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MATHEMATICAL MODELING OF COVID-19 WITH INTERVENTION: A CASE STUDY OF NIGERIA

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MATHEMATICAL MODELING OF COVID-19 WITH INTERVENTION: A CASE STUDY OF NIGERIA

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Dedication

I dedicate this thesis to Almighty God and my parents.

Abstract

The outbreak of COVID-19 disease caused by the SARS-CoV-2 in 2019 has claimed over 6.3 million lives. The pandemic prompted many countries to set up some preventive measures as means to control the spread of the disease. In this thesis, a deterministic system of coupled differential equations is proposed to study the transmission of COVID-19 among a well-mixed population with intervention strategies. The existence and uniqueness of the classical solution of the COVID-19 model are proved. The equilibrium points of the model are analyzed and, the basic reproduction number is obtained. The local asymptotic stability and the global asymptotic stability of the model are carried out. An adaptive Dormand–Prince numerical method is used to obtain approximate solution of the model. The results shows that combined control parameters may reduce the burden of COVID-19 faster in the population. In addition, the outcomes of this study show that in order to mitigate the spread of COVID-19 in the overall population, non-pharmaceutical intervention strategies such as social distancing, self-isolation, and hand washing should be practiced at the maximum and people should be vaccinated.

Keywords: COVID-19, Local stability, Global stability, Basic reproduction number, Numerical simulation.

Nomenclature

The state variables and parameters are described below.

State variable and Description	
State variable	**Description**
S	Susceptible Individuals
E	Exposed Individuals
I	Infected Individuals
Q	Quarantined Individuals
R	Recovered Individuals
V	Vaccinated Individuals
N	Total population

Parameters and Description	
Parameter	**Description**
Λ	Recruitment rate
κ	Parameter corresponding to the combination of the following measures such as Social-distancing, Self-isolation, and Hand-washing
ϕ_1, ϕ_2, ϕ_3	Vaccination rate in the S, E, I compartment
θ, ω	Loss of vaccine immunity and natural immunity
μ, δ	Natural and Induced death rate respectively
β	Effective transmission rate
q	Population of the isolated Infected Individuals
α	Progression rate of Exposed Individuals to the infected compartment
γ_1, γ_2	Rate of recovery of Infected and Quarantined Individuals

Contents

Chapter 1

Introduction

1.1 Introduction to COVID-19

On 11 March 2020, the World Health Organization (WHO) declared COVID-19 a pandemic. Since the first case of infection with this new coronavirus was reported in China in December 2019, SARS-CoV-2, as we now know it to be called, has killed over 6 million people with total cases of more than 600 million reported cases [30]. Since the initial outbreak of the coronavirus, it has since spread to almost every country in the world. The broad family of viruses known as coronaviruses are known to cause illnesses ranging from the common cold to more serious conditions like Severe Acute Respiratory Syndrome (SARS) and Middle East Respiratory Syndrome (MERS).[3]. COVID-19 is spread by dust particles and fomites when the infector and the infected person come into close, dangerous contact. Airborne distribution has not been recorded for COVID-19 and is not known to be a significant transmission medium based on empirical evidence, although it can be imagined if such aerosol-generating practices are carried out in medical facilities. Faecal spread has been seen in certain patients, and the active virus has been reported in a small number of clinical studies. Furthermore, the faecal-oral route does not seem to be a COVID-19 transmission medium; its function and relevance for COVID-19 need to be identified. For about 18,738,580 laboratory-confirmed cases recorded as of the second week of April 2020, the maximum number of cases (77.8%) was between 30 and 69 years of age. Among the recorded cases, 21.6% are farmers or employees by profession, 51.1% are male, and 77.0% are Hubei [13].

Although, COVID-19 first appeared in 2019, it wasn't until February 27, 2020, that it reached Nigeria. Through the Federal Ministry of Health, the Nigerian government has been stepping up efforts to promptly contain and control any outbreaks there. In order to respond to this case and put strict control measures in place, the multi-sector Coronavirus Preparedness Group, coordinated by the Nigeria Centre for Disease Control (NCDC), launched its National Emergency Operations Centre. The first phase of lockdown in Nigeria was initiated in April 2020. Prior to the shutdown, crowd space and even religious homes and

gatherings were restricted. Later, a number of lock-downs occurred since it was difficult to stop the spread. As time went on, restrictions and a complete lockdown were declared. As the economy remained stagnant, the government was given palliatives, which were then dispersed throughout the states of Nigeria. Despite the abundance of the palliatives, numerous news outlets reported that many were receiving minimal amounts [25].

During lock down, some businesses were suspended. In other states, such as Lagos, people are only permitted to leave their homes during certain hours while wearing protective gear, such as face guards and nasal masks. With all the measures in place, the virus did not stop spreading while families, individuals, and businesses were significantly affected by the stringent measures put in place. COVID-19 vaccines was introduced to Nigeria in March 2021. The vaccination exercise was opened with the highest officials in the nation getting vaccinated. Despite the examples set by the officials, most Nigerians were very hesitant to take the jabs. In recent times, it has lately been noted that many people have taken both the first and second doses of the vaccination because the vaccination certificate is required to access facilities and some other privileges. Despite the federal government lifting the restriction certain places (particularly in the banking industry) continue to use the "no mask, no entry" policy. The vaccination certificate is now a must because it is one of the essential documents one needs to have.

Several articles have numerically studied COVID-19 and developed mathematical models to understand the spread of the deadly COVID-19 disease. Only a few of them, however, have talked about how vaccinations can help stop the spread of the virus. However, to restrict the spread of COVID-19, this study provides a mathematical model of the effects of vaccines and other non-pharmaceutical control measures.

1.2 Objectives of the Study

The general objective of this work is to develop a mathematical model that has the potential to predict the impact of both vaccination and non-pharmaceutical intervention strategies against COVID-19 mortality and morbidity in Nigeria.

The specific objectives of the study are as follows:

1. To formulate the mathematical model and establish its well-posedness.
2. To determine the existence of the equilibrium points of the formulated model.
3. To establish the basic reproduction number using the next-generation matrix approach.
4. To establish the local asymptotic stability of the disease-free equilibrium points.
5. To establish the global asymptotic stability of the disease-free equilibrium points using the Lyapunov function method.

6. To simulate numerically the model to determine the effects of the model parameters on the infection with COVID-19 within the Nigerian population.

1.3 Significance of the Study

The mathematical model developed in this thesis provides a potential approach to predict the impact of both vaccination and non-pharmaceutical intervention strategies against COVID-19 mortality and morbidity in Nigeria. This study will be a useful reference for researchers conducting future studies on the impact of vaccination with or without additional control intervention measures in reducing COVID-19 spread in Nigeria. The results from this study will assist vaccine developers in evaluating and assessing the impact of vaccinations in preventing COVID-19 in the health sector. As main outcome, the model may become a prediction tool for public health policymakers to determine the particular conditions in which the disease-modified vaccine will be effective, as well as different intervention measures that will be advantageous for epidemic control.

1.4 Definitions of Used Terms

The following are key definitions of terminology and concepts used in this study. Most of the definitions can be found in Martcheva's book [21].

Mathematical Model: A mathematical model is a description of a system or a process using mathematical concepts and language.

Autonomous System: An ordinary differential equation is called an autonomous system if it is of the form;

$$\dot{x} = f(x) \qquad x \in \mathbb{R}^n (n \in \mathbb{N}), \tag{1.1}$$

where $\dot{x}$ denotes the derivative with respect to a smooth function x.

Equilibrium Point: A point $x = x^0 \in \mathbb{R}^n$ is an equilibrium point of system (1.1) if $f(x^0) = 0$.

Stable Equilibrium Point: An equilibrium point $x^0 \in \mathbb{R}^n$ is said to be stable if given $\epsilon > 0$ there exists $\delta = \delta(\epsilon) > 0$ such that, any solution $y(t) \in C^1(t_0, \infty)$ of (1.1) satisfies

$$|x^0 - y(t)| < \epsilon, \quad \text{whenever} \quad |x^0 - y(t_0)| < \delta,$$

for $t > t_0 \in \mathbb{R}$.

Asymptotically Stable: The equilibrium x^0 is said to be asymptotically stable if there exists a constant $c > 0$ such that, for any solution $y(t) \in C^1(t_0, \infty)$ of (1.1) satisfying $|x^0 - y(t_0)| < c$, then

$$\lim_{t \to \infty} |x^0 - y(t)| = 0.$$

Unstable: An equilibrium point x^0 that is not stable is said to be unstable.

Jacobian matrix: The Jacobian matrix $J(x)$ of the function $f : \mathbb{R}^n \to \mathbb{R}^n$ is defined as the matrix,

$$J(x) = \begin{pmatrix} \frac{\partial f_1}{\partial x_1}(x) & \cdots & \frac{\partial f_1}{\partial x_n}(x) \\ \vdots & \ddots & \vdots \\ \frac{\partial f_n}{\partial x_1}(x) & \cdots & \frac{\partial f_n}{\partial x_n}(x) \end{pmatrix}.$$

Disease Free Equilibrium: The disease-free equilibrium (DFE) is defined as the equilibrium point at which there is absence of disease in the population.

Susceptible individuals: A susceptible individual is a member of a population who is at risk of becoming infected by a disease.

Exposed or Latent individuals: These are individuals who are infected with the disease but not yet infectious.

Infectious individuals: These are individuals who have acquired the disease and can infect others.

Quarantined individuals: These are individuals who have been restricted or separated from individuals who were exposed to the disease to see if they become sick.

Vaccinated individuals: These are individuals who received vaccine against a particular disease or those that are immunized.

Recovered or Removed individuals: These are individuals who have been infected and have either recovered from the disease and entered the removed compartment or died. It is assumed that the number of deaths is negligible with respect to the total population.

Prevalence: The prevalence of a disease is the number of people who have the disease at a specific time divided by the total population size.

Disease Induced Mortality: This is the number of people who have died from the disease in one unit of time (e.g., one year) divided by the entire population.

Chapter 2

Review of Literature

One of the most aggressive human pathogens affecting our nowadays society is the Coronavirus. This virus attacks the respiratory system of the human body, and this has remained a worldwide risk. The infection was at first called "novel coronavirus 2019" (2019-nCoV) on the 12th of January 2020 by the World Health Organization (WHO), and officially named "severe acute respiratory syndrome coronavirus-2" (SARS-CoV-2) by the international committee of the Coronavirus Study Group (CSG), and WHO calls the disease "coronavirus disease 2019" (COVID-19) [14], [20]. SARS-CoV-2 is a specific virus that can cause COVID-19, a disease. The first outbreak originated in Wuhan, China in December 2019. COVID-19 subsequentially spread globally to become the fifth documented pandemic since the 1918 flu pandemic. SARS-CoV-2 is an enveloped and spherical particle carrying a positive-sense single-stranded RNA genome and it belongs to the subfamily coronavirinae, family coronavirdae, and order Nidovirales [19]. Previous studies showed that every human coronavirus have animal origins called natural hosts of HCOV-229E, SARS-COV, HCOV-NL63, and MERS-COV. Bats are really important and they are the major natural reservoirs of alpha-coronaviruses and beta-coronaviruses [35].

Bats are presumably reservoir hosts for SARS-COV2 and there was no intermediate host sample obtained by Scientists in an initial cluster of infections at the Huanan Seafood and Wildlife Market in Wuhan, China, where the sale of wild animals may be the source of zoonotic infection [16]. SARS-COV-2 is transmitted from person to person via direct contact or through droplets spread by coughing or sneezing from an infected individual [35]. Typically, droplets can only travel a distance of two meters and hang in the air for a short period of time. However, SARS-COV-2 remains intact and contagious in droplets (less than five microns in diameter) and can be suspended in the air for up to three hours [11]. Transmission of SARS-COV-2 from mother to fetus child could not be ruled out as there was a recent studies that showed Immunoglobin M (IgM) antibodies to SARS-COV-2 were present in newborn infant blood [20], [10], [38]. The symptoms of COVID-19 infection appear after an incubation period of approximately 5.2 days [19]. The period from the onset of COVID-19 symptoms to death ranged from 6 to 41 days with a median of 14 days [35]. This period is dependent on the age of the patient and status of the patient's immune

system. It was shorter among patients above 70-years old compared with those under the age of 70 [35]. The majority of COVID-19 infection's clinical signs are comparable to those of the other two coronaviruses in the same family. Fever, headache, myalgia, diarrhoea, dry cough, nausea, chest discomfort, exhaustion, and dyspnea are some of the symptoms [16], [15]. In severe cases, symptomatically infected persons often have moderate-to-severe respiratory symptoms, which can lead to severe pneumonia [39]. Few COVID-19 infected patients have strong upper respiratory tract signs and symptoms such as sneezing or sore throat, in contrast to SARS-CoV and MERS-CoV infections, suggesting that SARS-CoV-2 prefers to infect the lower respiratory tract [16]. Arrhythmia, hypoxemia, acute cardiac injury, acute ARDS, acute kidney injury, and shock have also been observed in COVID-19 patients [16], [4].

