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A Modified SIVR Mathematical Model of Covid-19 Post-vaccination Stability Analysis in Kenya

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Author's contribution

The sole author designed, analyzed, interpreted and prepared the manuscript.

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Abstract

In this paper, the study focused on carrying out a stability analysis on a modified SIR model for the COVID-19 pandemic for the period after vaccination in Kenya. The purpose of the work was to show that whereas the rate and the extent of disease spread amongst the Kenyan people was not as wide spread as happened in other parts of the world, it was necessary for government and policy makers to roll out a robust civic education to convince the majority of the Kenyan population to embrace vaccination as a major containment measure in curbing the spread of COVID-19 and other infectious diseases since the study revealed that the infections drastically reduced to near zero after vaccination except for a few isolated cases that were and still continue to exhibit mild symptoms to none at all. This was attributed to the development of the vaccine which upon a massive campaign by the Kenyan government, led into a significant portion of the population being vaccinated. It is believed this vaccination drive enhanced herd immunity amongst the population. This development has had a significant effect in the control of more recent COVID-19 variants like JN-1 that have

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remained largely mild and undetected in the country. Both local and global post vaccination stabilities were analyzed for the system using the Lyapunov function. The main objective of the study was therefore to carry out a post vaccination stability analysis by estimating key parameters such as the basic reproduction number, (R_0) and herd immunity threshold (HIT) for infectious diseases by fitting data into the model to enable predictions of future dynamics of the disease. The study used the Next Generation matrix and the least square method besides the Python software to solve generated differential equations of the model for R_0 and HIT parameters. Results obtained showed that, there was a significant reduction in infections due to enhanced herd immunity attributed largely to the roll out of vaccination.

Keywords: SIVR model; vaccination; herd immunity; local stability; global stability.

2010 Mathematics subject classification: 92B05, 93D15

1 Introduction

1.1 Background of COVID - 19

COVID – 19 named as such by the World Health Organization (WHO), [1] is a disease caused by a virus that belongs to a family of Coronaviruses (CoVs) that cause respiratory and intestinal illnesses in humans and animals. A number of these viruses have been identified before, with COVID-19 first identified in the Wuhan province of china in December 2019[(12)]. The virus gets transmitted from human to human through body fluids. Patients infected with COVID-19 range from those who don't exhibit clinical symptoms (referred to as Asymptomatic patients) to those having such common symptoms as fever, cough, sore throat, general body weakness, fatigue, muscular pains etc.

The first case of COVID-19 in Kenya was confirmed on 12th March, 2020 in Nairobi city. Ever since, the disease continued to spread exponentially in nearly all the regions until the roll out of vaccination upon the discovery of the vaccine. Elsewhere in India, Piu S, et al. [2] in their study established that COVID-19 had precipitated a major global crisis, with 968,117 total confirmed cases, 612,782 total recovered cases and 24,915 deaths in India as of July 15, 2020. At this rate, it was very necessary that every effort was to be put in place to curb the disease, including but not limited to researching on it to understand every aspect of its dynamics. Piu S. et al. [2] in their study further found out that in the absence of any effective therapeutics or drugs and with an unknown epidemiological life cycle, predictive mathematical models can aid in understanding of both the coronavirus disease control and management.

On the onset of the COVID-19 pandemic, particularly before the development of the vaccine, the rate at which the disease spread amongst the people was largely uncontrolled. Whereas there was restricted movement of people in urban centers, it was not practically possible to do so in rural and informal settlements where people depend on day to day manual jobs for their upkeep. In search of these casual jobs, people moved from one point to the next and hence had unlimited interactions with others which exposed them and their families back home with the possibility of contracting the disease. In the post vaccination period, much of the cases reported were as a result of re-infections. Isaac M.W., et al, [3] in their findings detailed how reinfection led into a surge in mortality rates and accumulation of COVID-19 active cases which the Kenyan health system could not handle. They further dettermined that even in the presence of reinfections, the surge in COVID-19 cases could be prevented by various intervention mechanisms through detection of asymptomatic individuals who unknowingly transmit the disease. It should however be acknowledged that in a country where people are reluctant to present themselves for testing, the only practical way out was to roll out vaccination across the populace.

