Int. J. Open Problems Compt. Math., Vol. 18, No. 4, December 2025
Print ISSN: 1998-6262, Online ISSN: 2079-0376
Copyright ©ICSRS Publication, 2025, www.i-csrs.org

Dynamical Analysis and Modeling of COVID-19 and Related Diseases under Comorbidity in Complex Domains

Florence Adongo, Titus Aminer, Benard Okelo, Benson Onyango

School of Biological, Physical, Mathematics and Actuarial Sciences,
Jaramogi Oginga Odinga University of Science and Technology,
Box 210-40601, Bondo-Kenya.
e-mail: benard@aims.ac.za

Received 8 July 2025; Accepted 27 September 2025

Abstract

The COVID-19 infection is known for causing more challenges to people with underlying health conditions such as cardiovascular, cerebrovascular and diabetes among others. Due to these complications and outbreak of diseases, there is need to formulate a model for comorbidities. Those who have these two or more diseases died at higher rate, four times, compared to those who are suffering from one disease. For diabetes and COVID-19 infected patients, mortality rate is higher, this means the rate of recovery is low and more resources are used towards patients with diabetes and COVID-19 comorbidity. Containment measures for COVID-19 such as quarantine and social distancing may lead to a decline in exercising and lack of a balanced diet, which are key for managing diabetic complications such as vision loss and kidney failure. This note provides a dynamical analysis comparing the rate of recovery and death rate that is high in those co-morbidities compared to those who do not have co-morbidities, all under vaccinations.

Keywords: *COVID-19, Model, Comorbidity, Dynamical analysis.*
2010 Mathematics Subject Classification: Primary 92D30; secondary 34A34, 37N25.

1 Introduction

Studies on Corona Virus Disease-19 (COVID-19) have been carried out for sometimes since the time it emerged and it has been determined that as peo-

ple moved from one place to another, they interacted with each other, causing the spread of COVID-19 disease from China its origin to other parts of the world. Those who were sick with other diseases such as pneumonia, diabetes, and hypertension among others were also infected with this disease hence comorbidity. Later on in 2023, the disease was classified as an on-going disease; implying that the disease is still present [1].

Research nomenclature indicates that the viral pandemic was later referred to as coronavirus, Wuhan corona virus [3] or Wuhan pneumonia [4] owing to its geographical origin. In 2020, it was named SAAR-Cov2 given that a similar virus had caused an outbreak in 2003. On 11th February 2020, the official name was issued by WHO as COVID-19.

When the disease takes long before recovery, then it may lead to pneumonia, multiple failure of some organs and later resulting into death [11]. Currently, it takes time before appearance of the above symptoms running between $1-14$ days but most individuals can show symptoms within 5 or 6 days. There are two categories of infected individuals: those who are having virus in the system without symptoms and those with the virus and showing symptoms. Either of the categories above is potentially infectious [25].

From clinical observations, 81% of patients experience mild symptoms that can cause mild pneumonia, 14% patients undergo severe symptoms such as dyspnoea, while 5% patients undergo shock, respiratory failure or multi organ dysfunction. Given varying body immunity levels, infected individuals may experience different symptoms and which may take long before recovery with older people noted to be at a high risk [5]. Additionally, the common symptoms have been clustered as; respiratory, musculoskeletal and digestive.

In terms of pathophysiology, COVID-19 infection in humans can spread far and wide. This leads to decreased secretion of lung surfactant resulting in the respiratory symptoms above. The increased production of angiotensin, may cause COVID-19 to enter into the cells through membrane fusion. The above conditions among COVID-19 patients may lead to infection and severity of other disease like hypertension, diabetes and cardiovascular diseases [12]. This helps to explain the possible severity of comorbidity of C19 and diabetes hence the study.

As a new medical condition, there is yet no specific medicine(s) recommended to treat the infection according to WHO. The infected individuals need to receive appropriate and optimized supportive care to relieve, treat symptoms and observe WHO recommendations on prevention measures [6]. People should be vaccinated using recommended vaccines, some of which are given once or twice to lower the rate of infection, increase recovery rate (rr) and lower death rate (dr).

Diabetes is a metabolism malfunction condition that makes blood to have a lot of sugar in the human body. Hormones such as insulin are produced and used

by the body to regulate sugar levels by moving sugar into cells. Sugar taken into the cells can be stored or converted into energy [7]. The condition may cause the body not to generate adequate insulin or to ineffectively use what it has made. These two conditions usually lead to two categories of diabetes (Type 1 and 2). Uncontrolled conditions due to diabetes can be destructive to various organs such as nerves, eyes, kidneys and others. Gestation diabetes occurs where there is a lot of sugar in the body of a pregnant mother due to production of insulin-blocking hormones by placenta. There is a rare condition called diabetes insipidus where the kidney causes the removal of lots of fluids from the body [21].

Causes include genes inherited from parents, overweight, age of 45 or older, physical inactivity and pre-existing medical conditions. Diabetes is treated using a drug or a combination of drugs depending on the type one has. The major drug is insulin, besides diet and exercise [21]. Available statistics show that the number of people being infected are increasing worldwide causing the death of many people especially those in countries with low income (under-developed countries) [18].

In the study by [2], patients that were hospitalized were tested and results given as; more men were infected with disease compared to females; those who were infected with other diseases had higher percentage compared to those who were suffering from C19 only. Age distribution was considered and the middle age were infected more compared to other ages. Symptoms distribution among patients was as follows; 40 (98%) patients had fever, 31 (76%) patients had cough and 18 (44%) patients had fatigue [26]. Less common symptoms like; sputum production among 11 (28%) patients of 39, headache among 3 (8%) patients out of 38, haemoptysis among 3 (5%) patients of 39 and diarrhoea noted with 1 (3%) patient of 38. A given percentage was taken to Intensive Care Unit (ICU) and some died because of the disease.

Diabetic persons' condition worsened when they contracted COVID-19 especially in China [9].

It has been reported that most people admitted at ICU were with comorbidity and they have a double challenge compared to others[10]. Mathematical models for COVID-19, diabetes and comorbidities have been carried out and results documented. A Susceptible, Exposure, Infected and Recovery (SEIR) model was formulated and analyzed [13]. From the data, it was observed that the spread of the disease was increasing causing basic reproduction number, R_0, to increase. From their recommendation, the link between China and other cities should be closed or cut off to reduce the contact to prevent the infections. Based on the data at that time, more research was necessary and other factors to be put into consideration to lower R_0.

Another model was developed and analyzed that regulate the blood glucose level of diabetic persons [14]. The numerical solution presented the complex

situation of diabetic patients. Examination was done and numerical solution obtained on normal person, those who were added insulin and insulin concentration was considered. Results showed that when glucose was given to a normal person, the glucose concentration level became very high but with time it was stable while plasma insulin concentration and generalized insulin variable level remained the same even after some time [15].
For some patients, it showed that at the initial, the glucose level was very high, but after giving glucose to them, there was no major fall in glucose level same to plasma insulin concentration and generalized insulin variable. On the diabetic patients, there was no effect of glucose concentration after sometime compared to normal person. Diabetes management is one of the important issues in the field of glucose-insulin regulatory system hence more research on mathematical models for long-term diabetes progression should be done continuously [17]. Mathematical models for COVID-19 and Diabetes have been done separately in literature hence the need for a study and mathematical modeling of COVID-19 and Diabetes co-morbidity more so under vaccination.

2 Basic concepts

Some of basic concepts which are useful in this study are outlined. These include Mathematical model, dynamical system, COVID-19, diabetes, co-morbidity, ordinary differential equation, epidemiological model and basic reproduction number [29].