The COVID-19 epidemic has had far-reaching consequences, affecting not only people's lives and health, but also the environment. It has not only highlighted the poor health infrastructure in the world, even in the most developed countries, but has also had a negative impact on the global economy. Almost the entire planet is gridlocked, all economic and commercial activity came to a standstill. It has stunned the world's largest economies, namely China and the United States, where there has been an economic downturn since the coronavirus pandemic [33]. Third World countries, such as Nigeria, are among the hardest hit by the economic crisis. In recent months, millions of people have lost their jobs. Debt repayment is impossible for poor countries. Nigeria, the most populous country in Africa with over 200 million inhabitants [2], is the main target of economic devastation. Millions of people have not recovered from the poor economy caused by COVID-19. In Nigeria, as in other nations throughout the world, the COVID-19 pandemic poses a serious threat to human health and the economy. This virus is much more damaging in Nigeria because non-pharmaceutical therapies and vaccinations are extremely difficult to administer in a country like Nigeria. When the first case was confirmed, the government could not impose a nationwide lockdown. The first COVID-19 case was reported in Lagos State on February 27, 2020, when an Italian national tested positive for the virus [9]. Approximately 255,924 cases have been confirmed, 249,961 patients have been discharged, 5,152,011 samples have been examined, and 3,143 people have died due to the infection's spread [8].

Mathematical models are extremely important in understanding the transmission dynamics and control of emerging and re-emerging infectious diseases. Predicting the severity of the COVID-19 pandemic and suggesting appropriate public health response techniques are two of humanity's most pressing concerns today. A number of mathematical models have recently been presented to investigate the COVID-19 pandemic's transmission patterns. Okounghae et al. [27] presented a mathematical model to investigate the effects of non-pharmaceutical control measures on the population dynamics of the novel coronavirus disease 2019. In their study, they provided forecasts for the cumulative number of reported cases and active cases for different levels of the control measures used in their model. They were able to achieve the fundamental reproduction number, and their numerical simulations demonstrate that the disease will eventually die out in the population if at least 55% per cent of the population adheres to social distancing

and the use of face masks while in public. The authors advised policymakers and others in positions of authority to aggressively screen and test persons in the public for new cases of COVID-19 symptomatic or asymptomatic infection, as well as to strictly enforce the usage of face masks and social distance measures. During the early outbreaks in Kano, Nigeria, Salihu S. et al. [22] employed mathematical modelling to estimate the number of COVID-19 underascertained η and the basic reproduction number R_0. Their findings resemble an exponential development pattern that resembles the epidemic curve of COVID-19 in Kano. According to their research, the number of COVID-19 instances is underreported, and this occurred in the fourth week of April 2020. They suggested that epidemiological studies and mitigation actions should be prioritized in the near future, as this will help to prevent COVID-19 from spreading in Kano, Nigeria. Their findings also show that the basic reproduction number R_0, which is 2.74 (95% CI : 2.53–2.96), has the potential to cause massive epidemics. Their forecasts are crucial for future epidemic response. Iboi et al. [17] developed a mathematical model to investigate COVID-19 transmission dynamics and control in Nigeria. In several Nigerian jurisdictions, data from the Nigeria Centre for Disease Control (NCDC) was utilized to assess the community-wide impact of various control and mitigation techniques. Their research found that COVID-19 can be effectively controlled in Nigeria if control and mitigation techniques are able to achieve the $R_c < 1$ threshold quantity. Furthermore, COVID-19 will be impossible to produce substantial outbreaks in Nigeria if rigorous social distancing measures are established (and maintained for a lengthy period of time).

Deressa et al. [6] proposed a mathematical model to predict COVID-19 transmission dynamics in Ethiopia. Their findings revealed that the disease-free and endemic equilibrium points for $R_0 < 1$ and $R_0 > 1$, respectively, are asymptotically stable locally and globally. The basic reproduction number $R_0 = 1.5085$ is greater than 1. However, in order to minimize the R_0, the goal must be to lower the transmission rate from asymptotically infected to suspected individuals (α), at the very least lowering the parameter α to 0.47, which will reduce the coronavirus from Ethiopia. The ideal mix of public health education, personal preventative measures, and treatment of hospitalized or isolated cases, according to the results from the optimal analysis and simulation, would considerably limit the COVID-19 pandemic in Ethiopia. Finally, their research findings can be used as policy input by the Ethiopian government and other countries. The government must take the required steps to ensure that the following preventative techniques are implemented consistently throughout the epidemic. Adewole et al. [1] created a deterministic model that describes COVID-19 dynamics in Nigeria. The basic reproduction number was calculated and used to assess the model's disease-equilibrium solution's stability. The model's essential parameters were estimated using data from the Nigeria Centre for Disease Control (NCDC). The authors investigated cost-effective solutions for time-independent controls to suppress virus transmission within a given time frame using Pontryagin's maximal principle. Their findings suggest that rigorous adherence to WHO recommendations, good contact tracing, and population testing for COVID-19 may all be accomplished in a short amount of time to prevent the disease spread. Oke et al. [26] theoretically examined the impact of disregarding

asymptomatic patients on the transmission of COVID-19 in Africa. They measured the basic reproduction number and checked for backward bifurcation. Additionally, the Local and Global asymptotic Stabilities were established. Their findings indicate that increasing case detection to find infected people who are asymptomatic will be very successful in limiting and lessening the impact of COVID-19 in Africa. Enforcing a living arrangement where recovered individuals are not permitted to mix with the susceptible or exposed individuals will also aid in restricting the spread of COVID-19 because it is unknown if a recovered individual can become re-infected or not.

Chapter 3

Methodology

3.1 Formulation of the Governing Equations

We assume that the total human population at a time $t \geqslant 0$ denoted by $N(t)$ is divided into mutually exclusive sub-populations of Susceptible humans $S(t)$, Exposed humans $E(t)$, Infected humans $I(t)$, Quarantined humans $Q(t)$, Recovered humans $R(t)$, and Vaccinated humans $V(t)$. Thus,

$$N(t) = S(t) + E(t) + I(t) + Q(t) + R(t) + V(t) \text{ for all } t \geqslant 0.$$

Figure 3.1 shows the Epidemic Interactions among the human compartments. The Susceptible humans are represented as S and the human populations are recruited into the S-compartment at a constant rate $\Lambda \in (0, \infty)$. Introducing the parameter κ that represents intervention strategies into the force of infection ξ. The force of infection ξ is strictly positive and depends on κ. Susceptible humans become exposed when they come into contact with infectious humans at the transmission rate $\beta \in (0, 1)$. The parameters ϕ_1, ϕ_2, and ϕ_3 represent the vaccination rate for humans in the Susceptible, Exposed, and Infected compartments, respectively. The parameters γ_1 and γ_2 represent the recovery rate of infected humans and quarantined humans, respectively. Vaccines reduce the risk of COVID-19, including the risk of several illnesses and death among people who are fully vaccinated. Most people who get COVID-19 got it when they were unvaccinated. However, in this study, we assume that the vaccines are not 100% effective at preventing infection, some people who are fully vaccinated will still get COVID-19. The parameter $\theta \in (0, 1)$ is the loss of immunity gained from vaccination, the parameter δ is the induced death rate, and the parameter ω is the natural loss of immunity after recovery.

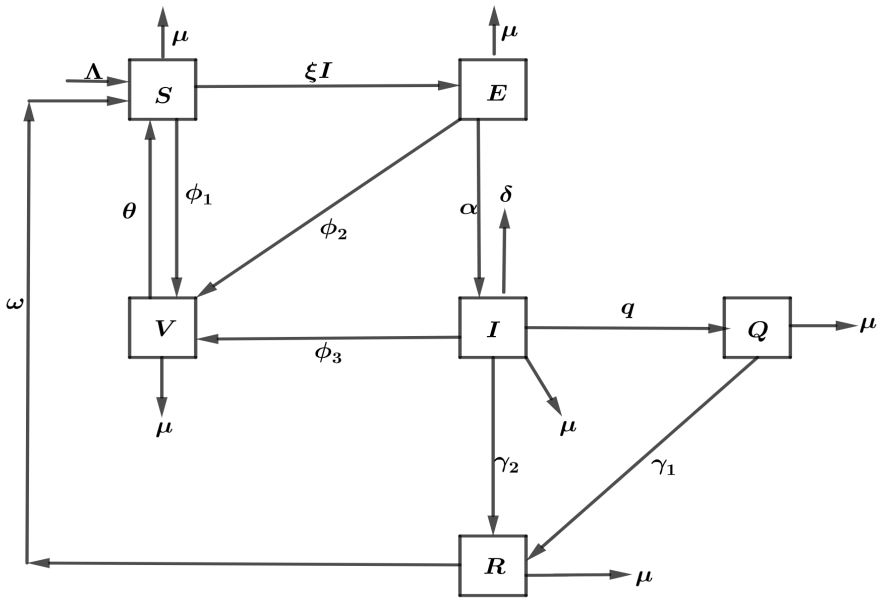

Figure 3.1: A scheme showing Epidemic Interactions among the human compartments.

The model for COVID-19 transmission dynamics in a population is given by the system of deterministic coupled non-linear differential equations (3.1), which is based on the assumptions utilized in the design of the COVID-19 model. We look for (S, E, I, Q, R, V) that are $(C^1(0,T) \times C^0(0,T))^6$ for some $T > 0$ that satisfy the ordinary differential equation

$$\begin{cases} \frac{dS}{dt} = \Lambda - \xi IS - \mu S - \phi_1 S + \theta V + \omega R, & t > 0 \\ \frac{dE}{dt} = \xi IS - \mu E - \phi_2 E - \alpha E, & t > 0 \\ \frac{dI}{dt} = \alpha E - (\gamma_2 + \mu + q + \phi_3 + \delta)I, & t > 0 \\ \frac{dQ}{dt} = qI - (\mu + \gamma_1)Q, & t > 0 \\ \frac{dR}{dt} = \gamma_2 I + \gamma_1 Q - (\mu + \omega)R, & t > 0 \\ \frac{dV}{dt} = \phi_1 S + \phi_2 E + \phi_3 I - (\mu + \theta)V, & t > 0 \end{cases} \tag{3.1}$$

with initial conditions

$$S(0) = s_0 > 0, E(0) = e_0 > 0, I(0) = i_0 > 0, Q(0) = q_0 > 0, V(0) = v_0 > 0, R(0) = r_0 > 0, \tag{3.2}$$

where $(\Lambda, \xi, \phi_1, \phi_2, \phi_3, \theta, \omega, \mu, \delta, \beta, q, \alpha, \kappa, \gamma_1, \gamma_2) \in (0, \infty)^{15}$ are positive real parameters, and the force of infection is

$$\xi = \frac{\beta(1-\kappa)}{N} \tag{3.3}$$

originally suggested by [27].

3.2 Qualitative Analysis of the Model

3.2.1 Positivity and Boundedness of Solutions

To investigate the dynamics of any disease, it is important to understand the behavior of the solution over time t. For human disease modeling, it is important to show that the solutions will be positive and bounded over a long time. Let $t_0 > 0$, then for all $t \in [0, t_0]$, the values of the parameters can be chosen so that the solution $S(t), E(t), I(t), Q(t), R(t)$, and $V(t)$ to ODE (3.1) with the positive initial conditions as defined in equation (3.2).

Lemma 3.2.1 (Boundedness)**.** *Positive constants $S_c, E_c, I_c, Q_c, V_c, R_c$, and V_c exists for the solutions $(S(t), E(t), I(t), Q(t), V(t), R(t))$, of the model equation (3.1)-(3.2) such that $\lim_{t\to\infty} \sup S(t) \leqslant S_c$, $\lim_{t\to\infty} \sup E(t) \leqslant E_c$, $\lim_{t\to\infty} \sup I(t) \leqslant I_c$, $\lim_{t\to\infty} \sup Q(t) \leqslant Q_c$, $\lim_{t\to\infty} \sup R(t) \leqslant R_c$, and $\lim_{t\to\infty} \sup V(t) \leqslant V_c$, for all $t \in [0, t_0]$, $t_0 > 0$ and the solution vector is positive.*

Proof. To show boundedness, we add all the equations in (3.1) and we obtain
$\frac{dN}{dt} = \Lambda - \mu N - \delta I \leqslant \Lambda - \mu N$.
It then follows that $\frac{dN}{dt} \leqslant 0$, if $N \geqslant \frac{\Lambda}{\mu}$. Thus solving $\frac{dN}{dt} \leqslant \Lambda - \mu N$ by applying Gronwall's inequality leads to,

$$N(t) \leqslant N(0)e^{-\mu t} + (1 - e^{-\mu t})\frac{\Lambda}{\mu} \leqslant N(0) + \frac{\Lambda}{\mu}, \text{ which is bounded.}$$

In particular, if $N(0) \leqslant \frac{\Lambda}{\mu}$, then

$$N(t) \leqslant N(0)e^{-\mu t} + (1 - e^{-\mu t})\frac{\Lambda}{\mu} \leqslant \frac{\Lambda}{\mu}e^{-\mu t} + \frac{\Lambda}{\mu} - \frac{\Lambda}{\mu}e^{-\mu t} = \frac{\Lambda}{\mu}.$$

Thus, the region

$$D = [(S, E, I, Q, R, V) \in \mathbb{R}^6_+ : S(t) + E(t) + I(t) + Q(t) + R(t) + V(t) \leqslant \frac{\Lambda}{\mu}],$$

is positively invariant region for the model (3.1). Furthermore, If $N(0) > \frac{\Lambda}{\mu}$ then either the solution of the model (3.1) enters D in a finite time or $N(t)$ approaches $\frac{\Lambda}{\mu}$ asymptotically.
Hence, region D attracts all solutions of the model (3.1) in $\mathbb{R}^6_+$. □

