Modeling the Effects of Vaccination and Incubation on Covid-19 Transmission Dynamics

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MODELING THE EFFECTS OF VACCINATION AND INCUBATION ON COVID-19 TRANSMISSION DYNAMICS

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A Thesis Submitted to the Board of Graduate Studies in Partial Fulfillment of the Requirements for the Conferment of a Master of Science Degree in Applied Mathematics of the University of Kabianga

UNIVERSITY OF KABIANGA

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DECLARATION AND APPROVAL

Declaration

I declare that this thesis is my original work and has not been presented for award or conferment of any diploma or degree in this or any other institution.

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ABSTRACT

The Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-COV-2) is a strain of Coronavirus that causes Coronavirus Disease 2019 (COVID-19). The respiratory illness responsible for the COVID-19 pandemic began in December 2019 in Wuhan city, China. Mathematical modeling has enabled the epidemiologist to understand the dynamics of the disease, its impact and future predictions in order to provide the governments with the best policies and strategies to curb the spread of the virus. Deterministic susceptible-vaccinated-asymptomatic-infectious-recovered (SVAIR) model was formulated incorporated with time delay. The delay accounts for the time lapsed between exposure and infectious period. In this study delay differential equations (DDEs) were formulated for the purposes of determining the stability of both disease free equilibrium (DFE) and endemic equilibrium point (EEP). It was found that the model was stable at both Disease Free Equilibrium (DFE) and Endemic Equilibrium Point (EEP) and was attained whenever $R_0 < 1$ and $R_0 > 1$ respectively. Calculations based on secondary data from various works of literature and the WHO dashboard was used. The basic reproduction number (R_0) was computed using the next generation matrix (NGM) approach. The sensitivity analysis was carried out, it was found out that for all model parameters under study, the signs of the sensitivity indices of R_0 were all negative. This implied that the model parameters under consideration contributed to the decline in the spread of the COVID-19 infection. It was noted that the rate of progression from asymptomatic to infectious δ_1 was more sensitive than other model parameters since it contributed the most negative sensitivity index of -0.9265. Finally, numerical simulation was done using MATLAB for validation of the analytical results. Graphical representation shows that stability is achieved when $\tau >$5 days and that R_0 = 0.95 at DFE. Furthermore at EEP it was noted that R_0 = 1.02 hence stability was guaranteed.

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DEDICATION

I devote this thesis to my wife Monicah and children Kipchirchir, Sandra, Chebii and Chesang. I thank you for your love and moral support.

ACKNOWLEDGEMENT

I want to start by giving thanks to God for this amazing chance to attend the University of Kabianga. Secondly, my sincere gratitude and appreciation goes to my supervisors; Dr. Wesley Kirui, Dr. Reuben Langat and Dr. Benard Tonui for their endless support, guidance and encouragement throughout the period of research writing, may God bless you all as you continue helping other students. I also take this opportunity to thank the Department of Mathematics, Actuarial and Physical Sciences lecturers as a whole for their encouragement throughout. Finally, I am grateful to my friends and postgraduate colleagues for giving me their valuable time ranging from academic discussions to social interaction. To my family, thank you for your moral and financial support, God bless you.

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LIST OF ABBREVIATIONS

COVID-19	Coronavirus Disease 2019
DFE	Disease Free Equilibrium
DDEs	Delay Differential Equations
EEP	Endemic Equilibrium Point
MATLAB	Mathematics Laboratory
NGM	Next Generation Matrix
ODEs	Ordinary Differential Equations
PDEs	Partial Differential Equations
SIR	Susceptible Infected Recovered
SEIR	Susceptible Exposed Infected Recovered
SVAIR	Susceptible Vaccinated Asymptomatic Infected Recovered
SARS-COV-2	Severe Acute Respiratory Syndrome Coronavirus 2
WHO	World Health Organization

LIST OF SYMBOLS

τ	Incubation period
γ	Natural death rate
α	Rate of recovery
S	Susceptible individuals
V	Vaccinated individuals
A	Asymptomatic individuals
I	Infectious
R	Removed
N	Total population
R_0	Basic Reproduction Number
μ	Disease induced death rate
P	Recruitment rate into susceptible
ω	Proportion of recruited individuals who are vaccinated
β_1	Transition rate of susceptible individuals to infectious class per contact time.
β_2	Transition rate of susceptible individuals to asymptomatic class per contact time
δ_1	Transition rate of asymptomatic individuals into infectious class
δ_2	Recovery rate of asymptomatic individuals
ε_1	Transition rate of vaccinated individuals to infectious class per unit time
ε_2	Transition rate of vaccinated individuals to asymptomatic class per unit time
ε_3	Transition rate of vaccinated individuals to recovered class per unit time

DEFINITIONS OF TERMS

Basic reproduction number: The mean number of new infections arising from a single infectious person introduced into a purely susceptible population, (Martcheva M et al.., 2015.)

Delay Differential Equation (DDEs) : Are a type of differential equation in which the derivative of the unknown function at a given time is given in terms of the values of the function at previous time.

Epidemiology: This is a branch of medical science that studies the distribution of disease in human populations and the factors determining that distribution, chiefly by the use of statistics

Infectious disease: Infectious diseases are the diseases caused by various pathogenic microorganisms such as virus, bacteria, protozoan, fungi, and other parasites.

Incubation period:The period between exposure to an infectious agent and the onset of symptoms of the disease.

Mathematical modeling:The art of translating problems from the real life phenomena into tractable mathematical formulations whose theoretical and numerical analysis provides insights answers and guidance useful for any particular phenomenon, (McKendrick et al., 1997.)

Prevalence: The number of people who have the disease at a specific time.

CHAPTER ONE

INTRODUCTION

1.1 Overview

Background data on the kinetics of COVID-19 transmission is covered in this chapter. Additionally, the definition of delay differential equations is provided. This includes the next-generation matrix for calculating the basic reproduction number R_0, as well as the dynamic equations of an example SIR and SEIR model. Lastly, the statement of the problem, objectives of the study, justification of the study and significance of the study are considered.

1.2 Background information

The devastating Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-COV-2) is a strain of Coronavirus that causes Coronavirus Disease 2019 (COVID-19). The respiratory illness responsible for the COVID-19 pandemic which began in December 2019 (WHO, 2020) in Wuhan City, China and spread to many countries in the world (Shen et al., 2020). Kenya reported its first case of COVID-19 in March 2020. This resulted in the restriction of movement by the government. This included the closure of all learning institutions (McCloskey et al., 2020)

The COVID-19 pandemic has caused a public health and economic crisis worldwide. In response to the pandemic, countries implemented non-pharmaceutical interventions (NPIs), but social and economic consequences of certain NPIs made them unsustainable in the long-term. The rollout of COVID-19 vaccines following approval in December 2020 and in early 2021 contributed to a substantial reduction in pandemic burden, thus raising the possibility of relaxing NPIs. While emergence of more transmissible variants presented new challenges, many countries, such as Switzerland, were faced with having to make decisions on when and how to relax NPIs while

continuing to protect their population.

As vaccination coverage against SARS-CoV-2 increases amidst the emergence and spread of more infectious and potentially more deadly viral variants, decisions on timing and extent of relaxing effective, but unsustainable, non-pharmaceutical interventions (NPIs) need to be reviewed. Non-pharmaceutical interventions (NPIs) can curb the spread of SARS-CoV-2 in an otherwise unprotected population by reducing the number of potentially transmissible pairwise contacts. In Kenya, NPIs have targeted several aspects of public life, including closure of learning institutions, restaurants and other entertainment venues, restrictions in sizes of spontaneous gatherings, the cancellation of events and facemask mandates in publicly accessible spaces.

As of 18th April 2022, COVID-19 had spread to nearly every country in the world, resulting in 500,186,525 confirmed cases of infection and 6,190,349 deaths globally, according to the WHO's pandemic declaration from March 2020 (WHO, 2020). The number of COVID-19 cases reported to WHO by selected countries as at April 2022 include ; USA had 79,716,960 confirmed cases and 2,711,776 deaths, United Kingdom had reported 21,715,120 confirmed cases and 979,321 deaths, Africa had reported cumulative of 8,676,141 confirmed cases and 171,350 deaths and Kenya had reported 323,588 confirmed cases and 5649 deaths, (Shen et al., 2020).

Over time, it became evident that the Kenyan government's non-pharmaceutical mitigation measures to curb the spread of COVID-19 were not effective. Use of face masks, keeping of social distance, routine hand washing, isolation of suspected cases and contact tracing are a few examples of such intervention measures. Because of the rapid spread of COVID-19 globally, vaccination has been identified as the best containment strategy by several countries. The WHO approved COVID-19 vaccines, but these vaccines remain inadequate in supply and developing nations continue to experience the shortages of COVID-19 vaccines.

Kenya used different types of vaccines such as Astra Zeneca, Modena, Pfizer and Johnson and Johnson. About 15% of the whole population had received further booster vaccinations totaling to about 285,700 individuals, one year after Kenya began administering vaccines on March 5, 2021. The public delayed uptake of the COVID-19 vaccine led to daily reports of COVID-19 cases to fluctuate, making it difficult to forecast how many COVID-19 cases may occur in the

future. The coronaviruses had a high risk of being transmitted from person to person (Wangari et al., 2021).

Some developed nations manufactured their own COVID-19 vaccines which became a greater challenge for developing nations to obtain enough vaccines to administer to their populations because of the growing demands for the manufactured vaccines (Prieto Curiel et al., 2021), this lowered the rate of vaccination. Less than 20% of the population in Kenya received a full vaccination, one and a half year after vaccination program began. The mathematical model would take these therapeutic approaches into account; including vaccination for those who are susceptible and treatment for those who are infectious.

1.3 Vaccines

Fully susceptible, partially susceptible and actively infected individuals not in hospital can potentially receive a vaccine. Vaccination was modeled using two assumptions: first, to trigger an immune response that blocks transmission for a proportion of exposure events, and second, to reduce the probability of developing symptoms if infection does occur. Upon vaccination, there is a time delay until the full efficacy of the vaccine is realized. Kenya used different types of vaccines such as Astra Zeneca, Modena, Pfizer and Johnson and Johnson in which in this study, the immune response for all vaccines was assumed to be equal. We assume the vaccine is 80% transmission blocking and has a further 75% probability of preventing symptoms leading to the observed 95% vaccine efficacy reported in clinical trials (Baden et al., 2021). In this study, we do not model decay of vaccine efficacy over time due to variants mutation.

The COVID-19 pandemic has caused a public health and economic crisis worldwide. In response to the pandemic, countries implemented non-pharmaceutical interventions (NPIs), but social and economic consequences of certain NPIs made them unsustainable in the long-term. The rollout of COVID-19 vaccines following approval in December 2020 and in early 2021 contributed to a substantial reduction in pandemic burden, thus raising the possibility of relaxing NPIs. While emergence of more transmissible variants presented new challenges, many countries, such as Kenya, were faced with having to make decisions on when and how to relax NPIs while continuing to protect their population. Mathematical models have proven to be useful in

providing insights to support this type of country-wide decision-making around different control strategies and public health objectives (Hall et al 2022). Here, we present a deterministic COVID-19 disease model which captures COVID-19 transmission dynamics using delay differential equations. The total population was subdivided into five compartments namely: Susceptible (S), Vaccinated (V), Asymptomatic (A), Infectious (I) and Recovered (R). In this study therefore, a SVAIR mathematical model for COVID-19 disease transmission dynamics was developed.

1.4 Mathematical Models

Epidemiologists use mathematical models to describe the dynamics of infectious diseases. There are two types of mathematical models namely; deterministic and stochastic models.
A stochastic, deterministic, individual-based transmission model of SARS-CoV-2 infection and COVID-19 disease model are just a few examples of mathematical models among many. The COVID-19 disease model tracks the characteristics of individuals such as age, risk-group (namely those with comorbidity), SARS-CoV-2 infection status, COVID-19 disease state, level of immunity, and vaccination details. If infected, an individual's viral load is tracked as a function of the time since the infection, along with the viral variant with which an individual is infected. The model captures viral transmission as infectious and susceptible people come into contact with each other. The probability of transmission is dependent on the viral load of the infectious individual, the variant of the virus being transmitted, and any partial immunity acquired by the susceptible individual either through previous infection or vaccination (Baden et al., 2021)). Warning immunity was not considered in this study.

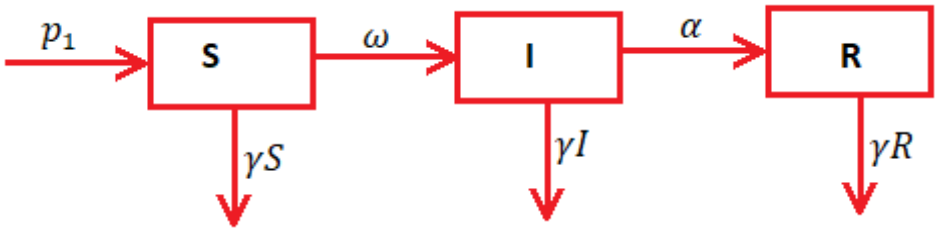
1.4.1 Deterministic model

These models provide the actual prediction of future events without the use of probability. The deterministic occurrence contains all the information required to confidently predict the results.

1.4.2 Stochastic model

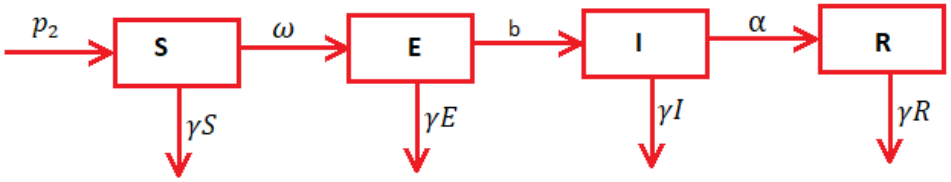
A stochastic model randomly addresses the likelihood of an event happening. Deterministic and stochastic models have various compartments, they include; SI, SIR, SEIR, SEIRS among many

others by considering different compartments. The prediction of these models is determined entirely by their initial conditions set of underlying delay differential equations and input of parameter values. The following are some of the examples of deterministic models used in studying the dynamics of infectious diseases. Susceptible – Infectious – Recovered (SIR) models are models that considers that transmission of a disease moves from susceptible class to infectious class then to recovered class as shown in **Figure 1.1** below.



Susceptible – Infectious – Recovered (SIR)

On the other hand the Susceptible – Exposed – Infectious – Recovered (SEIR) models are models that consider that transfer of a disease moves from susceptible compartment to exposed compartment to infectious compartment to recovered compartment as shown in **Figure 1.2** below.



Susceptible – Exposed – Infectious – Recovered (SEIR)

In this study a deterministic model for COVID-19, which incorporate vaccination and time delay was formulated because when an individual is vaccinated or infected there is a latent period laps before herd immunity is attained or being infectious.

1.5 Basic reproduction number R_0

In epidemiology, the basic reproduction number of an infection is the mean number of new infections arising from a single infectious person introduced into a purely susceptible population during infectious period. The basic reproduction number R_0 is the threshold for many epidemiological models. When $R_0 > 1$ the disease-free equilibrium point is unstable, the infection

will be able to spread in a population and locally stable for $R_0 < 1$, the infection dies out in the long run. To obtain R_0 the dominant eigenvalue of the next generation matrix (NGM) is considered,(Antonini et al., 2020)

1.6 Next-Generation Matrix (NGM)

In the mathematical model, the next generation matrix is used to calculate the fundamental reproductive number from the Jacobian matrix. The Jacobian matrix is derived from compartmental diagrams and is deduced from the first partial derivatives of sets of equations. By decomposing the Jacobian matrix for the infected sub-population into the transmission matrix and the transition matrix that describe compartment mobility and account for the number of new infections, the next-generation matrix can be used to calculate the basic reproductive number R_0. Throughout the computation of the NGM, the transmission and transition matrices are employed. The dominant eigenvalues obtained by picking the largest eigenvalue of the NGM is what gives the R_0, (Diekmann et al.., 2010).Between exposure and infectious there is time lapsed called incubation period in which the infected individual takes to become infectious and transmit the virus to others, this period is considered as time delay in mathematical modeling. The effects of time delay during quarantine and isolation period were not effectively considered and therefore time delay should be incorporated in COVID-19 models.

1.7 Delay Differential Equations (DDEs)

Delay differential equations (DDEs) are a type of differential equation in which the derivative of the unknown function at a certain time is given in terms of the values of the function at previous times. A general form of the time-delay differential equation for $x(t) \in \Re^n$ is

$$\frac{dy}{dx} = f(y(t)), y(t-\tau_1), y(t-\tau_2), \ldots, y(t-\tau_n)) \tag{1.1}$$

Which is solved on $a \leq t \leq b$ with given history and where;

t is the time

τ is the time delay

a and b are values of t

f is a given function that may depend on the current time t, the current state y(t) and the delayed state y(t-τ)

There are two types of delay differential equations; Constant DDEs and Variable DDEs. In this study, constant DDEs were considered. This differential equation takes the form;

$$\frac{dx}{dt} = f(x(t), x(t-\tau), t) \tag{1.2}$$

Where $x \in R^n$ and the quantity τ is a positive constant and t is time , (Thompson et al.., 2007)

1.8 Statement of the Problem

The main problem facing the health sector is prevention, treatment, the increased number of asymptomatic individuals and the time delays due to incubation period. This has increased the overall number of infected individuals and deaths that have been reported over time. The COVID-19 pandemic has been mostly controlled by vaccination (Starr et al., 2021). The effects of time delay on the COVID-19 transmission dynamics should be incorporated. Its worth to note that between the exposure and being infectious there is a time delay usually referred to as incubation period. In this study a Susceptible-Vaccinated-Asymptomatic-Infectious-Recovered (SVAIR) mathematical model that incorporates the effects of vaccination and incubation on COVID-19 transmission dynamics in Kenya is developed.