In a study carried out by Iyaya C. W. et al. [4] they showed that COVID-19 does not affect all population groups equally. In their findings, they noted that age is the strongest risk factor for severe COVID-19 outcomes. Additionally, some chronic medical conditions occur more frequently in certain population groups and the risk of severe COVID-19 increases as the number of underlying medical conditions increase in an individual. Thus, old people and those with underlying medical conditions such as cardiovascular disease, diabetes, hypertension, chronic respiratory disease and cancer are more likely to experience serious illness from COVID-19. This called for a more urgent intervention mechanism to protect this group of persons because in Kenya, the Ministry of

Health estimated that 1 out of 3 people aged 58 years and above were either diabetic or hypertensive or both. This group of people alongside the frontline health workers needed protection. The government therefore prioritized groups of individuals for vaccination and other intervention measures to protect them against contracting the disease. It was estimated that the bulk of the reported fatalities in Kenya comprised of people living with underlying medical conditions due to old age.

The post vaccination period had two groups of people, the vaccinated and unvaccinated both of whom were assumed to have acquired herd immunity. Initially, it wasn't clear whether or not the infected and recovered had acquired everlasting or partial protective immunity. In the works of Bendadavid E., et al, [5] they pointed out that at the time, researchers believed, that the infected acquired 'passport' immunity and therefore required to be allowed to relax COVI-19 containment measures, including the freedom to mingle freely with the general public.

Edridge A.W.D., et al, [6] in a further research on serological testing for seasonal Human Coronavirus (HCoV-229E), found that the majority of patients lost 50% of the acquired antibodies after six months, 75% after a year and completely returned to baseline after four years pointing out to the need for a more reliable prevention mechanism such as the development of a vaccine for the disease.

This study sought to show that by carrying out a stability analysis, post the vaccination roll out, the extent of transmissions drastically reduced compared to the period before vaccination. The end result showed that vaccination indeed enhanced herd immunity amongst the Kenyan people, both rural and urban and there is therefore need to depart from their traditional and/or cultural beliefs and practices that had negative attitudes that slowed down vaccination rollout amongst the people with a majority of them in rural and informal settlements.

1.2 Model formulation

According to Diekmann O., et al, [7] mathematical modeling of transmission trends of infectious diseases has extensively been studied. These models have been used by many researchers to understand and predict transmission dynamics of infectious diseases. This study proposed a deterministic modified SIVR ordinary differential equation model that captures the COVID-19 dynamics, a member of the family of novel coronavirus or SARS-CoV-2. The study classified the Kenyan human population into four compartments, namely susceptible individuals (S), infected individuals (I), Vaccinated individuals (V) and Recovered individuals (R) to formulate, the SIVR (susceptible or uninfected (S) → infected individuals (I) → Vaccinated individuals (V) → Recovered individuals (R) model. The total size of the population is N(t) = S(t) + I(t) + V(t) + R(t). The study presupposed that it's only a section of the susceptibles that were vaccinated. Further, that only interactions between the infected and the susceptibles caused the transmission of the viruses. The proposed modified SIVR model for the Kenyan COVID-19 transmission dynamics is illustrated in Fig. 1. The model consists of the following set of nonlinear differential equations;

1.2.1 Differentia equations

The compartmental SIVR model together with the rates of change amongst the different parameters as shown in the diagram above yields the following differential equations

$$\frac{dS}{dt} = \Lambda - \beta SI - \rho S - \mu S \quad [1]$$

$$\frac{dI}{dt} = \beta SI - \mu I - \gamma I \quad [2]$$

$$\frac{dV}{dt} = \rho S - \tau V \quad [3]$$

$$\frac{dR}{dt} = \gamma I + \tau V - \mu R \quad [4]$$

with S(t) + I(t) + V(t) + R(t) = N(t)

List 1. Description of variables and parameters used in the model

Λ: Per-capita entry rate.	β: Disease transmission rate.
S: Susceptible individuals.	γ: Per- capita recovery rate.
I: Infectious individuals.	ρ: Vaccination rate
V: The vaccinated individuals.	μ: Per-capita removal rate
R: Recovered individuals	τ: Recovery rate for the vaccinated
N(t): total size of the population	

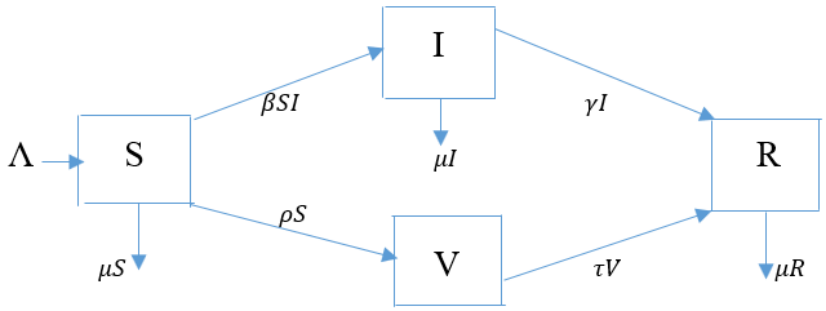

Fig. 1. Diagrammatic Representation of the SIVR Model with Vaccination

1.3 SIVR model

1.3.1 Assumptions

The following assumptions hold:

1. Closed population size, N.
2. Transmission and removal rates are regarded constant
3. A well-mixed population i.e. one where any susceptible individual can get infected.
4. Birth rate or entry population is equal to death rate

1.3.2 Epidemiology

Epidemiology is the study of the distribution and determinants of disease prevalence in humans, Ma S., et al, [8]

Murray J. [9] details in his book how the compartments, Susceptible, Infected, and Removed model's equations form a dynamical system. Since all three variables vary over time. Analyzing stability helps us to establish whether or not we have constant solutions, whether these solutions near the equilibrium points move towards or away from the equilibrium points, how the solutions behave as time, t, approaches infinity and if any of the solutions oscillate.

If the solutions tend toward the equilibrium value, such point will be considered *stable or an attractor*. In dynamical systems, an attractor refers to a set of states towards which a system tends to evolve, for a wide variety of starting conditions of the system. The system solutions get close enough to the attractor values and remain close even if slightly disturbed.

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 equilibrium point***.**

1.4 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.

1.5 Global stability

Global stability means that all solutions of the system approach the equilibrium point independent of the initial values. The case where both eigenvalues are real, negative, and distinct produces a phase portrait that shows all trajectories tending toward the equilibrium point as $t \rightarrow \infty$, the value of x(t) gets small, so it is a globally stable equilibrium point.

1.6 Herd immunity

In their brief history on vaccines [10], the World Health Organization defines vaccination as a simple, safe, and effective way of protecting an individual against harmful diseases before they come into contact with them. For centuries, people have looked for ways of protecting themselves against diseases and infections. Vaccination has stood the test of time as an effective method. In earlier times, this was done by exposing healthy people to the infection, as in the case of smallpox in the 15th century, in the hope that they would develop immunity against the pathogens. However, with time, proper vaccines for different diseases have been developed, and proper trials done to ascertain their suitability before administering them to humans. Herd immunity is defined as the immunity developed by the majority of a population against contagious diseases.

The term herd immunity was first used by Topley W., et al, [11]. 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 vaccination or via natural infection with a pathogen, Randolph H., et al, [12]. 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 immunocompromised are still protected against the disease.

The herd immunity threshold (HIT) depends on a single parameter known as the Basic Reproduction number, R_0. The R_0 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 R_0 of 2 is considered for example, it means on average, one infected person is capable of infecting two others on average during the infectious period.

In his book, Murray J. [9]. The basic reproduction number, R_0, is a necessary parameter when dealing with an epidemic under control with vaccination. 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, according to Anderson R., et al, [13] it was successful in eradicating smallpox in the world in 1979. 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, according to the European Union Centre for Disease Control, 125 cases caused by wild poliovirus were reported globally.

Recently, in their findings, Isaac M.W., et al, [3] found that re-infection with COVID-19 led into an increase in the cumulative deaths. Further, they found that the comparison on the impact of non-pharmaceutical interventions such has treatment and/or vaccination on curbing the spread of the disease, suggested that the wearing of face masks reduced COVID 19 prevalence compared with social/physical distancing. Their study revealed that early detection of asymptomatic cases through contact tracing, testing and isolating drastically reduced the disease surge

1.7 Study population

The sample data used in the study as obtained from the Ministry of Health, and the World statistics [14] Kenya, was distributed across the country. The population of Kenya as of the year 2022 was approximately 54, 027, 487. 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 where its believed people had a form of unexplained immunity attributed to their feeding habits [15].

Table 1. COVID-19 data for the first twenty days in kenya after the introduction of the vaccination

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

Source: Ministry of Health, Kenya

2 Post-Vaccination Findings

To arrive at these findings, the COVID-19 data from April 2021 to March 2022, readily available in the Kenyan Ministry of Health website, was fed into a computer software, python, using recorded computation of parameters necessary to determine the R_0 number. A remarkable observation under the circumstances is that the data available for use relates to the period during which the vaccination drive against COVID-19 was underway and the same data has varied over time.