3.2.2 Existence and Uniqueness Theorems

Equation (3.1) is an autonomous system with f defined in equation (3.4). Thus, we have,

$$\begin{cases} f_1(S,E,I,Q,R,V) = \Lambda - \xi IS - \mu S - \phi_1 S + \theta V + \omega R, \\ f_2(S,E,I,Q,R,V) = \xi IS - \mu E - \phi_2 E - \alpha E, \\ f_3(S,E,I,Q,R,V) = \alpha E - (\gamma_2 + \mu + q + \phi_3 + \delta)I, \\ f_4(S,E,I,Q,R,V) = qI - (\mu + \gamma_1)Q, \\ f_5(S,E,I,Q,R,V) = \gamma_2 I + \gamma_1 Q - (\mu + \omega)R, \\ f_6(S,E,I,Q,R,V) = \phi_1 S + \phi_2 E + \phi_3 I - (\mu + \theta)V. \end{cases} \tag{3.4}$$

We use the following theorem to establish the existence and uniqueness of solution for the model (3.1). To prove this, we need the following standard results

Theorem 3.2.2 (Existence and Uniqueness Theorem)**.** *Define the domain D such that $D = \{|t - t_0| \leqslant a, \quad \|X - X_0\| \leqslant b\} \subseteq C^1$ where $X = (S,E,I,Q,R,V)^T$ and $X_0 = (s_0, e_0, i_0, q_0, r_0, v_0)^T$.*
Then, the problem (3.1) written in matrix form

$$\dot{X} = f(X)$$

has a unique solution in D if $f(X)$ is continuous in D and there exist constants $K_1, K_2, K_3, K_4, K_5, K_6$ such that
$\left|\frac{\partial f}{\partial S}\right| < K_1$, $\left|\frac{\partial f}{\partial E}\right| < K_2$, $\left|\frac{\partial f}{\partial I}\right| < K_3$, $\left|\frac{\partial f}{\partial Q}\right| < K_4$, $\left|\frac{\partial f}{\partial R}\right| < K_5$, $\left|\frac{\partial f}{\partial V}\right| < K_6$

Proof. We shall show that the solution to the system (3.4) exists and the solution is unique.

Existence Theorem. Clearly, the functions $f_1, \cdots, f_6$ as defined in (3.4) are continuous and bounded in D, hence, there exists a solution in D.

Uniqueness Theorem. It is left to prove the uniqueness of the solution. To show that there is a unique solution to the system of the equations (3.4), we shall show that $\frac{\partial f_i}{\partial x_j}, i, j = 1, 2...6$ are continuous and bounded. We explore the following partial derivatives for all the model equations (3.4). Starting from the first equation in the functions (3.4), we have

$$\frac{\partial f_1}{\partial S} = -\xi I - (\mu + \phi_1), \quad \left|\frac{\partial f_1}{\partial S}\right| = |-\xi I - (\mu + \phi_1)| \leqslant K < \infty$$
$$\frac{\partial f_1}{\partial E} = 0, \quad \left|\frac{\partial f_1}{\partial E}\right| = |0| < \infty, \quad \frac{\partial f_1}{\partial I} = -\xi S - (\mu + \phi_1)S$$
$$\left|\frac{\partial f_1}{\partial I}\right| = |-\xi S - (\mu + \phi_1)S| < \infty \quad \frac{\partial f_1}{\partial Q} = 0, \quad \left|\frac{\partial f_1}{\partial Q}\right| = |0| < \infty$$
$$\frac{\partial f_1}{\partial R} = \omega, \quad \left|\frac{\partial f_1}{\partial R}\right| = |\omega| < \infty, \quad \frac{\partial f_1}{\partial V} = \theta, \quad \left|\frac{\partial f_1}{\partial V}\right| = |\theta| < \infty.$$

Similarly by differentiating the function f_2 in (3.4), we have

$$\frac{\partial f_2}{\partial S} = \xi I, \quad \left|\frac{\partial f_2}{\partial S}\right| = |\xi I| \leqslant K < \infty$$
$$\frac{\partial f_2}{\partial E} = -(\mu + \phi_2 + \alpha), \quad \left|\frac{\partial f_2}{\partial E}\right| = |-(\mu + \phi_2 + \alpha)| \leqslant K < \infty$$
$$\frac{\partial f_2}{\partial I} = \xi S, \quad \left|\frac{\partial f_2}{\partial I}\right| = |\xi S| \leqslant K < \infty$$
$$\frac{\partial f_2}{\partial Q} = 0, \quad \left|\frac{\partial f_2}{\partial Q}\right| = |0| < \infty, \quad \frac{\partial f_2}{\partial R} = \omega, \quad \left|\frac{\partial f_2}{\partial R}\right| = |\omega| < \infty, \quad \frac{\partial f_2}{\partial V} = \theta, \quad \left|\frac{\partial f_2}{\partial V}\right| = |\theta| < \infty.$$

By differentiating the third function equation in (3.4), we have

$$\frac{\partial f_3}{\partial S} = 0, \quad \left|\frac{\partial f_3}{\partial S}\right| = |0| < \infty, \quad \frac{\partial f_3}{\partial E} = \alpha, \quad \left|\frac{\partial f_3}{\partial E}\right| = |\alpha| \leqslant K < \infty,$$
$$\frac{\partial f_3}{\partial I} = -(\gamma_2 + \mu + q + \phi_3 + \delta), \quad \left|\frac{\partial f_3}{\partial I}\right| = |-(\gamma_2 + \mu + q + \phi_3 + \delta)| \leqslant K < \infty$$
$$\frac{\partial f_3}{\partial Q} = 0, \quad \left|\frac{\partial f_3}{\partial Q}\right| = |0| < \infty, \quad \frac{\partial f_3}{\partial R} = 0, \quad \left|\frac{\partial f_3}{\partial R}\right| = |0| < \infty, \quad \frac{\partial f_3}{\partial V} = 0, \quad \left|\frac{\partial f_3}{\partial V}\right| = |0| < \infty.$$

In the functions, the fourth equation in (3.4) is taken into consideration, and by differentiating, we have

$$\frac{\partial f_4}{\partial S} = 0, \quad \left|\frac{\partial f_4}{\partial S}\right| = |0| < \infty, \quad \frac{\partial f_4}{\partial E} = 0, \quad \left|\frac{\partial f_4}{\partial E}\right| = |0| < \infty, \quad \frac{\partial f_4}{\partial I} = q, \quad \left|\frac{\partial f_4}{\partial I}\right| = |q| \leqslant K < \infty,$$
$$\frac{\partial f_4}{\partial Q} = (\mu + \gamma_1), \quad \left|\frac{\partial f_4}{\partial Q}\right| = |(\mu + \gamma_1)| \leqslant K < \infty \quad \frac{\partial f_4}{\partial R} = 0, \quad \left|\frac{\partial f_4}{\partial R}\right| = |0| < \infty \quad \frac{\partial f_4}{\partial V} = 0, \quad \left|\frac{\partial f_4}{\partial V}\right| = |0| < \infty$$

In the functions, beginning with the fifth equation in (3.4), and by differentiating we have

$$\frac{\partial f_5}{\partial S} = 0, \quad \left|\frac{\partial f_5}{\partial S}\right| = |0| < \infty \quad \frac{\partial f_5}{\partial E} = 0, \quad \left|\frac{\partial f_5}{\partial E}\right| = |0| < \infty \quad \frac{\partial f_5}{\partial I} = \gamma_2, \left|\frac{\partial f_5}{\partial I}\right| = |\gamma_2| \leqslant K < \infty \quad \frac{\partial f_5}{\partial Q} = \gamma_1,$$
$$\left|\frac{\partial f_5}{\partial Q}\right| = |\gamma_1| \leqslant K < \infty \quad \frac{\partial f_5}{\partial R} = -(\mu + \omega), \quad \left|\frac{\partial f_5}{\partial R}\right| = |-(\mu + \omega)| \leqslant K < \infty \quad \frac{\partial f_5}{\partial V} = 0, \quad \left|\frac{\partial f_5}{\partial V}\right| = |0| < \infty.$$

Finally, based on the sixth equation in the functions (3.4), and by differentiating we have

$$\frac{\partial f_6}{\partial S} = \phi_1, \quad \left|\frac{\partial f_6}{\partial S}\right| = |\phi_1| \leqslant K < \infty, \quad \frac{\partial f_6}{\partial E} = \phi_2, \quad \left|\frac{\partial f_6}{\partial E}\right| = |\phi_2| \leqslant K < \infty$$
$$\frac{\partial f_6}{\partial I} = \phi_3, \quad \left|\frac{\partial f_6}{\partial I}\right| = |\phi_3| \leqslant K < \infty \quad \frac{\partial f_6}{\partial Q} = 0, \quad \left|\frac{\partial f_6}{\partial Q}\right| = |0| < \infty$$
$$\frac{\partial f_6}{\partial R} = 0, \quad \left|\frac{\partial f_6}{\partial R}\right| = |0| < \infty \quad \frac{\partial f_6}{\partial V} = -(\mu + \theta), \quad \left|\frac{\partial f_6}{\partial V}\right| = |-(\mu + \theta)| < \infty$$

Hence, we have clearly established that all these partial derivatives are continuous and bounded.

Therefore, there exists a unique solution of model equation (3.4).

□

3.2.3 Equilibrium Points

Disease Free Equilibrium (DFE) in the COVID-19 model (3.1)-(3.2), i.e. an equilibrium without the virus. In this case, $I = E = 0$ and $\dot{X} = 0$ are equivalent to $f(X) = 0$ where $X = (S, E, I, Q, R, V)$.

Hence, we need to solve the following system to find the corresponding DFE.

$$\begin{cases} \Lambda - (\mu + \phi_1)S + \theta V = 0 \\ \phi_1 S - (\mu + \theta)V = 0 \\ qI - (\mu + \gamma_1)Q = 0 \\ \gamma_2 I + \gamma_1 Q - (\mu + \omega)R = 0 \end{cases} \tag{3.5}$$

Solving equation (3.5), we obtain

$$S = \frac{\Lambda(\mu + \theta)}{\mu(\mu + \theta + \phi_1)}, R = 0, Q = 0, \text{ and } V = \frac{\phi_1 \Lambda}{\mu(\mu + \theta + \phi_1)}.$$

Hence, we have

$$\varepsilon^* = (S^*, E^*, I^*, Q^*, R^*, V^*) = \left(\frac{\Lambda(\mu + \theta)}{\mu(\mu + \theta + \phi_1)}, 0, 0, 0, 0, \frac{\phi_1 \Lambda}{\mu(\mu + \theta + \phi_1)} \right).$$

An endemic disease equilibrium (EDE) of the model (3.1)-(3.2) is obtained by setting all states $\dot{X} = 0$ which is equivalent to $f(X) = 0$ and we have;

$$\begin{cases} \Lambda - \xi I^{**} S^{**} - \mu S^{**} - \phi_1 S^{**} + \theta V^{**} + \omega R^{**} = 0 \\ \xi I^{**} S^{**} - \mu E^{**} - \phi_2 E^{**} - \alpha E^{**} = 0 \\ \alpha E^{**} - (\gamma_2 + \mu + q + \phi_3 + \delta) I^{**} = 0 \\ qI^{**} - (\mu + \gamma_1) Q^{**} = 0 \\ \gamma_2 I^{**} + \gamma_1 Q^{**} - (\mu + \omega) R^{**} = 0 \\ \phi_1 S^{**} + \phi_2 E^{**} + \phi_3 I^{**} - (\mu + \theta) V^{**} = 0 \end{cases} \tag{3.6}$$

From the third and the fourth equations, we have

$$E^{**} = \frac{\gamma_2 + \mu + q + \phi_3 + \delta}{\alpha} I^{**} \tag{3.7}$$

$$Q^{**} = \frac{qI^{**}}{\mu + \gamma_1} \tag{3.8}$$

Rearranging the fifth equation of the system (3.6) and substituting Q^{**}, we have

$$R^{**} = \frac{\gamma_2 I^{**} + \gamma_1 Q^{**}}{\mu + \omega} \tag{3.9}$$

$$= \frac{((\mu + \gamma_1)\gamma_2 + q\gamma_1) I^{**}}{(\mu + \gamma_1)(\mu + \omega)} \tag{3.10}$$

Adding the first and the second equations of the system (3.6), we have

$$\Lambda - (\mu + \phi_1) S^{**} + \theta V^{**} + \omega R^{**} - (\mu + \phi_2 + \alpha) E^{**} = 0 \tag{3.11}$$

Rearranging the sixth equation of the system (3.6) and also rearranging equation (3.11), we have the system of two equations

$$(\mu + \phi_1)\, S^{**} - \theta V^{**} = \Lambda + \omega R^{**} - (\mu + \phi_2 + \alpha)\, E^{**} \tag{3.12}$$

$$-\phi_1 S^{**} + (\mu + \theta) V^{**} = \phi_2 E^{**} + \phi_3 I \tag{3.13}$$

Solving the equations for S^{**} and V^{**}, we have

$$S^{**} = \frac{\Lambda(\mu+\theta) - (\mu(\mu+\phi_2+\alpha+\theta)+\theta\alpha)E^{**} + \theta\phi_3 I^{**} + (\mu+\theta)\omega R^{**}}{\mu(\mu+\phi_1+\theta)} \tag{3.14}$$

$$V^{**} = \frac{\phi_1\Lambda(\mu+\theta) - (\phi_2 - \phi_1\mu(\mu+\phi_2+\alpha+\theta)+\phi_1\theta\alpha)E^{**} + \phi_3(\theta\phi_1+1)I^{**} + (\mu+\theta)\omega\phi_1 R^{**}}{\mu(\mu+\phi_1+\theta)(\mu+\theta)} \tag{3.15}$$