1.9 Objective of the study

1.9.1 General objective

To model the effects of vaccination and incubation on COVID-19 transmission dynamics using delay differential equations.

1.9.2 Specific Objectives

(i) To formulate a mathematical model that incorporate vaccination and incubation period on COVID-19 transmission dynamics

(ii) To determine the equilibrium points of the SVAIR mathematical model developed and their stabilities.

(iii) To perform sensitivity analysis of the SVAIR model to the parameters of the model.

(iv) To carry out numerical simulation of the SVAIR model developed using MATLAB.

1.10 Justification of the study

The deterministic SVAIR mathematical model developed in this study is important in addressing the transmission dynamics of COVID-19. The study took into consideration the effects of vaccination and time delay on the basic reproduction number. During the early spread of COVID-19, non-pharmaceutical measures were not effective, therefore vaccination was introduced as the best alternative measure to curb its spread but time delay was not considered in the previous mathematical models.

1.11 Significance of the study

Infectious diseases like COVID-19 caused of sickness and death globally. This study is of great significance in the following areas: health sector and economy of the country. The model access the likely effects of the mitigation measures being applied and determine the expected epidemic trajectories if the vaccination program may not be fully implemented. Non-pharmaceutical measures such as face masking, keeping social distance, isolation and quarantine of suspected cases, have not been effective methods in curbing the spread of COVID 19. This model identify the immediate response on the best interventions that can be put in place to ensure normal operations even during the time of pandemic. The impact of COVID-19 pandemic on both global and local economy has been greatly felt. The model will inform the formulation of policies that will ease the impact of COVID-19 in the country.

CHAPTER TWO

LITERATURE REVIEW

2.1 Introduction

Many epidemiologists have sought to understand the dynamics of infectious diseases using mathematical models. In both developed and less developed countries, the COVID-19 pandemic has recently caused loss of lives. This chapter will provide an overview on the previous related studies which have been done by different scholars on COVID-19 dynamics. Finally this chapter also identify the knowledge gaps that exist on the previous studies and thus the need to carry out this study with an aim of bridging the existing gap.

2.2 Review of Related Literature

2.2.1 Mathematical models

Starting with the work of Daniel Bernoulli in 1760, the development of mathematical models has been critical in our understanding of the dynamics of infectious diseases. With the work of Kermack and McKendrick(1927), mathematical models have since been used to provide framework for understanding the dynamics of infectious diseases. COVID-19 being an infectious disease, its transmission dynamics models are flourishing and exist in many literature.

Early work on mathematical modeling was a study by Daniel Bernoulli on the deaths from smallpox that published in 1766.The use of mathematical models to model infectious disease gained traction in early 20th century beginning with Hamer's model on the recurrence of measles epidemic in 1906. This was followed by a study on the transmission dynamics of malaria between mosquitoes and humans, (Rose 1911). McKendrick et al., 1927 extended malaria model and studied the conditions for the spread of the disease and control mechanism.

Different types of epidemics that have since hit the World in the recent past include SARS-CoV in 2002-2003, Middle East respiratory syndrome coronavirus (MERS-CoV) in 2012, Ebola in 1976 and 2013, H1N1 in 2009 and finally SARS-Cov-2 (COVID-19) in December 2019 (Alzubadi et al., 2023). Mathematical analysis of the spread and control of the novel coronavirus (COVID-19) in China informed the WHO (World Health Organization) of the outbreak. Later, on January 20, 2020, scientists from China's National Health Commission expressed its potential to spread from one human to another upon the confirmation of two cases in Guangdong upon visiting Wuhan. Later, the Wuhan authorities announced new measures for the control and prevention of this epidemic.

The novel coronavirus identified as Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-COV-2) is a strain of Coronavirus that causes Coronavirus Disease 2019. This disease was named by the World Health Organization (WHO) as Coronavirus Disease 2019 (COVID-19) (Sonani et al.,2020). WHO declared COVID-19 a pandemic in March 2020. Since March 2020, the pandemic in China has been substantially under control, yet, it continues to be a significant threat to global health and other sectors in the world. The effectiveness of China's healthcare system and awareness campaigns, helped the country to contained the COVID-19 pandemic and were one of the main factors contributing to its success in the fight against the disease, (Musa et al., 2022).

The most populated country in Africa, Nigeria, was affected by COVID-19 and other infectious and non-infectious diseases. Even in a normal circumstance, Nigeria healthcare system falls short of providing its people with the fundamental and necessary healthcare they required. As of January, 2020, the Nigeria Center for Disease Control (NCDC) had tallied 122,996 active cases and 1,507 fatalities at the time COVID-19 infection was not then treatable with vaccination or antiviral medication. As a result, a variety of non-pharmaceutical interventions including social isolation, face-masking, contact tracing, community-wide lock-downs, quarantining of suspected cases and the dissemination of health information about COVID-19 were used to control the spread of COVID-19 at that time.

Musa et al., (2022), investigated the infection fatality rate and infection rate of COVID-19

in Nigeria using a susceptible-exposed-infectious-recovered (SEIR) based model with a time-varying transmission rate controlled by systems of delayed differential equations (DDEs). They discovered that the awareness campaigns, in particular the use of non-pharmaceutical measures in mitigating the spread of COVID-19 were effective during the earliest period of infection. This study fails to incorporate effects of vaccination in transmission dynamics.

Jayaweera et al., (2020), in their studies of the mathematical modeling of COVID-19 with diffusion and control using systems of ordinary differential equations (ODEs), found out that it was important to understand the dynamics of the infection in the period of early spread. They also pointed out the effectiveness of control measures to established whether sustained transmission was likely to occur in new locations. The model was used to examine the COVID-19 spread in Wuhan, China from both inside and outside the city. The outbreak variations in Wuhan between January and February 2020 were computed. These computations were used to determine whether the disease carriers could be brought outside Wuhan city in China and could lead to a sustained human-to-human spread.

The SEIR model formulated used data on cases of COVID-19 in China and cases abroad that started in Wuhan city to assess how the disease transmission varied over time in January and February 2020. The probability that newly found cases could spark an outbreak in additional locations was computed. The mean daily basic reproduction number R_0 for Wuhan was also computed (the expected number of secondary cases following the introduction of one infected individual into a fully susceptible population). According to the study, a disease must have $R_0 > 1$ in order to spread among a host population because R_0 has a threshold value of this magnitude. The basic reproduction rate dropped from 2.45 one week prior to the introduction of travel restrictions on January 23, 2020, to 1.05 one week thereafter.

As of August 2020, there were 19,187,943 confirmed cases of COVID-19 worldwide, including 716,075 fatalities, according to the report by (Sonani et al.,2020). In India, there were 2,088,611 confirmed cases and 42,518 fatalities; in the United States, there were 4,836,930 confirmed cases and 158,606 fatalities; in Spain, there were 314,362 confirmed cases and 28,503 fatalities and in South Africa, there were 545,476 confirmed cases and 9909 fatalities. The majority of

near-the-beginning holder's organisms were found in Wuhan, China in December 2019, while the majority of worldwide transmitted overseas holders were found to have traveled to and from Wuhan (Cohen et al., 2020).

A time delay dynamic model with a set of nonlinear DDEs were used to model and analyze COVID-19 transmission by (Cong Y et al., 2020. The study compared the global spread of the new coronary pneumonia epidemic at the beginning of 2020 to COVID-19 in order to mimic COVID-19 and assess the escalation of the epidemic in Beijing and Wuhan cities of China. They found out that during a short time, Wuhan city experienced a sharp rise in the number of infections.The analysis discovered that the pandemic was firmly under control and that the known cases had cleared up after several months of isolation and quarantine. After several months of reporting no new local cases, a new round of coronary pneumonia broke out in Beijing city on June 10, 2020. Several national and local governments ignored WHO recommendations but revived the economy and eased restrictions before the pandemic situation had been successfully controlled. Because the relationship between epidemic prevention and control and the reviving of the economy is difficult to balance, the model predicted high possibilities of future outbreak. It may be crucial to fully deliver antiviral medications and vaccines to Kenya's population in order to curb the spread of the pandemic and control it. In the absence of vaccines, early detection, isolation and treatment were then the most efficient intervention measures (Xu et al., 2021). The study only concentrated on non-pharmaceutical approaches for containing and lessening the pandemic impact in the absence of reliable vaccines or antivirals. These mitigation measures were temporary, since over time, the general populace has a tendency to loosen them, which would accelerate the spread of COVID-19. This study did not consider how the COVID-19 incubation period affected the isolated and confined patients.

Wangari et al., (2021), studied the mathematical modeling of COVID-19 transmission in Kenya, a model with a re-infection transmission mechanism. They found out that a large pool of infectious people who were asymptomatic would eventually increase symptomatic people with mild and severe symptoms, which may eventually increase the overall number of reported deaths. The study suggested that re-infection of recovered people who had lost their protective immunity caused this situation. The model uses deterministic ordinary differential equations (ODEs) for

computation of basic reproduction number.

Using face masks significantly lowered COVID-19 prevalence when compared to merely maintaining social distance, according to the model used to evaluate the efficacy of multiple non-pharmaceutical strategies for lowering COVID-19. Furthermore, it showed that the COVID-19 surge could be greatly decreased by raising the rate at which asymptomatic patients were discovered using contact tracing, testing and isolation. The peak COVID-19 infection was greatly delayed with only the use of face masks and keeping social distance. This has received substantial support from numerical simulations, which showed that COVID-19 transmission could be reduced more successfully when parameters that account for routine face masking and keeping social distance were combined.

Unfortunately, it's model did not take into account the incubation period during asymptomatic period. It did not state how asymptomatic cases would affect Kenya's low rate of vaccination program, including whether or not the program would successfully stop the spread of COVID-19. In addition, whether the people who received the booster shots would be fully protected. The study gives predictions about the possibility of disease burden, morbidity and death. It called for further research on the problems presented by asymptomatic patients in connection with reinfection.

Diagne et al., (2021), mathematically modeled the transmission mechanism of COVID-19. They featured the fundamental dynamics of the disease as well as two critical therapeutic approaches, that is, vaccination of those at risk and treatment of those already sick. On whether the effective reproduction number R_0 is less than or greater than a unit, they found out that both the endemic equilibrium and the disease-free equilibrium were globally stable. The nonlinear system of equations utilized in this study to model the transmission dynamics of COVID-19 was developed using ordinary differential equations (ODEs).

The model was fitted using daily Senegal cumulative data at the start of the COVID-19 pandemic and a basic reproduction number $R_0 = 1.31$. The simulation outcomes demonstrated that further efforts such as non-pharmaceutical public health programs were to continue being applied when $R_0 > 1$, regardless of how well the COVID-19 vaccine and therapy prevent the disease from

spreading. The study did not consider the time delay in the transmission dynamics of COVID-19. The study formulated SVEIAHR mathematical model where the impact of model parameters on the spread of disease were examined through sensitivity analysis.

Jayaweera et al., (2020), in their study emphasized that forced social isolation and entire or partial lockdowns were effective ways to stop the virus from being transmitted. Their model showed that there was a significant time delay when people were subjected to mandatory social isolation. The crucial time interval between infection containment and transmission was two weeks. The mathematical model formulated uses a system of ordinary differential equations (ODE).

Aldila et al., (2020), in their study, considered the undiscovered asymptomatic individuals who actively contributed to the transmission of COVID-19 despite having no symptoms. The study also demonstrated that the inadequate healthcare resources contributed to the spread of COVID-19 virus. SVEIAHR mathematical model were developed, sensitivity analysis was used to determine how the model parameters affected the spread of the disease. The study found out that although it was necessary to reduced the basic reproductive number below one in order to stop the spread of infection, it was not sufficient because several additional intervention measures were required.

Lemaitre et al., (2020), in their study, the effects of non-pharmaceutical strategies, according to their studies, there was a significant amount of virus transmission through contaminated surfaces, depending on the nature of the virus. This study expressed concern about the fact that public awareness of COVID-19 was one of the main challenges in curbing the spread of COVID-19. Many people choose not to follow the safety precautions already in place including wearing face masks, regularly sanitizing, avoiding crowded areas and maintaining social distance. The mathematical model did not considered vaccination as the mitigation measure against the spread of COVID-19.

Shattock et al., (2022), studied the mathematical model based on ordinary differential equations (ODEs). Their study subdivided the total population into; susceptible, vaccinated, infectious, asymptomatic and recovered (SVIAR). Several simplifying assumptions were made in this

modeled scenarios. At the time of the study, the extent to which immunity wanes over time was not well understood, thus it assumed that no loss of naturally acquired or vaccine-induced immunity for the short period of simulation. It was important to monitor waning immunity for a period of 6 to 12-months after recovery or being vaccinated, as well as vaccine efficacy on novel variants. Vaccinating people under 18 years of age was not in progress since at that time, vaccine safety in children had not yet tested and delaying of second vaccine doses to increase coverage of first doses or because of dosing shortages were not explored, nor were third dose booster vaccines for those over 65 years of age were not approved at the time of their study. They did not consider potential changes in behavior among people once they were vaccinated.Their study showed that vaccinations contributed more to reduction in the transmission rate of COVID-19 compared to using NPIs strategy only,but did not state how the asymptomatic individuals affects the transmission dynamic of COVID-19 and the time delay.

2.3 Identification of the Knowledge Gap

The above studies did not consider the time delay between asymptomatic and infectious time which requires the use of delay differential equations (DDEs) in modeling the transmission dynamics of COVID-19.Their numerical simulations indicated that COVID-19 could be controlled in the community with the implementation of nonpharmaceutical public health interventions such as face masks, hand washing and social distancing should continue to be encouraged. Also in presence of reinfection, if Kenya had increased the detection rate to those who were asymptomatic through contact tracing and isolating the positive cases, COVID-19 surge could have be curbed. Detection of asymptomatic cases during the period when Kenya was not vaccinating the general public on a large scale was effective in curbing COVID-19. These studies did not exhaustively considered the impact of both therapeutic and nontherapeutic measures on the dynamics of COVID-19. This study formulated a SVAIR mathematical model incorporating time delay for the transmission dynamics of COVID-19 in Kenya in which incubation period is considered.

CHAPTER THREE

RESEARCH METHODOLOGY

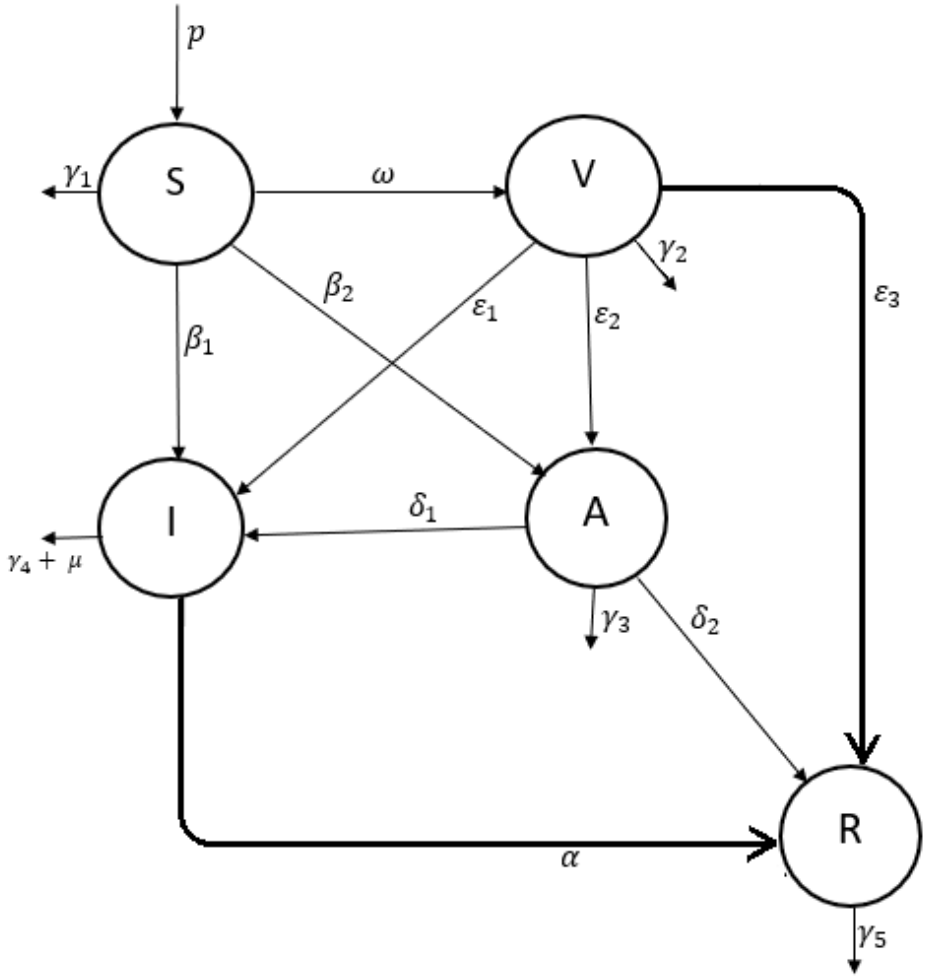
3.1 Introduction

This chapter outlines the methodology used in the thesis. First, the SVAIR model for COVID-19 is presented, model assumptions and equations are given. The model preliminary analysis, computation of the basic reproduction number, equilibrium points and stability analysis are provided. Finally, sensitivity analysis of the basic reproduction number on parameters is examined.

