MODELLING COVID-19 DYNAMICS (SPREAD AND CONTROL) AND THE EFFECTS OF A PREVENTIVE VACCINE

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DECLARATION

Declaration by the Student

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This thesis is dedicated to my son Xavier Ethan.

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ABSTRACT

Corona virus 2019 (COVID-19) have been pandemic both in Africa and the whole world. This work formulated and analyzed mathematical model of COVID-19 that monitors the temporal dynamics of the disease in the presence of preventive vaccine. The most effective ways of controlling the transmission of infectious disease is through vaccination and treatment. Due to transmission characteristics of COVID-19 , the population was divided into six classes. That is; susceptible(S), vaccinated (V), infective (I), hospitalized (H), home based care (H_B) and recovery(R). In this thesis, non-linear system of differential equations governing the model was formulated to compute and were solved using quantitative analysis. Feasibility region and positivity of model variable was worked out in which the model is bounded so as to obtain the feasibility solution of the set and positivity of variables. The disease free equilibrium, local and global stability of the disease free equilibrium are discussed. The endemic equilibrium , local and global endemic equilibrium are determined. The model monitor reproduction number R_O using next generation matrix method which describe the dynamics of the COVID-19.The disease free equilibrium is local asymptotically stable when basic reproduction number $R_o < 1$ and unstable when basic reproduction number $R_o > 1$. The numeric results obtained are determined graphically by use of MAPLE simulation method. The solution has been computed using numerical classical fourth order Runge Kutta integration method to gauge its effectiveness . The results indicated that; high vaccination coverage of φ =0.9 leads to high number of individuals recovering and low vaccination coverage of $\varphi = 0.1$ leads to high reproduction number hence the disease may not be eradicated .

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ABBREVIATION

DFE	Disease Free Equilibrium
EE	Endemic Equilibrium
ODE	Ordinary Differential Equation
SVIHH_BR	Susceptible, Vaccinated, Infective, Hospitalized, Home base care and recovered
WHO	World Health Organization
COVID-19	Corona virus 2019
SEIRS	Susceptible, Exposed, Infective, Recovered and Susceptible
EUL	Emergency Use Listing
SARS-CoV-2	Severe acute respiratory syndrome coronavirus2
SEIAR	Susceptible, Vaccinated, Exposed, Asymptomatically infected, symptomatically infected and Removes/immune
SVEITR	Susceptible, Vaccination, Exposed, Infective, Treated, Recovered
RMSE	Root-mean square error
SEIQRDV	Susceptible, Exposed, Infectious, Quarantined, Recovered, Deaths and Vaccinated

NOMENCLATURE

Λ Recruitment rate

N Total population

S Susceptible

V Vaccinated

I Infective

H Hospitalized

H_B Home base care

R Recovered

β Rate of recruitment to infective class from susceptible

q Rate of recruitment to vaccination from susceptible

K Rate of recruitment to hospital treatment from infective class

$\boldsymbol{\omega}$ Rate of recruitment to home based care from infective class

α Death rate as a result of COVID-19

$\boldsymbol{\mu}$ Death rate as a result of natural calamities

$\boldsymbol{\varepsilon}$ Recovery rate of hospitalized COVID-19 patients

$\boldsymbol{\chi}$ Recovery rate of home-based care individual

$\boldsymbol{\pi}$ Rate at which recovery individual can be susceptible

$\boldsymbol{\delta}$ Rate at which infective individual can recover

$\boldsymbol{\rho}$ Rate at which vaccinated individual can be infective.

φ vaccination coverage

R_V Reproduction vaccination number

R_O Basic reproduction number

$R_O(V)$ Basic reproduction number for vaccinated

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CHAPTER ONE

INTRODUCTION

1.1 Introduction

The increasing study of realistic and practically useful mathematical models in population, whether we are dealing with a human population with or without its age distribution, population of an endangered species, bacterial or viral growth and so on, is a reflection of their use in helping to understand the dynamic processes involved and in making practical predictions. Kermack et-al (1927), Varotsos et-al (2020) and Maini et-al (2022).

1.1.1 Background Information and Causes of COVID -19

Coronavirus disease (COVID-19) is an infectious disease caused by the SARS-CoV-2 virus. The best way to prevent and slow down transmission is to be well informed about the disease and how the virus spreads. Most people infected with the virus will experience mild to moderate respiratory illness and recover without requiring special treatment. However, some will become seriously ill and require medical attention. Older people and those with underlying medical conditions like cardiovascular disease, diabetes, chronic respiratory disease, or cancer are more likely to develop serious illness. Anyone can get sick with COVID-19 and become seriously ill or die at any age. The virus can spread from an infected person's mouth or nose in small liquid particles when they cough, sneeze, speak, sing or breathe. These particles range from larger respiratory droplets to smaller aerosols. It is important to practice respiratory etiquette, for example by coughing into a flexed elbow, and to stay at home and self-isolate until one recover .If one feel unwell and wear a face mask to prevent droplet from falling on surface as in Jaguga et-al (2020).

To prevent infection and to slow down the transmission of COVID-19, the following should be done: one should get vaccinated, maintaining one meter apart from others, wearing a properly fitted mask, wash the hands regularly with soap and water or clean with alcohol-based hand rub, Cover the mouth and nose when coughing or sneezing and if one feel unwell, stay home and self-isolate until you recover. Most infected people will develop mild to moderate illness and recover without hospitalization.

Vaccination is a simple, safe, and effective way of protecting one against harmful diseases, before coming into contact with them. It uses the body's natural defenses to build resistance to specific infections and makes the immune system stronger. Vaccines train the immune system to create antibodies, just as it does when it's exposed to a disease. However, because vaccines contain only killed or weakened forms of germs like viruses or bacteria, they do not cause the disease or put one at the risk of its complications

The first mass vaccination programmed started in early December 2020 and the number of vaccination doses administered was updated on a daily basis on the website of world health organization the date was provided by department of the Ministry of Health in each country. At least 13 different vaccines (across 4 platforms) have been administered. The Pfizer/BioNtech Comirnaty vaccine was listed for WHO Emergency Use Listing (EUL) on 31 December 2020. The SII/Covishield and AstraZeneca/AZD1222 vaccines (developed by AstraZeneca/Oxford and manufactured by Serum Institute of India and SK Bio respectively) were given EUL on 16 February 2021. The Janssen/Ad26.COV 2.S developed by Johnson & Johnson, was listed for EUL on 12 March 2021. The Moderna COVID-19 vaccine (mRNA 1273) was listed for EUL on 30 April 2021 and the Sinopharm COVID-19 vaccine was listed for EUL on 7 May 2021. Beijing Bio-Institute of Biological Products Co

Ltd, subsidiary of China National Biotec Group (CNBG), produces the Sinopharm vaccine. The Sinovac-CoronaVac was listed for EUL on 1 June 2021.

In Kenya, there were over 250K confirmed cases of COVID-19 and more than 5150 deaths while in the whole world its 219M total cases and 4.55m deaths case reported to WHO. As of 27 September 2021, a total of 3712030 vaccine doses had been administered. Since the vaccination was introduced in Kenya daily cases reduced. Up to date over 1M have received the first dose and over 450k have been vaccinated fully.

This study develops and analyzed the COVID-19 model for monitoring the disease in presence of consistent preventive vaccination This study monitored the recovery rate of those getting treatments after being vaccinated. Those with COVID-19 can take medication in home-based care or in the hospital.

1.2 Statement of the Problem

A six compartment model namely; susceptible (S), vaccinated (V), infective (I), hospitalized (H), home base care (H_B) and recovered(R) ($SVIHH_BR$) was develop to determine the spread and control of COVID-19 and the effects of a preventive vaccine. Many studies and literatures show that, scholars have study much on safe ways of treatment and controlling the spread of COVID-19 disease, determination of age to be vaccinated and more so time interval between the first dose and the second dose. Recent studies (Gonzalez, 2021) focused on varying population size to model of COVID-19 with interested of the impact of the temporal regime of vaccination that is varying the time between the first and the second dose without considering effect of vaccination since it incorporates births and deaths due to fatal diseases. Hence introducing a effect vaccination on the spread and control of COVID-19 will help to eradicate these diseases.

1.3 Research Objectives

1.3.1 General objective

The main objective of this study is to develop and analyze COVID-19 model that monitors the temporal dynamics of the disease in the presence of preventive vaccine.

1.3.2 Specific objectives

Specific objectives was to;

i. Formulate the SVIHH_BR model incorporating the impact of hospitalization and home-based care in the treatment of COVID-19.
ii. Determine the model disease-free equilibrium (DFE) points and the existence of DFE local and global stabilities using basic reproduction number R_O criteria.
iii. Determine the model endemic equilibrium (EE) and the existence of EE local and global stability using basic reproduction number R_O criteria.
iv. Perform quantitative analysis of the model and determine the effect of vaccine in the eradication of COVID-19 disease.

1.4 Justification of the study

The study focusses on COVID-19 in a fraction population size where the susceptible individual recruited and later vaccinated. Many scholars i.e Yavuz et-al (2021) ,Vespignani et-al(2020), Samui et-al(2020) and Ndairou et-al(2020) have formulated mathematical models to describe the optimal control and preventive measures of Corona virus to eradicate this disease, more births than death would be a better assumption. Recent studies (Gonzalez, 2021) focused on varying population size to model of COVID-19 with interested of the impact of the temporal regime of vaccination that is varying the time between the first and the second dose without

considering constant vaccination since it incorporates births and deaths due to fatal diseases. To push the models to further realism, we consider addition of varying population size and infective individual which have been vaccinated. This study formulates a mathematical model that can be used to analyze COVID-19 diseases effectively which contributes to the field of mathematical epidemiology. It develops a framework that would predict the disease dies out and needs to prevent the spread of these diseases. This study will help policy makes in their decision for the use of vaccination approach thus helping the government on annual vaccination routines. It also acts as a basis for further research by students and researchers on modeling other diseases and on building a model. Its help to discover factors which govern the system and how the aspects of the system are related.