By compiling the results, the endemic equilibrium point is

$$\begin{cases} S^{**} = \frac{\Lambda(\mu+\theta) - \left(\mu(\mu+\phi_2+\alpha+\theta)+\theta\alpha\right)E^{**} + \theta\phi_3 I^{**} + (\mu+\theta)\omega R^{**}}{\mu(\mu+\phi_1+\theta)} \\ E^{**} = \frac{(\gamma_2+\mu+q+\phi_3+\delta)I^{**}}{\alpha}, \quad Q^{**} = \frac{qI^{**}}{(\mu+\gamma_1)} \quad R^{**} = \frac{((\mu+\gamma_1)\gamma_2+q\gamma_1)I^{**}}{(\mu+\gamma_1)(\mu+\omega)} \\ V^{**} = \frac{\phi_1\Lambda(\mu+\theta) - \left(\phi_2 - \phi_1\mu(\mu+\phi_2+\alpha+\theta)+\phi_1\theta\alpha\right)E^{**} + \phi_3(\theta\phi_1+1)I^{**} + (\mu+\theta)\omega\phi_1 R^{**}}{\mu(\mu+\phi_1+\theta)(\mu+\theta)} \end{cases} \tag{3.16}$$

3.2.4 Basic Reproduction Number

The basic reproduction number R_0, is one of the most important key parameters, that study the ease-time dynamics of infectious disease. It helps in determining whether or not an infectious disease will spread through the population. It is defined as the number of secondary infections caused by a single infected person in a completely susceptible population.
Essentially, it holds:

- If $R_0 < 1$, then the disease will die out.
- If $R_0 > 1$, this is called an epidemic, that is, the disease will spread.

This study employs the Van den Driessche and Watmough methods for establishing R_0 [34]. A strategy for obtaining the next-generation matrix using ordinary differential equations compartmental models for disease transmission is included in this method. Infected compartments and non-infected (healthy) compartments will be separated into two categories. If the people in a compartment are infected, it's called an infected compartment. If the people in a compartment are infected but not infectious, it's called a latent compartment. The non-infected compartments are the rest of the compartments.
In our model (3.1)-(3.2) there are 2 infected compartments and 4 non-infected compartments, the entire ordinary differential equation model has $4 + 2$ dependent variables. Let z be the vector of the dependent

variables in the infected compartments, and let y be the vector of variables in the non-infected compartments.We have $z = (E, I)^T \in \mathbb{R}^2$ and $y = (S, Q, R, V)^T \in \mathbb{R}^4$. The method below was introduced in [21, 34]. The following steps illustrate how to establish the basic reproduction number R_0.

Step 1: We arrange the equations such that the first 2 components of the ODE system correspond to the infected compartments. Thus, we denote by $\dot{z}$ and $\dot{y}$ the original ODE of the system

$$\dot{z} = \begin{pmatrix} \dot{z}_1 \\ \dot{z}_2 \end{pmatrix} = \begin{cases} \xi IS - (\mu + \alpha + \phi_2)E, \\ \alpha E - (\gamma_2 + \mu + q + \phi_3 + \delta)I \end{cases} \tag{3.17}$$

$$\dot{y} = \begin{pmatrix} \dot{y}_1 \\ \dot{y}_2 \\ \dot{y}_3 \\ \dot{y}_4 \end{pmatrix} = \begin{cases} \Lambda - \xi IS - \mu S - \phi_1 S + \theta V + \omega R, \\ qI - (\mu + \gamma_1)Q, \\ \gamma_2 I + \gamma_1 Q - (\mu + \omega)R, \\ \phi_1 S + \phi_2 E + \phi_3 I - (\mu + \theta)V \end{cases} \tag{3.18}$$

Step 2: We split the right-hand side in the infected compartments in the following way:

$$\dot{z}_i = \mathcal{F}_i - \mathcal{V}_i, \qquad \dot{y}_j = g_j \tag{3.19}$$

$i = 1, ...2$, $j = 1, ...4$ and $\mathcal{V}_i = \mathcal{V}_i^- - \mathcal{V}_i^+$

where

- the function $\mathcal{F}_i$ represents the rate of appearance of new infections in compartment i.
- the function $\mathcal{V}_i$ incorporates the remaining transitional terms, namely births, deaths, disease progression, etc.
- the function $\mathcal{V}_i^+ \geqslant 0$ represents the rate of transfer of individuals into compartment i, and
- the function $\mathcal{V}_i^- \geqslant 0$ represents the rate of transfer of individuals out of compartment i.

The decomposition satisfies the following properties:

Property 1. $\mathcal{F}_i(0, y) = 0$ and $\mathcal{V}_i(0, y) = 0$ for $y \geqslant 0$ and $i = 1, ...2$. The first condition says that all new infections are secondary infections arising from infected hosts. The second conditions says that there is no immigration of susceptible individuals into the disease compartments.

Property 2. $\mathcal{F}_i(z, y) \geqslant 0 \ \forall \ z, y \geqslant 0$

Property 3. $\mathcal{V}_i(z, y) \leqslant 0$ whenever $z_i = 0$ for $i = 1, ...2$. Each component $\mathcal{V}_i$ represents the net outflow of a compartment and must give inflow only (that is, be negative) if the compartment is empty.

Property 4. $\sum_{i=1}^{n} \mathcal{V}_i(z, y) \geqslant 0 \ \forall \ z, y \geqslant 0$. The total outflow of all infected compartments is positive.

Step 3: Assume that the disease-free system

$$\dot{y} = g(0, y)$$

has a unique disease free-equilibrium $\epsilon^* = (0, y_0)$ such that all solutions with initial conditions of the form $(0, y)$ approach $(0, y_0)$ as $t \to \infty$. Determine the disease-free equilibrium ϵ_0.

Step 4: Determine the matrices F and V with components

$$f_{ii} = \left[\frac{\partial \mathcal{F}_i(0,y_0)}{\partial z_j}\right] \quad \text{and} \quad v_{ii} = \left[\frac{\partial \mathcal{V}_i(0,y_0)}{\partial z_j}\right] \tag{3.20}$$

These matrices appear from the linearization of the system (3.19) around the DFE. Since $\mathcal{F}_i(0, y) = 0$ and $\mathcal{V}_i(0, y) = 0$ by Property 1, it holds that

$$\frac{\partial \mathcal{F}_i(0, y_0)}{\partial y_j} = \frac{\partial \mathcal{V}_i(0, y_0)}{\partial y_j} = 0$$

for every pair (i, j). This implies that the linearized equations for the infected compartments z while computed at the DFE are decoupled from the remaining equations. The linearized system for the infected compartments can be written as

$$\dot{z} = (F - V)z,$$

where the F and V matrices are defined above.

Step 5: The next generation matrix is defined as

$$R_0 = \rho(\mathbf{F}\mathbf{V}^{-\mathbf{1}}) = \rho(K),$$

where $\mathbf{F}$, $\mathbf{V}$ are the Jacobian of F, V respectively and $\rho(K)$ denotes the spectral radius of K.

Spectral radius of a matrix: The spectral radius of a square matrix is the maximum of the absolute values of its eigenvalues.

For the model under study, we find the Basic Reproduction number R_0 following the 5 steps illustrated above. First, we regroup the model (3.1) into the Infected compartment and Non-Infected compartment. The infected compartments are:

$$\begin{cases} \frac{dE}{dt} = \xi IS - (\mu + \alpha + \phi_2)E \\ \\ \frac{dI}{dt} = \alpha E - (\gamma_2 + \mu + q + \phi_3 + \delta)I. \end{cases} \tag{3.21}$$

The Non-infected compartments are:

$$\begin{cases} \frac{dS}{dt} = \Lambda - \xi IS - \mu S - \phi_1 S + \theta V + \omega R \\ \\ \frac{dQ}{dt} = qI - (\mu + \gamma_1)Q \\ \\ \frac{dR}{dt} = \gamma_2 I + \gamma_1 Q - (\mu + \omega)R \\ \\ \frac{dV}{dt} = \phi_1 S + \phi_2 E + \phi_3 I - (\mu + \theta)V. \end{cases} \tag{3.22}$$

The compartments that are infected and not infected are described in the F and V matrix below.

$$F := \begin{pmatrix} \xi IS \\ \\ 0 \end{pmatrix} \text{ and } V := \begin{pmatrix} -(\mu + \alpha + \phi_2)E \\ \\ +\alpha E - (\gamma_2 + \mu + q + \phi_3 + \theta)I \end{pmatrix}.$$

The Jacobian of F and V are computed as follows

$$\mathbf{F}(\varepsilon^*) = \boldsymbol{\nabla} F(\varepsilon^*) = \begin{pmatrix} 0 & \xi S \\ 0 & 0 \end{pmatrix}, \ \mathbf{V} = \boldsymbol{\nabla} V = \begin{pmatrix} (\mu + \alpha + \phi_2) & 0 \\ -\alpha & (\gamma_2 + \mu + q + \phi_3 + \delta) \end{pmatrix}.$$

Hence, at the Disease Free Equilibrium,

$$\begin{aligned} \mathbf{FV^{-1}}(\varepsilon^*) &= \begin{pmatrix} 0 & \xi S^* \\ \\ 0 & 0 \end{pmatrix} \begin{pmatrix} \frac{1}{(\mu+\alpha+\phi_2)} & 0 \\ \\ \frac{\alpha}{(\mu+\alpha+\phi_2)(\gamma_2+\mu+q+\phi_3+\delta)} & \frac{1}{(\gamma_2+\mu+q+\phi_3+\delta)} \end{pmatrix} \\ &= \begin{pmatrix} \frac{\alpha\xi S^*}{(\mu+\alpha+\phi_2)(\gamma_2+\mu+q+\phi_3+\delta)} & \frac{\xi S^*}{(\gamma_2+\mu+q+\phi_3+\delta)} \\ \\ 0 & 0 \end{pmatrix}. \end{aligned}$$

Computing eigenvalues of $(\mathbf{FV^{-1}})$, and replacing the results of the disease-free equilibrium ε^*, we have $\lambda_1 = 0$ and

$$\lambda_2 = \frac{\alpha\xi(\mu + \theta)}{(\mu + \theta + \phi_1)(\mu + \alpha + \phi_2)(\gamma_2 + \mu + q + \phi_3 + \delta)}$$

By picking the highest dominant value, then we get the basic reproduction number

$$R_0 = \frac{\alpha\xi(\mu + \theta)}{(\mu + \theta + \phi_1)(\mu + \alpha + \phi_2)(\gamma_2 + \mu + q + \phi_3 + \delta)}.$$

3.3 Local Stability

Theorem 3.3.1. *The DFE is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.*

Proof. The Jacobian matrix evaluated at the DFE state for system (3.1) is given by

$$J(\varepsilon^*) = \begin{pmatrix} -(\mu+\phi_1) & 0 & \xi S^* & 0 & \omega & \theta \\ 0 & -(\mu+\alpha+\phi_2) & \xi S^* & 0 & 0 & 0 \\ 0 & \alpha & -(\gamma_2+\mu+q+\phi_3+\delta) & 0 & 0 & 0 \\ 0 & 0 & q & -(\mu+\gamma_1) & 0 & 0 \\ 0 & 0 & \gamma_2 & \gamma_1 & -(\mu+\omega) & 0 \\ \phi_1 & \phi_2 & \phi_3 & 0 & 0 & -(\mu+\theta) \end{pmatrix}$$

For simplicity, Let:= $a_1 = (\mu+\phi_1)$, $a_2 = \xi S^*$, $a_3 = (\mu+\alpha+\phi_2)$, $a_4 = (\gamma_2+\mu+q+\phi_3+\delta)$, $a_5 = (\mu+\gamma_1)$, $a_6 = (\mu+\omega)$, and $a_7 = (\mu+\theta)$.

By substituting the notation above, we have

$$det(J(\varepsilon^*) - \lambda I) = \begin{vmatrix} -a_1-\lambda & 0 & a_2 & 0 & \omega & \theta \\ 0 & -a_3-\lambda & a_2 & 0 & 0 & 0 \\ 0 & \alpha & -a_4-\lambda & 0 & 0 & 0 \\ 0 & 0 & q & -a_5-\lambda & 0 & 0 \\ 0 & 0 & \gamma_2 & \gamma_1 & -a_6-\lambda & 0 \\ \phi_1 & \phi_2 & \phi_3 & 0 & 0 & -a_7-\lambda \end{vmatrix}$$

Next, to compute the eigenvalues of $J(\varepsilon^*)$, one obtains the following results for the eigenvalues and the remaining terms in the characteristics equation:

$\lambda_1 = -a_1$, $\lambda_2 = -a_5$, $\lambda_3 = -a_6$, $\lambda_4 = -a_7$, and $\lambda^2 + (a_3+a_4)\lambda + (a_3a_4 - a_2\alpha) = 0$.