3.2 SVAIR Model for COVID-19

In order to formulate the model for the COVID-19 we describe the total population of humans by N(t) where the population was subdivided into five compartments for the SVAIR model namely: The susceptible (S) describes healthy people who have not yet been exposed to the COVID-19 virus. The vaccinated (V) are the susceptible individuals who have received COVID-19 vaccine, the infected asymptomatic (A), the symptomatic infected individuals (I) and the recovered (R) from COVID-19. The deterministic model that describes the transmission dynamics of COVID-19 in the community is shown by the following nonlinear delay differential equations (3.2):

SVAIR Model Formulation



SVAIR Model Flow Diagram

3.3 Model Assumptions

The following are the assumptions useful in the derivation of the model.

(i) There is homogeneous interaction of individuals in the population, which means that every individual who is uninfected has an equal chance of being infected provided he or she comes in contact with the infected individual.

(ii) The recruitment rate into the susceptible class is constant $p > 0$.

(iii) There is disease induced death and death due to natural causes.

(iv) Dealing with closed population, no immigration nor emigration.

(v) Permanent immunity upon vaccination is attainable.

(vi) A fraction of asymptomatic individuals goes to infectious class.

(vii) There is time lapse between exposure and being symptomatic.

(viii) The rate of mortality is higher in infectious class followed by asymptomatic then recovered group and least in the vaccinated and susceptible class, $\gamma_1 < \gamma_2 < \gamma_5 < \gamma_3 < \gamma_4$

(ix) All the new borns are assumed to be susceptible.

Delay differential equations are used to develop the model because the incubation period causes the transition rates between compartments to be mathematically stated as derivatives. The deterministic nature of the illness transition and the differentiability of the population size in a compartment with regard to time are assumed when creating such models. Thus, a compartment's population changes can be computed based solely on the model's development history. The COVID-19 mathematical model is based on SVAIR transmission model in which the total population size N(t) is given by:

$$N(t) = S(t) + V(t) + A(t) + I(t) + R(t) \tag{3.1}$$

All newborns are assumed to be susceptible. The natural recruitment and the natural death are denoted by P and γ respectively. Susceptible individuals are vaccinated at constant rate ω. The parameters β_1 and β_2 are the infecting rates of infectious and asymptotic individuals respectively. The rate at which the infectious and asymptomatic individuals recovered and acquire temporary immunity is represented by α and δ_2.

The vaccinated individuals need a period of time to develop the immunity against the virus represented by $\frac{1}{\varepsilon_3}$ and $\frac{1}{\delta_1}$ represent the incubation period for asymptomatic individuals takes to develop symptoms. The virus may infect the vaccinated individuals but at a lower rate than the susceptible individuals who are not vaccinated. Based on the above assumptions and the model parameters, the transmission dynamics of COVID-19 is modeled by the following system of delay differential equations. The term with ε_3 appears in the $\frac{dV(t)}{dt}$ and $\frac{dR(t)}{dt}$ equations is epidemiologically logical ,(Ismail et al., 2024)

3.4 Model Equations

using the model assumptions above, the SVAIR model equations are:

$$\left.\begin{aligned}
\frac{dS(t)}{dt} &= p - \omega S - \beta_1 S I_\tau - \beta_2 S I_\tau - \gamma_1 S \\
\frac{dV(t)}{dt} &= \omega S - \varepsilon_1 V_\tau I_\tau - \varepsilon_2 V_\tau I_\tau - \varepsilon_3 V_\tau - \gamma_2 V \\
\frac{dA(t)}{dt} &= \beta_2 S I_\tau + \varepsilon_2 V_\tau I_\tau - \delta_1 A_\tau - \delta_2 A_\tau - \gamma_3 A \\
\frac{dI(t)}{dt} &= \beta_1 S I_\tau + \varepsilon_1 V_\tau I_\tau + \delta_1 A_\tau - \alpha I_\tau - (\gamma_4 + \mu) I \\
\frac{dR(t)}{dt} &= \alpha I_\tau + \delta_2 A_\tau + \varepsilon_3 V_\tau - \gamma_5 R
\end{aligned}\right\} \tag{3.2}$$

where S(0) $\geq$ 0, V(0) $\geq$ 0, A(0) $\geq$ 0, I(0) $\geq$ 0 and R(0) $\geq$ 0 are the given initial states. Where p represents new birth rate into susceptible human population, β_2 represents the transmission coefficient from susceptible individuals to asymptomatic infected cases, ω represents the transmission coefficient from susceptible individuals to vaccinated class γ represents the natural death rate in all compartments. δ_1 represents the rate of transmission of asymptomatic infected cases to infected individuals with symptoms. α is the transmission coefficient of the infectious cases to the recovered cases. δ_2 represents the rate of transmission of asymptomatic infected cases with mild symptoms to the recovered cases due the strong immunity of these individuals. μ respectively represent the death rate of the infected individuals. ε_3 represent the rate of progression from vaccinated class to recovery without being infected.

3.4.1 Model Preliminary Analysis

3.4.2 Positivity and Boundedness of the model

Positivity and boundedness of the model is shown before doing result analysis for equation 3.2 to be epidemiologically meaningful. Thus, we show that all state variables of the model (3.2) are nonnegative for all time $t > 0$ and bounded and the existence of solutions are underscored by these properties.

Let C = C [-τ,0], $\Re^5$ be a Banach space of continuous functions mapping the interval [-τ,0] into $\Re^5$ with the topology of uniform convergence. By fundamental theory of differential equations

it can be shown that there exists a unique solution (S(t),V(t), A(t), I(t), R(t)) of the system 3.2 with initial data.

$$(S(0) > 0, V(0) > 0, A(0) > 0, I(0) > 0, R(0) > 0 \in C) \tag{3.3}$$

In addition, we assume that the initial data for the system 3.2 satisfies;

$$S_0(p) \geq 0, V_0(p) \geq 0, A_0(p) \geq 0, I_0(p) \geq 0, R_0(p) \geq 0, \in [-\tau, 0] \tag{3.4}$$

The following theorem establishes the positivity and boundedness of the solution with initial functions satisfying (3.3) and (3.4)

Theorem 3.1

Let SVAIR be the solution of the system 3.2 satisfying the condition 3.3 and 3.4. then S,V,A,I and R are all positive and bounded for all $t > 0$ at which the solution exist.

Proof

From the system 3.2 we have

$$S(t) = S(0)e^{-\int_0^t(\omega+\beta_1 I_\tau+\beta_2 I_\tau+\gamma_1)d\theta} + \int_0^t (p+(\omega+\beta_1 I_\tau+\beta_2 I_\tau+\gamma_1))S(\varphi)e^{\int_0^t(\omega+\beta_1 I_\tau+\beta_2 I_\tau+\gamma_1)d\theta}d\varphi \tag{3.5}$$

From 3.5 it is clearly seen that $S(t) > 0$ hence positive. Similarly, it can be shown that

$$V(t) > 0, A(t) > 0, I(t) > 0, R(t) > 0 \tag{3.6}$$

Using equation 3.3 and 3.4, positivity follows immediately from the above integral forms. For boundedness we define

$$N(t) = S(t) + V(t) + A(t) + I(t) + R(t) \tag{3.7}$$

$$\frac{dN(t)}{dt} = \frac{d(S(t)+V(t)+A(t)+I(t)+R(t))}{dt} \tag{3.8}$$

$$\frac{dN(t)}{dt} = p - \gamma(S(t)+V(t)+A(t)+I(t)+R(t)) \tag{3.9}$$

Thus

$$\frac{(dN(t))}{dt} \leq p - \Sigma \tag{3.10}$$

Where

$$\Sigma = \gamma S + \gamma V + \gamma A + \gamma I + \gamma R \tag{3.11}$$

This implies that N(t) is bounded and so are S(t)+V(t)+A(t)+I(t)+R(t).

This completes the proof of theorem 3.1

3.5 Computation of Basic Reproduction Number R_0

Infections produced by a single infectious individual during the course of their infectious period are known as the basic reproduction number. R_0 is the threshold value for infectious disease models because when $R_0 < 1$, the illness dies off, indicating that the disease free equilibrium state (DFE) is stable, when $R_0 > 1$, the DFE is unstable, meaning that the disease spread throughout the population.

In this study we adopted the next generation matrix (NGM) approach in computation of R_0 such that $R_0 = \rho FV^{-1}$ where ρ is the spectral radius for NGM while F is the matrix of new infection by the disease and V is the matrix of transfer of infection in and out of the compartment by other means. The dominant eigenvalue of the NGM is considered as R_0. In the equation 3.2, there are two infectious classes, A and I hence at disease free equilibrium, the matrix of new infection is given by matrix F and V.

Matrix F_i is obtained by considering the rate of new infections entering the compartment i is given by;

$$F_i = \begin{bmatrix} \beta_2 S I_\tau + \varepsilon_2 V_\tau I_\tau \\ \beta_1 S I_\tau + \varepsilon_1 V_\tau I_\tau + \delta_1 A_\tau \end{bmatrix} \tag{3.12}$$

The matrix that represents the rate of transfer into and out of compartment by other means at DFE is given by;

$$V_i = \begin{bmatrix} \delta_1 A_\tau + \delta_2 A_\tau + \gamma_3 A \\ \alpha I_\tau + (\gamma_4 + \mu) I \end{bmatrix} \tag{3.13}$$

We then differentiate F_i partially with respect to the state variables to obtain a 2×2 matrix

$$F_i = \begin{bmatrix} 0 & (\varepsilon_2 V_\tau + \beta_2 S)e^{-\lambda_\tau} \\ \delta_1 e^{-\lambda_\tau} & (\varepsilon_1 V_\tau + \beta_1 S)e^{-\lambda_\tau} \end{bmatrix} \tag{3.14}$$

Similarly, differentiating matrix V_i partially with respect to the state variables, we obtain a 2×2 matrix,

$$V_i = \begin{bmatrix} (\delta_1 + \delta_2)e^{-\lambda_\tau} + \gamma_3 & 0 \\ 0 & \alpha e^{-\lambda_\tau} + (\gamma_4 + \mu) \end{bmatrix} \tag{3.15}$$

The inverse of V is computed as

$$V^{-1} = \begin{bmatrix} \frac{1}{(\delta_1+\delta_2)e^{-\lambda_\tau}+\gamma_3} & 0 \\ 0 & \frac{1}{\alpha e^{-\lambda_\tau}+(\gamma_4+\mu)} \end{bmatrix} \tag{3.16}$$

Using the next generation matrix (NGM) to determine the basic reproduction number R_0 , we have

$$NGM = FV^{-1} = \begin{bmatrix} 0 & \frac{(\varepsilon_2 V_\tau+\beta_2 S)e^{-\lambda_\tau}}{\alpha e^{-\lambda_\tau}+(\gamma_4+\mu)} \\ \frac{\delta_1 e^{-\lambda_\tau}}{(\delta_1+\delta_2)e^{-\lambda_\tau}+\gamma_3} & \frac{(\varepsilon_1 V_\tau+\beta_1 S)e^{-\lambda_\tau}}{\alpha e^{-\lambda_\tau}+(\gamma_4+\mu)} \end{bmatrix} \tag{3.17}$$

To determine the eigenvalues, we have the characteristic matrix of FV^{-1} given by;

$$\begin{vmatrix} -\lambda & \frac{(\varepsilon_2 V_\tau+\beta_2 S)e^{-\lambda_\tau}}{\alpha e^{-\lambda_\tau}+(\gamma_4+\mu)} \\ \frac{\delta_1 e^{-\lambda_\tau}}{(\delta_1+\delta_2)e^{-\lambda_\tau}+\gamma_3} & \frac{(\varepsilon_1 V_\tau+\beta_1 S)e^{-\lambda_\tau}}{\alpha e^{-\lambda_\tau}+(\gamma_4+\mu)} - \lambda \end{vmatrix} = 0 \tag{3.18}$$

Solving for the eigenvalues we obtain;

$$\lambda_2 = \frac{(\varepsilon_1 V_\tau + \beta_1 S)e^{-\lambda_\tau}}{\alpha e^{-\lambda_\tau} + (\gamma_4 + \mu)} \tag{3.19}$$

The dominant eigenvalue is;

$$\lambda_2 = \frac{(\varepsilon_1 V_\tau + \beta_1 S)e^{-\lambda_\tau}}{\alpha e^{-\lambda_\tau} + (\gamma_4 + \mu)} \tag{3.20}$$

The R_0 which is the dominant eigenvalue is given by;

$$Therefore, R_0 = \frac{(\varepsilon_1 V_\tau + \beta_1 S)e^{-\lambda_\tau}}{\alpha e^{-\lambda_\tau} + (\gamma_4 + \mu)} \tag{3.21}$$

3.6 Disease Free Equilibrium Point and its Stabilities

We investigate the stability of equation 3.2 in order to have a good understanding of the dynamics of COVID-19. The Disease Free Equilibrium Point (DFE) and the Endemic Equilibrium Point (EEP) are the two equilibria points taken into account in mathematical epidemiology. In this study, we examined the stabilities of the disease-free equilibrium found in equation 3.2.

An equilibrium point is determined by setting the right-hand side of each equation 3.2 to zero, then solving each equation for the constant solution. Disease free equilibrium point is a feasible region in the solution of equation 3.2 in the absence of viral infection.

For the model equation 3.2, DFE is set of S(0),V(0),A(0),I(0),R(0)=$(\frac{p}{\omega+\beta_1 I_\tau+\beta_2 I_\tau+\gamma_1}$,0,0,0,0) obtained by simple algebraic computation in the absence of COVID-19 viruses.

3.6.1 Local Stability of Disease Free Equilibrium

The model's variable state when there is no disease is known as the disease-free equilibrium. The eigenvalues of the Jacobian matrix formed during DFE, where the reproduction number is less than one $R_0 < 1$, can be used to assess the stability. Deduced from the first partial derivatives of equation 3.2 and derived from compartmental classes, the Jacobian matrix is provided by;

$$\left.\begin{aligned} &p-\omega S-\beta_1 SI_\tau-\beta_2 SI_\tau-\gamma_1 S=0\\ &\omega S-\varepsilon_1 V_\tau I_\tau-\varepsilon_2 V_\tau I_\tau-\varepsilon_3 V_\tau I_\tau-\gamma_2 V=0\\ &\beta_2 SI_\tau+\varepsilon_2 V_\tau I_\tau-\delta_1 A_\tau-\delta_2 A_\tau-\gamma_3 A=0\\ &\beta_1 SI_\tau+\varepsilon_1 V_\tau I_\tau+\delta_1 A_\tau-\alpha I_\tau-(\gamma_4+\mu)I=0\\ &\alpha I_\tau+\delta_2 A_\tau+\varepsilon_3 V_\tau I_\tau-\gamma_5 R=0 \end{aligned}\right\} \tag{3.22}$$

The linearization matrix is given by;

$$\begin{bmatrix} -(\omega+\beta_1 I_\tau+\beta_2 I_\tau+\gamma_1) & 0 & 0 & 0 & 0 \\ \omega & -(\varepsilon_1+\varepsilon_2+\varepsilon_3)I_\tau e^{-\lambda_\tau}-\gamma_2 & 0 & -(\varepsilon_1+\varepsilon_2+\varepsilon_3)v_\tau e^{-\lambda_\tau} & 0 \\ \beta_2 I_\tau & \varepsilon_2 I_\tau e^{-\lambda_\tau} & -(\delta_1+\delta_2)e^{-\lambda_\tau}-\gamma_3 & (\beta_2 S+\varepsilon_2 V_\tau)e^{-\lambda_\tau} & 0 \\ \beta_1 I_\tau & \varepsilon_1 I_\tau e^{-\lambda_\tau} & \delta_1 e^{-\lambda_\tau} & Q & 0 \\ 0 & \varepsilon_3 I_\tau e^{-\lambda_\tau} & \delta_2 e^{-\lambda_\tau} & (\alpha+\varepsilon_3 v_\tau)e^{-\lambda_\tau} & -\gamma_5 \end{bmatrix} \tag{3.23}$$

Where Q = -$(\alpha-\beta_1 s-\varepsilon_1 v_\tau)e^{-\lambda_\tau}-(\gamma_4+\mu)$

The equation 3.2 is locally asymptotically stable if all the eigenvalues of the linearization matrix of the equation 3.2 are negative.

$$\begin{vmatrix} A-\lambda & 0 & 0 & 0 & 0 \\ \omega & B-\lambda & 0 & -(\varepsilon_1+\varepsilon_2+\varepsilon_3)v_\tau e^{-\lambda_\tau} & 0 \\ \beta_2 I_\tau & \varepsilon_2 I_\tau e^{-\lambda_\tau} & C-\lambda & (\beta_2 S+\varepsilon_2 V_\tau)e^{-\lambda_\tau} & 0 \\ \beta_1 I_\tau & \varepsilon_1 I_\tau e^{-\lambda_\tau} & \delta_1 e^{-\lambda_\tau} & D-\lambda & 0 \\ 0 & \varepsilon_3 I_\tau e^{-\lambda_\tau} & \delta_2 e^{-\lambda_\tau} & (\alpha+\varepsilon_3 v_\tau)e^{-\lambda_\tau} & E-\lambda \end{vmatrix} \tag{3.24}$$

Where;

$$\left.\begin{aligned} A &= -(\omega+\beta_1 I_\tau+\beta_2 I_\tau+\gamma_1) \\ B &= -(\varepsilon_1+\varepsilon_2+\varepsilon_3)I_\tau e^{-\lambda_\tau}-\gamma_2 \\ C &= -(\delta_1+\delta_2)e^{-\lambda_\tau}-\gamma_3 \\ D &= -(\alpha-\beta_1 s-\varepsilon_1 v_\tau)e^{-\lambda_\tau}-(\gamma_4+\mu) \\ E &= -\gamma_5 \end{aligned}\right\} \tag{3.25}$$

The eigenvalues are;

$$\left.\begin{aligned}
\lambda_{**1} &= -(\omega + \beta_1 I_\tau + \beta_2 I_\tau + \gamma_1) \\
\lambda_{**2} &= -(\varepsilon_1 + \varepsilon_2 + \varepsilon_3) I_\tau e^{-\lambda_\tau} - \gamma_2 \\
\lambda_{**3} &= -(\delta_1 + \delta_2) e^{-\lambda_\tau} - \gamma_3 \\
\lambda_{**4} &= -(\alpha - \beta_1 s - \varepsilon_1 v_\tau) e^{-\lambda_\tau} - (\gamma_4 + \mu) \\
\lambda_{**5} &= -\gamma_5
\end{aligned}\right\} \tag{3.26}$$

For the equation 3.2, it is clear that the dominant eigenvalue is;

$$\lambda_{**4} = -(\alpha - \beta_1 s - \varepsilon_1 v_\tau) e^{-\lambda_\tau} - (\gamma_4 + \mu) \tag{3.27}$$

Theorem 3.2

Disease free equilibrium is stable whenever $R_0 < 1$, otherwise it's unstable.