Definition 2.1 *([39], Definition 3.7) A deterministic model is an established correlation between the input and output of a given structure. Such correlations may or may not change over time. In this type of model, we formulated deterministic model where product of the simulation is fully regulated by the parameter rates and the initial state.*

Definition 2.2 *([16], Definition 2.2) A stochastic model is correlations between input and output of a given structure where both the inputs and outputs are arbitrary.*

Definition 2.3 *([20], Definition 3.3) Sets of equations conveying the level of variation in terms of the variables and time are known as dynamical systems. Examples of dynamical systems are:*
(i). Non-autonomous- $x' = G(t, y)$, *where* $G : \mathcal{R}^{n+1} \to \mathcal{R}^n$.
(ii). Discrete dynamical system- $x[m+1] = G_m(x[m])$, *where* $G_m : \mathcal{R}^n \to \mathcal{R}^n \ \ \forall m \in \mathcal{F}$,
(iii). Autonomous discrete dynamical system-$y[m+1] = G(y[m])$

Definition 2.4 *([19], Definition 5.3) Diabetes is a metabolism malfunction condition that make blood to have a lot of sugar in the human body.*

Definition 2.5 *([22], Definition 4.4) Co-morbidity is the presence of more than one disease in the same person. For example; diabetes and hypertension, diabetes and kidney failure or diabetes and COVID-19, malaria and pneumonia among others.*

Definition 2.6 *([23], Definition 2.3) The basic reproduction number (R_0) is the number of times infected individual infect other people in their entire infectious life.*

3 Research methodology

These are some of the methods, inequalities,theories,programming and criteria that will be used in the model formation and the analysis of the model formulated. Deterministic differential equation will be used on model formation.

3.1 The Kermack-MC kendrick model

This is one of the comparative models using time as independent variable (t) and mathematic expression for the rate of transfer between the compartments as derivatives with respect to time. This mathematical expression give differential equations which form a model. Example of compartments are susceptible (those can be infected), infection (those who have the disease), recovery/ removed (those who recover from disease),vaccination(those have been vaccinated), exposed(those among the infected people) among others. From the compartments we can come up with models such as SIR, SEIR , and SEIVR among others. Using differential equations example of models is as follows:

$$\begin{aligned} \frac{dS}{dt} &= -\beta SI \\ \frac{dI}{dt} &= \beta SI - \gamma I \\ \frac{dR}{dt} &= \gamma I \end{aligned}$$

3.2 Gronwall's inequality

Gronwalls inequality is a deterministic analytical statement that converts an inequality involving a function and its integral into an explicit, computable bound. It is applicable when a nonnegative function is constrained by an additive constant plus the integral of a nonnegative coefficient times the function

itself. The inequality yields an exponential-type upper bound that controls the functions growth in terms of the given coefficient and initial constant.

3.3 Routh-Hurwitz stability criterion

The RouthHurwitz stability criterion is an algebraic method for determining whether all roots of a polynomial lie strictly in the open left half of the complex plane. Since the locations of the roots of the characteristic polynomial dictate the stability of a linear time-invariant system, the criterion provides a systematic procedure to test stability without explicitly computing the roots.

3.4 Lyapunov technique

The Lyapunov technique is used to investigate stability properties of equilibrium points of dynamical systems without requiring explicit solutions of the governing equations. The method is based on the construction of an auxiliary scalar function, called a Lyapunov function, whose behavior along system trajectories provides information about stability.

3.5 The next generation matrix

This involves determining the rate at which new infections are generated denoted as G; and determining the transmission terms represented as M compartments because of recovery, death and any other factors to other compartments [34]. From next generation matrix ($\mathcal{GM}^{-1}$), we construct R_0 [33].

3.6 Jacobian Matrix, Determinant and Traces

Evaluating Jacobian Matrix [37] to come up with characteristic equations which give eigenvalues. The negative eigenvalues will make $R_0 < 1$ making local stability of DFE stable, if eigenvalues are positive then $R_0 > 1$ hence unstable. From the jacobian matrix, we can solve determinant and trace then applying Routh-Hurwitz condition. If Routh-Hurwitz condition holds then there is stability of DFE. Consider

$$y = (\, g \,)_1 (X) g_2(X) .. g_n(X) \tag{1}$$

The determinant for the above jacobian matrix is called a Jacobian [40].

3.7 Normalized forward sensitivity index

This indicate how other parameters indicated in the study affect the R_0. These parameters include death, rate of recovery, transmission rate among others.

Using set of assumptions, sensitivity analysis can be constructed to show how parameters affect dependent variables such basic reproductive number in the model. There two type of sensitivity analysis that is local and global. In local sensitivity analysis one parameter is observed while others are put constant some time refer as one-factor-at-a-time (OAT). In this work we are using global sensitivity analysis as it was used in [41] which is calculated using normalized forward sensitivity index. Using example of [42] as $S_K^{R_0} = \frac{dR_0}{dK} \times \frac{K}{R_0}$ where K is the parameter being observed over basic reproduction number.

3.8 Numerical simulation technic

Numerical simulations can be done using Python which has jupyter note book programming language or any other programming language in studies of this nature [43]. After imputing differential equations, parameters and their values, initial values and plotting. The command run that give graphs that are used to compare the effect of COVID-19 and Diabetes co-morbidity under vaccination on recovery of patients. The graphs will be used to compare the rate of recovery for the two models in our next paper [25].

4 Main results

4.1 Model formulation

We formulate mathematical models using the system of non-linear differential equation for COVID-19 under vaccination and another model of diabetic population but infected with COVID-19 as co-morbidity considering vaccination. There are those who are recovering naturally from the disease and those who are using other controls such as use of drugs or proper management of the disease to get well thus forming the recovery class.
The recovery class is increased by those individuals recovering from L and I classes and decreased by death μ, those who got vaccinated and move to V class and re-infected individuals back to L and I classes. The effect of vaccination on susceptible and recovery individuals reduces chances of infection hence we assumed less than a unit. Immigration or emigration, birth and death makes the population not stable or constant. Therefore we assumed a constant recruitment ρ into susceptible class. Susceptible persons are infected to asymptomatic and symptomatic classes at the rate of λ_1 and λ_2 respectively. Susceptible persons are vaccinated at the rate θ. The COVID-19 infected and infectious with COVID-19 but asymptomatic L class and infectious with COVID-19 symptomatic I class take place at the rate γ_1 and γ_2 respectively. After recovery from COVID-19, it is necessary for those who have recovered to be vaccinated, this is done at the rate κ. Re-infection occurs from those

Table 1: parameters and Interpretations

Parameter	**Interpretations**
S_H	Susceptible population
I	Infected population (symptomatic)
L	Carriers population (asymptomatic)
R	Recovered population
V	Vaccinated population
Λ_H	Rate of recruitment to the susceptible population
λ_1	Rate of movement from susceptible to carrier individuals
λ_2	Rate of movement from susceptible to infected individuals
θ	Rate of movement from susceptible to the vaccinated individuals
κ	Rate of movement from recovered to the vaccinated individuals
μ	Natural death rate
γ_1	Rate of recovery for carrier population
γ_2	Rate of recovery for infected population
τ	Rate of transfer of carrier individuals to the infected population
β_L	Effective contact rate for COVID-19 transmission to asymptomatic
δ	Death rate due to corona virus
β_I	Effective contact rate for COVID-19 transmission to symptomatic
ψ_1	Rate of reinfections for carrier population from recovery population
ψ_2	Rate of reinfections for infected population from recovery population

who have recovered from infection both asymptomatic or symptomatic at ψ_1 and ψ_2 respectively. Asymptomatic individuals become symptomatic at τ. In a population, some people are limited naturally through death, not because of infection, at the rate of μ.
The force of transmission is give as $\lambda S_H = (\lambda_1 + \lambda_2)S_H = (\beta_L L + \beta_I I)S_H$. The dynamical system obtained is:

$$\frac{dS_H}{dt} = \rho_H - (\lambda_1 + \lambda_2 + \theta + \mu)S_H \tag{2}$$

$$\frac{dL}{dt} = \lambda_1 S_H + \psi_1 R - (\mu + \delta + \tau + \gamma_1)L \tag{3}$$

$$\frac{dV}{dt} = \kappa R + \theta S_H - \mu V \tag{4}$$

$$\frac{dI}{dt} = \lambda_2 S_H + \psi_2 R + \tau L - (\mu + \delta + \gamma_2)I \tag{5}$$

$$\frac{dR}{dt} = \gamma_2 I + \gamma_1 L - (\mu + \psi_2 + \psi_1 + \kappa)R \tag{6}$$