By applying the Routh–Hurwitz criterion, the quadratic equation will have roots with negative real parts if and only if $(a_3+a_4) > 0$, and $(a_3a_4 - a_2\alpha) > 0$.

Now, considering $(a_3a_4 - a_2\alpha) > 0$, by substituting a_3, a_4, a_2, and S^* back into the inequality expression, we obtain

$$\frac{\alpha\xi(\mu+\theta)}{(\mu+\theta+\phi_1)(\mu+\alpha+\phi_2)(\gamma_2+\mu+q+\phi_3+\delta)} < 1.$$

This implies that, $R_0 < 1$. As a result, the disease-free equilibrium, ε^* is locally asymptotically stable if $R_0 < 1$.

□

The threshold quantity R_0 is the control reproduction number for the model (3.1)-(3.2). It represents the average number of secondary COVID-19 infections generated by a typical infectious individual (infected,

exposed, and vaccinated) in a completely susceptible population where control measures are present. As a consequence of Theorem (3.3.1), biologically speaking, COVID-19 spread can be reduced from the population when $R_0 < 1$ if the initial sizes of the sub-populations of the model are in the region of attraction of the DFE.

3.4 Global Stability of the Disease-Free Equilibrium via Lyapunov Method

Definition 3.4.1. *Let L be a continuous scalar function, $L : \mathbb{R}^n \to \mathbb{R}$, then the function L is called positively definite (or globally positively definite) on the entire space if $L(x^*) = 0$ and $L(x) > 0$ for $x \neq x^*$, where x^* is an equilibrium point of the autonomous system $\frac{dx}{dt} = f(x)$.*

We define the derivative of $L(x)$ along the solutions of the system of differential equations as

$$L'(x) = \frac{d}{dt}L(x(t)) = \frac{\partial L}{\partial x}\frac{dx}{dt}.$$

Now, Lyapunov's theorem for global stability of the equilibrium x^* is given as the following theorem:

Theorem 3.4.1 (Lyapunov's Stability)**.** *If a function $L(x)$ is globally positively definite and radially unbounded, and its time derivative is globally negative definite. $L'(x) < 0$ for all $x \neq x^*$, then the equilibrium x^* is globally stable. We refer the reader to [28], e.g. for a proof of Theorem 3.4.1.*

There are no established rules for finding a Lyapunov function, and finding a Lyapunov function is tricky and computationally intensive. In this work, we adopt an approach by Shuai and Van den Driessche [31] to prove the global asymptotic stability of disease-free equilibrium points (DFE).

Theorem 3.4.2. *Let F and V be defined such that*

$$F = \frac{\partial \mathcal{F}_i}{\partial x_j}(\bar{x}) \geq 0, \qquad V = \frac{\partial \mathcal{V}_i}{\partial x_j}(\bar{x}) \geq 0, \quad \text{with} \quad 1 \leqslant i, j \leqslant m, \tag{3.23}$$

and set

$$f(x, y) = (F - V)x - \mathcal{F}(x, y) + \mathcal{V}(x, y). \tag{3.24}$$

If $f(x, y) \geqslant 0$ in $\Gamma \subset \mathbb{R}^{n+m}_+$, $F \geqslant 0$, $V^{-1} \geqslant 0$, and $R_0 \leqslant 1$, the the function $Q = wV^{-1}x$ is a Lyapunov function for the model (3.1) on Γ.

Proof. To determine the Lyapunov candidate for the Global Asymptotically Stability (GAS) of the DFE, we assume [1]

$$L(E, I) = A_1E + A_2I, \tag{3.25}$$

[1]The disease classes are usually considered when constructing Lyapunov function of infectious diseases modeling.

where the constants A_1 and A_2 are to be determined: Corresponding to the infected classes of the model(3.1), we assume a Peron eigenvector as follows, $w = (w_1, w_2)$. Evaluating $V^{-1}F$ and $w.V^{-1}F = R_0 w^T$, we obtain the choice of the constants

$$A_1 = \frac{R_0\alpha}{(\mu+\alpha+\phi_2)(\gamma_2+\mu+q+\phi_3+\delta)} \text{ and } A_2 = \frac{R_0}{(\gamma_2+\mu+q+\phi_3+\delta)}$$

Hence, the Lyapunov candidate becomes

$$L = \frac{R_0\alpha}{(\mu+\alpha+\phi_2)(\gamma_2+\mu+q+\phi_3+\delta)}E + \frac{R_0}{(\gamma_2+\mu+q+\phi_3+\delta)}I.$$

Taking the derivative with respect to t gives

$$\dot{L} = \frac{R_0\alpha}{(\mu+\alpha+\phi_2)(\gamma_2+\mu+q+\phi_3+\delta)}\dot{E} + \frac{R_0}{(\gamma_2+\mu+q+\phi_3+\delta)}\dot{I}.$$

By substituting the differential equation E and I, we obtain.

$$\begin{aligned}\dot{L} &= \frac{R_0\alpha}{(\mu+\alpha+\phi_2)(\gamma_2+\mu+q+\phi_3+\delta)}\Big[\xi I S^0 - \mu E - \phi_2 E - \alpha E\Big] + \\ &\quad \frac{R_0}{(\gamma_2+\mu+q+\phi_3+\delta)}\Big[\alpha E - (\gamma_2+\mu+q+\phi_3+\delta)I\Big] \\ &= \frac{R_0\alpha\xi S^0 I}{(\mu+\alpha+\phi_2)(\gamma_2+\mu+q+\phi_3+\delta)} - R_0 I.\end{aligned}$$

If we factor out R^0 and substituting S^0, and N^0, we get

$$\dot{L} < R_0\left(\frac{\alpha\xi(\mu+\theta)}{(\mu+\theta+\phi_1)(\mu+\alpha+\phi_2)(\gamma_2+\mu+q+\phi_3+\delta)} - 1\right)I.$$

Again, recall that

$$R_0 = \frac{\alpha\xi(\mu+\theta)}{(\mu+\theta+\phi_1)(\mu+\alpha+\phi_2)(\gamma_2+\mu+q+\phi_3+\delta)}$$

Thus,

$$\dot{L} < R_0(R_0-1)I.$$

Hence, we have that $\dot{L} < 0$ for $R_0 < 1$ with $\dot{L} = 0$ if $I = 0$. Hence, L is a Lyapunov function on D. Thus, by the La Salle's Invariance Principle [18], every solution to the equations (3.1), with initial conditions in D, approaches the DFE ε^* as $t \to 0$ whenever $R_0 < 1$. □

3.5 Sensitivity Analysis of R_0

The sensitivity analysis is used to figure out how important each model parameter is with respect to disease transmission. It is vital to understand the relative relevance of the many elements associated with COVID-19 transmission in Nigeria in order to determine how effectively to prevent human mortality and morbidity caused by its spread. In this thesis, numerical sensitivity indices are used to identify parameters that have a large impact on the basic reproduction number R_0 and should be targeted by intervention

strategies. In conducting the sensitivity analysis, this thesis follows the method introduced by Chen et al.[5].

Definition 3.5.1. *The normalized forward sensitivity index of a function, u that depends differentiably on a parameter p is defined as*

$$\Gamma_p^u = \frac{\partial u}{\partial p}\frac{p}{u}.$$

Given that R_0 has an explicit formula. We calculate the sensitivity of R_0 as

$$\Gamma_p^{R_0} = \frac{\partial R_0}{\partial p} \times \frac{p}{R_0},$$

to each of the fifteen different parameters described in Table 3.1. For example, the sensitivity index of R_0 with respect to α is

$$\Gamma_\alpha^{R_0} = \frac{\partial R_0}{\partial \alpha}\frac{\alpha}{R_0} = 1 - \frac{\alpha}{(\alpha+\mu+\phi_2)} > 0.$$

The sensitivity index of R_0 with respect to θ is

$$\Gamma_\theta^{R_0} = \frac{\partial R_0}{\partial \theta}\frac{\theta}{R_0} = \theta - \frac{\theta}{(\theta+\mu+\phi_1)} > 0, \text{where}(\theta>0, \mu>0, \phi_1>0).$$

The sensitivity index of R_0 with respect to μ is

$$\Gamma_\mu^{R_0} = \frac{\partial R_0}{\partial \mu}\frac{\mu}{R_0} = \mu\left(\frac{1}{(\theta+\mu)} - \frac{1}{(\theta+\mu+\phi_1)} - \frac{1}{(\gamma_2+\mu+q+\delta+\phi_3)} - \frac{1}{(\alpha+\mu+\phi_2)}\right) > 0.$$

The sensitivity index of R_0 with respect to ϕ_1 is

$$\Gamma_{\phi_1}^{R_0} = \frac{\partial R_0}{\partial \phi_1}\frac{\phi_1}{R_0} = \frac{-\phi_1}{(\theta+\mu+\phi_1)} < 0.$$

The sensitivity index of R_0 with respect to ϕ_2 is

$$\Gamma_{\phi_2}^{R_0} = \frac{\partial R_0}{\partial \phi_2}\frac{\phi_2}{R_0} = \frac{-\phi_2}{(\alpha+\mu+\phi_2)} < 0.$$

The sensitivity index of R_0 with respect to ϕ_3 is

$$\Gamma_{\phi_3}^{R_0} = \frac{\partial R_0}{\partial \phi_3}\frac{\phi_3}{R_0} = \frac{-\phi_3}{(\gamma_2+\mu+q+\delta\phi_3)} < 0.$$

The sensitivity index of R_0 with respect to γ_2 is

$$\Gamma_{\gamma_2}^{R_0} = \frac{\partial R_0}{\partial \gamma_2}\frac{\gamma_2}{R_0} = \frac{-\gamma_2}{(\gamma_2+\mu+q+\delta+\phi_3)} < 0.$$

The sensitivity index of R_0 with respect to δ is

$$\Gamma_\delta^{R_0} = \frac{\partial R_0}{\partial \delta}\frac{\delta}{R_0} = \frac{-\delta}{(\gamma_2+\mu+q+\delta+\phi_3)} < 0.$$

The sensitivity index of R_0 with respect to q is

$$\Gamma_q^{R_0} = \frac{\partial R_0}{\partial q}\frac{q}{R_0} = \frac{-q}{(\gamma_2+\mu+q+\delta\phi_3)} < 0.$$

All the results obtained above are shown in Table 3.1.

Table 3.1: Numerical values of sensitivity indices of R_0.

Parameter symbol	**Sensitivity Index**
β	1
ξ	1
κ	Negative
α	Positive
θ	Positive
μ	Positive
ϕ_1	Negative
ϕ_2	Negative
ϕ_3	Negative
γ_2	Negative
δ	Negative
q	Negative
R_0	0.00543

Chapter 4

Discussion of Results

4.1 Analysis and Discussion of Results

To illustrate the behavior of the solution of our model, numerical simulations of the model (3.1)-(3.2) are carried out using the parameter values provided in Table 4.1. We simulate the model by using ode45 solver in MATLAB. Here, we use the choice

$$\xi = \frac{\beta(1-\kappa)}{N}, \text{where } N \text{ is the total population.} \tag{4.1}$$

Given that the force of infection is ξ at (4.1), the sensitivity indexes of R_0 with respect to β and ξ are both 1. Numerical simulations and graphical illustrations are carried out to verify some of the analytical results on the stability system (3.1). The initial conditions used in simulating the COVID-19 model can be found in Appendix (A). We simulate the COVID-19 model, a case study of Nigeria with intervention strategies, and analyze the effect of varying key parameters in the model with intervention strategies. The reported COVID-19 data used for the model fitting in Table 1 can be found in [23].

Figure 4.1 shows a good agreement between the fitted data and the reported data. This validates the model as a reliable tool for the existing phenomenon and surely predicts the future. It should be noted that the small deviation in the curve fitting between 0 to 15 days can be traced to the uncertainty present in real-life situations, however, both the fitted and the reported data are in perfect agreement after the 15 days. The model may advise the policymakers on COVID-19 controls and on short-time strategies employable to meet the demand of the population.

Table 4.1: Parameter values for COVID-19 Model for Nigeria Case.