Proof

λ_{**4} should be negative. This can only be negative if,

$$-(\alpha - \beta_1 S - \varepsilon_1 V_\tau) e^{-\lambda_\tau} - (\gamma_4 + \mu) = 0$$

$$(\beta_1 S + \varepsilon_1 v_\tau) e^{-\lambda_\tau} < \alpha e^{-\lambda_\tau} + (\gamma_4 + \mu) \tag{3.28}$$

This implies that;

$$\frac{(\beta_1 S + \varepsilon_1 v_\tau) e^{-\lambda_\tau}}{\alpha e^{-\lambda_\tau} + (\gamma_4 + \mu)} < 1 \tag{3.29}$$

Therefore $R_0 < 1$ is obtained for DFE to be stable.

3.6.2 Global stability analysis of disease free equilibrium points

Global stability of the equation 3.2 was done by constructing Lyapunov functions for disease free equilibrium point.

Theorem 3.3

If $R_0 < 1$ then the disease free equilibrium of the equation 3.2 is globally asymptotically stable, otherwise it is unstable for $R_0 > 1$.

Proof

Consider the following Lyapunov functions;

$$V(S,V,A,I,R) = (S-S_0)^2 + (V-0)^2 + (A-0)^2 + (I-0)^2 + (R-0)^2 \tag{3.30}$$

$$= (S-S_0)^2 + (V)^2 + (A)^2 + (I)^2 + (R)^2 \tag{3.31}$$

For the equation 3.2, we have

$$p - \omega S - \beta_1 S I_\tau - \beta_2 S I_\tau - \gamma_1 S = 0 \tag{3.32}$$

This implies;

$$S = \frac{p}{\omega + \beta_1 I_\tau + \beta_2 I_\tau + \gamma_1} \tag{3.33}$$

Implying that $S > 0$ hence positive.

It follows that

$$V = V_n = A = I = R > 0 \tag{3.34}$$

The derivative of the equation 3.2 with respect to time is given as;

$$\frac{dV}{dt}(S,V,A,I,R) = 2(S-S_0)\frac{dS}{dt} + 2V\frac{dV}{dt} + 2A\frac{dA}{dt} + 2I\frac{dI}{dt} + 2R\frac{dR}{dt} \tag{3.35}$$

At disease free equilibrium point, $V = V_n = A = I = R = 0$, therefore equation (3.35) above becomes;

$$V^1 = 2(S-S_0) \tag{3.36}$$

For global stability, $S < S_0$ so that $V^1 < 0$.

If $S \leq S_0$ then $V^1 \leq 0$ implying that $R_0 \leq 1$, implying the disease free equilibrium point is globally stable.

3.7 The Endemic Equilibrium Point

In this section, we study the conditions for the existence of endemic equilibrium point for equation 3.2. The analysis of the equilibrium point is done at the critical point of equation 3.2 which exist

when $S > 0, V > 0, A > 0, I > 0, R > 0$. The zero solution of the nonlinear equation 3.2 at EEP is asymptotically stable if and only if the zero solutions of the linear system obtain from equation 3.2 is asymptotically stable. Therefore, the asymptotic stability of equation 3.2 at EEP is established by examining the signs of the eigenvalues of the zero solutions of the linearized system.

$$\left.\begin{aligned} & p - \omega S - \beta_1 S I_\tau - \beta_2 S I_\tau - \gamma_1 S = 0 \\ & \omega S - \varepsilon_1 V_\tau I_\tau - \varepsilon_2 V_\tau I_\tau - \varepsilon_3 V_\tau I_\tau - \gamma_2 V = 0 \\ & \beta_2 S I_\tau + \varepsilon_2 V_\tau I_\tau - \delta_1 A_\tau - \delta_2 A_\tau - \gamma_3 A = 0 \\ & \beta_1 S I_\tau + \varepsilon_1 V_\tau I_\tau + \delta_1 A_\tau - \alpha I_\tau - (\gamma_4 + \mu) I = 0 \\ & \alpha I_\tau + \delta_2 A_\tau + \varepsilon_3 V_\tau I_\tau - \gamma_5 R = 0 \end{aligned}\right\} \tag{3.37}$$

Defining the endemic equilibrium point as;

$E = S^*, V^*, A^*, I^*, R^*$ which satisfies system 3.37. Then with simple algebraic calculations we obtain the values of endemic equilibrium points as;

$$\left.\begin{aligned} S^* &= \frac{p}{\omega + \beta_1 I_\tau + \beta_2 I_\tau + \gamma_1} \\ V^* &= \frac{\frac{\omega p}{\omega + \beta_1 I_\tau + \beta_2 I_\tau + \gamma_1} - (\varepsilon_1 + \varepsilon_2 + \varepsilon_3) V_\tau I_\tau}{\gamma_2} \\ A^* &= \frac{\frac{\beta_2 I_\tau p}{\omega + \beta_1 I_\tau + \beta_2 I_\tau + \gamma_1} + \varepsilon_2 V_\tau I_\tau - (\delta_1 + \delta_2) A_\tau}{\gamma_3} \\ I^* &= \frac{\frac{\beta_1 I_\tau P}{\omega + \beta_1 I_\tau + \beta_2 I_\tau + \gamma_1} + \varepsilon_1 V_\tau I_\tau + \delta_1 A_\tau - \alpha I_\tau}{(\gamma_4 + \mu)} \\ R^* &= \frac{\alpha I_\tau + \delta_2 A_\tau + \varepsilon_3 V_\tau I_\tau}{\gamma_5} \end{aligned}\right\} \tag{3.38}$$

3.7.1 Stability of endemic equilibrium point

The EEP is guaranteed to be stable, when all of the linearization matrix's eigenvalues for EEP are negative, equation 3.2 is considered locally asymptotically stable. It is necessary to convert the

equilibrium points to the origin for stability analysis. In order to begin, we centralize equation 3.2 at the endemic equilibrium $E(S^*,V^*,A^*,I^*,R^*)$ by introducing new variables.

$$\left.\begin{aligned} L_1 &= S - S^* \\ L_2 &= V - V^* \\ L_3 &= A - A^* \\ L_4 &= I - I^* \\ L_5 &= R - R^* \end{aligned}\right\} \tag{3.39}$$

We then rewrite model equation 3.2 in terms of the new variables and because $E(S^*,V^*,A^*,I^*,R^*)$ is an equilibrium point, the constant terms cancel. We also discard the quadratic terms because their partial derivatives at the origin is zero. The equation 3.2 with the new variables becomes;

$$\left.\begin{aligned} L_1^* &= p - (\omega + \beta_1 I_\tau + \beta_2 I_\tau + \gamma_1)(L_1 + S^*) \\ L_2^* &= \omega(L_1 + S^*) - (\varepsilon_1 I_\tau + \varepsilon_2 I_\tau + \varepsilon_3 I_\tau)(L_{2\tau} + V_\tau^*) - \gamma_2(L_2 + V^*) \\ L_3^* &= \beta_2 I_\tau(L_1 + S^*) + \varepsilon_2 I_\tau(L_{2\tau} + V_\tau^*) - (\delta_1 + \delta_2)(L_{3\tau} + A_\tau^*) - \gamma_3(L_3 + A^*) \\ L_4^* &= \beta_1 I_\tau(L_1 + S^*) + \varepsilon_1 I_\tau(L_{2\tau} + V_\tau^*) + \delta_1(L_{3\tau} + A_\tau^*) - \alpha(L_{4\tau} + I_\tau^*) - (\gamma_4 + \mu)(L_4 + I^*) \\ L_5^* &= \alpha(L_{4\tau} + I_\tau^*) + \delta_2(L_{3\tau} + A_\tau^*) + \varepsilon_3 I_\tau(L_{2\tau} + V_\tau^*) - \gamma_5(L_5 + R^*) \end{aligned}\right\} \tag{3.40}$$

A solution of the form $L(t) = L_0 e^{-\lambda t}$ and equation 3.40 is linearized about the equilibrium $(L_1,L_2,L_3,L_4,L_5) = (S^*,V^*,A^*,I^*,R^*)$ to obtain;

$$L^{*'}(t) = BL^*(t) \tag{3.41}$$

Where B is a 5×5 matrix. Differentiating the expanded equations of equation 3.40 partially with respect to the state variables, we obtain a 5×5 matrix Jacobian J_2, and is given as;

$$\begin{bmatrix} -(\omega+\beta_1 I_\tau+\beta_2 I_\tau+\gamma_1) & 0 & 0 & 0 & 0 \\ \omega & -I_\tau(\varepsilon_1+\varepsilon_2+\varepsilon_3)e^{-\lambda_\tau}-\gamma_2 & 0 & 0 & 0 \\ \beta_2 I_\tau & \varepsilon_2 I_\tau e^{-\lambda_\tau} & -(\delta_1+\delta_2)e^{-\lambda_\tau}-\gamma_3 & 0 & 0 \\ \beta_1 I_\tau & \varepsilon_1 I_\tau e^{-\lambda_\tau} & \delta_1 e^{-\lambda_\tau} & -(\gamma_4+\mu+\alpha e^{-\lambda_\tau}) & 0 \\ 0 & \varepsilon_3 I_\tau e^{-\lambda_\tau} & \delta_2 e^{-\lambda_\tau} & \alpha e^{-\lambda_\tau} & -\gamma_5 \end{bmatrix} \tag{3.42}$$

The characteristic equation of the Jacobian matrix is given by; $|J-I\lambda|=0$, where I is 5×5 identity matrix. To find the eigenvalues we have;

$$\begin{vmatrix} A-\lambda & 0 & 0 & 0 & 0 \\ \omega & B-\lambda & 0 & 0 & 0 \\ \beta_2 I_\tau & \varepsilon_2 I_\tau e^{-\lambda_\tau} & C-\lambda & 0 & 0 \\ \beta_1 I_\tau & \varepsilon_1 I_\tau e^{-\lambda_\tau} & \delta_1 e^{-\lambda_\tau} & D-\lambda & 0 \\ 0 & \varepsilon_3 I_\tau e^{-\lambda_\tau} & \delta_2 e^{-\lambda_\tau} & \alpha e^{-\lambda_\tau} & E-\lambda \end{vmatrix} = 0 \tag{3.43}$$

Where;

$$\left.\begin{aligned} A &= -(\omega+\beta_1 I_\tau+\beta_2 I_\tau+\gamma_1) \\ B &= -I_\tau(\varepsilon_1+\varepsilon_2+\varepsilon_3)e^{-\lambda_\tau}-\gamma_2 \\ C &= -(\delta_1+\delta_2)e^{-\lambda_\tau}-\gamma_3 \\ D &= -(\gamma_4+\mu+\alpha e^{-\lambda_\tau}) \\ E &= -\gamma_5 \end{aligned}\right\} \tag{3.44}$$

The equation 3.40 is locally asymptotically stable if all the eigenvalues of the linearization matrix of equation 3.40 are negative. Therefore the eigenvalues are;

$$\left.\begin{aligned} \lambda_{***1} &= -(\omega+\beta_1 I_\tau+\beta_2 I_\tau+\gamma_1) \\ \lambda_{***2} &= -I_\tau(\varepsilon_1+\varepsilon_2+\varepsilon_3)e^{-\lambda_\tau}-\gamma_2 \\ \lambda_{***3} &= -(\delta_1+\delta_2)e^{-\lambda_\tau}-\gamma_3 \\ \lambda_{***4} &= -(\gamma_4+\mu+\alpha e^{-\lambda_\tau}) \\ \lambda_{***5} &= -\gamma_5 \end{aligned}\right\} \tag{3.45}$$

The existence of endemic equilibrium is determined by the existence of positive solutions of the quadratic equation. Using λ_{***3} and λ_{***4} being the dominant eigenvalues, we obtain the characteristics equation below;

$$[((\varepsilon_1+\varepsilon_2+\varepsilon_3)e^{-\lambda_\tau}-\gamma_2)-\lambda][((\delta_1+\delta_2)e^{-\lambda_\tau}-\gamma_3)-\lambda]=0 \tag{3.46}$$

$(x-\lambda)(y-\lambda)=0$

Where,

$x=((\varepsilon_1+\varepsilon_2+\varepsilon_3)e^{-\lambda_\tau}-\gamma_2)$

$y=((\delta_1+\delta_2)e^{-\lambda_\tau}-\gamma_3)$

This implies,

$$\lambda^2-(x+y)\lambda+xy=0 \tag{3.47}$$

Which simplifies as;

$$a\lambda^2-b\lambda+c=0 \tag{3.48}$$

Therefore , the reproduction number at EEP is given by;

$$\lambda_{***}=\frac{-b\pm\sqrt{(b^2-4ac)}}{2a}$$

$$R_0=\lambda_{***}=\frac{-b+\sqrt{(b^2-4c)}}{2} \tag{3.49}$$

Clearly,

$$R_0=\frac{b^2}{(b^2-4c)}>1 \tag{3.50}$$

The basic reproduction number at EEP is greater than one $(R_0>1)$, which implies that the disease existence persists and is therefore endemic.

CHAPTER FOUR

RESULTS AND DISCUSSION

4.1 Introduction

In this chapter, numerical simulation were carried out using MATLAB to verify the analytic results on stability of model equations 3.2 considered in chapter three.The values for the parameters were taken from the literature. In simulation of the model equations 3.2, the following initial values in each compartment at the onset of infection is assumed to apply; $(S(0),V(0),A(0),I(0),R(0)) = (5.036\times 10^7,0,0,0,0)$ on the interval $[-\tau,0]$.

4.2 Numerical Simulations and Discussions

In the numerical solution, we used MATLAB to plot the system of Equation (3.2) to verify the analytical results on the stability of the model. The plot gives us an idea of how the different compartment of people; susceptible, vaccinated, asymptomatic infected, or recovered, rise or fall during a period of weeks. Figure 4.1 illustrates the smooth curve, representing susceptibility with time which drastically decreases within the first 150 days the stabilized after. Figure 4.2 represents the smooth curve for the vaccinated against time, which rose slowly due to vaccine shortage within the first 150 days then steadily rose. The figure 4.3 represents the smooth curve for recovery with time, which rose steadily within the first 150 days before it stabilized because of increased vaccination coverage. Figure 4.4 represents the infections with time, the number of infections rose to the peak in the first 200 days then gradually decreased. First, everyone is susceptible, then the number decrease and some of the infected progress to asymptomatic or infectious class but after a series of weeks, the infected becomes sick, but they recovery from the disease or die.