1.5 Assumptions

In this thesis, we have study SVIHH_BR model with fraction population size. The model has a susceptible group denoted by S, vaccinated group donated by V, infective group donated by I, hospitalized group donated by H, home base care group donated by $H_B$and recovered group denoted by R. The SVIHH_BR model of the COVID-19 diseases considers the following assumptions:

i. The rate of recruiting new members to a system is Λ
ii. The death rate μ due to nature calamities in each class hence population size N remains constant.
iii. There were restrictions on human behavior such as quarantine, wearing masks, keeping distance and washing of hands with hand sanitizer.
iv. A susceptible individual will move into the vaccinated class at average contact rate qS and to infective class at average contact rate βSI.
v. Vaccinated individual can move to infective class at contact rate of ρVI.

vi. An infective individual will get treatment at home base care at a rate of kI or in hospital at contact rate of ωI and other will recover at the rate $\boldsymbol{\delta I}$

vii. Individual in treatment at home base care progresses to recovered group due to treatment at a rate χH_B and those in hospitalization progress to recovery group at a rate εH

viii. We assume α to be death rate due to COVID-19 disease infection.

ix. All the variables are positive at all the time.

x. The model assumes that the efficacy of the vaccine is 100%.

CHAPTER TWO

LITERATURE REVIEW

2.1 Introduction

Mathematical analysis and modelling is an important part of infectious disease epidemiology. The mathematical description of disease epidemics immediately leads to several useful results, including the expected size of an epidemic and the critical level that is needed for an intervention to achieve effective disease control. This chapter gives relevant scholars models which have been improved from SIR model. The model consisted of Susceptible, Infective and Recovered which was used to determine the spread of an infectious disease over a given period of time. (David et-al 2014)

2.2 Literature relevant to this study

M.L Diagne et-al (2021) formulated a deterministic model of the transmission dynamics of COVID-19 with an imperfect vaccine. Their model was theoretically analyzed; its effective and basic reproduction numbers were derived. The disease-free equilibrium is globally asymptotically stable, and the disease could be eradicated when the reproduction number is below unity (R_O<1) .They introduced into the model system one time-dependent control variables $u_1(t)$ representing vaccination and $u_2(t)$ representing treatment of hospitalized individuals and applied the Pontryagin maximum principle to determine the optimal control strategy for mitigating the spread of the disease. The model fitted well with the observed daily data from Senegal early COVID-19 epidemic. Numerical simulations of the optimal control of the full model were carried out using a set of model parameter values which indicated that COVID-19 can be controlled in the community with the

implementation of vaccination and treatment. While their results suggest that vaccination and treatment were very effective in mitigating the spread of COVID-19, more efforts are needed to eradicate the disease.

They also performed a sensitivity analysis using the partial rank correlation coefficient in conjunction with the Latin hypercube sampling technique, to identify the model parameters that significantly influence the initial disease transmission. Early identification of parameters with greater influence on disease transmission is important to inform policy decision on which parameters to focus either for data collection or to mitigate the spread of the disease. The critical vaccination threshold was derived, and it was noted that if the vaccine efficacy is low and the disease reproduction number is high, the disease may not be eradicated even if a large proportion of the population is vaccinated. That is, additional efforts will be needed to reduce $R_0(V)$ below unity even if vaccine coverage was high.

Gonzalez et-al (2021) Studied the impact of different vaccination regime on main health outcome such as death. They developed mathematical model of COVID-19 that divides the total population into different classes depending on the COVID-19 progression and vaccination status. Thus, they considered classes or subpopulations as: susceptible, latently infected (not yet Infectious), pre-symptomatic (and infectious), infected (able to infect others), asymptomatic (able to infect others), hospitalized, recovered (not infectious), vaccinated with one dose, and, lastly, vaccinated with two doses. They constructed a mathematical model that includes ordinary differential equations. The mathematical model constructed considers transitions of individuals through the aforementioned classes. They assumed that recovered individuals have long immunity against reinfection during the period of study that is shorter than a year. They also assume that only susceptible individuals

and those vaccinated with one dose are the only ones that can be vaccinated. Parameter values used for this study were taken from scientific literature. Their model assumed constant vaccination per day. They carry out numerical simulation and analyze under different scenarios and plots were processed using python programming language, in particular the scipy.odeint () function, which is python wrapper for the ODEPACK solver. Their study was mainly interested with impact of the temporal regime of vaccination that is varying the time between the first and the second dose. However, from their research, vaccinated population was not considered.

Jaharuddin et-al (2020) used an autonomous nonlinear differential equation system for measles dynamics, which incorporates constant vaccination, therapy, and treatment rates, is considered first of all. They developed SVEITR model consists of six compartments where S-susceptible, V-vaccinated, E-exposed, I-infected, T-treated, and R-recovered. From this compartment model they formulated equation from mathematical model. From the equation developed, they carried out disease-free and endemic equilibrium points using Routh–Hurwitz criteria and bifurcation theory to find out the stability of the model. They find out that the stability of the equilibrium points depends on the basic reproduction numberR_O. If, the disease-free equilibrium point will be stable, otherwise unstable. The endemic equilibrium point is in an asymptotically stable condition ifR_O <1, otherwise unstable. Their sensitivity analysis of the model reveals that the effective contact rate was the most influencing parameter to the model. They extended their research with constant controls including time-dependent vaccination, therapy, and treatment rates, resulting in the model with optimal controls. The Pontryagin maximum principle was employed to derive the necessary conditions for existence of an optimal controlled pair that minimizes the

number of exposed and infected individuals jointly with the control effort. For more accuracy, they compared and evaluated their results using the forward-backward sweep method and the fourth-order Runge–Kutta algorithm. It is demonstrated that moderate and high levels of coverage can effectively reduce the measles cases. However, the drug resistance individual was no considered as the case of re-infection.

Intissar et-al (2021) research on modeling the effect of population –wide vaccination on the evolution of COVID-19.in their study, the total population was partitioned into 4 sub-populations: S, susceptible (non-infected without immunity); I, infected (active cases); R, removed (closed cases, recovered or dead); V, effectively vaccinated (non-infected individuals that were effectively vaccinated, immune). In their results, the daily vaccination rates were used for their epidemic predictions representing the daily vaccination rate of fully vaccinated individuals (i.e., non-infected individuals that received the two doses of the vaccine). From their compartment, equation was formulated on the flow of COVID-19 disease where Variable and parameters were assumed constant. They further worked out the basic reproduction number and found it to be greater than one (R_O>1) which indicates that every person would cause a proper epidermic outbreak. They used root-mean square error (RMSE) method and trapezoidal integration rule to stimulate and discussed their solution. They made a conclusion that vaccination should start as soon as possible and the vaccination campaign should stop only when there is no susceptible sub population left. This study, did not take account of vital dynamics (demographic births and nature deaths) since their population remains constant.

Zhang et-al (2010) in their research article of the analysis of Epidemic Network Model with infective force in latent and infected period, formulated SEIAR model and classify the population as susceptible (S), exposed (E), asymptomatically infected

(A), symptomatically infected (I), and removed/immune (R).Their model where in form of S-E-I-A-R from which they develop differential equation .Their mathematical formulation of their epidemic problem were completed using positive parameters. From their model, they worked out on global stability and basic reproduction number R_O using Next Generation Matrix. Their discussion concluded that, the infection disease free equilibrium is locally asymptotically stable when basic reproduction no is less than $(R_O < 1)$ since their where no vaccination and the infection disease free equilibrium is unstable when basic reproduction no is greater than (R_O>1). Their derivation depends on an explicit formula for the Basic reproduction number of networks of disease transmission model without considering those who have been vaccinated before recruitment.

Ghostine et-al (2021), in their SEIQRDV model with a vaccination compartment were proposed to simulate the novel coronavirus disease (COVID-19) spread in Saudi Arabia. Their model considers seven stages of infection: susceptible (S), exposed (E), infectious (I), quarantined (Q), recovered (R), deaths (D), and vaccinated (V). From these compartments they formulated equation from their SEIR model. They carried out mathematical analysis to illustrate the non-negativity, boundedness, epidemic equilibrium, existence, and uniqueness of the endemic equilibrium, and the basic reproduction number of the model. From their model, an epidemic was expected to increase exponentially if $R_O > 1$ and end if $R_O < 1$ also the disease –free equilibrium is locally and globally asymptotically stable if $R_O < 1$ and unstable if $R_O > 1$. The results were computed and solved using a fourth –order Runge kutta method. They then presented the numerical results in form of graphs. They however in their model did not take into account the impact of recruitment population being vaccinated before instead they work on effect on vaccination on the spread of disease.