Data from Kenya's ministry of health website for the period between April 2021 to March 2022 was fed into the software for computation R_0, and HIT at intervals of 30 days to give a summary shown in Table 2 below;

Table 2. R_0 and herd immunity threshold of COVID-19 during 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

From Table 2, an average value of R_0 was calculated and determined as 1.080045 and an average HIT value of 0.07, correct to two decimal places. Applying the formulae, $HIT = 1 - \frac{1}{R_0}$, with R_0 = 1.080045, HIT is obtained as 0.07, correct to two decimal places which agrees with the table average value.

The value of R_0 is obtained as; R_0 = 1.08, and an HIT value of 0.07, both correct to two decimal places. This implied that only a paltry 7% of the total Kenyan population needed to be vaccinated to bring the disease to a halt.

2.1 Stability analysis for the modified SIR model with vaccination

In Fig. 1, the diagrammatic representation of the modified SIR Model with Vaccination, an analysis of the stability of the model with induced vaccination is carried out. With vaccination, the system of equations [1- 4] were obtained;

With assumption 4) above, the population size N remains constant over time. Thus, $\frac{dS}{dt} + \frac{dI}{dt} + \frac{dV}{dt} + \frac{dR}{dt} = 0$. This implies that $S(t) + I(t) + V(t) + R(t) = N$. For purposes of the current analysis, it will be assumed that during the transmission of COVID-19 viruses, the no births equals the number of deaths since the epidemic dynamics occur on a relatively faster time scale than the rate of change of human population. Hence the population will be treated as constant.

From $S(t) + I(t) + V(t) + R(t) = N$, $R(t)$ can be determined from the other variables, hence it sufficient to consider the other three variables.

The rearranged equations now become

$$\frac{dS}{dt} = \Lambda - \beta SI - \rho S - \mu S \quad [5]$$

$$\frac{dI}{dt} = \beta SI - \mu I - \gamma I \quad [6]$$

$$\frac{dV}{dt} = \rho S - \tau V \quad [7]$$

Incorporating assumption 4) above, the system of equations become

$$\frac{dS}{dt} = \mu - \beta SI - \rho S - \mu S$$
$$\frac{dI}{dt} = \beta SI - \mu I - \gamma I$$
$$\frac{dV}{dt} = \rho S - \tau V$$

or

$$\frac{dS}{dt} = \mu(1 - S) - \beta SI - \rho S \quad [8]$$
$$\frac{dI}{dt} = \beta SI - \mu I - \gamma I \quad [9]$$
$$\frac{dV}{dt} = \rho S - \tau V \quad [10]$$

Making the set below positively invariant

$$\phi = \{(S(t), I(t), V(t)) \in \Re_+^3, S(t) + I(t) + V(t) \leq 1\}$$

2.2 Local stability of the system

Two equilibrium points exist for the above model:

The *Disease Free Equilibrium* point E_0 $(S = 1 - \rho, I = 0, V = \rho)$ and the *Endemic Equilibrium point*

$$E^*\left(S = \frac{\mu+\gamma}{\beta}, I = \frac{\mu(\beta(1-\rho)-\mu-\gamma)}{\beta(\mu+\gamma)}, V = \rho\right).$$

A determination of I from [8] shows the existence of an Endemic equilibrium point. Substituting this value in [9], we obtain

$$S^2 - S\left(1 - \rho + \frac{\mu}{\beta} + \frac{\gamma}{\beta}\right) + \frac{(1-\rho)\mu}{\beta} + \frac{(1-\rho)\gamma}{\beta} = 0 \quad [11]$$

The discriminant to [11] is

$$D = \left(1 - \rho + \frac{\mu}{\beta} + \frac{\gamma}{\beta}\right)^2 - 4\left(\frac{(1-\rho)\mu}{\beta} + \frac{(1-\rho)\gamma}{\beta}\right)$$

whose positive solution, $D \geq 0$

i.e. $\left(1 - \rho + \frac{\mu}{\beta} + \frac{\gamma}{\beta}\right)^2 \geq 0$

giving a reproduction number for the vaccinated as $R_v = R_0(1-\rho)$ and E^* will only exist if $R_v > 1$

Applying the Jacobian in determining the stability of the equilibrium points, we have,

$$J(S,I,V) = \begin{pmatrix} \frac{\partial}{\partial S}\left((1-\rho)\mu - \beta SI - \mu S\right) & \frac{\partial}{\partial I}\left((1-\rho)\mu - \beta SI - \mu S\right) & \frac{\partial}{\partial V}\left((1-\rho)\mu - \beta SI - \mu S\right) \\ \frac{\partial}{\partial S}(\beta SI - \mu I - \gamma I) & \frac{\partial}{\partial I}(\beta SI - \mu I - \gamma I) & \frac{\partial}{\partial V}(\beta SI - \mu I - \gamma I) \\ \frac{\partial}{\partial S}(\rho S - \tau V) & \frac{\partial}{\partial I}(\rho S - \tau V) & \frac{\partial}{\partial V}(\rho S - \tau V) \end{pmatrix} \quad [12]$$