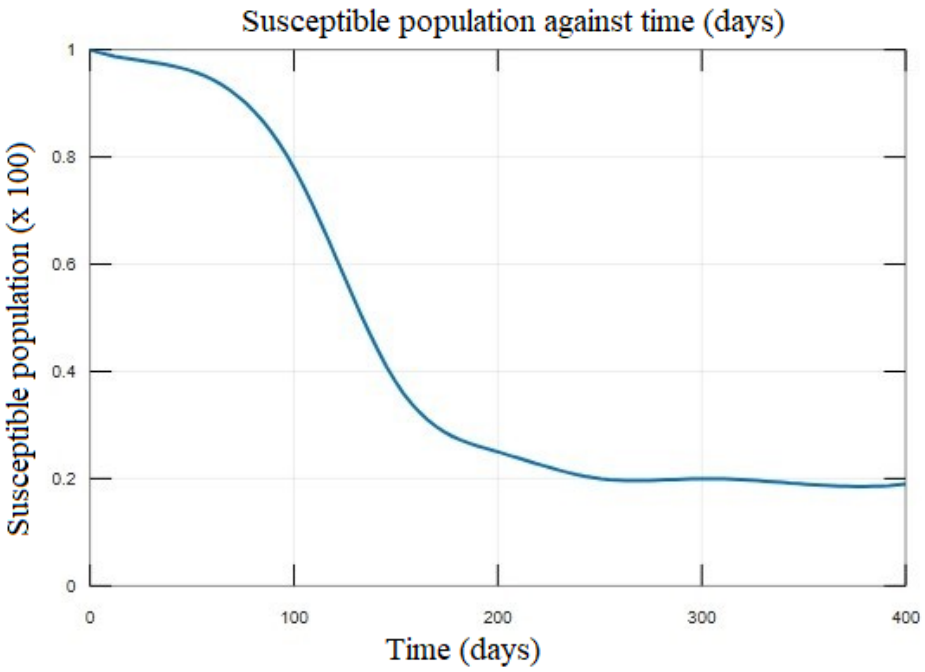
Numerical simulations of the model equations are obtained using the list of parameters and the estimated values given in Table 4.1

Table 4.1: Table of parameters, parameter description, values and source

Table 4.1 Table of parameters and values

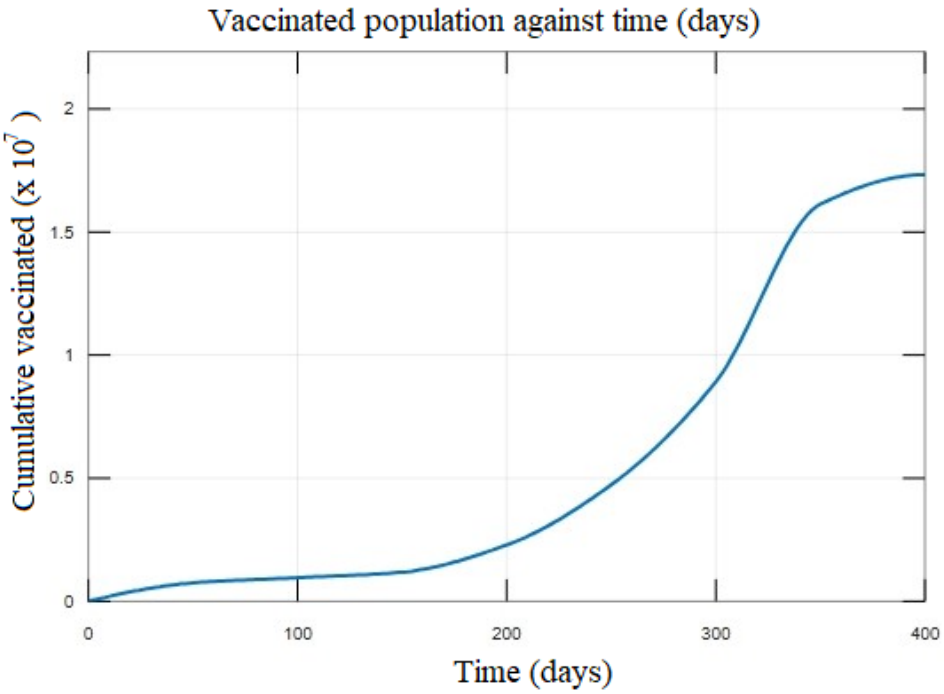
Parameter	**Description**	**Value**	**Source**
S	Susceptible class	$5.036 \times 10^7 years^{-1}$	Estimated from (Muchiri *et al*., 2022)
V	Coronavirus disease vaccinated class	$1.8 \times 10^7 years^{-1}$	(Carpio *et al*., 2021)
A	Asymptomatic infected class	7322 $years^{-1}$	(Carpio *et al*., 2021)
I	Symptomatic infected class	7545 $years^{-1}$	Estimated from (Muchiri *et al*., 2022)
R	Recovered class	203968 $years^{-1}$	Estimated from (Muchiri *et al*., 2022)
p	Recruitment rate	1762 $days^{-1}$	Estimated from (Muchiri *et al*., 2022)
ω	Rate of progression from S to V	0.002 $days^{-1}$	Assumed
β_1	Transition rate from susceptible to infectious class	0.233 $days^{-1}$	(Chae *et al*., 2020)
β_2	Transition rate from susceptible to asymptomatic class	0.09786 $days^{-1}$	(Algarni *et al*., 2022)
ε_1	Rate of progression from V to I	0.462 $days^{-1}$	(Chae *et al*., 2020)
ε_2	Rate of progression from V to A	0.039 $days^{-1}$	(Li *et al*., 2020)
$\frac{1}{\varepsilon_3}$	Immunity development period	0.0714 $days^{-1}$	(Algarni *et al*., 2022)
δ_1	Rate of progression from A to I	0.1428 $days^{-1}$	(Algarni *et al*., 2022)
δ_2	Rate of progression from A to R	0.165 $days^{-1}$	(Buonomo *et al*., 2022)
α	Rate of progression from I to R	0.1 $days^{-1}$	(Algarni *et al*., 2022)
γ	Natural death rate	1.07×10^{-2} $years^{-1}$	(Buonomo *et al*., 2022)
μ	Disease induced death rate	6.248×10^{-4} $days^{-1}$	(Buonomo *et al*., 2022)
τ	Time delay	To be estimated	

SVAIR Model Population vs Time(days)



A plot of Susceptible Population against time (days)

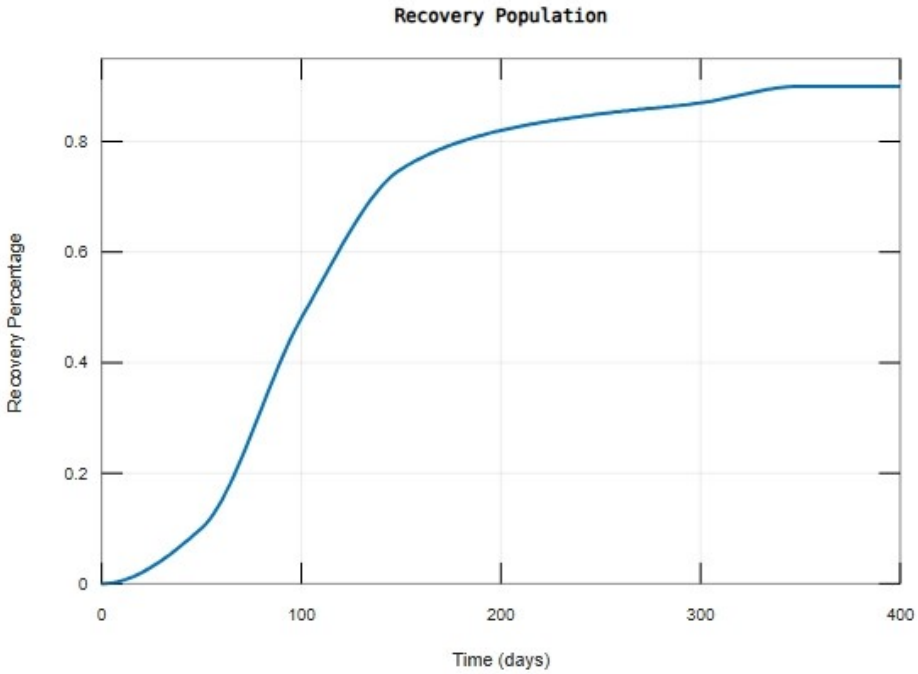
From the graph in the **Figure 4.1** above , it is evident that susceptible population is inversely proportional to time, nearly the entire population were susceptible from the onset then drastically decreases. S(t) decreases over time steeply at first then levels off near the end as the epidemic slows. The sharp drop was due to the rising number of infection cases and the stabilized at the lower level was because increased vaccination coverage which boost the immune protection.



A plot of Vaccinated population against time (days)

From the graph **Figure 4.2** above, it is evident that vaccinated individuals are directly proportional to time. The number of vaccinated individuals gradually increases.

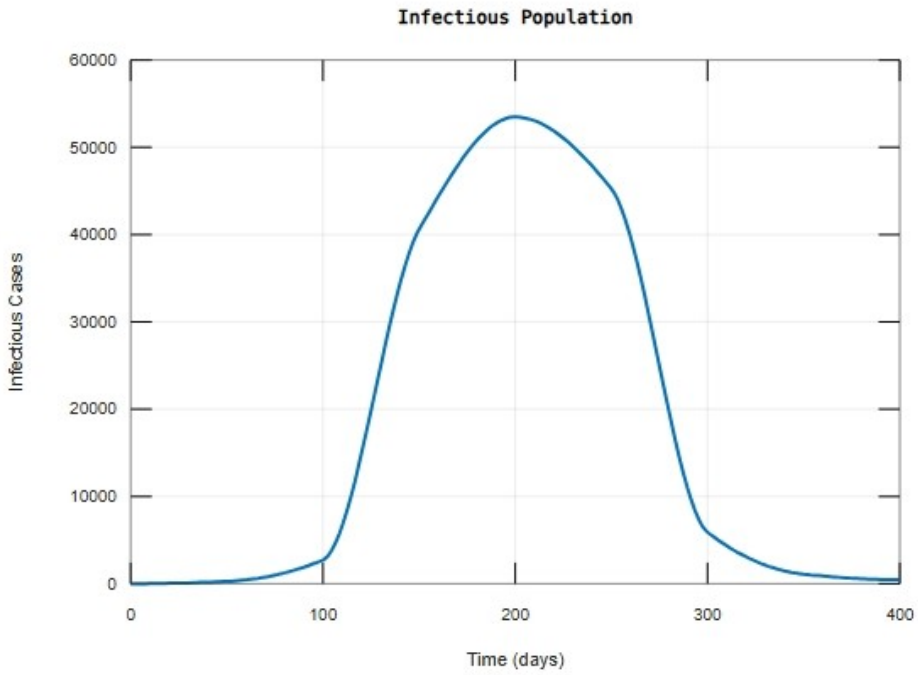
A solution to COVID-19 could be achieved when we increased the number of vaccinated people to half or more of the population. These results reduced the number of infected people and increased the number of susceptible individuals.



A plot of Recovered against Time

From the graph **Figure 4.3** above, it is evident that the number of recovered individuals is directly proportional to time. Recovered individuals increases because of increased vaccination coverage.

Following a period of infection, surviving individuals recovered to a state in which viral shedding no longer occurs. These recovered individuals are assumed to be susceptible to reinfection but are assumed to retain some level of partial immunity, which reduces susceptibility to subsequent exposure. Gradual loss of protection against infection over time may result in immunity decay, but for this study we optimistically assume no immunity decay following naturally acquired or vaccine-induced immunity due to the short-term nature of the simulation period. For naturally acquired immunity due to infection, we assumed an 90% transmission blocking effect when or if re-exposed (Shattock et al., 2022). If a previously infected but recovered individual is later vaccinated, the level of transmission blocking immunity is taken to be the highest of the two independent effects. No combined resultant effect was considered. Although the transmission blocking effect of naturally acquired immunity and vaccine-induced-acquired immunity can be of different strengths, they are both considered enhancing the immune state of an individual.



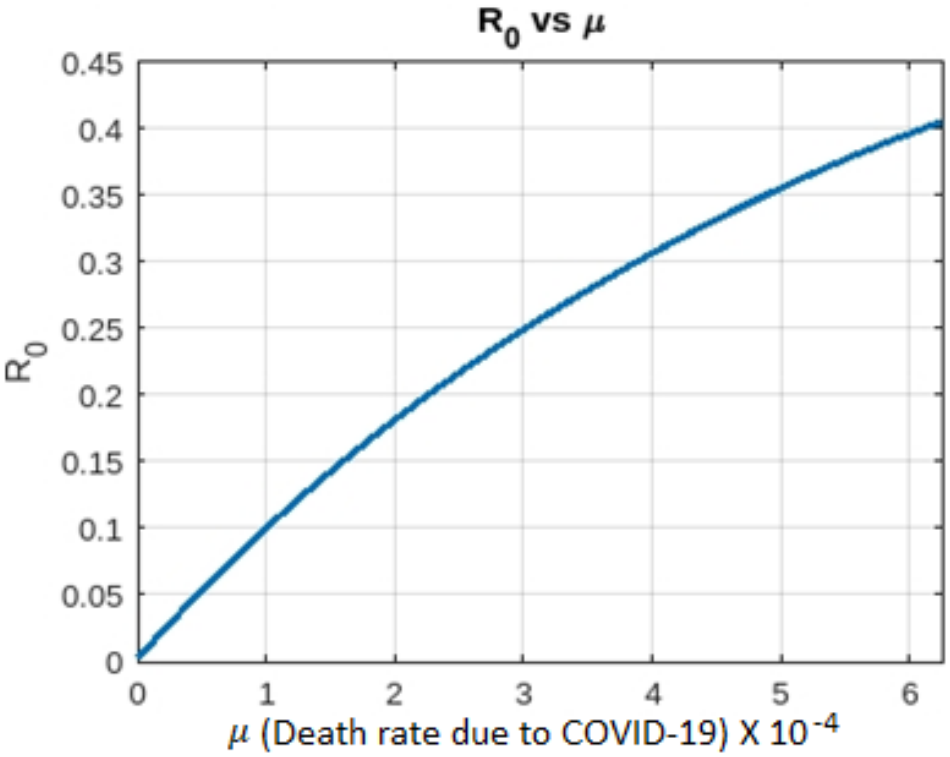
A plot of Infectious against Time

From the graph **Figure 4.4** above, it is evident that the rate of infections rose with time to the peak. Increased in number due to absent of vaccination and gradually decreased when vaccination started which caused delayed infections.

Early phase, infections start low and increase exponentially because many people are susceptible. The peak phase demonstrates that infection growth slows as more people develop immune naturally or due to vaccines, the curve reaches maximum point called the epidemic peak. The decline phase; infections decrease as fewer susceptible individuals remain and the number of recovered reduces the spread of COVID-19. The end phase; the curve flattens or stabilizes at a lower endemic level meaning there was less infections but more vaccination coverage. Figure 4.1 shows the dynamics of population of various compartments of SVAIR against time in days. From the graph, it is evident that nearly the entire population is susceptible from the onset and as the infected number increases, the number of susceptible individuals drastically decreases to about 20 percent, because vaccination, treatment and time delay played a major role in stabilizing the rate of infection. Increasing vaccination coverage means immunity of the susceptible individual is improved hence the infectious period delayed and therefore the basic reproduction number is lowered, minimizing the rate of infection.

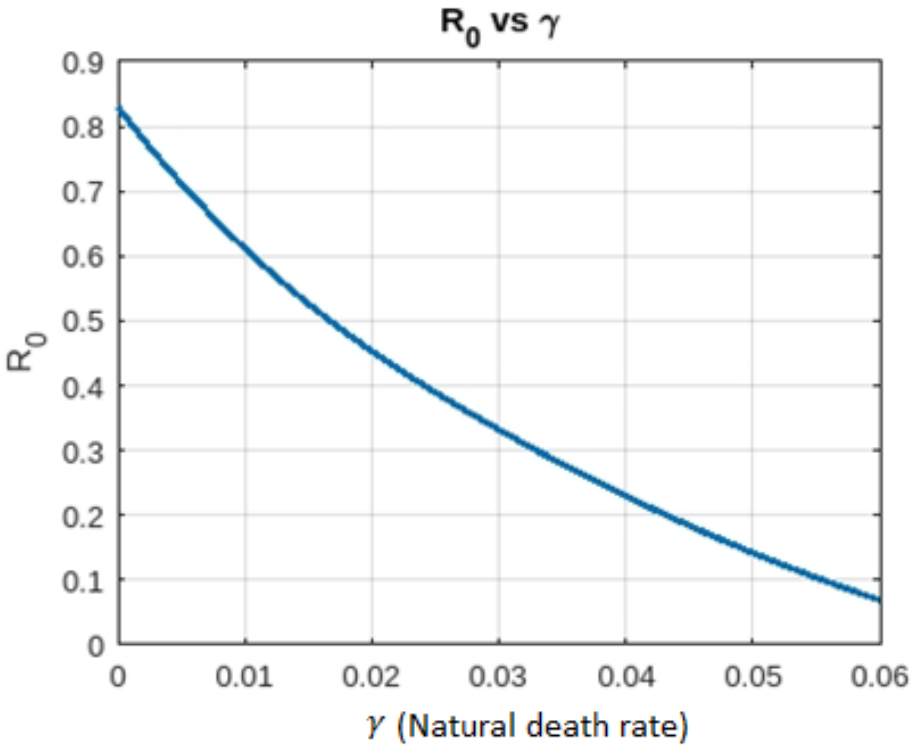
4.3 Analysis of Basic Reproduction Number at DFE and EEP

At Disease Free Equilibrium (DFE), $R_0 < 1$ which implies that the disease dies out in the population. Public health interventions aim at reducing and maintaining the value of R_0 below 1 which can be attained through vaccination which lowers the rate of infection by increasing time delay hence R_0 is reduced.



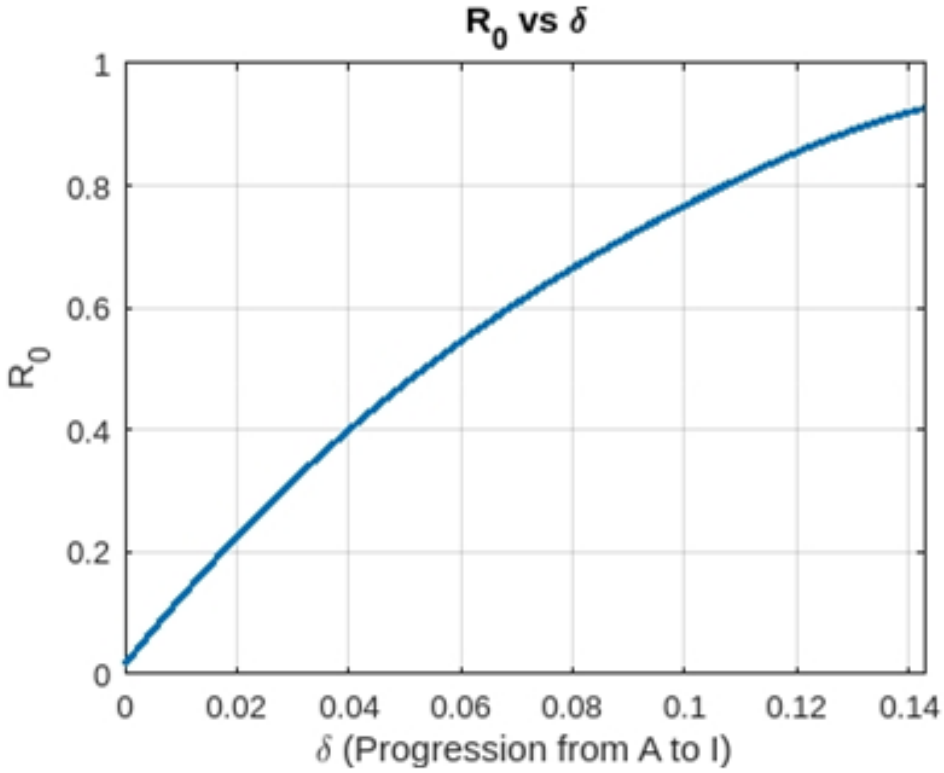
R_0 against Death rate due to COVID-19 (μ)

Figure 4.5 above shows that the reproduction number R_0 is directly proportion to the rate of death. It is evident from the graph that when the number of individuals with the disease is high, those who die also goes up and vice versa unless intervention measure like vaccination is rolled out which lowers the spread by increasing time delay hence R_0 is reduced resulting in reduced death rate.