In their study Yin et-al (2020), were concerned with the stability of an age-structured susceptible–exposed–infective–recovered–susceptible (SEIRS) model with time delay. They investigated the existence and uniqueness of the continuous traveling wave solution under some hypotheses. Moreover, the age-structured SEIRS system was reduced to the nonlinear autonomous system of delay ODE using some insignificant simplifications. In their studied, they investigated on dimensionless indexes for the existence of one disease-free equilibrium point and one endemic equilibrium point of the model. Furthermore, they established the local stability for the disease-free equilibrium point(DFE) and the endemic equilibrium (EE) point of the infection-induced disease model using Hurwitz's criterion and Descartes' rule. They found that, disease free equilibrium point (DFE) of the system is locally asymptotically stable and has unique endemic equilibrium point and they found to be locally asymptotically stable. Finally, some numerical simulations were carried out to illustrate their theoretical results and display their results using graphical solution using MABLE. In general, their studied provides the practical understanding of the different dynamic behavior of an age structured SEIRS model, without considering the vaccination strategy.

Idris Ahmed et-al. (2021) attempted described the outbreak of coronavirus disease 2019(COVID-19) with help of a mathematical model using both the ordinary differential equation (ODE) and fractional differential equation. The spread of the disease has been on the increase across the global for some time with no end in sight. The research used the daily data of COVID-19 cases posted in Nigeria WHO website for numerical simulation which has been fitted to the model. The model bought into consideration both asymptomatic and symptomatic infected individuals with the fact

that an exposed individual is either sent to quarantine first or move to one of the infected classes with possibility that susceptible individual can also move to quarantine class directly. The model had two equilibrium points; the disease-free equilibrium point (DFE) and the endemic equilibrium point (EE). Stability analysis of their endemic equilibrium points show was locally asymptotically stable whenever Ro<1. The existence and uniqueness of solution established via the technique of fixed-point theorem. Furthermore, they solved the fractional model numerically using Atangana-Toufik numerical scheme and presented in different forms of graphical results to minimize the infection. However, their model didn't take in consideration patients taking their treatment in hospital or under home base care facilities as well as the vaccinated individuals.

Concluding this chapter we can observe that the models of Yin et-al (2020), Ghostine et-al (2021), Zhang et-al (2010), Jaharuddin et-al (2020) and Gonzalez et-al (2021) were very similar to the model we present in this work. This research extend the work of Zhang et-al (2010) from a SEIAR model to a SVIHH_BR model and study the temporal dynamics. The SVIHH_BR model formulated in this research advances from previous studies by incorporating home base care individual for treatment from infectives individual and those under treatments in hospitals. Assumptions are always made to improve the model analysis and make it more realistic. The solution will be computed using fourth order Runge Kutta integration method to gauge its effectiveness and impact of vaccination in Covid 19. The main question to be addressed is whether vaccination coverage can influence disease spreading and control and inform health authorities on prevention and eradication strategies.

In the next chapter, the methodologies used will be discussed and analysis the equation governing model.

CHAPTER THREE

METHODOLOGY

3.1 Introduction

The SEIR model formulated was improved to include those individuals who are receiving treatment in hospital and those in home-based care as in Zhou et-al(2011). The model developed gave reasonable and normal results. Assumption was also made to improve the model formulation analysis and to predict the disease dies out.

3.2 Model Description and Formulation

The model was divided into six sub-classes according to their disease status and the movement between them from the human population (N); that is, susceptible(S), vaccinated (V), infective (I), hospitalized (H), home base care (H_B) and recovered(R) compartment.. The S-V-I-H-H_B-R model developed is described in figure 3.1 below

.

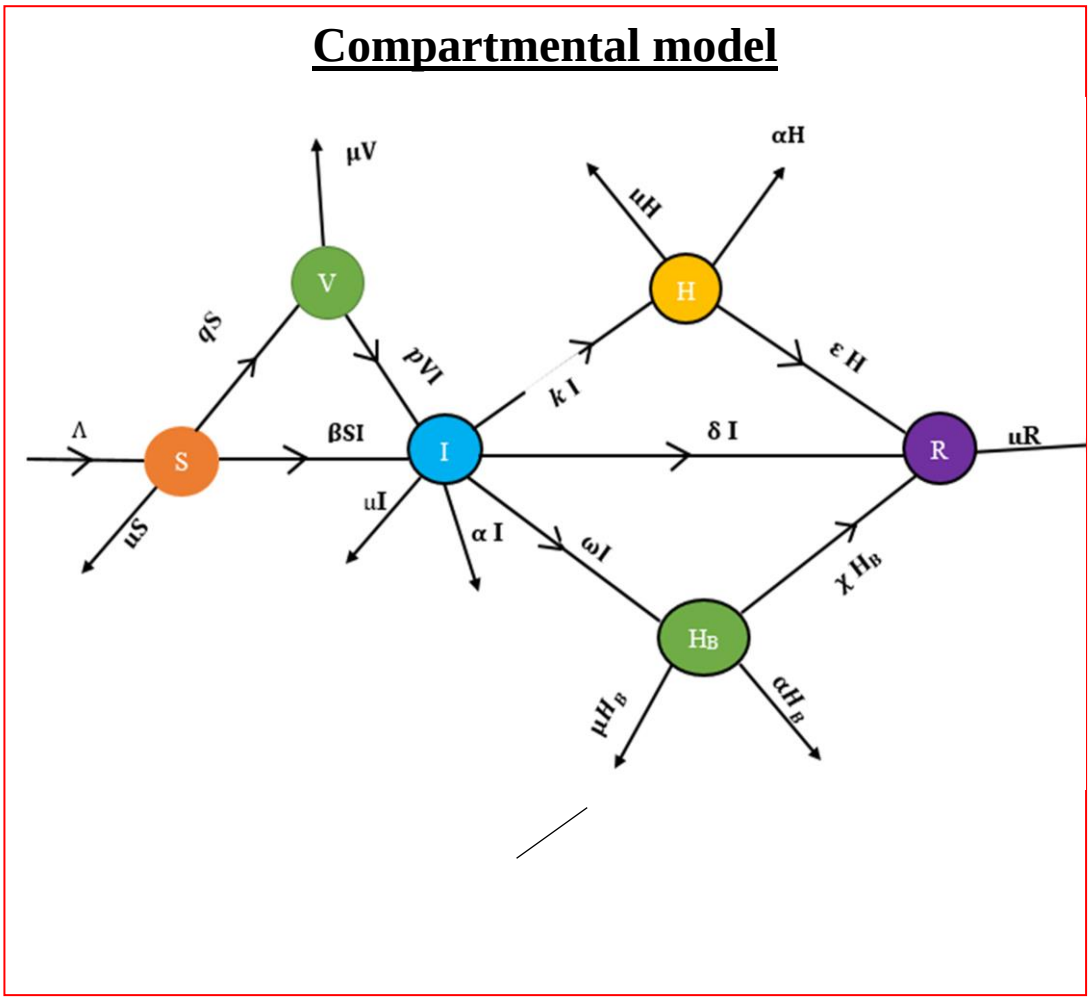

Figure 3.1 An S-V-I-H-H_B-R compartment COVID-19 model

Arrows indicate the movement from one compartment to another and others exits from the population. The model assumes that a fraction of the population has been recruited into susceptible individuals which are those likely to be affected by COVID-19. The vaccinated individuals are those susceptible who get COVID-19 vaccine. The infective individuals are those who have been infected with COVID-19. The home-based care individuals had contracted the COVID-19 disease and were taking medication at home as prescribed by qualified medical personnel. The hospitalized individuals were those infected by the COVID-19 disease and admitted in approved medical facility. Modification of assumptions lead to a model where infected

individuals recover. The recovered individuals were those who got well after a COVID-19 infection and tested negative.

Susceptible individual recruited at the rate of **Λ.** The susceptible individual were vaccinated at the rate of **qS** and those vaccinated become infective at the rate of **pVI**. The infected individual were recruited at the rate of $\boldsymbol{\beta SI}$ from susceptible class. The rate at which the infected individuals were either treatment over COVID-19 at home-based care and being attend by qualified doctors at the rate of $\boldsymbol{\omega}$ or received treatment in hospital at the rate $\boldsymbol{\kappa}$. The rate at which the hospitalizes recovered was $\boldsymbol{\varepsilon}$ while the rate at which those on home –based care recovered was $\boldsymbol{\chi}$**.**The infected individual recovered without getting treatment at contact rate of $\boldsymbol{\delta}$ .The model took into account deaths caused by nature μ and those caused by corona virus at the rate of α under the assumption that all the parameters are constant. The total population **N= S+V+I+H+**H_B**+R** and all recruitment rates were between zero to one.

The following values in table 3.1 were the Model parameters corresponded to COVID-19 case in Kenya for the average month of January to march 2021.They were obtained from the Ministry of health website for daily reporting of COVID-19 outbreak in Kenya. The data obtained help in calculating some parameters values by doing the average of the 3 months.

Table 3. 1 Summary of parameter descriptions

Parameter interpretation	Values per day	Source
Λ Recruitment rate	10	Estimated
β Rate of recruitment to infective class from susceptible	0.5787	Estimated
q Rate of recruitment to vaccination from susceptible	0.4213	Calculated
κ Rate of recruitment to vaccination	0.5493	Calculated
$\boldsymbol{\omega}$ Rate of recruitment to home based care	0.9506	Calculated
α Death rate as a result of COVID-19	0.00961	(parra, 2021)
$\boldsymbol{\mu}$ Death rate as a result of natural calamities	0.00411	(Deressa, 2020)
$\boldsymbol{\varepsilon}$ Recovery rate of hospitalized COVID-19 patients	0.15	Calculated
$\boldsymbol{\chi}$ Recovery rate of home-based care individual	0.1612	Calculated
$\boldsymbol{\delta}$ Rate at which infective individual can recover	0.189	Calculated
$\boldsymbol{\rho}$ Rate at which vaccinated individual can be infective	0.2	Estimated

The following table 3.2 were the initial condition used of members of individuals.