Parameter symbol	**Baseline Value**	**Source**
Λ	0.0107	[36, 37]
κ	0.5	Assumed
ϕ_1	0.0571	Fitted
ϕ_2	0.00134	Fitted
ϕ_3	0.0153	Fitted
θ	0.0009	Assumed
ω	0.01	[7]
μ	0.0011188	[24]
β	0.1086	[1]
q	1/7	[29]
α	1/5.2 /day	[27, 35]
δ	0.015 /day	[12, 27]
γ_1	0.176	[32]
γ_2	0.142/day	[17]

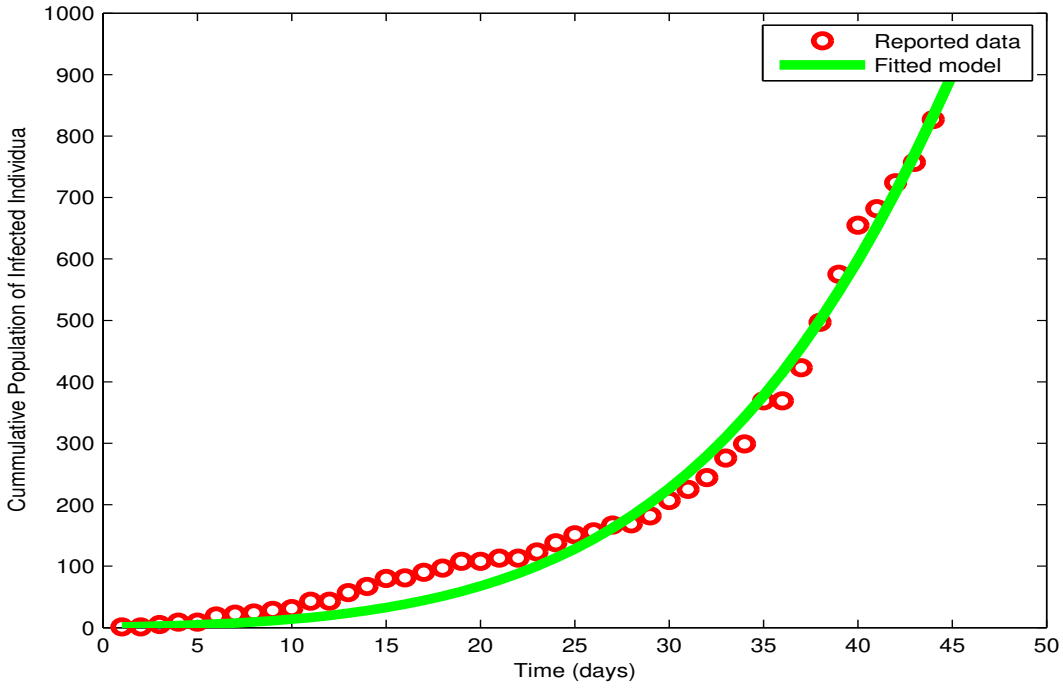

Figure 4.1: Model fitted to Covid-19 data in Lagos, Nigeria.

Figures 4.2 show the effect of adjusting the rate of the non-pharmaceutical intervention strategies on infected individuals and it is observed that as the rate of non-pharmaceutical intervention strategies increases, the infected class decreases. This implies that non-pharmaceutical intervention strategies have significant effects on the transmission of the disease and can therefore be used as a means of controlling the spread of COVID-19.

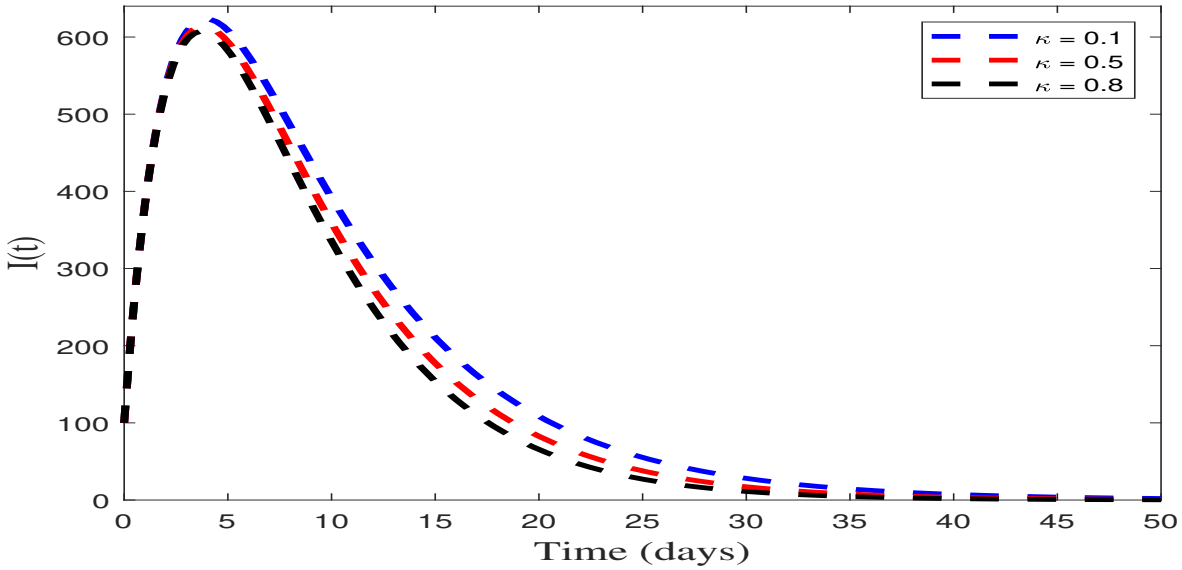

Figure 4.2: Effect of combination of intervention strategies (κ) on the Infected Individuals.

The effect of varying the vaccination rate ϕ_1 of the susceptible compartment on infected individuals is shown in Figure 4.3. It can be seen that with an increase in the vaccination rate of the susceptible compartment, the number of infected individuals decreases. This implies that when we implement vaccination as a measure, many people adhere to it and accept it, and get vaccinated. When people get vaccinated, the number of people who will be susceptible or who will be highly prone to be infected by COVID-19 will be reduced drastically and it can be seen that those people move from the Susceptible compartment to the Vaccinated compartment.

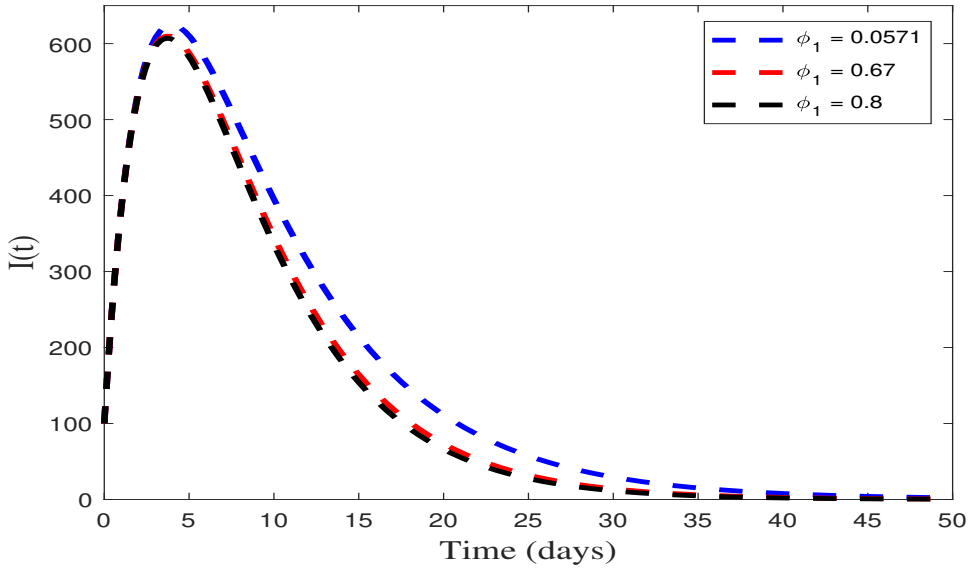

Figure 4.3: Effect of vaccination rate in the susceptible class (ϕ_1) on the Infected Individuals.

The effect of varying the vaccination rate ϕ_2 of the exposed compartment on infected individuals is shown in Figure 4.4. It can be seen that when we increased the vaccination rate with good efficacy in the exposed compartment, the number of infected, and the total population of individuals decreases. This suggests that when vaccination is implemented as a measure, many people will stick to it, accept it, and get themselves vaccinated. When people are vaccinated, it suggests that the number of people who are exposed or who are highly likely to get COVID-19 will substantially decrease, and it is evident that those individuals migrate from the Exposed compartment to the Vaccinated compartment.

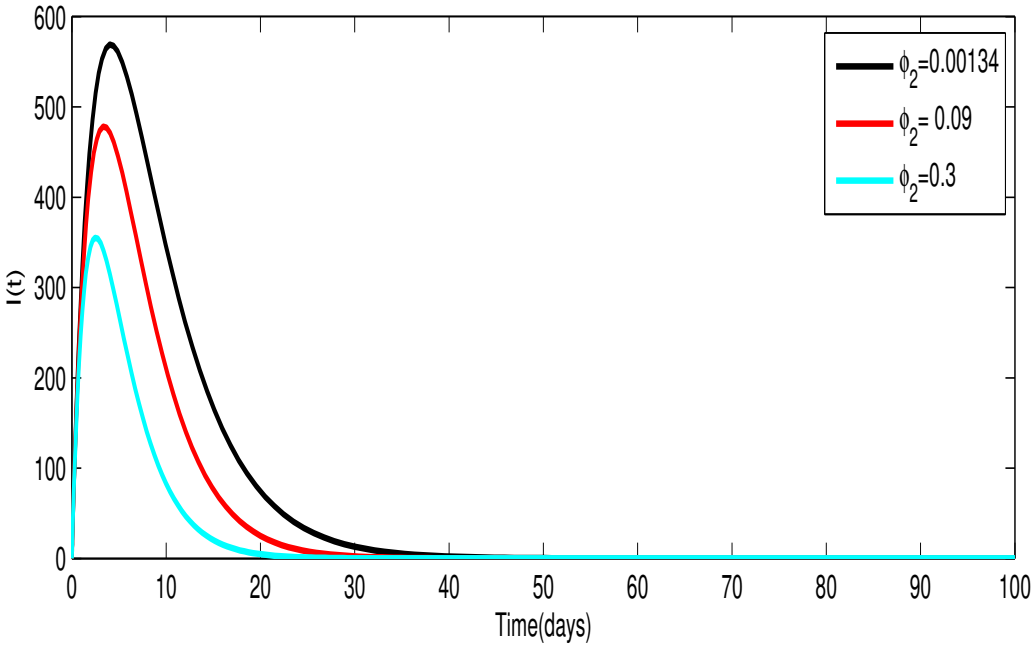

Figure 4.4: Effect of vaccination rate in the exposed class (ϕ_2) on the Infected Individuals.

A similar result is observed in the infected compartment as shown in Figure 4.5. Vaccination is a preventive measure that is targeted at reducing the infection rate by building human body immunity. As a result, infected individuals would reduce as the abundance of exposed humans decreases. In other words, as the number of exposed individuals reduces, the number of infectious humans numbers reduces.

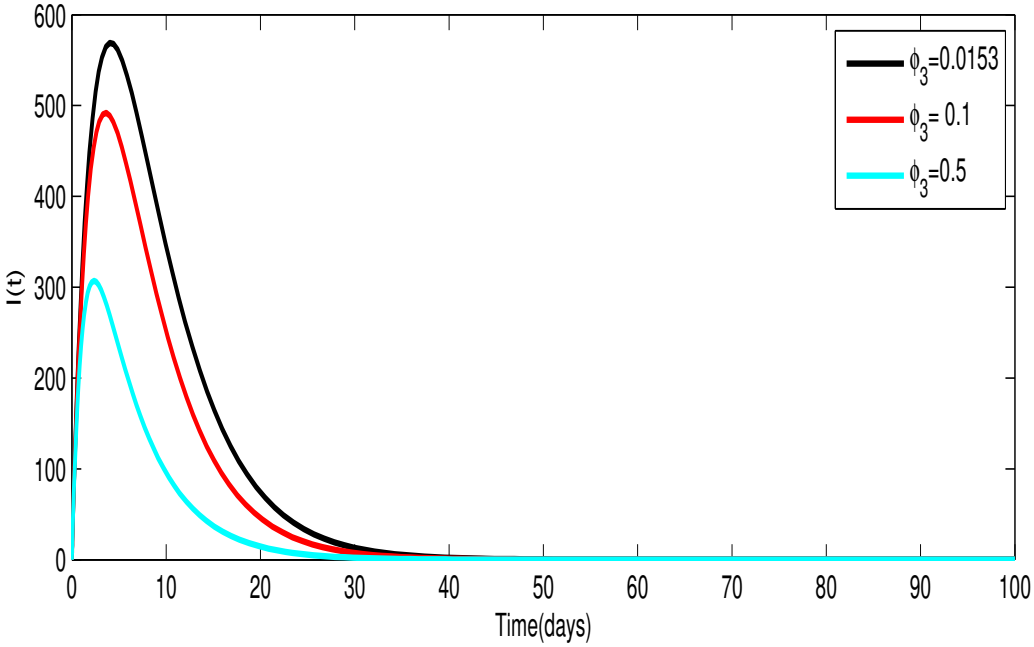

Figure 4.5: Effect of vaccination rate in the infected class (ϕ_3) on the Infected Individuals.

The effect of varying the initial condition $I(0)$ on the vaccinated compartment is shown in Figure 4.6. It can be seen that when we increased the initial condition, the vaccinated compartment increases. The vaccinated population increases as the number of initially infected individuals increase. This could be because of the fear of death that prompted the infected population to seek vaccination to reduce the chance of death. Hence, vaccinated compartment increase with increasing number of infected individuals.