This is the model of COVID-19 free diabetes under vaccination. The dynamical analysis of the formulated model includes positivity of the solution, boundedness of solution, two equilibria and their stabilities.

4.2 Positivity of solution

Proposition 4.1 *From the model Let the initial conditions be* $(S_H, V, L, I and R)(0) > 0$*, then the solution set* $(S_H, V, L, I and R)(t)$ *of the model is positive* $\forall t > 0$

Proof. Taking model

$$\frac{dS_H}{dt} = \rho_H - (\lambda_1 + \lambda_2 + \theta + \mu)S_H \tag{7}$$
$$\frac{dL}{dt} = \lambda_1 S_H + \psi_1 R - (\mu + \delta + \tau + \gamma_1)L \tag{8}$$
$$\frac{dV}{dt} = \kappa R + \theta S_H - \mu V \tag{9}$$
$$\frac{dI}{dt} = \lambda_2 S_H + \psi_2 R + \tau L - (\mu + \delta + \gamma_2)I \tag{10}$$
$$\frac{dR}{dt} = \gamma_2 I + \gamma_1 L - (\mu + \psi_2 + \psi_1 + \kappa)R \tag{11}$$

From this model, let us take the first equation $\frac{dS_H}{dt} = \rho_H - (\lambda_1 + \lambda_2 + \theta + \mu)S_H$, then we have $\frac{dS_H}{dt} = \rho_H - (\lambda_1 + \lambda_2 + \theta + \mu)S_H$ which implies that $\frac{dS_H}{dt} \geq (\lambda_1 + \lambda_2 + \theta + \mu)S_H$. Seperating the variables yields $\frac{dS_H}{S_H} \geq (\lambda_1 + \lambda_2 + \theta + \mu)dt$. Integrating the differential inequality $(\int_{S_H 0}^{S_H})\frac{dS_H}{S} \geq \int(\lambda_1 + \lambda_2 + \theta + \mu)dt$ yields

$$\ln S_H - \ln S_{H0} \geq \int(\lambda_1 + \lambda_2 + \theta + \mu)dt \tag{12}$$

$$\ln \frac{S_H}{S_H 0} \geq \int(\lambda_1 + \lambda_2 + \theta + \mu)dt \tag{13}$$

$$\frac{S_H}{S_H 0} \geq \exp \int(\lambda_1 + \lambda_2 + \theta + \mu)dt \tag{14}$$

$$S_H \geq S_H 0 \exp \int(\lambda_1 + \lambda_2 + \theta + \mu)dt. \tag{15}$$

Therefore, we can conclude that equation 15 is a non-negative of t, S_H stays positive. Applying same procedure to the remaining variables, $(V, L, I$ and$R)$ are also positive $\forall t > 0$.

4.3 Boundedness of the solution

The total population is given by summing up the four equations of the model. This gives,

$$N = S_H + V + I + L + R. \tag{16}$$

$$\frac{dN}{dt} = \frac{dS_H}{dt} + \frac{dV}{dt} + \frac{dI}{dt} + \frac{dL}{dt} + \frac{dR}{dt}, \tag{17}$$

from the original model, we obtained by simplifying

$$\frac{dN}{dt} = \rho_H - \delta I - \delta L - (\mu S + \mu V + \mu I + \mu L + \mu R). \tag{18}$$

Thus

$$\frac{dN}{dt} = \rho_H - \delta I - \delta L - \mu(S + V + I + L + R) \tag{19}$$

hence

$$\frac{dN}{dt} = \rho_H - \mu N - \delta I. \tag{20}$$

therefore we have

$$\frac{dN}{dt} \leq \rho_H - \mu N, \tag{21}$$

which implies that

$$\frac{dN}{dt} + \mu N \leq \rho_H \tag{22}$$

Using integrating factor/separation of variables to solve Inequality 22 we obtain

$$N(t) \leq \frac{\rho_H}{\mu} + N(0)\exp^{-\mu t} \tag{23}$$

From inequality 23, we observe that

$$0 \leq N(t) \leq \frac{\rho_H}{\mu} + N(0)e^{-\mu t}, \tag{24}$$

where $N(0)$ represents initial population. As $t \to \infty$, then

$$0 \leq N(t) \leq \frac{\rho_H}{\mu}. \tag{25}$$

4.4 The Disease Free Equilibrium (DFE), $E^0(S^0, L^0, V^0, I^0, R^0)$

Consider the dynamical system

$$\begin{aligned}
\frac{dS_H}{dt} &= \rho_H - (\lambda_1 + \lambda_2 + \theta + \mu)S_H && (26)\\
\frac{dL}{dt} &= \lambda_1 S_H + \psi_1 R - (\mu + \delta + \tau + \gamma_1)L && (27)\\
\frac{dV}{dt} &= \kappa R + \theta S_H - \mu V && (28)\\
\frac{dI}{dt} &= \lambda_2 S_H + \psi_2 R + \tau L - (\mu + \delta + \gamma_2)I && (29)\\
\frac{dR}{dt} &= \gamma_2 I + \gamma_1 L - (\mu + \psi_2 + \psi_1 + \kappa)R. && (30)
\end{aligned}$$

To determine the DFE of COVID-19, only susceptible and vaccination populations or variables are considered. We solve for the variables S^0 and V^0 since $I^0 = 0$, $L^0 = 0$ and $R^0 = 0$ but $\lambda_1 = \beta_L L$ and $\lambda_2 = \beta_I I$.

$$S^0 = \frac{\rho_H}{(\theta + \mu)}, \tag{31}$$

Also,

$$S^0 = \frac{\mu V^0}{\theta} \tag{32}$$

Equating equations and calculating the value of V^0,

$$V^0 = \frac{\theta \rho_H}{\mu(\theta + \mu)}. \tag{33}$$

Using equations , the DFE E^0, of model is then calculated as

$$E^0(S^0, L^0, V^0, I^0, R^0,) = \left(\frac{\rho_H}{(\theta + \mu)}, 0, \frac{\theta \rho_H}{\mu((\theta + \mu)}, 0, 0 \right). \tag{34}$$