Table 3. 2 Initial conditions

Compartment	Number of individuals
S(0)	1400
V(0)	300
I(0)	150
$H_B(0)$	200
H(0)	250
R(0)	100

3.3 Model Equations

According to the inflows and outflows in Figure 3.1 of the COVID-19 and disease pathways with control measures, we can convert them into 1st order ordinary non-linear differential equation as follows;

$$\left.\begin{aligned}
&\frac{dS}{dt} = \Lambda - (\beta SI + qS + \mu S) \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots 3.1(a)\\
&\frac{dV}{dt} = qS - (pI + \mu)\mathrm{V} \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots 3.1(b)\\
&\frac{dI}{dt} = \beta SI + \mathrm{pVI} - (k + \omega + \delta + \mu + \alpha)I \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots 3.1(c)\\
&\frac{dH_B}{dt} = \omega\mathrm{I} - (\chi + \mu + \alpha)H_B \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots 3.1(d)\\
&\frac{dH}{dt} = \mathrm{kI} - (\varepsilon + \mu + \alpha)\mathrm{H} \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots 3.1(e)\\
&\frac{dR}{dt} = \varepsilon H + \chi H_B + \delta\mathrm{I} - \mu\mathrm{R} \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots 3.1(f)
\end{aligned}\right\} 3.3.1$$

Where $\frac{dS}{dt}, \frac{dV}{dt}, \frac{dI}{dt}, \frac{dH_B}{dt}, \frac{dH}{dt}$ and $\frac{dR}{dt}$ are SVIHH_BR model equations.

3.4 The Feasibility Region

This is the region whereby the solution to model equalized is non-negative and uniformly bounded.

Let (S, V, I, H, H_B, R) ϵ $\mathbb{R}^6$ be any solution with initial condition

S(0) $=S_0$, V(0)=V_0 $I(0)=0$ H(0) $=0$ $H_B(0)=0$ R(0)=0 and N=S+V+I+H+H_B+R ……..(3.4.1)

We can differentiate N both with respect to (w.r.t) time sides and to obtain the expression;

$\frac{dN}{dt}=\frac{dS}{dt}+\frac{dV}{dt}+\frac{dI}{dt}+\frac{dH}{dt}+\frac{dH_B}{dt}+\frac{dR}{dt}$ ……..... ……(3.4.2)

Substituting equation 3.1(a) ,3.1(b) …..to 3.1(f) into eq (3.4.2) to obtain

$$\frac{dN}{dt}=\Lambda+-(\mathrm{V}+\mathrm{S}+\mathrm{I}+\mathrm{H}+H_B+\mathrm{R})\mu+(H+I+H_B)\alpha$$

$$=\Lambda-\mu N+(H+I+H_B)\alpha \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots (3.4.3)$$

Ignore death caused by COVID-19 disease $\alpha=0$

$$\frac{dN}{dt}=\Lambda-\mu N$$

$intergrating$ Equation 3.4.3 w.r.t N

$$\frac{dN}{dt}=(\Lambda-\mu N)$$

$$lnN=(\Lambda-\mu N)t$$

$$N=N_O e^{(\Lambda-\mu N)t} \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots (3.4.4)$$

Here N_O is initial population with initial condition (S_0, V_0, I_0, H_0 .H_{B0}, R_0)

Hence t → ∞ the equation

N will be such that $0 \leq S+V+I+H+H_B+R \leq N_O e^{(1-\mu N)t}$ ……eq (3.4.5)

Thus

$(S, V, I, H, H_B, R) \epsilon \mathbb{R}^6: 0 \leq S + V + I + H + H_B + R \leq N_O e^{(1-\mu)t}$ ... ...…(3.4.6)

This means that the system is uniformly bounded at all time so that the SVIHH_BR model will be biologically feasible.

3.5 Positivity of the Model

The COVID-19 model developed was biologically and mathematically feasible since all the parameters and variables were positive.

3.5.1 Model Lemma

Let the initial condition be

$\{S_0, V_0, I_0, H_0, H_{B0}, R_0 \geq 0\} \epsilon \mathbb{R}^6$ then the solution set $\{ S_t, V_t, I_t, H_t, H_{Bt}, R_t \}$ of the model were positive for all t >0

Proof: From differential equation 3.1(a) of a system

$$\frac{dS}{dt} = \Lambda - (\beta SI + qS + \mu S)$$

Positivity implies that;

$\frac{dS}{dt} \geq (\beta SI + qS + \mu S)$ ... ...(3.5.1)

Or

$dS/S \geq (\beta I + q + \mu)dt.$

On integrating (3.5.2) we have

$lnS(t) \geq (\beta I + q + \mu)$dt

$S(t) \geq \exp(-(\beta I + q + \mu)dt$……(3.5.7)

When t=0, we obtain eq 3.5.7 will be;

$S(t) \geq S(o)\exp(-(\beta SI + qS + \mu S)dt)$……...(3.5.8)

Since

$(\beta I + q + \mu)dt \geq 0$ ……..…….....(3.5.9)

From eq 3.3(b)

$\frac{dV}{dt} = qS - (pI + \mu)V$

Positivity implies that:

$\frac{dV}{dt} \geq (pI + \mu)V$…….(3.5.10)

or

$\frac{dV}{V} \geq (pI + \mu)dt$

On integrating equation (3.5.10) we have

$lnV(t) \geq (pI + \mu)dt$

$V(t) \geq \exp(-(pI + \mu)dt$……..….(3.5.11)

When t=0, we obtain

$V(t) \geq V(o)\exp(-(pI + \mu)dt)$ ……..……..(3.5.12)

Since

$(pI + \mu)\,dt \geq 0$ ……..……....(3.5.13)

From eq 3.3(c)

$$\frac{dI}{dt} = \beta SI + \text{pVI} - (k + \omega + \delta + \mu + \alpha)I$$

For positivity of the equation can be written as;

$$\frac{dI}{dt} \geq (k + \omega + \delta + \mu + \alpha)I$$

or

$$\frac{dI}{I} \geq (k + \omega + \delta + \mu + \alpha)dt \text{……..(3.5.14)}$$

On integrating eq 3.5.14 we have

$$ln\text{I(t)} \geq (k + \omega + \delta + \mu + \alpha)\text{dt} \text{……..(3.5.15)}$$

$$\text{I(t)} \geq \exp(-(k + \omega + \delta + \mu + \alpha)dt \text{……..(3.5.16)}$$

When t=0, we obtain

$$\text{I(t)} \geq I(\text{o})\exp(-(k + \omega + \delta + \mu + \alpha)dt)$$

Since

$$(k + \omega + \delta + \mu + \alpha)\text{dt} \geq 0 \text{…….(3.5.11)}$$

From eq 3.3(d)

$$\frac{dH_B}{dt} = \omega\text{I} - (\chi + \mu + \alpha)H_B$$

For positivity of the equation can be written as;

$$\frac{dH_B}{dt} \geq -(\chi + \mu + \alpha)H_B \text{…… (3.5.17)}$$

or

$$\frac{dH_B}{H_B} = -(\chi + \mu + \alpha)dt \text{……..... (3.5.18)}$$

On integrating equation eq 3.5.18, we have

$lnH_B(t) \geq -(\chi + \mu + \alpha)dt$……...……..eq 3.5.19

$$H_B(t) \geq \ exp\ (-(\chi + \mu + \alpha)dt$$

When t=0 we obtain

$H_B(t) \geq H_{B_O}\ exp\ (-(\chi + \mu + \alpha)dt)$ … . (3.5.20)

Since

$(\chi + \mu + \alpha) \geq 0$ ……...……..……....(3.5.21)

From eq 3.3(e)

$\frac{dH}{dt} = \text{kI} - (\varepsilon + \mu + \alpha)\text{H}$

For positivity of the equation can be written as;

$\frac{dH}{dt} \geq -(\varepsilon + \mu + \alpha)\ H$……..….(3.5.22)

or

$\frac{dH}{H} = -(\varepsilon + \mu + \alpha)\ dt$…….……...(3.5.23)

On integrating the equation eq 3.5.23 we have

ln$H$(t) =exp $(-(\varepsilon + \mu + \alpha)\ dt$ ……(3.5.24)

When t=0 ,we obtain

H (t) = exp $(-(\varepsilon + \mu + \alpha)\ dt)$

Since $\varepsilon + \mu + \alpha \geq 0$……(3.5.25)

From eq 3.3(f)

$\frac{dR}{dt} = \varepsilon H + \chi H_B + \delta I - \mu R$

For positivity of the equation can be written as;

$\frac{dR}{dt} \geq -\mu R$……..…..(3.5.26)

or

$\frac{dR}{R} \geq -(\mu)dt$……...…(3.5.27)

On integrating equation eq 3.5.27 the left-hand side(LHS),the equation become

$lnR(\geq -(\mu)dt$…….. (3.5.28)

When t=0

$R(t) = R_0 \exp(-(\mu)dt)$…….……..(3.5.29)

Since $(\mu) \geq 0$)…….……..(3.5.30)

Hence $the\ system\ S(t), V(t), I(t), H_B(t)H(t), R(t)$ are positive for all t≥ 0

3.6 Disease Free Equilibrium (DFE)

The disease-free equilibrium points of the model are the steady state when there is no corona virus. To determine the D.F.E of this model, we set the entire derivative equal to zero and solve the model equation 3.3(a) to eq 3.3(f) .We also assume that the

susceptible individual receives constant vaccination against the COVID-19 disease for us to get DFE.