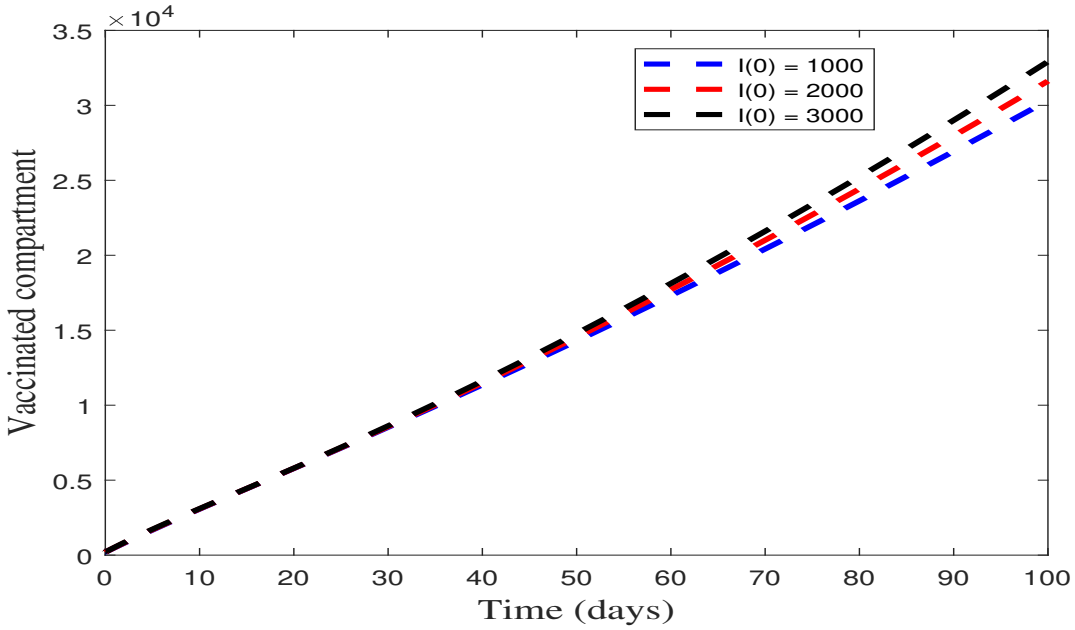

Figure 4.6: Effect of $I(0)$ on vaccinated compartment $V(t)$.

The effect of varying the recruitment rate Λ on the susceptible and vaccinated compartments is shown in Figure 4.7 and 4.8 respectively. It can be seen that when we increased the recruitment rate, the susceptible compartment increases. Also, it is worth noting that increasing the recruitment rate has no effect on the vaccinated compartment (see Figure 4.8).

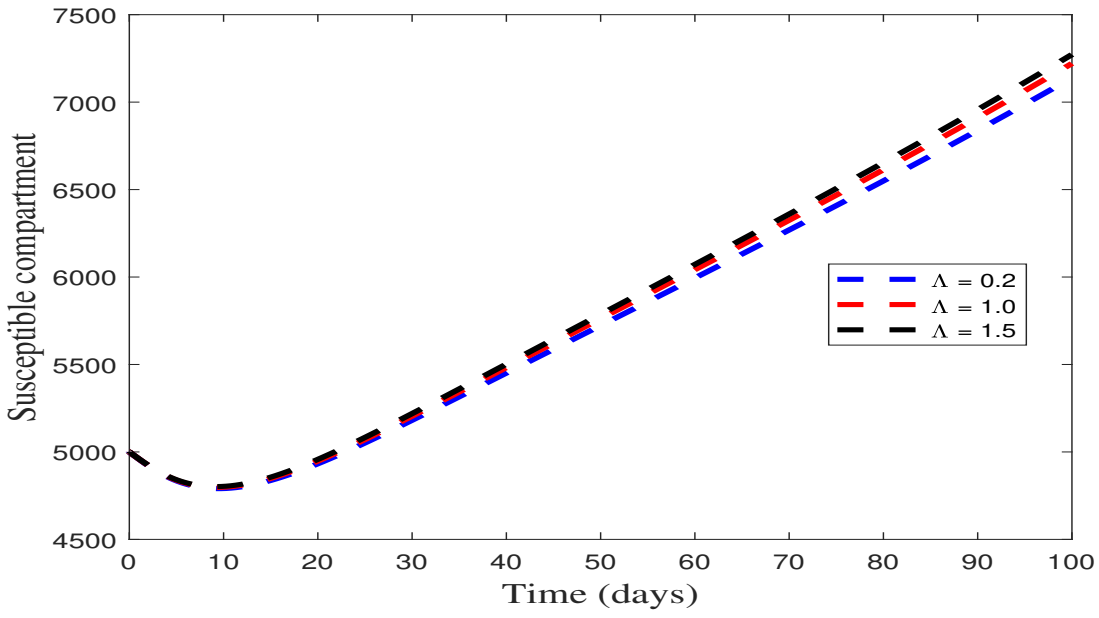

Figure 4.7: Effect of increasing the recruitment rate (Λ) on Susceptible compartment.

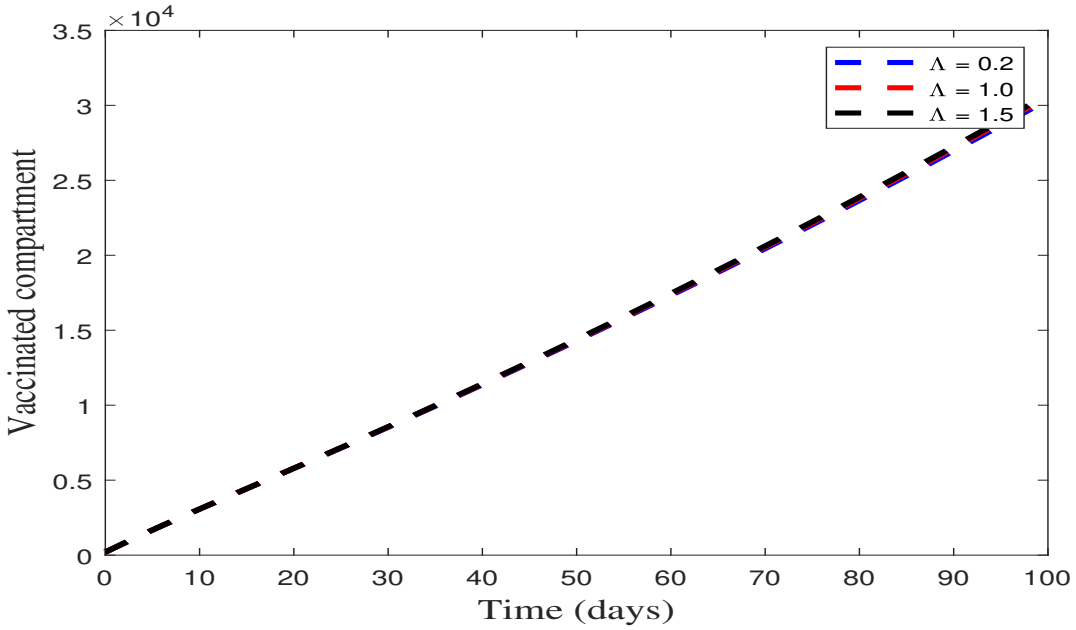

Figure 4.8: Effect of increasing the recruitment rate (Λ) on Vaccinated compartment.

The effect of varying the initial condition $R(0)$ on the susceptible compartment is shown in Figure 4.9. It can be seen that when we increased the initial condition, the susceptible compartment increases.

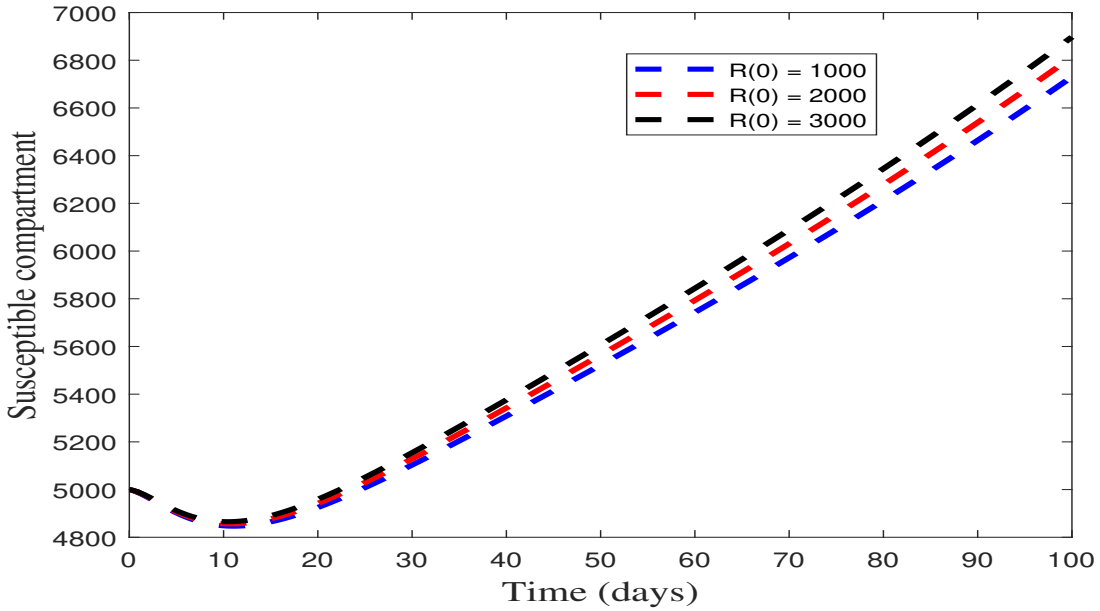

Figure 4.9: Effect of increasing $R(0)$ on the Susceptible compartment.

4.2 Discussion of the Sensitivity Analysis

Table 3.1 shows that the parameters (β, θ, μ, and α) with positivity indices have the potential to increase the endemicity of the infection in the population (i.e., the positive sensitivity parameter) because as they increase, the value of the reproduction number increases. Also, the parameters with negative sensitivity indices contribute to controlling the expansion of the infection in the population. This is because an increase in the parameters will result in a decrease in the value of the reproduction number.

Chapter 5

Conclusion and Future Work

5.1 Conclusion

In this study, we developed, analyzed a model to describe the transmission dynamics of the spread of COVID-19, a case study of Nigeria.

We sub-divided the human population into Susceptible humans $S(t)$, Exposed humans $E(t)$, Infected humans $I(t)$, Quarantined humans $Q(t)$, Recovered humans $R(t)$, and Vaccinated humans $V(t)$. We showed that the model is mathematically and epidemiologically meaningful by investigating the invariant region, the positivity of solutions, and the existence and uniqueness of solutions. The local and global stability of the model was analyzed, and we obtained the basic reproduction number R_0 using the next-generation matrix approach. The results show that the COVID-19 model equilibrium ϵ^* is locally and globally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$. A sensitivity analysis of the model parameters on the basic reproduction number was conducted to see the effect of key parameters on the disease dynamics. Overall, the result shows that the parameters (θ, μ, β, and α) with positivity indices have the potential of increasing the endemicity of the infection in the population. That is, as the positive sensitivity parameter increases, the values of the basic reproduction number increase. Numerical simulations were carried out and we explored the effect of controlled parameters on the infected class and the total population. Conclusively, as the model reproduces the results from the test case, it also suggests that taking control measures to decrease the force of infection is beneficial and this will mitigate the spread of COVID-19 in the population.

5.2 Recommendations

Based on the outcomes of this study, the following recommendations are suggested as means of containing and controlling the spread of COVID-19 in Nigeria:

1. Practicing intervention strategies will reduce the spread of the disease.

2. Constant practice of getting vaccines will help reduce the transmission in the population.

3. Enforcement of the control measures as well as penalties for noncompliance.

5.3 Future Work

It is well known that optimal control is useful in epidemiology. While mathematical modeling of infectious diseases has shown that isolation, quarantine, vaccination, and/or treatment are frequently required to completely eradicate infectious diseases. Therefore, it is crucial to examine the optimal control theory, which will inform us of how they should be administered by providing the right times for intervention and the right amounts. Additionally, stochastic simulations will be valuable for capturing the random character of COVID-19 in the population. Another key area that can be implemented in the model is continuous awareness through effective communication of COVID-19-related risks.

Acknowledgements

I would like to express my deepest gratitude to my supervisor, Professor Eddie Wadbro, for believing in me, and for all his academic and moral support throughout the year. This thesis would not have been possible without my co-supervisor, Dr. OKE Abayomi Samuel from Adekunle Ajasin University, Akungba Akoko, Nigeria, whose guidance from the initial step of research enabled me to develop an understanding of the subject. I am grateful to Professor Adrian Muntean from the Department of Mathematics and Computer Science, Karlstad University, for his valuable suggestions, and ever encouraging and motivating guidance. Many thanks to all of the members of staff at Karlstad University, Sweden, and the University of L'Aquila, Italy (Intermaths) for their kind support during my Master's study. I would also like to thank Dr. Olufemi Abolaji, Mr. Kehinde Ajeigbe, Mr. Godwin Osabutey, and Mr. Afeez for giving me the financial support and advice, you both made this journey a possible one for me. Very special thanks to Dr. Omame Andrew, Dr. Emmanuel Dansu, Mr. Ojo Mayowa Michael, Mr. Femi Ige, who were always there for me during the struggles of this research work. I am grateful to my parents whose constant love and support keep me motivated and confident. My accomplishments and success are because they believed in me. My deepest thanks to my siblings, who keep me grounded, remind me of what is important in life and are always supportive of my adventures. I would like to thank all my friends for encouraging and supporting me whenever I needed them.

Table 1: Reported cases of COVID-19 in Lagos Nigeria from 16/3/2020 to 30/4/2020.