Differentiating [12] respectively with respect to S, I and V, we obtain

$$J(S,I,V) = \begin{pmatrix} -\beta I - \mu & -\beta S & 0 \\ \beta I & \beta S - \mu - \gamma & 0 \\ \rho & 0 & -\tau \end{pmatrix} \quad [13]$$

2.2.1 Disease Free Equilibrium (DFE)

At DFE, $E_0\ (S = 1 - \rho, I = 0,\ V = \rho)$, [13] becomes
Thus,

$$J(S,I,V) = \begin{pmatrix} -\mu & -\beta(1-\rho) & 0 \\ 0 & \beta(1-\rho) - \mu - \gamma & 0 \\ \rho & 0 & -\tau \end{pmatrix} \quad [14]$$

The characteristic equation corresponding to [14] is

$$\begin{vmatrix} -\mu - \lambda & -\beta(1-\rho) & 0 \\ 0 & (\beta(1-\rho) - \mu - \gamma) - \lambda & 0 \\ \rho & 0 & -\tau - \lambda \end{vmatrix} = 0 \quad [15]$$

Simplifying and solving for λ, we obtain the eigenvalues $\lambda_1 = -\mu$, $\lambda_2 = (\beta(1-\rho) - \mu - \gamma)$, and $\lambda_3 = -\tau$.
From the results above, it's clear λ_1 and λ_3 are negative.

From $\lambda_2 = (\beta(1-\rho) - \mu - \gamma)$, there are two possibilities depending on the value of $(\beta(1-\rho) - \mu - \gamma)$

i) If $(\beta(1-\rho) - \mu - \gamma) > 0$, then
$R_v = R_0(1-\rho) > 1$
$R_v > 1$

The interpretation is that the DFE point is not asymptotically stable

ii) If $(\beta(1-\rho)-\mu-\gamma)<0$, then
$R_v = R_0(1-\rho)<1$
$R_v<1$

The interpretation is that the DFE point is asymptotically stable and the trajectories will approach the disease-free equilibrium point. The interpretation is that the disease will die out and there will be no epidemic.

2.2.2 Endemic equilibrium

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

At $E^*\left(S=\frac{\mu+\gamma}{\beta}, I=\frac{\mu(\beta(1-\rho)-\mu-\gamma)}{\beta(\mu+\gamma)}, V=\rho\right)$, making these substitutions into [13], we have,

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

Simplifying,

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

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

$$J(S,I,V)=\begin{pmatrix}\frac{\mu(\beta(1-\rho))}{(\mu+\gamma)} & -(\mu+\gamma) & 0\\ \left(\frac{\mu(\beta(1-\rho)-\mu-\gamma)}{(\mu+\gamma)}\right) & 0 & 0\\ \rho & 0 & -\tau\end{pmatrix} \quad [16]$$

From [16], the resulting characteristic equation is given by,

$$\begin{vmatrix}\frac{\mu(\beta(1-\rho))}{(\mu+\gamma)}-\lambda & -(\mu+\gamma) & 0\\ \left(\frac{\mu(\beta(1-\rho)-\mu-\gamma)}{(\mu+\gamma)}\right) & 0-\lambda & 0\\ \rho & 0 & -\tau-\lambda\end{vmatrix}=0 \quad [17]$$

Solving [17] for the eigenvalues λ, we obtain,

$$-(\tau+\lambda)\left\{\lambda^2-\left(\frac{\mu\beta(1-\rho)}{\mu+\gamma}\right)\lambda+\mu[(\beta(1-\rho)-\mu-\gamma)]\right\}=0 \quad [18]$$

Solving [18] for the eigenvalues, we obtain,

$$\lambda_1 = \tau$$

$$\lambda_2 = -\frac{\mu(\beta(1-\rho))}{(\mu+\gamma)} \pm \left\{ \frac{\mu^2(\beta^2(1-\rho)^2)}{(\mu+\gamma)^2} - 4\mu[\beta(1-\rho)-\mu-\gamma] \right\}^{\frac{1}{2}} \text{ which reduces to,}$$

$$\lambda_2 = -\mu R_v \pm \{\mu^2 R_v^2 - 4\mu(\mu+\gamma)(R_v-1)\}^{\frac{1}{2}} \quad [19]$$

Given that $\frac{(\beta(1-\rho))}{(\mu+\gamma)} = R_v(1-\rho) = R_v$ and given that $\mu(\beta(1-\rho)-\mu-\gamma)$ is positive, $\mu^2 R_v^2 - 4\mu(\mu+\gamma)(R_v-1)$ i.e. either greater or smaller than $\mu^2 R_v^2$.