To obtain equilibrium points we let S≠ 0 $V = 0$

$\frac{dS}{dt}\ and\ \frac{dV}{dt} = 0,\ \frac{dI}{dt} = \frac{dH}{dt} = \frac{dH_B}{dt} = \frac{dR}{dt} = 0$ ……..……..…..(3.6.1)

By setting the differential equation to be zero, we obtain;

$$\left.\begin{array}{c} \Lambda - (\beta SI + qS + \mu S) = 0 \\ qS - (pI + \mu)\mathrm{V} = 0 \\ \beta SI + \mathrm{pVI} - (k + \omega + \delta + \mu + \alpha)I) = 0 \\ \omega\mathrm{I} - (\chi + \mu + \alpha)H_B) = 0 \\ \mathrm{kI} - (\varepsilon + \mu + \alpha)\mathrm{H} = 0 \\ \varepsilon H + \chi H_B + \delta\mathrm{I} - \mu\mathrm{R} = 0 \end{array}\right\} \text{……. (3.6.2)}$$

Assuming that there is no disease, therefore, when $S \neq 0\ V = 0, I = 0, H_B = 0, H = 0\ and\ R = 0$

From the above equation 3.6.2 becomes;

$\Lambda = (\beta SI + qS + \mu S)$…… (3.6.3)

But $\beta SI = 0$ substituting in equation 3.6.3 we get,

$\Lambda = (q + \mu)S$…….……...……(3.6.4)

Hence

$\frac{\Lambda}{q+\mu} = S_0$..... (3.6.5)

Hence disease-free equilibrium =$(S^*V^*I^*H_B{}^*H^*R^*)$

D.F.E = $(\frac{\Lambda}{q+\mu}, \mathbf{0}, 0,0,0,0)$

3.7 Basic reproduction Number R_0;

Basic reproduction number can be defined as number of the new infections produced by a typical infective individual in a population at DFE. The number can predict whether the COVID-19 will be spread from susceptible population to other individuals. These basic reproduction number acts as a threshold parameter which tells whether the infection dies out or persists in a community. Epidemiologically, the reproductive number of the disease tells us how many secondary cases of infective individual will be produced in an entirely susceptible population during the individual period as an infective. The basic reproduction can be used to determine equilibrium stability as used in Van den et-al (2017).. The higher the R_0, the more likely the disease will become a pandemic. There are three different possibilities that can occur which can be classified using R_0;

i. If $R_0 < 1$,the COVID-19 will not spread and will eventually die out
ii. if R_0 =1, the COVID-19 will remain stable in the society but will not cause an epidemic
iii. If R_0>1, the COVID-19 will spread and cause a pandemic.

In this thesis, the mean number of new COVID-19 infections was accounted for the reproduction number in which a COVID-19 infected individual gets introduced to fully susceptible population or vaccinated population.

The basic reproduction number of the model was obtained using Next generation matrix. Where Heffernan et-al (2005) have used it. The method of obtaining R_0 was worked out as below;

$R_0 = \mathrm{F}V^{-1}$.....(3.7.1)

Where FV^{-1} define spectral radius.

Let X to be the vector function of classes which are infected (I), which are vaccinated and those under treatment that is, home Base care(H_B) and hospitalized individual (H).

Let Y to be the vector of classes which are uninfected which are susceptible individual, those who have been vaccinated and those who recover from the COVID-19.

$$X = \begin{bmatrix} I \\ H \\ H_B \end{bmatrix} \text{And Y} = \begin{bmatrix} S \\ V \\ R \end{bmatrix} \quad \text{.....(3.7.2)}$$

F (X, Y) becomes the vector function containing new infections rate .This same point is made by others (Heffernan et-al, 2005).

V(X, Y) become the vector function containing no new infection rate.

$$\text{F} = \begin{bmatrix} \beta SI \\ 0 \\ 0 \end{bmatrix} \quad \text{..... (3.7.3)}$$

$$\text{V} = \begin{bmatrix} \text{pVI} - (k + \omega + \delta + \mu + \alpha)I \\ \omega\text{I} - (\chi + \mu + \alpha)H_B \\ \text{kI} - (\varepsilon + \mu + \alpha)\text{H} \end{bmatrix} \quad \text{.....(3.7.4)}$$

Calculating the Jacobian of F and V with respect to t matrix of all 1^{st}order partial derivatives becomes;

$$\text{F} = \begin{bmatrix} \frac{dF^1}{dI} & \frac{dF^1}{dH} & \frac{dF^1}{dH_B} \\ \frac{dF^2}{dI} & \frac{dF^2}{dH} & \frac{dF^2}{dH_B} \\ \frac{dF^3}{dI} & \frac{dF^3}{dH} & \frac{dF^3}{dH_B} \end{bmatrix}$$

$$= \begin{bmatrix} \beta S_0 & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \quad \text{.....(3.7.5)}$$

$$V = \begin{bmatrix} \frac{dV^1}{dI} & \frac{dV^1}{dH} & \frac{dV^1}{dH_B} \\ \frac{dV^2}{dI} & \frac{dV^2}{dH} & \frac{dV^2}{dH_B} \\ \frac{dV^3}{dI} & \frac{dV^3}{dH} & \frac{dV^3}{dH_B} \end{bmatrix}$$

$$V = \begin{bmatrix} k + \omega + \delta + \mu + \alpha & 0 & \begin{matrix} 0 \\ 0 \end{matrix} \\ -\omega & \chi + \mu + \alpha & 0 \\ -k & 0 & \varepsilon + \mu + \alpha \end{bmatrix} \ldots \ldots \ldots \ldots \ldots \ldots \ldots \ldots \ldots . \ldots \ldots \ldots . \ldots (3.7.6)$$

Obtaining V^{-1} from equation 3.7.6 becomes;

$$V^{-1} = \begin{bmatrix} k + \omega + \delta + \mu + \alpha^{-1} & 0 & 0 \\ \frac{\omega}{(k+\omega+\delta+\mu+\alpha)(\chi+\mu+\alpha)} & \chi + \mu + \alpha^{-1} & 0 \\ \frac{p}{(k+\omega+\delta+\mu+\alpha)(\varepsilon+\mu+\alpha)} & 0 & \varepsilon + \mu + \alpha^{-1} \end{bmatrix} \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots (3.7.7)$$

We can form the next generation matrix (operator) FV^{-1} from matrix of partial derivates of F and V in equation 3.7.5 and equation 3.7.6.we get

$$FV^{-1} = \begin{bmatrix} \frac{\beta S_0}{k+\omega+\delta+\mu+\alpha} & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots (3.7.8)$$

The Eigenvalues of matrix

$$\frac{\beta S_0}{k+\omega+\delta+\mu+\alpha} \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots (3.7.9)$$

The basic reproduction number R_O and is given by the spectral radius (dominant eigenvalus) of the matrix FV^{-1}

$R_O=\frac{\beta S_0}{(k+\omega+\delta+\mu+\alpha)}$.....(3.7.10)

But $S_0 = \frac{\Lambda}{q+\mu}$

Hence

$R_O=\frac{\beta\Lambda}{(k+\omega+\delta+\mu+\alpha)(q+\mu)}$……..….(3.7.11)

3.8 Local Stability of Disease-Free Equilibrium

Local stability of an equilibrium point means that if you put the system somewhere nearby the point then it will move itself to the equilibrium point after some time.

Theorem

The disease-free equilibrium DFE is locally asymptotically stable if R_O<1

Proof;

Involved the analysis of the local stability of the disease-free equilibrium that is in absences of corona virus. From the model, Jacobian approach was used to form matrix at disease free equilibrium of the non-linear system given by;

$$J = \begin{bmatrix} -(q+\mu) & 0 & -\beta(\frac{\Lambda}{q+\mu}) & 0 & 0 & 0 \\ q & -(q+\rho+\mu) & 0 & 0 & 0 & 0 \\ 0 & \rho & \beta(\frac{\Lambda}{q+\mu}) - (k+\omega+\delta+\mu+\alpha) & 0 & 0 & 0 \\ 0 & 0 & \omega & -(x+\mu+\alpha) & 0 & 0 \\ 0 & 0 & k & 0 & -(\varepsilon+\mu+\alpha) & \delta \\ 0 & 0 & \delta & x & \varepsilon & -\mu \end{bmatrix}$$

.....(3.8.1)

=

$$\begin{bmatrix} -(q+\mu) & 0 & \beta S_0 & 0 & 0 & 0 \\ q & -(q+\rho+\mu) & 0 & 0 & 0 & 0 \\ 0 & \rho & \beta S_0 - (k+\omega+\delta+\mu+\alpha) & 0 & 0 & 0 \\ 0 & 0 & \omega & -(x+\mu+\alpha) & 0 & 0 \\ 0 & 0 & k & 0 & -(\varepsilon+\mu+\alpha) & \delta \\ 0 & 0 & \delta & x & \varepsilon & -\mu \end{bmatrix}$$

..(3.8.2)

Which yields the following eigenvalues;

$$\begin{pmatrix} -(q+\mu) \\ -(x+\mu+\alpha) \\ -(\varepsilon+\mu+\alpha) \\ -(\pi+\mu) \\ -(q+\rho+\mu) \\ \beta\frac{\Lambda}{q+\mu} - (k+\omega+\delta+\mu+\alpha) \end{pmatrix} \quad \text{.....(3.8.4)}$$

Five of the eigenvalues are negative hence to make the system stable we need to have

$$\beta\frac{\Lambda}{q+\mu} - (k+\omega+\delta+\mu+\alpha) > 0 \quad \text{.....(3.8.5)}$$

Hence

$$\beta\frac{\Lambda}{q+\mu} > (k+\omega+\delta+\mu+\alpha) \quad \text{.....(3.8.6)}$$

In conclusion, if $\beta\frac{\Lambda}{q+\mu} > (k+\omega+\delta+\mu+\alpha)$. This means the disease-free equilibrium is asymptotically locally stable.