The equation represents a compartmental model with different populations (compartments) including S_H (susceptible humans), L (Latent individuals), V (Vaccinated individuals), I (Infected individuals) and R (recovery individuals). The terms λ_1, λ_2, θ, μ, ρ_H, γ_2, γ_1, ψ_2, ψ_1, κ and τ are parameters governing the transitions between compartments. We carry out the matrix construction as follows: Let's denote the infected compartments $X_1 = L$ and$X_2 = I$

$$\frac{dL}{dt} = \lambda_1 S_H + \psi_1 R - (\mu + \delta + \tau + \gamma_1)L \tag{35}$$

$$\frac{dI}{dt} = \lambda_2 S_H + \psi_2 R + \tau L - (\mu + \delta + \gamma_2)I \tag{36}$$

$$\tag{37}$$

The infection rate matrix G can be constructed as; new infection is divided into two where a fraction of those who are causing new infection at L class and those who are causing new infection at I class at $1 - n$ and n respectively. Force of infection:$\lambda = \beta_L \frac{L}{N} + \beta_I \frac{I}{N}$ and new infections ;$(1 - n)\lambda S_H$ into L class and $(n)\lambda S_H$ into I. We have $G = \begin{pmatrix} (1-n)\lambda S_H \\ n\lambda S_H \end{pmatrix}$. Substituting λ we have

$$G = (1 - n)(\beta_L \frac{L}{N} + \beta_I \frac{I}{N})S_H n(\beta_L \frac{L}{N} + \beta_I \frac{I}{N})S_H \tag{38}$$

The Jacobian of G becomes $G = \frac{S_H}{N} \begin{pmatrix} (1-n)(\beta_L & (1-n)(\beta_I \\ n(\beta_L & n(\beta_I \end{pmatrix}$. For the non-new infection terms M for L and I, we differentiate with respect L and I

$$M = -(\mu + \delta + \tau + \gamma_1)L - (\mu + \delta + \gamma_2)I \tag{39}$$

Jacobian matrix is deduced as

$$J_{L,I} = \begin{pmatrix} -(\mu+\delta+\tau+\gamma_1) & 0 \\ \tau & -(\mu+\delta+\gamma_2) \end{pmatrix}. \tag{40}$$

Hence,

$$M = \begin{pmatrix} (\mu+\delta+\tau+\gamma_1) & 0 \\ -\tau & (\mu+\delta+\gamma_2) \end{pmatrix} \tag{41}$$

To calculate the inverse of M, we get the determinant
$detM = [(\mu+\delta+\tau+\gamma_1)][(\mu+\delta+\gamma_2)] = (\mu+\delta+\tau+\gamma_1)(\mu+\delta+\gamma_2)$

$$M^{-1} = \begin{pmatrix} -\frac{1}{(\mu+\delta+\tau+\gamma_1)} & 0 \\ \frac{\tau}{(\mu+\delta+\tau+\gamma_1)(\mu+\delta+\gamma_2)} & -\frac{1}{(\mu+\delta+\gamma_2)} \end{pmatrix} \tag{42}$$

$$GM^{-1} = \frac{S_H}{N}\begin{pmatrix} \frac{(1-n)\beta_L}{(\mu+\delta+\tau+\gamma_1)} + (1-n)(\beta_I\frac{\tau}{(\mu+\delta+\tau+\gamma_1)(\mu+\delta+\gamma_2)} & \frac{(1-n)\beta_I}{(\mu+\delta+\gamma_2)} \\ \frac{n\beta_L}{(\mu+\delta+\tau+\gamma_1)} + n\beta_I\frac{\tau}{(\mu+\delta+\tau+\gamma_1)(\mu+\delta+\gamma_2)} & \frac{n\beta_I}{(\mu+\delta+\gamma_2)} \end{pmatrix} \tag{43}$$

The two eigenvalues represent asymptomatic class L and symptomatic infected class I hence we have two R_0, L and R_0, I. We add the two and get the total basic reproduction number $R_0 = R_0, L + R_0, I$

$-\frac{S_H}{N}[\frac{(1-n)\beta_L}{(\mu+\delta+\tau+\gamma_1)} + (1-n)\beta_I\frac{\tau}{(\mu+\delta+\tau+\gamma_1)(\mu+\delta+\gamma_2)} - \eta$ and $-\frac{S_H}{N}\frac{n\beta_I}{(\mu+\delta+\gamma_2)}] - \eta$ hence $R_0, L = \frac{S_H}{N}[\frac{(1-n)\beta_L}{(\mu+\delta+\tau+\gamma_1)} + (1-n)\beta_I\frac{\tau}{(\mu+\delta+\tau+\gamma_1)(\mu+\delta+\gamma_2)}]$, which gives $R_0, I = \frac{S_H}{N}[\frac{n\beta_I}{(\mu+\delta+\gamma_2)}]$ adding the two, $R_0 = \frac{S_H}{N}[\frac{(1-n)\beta_L}{(\mu+\delta+\tau+\gamma_1)} + (1-n)\beta_I\frac{\tau}{(\mu+\delta+\tau+\gamma_1)(\mu+\delta+\gamma_2)} + \frac{n\beta_I}{(\mu+\delta+\gamma_2)}]$ rearranging R_0 $R_0 = \frac{S_H}{N}[\frac{(1-n)\beta_L}{(\mu+\delta+\tau+\gamma_1)} + \frac{\beta_I}{(\mu+\delta+\gamma_2)}(\frac{(1-n)\tau}{(\mu+\delta+\tau+\gamma_1)} + n)]$.
At DFE, E^0 where $S_H^0 = \frac{\rho_H}{(\theta+\mu)}$ and $N = \frac{\rho_H}{\mu}$. Substituting S_H^0 and N into R_0 we have $R_0 = \frac{\mu}{(\theta+\mu)}[\frac{(1-n)\beta_L}{(\mu+\delta+\tau+\gamma_1)} + \frac{\beta_I}{(\mu+\delta+\gamma_2)}(n + \frac{(1-n)\tau}{(\mu+\delta+\tau+\gamma_1)})]$. From R_0 we conclude that:
(i). $\frac{(1-n)\beta_L}{(\mu+\delta+\tau+\gamma_1)}$ infections caused by only those who are in L class.
(ii). $\frac{\beta_I}{(\mu+\delta+\gamma_2)}(n + \frac{(1-n)\tau}{(\mu+\delta+\tau+\gamma_1)})$ infections caused by those who went straight to I class $n\frac{\beta_I}{(\mu+\delta+\gamma_2)}$ and those who enter through L showed signs and symptoms then moved to I $\frac{\beta_I}{(\mu+\delta+\gamma_2)}(\frac{(1-n)\tau}{(\mu+\delta+\tau+\gamma_1)})$.
(iii). When $\tau = 0$ then we have $R_0 = \frac{\mu}{(\theta+\mu)}[\frac{(1-n)\beta_L}{(\mu+\delta+\tau+\gamma_1)} + \frac{\beta_I}{(\mu+\delta+\gamma_2)}n]$.

4.5 The Local Stability of DFE

Theorem 4.2 *The DFE of the model is locally asymptotically stable provided that $R_0 < 1$ and unstable when $R_0 > 1$.*

Proof. By evaluating Jacobian matrix of the model, the proof of local stability of DFE is obtained as follows. Let $A = (\lambda_1 + \lambda_2 + \theta + \mu)$, $B = (\mu+\delta+\tau+\gamma_1)$, $A = (\mu+\delta+\gamma_2)$ and $D = (\mu+\psi_2+\psi_1+\kappa)$ then we have

$$J_E^0 = \begin{pmatrix} -A & 0 & 0 & 0 & 0 \\ 0 & -B & 0 & 0 & \psi_1 \\ \theta & 0 & -\mu & 0 & \kappa \\ 0 & \tau & 0 & -C & \psi_2 \\ 0 & \gamma_1 & 0 & \gamma_2 & -D \end{pmatrix} \tag{44}$$

The characteristic equation is $[-(\lambda_1 + \lambda_2 + \theta + \mu) - \lambda][-(\mu + \delta + \tau + \gamma_1) - \lambda][-\mu - \lambda][-(\mu + \delta + \gamma_2) - \lambda][-(\mu + \psi_2 + \psi_1 + \kappa) - \lambda] = 0$

hence $\lambda = -(\mu + \delta + \gamma_2), -(\mu + \delta + \tau + \gamma_1) and - \mu$ making R_0 negative.