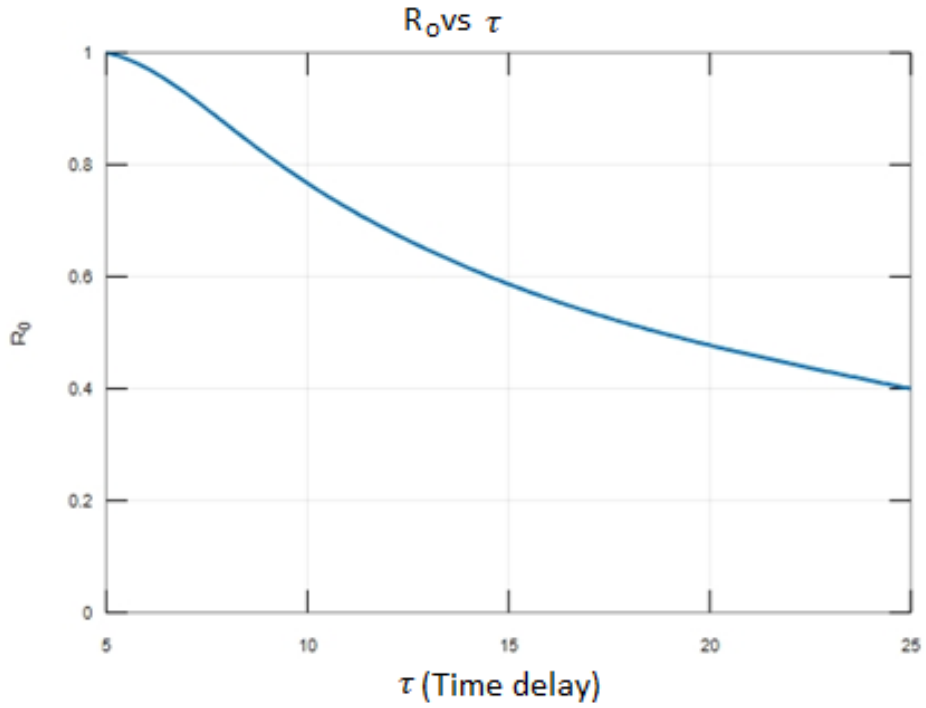
R_0 against Gamma (γ)

Figure 4.6 above shows a plot of reproduction number against the natural death rate. It is evident that R_0 is inversely proportional to the natural death rate, when the natural death rate (γ) is high it reduces the effective reproduction number R_0 slightly as some infected individuals die before transmitting the virus.



R_0 against Delta (δ)

Figure 4.7: above shows a plot of reproduction number against the rate of progression from asymptomatic (A) to infectious (I). It is evident that R_0 is directly proportional to the rate of progression δ. A higher progression rate (δA) implies a shorter asymptomatic infectious period (incubation period) this potentially increasing R_0.



R_0 against Time delay (τ)

Figure 4.8 above shows a plot of reproduction number against time delay at DFE. From the graph R_0 is inversely proportional to time delay. It is evident that a shorter τ leads to higher R_0 and the virus spread faster in a population. If τ is short, highly contagious variants spread faster before intervention measures can take effect.

4.4 Sensitivity Analysis of Basic Reproduction Number R_o

Sensitivity analysis for the basic reproduction number R_o is being studied in order to identify the parameter value that has the greatest impact on the spread of the disease. Because of this, we must ascertain the reproduction number's sensitivity index in relation to the model parameters. Sensitivity index analysis is done using partial derivatives when the variable is a differentiable function of the parameter, (Adebiyi et al.., 2016)

Through the examination of these indices, the most important parameter for the dynamics of COVID-19 transmission was identified. The ratio of the relative change in a variable to the relative change in a parameter is known as the normalized forward sensitivity index, (Adebiyi et

al .., 2016) Sensitivity index is defined using partial derivative as;

$$\eta = \frac{\partial R_o}{\partial x} \times \frac{x}{R_o} \tag{4.1}$$

where,

η is the sensitivity index

R_o is the basic reproduction number

x is the parameter under investigation

Therefore the derivation of the sensitivity index η of R_o with respect to each parameter is given below.

$\frac{\partial R_o}{\partial \gamma} \times \frac{\gamma}{R_o} = \frac{-\gamma}{\delta_1+\gamma+\mu} = -0.06942$

$\frac{\partial R_o}{\partial \mu} \times \frac{\mu}{R_o} = \frac{-\mu}{\delta_1+\gamma+\mu} = -0.004054$

$\frac{\partial R_o}{\partial \alpha} \times \frac{\alpha}{R_o} = \frac{-\alpha}{\delta_1+\gamma+\mu} = 0.6488$

$\frac{\partial R_o}{\partial \delta_1} \times \frac{\delta_1}{R_o} = \frac{-\delta}{\delta_1+\gamma+\mu} = 0.9265$

The sensitivity indices for the parameters in our model are as shown in the table 4.2 below.

Table 4.2 Table of parameters, parameter description, values and sensitivity indices of R_o

Parameter	Parameter description	Value	Sensitivity
γ	Natural death rate	0.0107 days^{-1}	- 0.06942
		0.005 days^{-1}	- 0.04744
μ	Disease induced death rate	0.0006248 years^{-1}	- 0.004054
		0.0004 years^{-1}	- 0.003795
α	Recovery rate of infectious class	0.1 days^{-1}	- 0.6488
		0.05 days^{-1}	- 0.4744
δ_1	Rate of progression of asymptomatic to infectious class	0.1428 days^{-1}	- 0.9265
		0.12 days^{-1}	- 0.8547

Sources of parameter values:(Algarni et al.., 2022)

From table 4.2, it is evident that for all model parameters, the signs of the sensitivity indices of R_o are all negative. This implies that all model parameters under consideration contributes to the decline in the spread of the COVID-19 infection. It is all notable that δ_1 is more sensitive than

other model parameters since it gives the most negative sensitivity index. Thus δ_1, which is rate of progression of asymptomatic individuals to infectious class, contributes more to the decline in the spread and transmission of COVID-19 infection.

CHAPTER FIVE

CONCLUSION, RECOMMENDATIONS AND SUGGESTIONS FOR FURTHER RESEARCH

5.1 Introduction

This chapter gives an overview of the thesis, it outlines the summary of the key findings and highlights the general conclusion. Finally the recommendations and suggestions for further research work.

5.2 Conclusion

In this study, we formulated and presented a SVAIR mathematical model of COVID-19 transmission dynamics, incorporating time delay. An expression of the basic reproduction number was derived from the model equation (3.2). Stability analysis of equilibrium points of the model was determined where, it was found out that the model was stable at both Disease Free Equilibrium (DFE) and Endemic Equilibrium Point (EEP) and was attained when $R_0 < 1$ and $R_0 > 1$ respectively. The parameter values were taken from the literature and the analytical findings were verified numerically using the Matlab dde 23 solver. From the study it is evident that for time delay $\tau > 5$ days DFE is stable otherwise unstable. Numerical simulation was carried out to validate the analytic results where it was found out that $R_0 = 0.95$ at DFE. Furthermore the results show that stability was guaranteed at EEP and is attained at $R_0 = 1.02$. Time delay, rate of vaccination and rate of progression from asymptomatic to infectious plays an important role in the transmission of COVID-19 and stability at DFE and EEP. The sensitivity analysis of the model parameters were performed to identify the parameter that has the greatest impact on the reproduction number R_o, it was found out that for all model parameters under study, the signs

of the sensitivity indices of R_o were all negative. This implied that the model parameters under consideration contributed to the decline in the spread of the COVID-19 infection. It was noted that the rate of progression from asymptomatic to infectious δ_1 was more sensitive than other model parameters since it contributed the most negative sensitivity index of -0.9265.

5.3 Recommendations

In our study on COVID-19 outbreak in Kenya, numerical simulation shows that incubation period for asymptomatic individuals was 5 days. This recommends it necessary to prolong the time delay by increasing the vaccination coverage against COVID-19 so that all potentially infected asymptomatic individuals progress to recovery class. The time delay was substantially shorter, suggesting that a significant number of secondary transmission may occur prior to illness onset. Also based on the high sensitivity index δ_1 interventions should be prioritize reducing contact rates of symptomatic individuals. Numerical simulations showed that the disease free equilibrium (DFE) was less than a unit, therefore second and third booster shot may be necessary to prevent gradual immune decay hence maintaining Ro below a unit.

5.4 Suggestions for further research

This study has not exhausted all about COVID-19 transmission dynamics. Further studies can be done on COVID-19 vaccine efficacy. The model may be extended to include co-infection between COVID-19 and TB or any other underlying conditions. Further studies can also be done on the effects of partially and fully vaccinated individuals on COVID-19 transmission dynamics. Future work could be done to incorporate waning immunity to study the impact of booster shots, which was beyond the scope of this model.

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APPENDICES

APPENDIX I : MATLAB Code: Dynamics of COVID-19 against time

```
% MATLAB script to plot SVAIR model data with smooth curves
% Time points (in days)
time = [0 50 100 150 200 250 300 350 400];
% Data from the table
susceptible = [1 0.96 0.78 0.38 0.25 0.2 0.2 0.19 0.19];
vaccinated = [0 750400 960500 1183400 2290800 4733800 8913700 16130700 17327200];
infectious = [0 270 2720 40590 53475 45250 5900 1100 450];
recovery = [0 0.1 0.48 0.75 0.82 0.85 0.87 0.9 0.9];
% Generate smooth time points
smooth_time = linspace(min(time), max(time), 200);
% Interpolate using spline for smooth curves
smooth_susceptible = interp1(time, susceptible,
smooth_time, 'spline');
smooth_vaccinated = interp1(time, vaccinated,
smooth_time, 'spline');
smooth_infectious = interp1(time, infectious,
smooth_time, 'spline');
smooth_recovery = interp1(time, recovery, smooth_time, 'spline');
% Plot Susceptible Population
figure;
plot(smooth_time,smooth_susceptible,'', 'LineWidth', 2);
xlabel('Time (days)');
ylabel('Susceptible Percentage');
```

```
    title('Susceptible Population');
grid on;
% Plot Vaccinated Population
figure;
plot(smooth_time, smooth_vaccinated, '-', 'LineWidth', 2);
xlabel('Time (days)');
ylabel('Cumulative Vaccinated');
title('Vaccinated Population');
grid on;
ylim([0 max(vaccinated)]); % Ensure axis starts at 0
% Plot Infectious Population
figure;
plot(smooth_time, smooth_infectious, '-', 'LineWidth', 2);
xlabel('Time (days)');
ylabel('Infectious Cases');
title('Infectious Population');
grid on;
% Plot Recovery Population
figure;
plot(smooth_time, smooth_recovery, '-', 'LineWidth', 2);
xlabel('Time (days)');
ylabel('Recovery Percentage');
title('Recovery Population');
grid on;
ylim([0 max(recovery)]); % Ensure axis starts at 0
```

APPENDIX II : MATLAB Code: R_0 variations with different model parameters

```
% MATLAB script to plot R0 variations with different parameters separately (without circles)
% (a) R0 vs Death rate due to COVID-19 (Î¼)
mu = [0.6248 0.5 0.4 0.3 0.2 0.1] * 10^-3;
R0_mu = [0.4054 0.3559 0.3067 0.2494 0.1815 0.1];
figure;
plot(mu, R0_mu, '-', 'LineWidth', 2);
xlabel('mu (Death rate due to COVID-19)');
ylabel('R_0');
title('R_0 vs mu');
grid on;
% (b) R0 vs Natural death rate (Î³)
gamma = [0.0107 0.02 0.03 0.04 0.05 0.06];
R0_gamma = [0.5994 0.4537 0.3325 0.23 0.1425 0.06942];
figure;
plot(gamma, R0_gamma, '-', 'LineWidth', 2);
xlabel('gamma (Natural death rate)');
ylabel('R_0');
title('R_0 vs gamma');
grid on;
% (c) R0 vs Recovery rate of Infectious (Î±)
alpha = [0.1 0.2 0.3 0.4 0.5 0.6];
R0_alpha = [0.6488 1.425 2.3 3.325 4.537 5.9945];
figure;
plot(alpha, R0_alpha, '-', 'LineWidth', 2);
xlabel('alpha (Recovery rate of Infectious)');
```

```
ylabel('R_0');
title('R_0 vs alpha');
grid on;
% (d) R0 vs Rate of progression from A to I (Î´)
delta = [0.1428 0.12 0.1 0.08 0.06 0.04];
R0_delta = [0.9265 0.8547 0.7669 0.665 0.5445 0.3996];
figure;
plot(delta, R0_delta, '-', 'LineWidth', 2);
xlabel('delta (Progression from A to I)');
ylabel('R_0');
title('R_0 vs delta');
grid on;
% (e) R0 vs Time delay (Ï„)
tau = [7 8.3 10.0 12.5 16.67 25];
R0_tau = [0.9265 0.8547 0.7669 0.665 0.5445 0.3996];
figure;
plot(tau, R0_tau, '-', 'LineWidth', 2);
xlabel('tau (Time delay)');
ylabel('R_0');
title('R_0 vs tau');
grid on;
ylim([0 1]);
```

APPENDIX III : Clearance to commence field work

UNIVERSITY OF KABIANGA
ISO 9001:2015 CERTIFIED
OFFICE OF THE DIRECTOR, BOARD OF GRADUATE STUDIES

REF: PGC/AM/001/20

DATE: 6TH MARCH, 2024

Peter Kibii Cheruiyot,
Mathematics, Actuarial and Physical Sciences Department,
University of Kabianga,
P.O Box 2030- 20200,
KERICHO.

Dear Mr. Cheruiyot,

RE: CLEARANCE TO COMMENCE FIELD WORK/DATA COLLECTION

I am pleased to inform you that the Board of Graduate Studies has considered and approved your MSc research proposal entitled **"Modelling the Effects of Vaccination, Incubation and Treatment on COVID – 19 Transmission Dynamics."**

Subsequently the Board has also approved the following supervisors for appointments.

1. Dr. Wesley Kirui
2. Dr. Reuben Langat
3. Dr. Benard Tonui

You may now proceed to commence field work/data collection on condition that you obtain a research permit from NACOSTI and /or an ethical review permit from a relevant ethics review board.

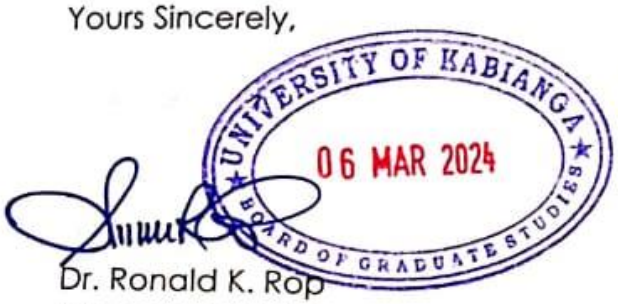
You are also required to publish one (1) article in a peer reviewed journal, with all your supervisors, before your oral defense of thesis.

You are required to submit through your supervisors, and HoD, progress reports every three months, to the Director, Board of Graduate Studies.

Please note that it is the policy of the University that you complete your studies within three years from the date of registration. Do not hesitate to consult this office in case of any difficulties encountered in the course of your studies.

I wish you all the best in your research and hope that your study will yield original contribution for the betterment of humanity.

Yours Sincerely,



Dr. Ronald K. Rop
DIRECTOR, BOARD OF GRADUATE STUDIES.
RKR/lc

cc 1. Dean, SST
2. HOD, MAPS
3. Supervisors

APPENDIX V : NACOSTI Research Permit

REPUBLIC OF KENYA

NATIONAL COMMISSION FOR
SCIENCE,TECHNOLOGY & INNOVATION

Ref No: **408657**

Date of Issue: **06/March/2025**

RESEARCH LICENSE

This is to Certify that Mr.. PETER KIBII CHERUIYOT of University of Kabianga, has been licensed to conduct research as per the provision of the Science, Technology and Innovation Act, 2013 (Rev.2014) in Bomet on the topic: MODELLING THE EFFECTS OF VACCINATION, INCUBATION AND TREATMENT ON COVID -19 TRANSMISSION DYNAMICS for the period ending : 06/March/2026.

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THE SCIENCE, TECHNOLOGY AND INNOVATION ACT, 2013 (Rev. 2014)
Legal Notice No. 108: The Science, Technology and Innovation (Research Licensing) Regulations, 2014

The National Commission for Science, Technology and Innovation, hereafter referred to as the Commission, was the established under the Science, Technology and Innovation Act 2013 (Revised 2014) herein after referred to as the Act. The objective of the Commission shall be to regulate and assure quality in the science, technology and innovation sector and advise the Government in matters related thereto.

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    ii. Adversely affect the lives of Kenyans
    iii. Be in contravention of Kenya's international obligations including Biological Weapons Convention (BWC), Comprehensive Nuclear-Test-Ban Treaty Organization (CTBTO), Chemical, Biological, Radiological and Nuclear (CBRN).
    iv. Result in exploitation of intellectual property rights of communities in Kenya
    v. Adversely affect the environment
    vi. Adversely affect the rights of communities
    vii. Endanger public safety and national cohesion
    viii. Plagiarize someone else's work
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15. Relevant Institutional Scientific and Ethical Review Committee shall monitor and evaluate the research periodically, and make a report of its findings to the Commission for necessary action.

