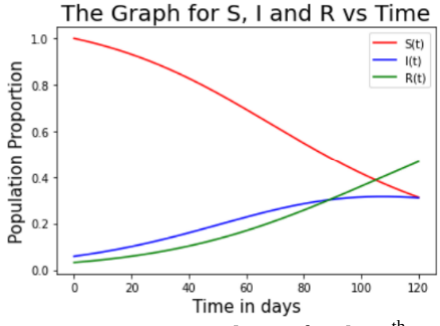


Figure 4.8: SIR Simulation for the 3rd Month.

Figure 4.9: SIR Simulation for the 4th Month.

Figure 4.10: SIR Simulation over time before vaccination drive.

Equilibrium Point is linearly stable. The graphs in 4.10 above depict the trend for the first four months.

4.5.2 Simulation For Period After Vaccinations

We use the same parameters as used in the above simulations but with the addition of a parameter, p, where p is the vaccination rate for COVID-19. The Endemic Equilibrium Points, E^* corresponding to the first four months after the inception of the vaccination drive in Kenya are given 4.11 below. It is noted that the susceptible population is re-

Endemic Equilibrium Points After Vaccination.	
Month	**Endemic Equilibrium Point,E*(S,I,V)**
1	E*(0.946022, 0.001599, 0.0031)
2	E*(0.745570, 0.062665, 0.0170)
3	E*(0.768893, 0.046577, 0.0200)
4	E*(0.756590, 0.040811, 0.0290)
5	E*(0.756967, 0.037387, 0.0348)

Figure 4.11: Endemic Equilibrium Points.

duced because of vaccination. The infected population reduces due to vaccination drive, whereas the vaccinated population increases. This is depicted in the graphs 4.17 below. The reproduction number, R_v, was obtained from:

$R_v = R_0(1 - p)$.

For the first instance above, $R_v = 0.946022(1 - 0.0031) = 0.9453089 < 1$ hence implies that the Disease Free Equilibrium is stable.

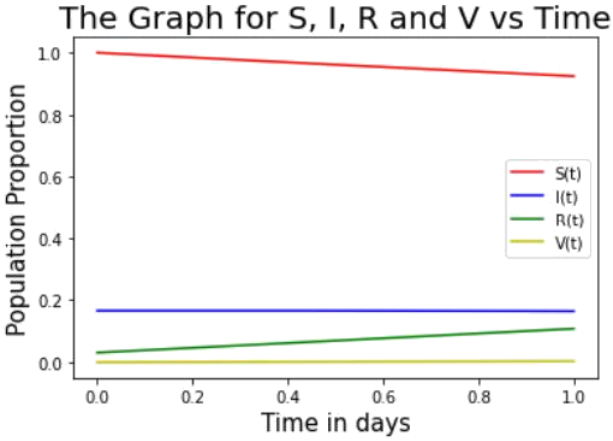


Figure 4.12: S, I, R and V relationship at DFE

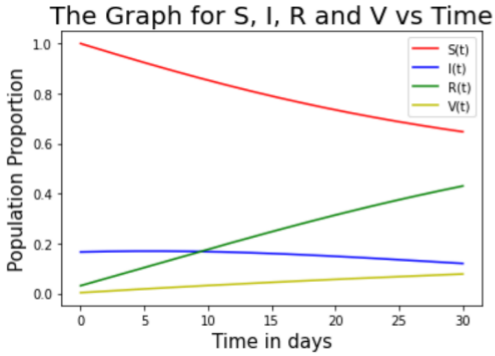


Figure 4.13: SIRV Simulation for the 1st Month.

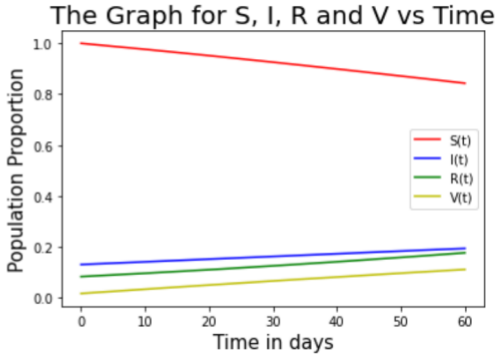


Figure 4.14: SIRV Simulation for the 2nd Month.