This will reduce matrix J_E^0 to

$$J_E^0 = \begin{pmatrix} -(\lambda_1 + \lambda_2 + \theta + \mu) & \\ 0 & -(\mu + \psi_2 + \psi_1 + \kappa) \end{pmatrix} \tag{45}$$

The trace of the Matrix 45 is $-(2\mu + \lambda_1 + \lambda_2 + \kappa + \theta + \psi_2 + \psi_1)$. Clearly, the DFE is locally asymptotically stable.

4.6 The Global Stability of DFE

The DFE of the model is GAS if $R_0 \leq 1$. Using technique by Castillo Chavez with stability conditions [27].

Theorem 4.3 *The DFE E^0 of the dynamical system is GS if $R_0 < 1$ and unstable whenever $R_0 > 1$, provided that the conditions H1 and H2 are satisfied.*

Proof. From the dynamical system, $X = (S, V)$ and $Z = (L, I, R)$, we get

$$H(X, 0) = \begin{pmatrix} \rho_H - (\lambda_1 + \lambda_2 + \theta + \mu)S_H \\ \theta S_H - \mu V \end{pmatrix} \tag{46}$$

This equation has a unique EP at $X = \frac{\rho_H}{(\lambda_1+\lambda_2+\theta+\mu)}, \frac{\theta\rho_H}{\mu(\lambda_1+\lambda_2+\theta+\mu)}$ which is GAS. Therefore we get

$$P = \begin{pmatrix} P & 0 & \psi_1 \\ \tau & Q & \psi_2 \\ \gamma_1 & \gamma_2 & -FR \end{pmatrix} \tag{47}$$

$$PZ = \begin{pmatrix} PL & 0 & \psi_1 R \\ 0 & QI & \psi_2 R \\ 0 & 0 & FR \end{pmatrix} \tag{48}$$

$$GZ = \begin{pmatrix} \beta_L S_H + \psi_1 R - (\mu + \delta + \tau + \gamma_1)L \\ \beta_I S_H + \psi_2 R + \tau L - (\mu + \delta + \gamma_2)I \\ \gamma_2 I + \gamma_1 L - (\mu + \psi_2 + \psi_1 + \kappa)R \end{pmatrix} \tag{49}$$

$$\widehat{G}Z = PZ - GZ = \begin{pmatrix} 0 \\ 0 \\ 0 \end{pmatrix} \tag{50}$$

So, $\widehat{G}Z = [0,0,0]^T$. Therefore, $\widehat{G}Z = 0$ hence the proof is complete. The conditions have been met and therefore E^0 is GAS.

4.7 The Endemic Equilibrium (EE), $E^*(S^*, V^*, L^*, I^*, R^*)$

We can use R_0 of the model to prove the existence of endemic equilibrium (EE) by testing the positivity of I^*, S^*, V^*, L^* and R^*.

Theorem 4.4 *The EE $E^*(S^*, V^*, L^*, I^*, R^*)$ exists provided that $R_0 > 1$.*

Proof. Using the dynamical system and equating each equation to zero.

$$\begin{aligned}
0 &= \rho_H - (\lambda_1 + \lambda_2 + \theta + \mu)S_H^* && (51)\\
0 &= \lambda_1 S_H^* + \psi_1 R^* - (\mu + \delta + \tau + \gamma_1)L^* && (52)\\
0 &= \kappa R^* + \theta S_H^* - \mu V^* && (53)\\
0 &= \lambda_2 S_H^* + \psi_2 R^* + \tau L^* - (\mu + \delta + \gamma_2)I^* && (54)\\
0 &= \gamma_2 I^* + \gamma_1 L^* - (\mu + \psi_2 + \psi_1 + \kappa)R^* && (55)
\end{aligned}$$

From the first equation of system, we solve

$$S_H^*(\lambda_1 + \lambda_2) = \rho_H - (\theta + \mu). \tag{56}$$

But $\lambda = \lambda_1 + \lambda_2 = \beta_L L + \beta_I I$, therefore $S_H^*(\lambda_1 + \lambda_2) = S_H^*\lambda$. Let $a = (\mu + \delta + \tau + \gamma_1)$, $a_1 = (\mu + \delta + \gamma_2)$, $a_2 = (\mu + \psi_2 + \psi_1 + \kappa)$. Solving for R^* we obtain

$$R^* = \frac{\gamma_2 I^* + \gamma_1 L^*}{a_2}. \tag{57}$$

Rearranging we have

$$\lambda_1 S_H^* = aL - \psi_1 R^*, \tag{58}$$

which is

$$\lambda_2 S_H^* = a_1 I - \tau L^* - \psi_2 R^*. \tag{59}$$

Substituting equation 57 into equation 58 and equation 59 to eliminate R^*.

$$\lambda_1 S_H^* = (a - \frac{\psi_1\gamma_1}{a_2})L - (\frac{\psi_1\gamma_2}{a_2})I \tag{60}$$

$$\lambda_2 S_H^* = (a_1 - \frac{\psi_2\gamma_2}{a_2})I - (\frac{\psi_2\gamma_1}{a_2} - \tau)L \tag{61}$$

Let $H = (a - \frac{\psi_1\gamma_1}{a_2})$, $M = (\frac{\psi_1\gamma_2}{a_2})$, $N = (a_1 - \frac{\psi_2\gamma_2}{a_2})$, $P = (\frac{\psi_2\gamma_1}{a_2} - \tau)$ then

$$\lambda_1 S_H^* = HL - MI \tag{62}$$

and

$$\lambda_2 S_H^* = NI - PL \tag{63}$$

Writing $A, [L, I]^T = S_H\lambda[\lambda_1, \lambda_2]^T$. With A matrix differentiated with respect to L and I.

$$A = \begin{pmatrix} H & -M \\ -P & N \end{pmatrix} \tag{64}$$

Let $W = detA = HN - PM$
$A^{-1} = \frac{1}{W}\begin{pmatrix} N & M \\ P & H \end{pmatrix}$
Using definition of λ
$\lambda = B_L L + B_I I = \frac{S_H\lambda}{W}[B_L(\lambda_1 N + \lambda_2 M) + B_I(\lambda_1 P + \lambda_2 H)]$.
Let $Z = [B_L(\lambda_1 N + \lambda_2 M) + B_I(\lambda_1 P + \lambda_2 H)]$
then

$$\lambda = \frac{S_H\lambda Z}{W} \tag{65}$$

At DFE $\lambda < 0$ and $R_0 < 1$ or at the endemic equilibrium EE then $\lambda > 0$ and $R_0 > 1$ dividing both sides of equation 65

$$1 = \frac{S_H Z}{W}, \tag{66}$$

this implies $S_H^* = \frac{w}{Z}$ Once there is infection, then Z is define and therefore $S_H^* = \frac{w}{Z}$,
$\lambda^* = \frac{Z\rho_H}{W} - (\theta + \mu)$,
$L^* = \frac{\lambda^*}{Z}(\lambda_1 N + \lambda_2 M)$
$I^* = \frac{\lambda^*}{Z}(\lambda_1 P + \lambda_2 H)$.
Since $\lambda > 0$, it follows that $L > 0$, $1 > 0$.