3.9 Global Stability of Disease-Free Equilibrium

Global stability means that the system will come to the equilibrium point from any possible starting point. The stability at the equilibrium points is analyzed based on the Lyapunov invariance principal. By using appropriate Lyapunov function, the uninfected equilibrium point is proven to be globally asymptotically stable when the reproduction number is less than one and unstable otherwise.(Li et-al 2000)

Systematically, we define a Lyapunov function L such that

$$L=\frac{1}{k+\omega+\delta+\mu+\alpha_1} l_c \qquad (3.9.1)$$

The

$$\frac{dL}{dt}=\frac{1}{(k+\omega+\delta+\mu+\alpha_1)(q+\mu)}\frac{dc}{dt} \qquad (3.9.2)$$

$$\frac{dL}{dt}=\frac{1}{k+\omega+\delta+\mu+\alpha_1}(Bsl_c-(k+\omega+\delta+\mu+\alpha_1)(q+\mu)l_c \qquad (3.9.3)$$

$$=\frac{Bsl_c}{(k+\omega+\delta+\mu+\alpha_1)(q+\mu)}-l_c \qquad (3.9.4)$$

$$\frac{dL}{dt}\leq\left(\frac{Bsl_c}{(k+\omega+\delta+\mu+\alpha_1)(q+\mu)}-1\right) l_c \qquad (3.9.5)$$

$$\frac{dL}{dt}\leq(R_O-1)l_c \qquad (3.9.6)$$

So $\frac{dL}{dt}\leq 0$, if $R_O\leq 1$

Furthermore, $\frac{dL}{dt}=0$ if $l_c=0\ or R_O=1$

Hence DFE is globally asymptotically stable if $R_O\leq 1$

3.10 The endemic equilibrium (EE)

This section, looked at the existence of endemic equilibrium point. Let denote **t**he endemic equilibrium by $E*$ and defined as a steady state solution for the Model. This can occur when there is a persistence of the disease. It was obtained by equating the system of equation to zero.

$$\begin{cases} \Lambda - (\beta S^* I^* + qS^* + \mu S^*) = 0 \\ qS^* - (p + \mu)V^* = 0 \\ \beta S^* I^* + pV^* I^* - (k + \omega + \delta + \mu + \alpha)I^* = 0 \\ \omega I^* - (\chi + \mu + \alpha){H_B}^* = 0 \\ kI^* - (\varepsilon + \mu + \alpha)H^* = 0 \\ \varepsilon H^* + \chi {H_B}^* + \delta I^* - \mu R^* = 0 \end{cases} \quad \text{.....(3.10.1)}$$

From the second, fourth, fifth and the sixth equations (eq 3.10.1) of the model the following equation were obtained;

$S* = \frac{(p+\mu)V*}{q}$ ..... (3.10.2)

$V* = \frac{qS*}{(p+\mu)}$ ..... (3.10.3)

$I^* = \frac{\mu R^* - \varepsilon H^* - \chi {H_B}^*}{\delta}$ ..... (3.10.4)

$H_B* = \frac{\omega I*}{(\chi+\mu+\alpha)}$ ..... (3.10.5)

$H* = \frac{kI*}{(\varepsilon+\mu+\alpha)}$ .....(3.10.6)

$R* = \frac{\varepsilon H* + \chi H_B* + \delta I*}{\mu}$ ..... (3.10.7)

Hence E∗= (S∗; V∗; H_B∗; H∗; I∗; R∗) is the endemic equilibrium of the model

3.11 Local stability of endemic equilibrium (EE)

Theorem If R_o>1 then the endemic equilibrium E∗=$(S^*, V^*, I^*, H_B{}^*, H^*, R^*)$of the governing model differential equation will be asymptotically stable.

Thus, the Jacobian matrix with respect to equation (3.3.1) is given by;

$$J_{E*} = \begin{bmatrix} -\beta I^* - q - \mu & 0 & -\beta S^* I^* & 0 & 0 & 0 \\ q & -(q+\rho+\mu) & 0 & 0 & 0 & 0 \\ \beta I^* & \rho & \beta S^* I^* - (k+\omega+\delta+\mu+\alpha) & 0 & 0 & 0 \\ 0 & 0 & \omega & -(x+\mu+\alpha) & 0 & 0 \\ 0 & 0 & k & 0 & -(\varepsilon+\mu+\alpha) & \delta \\ 0 & 0 & \delta & x & \varepsilon & -\mu \end{bmatrix}$$

.....(3.11.1)

We show the stability of the matrix J_{E*} by verifying the Rourth-Hurwitz conditions, that is, all the roots of the resulting characteristic equations must have negative real part. The characteristic polynomial of Jacobian matrix at $E^u$is given by det $J_{E*} - \lambda I = 0$, where λ is the eigenvalue and I is 6× 6 identity matrix. Thus,

$$(J_{E*} - \lambda I) = \begin{bmatrix} -\beta I^* - q - \mu & 0 & -\beta s^* I^* & 0 & 0 & 0 \\ q & -(q+\rho+\mu) & 0 & 0 & 0 & 0 \\ -\beta I^* & \rho & -\beta s^* I^* - (k+\omega+\delta+\mu+\alpha) & 0 & 0 & 0 \\ 0 & 0 & \omega & -(x+\mu+\alpha) & 0 & 0 \\ 0 & 0 & k & 0 & -(\varepsilon+\mu+\alpha) & \delta \\ 0 & 0 & \delta & x & \varepsilon & -\mu \end{bmatrix}$$

.

……..……(3.11.2)

$= -\beta s^* I^* - (k + \omega + \delta + \mu + \alpha) - \lambda 1[(x + \mu + \alpha) - \lambda 2[-(\varepsilon + \mu + \alpha) - \lambda 3][-\mu - \lambda 4]]$

$\lambda 1 = -(k + \omega + \delta + \mu + \alpha) < 0$

$\lambda 2 = -(x + \mu + \alpha) < 0$

$\lambda 3 = -(\varepsilon + \mu + \alpha) < 0$

Meaning $\lambda 4 \lambda$,5 and $\lambda 6 < 0$ (all roots are negative).

Hence by Routh-Hurwitz criteria as in Boyce et-al. (2017), we have that the eigenvalues of $J_{\mathrm{E}*}$ has negative real part when reproduction number $R_0 > 1$. This shows that the endemic equilibrium E∗ is locally asymptotically stable.

3.12 Global stability of endemic equilibrium

We note that there are no established procedures for calculating a Lyapunov function, and often finding a Lyapunov function is tedious and tricky when using trial and error approach Martcheva et-al (2015).We determine the global stability of the endemic equilibrium E∗ by defining the following Lyapunov function:

$$V(S^*V^*I^*{H_B}^*H^*R^*)=(S - S^* - S^* log \frac{S^*}{s})+(V - V^* - V^* log \frac{V^*}{V})+(I - I^* - I^* log \frac{I^*}{I})+$$

$$(H_B - {H_B}^* - {H_B}^* log \frac{{H_B}^*}{H_B}) + (H - H^* - H^* log \frac{H^*}{H}) + (R - R^* - R^* log \frac{R^*}{R}) \ldots. (3.12.1)$$

V is positive definite since V=0 when$(\mathrm{S}, \mathrm{V}, \mathrm{I}, \mathrm{H}, H_B, \mathrm{R}) = (S^*, V^*, I^*, {H_B}^*, H^*, R^*)$ and V>0 otherwise; V was radically unbounded. Hence, V is a Lyapunov function.

We prove that, the derivative of V with respect to t is negative as in korobeinikov (2006). The derivative of V, by calculating along the (eq 3.12.1) becomes;

$$V' = (\frac{S - S^*}{S})S' + (\frac{V - V^*}{V})V' + (\frac{I - I^*}{I})I' + (\frac{H_B - {H_B}^*}{H_B}){H_B}' + (\frac{H - H^*}{H})H' + (\frac{R - R^*}{R})R' \ldots\ldots\ldots\ldots (3.12.2)$$

Hence

$V' = (\frac{S-S^*}{S})[\Lambda - (\beta I + q + \mu)] + (\frac{V-V^*}{V})[(qS - (p + \mu)] + (\frac{I-I^*}{I})[\beta SI + pVI - (k + \omega + \delta + \mu + \alpha)] + (\frac{H_B - H_B{}^*}{H_B})[\omega I - (\chi + \mu + \alpha)] + (\frac{H-H^*}{H})[kI - (\varepsilon + \mu + \alpha)] + (\frac{R-R^*}{R})[\varepsilon H + \chi H_B + \delta I - \mu]$……..……...(3.12.3)

Or

$$V' = F - G$$

where F represent the positive terms and G the negative terms of the equation. Hence F<G in the equation above, then we have that $V' = 0$.