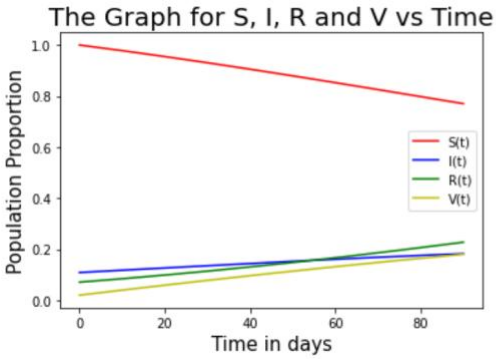


Figure 4.15: SIRV Simulation for the 3rd Month.

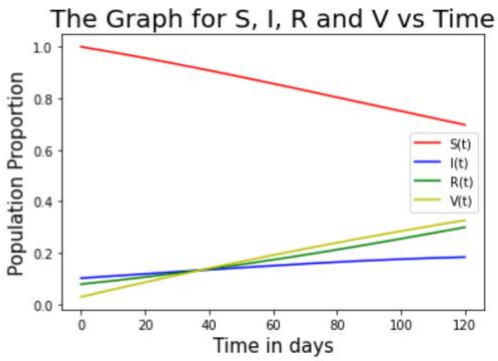


Figure 4.16: SIRV Simulation for the 4th Month.

Figure 4.17: SIRV Simulation over time after vaccination drive.

5. Conclusions and Recommendations

5.1 Introduction

The chapter uses the findings and results obtrained in chapter four to conclude the objectives as outlined in chapter two. Some possible recommendations for future research in this line are also outlined.

5.2 Conclusions

In summary, we tasked ourselves with establishing whether or not the Kenyan popula-tion gained herd immunity during the COVID-19 pandemic. Next, stability analysis was carried out on our SIR model.

5.2.1 Specific Objective One

First, we began by establishing the R_0 numbers for the distinct periods: before and after vaccinations. The R_0 number is at the core of determining herd immunity.

We established that the R_0 number for the Kenyan population before the vaccination drivewas 1.42874. This gave a herd immunity of 0.3001 when calculated, implying that 30.01% of the population needed to be vaccinated to have the virus spread under control. On the other hand, we got R_0 for the Kenyan population after the vaccination drive had started to be 1.06191, which translates to a Herd Immunity of 0.05829, implying that the population that now needs to be vaccinated to keep the pandemic under control is 5.829%.

5.2.2 Specific Objective Two

The reduction in herd immunity Threshold's figures in objective (1) above from 30.01% to 5.829% can be attributed to the herd immunity obtained via vaccination or naturally. Natural acquired Herd Immunity occours when an individual who got infected acquires immunity when he recovers from the illness without necessarily getting vaccinated.

5.2.3 Specific Objective Three

Lastly, we carried out the stability analysis on our SIR model without and with vaccination. It is noted that Vaccination contributes significantly to the acquisition of herd immunity. For both cases, it was noted that the dynamics of infection depended on the value of the R_0 number.

For the first model, SIR, without vaccination, it was established that the Disease Free Equilibrium (DFE) exists only when the $R_0 < 1$. In such scenarios, all the trajectories will be approaching the DFE. The stability of the local and global DFE points is also discussed. It was apparent that the two equilibriums, Disease Free Equilibrium and the Endemic Equilibrium, cannot coexist. Endemic Equilibrium exists only when $R_0 > 1$. Global and local stability of the Endemic Equilibrium was also explored.

For the second model, SIRV, vaccination was introduced, leading to a drastic change in the reproduction number, hence the Herd Immunity. As in the first model, the DFE is stable if $R_0 < 1$ and the trajectories approach the Endemic Equilibrium Point when $R_0 > 1$.

It was seen in the graph depictions, the effect of the infection rate, β, on the susceptible and the infected population. The susceptible population gradually decreases while the infected population on the other hand rises as the infection rate, β. Consequently, as the vaccination is introduced into the susceptible population, the infected population de-creases. This can be attributed to herd immunity which was facilitated by the vaccination drive.