4.8 The Local Stability of EE

Consider the system given as:

$$\frac{dS_H}{dt} = \rho_H - (\lambda_1 + \lambda_2 + \theta + \mu)S_H \tag{67}$$

$$\frac{dL}{dt} = \lambda_1 S_H + \psi_1 R - (\mu + \delta + \tau + \gamma_1)L \tag{68}$$

$$\frac{dV}{dt} = \kappa R + \theta S_H - \mu V \tag{69}$$

$$\frac{dI}{dt} = \lambda_2 S_H + \psi_2 R + \tau L - (\mu + \delta + \gamma_2)I \tag{70}$$

$$\frac{dR}{dt} = \gamma_2 I + \gamma_1 L - (\mu + \psi_2 + \psi_1 + \kappa)R \tag{71}$$

$a_4 = (\lambda_1 + \lambda_2 + \theta + \mu)$ others remained the same as they have been used.

$$J(E^*) = \begin{pmatrix} -a_4 & 0 & 0 & 0 & 0 \\ \lambda_1 & -a_1 & 0 & 0 & \psi_1 \\ \theta & 0 & -\mu & 0 & \kappa \\ \lambda_2 & \tau & 0 & -a_2 & \psi_2 \\ 0 & \gamma_1 & 0 & \gamma_2 & -a_3 \end{pmatrix} \tag{72}$$

From the matrix, there is one eigenvalue $-\mu$ which gives

$$J(E^*) = \begin{pmatrix} -a_4 & 0 & 0 & 0 \\ \lambda_1 & -a_1 & 0 & \psi_1 \\ \lambda_2 & \tau & -a_2 & \psi_2 \\ 0 & \gamma_1 & \gamma_2 & -a_3 \end{pmatrix} \tag{73}$$

Let's determine the trace and determinant of the above matrix.

$$J(E^*) = \begin{pmatrix} -a_4 & 0 & 0 & 0 \\ \lambda_1 & -a_1 & 0 & \psi_1 \\ \lambda_2 & \tau & -a_2 & \psi_2 \\ 0 & \gamma_1 & \gamma_2 & -a_3 \end{pmatrix} \tag{74}$$

we sum up the diagonals entries to get trace.
The trace $= -(a_4 + a_1 + a_2 + b_3)$. The trace is negative.
We use cofactor expansion along first row to calculate determinant

$$det = -a_4 det \begin{pmatrix} -a_1 & 0 & \psi_1 \\ \tau & -a_2 & \psi_2 \\ \gamma_1 & \gamma_2 & -a_3 \end{pmatrix}$$

det of $3x3$
$-a_1(a_2a_3 - \gamma_2\psi_2) + \psi_1(\tau\gamma_2 + a_2\gamma_1)$
$detJ(E^*) = a_4[a_1(a_2a_3 - \gamma_2\psi_2) - \psi_1(\tau\gamma_2 + a_2\gamma_1)]$

4.9 The Global Stability of EE

Theorem 4.5 *If $R_0 > 1$ then the EE $E^*(S_H^*, V^*, L^*, I^*, R^*)$ is GAS in the interior of Ω.*

Proof. Let the endemic equilibrium be $(S_H^*, V^*, L^*, I^*, R^*)$ where $S_H^*, V^*, L^*, I^*, R^*$ are the steady state values satisfying the system. Consider the non-linear Lyapunov function $P : (S_H, V, L, I, R) :\in \Omega \subset \Re_+^5 : S_H, V, L, I, R > 0$.

$$P : (S_H, V, L, I, R) = \frac{1}{2}(\frac{S_H}{S_H^*} - 1)^2 + \frac{1}{2}(\frac{V}{V^*} - 1)^2 + \frac{1}{2}(\frac{L}{L^*} - 1)^2 + \frac{1}{2}(\frac{I}{I^*} - 1)^2 + \frac{1}{2}(\frac{R}{R^*} - 1)^2 \tag{75}$$

Then P is in the interior of Ω. E^* is the global minimum of P on Ω and $P : (S_H, V, L, I, R) = 0$. the time derivative is given

$$\begin{aligned}\frac{dP}{dt} &= \frac{1}{S_H^*}(\frac{S_H}{S_H^*} - 1)\frac{dS_H}{dt} + \frac{1}{L^*}(\frac{L}{L^*} - 1)\frac{dL}{dt} \\ &+ \frac{1}{V^*}(\frac{V}{V^*} - 1)\frac{dV}{dt} + \frac{1}{I^*}(\frac{I}{I^*} - 1)\frac{dI}{dt} \\ &+ \frac{1}{R^*}(\frac{R}{R^*} - 1)\frac{dR}{dt}.\end{aligned}$$

With the derivatives of (S_H, V, L, I, R) we have

$$\begin{aligned}\frac{dP}{dt} &= \frac{1}{S_H^*}(\frac{S_H}{S_H^*} - 1)\rho_H - (\lambda_1 + \lambda_2 + \theta + \mu)S_H \\ &+ \frac{1}{L^*}(\frac{L}{L^*} - 1)\lambda_1 S_H + \psi_1 R - (\mu + \delta + \tau + \gamma_1)L \\ &+ \frac{1}{V^*}(\frac{V}{V^*} - 1)\kappa R + \theta S_H - \mu V \\ &+ \frac{1}{I^*}(\frac{I}{I^*} - 1)\lambda_2 S_H + \psi_2 R + \tau L - (\mu + \delta + \gamma_2)I \\ &+ \frac{1}{R^*}(\frac{R}{R^*} - 1)\gamma_2 I + \gamma_1 L - (\mu + \psi_2 + \psi_1 + \kappa)R.\end{aligned}$$

This results into

$$\begin{aligned}\frac{dP}{dt} &= (\frac{S_H}{S_H^*} - 1)\frac{\rho_H}{S_H^*} - (\lambda_1 + \lambda_2 + \theta + \mu)\frac{S_H}{S_H^*} \\ &+ (\frac{L}{L^*} - 1)\frac{\lambda_1 S_H}{L^*} + \frac{\psi_1 R}{L^*} - (\mu + \delta + \tau + \gamma_1)\frac{L}{L^*} \\ &+ (\frac{V}{V^*} - 1)\frac{\kappa R}{V^*} + \frac{\theta S_H}{V^*} - \mu\frac{V}{V^*} \\ &+ (\frac{I}{I^*} - 1)\frac{\lambda_2 S_H}{I^*} + \frac{\psi_2 R}{I^*} + \frac{\tau L}{I^*} - (\mu + \delta + \gamma_2)\frac{I}{I^*} \\ &+ (\frac{R}{R^*} - 1)\frac{\gamma_2 I}{R^*} + \frac{\gamma_1 L}{R^*} - (\mu + \psi_2 + \psi_1 + \kappa)\frac{R}{R^*}\end{aligned}$$

At the equilibrium point we have

$$\begin{aligned}\frac{\rho_H}{S_H^*} &= (\lambda_1 + \lambda_2 + \theta + \mu) \\ \frac{\lambda_1 S_H}{L^*} + \frac{\psi_1 R}{L^*} &= (\mu + \delta + \tau + \gamma_1) \\ \frac{\kappa R}{V^*} + \frac{\theta S_H}{V^*} &= \mu \\ \frac{\lambda_2 S_H}{I^*} + \frac{\psi_2 R}{I^*} + \frac{\tau L}{I^*} &= (\mu + \delta + \gamma_2) \\ \frac{\gamma_2 I}{R^*} + \frac{\gamma_1 L}{R^*} &= (\mu + \psi_2 + \psi_1 + \kappa)\end{aligned}$$