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

The interpretation is that since λ_1 and both real parts of the eigenvalues from [19] are negative, it can be concluded that the Endemic Equilibrium point is locally stable, implying that both the susceptible and the infected persons survive in either case.

2.3 Global stability of the system

Here, we also perform both the DFE and the EE of the system using the Lyapunov function

A function F(x,y) is negative definite on a region ϕ from the origin if $\forall\,(x,y),\ F(x,y) < 0$, whereas it is positive definite on a region ϕ, with the origin if $\forall\,(x,y),\ F(x,y) > 0$. The same function is considered Lyapunov on an open region ϕ, if it is continuous, positive definite and has continuous first order partial derivatives on the region ϕ

2.3.1 Disease free equilibrium

Theorem 2.1

The Disease Free Equilibrium point of the system is globally asymptotically stable on ϕ

Proof.

In analyzing the global stability of the Disease Free point, apply the following Lyapunov function L: $\phi \rightarrow R$ and L (S, I, V) = S(t) + I(t) + V(t), whose derivative is given by,

$$\frac{dL}{dt}(S,I,V) = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dV}{dt} \quad [20]$$

$$\frac{dL}{dt}(S,I,V) = \mu(1-S) - \beta SI - \rho S + \beta SI - \mu I - \gamma I + \rho S - \tau V \quad [21]$$

Simplifying, we have,

$$\frac{dL}{dt}(S,I,V) = \mu - \mu S - \beta SI - \rho S + \beta SI - \mu I - \gamma I + \rho S - \tau V$$

$$\text{or } \frac{dL}{dt}(S,I,V) = \mu - \mu S - \mu I - \gamma I - \tau V \quad [22]$$

Collecting like and factoring common terms,

$$\frac{dL}{dt}(S,I,V) = \mu(1-S) - (\mu-\gamma)I - \tau V \quad [23]$$

$$\text{or} \quad \frac{dL}{dt}(S,I,V) = \mu(1-S) - \tau V - (\mu+\gamma)I \tag{24}$$

$$\text{or} \quad \frac{dL}{dt}(S,I,V) = (\mu+\gamma)\left\{\frac{(1-S)\mu R_0}{\beta} - \frac{\tau R_0 V}{\beta} - I\right\} \tag{25}$$

$$\text{or} \quad \frac{dL}{dt}(S,I,V) = \frac{(\mu+\gamma)}{\beta}\left\{\left[\left((1-S)\mu R_0\right) - \tau R_0 V\right] - I\right\} \tag{26}$$

$$\text{or} \quad \frac{dL}{dt}(S,I,V) = \frac{(\mu+\gamma)}{\beta}\left\{R_0[\mu(1-S) - \tau V] - \beta I\right\} \tag{27}$$

Implication,

$$\text{If } R_0 < 0, \text{ then } \frac{dL}{dt}(S,I,V) < 0$$

The interpretation that the Disease Free Equilibrium is globally asymptotically stable.

2.3.2 Endemic equilibrium

Theorem 2.2

The Endemic Equilibrium Point $E^*(S^*, I^*, V^*)$ of the system is globally asymptotically stable on ϕ

Proof.

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

$$L(S,I,V) = \vartheta\left[S - S^* \ln\left(\frac{S}{S^*}\right)\right] + \varphi\left[I - I^* ln\left(\frac{I}{I^*}\right)\right] + \omega\left[V - V^* ln\left(\frac{V}{V^*}\right)\right] \tag{28}$$

where ϑ, φ and ω are constants.