5.3 Recommendation

The input brought about by the study above will be useful for managing and controlling epidemics in the future. The results, subsequent analysis, and simulations done above are not only restricted to COVID-19 but could also be applied to other epidemiological diseases that could break out in developing countries such as Kenya.

Future research could further examine the influence of herd immunity on a smaller geo-graphical area say, at the county level for example. This is because different counties were hit differently by the pandemic. It will also be phenomenal if thorough research on the comparative effects of herd immunity on different age groups e.g. how herd immunity compares between the young generation and the old generation.

The study can also be taken to the next level to determine how the two types of herd immunity compare in terms of effectiveness once acquired:

- Natural herd immunity
- Acquired herd immunity gained via vaccination.

References

[1] W. Topley, G. Wilson The Spread of Bacterial Infection. The Problem of Herd Immunity, Epidemiol. infect, Vol. **21**(1923), 243-249.
[2] H. Randolph, L. Barreiro, Herd Immunity: Understanding Covid-19, Journal of Math. Cellpress Immunity Primer, Vol. **52**(2020).
[3] J. Murray, Mathematical Biology. Volume I: Interdisciplinary Applied Mathematics, Springer-Verlag (2003)
[4] European Centre for Disease: COVID-19. Accessed April 18, 2022 from https://www.ecdc.europa.eu/en/coronavirus
[5] United Kingdom Research and Innovation. Accessed May 28, 2022 at: https://www.ukri.org
[6] World Health Organization's website: https://covid19.who.int/
[7] S. Ma, Y. Xia, H.Hethcote Mathematical Understanding of Infectious Disease Dynamics, National University of Singapore (2008)