Doing a substitution gives

$$\begin{aligned}\frac{dP}{dt} &= (\frac{S_H}{S_H^*}-1)((\lambda_1+\lambda_2+\theta+\mu)-(\lambda_1+\lambda_2+\theta+\mu)\frac{S_H}{S_H^*})\\ &+ (\frac{L}{L^*}-1)((\mu+\delta+\tau+\gamma_1)-(\mu+\delta+\tau+\gamma_1)\frac{L}{L^*})\\ &+ (\frac{V}{V^*}-1)(\mu-\mu\frac{V}{V^*})\\ &+ (\frac{I}{I^*}-1)((\mu+\delta+\gamma_2)-(\mu+\delta+\gamma_2)\frac{I}{I^*})\\ &+ (\frac{R}{R^*}-1)((\mu+\psi_2+\psi_1+\kappa)-(\mu+\psi_2+\psi_1+\kappa)\frac{R}{R^*}).\end{aligned}$$

Re-arranging gives

$$\begin{aligned}\frac{dP}{dt} &= (\frac{S_H}{S_H^*}-1)(\frac{S_H}{S_H^*}-1)(-(\lambda_1+\lambda_2+\theta+\mu))\\ &+ (\frac{L}{L^*}-1)(\frac{L}{L^*}-1)(-(\mu+\delta+\tau+\gamma_1))\\ &+ (\frac{V}{V^*}-1)(\frac{V}{V^*}-1)(-\mu)\\ &+ (\frac{I}{I^*}-1)(\frac{I}{I^*}-1)(-(\mu+\delta+\gamma_2))\\ &+ (\frac{R}{R^*}-1)(\frac{R}{R^*}-1)(-(\mu+\psi_2+\psi_1+\kappa))\end{aligned}$$

Hence, we have

$$\begin{aligned}\frac{dP}{dt} &= -(\lambda_1+\lambda_2+\theta+\mu)(\frac{S_H}{S_H^*}-1)^2\\ &- (\mu+\delta+\tau+\gamma_1)(\frac{L}{L^*}-1)^2\\ &- (\mu)(\frac{V}{V^*}-1)_2-(\mu+\delta+\gamma_2)(\frac{I}{I^*}-1)^2\\ &- (\mu+\psi_2+\psi_1+\kappa)(\frac{R}{R^*}-1)^2.\end{aligned}$$

Therefore, the Lyapunov function P is positive definite and its derivative P' is negative definite. Therefore the EE of the system is GAS.

4.10 Sensitivity Analysis

Parameter sensitivity shows how some parameters within the system, have higher or lower degree of influencing the model's stability with

$$R_0 = \frac{\mu}{(\theta+\mu)}[\frac{(1-n)\beta_L}{(\mu+\delta+\tau+\gamma_1)}+\frac{\beta_I}{(\mu+\delta+\gamma_2)}(n+\frac{(1-n)\tau}{(\mu+\delta+\tau+\gamma_1)})]$$

and using the formula as $S_P^{R_0=\frac{dR_0}{dP}} \times \frac{P}{R_0}$ some of the parameters that affect R_0 both positively and negatively are; $\mu, \theta, \delta, \gamma_1, \gamma_2, \tau, \beta_L, \beta_I, n$

For transmission rates of the disease on R_0,

$S_{\beta_L}^{R_0=\frac{\mu(1-n)}{(\theta+\mu)(\mu+\delta+\tau+\gamma_1)}} \times \frac{\beta_L}{R_0}$

$S_{\beta_I}^{R_0=\frac{\mu[n(\mu+\delta+\tau+\gamma_1)+(1-n)\tau]}{(\theta+\mu)(\mu+\delta+\tau+\gamma_1)(\mu+\delta+\gamma_2)}} \times \frac{\beta_I}{R_0}$

$S_{n}^{R_0=\frac{\mu[(\mu+\delta+\gamma_2)(\beta_I-\beta_L)-\tau]}{(\theta+\mu)(\mu+\delta+\tau+\gamma_1)(\mu+\delta+\gamma_2)}} \times \frac{n}{R_0}$

$S_{\tau}^{R_0=\frac{\mu[(1-n)\beta_I(\mu+\delta+\gamma_1)+(1-n)\beta_L(\mu+\delta+\gamma_2)]}{(\theta+\mu)(\mu+\delta+\tau+\gamma_1)^2(\mu+\delta+\gamma_2)}} \times \frac{\tau}{R_0}.$

Effect of recovery rates on R_0 :

$S_{\gamma_1}^{R_0=-\frac{\mu(1-n)(\tau\beta_I+(\mu+\delta+\gamma_2)\beta_L)}{(\theta+\mu)(\mu+\delta+\tau+\gamma_1)^2(\mu+\delta+\gamma_2)}} \times \frac{\gamma_1}{R_0}$

$S_{\gamma_2}^{R_0=-\frac{\mu\beta_I(n(\mu+\delta+\tau+\gamma_1)+(1-n)\tau)}{(\theta+\mu)(\mu+\delta+\tau+\gamma_1)(\mu+\delta+\gamma_2)^2}} \times \frac{\gamma_2}{R_0}$

Effect of vaccination rate on R_0 :

$S_{\theta}^{R_0=-\frac{\mu}{(\theta+\mu)^2}} \times \frac{\theta}{R_0}$

Effect of rate of natural death and death caused by COVID-19:

$S_{\mu}^{R_0=-\frac{\theta[(1-n)\beta_L(\mu+\delta+\gamma_2)^2+n\beta_I(\mu+\delta+\tau+\gamma_1)^2+(n\beta_I+\beta_I(1-n)\tau)(2\mu+2\delta+\gamma_1+\gamma_2+\tau)]}{(\theta+\mu)^2(\mu+\delta+\gamma_2)_2(\mu+\delta+\tau+\gamma_1)^2}} \times \frac{\mu}{R_0}$

$S_{\delta}^{R_0=-\frac{\mu[(1-n)\beta_L(\mu+\delta+\gamma_2)^2+n\beta_I(\mu+\delta+\tau+\gamma_1)^2+(n\beta_I+\beta_I(1-n)\tau)(2\mu+2\delta+\gamma_1+\gamma_2+\tau)]}{(\theta+\mu)^2(\mu+\delta+\gamma_2)_2(\mu+\delta+\tau+\gamma_1)^2}} \times \frac{\delta}{R_0}.$

Parameters such as β_L,β_I, n and τ which have positive index increase the R_0. These parameters should be controlled if not managed by reducing their effect in order to reduce R_0. Those parameters with negative index such as γ_2, γ_1, θ, δ and μ reduce R_0. An increase in these parameter will reduce R_0 and COVID-19 will be managed. By reduction of R_0, COVID-19 will be controlled and managed hence less people will have severe illness, less deaths and less admission on ICU or hospital. Rate of recovery and rate of vaccination play a vital role when its come to control of COVID-19 hence more people should be vaccinated. This will boost the rate of recovery hence more people will recover faster.

5 Open Problems

Two natural problems emanate from this work. The following problems can be considered for future research.**Problem 1:** Can one carry out a numerical analysis of the model to support the theory? **Problem 2:** There is need for bifurcation analysis of the model.

References

[1] Aleta A., Martin-Corral D., Pastore A. et al, Modelling the impact of testing,contact tracing and household quarantine on second waves of COVID-19, *Natural Human Behaviour* Vol.4,NO.9, (2020), 964-971.

[2] American Diabetes Association.,*H*ow COVID-19 Impacts People With Diabetes, https:www.diabetes.org/Covid-19-covid-19/ 2020.

[3] Anusha S. and Athithan S., *Mathematical Modelling Co-existence of Diabetes and COVID-19: deterministic and Stochastic Approach.*, Department of Mathematics, Faculty of Engineeringand Technology, SRM institute of Science and Technology,Kattankulathur,tamil nadu-603203, India , 2020.