Differentiating the function with respect to time, t, we have

$$\frac{dL}{dt} = \frac{\partial L}{\partial S}\frac{dS}{dt} + \frac{\partial L}{\partial I}\frac{dI}{dt} + \frac{\partial L}{\partial V}\frac{dV}{dt} \tag{29}$$

$$\frac{dL}{dt} = \vartheta\left[\left(1 - \frac{S^*}{S}\right)\left((1-\rho)\mu - \beta SI - \mu S\right)\right] + \varphi\left[\left(1 - \frac{I^*}{I}\right)(\beta SI - \mu I - \gamma I)\right] + \omega\left[\left(1 - \frac{V^*}{V}\right)(\mu S - \tau V)\right] \tag{30}$$

$$\frac{dL}{dt} = \vartheta\left[\left(\frac{S-S^*}{S}\right) * S\left(\frac{(1-\rho)\mu}{S} - \beta I - \mu\right)\right] + \varphi\left[\left(\frac{I-I^*}{I}\right) * I(\beta S - \mu - \gamma)\right] + \omega\left[\left(\frac{V-V^*}{V}\right) * V\left(\frac{\mu S}{V} - \tau\right)\right] \tag{31}$$

$$\frac{dL}{dt} = \vartheta\left[(S-S^*)\left(\frac{(1-\rho)\mu}{S} - \beta I - \mu\right)\right] + \varphi[(I-I^*)(\beta S - \mu - \gamma)] + \omega\left[(V-V^*)\left(\frac{\mu S}{V} - \tau\right)\right] \tag{32}$$

At equilibrium point

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

Making these substitutions, we have,

$$\frac{dL}{dt} = -\mu\vartheta(1-\rho)\left(\frac{(S-S^*)^2}{SS^*}\right) + \beta(\vartheta - \varphi)(I-I^*)(S-S^*) - \tau\omega[(V-V^*)]^2 \tag{33}$$

Additionally,

If $\vartheta = \varphi + \omega = 1$,

then $\frac{dL}{dt} = \mu\vartheta\left(\frac{(S-S^*)^2}{SS^*}\right) - \tau\omega[(V-V^*)]^2 \leq 0$ and if $S = S^*$, $V = V^*$, $\frac{dL}{dt} = 0$

The interpretation is that by LaSalle's Invariance Principle, the Endemic Equilibrium Point is globally asymptotically stable, since in other words, both eigenvalues are real, negative, and distinct producing 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 Post Vaccination Projections

For post vaccination projection dynamics, the parameters as illustrated on the flow diagram are used. The Endemic Equilibrium Points, $E*$ corresponding to the first four months after the inception of the vaccination drive in Kenya are given in Table 3.

The data used to simulate the results shown in the graphs depicted in Fig. 1 through 7 was obtained from Kenya's Ministry of Health (MoH) website. The same was therefore readily available as was captured and tabulated by the Ministry of Health officials on a daily basis.

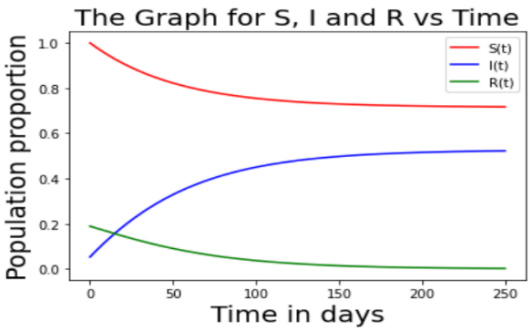

Fig. 2. S, I, and R relationship after 250 days

Table 3. Endemic Equilibrium Points, E*(S,I,V) for the first five months after commencement of COVID-19 Vaccination drive in Kenya

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)

As is depicted in the following graphs, as the vaccination numbers increase, those of susceptibles and infected reduce.

This is depicted in Fig. 3, 4, 5, 6, and 7.
The reproduction number, Rv, was obtained from: $Rv = R_0(1-p)$.

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

Fig. 3 shows a somewhat sharp increase in the number of the vaccinated, whereas those for susceptibles and recovered are both decreasing respectively.

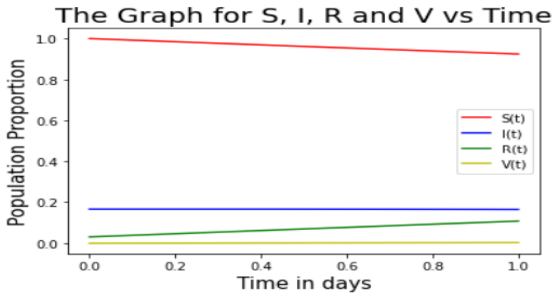

Fig. 3. S, I, R and V relationship at DFE

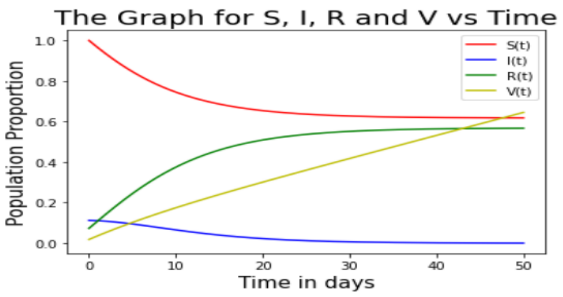

Fig. 4. S I R and V relationship after 50 days

Figs. 4 and 5 below show a reduction in the number of susceptibles whereas the number of recovered is increasing. These numbers are seen stabilizing at some point. On the other hand, the infected numbers remain low.