[8] H. Hethcote, J. Yorke, Gonorrhea Transmission Dynamics and Control, Lecture Notes in Biomathematics, vol. 56, Springer-Verlag, Berling (1984)
[9] R. Anderson, R. May, Vaccination and herd Immunity to Infectious Diseases, Nature vol 318 (1991).
[10] Roy M. Anderson, Population Dynamics of Infectious Diseases: Theory and Applications, Chapman and Hall, 1st Edition (1982).
[11] Fred Braver, Mathematical Models in Population Biology and Epidemiology, Springer, 2nd Edition (2010).
[12] J. Ochoche, R. Gweryina, A Mathematical Model of Measles with Vaccination and Two Phases of Infectiousness, IOSR Journal of Mathematics(IOSR-JM) e-ISSN: 2278-5728, p-ISSN:2319-765X. Vol. **10**, Issue 1 Ver. IV. (Feb 2014), 95-105. www.iosrjournals.org
[13] M. Nyamu, B. Mutwiri, B. Lawrence, modelling of COVID-19(Coronavirus in Kenya using SIR Model, International Journal of Statistics and Applied Mathematics **5** (5) (2020),20-24, DOI: https://doi.org/10.22271/maths.2020.v5.i5a.579
[14] I. Faizal, N. Nugrahani, Herd Immunity and COVID-19 in Indonesia, Jurnal Teknologi Laboratorium Vol. **9** No. 1 (2020), 21-28.
[15] J. Robinson. l Banerjee, B. Sathian COVID-19: Is Herd Immunity the answer?, JAIM **18**,Number 2 (July-December 2020).
[16] R. Caga-anan, M. Raza, G. Shelda, E. Metillo, P. Castillo, Y. Mammeri, Effect of Vaccination to Covid-19 Disease Progression and Herd Immunity, De Gruyter, Vol. **9**(2020),262-272
[17] E. Hoque, S. Islam, S. Kumar, D. Mitra, M. Amin, Adjusted Dynamics of COVID-19 Pandemic due to Herd Immunity in Bangladesh, Department of Computer and Information Science, Fordham University, 441 E Fordham Rd, The Bronx, 10458 New York, USA.(2020).
[18] S. Chowdhury, S. Roychowdhury, I Chaudhuri, Universality and herd immunity thresh-old: Revisiting the SIR model for COVID-19, Department of Physics, St. Xavier's Col-lege, 30 Mother Teresa Sarani, Kolkata-700016, West Bengal, India, 2021, International Journal of Modern Physics.
[19] M. Soni, R. Sharma, S. Sharma, The basic reproduction number and herd immunity for COVID-19 in India, Indian Journal of Science and Technology **14** (35): 2773-2777. https://doi.org/10.17485/IJST/v14i35.797 (2021)
[20] D. Garcia-Garcia,E. Morales, E. Fonfria,I. Vigo, C. Bordehore, Caveats on COVID-19 Herd Immunity Threshold: the Spain Case, Department of Applied Mathematics, University of Alicante, Alicante, Spain, Multidisciplinary Institute for Environmental Studies "Ramon Margalef", University of Alicante, Spain, Department of Ecology, University of Alicante, Spain, June 2021.
[21] Ministry of Health, Kenya, 9th May report: https://www.health.go.ke/wp-content/uploads/2022/05/MINISTRY-OF-HEALTH-KENYA-COVID-19-IMMUNIZATION-STATUS-REPORT-9TH-MAY-2022.pdf
[22] J.M Heffernan, R.J Smith and L.M Wahl Perspectives on the basic reproductive ratio, Journal of the Royal Society Interface.
[23] Beau L. et al. " Modelling the Spread of Measles" (2020), Electronic Theses, Projects, and Dissertations. 1110.
[24] Clemente-Suarez V.J. et al. Dynamics of Population Immunity Due to the Herd Effect in the COVID-19 Pandemic, Vaccines, Vol. **8**, (2020), 236; doi:10.3390/vaccines8020236
[25] Diekman O. et al. On the definition and the Computation of the Basic Reproduction Ratio R0 in Models For Infectious-Diseases in Heterogeneous Populations, Article in Journal of Mathematical Biology.**28**, (1990), 365-382.
[26] Dolbeault J. et al. Heterogenoous Social Interactions and the COVID-19 Lockdown Out-come in a Multi-Group SEIR Model, PSL University: arXiv:2005.00049v2 [q-bio.PE] 23 Jun 2020
[27] Fosu G.O. et al. Next-Generation Matrices and Basic Reproductive Numbers for all Phases of the Coronavirus Disease, Open Journal of Mathematical Sciences, Vol **4**, (2020), 261-272; doi:10.30538/oms2020.0117
[28] Heffernan J.M. et al. Perspectives on the Basic Reproductive Ratio, Journal of the Royal Society Interface **2** (2005), 281-293 doi: 10.1089/rsif.2005.0042
[29] Jones J.H. et al. Notes on R0, Department of Anthropological Sciences, May 1, 2007, Stanford University.
[30] Kimathi M. et al. Age-structured model for COVID-19: Effectiveness of social distancing and contact reduction in Kenya, Infectious Disease Modelling. **6**,(2021),15-23
[31] KWOK K. O. et al. Herd immunity-estimating the level required to halt the COVID-19 epidemics in affected countries, Journal of Infection (2020), doi: https://doi.org/10.1016/j.jinf.2020.03.027

[32] Mallory ML. et al. Vaccination-Induced Herd Immunity: Successes and Challenges, Journal of Allergy and Clinical Immunology, (2018), doi:10.1016/j.jaci.2018.05.007.
[33] Matthias E. et al. SIR-based Mathematical Modeling of Infectious Diseases with Vaccination and Waning Immunity, Journal of Computational Science, (2019), doi: https//doi.org/10.1016/j.jocs.2019.101027
[34] Mbogo R.W. et al. SARS-COV-2 Outbreak and control in Kenya-Mathematical Model Analysis, Infectious Disease Modelling (2021), doi: https://doi.org/10.1016/j.idm.2021.01.009
[36] Momoh A.A. et al. Mathematical Model for Control of Measles Epidemiology, Indian Journal of Pure and Applied Mathematics, Vol. **87**, No. 5 (2013), 707-718
[37] Paul E.M. et al. Herd Immunity: History, Theory, Practice, Epidemiologic Reviews The John Hopkins University School of Hygiene and Public Health, Volume **15** No. 2(1993).
[38] Statista website: https://www.statista.com/statistics/1136519/cumulative-coronavirus-cases-in-kenya-by-county/
[39] World Population Review's website: https://worldpopulationreview.com/world-cities/nairobi-population
[40] World statistics: www. Worldometers. Info
[41] Supida Chauhan, Om Prakash Misra, Joydip Dhar. Stability Analysis of SIR Model with Vaccination, Amity Institute of Applied Sciences, American Journal of Computational and Applied Mathematics 2014, 4(1): 17-23 DOI: 10.5923/j.ajcam.20140401.03
[42] Youssef H. M. et al. Modified SEIR Model with Global Analysis Applied to the Data of Spreading COVID-19 in Saudi Arabia, Mechanical Engineering Department, College of Engineering and Islamic Architecture, Umm Al-Qura University, Makkah 21955, Saudi Arabia (2020), doi:10.1063/5.0029698