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Innovation(NACOSTI),
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APPENDIX VI : Publication from the Thesis

Journal of Advances in Mathematics and Computer Science

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Modelling the Effects of Vaccination and Incubation on Covid-19 Transmission Dynamics

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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Original Research Article

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Abstract

The Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-COV-2) is a strain of Coronavirus that causes Coronavirus Disease 2019 (COVID-19). The respiratory illness responsible for the COVID19 pandemic began in December 2019 in Wuhan city, China. Mathematical modeling has enabled the epidemiologist to understand the dynamics of the disease, its impact and future predictions in order to provide the governments with the best policies and strategies to curb the spread of the virus. Deterministic susceptible-vaccinated-asymptomatic-infectious-recovered (SVAIR) model was formulated incorporated with time delay. The delay accounts for the time lapsed between exposure and infectious period. Furthermore, time delay and vaccination inversely affects

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the basic reproduction number hence play a major role in stabilizing the rate of infection. In this study delay differential equations (DDE) were formulated for the purposes of determining the stability of both disease free equilibrium (DFE) and endemic equilibrium point (EEP). It was found out that the model was stable at both Disease Free Equilibrium (DFE) and Endemic Equilibrium Point (EEP) and was attained whenever $R_0 < 1$ and $R_0 > 1$ respectively. Calculations based on secondary data from various works of literature and the WHO dashboard was used. The basic reproduction number (R_0) was computed using the next generation matrix (NGM) approach. Finally, numerical simulations were carried out using MATLAB for validation of the analytical results. Graphical representation shows that stability is achieved when $\tau > 5$ days and that $R_0 = 0.95$ at DFE. Furthermore at EEP it was noted that $R_0 = 1.02$ hence stability was guaranteed.

Keywords: COVID-19; reproduction number; delay differential equations; stability; disease free equilibrium.

1 Introduction

The devastating Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-COV-2) is a strain of Coronavirus that causes Coronavirus Disease 2019 (COVID-19). The respiratory illness responsible for the COVID-19 pandemic which began in December 2019 (WHO, 2020) in Wuhan City, China and spread to many countries in the world (Shen et al., 2020). Kenya reported its first case of COVID-19 in March 2020. This resulted in the restriction of movement by the government, which included the closure of all learning institutions (McCloskey et al., 2020).

As of 18th April 2022, COVID-19 had spread to every country in the world, resulting in 500,186,525 confirmed cases of infection and 6,190,349 deaths globally, according to the WHO's pandemic declaration from March 2020 (WHO, 2020). The number of COVID-19 cases reported to WHO by selected countries as at April 2022 include; USA had 79,716,960 confirmed cases and 2,711,776 deaths, United Kingdom had reported 21,715,120 confirmed cases and 979,321 deaths, Africa had reported cumulative of 8,676,141 confirmed cases and 171,350 deaths and Kenya had reported 323,588 confirmed cases and 5649 deaths, (Shen et al., 2020).

Over time, it became evident that the Kenyan government's non-pharmaceutical mitigation measures to curb the spread of COVID-19 were not effective. Use of face masks, keeping of social distance, routine hand washing, isolation of suspected cases and contact tracing were a few examples of such intervention measures. Because of the rapid spread of COVID-19 globally, vaccination has been identified as the best containment strategy by several countries. The WHO approved COVID-19 vaccines where Kenya used different types of vaccines such as Astra Zeneca, Modena, Pfizer and Johnson and Johnson. In this study, an SVAIR mathematical model was formulated using delay differential equations (DDE) in which delay is incorporated to ascertain for the incubation period in the COVID-19 transmission dynamics.

2 Review of Related Literature

The novel coronavirus identified as Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-COV-2) is a strain of Coronavirus that causes Coronavirus Disease 2019. This disease was named by the World Health Organization (WHO) as Coronavirus Disease 2019 (COVID-19) (WHO, 2020). WHO declared COVID-19 a pandemic in March 2020.

Musa et al., (2022), studied the infection fatality rate and infection rate of COVID-19 in Nigeria using a susceptible-exposed-infectious-recovered (SEIR) based model with a time-varying transmission rate controlled by systems of delayed differential equations (DDEs). They discovered that the awareness campaigns, in particular the use of non-pharmaceutical measures in mitigating the spread of COVID-19 were effective during the earliest period of infection.

Xu et al., 2021, in their study found out that in the absence of vaccines, early detection, isolation and treatment were then the most efficient intervention measures. The study only concentrated on non-pharmaceutical approaches for containing and lessening the pandemic impact in the absence of reliable vaccines or antivirals. These mitigation measures were temporary, since over time, the general populace has a tendency to loosen them, which would accelerate the spread of COVID-19. This study did not consider how the COVID-19 incubation period affected the isolated and confined patients.

Wangari et al., (2021), studied the mathematical modeling of COVID-19 transmission in Kenya, a model with a re-infection transmission mechanism. They found out that a large pool of infectious people who were asymptomatic would eventually increase symptomatic people with mild and severe symptoms, which may eventually increase the overall number of reported deaths. The model used deterministic ordinary differential equations (ODEs) for computation of basic reproduction number. Unfortunately, the model did not take into account the incubation period during asymptomatic period but it called for further research on the problems presented by asymptomatic patients in connection with reinfection.

Aldila et al., (2020), in their study, they considered the undiscovered asymptomatic individuals who actively contributed to the transmission of COVID-19 despite having no symptoms. The study also demonstrated that the inadequate healthcare resources contributed to the spread of COVID-19 virus. SVEIAHR mathematical model were developed, sensitivity analysis was used to determine how the model parameters affected the spread of the disease. The study found out that although it was necessary to reduce the basic reproductive number below one in order to stop the spread of infection, it was not sufficient because several additional intervention measures were required.

Lemaitre et al., (2020), in their study, they investigated the effects of non-pharmaceutical strategies, according to their studies, there was a significant amount of virus transmission through contaminated surfaces, depending on the nature of the virus. This study expressed concern over the fact that public awareness of COVID-19 was one of the major challenge in curbing the spread of COVID-19. Many people choose not to follow the safety precautions already in place including wearing face masks, regularly sanitizing, avoiding crowded areas and maintaining social distance. The mathematical model did not considered vaccination as the mitigation measure against the spread of COVID-19.

Shattock et al., (2022), studied the mathematical model based on ordinary differential equations (ODEs). Their study subdivided the total population into; susceptible, vaccinated, infectious, asymptomatic and recovered (SVIAR). Their study showed that vaccinations could assist to reduce transmission rates, but they also stated that during the COVID-19 pandemic, proper waste management was essential. In addition, it did not state how the asymptomatic individuals affect the transmission dynamic of COVID-19 and the time delay.

The above studies did not consider the use of delay differential equations (DDEs) in modeling the transmission dynamics of COVID-19. This study formulated a mathematical model incorporating DDEs for the transmission dynamics of COVID-19 in Kenya in which incubation period is considered.

3 Methodology

3.1 Introduction

In epidemiology, the basic reproduction number of an infection is the mean number of new infections arising from a single infectious person introduced into a purely susceptible population during infectious period. The basic reproduction number R_0 is the threshold for many epidemiological models. When $R_0 > 1$ the disease-free equilibrium point is unstable, the infection will be able to spread in a population and locally stable for $R_0 < 1$, the infection dies out in the long run.

To obtain R_0 the dominant eigenvalue of the next generation matrix is considered such that $R_0 = \rho FV^{-1}$. Where ρ is the spectral radius of the next generation matrix. Matrix F_i represents the rate of new infection entering compartment i, and matrix V_i represents the rate of transfer into and out of compartment i by other means. Since we are dealing with large population, compartmental mathematical model is used. The total population is subdivided into compartments each representing a specific stage of epidemic. The Susceptible-Vaccinated-Asymptomatic- Infected- Recovered (SVAIR) mathematical models are utilized to study and analyze the COVID-19 transmission dynamics.

3.2 Methods of solution

The stability of the model has been determined using the Jacobian matrix which is a system of partial derivative of equations for checking the stability of Disease Free Equilibrium (DFE) and numerical simulation was done using MATLAB to validate the analytic results.

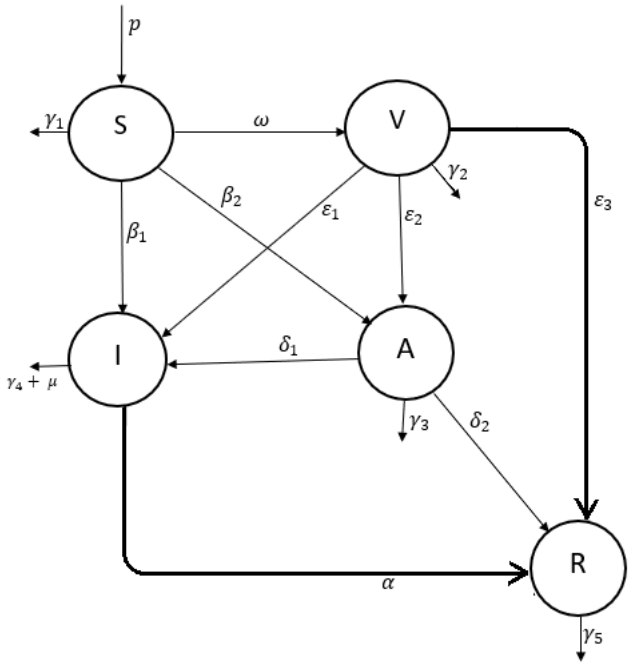

Fig. 1. SVAIR model flow diagram

3.3 Model equations

$$\left.\begin{aligned}
\frac{dS(t)}{dt} &= p - \omega S - \beta_1 S I_\tau - \beta_2 S I_\tau - \gamma_1 S \\
\frac{dV(t)}{dt} &= \omega S - \varepsilon_1 V_\tau I_\tau - \varepsilon_2 V_\tau I_\tau - \varepsilon_3 V_\tau I_\tau - \gamma_2 V \\
\frac{dA(t)}{dt} &= \beta_2 S I_\tau + \varepsilon_2 V_\tau I_\tau - \delta_1 A_\tau - \delta_2 A_\tau - \gamma_3 A \\
\frac{dI(t)}{dt} &= \beta_1 S I_\tau + \varepsilon_1 V_\tau I_\tau + \delta_1 A_\tau - \alpha I_\tau - (\gamma_4 + \mu) I \\
\frac{dR(t)}{dt} &= \alpha I_\tau + \delta_2 A_\tau + \varepsilon_3 V_\tau I_\tau - \gamma_5 R
\end{aligned}\right\} \tag{3.1}$$

3.4 Model preliminary analysis

3.4.1 Positivity and boundedness of the model

Positivity and boundedness of the model is shown before doing result analysis for model equation 3.1 to be epidemiologically meaningful. Thus, we show that all state variables of the model equation (3.1) are nonnegative for all time $t > 0$ and bounded and the existence of solutions are underscored by these properties.

Let C = C [-τ, 0], $\Re^5$ be a Banach space of continuous functions mapping the interval [-τ, 0] into $\Re^5$ with the topology of uniform convergence. By fundamental theory of differential equations, it can be shown that there exists a unique solution (S(t), V(t), A(t), I(t), R(t)) of the model equation 3.1 with initial data.

$$(S(0) > 0, V(0) > 0, A(0) > 0, I(0) > 0, R(0) > 0 \in C) \tag{3.2}$$

In addition, we assume that the initial data for the model equation 3.2 satisfies;

$$S_0(p) \geq 0, V_0(p) \geq 0, A_0(p) \geq 0, I_0(p) \geq 0\ , R_0(p) \geq 0\ , \in\ [-\tau, 0] \tag{3.3}$$

The following theorem establishes the positivity and boundedness of the solution with initial functions satisfying (3.2) and (3.3)

Theorem 3.1

Let (SVAIR) be the solution of the model equation 3.1 satisfying the condition 3.2 and 3.3. then S,V,A,I and R are all positive and bounded for all $t > 0$ at which the solution exist.

Proof

From the equation 3.1 we have

$$S(t) = S(0)e^{-\int_0^t(\omega+\beta_1 I_\tau+\beta_2 I_\tau+\gamma_1)d\theta} + \int_0^t\left(p + (\omega + \beta_1 I_\tau + \beta_2 I_\tau + \gamma_1)\right)S(\varphi)e^{\int_0^t(\omega+\beta_1 I_\tau+\beta_2 I_\tau+\gamma_1)d\theta}d\varphi \quad (3.4)$$

From 3.4 it is clearly seen that $S(t) > 0$ hence positive. Similarly, it can be shown that

$$V(t) > 0, A(t) > 0, I(t) > 0, R(t) > 0 \quad (3.5)$$

Using equation 3.2 and 3.3, positivity follows immediately from the above integral forms.

For boundedness we define

$$N(t) = S(t) + V(t) + A(t) + I(t) + R(t) \quad (3.6)$$

$$\frac{dN(t)}{dt} = \frac{d(S(t) + V(t) + A(t) + I(t) + R(t))}{dt} \quad (3.7)$$

$$\frac{dN(t)}{dt} = p - \gamma\big(S(t) + V(t) + A(t) + I(t) + R(t)\big) \quad (3.8)$$

Thus $\frac{dN(t)}{dt} \leq p - \Sigma$ (3.9)

Where $\Sigma = \gamma S + \gamma V + \gamma A + \gamma I + \gamma R$ (3.10)

This implies that $N(t)$ is bounded and so are

$S(t) + V(t) + A(t) + I(t) + R(t)$. This complete the proof of theorem 3.1

3.4.2 Computation of basic reproduction number R_0

Infections produced by a single infectious individual during the course of their infectious period are known as the basic reproduction number. R_0 is the threshold value for infectious disease models because when $R_0 < 1$, the disease dies off, indicating that the disease free equilibrium state (DFE) is stable and when $R_0 > 1$, the DFE is unstable, meaning that the disease spread throughout the population.

In this study we adopted the next generation matrix (NGM) approach in computation of R_0 such that $R_0 = \rho FV^{-1}$ where ρ is the spectral radius for NGM while F is the matrix of new infection by the disease and V is the matrix of transfer of infection in and out of the compartment by other means. The dominant eigenvalue of the NGM is considered as R_0. In the model equation 3.1, there are two infectious classes; A and I hence at disease free equilibrium, the matrix of new infection is given by matrix F and V.

Matrix F_i is obtained by considering the rate of new infections entering the compartment i is given by;

$$F_i = \begin{bmatrix} \beta_2 S I_\tau + \varepsilon_2 V_\tau I_\tau \\ \beta_1 S\, I_\tau + \varepsilon_1 V_\tau I_\tau + \delta_1 A_\tau \end{bmatrix} \quad (3.11)$$

The matrix that represents the rate of transfer into and out of compartment by other means at DFE is given by;

$$V_i = \begin{bmatrix} \delta_1 A_\tau + \delta_2 A_\tau + \gamma_3 A \\ \alpha I_\tau + (\gamma_4 + \mu) I \end{bmatrix} \tag{3.12}$$

We then differentiate F_i partially with respect to the state variables to obtain a 2×2 matrix

$$F_i = \begin{bmatrix} 0 & (\varepsilon_2 V_\tau + \beta_2 S\,) e^{-\lambda_\tau} \\ \delta_1 e^{-\lambda_\tau} & (\varepsilon_1 V_\tau + \beta_1 S) e^{-\lambda_\tau} \end{bmatrix} \tag{3.13}$$

Similarly, differentiating matrix V_i partially with respect to the state variables, we obtain a 2×2 matrix,

$$V_i = \begin{bmatrix} (\delta_1 + \delta_2) e^{-\lambda_\tau} + \gamma_3 & 0 \\ 0 & \alpha e^{-\lambda_\tau} + (\gamma_4 + \mu) \end{bmatrix} \tag{3.14}$$

The inverse of V is computed as

$$V^{-1} = \begin{bmatrix} \frac{1}{(\delta_1+\delta_2)e^{-\lambda_\tau}+\gamma_3} & 0 \\ 0 & \frac{1}{\alpha e^{-\lambda_\tau}+(\gamma_4+\mu)} \end{bmatrix} \tag{3.15}$$

Using the next generation matrix (NGM) to determine the basic reproduction number R_0

$$NGM = FV^{-1} = \begin{bmatrix} 0 & \frac{(\varepsilon_2 V_\tau+\beta_2 S\,)e^{-\lambda_\tau}}{\alpha e^{-\lambda_\tau}+(\gamma_4+\mu)} \\ \frac{\delta_1 e^{-\lambda_\tau}}{(\delta_1+\delta_2)e^{-\lambda_\tau}+\gamma_3} & \frac{(\varepsilon_1 V_\tau+\beta_1 S)e^{-\lambda_\tau}}{\alpha e^{-\lambda_\tau}+(\gamma_4+\mu)} \end{bmatrix} \tag{3.16}$$

The eigenvalues of matrix (3.16) are computed by $|J - I\lambda| = 0$, where J is the matrix FV^{-1} and I is 2×2 identity matrix.

$$\begin{vmatrix} 0-\lambda & \frac{(\varepsilon_2 V_\tau+\beta_2 S\,)e^{-\lambda_\tau}}{\alpha e^{-\lambda_\tau}+(\gamma_4+\mu)} \\ \frac{\delta_1 e^{-\lambda_\tau}}{(\delta_1+\delta_2)e^{-\lambda_\tau}+\gamma_3} & \frac{(\varepsilon_1 V_\tau+\beta_1 S)e^{-\lambda_\tau}}{\alpha e^{-\lambda_\tau}+(\gamma_4+\mu)} - \lambda \end{vmatrix} \tag{3.17}$$