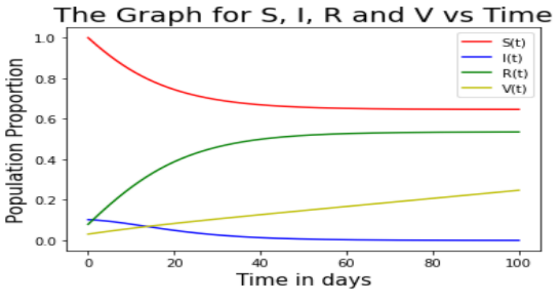

Fig. 5. S I R and V relationship after 100 days

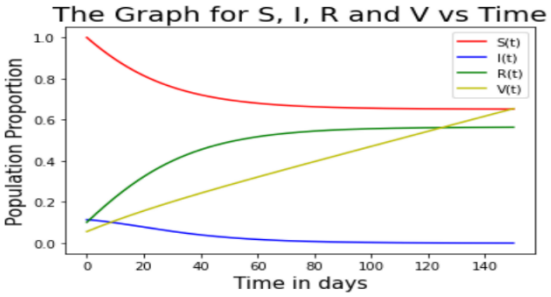

Fig. 6. S I R and V relationship after 150 days

Figs. 6 and 7 show that the numbers of the vaccinated increasing while those for recovered remain low and stable as the number of days increase.

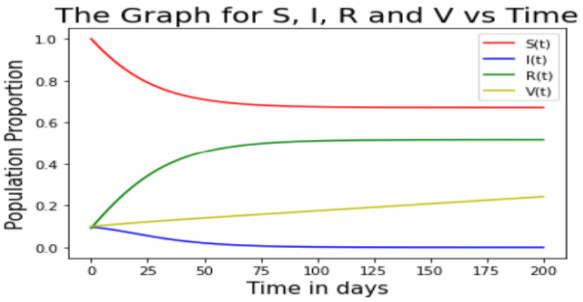

Fig. 7. S I R and V relationship after 200 days.

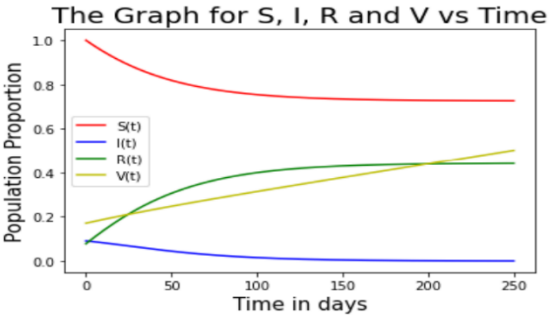

Fig. 8. S I R and V relationship after 250 days.

5 Conclusions and Recommendations

5.1 Conclusions

With an estimated R_o value approaching 1.08, translating into a Herd Immunity Threshold of 0.07, implied that the population that needed to be vaccinated to keep the pandemic under control was 7%. This was a huge milestone achieved in curbing the spread of the disease. This is believed to be attributed to the herd immunity obtained through vaccination. or naturally. From the post vaccination stability analysis carried out, it can be concluded that vaccination significantly contributed to the acquisition of herd immunity.

Upon vaccination, results show that the susceptible population gradually decreased while the infected population declined steadily as shown by the infection rate, β. This is believed to be a result of enhanced herd immunity due to the robust vaccination roll out.

5.2 Recommendations

Its highly recommended that stability analyses be carried out amongst people of different age groups with a view to determining which group is most vulnerable. It's not lost on the study that a large portion of Kenyans never got vaccinated due to their cultural beliefs, yet they continue to exhibit characteristics of herd immunity, pointing out to the fact that a large portion acquired natural herd immunity upon infection. It might be necessary to determine, if possible, which between natural immunity or one acquired due to vaccination is more effective in curbing the spread of COVID-19 and other similar pandemics and how the same can be enhanced.

Disclaimer (Artificial Intelligence)

Author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc) and text-to-image generators have been used during writing or editing of this manuscript.

Competing Interests

Author has declared that no competing interests exist.

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