[4] Anwar Z., Ebraheem A., Vedat S. and Gul J., Mathematical Model for Coronavirus disease 2019 (COVID-19) Containing Isolstion class, *Hindawi; BIomed Research international*

[5] Boutayeb A., Twizell E., Achouayb K. and Chetouani A., A mathematical model for the burden of diabetes and its complications, *Biomedical Engineering Online*, 2004.

[6] Chan J., Yuan S., Kok K., Jianping Z. et al Clinical features of patients infected with 2019 novel coronavirus in wuhan, China, *The lancet*,Vol.395, No.10223, 497-506, 2020.

[7] Chaolin H., Yeming W., Xingwang L. and Gul J., Mathematical Model for Coronavirus disease 2019 (COVID-19) Containing Isolstion class, *Hindawi; BIomed Research international* https:doi.org/10.1155/2020/3452402.

[8] Eryarsoy E., Delen D., Davazdahemami B. and Topuz K., A novel diffusion-based model for estimating cases,and fatalital epidemics the case of COVID-19, *journal of business research* Vol.124,NO.9, (2021), 163-178.

[9] Ikram I., Nadia I. and Saloua E., A new model of COVID-19 among diabetes population: a mathematical analysis and optimal control approach for intervention strategies,*Communications in Mathematical biology and Neuroscience*, 123(2023), 89-102.

[10] Jiang S., Xia S., Ying T. and Lu L., A novel coronavirus (2019-nCoV) causing pneumonia-associated respiratory syndrome, *Cellular and Molecular Immunology*, 17(2020), 55-64.

[11] Joan L. and Ira B., Seasonality and period-doubling bifurcations in an epidemic model, *Journal of theoretical biology*, 110(1984), 665-679.

[12] JohnF. and Brownlee M., Statistical studies in immunity: the theory of an epidemic, *Proceedings of the Royal Society of Edinburgh*, 26(1907),484-521.

[13] Jomar F., Insights from mathematical models of 2019-nCoV acute respiratory disease (COVID19) dynamics, *Early models of 2019-nCov ARD dynamics*, 2020.

[14] Julien A. and Stephanie P., A simple model for COVID-19, Infectious Disease Modelling, *scienceDirect* , 5(2020), 309-316.

[15] Kassa S., Njagarah H. and Terefe Y., Analysis of the mitigation strategies for COVID-19 from mathematical modelling perspective, *medRxiv preprint doi:https://doi.org/10.1101/2020.04.15.20066308v1.*

[16] Kermack W. and Mckendrick A., *Contribution to the mathematicaltheorem of epidemics. ii. further studies of the problem of endemicit*, Bulletin of mathematical biology, Vol.53, No.1, (1991),57-87.

[17] Kenneth A. and Dexter J., *Engineering mathematics*, Palgrave, Macmillan, 2013.

[18] www.ne.jp/asahi/kishimoto/clinic/cash/ Kishimoto Clinic, *COVID19.*, 2020.

[19] Kwach B., Ongati O. and Simwa R., Mathematical Model for Detecting Diabetes in the blood, *Applied Mathemticl science*, Vol.5, No 6, (2014), 279-286.

[20] La Salle J., Lefschetz G. and Solomon N., *Stability by Liapunov's Direct Method: With application* , New York: Academic Press, (1996).

[21] Lauren M., Nadia N. and Caroline O., *Modeling challenges in context:Lessons from malaria,HIV and tuberculosis*, Epidemics, Vol.10, (2015), 102-107.

[22] Lawi G., Ongati O. and MUgisha J., Modeling Co-infection of Paediatric Malaria and pneumonia, *International Journal of Math*, 7(2013), 413-424.

[23] Letko M., Marzi A. and Munster V., Functional assessment of cell entry and receptor usage for SARS-CoV-2 and other lineage B betacoronaviruses, *Nature Microbiology*, 5(2020), 562-569.

[24] Martinez D., Gonzalez G. and Villanueva R, Analysis of key factors of a SARS-CoV-2 vaccination program: a mathematical modeling approach, *Epidemiologa*, 2(2021), 140-161.

[25] Norman T. and Bailey J., *The mathematical theory of epidemics*, London, 1957.

[26] Odo D., Heesterbeek J. and Johan A., On the definition and the computation of the basic reproduction ratio Ro in motels for infectious diseases in heterogeneous population , *Journal of mathematical biology*, 43(1990), 45-78.

[27] Onyango L., Ogada E., Thirika A. and Lawi G., *Modeling malaria and ratavirus co-infection*, Neural, parallel and computation, 26(2018) 143-168.

[28] Omame A., Sene N., Analysis of COVID-19 and comorbidity co-infection Model with Optimal Control, *medRxiv preprint doi:https://doi.org/10.1101/2020.08.04.20168013*.

[29] OShea D., *An introduction to Dynamical Systems and Mathematical Modelling*, Research Foundation, 1992.

[30] Patrick C., *A new proof of the routh hurwitz stability criterion using the second method of liapunov*, In mathematical proceedings of the Cambridge Philosophical society,Cambridge university Press, (1962), 694-702.

[31] Rex C. and Robinson B., *An introduction to dynamical systems:continuos and discrete*, American Mathematical Society, 2012.

[32] Ronald T. and Ross K., *The prevention of malaria*, Springer, Dutton, 1910.

[33] Samuel O. and Joseph A., *A Mathematical model Model of Transmission Dynamics of SARS-CoV-2(COVID-19) with an Underlying Condition of diabetes*, International Journal of Mathematics and Mathematical Sciences Article ID 7984818https:doi.org/10.1155/2022/7984818 (2022).

[34] Sandhya and Deepak K., Mathematical Model for Glucose-Insulin Regulatory System of Diabetes Mellitus, *Advances in Applied Mathematical Biosciences*, Vol.2, (2011), 39-46.

[35] Sileshi S., Henok. D and Tadesse A., Mathematical Modeling of COVID-19 Transmission Dynamics with vaccination:A case Study In Ethiopia, Hindawi:Discrete Dynamics in Nature and Society, 2023.

[36] Sirajo A., Niniuolu J., Omotayo B. and Usman Y., Sensitivity analysis of the parameters of mathematical model of hepatatis B virus transmission, *Universal Journal of applied Mathematics*, 4(2013), 230-241.

[37] Verdecchia P., Cavallini C., Spanevello A. and Angeli F., The pivotal link between ACE2 deficiency and SARS-CoV-2 infection, *European Journal of Internal Medicine*, 76(2020), 14-20.

[38] Vitaly V., Malay B. and Sergei P., On a quarantine model of Covid-19 infection and data analysis, arXiv:2003.0944v1[q-bio.PE], Vol.41, 2020.

[39] William O. and Anderson G., Contribution to the mathematicaltheorem of epidemics III. further studies of the problem of endemicity, Proceedings of the Royal Society of London. Series A containing Papers of a mathematical and Physical Characters, 90(1933), 94-122.

[40] Won Y., Mortality rate of patients COVID-19 based on underlying health conditions,*Diaster Medicine and Public Health Preparedness*, 3(2021), 1-16.

[41] WuJJ L., Nowcasting and forecasting the potential domestic and international spread of 2019-ncov outbreak originating in Wuhan china, *Modeling study Lancet*, 97(2020), 395-689.

[42] Xuefei L. and Emanuele B., Is time to intervention the COVID-19 outbreak really important? A global sensitivity analysis approach, arXiv:2005.01833vI [stat.AP] 2020.

[43] Yang C. and Wang J., COVID-19 and underlying health conditions:a modeling investigation, *Mathematical biosciences and Engineering*, 18(4), (2021), 3790-3812.