Clearly $V' = 0$ if and only if $S = S^*, V = V^*, I = I^*, H_B = H_B{}^*, H = H^*, R = R^*$.Thus E∗ is the endemic equilibrium of the model and was globally asymptotically stable if F<G . Boyce et-al. (2021) made this same point.

CHAPTER FOUR

RESULTS AND DISCUSSIONS

4.1 Introduction

This section outlines the statistical output obtained based on methodologies used so as to achieve each of the study objectives.

4.2 The Feasibility Region

The total population N is the sum of the population in the susceptible, vaccinated, infective, hospitalized, home base care and recovered compartment i.e., N=S+V+I+H+H_B+R.

This proof that the model is non-negative and uniformly bounded.

4.3 Positivity of a Model

We are dealing with human being population hence the COVID-19 model is biologically and mathematically feasible since all the parameters and variables are positive.

$(\beta I + q + \mu)$dt≥ 0 from equation (3.5.9)

replacing the parameters with the values in table 3.1

βI =0.5787x150

q =0.4213

μ =0.00411

Substituting the above values in the equation we get 87.2304≥ 0 hence the model has positive parameters and variables.

$(pI + \mu)$ dt≥ 0 from equation (3.5.13)

replacing the parameters with the values in table 3.1

pI =0.2x 150

μ =0.00411

Substituting the above values in the equation we get $60.00411 \geq 0$ hence the model has positive parameters and variables.

$(k + \omega + \delta + \mu + \alpha)dt \geq 0$ from equation (3.5.11)

replacing the parameters with the values in table 3.1

k =0.5493

ω =0.9506

$\delta = 0.189$

μ =0.00411

$\alpha = 0.00961$

Substituting the above values in the equation we get $1.15322 \geq 0$hence the model has positive parameters and variables.

$(\chi + \mu + \alpha) \geq 0$ from equation (3.5.21)

replacing the parameters with the values in table 3.1

χ=0.1612

μ =0.00411

$\alpha = 0.00961$

Substituting the above values in the equation we get $0.17492 \geq 0$hence the model has positive parameters and variables.

$\varepsilon + \mu + \alpha \geq 0$ from equation (3.5.25)

replacing the parameters with the values in table 3.1

ε=0.15

μ =0.00411

$\alpha = 0.00961$

Substituting the above values in the equation we get $0.16372 \geq 0$ hence the model has positive parameters and variables.

$(\mu) \geq 0$)from equation (3.5.30)

μ =0.00411

Substituting the above values in the equation we get 0.00411≥ 0 hence the model has positive parameters and variables.

4.4 The Basic Reproduction Number

The basic reproduction number $R_O$is an estimation which determines if there will be an outbreak of COVID-19 or not.

Since R_O was an estimation, the most dominant Eigen-value was picked.

R_O=$\frac{\beta S_O}{(k+\omega+\delta+\mu+\alpha)}$ from equation (3.7.10)

Where $\frac{\Lambda}{q+\mu} = S_0$

R_O=$\frac{\beta\Lambda}{(k+\omega+\delta+\mu+\alpha)(q+\mu)}$

replacing the parameters with the values in table 3.1

where, $\beta = 0.5787$

k =0.5493

ω =0.9506

$\delta = 0.189$

μ =0.00411

$\alpha = 0.00961$

Substituting in the equation above we get R_0 =0.7584 hence the $R_0 < 1$

Hence, it's proof that the disease-free equilibrium is asymptomatically locally stable

4.5. The Disease-Free Equilibrium

The estimation of the basic reproduction number determines the disease-free equilibrium. At the DFE, the determinant of the Jacobian matrix is positive at $R_0 > 1$ then the model is stable.

Hence disease-free equilibrium =($S^*V^*I^*{H_B}^*H^*R^*$) = ($\frac{\Lambda}{q+\mu}$, 0,0,0,0,0)

S=$\frac{\Lambda}{q+\mu}$ .from equation (3.6.5)

The susceptible population is the total population which is free of the disease while V= $I = H_B = H = R = 0$.

This means that the infected, home-based care and hospitalized are not there because there is no disease in the equilibrium. Since there are no diseases, no one recovers.

4.6 Local Stability of the Disease-free equilibrium.

The equation $\beta\frac{\Lambda}{q+\mu} > (k + \omega + \delta + \mu + \alpha)$ from equation (3.8.6)

Proof; replacing the parameters with the values in table 3.1;

$\beta = 0.5787$

$\Lambda = 10$

$q = 0.4213$

k =0.5493

ω =0.9506

$\delta = 0.189$

μ =0.00411

$\alpha = 0.00961$

13.603>1.15322 was obtained.

This proof that the disease-free equilibrium was asymptomatically locally stable.

4.7 Global Stability of Disease-Free Equilibrium

DFE is globally stability is $R_O \leq 1$

But reproduction number R_0 =0.7584

Hence 0.7584≤ 1

This proof that the disease-free equilibrium is globally asymptomatically stable.

4.8 **The endemic equilibrium (EE)**

$S* = \frac{(p+\mu)V*}{q}$ from equation (3.10.2)

Replacing values of parameters in table 3.1

p =0.2

q =0.4213

μ =0.00411

we get: $S* = 0.4845\ V*$

$V* = \frac{qS*}{(p+\mu)}$ from equation (3.10.3)

Replacing values of parameters in table 3.1

p =0.2

q =0.4213

μ =0.00411

we get: $V* = 2.064S*$

$I^* = \frac{\mu R^* - \varepsilon H^* - \chi H_B{}^*}{\delta}$ from equation (3.10.4)

Replacing values of parameters in table 3.1

χ=0.1612

μ =0.0041

$\delta = 0.189$

$\varepsilon = 0.15$

we get: $I^* = \frac{0.00411R^* - 0.15H^* - 0.1612H_B{}^*}{0.189}$

$H_B * = \frac{\omega I*}{(\chi+\mu+\alpha)}$ from equation (3.10.5)

Replacing values of parameters in table 3.1

ω =0.95061

χ=0.1612

μ =0.0041

$\alpha = 0.00961$

we get: $H_B * = \frac{0.9506I*}{0.17492}$

H $* = \frac{\mathrm{k}I*}{(\varepsilon+\mu+\alpha)}$ from equation (3.10.6)

Replacing values of parameters in table 3.1

k = 0.5493

ε=0.15

μ =0.00411

$\alpha = 0.00961$

we get: $H * = \frac{0.5493I*}{0.16372}$

$R * = \frac{\varepsilon H* + \chi H_B* + \delta I*}{\mu}$ from equation (3.10.7)

Replacing values of parameters in table 3.1

ε=0.15

$\delta = 0.189$

$x = 0.1612$

μ =0.00411

we get: $R* = \frac{0.15H* + 0.1612H_B* + 0.189I*}{0.00411}$

Hence E∗= (S∗; V∗; H_B∗; H∗; I∗; R∗) is the proof of endemic equilibrium of the model

4.9 Local stability of endemic equilibrium (EE)

The equation

$= -\beta s^* I^* - (k + \omega + \delta + \mu + \alpha) - \lambda 1[(x + \mu + \alpha) - \lambda 2[-(\varepsilon + \mu + \alpha) - \lambda 3][-\mu - \lambda 4]]$

$\lambda 1 = -(k + \omega + \delta + \mu + \alpha) < 0$

$\lambda 2 = -(x + \mu + \alpha) < 0$

$\lambda 3 = -(\varepsilon + \mu + \alpha) < 0$ Is true ,

Proof: k =0.5493

ω =0.9506

$\delta = 0.189$

μ =0.00411

$\alpha = 0.00961$

$x = 0.1612$

$\varepsilon = 0.15$

we have that the eigenvalues of J_{E*} has negative real part when reproduction number $R_0 > 1$. This shows that the endemic equilibrium E∗ is locally asymptotically stable.

4.10 Global stability of endemic equilibrium

$V' = (\frac{S-S^*}{S})[\Lambda - (\beta I + q + \mu)] + (\frac{V-V^*}{V})[(qS - (p + \mu)] + (\frac{I-I^*}{I})[\beta SI + pVI - (k + \omega + \delta + \mu + \alpha)] + (\frac{H_B - H_B{}^*}{H_B})[\omega I - (\chi + \mu + \alpha)] + (\frac{H-H^*}{H})[kI - (\varepsilon + \mu + \alpha)] + (\frac{R-R^*}{R})[\varepsilon H + \chi H_B + \delta I - \mu]$ from equation (3.12.3)

Proof: the initial condition in table 3.2 S=1400

V=300

I=150

$H_B = 200$

$H = 250$

$R = 100$

Replacing the above parameters and $S = S^*, V = V^*, I = I^*, H_B = H_B{}^*, H = H^*, R = R^*$.

$V' = 0$ was obtained.

Thus, this the proof that the endemic equilibrium was globally asymptotically stable

4.11 Quantitative analysis of the model.

The numerical solution of the COVID-19 model problem was computed using the classical fourth order Runge-Kutta method Atkinson, (2008); Boyce et-al., (2021); Martcheva, (2015) code using Maple mathematics tool. The pure numerical technique was used to solve the differential equations of system (3.3.1) of the form:

$$\frac{dS}{dt} = f(R, S, I),$$

$$\frac{dV}{dt} = \boldsymbol{f}(S, \mathrm{V})$$

$$\frac{dI}{dt} = f(S, I, \mathrm{V}),$$

$$\frac{dH_B}{dt} = f(\mathrm{I}, H_B)$$

$$\frac{dH}{dt} = f(\mathrm{I},\mathrm{H})$$

$$\frac{dR}{dt} = f(H, H_B, \mathrm{V}, \mathrm{I}, \mathrm{R})$$

Satisfying

$S(0) = S_0; I S_0 = I S_0; V_s(0) = V S_0 H(0) = H r_0 \quad H_B(0) = H_B r_0$ and $R(0) = R0$.