Solving for the eigenvalues we obtain;

$$\left.\begin{aligned} \lambda_{*1} &= 0 \\ \lambda_{*2} &= \frac{(\varepsilon_1 V_\tau+\beta_1 S)e^{-\lambda_\tau}}{\alpha e^{-\lambda_\tau}+(\gamma_4+\mu)} \end{aligned}\right\} \tag{3.18}$$

The dominant eigenvalue is;

$$\lambda_{*2} = \frac{(\varepsilon_1 V_\tau+\beta_1 S)e^{-\lambda_\tau}}{\alpha e^{-\lambda_\tau}+(\gamma_4+\mu)} \tag{3.19}$$

$$\text{Therefore, } R_{0\ (model\ 3.1)} = \lambda_{*2} = \frac{(\varepsilon_1 V_\tau+\beta_1 S)e^{-\lambda_\tau}}{\alpha e^{-\lambda_\tau}+(\gamma_4+\mu)} \tag{3.20}$$

3.5 Local stability of disease free equilibrium

Mathematical epidemiology considers two equilibria points; Disease Free Equilibrium Point (DFE) where $R_0 < 1$ and Endemic Equilibrium Point (EEP) where $R_0 > 1$. DFE occurs in absence of the disease while EEP occurs

in presence of a disease. A system is stable if all the eigenvalues of the linearized matrix about a fixed point have negative real parts. The dominant eigenvalue of the Jacobian matrix obtained during disease DFE yield the basic reproduction number R_0 which determine the stability of the disease. DFE is an equilibrium point described when the rate of change is equal to zero. It is evaluated by equating the system of delay differential equations (3.1) to zero. The Jacobian matrix is obtained from the first partial derivative of the model equations 3.1 in which the DFE is locally asymptotically stable if $R_0 < 1$, otherwise unstable. To obtain the Jacobian matrix we differentiate equation (3.1) with respect to state variables at DFE.

$$\left.\begin{array}{l} p - \omega S - \beta_1 S I_\tau - \beta_2 S I_\tau - \gamma_1 S = 0 \\ \omega S - \varepsilon_1 V_\tau I_\tau - \varepsilon_2 V_\tau I_\tau - \varepsilon_3 V_\tau I_\tau - \gamma_2 V = 0 \\ \beta_2 S I_\tau + \varepsilon_2 V_\tau I_\tau - \delta_1 A_\tau - \delta_2 A_\tau - \gamma_3 A = 0 \\ \beta_1 S I_\tau + \varepsilon_1 V_\tau I_\tau + \delta_1 A_\tau - \alpha I_\tau - (\gamma_4 + \mu) I = 0 \\ \alpha I_\tau + \delta_2 A_\tau + \varepsilon_3 V_\tau I_\tau - \gamma_5 R = 0 \end{array}\right\} \quad (3.21)$$

The linearization matrix is given by;

$$\begin{bmatrix} -(\omega + \beta_1 I_\tau + \beta_2 I_\tau + \gamma_1) & 0 & 0 & 0 & 0 \\ \omega & -(\varepsilon_1 + \varepsilon_2 + \varepsilon_3) I_\tau e^{-\lambda_\tau} - \gamma_2 & 0 & -(\varepsilon_1 + \varepsilon_2 + \varepsilon_3) v_\tau e^{-\lambda_\tau} & 0 \\ \beta_2 I_\tau & \varepsilon_2 I_\tau e^{-\lambda_\tau} & -(\delta_1 + \delta_2) e^{-\lambda_\tau} - \gamma_3 & (\beta_2 S + \varepsilon_2 V_\tau) e^{-\lambda_\tau} & 0 \\ \beta_1 I_\tau & \varepsilon_1 I_\tau e^{-\lambda_\tau} & \delta_1 e^{-\lambda_\tau} & -(\alpha - \beta_1 s - \varepsilon_1 v_\tau) e^{-\lambda_\tau} - (\gamma_4 + \mu) & 0 \\ 0 & \varepsilon_3 I_\tau e^{-\lambda_\tau} & \delta_2 e^{-\lambda_\tau} & (\alpha + \varepsilon_3 v_\tau) e^{-\lambda_\tau} & -\gamma_5 \end{bmatrix} \quad (3.22)$$

The equation 3.1 is locally asymptotically stable if all the eigenvalues of the linearization matrix of the equation 3.1 are negative. The eigenvalues are:

$$\begin{aligned} \lambda_{**1} &= -(\omega + \beta_1 I_\tau + \beta_2 I_\tau + \gamma_1) \\ \lambda_{**2} &= -(\varepsilon_1 + \varepsilon_2 + \varepsilon_3) I_\tau e^{-\lambda_\tau} - \gamma_2 \\ \lambda_{**3} &= -(\delta_1 + \delta_2) e^{-\lambda_\tau} - \gamma_3 \\ \lambda_{**4} &= -(\alpha - \beta_1 s - \varepsilon_1 v_\tau) e^{-\lambda_\tau} - (\gamma_4 + \mu) \\ \lambda_{**5} &= -\gamma_5 \end{aligned} \quad (3.23)$$

For the model 3.1, it is clear that the dominant eigenvalue is;

$$\lambda_{**4} = -(\alpha - \beta_1 s - \varepsilon_1 v_\tau) e^{-\lambda_\tau} - (\gamma_4 + \mu) \quad (3.24)$$

Theorem 3.2

Disease free equilibrium is stable whenever $R_0 < 1$, otherwise it's unstable.

Proof

λ_{**4} should be negative. This can only be negative if,

$$-(\alpha - \beta_1 s - \varepsilon_1 v_\tau) e^{-\lambda_\tau} - (\gamma_4 + \mu) = 0$$

Which simplifies to

$$(\beta_1 s + \varepsilon_1 v_\tau) e^{-\lambda_\tau} < \alpha e^{-\lambda_\tau} + (\gamma_4 + \mu) \quad (3.25)$$

This implies that;

$$R_0 = \frac{(\beta_1 s + \varepsilon_1 v_\tau) e^{-\lambda_\tau}}{\alpha e^{-\lambda_\tau} + (\gamma_4 + \mu)} < 1 \quad (3.26)$$

Therefore $R_0 < 1$ is obtained for DFE to be stable.

4 Numerical Analysis and Results

This section comprises of parameter values obtained from literature and graphs obtained using Matlab. (Table 4.1)

Table 1. Table of parameters, parameter description, values and source

Parameter	Description	Value	Source
S	Susceptible class	$5.036 \times 10^7\ years^{-1}$	Estimated from (Muchiri et al., 2022)
V	Coronavirus disease vaccinated class	0	(Carpio et al., 2021)
A	Asymptomatic infected class	$7322\ years^{-1}$	(Carpio et al., 2021)
I	Symptomatic infected class	$7545\ years^{-1}$	Estimated from (Muchiri et al., 2022)
R	Recovered class	$203968\ years^{-1}$	Estimated from (Muchiri et al., 2022)
p	Recruitment rate	$1762\ days^{-1}$	Estimated from (Muchiri et al., 2022)
ω	Rate of progression from S to V	$0.002\ days^{-1}$	Assumed
β_1	Transition rate from susceptible to infectious class	$0.233\ days^{-1}$	(Chae et al., 2020)
β_2	Transition rate from susceptible to asymptomatic class	$0.09786\ days^{-1}$	(Algarni et al., 2022)
ε_1	Rate of progression from V to I	$0.462\ days^{-1}$	Chae et al., 2020
ε_2	Rate of progression from V to A	$0.039\ days^{-1}$	(Li et al., 2020)
$\frac{1}{\varepsilon_3}$	Immunity development period	$0.0714\ days^{-1}$	(Algarni et al., 2022)
δ_1	Rate of progression from A to I	$0.1428\ days^{-1}$	(Algarni et al., 2022)
δ_2	Rate of progress from A to R	$0.165\ days^{-1}$	(Buonomo et al., 2022)
α	Rate of progression from I to R	$0.1\ days^{-1}$	(Algarni et al., 2022)
γ	Natural death rate	$1.07 \times 10^{-2}\ years^{-1}$	(Buonomo et al., 2022)
μ	Disease induced death rate	$6.248 \times 10^{-4}\ days^{-1}$	(Buonomo et al., 2022)
τ	Time delay	To be determined	

Fig. 2 shows the dynamics of population of various compartments against time in days. From the graph, it is evident that nearly the entire population were susceptible from the onset and as the infections number increases, the number of susceptible individual drastically decreases to about 20 percent because vaccination, treatment and time delay played a major role in stabilizing the rate of infection. Increasing vaccination coverage means immunity of the susceptible individual is improved hence the infectious period delayed and therefore the basic reproduction number is lowered, minimizing the rate of infection.

Fig. 3 shows that the reproduction number R_0 is directly proportion to the rate of death due to COVID-19 (μ) unless intervention measures like vaccination is rolled out which increases the latent period hence R_0 is lowered resulting in reduced death rate.

Fig. 4 shows a plot of reproduction number against the rate of progression from asymptomatic (A) to infectious (I). It is evident that R_0 is directly proportional to the rate of progression δ. A shorter asymptomatic infectious period potentially increases the value of R_0 hence higher rate of progression from A to I.

Fig. 5 shows a plot of reproduction number against time delay at DFE. From the graph R_0 is inversely proportional to time delay τ. A shorter τ leads to a higher R_0, therefore highly contagious variants spread faster before intervention measures can take effect.

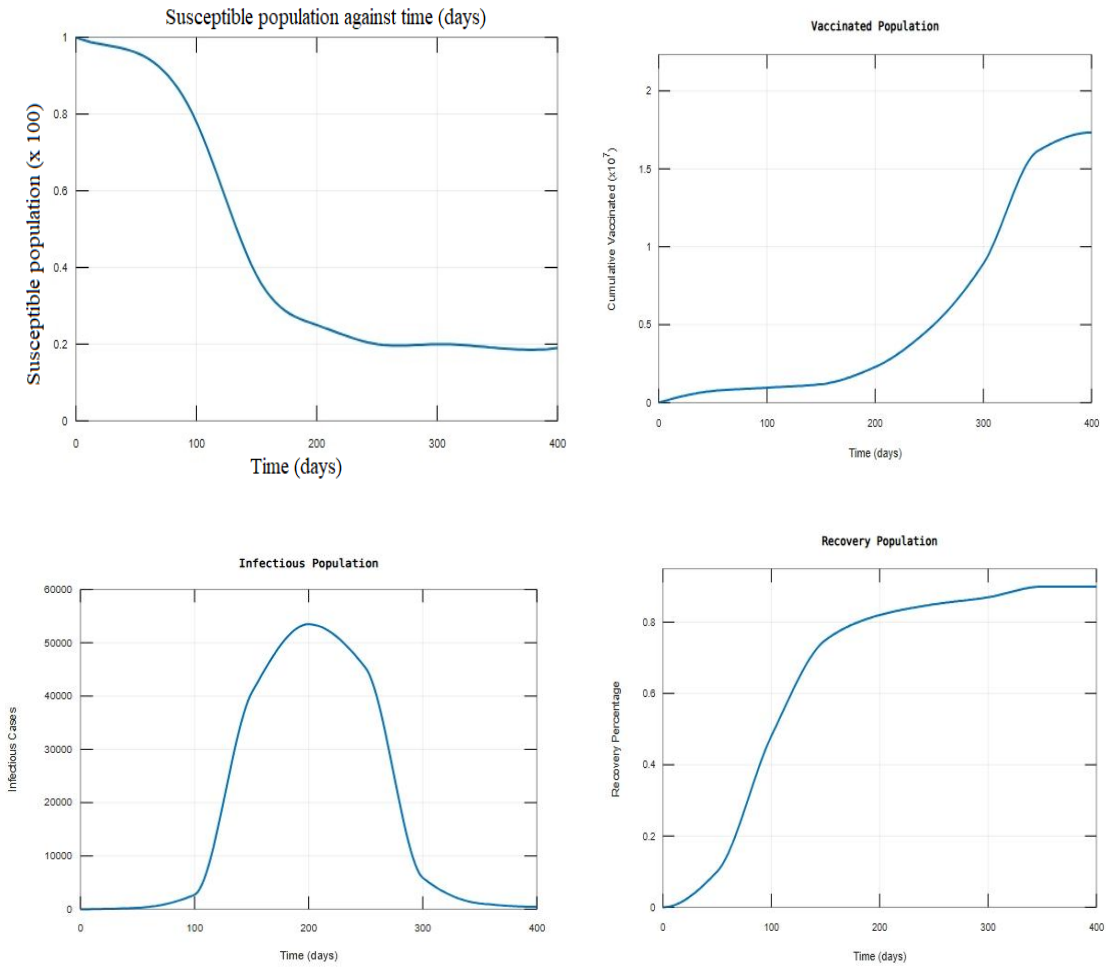

Fig. 2. A plot of SVAIR model population against time

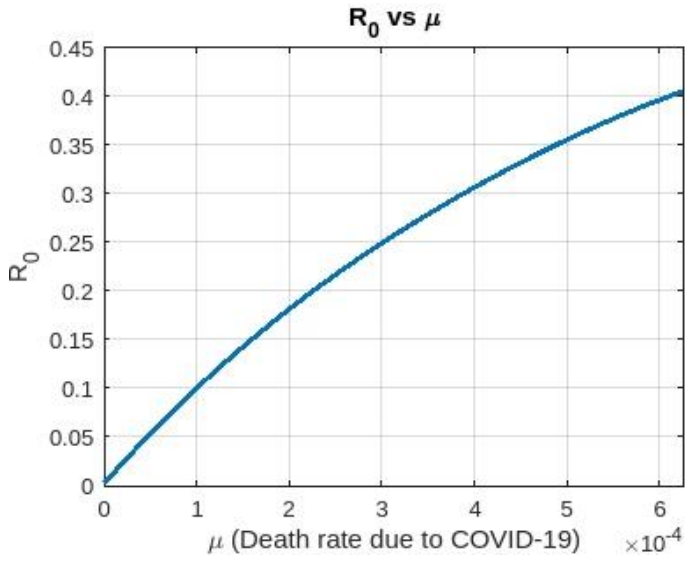

Fig. 3. A plot of R_0 against Death rate due to COVID-19 (μ)

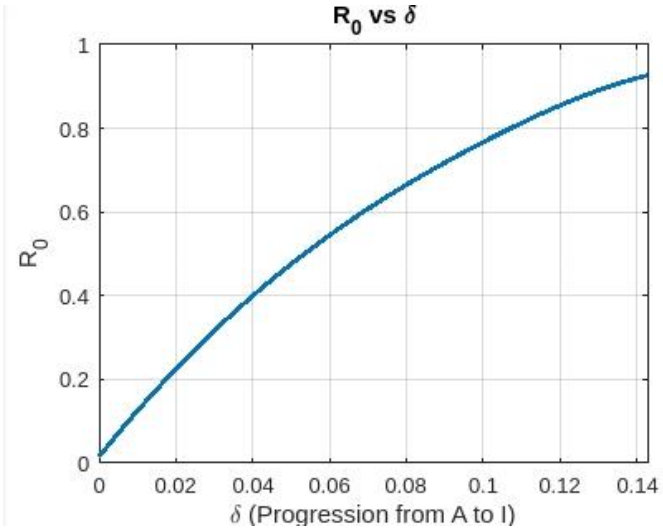

Fig. 4. A plot of R_0 at EEP against Delta (δ)

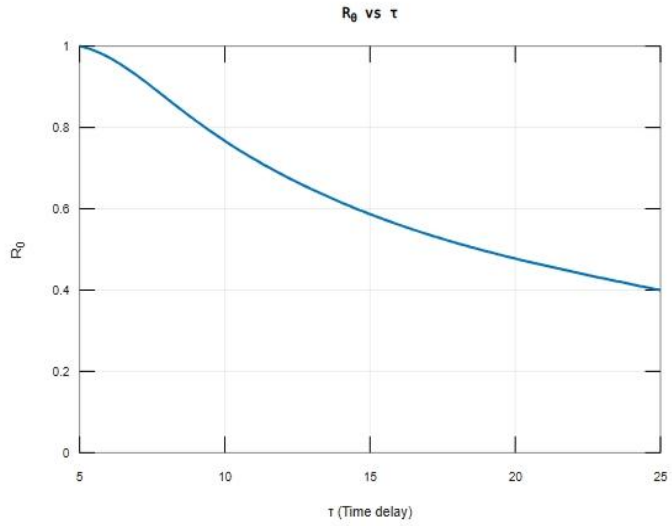

Fig. 5. A plot of R_0 against Time delay (τ)

5 Conclusion

It was found out that the model was stable at both Disease Free Equilibrium (DFE) and Endemic Equilibrium Point (EEP) and was attained when $R_0 < 1$ and $R_0 > 1$ respectively. Parameter values were taken from the literature, and the analytical findings were verified numerically using the Matlab dde 23 solver. This was affected by vaccination, time delay and the rate of progression from asymptomatic class to infectious compartment. From the study it is evident that for time delay $\tau > 5$ days, DFE is stable otherwise unstable. Numerical simulation was carried out to validate the analytic results where it was found out that $R_0 = 0.95$ at DFE. Furthermore the results show that stability was guaranteed at EEP and is attained at $R_0 = 1.02$. Time delay, rate of vaccination and rate of progression from asymptomatic to infectious plays an important role in the transmission of COVID-19 and stability at DFE and EEP.

Suggestions for Further Research

This study has not exhausted all about COVID-19 transmission dynamics. Further studies can be done on COVID-19 vaccine efficacy. The model can be extended to include co-infection between COVID-19 and TB or any other

underlying conditions. Further studies can be done on the effects of partially and fully vaccinated individuals on COVID-19 transmission dynamics.

Disclaimer (Artificial Intelligence)

I 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

Authors have declared that no competing interests exist.

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