Date	Cases	Cumulative Cases	Date	Cases	Cumulative Cases
16/03/2020	1	1	13/04/2020	13	182
17/03/2020	0	1	14/04/2020	25	207
18/03/2020	4	5	15/04/2020	18	225
19/03/2020	4	9	16/04/2020	19	244
20/03/2020	0	9	17/04/2020	32	276
21/03/2020	10	19	18/04/2020	23	299
22/03/2020	3	22	19/04/2020	70	369
23/03/2020	2	24	20/04/2020	0	423
24/03/2020	4	28	21/04/2020	54	497
25/03/2020	3	31	22/04/2020	74	1
26/03/2020	12	43	23/04/2020	78	575
27/03/2020	0	43	24/04/2020	80	655
28/03/2020	14	57	25/04/2020	27	682
29/03/2020	10	67	26/04/2020	42	724
30/03/2020	13	80	27/04/2020	33	757
31/03/2020	1	81	28/04/2020	80	827
01/04/2020	9	90	29/04/2020	87	914
02/04/2020	7	97	30/04/2020	45	959
03/04/2020	11	108			
04/04/2020	0	108			
05/04/2020	5	113			
06/04/2020	0	113			
07/04/2020	10	123			
08/04/2020	15	138			
09/04/2020	13	151			
10/04/2020	5	156			
11/04/2020	11	167			
12/04/2020	2	169			

List of Figures

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Appendix A

First Appendix

```
% Simulation showing various dynamics of the model as  I(0)  is varied on V(t)
% from 1000 to 3000
options = odeset('RelTol',1e-4,'AbsTol',[1e-9 1e-9 1e-9 1e-9 1e-9 1e-9]);
for i = [1, 2, 3]
    I0 = 1000*i;
    [T,Y] = ode45(@A3a,[0 100],[5000 2003 I0 0 0 200],options);
    if i == 1
        txt = "b--";
    elseif i == 2
        txt = "r--";
    elseif i == 3
        txt = "k--";
    end

    figure(1)
    plot(T,Y(:,6), txt,  'MarkerSize',5, 'LineWidth',2.5)
    hold on
end
xlabel('Time (days)','fontsize',14,'FontName','times new roman','LineWidth',2)
ylabel('Vaccinated compartment','fontsize',14,'FontName','times new roman','LineWidth',2)
%legend('I with age', 'I without age');

function dZ = A3a(t,Z)
dZ = zeros(6,1);    % a column vector
```

```
lambda = 0.017; mu = 0.0011188; phi1 = 0.0571; phi2 = 0.00134;
phi3 = 0.0153; q = 0.143; gamma1 = 0.176; gamma2 = 0.142; kappa = 0.5;
beta = 0.1018; delta = 0.015; alpha = 0.2; omega = 0.0111; theta = 0.0009;
epsilon = (beta*(1-kappa))/(5000+2003+0+0+0+200);

dZ(1) = lambda - epsilon*Z(3)*Z(1) - mu*Z(1) + theta*Z(6) + omega*Z(5);
dZ(2) = epsilon*Z(3)*Z(1) - mu*Z(2) - phi2*Z(2) - alpha*Z(2);
dZ(3) = alpha*Z(2) - (gamma2 + mu + q + phi3 + delta)*Z(3);
dZ(4) = q*Z(3) - (mu + gamma1)*Z(4);
dZ(5) = gamma2*Z(3) + gamma1*Z(4) - (mu+omega)*Z(5);
dZ(6) = phi1*Z(1) + phi2*Z(2) + phi3*Z(3) - (mu+theta)*Z(6);
end

% Simulation showing various dynamics of the model as  I(0)  is varied on V(t)
% from 1000 to 3000
global lambda
options = odeset('RelTol',1e-4,'AbsTol',[1e-9 1e-9 1e-9 1e-9 1e-9 1e-9]);
i = 1;
for lambda = [0.2, 1.0, 1.5]
    [T,Y] = ode45(@A3a,[0 100],[5000 2003 1000 0 0 200],options);
    if i == 1
        txt = "b--";
    elseif i == 2
        txt = "r--";
    elseif i == 3
        txt = "k--";
    end
    figure(1)
    plot(T,Y(:,1),txt,  'MarkerSize',5, 'LineWidth',2.5)
    xlabel('Time (days)','fontsize',14,'FontName','times new roman','LineWidth',2)
   ylabel('Susceptible compartment','fontsize',14,'FontName','times new roman','LineWidth',2)
    %%legend('I with age', 'I without age');
    hold on

    figure(2)
    plot(T,Y(:,2),txt,  'MarkerSize',5, 'LineWidth',2.5)
```

```
    xlabel('Time (days)','fontsize',14,'FontName','times new roman','LineWidth',2)
    ylabel('Exposed compartment','fontsize',14,'FontName','times new roman','LineWidth',2)
    %%legend('I with age', 'I without age');
    hold on

    figure(3)
    plot(T,Y(:,3),txt,  'MarkerSize',5, 'LineWidth',2.5)
    xlabel('Time (days)','fontsize',14,'FontName','times new roman','LineWidth',2)
    ylabel('Infected compartment','fontsize',14,'FontName','times new roman','LineWidth',2)
    %%legend('I with age', 'I without age');
    hold on

    figure(4)
    plot(T,Y(:,4),txt,  'MarkerSize',5, 'LineWidth',2.5)
    xlabel('Time (days)','fontsize',14,'FontName','times new roman','LineWidth',2)
   ylabel('Quarantined compartment','fontsize',14,'FontName','times new roman','LineWidth',2)
    %%legend('I with age', 'I without age');
    hold on

    figure(5)
    plot(T,Y(:,5),txt,  'MarkerSize',5, 'LineWidth',2.5)
    xlabel('Time (days)','fontsize',14,'FontName','times new roman','LineWidth',2)
    ylabel('Recovered compartment','fontsize',14,'FontName','times new roman','LineWidth',2)
    %%legend('I with age', 'I without age');
    hold on

    figure(6)
    plot(T,Y(:,6), txt,  'MarkerSize',5, 'LineWidth',2.5)
    hold on
    xlabel('Time (days)','fontsize',14,'FontName','times new roman','LineWidth',2)
   ylabel('Vaccinated compartment','fontsize',14,'FontName','times new roman','LineWidth',2)

    i = i + 1;
end
```

```
function dZ = A3a(t,Z)
global lambda
dZ = zeros(6,1);    % a column vector

%lambda = 0.017;
mu = 0.0011188; phi1 = 0.0571; phi2 = 0.00134;
phi3 = 0.0153; q = 0.143; gamma1 = 0.176; gamma2 = 0.142; kappa = 0.5;
beta = 0.1018; delta = 0.015; alpha = 0.2; omega = 0.0111; theta = 0.0009;
epsilon = (beta*(1-kappa))/(5000+2003+0+0+0+200);

dZ(1) = lambda - epsilon*Z(3)*Z(1) - mu*Z(1) + theta*Z(6) + omega*Z(5);
dZ(2) = epsilon*Z(3)*Z(1) - mu*Z(2) - phi2*Z(2) - alpha*Z(2);
dZ(3) = alpha*Z(2) - (gamma2 + mu + q + phi3 + delta)*Z(3);
dZ(4) = q*Z(3) - (mu + gamma1)*Z(4);
dZ(5) = gamma2*Z(3) + gamma1*Z(4) - (mu+omega)*Z(5);
dZ(6) = phi1*Z(1) + phi2*Z(2) + phi3*Z(3) - (mu+theta)*Z(6);
end



% Simulation showing various dynamics of the model as  recruitment rate (Lambda) on S(t) and V(t)  is varied
%
global lambda
options = odeset('RelTol',1e-4,'AbsTol',[1e-9 1e-9 1e-9 1e-9 1e-9 1e-9]);
i = 1;
for lambda = [0.2, 1.0, 1.5]
    [T,Y] = ode45(@A3a,[0 100],[5000 2003 1000 0 0 200],options);
    if i == 1
        txt = "b--";
    elseif i == 2
        txt = "r--";
    elseif i == 3
        txt = "k--";
    end
    figure(1)
    plot(T,Y(:,1),txt,  'MarkerSize',5, 'LineWidth',2.5)
    xlabel('Time (days)','fontsize',14,'FontName','times new roman','LineWidth',2)
```

```
ylabel('Susceptible compartment','fontsize',14,'FontName','times new roman','LineWidth',2)
 %%legend('I with age', 'I without age');
 hold on

 figure(2)
 plot(T,Y(:,2),txt,  'MarkerSize',5, 'LineWidth',2.5)
 xlabel('Time (days)','fontsize',14,'FontName','times new roman','LineWidth',2)
 ylabel('Exposed compartment','fontsize',14,'FontName','times new roman','LineWidth',2)
 %%legend('I with age', 'I without age');
 hold on

 figure(3)
 plot(T,Y(:,3),txt,  'MarkerSize',5, 'LineWidth',2.5)
 xlabel('Time (days)','fontsize',14,'FontName','times new roman','LineWidth',2)
 ylabel('Infected compartment','fontsize',14,'FontName','times new roman','LineWidth',2)
 %%legend('I with age', 'I without age');
 hold on

 figure(4)
 plot(T,Y(:,4),txt,  'MarkerSize',5, 'LineWidth',2.5)
 xlabel('Time (days)','fontsize',14,'FontName','times new roman','LineWidth',2)
ylabel('Quarantined compartment','fontsize',14,'FontName','times new roman','LineWidth',2)
 %%legend('I with age', 'I without age');
 hold on

 figure(5)
 plot(T,Y(:,5),txt,  'MarkerSize',5, 'LineWidth',2.5)
 xlabel('Time (days)','fontsize',14,'FontName','times new roman','LineWidth',2)
 ylabel('Recovered compartment','fontsize',14,'FontName','times new roman','LineWidth',2)
 %%legend('I with age', 'I without age');
 hold on

 figure(6)
 plot(T,Y(:,6), txt,  'MarkerSize',5, 'LineWidth',2.5)
 hold on
 xlabel('Time (days)','fontsize',14,'FontName','times new roman','LineWidth',2)
```

```
   ylabel('Vaccinated compartment','fontsize',14,'FontName','times new roman','LineWidth',2)

    i = i + 1;
end



function dZ = A3a(t,Z)
global lambda
dZ = zeros(6,1);    % a column vector

%lambda = 0.017;
mu = 0.0011188; phi1 = 0.0571; phi2 = 0.00134;
phi3 = 0.0153; q = 0.143; gamma1 = 0.176; gamma2 = 0.142; kappa = 0.5;
beta = 0.1018; delta = 0.015; alpha = 0.2; omega = 0.0111; theta = 0.0009;
epsilon = (beta*(1-kappa))/(5000+2003+0+0+0+200);

dZ(1) = lambda - epsilon*Z(3)*Z(1) - mu*Z(1) + theta*Z(6) + omega*Z(5);
dZ(2) = epsilon*Z(3)*Z(1) - mu*Z(2) - phi2*Z(2) - alpha*Z(2);
dZ(3) = alpha*Z(2) - (gamma2 + mu + q + phi3 + delta)*Z(3);
dZ(4) = q*Z(3) - (mu + gamma1)*Z(4);
dZ(5) = gamma2*Z(3) + gamma1*Z(4) - (mu+omega)*Z(5);
dZ(6) = phi1*Z(1) + phi2*Z(2) + phi3*Z(3) - (mu+theta)*Z(6);
end

% Simulation showing various dynamics of the model as  R(0)  is varied
% from 1000 to 3000
options = odeset('RelTol',1e-4,'AbsTol',[1e-9 1e-9 1e-9 1e-9 1e-9 1e-9]);
for i = [1,2,3]
    R0 = 1000*i;
    [T,Y] = ode45(@A3a,[0 100],[5000 2003 100 0 0 R0],options);
    if i == 1
        txt = "b--";
    elseif i == 2
        txt = "r--";
    elseif i == 3
        txt = "k--";
```

```
    end

    figure(1)
    plot(T,Y(:,1), txt,  'MarkerSize',5, 'LineWidth',2.5)
    hold on
end
xlabel('Time (days)','fontsize',14,'FontName','times new roman','LineWidth',2)
ylabel('Susceptible compartment','fontsize',14,'FontName','times new roman','LineWidth',2)
%legend('I with age', 'I without age');

function dZ = A3a(t,Z)
dZ = zeros(6,1);    % a column vector

lambda = 0.017; mu = 0.0011188; phi1 = 0.0571; phi2 = 0.00134;
phi3 = 0.0153; q = 0.143; gamma1 = 0.176; gamma2 = 0.142; kappa = 0.5;
beta = 0.1018; delta = 0.015; alpha = 0.2; omega = 0.0111; theta = 0.0009;
epsilon = (beta*(1-kappa))/(5000+2003+0+0+0+200);

dZ(1) = lambda - epsilon*Z(3)*Z(1) - mu*Z(1) + theta*Z(6) + omega*Z(5);
dZ(2) = epsilon*Z(3)*Z(1) - mu*Z(2) - phi2*Z(2) - alpha*Z(2);
dZ(3) = alpha*Z(2) - (gamma2 + mu + q + phi3 + delta)*Z(3);
dZ(4) = q*Z(3) - (mu + gamma1)*Z(4);
dZ(5) = gamma2*Z(3) + gamma1*Z(4) - (mu+omega)*Z(5);
dZ(6) = phi1*Z(1) + phi2*Z(2) + phi3*Z(3) - (mu+theta)*Z(6);
end
```

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