We let $h = t_{n+1} - t_n, \quad n = 0.1.2$ ....

So that the Taylor series of S (t_{n+1}) = S_{n+1} about S_n is given by,

$$\mathrm{S}(t_{n+1}) = S_n + hf(t_n, S_n) + \frac{1}{2}!h^2 f(t_n, S_n) + ..$$

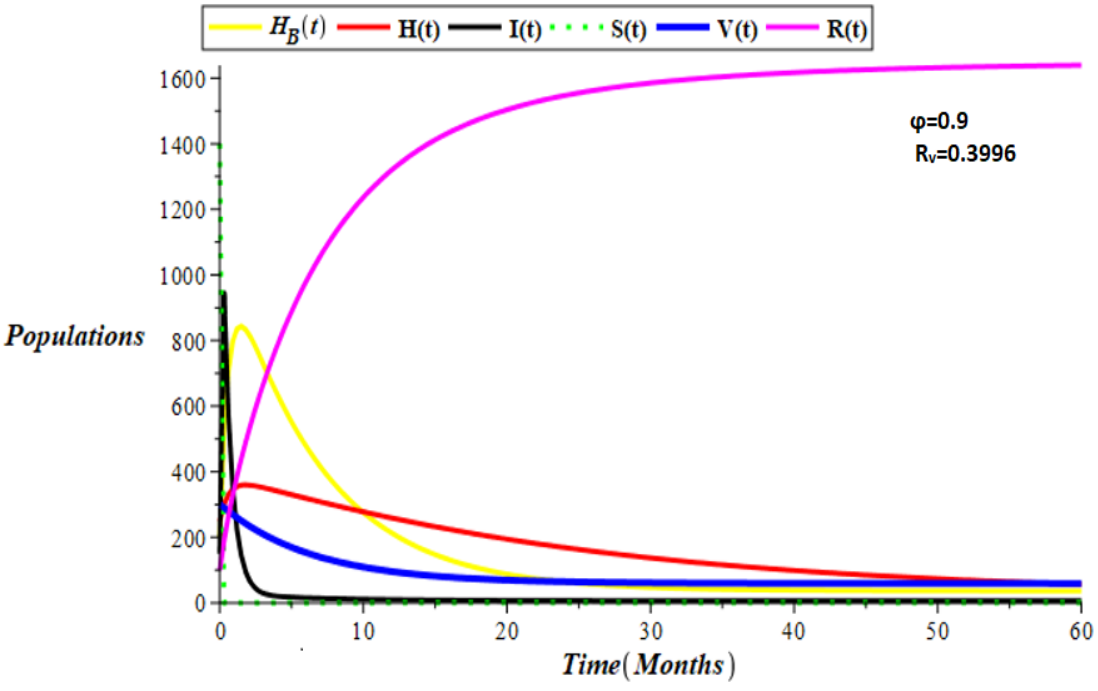

Figure 4. 1 Simulation for population dynamics with high vaccination coverage

Figure 4.1 demonstrates the impact of high vaccination coverage on the disease-free initial population dynamics. The total population is assumed that at the initial year all the human population is susceptible to the COVID-19 disease; this implies that all individuals are likely to be affected by the disease. We note that the population of the susceptible individuals decreases with time while that of the Recovered group gradually increases due to recruitment of vaccinated susceptible individuals and infective susceptible individuals. Vaccinated individual gradually decreases with time due to recruitment to infective individual. The population of individuals under treatment both in hospital and those under home based care decrease gradually due to high recovary rate. The recovery group display a sharp increase as time increases due to more recruitment through vaccination. It is interesting to note that with an initial low number of infective populations the COVID-19 disease gradually grows until it attains a peak value then decreases gradually to disease Free State. The exponential rise of infections initially is due to recruitment of susceptible as a result of high force of infection witnessed in COVID-19 disease. The sharp decrease from peak to disease free state is due to treatment and high vaccination coverage ($\varphi = 0.9$). The Population eventually attains disease free with all the time.

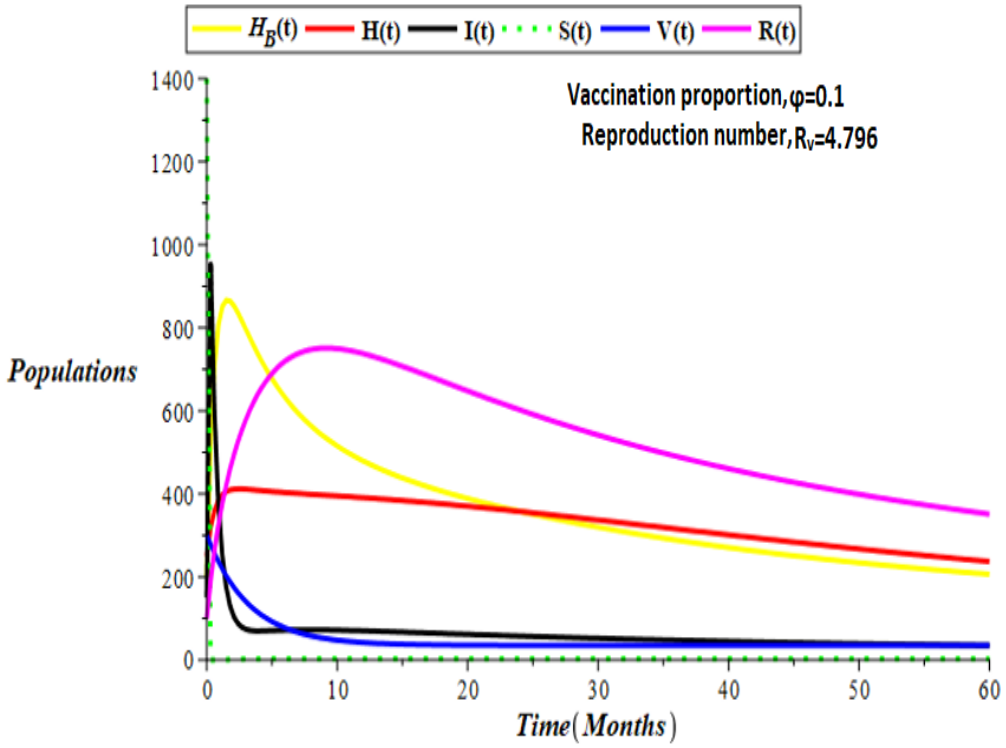

Figure 4. 2 Simulation for population dynamics with low vaccination coverage

Figure 4.2 shows the effect of low vaccination proportion on the dynamics of population with low number of infective present in the community. The population of the susceptible group reduces gradually with time as well as small increase in the population of recovered group is noted. There will be high reproduction number. However, it is important to observe that the population of infective may never disappear with time and the endemic equilibrium state will not be achieved. This demonstrates that a disease-free equilibrium only occurs when the reproduction number $R_O > 0$. Hence, low vaccination coverage level the reproduction number is $R_O < 0$. leads to persistence of the COVID-19 disease in the Community with the endemic state being stable asymptotically.

CHAPTER FIVE

CONCLUSION AND RECOMMENDATIONS

5.1 Conclusion

A $SVIHH_BR$ deterministic model formulated that monitors the temporal dynamics of COVID-19 disease in the presence of preventive vaccination. The mathematical analysis was done using ordinary differential equation (ODE). The data used in simulation is based on the disease spread in Ke nya early 2022 and the findings revealed the presence knowledge and acceptability among medical staff towards COVID-19 vaccination. The model incorporates the fact that susceptible population are infectious to the community they spread COVID-19 to other individuals.

The study proves the existence of the feasible region that is mathematically is non-negative and uniformly bounded. The model has positive parameters and variables since we are dealing with human population. The existences of diseases free and endemic equilibrium are asymptomatically locally stable.

The model is theoretically analyzed; its effective and basic reproduction numbers are derived. It is observed that, when $R_O < 1$ the disease-free equilibrium is locally asymptotically stable and the disease could be eradicated otherwise was unstable. The equations indicate that the COVID-19 disease is declining with a very high number of individual's recovery and it is noted that if the vaccine efficiency is low and the disease reproduction number is high, the disease may not be eradicated even if a large proportion of the population is vaccinated. The global stability of endemic equilibrium is attained if vaccination reproduction number is greater than unity.

The computations using Fourth order Runge-Kutta method indicates that COVID-19 can be controlled in the community with the implementation of high vaccination rate

and treatment while our results suggest that vaccination and treatment are very effective in reducing the spread of disease as stated by Atangana (2020).

5.2 Recommendation

Based on finding in figure 4.1 and 4.2 we recommend high vaccination rate to achieve a disease-free state in our community.More so, policy makers should take steps to encouragement individuals to have positive perceptions toward vaccination and improved acceptability towards COVID-19 vaccinations in order to reduce the vaccine hesitancy and the spread of the disease.

This study is not exhaustive students and researchers can investigate the chances at which the vaccinated individual and the recovery individual can be carries to COVID-19.

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APPENDIX I: Similarity Report