Appendix

Python code for obtaining R_0 before vaccination drive

```
# -*- coding: utf-8 -*-
"""
Created on Thu Apr 27 11:27:59 2023

@author: RALPH
"""

import pandas as pd
import numpy as np
import matplotlib.pyplot as plt
from statsmodels.formula.api import ols
df=pd.read_csv('cpre.csv')
df=df.sample(frac=1)
df.rename(columns={'day':'t','infected':'I'},inplace=True)
R=df['discharged']+df['deaths']
df['R']=R
logI= df['I'].transform([np.log])
df['logI']=logI
df1=df[['t','I','logI','R']]
df1=df1.iloc[0:275,:]
print(df1.head(5))

reg=ols('logI~t',data=df1).fit()
print(reg.summary())
k=reg.params['t']
b=reg.params['Intercept']

plt.plot(df1['t'],df1['logI'],'o')
plt.plot(df1['t'],(k*df1['t']+b))
plt.title('m estimate:%s'%k)
plt.grid()
plt.figure()

gr=[]
for i in range(0,272):
    oo=(df1['R'][i+1]-df1['R'][i])/df1['I'][i]
    gr.append(oo)
plt.plot(gr)
plt.grid(1)

gamma=np.mean(gr)
plt.title('gamma estimate: %s'%gamma)
plt.legend(fontsize=20,loc='upper left')
beta=k+gamma
print('gamma=%g beta=%g R0=%g' %(gamma, beta, beta/gamma))
```

Figure 5.1: Python Code for estimation of R_0 before Covid-19 vacccination practice.

```
4
5   @author: RALPH
6   """
7
8   import pandas as pd
9   import numpy as np
10  import matplotlib.pyplot as plt
11  from statsmodels.formula.api import ols
12  df=pd.read_csv('cpost.csv')
13  df=df.sample(frac=1)
14  df.rename(columns={'day':'t','infected':'I'},inplace=True)
15  R=df['discharged']+df['deaths']
16  df['R']=R
17  logI= df['I'].transform([np.log])
18  df['logI']=logI
19  df1=df[['t','I','logI','R']]
20  df1=df1.iloc[0:337,:]
21  print(df1.head(5))
22
23  reg=ols('logI~t',data=df1).fit()
24  print(reg.summary())
25  k=reg.params['t']
26  b=reg.params['Intercept']
27
28  plt.plot(df1['t'],df1['logI'],'o')
29  plt.plot(df1['t'],(k*df1['t']+b))
30  plt.title('m estimate:%s'%k)
31  plt.grid()
32  plt.figure()
33
34  gr=[]
35  for i in range(0,335):
36      oo=(df1['R'][i+1]-df1['R'][i])/df1['I'][i]
37      gr.append(oo)
38  plt.plot(gr)
39  plt.grid(1)
40
41  gamma=np.mean(gr)
42  plt.title('gamma estimate: %s'%gamma)
43
44  beta=k+gamma
45  print('gamma=%g beta=%g R0=%g' %(gamma, beta, beta/gamma))
46
47
```

Figure 5.2: Python Code for estimation of R_0 during/after vaccines Covid-19 vacccination practice.